# The effects of water immersion on recovery in Australian Football

by

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This thesis is submitted in partial fulfilment of the requirements for the award of

#### DOCTOR OF PHILOSOPHY

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### ABSTRACT

Participation in a physically demanding team sport, such as Australian football (AF), can result in post-exercise physical performance decrements as well as increases in psychometric measures, muscle damage and inflammation. The use of both cold water immersion (CWI) and contrast water therapy (CWT) as a means of post-exercise recovery is becoming more prevalent in the team sport environment. Both modalities are utilised in an attempt to enhance physical performance recovery while reducing perceived muscle soreness and fatigue. Although increasing in popularity, conflicting evidence exists regarding their effectiveness. Comparisons within the same team sport population are lacking, while little information on the efficacy of either intervention exists in AF. Therefore, the effects of a single 14 min exposure of both CWI and CWT on restoring repeat 20 m sprint time, jump performance and psychometric measures was investigated following AF training (Study 1) and an AF match (Study 2). The effects of both modalities post-match on moderating the appearance of markers of muscle damage and inflammation was also investigated (Study 3).

Australian football training and match participation reduced repeat sprint, countermovement and squat jump performance and increased perceptions of fatigue and muscle soreness. Match participation also greatly increased inflammatory and muscle damage markers. Both CWI and CWT were more successful than a passive recovery at attenuating decrements in physical performance and reducing exercise induced increases muscle soreness, fatigue and markers of muscle damage, with CWI being the most effective. Neither CWI nor CWT altered the post-exercise inflammatory response and therefore most likely had no effect on blunting the inflammatory pathways involved in muscle repair.

Results demonstrate that AF participation leads to post-exercise declines in physical performance, an acute phase inflammatory response, muscle damage and heightened perceptions of soreness and fatigue. It was established that in well trained AF players, both modalities were more effective at attenuating these changes than a passive recovery with CWI being more effective. These findings add to the body of knowledge regarding the use and efficacy of CWI and CWT in team sport and would be of benefit to team sport athletes/coaches in decreasing any confusion regarding their use and in helping them to determine the best modality for their recovery needs.

# **STUDENT DECLARATION**

"I, George Peter Elias, declare that the PhD thesis entitled "The effects of water immersion on recovery in Australian Football" is no more than 100,000 words in length including quotes and exclusive of tables, figures, appendices, bibliography, references and footnotes. This thesis contains no material that has been submitted previously, in whole or in part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work. I declare that all the tables, figures and statistical analysis presented in this thesis and that all work on assays for muscle damage and inflammatory markers was completed by myself."

Signature

Date

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# ABBREVIATIONS

Abbreviation	Definition
AF	Australian football
ATP	Adenosine triphosphate
Ca <sup>2+</sup>	Calcium
СК	Creatine Kinase
cm	Centimetre
СМЈ	Countermovement jump
CWI	Cold water immersion
CWT	Contrast water therapy
deg.sec	Degrees per second
DOMS	Delayed onset muscle soreness
E-C	Excitation contraction
EIMD	Exercise induced muscle damage
ES	Effect size
F:C	Flight time:contraction time ratio
FT	Flight time
h	Hour
IL-1	Interleukin 1
IL-10	Interleukin 10
IL-1ra	Interleukin 1 receptor agonist
IL-1β	Interleukin 1 beta
IL-6	Interleukin 6
IU.L	International units per litre
K <sup>+</sup>	Potassium
kDa	Kilodalton
Km	Kilometre
lbs	Pounds
m	Metre
m.min <sup>-1</sup>	Metres per minute
Mb	Myoglobin
min	Minute
MEP	Motor evoked potential
Na <sup>+</sup>	Sodium
Na <sup>+</sup> -K <sup>+</sup> -ATPase	Sodium potassium ATPase
ng.ml	Nanograms per millilitre
PAS	Passive
PCr	Phosphocreatine
P <sub>i</sub>	Inorganic phosphate
RPE	Rating of perceived exertion
RSA	Repeat-sprint ability
S	Second
SD	Standard deviation
SJ	Static jump
SR	Sarcoplasmic reticulum
SSC	Stretch shortening cycle
SSG	Small sided games
TNF	Tumor necrosis factor
TNF-α	Tumor necrosis factor alpha
TWI	Thermoneutral water immersion
°C	Degrees celcius

# **PUBLICATIONS**

The following publications and conference abstracts are in support of this thesis:

Peer review publications arising directly from this thesis:

- Elias, G. P., Varley, M. C., Wyckelsma, V. L., McKenna, M. J., Minahan, C. L., & Aughey, R.
   J. Effects of water immersion on post-training recovery in Australian footballers. *International Journal of Sports Physiology and Performance*. 7(4), 357-66 (2012) (Chapter 4)
- Elias, G. P., Wyckelsma, V. L., Varley, M. C., McKenna, M. J. & Aughey, R. J. (In Press).
   Effectiveness of water immersion on post-match recovery in elite professional footballers.
   *International Journal of Sports Physiology and Performance*. (Chapter 5)

Additional peer reviewed publications arising during the candidature process:

 Varley, M.C., Elias, G., & Aughey, RJ. (2012). Current match analysis techniques can underestimate intense periods of high-velocity running. *International Journal of Sports Physiology and Performance*. 7 (2), 183-85

#### Peer reviewed abstracts:

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### CHAPTER 1. INTRODUCTION

Participation in elite team sport training and matches can often result in athletes experiencing muscular damage and inflammation, heightened perceptions of both muscle soreness and fatigue and a reduction in physical capacities in the days following exercise (Ascensao et al., 2008, Aughey, 2010, Fatouros et al., 2010, Ispirlidis et al., 2008, Magalhaes et al., 2010, Montgomery et al., 2008b). As a result, in sports where athletes compete regularly, post-exercise recovery can play an important role in assisting athletes to overcome both training and competition demands (Halson, 2011).

Post-exercise recovery should allow for a period of physical and psychological regeneration and the principal aim of any recovery session should be to promote an athlete's readiness to train or compete again. In particular, recovery should maximise between-competition performance restoration and minimise/alleviate fatigue and soreness, therefore attaining a balance between recovery and training/competition stresses is important.

Two post-exercise recovery modalities commonly utilised in the team sport setting are cold water immersion and contrast water therapy (Ascensao et al., 2011, Dawson et al., 2005, Ingram et al., 2009, King and Duffield, 2009, Kinugasa and Kilding, 2009, Montgomery et al., 2008b, Rowsell et al., 2009). They are both utilised in an attempt to enhance the recovery process by attenuating the decline in physical performance after exercise and reducing psychometric measures such as perceived muscle soreness and fatigue. The use of both modalities in team sport is increasing in popularity and both have been successful in restoring physical and psychometric recovery (Ascensao et al., 2011, Ingram et al., 2009, King and Duffield, 2009, Montgomery et al., 2008b, Rowsell et al., 2009, Rowsell et al., 2011); however conflicting evidence exists regarding the efficacy of either modality in team sport (Higgins et al., 2011, Kinugasa and Kilding, 2009). Methodological differences have led to unclear results which can lead to confusion for athletes and coaches regarding the most appropriate intervention, however it appears that a total immersion time of 10 min or more may be an important factor in enhancing the overall effectiveness of either modality (Ascensao et al., 2011, Ingram et al., 2009, King and Duffield, 2009) while the use of hot showers may limit their effectiveness (Halson, 2011). Comparisons on the

effectiveness of both recovery modalities within the same athlete population using similar immersion times are lacking. Consequently, the efficacy of cold versus contrast therapies after a competitive match is extremely limited while the effectiveness of either modality post-training is unknown.

Australian football is a high-intensity intermittent full-contact team sport that imposes great physical demands on players. It requires a combination of ball skills, speed and athleticism and places high demands on a player's high-intensity and prolonged running capacity (Aughey, 2010, Coutts et al., 2010). Over the length of a long pre-season and competition season, post-exercise recovery may become important in assisting players to better prepare for their next exercise bout. In Australian football, both cold water immersion and contrast water therapy are widely utilised and during the weekly cycle, effective use of either/both interventions may help players to recover from the increasing physical and running demands of match play. One investigation has examined the post-match effects of contrast water therapy combined with a pool recovery session (Dawson et al., 2005), however the effectiveness of a single acute application of cold water immersion or contrast water therapy after a game or training remains unknown.

This thesis will therefore investigate the impact of both cold water immersion and contrast water therapy in well trained Australian footballers after both training and a match. Specifically, the effects of both cold water immersion and contrast water therapy in restoring perceived muscle soreness and fatigue and physical performance will be investigated after Australian football training and match participation. Additionally, the effectiveness of both modalities in reducing the appearance of markers of post-match muscle damage and inflammation will also be investigated.

### **CHAPTER 2. REVIEW OF LITERATURE**

#### 2.1 Australian Football

Australian football (AF) is a contact sport played over four 20 minute quarters by two teams of 18 players and 4 substitutes (Gray and Jenkins, 2010). The game allows for unlimited substitutions for three of the players while the fourth player is allowed onto the field only after one of the outfield players is taken off/removed from the field permanently. The sport requires a combination of ball skills, speed and athleticism and places high demands on a player's prolonged running capacity (Coutts et al., 2010, Dawson et al., 2004b), particularly high-intensity running (Aughey, 2010). The game also includes a substantial amount of physical contact as players regularly collide with each other when tackling, shepherding, contesting ground balls, bumping each other and during marking and ruck contests. The following sections will review the activity profile of both AF matches and training and compare these to other team sports.

#### 2.1.1 The activity profile of an AF game

The demands of AF at the elite level have changed considerably in recent years, with evidence that the game is getting faster and more arduous (Gray and Jenkins, 2010). Australian football places great demands on an athlete's running ability, with players requiring well developed endurance, speed-endurance, repeat-sprint ability, maximal running velocity and acceleration capacities (Wisbey et al., 2010). With demands on running increasing through the period from 2005-2009 (Aughey, 2010, Aughey, 2011a, Aughey, 2011b, Coutts et al., 2010, Gray and Jenkins, 2010), players are generally more fatigued during the second half of games as demonstrated by a reduction in high intensity running (Aughey, 2010).

#### 2.1.1.1 Total distance covered during AF matches

Elite AF players can cover between 12,700-13,000 m during a game and up to 18,000 m during finals (Aughey, 2010, Coutts et al., 2010). Although average distances covered in games did not increase

during the 2005-2009 period, total game time decreased by 9% (Wisbey et al., 2010). When expressed as metres covered per minute of game play and when factoring in the drop in game time, distance covered during games has indeed increased. From 2005-2007, players on average covered 113 m.min<sup>-1</sup> (Coutts et al., 2010), with this figure reaching 127 m.min<sup>-1</sup> during 2008-2009 (Aughey, 2010). During the finals, match intensity increases further as players can cover up to 150 m.min<sup>-1</sup> (Aughey, 2011b).

#### 2.1.1.2 High and mean velocity running during AF matches

High velocity running (4–10 m.sec<sup>-1</sup>) is an important component of AF football as it allows players to get into attacking/defending positions, get to the next contest ahead of their opponent or simply break away from them. Throughout an AF match, players cover approximately 30% of game distance as high velocity running (Coutts et al., 2010) and during finals matches, this figure increases by approximately 9% (Aughey, 2011b).

#### 2.1.1.3 Accelerations during AF matches

The highly intermittent nature of AF is evident by the number of moderate  $(1.10-2.78 \text{ m.s}^{-2})$  and maximal accelerations (2.78-10 m.s<sup>-2)</sup> that occur during the game (Aughey, 2010). Moderate accelerations increased by an average of 3.6% in the 3 years after 2005 indicating that the demands of an AF match are changing. During a regular season match, players are involved in 96 maximal accelerations (Aughey, 2010) and during finals match, the number of maximal accelerations almost doubles (Figure 2.1) (Aughey, 2011b). This equates to an extra distance covered in maximal accelerations of approximately 450 m.

When combined with the drop in average game time over the last few years, as well as the increase in metres covered per minute, mean velocity and high intensity running, AF players are working harder than previous seasons during games (Wisbey et al., 2010).

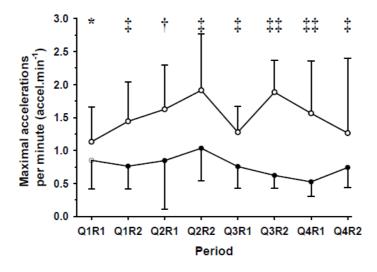


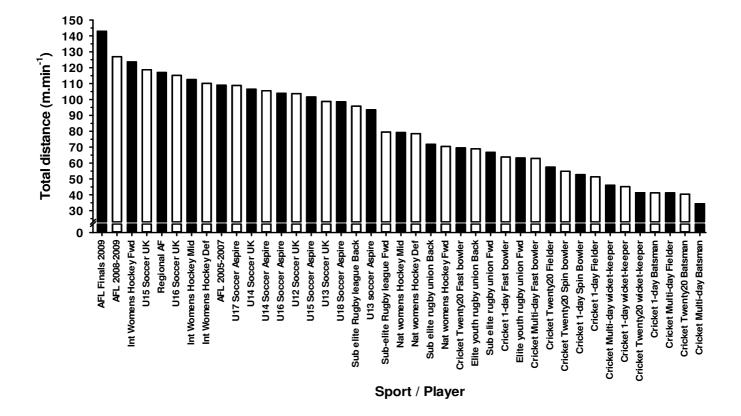
Figure 2.1: The number of maximal accelerations  $(2.78-10.00 \text{ m.s}^{-2})$  per minute, expressed per period of time (rotation) on the field during AF games. Periods were named for the quarter of play they occurred in, and the sequential number of the rotation in that quarter (Q1R1; Q1R2; Q2R1; 479 Q2R2; Q3R1; Q3R2; Q4R1; AND Q4R2 respectively). Closed circles are for regular season games, open circles are for finals games. Data is 480 Mean ± SD, \* denotes a small magnitude increase from regular season games; † denotes a moderate increase from regular season games; ‡ 481 denotes a large increase from regular season games and ‡‡ a very large increase, n=24 (Aughey, 2011b).

#### 2.1.2 The activity profile of other team sport matches compared to AF game.

In comparison to other team sport athletes, the activity profile of AF players is higher (Figure 2.2). Below is a review of the activity profile of players from other team sports compared to AF.

#### 2.1.2.1 Total distance covered during matches

During the 2005-2006 English FA premier league season, players (excluding goalkeepers) covered an average of 10,714 m (Bradley et al., 2009). When expressed as metres covered per minute over an entire 90 min match, this equates to players covering 119.0 m.min<sup>-1</sup>, or ~7-26% lower than AF. Results in field hockey also demonstrate lower distances when compared to AF games. Over the course of 13 international matches, elite field hockey players covered an average of 5,541 m or 115.4 m.min<sup>-1</sup> during the 48 min of game time they participated in (Macutkiewicz and Sunderland, 2011). In 32 games of a domestic competition, elite field hockey players participating in the Australian hockey league covered an average of 6,576 m or 93.9 m.min<sup>-1</sup> during a 70 min match (Gabbett, 2010). The activity profile in both rugby codes is also less than that reported in AF. During 16 national rugby league matches, players covered between 93 and 101 m.min<sup>-1</sup> depending on playing position (Gabbett et al., 2012)



while junior rugby union players covered an average of 4000 m per hour of game play or 66.6 m.min<sup>-1</sup>

(Hartwig et al., 2011).

Figure 2.2: Total distance per minute of matches (m.min-1) for various team sport athletes (Aughey, 2011a).

#### 2.1.2.2 High velocity running during matches

During AF matches, players cover approximately 3885 m at a high velocity (Coutts et al., 2010). When compared to distances in other sports, and allowing for variations in the classification of velocity bands, AF players covered greater distances than those recorded in English premier league matches (2549m) (Bradley et al., 2009) during Italian and Danish first division soccer games from the early 2000's (3080 and 2310 m respectively) (Mohr et al., 2003).

#### 2.1.3 Activity profile of AF training

The activity profile of AF training differs greatly from that of matches. Typical AF training sessions are much shorter in duration than matches (Dawson et al., 2004a). Players typically have more kicks and handballs during training, however training typically has less tackling, shepherding, ground balls and contested marks (Dawson et al., 2004a). The running requirements of training will be addressed below.

#### 2.1.3.1 Running requirements of AF training

Some of the running requirements during training are similar to a match, however, overall distance and intensity of running (metres covered per minute) fail to match games (Dawson et al., 2004a, Farrow, 2008). Australian footballers can cover between 1815-1930 m or 92-102 m.min<sup>-1</sup> during a typical AF training session (Farrow, 2008). These values reflect ~72-80% of regular season matches and only ~61-68% of finals and is indicative of shorter training durations compared to matches (Dawson et al., 2004a). The duration of fast running (purposeful running but not near maximal speed) and sprinting efforts (running at top speed or very close to it) during training were almost all <6 sec which closely matched games as did changes of direction when sprinting (Dawson et al., 2004a). Fast running and sprint bouts however were not performed as frequently during training (every 76 sec) compared to during training during training (Dawson et al., 2004a).

#### 2.1.3.2 Accelerations during AF training

The number of accelerations performed at training is well below that seen during matches. During regular season matches, players perform up to 96 accelerations (> $2.78m.sec^{-2}$ ) per match (Aughey, 2010), however during training, this value is 5 (> $4.0m.sec^{-2}$ ) (Farrow, 2008). The difference in the threshold at which these values was calculated will no doubt have contributed to the large discrepancy in values, however, as both the duration and distances covered during training are well below matches (Dawson et al., 2004a, Farrow, 2008), it is still reasonable to assume that the number of accelerations would be less than during a match.

#### 2.1.4 Activity profile of team sport training

The activity profiles of training often fail to meet those of matches. Over the course of rugby league season, the intensity and distance covered was lower when comparing skills sessions to games (Gabbett et al., 2012). Compared to training, players covered a greater distance per minute during games (166%), covered more high speed distance (200%) and performed more repeated high-intensity effort bouts (167%). In rugby union, junior players were followed between 2003-2008 with data also indicating that games were far more intense than training (Hartwig et al., 2011). For every hour of game play compared to training, players performed more sprints (21.8 vs. 1.0), spent more time sprinting (45 vs. 2 sec) and covered more distance while sprinting (324 vs. 11 m). In field hockey, during the course of 19 training sessions and 32 elite level matches, players spent more time performing low intensity activities and less time performing moderate and high intensity activities during training (Gabbett, 2010). With reference to velocity of movements performed, players spent approximately 60% of training but only 35% of games moving between 0-1 m.s<sup>-1</sup>. While approximately 60% of game movements and 37% of training movements took place between 1-3 m.s<sup>-1</sup> and 3-5 m.s<sup>-1</sup>.

#### 2.1.5 Small sided games

In order to introduce some intensity to training while still developing game sense and sport specific skills, coaches will often incorporate small sided games (SSG) into training. This form of training is a modified version of game play and is utilised by sports such as soccer (Coutts et al., 2009, Gabbett and Mulvey, 2008, Hill-Haas et al., 2009, Hill-Haas et al., 2011), handball (Buchheit et al., 2009) and rugby league (Gabbett, 2006) to simulate the activity profile of matches, add pressure to training drills and to improve physiological components such as aerobic capacity whilst still working on an athlete's games specific skills (Buchheit et al., 2009,

Gabbett and Mulvey, 2008, Rampinini et al., 2007). Games typically take place on a reduced pitch, often using modified rules and involve a smaller number of players than a traditional game (5 vs. 5, 4 vs. 4, 4 vs. 3 and 3 vs. 3) (Hill-Haas et al., 2011). The intensity of training is influenced by the pitch size used, the length of games, the rest period between games and the number of participants on each team (Rampinini et al., 2007). Games typically last between 2 and 10 min and have a work to rest ratio between 1:1 and 4:1 (Hill-Haas et al., 2011). The games themselves are played at a high intensity with athletes reaching 90-95% of maximal heart rate when games are used as a physical conditioning tool (Hoff et al., 2002) and player rating of perceived exertion during games can reach 8.5/10 (Rampinini et al., 2007). However, similar to other training methods, SSG training may not always simulate the same high intensity, repeated running efforts that actual match participation provides (Gabbett et al., 2009).

As well as working on sports specific skills, using SSG as a part of training can improve a number of desired physiological attributes. In handballers, training which included SSG improved time to exhaustion during intermittent fitness tests, improved repeat-sprint ability and decreased the percent decline during the repeat-sprint ability test (Buchheit et al., 2009). In soccer, 7 weeks of SSG training improved distance covered in the Yo-Yo intermittent recovery test in youth players (Hill-Haas et al., 2009) while 12 weeks of SSG training substantially improved both maximal oxygen uptake and distance covered in the Yo-Yo intermittent recovery test (Krustrup et al., 2010).

Similar to other sports, SSG are used in AF as a training tool to replicate game play and improve physiological capacities whilst working on game based skills. Although the effects of SSG on physical capacities in AF is yet to be elucidated, it is reasonable to assume that effects similar to those seen in other sports take place.

Although training is physically demanding on players, it is understandable that the demands are not entirely similar to games. During training, athletes typically cover less distance per training minute, cover less high speed distance, performed less repeated high-intensity effort bouts, spend less time performing moderate and high intensity activities and more time performing low intensity activities and perform less sprints. Competitive games are of a highly intense nature and when combined with the limited recovery time between matches in a weekly competition, training cannot and should not entirely match game demands

#### 2.1.6 Physiological effects resulting from game/training participation

It is clear that team sport games and training, including AF, can be demanding. These demands can lead to a number of physiological changes. Team sports participation can lead to fatigue resulting in performance declines both in-game and in the days following. Temporary or transient fatigue can lead to decreased match running performance, while prolonged fatigue, which can take hours or days to recover from, can also negatively influence physical performance and perceptions of recovery in the days after exercise (see section 2.2). Match participation can also result in muscle soreness and damage (see section 2.4) as well as an acute inflammatory response (see section 2.5) which similar to fatigue, can lead to reduced physical performance in the days following exercise.

#### 2.1.7 *Recovering from games/training*

As the demand and physical stress on team sport athlete's increases, so does the importance of recovering effectively from these demands. Recovery plays a vital role in any training program. Introducing adequate recovery procedures and modalities can allow athletes to overcome the demands of both training and competition, particularly those in week to week competitions (Halson, 2011) (see section 2.6). Given the potential decrements associated with AF participation, it is important that athletes are afforded adequate recovery time in order to be at their best when they are required to play and train next.

Post-match recovery should be aimed at reducing the magnitude of physiological disturbance associated with exercise. Sections 2.7-2.12 will review two recovery modalities commonly used in team sport settings (cold water immersion, CWI and contrast water therapy, CWT) and

their effectiveness on restoring physical function as well as on the fatigue, muscle damage/soreness and inflammation commonly seen after team sport participation.

#### 2.1.8 Conclusion

It is clear in many sports that both training and competition are physically taxing. Australian football is a highly challenging sport which has been increasing in intensity over the last 5 seasons. Players cover more high intensity running and total distance per minute of game time and undertake more accelerations than ever before. Matches result in decreased physical capacities as well as increased fatigue, muscle soreness and muscle damage. It is important that players are given enough time to recover from training and game stresses, however this may not be practical during a long season. Therefore employing a recovery modality which can increase player recovery and reduce the deleterious effects of AF participation is of paramount importance.

#### 2.2 Fatigue in team sport athletes

During athletic or sports participation, participants often experience some form of physical or muscular fatigue. Muscle fatigue has been defined as a transient and recoverable decline in muscle force and/or power with repeated or continuous muscle contractions (McKenna et al., 2008) and within sport competition it has been described as an exercise induced impairment of performance (Knicker et al., 2011). Fatigue experienced during exercise can lead to changes in physical abilities such as: lowered mean and peak power production, decreased muscle force, a decrease in stride or pedal frequency, slower sprint and agility times, a decrease in the ability to execute motor skills, and an increase in subjective sensations like tiredness and general fatigue (Knicker et al., 2011). Fatigue in team sport athletes manifests in a number of ways including; a loss of exercise capacity or reduced running during matches (Aughey, 2010, Bradley et al., 2009, Mohr et al., 2003), a reduction in force and power production resulting in reduced physical performance in the day/s post-game (Andersson et al., 2008, Ascensao et al., 2011,

Ascensao et al., 2008, Cormack et al., 2008b, Ispirlidis et al., 2008) and an increase in the sense of effort (Montgomery et al., 2008b, Rowsell et al., 2009, Rowsell et al., 2011).

Fatigue can be temporary or more prolonged. Temporary or acute fatigue results in a transient but recoverable loss of muscle force and is most likely responsible for reductions of performance within and immediately after a game/training. Performance related declines resulting from temporary fatigue recover mostly during the first hour after activity (Allen et al., 2008b). Prolonged or accumulated fatigue however can take hours or even days to subside after exercise (Keeton and Binder-Macleod, 2006). In situations where repeated bouts of intense activity are performed over several days, such as during a heavy training period, during a competitive season or tournament scenario, fatigue can accumulate and negatively impact on the ability of muscles to function properly. This may be due to low-frequency fatigue (Balog, 2010, Ferguson, 2010, Keeton and Binder-Macleod, 2006), which is characterized by a proportionately greater loss of force in response to low-versus high-frequency muscle stimulation. Such a loss in force will play a significant role in the decline in the force-generating capabilities of skeletal muscle (Keeton and Binder-Macleod, 2006). Accumulated fatigue may play a role during a competitive team sport season, such as AF, where players are expected to train several times per week and compete on weekends. Continued training and competition in a fatigued state can lead to overreaching, a state of temporary performance decrement or a more severe condition called overtraining which is characterised by long-term performance decrements (Fry et al., 1991). This can ultimately lead to maladaptation and compromised and reduced performance. It is therefore imperative that athletes adequately recover in order to limit the effects of this type of fatigue on athletic performance.

The effects of acute and prolonged/accumulated fatigue can be measured in a number of ways. During a game, reductions in specific skills or running are typically used as indirect measures of acute fatigue, while strength, repeat-sprint ability and countermovement jump measures are used in team sport to measure prolonged fatigue immediately and in the days after a game. The following sections will review the effects of temporary/acute fatigue on match performance (i.e. running and passing) and the effects prolonged/accumulated fatigue can have on match related physical performance (i.e. sprinting and jumping) in the days following exercise and during a tournament/team sport season.

#### 2.2.1 Effect of acute fatigue on physical performance

#### 2.2.1.1 Acute fatigue decreases running performance in games

The fatigue experienced during competitive play is typically measured as a drop in running performance and may be an indicator of players experiencing temporary fatigue (Bangsbo, 1994, Bangsbo et al., 2007, Bangsbo et al., 2006, Bradley et al., 2009, Mohr et al., 2003, Rampinini et al., 2009, Mohr et al., 2005). In soccer, fatigue during games leads to decrements in performance throughout a game (Bangsbo, 1994, Bangsbo et al., 2007, Bangsbo et al., 2006, Bradley et al., 2007, Bangsbo et al., 2006, Bradley et al., 2007, Bangsbo et al., 2003, Rampinini et al., 2009, Mohr et al., 2009, Mohr et al., 2003, Rampinini et al., 2009, Mohr et al., 2005). Decrements in distance covered, running intensity and technical performance have been reported (Bangsbo et al., 2007, Bangsbo et al., 2006, Bradley et al., 2009, Mohr et al., 2003, Rampinini et al., 2009). Elite players cover greater distances in the first half of games in both running (2172 m vs. 2052 m) and jogging (879m vs. 827 m) when compared to the second half, while walking distances in the second half increase when compared to the first (1929 vs. 1889) (Bradley et al., 2009). When combined, the overall drop in running and increase in player walking is indicative of player fatigue.

The total amount of high intensity running also declines towards the end of a soccer game and after periods of increased high-intensity movement (Bradley et al., 2009, Mohr et al., 2003). During the last 15 min of a game, high intensity running can drop by 14-45% when compared to the first 60 min (Mohr et al., 2003). Similarly, when compared to the first 15 min of each half, high intensity running can drop in by 17% in the last 15 min of the first half and by 21% in the final 15 min of the second half (Bradley et al., 2009). Following the most intense 5 min period of a game, distance covered in the ensuing 5 min can drop by over 50% and this can become a

12% reduction when compared to distance covered during all 5 min intervals in a game (Bradley et al., 2009, Mohr et al., 2003).

Technical performance is also affected by match fatigue. In the second half of games, short passing attempts (-10%), short pass success (-9.9%) and the number of involvements with the ball (-8.3%) all decline (Rampinini et al., 2009). A 5.2-5.7% decline in the high intensity running distance whilst in possession of the ball is also evident in the second half (Bradley et al., 2009, Rampinini et al., 2009).

A game of AF places great demands on the running endurance, speed-endurance, repeat-sprint ability, maximal running velocity and acceleration of players and over the course of a game players will experience some fatigue (Wisbey et al., 2010, Aughey, 2010, Coutts et al., 2010). Players can cover between 12,700-13000 m during matches and up to 18,000 m during finals matches (Aughey, 2011b, Aughey, 2010, Coutts et al., 2010) and when compared to the first quarter, total and high-intensity running distances covered in the second, third and fourth quarter of games decreased (Coutts et al., 2010). High-intensity running and maximal accelerations also decreased within each quarter and this is indicative of significant player fatigue (Aughey, 2010) (Figure 2.3).

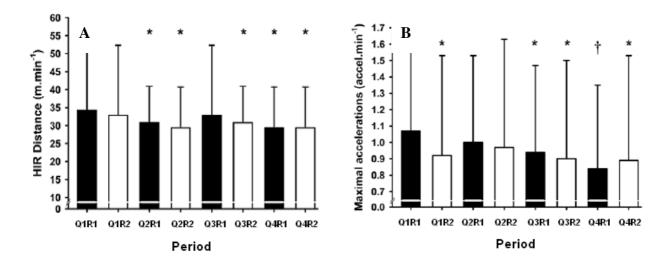


Figure 2.3: Distance per rotation expressed in metres per minute of AF game time for (A) high intensity running and (B) accelerations. \* Denotes a small reduction from Q1R1. † Denotes a moderate reduction from Q1R1 (Aughey, 2010).

Although it is clear that match running performance can decline across many team sports, care must be taken when assessing such declines. Changes in running performance may be independent of fatigue as match running may be influenced by a number of factors including; team or opposition tactics (i.e. playing with an extra defender to slow down attacks on goal or block space, or playing with one less attacker in order to create more space to run into), weather conditions (i.e. wet or muddy conditions), ground size (i.e. smaller ground size may lead to less overall running due to less open space) or in reaction to a direct opponent (i.e. marking an opponent may lead to the opponent running less as they are effectively taken out of the match).

#### 2.2.1.2 Effect of acute match related fatigue on post-match physical performance

In soccer, the ability to perform repeated sprints declines substantially post game. Repeat-sprint time increased considerably (3-8%) (Andersson et al., 2008, Ascensao et al., 2008) and remained elevated by 5-12% over 72 h after a soccer match (Ascensao et al., 2011, Ascensao et al., 2008, Ispirlidis et al., 2008, Fatouros et al., 2010). The ability to produce maximal strength and force from the quadriceps and hamstrings as well as vertical jump height also decline post-match (Andersson et al., 2008, Ascensao et al., 2011, Ascensao et al., 2008). After a soccer match, hamstring and quadriceps peak torque (at 90 deg.sec<sup>-1</sup>) were both reduced post-game (Ascensao et al., 2008). Hamstring strength decreased 15% over the first 24 h and remained 10% lower than baseline at 48 and 72 h. Quadriceps strength followed a similar pattern 48 (10%) and 72 h (5%) post-match. These strength reductions corresponded with a 7% decline in 20 m sprint time post-match which after 72 h remained reduced by 5%. In another soccer study, post-match declines in 20 m sprint performance (3.4%) and countermovement jump (4.7%) corresponded with reductions in peak torque for both knee extension (7.8%) and knee flexion (8.7%) (Andersson et al., 2008).

In AF, players experience reductions in physical performance as a result of participating in a match (Cormack et al., 2008a, Cormack et al., 2008b, Dawson et al., 2005). When compared to

pre-match measures, vertical jump height was 9.6% lower 15 h post-match (Dawson et al., 2005) while the ratio of flight time:contraction time decreased 7.5% immediately post-match and was 7.8% lower after 24 h (Cormack et al., 2008a). Peak power and total work as measured on a cycle ergometer also declined post-match (Dawson et al., 2005) and although sprint cycling is not directly related to the game of AF, the ability to produce and maintain power can affect sprinting performance during games. During a 6 s sprint test, peak power declined 9.7% 15 h post-match and remained 1.6% lower after 48 h (Dawson et al., 2005). Total work also declined 10.7% after 15 h and 1.3% after 48 h (Dawson et al., 2005). In an AF match, declines in jumping ability and peak power output will be detrimental to performance in athletes who are repeatedly involved in marking and ruck contests (repeated jumping) as well as negatively impact change of direction, kicking and sprinting.

#### 2.2.2 Effect of accumulated fatigue on physical performance

#### 2.2.2.1 Accumulated fatigue decreases running performance during matches

When multiple training sessions or games are played over a short period of time such as during a training camp, a tournament or a heavy pre-season training program such as an AF pre-season, fatigue can accumulate. Over the course of a 3 game, 4 day field hockey tournament, the percent of total game time spent standing by players significantly increased across all three games (7.4%, 11.2% and 15.6%) while the frequency of repeat-sprints decreased (Spencer et al., 2005). When compared to distances covered during the first game of a 6 game, 9 day field hockey tournament, strikers were unable to match total distance (10,787 m) or high speed running distance (2,706 m) in any of the 5 subsequent matches (Jennings et al., 2012). Strikers experienced a 2.4-20.6% drop in total distance and an 8.5-36.9% drop in high speed running distance. Midfielders closely matched game 1 total distance (0.1-4.6% decrease) however they were not able to match high speed running distances (7.3-13.2% decrease). Total distance for defenders varied the most. Compared to game 1, total distance both increased (3.4-7.5%) and decreased (0.6-9.9%), however similar to both the strikers and midfielders, high speed running

decreased by between 1.0-33.3% across the tournament. The differences observed in total distance between playing position may be due to the number of times each player was substituted as the tournament progressed or specific game tactics. However, high speed running, which is important for getting into scoring positions (Faude et al., 2012) and closing down attacking plays, decreased across the tournament in all positions and is evidence that any accumulation of fatigue results in players being unable to sustain the demands for repeat running across a number of games.

The inability to sustain high speed running across a number of days can have implications for all team sport athletes. This drop in high speed running could affect the outcome of competitive games particularly in AF, where the activity profile of athletes is greater than other team sports (see section 2.1). Therefore, without adequate recovery between training and games, it is likely that AF players will experience declines in high speed running performance.

## 2.2.2.2 Effect of accumulated fatigue on post-match physical performance

During a competitive AF season, muscle fatigue can accumulate. Countermovement flight time:contraction time ratio was used as a measure of fatigue throughout an entire 22 week AF season (Cormack et al., 2008b). When measured 72-96 h post match, flight time:contraction time was substantially lower (60%) than pre-match values (Cormack et al., 2008b). This highlights the fact that post-match fatigue can indeed accumulate across an AF season and emphasizes the need for adequate recovery protocols to allow athletes to overcome the fatiguing effects of weekly match play.

Muscle fatigue can also accumulate in athletes participating in tournaments and adversely affect physical performance. In handball players participating in a 5 day training camp followed by a 3 day tournament, both jump and sprint performance were negatively affected (Ronglan et al., 2006). During the training camp jump height decreased by 6.9% while 20 m sprint time increased by 2%. During the tournament, jump height was reduced by 6.7% with a 3.7 % increase observed in sprint time. Cumulative fatigue was also evident over three consecutive

days of basketball play (Montgomery et al., 2008b). Athletes had small impairments in a linedrill ability test, a moderate decrement in 20 m acceleration and a large to very large decrement in agility. General fatigue in these athletes also increased substantially. Similar to handball, during a 3 day basketball tournament, there was a small effect indicating that vertical jump decreased post game (Montgomery et al., 2008b).

# 2.2.3 Perceived fatigue as a measure of fatigue in team sport

A commonly used tool for assessing post-exercise fatigue in the team sport setting is the measurement of a player's perception of fatigue. Perceptions of general and/or prolonged fatigue can be assessed using a visual analogue scale (VAS). The VAS is an instrument that measures a characteristic or attitude that is believed to range across a continuum of values and cannot easily be directly measured (Gould et al., 2001). During several 4 game, 4 day soccer tournaments (Rowsell et al., 2009, Rowsell et al., 2011), general fatigue in two recovery groups (cold water immersion and thermoneutral immersion) was measured 22 h after completing a game (prior to next game). Across the tournament, both groups indicated they felt fatigued prior to games 2, 3 and 4, with perceived fatigue 30-45% lower in the cold water immersion group. Perceived fatigue has also been measured during a 3 game, 3 day basketball tournament (Montgomery et al., 2008a, Montgomery et al., 2008b). Perceived fatigue was measured preand immediately post-games and increased in 3 groups (cold water immersion 27%, compression garment 28% and control 31%) across the 3 days.

Post-match physical performance can be adversely affected in athletes experiencing higher levels of perceived fatigue in the days after exercise. Over the course of a 4 day soccer tournament, the group of athletes with the lowest perception of post-match fatigue, (cold water immersion) exhibited smaller reductions in total game distance (580 m) when compared to a thermoneutral group (875 m) (Rowsell et al., 2011). During a basketball tournament, the group with the smallest increase in perceived fatigue (cold water immersion, 1.0) performed best in line drill (repeat-sprint between lines on court) and 20 m sprint tests when compared to a

control group (perceived fatigue of 2.2) and a group wearing compression garments (perceived fatigue of 1.1) (Montgomery et al., 2008b).

Mental fatigue can also contribute to a decrease in physical performance by limiting exercise tolerance through higher perceptions of effort (Marcora et al., 2009). In participants performing cycling efforts, subjects who were mentally fatigued prior to exercise reached their maximal level of perceived exertion and disengaged from their cycling task earlier than a control group condition (Marcora et al., 2009). This has implications for team sport athletes who feel tired or fatigued going into a game or in the day/s after a game of heavy training session. Their perception of effort is likely to increase quicker than less fatigued athletes and result in decreased physical performance.

Adequate recovery will allow athletes to overcome game and training related perceptions of fatigue, therefore monitoring athletes for increased tiredness should be part of any team sport monitoring program.

#### 2.3 Introduction to mechanisms contributing to exercise-induced fatigue

Fatigue can be temporary or prolonged (Allen et al., 2008b, Davis and Walsh, 2010) and result in reduced running performance during games, reduced physical performance in the days after a game or decreased performance across a tournament or competitive season (see section 2.2.1 and 2.2.2).

Fatigue, leading to reduced running during games or post-exercise physical performance, is a multifaceted phenomenon comprising numerous components acting at multiple sites within both the central nervous system and within the muscle itself (Figure 2.4) (McKenna, 2003). Fatigue is considered to fall into two broad categories: central, which involves disturbance to neuromuscular transmission between the central nervous system and muscle membrane, and peripheral factors, which would lead to alteration within the muscle (Giannesini et al., 2003, McKenna, 2003). Both central and peripheral fatigue work collectively to reduce muscle power however it appears that a significant portion of fatigue is of a peripheral origin with central

factors contributing to approximately 10-20% (Allen and Westerblad, 2001, Kent-Braun, 1999, Westerblad et al., 1998, McKenna, 2003).

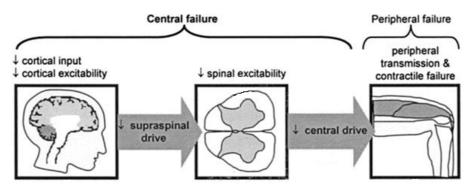


Figure 2.4: Contributors to central and peripheral fatigue. Adapted from (Kalmar and Cafarelli, 2004).

# 2.3.1 Central Fatigue

Central fatigue has been described as a progressive, exercise-induced decline in voluntary activation of a muscle (Gandevia et al., 1996) or as a force generated by voluntary muscular effort that is less than that produced by electrical stimulation (Davis and Bailey, 1997). Central fatigue causes a progressive decrease in the maximal voluntary force produced by a muscle/muscle group which occurs through inadequate activation of motoneurons (Taylor et al., 2000) and as a result, motor unit firing rates decline (Gandevia, 2001). As exercise progresses, the ability to generate maximal voluntary force declines, and the central drive required to maintain a given intensity of exercise increases (Kalmar and Cafarelli, 2004). The net result of this is a reduction in muscle force.

Contributors to central fatigue include changes at both the supraspinal and spinal level (Figure 2.4) (Davis and Bailey, 1997, Davis and Walsh, 2010, Gandevia, 2001, Kalmar and Cafarelli, 2004, Taylor et al., 2000). A decrease in supraspinal drive, resulting from a drop in cortical input and cortical excitability/stimulation, can contribute to central fatigue (Gandevia, 2001, Kalmar and Cafarelli, 2004, Taylor et al., 2000). Transcranial magnetic or electrical stimulation produces nervous propagation along the corticospinal tract and spinal roots, and stimulation over the primary motor cortex produces excitatory EMG responses in most muscles at short

latency (termed motor evoked potentials; MEP) (Gandevia, 2001, Gandevia et al., 1996). The degree of force increment of a muscle evoked by stimulation of the motor cortex depends on the size of the MEP induced (Gandevia, 2001, Gandevia et al., 1996, Kalmar and Cafarelli, 2004, Taylor et al., 2000). An increase in MEP amplitude during voluntary contraction (as measured by increased force during) is an indicator of reduced cortical drive and the greater the decrease in force, the greater the decrease in cortical drive (Gandevia, 2001, Gandevia et al., 1996, Kalmar and Cafarelli, 2004, Taylor et al., 2000). Following a series of fatiguing intermittent maximal voluntary contractions of the elbow flexors, a loss of ~7% in voluntary torque was measured (Taylor et al., 2000). Following cortical stimulation, the torque increment increased between 1.5-7% of ongoing torque and this demonstrates a supraspinal component to the central fatigue produced by the elbow flexor contractions (Taylor et al., 2000). Central fatigue was also developed following sustained (3 min) maximal voluntary contraction of the biceps fell to 90% after 3 min, however following cortical stimulation, force increased by 9.8%.

Transcranial magnetic stimulation can also generate separate inhibitory effects at cortical level following exhaustive exercise where a decrease in MEP amplitude is indicative of intracortical inhibition (Gandevia, 2001, Verin et al., 2004). Following an incremental treadmill test till exhaustion (lasting ~18 min) MEP amplitude for the quadriceps was reduced. When measured 5, 20 and 40 min post-test, quadriceps MEP values were 59, 74 and 73% of baseline (Verin et al., 2004). These values are suggestive of a role for central fatigue in the task failure and this fatigue can persist for 40 min post exercise.

Changes at the spinal level leading to impaired alpha motoneuron activity also contribute to central fatigue (Gandevia, 2001, Garland, 1991, Garland and Kaufman, 1995, Macefield et al., 1991). A decreased firing rate of muscle spindles is one such contributor. During a sustained submaximal voluntary contraction (~30%) lasting 1 min, the rate of spindle firing of the ankle dorsiflexors declined by 72% and is evidence of fatigue (Macefield et al., 1991). Reflex inhibition of small diameter or group III and IV afferents can also lead to a decline in

motoneuron activity (Garland, 1991, Garland and Kaufman, 1995). Following a fatiguing protocol of the soleus muscle (4-5 min stimulation) and a compressive block of large diameter afferents, maximal voluntary contraction declined by 56.8% and was associated with an inhibitory reflex of the group III and IV afferents and resulted in decreased motoneuron output (Garland, 1991).

During sustained and exhaustive exercise, such as sprinting and repeated sprinting, changes at the cortical and spinal level can lead to suboptimal recruitment of motoneurons resulting in decreases in force. For team sport athletes, any decrement in motoneuron recruitment may negatively impact on match or training performance. Although this may be true, central factors contribute to ~10-20% of exercise induced fatigue leaving peripheral fatigue as the major contributor (Allen and Westerblad, 2001, Kent-Braun, 1999, Westerblad et al., 1998, McKenna, 2003). Proposed peripheral mechanisms contributing to fatigue will be reviewed in section 2.3.2.

#### 2.3.2 Peripheral fatigue

Peripheral fatigue has been described as arising from the failure of mechanisms at or beyond the neuromuscular junction (McKenna, 2003) and is thought to be made up of many facets. The degree of central fatigue is greater for exercise bouts of longer duration (i.e. greater than 2 h) than for shorter, more intense bouts (Millet and Lepers, 2004), therefore, it seems that peripheral fatigue may play a larger role in leading to performance reductions during and following team-sport performance. Below is review of some of the contributing mechanisms.

#### 2.3.2.1 Sodium/Potassium disturbances

The transport of both sodium (Na<sup>+</sup>) and potassium (K<sup>+</sup>) is critical to ensuring cell membrane excitability (Allen et al., 2008b). The ability to sustain an action potential during intense activity or high frequency is primarily dependent on the ability to restore K<sup>+</sup> ions from interstitial space back into the cell and also to eject Na<sup>+</sup> ions back to the interstitial space (Green, 1997, Fowles et

al., 2002). Re-establishing this gradient is under the control of the Na<sup>+</sup>-K<sup>+</sup> pump which expends energy (ATP) to pump these ions against their concentration gradient (Fowles et al., 2002, Clausen, 2003). The enzyme required for this process to occur is embedded in the membrane and is called Na<sup>+</sup>-K<sup>+</sup>-ATPase (Green, 1997, Clausen, 2003). The capacity of the Na<sup>+</sup>-K<sup>+</sup> pump can be challenged by heavy contractile demands or intense activity (Fowles et al., 2002, Green, 1997, McKenna, 2003, Clausen, 2003). In order to maintain action potentials during intense activity, the pump must possess a high ATPase activity and a high capacity for rapid ATP hydrolysis (Fowles et al., 2002, Green, 1997). During fatiguing exercise, maximal ATPase activity can become reduced (Fowles et al., 2002, Fraser et al., 2002) and may result in an impairment to membrane potential due to changes in K<sup>+</sup> uptake and Na<sup>+</sup> efflux (Clausen, 2003). This can then manifest in a drop in muscle force production (McKenna, 2003) and can impact on actions such as kicking, jumping, directional changes and sprinting which are common to team sports such as AF and soccer.

#### 2.3.2.2 E-C coupling

Alterations to excitation-contraction (E-C) coupling can also contribute to fatigue during team sport participation (Giannesini et al., 2003, Place et al., 2010). Excitation-contraction coupling is the sequence of events from the generation of an action potential at the surface of a muscle fibre, right through to the sliding of myofilaments (Powers and Howley, 2007). Calcium (Ca<sup>2+</sup>) plays a key role in muscle contraction (cross bridge cycling) and modulates the force of these contractions (Martonosi, 1984). In muscle, Ca<sup>2+</sup> is stored in and released during contraction from membranous channels known as the sarcoplasmic reticulum (SR) (Martonosi, 1984). Any disruption to the release of Ca<sup>2+</sup> from the SR and subsequent decline in intracellular Ca<sup>2+</sup> or a decrease in myofibrillar Ca<sup>2+</sup> sensitivity can negatively impact cross bridge activity (Place et al., 2010). This will impact on the ability of muscle to produce force (McKenna, 2003) and therefore negatively impact the physical performance of team sport athletes during games.

# 2.3.2.3 Inorganic phosphate

The accumulation of inorganic phosphate ( $P_i$ ) in working muscles has been linked to fatigue and to a reduction in the force generating capacity of muscle. This is a major factor thought be responsible for the depression of SR Ca<sup>2+</sup> release (Allen et al., 2008a, Allen et al., 2008b, McKenna, 2003). Adenosine triphosphate (ATP) and phosphocreatine (PCr) are broken down to supply energy for muscle contractions (Allen et al., 2008b). During periods of fast energy consumption, the breakdown of PCr results in an accumulation of  $P_i$  (Allen et al., 2008b, Allen et al., 2008a, Allen and Westerblad, 2001). This  $P_i$  can then enter the SR and precipitate with Ca<sup>2+</sup> leading to decreased SR Ca<sup>2+</sup> release, decreased myofilament Ca<sup>2+</sup> sensitivity, impairments to cross bridge attachment leading to a reduction in muscle force due to alterations in E-C coupling and ultimately negatively influence physical performance (Allen et al., 2008b, Allen et al., 2008a, Allen and Westerblad, 2001, Duke and Steele, 2001, Fitts, 1994, Westerblad et al., 2002). During an AF game, the accumulation of  $P_i$  and resultant drop in muscle force may impact on a player's ability to jump, sprint or repeat sprint or negatively impact on their ability to outmuscle an opponent or win a contested situation.

# 2.3.2.4 Phosphocreatine/ATP depletion

High intensity exercise lasting several minutes through till exhaustion results in almost total depletion of muscle phosphocreatine (PCr) (Hultman et al., 1967). This has been confirmed following electrical stimulation of the quadriceps under anaerobic conditions (Spriet et al., 1987) and after repeated bouts of intense cycling (McCartney et al., 1986). Although under these conditions the maximum rate of ATP resynthesis is reduced, it is the rate of PCr hydrolysis which appears to be more important as this indicates the rate of ATP resynthesis (McKenna, 2003). Following a 6 sec sprint, muscle PCr and ATP content decreased by 57% and 13% respectively (Gaitanos et al., 1993). Were this to continue, PCr would be depleted after 10 sec and ATP after 46 sec, meaning that during an event like a 400 m sprint, athletes

would go into muscle rigor. This does not occur, therefore, the rate of PCr hydrolysis to help resynthesise ATP is important during brief high intensity exercise (McKenna, 2003).

