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
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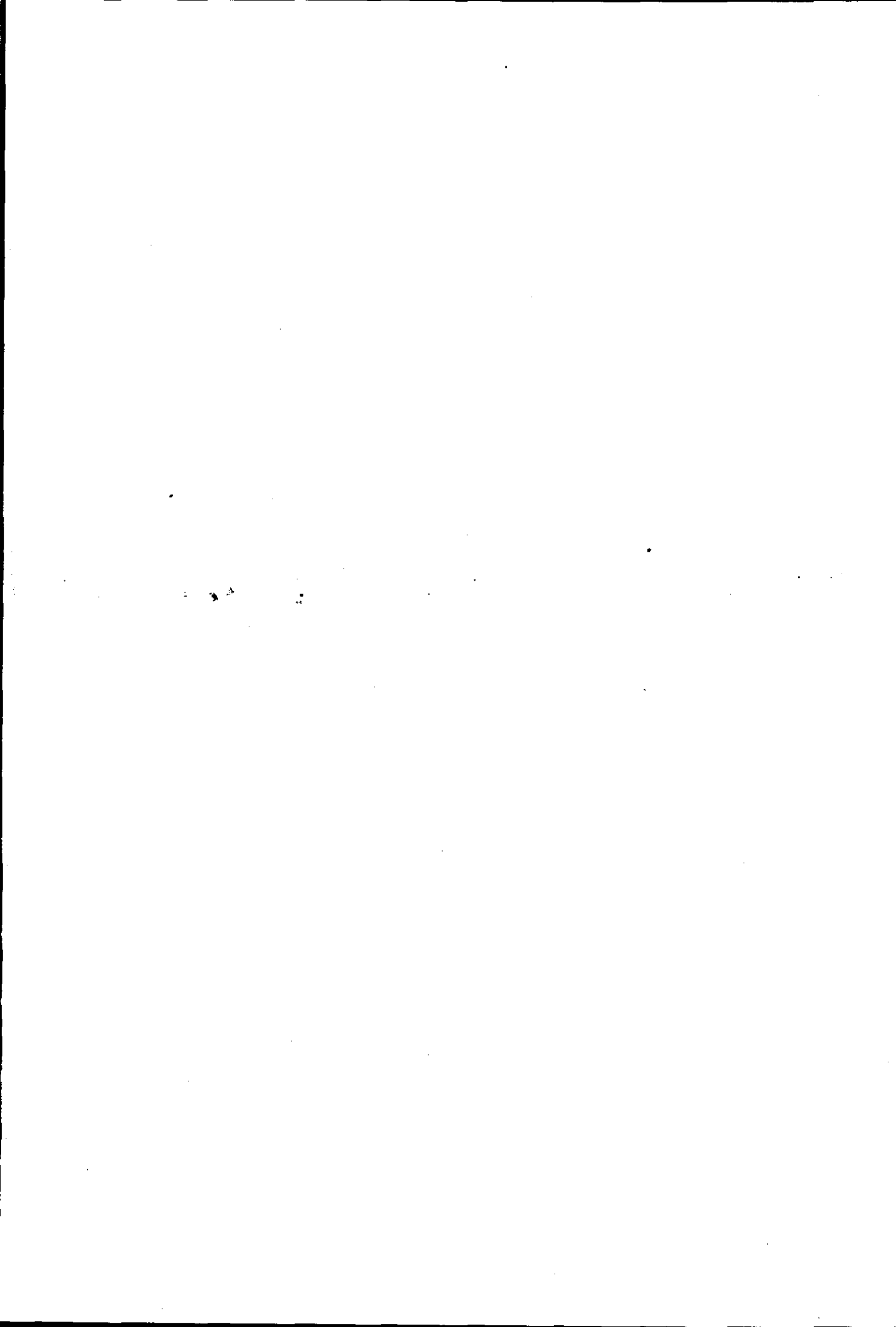
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# **Fitness assessment and recovery strategies for soccer**

By


**Samuel Erith**

A Doctoral Thesis

Submitted in partial fulfilment of the requirements for the reward of Doctor  
of Philosophy of Loughborough University

**April 2007**

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## ABSTRACT

In recent years our understanding of the physical demands of soccer has improved. We know that the intensity at which the game is played has increased and that the fixture schedules for professional teams can often be very congested. These factors are likely to have increased the importance placed on the physical condition of players. Therefore, the process of monitoring the fitness levels of players is likely to be an important task within clubs. Any fitness assessments that are employed need to be sensitive enough to detect changes that may result from different training stimuli. A further critical consideration for clubs is what are the best practices to implement in order to maximise recover between matches? The two areas that are central to successful recovery of performance are the restoration of muscle and liver glycogen stores and the rapid reduction of muscle soreness.