During short repeated sprints, reductions in PCr can also affect performance. In subjects performing 10 x 6 sec sprints (30 sec rest between), PCr concentration [PCr] decreased to 57% of resting values after the first sprint and progressively declined to 16% after the final sprint (Gaitanos et al., 1993). Similarly, during 5 x 6 sec sprints (departing every 30 sec) [PCr] was reduced (Dawson et al., 1997). During the post-exercise recovery period [PCr] was 27.4% of pre-exercise levels after 10 sec and 44.7% after 30 sec recovery. After 3 min however, [PCr] was close to pre-exercise levels (83.7%) (Dawson et al., 1997). These results indicate that following repeated 6 sec sprints, short recovery periods do not allow for complete resynthesis of PCr, however longer recovery durations can increase levels of resynthesis. The repeat sprint nature of team sports means that players will be utilising and depleting their PCr stores during high intensity bouts in games. However the intermittent nature of a team sport like AF, where there are numerous breaks in play (for ball ups, boundary throw ins, set scoring shots on goal as well as time between scores to the resumption of play) means that players are regularly afforded breaks allowing PCr to partially recover. Additionally, players have 3 extended breaks during games (6 min for both 1/4 and 3/4 time, and 20 min for 1/2 time) meaning full repletion of PCr will have occurred. Consequently, PCr depletion is likely to be responsible only for transient fatigue during games. Any depletion of PCr during games is unlikely to contribute to the prolonged fatigue after games, as measured by drops in next day physical performance, as it will have fully recovered.

#### 2.3.2.5 Muscle glycogen

The depletion of intra-muscular glycogen stores has also been implicated in fatigue (Allen et al., 2008b, Ortenblad et al., 2011, Nielsen et al., 2011, Helander et al., 2002). Lowered muscle glycogen concentrations (muscle [glycogen]) can lead to exercise-induced impairment of the SR (Ortenblad et al., 2011). During repeated muscle stimulation,  $Ca^{2+}$  release from the SR

decreases (Allen et al., 2008b) which can be affected by muscle [glycogen] (Helander et al., 2002). In untrained subjects, an association has been demonstrated between low muscle [glycogen] and a reduction in SR  $Ca^{2+}$  release leading to reduced force during muscle contraction (Duhamel et al., 2006).

During intense all out exercise, decreased muscle [glycogen] can affect energy production. During a 6 sec all out sprint, muscle [glycogen] can decrease by 14% and by 25% after a 30 sec sprint (Cheetham et al., 1986). These reductions may negatively impact sprint and repeat sprint performance typical to team sports, as reduced glycogen availability would restrict the rate of ATP production through glycolysis (McKenna, 2003).

During both short and prolonged high intensity intermittent exercise, muscle glycogen availability affects physical performance (Balsom et al., 1999). Fatigue during the second half of a soccer game is generally higher than the first half which has been linked to the depletion of muscle glycogen (Bangsbo et al., 2007, Green, 1997, Krustrup et al., 2006, Reilly, 1997, Reilly et al., 2008, Mohr et al., 2003). Glycogen concentration in muscle is reduced by up to 90% during a game (Bangsbo et al., 2007, Reilly et al., 2008) and after a soccer match, up to 47% of quadriceps muscle fibres may be almost depleted of glycogen (Krustrup et al., 2006). This has negative implications for energy production as muscle triglycerides, blood free fatty acids and glucose are progressively used during oxidative metabolism (Bangsbo et al., 2007, Reilly et al., 2008). As a result, substantial glycogen depletion may lead to a drop in athletic performance, particularly in the second half of games.

Reduced muscle [glycogen] may also influence exercise performance in days after a game. Following a soccer match, muscle [glycogen] in players was 43% lower immediately postmatch and 27% lower after 24 h when compared to pre-exercise levels (Krustrup et al., 2011). Although no direct evidence is available, such reductions are also likely to have an effect on AF players as the running completed and therefore energy requirements of AF are greater than those of other team sports (see section 2.1). In the days after an AF match, it is possible that game induced muscle [glycogen] reductions would impact exercise performance. Post-match meals consumed by AF players are typically high in carbohydrates and they are encouraged to consume high carbohydrate drinks post-match and in the day after. Although players would be restoring their glycogen levels, repletion of glycogen stores following 2 h of exercise (the approximate duration of an AF match) can take up to 46 h post-exercise to be complete (Piehl, 1974). Therefore in the day/s following a match, incomplete muscle glycogen repletion is likely to be a contributor to post-exercise performance reductions.

## 2.3.3 Conclusions

Fatigue is a complex phenomenon related to both central and peripheral factors including; impaired motoneuron activation, reduced cortical drive, glycogen depletion, disturbances in  $Na^+$  and  $K^+$  regulation, accumulation of  $P_i$ , the depletion of PCr and ATP and E-C coupling alterations. Fatigue can result in short term reductions in athletic performance or have a more prolonged effect. The inability to overcome both short term and prolonged fatigue can lead to athletes displaying symptoms of overreaching (Halson and Jeukendrup, 2004). In order for athletes to overcome residual fatigue, it is of paramount importance that they recover effectively. This will enable them to perform to their maximum during their next training session or competition. Effective recovery is important for all athletes, but is particularly important for athletes involved in weekly competitions such as AF. In AF, little evidence exists on the efficacy of recovery interventions to reduce fatigue and therefore further investigation is required.

Team sport participation can not only result in athletes experiencing temporary or more prolonged fatigue, but can also result in athletes experiencing muscle soreness and muscle damage. This will be discussed in the following section.

# 2.4 Muscle damage from sport participation

Clear evidence exists that muscle damage occurs following sports participation, unaccustomed physical activity, activity of a high intensity or that which is prolonged and metabolically

demanding (Ascensao et al., 2008, Byrne et al., 2004, Clarkson and Sayers, 1999, Howatson and Van Someren, 2008, Ispirlidis et al., 2008, Takarada, 2003, Tee et al., 2007). This is commonly known as exercise-induced muscle damage (EIMD) (Byrne et al., 2004, Clarkson and Sayers, 1999, Howatson and Van Someren, 2008).

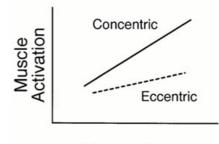
Muscle is composed of two main components; myofibres and connective tissue (Marieb et al., 2008). The myofibres and their accompanying nerves are responsible for the contractile function of the muscle while the muscle cells are bound together by connective tissue. Physical activity can result in varying degrees of damage to working muscles due to increased mechanical stress (Allen and Westerblad, 2001, Clarkson and Sayers, 1999).

Muscle damage induced by exercise can lead to heightened sensations of muscle soreness as well as compromised physical performance in the days after damage has been sustained. For team sport athletes, such as soccer, rugby, hockey and AF, the ability to sprint, jump and change direction can influence the outcome competitive situations. The causes of muscle damage and the effects it can have on athletic and team sport performance will be discussed in sections 2.4.1 and 2.4.3.

#### 2.4.1 Causes of muscle damage

Mechanical stress is thought to be a major cause of damage to myofibrils during eccentric contractions (Allen, 2001, Clarkson and Sayers, 1999, Howatson and Van Someren, 2008, Morgan and Allen, 1999, Proske and Morgan, 2001). The loading profile of eccentric contractions (high force with low fibre recruitment) (Figure 2.5) places a higher mechanical stress on muscle fibres than concentric contractions therefore causing a higher degree of damage (Enoka, 1996). The mechanism of force generation during eccentric actions also differs, whereby the cross-bridges are detached mechanically and with greater force rather than undergoing detachment involving ATP splitting, as with concentric actions (Eston et al., 2003). The degree of muscle damage experienced increases with an increasing number of eccentric

contractions as well as the degree of stretch occurring (Allen, 2001, Lieber and Friden, 1999, Morgan and Allen, 1999).



Muscle Force

# Figure 2.5: Difference between concentric and eccentric contractions in required muscle activation (electromyography) to achieve a given muscle force (Enoka, 1996).

Within a myofibril, some sarcomeres are stronger than others and it is hypothesized that when the myofibrils are stretched during an eccentric contraction, the weaker sarcomeres absorb more of the stretch (Morgan and Allen, 1999, Proske and Allen, 2005, Proske and Morgan, 2001). Consequently, these sarcomeres become progressively weaker and when they reach their yield point, they lengthen uncontrollably and rapidly to the point of little or no overlap (Clarkson and Sayers, 1999, Peake et al., 2005b, Proske and Morgan, 2001). During repeated contractions, first the weaker, then the stronger sarcomeres are over-stretched and this process can happen repeatedly as the next weakest structures are placed under increased tension (Peake et al., 2005b, Proske and Allen, 2005). During muscle relaxation, most of these overstretched sarcomeres re-integrate and resume normal function, however a few may fail and become disrupted (Clarkson and Sayers, 1999, Allen, 2001, Proske and Allen, 2005, Proske and Morgan, 2001). This disruption can lead to damage of nearby elements of the muscle such as the sarcoplasmic reticulum, transverse tubules or muscle membrane (Proske and Morgan, 2001). This can lead to a loss of membrane integrity therefore allowing intramuscular proteins such as creatine kinase (CK) and myoglobin (Mb) to leak out (Allen, 2001, Clarkson and Sayers, 1999).

As a result of muscle fibre damage, disruption of excitation-contraction coupling may occur and Ca<sup>2+</sup>moves freely into the sarcoplasm stimulating proteolytic pathways related to fibre degradation and repair (Peake et al., 2005b, Proske and Allen, 2005). This appears to create some of the symptoms related to muscle damage such as soreness and loss of muscle function, as well as an amplification of circulating muscle proteins which represents damage to the plasma membrane (Peake et al., 2005b, Proske and Morgan, 2001).

#### 2.4.1.1 Muscle damage leads to swelling and inflammation

An inflammatory response occurs after muscle damage or injury in order to clear waste from the injured area in preparation for healing (Clarkson and Sayers, 1999, Clarkson and Hubal, 2002). This response is characterised by increased fluid transfer to the area of damage as well as an infiltration of plasma proteins (Clarkson and Hubal, 2002, Clarkson and Sayers, 1999). Along with the infiltration of blood, the products of muscle breakdown can attract water causing an increase to the interstitial fluid surrounding the damaged muscle known as swelling or oedema (Clarkson and Sayers, 1999, Vander et al., 1994, Clarkson and Hubal, 2002). Infiltration inflammatory cells (macrophages and phagocytes) enter the site of damage during the inflammatory response (Clarkson and Sayers, 1999, Vander et al., 1994). The proliferation of these cells is thought to amplify the initial muscle damage through an increased release of proteases and reactive oxygen species (Clarkson and Hubal, 2002). Reactive oxygen species play a role in removing damaged skeletal muscle cells but when present in large numbers have also been implicated in secondary muscle damage(Aoi et al., 2004, Basu et al., 1999, Close et al., 2005, Kon et al., 2007).

# 2.4.1.2 Muscle damage causes a delayed onset muscle soreness

Muscle damage and the resultant post-exercise swelling can contribute to the sensation of delayed onset muscle soreness (DOMS) (Chleboun et al., 1998, Nosaka et al., 2002). This typically presents as a sensation of muscular discomfort, a dull ache and/or pain that usually

begins 12 h post exercise (Close et al., 2005, Friden and Lieber, 2001). Depending on the amount of damage, DOMS can progress from mild to severe or even debilitating soreness (Close et al., 2005) and usually peaks between 48-72 h post-exercise before diminishing with pain typically ending after 5-7 days. Soreness aside, the result of this damage can manifest in a number of ways. Swelling can increase in a muscle or group of muscles (Chleboun et al., 1998, Vaile et al., 2007) and can be accompanied by increased stiffness (Chleboun et al., 1998), tenderness and a loss of function (Byrne et al., 2004, Close et al., 2004, MacIntyre et al., 1996, Vaile et al., 2007, Peake et al., 2005b, Proske and Morgan, 2001).

The severity of DOMS is generally associated with the degree of muscle and/or connective tissue damage and subsequent inflammation/swelling (Nosaka et al., 2002). Muscle damage and soreness are often placed in the same category and generally it is considered that the greater the amount of muscle damage, the larger the DOMS experienced (Nosaka et al., 2002). Although both DOMS and changes in indirect markers of muscle damage (such as blood increases in muscle proteins, muscle swelling and loss of strength) are induced by eccentric damage it is possible that they are not closely related. Poor correlations exist between DOMS and other indicators of muscle damage while DOMS has been considered as a poor reflector of eccentric EIMD (Nosaka et al., 2002). Therefore the amount of DOMS experienced should not be confused with the amount of muscle damage.

#### 2.4.1.3 Eccentric exercise increases exercise induced muscle damage

The amount of EIMD experienced depends on the length and type of the exercise bout as well as the intensity of muscle contraction (Clarkson and Sayers, 1999, Close et al., 2004, Friden and Lieber, 2001, Kon et al., 2007). Muscle damage is evident following eccentric contractions (Clarkson and Sayers, 1999, Close et al., 2004, Friden and Lieber, 2001, Kon et al., 2007) and that damage increases with the number of contractions and with the length of stretch (Allen, 2001). In comparison to concentric contractions, eccentric contractions recruit fewer motor units meaning that a smaller cross-sectional area of the muscle is activated to handle the

imposed load (Enoka, 1996). In exercise where the eccentric component is unaccustomed, substantial or repeated, such as that experienced in competition by many team sport athletes, the pain, tenderness, swelling and stiffness develop more slowly and are most prominent over the days subsequent to the causative exercise (Allen, 2001).

A sequence of active eccentric action followed by active concentric action is known as a stretch shortening cycle (SSC) (Komi, 2000). This occurs when body segments are subjected to stretch, and takes place in most sporting activities (Eston et al., 2003). It is not surprising therefore that muscle damage is a common occurrence during prolonged or intense team sport activity exercise involving repeated SSC's such as jumping, sprinting or changing direction. Additional symptoms of EIMD include DOMS, swelling, decreased range of motion, increased blood proteins and impaired neuromuscular control (Byrne et al., 2004). Athletes may be able to tolerate some soreness while a diminished range may not adversely impact performance, but of greatest consequence to an athlete is the negative influence that muscle damage may have on muscle function and any implications for athletic performance.

# 2.4.1.4 The repeated bout effect

It is clear that unaccustomed and eccentric exercise can induce muscle damage. Following recovery from this exercise, a repeated bout of the same exercise results in minimal symptoms of muscle damage and this has been referred to as the repeated bout effect (Nosaka and Clarkson, 1995). This repeat bout effect has a protective effect against excessive muscle damage following the initial damaging bout and may be of importance in running based activities such as AF. Two weeks after completing 100 maximal eccentric activations of the knee extensors, male subjects performed 5 x 8 min bouts of downhill treadmill running (Eston et al., 1996). Following the running bouts, muscle tenderness was lower (55%), peak creatine kinase activity was lower (50%) and both concentric (25%) and eccentric (30%) maximal torque were reduced when compared to a control group who did not perform any prior eccentric knee extensor movements. A group of male subjects (7) performed 2 downhill runs

(30 min, -10% gradient at 40% peak  $\dot{V}O_{2max}$ ) separated by 2 weeks (Westerlind et al., 1992). Creatine kinase increased more following bout 1 (61%) than after bout 2 (11%), while soreness ratings were 400% lower following bout 2 compared to bout 1. These results indicate that muscle damage had been reduced during bout 2. The repeated bout effect has also been demonstrated following 8 weeks of downhill walking (Balnave and Thompson, 1993). Participants walked on a treadmill at 6.4 km/h once a week for 8 weeks. Peak serum myoglobin and creatine kinase were much lower in week 8 than in week 1 (-25% and -250%), while maximal voluntary contraction of the quadriceps was reduced by a much smaller magnitude after week 8 (-11%) compared to week 1 (-26%).

These results may be of benefit in athletes such as AF players where training and competition occurs at high intensities. The repeat bout effect may reduce the severity of symptoms following eccentric movements and also reduce the degree of loss in physical function which will only be of benefit to athletes during ensuing exercise bouts.

#### 2.4.2 Measures of muscle damage

Muscle damage can be measured directly or indirectly. Direct assessment is possible through analysis of muscle biopsies or through magnetic resonance imaging (Clarkson and Hubal, 2002). Inherent problems exist with using muscle biopsies as small samples are used to estimate damage over an entire muscle. Consequently, over- or under-estimation of the extent of damage can occur as muscle damage tends to be localised and not evenly distributed throughout a muscle (Clarkson and Hubal, 2002). Additionally, the act of taking a biopsy is in itself damaging and may exacerbate any damage present. Imaging techniques are able to assess oedema formation in whole muscles and although it is non-invasive, it remains unclear what the changes in images indicate (Clarkson and Hubal, 2002).

During muscle damage, fibres are damaged and proteins are released into the interstitial space, making their way into circulation (Lippi et al., 2008). Indirectly, EIMD is typically assessed through measuring the concentration of intracellular proteins in plasma, such as creatine kinase [CK] and myoglobin [Mb] (Bailey et al., 2007, Friden and Lieber, 2001, Peake et al., 2005b, Takarada, 2003).

Following eccentric exercise, CK has a delayed appearance in the blood, such that it does not begin to increase until 24–48 h and peaks 96–120 h post-exercise (Clarkson et al., 1992). Myoglobin however, is a much smaller protein than CK (17kDa vs. 80kDa) and appears in the blood earlier (0.5-1h post) and peaks sooner (24–72 h post) (Nosaka and Clarkson, 1996). Differing routes of delivery into circulation can explain the different time courses in the appearance and peaks of both [Mb] and [CK]. Myoglobin is a smaller protein which appears in the blood faster than larger molecular weight proteins as it has a more direct route into the micro-vascular endothelium (Mair, 1999). However, CK, being a larger protein, cannot easily enter the micro-vascular endothelium and is released into the interstitial space where it is taken up by lymphatic vessels (Lindena et al., 1979). Compared to the circulatory system, the lymphatic system circulates more slowly and relies on external forces (such as muscular activity) to facilitate movement (Lindena et al., 1979).

In the days following damaging exercise, [Mb] and/or [CK] fluctuate. Increases in both markers have been observed following a number of exercise protocols (Table 2.1).

Although team sport athletes are not subject to the magnitude of eccentric loading as those outlined in Table 2.1, participation in matches also leads to increased serum/plasma [CK] and [Mb]. Following team sport activity, [CK] peaks between 24-48 h after damage (Andersson et al., 2008, Ascensao et al., 2011, Ascensao et al., 2008, Fatouros et al., 2010, Ispirlidis et al., 2008) while [Mb] peaks between 30 min to 1 h post exercise (Ascensao et al., 2011, Ascensao et al., 2008, Boyd et al., Unpublished observations, Montgomery et al., 2008a, Takarada, 2003) (see section 2.4.4.2).

Exercise type	Participants	Time course and magnitude of post- exercise change in [Mb] and [CK] compared to pre-exercise		
Eccentric leg extension (10 x 10 at 90 deg.sec <sup>-1</sup> ) (Byrne et al., 2001)	8 healthy participants (5 male, 3 female)	CK (IU.L <sup>-1</sup> )	0 h post 1 day 2 days 4 days 7 days	102.0% 250.0% 320.0% 660.0%* 805.0%*
45-min of downhill running (Peake et al., 2005b)	10 well trained male runners and triathletes	Mb (ng.ml <sup>-1</sup> )	0 h post 1 h 24 h	1100.0% * 1800.0%* 200.0%*
		CK (IU.L <sup>-1</sup> )	0 h post 1 h 24 h	140.0%* 156.0% * 420.0%*
Countermovement jump protocol (10 x 10) (Twist and Eston, 2005)	10 male participants	CK (IU.L <sup>-1</sup> )	0.5 h post 24 h 48 h 72 h	94.7% 157.2%* 161.2%* 100.0%
30 min downhill running (Chen et al., 2007)	10 male soccer players	Mb (ng.ml <sup>-1</sup> )	1 h post 1 days 2 days 3 days	510.1% * 641.4% * 621.2% * 475.8%
		CK (IU.L <sup>-1</sup> )	1 h post 1 days 2 days 3 days	216.2% 400.0% * 440.0% * 400.0% *
Heavy eccentric squatting protocol (6 x 10 parallel squats at 100% body weight with 5-s one repetition maximum eccentric squat superimposed onto each set) (French et al., 2008)	26 healthy young males	Mb (ng.ml <sup>-1</sup> )	1 h post 24 h 48 h	783.3% * 220.4% 132.3%
		CK (IU.L <sup>-1</sup> )	1 h post 24 h 48 h	237.3% 418.5% * 215.1%
Eccentric leg extension (3 x 50 at 30 deg.sec <sup>-1</sup> ) (Magal et al., 2010)	17 untrained males	CK (IU.L <sup>-1</sup> )	0 h post 24 h 48 h 72 h 96 h	106.8% 217.2% * 165.5% 148.3% 196.6%
Drop jump protocol (5 x 20 drop jumps) (Howatson et al., 2009)	16 physically active males	CK (IU.L <sup>-1</sup> )	24 h post 48 h 72 h	375.0% * 200.0% 120.0%

# Table 2.1: Time course and magnitude of change in [Mb] and [CK] after various leg exercise protocols. \*P<0.05

Although both [Mb] and [CK] are commonly used as markers of damage, large variability in the CK response to damaging exercise has been reported, therefore [CK] may not be the most appropriate marker to assess. Large inter-subject variability and large variances in serum levels have been reported for [CK] (Nosaka et al., 1991, Nosaka and Clarkson, 1996). Following a

bout of eccentric elbow flexion, post-exercise [CK] ranged between 239 and 25,244 IU.L<sup>-1</sup> while [Mb] variations were much lower (70-3100 ng.ml<sup>-1</sup>) (Nosaka and Clarkson, 1996). The amount of activity undertaken by subjects in the days post-exercise can influence [CK] (Chen et al., 2005, Sayers et al., 2000b, Sayers and Clarkson, 2003). Compared to participants involved in daily activities, immobilised participants have much smaller changes in [CK] following damaging exercise (Chen et al., 2005, Sayers et al., 2000b, Sayers and Clarkson, 2003). Following 50 eccentric elbow flexor movements, the exercised/damaged arm was immobilised for 4 days (Sayers and Clarkson, 2003). Peak [CK] for immobilised subjects was 800 IU.L<sup>-1</sup> with a control group reaching 2800 IU.L<sup>-1</sup>. Similarly, 50 eccentric elbow flexor movements resulted in a [CK] of 4200 for subjects immobilised for 4 days and 600 IU.L<sup>-1</sup> for controls (Sayers et al., 2000b). A third group in the same study performed 50 concentric bicep curls with a light weight (5 lbs) (Savers et al., 2000b). In this group [CK] peaked at 2600IU.L<sup>-1</sup> which was 433% higher than the immobilised group but only 62% of the concentration observed in controls. The variance in [CK] may be due to the activity of the lymphatic system. Immobilisation decreases lymphatic transport, therefore the movement of CK from the interstitium into circulation is reduced thus giving a lower [CK] value (Chen et al., 2005). Conversely, light activity speeds lymphatic transport above that in controls which can assist in enhancing CK clearance. As such, the high variability in [CK] may be a product of the amount of post-exercise activity (or lack of) by participants and the rate of lymphatic transport. This response however is not true for [Mb] (Sayers and Clarkson, 2003), therefore CK may be an inappropriate marker of muscle damage.

Measuring the circumference of a limb for increased swelling following EIMD has been used as an indirect measure of muscle damage (Vaile et al., 2008c) while measuring physical performance prior to and in the days post-exercise can give an indication of the effects of damaged muscle. Physical performance measures include; strength (Bailey et al., 2007, Byrne and Eston, 2002b, Goodall and Howatson, 2008, Howatson and Van Someren, 2008), power (Elmer and Martin, 2010, Twist and Eston, 2005, Twist and Eston, 2007), speed (Highton et al., 2009, Twist and Eston, 2005), agility (Highton et al., 2009) and vertical jump (Bailey et al., 2007, Byrne and Eston, 2002a, Twist and Eston, 2007).

Muscle damage impacts on an athlete's ability to produce strength, power, speed and agility as well as jumping ability which are all important components of physical performance for team sport athletes. Athletes need to be aware of the effect that muscle damage can have on muscle function and how recovering adequately from this damage can influence physical performance.

# 2.4.3 *Effects of muscle damage on athletic performance*

Eccentric contractions can lead to the disruption and subsequent damage of muscle fibres (Allen, 2001, Clarkson and Sayers, 1999, Howatson and Van Someren, 2008) which can compromise muscle function and athletic performance (Eston et al., 1996, Highton et al., 2009). A primary objective of athletic training should be to improve physical characteristics that increase performance, however, if training is regularly negatively affected by muscle damage, training outcomes cannot be maximised. For team sport athletes, the ability to sprint, change direction, jump and kick are important in determining success and any reduction is these capacities may negatively influence the result of competitive matches. The following section will address the effects that muscle damage can have on the ability to produce muscle force, as well as the potential to influence vertical jump, sprint and agility performance.

#### 2.4.3.1 Muscle damage leads to reductions in muscle force

Eccentric contractions lead to more muscle damage than concentric contractions (Clarkson and Sayers, 1999), and a greater reduction in force production (Proske and Allen, 2005). Following concentric contractions, recovery of the decline in muscle force is complete within 1-2 h, however force can remain depressed for up to a week after eccentric contractions (Figure 2.6) (Proske and Allen, 2005). Therefore a force deficit measured after 2 h is likely to be caused by damage to the muscle (Proske and Allen, 2005) along with neuromuscular fatigue contributions (see section 2.5).

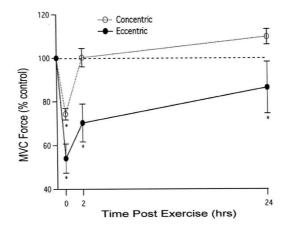


Figure 2.6: Force changes after concentric and eccentric exercise. Changes in force measured as maximum voluntary contraction (Proske and Allen, 2005).

Many team sport movements involve high movement or muscular contraction velocities (i.e. sprinting, jumping and kicking) and damage sustained during exercise can negatively impact on force production at these high velocities. Concentric isokinetic peak torque of the knee extensors at 270 deg.sec<sup>-1</sup> was measured following a muscle damaging exercise protocol (10 sets x 10 reps maximal vertical jumps) (Highton et al., 2009). Compared to a control group, isokinetic strength was substantially reduced after both 24 (8.8%) and 48 h (12.3%) post-exercise and this negatively impacted agility and both 5 and 10 m sprint performance (see section 2.4.3.2). After performing 100 eccentric knee extensor movements (10 sets x 10 reps at 90 deg.sec<sup>-1</sup>) peak concentric torque (180 deg.sec<sup>-1</sup>) was reduced 30% immediately post-exercise as well as 1 (26.5%), 2 (15.5%), 4 (18.0%) and 7 days (9.5%) post-exercise (Byrne et al., 2001). Both concentric and eccentric strength at 180 deg.sec<sup>-1</sup> have been assessed after 100 continuous maximal voluntary eccentric contractions (30 deg.sec<sup>-1</sup>) (Eston et al., 1996). Eccentric strength was substantially reduced immediately post-test (23%) as well as after 2 days (25%) with concentric strength also reduced post-test (15%) and after 2 days (13%).

For up to a week following damaging exercise, a muscle's ability to produce both concentric and eccentric force at high velocity can be compromised. This is of great concern to team sport athletes. Team sport seasons, such as in AF, consist of weekly cycles of training followed by competition and if athletes have not fully recovered from the damaging effects of either training or games during each week, any loss of concentric and/or eccentric force can have implications for power/force production and ultimately performance. For AF players, the inability to overcome muscle damage and associated reductions in concentric or eccentric strength may negatively influence on field performance of several important characteristics, these being sprinting, changing direction and jumping. The effects of muscle damage on speed and agility and vertical jump performance will now be discussed.

# 2.4.3.2 Muscle damage leads to decreased sprint and agility performance

There is mounting evidence suggesting that muscle damage has a detrimental effect on sprint and agility performance. For team sport athletes this can negatively impact on their ability to train in the days following heavy training sessions or games. Specifically, for AF players undergoing several high intensity game specific sessions per week during the pre-season, this may result in a drop in the quality/speed of training and ultimately, game performance

Sprint (5 and 10 m) and agility performance and ground contact time (agility turn point) have been assessed following a damage inducing jump protocol (10 sets x 10 reps maximal vertical jumps) in 20 recreationally active males (Highton et al., 2009). Sprint times were substantially slower for 5 and 10 m after 24 (both 5%) and 48 h (6% and 5 % respectively) before returning to baseline after 7 days. Time taken to complete a 20 m agility test (1 change of direction) increased after both 24 (5%) and 48 h (8%). Ground contact time during the agility run also increased by 16% at 24 h and by 21 % after 48 h. After performing a similar muscle damaging protocol (10 sets x 10 reps maximal vertical jumps), sprint time over 10 m in 10 male university level athletes was substantially slower after 30 min (3%), 24 (3%) and 48 h (3%) before returning near baseline after 72 h (Twist and Eston, 2005)

It must be noted that participants in the afore mentioned studies were recreationally active or university level athletes and all undertook a level of plyometric exercise not representative of team sport movement patterns, however, these results will be of concern to team sport athletes. The effects of team sport participation on sprint times in team sport athletes is discussed below (see section 2.4.4.3). However, it is clear that muscle damage decreases sprint and agility times in the days following exercise. This could impact running/training performance of highly trained multi-sprint team sport athletes such as soccer, rugby and hockey players particularly if training is compromised over a number of weeks. These results are also relevant to AF players. During the pre-season, AF players typically train within 24 h of a previous session with no more than 48 h between highly intense game-specific sessions. Therefore, adequate recovery is needed to ensure the detrimental effects of muscle damage on sprint and agility are overcome.

#### 2.4.3.3 Muscle damage decreases vertical jump performance

Muscle damage is also detrimental to vertical jump performance. Jumping can utilise the SSC (such as a countermovement or drop jump), or not (such as a squat jump). In most team sports, athletes will utilise the SSC when jumping (i.e. AF players in a ruck or marking contest, soccer players jumping to head the ball or rugby and American football players jumping to catch the ball). Therefore, this section of the literature review will focus on the effect of EIMD on vertical jump performance using the SSC only.

Vertical jump performance was assessed in 8 healthy participants following 100 squats (10 sets x 10 reps at 80% concentric one repetition maximum) (Byrne and Eston, 2002a). Countermovement jump height was reduced 9% after 1 h and remained 10% lower after 1, 2 (7%) and 3 days (6%). Similarly, drop-jump performance decreased 10% after 1 h and remained reduced after 1 (7%), 2 (9%) and 3 days (5%). Drop jump performance has also been compromised following plyometric exercise (10 sets x 10 reps maximal vertical jumps) in 19 healthy participants (Twist and Eston, 2007). Jump height was reduced at 24 (7%), 48 (7%) and 72 h (6%) while corresponding ground contact times increased after 48 (11%) and 72 h (13%). In the days after exercise, muscle damage in healthy/recreationally trained individuals or non-team sport trained athletes leads to increased perceptions of soreness and serum/plasma [Mb] and [CK]. Muscle force, sprinting and agility as well as jump performance are also compromised and a role for neuromuscular fatigue may also contribute to this (see section 2.5).

The decline in performance and increased soreness have resulted mostly from movements not typically undertaken by team sport athletes, therefore, the effects of muscle damage on team sport athletes resulting from team sport participation are yet to be fully elucidated. These will be reviewed in the section below.

# 2.4.4 Effects of muscle damage on team sport athletes

Apart from an athlete's skill level, team sport success, such as in AF is strongly linked to a player's ability to out-jump, out-manoeuvre or out-sprint direct opponents. Success against opponents can lead to an athlete scoring, creating or breaking up scoring opportunities, defending and creating turnovers. Strength and power are both important characteristics for team sport athletes, therefore any loss in these qualities will negatively impact match and training performance.

Eccentric and stretch shortening cycle actions increase the likelihood that an athlete will suffer muscle damage (Eston et al., 2003). During games, team sports athletes often perform actions with high eccentric loads such as sprinting, jumping, kicking and changing direction, therefore it is likely that the nature of team sport performance will cause some degree of muscle damage and/or soreness and result in a drop performance.

#### 2.4.4.1 Muscle soreness resulting from team sport

Physically demanding team sport or simulated team sport exercise, results in athletes experiencing substantial increases in muscle soreness of between 150-1200% in the days following activity (Table 2.2). This soreness can persist for up to 72 h post-exercise. Increased perceived pain can lead to reduced maximal strength and exercise performance (Trost et al., 2011) and in team sport athletes, this soreness can lead to a drop in athletic performance (see section 2.3.4.2).

It is clear that participating in team sports or simulated team sport activities results in substantial and prolonged perceptions of soreness. This can lead to reduced muscle strength and

affect an athlete's ability to maintain intensity in training. In AF, players train 24-72 h after their last exercise bout and during pre-season, athletes typically train within 24 h. Therefore, limiting and recovering from any match/training induced soreness and improving the ability of players to maintain training intensity will allow players to be better prepared for any upcoming matches or training sessions.

Exercise type	Participants	Time course and magnitude of post-exercise muscle soreness compared to pre-exercise		
90-min intermittent shuttle running (Loughborough Intermittent Shuttle Test: LIST) (Thompson et al., 1999)	16 male students	24 h post-exercise 48 h 72 h	1180% * 1040% * 420% *	
Australian football match (Dawson et al., 2005)	17 semi-professional AF players	15 h post-match 48 h	157% * 139% *	
90-min of intermittent shuttle running and walking (LIST) (Bailey et al., 2007)	20 healthy males	0 h post-exercise 1 h 24 h 48 h	397% * 320% * 394% * 325% *	
Soccer match (Ispirlidis et al., 2008)	24 elite male soccer players	0 h post-match 24 h 48 h	710% * 830% * 850% *	
Soccer match (Ascensao et al., 2008)	16 males soccer players	0.5 h post-match 24 h 48 h	480% * 390% * 200% *	
Soccer match (Andersson et al., 2008)	22 elite female soccer players	21 h post-match 27 h 45 h 51 h	129% * 132% * 126% * 122% *	
80-min of simulated team sports running followed by shuttle run test to exhaustion (Ingram et al., 2009)	11 male team sport participants	0 h post-exercise 24 h 48 h	700% * 500% * 500% *	
Soccer match (Fatouros et al., 2010)	20 male soccer players	0 h post-match 24 h 48 h	750% * 830% * 500% *	
Soccer match and 90-min of intermittent shuttle running and walking (LIST) (Magalhaes et al., 2010)	16 male soccer players	0.5 h post-match 24 h 48 h 0.5 h post-shuttle run 24 h 48 h	600% * 500% * 240% * 480% * 400% * 190% *	

Table 2.2: Muscle soreness following team sport and simulated team sport exercise. \*P<0.05

Muscle damage increases with the number of eccentric movements (Allen, 2001). This type of movement is common in team sports and as a result, team sport participation often results in an increased presence of muscle damage markers. In the days following a soccer match marked increases in [Mb] (1380-1400%) and [CK] (180-900%) post game have been observed (Andersson et al., 2008, Ascensao et al., 2011, Ascensao et al., 2008, Ispirlidis et al., 2008) (Table 2.3).

Exercise type	Participants	Time course and magnitude of post-match change in [Mb] and [CK] compared to pre- match		
Soccer match	24 elite male soccer players	CK	0 h post	359.1% *
(Ispirlidis et al., 2008)		$(IU.L^{-1})$	24 h	440.1% *
			48 h	854.5% *
			72 h	772.7 % *
			96 h	472.7% *
Soccer match	16 males soccer players	Mb	0.5 h post	1380.0% *
(Ascensao et al., 2008)	1 5	$(ng.ml^{-1})$	24 h	240.0%
			48 h	120.0%
		СК	0.5 h post	182.5%*
		$(IU.L^{-1})$	24 h	402.5% *
			48 h	407.5%*
			72 h	302.5% *
Soccer match	22 elite female soccer players	СК	0 h post	224.0% *
(Andersson et al., 2008)		$(IU.L^{-1})$	21 h	308.2% *
			45 h	202.1% *
			69 h	107.5 %
Soccer match	20 male soccer players	СК	0 h post	366.7% *
(Fatouros et al., 2010)		$(IU.L^{-1})$	24 h	533.3% *
			48 h	911.1% *
			72 h	772.2% *
Soccer match	16 male soccer players	Mb	0.5 h post	1400.0% *
(Magalhaes et al., 2010)		$(ng.ml^{-1})$	24 h	180.0%
			48 h	120.0%
		СК	0.5 h post	274.2%*
		$(IU.L^{-1})$	24 h	685.7% *
			48 h	468.6%*
			72 h	342.9% *

Table 2.3: Time course and magnitude of change in [Mb] and [CK] following a soccer match.  $*P{<}0.05$ 

Basketball participation can also be damaging. Over the course of a short basketball tournament (3 games in 3 days), plasma [Mb] increased 460-510% immediately after each game before returning near baseline the next day (Montgomery et al., 2008a).

The game of rugby has also proved to be muscle damaging. Fifteen rugby players were tracked over 2 consecutive competitive games with blood markers of muscle damage being measured following each match (Takarada, 2003). Games were associated with structural muscle damage caused primarily by tackles, and substantial [Mb] increases were evident immediately post-game (1200%), as well as 45 min (2100%), 90 min (1500%) and 24 h (300%) after the game. In AF, post-training data suggests that AF is also damaging. After completing an AF training session which included SSG (4 x 3 min contact and non-contact SSG with 2 min recovery between), muscle damage as indicated by an increase in plasma [Mb], was elevated 240% immediately post-exercise and was still 165% higher than baseline after 24 h (Boyd et al., Unpublished observations). Currently, the extent to which a game of AF can induce muscle

damage and inflammation is unknown. However based on training data, evidence suggesting games are more demanding than training and the fact that the game of AF involves frequent direct contact between players in the form of tackles, bumps and marking/ruck contests, it is highly likely that match participation will result in players experiencing muscle damage.

#### 2.4.4.3 Muscle damage in team sport results in reduced physical performance

The effect of muscle damage as a result of team sport participation on subsequent athletic performance has also been investigated. Table 2.4 outlines the decline in team sport related performance in studies where [Mb] and/or [CK] have substantially increased post-match.

Exercise type	Participants 24 elite male soccer players	Time course and magnitude of post- match change in physical performance compared to pre-match		
Soccer match (Ispirlidis et al., 2008)		20 m sprint	24 h post 48 h 72 h	-2.0% * -2.5% * -1.7% *
		Vertical jump	24 h post 48 h 72 h	-12.5% * -10.4% -8.3%
Soccer match (Ascensao et al., 2008)	16 males soccer players	20 m sprint	0.5 h post 24 h 48 h 72 h	-9.1% * -6.1% * -4.9% * -5.5% *
Soccer match (Andersson et al., 2008)	22 elite female soccer players	20 m sprint	0 h post 69 h	-3.5% * 0.6%
		Vertical jump	0 h post 69 h	-4.7% * -3.0%
Soccer match (Fatouros et al., 2010)	20 male soccer players	20 m sprint	24 h post 48 h 72 h	-7.6% * -5.3% * -4.7% *
		Vertical jump	24 h post 48 h 72 h	-12.8% * -5.3% -2.1%
Soccer match (Magalhaes et al., 2010)	16 male soccer players	20 m sprint	0.5 h post 24 h 48 h 72 h	-8.8% * -6.8% * -5.8% * -5.1% *
		Vertical jump	0.5 h post 24 h 48 h 72 h	-12.2% * -7.8% * -7.8% * -8.9% *

Table 2.4: Time course and magnitude of change in physical performance following team sport participation. \* Indicates performance is significantly worse than pre-exercise (p<0.05).

It is clear that the muscle damage caused by a soccer game can negatively influence physical performance and it is highly likely that similar results would be evident after an AF match. Muscle damage exists after AF training (Boyd et al., Unpublished observations) and although markers of muscle damage have not been measured after an AF game, match participation resulted in decreased physical performance (Cormack et al., 2008a, Cormack et al., 2008b, Dawson et al., 2005). Vertical jump performance was reduced 15 h post-match (9.6%) when compared to pre-match measures (Dawson et al., 2005) while the ratio of flight time:contraction time was reduced immediately post-match (7.5%) and after 24 h (7.8%)

(Cormack et al., 2008a). Peak power as measured on a cycle ergometer also declined 15 h postmatch (9.7%) and remained lower after 48 h (1.6%) (Dawson et al., 2005).

Muscle damage induced by team sport matches and training can result in performance decrements during the ensuing post exercise period. In AF, players are expected to train in the 24-48 h post-match, therefore overcoming the performance limiting effects of muscle damage is important in preparing players for their next match.

# 2.4.5 Conclusions

It is clear that sports participation, unaccustomed physical activity, prolonged and metabolically demanding activity, high intensity movements and in particular eccentric contractions can each lead to muscle damage. It is also clear that this damage results in increased plasma levels of intracellular proteins, prolonged soreness and can negatively impact one's ability to produce muscle force, reduce sprint speeds and reduce both vertical jump ability and agility. As a result, sports and team sport participation can be negatively impacted, and in order for athlete's to effectively overcome the effects of muscle damage, an adequate recovery protocol and/or recovery period is essential. This is particularly important for team sport athletes competing from week to week, therefore the effect of recovery on muscle damage requires investigation.

#### 2.5 Inflammation following exercise

The typical physiological reaction to muscle damage or injury is and acute inflammatory response which can be caused by specific and non-specific immune responses (Watkins et al., 1995). Inflammation is the process that mobilises the body's defensive systems and it represents the sum of the body's tissue reactions to cell injury. Inflammation may be triggered by factors such as infection, heat, chemical irritation, mechanical trauma, tissue injury or other irritants (Prentice, 1999, Starkey, 1999, Watkins et al., 1995) and presents typically as swelling, pain, redness and heat (Tracey, 2002). In team sports, particularly high physical contact sports

such as AF, muscle trauma and injury could be major contributors to post-match inflammation, with the purpose of inflammation being to control the effects of the injurious agent and assist in the repair and adaptation of muscle tissue (Starkey, 1999, Tidball, 2005). This process contains, dilutes or destroys the injurious agent/s in an attempt to protect the area from further insult or injury (Starkey, 1999).

During the acute inflammatory phase, leukocytes and other phagocytic cells are delivered to the injured tissue (Prentice, 1999). Phagocytic cells (macrophages) are specialised to engulf and destroy particles and infective agents and their release is generally a protective cellular reaction which tends to localise or dispose of injury by-products, such as blood and damaged cells, and set the scene for tissue repair (Prentice, 1999, Watkins et al., 1995). During this process, local vascular effects (vasoconstriction and vascular spasm), fluid exchange disturbances and migration of leukocytes from the blood to the injured tissue occur (Prentice, 1999). Upon engulfing a particle, macrophages produce, then sequentially release, a series of pro-inflammatory cytokines (Watkins et al., 1995).

Inflammation is a local response which can be caused by a number of factors including inflection, numerous irritants, tissue damage or simply by the common cold (Watkins et al., 1995). As a result, the focus of the following section is to discuss inflammation and the release/role of both pro- and anti-inflammatory cytokines resulting from muscle injury. It will critically discuss inflammation after exercise induced muscle damage and also resulting from team sport participation.

# 2.5.1 Pro- and anti-inflammatory cytokine response during acute phase inflammation

Cytokines are soluble hormone-like substances produced by a variety of cells such as immune cells and endothelial cells, which are involved in the control of the immune and acute-phase response, inflammatory reactions and the repair process of tissue (Dinarello, 1997b, Smith, 2000). Cytokines have the capacity to stimulate surrounding cells (paracrine) or themselves

(autocrine) which can lead to further cytokine production and amplification of a particular response (Smith, 2000). They mediate communication throughout the body within and between non-immune and immune cells, organs and organ systems (Pedersen et al., 1998, Shephard, 2002).

Cytokines are produced by a variety of cells including circulating leukocytes, tissue-resident and vascular endothelial cells (Dinarello, 1997b, Cavaillon, 1994, Thijs and Hack, 1995). Based on their predominant action, cytokines may be characterized as either pro- or antiinflammatory (Smith et al., 2000). Some cytokines clearly promote inflammation and are called pro-inflammatory cytokines, whereas other cytokines suppress the activity of pro-inflammatory cytokines and are called anti-inflammatory cytokines (Dinarello, 2000). Plasma or serum anti-/pro-inflammatory cytokines are frequently measured as biomarkers of inflammation (Flynn et al., 2007) with interleukin 6 (IL-6) (both pro- and anti-inflammatory cytokine), tumor necrosis factor alpha (TNF- $\alpha$ ) (pro-inflammatory cytokine), interleukin 1-beta (IL-1 $\beta$ )(pro-inflammatory cytokine) and interleukin 10 (IL-10) (anti-inflammatory cytokine) frequently measured (Flynn et al., 2007, Petersen and Pedersen, 2005, Stewart et al., 2007).

At the onset of inflammation, there is an up-regulation of the pro-inflammatory cytokines TNF- $\alpha$ , IL-1 $\beta$  and IL-6 (Dinarello, 1997b, Cavaillon, 1994). At the site of injury or infection the inflammatory response is initiated by TNF- $\alpha$  and IL-1 $\beta$ , released by resident macrophages (Dinarello, 1997b, Tidball, 1995). These cytokines then stimulate the synthesis of IL-6, typically by the local endothelium (Smith et al., 2000). In addition to the up-regulation and amplification of acute inflammation by pro-inflammatory cytokines, a number of anti-inflammatory cytokines play a crucial role in the containment and resolution of this process, such as the IL-1 receptor antagonist (IL-1ra) and IL-10 (Dinarello, 1997b, Petersen and Pedersen, 2005).

The production of anti-/pro-inflammatory cytokines is up-regulated rapidly in response to strenuous physical activity and other forms of stress (Shephard, 2002, Peake et al., 2005c, Ostrowski et al., 1999). During and following a bout of strenuous exercise, the normal pattern

of cytokine release is thought to include a well-ordered release of pro-inflammatory, antiinflammatory cytokines and cytokine inhibitors, with IL-6 playing a dominant role. During intense activity, TNF- $\alpha$  is typically secreted first, followed by IL-1, IL-6, TNF receptors and IL-10 (Drenth et al., 1995, Pedersen et al., 1998). The magnitude of the acute cytokine response to exercise appears to be influenced by both the duration and the intensity of the activity. Other important variables are the mode of exercise, the fitness of the individual and the progressive increase in core temperature and environment (Shephard, 2002).

#### 2.5.1.1 Interleukin-6

Interleukin-6 is released from working muscle into the circulation, where it can exert its effect in other organs in a hormone-like fashion (Nielsen and Pedersen, 2007). It increases after exercise as a function of exercise intensity and duration (Pedersen and Febbraio, 2005, Pedersen et al., 2003, Nielsen and Pedersen, 2007, Febbraio and Pedersen, 2002). The roles of IL-6 during and after exercise include regulation of carbohydrate utilization, stimulation of the restoration of damaged or depleted muscle proteins, mobilization and activation of neutrophils, and suppression of further muscle damage (Shephard, 2002). Interleukin-6 stimulates the production of the anti-inflammatory cytokines such as IL-10 (Petersen and Pedersen, 2005) and suppresses the pro-inflammatory cytokines TNF- $\alpha$  and IL-1 production in humans (Smith, 2000). Although some production of IL-6 occurs in peripheral blood mononuclear cells, exercising muscle appears to be the main source (Petersen and Pedersen, 2005), therefore it would be expected that during strenuous team sport competition, such as AF, [IL-6] would increase.

#### 2.5.1.2 Interleukin-10

Interleukin 10 plays an important role in containing and resolving the inflammatory process via suppression of pro-inflammatory cytokines (Petersen and Pedersen, 2005). Secretion of IL-10 is mediated by several cytokines including IL-6 (Cyktor and Turner, 2011) and is considered to

be one of the most important cytokines with anti-inflammatory properties (Sabat et al., 2010). The action of IL-10 leads to inhibition of secretion of inflammatory cytokines, including TNF and IL-1, therefore, after the generation of a pro-inflammatory immune response, IL-10 serves to dampen inflammation and help eliminate inflammatory responses that could be deleterious to the host, limiting potential tissue damage (Cyktor and Turner, 2011, Moore et al., 2001, Dinarello, 2000). In athletes, muscle damage leads to a loss of muscle function in the days post-exercise (see section 2.4.4.3), therefore the potential role of IL-10 to limit tissue damage may help reduce the time taken to recover from damaging exercise and should benefit performance.

#### 2.5.1.3 Interleukin-1 and tumor necrosis factor

During the inflammatory process, IL-1 and TNF are produced and function primarily as proinflammatory cytokines, as do their derivatives IL-1 $\beta$  and TNF- $\alpha$  (Dinarello, 1997b, Dinarello, 2005). Biologically, IL-1 and TNF are closely related and as such they act synergistically and are produced at sites of local inflammation (Dinarello, 2000, Dinarello, 1997a). Interleukin-1 and TNF initiate a cascade of inflammatory mediators by targeting the endothelium which can ultimately lead to tissue destruction and loss of function (Dinarello, 2000).

Tumor necrosis factor is a potent multifunctional pro-inflammatory cytokine that has a role in immunity, inflammation, control of cell proliferation, differentiation, and apoptosis lipid metabolism, and coagulation (Wallach et al., 1999). Tumor necrosis factor-alpha promotes the inflammatory response and plays an important role in various aspects of the immune-response regulation and tissue injury (Stubgen, 2011). It is produced mainly by activated macrophages, but also by monocytes, neutrophils and endothelial cells (Stubgen, 2011). Production of TNF- $\alpha$  is stimulated by IL-1, but can also be inhibited by IL-6 (Stubgen, 2011). Tumor necrosis factor-alpha has been associated with the pathogenesis of several diseases, including rheumatoid arthritis, Crohn's disease, atherosclerosis, psoriasis, sepsis, diabetes and obesity (Parameswaran and Patial, 2010). Tumor necrosis factor-alpha plays a pivotal role in coordinating the cytokine

cascade in many inflammatory diseases and has been viewed as a "master-regulator" of inflammatory cytokine production (Parameswaran and Patial, 2010).

Interleukin-1 is a highly inflammatory molecule whose primary source is from macrophages and is a major mediator of the acute phase response (Dinarello, 1988, Schweizer et al., 1988). Interleukin-1 mediates a coordinated set of host adaptations that develop in the first few hours or days following infection or injury (Cannon et al., 1986). Interleukin-1 activates lymphocytes and plays an important role in the initiation of the immune response as well as affecting mesenchymal tissue remodelling through its contribution to the destructive and repair process (Dinarello, 1988). Interleukin-1 and IL-1 $\beta$  are involved in enhancing pain sensitivity or lowering one's pain threshold (Dinarello, 2000, Schweizer et al., 1988). Increased levels of both/either cytokine increase prostaglandin production (Schweizer et al., 1988). Prostaglandin (PG)-E<sub>2</sub> is associated with enhanced pain sensitivity, and increased levels of circulating IL-1 $\beta$ cause a tenfold increase in pain (Schweizer et al., 1988). This increase was accompanied by enhanced pain reflexes. Therefore, in addition to mediating the inflammatory and acute phase response, IL-1 and IL-1 $\beta$  also play a role in peripheral pain sensation.

During an acute phase inflammatory response, both pro- and anti-inflammatory cytokines are released (Dinarello, 2000). Pro-inflammatory cytokines increase the inflammatory response and contribute to the destruction of tissue in preparation for tissue remodelling. Anti-inflammatory cytokines serve to restrict the production of pro-inflammatory cytokines which helps to dampen inflammation and ultimately eradicate the inflammatory responses. Muscle damage can occur as a result of team sport participation (see section 2.4.4.3) and the section below will review the role of cytokines following muscle damage and their response to damage caused by exercise and team sport exercise.

# 2.5.2 The role of inflammation following muscle damage

Following exercise or team sport participation, muscle can become damaged. This mechanical damage is commonly referred to as exercise-induced muscle damage (EIMD) (Byrne et al.,

2004, Clarkson and Sayers, 1999, Howatson and Van Someren, 2008). This insult may initiate a generalized acute inflammatory response in response to the damage, which involves the release of various cytokines responsible for the initiation and moderation of the inflammatory response (Malm, 2001, Cannon and St Pierre, 1998, Montgomery et al., 2008a).

The severity and extent of inflammation depends on the type, duration and intensity of exercise (Malm, 2001). Eccentric exercise produces more muscle damage than concentric exercise (Clarkson and Sayers, 1999) and as such, exercise with eccentric contractions, such as those experienced during AF participation, will cause more damage and inflammation than concentric exercise of equal intensity and duration (Malm, 2001).

The major value of the inflammatory response is the removal of cellular debris or necrotic tissue and the subsequent promotion of tissue repair, muscle regeneration and adaptation (Cannon and St Pierre, 1998, Clarkson and Hubal, 2002, Clarkson and Sayers, 1999, Tidball, 2005). The inflammatory response is characterised by an increase in fluid transfer to the damaged area, an infiltration of plasma proteins, attraction and migration of blood cells to the injured area and stimulating various cell types leading to the production of inflammatory mediators (Cannon and St Pierre, 1998, Clarkson and Hubal, 2002, Clarkson and Sayers, 1999). Muscle trauma such as that occurring in collision sports results in activation of circulating monocytes, which, in turn, produce large quantities of pro-inflammatory cytokines IL-1, IL-6, and TNF- $\alpha$  (Smith, 2000). These cytokines play a major role in the acute phase response and help to augment inflammation, activate macrophages and increase the expression of adhesion molecules (Shephard and Shek, 1998). Interleukin-1, IL-6, and TNF- $\alpha$  also play a role in the degradation of muscle protein, resulting in amino-acid release into the circulation and help to stimulate hepatocytes to absorb these amino acids to synthesize new acute-phase proteins (Ramadori and Christ, 1999).

Interleukin-1 $\beta$  and TNF- $\alpha$  play a role in initiating the breakdown of damaged muscle tissue and are secreted at the onset of inflammation where one of their functions is to activate endothelial cells of local blood vessels (Smith, 2000, Peake et al., 2005a). Adhesion molecules are then

expressed by endothelial cells, and are critical in the influx of neutrophils to the injured site, which in turn starts the process of cell destruction (Cannon and St Pierre, 1998). Interleukin-1 and TNF help with the up-regulation of the adhesion molecules and IL-1 $\beta$  and TNF- $\alpha$  help to stimulate the production of IL-6 (Smith, 2000, Petersen and Pedersen, 2005). Although associated as part of the acute phase response, IL-6 however does not directly induce inflammation (Pedersen and Hoffman-Goetz, 2000) but actually helps to stimulate the production of anti-inflammatory cytokines such as IL-10 and helps to suppress further muscle damage (Shephard, 2002, Petersen and Pedersen, 2005). The magnitude of IL-6 release is related to the degree of muscle injury but more closely related to exercise intensity (Smith, 2000, Petersen and Pedersen, 2005).

During the repair phase of an injury, satellite cells are released and begin to proliferate (Seale et al., 2003). Satellite cells are mononucleated stem cells that undergo asymmetric divisions leading to the formation of undifferentiated cells (Pallafacchina et al., 2012). The activation and subsequent proliferation of satellite cells is a necessary step contributing to the growth and formation of new myofibres (Wang and Rudnicki, 2012, Pallafacchina et al., 2012). Satellite cells therefore play an important role in the growth, maintenance and regeneration of skeletal muscle. Interleukin-6 has been identified as an essential and critical regulator of satellite cell proliferation in skeletal muscle (Serrano et al., 2008) and when uninhibited, the release of muscle produced IL-6 assists in myonuclear accretion leading to muscle growth and repair (Serrano et al., 2008). However, a reduction or deficiency in IL-6 can result in reduced satellite cell-derived myoblast proliferation and therefore muscle repair and growth may be blunted (Serrano et al., 2008). As a result, a reduction in IL-6 following damaging exercise may inhibit the normal repair process of muscle leading to a longer period of recovery or a reduction in muscle growth.

### 2.5.3 The inflammatory response and exercise

Exercise can stimulate increased plasma levels of multiple cytokines (Pedersen, 2000). The level of circulating IL-6 increases in an exponential fashion (up to 100 fold) in response to exercise (Febbraio and Pedersen, 2002, Pedersen et al., 2001) while the pro-inflammatory cytokines, TNF-a and IL-1b, in general do not increase to the same extent or do not increase at all with exercise (Petersen and Pedersen, 2006, Ostrowski et al., 1998).

Exercise induces increases in [IL-1 $\beta$ ], [TNF- $\alpha$ ] and [IL-6] following acute, prolonged and intense exercise (Abbey and Rankin, 2011, Brenner et al., 1999, Meckel et al., 2009, Ostrowski et al., 1998, Ostrowski et al., 1999, Pournot et al., 2011, Suzuki et al., 2003). Interleukin-6 is produced in larger amounts than any other cytokine in relation to exercise (Figure 2.7) (Pedersen, 2000), and the increased production of this and other anti-inflammatory cytokines during exercise may serve to restrict pro-inflammatory reactions to exercise-induced muscle damage or the magnitude and duration of the inflammatory response after exercise (Toft et al., 2002, Ostrowski et al., 1999). As a result, the relative concentration of both TNF- $\alpha$  and IL-1 are lower than IL-6 following strenuous exercise (Petersen and Pedersen, 2006, Ostrowski et al., 1998).

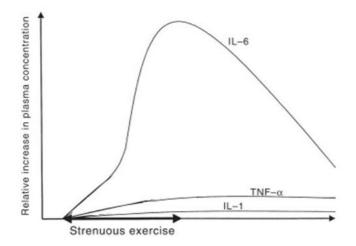


Figure 2.7: Cytokine changes in relation to strenuous exercise. Adapted from (Pedersen, 2000).

### 2.5.4 The inflammatory response to prolonged exercise

Field based team sport matches can last up to 120 min, therefore the inflammatory response following exercise of this duration is of particular interest. Following prolonged exercise, antiinflammatory cytokines increase substantially, however the appearance of pro-inflammatory cytokines is more variable and increases may not always be detected. Evidence of the relatively minor changes in IL-1 $\beta$  and TNF- $\alpha$  following prolonged exercise compared to IL-6 and IL-10 can be seen after a marathon, prolonged treadmill running and prolonged cycling (Brenner et al., 1999, Jenkins et al., 1994, Ostrowski et al., 1998, Ostrowski et al., 1999, Starkie et al., 2001, Suzuki et al., 2003). After a marathon, the concentration of IL-1 $\beta$ , TNF- $\alpha$ , IL-6 and IL-10 all increased (Ostrowski et al., 1999). Immediately post-race, [TNF- $\alpha$ ] and [IL-1 $\beta$ ] increased only by 2·3-fold and 2·1-fold respectively which was the peak concentration for both cytokines. Interleukin-6 and [IL-10] also peaked immediately post exercise at 128-fold and 27fold higher than pre-race. Similarly, after participants completed a full marathon, [IL-6] and [TNF- $\alpha$ ] increased immediately post-race (118-fold and 4.8-fold) (Starkie et al., 2001).

Following 2 h of cycling (between 60-65%  $\dot{V}O_{2max}$ ) [IL-6] increased immediately post exercise (4.3-fold) and after 3 h recovery (5.5-fold), while [TNF- $\alpha$ ] after 3 h was increased by only 1.4-fold and after 24 h was only elevated by 1.35-fold (Brenner et al., 1999). After a marathon, plasma [IL-1 $\beta$ ] remained at baseline levels however both [IL-10] and [IL-6] increased 4.1-fold and 80-fold respectively (Suzuki et al., 2003). Similarly, following 2.5 h of treadmill running no changes in [IL-1 $\beta$ ] and [TNF- $\alpha$ ] were detected while a 35-fold increase in [IL-6] was evident immediately post exercise (Ostrowski et al., 1998).

Increases in pro-inflammatory cytokines are balanced by the release of the anti-inflammatory cytokine IL-10 (Jenkins et al., 1994). This in turn can up-regulate the release of the cytokine inhibitor IL-1ra and cause a down regulation of IL-1 $\beta$  and TNF- $\alpha$  (Drenth et al., 1995, Jenkins et al., 1994). Immediately following a 6 h endurance run (~65 km), IL-6 increased 3.9-fold while IL-1ra increased 4.7-fold, however no change was detected in [IL-1 $\beta$ ] or [TNF- $\alpha$ ]

(Drenth et al., 1995). Similarly, [IL-1 $\beta$ ] and [TNF- $\alpha$ ] were unable to be detected after 1 h of cycling (at 75%  $\dot{V}O_{2max}$ ) while [IL-6] increased ~2-fold (Ullum et al., 1994). Following 60 min of treadmill running (between 55-75%  $\dot{V}O_{2max}$ ), no change was detected in either [IL-1 $\beta$ ] or [TNF- $\alpha$ ] while [IL-6] was increased (5-7.5 fold) both immediately post and 30 min after exercise and [IL-1ra] increased (2-2.5-fold) after 1 and 2 h (Scott et al., 2011).

The findings above suggest that anti-inflammatory cytokines can restrict the magnitude and duration of the inflammatory response to exercise. Prolonged exercise can increase anti-inflammatory cytokines, however [IL-1 $\beta$ ] and [TNF- $\alpha$ ] may remain relatively unchanged. This may also be the case in prolonged team sport competitions such as AF, where matches can last up to 2 h. Although AF is similar in duration to a marathon (i.e. 2-2.5 h), match demands differ greatly. An AF match has frequent breaks in play as well as 3 extended breaks throughout (i.e. <sup>1</sup>/<sub>4</sub> time, <sup>1</sup>/<sub>2</sub> time and <sup>3</sup>/<sub>4</sub> time). Team sport matches have much shorter running bouts than a marathon does and athletes are frequently required to perform a number of sprints over the course of a few minutes during match play, therefore the section below will review the inflammation resulting from shorter, repeat sprint bouts.

### 2.5.5 Inflammatory response after short duration and repeat sprint exercise

Running bouts much shorter than marathons (i.e. 30-250 m or 5 s to 3 min) can also increase circulating inflammatory cytokines (Table 2.5). This type of running, including a recovery period after each bout, is more representative of team sport running. Although increases are evident, it is clear that magnitude of change in cytokine concentration is not as great as that seen in more prolonged running efforts (see section 2.5.3). Also of note is the relatively minor increase in pro-inflammatory cytokines after this type of running (similar to prolonged exercise) and that substantial increases in anti-inflammatory cytokines may restrict increases in pro-inflammatory cytokines.

Exercise type	Participants	Time course and magnitude of post-exercise cytokine change compared to pre-exercise			
Repeat 250-m sprints (4 x 250-m sprint at 80% of 100-m max speed, 3-min recovery between sprints)	20 elite junior handball players	IL-1 $\beta$ (pg.ml <sup>-1</sup> )	0 h post 1 h	133.3% 83.3%	
(Nemet et al., 2009)		IL-6 (pg.ml <sup>-1</sup> )	0 h post 1 h	121.4%* 150.0% *	
		IL-10 (pg.ml <sup>-1</sup> )	0 h post 1 h	100.0% 128.6%	
Repeat 250-m sprints (4 x 250-m sprint at 80% of 100-m max	12 elite junior handball players	IL-1 $\beta$ (pg.ml <sup>-1</sup> )	0 h post	107.7%	
speed, 3-min recovery between sprints) (Meckel et al., 2009)		IL-6 (pg.ml <sup>-1</sup> )	0 h post 1 h	153.8%* 169.2% *	
		IL-10 (pg.ml <sup>-1</sup> )	0 h post	100.0%	
Repeat 30-m sprints (12 x 30-m sprints every 35-s) (Abbey and Rankin, 2011)	15 healthy, male, team- sport-trained athletes	IL-6 (pg.ml <sup>-1</sup> )	0 h post 1 h	172.0%* 158.0% *	
45-min simulated trail run (5 x 3-min flat + 3-min uphill + 3min	11 well trained runners	IL-1 $\beta$ (pg.ml <sup>-1</sup> )	0 h post	138.0% *	
downhill running) (Pournot et al., 2011)		IL-6 (pg.ml <sup>-1</sup> )	0 h post	1600.0%*	
		IL-10 (pg.ml <sup>-1</sup> )	0 h post	700.0%*	

Table 2.5: Time course and magnitude of cytokine change after repeat short running bouts. \*P<0.05

### 2.5.6 Inflammation resulting from team sport or simulated team sport exercise

Along with running bouts, changes in both pro and anti-inflammatory cytokines can occur as a result of team sport and simulated team sport participation with marked increases in both [IL-6] and [IL-10] evident immediately post-match and up to 1 h post (Table 2.6). However, similar to both prolonged and shorter bout exercise, [IL-1 $\beta$ ] and [TNF- $\alpha$ ] are more variable with several studies indicating no substantial increase.

Exercise type	Participants	Time course and magnitude of post-exercise cytokine change compared to pre-exercise			
Soccer match	24 male soccer	IL-6 (pg.ml <sup>-1</sup> )	0 h post	600% *	
(Ispirlidis et al., 2008)	players		24 h	110%	
3 day, 3 game basketball	29 male basketballers	IL-6 (pg.ml <sup>-1</sup> )	Game 1 - 0 h post	450% *	
tournament			Game 2 - 0 h post	400% *	
(Montgomery et al., 2008a)			Game 3 - 0 h post	375% *	
		IL-10 (pg.ml <sup>-1</sup> )	Game 1 - 0 h post	300% *	
			Game 2 - 0 h post	250% *	
			Game 3 - 0 h post	300% *	
Soccer match	22 elite female soccer	IL-6 (pg.ml <sup>-1</sup> )	0 h post	181% *	
(Andersson et al., 2010)	players		21 h	119%	
		IL-10 (pg.ml <sup>-1</sup> )	0 h post	123% *	
			21 h	115%	
		IL-1 $\beta$ (pg.ml <sup>-1</sup> )	0 h post	96%	
		140	21 h	117%	
		TNF- $\alpha$ (pg.ml <sup>-1</sup> )	0 h post	277% *	
			21 h	155%	
Handball match	14 elite male	IL-6 (pg.ml <sup>-1</sup> )	0 h post	170% *	
(Marin et al., 2011)	handballers		24 h post	108%	
		TNF- $\alpha$ (pg.ml <sup>-1</sup> )	0 h post	58%	
			24 h post	46%	
Loughborough intermittent shuttle	38 males	IL-6 (pg.ml <sup>-1</sup> )	0 h post	594% *	
test (90 min simulated soccer			1 h post	388% *	
running)			24 h post	131%	
(Bailey et al., 2011)			48 h post	113%	
		TNF- $\alpha$ (pg.ml <sup>-1</sup> )	0 h post	107%	
			1 h post	107%	
			24 h post	100%	
			48 h post	100%	

## Table 2.6: Time course and magnitude of cytokine change after team sport or simulated team sport activity. \*P<0.05

In addition to the eccentric damage resulting from locomotion, the physical contact sustained by players during high-impact collision sports matches may elicit a greater inflammatory response. Following an international rugby union game, [IL-6] increased 320.9% immediately post-match before returning to pre exercise level after 14 h (Cunniffe et al., 2010), however this response is not substantially different from other team sport participants (Table 2.6). In AF, players make regular contact with each other whilst tackling, during marking and rucking contests and when competing for ground balls as well as making regular contact with the ground. Although no evidence exists in AF regarding the inflammatory response to match play, based on similar team sport participation, it is highly likely that AF matches would elicit an acute phase response. The effect of AF match play on the inflammatory response is an area which needs addressing.

### 2.5.7 Conclusions

The acute inflammatory response is a critical biological response. It helps to contain, dilute or destroy injurious agents in an attempt to protect the area from further insult or injury (Starkey, 1999a). It stimulates the process of removing necrotic tissue and cellular debris and helps with the subsequent promotion of tissue repair (Cannon and St Pierre, 1998, Clarkson and Hubal, 2002, Clarkson and Sayers, 1999). It is clear that muscle damage and exercise can increase the levels circulating cytokines and that these elevated levels can persist over several days. It is also apparent that anti-inflammatory cytokines can increase substantially after prolonged and short running bouts as well as team sport activities, while pro-inflammatory cytokines appear to be far more variable. Recovery from heavy exercise bouts for athletes training and competing regularly can become important. If left unresolved, an acute inflammatory response can become a chronic issue and even a systemic issue, therefore adequate recovery from such exercise should be an important part of an overall exercise plan. Failure by athletes to effectively recover from the demands of their chosen sport can lead to a state of overreaching, which has been suggested to occur more in team sports and explosive/power sports, which includes AF (Halson and Jeukendrup, 2004).

### 2.6 The effectiveness of post-exercise recovery

Recovery has been defined as the return of a muscle to its pre-exercise state following exercise (Tomlin and Wenger, 2001). It is important for athletes wanting to improve or maximise performance that they achieve an adequate balance between the stresses of training and competition and recovery from these stresses. Adequate recovery from sports participation allows athletes to compete during subsequent games/competition without residual soreness or

fatigue, thus potentially minimising the athlete's chance of injury (Hing et al., 2008). For team sport athletes competing weekly, like AF, the ability to reduce the chance of injury as well as soreness and fatigue can only be of benefit during a long and demanding season.

A number of strategies are utilised by athletes, coaches, medical staff and trainers to enhance the post exercise recovery process (Wilcock et al., 2006b). These include massage, active recovery, relaxation, compression garments, stretching, and anti-inflammatory drugs (Barnett, 2006, Hemmings et al., 2000) however conclusive evidence is lacking as to the overall effectiveness of these modalities (Barnett, 2006). Another frequently used recovery modality is water immersion (WI) and this strategy is regularly employed in athletic settings (Ascensao et al., 2011, Dawson et al., 2005, Halson, 2011, Hing et al., 2008, Ingram et al., 2009, Montgomery et al., 2008b, Rowsell et al., 2009, Rowsell et al., 2011, Vaile et al., 2008b, Vaile et al., 2008a).

Typically, WI involves an athlete being immersed in water to hip level or mid sternal level (Ingram et al., 2009, Rowsell et al., 2009, Vaile et al., 2007, Vaile et al., 2008a). There are four major methods of WI utilised: (1) contrast water therapy (CWT) involves alternating between hot and cold water, (2) cold water immersion (CWI) uses cold water only, (3) thermotherapy, which uses warm water and (4) thermoneutral immersion (TWI), which involves using water temperatures between 34-35°C (Halson, 2011, Wilcock et al., 2006a).

Immersion in water may produce a number of physiological changes. These can include changes such as fluid shifts from high to low pressure areas, pain control, vasoconstriction and reductions in blood flow, oedema and swelling, which are addressed in the sections below (Algafly and George, 2007, Isii et al., 2007, Nadler et al., 2004, Peiffer et al., 2009, Skiveren et al., 2008, Vaile et al., 2008c, Vaile et al., 2010, Wilcock et al., 2006a, Yanagisawa et al., 2003). It has been theorised that these changes could improve recovery post exercise and these changes have been attributed principally to the effects of hydrostatic pressure and temperature (Wilcock et al., 2006a).

### 2.6.1 Mechanisms of water immersion during recovery

When a body is immersed, hydrostatic pressure is exerted on the body (Wilcock et al., 2006a). The magnitude of this pressure is increased with the increased depth of submersion and is described by the equation:  $P = P_{atm} + g \bullet \rho \bullet h$ 

Where P = water pressure,  $P_{atm}$  = atmospheric pressure (standard sea level 1013 hPa), g = gravity (9.81m.s<sup>-2</sup>),  $\rho$  = water density (1000 kg.m<sup>-3</sup>) and h = water height (m<sup>3</sup>) (Wilcock et al., 2006a).

This proportional change in pressure with depth, causes an upward squeeze on the body which at a depth of 1 m is 74 mmHg and as the depth of immersion increases, central venous pressure increases along with it (Figure 2.8) (Risch et al., 1978, Wilcock et al., 2006a). When external pressure on the body increases, fluids and gases are displaced from high pressure to low pressure areas (Lollgen et al., 1981). Fluid from the extravascular space moves into the vascular compartment and reduces exercise-induced increases in muscle volume (Wilcock et al., 2006a).

Increased external pressure causes shifting of fluids from the lower limbs into the thoracic region, as indicated by increases in stroke volume and overall blood flow (Lollgen et al., 1981, Risch et al., 1978). These increases are proportional to the depth of immersion with greater changes seen as the depth of immersion increases (Figure 2.8 and Figure 2.9) (Lollgen et al., 1981, Risch et al., 1978).

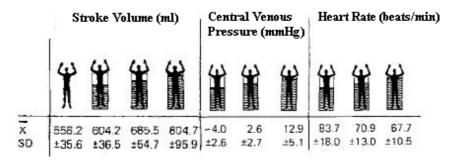


Figure 2.8: Increase in stroke volume, central venous pressure and decrease in heart rate with increasing immersion depth. Adapted from (Risch et al., 1978).

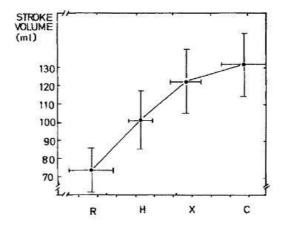


Figure 2.9: Increase in stroke volume with increasing immersion depth. R = rest (no immersion), H = immersion to hip level, X = immersion to xiphoid, C = immersion to chin level. Adapted from (Lollgen et al., 1981).

An important role for hydrostatic pressure in the recovery process is in the reduction of oedema (Friden and Lieber, 2001). Typically, swelling and loss of motion and function follow after exercise-induced muscle damage (Howell et al., 1993). Water immersion can assist the recovery process by increasing the pressure gradient between the interstitial compartment of the legs and the intravascular space (Wilcock et al., 2006a). Subsequently, the space available for swelling to accumulate in tissue is reduced and can decrease the extent of swelling typically associated with inflammation (Kraemer et al., 2004). An increase in the gradient between capillary filtration pressure and internal tissue pressure can also improve the re-absorption of interstitial fluids and further reduce oedema (Friden and Lieber, 2001).

The formation of oedema can negatively impact recovery and increase recovery times (Prentice, 1999). Oedema, formed during and after muscle damaging exercise, can reduce the amount of oxygen delivered to local cells by increasing the transport route of localised capillaries as a result of increased internal compression (Wilcock et al., 2006a). This lack of oxygen can cause cell damage (Friden and Lieber, 2001). Oedema can also cause an increase in infiltration of leukocytes and monocytes, as well as increased circulating inflammatory cells (Cesari et al., 2004, Mishra et al., 1995). These cells can cause further tissue degradation or secondary muscle damage (Mishra et al., 1995, Cesari et al., 2004). Hydrostatic pressure can promote the clearance of inflammatory mediators from a damaged or injured area (Kraemer et

al., 2004) and by reducing oedema and reducing the inflammatory process, secondary muscle damage is likely to be reduced (Sayers et al., 2000a). This can enhance an athlete's ability to recover from muscle damaging exercise (Sayers et al., 2000a) and can promote the physiological actions of healing (Kraemer et al., 2004).

Water immersion can benefit the recovery of athletes post-exercise (Gill et al., 2006, Hamlin, 2007, Vaile et al., 2008b, Vaile et al., 2008a, Wilcock et al., 2006b, Ascensao et al., 2011, Dawson et al., 2005, Halson, 2011, Hing et al., 2008, Ingram et al., 2009, Montgomery et al., 2008b, Rowsell et al., 2009, Rowsell et al., 2011). Ten to 20 minutes of both CWI and CWT can benefit athletes who have undertaken intense exercise sessions causing prolonged muscle fatigue and muscle damage, particularly in athletes competing week to week (Halson, 2011, Wilcock et al., 2006b).

### 2.7 Cold water immersion (CWI)

Cryotherapy is a term used to describe the application of cold modalities and is the local or general use of low temperatures for the removal of heat from a body part or in medical therapy (Prentice, 1999, Starkey, 1999). Cryotherapy is widely used to treat acute and sport-related injuries (Bleakley et al., 2004, Starkey, 1999) and CWI is a form of cryotherapy where a body part is submerged into a mixture of ice and water or cooled water for a set period of time (Starkey, 1999).

### 2.7.1 Physiological effects of cold application

It has been proposed that by decreasing tissue temperature, the application of ice/cold can diminish muscle spasm and minimise swelling inflammation, thereby aiding recovery after soft-tissue trauma (Knight, 1995, Nadler et al., 2004, Prentice, 1999, Tsang et al., 1997). In response to tissue injury, specialised nerve endings called nociceptors are activated (Nadler et al., 2004). The sensation of pain is recognised as nociceptors transmit nerve signals that travel via the spinal cord to the brain. At the same time, increased muscle motor activity at the

site of injury lead to a reflexive muscle contraction due to spinal reflex initiated by neurotransmitters (Nadler et al., 2004). If persistent, this increase in muscle tone can cause painful muscle spasms, which can lead to further tissue damage and in turn, increase pain further. This process is called the pain-spasm-pain cycle (Figure 2.10) (Nadler et al., 2004). The use of cryotherapy can interrupt this cycle to prevent further tissue injury and to reduce the sensation of pain however it is not known whether this phenomenon occurs in team sport athletes or in team sport situations.

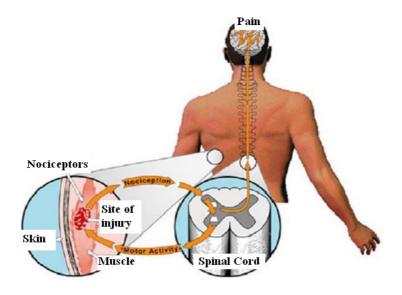


Figure 2.10: Pain-spasm-pain cycle (Nadler et al., 2004).

Cryotherapy produces a 3 to 4 stage sensation (Prentice, 1999). The first stage is the sensation of cold which is closely followed by a stinging sensation then a burning or aching sensation and finally a numbness or analgesic sensation. This can occur over a 5-15 min period (Prentice, 1999). The first 3 stages (cold, stinging, burning/aching) occur during the initial stages of CWI/cryotherapy particularly during very cold temperatures (i.e. 5°C or lower) and are caused by temporary irritation to nerve endings in the skin (Kowal, 1983, Prentice, 1999, Starkey, 1999, Knight et al., 1981). During CWI, if the water temperature is too low and causes pain and discomfort, it is unlikely to be tolerated and its use is likely to be discontinued. In athletic populations, a temperature range of between 10-15°C has been recommended to ensure that CWI protocols are well tolerated (Halson, 2011).

The use of cryotherapy can decrease muscle temperature to between 22-28°C (Figure 2.11) (Beelen and Sargeant, 1991, de Ruiter et al., 1999, Sargeant, 1987, Markovic et al., 2004) and skin temperature to between 9.2-10.2°C (Isii et al., 2007, Kanlayanaphotporn and Janwantanakul, 2005).

# Mean Temperature Change Over Time

Figure 2.11: Intramuscular temperature reduction resulting from cryotherapy over 20 min. Adapted from (Markovic et al., 2004)

As the temperature of skin and muscle decreases, cold can reduce the rate of nerve conduction velocity (Algafly and George, 2007). Substantial decreases in tibial nerve conduction velocity occurred after submersion in ice water when compared to a non-immersed ankle (Figure 2.12) (Algafly and George, 2007). Such reductions in nerve conduction velocity can interrupt the cycle of pain (Sauls, 1999). Additionally, the slower neural firing rate can decrease motor activity by 30% (Algafly and George, 2007) which can lead to a reduction in muscle tone and contribute to reducing muscle spasm (Sauls, 1999).

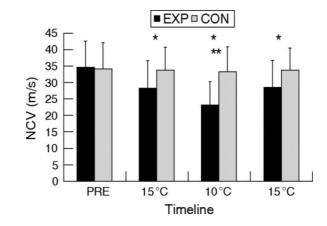


Figure 2.12: Influence of cold application on nerve conduction velocity (NCV) at different skin temperatures. Adapted from (Algafly and George, 2007). EXP = experimental/cooled ankle, CON = control ankle. \* Significant difference between EXP and CON (p<0.05), \*\* Significant difference between 15°C and 10°C (p<0.05).

Cold application is also known to produce an analgesic effect as well as increase pain tolerance and pain threshold (Algafly and George, 2007, Bugaj, 1975, Skiveren et al., 2008). Following the use of 10 min of ice massage, analgesia was elicited only after the localized region had been cooled to, and maintained below 13.6°C (Bugaj, 1975). Analgesia following 5 min of cold application (gel pack) to the axilla has also been reported (Skiveren et al., 2008). Cold application also increased pain threshold and pain tolerance (Algafly and George, 2007). Following immersion in ice water until skin temperature reached 15°C or 10°C, pain threshold and tolerance in the tibial nerve increased substantially (Figure 2.13). The increase in pain tolerance and threshold was associated with a 33% drop in nerve conduction velocity (Nadler et al., 2004, Swenson et al., 1996). For athletes using CWI post-exercise, analgesia can help to reduce perceptions of muscle soreness (see section 2.5.5).

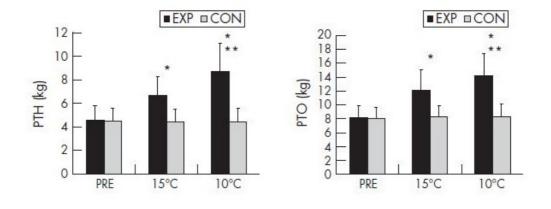


Figure 2.13: Influence of cold application on pain threshold (PTH) and pain tolerance (PTO) at different skin temperatures. Adapted from (Algafly and George, 2007). EXP = experimental/cooled ankle, CON = control ankle. \* Significant difference between EXP and CON (p<0.05), \*\* Significant difference between  $15^{\circ}$ C and  $10^{\circ}$ C (p<0.05).

Cold water immersion also reduced blood flow through peripheral vasoconstriction (Peiffer et al., 2009). After performing 90 min of cycling followed by a 16.1 km time trial in the heat on two separate occasions (32.3°C and 55.5% relative humidity), the effects of 20 min of CWI (14.3°C) on femoral venous vessel diameter was measured in 10 cyclists (Peiffer et al., 2009). When compared to the control condition, CWI decreased femoral vessel diameter ~12% both 45 and 90 min post-exercise. This decrease reduced blood flow by ~24%. Blood flow to the arms and legs was also reduced following 15 min of CWI (15°C) (Vaile et al., 2010). After completing 35 min of cycling, participants undertook an active recovery (15 min of cycling at 40% peak power output) or 15 min of CWI. This was followed by a further 40 min of passive rest prior to completing another 35 min of cycling. After CWI, blood flow to the arms and legs was substantially reduced when compared to that seen in the active recovery group (Figure 2.14).

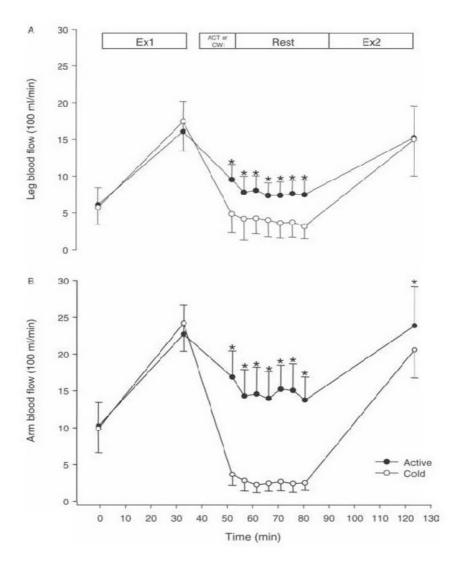


Figure 2.14: A) Leg blood flow and B) Arm blood flow following a cycling performance trial (Ex1), a 15-min period of either active recovery (ACT) or cold water immersion recovery (CWI) followed by a 40-min rest period, and a second cycle performance trial (Ex2). Adapted from (Vaile et al., 2010). \*P<0.05

The application of CWI leads to vasoconstriction, reduced blood flow and an improved ability to re-absorb interstitial fluids as a result of hydrostatic pressure. Together, these physiological outcomes can act synergistically to reduce oedema and localised swelling (Yanagisawa et al., 2003, Vaile et al., 2008c). Following 14 min of CWI ( $15^{\circ}$ C), the degree of post-exercise swelling after an eccentric leg exercise protocol (5 x 10 eccentric leg press) was substantially reduced after 24 (0.85%), 48 (0.90%) and 72 h (0.60%) when compared to a control (Figure 2.15) (Vaile et al., 2008c). Similarly, following a muscle damaging plantar flexion protocol (5 x 12 concentric/eccentric contractions), 15 min of CWI ( $5^{\circ}$ C) substantially reduced muscle

oedema 48 h post-exercise (10.5%) when compared to controls (Yanagisawa et al., 2003). Reduced oedema may also have a positive effect on reducing perception of pain as the pressure on pain receptors caused by oedema is reduced (Wilcock et al., 2006a).

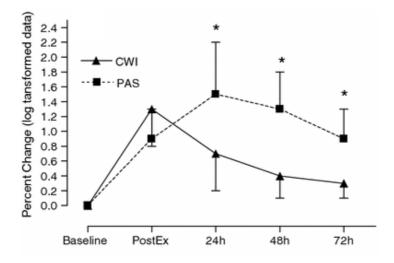


Figure 2.15: Percentage change in post-exercise swelling of the thigh after muscle damaging exercise (Vaile et al., 2008c). CWI = cold water immersion, PAS = passive recovery. \*P<0.05.

The use of CWI also improved thermal comfort as well as decreased body temperature (Vaile et al., 2008a). When compared to an active recovery, 4 differing CWI protocols (5 x 1 min immersion with 2 min rest at 10°C, 15°C and 20°C and a continuous exposure 1 x 15 min exposure to 20°C) substantially reduced mean body temperature between 2 cycling bouts separated by 55 min (Figure 2.16) (Vaile et al., 2008a). These CWI protocols also reduced the thermal sensation (Figure 2.17) (Vaile et al., 2008a). The decrease in both led to all CWI groups performing substantially more work during the second cycling bout compared to the active recovery (10°C 99.4%, 15°C 100.5%, 20°C 99.2%, 20°C+ 99.3% and active recovery 95.9%).

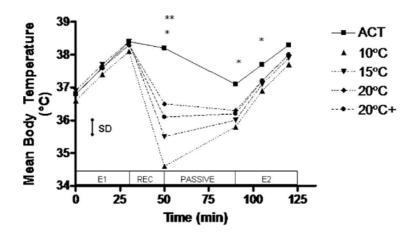


Figure 2.16: Effect of water temperature on change in mean body temperature (°C) during a 15 min recovery (REC) after completing a bout of cycling (E1) and prior to completing a second bout of cycling (E2). Adapted from (Vaile et al., 2008a). ACT = active recovery (no immersion in water). \* Significant difference between all 4 recovery interventions (p<0.05), \*\*Significant difference between all vater based recovery interventions and active recovery

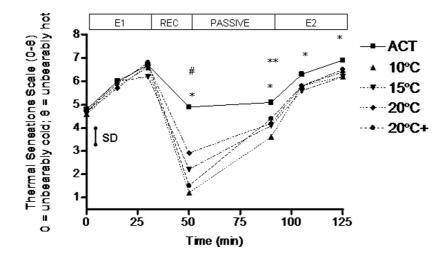


Figure 2.17: Effect of water temperature on change in thermal sensation during a 15 min recovery (REC) after completing a bout of cycling (E1) and prior to completing a second bout of cycling (E2) (Vaile et al., 2008a). ACT = active recovery (no immersion in water). \* Significant difference between ACT and all four CWI treatments (p<0.05), \*\*Significant difference between CWI protocols 10°C vs. 15°C, 10C vs. 20°C, 10°C vs. 20°C+, and 15°C vs. 20°C (p<0.01), #Significant difference between CWI protocols 10°C vs. 15°C, 10C vs. 15°C, 10C vs. 20°C, and 20°C vs. 20°C+(p<0.05).

Cold water immersion can have positive effects on the perception of pain, thermal comfort and oedema reduction. It can also have positive benefits on reducing perceived muscle soreness and fatigue (see sections 2.10 and 2.11) (Bailey et al., 2007, Connolly et al., 2005, Hubbard and Denegar, 2004, Ingram et al., 2009, Montgomery et al., 2008b, Rowsell et al., 2009, Rowsell et

al., 2011). These effects may enable athletes to return to exercise sooner than those not undertaking CWI and may also play a role in enhancing the recovery of physical performance.

### 2.8 Contrast Water Therapy (CWT)

### 2.8.1 Physiological effects of heat application

The application of superficial heat can have a number of physiological effects including increased subcutaneous and muscle temperatures (Myrer et al., 1994, Myrer et al., 1997), increased heart rate and cardiac output and decreased total peripheral resistance (Bonde-Petersen et al., 1992). These effects can subsequently increase short term blood flow (Song, 1984) due to peripheral vasodilation (Cochrane, 2004, Wilson et al., 2002). Following 5 min of heat application (hot-pack) to the calf, both muscle (0.74°C) and subcutaneous (8.13°C) temperatures increased substantially (Myrer et al., 1997). Similarly, muscle temperature increased substantially (2.83°C) throughout 20 min of immersion in hot water (40°C) (Myrer et al., 1994). Such increases can lead to increased skin and muscle blood flow (Figure 2.18) (Song, 1984) and are likely to occur due to an increase in vasodilation. Following a bout of either whole-body or local heating, cutaneous vascular conductance increased substantially in both groups (whole-body 380%, local 361%) with the increase determined to be evidence of a vasodilation response (Wilson et al., 2002). This response is thought to occur as a result of the increase in local temperature decreasing sympathetic nerve drive and increasing vessel diameter (Cochrane, 2004).

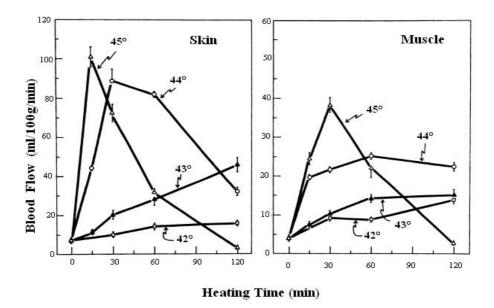


Figure 2.18: Change in leg skin and muscle blood flow resulting from exposure to different hot temperatures. Adapted from (Song, 1984).

Heart rate and cardiac output increased while total peripheral resistance decreased as a result of 15-20 min immersion in 43.8°C water (Figure 2.19) (Bonde-Petersen et al., 1992). The increase in blood flow is thought increase oxygen delivery which may assist in tissue repair and recovery (Cote et al., 1988, Wilcock et al., 2006a).

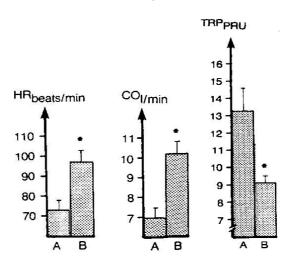


Figure 2.19: Change in heart rate (HR), cardiac output (CO) and total peripheral resistance per 100g of tissue (TRP<sub>PRU</sub>) resulting from passive rest (A) and hot water immersion (B). Adapted from (Bonde-Petersen et al., 1992). \* P<0.05

Several contraindications exist with the use of hot water. High temperatures can lead to skin burns (Prentice, 1999) while compared to cold, heat can increase swelling and the inflammatory response (Figure 2.20) (Cote et al., 1988). In turn, this may increase recovery time from damaging exercise. For AF players or other team sport athletes experiencing high physical contact or high eccentric loads causing damage, an increase in swelling and recovery time would not be desirable and may hamper match/training preparation.

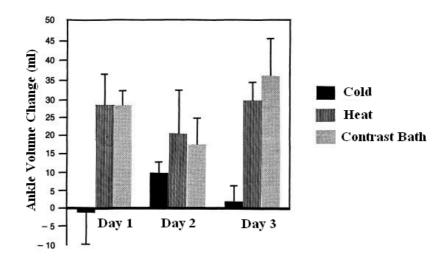


Figure 2.20: Change in ankle volume (swelling) over 3 days of treatment after cold, heat and contrast bathing (Cote et al., 1988).

### 2.8.2 Physiological effects of contrast water therapy

During CWT, athletes alternate between hot and cold water immersion at regular intervals (i.e. 3:1 ratio of hot to cold) (Vaile et al., 2007). It is believed that CWT provides some of the benefits of both cold and hot immersion, including temperature changes which promote vasodilation and vasoconstriction, leading to a 'vaso-pumping' action and an increase in blood flow. It is theorised that this pumping action improves venous return and can potentially reduce the oedema/swelling associated with injury (Fiscus et al., 2005, Prentice, 1999, Starkey, 1999). The effect of CWT on blood flow has been examined (Fiscus et al., 2005). Following a 20 min CWT protocol (4 min at 40°C alternating with 1 min at 13°C) lower leg blood flow showed substantial fluctuations compared to a control group. The increases were attributed to a substantial increase in blood flow (Figure 2.21). Although the 4:1 ratio indicated substantial blood flow changes resulting from water temperature changes, a ratio of 1:1 or 2:1 hot/cold is much

more common than the 4:1 ratio used by the authors, particularly in a team sport setting (Dawson et al., 1997, Higgins et al., 2011, King and Duffield, 2009, Kinugasa and Kilding, 2009).

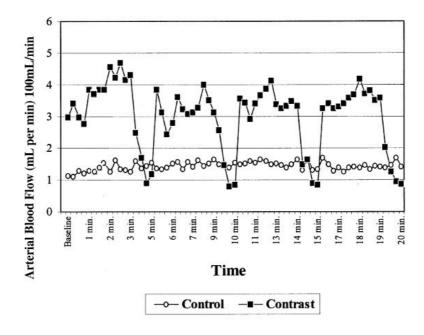


Figure 2.21: Change in arterial blood flow during contrast water immersion (Fiscus et al., 2005). Note that baseline indicates the point when blood flow measurements began.

In order for vaso-pumping to occur, muscle temperature and not just skin temperature needs to change (Wilcock et al., 2006a). The effects of CWT on increasing/decreasing muscle temperature has been investigated (Myrer et al., 1994). Muscle temperature following either 20 min of CWT (4 x 4 min hot at 40.6°C followed by 1 min cold at 15.6°C) or hot water immersion (20 min at 40.6°C) was measured. Results indicate that hot water immersion substantially increased baseline muscle temperature, however, during CWT, muscle temperature failed to increase (Figure 2.22) (Myrer et al., 1994). The effects of heat application may be counteracted by the application of cold (i.e. lowering muscle temperature) therefore muscle temperature during CWT may remain unchanged.

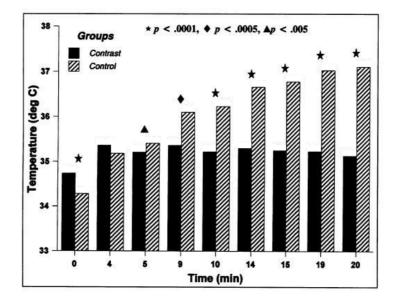


Figure 2.22: Change in muscle temperature resulting from contrast water immersion. Adapted from (Myrer et al., 1994).

As the exposure to hot temperatures during typical team sport application is limited to a 1:1 or 2:1 ratio it is possible that the substantial increases in blood flow demonstrated by Fiscus *et al.*, 2005 will not be seen. Vaso-pumping occurs at slow rates (Wilcock et al., 2006a), and as team sport participants typically alter between hot and cold water 2-6 times, pumping may occur only 2-6 times over the total duration of a recovery intervention (Dawson et al., 1997, Higgins et al., 2011, King and Duffield, 2009, Kinugasa and Kilding, 2009). Muscle temperatures do not change greatly during CWT and if athletes spend limited time in hot water, it seems probable that vaso-pumping would play only a minor role in reducing exercise/contact induced oedema and assisting recovery.

The effects of hydrostatic pressure however may help to explain why CWT may be effective in oedema reduction. Following an eccentric leg press protocol, effectiveness of 15 min CWT (1 min at 8-10°C alternating with 2 min at 40-42°C) on reducing thigh volume over 72 h (Vaile et al., 2007) was compared to a passive recovery. Contrast water therapy produced smaller increases and faster reductions in thigh circumference compared to the passive treatment after 24 (CWT 3.2%, PAS 6.2%), 48 (3.1%, PAS 6.6%) and 72 h (1.0%, PAS 3.9%). As subjects were exposed to short periods of hot water during the 2:1 hot/cold recovery protocol, it is likely

that hydrostatic pressure played a larger role in reducing oedema than vaso-pumping. Similarly, following an eccentric leg press protocol, 14 min of CWT (1 min at 15° C alternating with 1 min at 38° C) ameliorated increases in thigh circumference/swelling after 24 (CWT 0.35%, PAS 1.4%), 48 (CWT 0.2%, PAS 1.4%) and 72 h (CWT 0.2%, PAS 1.1%) (Vaile et al., 2008c).

Contrast water therapy is a commonly used modality in athletic settings and even though it can help to reduce swelling and oedema, athletes will be primarily concerned with the ability of the modality to restore physical function. This will be discussed below.

### 2.9 Effects CWI and CWT on recovery following team sport activity

### 2.9.1 Effects CWI and CWT on physical performance following team sport activity

The following section will only review the effectiveness of both CWI and CWT on the recovery of physical performance measures, such as sprint and jump performance, after participation in team sport and team sport related activities.

Equivocal data exists regarding the effectiveness of CWI and CWT as methods for attenuating physical performance following team sport exercise. Although this is the case, the efficacy of CWI appears to be enhanced by the total time of immersion and by the temperature of water utilised. This will be reviewed below.

### 2.9.1.1 Effect of total immersion time on recovery of physical performance

In team sports, evidence on the overall effectiveness of CWI and CWT is limited and equivocal. It appears however that the total time of immersion may be influential with a total immersion duration of at least 10 min being more effective than shorter durations (i.e. 5-6 min) at inducing substantial positive changes (Table 2.7). In netballers 10 min of CWI and 15 min of CWT (2 x 5 min) helped ameliorate declines in jump and repeat 20 m sprint performance (King and Duffield, 2009) while in soccer players, CWI was more effective than a thermoneutral

protocol at attenuating or improving sprint, repeat sprint and jump performance (Ascensao et al., 2011).