We have a good understanding of the importance of carbohydrate feeding in the immediate hours following the completion of exercise, furthermore that high levels of carbohydrate consumed during short recovery periods can improve subsequent endurance running capacity in both continuous and intermittent exercise. However, there is dearth of literature investigating the effects that different types (glycemic index) of carbohydrates have on subsequent performance of high intensity intermittent exercise. Furthermore, we know that the movement patterns experienced in soccer commonly induce symptoms of muscle damage. Despite this there is little research-based information on modalities that reduce these potentially detrimental side-effects (Barnett, 2006).

For these reasons the series of investigations that have been conducted in this thesis were designed with the intent to examine areas that are critical to the preparation and recovery of soccer players.

The first of five experimental chapters collated information on the use of fitness testing within English professional football. It was concluded that the practise of fitness testing players is extremely commonplace and that field-based testing protocols were far more popular an assessment method. The second experimental chapter went on to demonstrate that the most commonly used fitness test within professional football

(MSFT) was sensitive enough to detect performance changes that occur as a result of training. A further finding within the context of the question was that it is possible for female players to significantly improve aerobic capabilities with additional high intensity aerobic training.

The third experimental chapter investigated the effect different glycaemic index high CHO diets could have on recovery of performance following 90 min of intermittent soccer type exercise. This study concluded that consuming either predominately high or low GI CHO mixed meals in the 24h recovery period between bouts of high intensity prolonged intermittent exercise had no difference on measures of performance.

The final two experimental chapters went on to investigate the effects of cold water immersion on indices of muscle damage following intermittent exercise. Results from these investigations suggest that submerging individuals in 10°C water immediately following high intensity intermittent exercise reduces some but not all indices of muscle damage.

In summary, fitness assessments of players are commonly made within professional football clubs. The most common test used was the MSFT and this appears to be sensitive to changes that result as a consequence of training. During recovery from high intensity intermittent exercise the importance of carbohydrates is apparent although the type of carbohydrate appears to be less important, furthermore, cold-water immersion may be effective in reducing some but not all indices of muscle damage.

**Keywords:** *soccer, intermittent exercise, fitness assessment, recovery, glycaemic index, muscle soreness, cryotherapy.*



## PUBLICATIONS AND CONFERENCE PROCEEDINGS

Erith S. J (2004). An Overview of Fitness Testing within English Professional Football. *Journal of Sports Sciences (Annual Conference of the British Association of Sport and Exercise Sciences)*, 22 (3): p247-248.

Erith S.J., C. Williams, E. Stevenson, S. Chamberlain, P. Crews and I. Rushbury (2004). The effect of high carbohydrate meals with different glycaemic indices on 22h recovery from prolonged intermittent shuttle running. *Conference Proceedings: 9th Annual Congress. European College of Sport Science*.

Erith, S. and C. Williams (2005). Fitness Testing Within English Professional Soccer And Training Induced Changes In Elite Female Soccer Players. *Med Sci Sports* 37(5): S78.

Bailey, D. M, S. J. Erith, P.J. Griffin, D.S. Brewer, A. Dowson and C. Williams (2005) Influence of cold water immersion on indices of muscle damage following prolonged intermittent shuttle-running exercise. *J Physiol* 565P, PC22 Communications.

Erith S., D. Bailey, N. Gant, J. Hupton, A. Thomas and C. Williams (2005). The influence of cold-water immersion on the indices of muscle damage following prolonged high intensity intermittent exercise. *Journal of Sports Sciences (Annual Conference of the British Association of Sport and Exercise Sciences)*, 23 (11/12): p1187.

Erith S.J., C. Williams, E. Stevenson, S. Chamberlain, P. Crews and I. Rushbury (2006) The effect of high carbohydrate meals with different glycemic indices on recovery of performance during prolonged intermittent high-intensity shuttle running. *International Journal of Sports Nutrition and Exercise Metabolism*, 16, 393-404.