Multiple CWI exposures can also assist recovery. Following 80 min of simulated team-sport running (4 x 20 min quarters), CWI utilised immediately post exercise and again after 24 h, proved more effective at restoring and improving sprint performance than a passive recovery (Ingram et al., 2009).

Task	Participan ts	ipan Recovery protocol		Performance measure	Change in performance compared to pre-exercise			
Simulated netball circuit (King and Duffield, 2009)	10 well trained female netballers	PAS 15 min passive rest	CWI 2 x 5 min at 9.3°C with 2.5 min between	Vertical Jump	24 h post	PAS -8.1%	CWI -4.4% **	
80 min simulated team sport exercise (Ingram et al., 2009)	11 male team sport athletes	PAS 15 min passive rest	CWI 2 x 5 min at 10°C with 2.5 min between	20 m sprint Repeat sprint (10 x 20 m)	48 h post 48 h post	PAS -0.75% -1.69%	CWI 0.50% *# 0.03% *#	
Soccer match (Ascensao et al., 2011)	20 male soccer players	TWI 1 x 10 min at 35°C	CWI 1 x 10 min at 10°C	20 m sprint Countermovement	24 h post 48 h 24 h post	TWI -9.6% -10.0% -26.7%	CWI -5.2% -5.4% -13.2%	
				Jump Squat Jump	48 h 24 h post 48 h	-21.1% -8.9% -8.4%	-7.7% -2.8% 4.1% #	

Table 2.7: Time course and magnitude of change in physical performance following 10 min of cold water immersion (CWI) compared to either passive recovery (PAS) or thermoneutral immersion (TWI) after team sport participation. \* Significant difference to PAS or TWI (p<0.05), \*\* Indicates large effect between CWI and PAS (>0.7), # Improvement in performance compared to baseline.

Several investigations have been able to demonstrate the positive benefits of short term (i.e. less than 10 min) water immersion (Table 2.8) however results have been inconsistent. In netballers, CWT (5 min total CWI immersion) undertaken after completing a simulated netball circuit had a large effect (effect size 0.74) in attenuating the decline in repeat 20 m sprint time when compared to a passive recovery. After a 3 day basketball tournament, 5 min of cold water immersion (5 x 1 min) improved time taken to perform a line drill (repeat sprint between line

markings on the court) and also assisted in maintaining 20 m sprint performance when compared to a control condition (Montgomery et al., 2008b). Similarly, during a soccer tournament, CWI (5 x 1 min) allowed athletes to better maintain running performance (total and high intensity running distance) over a series of 4 matches compared to TWI (Rowsell et al., 2011).

Task Participants		<b>Recovery protocol</b>		Performance measure	Change in performance compared to pre-exercise					
Effect of CWI										
3 day 3 game basketball tournament (Montgomery et al., 2008b)	29 male basketball players	CON Carbohydrate ingestion + stretching	CWI 5 x 1 min at 11°C with 2 min between	Line Drill (repeat sprint between line markings on court)	Pre- tournament	CON -0.4%	CWI 1.4% #			
				20 m sprint	Pre- tournament	CON -0.7%	CWI -0.5%			
4 day 4 game soccer tournament (Rowsell et al., 2011)	20 junior male soccer players	TWI 5 x 1 min at 34°C with 1 min between	CWI 5 x 1 min at 10°C with 1 min between	Drop in total run distance covered compared to Game 1	Game 3 Game 4	TWI -8.5% -9.6%	CWI -5.8% * -5.0% *			
				Drop in total high intensity running (>15 km/h) distance compared to Game 1	Game 3 Game 4	-12.3% -26.8%	-10.9% -7.7% *			
			Effect of C	CWT						
Simulated netball circuit (King and Duffield, 2009)	10 well trained female netballers	PAS 15 min passive rest	CWT 5 x 1 min at 9.7°C alt with 2 x1 min shower at 39.1°	5 x 20 m sprint	24 h post	PAS -5.8%	CWT -4.4% **			

Table 2.8: Time course and magnitude of change in physical performance following immersion of 5 min during cold water immersion (CWI) or contrast water therapy (CWT) compared to either passive recovery (PAS), control condition (CON) or thermoneutral immersion (TWI) after team sport participation. \* Significant difference to PAS or TWI (p<0.05), \*\* Indicates large effect between CWI/CWT and PAS (>0.7), # Improvement in performance compared to baseline.

The efficacy of short term immersion strategies in team sports has not always been evident however. Throughout a 4 day junior soccer tournament, CWI (5 x 1 min at  $10^{\circ}$ C) provided no additional benefit than TWI (5 x 1 min at  $34^{\circ}$ C) in the recovery of 20 m sprint time, repeat-

sprint time (12 x 20 m) or vertical jump height (Rowsell et al., 2009). Although no difference was observed between the two water therapies, the use of a control group was lacking, therefore, fully understanding the effectiveness of CWI is not possible. In youth soccer players, CWT was ineffective at restoring post-match vertical jump performance compared to a passive recovery (Kinugasa and Kilding, 2009). Players undertook 9 min of CWT which consisted of 3 x 1 min at 12°C alternating with 3 x 2 min shower at 38°C. The use of showers has not been recommended as an effective means of exposure to hot water (Halson, 2011) as it fails to provide the hydrostatic benefits associated with water immersion (section 2.7). When combined with only 3 min of water immersion it is possible to see how the 9 min of CWT failed to effectively aid post-match jump performance.

In rugby union, CWI has also failed to elicit a positive recovery benefit. Prior to and after 4 weeks of training and match play, 300 m and repeat sprint test performance (phosphate decrement test consisting of 7 x 7 sec sprint with 21 sec recovery) was assessed in youth players (Higgins et al., 2011). During the 4 week period players undertook 5 min of CWI (1 x 5 min at 10-12°C), 14 min of CWT (7 x 1 min cold at 10-12°C alternating with 7 x 1 min hot at 38-40°C) or a passive recovery. After the 4 weeks, CWT provided no additional performance benefit compared to the passive recovery while CWI was assessed as being detrimental to repeat sprint performance. Additionally, CWT was more successful than CWI at attenuating both 300 m sprint time (Effect size 0.53) and the phosphate decrement test (0.99). Several methodological issues may explain these results. A large discrepancy between total immersion times existed between the CWT and CWI protocol whereby players in the CWT were exposed to a greater length of cold immersion than the CWI group. Readers are also unaware of the physical status of participants prior to the re-test. A total training load was given for players during the four week investigation, however no breakdown of each participant's game time, game load or number of games played was presented. This leaves readers unaware whether the CWI group played more game time or played in more physically demanding positions (i.e. sustained more physical contact and more muscle damage/soreness) than participants in other recovery groups. Additionally, readers are only given data on test results after 4 weeks. Players were not tested at any other stage during the 4 weeks, therefore the effectiveness of CWI throughout this period is unknown. As no evidence of match/training related fatigue or damage was presented and participants were re-tested only after 4 weeks, being able to accurately determine the effectiveness of CWI is difficult and the notion that CWI is detrimental to performance should be taken with caution.

### 2.9.1.2 Effect of water temperature on recovery of physical performance

The temperature of water utilised by athletes may also influence the effectiveness of CWI or CWT. In athletic populations, a temperature range of between 10-15°C has been recommended to ensure that CWI protocols are well tolerated (Halson, 2011) and although not a team sport related activity, the only investigation looking at multiple water temperatures has been carried out on cyclists (Vaile et al., 2008a). Participants completed an initial 30 min cycling task followed by one of 5 recovery interventions and 40 min of passive rest before completing the same cycling task. The recovery interventions included an active recovery (15 min cycling at 40% VO<sub>2peak</sub>) and 4 water based recoveries at 10°C, 15°C and 20°C (all 5 x 1 min immersion with 2 min between) and a continuous exposure to 20°C (20°C+, 1x15 min). Performance in the second exercise bout was determined by the percentage of work done in exercise bout 2 compared to exercise bout 1 and it was clear that CWI allowed cyclists to perform substantially more work in the second bout when compared to the active recovery (Figure 2.23). Immersion in the 10-15°C temperature range was the most effective for restoring physical performance with immersion in 15°C improving performance in the second bout of exercise. This temperature range has been put forward as the most effective range for improving athletic performance (Halson, 2011) and although no evidence on the effectiveness of differing cold temperatures within a CWT protocol is available, based on these results, it is likely that a range of 10-15°C would provide greatest benefit. Temperatures below this (i.e. 5°C) can induce sensations of stinging, burning and aching due to increased neural sensitivity (Knight et al., 1981, Kowal, 1983, Prentice, 1999, Starkey, 1999). This can become uncomfortable and consequently, athletes are likely miss out on the full and potential benefit that CWI may provide if they fail to stay immersed and adhere to any CWI prescribed.

The effects of multiple water temperatures in the same investigation is yet to be clarified in team sport athletes, the information obtained is highly applicable to team sport athletes and should be taken into account by athletes/coaches/medical staff in helping to shape their recovery protocols.

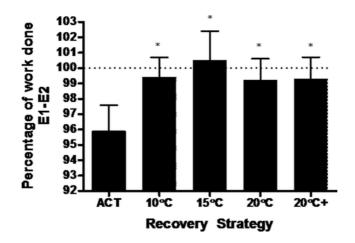


Figure 2.23: Percentage of work done in exercise bout 2 (E2) compared to exercise bout 1 (E1) following various recovery strategies (Vaile et al., 2008a). ACT = active recovery. \* Significantly different to ACT (p<0.05).

### 2.9.1.3 Effects of water recovery in AF

The effect of recovery in AF has received very little attention. Only one investigation has been conducted in recovery on AF players, where the effectiveness of immediate post-game recovery on soreness, power and flexibility was measured over the 48 h post game (Dawson et al., 2005). After playing an AF game, players undertook one of a number of recovery interventions; 15 min of stretching, 15 min of pool walking or a CWT protocol (5 x 2 min hot shower at 45°C alternating with 4 x 1min at 12°C) where players alternated between standing in a hot shower and standing in waist deep cold water. In addition to this players also undertook a next day standard 25-30 min pool recovery session. Data collected 15 and 48 h post games suggest that performing an immediate post-match CWT or pool recovery session did not

significantly enhance vertical jump or 6 sec cycle sprinting compared to the next day pool recovery session alone. Previous team sport research indicates that a total of 10 min of immersion can enhance the effectiveness of a water recovery protocol (Table 2.6) and although the AF players undertook 14 min of post-match CWT recovery, they were immersed for only 4 min. This short period may not have been enough to allow for the maximum benefits that immersion in water offers. Additionally, the use of showers, which has not been recommended (Halson, 2011), made up 10 of the 14 min duration of the CWT recovery, therefore, when combined with the low exposure to hydrostatic pressure, it is possible to see how the CWT protocol may have been ineffective.

Australian footballers typically train 24-72 h after a game or previous training session, however during the study by Dawson et al., no data was collected 24 h post-match. Consequently, the benefits of a hot and cold or pool recovery session remain unclear a full day after the match. During the pre-season, AF players are more than likely to train less than a day after any previous session, therefore having an understanding of the effectiveness after 24 h of a recovery modality will become crucial in determining its use.

### 2.9.1.4 Conclusions

The overall evidence concerning the effects of water immersion on post-exercise recovery in team sport is lacking and inconsistent. It appears that the use of hot showers is ineffective, however a total immersion time of at least 10 min may enhance effectiveness and an immersion time of 14-15 min has been recommended (Halson, 2011). This has been demonstrated after a soccer game where recovery of 20 m sprint performance along with squat and countermovement jumps was enhanced by CWI (Ascensao et al., 2011). Similarly, 10 min of CWI and 15 min of CWT in netballers helped ameliorate declines in jump and repeat 20 m sprint performance (King and Duffield, 2009). Multiple exposures of 10 min can also assist recovery. Following simulated team-sport running, multiple CWI exposures facilitated a more rapid return of 20 m sprint performance (Ingram et al., 2009).

### 2.10 Effects of CWI and CWT on perceptions of fatigue

Increased perceptions of fatigue can result in decreased post-match physical performance in the days after exercise while increases in mental fatigue prior to exercise have also led to reduced performance (see section 2.2.2). This increased perception can have a negative impact on training effort and is likely to increase quicker in more fatigued athletes

Overcoming this fatigue is important and both CWI and CWT have been effective in reducing the fatigue associated with exercise. Cold water immersion (5 x 1 min at  $10^{\circ}$ C) effectively reduced the perception of general fatigue when used post-match during a 4 game, 4 day soccer tournament in several investigations (Rowsell et al., 2009, Rowsell et al., 2011). When compared to TWI (5 x 1 min at 34°C), the use of CWI (5 x 1 min at 10°C) led to substantially lower increases in general fatigue (p < 0.007) however data indicating the magnitude of change between groups was not included (Rowsell et al., 2011). The same immersion protocols also led to CWI being more effective than TWI at mediating fatigue post-match in a similar 4 day tournament (Mean fatigue game 1 = CWI 2.8, TWI 4.8; Mean fatigue game 2 = CWI 3.1, TWI 5.7; Mean fatigue game 3 = 3.4, TWI 5.7; Mean fatigue game 4 = CWI 4.6, TWI 5.6) (Rowsell et al., 2009). Similarly, during a 3 day basketball tournament when compared to a control stretching condition, CWI (5 x1 min at 11°C) effectively reduced perceptions of fatigue across the tournament (change in fatigue score CWI 1.0, control 2.2) (Montgomery et al., 2008b) with the authors attributing reductions in fatigue to enhancing physical performance over a number of days. The effects of CWI on fatigue reduction has been investigated in cyclists exercising in the heat (Halson et al., 2008). After completing a simulated 40 min time trial on 2 separate occasions, CWI (3 x 1 min at 11.5°C) effectively reduced perceptions of general fatigue compared to a control condition (CWI 5.3, PAS 6.3) as well as increasing the rating of mental recovery (CWI 6.7, PAS 6.1). In a dose response investigation, CWT (1 min hot 38.4C alternating with 1 min cold at 14.6C) of 6, 12 or 18 min helped to reduce whole body fatigue between 2 cycling bouts of 75 min separated by 2 h when compared to a control group (Figure 2.24) (Versey et al., 2011).

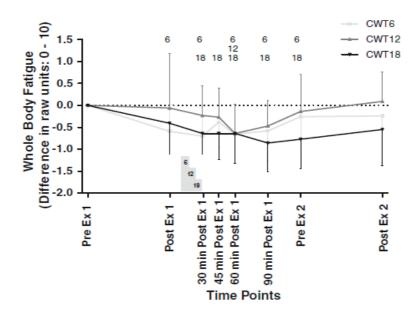


Figure 2.24: Change in whole body fatigue for 6, 12 and 18 min of CWT compared to a control condition. Adapted from (Versey et al., 2011). Shaded boxes indicate timing and duration of CWT protocols. Substantial effects for CWT trial compared with control are indicated by *6*, *12 or 18*.

Although both CWI and CWT can be effective in reducing perceptions of fatigue which in turn, may have a flow on effect to athletic performance, very little data exists. The efficacy of CWI or CWT as a recovery modality in a non-tournament situation is yet to be elucidated in team sport match such as Australian football, while no data exists after training. Reductions in fatigue post-match or training may benefit players by enhancing their overall recovery, their ability to train and their ability to play matches and as such, is an area that needs to be investigated.

### 2.11 Effects of CWI and CWT on muscle soreness after team sport activities

For team sport athletes, participating in their chosen sport often leads to increased perceptions of muscle soreness in the days following exercise (see section 2.3.4.1). The ability to overcome and recover from this soreness can help athletes in their preparation for their next match, particularly in sports competing weekly. As such, this section will look at the effectiveness both CWI and CWT in reducing soreness in team sport and simulated team sport events. It is clear that both CWT and CWI can have positive effects on reducing post-exercise muscle soreness

after team sport or simulated team sport participation, as well as and between games during tournament situations (Table 2.9). In the majority of investigations listed below, both CWI and CWT have successfully ameliorated increases in soreness. In AF however, no clear differences were seen (Dawson et al., 2005). This may be due to both the CWT and PAS protocols using a next day pool recovery session and not investigating either protocol in isolation. Additionally, CWI was not utilised during the investigation, therefore the effectiveness of both interventions is unclear in AF and this needs investigating.

Task	Participants	Total immersion/passive recovery time			Time course of change in perceived muscle soreness				
Australian football match (Dawson et al., 2005)	17 semi- professional Australian footballers	CWT 15 min (5 x 2 min shower at 45°C alt with 1 min at 12°C) + next day pool recovery		PAS 15 min passive rest + next day pool recovery	Baseline 15 h post 48 h	CWT 2.6 4.6 4.0	2	PAS 2.8 4.4 3.9	
Loughborough intermittent shuttle test (90 min) (Bailey et al., 2007)	20 healthy males	CWI 1 x 10 min at 10°C		PAS 10 min passive rest	Baseline 0 h post 1 h 24 h 48 h	CWI 1.2 6.2 3.5* 4.6* 4.3*	PA 1. 6. 5. 6. 5.	7 8 5 7	
3 game, 3 day basketball tournament (Montgomery et al., 2008b)	29 male basketballers	CWI 5 x 1min at 11°C		CON 2 x (10 x 15 sec stretches)	Pre tournament Post tournament	CWI 1.4 3.3*	CC 1. 4.	.6	
4 game, 4 day soccer tournament (Rowsell et al., 2009)	20 high performance junior male soccer players	CWI 5 x 1 min at 10°C		TWI 5 x 1min at 34°C	<b>24 h after</b> Match 1 Match 2 Match 3 Match 4	CWI 4.2 4.2 4.2 5.6	5 6 6	WI 5.4 5.2 5.8 5.1	
80 min simulated team sport running (Ingram et al., 2009)	11 male athletes	CWI 10 min (2 x 5 min at 10°C)	CWT 12 min (3 x 2 min at 10°C alt with 2 min at 40°C)	PAS 15 min passive rest	Baseline 0 h post 24 h 48 h	CWI 1 7 3*# 3*	CWT 1 7 4* 4	PAS 1 7 5 5	
Simulated netball circuit (4 x 15 min quarters) (King and Duffield, 2009)	11 male athletes	CWI 10 min (2 x 5 min at 9.3°C)	CWT 15 min (5 x 1 min at 9.7°C alt with 2 min shower at 39.1°C)	PAS 15 min passive rest	Baseline 0 h post Post- intervention	CWI 0.3 4.1 2.5*	CWT 0.1 4.1 2.6*	PAS 0.3 3.8 4.0	
Soccer match (Ascensao et al., 2011)	20 male junior soccer players	CWI 1 x 10 min at 10°C		TWI 1 x 10 min at 35°C	Quadriceps Baseline 30 min 24 h 48 h Calf Baseline 30 min 24 h 48 h	CW2 0.0 3.4 3.5* 3.1 0.0 2.1 1.4* 1.7	0. 4. * 6. 4. 0. 3.	0 0 6 0 4 7	

Table 2.9: Time course and magnitude of change in perceived muscle soreness after different recovery modalities. PAS = passive recovery, CWI = cold water immersion, CWT = contrast water immersion, TWI = thermoneutral immersion, CON = control. \* Significant difference to PAS (p<0.05), \*\* Significant difference to TWI (p<0.05), # Significant difference to CWT (p<0.05)

# 2.12 Effects of CWI and CWT on inflammatory and muscle damage markers after team sport activities

It is clear that following team sport activity, both muscle damage markers and inflammatory markers can increase substantially (see section 2.4.4.2 and 2.5.5). Muscle damage can impair physical performance in the days post-exercise while an acute inflammatory response that remains unresolved can lead to chronic inflammation (see section 2.4.4.3 and 2.5.2). Failure to recover from this can lead to a state of overreaching in athletes (Halson and Jeukendrup, 2004) and water immersion may play a role in decreasing the increases in inflammatory and muscle damage markers associated with exercise. For team sport athletes, this may become vital during the recovery period after weekly games or during a long season.

Both CWI and CWT have been effective in reducing the appearance of Mb and CK following team sport activity (Table 2.10). Cold water immersion is more effective than a passive recovery for reducing [Mb] in the hour after exercise (when [Mb] peaks, see section 2.4.2 and 2.4.4.2) while both CWI and CWT are more effective at attenuating peak increases in [CK] in the days following exercise.

Task	Participants	Total immersion/passive recovery timeTime course and mag match change in [Mb] compared to pre-exer					b] and [CK]		
Rugby matches (Gill et al., 2006)	23 elite male rugby players	CWT 9 min (3x 8-10°C alt 3 x 2 min a	with	PAS 9 min passive rest	CK (IU.L <sup>-1</sup> ) 36 h post 84 h	Percent CWT 55% 85%	age recovery PAS 18% 39%		
Loughborough intermittent shuttle test (90 min) (Bailey et al., 2007)	20 healthy males	CWI 1 x 10 min at 10°C		PAS 10 min passive rest	Mb (ng.ml <sup>-1</sup> ) 0 h post 1 h 24 h	CWI 660% 590% * 100%	PAS 720% 920% 160%		
80 min simulated team sport running (Ingram et al., 2009)	11 male athletes	CWI 10 min (2 x 5 min at 10°C)	CWT 12 min (3 x 2 min at 10°C alt with 2 min at 40°C)	PAS 15 min passive rest	CK (IU.L <sup>-1</sup> ) 24 h 48 h	261% 2.	WT PAS 38% 310 % 00% 135 %		
Soccer match (Ascensao et al., 2011)	20 male junior soccer players	CWI 1 x 10 min at 10°C		TWI 1 x 10 min at 35°C	Mb (ng.ml <sup>-1</sup> ) 0.5 h post 24 h CK (IU.L <sup>-1</sup> ) 0.5 h post 24 h 48 h	CWI 950% * 100% 292% 483% * 543% *	TWI 1100% 100% 308% 617% 623%		

Table 2.10: Time course and magnitude of change in [Mb] and [CK] after different recovery modalities. PAS = passive recovery, CWI = cold water immersion, CWT = contrast water immersion, TWI = thermoneutral immersion. \* Significant difference to PAS/TWI (p<0.05)

It is clear that CWI and CWT are effective at attenuating both [CK] and [Mb] in team sport athletes, however they appear to not substantially moderate the inflammatory process. Several studies have investigated the effects of CWT and/or CWI on both markers of muscle damage and inflammation. Following a DOMS-inducing leg press protocol, the effectiveness of 14 min of CWI (1 x 14 min at 15°C) or CWT (7 x 1 min at 15°C alternating with 7 x 1 min hot at 38°C) in reducing [CK], [Mb] and [IL-6] was monitored (Vaile et al., 2008c). Compared to a passive recovery CWI substantially reduced increases in [CK] at 24 (CWI 3.6% vs. PAS 201.6%) and lowered [CK] to below baseline after 72 h (CWI -8.5% vs. PAS 56.6%). After 24 h however, both interventions provided no difference in moderating increases in either [Mb] (CWI 23.4% vs. PAS 41.5%; CWT 52.4% vs. PAS 45.4%) or [IL-6] (CWI 2.8% vs. PAS 7.7%;

CWT 0.0% vs. PAS 11.8%). During a 3 day basketball tournament, CWI (5 x 1 min at 11°C) was used post-game and its impact on ameliorating markers of muscle damage and inflammatory cytokines was measured against compression garments and a control group (Montgomery et al., 2008a). Immediately following each game, [Mb], [CK], [IL10] and [IL-6] all increased substantially (between 200-450%). Measures 6 h post-game revealed no difference between groups for any of the markers measured. When comparing pre- to post-tournament measures however, CWI demonstrated a moderate effect in reducing [Mb] (effect size -0.80±0.55) and a small effect in reducing [CK] (effects size -0.24±0.33) compared to the control group. Additionally, CWI demonstrated a small effect in reducing [Mb] (effect size -0.58±0.51) when compared to the compression group. Comparisons between interventions for cytokine measures across the tournament were unclear.

For athletes training and competing regularly, the ability of water immersion to reduce muscle damage and inflammatory responses may be important in the avoidance of chronic inflammation and injury. Training and competition results in degrees of micro trauma to muscle and also connective tissue (Smith, 2000). This type of injury may be regarded as an initial phase along an injury continuum that can eventually lead to overtraining and further injury should the athlete not be afforded enough time to recover and these type of injuries not be allowed to heal (Smith, 2000). The inflammatory process is involved in muscle regeneration and adaptation (Tidball, 2005) and the lack of substantial disruption to the inflammatory process in the above team sport studies indicates that CWI and/or CWT may not disrupt the normal regenerative process.

#### 2.13 Conclusions

It has been well established that eccentric exercise can cause muscle damage leading to decreases in eccentric and concentric strength. It can also negatively impact events where power production is important, such as sprinting, jumping and changing direction, while perceived fatigue can also lead to a drop in physical performance.

The application of CWI and CWT can be effective for reducing swelling, muscle soreness, perceived fatigue, thermal comfort and markers of inflammation and muscle damage. Both modalities can also be effective at ameliorating or enhancing jumping and repeat-sprint performance, dynamic power, isokinetic force and maintaining cycling performance.

Despite the increase in use of CWI and CWT in the team sport setting, conjecture still remains regarding the overall efficacy of both modalities. It appears that a total immersion time of 10 or more minutes may enhance the effectiveness of CWI and CWT, however methodological differences between investigations have led to inconsistent results. Differences such as the method of hot exposure (hot showers involve no hydrostatic pressure and are not recommended vs. hot tub), the non-use of a control group, the level of athlete/participant used and differences in the temperature of cold water can only lead to confusion. For practitioners or athletes, such confusion and inconsistency in results can cloud decisions in choosing which modality to use, or how long an individual needs to be immersed to obtain the greatest benefits. Adding further to this confusion is the lack of consistency in the total immersion times employed within an investigation. Comparisons have been made between 10 min of CWI and 15 min of CWT (King and Duffield, 2009) or 10 min of CWI and 12 min of CWT (Ingram et al., 2009). This lack of consistent immersion times makes effective comparisons within and between studies more complicated and only adds further confusion concerning the effectiveness of either/both interventions.

In the team sport setting, there is a lack of uniform information on water immersion, particularly comparing the efficacy a single CWI and CWT application within the same athletic population. A gap currently exists on the use and efficacy of CWI and CWT in the sport of Australian football and also following team sport training. Therefore, the aim of this thesis is to determine the effectiveness of both CWI and CWT in restoring/attenuating physical performance, perceived muscle soreness and fatigue, reducing markers of muscle damage and the impact on the normal inflammatory response in Australian football players after training as well as match participation.

# **CHAPTER 3. GENERAL METHODS**

Described below are the general methods used during the completion of this thesis and a reference to which chapters they were utilised in. Chapter specific methods have been detailed within the methods section of the relevant chapter.

# 3.1 Psychometric measures

Perceptions of muscular soreness, fatigue and exertion were measured 90 min prior to exercise as well as immediately post, 24 and 48 h post-exercise in chapters 4 and 5.

# 3.1.1 Perceived fatigue and muscle soreness

Perceived fatigue and muscular soreness were assessed using two independent visual analogue scales. Players were asked to indicate their soreness and fatigue on an unmarked horizontal line (100 mm in length) with 'No pain/No fatigue' and 'Very-severe pain/Very-severe fatigue' at opposing ends. Scores were calculated based on the number of millimetres the 'ranking' (in the form of a marked line on the scale provided) was placed from the 'No soreness/fatigue' end of the scale (Gould et al., 2001).

## 3.1.2 Rating of perceived exertion

Perceived training intensity was measured via a rating of perceived exertion (RPE). Immediately post-exercise, players rated the global intensity of sessions based on a modified Borg scale of 1-10 (Borg, 1990). This measure has previously been considered to be a good indicator in team-sport of global internal load (Impellizzeri et al., 2011).

# 3.2 Physical performance testing

Physical performance measures were undertaken 90-min prior to exercise as well as immediately post, 24 and 48 h post-exercise in chapters 4 and 5.

In chapters 4 and 5, jump performance in the form of static jumps (SJ) and countermovement jumps (CMJ) was assessed upon completion of the psychometric measures. Following a 5-min standardised warm-up consisting of cycling and dynamic stretching, participants performed several sub-maximal practice jumps. Testing then commenced with two SJ's separated by 15 s. Players were required to place their hands on hips, lower themselves into a self-selected squat position (same for all jumps) and maintain this position for 3 s before jumping as high as possible. The SJ has been previously validated as a reliable measure of lower limb power (Markovic et al., 2004, Randell et al., 2011). The SJ's was followed by two CMJ's separated by 15 s. Players started with hands on hips in a fully upright position, dipped downward to a selfselected depth and then jumped as high as possible without pausing. The mean of the two jumps was then calculated and jump performance was assessed through flight-time:contraction time ratio (SJ FC:CT and CMJ FC:CT) (chapters 4 and 5) and flight time (SJ FT and CMJ FT) (chapter 5). This measure has been demonstrated as the most sensitive and useful variable for the assessment of neuromuscular fatigue in elite AF players (Cormack et al., 2008a). Trials were performed on a commercially-available force plate (400 Series Isometric Force Plate; Fitness Technology, Adelaide, Australia) connected to computer software (Ballistic Measurement System; Fitness Technology) capable of recording vertical ground reaction forces.

## 3.2.2 Repeat-sprint performance

In chapters 4 and 5, players undertook a repeat-sprint ability test within 90 s of concluding their jumps. This consisted of 6 x 20 m maximal sprints departing every 30 s. Sprints were conducted in an indoor stadium on a wooden floor. Each sprint involved a stationary start with players given a countdown to signify the start of the sprint. Time was recorded from when players first broke a light beam and was not a reflection of their reaction to the countdown. Players walked back to the start position between sprints ready to commence their next effort.

Players performed two practice sprints prior to testing. Total time (sum of 6 sprints) was recorded using wireless single-beam light gates (Smart Speed, Fusion Sport, Grabba International Pty Ltd, Queensland, Australia). Total sprint time has been used as it has previously been reported as a reliable method of presenting repeat-sprint data (Spencer et al., 2006).

### **3.3** Recovery interventions

The recovery interventions outlined below were utilised in chapters 4, 5 and 6. Within 12-min of completing exercise and immediately upon completion of the psychometric and physical testing, all athletes commenced their assigned recovery intervention. During COLD and CWT, all players were seated with legs stretched out and immersed up to their xiphoid process. Cold water immersion involved players being submerged in tubs in 12°C water for 14 consecutive minutes. Contrast water therapy required players to alternate between 1-min hot (38°C) and 1-min cold water (12°C) immersion for 7 cycles (total of 14-min). Hot and cold tubs (iCool Australia Pty Ltd, Queensland, Australia) were placed next to each other to minimize time spent changing between tubs (3-s). Heating and cooling units attached to the tubs measured and adjusted the water temperature in order to maintain the desired temperature. The PAS group was seated for 14-min with minimal movement in a similar posture to the immersed groups.

# 3.4 Activity profile analysis

During chapters 5 and 6, player movement data was recorded from a sub-sample of 5 players during an AF match using 5-Hz Global Positioning Systems (MinimaxX V2.0, Catapult, Australia). Players were selected for activity profile analysis based on playing position and included players from all positions (forward/defender, midfield, midfield/forward, ruck, and defender/midfield). Data was analysed and reported as metres covered per minute of match time (m.min<sup>-1</sup>) (Aughey, 2010).

## 3.5 Statistical analysis

The statistical analysis described was utilised in chapters 4 and 5. Data is expressed as mean  $\pm$  SD and effect size (ES)  $\pm$  90% confidence intervals. Magnitude of change was calculated using ES  $\pm$  90% CI and % change using a custom spreadsheet. Repeat-sprint and jumps data was log transformed to reduce bias due to non-uniformity of error. Effects were characterised for their practical (clinical) significance. Effect size was assessed using the following criteria: <0.2 trivial, 0.2–0.6 small, 0.6–1.2 moderate, 1.2–2.0 large, and >2.0 very large (Hopkins et al., 2009). A substantial change was accepted when there was more than 75% likelihood that the true value of the standardised mean difference was greater than the smallest worthwhile (substantial) change and data not meeting these criteria was classified as unclear. Raw data and change in means were reported for soreness and fatigue.

# CHAPTER 4. STUDY 1: EFFECTS OF WATER IMMERSION ON POST-TRAINING RECOVERY IN AUSTRALIAN FOOTBALLERS

Elias, G. P., Varley, M. C., Wyckelsma, V. L., McKenna, M. J., Minahan, C. L., & Aughey, R. J. Effects of water immersion on post-training recovery in Australian footballers. *International Journal of Sports Physiology and Performance*. **7**(4), 357-66 (2012)

## 4.1 Introduction

Post-exercise recovery, referring to a period of psychological and physical regeneration (Vaile et al., 2008b), is a vital part of any training program as it can allow athletes to overcome the demands of training and competition. Muscle damage can have a limiting effect on muscular performance in the days following exercise (Clarkson and Hubal, 2002). Recovery should be prioritised for athletes competing regularly (i.e. weekly), as it contributes to the minimisation of fatigue between competition (Halson, 2011). Strategies that optimise recovery after intense activity may help reduce physical performance decrements after exercise and therefore benefit subsequent training and athletic performance.

Cold water immersion (CWI) and contrast water therapy (CWT) are recovery methods commonly employed in athletic settings (Ascensao et al., 2011, Dawson et al., 2005, Ingram et al., 2009, King and Duffield, 2009, Montgomery et al., 2008b, Rowsell et al., 2009). It has been suggested that both can assist in reducing fatigue between exercise bouts (Vaile et al., 2008b), alleviate symptoms of exercise-induced muscle soreness (Ingram et al., 2009, Vaile et al., 2007, Bailey et al., 2007) and attenuate post-exercise power and strength reductions (Ingram et al., 2009, Vaile et al., 2007). Reductions in oedema, localised swelling and indices of exercise-induced muscle damage have also been attributed to CWI (Bailey et al., 2007, Yanagisawa et al., 2003). Additionally, immersion in water can

There is inconsistent evidence concerning the effects of water immersion on post-exercise recovery in team-sport, however, a total immersion time of at least 10-min for CWI and CWT may enhance the effectiveness of these interventions. After a soccer game, 10-min of CWI (1 x 10-min,  $10^{\circ}$ C) enhanced recovery of 20-m sprint performance and squat and countermovement jumps (Ascensao et al., 2011). In netballers, CWI (2 x 5-min, 9.3°C) helped ameliorate declines in 20-m repeat-sprint time and jump performance (King and Duffield, 2009). Multiple exposures of 10-min or more can also assist recovery. Following simulated team-sport running, multiple CWI (2 x 5-min,  $10^{\circ}$ C) and CWT (3 x 2-min,  $10^{\circ}$ C alternating with 3 x 2-min,  $40^{\circ}$ C) exposures reduced soreness, with CWI also facilitating a more rapid return of 20-m sprint performance (Ingram et al., 2009).

Investigations utilising less than 10-min immersion have demonstrated mixed results. A 5-min CWI protocol (5 x 1-min, 10°C) did not affect physical performance, indices of muscle damage or inflammation between successive matches in high-performance junior soccer players (Rowsell et al., 2009). In rugby, CWI (1 x 5-min, 10-12°C) had a detrimental effect on sprint performance (Higgins et al., 2010) while CWT (3 x 1-min, 8-10°C alternating with 3 x 1-min shower, 38°C) had little effect on 40-m repeat-sprint performance (Hamlin, 2007). Conversely, 5 minutes of CWI (5 x 1-min, 11°C) assisted restoration of physical performance during a basketball tournament (Montgomery et al., 2008b), while in netballers CWT (5 x 1-min, 9.7°C alternating with 5 x 2-min shower, 39.1°C) helped restore drops in jump performance and 20-m repeat-sprint time (King and Duffield, 2009).

These results lend support to the effectiveness and the use of longer submersion times. Indeed, a recent review (Halson, 2011) has established that 14 to 15-min of CWI or CWT can improve performance and recommends at least 10-min of immersion be utilised.

Cold water immersion may promote recovery quicker than CWT as exposure to cold temperatures induces a number of physiological changes. These include analgesia, reductions in fluid diffusion, vascular permeability and oedema, decreased localised vasoconstriction, as well as reduced acute inflammatory responses resulting from muscle damage (Eston and Peters, 1999, Cote et al., 1988, Koc et al., 2006). Cold water immersion can also have an additional impact on the cardiovascular system and the circulating blood volume (Bonde-Petersen et al., 1992, Peiffer et al., 2009, Vaile et al., 2010, Yanagisawa et al., 2003). When compared to hot water exposure, such as in CWT, cold water can reduce heart rate and cardiac output (Bonde-Petersen et al., 1992), reduce blood flow to both the upper and lower extremities (Peiffer et al., 2009, Vaile et al., 2010), increase peripheral vasoconstriction (Peiffer et al., 2009), decrease vessel diameter (Peiffer et al., 2009) and increase mean arterial pressure and total muscle peripheral resistance (Bonde-Petersen et al., 1992). When combined, the effects of CWI can further reduce oedema and localised swelling when compared to CWT. Conversely, the application of heat may be detrimental as it can increase the inflammatory response and oedema (Wilcock et al., 2006a, Cote et al., 1988) and therefore potentially decrease CWT's effectiveness when compared to CWI. This was demonstrated when CWI ameliorated declines in repeat-sprint ability, muscle soreness and leg strength more effectively than CWT in team-sport running (Ingram et al., 2009). In netballers, CWI was more effective at reducing soreness and jump and sprint decrements between training sessions (King and Duffield, 2009).

A lack of consistency in immersion durations employed within the same team-sport investigation makes it difficult to compare the effects of CWI and CWT (Ingram et al., 2009, King and Duffield, 2009). This may lead to confusion and/or difficulty for athletes/practitioners in deciding which protocol to use and how long individuals need to be immersed to obtain the greatest benefits. This is compounded by a lack of dose-response studies in team-sport athletes. Australian football (AF) is a high-intensity intermittent full-contact team sport. It requires a combination of ball skills, speed and athleticism and places high demands on a player's prolonged running capacity, particularly high-intensity running (Wisbey et al., 2010). As such, within-game fatigue (Wisbey et al., 2010) and subsequent muscle soreness (Dawson et al., 2005) and damage must be overcome by players. Recovery may play a vital role in this.

The difficulty in definitively determining the efficacy of water immersion for team-sport recovery, has been the predominant use of simulated running or tournament scenarios as exercise stressors rather than matches or training. For a sport like AF, tournaments are uncommon and the direct applicability of simulated running is questionable. It does not provide the same chaotic running patterns and is characterized by the absence of game specific movements such as jumping or kicking. It also lacks the presence of a ball, an opponent, direct physical contact and game/training pressures. Therefore making an informed decision on water based interventions using this data is difficult. Recovery after a game of AF has been investigated (Dawson et al., 2005), however the results of performing a hot (5 x 2-min shower, 45°C) and cold (4 x 1-min, 12°C) recovery or pool recovery session directly post-game did not significantly enhance physical recovery over performing a next day pool recovery session. Although water recovery post AF game has been previously investigated (Dawson et al., 2005), the efficacy of a single exposure to CWI or CWT immediately post AF activity is lacking, as is data on the use of water immersion per-se immediately post AF training. During an AF season players are generally expected to train 24 to 72 h after their previous session, while during the pre-season this is likely to be less than 24 h. It is believed that acute post-training recovery may enable athletes to better prepare for their next training stimulus.

This study aims to investigate the effectiveness of immediate and acute CWI and CWT immersion times on recovery of physical and psychometric performance post AF training, due to the inconsistency in CWI and CWT immersion times in team sports, and the limited research on CWI and CWT recovery in AF. It is hypothesized firstly, that AF training would lead to reduced physical capacity as well as increased fatigue and soreness over a 48 h period. Secondly it is believe that an acute, single exposure to both CWI and CWT would promote recovery more effectively than a passive recovery after AF training. It is further hypothesized that CWI would be more effective than CWT as an acute recovery intervention after AF training.

## 4.2 Methods

### 4.2.1 Subjects

Fourteen professional male AF players (age  $20.9 \pm 3.3$  years; body mass  $79.6 \pm 6.7$  kg; height  $186 \pm 7.2$  cm) with at least 4 years of specific AF training, volunteered and provided written informed consent to participate in this 3 week study. All players were injury and illness free throughout the investigation. The study was approved by the Victoria University Human Research Ethics Committee and conformed to the Declaration of Helsinki.

# 4.2.2 Design

This counterbalanced crossover study, investigated the effectiveness of two commonly-used water-based recoveries following a mid-week pre-season AF training session.

Repeat-sprint ability (RSA), jump performance (counter-movement jump (CMJ) and squat jump (SJ)), perceived soreness and fatigue were measured 45 min before training and immediately after. Soreness and fatigue were measured after 1 h, with all measures repeated 24 and 48 h post-training. Immediately following post-training measures, all players undertook one of three recovery protocols; passive recovery (PAS), cold water immersion (CWI) or contrast water therapy (CWT). During week 1, all players undertook PAS. This was unavoidable and mandated by the football club due to continuing facility construction. In week 2, CWI or CWT was randomly assigned with players undertaking the opposing recovery during week 3.

During the 48 h following standardized training, players only undertook their prescribed recovery intervention and participated in no further physical training.

## 4.2.3 AF training

Training took place at the same time/day over three consecutive weeks in similar environmental conditions (Week 1, 24.5°C; week 2, 24.0°C; week 3, 24.2°C) and was replicated and standardised for time, load and distance covered in drills. This was the main weekly

football session and took place 48 h after the previous training session. Training consisted of a 10-min standard jog warm-up, 30 min standardised skill development consisting of kicking, handball and positioning drills, and 4 x 2.5 min small-sided games interspersed with 2.5 min rest. Small-sided games involved two groups of eight players playing a hand passing game on a  $25 \times 15$  m pitch. Goals were placed at each end with the highest scoring team winning. Players were encouraged verbally by coaches throughout. Training was non-contact and this type of team-sport training has proved to be a reliable option for assessing team-sport performance (Singh et al., 2010). Small-sided games were a regular part of the training program at this point of the pre-season. Players had partaken in them on numerous occasions and were familiar with the demands and the set-up.

Immediately after training was completed, all players headed directly into the testing facility (100 m away) for post-training testing. This was to avoid athletes cooling down prior to post-exercise measures.

## 4.2.4 Psychometric measures

The methods used to determine rating of perceived exertion, muscle soreness and fatigue are outlined in chapter 3.

## 4.2.5 Jump performance

A description of the jumps required to be performed by athletes and the method of determining jump performance is outlined in chapter 3.

# 4.2.6 Repeat-sprint testing

A description of the repeat-sprinting protocol performed by athletes and the methods used to determine repeat-sprint performance is outlined in chapter 3.

#### 4.2.7 Recovery interventions

A description of the post-training recovery interventions performed by athletes is outlined in chapter 3.

# 4.2.8 Statistical analysis

A description of the statistical analysis performed is outlined in chapter 3. Data is expressed as mean  $\pm$  SD and effect size (ES)  $\pm$  90% confidence intervals.

# 4.3 Results

Pre and post-training psychometric and physical performance data are presented in Table 4.1. Table 4.2 indicates change in mean and percentage change differences for soreness, fatigue and physical performance between interventions. Figure 4.1 indicates change in mean scores between interventions for psychometric measures. Figure 4.2 indicates percentage change between interventions for physical performance measures. Training intensity (RPE) was comparable between groups over the 3 weeks (PAS  $7.1\pm1.1$ , CWI  $7.0\pm1.0$ , and CWT  $7.1\pm1.0$ ).

#### **4.3.1** Acute response to AF training

Immediately following training, acute decreases in sprint and jump performances were evident along with increased perceptions of soreness and fatigue, with no clear differences between groups (Table 4.1). Muscle soreness increased for all groups (PAS  $2.69\pm0.84$ , CWT  $1.39\pm0.32$ , and CWI  $2.50\pm0.39$ ) as did perceived fatigue (PAS  $4.77\pm0.74$ , CWT  $2.82\pm0.49$ , and CWI  $2.75\pm0.41$ ). Although results were unclear, jump performance decreased slightly for all groups. Sprint times deteriorated similarly for PAS ( $1.10\pm0.35$ ), CWT ( $0.71\pm0.21$ ) and CWI ( $0.80\pm0.24$ ).

## 4.3.2 Response to AF training over 48 h

Declines in performance and psychometric measures persisted for PAS during the 48 h posttraining period (Table 4.1). Muscle soreness rose at 1 h (3.21±0.87), increased markedly at 24 h  $(5.86\pm0.65)$  and remained elevated at 48 h (4.28\pm0.58). Similarly, perceived fatigue remained high at 1 (3.93±1.11), 24 (2.75±1.07) and 48 h (2.96±0.77). Jump performances were slightly blunted immediately post-training and remained so throughout the ensuing 48 h. Sprint times declined further after 24 h (1.85±0.51) but were back to baseline at 48 h.

## 4.3.3 Effect of recovery on muscle soreness

Muscle soreness was effectively attenuated by both CWT and CWI (Table 4.1 and figure 4.1). Contrast water therapy reduced soreness at 1 ( $0.51\pm0.57$ ), 24 ( $1.25\pm0.49$ ) and 48 h ( $0.69\pm0.45$ ) while CWI reduced soreness close to baseline at 1 h, was effective at 24 h ( $0.58\pm0.53$ ) and reduced soreness to pre-training levels after 48 h.

Cold water immersion was the most effective modality at reducing training-induced soreness when evaluating its effectiveness against CWT and PAS (Table 4.2). Of the two water based modalities, CWI was more effective than CWT at 24 (-0.95 $\pm$ 0.50) and 48 h (-0.78 $\pm$ 0.47-). Compared to PAS, CWT was better at alleviating muscle soreness at 1 (-1.60 $\pm$ 0.78), 24 (-2.58 $\pm$ 0.61) and 48 h (-2.12 $\pm$ 0.63) and CWI was substantially lower at 1 (-1.99 $\pm$ 0.65), 24 (-3.53 $\pm$ 0.58) and 48 h (-2.90 $\pm$ 0.54).

# 4.3.4 Effect of recovery on perceived fatigue

Both CWT and CWI were effective at moderating perceived fatigue (Table 4.1 and figure 4.1). Fatigue was attenuated by CWT at 1 ( $0.67\pm0.38$ ), 24 ( $0.63\pm0.46$ ) and 48 h ( $0.56\pm0.48$ ). Cold water immersion also reduced fatigue after 1 ( $0.79\pm0.38$ ) and 24 h ( $0.43\pm0.48$ ) while restoring fatigue to pre-training levels after 48 h.

Compared to CWT and PAS, CWI was the most effective intervention at reducing perceptions of fatigue (Table 4.2). At 48 h, CWI was more effective than CWT ( $-0.72\pm0.46$ ). Compared to PAS, both interventions were more successful at 1 (CWT  $-1.26\pm0.46$ ; CWI  $-1.28\pm0.60$ ), 24 (CWT  $-0.77\pm0.58$ ; CWI  $-0.99\pm0.67$ ) and 48 h (CWT  $-0.91\pm0.50$ ; COLD  $-1.46\pm0.52$ ).

# **4.3.5** *Effect of recovery on jump performance*

Comparisons for jump performances indicated no clear differences between groups (Table 4.1 and figure 4.2). At 48 h, CMJ was ameliorated effectively by CWT and CWI, while SJ was improved above baseline slightly at 48 h by CWI. Comparisons between interventions revealed CWI to be the most effective (Table 4.2).

## **4.3.6** Effect of recovery on sprint performance

Both water recoveries were effective at attenuating RSA (Table 4.1 and figure 4.2). Sprint times were attenuated by CWT at 24 ( $0.40\pm0.29$ ) and 48 h ( $0.32\pm0.25$ ). For CWI, sprint time was restored back to baseline at 24 h, and near baseline after 48 h.

Direct comparisons between interventions revealed CWI to be the most successful for restoring RSA at 24 h, with PAS being the poorest (Table 4.2). Cold water immersion was more effective than CWT at both 24 ( $-0.37\pm0.27$ ) and 48 h ( $-0.23\pm0.22$ ). In comparison to PAS, CWT ( $-1.29\pm0.56$ ) and CWI ( $-1.52\pm0.40$ ) were more effective after 24 h, with no differences evident at 48 h.

Table 4.1: Muscle soreness, perceived fatigue, jump performances and total sprint time (6 x 20-m sprints) prior to standardised Australian football training, immediately after and up to 48 h post-training (n = 14) (mean  $\pm$  SD) (Effect size data indicates difference compared to pre-training values). PAS = passive recovery, CWI = cold water immersion, CWT = contrast water therapy

Measure	Group	Pre-training	Post-training	1 h	24 h	48 h
		Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Muscle soreness	PAS	$2.4 \pm 0.9$	$4.9 \pm 1.5^{d}$	$5.4 \pm 1.9^{d}$	$7.9 \pm 0.8^{\rm d}$	$6.4 \pm 1.3^{d}$
	CWI	$2.8 \pm 1.0$	$5.5 \pm 0.9^{d}$	$3.0 \pm 1.2$	$3.4 \pm 1.3^{a}$	$2.8 \pm 1.2$
	CWT	$2.5 \pm 1.4$	$4.5 \pm 1.2^{\circ}$	$3.2 \pm 1.6^{a}$	$4.3 \pm 1.8^{b}$	$3.5 \pm 1.5^{b}$
Perceived fatigue	PAS	$2.5 \pm 0.7$	$6.1 \pm 2.0^{d}$	$5.7 \pm 2.2^{d}$	$4.7 \pm 1.7^{d}$	$4.9 \pm 1.7^{\rm d}$
	CWI	$2.6 \pm 1.3$	$6.3 \pm 0.9^{d}$	$3.7 \pm 1.2^{b}$	$3.2 \pm 1.3^{a}$	$2.6 \pm 1.3$
	CWT	$2.3 \pm 1.2$	$5.7 \pm 1.1^{d}$	$3.1 \pm 1.1^{b}$	$3.1 \pm 1.3^{b}$	$3.0 \pm 1.4^{a}$
Countermovement	PAS	$0.67 \pm 0.19$	$0.66 \pm 0.17$	-	$0.66 \pm 0.13$	$0.64 \pm 0.18$
Jump*	CWI	$0.69 \pm 0.14$	$0.69 \pm 0.11$	-	$0.68\pm0.09$	$0.68\pm0.12$
	CWT	$0.68 \pm 0.16$	$0.68 \pm 0.13$	-	$0.67 \pm 0.14$	$0.68 \pm 0.15$
Squat Jump*	PAS	$0.69 \pm 0.17$	$0.67 \pm 0.23$	-	$0.67 \pm 0.17$	$0.67 \pm 0.15$
	CWI	$0.66 \pm 0.14$	$0.62 \pm 0.11$	-	$0.66 \pm 0.09$	$0.67 \pm 0.12$
	CWT	$0.67 \pm 0.17$	$0.63 \pm 0.23$	-	$0.66 \pm 0.17$	$0.66 \pm 0.15$
Total sprint time (s)	PAS	$18.53 \pm 0.38$	$18.97 \pm 0.41$ <sup>b</sup>	-	$19.28 \pm 0.55$ <sup>c</sup>	$18.62 \pm 0.70$
	CWI	$18.62 \pm 0.46$	$19.01 \pm 0.56^{b}$	-	$18.62 \pm 0.49$	$18.66 \pm 0.46$
	CWT	$18.63 \pm 0.45$	$18.97 \pm 0.46^{b}$	_	$18.82 \pm 0.51^{a}$	$18.78 \pm 0.53$

 $^{\rm a}$  Small change (ES = 0.2–0.6);  $^{\rm b}$  Moderate change (ES 0.6–1.2);  $^{\rm c}$  Large change (ES 1.2–2.0);  $^{\rm d}$  Very large change (ES >2.0)

\* Flight time:contraction time ratio (s)

Measure	Group	1 h	24 h	48 h
		Change in mean	Change in mean	Change in mean
Muscle soreness	CWI v PAS	$\downarrow 2.8 \pm 0.9^{\circ}$	$\downarrow 4.9 \pm 0.8^{d}$	$\downarrow 4.0 \pm 0.8^{d}$
	CWT v PAS	$\downarrow 2.3 \pm 1.0^{\circ}$	$\downarrow 3.7 \pm 0.9^{d}$	$\downarrow 3.0 \pm 0.9^{\circ}$
	CWI v CWT	$\downarrow 0.5 \pm 0.7$	$\downarrow 1.2 \pm 0.6^{b}$	$\downarrow 1.0 \pm 0.6^{b}$
Perceived fatigue	CWI v PAS	$\downarrow 2.1 \pm 1.0^{\circ}$	$\downarrow 1.6 \pm 1.1^{\mathrm{b}}$	$\downarrow 2.4 \pm 0.8^{\circ}$
	CWT v PAS	$\downarrow 2.4 \pm 0.8^{\circ}$	$\downarrow 1.4 \pm 1.0^{b}$	$\downarrow 1.7 \pm 0.9^{b}$
	CWI v CWT	$\uparrow 0.3 \pm 0.7$	$\downarrow 0.2 \pm 0.7$	$\downarrow 0.7 \pm 0.5^{b}$
		% Change	% Change	% Change
Countermovement	CWI v PAS	-	↑0.3 ± 22.1	↑3.1 ± 29.2
Jump*	CWT v PAS	-	$\downarrow 0.9 \pm 22.5$	↑3.1 ± 31.5
	CWI v CWT	-	↑1.2 ± 13.2	$0.0 \pm 12.8$
Squat Jump*	CWI v PAS	-	↑4.3 ± 32.5	$^{4.9 \pm 18.8}$
	CWT v PAS	-	11.4 ± 17.8	$\uparrow 0.7 \pm 18.4$
	CWI v CWT	-	$\uparrow 2.9 \pm 30.9$	$\uparrow$ 4.2 ± 20.4
Total sprint time (s)	CWI v PAS	-	$\downarrow 3.9 \pm 1.1^{\circ}$	$\downarrow 0.3 \pm 1.3$
	CWT v PAS	-	$\downarrow 2.9 \pm 1.3^{\circ}$	↑0.3 ± 1.3
	CWI v CWT	-	$\downarrow 1.0 \pm 0.7^{a}$	$\downarrow 0.6 \pm 0.4^{a}$

Table 4.2: Change in mean between treatment groups for muscle soreness and perceived fatigue post-training, and percentage change between treatment groups for jump performances and total sprint time (6 x 20-m sprints) (n = 14). PAS = passive recovery, CWI = cold water immersion, CWT = contrast water therapy.

 $^{\rm a}$  Small change (ES = 0.2–0.6);  $^{\rm b}$  Moderate change (ES 0.6–1.2);  $^{\rm c}$  Large change (ES 1.2–2.0);  $^{\rm d}$  Very large change (ES >2.0)

\* Flight time:contraction time ratio (s)

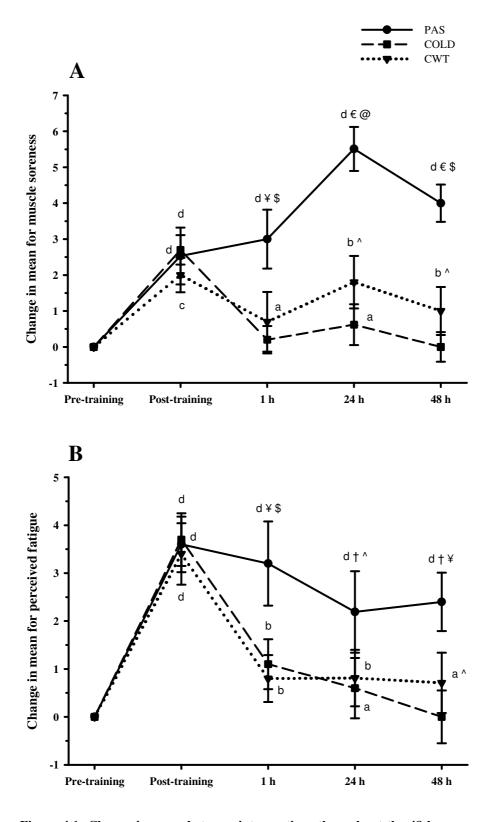


Figure 4.1: Change in mean between interventions throughout the 48 h recovery period for (A) muscle soreness and (B) perceived fatigue. <sup>a</sup> indicates small effect compared to pre-training; <sup>b</sup> indicates moderate effect to pre-training; <sup>c</sup> indicates large effect compared to pre-training; <sup>d</sup> indicates very large effect compared to pre-training; <sup>^</sup> indicates moderate effect compared to CWI; <sup>†</sup> indicates moderate difference compared to CWT; <sup>¥</sup> indicates large effect compared to CWI; <sup>§</sup> indicates large effect compared to CWT; <sup>§</sup> indicates large effect compared to CWT; <sup>©</sup> indicates very large effect compared to CWT.

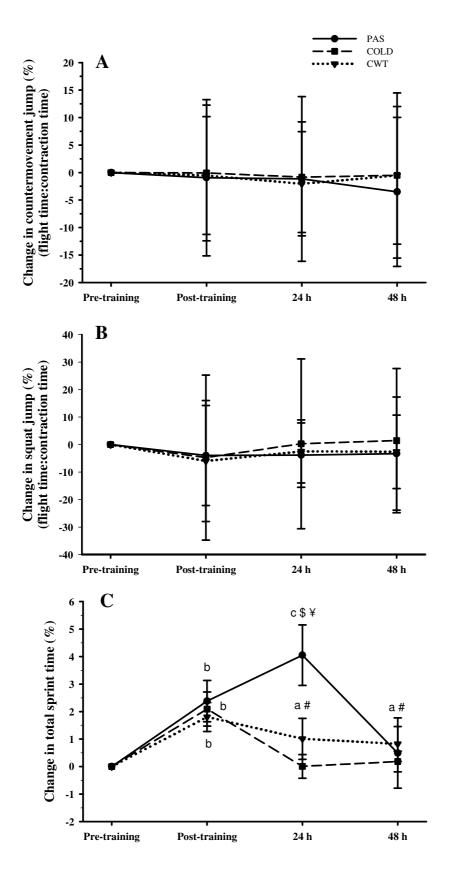


Figure 4.2: Percent change between interventions throughout the 48 h recovery period for (A) countermovement jump, (B) squat jump performance and (C) total sprint time (6 x 20 m sprints). <sup>a</sup> indicates small effect compared to pre-training; <sup>b</sup> indicates moderate effect compared to pre-training; <sup>c</sup> indicates large effect compared to pre-training; <sup>#</sup> indicates small effect compared to CWI; <sup>§</sup> indicates large effect compared to CWI; <sup>§</sup>

## 4.4 Discussion

The major finding of this investigation was that the acute application of CWI was a more effective recovery method than CWT or PAS for restoring repeat sprint performance and psychometric perceptions following AF training. Furthermore, it was established that AF training leads to functional and perceptual deficits lasting up to 48 h. Finally, both CWI and CWT restored sprint and perceptual measures during a typical AF training week. For the first time in team-sport, groups were matched for time immersed in water.

Restoring and promoting performance should be the primary aim of any recovery session. It was demonstrated that repeat-sprint performance declined substantially after training, and that after 24 h, 14-min of both CWI and CWT were effective at ameliorating these declines, with CWI restoring sprint time back to baseline. This may allow AF players to be better prepared for any running and/or speed/agility work which typically occurs within 24 h after a skills session during the pre-season. The effectiveness of both interventions concurs with previous findings where CWI and CWT were effective at reducing declines in RSA in netballers (King and Duffield, 2009). Cold water immersion also better maintained 20-m acceleration and sprint time after three days of basketball competition (Montgomery et al., 2008b), and enhanced 20-m sprint and jumping performance after a soccer game (Ascensao et al., 2011). Interestingly, after 48 h, CWI was more effective than CWT for restoring repeat-sprint ability, displaying similar results post simulated team-sport running (Ingram et al., 2009). When compared to PAS at 48 h however, no clear differences existed for CWI or CWT. This is in contrast to results where CWI was more effective than PAS after simulated team-sport running (Ingram et al., 2009). Differences may lie in the fact that following simulated running, athletes were subject to multiple CWI exposures (immediately and 24 h post) and not a single exposure as was the case in this study, or that simulated running resulted in greater muscle damage experienced by athletes. Differences may also be due to residual effects of central or peripheral fatigue which can take hours or even days to subside after exercise (Keeton and Binder-Macleod, 2006). Low-frequency fatigue (a proportionately greater loss of force in response to low- versus high-frequency muscle stimulation) can play a substantial role in reducing the force-generating capabilities of skeletal muscle (Keeton and Binder-Macleod, 2006).

Training induced a pronounced increase in post-exercise muscle soreness, which persisted during the 48 h recovery period for the PAS group. This is consistent with previous research and indicates the acute effects of exercise can persist throughout the ensuing recovery period (Bailey et al., 2007, Ingram et al., 2009, Montgomery et al., 2008b, Vaile et al., 2007, Vaile et al., 2008b, Ascensao et al., 2011). Both CWI and CWT had a marked positive effect on alleviating post-exercise muscle soreness with CWI fully restoring soreness to pre-exercise levels. Delayed onset muscle soreness can lead to an increased injury risk (Cheung et al., 2003) and with CWI and CWT reducing soreness effectively, post-training recovery may have an important role in preventing injury. It is slightly surprising that muscle soreness persisted in the PAS group. The repeat bout effect has previously been shown to reduce the degree of muscle soreness in subsequent bouts of exercise (Eston et al., 1996, Westerlind et al., 1992) and may played more of a role in reducing this soreness, however much of the research involving running based activities re-tests participants a number of days or weeks after the initial bout, and not 24 h as was the case in this thesis. The fact that physical testing was conducted so soon after training (and also the match) may have played a role in physical activity still being reduced. Additionally, although physical performance declined, it is still possible and likely that there may have been some influence from previous exercise bouts and that the repeated bout effect may have actually reduced the degree of soreness exhibited. There was no way of me measuring the potential effects of any repeated bout effect without following and repeatedly testing this group of athletes over the 4 months of training prior to this study being conducted.

Similar to soreness, the perception of fatigue rose markedly post-training. Both CWI and CWT were effective at diminishing fatigue post-training, with CWI returning fatigue to preexercise values. This concurs with earlier research. Previously, CWI moderated fatigue between successive soccer matches (Rowsell et al., 2009) and attenuated cumulative fatigue over a three-day basketball tournament (Montgomery et al., 2008b), while CWI and CWT reduced fatigue over five days of cycling (Vaile et al., 2008b).

After a game of AF, countermovement jump flight-time:contraction time ratio has been established as the most sensitive and useful variable for the assessment of neuromuscular fatigue (Cormack et al., 2008a). However, the results in this study suggest that jump performance was not affected by training and may not be the best indicator of fatigue after training; therefore it may not be an appropriate post-training measure. Australian football training contains less high-intensity movements and physical pressure than games (Dawson et al., 2004a), therefore it should fail to elicit the fatigue and subsequent reductions in jump performance as reported after games (Cormack et al., 2008a). It has also been suggested that even when sore, players may still be able to produce one-off efforts equal or close to their best, and that repeat-effort tests may be a better way to assess functional decrements (Dawson et al., 2005). This may have been the case with the PAS group.

The effectiveness of CWT and CWI over PAS may be explained by the physiological effects of hydrostatic pressure. Due to increased fluid shifts from the extremities to the central cavity, hydrostatic pressure can help restrict oedema accumulation within muscle post-exercise (Wilcock et al., 2006a). This can help decrease inflammation which can alter force generating capacity(Smith, 1991) and subsequent performance (Wilcock et al., 2006a). Being submerged can also induce a feeling of weightlessness, which can provide greater relaxation and help reduce perceptions of fatigue (Wilcock et al., 2006a).

The physiological effects of temperature may also explain the effectiveness of CWI and CWT. Cold is often used in the treatment of inflammation to improve the rehabilitation process and has a number of other effects including: analgesia, localised vasoconstriction, decreased rate of muscle metabolism, reduced fluid diffusion and vascular permeability, reduced oedema formation and decreasing acute inflammation responses from muscle damage (Eston and Peters, 1999, Cote et al., 1988, Koc et al., 2006). These effects can help to reduce the loss of force generation, pain and swelling that is often associated with inflammation (Smith, 1991) and attenuate any physical performance decrease (Wilcock et al., 2006a). Cold

water is effective at reducing muscle temperature (Myrer et al., 1994). In contrast, superficial heat can increase the inflammatory response and oedema, therefore heat application during CWT may be detrimental to athletes (Wilcock et al., 2006a, Cote et al., 1988). Although this may be counteracted by the subsequent application of cold, the overall decrease in inflammation and oedema may not be as great as 14 continuous minutes of cold. This may then diminish CWT's effectiveness at reducing pain and swelling. Consequently, the effects of increased exposure to cold combined with continuous hydrostatic pressure may explain why CWI was more effective than CWT throughout this investigation.

It is acknowledged that this study had some limitations. The first was that due to continued construction of facilities and therefore limited access; all players were forced to perform the PAS protocol during week 1. Although this was the case, all players at the time of testing were familiar with the training and the small-sided games that were utilised. Players had also partaken in this type of training on numerous occasions during the 16-17 previous weeks of pre-season. Due to the nature of water immersion, it was not possible to incorporate a placebo control for each of the water conditions and immersion durations therefore we cannot dismiss a treatment effect. In sedentary subjects, regular use of CWI interventions blunted some training adaptations over a 4-6 week period (Yamane et al., 2006). Subjects performed multiple 20-min CWI immersion at 5°C, and although not fully representative of protocols and temperatures used by athletic populations, this result may be of some concern. As this investigation was concerned with the acute single application of CWI/CWT, it is not able to be determined if this induced any long term impairments, and further investigation regarding this possibility is warranted. Endurance fitness remains important for elite AF players, however we were unable to measure the effects of CWI and CWT on this. As this study used professional athletes, it was not possible to include endurance testing, which would have caused too much disruption to the training and upcoming games schedule. During the course of this investigation, the effects of a thermoneutral immersion were unable to be investigated, and as such determining the degree of impact of both temperature and hydrostatic pressure is difficult. It is however clear that CWI is a more effective modality that CWT, therefore it is

likely that the effect of temperature played a more important role in determining the success of each modality rather than the effect of hydrostatic pressure.

# 4.5 Practical Applications

Players, coaches, sport science and medical staff should be aware that AF training leads to reductions in physical and psychometric measures over a 24 to 48 h period and that the use of CWI or CWT can attenuate post-training physical and psychometric reductions.

Staff and athletes also need to be aware that after AF training, CWI is more effective than CWT or PAS for restoring RSA as well as reducing perceptions of soreness and fatigue. Further research is needed to determine the effects of a single application of CWI and/or CWT after a game of AF.

## 4.6 Conclusion

Improving physical performance may be important during an AF season. Players are generally expected to train 24 to 72 h after their previous session; while during the pre-season players are generally expected to train within 24 h. Therefore attenuating the decline in physical performance between sessions is critical to these athletes. The unique nature of this study effectively demonstrated that 14-min of either CWI or CWT can assist athletes recover from typical AF training conditions. Through this study, an acute application of CWI was established as the superior modality for AF players to utilise following a training session. It clearly outperformed both CWT and PAS in minimising muscle soreness and perceived fatigue, as well as helping to more rapidly restore repeat-sprint performance.

# CHAPTER 5. STUDY 2: EFFECTIVENESS OF WATER IMMERSION ON POST-MATCH RECOVERY IN ELITE PROFESSIONAL FOOTBALLERS

Elias, G. P., Wyckelsma, V. L., Varley, M. C., McKenna, M. J. & Aughey, R. J. (In press). Effectiveness of water immersion on post-match recovery in elite professional footballers. *International Journal of Sports Physiology and Performance*.

## 5.1 Introduction

During a competitive team sport season, large physical and mental demands are placed on players. During the weekly cycles that make up a season, players are expected to train, compete and then recover in time to start the cycle again. In some sports, athletes compete several times during this cycle therefore a greater importance should be placed on recovering after matches.

Attaining a balance between recovery and training/competition stresses is important. In sports where athletes compete regularly, recovery is needed to maximise between-competition restoration and minimise fatigue (Halson, 2011). Optimising recovery after intense and exhaustive activity can help alleviate post-exercise physical performance reductions (see chapter 4) (Ascensao et al., 2011, Ingram et al., 2009), and in turn, may benefit subsequent training and performance of athletes.

Cold water immersion (CWI) and contrast water therapy (CWT) are recovery methods commonly employed in athletic settings (see chapter 4) (Ingram et al., 2009, Rowsell et al., 2009). Both methods can assist in attenuating post-exercise power and strength reductions (Ingram et al., 2009, Vaile et al., 2007), alleviate symptoms of exercise-induced muscle soreness (see chapter 4) (Ingram et al., 2009, Vaile et al., 2007, Bailey et al., 2007) and reduce fatigue between exercise bouts (see chapter 4) (Vaile et al., 2008b). Reductions in localised swelling, oedema and indices of exercise-induced muscle damage have also been attributed to CWI (Ascensao et al., 2011, Bailey et al., 2007).

The use of cold temperatures may promote quicker recovery than applying heat. The application of heat can increase oedema and the inflammatory response (Wilcock et al., 2006a, Cote et al., 1988, Swenson et al., 1996), which are both counter-productive to recovery. Conversely, cold exposure can induce numerous positive physiological changes including analgesia, oedema reduction through reduced fluid diffusion, vascular permeability and decreased localised vasoconstriction, as well as reduced acute inflammatory responses resulting from muscle damage (Cote et al., 1988, Wilcock et al., 2006a). Cold water immersion can also have an additional impact on the cardiovascular system and the circulating blood volume (Bonde-Petersen et al., 1992, Peiffer et al., 2009, Vaile et al., 2010, Yanagisawa et al., 2003). When compared to hot water exposure, such as in CWT, cold water can reduce heart rate and cardiac output (Bonde-Petersen et al., 1992), reduce blood flow to both the upper and lower extremities (Peiffer et al., 2009, Vaile et al., 2010), increase peripheral vasoconstriction (Peiffer et al., 2009), decrease vessel diameter (Peiffer et al., 2009) and increase mean arterial pressure and total muscle peripheral resistance (Bonde-Petersen et al., 1992). When combined, the effects of CWI can further reduce oedema and localised swelling when compared to CWT. The effectiveness of cold temperatures is demonstrated where CWI ameliorated declines in repeat-sprint ability, muscle soreness and leg strength more effectively than CWT (Ingram et al., 2009) and also promoted physical and psychometric recovery quicker than CWT following team-sport training (see chapter 4).

In team sports, increasing evidence supports the use of both cold water and contrast immersion therapies and it appears that total immersion time may be influential. An immersion duration of at least 10 min is required to induce substantial changes, with 14-15 min being recommended (Halson, 2011). After training, 14-min of both COLD (1 x 14-min,  $12^{\circ}$ C) and CWT (7 x 1-min, 38°C alternating with 7 x 1-min,  $12^{\circ}$ C) were effective at ameliorating the decline in 20-m sprint performance, muscle soreness and fatigue after

Australian football (AF) training (see chapter 4). After a soccer match, 10-min of cold water immersion (1 x 10-min, 10°C) enhanced recovery of 20-m sprint performance and squat and countermovement jumps (Ascensao et al., 2011). Cold water immersion (2 x 5-min, 9.3°C) and CWT (5 x 1-min, 9.7°C alternating with 5 x 2-min shower, 39.1°C) also helped ameliorate the decline in jump performance and 20-m repeat-sprint time in netballers (King and Duffield, 2009). Multiple exposures of 10-min or more can also assist recovery. Following simulated team-sport running, multiple CWI (2 x 5-min, 10°C) and CWT (3 x 2-min, 10°C alternating with 3 x 2-min, 40°C) exposures assisted in reducing soreness, with cold water facilitating a more rapid return of 20-m sprint performance and leg strength than either a CWT or passive recovery (Ingram et al., 2009).

Evidence of recovery benefits is equivocal when total submersion times are below 10 minutes. During a basketball tournament, 5-min of cold water immersion (5 x 1-min, 11°C) assisted restoration of physical performance (Montgomery et al., 2008b). Conversely, in rugby players, 40-m repeat-sprint performance was unaffected by CWT (3 x 1-min, 8-10°C alternating with 3 x 1-min shower, 38°C) (Hamlin, 2007), while COLD (1 x 5-min, 10-12°C) was detrimental to sprint performance (Higgins et al., 2010). In high-performance junior soccer players, 5-min of cold water immersion (5 x 1-min, 10°C) had no effect on physical performance, indices of muscle damage or inflammation between successive matches (Rowsell et al., 2009).

While research into team sport athlete recovery is growing, limited data exists directly comparing the effectiveness of CWT and CWI with the same group of participants.(Ingram et al., 2009, King and Duffield, 2009). For coaches, athletes and team staff, this can lead to confusion in effectively determining the most appropriate modality to use. Adding further uncertainty is that there appears to be a lack of homogeny with respect to the method of exposure utilised (showers vs. tubs) or the duration of exposure (10 min CWI vs. 12 to 15 min CWT, and not 10 min vs. 10 min). Additionally, comparisons between studies is further complicated by the lack of uniformity in the experience of participants used (novice vs.

experienced), the age of participants (junior vs. senior) or the level of competition (elite vs. amateur).

During the weekly cycle in AF, effective use of cold or contrast immersion modalities may help athletes to recover from the increasing physical and running demands of match play (Wisbey et al., 2010). During matches, both the high-intensity and prolonged running capacity of players are significantly stressed (Wisbey et al., 2010) and players can cover distances at a rate of between 113 and 127 m.min<sup>-1</sup> (Aughey, 2011b). In the days following exercise, muscle damage as well as post-exercise fatigue can limit muscular performance (see chapter 4) (Clarkson and Hubal, 2002) therefore, CWI or CWT recovery may play an important role in overcoming the fatigue experienced during matches (Aughey, 2010) and any associated muscle damage or soreness.

In AF, one post-training investigation has addressed the effectiveness of CWI and CWT (see chapter 4), however limited post-match data exists. In the only post-match investigation, players undertook multiple water exposures in the form of a post-match pool recovery session or a hot shower/cold water submersion plus a next day pool recovery session, or completed only a next day pool recovery session (Dawson et al., 2005). Multiple water exposures failed to significantly enhance physical recovery compared to a next day pool recovery session alone (Dawson et al., 2005). However, in AF, evidence demonstrating the effectiveness of CWI vs. CWT post-match is lacking, as is data on the efficacy of a single exposure to CWI or CWT immediately after a match.

Given the lack of uniformity investigating the effectiveness of CWI and CWT following team sport participation, the lack of overall evidence post-match in AF and an absence of data on the effectiveness of CWI following an AF match, the purpose of this study was therefore to examine the effects of a single 14 min acute application of CWI and CWT on physical performance, muscle soreness and perceived fatigue after an AF match. It was hypothesized that reductions in physical capacity as well as increased fatigue, muscle damage and soreness would result from participation in an AF match and that both CWI and CWT would promote recovery better than a passive recovery, with CWI most effective.

## 5.2 Methods

## 5.2.1 Subjects

Twenty-four professional male AF players ( $19.9 \pm 2.8$  years;  $80.8 \pm 8.1$  kg;  $186.4 \pm 6.4$  cm mean  $\pm$  SD) volunteered and provided written consent to participate in this study. All players were free of injury and illness at the time of testing and during match participation. The study was approved by the Victoria University Human Research Ethics Committee and conformed to the Declaration of Helsinki.

#### 5.2.2 Design

The study evaluated the effectiveness of two commonly-used water recovery methods and a passive recovery on post-match physiological and psychometric restoration after an Australian football match. Two teams of 19 players participated in a full practice match (mean temperature 25.8°C; mean relative humidity 63%) consisting of 4 quarters with a total match duration of 75 minutes.

Ninety minutes prior to the match, each player was required to indicate levels of muscle soreness and perceived fatigue and physical performance consisting of static jump (SJ), counter-movement jump (CMJ) and repeat-sprint ability (RSA) was then measured. Physical performance was tested immediately, 24 and 48 h post-match. Muscle soreness and perceived fatigue was measured immediately, and at 1, 24 and 48 h post-match.

Prior to the match, 24 of the competing players (12 from each team) were pre-selected for participation in the study. Selection was based on including players from all playing positions and then pair matching these selections. This was done to ensure an equal number of players participating in the investigation competed on both teams. Upon completion of the match, the 24 players were randomly assigned to one of three recovery interventions; Passive recovery (PAS) (n=8), CWI (n=8) or CWT (n=8). Players undertook their assigned recovery intervention immediately after they completed their post-match measures.

During the 48 h following the practice match, all participating players undertook no further physical activity and were only allowed to undertake their prescribed recovery intervention. Players also were given both fluid and food recommendations to adhere to in the days prior to and following the match.

# 5.2.3 Psychometric measures

The methods used to determine rating of perceived exertion, muscle soreness and fatigue are outlined in chapter 3.

## 5.2.4 Jump performance

A description of the jumps required to be performed by athletes and the method of determining jump performance is outlined in chapter 3.

# 5.2.5 Repeat-sprint testing

A description of the repeat-sprinting protocol performed by athletes and the methods used to determine repeat-sprint performance is outlined in chapter 3.

### 5.2.6 Recovery interventions

A description of the post-training recovery interventions performed by athletes is outlined in chapter 3.

# 5.2.7 Movement data

A description of the method and equipment used to obtain movement data is outline in chapter 3.

# 5.2.8 Statistical analysis

A description of the statistical analysis performed is outlined in chapter 3. Data is expressed as mean  $\pm$  SD and effect size (ES)  $\pm$  90% confidence intervals.

## 5.3 Results

Psychometric and physical performance data for pre and post-match are presented in Table 5.1. The change in mean and percentage change differences for soreness, fatigue and performance between interventions is presented in Table 5.2. Figure 5.1 compares absolute change in mean scores between interventions for psychometric measures. Figure 5.2 compares percentage change between interventions for jump performances. Figure 5.3 compares percentage change between interventions for repeat-sprint ability.

Perceived exertion was comparable between groups (PAS  $8.4\pm0.74$ , COLD  $8.4\pm0.52$ , and CWT  $8.6\pm0.52$ ). Players covered an average 8,012 m or  $107 \text{ m.min}^{-1}$  (range 102.8-112.1 m.min<sup>-1</sup>) during the 75 min of game time with players playing an average of 71 min (range 69-75 min).

## 5.3.1 Acute response to AF match

Immediately post-match, all groups displayed similar increased muscle soreness and perceived fatigue as well as decreased physical performance (Table 5.1). Countermovement jump (F:C) decreased for all groups with no clear differences for all other jump measures. Sprint performance deteriorated substantially for all groups.

# 5.3.2 Effects of recovery on perceived muscle soreness and fatigue

Post-match and throughout the 48 h recovery period, muscle soreness was highest in the PAS group with the lowest post-match ratings seen after CWI (Table 5.1). Compared to PAS, both CWI and CWT were more effective at alleviating soreness after 24 and 48 h, with CWI also being more effective 1 h post-exercise (Table 5.2 and Figure 5.1). Additionally, CWI was more effective when compared to CWT in reducing soreness after 1, 24 and 48 h (Table 5.2 and Figure 5.1).

Similar to soreness, perceived fatigue post-match and over the following 48 h was highest in PAS and lowest in CWI (Table 5.1). Cold water immersion was the most effective modality at reducing perceptions of fatigue when compared to PAS and CWT, particularly after 24 h

(Table 5.2 and Figure 5.1). Cold water reduced fatigue more effectively after 1, 24 and 48 h than PAS and after 24 and 48 h when compared to CWT. Contrast immersion was more effective compared to PAS only after 24 h.

## 5.3.3 Effect of recovery on physical performance

Both COLD and CWT were more effective at restoring jump performance throughout the recovery period than PAS (Table 5.1), with CWI the most effective. After 24 h, CWI was more effective than PAS in all jump measures, while CWT was more effective than PAS for CMJ (F:C) and SJ (FT) (Table 2 and Figure 2). Additionally, CWI was more effective at reducing declines in SJ (FT) than CWT after 24 h. At 48 h, both CWI and CWT were more effective than PAS at restoring SJ (FT).

Cold water immersion was the most effective at attenuating the decline in sprint performance with PAS being the poorest (Table 5.1). Compared to PAS, both CWI and CWT were more effective throughout the 48 h, and in comparison to CWT, CWI was the superior intervention (Table 5.2 and figure 5.3).

Measure	Group	Pre-match	Post-match		1 hr		24 hr		48 hr	
		Mean ± SD	Mean ± SD	Effect size	Mean ± SD	Effect size	Mean ± SD	Effect size	Mean ± SD	Effect size
Muscle	PAS	$2.1 \pm 0.8$	$6.6 \pm 1.6$	$5.32 \pm 1.24$	$6.6 \pm 1.7$	$5.32 \pm 1.29$	$7.5 \pm 1.4$	$6.40 \pm 0.83$	$5.5 \pm 1.6$	$4.01 \pm 0.97$
Soreness	CWI	$2.3 \pm 0.7$	$6.7 \pm 1.4$	$5.42 \pm 1.59$	$4.5 \pm 1.0$	$2.74 \pm 1.01$	$4.7 \pm 0.7$	$2.99 \pm 0.62$	$2.8 \pm 0.7$	$0.59 \pm 0.10$
	CWT	$2.3 \pm 0.7$	$7.2 \pm 1.7$	$6.37 \pm 1.46$	$6.3 \pm 1.3$	$5.21 \pm 1.36$	$6.5 \pm 0.7$	$5.46 \pm 0.90$	$4.1 \pm 0.7$	$2.39 \pm 0.29$
Perceived	PAS	$2.2 \pm 1.0$	$7.0 \pm 1.2$	$4.40 \pm 0.77$	$6.8 \pm 1.3$	$4.20 \pm 0.80$	$6.4 \pm 1.2$	$3.85 \pm 0.80$	$4.3 \pm 1.5$	$1.91 \pm 0.67$
Fatigue	CWI	$2.2 \pm 0.9$	$7.1 \pm 1.0$	$4.89 \pm 1.32$	$5.3 \pm 1.4$	$3.12 \pm 0.92$	$3.9 \pm 1.6$	$1.67 \pm 0.78$	$3.2 \pm 1.2$	$1.02 \pm 0.72$
	CWT	$2.3 \pm 1.2$	$7.5 \pm 1.5$	$3.90\pm0.76$	$6.0 \pm 1.7$	$2.80\pm0.92$	$5.3 \pm 1.3$	$2.25\pm0.69$	$3.9 \pm 1.4$	$1.22 \pm 0.38$
CMJ (F:C)	PAS	$0.81 \pm 0.14$	$0.74 \pm 0.20$	$-0.51 \pm 0.68$	-	-	$0.66 \pm 0.12$	$-0.92 \pm 0.52$	$0.72 \pm 0.11$	$-0.49 \pm 0.38$
(s)	CWI	$0.82 \pm 0.14$	$0.68 \pm 0.18$	$-1.12 \pm 1.01$	-	-	$0.77 \pm 0.14$	unclear	$0.82 \pm 0.14$	unclear
	CWT	$0.77 \pm 0.22$	$0.68 \pm 0.29$	$-0.59 \pm 0.67$	-	-	$0.73 \pm 0.21$	unclear	$0.77 \pm 0.24$	unclear
SJ (F:C)	PAS	$0.73 \pm 0.21$	$0.61 \pm 0.17$	unclear	-	-	$0.52 \pm 0.25$	1.19±0.68	$0.72 \pm 0.25$	unclear
(s)	CWI	$0.77 \pm 0.23$	$0.65 \pm 0.20$	unclear	-	-	$0.73 \pm 0.12$	unclear	$0.78 \pm 0.20$	unclear
	CWT	$0.74 \pm 0.26$	$0.60\pm0.09$	unclear	-	-	$0.55 \pm 0.19$	unclear	$0.74 \pm 0.27$	unclear
CMJ (FT)	PAS	$0.57 \pm 0.03$	$0.57 \pm 0.04$	unclear	-	-	$0.55 \pm 0.03$	0.55±0.54	$0.56 \pm 0.03$	unclear
(s)	CWI	$0.58 \pm 0.02$	$0.57 \pm 0.04$	unclear	-	-	$0.57 \pm 0.03$	unclear	$0.58 \pm 0.02$	unclear
	CWT	$0.57\pm0.02$	$0.56\pm0.04$	unclear	-	-	$0.56 \pm 0.04$	unclear	$0.57\pm0.02$	unclear
SJ (FT)	PAS	$0.53 \pm 0.02$	$0.52 \pm 0.03$	unclear	-	-	$0.51 \pm 0.02$	$1.37 \pm 0.79$	$0.52 \pm 0.02$	$0.90 \pm 0.61$
(s)	CWI	$0.55 \pm 0.03$	$0.54 \pm 0.03$	unclear	-	-	$0.56 \pm 0.02$	unclear	$0.55 \pm 0.02$	unclear
	CWT	$0.55 \pm 0.02$	$0.54\pm0.02$	unclear	-	-	$0.54\pm0.02$	unclear	$0.55\pm0.03$	unclear
Total sprint	PAS	$18.66 \pm 0.37$	$19.14 \pm 0.28$	$1.15 \pm 0.40$	-	-	$19.39 \pm 0.29$	1.74±0.58	$19.02 \pm 0.35$	$0.87 \pm 0.55$
time (s)	CWI	$18.50 \pm 0.47$	$19.14 \pm 0.67$	$1.19 \pm 0.55$	-	-	$18.54 \pm 0.41$	unclear	$18.50 \pm 0.59$	unclear
	CWT	$18.68 \pm 0.39$	$19.22 \pm 0.46$	$1.19 \pm 0.34$	-	-	$18.99 \pm 0.44$	0.68±0.16	$18.85 \pm 0.44$	$0.36 \pm 0.15$

Table 5.1: Mean  $\pm$  SD and effect size data compared to pre values for muscle soreness, perceived fatigue, jump performances and total sprint time (6 x 20 m sprints) prior to a match, immediately after and over 48 h post-match (mean  $\pm$  SD)

CMJ = countermovement jump; SJ = static jump; F:C = flight time:contraction time; FT = flight time PAS = passive recovery; CWI = cold water immersion; CWT = contrast water immersion

Measure	Group	1 hr		24 hr		48 hr	
		Change in mean	Effect Size	Change in mean	Effect Size	Change in mean	Effect Size
Muscle	CWI v PAS	$\downarrow 2.3 \pm 1.1$	$-3.06 \pm 1.49$	$\downarrow 3.0 \pm 0.5$	$-4.00 \pm 0.69$	$\downarrow 2.9 \pm 0.8$	$-3.87 \pm 1.09$
Soreness	CWT v PAS	↓0.5±1.6	unclear	↓1.2±1.1	$-1.68 \pm 1.54$	↓1.6±0.8	$-2.12 \pm 1.13$
	CWI v CWT	↓1.8±1.3	$-2.53 \pm 1.84$	↓1.8±0.7	$-2.51 \pm 1.02$	↓1.4±0.2	$-1.92 \pm 0.27$
Perceived	CWI v PAS	$\downarrow 1.5 \pm 1.4$	$-1.59 \pm 1.52$	$\downarrow 2.6 \pm 1.1$	$-2.70 \pm 1.20$	$\downarrow 1.1 \pm 1.0$	$-1.14 \pm 1.06$
Fatigue	CWT v PAS	$\downarrow 0.9 \pm 1.5$	unclear	$\downarrow 1.2 \pm 0.9$	$-1.13 \pm 0.86$	$\downarrow 0.5 \pm 0.6$	$-0.43 \pm 0.58$
0	CWI v CWT	$\downarrow 0.6 \pm 0.7$	$-0.57 \pm 0.63$	$\downarrow 1.3 \pm 1.2$	$-1.24 \pm 1.12$	$\downarrow 0.6 \pm 0.6$	$-0.57 \pm 0.59$
		% Change	Effect Size	% Change	Effect Size	% Change	Effect Size
CMJ (F:C)	CWI v PAS	-	-	15.5 ± 16.7	$0.78 \pm 0.88$	↑11.4 ± 17.1	unclear
(s)	CWT v PAS	-	-	$16.2 \pm 20.0$	$0.60 \pm 0.73$	1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	unclear
. /	CWI v CWT	-	-	$\downarrow 0.6 \pm 21.5$	unclear	$\uparrow 2.0 \pm 21.8$	unclear
SJ (F:C)	CWI v PAS	-	-	↑48.3 ± 28.2	$1.22 \pm 0.77$	↑5.5 ± 47.5	unclear
(s)	CWT v PAS	-	-	14.4 ± 79.2	unclear	$12.9 \pm 44.9$	unclear
	CWI v CWT	-	-	↑29.5 ± 49.9	unclear	$\uparrow 2.5 \pm 48.0$	unclear
CMJ (FT)	CWI v PAS	-	-	$2.6 \pm 2.3$	$0.52 \pm 0.46$	$1.4 \pm 2.7$	unclear
(s)	CWT v PAS	-	-	$12.4 \pm 3.7$	unclear	$1.2 \pm 2.0$	unclear
	CWI v CWT	-	-	$\uparrow 0.2 \pm 2.1$	unclear	$\downarrow 0.2 \pm 2.3$	unclear
SJ (FT)	CWI v PAS	-	-	↑6.7 ± 3.6	$1.44 \pm 0.79$	$13.6 \pm 2.5$	$0.79 \pm 0.55$
(s)	CWT v PAS	-	-	$14.2 \pm 2.8$	$1.09 \pm 0.74$	$13.2 \pm 2.3$	$0.84 \pm 0.61$
	CWI v CWT	-	-	$\uparrow 2.4 \pm 2.4$	$0.56 \pm 0.55$	$\uparrow 0.4 \pm 2.1$	unclear
Total sprint	CWI v PAS	-	-	$\downarrow 3.6 \pm 1.3$	$-1.53 \pm 0.53$	$\downarrow 1.9 \pm 1.5$	$-0.81 \pm 0.62$
time (s)	CWT v PAS	-	-	$\downarrow 2.2 \pm 1.3$	$-1.08 \pm 0.62$	$\downarrow 1.1 \pm 1.3$	$-0.51 \pm 0.63$
	CWI v CWT	-	-	$\downarrow 1.4 \pm 0.6$	$-0.56 \pm 0.23$	$\downarrow 0.8 \pm 0.6$	$-0.35 \pm 0.26$

Table 5.2: Between group change in mean and percent change with effect size for muscle soreness, perceived fatigue, jump performances and total sprint time (6 x 20 m sprints) prior to a match, immediately after and over 48 h post-match (mean ± SD)

CMJ = countermovement jump; SJ = static jump; F:C = flight time:contraction time; FT = flight time PAS = passive recovery; CWI = cold water immersion; CWT = contrast water immersion

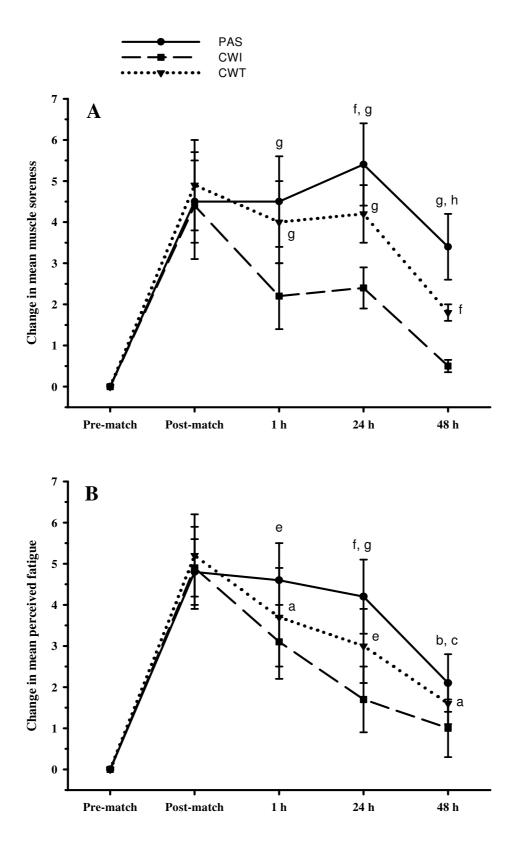


Figure 5.1: Comparison between groups for change in mean (compared to pre-match) throughout the 48 h recovery period for (A) muscle soreness and (B) perceived fatigue. <sup>a</sup> indicates small effect compared to CWI; <sup>b</sup> indicates small effect to CWT; <sup>c</sup> indicates moderate effect compared to CWI; <sup>d</sup> indicates moderate difference compared to CWT; <sup>e</sup> indicates large effect compared to CWI; <sup>f</sup> indicates large effect compared to CWI; <sup>g</sup> indicated very large effect compared to CWT; <sup>g</sup> indicates very large effect compared to CWT. PAS = passive recovery; CWI = cold water immersion; CWT = contrast water therapy.

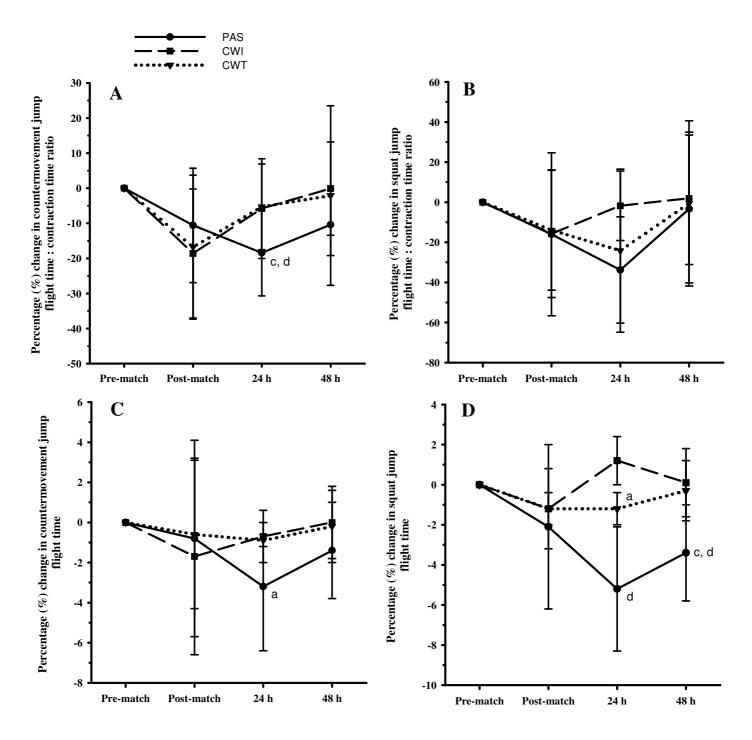


Figure 5.2: Comparison between groups for percent change (compared to pre-match) throughout the 48 h recovery period for (A) countermovement jump flight time:contraction time ratio (B) squat jump flight time:contraction time ratio (C) countermovement jump flight time (D) squat jump flight time.<sup>a</sup> indicates small effect compared to CWI; <sup>b</sup> indicates small effect to CWT; <sup>c</sup> indicates moderate effect compared to CWI; <sup>d</sup> indicates moderate difference compared to CWT. PAS = passive recovery; CWI = cold water immersion; CWT = contrast water therapy.

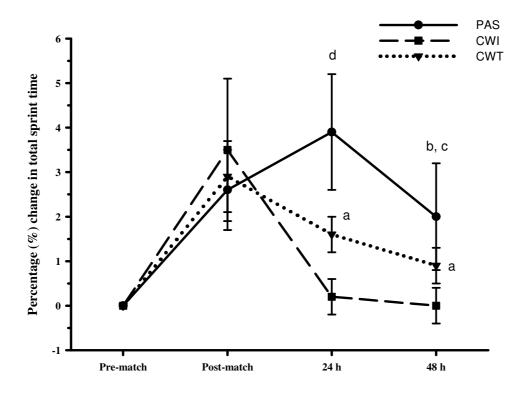


Figure 5.3: Comparison between groups for percent change (compared to pre-match) throughout the 48 h recovery period for total sprint time (6 x 20 m sprints). <sup>a</sup> indicates small effect compared to CWI; <sup>b</sup> indicates small effect to CWT; <sup>c</sup> indicates moderate effect compared to CWI; <sup>d</sup> indicates moderate difference compared to CWT. PAS = passive recovery, CWI = cold water immersion, CWT = contrast water therapy.

#### 5.4 Discussion

The major finding was that a single 14-min application of CWI following an AF match was the most effective for restoring physical performance, reducing perceived fatigue and alleviating muscle soreness than either CWT or PAS. Despite conflicting evidence regarding the effectiveness of CWI and CWT in typical team-sport situations (see chapter 4) (Hamlin, 2007, Higgins et al., 2010, Rowsell et al., 2009, Ascensao et al., 2011, Ingram et al., 2009) these findings demonstrate that both modalities help restore post-exercise physical and psychometric measures.

During the match in this study, players covered an average of 107 m.min<sup>-1</sup>. This distance is similar to the lower range of distances covered in Australian Football League competition matches (Aughey, 2011b) and indicates that the practice match was at a sufficient intensity to replicate the demands of a typical in-season match. These findings confirm that an AF match leads to prolonged functional deficits over 48 h which can be effectively attenuated by CWI and CWT.

The greatest impact on physical performance by either CWI or CWT was in ameliorating the decline in repeat sprint performance. The effectiveness of both interventions after a competitive match concurs with the finding where CWI and CWT were successful at ameliorating the decline in repeat sprint performance after AF training (see chapter 4). Ten min of CWI and 15 min CWT were effective at reducing declines in RSA in netballers (King and Duffield, 2009) while 10 min of CWI facilitated a more rapid return to baseline repeated sprint performances compared to a control group following simulated team-sport running (Ingram et al., 2009). In addition, 5 min CWI was better than control at maintaining 20 m acceleration and sprint time after three days of basketball competition (Montgomery et al., 2008b). Post-match, we have also demonstrated that compared to CWT, CWI was more effective after 24 (1.4%) and 48 h (0.8%). This is consistent with results seen after 5 consecutive days of training in cyclists (Vaile et al., 2008b) and with the previous finding

after AF training where CWI was more effective compared to CWT after 24 (1.0%) and 48 h (0.6%) (see chapter 4).

After the match, muscle soreness increased markedly in all groups and this endured throughout the 48 h recovery period for the PAS group. Similar increases (200-700%) and time course for muscle soreness have been reported in a control group following AF training (see chapter 4), 80 min of simulated team sport running (Ingram et al., 2009), 90 min of shuttle running (Bailey et al., 2007) and following an eccentric bout of leg exercise (Vaile et al., 2007), while during a 3 day basketball tournament, muscle soreness gradually increased (Montgomery et al., 2008b). Post-match, both CWI and CWT were effective at alleviating post-exercise muscle soreness when compared to the PAS treatment, in line with other investigations (see chapter 4) (Bailey et al., 2007, Ingram et al., 2009, Montgomery et al., 2008b, Vaile et al., 2007). Compared to CWT, CWI was more effective at alleviating soreness throughout this investigation. This has also been demonstrated post-training in AF throughout a 48 h recovery period (see chapter 4). Delayed muscle soreness can lead to an increased injury risk (Cheung et al., 2003), and as both CWI and CWT were effective at reducing soreness, post-match recovery may be important for injury prevention.

Post-match, perceived fatigue rose substantially in all groups, and remained elevated in the PAS group similar to increases previously reported (150-250%) (see chapter 4) (Montgomery et al., 2008b, Rowsell et al., 2009, Vaile et al., 2008b). Compared to PAS, this was effectively diminished by both CWI and CWT, with CWI being more successful. This concurs with previous studies where both CWI and CWT reduced fatigue after an AF training session (see chapter 4) and during five days of cycling (Vaile et al., 2008b) while CWI ameliorated cumulative fatigue over a three day basketball tournament (Montgomery et al., 2008b) and curbed fatigue between successive soccer matches (Rowsell et al., 2009). Additionally, CWI was more effective than CWT at diminishing perceived fatigue.

Countermovement jump flight time:contraction time is the most sensitive and useful variable for the assessment of neuromuscular status post-match in elite AF players (Cormack et al., 2008a). In this study, CMJ (F:C) deteriorated substantially post-match and compared to PAS, both CWI and CWT more successfully attenuated this decline. Static jump (F:C) also declined considerably immediately post match, with this effectively restored to pre-exercise values by CWI. Similarly, CWI was the more effective intervention at attenuating the decline in both CMJ (FT) and SJ (FT). It appears therefore that CWI is the more effective intervention for restoring jump performance. The changes in jump performance are likely to be a result of the muscle damage sustained during the match as well as the soreness and fatigue experienced by the athletes. The possible mechanisms responsible for this include low frequency fatigue (characterised by a proportionately greater loss of force in response to low- versus highfrequency muscle stimulation) which can reduce muscle force-generating capabilities by reducing low frequency force (Keeton and Binder-Macleod, 2006) and the disruption to muscle tissue caused by repeated eccentric loading of muscle fibres) (Clarkson and Hubal, 2002). Such muscle damage can lead to reductions in the muscle force and power produced by a muscle (Proske and Allen, 2005) caused possibly by disruptions to calcium regulation leading to reduced muscle excitability (Tee et al., 2007). This damage can also increase the degree of muscle soreness leading to reduced maximal strength and physical output (Trost et al., 2011). Muscle damaging exercise has also been directly linked with reductions in vertical jump performance (Byrne and Eston, 2002a). Therefore the combination of damage, soreness and low frequency fatigue may explain the reductions in jump performance observed.

The passive recovery was least successful in restoring all measures undertaken throughout this investigation. The physiological effect of hydrostatic pressure and the relaxation associated with water immersion may help explain the effectiveness of CWI and CWT. Increasing pressure can cause the displacement of fluids from the extremities towards the central cavity and decrease muscle oedema (Wilcock et al., 2006a). In turn, this reduction in oedema can decrease the pain and loss of force generating capacity associated with swelling (Smith, 1991) and as a result, help to better maintain physical performance (Wilcock et al., 2006a). Being immersed can also induce a feeling of greater relaxation and help reduce perceptions of fatigue and soreness compared to non-immersion strategies (Wilcock et al., 2006a). These effects may explain why PAS was less successful, but not CWI's superiority

over CWT. This may be partially explained by the inherent nature of each protocol. Both interventions required participants to be immersed for 14 minutes. The CWT protocol consisted of 14 one minute immersions; therefore, any benefits of hydrostatic pressure were experienced for only 1 min before being interrupted as players altered between tubs. With the CWI protocol however, participants are subjected to 14 continuous minutes of hydrostatic pressure. This continuous pressure proved more effective for attenuating declines in sprint and jump performance as well as lowering perceptions of fatigue and soreness. Therefore, in team sport athletes, it is possible that the intermittent nature of contrast immersion may be less effective as an intervention strategy when compared to a continuous immersion protocol. The positive benefits of continuous hydrostatic pressure as provided by CWI have also been noted in a previous investigation where CWI more effectively ameliorated declines in physical and psychometric performance than CWT after AF training (see chapter 4). Temperature may also explain the effectiveness of CWI over CWT. As well as being commonly used in the treatment of acute inflammation to enhance the recovery process, the application of cold has numerous physiological effects including: analgesia, localised vasoconstriction, reduced oedema, fluid diffusion and vascular permeability and decreased rate of muscle metabolism and these can assist in decreasing the acute inflammatory response after muscle damage (Cote et al., 1988, Wilcock et al., 2006a, Swenson et al., 1996). Collectively, this may attenuate physical performance decrease (Wilcock et al., 2006a) by reducing the pain, swelling and loss of force generation that is often associated with inflammation (Smith, 1991). Cold water immersion can also reduce thermal strain in athletes exercising in the heat (Vaile et al., 2008a) and reduces core temperature more effectively than CWT (Vaile et al., 2008b). As the AF match in this study took place at an ambient temperature of 25.8°C and with a relative humidity of 63%, a decrease in thermal strain and core temperature may have helped with reducing perceived soreness and fatigue. Indeed, using the heat stress index, players in this study were at a high risk of heat illness (Budd, 2008).

Whereas cold can inhibit the inflammatory process, the application of superficial heat may be detrimental, as heat can increase the inflammatory response and therefore increase oedema (Wilcock et al., 2006a, Cote et al., 1988). The subsequent application of cold after heat application during a CWT protocol may counteract this deleterious response, however, unlike 14 minutes of cold, the overall decrease in oedema may not be as great. This could ultimately diminish CWT's effectiveness at reducing pain and swelling, and in this investigation, may explain why COLD reduced soreness more effectively than CWT.

Several limitations associated with this study need to be acknowledged. Due to the nature of water immersion, and throughout this study, it was not possible to incorporate a placebo control for each of the water conditions and immersion durations. Because of this, a treatment effect cannot be dismissed. However Also, due to practical restrictions on player availability, we were unable to determine the effects of an AF match on endurance performance and therefore the efficacy of CWI or CWT on these parameters. During the course of this investigation, the effects of a thermoneutral immersion were unable to be investigated, and as such determining the degree of impact of both temperature and hydrostatic pressure is difficult. It is however clear that CWI is a more effective modality that CWT, therefore it is likely that the effect of temperature played a more important role in determining the success of each modality rather than the effect of hydrostatic pressure.

#### 5.5 Practical applications

Players, coaches, sport science and medical staff should be aware that an AF match leads to reduced physical and psychometric measures over a 24 to 48 h period and that the use of CWI or CWT can attenuate post-training physical and psychometric reductions.

The most effective strategy is CWI for restoring physical performance as well as reducing the perception of soreness and fatigue.

#### 5.6 Conclusion

Throughout this investigation, it was established that CWI and CWT can assist athlete recovery after an AF match. It is also demonstrated that CWI was more effective than CWT in each of the measures undertaken. This probably reflects the combined effects of uninterrupted hydrostatic pressure that 14 continuous minutes offers, the analgesic effects of cold, the ability of CWI to reduce thermal strain and core temperature efficiently and the ability of CWI to reduce swelling and pain more effectively than contrast therapies.

During an AF season, improving post-match physical performance is important as players generally train in the 24 to 72 h following a match in preparation for upcoming matches. Fourteen minutes of either CWI or CWT were advantageous in helping to rapidly restore physical performance as well as minimise muscle soreness and perceived fatigue post-match. Following a match of AF, CWI was established as the superior modality for players to utilise as it clearly outperformed both CWT and PAS in all variables measured.

# CHAPTER 6. STUDY 3: EFFECTIVENESS OF WATER IMMERSION ON POST-MATCH MUSCLE DAMAGE AND INFLAMMATION IN ELITE PROFESSIONAL FOOTBALLERS

#### 6.1 Introduction

This chapter represents a companion study to the one presented in chapter 5 with the same participants playing in the same AF match. Competitive team sport matches place substantial physical and mental stress on athletes, often resulting in participants experiencing fatigue (Aughey, 2010, Montgomery et al., 2008a, Ronglan et al., 2006, Rowsell et al., 2009), muscle soreness (see chapter 5) (Ascensao et al., 2011, Ascensao et al., 2008, Ispirlidis et al., 2008, Montgomery et al., 2008b, Rowsell et al., 2009), muscle damage (Ascensao et al., 2008, Ispirlidis et al., 2008, Ispirlidis et al., 2008, Montgomery et al., 2008a, Takarada, 2003) and an acute inflammatory response (Ispirlidis et al., 2008, Montgomery et al., 2008, Montgomery et al., 2008a).

In response to exercise, muscle damage or injury, a generalized acute inflammatory response is typically initiated involving the release of various cytokines responsible for the initiation and moderation of the inflammatory response leading to muscle repair and regeneration (Cannon and St Pierre, 1998, Malm, 2001, Montgomery et al., 2008a, Tidball, 1995). This includes the release of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- $\alpha$ ), interleukin 1-beta (IL-1 $\beta$ ) and interleukin-10 (IL-10) (Pedersen, 2000, Shephard, 2002). During the acute inflammatory phase, pro-inflammatory cytokines (IL-6, TNF- $\alpha$  and IL-1 $\beta$ ) help to attract and activate macrophages which are delivered to the injured tissue to help dispose of injury byproducts, such as blood and damaged cells, and set the scene for tissue repair (Watkins et al., 1995). In response to muscle injury, satellite cells are released and begin to proliferate (Seale et al., 2003). This activation and subsequent proliferation is a necessary step contributing to the growth and formation of new myofibres and therefore playing an important role in the growth, maintenance and regeneration of skeletal muscle (Wang and Rudnicki, 2012, Pallafacchina et al., 2012). Interleukin-6 has been identified as an essential and critical regulator of satellite cell proliferation in skeletal muscle (Serrano et al., 2008) and when uninhibited, the release of muscle produced IL-6 assists in myonuclear accretion leading to muscle growth and repair (Serrano et al., 2008). A deficiency or reduction in IL-6 however can result in reduced satellite cell-derived myoblast proliferation and lead to a blunting of muscle repair and growth (Serrano et al., 2008). Therefore a reduction in IL-6 following damaging exercise may increase the length of recovery or reduce muscle growth due to an inhibition the normal repair process of muscle.

Following exercise, changes in IL-1 and TNF- $\alpha$  tend to be subtle while changes in IL-6 are typically of a greater magnitude (Brenner et al., 1999, Pedersen, 2000, Petersen and Pedersen, 2006, Starkie et al., 2001, Suzuki et al., 2003). After prolonged and intense exercise, changes of 140-300% in IL-1 $\beta$ , 230-500% in TNF- $\alpha$ , 600-1200% fold in IL-6 and 400-700% in IL-10 have been reported (Abbey and Rankin, 2011, Ostrowski et al., 1999, Pournot et al., 2011, Shin and Lee, 2011, Starkie et al., 2001, Suzuki et al., 2003) however IL-1 $\beta$  and TNF- $\alpha$  have not always been detected following exercise (Drenth et al., 1995, Jenkins et al., 1994, Ostrowski et al., 1999, Toft et al., 2002, Ullum et al., 1994).

After team sport participation, changes in inflammatory cytokines are not as pronounced as those following a marathon or exercise of more than 2 hours (Abbey and Rankin, 2011, Ostrowski et al., 1999, Pournot et al., 2011, Shin and Lee, 2011, Starkie et al., 2001, Suzuki et al., 2003). Immediately following a handball match, IL-6 increased by 170% (Marin et al., 2011) while immediately after a soccer match, IL-6 increased 180-600% with IL-10 increased by 125% and TNF- $\alpha$  by 280% (Andersson et al., 2010, Ispirilidis et al., 2008). After each match during a 3 day, 3 game basketball tournament IL-6 rose by 375-450% and IL-10 increasing by 250-300% before both returning to baseline values within 24 h (Montgomery et al., 2008a).

Along with inducing an inflammatory response, high-intensity training and competition results in high mechanical stress placed on a muscle which can damage muscle and connective tissue (Clarkson and Sayers, 1999). Eccentric loading occurs frequently in field sports such as soccer, rugby, field hockey and Australian football (AF) and the loading profile of eccentric contractions (high force with low fibre recruitment) is considered as a major causative factor in muscle damage due to the high mechanical stress placed on muscle fibres (Enoka, 1996). Muscle damage can also occur in multi-sprint field sports as a direct consequence of players colliding or contacting each other such as rugby or AF (Takarada, 2003).

The presence of muscle damage in team sport is commonly assessed through measuring the concentration of serum/plasma myoglobin ([Mb]) (Ascensao et al., 2011, Ascensao et al., 2008, Montgomery et al., 2008a, Rowsell et al., 2009, Takarada, 2003). In soccer, 1380-1400% increases in [Mb] have been observed 30 min post-match (Ascensao et al., 2008, Magalhaes et al., 2010) and during a 3 game basketball tournament played over consecutive days, [Mb] increased 460-510% immediately post-match before returning to baseline prior to the next game 24 h later (Montgomery et al., 2008a). Participation in competitive rugby matches also induced structural damage to muscle tissue (Takarada, 2003). After participating in several matches, [Mb] rose 1000% immediately post-match and remained elevated after 24 h (100%). The increase was dependent on the number of tackles with a greater number of tackles leading to a greater increase. When muscle damage in team sport athletes, as measured by increased [Mb] has been reported, this has corresponded with physical performance decrements. Immediately following a soccer match, decrements post-match of 10% in 20 m sprint have been observed (Ascensao et al., 2011, Ascensao et al., 2008) while repeat 20 m sprint has declined 1.8% (Rowsell et al., 2009), countermovement jump by 5.8-27% (Ascensao et al., 2011, Rowsell et al., 2009) and static jump by 9% (Ascensao et al., 2011). Muscle damage can limit physical performance in the days following exercise (Clarkson and Hubal, 2002), therefore post-exercise recovery is an important tool in allowing athletes

overcome this and in assisting preparation for their next match. Cold water immersion (CWI)

and contrast water therapy (CWT) are recovery methods commonly employed in athletic settings to reduce the effects of muscle damaging and intense exercise (see chapter 4 and 5) (Ascensao et al., 2011, Bailey et al., 2007, Gill et al., 2006, Ingram et al., 2009, King and Duffield, 2009, Montgomery et al., 2008b, Rowsell et al., 2009, Rowsell et al., 2011, Vaile et al., 2007, Vaile et al., 2008b). Both methods can assist in enhancing recovery of physical performance (see chapter 4 and 5) (Ascensao et al., 2011, Ingram et al., 2009, King and Duffield, 2009, Montgomery et al., 2008b) while alleviating symptoms of exercise-induced muscle soreness (see chapter 4 and 5) (Ascensao et al., 2011, Bailey et al., 2007, Ingram et al., 2009, Vaile et al., 2007) and reducing fatigue between exercise bouts (see chapters 4 and 5) (Montgomery et al., 2008b, Vaile et al., 2008b). Additionally, cold exposure can induce a number of positive physiological changes including reductions in localised swelling and oedema and indices of exercise-induced muscle damage (Ascensao et al., 2011, Bailey et al., 2007, Cote et al., 1988, Wilcock et al., 2006a). Cold water immersion can also have an additional impact on the cardiovascular system and the circulating blood volume (Bonde-Petersen et al., 1992, Peiffer et al., 2009, Vaile et al., 2010, Yanagisawa et al., 2003). When compared to hot water exposure, such as in CWT, cold water can reduce heart rate and cardiac output (Bonde-Petersen et al., 1992), reduce blood flow to both the upper and lower extremities (Peiffer et al., 2009, Vaile et al., 2010), increase peripheral vasoconstriction (Peiffer et al., 2009), decrease vessel diameter (Peiffer et al., 2009) and increase mean arterial pressure and total muscle peripheral resistance (Bonde-Petersen et al., 1992). When combined, the effects of CWI can further reduce oedema and localised swelling when compared to CWT.

For all the potential psychometric and performance benefits that CWI and CWT provide, questions still remain surrounding their use and a potentially negative impact on muscle regeneration. The inflammatory process is involved in muscle regeneration and adaptation (Tidball, 2005) and the concern is that cold temperatures will blunt the normal inflammatory process and therefore diminish subsequent muscle repair and regeneration. For team sport athletes using CWI or CWT, blunting muscle repair would be of concern.

Australian football is a physically demanding full contact sport which can result in substantial increases in soreness and fatigue as well as decreases in physical performance both postmatch (see chapter 5) (Dawson et al., 2005) and after training (see chapter 4). However, the damaging effect of AF participation and any related increase in acute inflammation is currently unknown, while the magnitude to which CWI and CWT may attenuate or moderate any changes also remains unclear. Therefore the purpose of this study is to determine the effects of CWI and CWT on muscle damage markers and acute inflammation after an AF match.

#### 6.2 Methods

#### 6.2.1 Subjects

Twenty-four professional male AF players ( $19.9 \pm 2.8$  years;  $80.8 \pm 8.1$  kg;  $186.4 \pm 6.4$  cm) volunteered and provided written consent to participate in this study. All players were free of injury and illness at the time of testing and during match participation. The study was approved by the Victoria University Human Research Ethics Committee and conformed to the Declaration of Helsinki.

#### 6.2.2 Design

The study was designed to evaluate the effectiveness of two commonly-used water recovery methods on limiting post-match markers of muscle damage and inflammation in AF. Two teams of 19 players participated in a full practice match (mean temperature 25.8°C; mean relative humidity 63%) consisting of 4 quarters resulting in a total match duration of 75 minutes.

Prior to the match, 24 of the competing players (12 from each team) were pre-selected for participation in the study. Selection was based on ensuring players from all playing positions were included and then pair matching these selections. This was done to ensure an equal number of players participating in the investigation competed on both teams.

Throughout the course of this investigation, participants provided blood samples for analysis 90 min prior to the match, immediately post-match as well as after 1, 24 and 48 h.

Upon completion of the match, the 24 players were randomly and equally assigned to one of three recovery interventions; Passive recovery (PAS, n=8), cold water immersion (CWI, n=8) or contrast water therapy (CWT, n=8). Players undertook their assigned recovery intervention immediately after they completed their post-match measures. During the 48 h following the practice match, all participating players undertook no further physical activity and were only allowed to undertake their prescribed recovery intervention.

#### 6.2.3 Blood sampling and preparation

Venous blood samples (8 mL) were drawn through venipuncture of a forearm vein while players were seated. 1 mL was then pippetted into an eppendorf tube and placed on ice for subsequent determination of hematocrit (Micro hematocrit, H. I. Clements, North Ryde, New South Wales, Australia) and haemoglobin (Hb 201+; HemoCue, Ängelholm, Sweden) concentration. Changes in plasma volume were calculated using hematocrit and haemoglobin values (Dill and Costill, 1974). The remaining sample was then centrifuged (4°C) at 4500 rev.min<sup>-1</sup> for 10 min to obtain serum. The serum was then aliquotted into secondary eppendorf tubes and stored at -80°C.

#### 6.2.4 Biochemical assays

Using commercially available assay kits, the concentration of myoglobin (Myoglobin Enzyme ImmunoAssay Test Kit, Oxis International Inc, Foster City California, USA) and the inflammatory cytokines IL-6, IL-10, IL-1 $\beta$  and TNF- $\alpha$  (Bio-Plex Pro Human cytokine assay, Bio-Rad Laboratories Pty Ltd, Hercules, California, USA) was determined. To avoid variations in assay conditions, each assay was performed in duplicate. All assays were conducted following multiple steps as outlined by the manufacturer. Blood samples were brought to room temperature and shaken vigorously to ensure the serum was able to be analysed appropriately. Once this had occurred, 6-8 standards were mixed, diluted and

prepared ready to be placed into a 96 well plate. Similarly all samples were prepared and diluted to a 1:4 ratio. Samples and standards were then placed into a 96 well plate. An enzyme conjugate reagent was placed into the myoglobin plate and in the case of cytokine assay, magnetic couple beads were then prepared added into each well. Plates were allowed to incubate for 30-45 min and then was washed 2-3 times. Following this a TMB reagent was placed into the myoglobin wells and antibodies were placed into each cytokine. Plates were then incubated for a further 20 min before being washed 3 times. Streptavidian-PE was then added to cytokine plates with a further incubation time of 20 min. A stop solution was added to each myoglobin well and mixed for 30 seconds while all cytokine plates were then washed 3 times. All plates were then placed in a plate reader where myoglobin and cytokine levels were determined.

#### 6.2.5 Interassay variation

The interassay variation for all assays is presented in Table 6.1. Interassay variation was calculated as follows: (standard deviation of all replicate measurements divided by the mean of all replicate measurements) x 100.

#### 6.2.6 Recovery interventions

A description of the post-training recovery interventions performed by athletes is outlined in chapter 3.

#### 6.2.7 Movement Data

A description of the method and equipment used to obtain movement data is outline in chapter 3.

#### 6.2.8 Statistical analysis

Data is presented as mean  $\pm$  standard deviation. A repeated measures general linear model was used to establish differences between treatments over time. Adjustments for multiple

comparisons were applied using the Bonferoni method. Data was analysed using SPSS version 20.0 and statistical significance was set at P < 0.05.

Measure	Number of replicates	Interassay variation
IL-1β	105	9.5%
IL-6	105	8.9%
IL-10	105	8.0%
TNF-α	105	10.4%
Mb	105	6.7%

Table 6.1: Interassay variation for inflammatory cytokines and myoglobin.

#### 6.3 Results

All players finished the match covering an average 8,012 m or 107 m.min<sup>-1</sup> (range 102.8-112.1 m.min<sup>-1</sup>) during the 75 min match with players on field for an average of 71 min (range 69-75 min).

Inflammatory cytokine results for 2 participants were considered as outliers as their baseline values were between 2.5-3 fold above the standard deviation of the group mean, while only 4 of the 5 required blood samples were obtained from a third player (due to nausea and feeling like fainting). As a result, data from these 3 players was excluded from the study. Statistical analysis was performed on the blood data from the remaining 21 players with the final groups consisting of: PAS n = 7, CWI n = 7 and CWT n = 7.

#### 6.3.1 Myoglobin

Immediately following the match, [Mb] increased in all 3 groups (Figure 6.1). Compared to pre-match, [Mb] substantially increased in all 3 groups with PAS increasing by 1594% (333  $\mu$ g.L<sup>-1</sup>, *P* = 0.0003), CWI by 1392% (328  $\mu$ g.L<sup>-1</sup>, *P* = 0.0002) and CWT by 1442% (333  $\mu$ g.L<sup>-1</sup>, *P* = 0.0002). Following the recovery interventions CWI had the greatest impact on lowering [Mb] with PAS being least effective. After 1h, [Mb] increased by a further 6.6% in the PAS group (355  $\mu$ g.L<sup>-1</sup>, *P* = 0.29). A non-significant decrease was observed following CWT (9.0%, 303  $\mu$ g.L<sup>-1</sup>, *P* = 0.18) while [Mb] was reduced substantially by CWI (20.2%, 262  $\mu$ g.L<sup>-1</sup>, *P* =

0.013) following CWI. Additionally, CWI substantially lowered [Mb] compared to PAS (P = 0.048). Myoglobin in all three intervention groups returned to baseline after 24 h and remained at these levels at 48 h.

#### 6.3.2 Interleukin-1β

Interleukin-1 $\beta$  was undetected in 14 of the 21 participants and therefore due to the small data set available, no statistical analysis was performed on IL-1 $\beta$  data (data not shown).

#### 6.3.3 Interleukin-6

Interleukin-6 was detected in all participants only immediately after the match and after 1 h. As such, statistical analysis was performed only on this data. Pre-match, IL-6 was undetectable however large increases were evident post-match in all 3 groups (Figure 6.2). Compared to post-match, IL-6 had decreased after 1 h by 89.5.0% in PAS (P = 0.001), by 90.8% in CWI (P = 0.0001) and by 91.6% in the CWT group (P = 0.0001) with no differences observed between interventions. Interleukin-6 was undetectable after 24 and 48 h.

#### 6.3.4 Interleukin-10

Interleukin-10 increased significantly immediately post-match with PAS increasing 439% (P = 0.0003), CWI 447% (P = 0.0002) and CWT 454% (P = 0.0002) with no differences observed between the recovery groups (Figure 6.3). After 1h IL-10 had returned to baseline in all groups and remained at that level at 24 and 48 h.

#### 6.3.5 Tumor necrosis factor-a

Similar to IL-1 $\beta$ , TNF- $\alpha$  was undetected in 15 of the 21 participants and therefore due to the small data set available, no statistical analysis was performed on TNF- $\alpha$  data (data not shown).

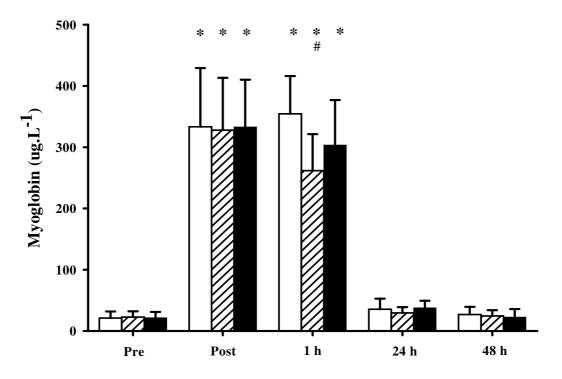


Figure 6.1: Comparison between groups for changes in myoglobin pre-match and throughout the 48 h recovery period. \* Significantly greater than pre-match (P < 0.001). \* Significant difference between PAS and CWI (P < 0.05). White bar = passive recovery, hatch bar = cold water immersion, lack bar = Contrast water immersion.

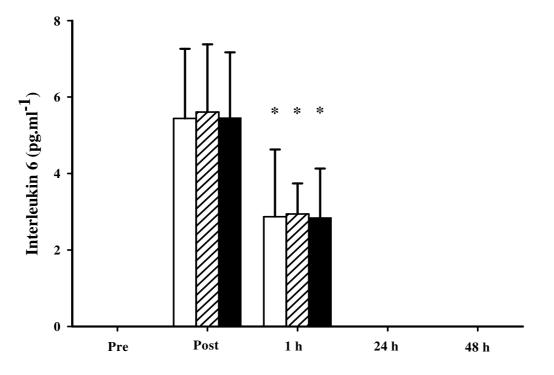


Figure 6.2: Comparison between groups for changes in interleukin-6 pre-match and throughout the 48 h recovery period. \* Significantly lower than post-match (P < 0.01). White bar = Passive recovery, Hatch bar = Cold water immersion, Black bar = Contrast water immersion.

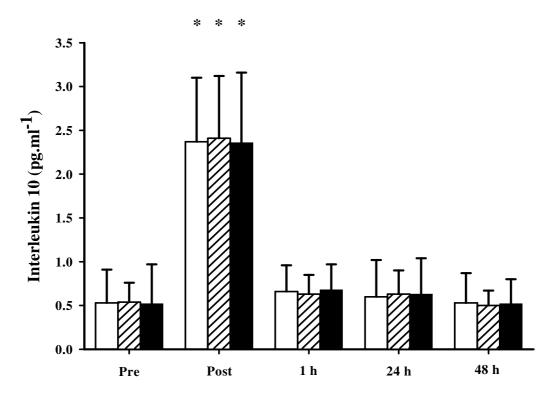


Figure 6.3: Comparison between groups for changes in interleukin-10 pre-match and throughout the 48 h recovery period. \* Significantly greater than pre-match (P < 0.001). White bar = Passive recovery, Hatch bar = Cold water immersion, Black bar = Contrast water immersion.

#### 6.4 Discussion

This is the first study to examine the effects of both CWI and CWT on muscle damage markers as well as on both pro- and anti-inflammatory cytokine concentrations after an AF match. Each of Mb, IL-10 and IL-6 increased substantially after an AF match and that the inflammatory process was unaffected by the type of recovery players undertook post-match.

Players covered an average of 107 m.min<sup>-1</sup> during the 75 min match which is similar to the lower range of distances covered in Australian Football League competition matches (Aughey, 2011b). This distance indicates that the practice match was at a sufficient intensity to replicate the demands of a typical in-season match.

Intense and damaging exercise results in an acute phase inflammatory response and it is clear than an AF match induces this response. Both [IL-6] and [IL-10] increased substantially in response to the match, and it was clear that compared to PAS, the magnitude and time course of the inflammatory response was not moderated by the use of either CWI or CWT. This lack of difference signifies that in well trained AF players, the application of cold temperatures via CWI and CWT did not disturb the normal inflammatory response. Interleukin-6 is an important moderator of satellite cell proliferation (Serrano et al., 2008) and as undertaking CWI and CWT led to no reduction in IL-6 compared to PAS, there was likely no blunting of the pathways leading to muscle repair and regeneration and no disturbance to the adaptive response resulting from the normal inflammatory response. This is a significant finding and indicates that both CWI and CWT should not be discounted as post-exercise recovery modalities. When used acutely, they do not have a negative impact on the normal inflammatory response and offer substantial benefits in reducing post-match soreness and fatigue improving physical performance (see chapter 5). Similar responses occur following repeated basketball matches where no differences in IL-6 and IL-10 were observed between a control or CWI group (Montgomery et al., 2008a) and also following a muscle damaging exercise protocol where CWI or CWT failed to elicit changes in IL-6 compared to a PAS recovery (Vaile et al., 2008c). Although likely similar to the acute use investigated here, the effects of prolonged use of either CWI or CWT are as yet unknown and requires further investigation.

Post-match changes in [IL-1 $\beta$ ] and/or [TNF- $\alpha$ ] were not detected in most players during this investigation. These results are not uncommon following prolonged and strenuous exercise, with low or no in [IL-1 $\beta$ ] and/or [TNF- $\alpha$ ] values being reported following 1, 2.5 and 6 h of running and following 2 h of strenuous cycling (Drenth et al., 1995, Ostrowski et al., 1998, Scott et al., 2011, Ullum et al., 1994). Additionally, the lack of detectable changes observed may have resulted from a lack of sensitivity in the kits used which has been confirmed by the manufacturer. Increases in pro-inflammatory cytokines are balanced by the release of the anti-inflammatory cytokine IL-10 (Jenkins et al., 1994). This in turn can up-regulate the release of the cytokine inhibitor IL-1ra and cause down regulation of IL-1 $\beta$  and TNF- $\alpha$  (Drenth et al., 1995, Jenkins et al., 1994) and may explain why both cytokines in this study were undetectable across all time points.

Increased serum/plasma [Mb] indicates damage to a muscle (Clarkson and Hubal, 2002, Clarkson and Sayers, 1999). During this investigation, substantial increases in [Mb] of between  $328-333 \ \mu g.L^{-1}$  were observed all 3 groups immediately following the match, similar in magnitude to that detected after several soccer matches (Ascensao et al., 2008, Magalhaes et al., 2010). The similarity of these results may be due to the activity profile where the AF players in this investigation covered 107 m.min<sup>-1</sup> compared to that of soccer matches where players can cover between 110 and 118 m.min<sup>-1</sup> (Bradley et al., 2009, Burgess et al., 2006).

The post-match use of both CWI and CWT both successfully reduced [Mb], similar to responses seen following after a soccer match and 90 min of shuttle running (Ascensao et al., 2011, Bailey et al., 2007). Furthermore, CWI substantially reduced [Mb] 1 h post-match compared to PAS similar to results following 90 min of team sport shuttle running (Bailey et al., 2007). The observed reductions in [Mb] for both CWI and CWT corresponded with improvements in post-match sprint performance (see chapter 5) where CWI was more successful at restoring repeat 20 m sprint and squat jump than CWT with both methods being more successful than PAS. It therefore appears that a reduction in muscle damage as measured by [Mb] corresponds with an improvement in sprint performance.

The mechanism(s) responsible for the attenuation of myoglobin by cold temperatures are unclear. Previously, cold application has been proposed to reduce the amount of post-exercise muscle damage and/or reduce the efflux of muscle proteins into the lymphatic system (Eston and Peters, 1999). Cold application is associated with a reduced permeability of cellular, lymphatic and capillary vessels and when combined with the physiological effects of hydrostatic pressure (Wilcock et al., 2006a), it is possible that CWI caused a reduction in protein efflux and may explain why CWI and CWT were more successful than PAS at attenuating [Mb] increases. Unsurprisingly, the application of heat can have an opposing effect by increasing blood flow and vasodilatation (Wilcock et al., 2006a). This can increase the efflux of proteins, and in turn increase the level of circulating Mb and may help explain the differences between the CWT and CWI groups in this investigation. Although the mechanisms leading to reduced [Mb] are not evident, it is clear that in well trained AF

players, reductions in [Mb] are positively related to physical performance (see chapter 5) which can only be of benefit for players in their preparation for their next training session or match.

#### 6.5 Conclusion

It is clear that participating in a game of AF induces muscle damage and an acute phase inflammatory response. This investigation established that in well trained team sport athletes, neither CWT nor CWI interfered with the regular post-exercise inflammatory response leading to muscle regeneration. This should allay any concerns that acute cold application will blunt the regenerative pathways associated with post-exercise inflammation. Throughout this investigation, CWI emerged as a more effective modality in attenuating post-match increases in myoglobin and when combined with the positive effects that CWI had on restoring physical performance (see chapter 5) it is recommended that 14 min of CWI be utilised as an acute recovery tool post-match in team sport athletes.

## CHAPTER 7. GENERAL DISCUSSION AND CONCLUSIONS

#### 7.1 Introduction

The effectiveness of water immersion modalities as a recovery method after exercise in team sports has been mixed. Therefore, this thesis investigated the efficacy of both cold water immersion and contrast water therapy as recovery tools after participation in Australian football training and a match. Specifically, the effectiveness of an acute application of CWI and CWT following an AF match and training in attenuating declines in physical performance post-exercise, reducing perceived muscle soreness and fatigue, as well as reducing markers of muscle damage and inflammation has been quantified for the first time. The findings of these investigations have been discussed in earlier chapters (chapters 4, 5 and 6); therefore this section will discuss the major results from the three investigations completed as part of this thesis and their practical applications.

#### 7.2 The effectiveness of water immersion in this thesis

This thesis determined that AF training and match participation leads to post-exercise decrements in psychometric and physical performance measures as well as inducing muscle damage and an acute inflammatory response. It also became clear that in well trained AF players, both CWI and CWT when used immediately post-exercise, were effective recovery modalities. Both interventions consistently proved more beneficial than a passive recovery in reducing muscle soreness and fatigue as well as enhancing physical performance, with CWI being the more effective. This outcome was consistent regardless of the stimulus/exercise load coming from a training session or from match play. Furthermore, both modalities reduced the appearance of muscle damage markers following an AF match with CWI again proving more successful.

Post-exercise recovery is aimed at promoting an athlete's readiness to train and compete by reducing factors that may be detrimental to performance while enhancing the recovery of those which are beneficial. Both recovery modalities were well tolerated by the AF players during this thesis and below is a summary of the effects of an acute post-exercise application of CWI and CWT on physical performance, psychometric measures and muscle damage and inflammation.

#### 7.2.1 Effects of water immersion on physical performance

It is clear that team sport activity results in reduced physical performance in the days following, with speed, agility and jump performance all reduced (Fatouros et al., 2010, Highton et al., 2009, Ispirlidis et al., 2008, Magalhaes et al., 2010, Twist and Eston, 2007). A reduced ability to perform these activities would most likely have a negative effect on match and training performance. This may manifest as an athlete not being able to out-sprint, out-jump or out-manoeuvre opponents leading to a turnover of possession, an opponent creating/breaking up a scoring opportunity or an opponent scoring, or simply an athlete not getting the full benefit of a training session/s.

In AF players, participation in both training and a match led to substantial decrements in repeat 20 m sprint performance while the match had a more pronounced effect on jump performance. The difference observed between jump performances from training to game may be related physical contact. Training involved no physical contact while the practice match was full contact therefore it is possible that this contact may have contributed to the post-match reductions in jump performance which were not seen following training. It was clear throughout this thesis that CWI was more successful than CWT in eliciting improvements in physical performance with PAS having the smallest effect. The results of this thesis support previous research where both CWI and CWT have been more effective than a thermoneutral/control or passive recovery at restoring sprint and jump performance following team sport or simulated team sport activity (Ascensao et al., 2011, Ingram et al., 2009, King and Duffield, 2009, Montgomery et al., 2008b).

#### 7.2.2 Effects of water immersion on psychometric measures

In athletes, reducing muscle soreness and/or perceptions of fatigue may lead to an overall increase in match/training performance. Increased mental fatigue prior to exercise can heighten maximal perceived exertion and cause participants to disengage from an exercise task early (Marcora et al., 2009). Poor lower limb comfort levels are strongly correlated with poor ratings of match performance in AF and rugby as measured by coach ratings ( $R^2 = 0.62$ ,  $P \le 0.001$ ), while good match ratings were not well correlated with lower limb comfort ( $R^2 = 0.25$ ) (Kinchington et al., 2012). This has implications for athletes feeling tired or sore going into a game or in the day/s after a game or heavy training session. Lessening fatigue is likely to result in a greater adherence to an exercise task while reducing soreness will diminish limb discomfort thus increasing physical output/performance.

Match participation and training induced considerable increases in perceived muscle soreness and fatigue immediately post-exercise which remained elevated in PAS throughout the 48 h recovery period. The acute application of CWI and CWT provided substantial benefits in reducing perceived muscle soreness and fatigue in well trained AF players, with CWI being more effective than CWT and PAS having the least effect. These results are in line with previous research where both interventions have proven more successful at reducing perceptions of soreness and fatigue after exercise (Bailey et al., 2007, Ingram et al., 2009, King and Duffield, 2009, Halson et al., 2008, Rowsell et al., 2009, Rowsell et al., 2011, Versey et al., 2011).

The magnitude of change in perceived muscle soreness and fatigue post-match appear to affect physical performance with improved sprint time being closely associated with reduced soreness or fatigue after 24 and 48 h. After 24 h, the percent change in total sprint time correlated highly (Hopkins, 2002) with changes in muscle soreness (r = 0.67) (Figure 7.1A) and fatigue (r = 0.66) (Figure 7.1B), while after 48 h, moderate to high correlations were observed in soreness (r = 0.53) (Figure 7.1C) and fatigue (r = 0.53) (Figure 7.1D). The magnitude of these correlations suggests that in a practical setting, there is a high (Hopkins,

2002) likelihood that repeat sprint ability will be negatively impacted by the presence of muscle soreness and a moderate-high (Hopkins, 2002) chance that sprint performance will be reduced by perceived fatigue. These correlations indicate that the higher the degree of soreness or fatigue experienced by an athlete, the greater the decrement in sprint performance. The effective reduction in muscle soreness and fatigue most likely contributed to enhanced physical performance in AF players. Australian football matches are longer in duration, have a greater running profile per minute of match play (Aughey, 2011a), and training can more closely match the activity profile of competitive matches than other team sports (Farrow, 2008, Hartwig et al., 2011) therefore it is highly probable that changes in soreness/fatigue would also contribute to enhancing physical performance in other team sports athletes. In this thesis, ratings of perceived exertion, muscle soreness and fatigue were higher following the match compared to training. This indicates that players found this task more taxing and more fatiguing and possibly induced more low frequency fatigue. This type of fatigue can take hours or days to resolve and the combination of this fatigue and the physical contact experienced during the match may have lead to these ratings being enhanced as well as the decrement in jump performance seen only post-match..



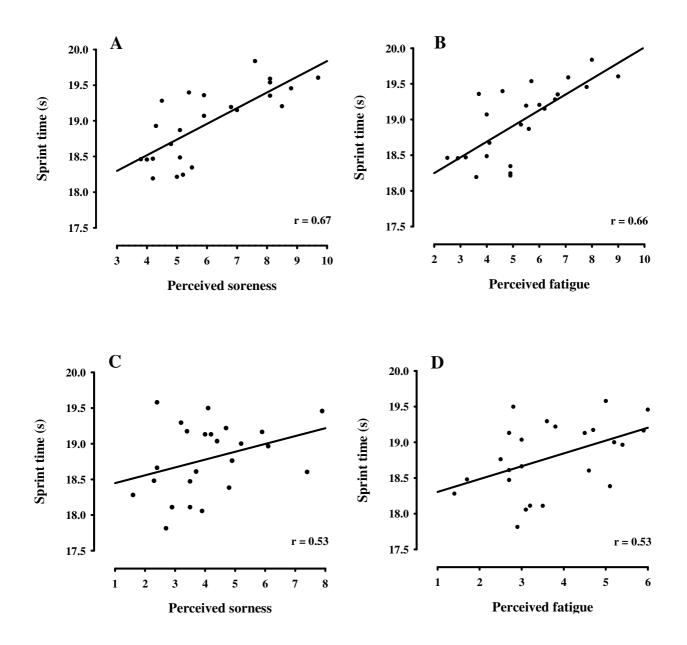


Figure 7.1: Correlations between perceived soreness and fatigue and sprint time. (A) Sprint time and perceived soreness after 24 h. (B) Sprint time and perceived fatigue after 24 h. (C) Sprint time and perceived soreness after 48 h. (D) Sprint time and perceived fatigue after 48 h.

#### 7.2.3 Effects of water immersion on muscle damage and inflammation

Immediately upon completion of the match, serum myoglobin was significantly elevated in all 3 recovery groups similar to levels detected after soccer match participation (Ascensao et al., 2008, Magalhaes et al., 2010). Cold water immersion had a greater impact on lowering [Mb] after 1 h than CWT, with CWI significantly reducing [Mb] compared to PAS (p = 0.48). This has also been observed following team sport and simulated team sport exercise (Ascensao et al., 2010).

al., 2011, Bailey et al., 2007). Myoglobin concentration in all three intervention groups returned to baseline after 24 h and remained at these levels at 48 h similar to previous research (Ascensao et al., 2008, Magalhaes et al., 2010).

Both IL-6 and IL-10 substantially increased in the hour post-match, however unlike Mb, the use of CWI and CWT did not moderate the magnitude and time course of the normal inflammatory response compared to PAS. Comparable responses have also been observed where CWI did not influence IL-6 or IL-10 after a series of basketball matches (Montgomery et al., 2008a) and where IL-6 was not moderated by CWT or CWI following muscle damaging leg exercise (Vaile et al., 2008c).

The degree of muscle damage may also be related to sprint performance in the days following an AF match. The percent change in Mb after 1h was highly correlated with total post-match sprint time at 24 h (r = 0.63) (Figure 7.2A) and moderately correlated with sprint performance after 48 h (r = 0.51) (Figure 7.2B). Therefore, an improvement in sprint performance is moderately related to a reduction in post-match [Mb].

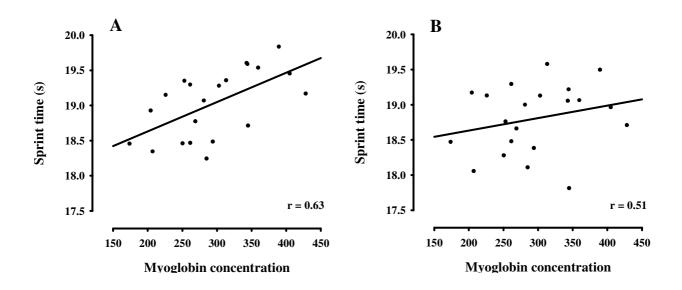


Figure 7.2: Correlations between myoglobin concentration and sprint time. (A) Sprint time after 24 h and myoglobin after 1 h. (B) Sprint time after 48 h and myoglobin after 1 h.

#### 7.3 The belief effect, water immersion and AF athletes

It was not possible to incorporate a placebo in this thesis due to the nature of water immersion or a control water condition due to limited space and time available with the athletes. Because of this, a treatment effect cannot be dismissed. Some of the athletes from the sample indicated their preference for using CWT, and others CWI. The use of CWI and CWT has become more prevalent in AF and based on this, and any beliefs or preconceptions regarding recovery, athletes may have expected their preferred modality to provide greater benefits over another intervention. In one study investigating the effects of belief on subjective ratings of wine, results indicate that increasing wine price improves subjective reports of flavour and pleasantness (Plassmann et al., 2008). Although that study was an investigation in the effect of marketing actions influencing subjective outcomes, it is possible that subjective ratings of soreness or fatigue may be influenced in athletes who have preconceptions or prefer one modality over another. Although beliefs into the effects of recovery were not measured during this thesis, an athlete's bias may impact psychometric measures such as pain (Wager, 2005). However any such preconceptions may have had a limited effect on influencing physical performance. It has been suggested that even when sore, players may still be able to produce one-off efforts equal or close to their best, and that repeat-effort tests may be a better way to assess functional decrements (Dawson et al., 2005). Repeat sprint and jump tests were used during this thesis with results indicating that as a group, players performed better in jump and sprint tests following CWI, therefore perceptions of recovery benefit may not be a main driver in subsequent athletic performance. Additionally, recovery beliefs would not affect the clearance of muscle damage markers post-match.

Although not utilised during this thesis, the use of a control water condition (thermoneutral immersion) has been previously investigated in team sport (Ascensao et al., 2011, Rowsell et al., 2009, Rowsell et al., 2011). During these studies, CWI was more effective than thermoneutral immersion at attenuating psychometric and physical performance and reducing [Mb] (Ascensao et al., 2011, Rowsell et al., 2009, Rowsell et al., 2011). Therefore it is

unlikely that the lack of a control water protocol played a major role in the outcomes observed in this thesis.

#### 7.4 The generalisability of findings from the exercise tasks utilised in this thesis

#### 7.4.1 Low total game time and distance covered compared to typical AF matches

The length of the match in this thesis was shorter than a typical AF match and this therefore needs to be considered when interpreting the effectiveness of CWI and CWT. Although the players covered distances per minute of match play considered to be on the lower end of Australian Football League competition matches (Aughey, 2011b) the total distance covered was lower than typical for AF (8012 m vs. 12,700-13,000 m). The degree of muscle damage sustained is related to an increase in running distance (Pandolf, 1998), therefore, an increase in total game time and subsequent running distance would likely result in players becoming more fatigued, sore and have greater muscle damage and inflammation post-match. The net result of this could be a greater reduction in physical performance in the days following a match and an increase in the time course for physical recovery. Therefore, the reduced game time in this thesis may have led to an underestimation of the effects of playing a full competitive AF match and in turn may underestimate the positive influence of both modalities on the recovery process. Alternatively, CWI/CWT may have only been successful due to the lower stimulus from the shorter total game time. This appears unlikely as both modalities have been successful following other team sport and simulated team sport events and during tournament scenarios (Ascensao et al., 2011, Bailey et al., 2007, Montgomery et al., 2008b, Rowsell et al., 2009, Rowsell et al., 2011), however the use of CWI and CWT after a full competition match is something which should be investigated in the future.

#### 7.4.2 Relatively young age of AF athletes in this thesis

The age of the participants used in this thesis is factor for consideration. The average age of the group was relatively young (20-21 yrs) compared to other AF squads (23-25 yrs), and as such, there may be an effect of training age on the magnitude of change in measures such as

soreness, fatigue and repeat-sprint performance. Two years of training in professional rugby union players (24.4-26.4 yrs) resulted in an overall increase in maximal upper and lower body strength (6.5-11.5%) with the magnitude of improvement in lower body strength positively related to a change in lean mass ( $p \le 0.05$ ) (Appleby et al., 2012). Sprint momentum, (a product of 10 m velocity and body mass) can discriminate between national and state level rugby league players (Baker and Newton, 2008) while in a more mature squad of professional rugby players (24.4 yrs), athletes effectively maintained upper body strength while slightly increasing lower body strength (8.5%) over the course of a 13 week competitive season (Argus et al., 2009). These results indicate that more developed and stronger athletes may be more resilient and able to withstand the loading and fatiguing nature of a team sport season while still enabling them to perform maximally more often in physical performance tests. Within the sample used in this thesis, 5 players were 23 yrs or older at the time of testing (27.8 yrs, 26.5 yrs, 24.8 yrs, 23.6 yrs and 23 yrs) nevertheless the group mean was relatively young. Such diversity in age in an AF squad is typical, however a more physically developed playing group may be more prepared to handle the demands of AF training/matches (Hrysomallis and Buttifant, 2012).

Changes in performance and psychometric variables may be less pronounced in more mature players and the effectiveness of CWI/CWT in attenuating these measures may have been enhanced due the young squad utilised in this thesis. Therefore, CWI/CWT may be of more benefit to younger athletes, less resilient athletes or athletes returning from injury who may be slightly detrained. Future research should include a broader age of athletes or athletes from a number of professional teams.

#### 7.4.3 Timing of studies in this thesis

The timing of the completed studies may have influenced the results obtained. All studies were conducted during the pre-season, which although physically taxing, may not induce the same physical loading and accumulation of fatigue that occurs during a competitive season. The effect of training and playing during a competitive AF season can have a negative impact

on power performance. The demands of a season resulted in upper body power in an AF squad being reduced by 4% in young players (1-3 yrs experience) while no effect was evident in older players (3+ yrs experience) (Hrysomallis and Buttifant, 2012). This indicates that younger players may be less able to cope with the demands of a season and this may be due to an overall accumulation of load or neuromuscular fatigue (Cormack et al., 2008b). Neuromuscular fatigue can accumulate during periods of an AF season and present as a reduction in lower body power (jump performance) even when regular weekly recovery takes place (Cormack et al., 2008b). When used acutely, both CWI and CWT effectively attenuated decreases in countermovement and static jump performance as well as repeat-sprint time, therefore using CWI/CWT during the season may be of greater benefit to younger athletes or the frequency of its use may have to increase during periods of greater neuromuscular fatigue.

#### 7.4.4 Environmental considerations

All training and match participation in this thesis took place in warm conditions (24.0-25.8°C, 63% humidity) and although not measured, this heat may have unduly increased thermal sensation and core temperature. Compared to AF matches in cooler conditions ( $17\pm4^{\circ}$ C) a small additional increase in core temperature occurs at the end of each quarter (0.22-0.40) during hot conditions ( $28\pm2^{\circ}$ C) (Aughey and McKenna, 2008). Similarly core temperature is additionally elevated following training in hot conditions ( $32.6\pm2.5^{\circ}$ C) versus cold conditions ( $12.3\pm2.4^{\circ}$ C) (Aughey and McKenna, 2008). The use of CWI during hot conditions is effective in reducing thermal sensation compared to an active recovery immediately post immersion (172-400%) and 40 min following immersion (113-145%) in cyclists (Vaile et al., 2008a). Therefore it is possible that the use of cold water may have provided an additional benefit in decreasing thermal sensation and in reducing perceived soreness and fatigue above that which may be expected in cooler climates. This cannot be dismissed, however both CWI and CWT have been effective at attenuating perceptual measures following 80 min of simulated team sport running in  $19.8\pm1.5^{\circ}$ C and  $41\pm12\%$  humidity (Ingram et al., 2009) and

also in indoor sports where temperatures would be cooler and less humid (King and Duffield, 2009, Montgomery et al., 2008a, Montgomery et al., 2008b).

Australian football pre-season training and early season competition matches take place during the summer months when temperatures would be similar if not warmer than those experienced during this thesis. A report into the effects of heat on AF players during matches and training recommends players undertake appropriate heat acclimatisation prior to playing in the heat (Aughey and McKenna, 2008). Given that the studies comprising this thesis were all conducted at the end of summer, it is reasonable to assume participants would have become accustomed to exercising in warm conditions and would have fully adapted to training/playing in these conditions (Armstrong and Maresh, 1991, Pandolf, 1998, Sunderland et al., 2008). It is not anticipated therefore that the acute application of CWI/CWT during these studies provided any additional benefits in decreasing thermal strain and/or perceptions of soreness or fatigue over that which would be expected in cooler climates.

The effect of training/playing in warm conditions is unlikely to have inflated the inflammatory response over that seen in cooler conditions, thus overestimating the effects of CWI/CWT. Inflammatory cytokines increased after prolonged exercise with environmental conditions ranging from 12°C with 60-70% humidity (Drenth et al., 1995, Suzuki et al., 2003), to 17°C (Ostrowski et al., 1999) and in 21.2°C and 70% humidity (Starkie et al., 2001). Therefore, based on these results, and the fact that all players would have been acclimatised to exercising in the heat, it is likely that ambient temperature played little role in further increasing the normal inflammatory response.

#### 7.5 Conclusions and practical applications

The findings of this thesis provide direct evidence on the effectiveness of CWI and CWT in assisting recovery in Australian footballers. This thesis concluded that a single application of 14 min of either CWI or CWT can enhance the recovery of jump performance after an AF match, while after training and a competitive match, both interventions attenuate decreases in repeat-sprint performance, reduced perceptions of fatigue and alleviated muscle soreness. It was also clear that CWI was the more successful. Neither CWI nor CWT affected post-match inflammation and as a result, it is likely that neither would blunt the normal inflammatory response leading to muscle repair following damaging exercise. Additionally, both modalities successfully attenuated the increase in [Mb] post-match compared to a passive recovery with CWI being the more successful. Furthermore, post-match changes in perceived fatigue, muscle soreness and muscle damage were strong indicators of repeat-sprint performance in the days following. It is clear from the results of this thesis that CWI was a more effective intervention after AF training and post-match than either CWT or a passive recovery for restoring physical performance, enhancing recovery of psychometric measures and limiting markers of muscle damage. The enhancement in physical and psychometric performance may lead to athletes being able to perform at their maximum in training, leading a greater training effect, or being able to perform maximally during a match with this enhancement unlikely to be restricted to just AF players.

The specific conclusions and practical applications of this thesis are:

#### Chapter 4/Study 1

#### Conclusions

1. Participation in AF training results in post-exercise increases in perceptions of muscle soreness and fatigue as well as a decline in repeat 20 m sprint performance which can persist for up to 48 h.

2. A single 14 min application of either CWI or CWT immediately following AF training can attenuate declines in repeat-sprint performance, reduce perceptions of fatigue and alleviate muscle soreness more effectively than a passive recovery during a 48 h recovery period.

3. In AF players, CWI post-training was more effective than CWT for enhancing physical and psychometric performance with a passive recovery being the poorest.

#### Practical Applications

1. Both CWI and CWT should be considered as effective modalities in restoring physical and psychometric deficits associated with AF.

2. Following training, CWI provides a greater recovery benefit than CWT, therefore the use of 14 min of CWI is recommended for enhancing recovery in AF players after training.

#### Chapter 5/Study 2

#### **Conclusions**

1. Participating in an AF match increases perceptions of muscle soreness and fatigue and results in post-exercise declines in countermovement and static jump performance and repeat 20 m sprint performance all of which can persist for up to 48 h post-match.

2. When applied as a single 14 min application immediately post-match, both CWI and CWT were more effective at relieving muscle soreness, diminishing fatigue and attenuating repeat-sprint and jump performance compared to a passive recovery during a 48 h recovery period.

3. Reductions in both perceived muscle soreness and fatigue correlates highly with improved sprint time 24 h post-match and correlates moderately-highly after 48 h.

4. Physical and psychometric recovery was more effectively enhanced by the use of CWI in AF players compared to CWT, with a passive recovery being the poorest.

#### Practical Applications

1. Both CWI and CWT should be considered as effective modalities in restoring physical and psychometric deficits associated with AF.

2. Cold water immersion provides a greater recovery benefit than CWT after an AF match, therefore use of 14 min of CWI is recommended for enhancing recovery in AF players

#### Chapter 6/Study 3

#### **Conclusions**

1. Australian football match participation results in substantial post-exercise increases in the markers of both acute inflammation (interleukin-6 and interleukin-10) and muscle damage (myoglobin).

2. One hour after the match, increases in myoglobin were more effectively attenuated by a single 14 min application of CWI and CWT than a passive recovery with CWI being the most effective.

3. Neither CWI nor CWT had any effect in attenuating IL-6 or IL-10 after 1 h compared to a passive recovery.

4. The magnitude of muscle damage (myoglobin) 1 h post-match is a strong indicator of sprint performance in the 48 following an AF match.

5. The normal post-exercise inflammatory response leading to muscle regeneration was not interrupted by either CWI or CWT compared to a passive recovery.

#### Practical Applications

1. Both CWI and CWT should be considered as effective modalities in decreasing markers of muscle damage and while not disrupting the normal inflammatory response in AF players after a match.

2. Following training, CWI provides a greater recovery benefit therefore, I recommend the use of 14 min of CWI for enhancing recovery in AF players

#### **CHAPTER 8. DIRECTIONS FOR FUTURE RESEARCH**

Research conducted for this thesis has investigated the efficacy of two commonly used water recovery interventions following AF training and a match. It was found that cold water immersion was the more effective modality after training and a match for attenuating performance declines, restoring psychometric deficits and clearing markers of muscle damage. Findings from this thesis provide important data and understanding in the area of team sport recovery as well as adding to the overall knowledge base of water recovery. The findings of this thesis can, however, be built on in the future. Potential areas of further investigations are discussed below:

### 8.1 Effects of multiple exposures of CWI and CWT on recovery in team sport athletes

It is clear from the findings in this thesis that a single 14 min application of CWI or CWT can enhance the recovery in AF players compared to a passive recovery over a 48 h period. One previous investigation in AF has looked at the effects of multiple water exposures on recovery. Players undertook an immediate post-match recovery (CWT or pool) followed by a next day pool recovery session with little difference between these protocols and a next day pool session alone (Dawson et al., 2005). Although multiple exposures have been previously investigated, the benefits of multiple CWI and/or CWT exposures in AF athletes are unknown. This may be particularly relevant during the pre-season phase of a team sport program where athletes typically train with a short turn around (i.e. 24 h) or train multiple times during the same day. Additionally, player recovery over a number of weeks could also be monitored.

# 8.2 Elucidating optimal CWI and/or CWT immersion times for recovery in team sport athletes

During this thesis 14 min of CWI or CWT enhanced the recovery of well-trained team sport athletes. However, it was not possible to test the efficacy of any other immersion times. As such, the efficacy of different periods of CWI and/or CWT needs to be investigated. Previous team sport investigations have utilised 5 and 6 min of CWI and CWT with mixed results, therefore potential time periods for examination include 8, 10 and 12 min with the aim being to elucidate their effectiveness compared to the 14 min utilised during this thesis. Athletes may have limited time for post-match recovery particularly when travel is required soon after the completion of matches therefore, determining the effectiveness of shorter time periods may allow for athletes to undertake an effective recovery given any imposed time constraints.

# 8.3 Determine effects of effects of multiple exposures of CWI and CWT on muscle adaptation in team sport athletes

Concerns currently exist surrounding the chronic use of CWI and possible detrimental effects with regards to muscle adaptation. In sedentary subjects, regular and numerous uses of 20 min CWI at 5°C blunted some training adaptations over a 4-6 week period (Yamane et al., 2006). This may be of some concern to athletes' and coaches, however the recovery protocol and length of immersion is not representative of typical team sport recovery.

After completing a match, the inflammatory process was not altered as a result of CWI or CWT in this thesis however the chronic effects of CWI or CWT is unknown. Therefore the effect of chronic CWI use on long term muscle adaptation (such as muscle strength) in well trained team sport athletes is an area I believe needs thorough investigation.

#### REFERENCES

ABBEY, E. L. & RANKIN, J. W. 2011. Effect of Quercetin Supplementation on Repeated-Sprint Performance, Xanthine Oxidase Activity, and Inflammation. *International Journal of Sport Nutrition and Exercise Metabolism*, 21, 91-96.

ALGAFLY, A. A. & GEORGE, K. P. 2007. The effect of cryotherapy on nerve conduction velocity, pain threshold and pain tolerance. *British Journal of Sports Medicine*, 41, 365-369.

ALLEN, D. G. 2001. Eccentric muscle damage: mechanisms of early reduction of force. *Acta Physiologica Scandanavica*, 171, 311-319.

ALLEN, D. G., LAMB, G. D. & WESTERBLAD, H. 2008a. Impaired calcum release during fatigue. *Journal of applied physiology*, 104, 296-305.

ALLEN, D. G., LAMB, G. D. & WESTERBLAD, H. 2008b. Skeletal muscle fatigue: cellular mechanisms. *Physiological Reviews*, 88, 287-332.

ALLEN, D. G. & WESTERBLAD, H. 2001. Role of phosphate and calcium stores in muscle fatigue. *Journal of Physiology*, 536, 657-65.

ANDERSSON, H., BOHN, S. K., RAASTAD, T., PAULSEN, G., BLOMHOFF, R.
 & KADI, F. 2010. Differences in the inflammatory plasma cytokine response following two elite female soccer games separated by a 72-h recovery. *Scandinavian Journal of Medicine & Science in Sports*, 20, 740-747.

ANDERSSON, H., RAASTAD, T., NILSSON, J., PAULSEN, G., GARTHE, I. & KADI, F. 2008. Neuromuscular fatigue and recovery in elite female soccer: effects of active recovery. *Medicine & Science in Sports & Exercise*, 40, 372-380.

AOI, W., NAITO, Y., TAKANAMI, Y., KAWAI, Y., SAKUMA, K., ICHIKAWA, H., YOSHIDI, N. & YOSHIKAWA, T. 2004. Oxidative stress and delayedonset muscle damage after exercise. *Free Radical Biology & Medicine*, 37, 480-487.

APPLEBY, B., NEWTON, R. U. & CORMIE, P. 2012. Changes in Strength over a 2-Year Period in Professional Rugby Union Players. *Journal of strength and conditioning research / National Strength & Conditioning Association*, 26, 2538-46.

ARGUS, C. K., GILL, N. D., KEOGH, J. W., HOPKINS, W. G. & BEAVEN, C. M. 2009. Changes in strength, power, and steroid hormones during a professional rugby union competition. *Journal of strength and conditioning research / National Strength & Conditioning Association*, 23, 1583-92.

ARMSTRONG, L. E. & MARESH, C. M. 1991. The induction and decay of heat acclimatisation in trained athletes. *Sports Medicine*, 12, 302-12.

ASCENSAO, A., LEITE, M., REBELO, A. N., MAGALHAES, S. & MAGALHAES, J. 2011. Effects of cold water immersion on the recovery of physical performance and muscle damage following a one-off soccer match. *Journal of Sports Sciences*, 29, 217-25.

ASCENSAO, A., REBELO, A., OLIVEIRA, E., MARQUES, F., PEREIRA, L. & MAGALHAES, J. 2008. Biochemical impact of a soccer match - analysis of

oxidative stress and muscle damage markers throughout recovery. *Clinical biochemistry*, 41, 841-851.

- AUGHEY, R. J. 2010. Australian football player work rate: evidence of fatigue and pacing? *International Journal of Sports Physiology and Performance*, **5**, 394-405.
- AUGHEY, R. J. 2011a. Applications of GPS technologies to field sports. International Journal of Sports Physiology and Performance, 6, 295-310.
- AUGHEY, R. J. 2011b. Increased High-Intensity Activity in Elite Australian Football Finals Matches. *International Journal of Sports Physiology and Performance*, 6, 367-379.
- AUGHEY, R. J. & MCKENNA, M. J. 2008. AFL research report The effects of heat on AFL plyers' during matches and training.
- BAILEY, D., ERITH, S., GRIFFIN, P., DOWSON, A., BREWER, D., GANT, N. & WILLIAMS, C. 2007. Influence of cold-water immersion on indicies of muscle damage following prolonged intermittent shuttle running. *Journal of Sports Sciences*, 25, 1163-1170.
- BAILEY, D. M., WILLIAMS, C., BETTS, J. A., THOMPSON, D. & HURST, T. L. 2011. Oxidative stress, inflammation and recovery of muscle function after damaging exercise: effect of 6-week mixed antioxidant supplementation. *European Journal of Applied Physiology*, 111, 925-936.
- BAKER, D. G. & NEWTON, R. U. 2008. Comparison of lower body strength, power, acceleration, speed, agility, and sprint momentum to describe and compare playing rank among professional rugby league players. *Journal of strength and conditioning research / National Strength & Conditioning Association*, 22, 153-8.
- BALNAVE, C. D. & THOMPSON, M. W. 1993. Effect of training on eccentric exercise-induced muscle damage. *Journal of applied physiology*, 75, 1545-51.
- BALOG, E. M. 2010. Excitation-Contraction Coupling and Minor Triadic Proteins in Low-Frequency Fatigue. *Exercise and Sport Sciences Reviews*, 38, 135-142.
- BALSOM, P. D., GAITANOS, G. C., SODERLUND, K. & EKBLOM, B. 1999. High-intensity exercise and muscle glycogen availability in humans. *Acta Physiologica Scandanavica*, 165, 337-45.
- BANGSBO, J. 1994. The physiology of soccer with special reference to intense intermittent exercise. *Acta Physiologica Scandanavica*, 151, 1-156.
- BANGSBO, J., IAIA, F. & KRUSTRUP, P. 2007. Metabolic response and fatigue in soccer. *International Journal of Sports Physiology and Performance*, 2, 111-127.
- BANGSBO, J., MOHR, M. & KRUSTRUP, P. 2006. Physical and metabolic demands of training and match-play in the elite football player. *Journal of Sports Sciences*, 24, 665-74.
- BARNETT, A. 2006. Using recovery modalities between training sessions in elite athletes does it help? *Sports Medicine*, 36, 781-796.
- BASU, T. K., TEMPLE, N. J., GARG, M. L. & (EDS.) 1999. Antioxidants in Human Health and Disease, New York, CABI Publishing.
- BEELEN, A. & SARGEANT, A. J. 1991. Effect of lowered muscle temperature on the physiological response to exercise in men. *European Journal of Applied Physiology and Occupational Physiology*, 63, 387-92.
- BLEAKLEY, C., MCDONOUGH, S. & MACAULEY, D. 2004. The use of ice in the treatment of acute soft-tissue injury: a systematic review of randomised control trials. *American Journal of Sports Medicine*, 32, 251-261.

- BONDE-PETERSEN, F., SCHULTZ-PEDERSEN, L. & DRAGSTED, N. 1992. Peripheral and central blood flow in man during cold, thermoneutral, and hot water immersion. *Aviation, Space, And Environmental Medicine* 63, 346-50.
- BORG, G. 1990. Psychophysical scaling with applications in physical work and the perception of exertion. *Scandinavian Journal of Work, Environment & Health,* 16 Suppl 1, 55-8.
- BOYD, L. J., BALL, K., GALLAHER, E. L., STEPTO, N. K. & AUGHEY, R. J. Unpublished observations.
- BRADLEY, P. S., SHELDON, W., WOOSTER, B., OLSEN, P., BOANAS, P. & KRUSTRUP, P. 2009. High-intensity running in English FA Premier League soccer matches. *Journal of Sports Sciences*, 27, 159-68.
- BRENNER, I. K., NATALE, V. M., VASILIOU, P., MOLDOVEANU, A. I., SHEK, P. N. & SHEPHARD, R. J. 1999. Impact of three different types of exercise on components of the inflammatory response. *European journal of applied physiology and occupational physiology*, 80, 452-60.
- BUCHHEIT, M., LAURSEN, P. B., KUHNLE, J., RUCH, D., RENAUD, C. & AHMAIDI, S. 2009. Game-based Training in Young Elite Handball Players. *International journal of sports medicine*, 30, 251-258.
- BUDD, G. M. 2008. Wet-bulb globe temperature (WBGT) its history and its limitations. *Journal of Science and Medicine in Sport*, 11, 20-32.
- BUGAJ, R. 1975. The cooling, analgesic, and rewarming effects of ice massage on localized skin. *Physical therapy*, 55, 11-9.
- BURGESS, D. J., NAUGHTON, G. & NORTON, K. I. 2006. Profile of movement demands of national football players in Australia. *Journal of science and medicine in sport / Sports Medicine Australia*, 9, 334-41.
- BYRNE, C. & ESTON, R. 2002a. The effect of exercise-induced muscle damage on isometric and dynamic knee extensor strength and vertical jump performance. *Journal of Sports Sciences*, 20, 417-25.
- BYRNE, C. & ESTON, R. 2002b. Maximal-intensity isometric and dynamic exercise performance after eccentric muscle actions. *Journal of Sports Sciences*, 20, 951-9.
- BYRNE, C., ESTON, R. G. & EDWARDS, R. H. 2001. Characteristics of isometric and dynamic strength loss following eccentric exercise-induced muscle damage. *Scandinavian Journal of Medicine and Science in Sports*, 11, 134-40.
- BYRNE, C., TWIST, C. & ESTON, R. 2004. Neuromuscular function after exerciseinduced damage. *Sports Medicine*, 31, 49-69.
- CANNON, J. G., EVANS, W. J., HUGHES, V. A., MEREDITH, C. N. & DINARELLO, C. A. 1986. Physiological mechanisms contributing to increased interleukin-1 secretion. *Journal of applied physiology*, 61, 1869-74.
- CANNON, J. G. & ST PIERRE, B. A. 1998. Cytokines in exertion-induced skeletal muscle injury. *Molecular and cellular biochemistry*, 179, 159-67.
- CAVAILLON, J. M. 1994. Cytokines and macrophages. *Biomedicine & pharmacotherapy*, 48, 445-53.
- CESARI, M., PENNINX, B. W., PAHOR, M., LAURETANI, F., CORSI, A. M., RHYS WILLIAMS, G., GURALNIK, J. M. & FERRUCCI, L. 2004. Inflammatory markers and physical performance in older persons: the InCHIANTI study. *The journals of gerontology. Series A, Biological sciences and medical sciences*, 59, 242-8.

- CHEETHAM, M. E., BOOBIS, L. H., BROOKS, S. & WILLIAMS, C. 1986. Human muscle metabolism during sprint running. *Journal of applied physiology*, 61, 54-60.
- CHEN, T. C., NOSA, K. & TU, Y. H. 2007. Changes in running economy following downhill running. *Journal of Sports Sciences*, 25, 55-63.
- CHEN, T. C., NOSAKA, K. & JUNG-CHARNG, L. 2005. Effects of immobilization and active mobilization on recovery of muscle after eccentric exercise. *Journal of exercise science and fitness*, 3.
- CHEUNG, K., HUME, P. & MAXWELL, L. 2003. Delayed onset muscle soreness : treatment strategies and performance factors. *Sports Medicine*, 33, 145-64.
- CHLEBOUN, G. S., HOWELL, J. N., CONATSER, R. R. & GIESEY, J. J. 1998. Relationship between muscle swelling and stiffness after eccentric exercise. *Medicine & Science in Sports & Exercise*, 30, 529-535.
- CLARKSON, P. M. & HUBAL, M. J. 2002. Exercise-induced muscle damage in humans. *American Journal of Physical Medicine and Rehabilitation*, 81, S52-S69.
- CLARKSON, P. M., NOSAKA, K. & BRAUN, B. 1992. Muscle Function after Exercise-Induced Muscle Damage and Rapid Adaptation. *Medicine and Science in Sports and Exercise*, 24, 512-520.
- CLARKSON, P. M. & SAYERS, S. P. 1999. Etiology of exercise-induced muscle damage. *Canadian Journal of Applied Physiology*, 24, 234-248.
- CLAUSEN, T. 2003. Na+-K+ pump regulation and skeletal muscle contractility. *Physiological reviews*, 83, 1269-324.
- CLOSE, G. L., ASHTON, T., CABLE, T., DORAN, D. & MACLAREN, D. P. M. 2004. Eccentric exercise, isokinetic muscle torque and delayed onset muscle soreness: the role of reactive oxygen species. *European Journal of Applied Physiology*, 91, 615-621.
- CLOSE, G. L., ASHTON, T., MCARDLE, A. & MACLAREN, D. P. M. 2005. The emerging role of free radicals in delayed onset muscle soreness and contraction-induced muscle injury. *Comparative Biochemistry and Physiology*, Part A 142, 257-266.
- COCHRANE, D. J. 2004. Alternating hot and cold water immersion for athlete recovery: a review. *Physical Therapy in Sport*, 5, 26-32.
- CONNOLLY, D. A. J., SAYERS, S. P. & MCHUGH, M. P. 2005. Treatment and prevention of delayed onset muscle soreness. *Journal of Strength and Conditioning Research*, 17, 197-208.
- CORMACK, S. J., NEWTON, R. U. & MCGUIGAN, M. R. 2008a. Neuromuscular and endocrine responses of elite players to an Australian Rules football match. *International Journal of Sports Physiology and Performance*, 3, 439-453.
- CORMACK, S. J., NEWTON, R. U., MCGUIGAN, M. R. & CORMIE, P. 2008b. Neuromuscular and endocrine responses of elite players during an Australian Rules football season. *International Journal of Sports Physiology and Performance*, 3, 359-374.
- COTE, D. J., PRENTICE, W. E., JR., HOOKER, D. N. & SHIELDS, E. W. 1988. Comparison of three treatment procedures for minimizing ankle sprain swelling. *Physical Therapy*, 68, 1072-6.
- COUTTS, A. J., QUINN, J., HOCKING, J., CASTAGNA, C. & RAMPININI, E. 2010. Match running performance in elite Australian Rules Football. *Journal of Science and Medicine in Sport*, 13, 543-8.

- COUTTS, A. J., RAMPININI, E., MARCORA, S. M., CASTAGNA, C. & IMPELLIZERI, F. M. 2009. Heart rate and blood lactate correlates of perceived exertion during small-sided soccer games. *Journal of Science and Medicine in Sport*, 12, 79-84.
- CUNNIFFE, B., HORE, A. J., WHITCOMBE, D. M., JONES, K. P., BAKER, J. S. & DAVIES, B. 2010. Time course of changes in immuneoendocrine markers following an international rugby game. *European Journal of Applied Physiology*, 108, 113-122.
- CYKTOR, J. C. & TURNER, J. 2011. Interleukin-10 and immunity against prokaryotic and eukaryotic intracellular pathogens. *Infection and immunity*, 79, 2964-73.
- DAVIS, J. M. & BAILEY, S. P. 1997. Possible mechanisms of central nervous system fatigue during exercise. *Medicine and science in sports and exercise*, 29, 45-57.
- DAVIS, M. P. & WALSH, D. 2010. Mechanisms of fatigue. *Journal of Supportive Oncology*, 8, 164-74.
- DAWSON, B., GOODMAN, C., LAWRENCE, S., PREEN, D., POLGLAZE, T., FITZSIMONS, M. & FOURNIER, P. 1997. Muscle phosphocreatine repletion following single and repeated short sprint efforts. *Scandinavian Journal of Medicine & Science in Sports*, 7, 206-213.
- DAWSON, B., GOW, S., MODRA, S., BISHOP, D. & STEWART, G. 2005. Effects of immediate post-game recovery procedures on muscle soreness, power and flexibility levels over the next 48 hours. *Journal of Science and Medicine in Sport*, 8, 210-221.
- DAWSON, B., HOPKINSON, R., APPLEBY, B., STEWART, G. & ROBERTS, C. 2004a. Comparison of training activities and game demands in the Australian Football League. *Journal of Science and Medicine in Sport*, 7, 292-301.
- DAWSON, B., HOPKINSON, R., APPLEBY, B., STEWART, G. & ROBERTS, C. 2004b. Player movement patterns and game activities in the Australian Football League. *Journal of Science and Medicine in Sport*, 7, 278-91.
- DE RUITER, C. J., JONES, D. A., SARGEANT, A. J. & DE HAAN, A. 1999. Temperature effect on the rates of isometric force development and relaxation in the fresh and fatigued human adductor pollicis muscle. *Experimental Physiology*, 84, 1137-50.
- DILL, D. B. & COSTILL, D. L. 1974. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol*, 37, 247-8.
- DINARELLO, C. A. 1988. Biology of interleukin 1. *The FASEB journal : official publication of the Federation of American Societies for Experimental Biology*, 2, 108-15.
- DINARELLO, C. A. 1997a. Proinflammatory and anti-inflammatory cytokines as mediators in the pathogenesis of septic shock. *Chest*, 112, 321S-329S.
- DINARELLO, C. A. 1997b. Role of pro- and anti-inflammatory cytokines during inflammation: experimental and clinical findings. *Journal of biological regulators and homeostatic agents*, 11, 91-103.
- DINARELLO, C. A. 2000. Proinflammatory cytokines. Chest, 118, 503-8.
- DINARELLO, C. A. 2005. Interleukin-1beta. Critical care medicine, 33, S460-2.
- DRENTH, J. P., VAN UUM, S. H., VAN DEUREN, M., PESMAN, G. J., VAN DER VEN-JONGEKRIJG, J. & VAN DER MEER, J. W. 1995. Endurance run increases circulating IL-6 and IL-1ra but downregulates ex vivo TNF-alpha and IL-1 beta production. *Journal of applied physiology*, 79, 1497-503.

- DUHAMEL, T. A., PERCO, J. G. & GREEN, H. J. 2006. Manipulation of dietary carbohydrates after prolonged effort modifies muscle sarcoplasmic reticulum responses in exercising males. *American Journal of Physiology - Regulatory, Integrative and Comparative Physiology*, 291, R1100-10.
- DUKE, A. M. & STEELE, D. S. 2001. Mechanisms of reduced SR Ca(2+) release induced by inorganic phosphate in rat skeletal muscle fibers. *American Journal of Cell Physiology*, 281, C418-29.
- ELMER, S. J. & MARTIN, J. C. 2010. Joint-specific power loss after eccentric exercise. *Medicine and science in sports and exercise*, 42, 1723-30.
- ENOKA, R. M. 1996. Eccentric contractions require unique activation strategies by the nervous system. *Journal of Applied Physiology*, 81, 2339-2346.
- ESTON, R., BYRNE, C. & TWIST, C. 2003. Muscle function after exercise-induced muscle damage: considerations for athletic performance in children and adults. *Journal of Exercise Science and Fitness*, 1, 85-96.
- ESTON, R. & PETERS, D. 1999. Effects of cold water immersion on the symptoms of exercise-induced muscle damage. *Journal of Sports Sciences*, 17, 231-238.
- ESTON, R. G., FINNEY, S., BAKER, S. & BALTZOPOULOS, V. 1996. Muscle tenderness and peak torque changes after downhill running following a prior bout of isokinetic eccentric exercise. *Journal of Sports Sciences*, 14, 291-9.
- FARROW, D. 2008. Skill and physiological demands of open and closed training drill in Australian footbal. *International Journal of Sports Science & Coaching*, 3, 489-499.
- FATOUROS, I. G., CHATZINIKOLAOU, A., DOUROUDOS, I. I., NIKOLAIDIS, M. G., KYPAROS, A., MARGONIS, K., MICHAILIDIS, Y., VANTARAKIS, A., TAXILDARIS, K., KATRABASAS, I., MANDALIDIS, D., KOURETAS, D. & JAMURTAS, A. Z. 2010. Time-Course of Changes in Oxidative Stress and Antioxidant Status Responses Following a Soccer Game. Journal of Strength and Conditioning Research, 24, 3278-3286.
- FAUDE, O., KOCH, T. & MEYER, T. 2012. Straight sprinting is the most frequent action in goal situations in professional football. *Journal of Sports Sciences*, 30, 625-631.
- FEBBRAIO, M. A. & PEDERSEN, B. K. 2002. Muscle-derived interleukin-6: mechanisms for activation and possible biological roles. *Faseb Journal*, 16, 1335-1347.
- FERGUSON, R. A. 2010. Limitations to performance during alpine skiing. *Experimental Physiology*, 95, 404-410.
- FISCUS, K. A., KAMINSKI, T. W. & POWERS, M. E. 2005. Changes in lower-leg blood flow during warm-, cold-, and contrast-water therapy. *Archives of Physical Medicine and Rehabilitation*, 86, 1404-10.
- FITTS, R. 1994. Cellular mechanisms of muscle fatigue. *Physiological Reviews*, 74, 49-83.
- FLYNN, M. G., MCFARLIN, B. K. & MARKOFSKI, M. M. 2007. State of the Art Reviews: The Anti-Inflammatory Actions of Exercise Training. *American Journal of Lifestyle Medicine*, 1, 220-235.
- FOWLES, J. R., GREEN, H. J., SCHERTZER, J. D. & TUPLING, A. R. 2002. Reduced activity of muscle Na(+)-K(+)-ATPase after prolonged running in rats. *Journal of applied physiology*, 93, 1703-8.
- FRASER, S. F., LI, J. L., CAREY, M. F., WANG, X. N., SANGKABUTRA, T., SOSTARIC, S., SELIG, S. E., KJELDSEN, K. & MCKENNA, M. J. 2002. Fatigue depresses maximal in vitro skeletal muscle Na(+)-K(+)-ATPase

activity in untrained and trained individuals. *Journal of applied physiology*, 93, 1650-9.

- FRENCH, D. N., THOMPSON, K. G., GARLAND, S. W., BARNES, C. A., PORTAS, M. D., HOOD, P. E. & WILKES, G. 2008. The effects of contrast bathing and compression therapy on muscular performance. *Medicine and science in sports and exercise*, 40, 1297-306.
- FRIDEN, J. & LIEBER, R. L. 2001. Eccentric exercise-induced injuries to contractile and cytoskeletal muscle fibre components. *Acta Physiologica Scandanavica*, 171, 321-326.
- FRY, R. W., MORTON, A. R. & KEAST, D. 1991. Overtraining in athletes. An update. *Sports medicine (Auckland, N.Z.)*, 12, 32-65.
- GABBETT, T., JENKINS, D. & ABERNETHY, B. 2009. Game-Based Training for Improving Skill and Physical Fitness in Team Sport Athletes. *International Journal of Sports Science & Coaching*, 4, 273-283.
- GABBETT, T. J. 2006. Skill-based conditioning games as an alternative to traditional conditioning for rugby league players. *Journal of Strength and Conditioning Research*, 20, 309-315.
- GABBETT, T. J. 2010. GPS analysis of elite women's field hockey training and competition. *Journal of strength and conditioning research / National Strength & Conditioning Association*, 24, 1321-4.
- GABBETT, T. J., JENKINS, D. G. & ABERNETHY, B. 2012. Physical demands of professional rugby league training and competition using microtechnology. *Journal of science and medicine in sport / Sports Medicine Australia*, 15, 80-6.
- GABBETT, T. J. & MULVEY, M. J. 2008. Time-motion analysis of small-sided training games and competition in elite women soccer players. *Journal of strength and conditioning research / National Strength & Conditioning Association*, 22, 543-52.
- GAITANOS, G. C., WILLIAMS, C., BOOBIS, L. H. & BROOKS, S. 1993. Human muscle metabolism during intermittent maximal exercise. *Journal of applied physiology*, 75, 712-9.
- GANDEVIA, S. C. 2001. Spinal and supraspinal factors in human muscle fatigue. *Physiological Reviews*, 81, 1725-1789.
- GANDEVIA, S. C., ALLEN, G. M., BUTLER, J. E. & TAYLOR, J. L. 1996. Supraspinal factors in human muscle fatigue: evidence for suboptimal output from the motor cortex. *The Journal of physiology*, 490 (Pt 2), 529-36.
- GARLAND, S. J. 1991. Role of small diameter afferents in reflex inhibition during human muscle fatigue. *The Journal of physiology*, 435, 547-58.
- GARLAND, S. J. & KAUFMAN, M. P. 1995. Role of muscle afferents in the inhibition of motoneurons during fatigue. *Advances in Experimental Medicine and Biology*, 384, 271-8.
- GIANNESINI, B., COZZONE, P. J. & BENDAHAN, D. 2003. Non-invasive investigations of muscular fatigue: metabolic and electromyographic components. *Biochimie*, 85, 873-83.
- GILL, N. D., BEAVEN, C. M. & COOK, C. 2006. Effectiveness of post-match recovery strategies in rugby players. *Br J Sports Med*, 40, 260-263.
- GOODALL, S. & HOWATSON, G. 2008. The effects of multiple cold water immersion on indices of muscle damage. *Journal of Sports Sciences and Medicine*, 7, 235-241.

- GOULD, D., KELLY, D., GOLDSTONE, L. & GAMMON, J. 2001. Examining the validity of pressure ulcer risk assessment scales: developing and using illustrated patient simulations to collect the data. *Journal of Clinical Nursing*, 10, 697-706.
- GRAY, A. J. & JENKINS, D. G. 2010. Match analysis and the physiological demands of Australian football. *Sports Med*, 40, 347-60.
- GREEN, H. J. 1997. Mechanisms of muscle fatigue in intense exercise. *Journal of Sports Sciences*, 15, 247-256.
- HALSON, S. 2011. Does the time frame between exercise influence the effectiveness of hydrotherapy for recovery? *International Journal of Sports Physiology and Performance*, 6, 147-159.
- HALSON, S. L. & JEUKENDRUP, A. E. 2004. Does overtraining exist? An analysis of overreaching and overtraining research. *Sports medicine*, 34, 967-81.
- HALSON, S. L., QUOD, M. J., MARTIN, D. T., GARDNER, A. S., EBERT, T. R. & LAURSEN, P. B. 2008. Physiological responses to cold water immersion following cycling in the heat. *International Journal of Sports Physiology and Performance*, 3, 331-46.
- HAMLIN, M. J. 2007. The effect of contrast temperature water therapy on repeated sprint performance. *Journal of Science and Medicine in Sport*, 10, 398-402.
- HARTWIG, T. B., NAUGHTON, G. & SEARL, J. 2011. Motion analyses of adolescent rugby union players: a comparison of training and game demands. *Journal of strength and conditioning research / National Strength & Conditioning Association*, 25, 966-72.
- HELANDER, I., WESTERBLAD, H. & KATZ, A. 2002. Effects of glucose on contractile function, [Ca2+]i, and glycogen in isolated mouse skeletal muscle. *American Journal of Physiology Cell Physiology*, 282, C1306-12.
- HEMMINGS, B., SMITH, M., GRAYDON, J. & DYSON, R. 2000. Effects of massage on physiological restoration, perceived recovery, and repeated sports performance. *British Journal of Sports Medicine*, 34, 109-14; discussion 115.
- HIGGINS, T. R., HEAZLEWOOD, I. T. & CLIMSTEIN, M. 2010. A Random Control Trial of Contrast Baths and Ice Baths for Recovery during Competition in U/20 Rugby Union. *J Strength Cond Res*.
- HIGGINS, T. R., HEAZLEWOOD, I. T. & CLIMSTEIN, M. 2011. A random control trial of contrast baths and ice baths for recovery during competition in U/20 rugby union. *The Journal of Strength & Conditioning Research*, 25, 1046-51.
- HIGHTON, J., TWIST, C. & ESTON, R. 2009. The effects of exercise-induced muscle damage on agility and sprint running performance. *Journal of Exercise Science and Fitness*, 7, 24-30.
- HILL-HAAS, S. V., COUTTS, A. J., ROWSELL, G. J. & DAWSON, B. T. 2009. Generic versus small-sided game training in soccer. *International journal of sports medicine*, 30, 636-42.
- HILL-HAAS, S. V., DAWSON, B., IMPELLIZZERI, F. M. & COUTTS, A. J. 2011. Physiology of Small-Sided Games Training in Football A Systematic Review. *Sports Medicine*, 41, 199-220.
- HING, W. A., WHITE, S. G., BOUAAPHONE, A. & LEE, P. 2008. Contrast therapy a systemic review. *Physical Therapy in Sport*, 9, 148-161.
- HOFF, J., WISLOFF, U., ENGEN, L. C., KEMI, O. J. & HELGERUD, J. 2002. Soccer specific aerobic endurance training. *British Journal of Sports Medicine*, 36, 218-221.

- HOPKINS, W. G. 2002. A Scale of Magnitudes for Effect Statistics [Online]. Available: http://www.sportsci.org/resource/stats/effectmag.html.
- HOPKINS, W. G., MARSHALL, S. W., BATTERHAM, A. M. & HANIN, J. 2009. Progressive statistics for studies in sports medicine and exercise science. *Medicine and science in sports and exercise*, 41, 3-13.
- HOWATSON, G., GOODALL, S. & VAN SOMEREN, K. A. 2009. The influence of cold water immersions on adaptation following a single bout of damaging exercise. *European Journal of Applied Physiology*, 105, 615-621.
- HOWATSON, G. & VAN SOMEREN, K. A. 2008. The prevention and treatment of exercise-induced muscle damage. *Sports Medicine*, 38, 483-503.
- HOWELL, J. N., CHLEBOUN, G. & CONATSER, R. 1993. Muscle stiffness, strength loss, swelling and soreness following exercise induced injury in humans. *Journal of Physiology*, 464, 183-196.
- HRYSOMALLIS, C. & BUTTIFANT, D. 2012. Influence of training years on upperbody strength and power changes during the competitive season for professional Australian rules football players. *Journal of science and medicine in sport / Sports Medicine Australia*, 15, 374-8.
- HUBBARD, T. J. & DENEGAR, C. R. 2004. Does Cryotherapy Improve Outcomes With Soft Tissue Injury? *Journal of Athletic Training*, 39, 278-279.
- HULTMAN, E., BERGSTROM, J. & ANDERSON, N. M. 1967. Breakdown and resynthesis of phosphorylcreatine and adenosine triphosphate in connection with muscular work in man. *Scandinavian Journal of Clinical and Laboratory Investigation*, 19, 56-66.
- IMPELLIZZERI, F. M., BORG, E. & COUTTS, A. J. 2011. Intersubjective comparisons are possible with an accurate use of the Borg CR scales. *International Journal of Sports Physiology and Performance*, 6, 2-4; author reply 4-5.
- INGRAM, J., DAWSON, B., GOODMAN, C., WALLMAN, K. & BEILBY, J. 2009. Effect of water immersion methods on post-exercise recovery from simulated team sport exercise. *Journal of Science and Medicine in Sport*, 12, 417-421.
- ISII, Y., MATSUKAWA, K., TSUCHIMOCHI, H. & NAKAMOTO, T. 2007. Icewater hand immersion causes a reflex decrease in skin temperature in the contralateral hand. *The Journal of Physiological Sciences*, 57, 241-248.
- ISPIRLIDIS, I., FATOUROS, I. G., JAMURTAS, A. Z., NIKOLAIDIS, M. G., MICHAILIDIS, I., DOUROUDOS, I., MARGONIS, K., CHATZINIKOLAOU, A., KALISTRATOS, E., KATRABASAS, I., ALEXIOU, V. & TAXILDARIS, K. 2008. Time-course of changes in inflammatory and performance responses following a soccer game. *Clinical Journal of Sport Medicine*, 18, 423-431.
- JENKINS, J. K., MALYAK, M. & AREND, W. P. 1994. The effects of interleukin-10 on interleukin-1 receptor antagonist and interleukin-1 beta production in human monocytes and neutrophils. *Lymphokine and cytokine research*, 13, 47-54.
- JENNINGS, D., CORMACK, S. J., COUTTS, A. J. & AUGHEY, R. J. 2012. GPS analysis of international field hockey tournament. *International Journal of Sports Physiology and Performance*, In Press.
- KALMAR, J. M. & CAFARELLI, E. 2004. Caffeine: a valuable tool to study central fatigue in humans? *Exercise and sport sciences reviews*, 32, 143-7.

- KANLAYANAPHOTPORN, R. & JANWANTANAKUL, P. 2005. Comparison of skin surface temperature during the application of various cryotherapy modalities. *Archives of Physical Medicine and Rehabilitation*, 86, 1411-1415.
- KEETON, R. B. & BINDER-MACLEOD, S. A. 2006. Low-frequency fatigue. *Physical therapy*, 86, 1146-50.
- KENT-BRAUN, J. A. 1999. Central and peripheral contributions to muscle fatigue in humans during sustained maximal effort. *European journal of applied physiology and occupational physiology*, 80, 57-63.
- KINCHINGTON, M., BALL, K. & NAUGHTON, G. 2012. Relation between lower limb comfort and performance in elite footballers. *Physical Therapy in Sport*, 13, 27-34.
- KING, M. & DUFFIELD, R. 2009. The effects of recovery interventions on consecutive days of intermittent sprint exercise. *The Journal of Strength & Conditioning Research*, 23, 1795-1802.
- KINUGASA, T. & KILDING, A. E. 2009. A comparison of post-match recovery strategies in youth soccer players. *The Journal of Strength & Conditioning Research*, 23, 1402-07.
- KNICKER, A. J., RENSHAW, I., OLDHAM, A. R. & CAIRNS, S. P. 2011. Interactive processes link the multiple symptoms of fatigue in sport competition. *Sports Medicine*, 41, 307-28.
- KNIGHT, K. L. 1995. *Cryotherapy in sports injury management,* Champaign, Illinois, Human Kinetics.
- KNIGHT, K. L., BRYAN, K. S. & HALVORSEN, J. M. 1981. Circulatory changes in the forearm in 1, 5, 10 and 15°C water. *International journal of sports medicine*, 4.
- KOC, M., TEZ, M., YOLDAS, O. & GOCMEN, E. 2006. Cooling for the reduction of postoperative pain: prospective randomized study. *Hernia*, 10, 184-186.
- KOMI, P. V. 2000. Stretch-shortening cycle: a powerful model to study normal and fatigued muscle. *Journal of Biomechanics*, 33, 1197-206.
- KON, M., TANABE, K., LEE, H., KIMURA, F., AKIMOTO, T. & KONO, I. 2007. Eccentric muscle contractions induce greater oxidative stress than concertic contractions in skeletal muscle. *Applied Physiology, Nutrition, and Metabolism*, 32, 273-281.
- KOWAL, M. A. 1983. Review of physiological effects of cryotherapy. *The Journal of* orthopaedic and sports physical therapy, 5, 66-73.
- KRAEMER, W. J., FRENCH, D. N. & SPIERING, B. A. 2004. Compression in the treatment of acute muscle injuries in sport. *International SportMed Journal*, 5, 200-209.
- KRUSTRUP, P., CHRISTENSEN, J. F., RANDERS, M. B., PEDERSEN, H., SUNDSTRUP, E., JAKOBSEN, M. D., KRUSTRUP, B. R., NIELSEN, J. J., SUETTA, C., NYBO, L. & BANGSBO, J. 2010. Muscle adaptations and performance enhancements of soccer training for untrained men. *European Journal of Applied Physiology*, 108, 1247-1258.
- KRUSTRUP, P., MOHR, M., STEENSBERG, A., BENCKE, J., KJAER, M. & BANGSBO, J. 2006. Muscle and blood metabolites during a soccer game: implications for sprint performance. *Medicine & Science in Sports & Exercise*, 38, 1165-1174.
- KRUSTRUP, P., ORTENBLAD, N., NIELSEN, J., NYBO, L., GUNNARSSON, T. P., IAIA, F. M., MADSEN, K., STEPHENS, F., GREENHAFF, P. & BANGSBO, J. 2011. Maximal voluntary contraction force, SR function and

glycogen resynthesis during the first 72 h after a high-level competitive soccer game. *European journal of applied physiology*, 111, 2987-95.

- LIEBER, R. L. & FRIDEN, J. 1999. Mechanisms of muscle injury after eccentric contraction. *Journal of science and medicine in sport / Sports Medicine Australia*, 2, 253-65.
- LINDENA, J., KUPPER, W. & TRAUTSCHOLD, I. 1979. Lymphatic transport of cellular enzymes from muscle into the intravascular compartment. *Enzyme*, 24.
- LIPPI, G., SCHENA, F., SALVAGNO, G. L., MONTAGNANA, M., GELATI, M., TARPERI, C., BANFI, G. & GUIDI, G. C. 2008. Acute variation of biochemical markers of muscle damage following a 21-km, half-marathon run. *Scandinavian journal of clinical and laboratory investigation*, 68, 667-72.
- LOLLGEN, H., VON NIEDING, G., KOPPENHAGEN, K., KERSTING, F. & JUST, H. 1981. Hemodynamic response to graded water immersion. *Klinische Wochenschrift*, 59, 623-8.
- MACEFIELD, G., HAGBARTH, K. E., GORMAN, R., GANDEVIA, S. C. & BURKE, D. 1991. Decline in Spindle Support to Alpha-Motoneurons during Sustained Voluntary Contractions. *Journal of Physiology-London*, 440, 497-512.
- MACINTYRE, D. L., REID, W. D., LYSTER, D. M., SZASZ, I. J. & MCKENZIE, D. C. 1996. Presence of WBC, decreased strength, and delayed soreness in muscle after eccentric exercise. *Journal of Applied Physiology*, 80, 1006-1013.
- MACUTKIEWICZ, D. & SUNDERLAND, C. 2011. The use of GPS to evaluate activity profiles of elite women hockey players during match-play. *Journal of Sports Sciences*, 29, 967-973.
- MAGAL, M., DUMKE, C. L., URBIZTONDO, Z. G., CAVILL, M. J., TRIPLETT, N. T., QUINDRY, J. C., MCBRIDE, J. M. & EPSTEIN, Y. 2010. Relationship between serum creatine kinase activity following exerciseinduced muscle damage and muscle fibre composition. *Journal of Sports Sciences*, 28, 257-266.
- MAGALHAES, J., REBELO, A., OLIVEIRA, E., SILVA, J. R., MARQUES, F. & ASCENSAO, A. 2010. Impact of Loughborough Intermittent Shuttle Test versus soccer match on physiological, biochemical and neuromuscular parameters. *European Journal of Applied Physiology*, 108, 39-48.
- MAIR, J. 1999. Tissue release of cardiac markers: From physiology to clinical applications. *Clinical Chemistry and Laboratory Medicine*, 37, 1077-1084.
- MALM, C. 2001. Exercise-induced muscle damage and inflammation: fact or fiction? *Acta Physiologica Scandanavica*, 171, 233-239.
- MARCORA, S. M., STAIANO, W. & MANNING, V. 2009. Mental fatigue impairs physical performance in humans. *Journal of Applied Physiology*, 106, 857-864.
- MARIEB, E. N., MALLATT, J. & WILHELM, P. B. 2008. *Human Anatomy*, San Fransisco, Pearson Benjamin Cummings.
- MARIN, D. P., DOS SANTOS, R. D. M., BOLIN, A. P., GUERRA, B. A., HATANAKA, E. & OTTON, R. 2011. Cytokines and Oxidative Stress Status Following a Handball Game in Elite Male Players. Oxidative Medicine and Cellular Longevity.
- MARKOVIC, G., DIZDAR, D., JUKIC, I. & CARDINALE, M. 2004. Reliability and factorial validity of squat and countermovement jump tests. *Journal of strength and conditioning research / National Strength & Conditioning Association*, 18, 551-5.

- MARTONOSI, A. N. 1984. Mechanisms of Ca2+ release from sarcoplasmic reticulum of skeletal muscle. *Physiological reviews*, 64, 1240-320.
- MCCARTNEY, N., SPRIET, L. L., HEIGENHAUSER, G. J., KOWALCHUK, J. M., SUTTON, J. R. & JONES, N. L. 1986. Muscle power and metabolism in maximal intermittent exercise. *Journal of applied physiology*, 60, 1164-9.
- MCKENNA, M. J. 2003. Mechanisms of muscle fatigue. *In:* HARGREAVES, M. & HAWLEY, J. A. (eds.) *Physiological bases of sports performance*. Sydney: McGraw Hill.
- MCKENNA, M. J., BANGSBO, J. & RENAUD, J. M. 2008. Muscle K+, Na+, and Cl disturbances and Na+-K+ pump inactivation: implications for fatigue. *Journal of Applied Physiology*, 104, 288-95.
- MECKEL, Y., ELIAKIM, A., SERAEV, M., ZALDIVAR, F., COOPER, D. M., SAGIV, M. & NEMET, D. 2009. The Effect of a Brief Sprint Interval Exercise on Growth Factors and Inflammatory Mediators. *Journal of Strength and Conditioning Research*, 23, 225-230.
- MILLET, G. Y. & LEPERS, R. 2004. Alterations of neuromuscular function after prolonged running, cycling and skiing exercises. *Sports medicine*, 34, 105-16.
- MISHRA, D. K., FRIDEN, J., SCHMITZ, M. C. & LIEBER, R. L. 1995. Antiinflammatory medication after muscle injury. A treatment resulting in shortterm improvement but subsequent loss of muscle function. *Journal of Bone and Joint Surgery, American Volume,* 77, 1510-9.
- MOHR, M., KRUSTRUP, P. & BANGSBO, J. 2003. Match performance of highstandard soccer players with special reference to development of fatigue. *Journal of Sports Sciences*, 21, 519-528.
- MOHR, M., KRUSTRUP, P. & BANGSBO, J. 2005. Fatigue in soccer: a brief review. *Journal of Sports Sciences*, 23, 593-599.
- MONTGOMERY, P., PYNE, D., COX, A. J., HOPKINS, W. G., MINAHAN, C. L. & HUNT, P. H. 2008a. Muscle damage, inflammation, and recovery interventions during a 3-day basketball tournament. *European Journal of Sport Science*, 8, 241-250.
- MONTGOMERY, P. G., PYNE, D. B., HOPKINS, W. G., DORMAN, J. C., COOK, K. & MINAHAN, C. L. 2008b. The effect of recovery strategies on physical performance and cumulative fatigue in competitive basketball. *Journal of Sports Sciences*, 26, 1135-45.
- MOORE, K. W., DE WAAL MALEFYT, R., COFFMAN, R. L. & O'GARRA, A. 2001. Interleukin-10 and the interleukin-10 receptor. *Annual review of immunology*, 19, 683-765.
- MORGAN, D. L. & ALLEN, D. G. 1999. Early events in stretch-induced muscle damage. *Journal of applied physiology*, 87, 2007-15.
- MYRER, J. W., DRAPER, D. O. & DURRANT, E. 1994. Contrast therapy and intramuscular temperature in the human leg. *Journal of athletic training*, 29, 318-22.
- MYRER, J. W., MEASOM, G., DURRANT, E. & FELLINGHAM, G. W. 1997. Cold- and hot-pack contrast therapy: subcutaneous and intramuscular temperature change. *Journal of athletic training*, 32, 238-41.
- NADLER, S. F., WEINGAND, K. & KRUSE, R. J. 2004. The physiologic basis and clinical applications of cryotherapy and thermotherapy for the pain practitioner. *Pain physician*, 7, 395-9.
- NEMET, D., MECKEL, Y., BAR-SELA, S., ZALDIVAR, F., COOPER, D. M. & ELIAKIM, A. 2009. Effect of local cold-pack application on systemic

anabolic and inflammatory response to sprint-interval training: a prospective comparative trial. *European Journal of Applied Physiology*, 107, 411-417.

- NIELSEN, A. R. & PEDERSEN, B. K. 2007. The biological roles of exerciseinduced cytokines: IL-6, IL-8, and IL-15. *Applied physiology, nutrition, and metabolism*, 32, 833-9.
- NIELSEN, J., HOLMBERG, H. C., SCHRODER, H. D., SALTIN, B. & ORTENBLAD, N. 2011. Human skeletal muscle glycogen utilization in exhaustive exercise: role of subcellular localization and fibre type. *The Journal of Physiology*, 589, 2871-85.
- NOSAKA, K. & CLARKSON, P. M. 1995. Muscle damage following repeated bouts of high force eccentric exercise. *Medicine and science in sports and exercise*, 27, 1263-9.
- NOSAKA, K. & CLARKSON, P. M. 1996. Variability in serum creatine kinase response after eccentric exercise of the elbow flexors. *International journal of sports medicine*, 17, 120-127.
- NOSAKA, K., CLARKSON, P. M., MCGUIGGIN, M. E. & BYRNE, J. M. 1991. Time Course of Muscle Adaptation after High Force Eccentric Exercise. *European journal of applied physiology and occupational physiology*, 63, 70-76.
- NOSAKA, K., NEWTON, M. & SACCO, P. 2002. Delayed-onset muscle soreness does not reflect the magnitude of eccentric exercise-induced muscle damage. *Scandanavian Journal of Medicine & Science in Sports*, 12, 337-46.
- ORTENBLAD, N., NIELSEN, J., SALTIN, B. & HOLMBERG, H. C. 2011. Role of glycogen availability in sarcoplasmic reticulum Ca2+ kinetics in human skeletal muscle. *The Journal of Physiology*, 589, 711-25.
- OSTROWSKI, K., HERMANN, C., BANGASH, A., SCHJERLING, P., NIELSEN, J. N. & PEDERSEN, B. K. 1998. A trauma-like elevation of plasma cytokines in humans in response to treadmill running. *The Journal of physiology*, 513 (Pt 3), 889-94.
- OSTROWSKI, K., ROHDE, T., ASP, S., SCHJERLING, P. & PEDERSEN, B. K. 1999. Pro- and anti-inflammatory cytokine balance in strenuous exercise in humans. *Journal of Physiology-London*, 515, 287-291.
- PALLAFACCHINA, G., BLAAUW, B. & SCHIAFFINO, S. 2012. Role of satellite cells in muscle growth and maintenance of muscle mass. *Nutrition, metabolism, and cardiovascular diseases : NMCD*.
- PANDOLF, K. B. 1998. Time course of heat acclimation and its decay. *International journal of sports medicine*, 19 Suppl 2, S157-60.
- PARAMESWARAN, N. & PATIAL, S. 2010. Tumor necrosis factor-alpha signaling in macrophages. *Critical reviews in eukaryotic gene expression*, 20, 87-103.
- PEAKE, J., NOSAKA, K. & SUZUKI, K. 2005a. Characterization of inflammatory responses to eccentric exercise in humans. *Exercise Immunology Review*, 11, 64-85.
- PEAKE, J., SUZUKI, K., WILSON, G., HORDERN, M., NOSAKA, K., MACKINNON, L. & COOMBES, J. S. 2005b. Exercise-induced muscle damage, plasma cytokines, and markers of neutrophil activation. *Medicine & Science in Sports & Exercise*, 37, 737-745.
- PEAKE, J. M., SUZUKI, K., HORDERN, M., WILSON, G., NOSAKA, K. & COOMBES, J. S. 2005c. Plasma cytokine changes in relation to exercise intensity and muscle damage. *European Journal of Applied Physiology*, 95, 514-521.

- PEDERSEN, B. K. 2000. Exercise and cytokines. *Immunology and Cell Biology*, 78, 532-535.
- PEDERSEN, B. K. & FEBBRAIO, M. 2005. Muscle-derived interleukin-6--a possible link between skeletal muscle, adipose tissue, liver, and brain. *Brain, Behavior, and Immunity*, 19, 371-6.
- PEDERSEN, B. K. & HOFFMAN-GOETZ, L. 2000. Exercise and the immune system: regulation, integration, and adaptation. *Physiological reviews*, 80, 1055-81.
- PEDERSEN, B. K., OSTROWSKI, K., ROHDE, T. & BRUUNSGAARD, H. 1998. The cytokine response to strenuous exercise. *Canadian Journal of Physiology and Pharmacology*, 76, 505-511.
- PEDERSEN, B. K., STEENSBERG, A., KELLER, P., KELLER, C., FISCHER, C., HISCOCK, N., VAN HALL, G., PLOMGAARD, P. & FEBBRAIO, M. A. 2003. Muscle-derived interleukin-6: lipolytic, anti-inflammatory and immune regulatory effects. *Pflugers Arch*, 446, 9-16.
- PEDERSEN, B. K., STEENSBERG, A. & SCHJERLING, P. 2001. Muscle-derived interleukin-6: possible biological effects. *Journal of Physiology-London*, 536, 329-337.
- PEIFFER, J. J., ABBIS, C. R., NOSAKA, K., PEAKE, J. M. & LAURSEN, P. B. 2009. Effect of cold water immersion after exercise in the heat on muscle function, body temperatures, and vessel diameter. *Journal of Science and Medicine in Sport*, 12, 91-96.
- PETERSEN, A. M. & PEDERSEN, B. K. 2005. The anti-inflamatory effect of exercise. *Journal of Applied Physiology*, 98, 1154-1162.
- PETERSEN, A. M. W. & PEDERSEN, B. K. 2006. The Role of II-6 in Mediating the Anti-Inflammatory Effects of Exercise. *Journal of Physiology and Pharmacology*, 57, 43-51.
- PIEHL, K. 1974. Time course for refilling of glycogen stores in human muscle fibres following exercise-induced glycogen depletion. *Acta physiologica Scandinavica*, 90, 297-302.
- PLACE, N., YAMADA, T., BRUTON, J. D. & WESTERBLAD, H. 2010. Muscle fatigue: from observations in humans to underlying mechanisms studied in intact single muscle fibres. *European journal of applied physiology*, 110, 1-15.
- PLASSMANN, H., O'DOHERTY, J., SHIV, B. & RANGEL, A. 2008. Marketing actions can modulate neural representations of experienced pleasantness. *Proceedings of the National Academy of Sciences of the United States of America*, 105, 1050-1054.
- POURNOT, H., BIEUZEN, F., LOUIS, J., FILLARD, J. R., BARBICHE, E. & HAUSSWIRTH, C. 2011. Time-course of changes in inflammatory response after whole-body cryotherapy multi exposures following severe exercise. *PLoS One*, 6, e22748.
- POWERS, S. K. & HOWLEY, E. T. 2007. *Exercise Physiology: Theory and Application to Fitness and Performance,* New York, McGraw-Hill.
- PRENTICE, W. E. 1999. *Therapeutic Modalities in Sports Medicine, fourth edition,* Boston, WCB/McGraw-Hill.
- PROSKE, U. & ALLEN, T. J. 2005. Damage to skeletal muscle from eccentric exercise. *Exercise and Sport Sciences Reviews*, 33, 98-104.
- PROSKE, U. & MORGAN, D. L. 2001. Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. *Journal of Physiology*, 537, 333-45.

- RAMADORI, G. & CHRIST, B. 1999. Cytokines and the hepatic acute-phase response. *Seminars in liver disease*, 19, 141-55.
- RAMPININI, E., IMPELLIZERI, F. M., CASTAGNA, C., ABT, G., CHAMARI, K., SASSI, A. & MARCORA, S. M. 2007. Factors influencing physiological responses to small-sided soccer games. *Journal of Sports Sciences*, 25, 659-666.
- RAMPININI, E., IMPELLIZZERI, F. M., CASTAGNA, C., COUTTS, A. J. & WISLOFF, U. 2009. Technical performance during soccer matches of the Italian Serie A league: effect of fatigue and competitive level. *Journal of Science and Medicine in Sport*, 12, 227-33.
- RANDELL, A. D., CRONIN, J. B., KEOGH, J. W., GILL, N. D. & PEDERSEN, M. C. 2011. Reliability of performance velocity for jump squats under feedback and nonfeedback conditions. *Journal of strength and conditioning research / National Strength & Conditioning Association*, 25, 3514-8.
- REILLY, T. 1997. Energetics of high-intensity exercise (soccer) with particular reference to fatigue. *Journal of Sports Sciences*, 15, 257-263.
- REILLY, T., DRUST, B. & CLARKE, N. 2008. Muscle fatigue during football-match play. *Sports Medicine*, 38, 357-367.
- RISCH, W. D., KOUBENEC, H. J., BECKMANN, U., LANGE, S. & GAUER, O. H. 1978. The effect of graded immersion on heart volume, central venous pressure, pulmonary blood distribution, and heart rate in man. *Pflugers Archiv : European journal of physiology*, 374, 115-8.
- RONGLAN, L. T., RAASTAD, T. & BORGESEN, A. 2006. Neuromuscular fatigue and recovery in elite female handball players. *Scandanavian Journal of Medicine & Science in Sports*, 16, 267-273.
- ROWSELL, G. J., COUTTS, A. J., REABURN, P. & HILL-HAAS, S. 2009. Effects of cold-water immersion on physical performance between successive matches in high-performance junior male soccer players. *Journal of Sports Sciences*, 27, 565-73.
- ROWSELL, G. J., COUTTS, A. J., REABURN, P. & HILL-HAAS, S. 2011. Effect of post-match cold-water immersion on subsequent match running performance in junior soccer players during tournament play. *Journal of Sports Sciences*, 29, 1-6.
- SABAT, R., GRUTZ, G., WARSZAWSKA, K., KIRSCH, S., WITTE, E., WOLK, K. & GEGINAT, J. 2010. Biology of interleukin-10. *Cytokine & growth factor reviews*, 21, 331-44.
- SARGEANT, A. J. 1987. Effect of muscle temperature on leg extension force and short-term power output in humans. *European Journal of Applied Physiology and Occupational Physiology*, 56, 693-8.
- SAULS, J. 1999. Efficacy of cold for pain: Fact or fallacy? Online Journal of Knowledge Synthesis for Nursing, 6.
- SAYERS, S. P. & CLARKSON, P. M. 2003. Short-term immobilization after eccentric exercise. Part II: Creatine kinase and myoglobin. *Medicine and science in sports and exercise*, 35, 762-768.
- SAYERS, S. P., CLARKSON, P. M. & LEE, J. 2000a. Activity and immobilization after eccentric exercise: I. Recovery of muscle function. *Medicine and science in sports and exercise*, 32, 1587-92.
- SAYERS, S. P., CLARKSON, P. M. & LEE, J. 2000b. Activity and immobilization after eccentric exercise: II. Serum CK. *Medicine and science in sports and exercise*, 32, 1593-1597.

- SCHWEIZER, A., FEIGE, U., FONTANA, A., MULLER, K. & DINARELLO, C. A. 1988. Interleukin-1 enhances pain reflexes. Mediation through increased prostaglandin E2 levels. *Agents and actions*, 25, 246-51.
- SCOTT, J. P., SALE, C., GREEVES, J. P., CASEY, A., DUTTON, J. & FRASER,
   W. D. 2011. Effect of exercise intensity on the cytokine response to an acute bout of running. *Medicine and science in sports and exercise*, 43, 2297-306.
- SEALE, P., POLESSKAYA, A. & RUDNICKI, M. A. 2003. Adult stem cell specification by Wnt signaling in muscle regeneration. *Cell cycle*, 2, 418-9.
- SERRANO, A. L., BAEZA-RAJA, B., PERDIGUERO, E., JARDI, M. & MUNOZ-CINOVES, P. 2008. Interleukin-6 is an essential regulator of satellite cellmediated skeletal muscle hypertrophy. *Cell Metabolism*, 7, 33-44.
- SHEPHARD, R. J. 2002. Cytokine responses to physical activity, with particular reference to IL-6: sources, actions, and clinical implications. *Critical Reviews in Immunology*, 22, 165-82.
- SHEPHARD, R. J. & SHEK, P. N. 1998. Acute and chronic over-exertion: do depressed immune responses provide useful markers? *International journal of sports medicine*, 19, 159-71.
- SHIN, Y. O. & LEE, J. B. 2011. Effects of exercise training on plasma cytokine and chemokine levels, and thermoregulation. *Journal of Thermal Biology*, 36, 219-224.
- SINGH, T. K. R., GUELFI, K. J., LANDERS, G., DAWSON, B. & BISHOP, D. 2010. Reliability of a contact and non-contact simulated team game circuit. *Journal of Sports Science and Medicine*, 9, 638-642.
- SKIVEREN, J., KJAERBY, E. & LARSEN, H. N. 2008. Cooling by frozen gel pack as pain relief during treatment of axillary hyperhidrosis with botulinum toxin A injections. *Acta Dermato-Venereologica*, 88, 366-369.
- SMITH, L. L. 1991. Acute inflammation: the underlying mechanism in delayed onset muscle soreness? *Medicine and science in sports and exercise*, 23, 542-51.
- SMITH, L. L. 2000. Cytokine hypothesis of overtraining: a physiological adaptation to excessive stress? *Medicine and science in sports and exercise*, 32, 317-31.
- SMITH, L. L., ANWAR, A., FRAGEN, M., RANANTO, C., JOHNSON, R. & HOLBERT, D. 2000. Cytokines and cell adhesion molecules associated with high-intensity eccentric exercise. *European journal of applied physiology*, 82, 61-7.
- SONG, C. W. 1984. Effect of local hyperthermia on blood flow and microenvironment: a review. *Cancer research*, 44, 4721s-4730s.
- SPENCER, M., FITZSIMONS, M., DAWSON, B., BISHOP, D. & GOODMAN, C. 2006. Reliability of a repeated-sprint test for field-hockey. *Journal of Science and Medicine in Sport*, 9, 181-4.
- SPENCER, M., RECHICHI, C., LAWRENCE, S., DAWSON, B., BISHOP, D. & GOODMAN, C. 2005. Time-motion analysis of elite field hockey during several games in succession: a tournament scenario. *Journal of Science and Medicine in Sport*, 8, 382-91.
- SPRIET, L. L., SODERLUND, K., BERGSTROM, M. & HULTMAN, E. 1987. Anaerobic energy release in skeletal muscle during electrical stimulation in men. *Journal of applied physiology*, 62, 611-5.
- STARKEY, C. 1999. Therapeutic Modalities, Philadelphia, F. A. Davis Company.
- STARKIE, R. L., ROLLAND, J., ANGUS, D. J., ANDERSON, M. J. & FEBBRAIO, M. A. 2001. Circulating monocytes are not the source of elevations in plasma

IL-6 and TNF-alpha levels after prolonged running. *American Journal of Physiology-Cell Physiology*, 280, C769-C774.

- STEWART, L. K., FLYNN, M. G., CAMPBELL, W. W., CRAIG, B. A., ROBINSON, J. P., TIMMERMAN, K. L., MCFARLIN, B. K., COEN, P. M. & TALBERT, E. 2007. The influence of exercise training on inflammatory cytokines and C-reactive protein. *Medicine and science in sports and exercise*, 39, 1714-1719.
- STUBGEN, J. P. 2011. Tumor necrosis factor-alpha as a potential therapeutic target in idiopathic inflammatory myopathies. *Journal of neurology*, 258, 961-70.
- SUNDERLAND, C., MORRIS, J. G. & NEVILL, M. E. 2008. A heat acclimation protocol for team sports. *British Journal of Sports Medicine*, 42, 327-33.
- SUZUKI, K., NAKAJI, S., YAMADA, M., LIU, Q., KURAKAKE, S., OKAMURA, N., KUMAE, T., BUMEDA, T. & SUGAWARA, K. 2003. Impact of a competitive marathon race on systemic cytokine and neutrophil responses. *Medicine & Science in Sports & Exercise*, 35, 348-355.
- SWENSON, C., SWARD, L. & KARLSSON, J. 1996. Cryotherapy in sports medicine. *Scandanavian Journal of Medicine & Science in Sports*, 6, 193-200.
- TAKARADA, Y. 2003. Evaluation of muscle damage after a rugby match with special reference to tackle plays. *British Journal of Sports Medicine*, 37, 416-419.
- TAYLOR, J. L., ALLEN, G. M., BUTLER, J. E. & GANDEVIA, S. C. 2000. Supraspinal fatigue during intermittent maximal voluntary contractions of the human elbow flexors. *Journal of Applied Physiology*, 89, 305-313.
- TEE, J. C., BOSCH, A. N. & LAMBERT, M. I. 2007. Metabolic consequences of exercise-induced muscle damage. *Sports Medicine*, 37, 827-836.
- THIJS, L. G. & HACK, C. E. 1995. Time course of cytokine levels in sepsis. *Intensive care medicine*, 21 Suppl 2, S258-63.
- THOMPSON, D., NICHOLAS, C. W. & WILLIAMS, C. 1999. Muscular soreness following prolonged intermittent high-intensity shuttle running. *Journal of Sports Sciences*, 17, 387-395.
- TIDBALL, J. G. 1995. Inflammatory cell response to acute muscle injury. *Medicine* and science in sports and exercise, 27, 1022-32.
- TIDBALL, J. G. 2005. Inflammatory processes in muscle injury and repair. *American Journal of Physiology-Regulatory Integrative and Comparative Physiology*, 288, R345-R353.
- TOFT, A. D., JENSEN, L. B., BRUUNSGAARD, H., IBFELT, T., HALKJAER-KRISTENSEN, J., FEBBRAIO, M. & PEDERSEN, B. K. 2002. Cytokine response to eccentric exercise in young and elderly humans. *American Journal* of Physiology-Cell Physiology, 283, C289-C295.
- TOMLIN, D. L. & WENGER, H. A. 2001. The relationship between aerobic fitness and recovery from high intensity intermittent exercise. *Sports Medicine*, 31, 1-11.
- TRACEY, K. J. 2002. The inflammatory reflex. Nature, 420, 853-9.
- TROST, Z., FRANCE, C. R. & THOMAS, J. S. 2011. Pain-related fear and avoidance of physical exertion following delayed-onset muscle soreness. *Pain*, 152, 1540-1547.
- TSANG, K. K. W., BUXTON, B. P., GUION, W. K., JOYNER, A. B. & BROWDER, K. D. 1997. The effects of cryotherapy applied through various barriers. *Journal of Sport Rehabilitation*, 6, 343-354.

- TWIST, C. & ESTON, R. 2005. The effects of exercise-induced muscle damage on maximal intensity intermittent exercise performance. *European Journal of Applied Physiology*, 94, 652-8.
- TWIST, C. & ESTON, R. 2007. The effect of muscle-damaging exercise on maximal intensity cycling and drop jump performance. *Journal of Exercise Science and Fitness*, 5, 79-87.
- ULLUM, H., HAAHR, P. M., DIAMANT, M., PALMO, J., HALKJAERKRISTENSEN, J. & PEDERSEN, B. K. 1994. Bicycle Exercise Enhances Plasma II-6 but Does Not Change II-1-Alpha, II-1-Beta, II-6, or Tnf-Alpha Pre-Messenger-Rna in Bmnc. *Journal of Applied Physiology*, 77, 93-97.
- VAILE, J., GILL, N. D. & BLAZEVICH, A. J. 2007. The effect of contrast water therapy on symptoms of delayed onset muscle soreness. *The Journal of Strength & Conditioning Research*, 21, 697-702.
- VAILE, J., HALSON, S., GILL, N. D. & DAWSON, B. 2008a. Effect of cold water immersion on repeat cycling performance and thermoregulation in the heat. *Journal of Sports Sciences*, 26, 434-440.
- VAILE, J., HALSON, S., GILL, N. D. & DAWSON, B. 2008b. Effect of hydrotherapy on recovery from fatigue. *International journal of sports medicine*, 29, 539-544.
- VAILE, J., HALSON, S., GILL, N. D. & DAWSON, B. 2008c. Effect of hydrotherapy on the signs and symptoms of delayed onset muscle soreness. *European Journal of Applied Physiology*, 102, 447-455.
- VAILE, J., O'HAGAN, C., STEFANOVIC, B., WALKER, M., GILL, N. & ASKEW, C. D. 2010. Effect of cold water immersion on repeated cycling performance and limb blood flow. *British Journal of Sports Medicine*.
- VANDER, A. J., SHERMAN, J. H. & LUCIANO, D. S. 1994. *Human physiology: The mechanisms of Body Function,* New York, McGraw-Hill.
- VERIN, E., ROSS, E., DEMOULE, A., HOPKINSON, N., NICKOL, A., FAUROUX, B., MOXHAM, J., SIMILOWSKI, T. & POLKEY, M. I. 2004. Effects of exhaustive incremental treadmill exercise on diaphragm and quadriceps motor potentials evoked by transcranial magnetic stimulation. *Journal of applied physiology*, 96, 253-9.
- VERSEY, N., HALSON, S. & DAWSON, B. 2011. Effect of contrast water therapy duration on recovery of cycling performance: a dose-response study. *European Journal of Applied Physiology*, 111, 37-46.
- WAGER, T. D. 2005. The neural bases of placebo effects in pain. *Current Directions* in *Psychological Science*, 14, 175-179.
- WALLACH, D., VARFOLOMEEV, E. E., MALININ, N. L., GOLTSEV, Y. V., KOVALENKO, A. V. & BOLDIN, M. P. 1999. Tumor necrosis factor receptor and Fas signaling mechanisms. *Annual review of immunology*, 17, 331-67.
- WANG, Y. X. & RUDNICKI, M. A. 2012. Satellite cells, the engines of muscle repair. *Nature reviews. Molecular cell biology*, 13, 127-33.
- WATKINS, L. R., MAIER, S. F. & GOEHLER, L. E. 1995. Immune activation: the role of pro-inflammatory cytokines in inflammation, illness responses and pathological pain states. *Pain*, 63, 289-302.
- WESTERBLAD, H., ALLEN, D. G., BRUTON, J. D., ANDRADE, F. H. & LANNERGREN, J. 1998. Mechanisms underlying the reduction of isometric force in skeletal muscle fatigue. *Acta Physiologica Scandinavica*, 162, 253-60.

- WESTERBLAD, H., ALLEN, D. G. & LANNERGREN, J. 2002. Muscle fatigue: lactic acid or inorganic phosphate the major cause? *News in Physiological Sciences*, 17, 17-21.
- WESTERLIND, K. C., BYRNES, W. C. & MAZZEO, R. S. 1992. A comparison of the oxygen drift in downhill vs. level running. *Journal of applied physiology*, 72, 796-800.
- WILCOCK, I. M., CRONIN, J. B. & HING, W. A. 2006a. Physiological response to water immersion: a method for sport recovery? *Sports Medicine*, 36, 747-765.
- WILCOCK, I. M., CRONIN, J. B. & HING, W. A. 2006b. Water immersion: does it enhance recovery from exercise? *International Journal of Sports Physiology and Performance*, 1, 195-206.
- WILSON, T. E., CUI, J. & CRANDALL, C. G. 2002. Effect of whole-body and local heating on cutaneous vasoconstrictor responses in humans. *Autonomic neuroscience : basic & clinical*, 97, 122-8.
- WISBEY, B., MONTGOMERY, P. G., PYNE, D. B. & RATTRAY, B. 2010. Quantifying movement demands of AFL football using GPS tracking. *Journal* of Science and Medicine in Sport, 13, 531-6.
- YAMANE, M., TERUYA, H., NAKANO, M., OGAI, R., OHNISHI, N. & KOSAKA, M. 2006. Post-exercise leg and forearm flexor muscle cooling in humans attenuates endurance and resistance training effects on muscle performance and on circulatory adaptation. *European Journal of Applied Physiology*, 96, 572-80.
- YANAGISAWA, O., NIITSU, M., TAKAHASHI, H., GOTO, K. & ITAI, Y. 2003. Evaluations of cooling exercised muscle with MR imaging and 31P MR spectroscopy. *Medicine and science in sports and exercise*, 35, 1517-23.

# **APPENDIX 1: INFORMATION FOR PARTICIPANTS**

#### 8.4 Information for participants in study 1, 2 and 3



# INFORMATION TO PARTICIPANTS INVOLVED IN RESEARCH

#### You are invited to participate

You are invited to participate in a research project entitled "The effects of water immersion and antioxidant supplementation on recovery in team sport".

This project is being conducted by a student researcher, Mr George Elias, as part of a PhD study at Victoria University under the supervision of Dr Robert Aughey and Professor Michael McKenna from the faculty of Arts, Education and Human Development.

#### **Project explanation**

This project has been designed to investigate the effectiveness of water immersion as a recovery tool in elite male team sport athletes in typical sporting scenarios. Water immersion has been shown to help speed up and improve recovery between exercise sessions. Through an enhanced recovery it is proposed that you would be able to train/compete at a greater intensity at the next session. The value of the water immersion to enhance recovery will be assessed through simple performance measures as well as physical and psychometric markers (see below).

#### What will I be asked to do?

As a participant in this research project you will be required to participate in:

 <u>Baseline testing of your performance:</u> it is important to measure your baseline performance to see if any changes occur as a result of the recovery protocol you undertake. Baseline testing will involve completing two simple performance tests: 1) a countermovement jump test and 2) a repeat sprint ability test, both of which are explained later in this document. Baseline testing of both of these measures will be performed on the same day. Two separate baseline testing sessions will be completed. Each baseline testing session will take approximately 15 minutes. This will be conducted during your normal training schedule.

- 2) <u>Small sided games</u>: This is the first testing phase of the project. You will be required to participate in Australian Football drills which are referred to as small sided games (to simulate match play). Each small sided game consists of four x five minute quarters. One game will be designed to represent a non-contact scenario of the game. Following each game, you will be asked to either wear a pair of compression pants or normal (non-compressive) shorts/pants until the next training session. The total time required per visit (including pre and post game measures) is estimated to be approximately 1 hour. Each small sided game will be separated by one week. Each of the small sided games (including taking measurements) will be completed within your normal training schedule.
- 3) <u>Diet and exercise control</u>: During each week of the study you will be asked to train normally within the team's training schedule. You will also be asked to avoid alcohol, caffeinated drinks and supplements as well as any exercise other than that given to you by the club for the 48 h prior to each SSG session.
- 4) <u>Recovery protocols</u>: This will be done straight after training and will take about 15 min just like your normal recovery. You will be asked to undertake one of 2 different recovery protocols. These being cold water immersion which requires you to be submerged in cool water for 14 min continuously and a hot/cold water immersion where you will alternate between 1 min of hot and 1 min of cold water. You will repeat this 7 times giving you a total of 14 min.
- 5) <u>Testing requirements</u>: All testing will be conducted during your normal training regime, no additional time commitments will be required. The testing will require you to perform a number of activities including psychometric measures, performance measures, blood measures and ingesting an Antioxidant in 90 min prior to exercise. These are listed in much more detail below:
- Please Note: All tasks within this project will be completed within the normal training and competition completed as part of being a player at your club and thus should not disrupt your day to day schedule in any considerable manner.

Psychometric measures. This will be done prior to and after training and before other testing sessions. This is similar to what you currently do and will take a minute to complete
 Your perceived muscular soreness and your perceived level of fatigue will be assessed using a visual analogue scale (VAS). This is essentially a way of quantifying your level of soreness and fatigue. You will be asked to rank how sore you feel on a scale of "no soreness" to "extremely sore". You will also be asked to rank how fatigued you feel on a scale of "no fatigue" to "extremely fatigued". This measurement will be taken immediately before and after exercise sessions you conduct during the project. The VAS is non-invasive and has been used previously at the Australian Institute of Sport to measure changes in perceived muscular soreness and fatigue following exercise.

In order to quantify how hard you felt each exercise session was, a session rating of perceived exertion (RPE) will be used. You will be asked to rank your perception of the exertion or intensity of each session based on a scale of 1-10 (with 1 representing the lowest ranking of intensity, and 10 representing the highest ranking of intensity).

 Blood samples: This will be done just prior to and after training and before the other scheduled testing sessions and will take 3-5 minutes to complete A small blood sample will be taken from a forearm vein by a qualified professional. This will be taken in order to determine the level of muscle damage occurring as a result of exercise and any intervention. You will be asked to sit quietly for ten minutes prior to the sample being taken. Blood samples will be taken before and after exercise session and at the next day training session. A total of four blood samples will be taken across the study period. Each blood sample with consist of 10 ml of blood. A total of 40 ml of blood will be taken across the study period. On any single day a maximum of two blood samples (10 ml each) will be taken. This equates to 20 ml of blood being taken on any one day. The blood samples will be analysed for markers of muscle damage and your anti-oxidant status.

- Performance measures: This will be done just prior to training and other scheduled testing. It is similar to what you already do and should take 5-10 min to complete both tests
   You will be required to complete two simple performance tests. This is to assess whether or not an intervention has an effect on your performance. A repeat sprint ability test (RSA) will be used to assess your ability to perform six short repeated sprints. The RSA test is commonly used among team sports. You will also be required to complete a simple jump test, known as the countermovement jump test (CMJ) following exercise. This is a simple jump test performed on a mat that records a multitude of variables during the jump.
- 6) <u>Complete a medical questionnaire:</u> this will be given to you prior to any testing taking place. If you have any questions or problems filling this questionnaire, please speak to your club doctor, club physio or the principle Victoria University investigator.

#### What will I gain from participating?

- No payment or reimbursement will be provided for participation in this project. As a participant you will gain a greater insight into your responses to the various recovery procedures you undertake. This can help you to better prepare yourself during the week for both training and matches,
- If you would like to know what your individual results or responses to any of the tests conducted, all you need to do is approach a member of the Victoria University staff and they will be happy to provide you with the information you may be seeking.

#### How will the information I give be used?

- The information you provide to the researcher (through personal details and the results of your participation in the project) will be kept strictly confidential. Only group data will be reported and presented. This data may be presented through written publication, posters and conference presentations.
- Your personal information will not be passed onto any people or organisations other than the principal investigators.

#### What are the potential risks of participating in this project?

#### Repeat sprint ability test and high intensity exercise during small-sided games and training and competition-

The performance of high intensity exercise when participating in the small sided games, training and competition involves a risk of sudden death due to myocardial infarct (heart attack) or a vasovagal episode (slow pulse, a fall in blood pressure, and sometimes convulsions). Signs and symptoms may include: sudden drop in heart rate during recovery (common) or exercise (rare); drop in blood pressure;

pale complexion; fixed facial expression; pupils constricted; participant becomes uncommunicative or slurring of words; restless and irritability; sweating; fatigue (if exercising). While vasovagal episodes are not uncommon, they are reversed quickly when employing vasovagal management plan, and long-term risks are minimal. Exercise that includes running and physical contact carries the risk of muscle soreness and stiffness.

- <u>Blood sampling</u> –Risks of blood sampling include unintentional (i) use of out-of-date sterile saline solution (saline is used during the blood sampling procedure), (ii) injection of an unintended compound / solution, (iii) transmission of infection to the participant due to lack of use of aseptic (free of microbiological organisms) techniques, (iv) discomfort, bruising and infection (for example puss, tenderness and/or redness). More serious complications such as bleeding, arterial spasm, distal arterial thromboembolism, thrombosis, and infection are theoretically possible, but rare.
- (<u>Arterial spasm</u>: temporary, sudden contraction in one location in the muscles in the wall of an artery; <u>distal arterial thromboembolism</u>: formation of a clot (thrombus) in a blood vessel that breaks loose and is carried by the blood stream to plug another vessel. This form of thromboembolism occurs in the distal section of the artery; <u>thrombosis</u>: a clot within a blood vessel which obstructs blood flow through the circulatory system)

#### Who is not eligible to participate in this project?

You will be ineligible to participate if you are injured or do not have approval by the club medical/physiotherapy staff. You will also be ineligible to participate if you are asthmatic, suffer from diabetes, have suffered with kidney or bladder stones, have kidney of liver disease, have been an organ transplant recipient or if you suffer from adverse reactions to cold water.

#### Who is conducting the study?

#### Organisations Involved in the Project:

- Victoria University (Footscray Park Campus)

#### Principal Researcher:

Dr Robert Aughey Victoria University Footscray Park Campus <u>Robert.aughey@vu.edu.au</u> 9919 5551

#### **Student Researcher:**

Mr George Elias Victoria University Footscray Park Campus george.elias@live.vu.edu.au 9919 4207

Any queries about your participation in this project may be directed to the Principal Researcher listed above.

If you have any queries or complaints about the way you have been treated, you may contact the Secretary, Victoria University Human Research Ethics Committee, Victoria University, PO Box 14428, Melbourne, VIC, 8001 phone (03) 9919 4781.

## **APPENDIX 2: INFORMED CONSENT FORM**

#### 8.5 Information for participants in study 1, 2 and 3

# Consent Form for Subjects Involved in Research

#### **INFORMATION TO PARTICIPANTS:**

We would like to invite you to be a part of a study examining the physiological and performance responses to recovery strategies following exercise.

#### **CERTIFICATION BY SUBJECT**

I,

of

certify that I am voluntarily giving my consent to participate in the study titled:

The effects of water immersion and antioxidant supplementation on recovery in team sport

being conducted at Victoria University by:

Dr Rob Aughey (Principal investigator) Professor Michael McKenna (Co Supervisor) Mr George Elias (PhD student)

I certify that the objectives of the study, together with any risks and safeguards associated with the procedures listed hereunder to be carried out in the research, have been fully explained to me by:

Dr Rob Aughey (Principal investigator) Professor Michael McKenna (Co Supervisor) Mr George Elias (PhD student)

and that I freely consent to participation involving the use on me of these procedures.

#### **Procedures:**

- Small-sided games
- Recovery intervention (cold water immersion or contrast water therapy or antioxidant supplementation).
- Performance test (repeat sprint ability and countermovement jump)
- Blood sampling
- Psychometric testing

I certify that I have had the opportunity to have any questions answered and that I understand that I can withdraw from this study at any time and that this withdrawal will not jeopardise me in any way.

I have been informed that the information I provide will be kept confidential.

Signed: ..... Date: .....

Any queries about your participation in this project may be directed to the researcher (Name: Dr Rob Aughey ph. 03-9919 5551). If you have any queries or complaints about the way you have been treated, you may contact the Secretary, University Human Research Ethics Committee, Victoria University, PO Box 14428, Melbourne, 8001 (telephone no: 03-9919 4710).

## **APPENDIX 3: MEDICAL QUESTIONNAIRE**

8.6 Medical questionnaire for participants in study 1, 2 and 3



**QUESTIONNAIRE** Responses to this questionnaire will be kept strictly confidential. The responses from this questionnaire will provide the investigators with appropriate information to establish suitability of your participation in this study. Anyone who is currently carrying a musculo skeletal injury or has a history of past, serious musculo skeletal

**MEDICAL** 

carrying a musculo-skeletal injury or has a history of past, serious musculo-skeletal injuries may be excluded from the study for health and safety reasons.

Please complete the following preliminary questionnaire.

Are you currently undertaking any form of regular exercise? YES NO (If yes, briefly describe the type and amount (i.e. Frequency, duration) of exercise you perform)

Are you a smoker?	YES	NO
Has anyone ever told you that you:		
• are overweight?	YES	NO
	DON'T K	NOW
• have high blood pressure?	YES	NO
	DON'T K	NOW

• have a heart murmur?	YES	NO			
	DON'T K	DON'T KNOW			
• are asthmatic?	YES	NO			
	DON'T K	NOW			
• are Haemophiliac?	YES	NO			
	DON'T K	NOW			
• have type 2 diabetes?	YES	NO			
	DON'T K	NOW			
• heart palpitations (sensation of abnormally	/ fast and/or irreg	ular heart beat)?			
	YES	NO			
	DON'T K	NOW			
• episodes of fainting, collapse or loss of con	nsciousness?				
	YES	NO			
	DON'T K	NOW			
		NG			
• abnormal bleeding or bruising?	YES	NO			
	DON'T K	NOW			
• gastrointestinal problems?	YES	NO			
	DON'T KNOW				
Do you, or any of your family have a history	y of cardiovascul	ar disease?			
	YES	NO			
(e.g. Heart attack, chest pain, stroke, rheumat	ic vascular disea	se)			
If YES, please elaborate:					
(e.g. Heart attack, chest pain, stroke, rheumat If YES, please elaborate:					

If YES, please elaborate:

Have you suffered any musculoskeletal injury in the last 6 months?

If YES, please elaborate:	YES	NO
Do you have any allergies (including to medications)	YES	NO
DON'T KNOW		
If YES, please elaborate:		

Have you ever experienced difficulty swallowing or any other gastrointestinal problem?

	YES	NO
	DON'T F	KNOW
11 /		

If YES, please elaborate:

Are you currently taking any medications including the following?

•	Anti-coagulants	YES	NO
	DON'T KNOW		
•	Anti-inflammatories	YES	NO
	DON'T KNOW		
•	Aspirin	YES	NO
	DON'T KNOW		

• Others, please specify:

If YES, please elaborate:

Do you have any other reason which you know of which you think may prevent you from undertaking exercise or any of the other proposed tests? YES NO

If YES, please elaborate:

I believe the information I have provided to be true and correct.

\_\_\_\_\_

Signed:\_\_\_\_\_

Date:\_\_\_\_\_

COMMENTS ON MEDICAL EXAMINATION (where appropriate):

\_\_\_\_

# **APPENDIX 4: FATIGUE, RPE & MUSCLE SORENESS**

### 8.7 Visual analogue scale used in study 1 and 2

General Fa Muscle So	atigue, RPE and reness			A NEW School of TY THOUGHT
Name:		I	Date:	
	PART ONE: Gener	al Fatigue		
<b>INSTRUCTIONS:</b>				
How severe is your	level of GENERAL FATIG	UE today? Place a	vertical mark on	
the line below to ind	licate how bad you feel your <b>G</b>	ENERAL FATIG	UE is today.	
No			Very	severe
Fatigue			fatig	ue
	PART TWO: Muscl	e Soreness		
the line below to ind	level of <b>MUSCLE SORENE</b> licate how bad you feel your <b>N</b>	-		
No			Very	severe
Soreness			sorei	iess
	PART THREE: Ses	sion RPE		
				]
	[]	RPE SO	DESCRIPTOR	
Session RP	<b>F</b> •	0	Rest	
Session RE		1	Very very easy	
		2	Easy	
		3	Moderate	
		4 5	Somewhat hard Hard	
		6		
		7	Very Hard	
		8		
		9		
		10	Maximum	

# APPENDIX 5: RAW PERFORMANCE AND PSYCHOMETRIC DATA FOR CHAPTER 4/STUDY 1

#### 8.8 Raw data version of Table 4.1

Measure	Group	Pre-training	Post-training	1 h	24 h	48 h
		Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Muscle soreness	PAS	$2.5 \pm 0.8$	$4.9 \pm 1.7$	$5.4 \pm 1.9$	$7.9 \pm 0.8$	$6.5 \pm 1.5$
	COLD	$2.8 \pm 1.2$	$5.5 \pm 0.9$	$3.0 \pm 1.2$	$3.4 \pm 1.3$	$2.8 \pm 1.2$
	CWT	$2.5 \pm 1.2$	$4.5 \pm 1.2$	$3.3 \pm 1.7$	$4.3 \pm 1.8$	$3.5 \pm 1.5$
Perceived fatigue	PAS	$2.5 \pm 0.7$	$7.1 \pm 2.0$	$5.7 \pm 2.2$	$4.7 \pm 1.7$	$4.9 \pm 1.7$
	COLD	$2.6 \pm 1.3$	$6.3 \pm 0.9$	$3.7 \pm 1.2$	$3.2 \pm 1.3$	$2.6 \pm 1.3$
	CWT	$2.3 \pm 1.2$	$5.7 \pm 1.1$	$3.1 \pm 1.1$	$3.1 \pm 1.3$	$3.0 \pm 1.4$
Countermovement	PAS	$0.67 \pm 0.19$	$0.67 \pm 0.18$	-	$0.67 \pm 0.15$	$0.65 \pm 0.18$
Jump*	COLD	$0.70 \pm 0.14$	$0.70 \pm 0.11$	-	$0.68\pm0.09$	$0.68 \pm 0.12$
	CWT	$0.69 \pm 0.15$	$0.69 \pm 0.16$	-	$0.67 \pm 0.16$	$0.69 \pm 0.12$
Squat Jump*	PAS	$0.69 \pm 0.17$	$0.67 \pm 0.23$	-	$0.67 \pm 0.17$	$0.67 \pm 0.15$
	COLD	$0.67 \pm 0.15$	$0.62 \pm 0.11$	-	$0.67 \pm 0.11$	$0.67 \pm 0.12$
	CWT	$0.67 \pm 0.17$	$0.63 \pm 0.23$	-	$0.66 \pm 0.17$	$0.66 \pm 0.15$
Total sprint time	PAS	$18.53 \pm 0.38$	$18.98 \pm 0.44$	-	$19.28 \pm 0.54$	$18.62 \pm 0.71$
(sec)	COLD	$18.63 \pm 0.49$	$19.01 \pm 0.56$	-	$18.63 \pm 0.50$	$18.66 \pm 0.44$
	CWT	$18.64 \pm 0.46$	$18.97 \pm 0.48$	-	$18.82 \pm 0.51$	$18.78 \pm 0.53$

# APPENDIX 6: HAEMOGLOBIN AND HEMATOCRIT RAW DATA FOR CHAPTER 6/STUDY 3

#### 8.9 Haemoglobin and hematocrit data for each participant in chapter 6/study 3

PARTICIPANT										
NUMBER	PF	RE	PO	ST	1	IR	24	HR	48	HR
	Hb	Hct								
1	166	50	163	50	133	48	150	48	145	45
2	153	48	152	49	94	32	134	44	144	45
3	162	47	162	47	133	38	121	43	156	43
4	159	46	155	48	137	49	165	51	154	48
5	155	47	152	48	156	48	166	47	160	46
6	151	46	156	45	132	42	150	47	147	54
7	157	49	152	47	140	46	174	47	153	46
8	180	52	181	48	132	44	174	55	161	50
9	176	51	155	48	164	51	147	49	156	48
10	160	50	149	42	146	44	135	53	174	50
11	166	52	149	48	157	52	171	48	146	44
12	171	53	175	52	172	51	183	51	177	47
13	153	47	147	44	127	50	116	47	160	46
14	168	48	165	46	106	49	151	46	149	44
15	165	49	168	49	158	48	158	48	155	47
16	158	52	166	50	160	49	133	50	126	48
17	166	51	171	54	160	52	147	49	161	44
18	152	50	166	49	164	42	117	54	177	51
19	171	47	161	40	183	49	175	48	171	47
20	167	52	172	52	148	46	139	48	175	52
21	167	44	160	48	118	47	128	54	120	44

## **APPENDIX 7: ANALYSIS OF ADDITIONAL JUMP**

# VARIABLES FOR CHAPTER 4/STUDY 1

#### 8.10 Raw data for peak jump height, peak jump power and peak jump force for

#### chapter 4/study1

		Static Jump						
		Pre	Post	24 h	48 h			
	PAS	0.6 ± 0.1	0.6 ± 0.1	0.6 ± 0.1	0.6 ± 0.2			
Peak Distance	CWI	0.6 ± 0.2	$0.6 \pm 0.1$	$0.6 \pm 0.1$	0.6 ± 0.1			
	CWT	0.6 ± 0.2	$0.6 \pm 0.1$	$0.6 \pm 0.1$	0.6 ± 0.1			
	PAS	1159.8 ± 349.8	1348.9 ± 453.1	1189.1 ± 360.1	1258.2 ± 447.8			
Peak Force	CWI	1138.7 ± 328.0	1331.1 ± 387.3	1205.0 ± 347.1	1239.2 ± 446.5			
	CWT	1146.8 ± 361.0	1322.5 ± 352.0	1174.2 ± 358.5	1226.5 ± 348.4			
	PAS	4004.4 ± 620.2	4203.6 ± 571.3	4127.4 ± 564.7	4171.6 ± 649.1			
Peak Power	CWI	4043.9 ± 547.7	4191.3 ± 568.6	4144.9 ± 589.1	4200.6 ± 576.4			
	CWT	3991.3 ± 567.9	4196.6 ± 552.4	4104.8 ± 491.9	4181.4 ± 529.1			

	Counter Movement Jump							
		Pre	Post	24 h	48 h			
	PAS	0.7 ± 0.1	0.6 ± 0.1	0.7 ± 0.1	0.7 ± 0.2			
Peak Distance	CWI	0.7 ± 0.2	0.6 ± 0.1	0.7 ± 0.1	0.7 ± 0.1			
	CWT	0.7 ± 0.1	0.7 ± 0.1	0.7 ± 0.1	0.7 ± 0.1			
	PAS	1498.3 ± 449.9	1521.5 ± 439.7	1464.5 ± 408.8	1516.9 ± 354.4			
Peak Force	CWI	1465.5 ± 436.2	1530.1 ± 390.1	1479.3 ± 414.8	1522.5 ± 433.1			
	CWT	1484.3 ± 375.9	1498.3 ± 423.9	1481.9 ± 386.8	1486.3 ± 394.9			
	PAS	4157.9 ± 705.1	4180.0 ± 622.1	4146.8 ± 618.1	4194.1 ± 563.7			
Peak Power	CWI	4209.7 ± 522.4	4205.9 ± 542.2	4189.1 ± 469.2	4131.1 ± 584.5			
	CWT	4208.1 ± 530.2	4158.4 ± 553.7	4131.9 ± 499.5	4146.8 ± 524.0			