Bailey D.M., S. J. Erith, P. J. Griffin, A. Dowson, D.S. Brewer, N. Gant and C. Williams (In Press). Influence of cold-water immersion on indices of muscle damage following prolonged intermittent shuttle running. *Journal of Sports Science*.

## ACKNOWLEDGEMENTS

I am indebted to a number of people for their help and support in producing this Doctoral thesis.

First and foremost the greatest show of thanks should undoubtedly be given to Professor Clyde Williams. The support, guidance and encouragement Clyde has given me over the past 6 and a half years has been outstanding and unrelenting. Clyde's decision to allow me to carry on with this thesis on a part-time basis whilst advancing my professional career with full-time employment has given me the opportunity to gain some tremendous experiences, for which I will always be grateful.

Anyone who has conducted a research study in the area of exercise physiology will know the importance of help from within your research team. I would like to thank a number of colleagues for their support, guidance and most importantly friendship during the time I have spent researching at Loughborough, namely, Dr Ajmol Ali, Dr David Bailey, Dr Andy Foskett, Dr Nick Gant, Dr Ching-lin Wu, Dr Emma Stevenson and Dr James Betts. Furthermore, I would like to thank Maria Nute and Spencer Newport for their excellent help and assistance around the laboratory.

It is very important that I thank all those who have kindly volunteered to take part in the studies that are included in this thesis. There are too many of you to list but the sacrifices in time and lifestyle you made during these experiments is greatly appreciated.

I must thank Dr Richard Hawkins (Formerly of The Football Association) and Alan Hodson – Head of the Medical and Exercise Science Department for the belief they showed in me. The financial support that The Football Association have given me was critical in order to fund this Ph.D.

Finally, I would like to give particularly acknowledgement to my Fiancé, Jo who has stood by me and supported me throughout this work. I am indebted to her for her unbelievable level of understanding.

# TABLE OF CONTENTS

LIST OF FIGURES.....	x
LIST OF TABLES.....	xi
LIST OF ABBREVIATIONS.....	xii-xiii
Chapter 1 INTRODUCTION.....	1
Chapter 2 REVIEW OF LITERATURE	
2.1 Introduction.....	6
2.2 Fitness testing soccer players.....	6
2.2.1 Laboratory tests for endurance performance.....	7
2.2.2 Laboratory tests for strength and power parameters.....	9
2.2.3 Field based physiological assessment.....	9
2.3 Fitness training soccer players.....	13
2.3.1 Endurance training interventions within soccer.....	13
2.3.2 Strength training interventions within soccer.....	16
2.3.3 Concurrent training studies.....	18
2.4 Carbohydrate intake during recovery from exercise.....	23
2.4.1 The amount of carbohydrate required.....	23
2.4.2 The timing of carbohydrate.....	24
2.4.3 The type of carbohydrate.....	26
2.5 Exercise induced muscle damage.....	29
2.5.1 Aetiology of exercise induced muscle damage.....	30
2.5.2 Indices of muscle damage.....	32
2.6 Effect of cryotherapy on muscle damage.....	34
2.7 Summary.....	37
Chapter 3 GENERAL METHODS	
3.1 Introduction.....	42
3.2 Experimental testing procedures.....	43
3.2.1 Testing Facilities.....	43
3.2.2 Anthropometry.....	43
3.2.3 Estimation of maximal oxygen uptake.....	44
3.2.4 Expired air collection and analysis.....	44
3.2.4.1 Standard Douglas bag method.....	44
3.2.4.2 Analysis of expired air.....	45
3.2.5 Heart rate monitoring and RPE.....	45
3.2.6 Dietary analysis.....	46
3.2.7 The Loughborough Intermittent Shuttle Test .....	46
3.2.8 Blood sampling.....	47
3.2.9 Blood Analysis.....	48
3.2.9.1 Plasma glucose, free fatty acids and glycerol.....	48
3.2.9.2 Serum myoglobin and creatine kinase activity.....	48
3.2.9.3 Plasma volume.....	48
3.2.9.4 Serum insulin and cortisol.....	49

	3.2.9.5 Blood lactate.....	49
	3.2.9.6 Intra-assay variation.....	49
<b>Chapter 4</b>	<b>AN OVERVIEW OF FITNESS TESTING PRACTICES WITHIN ENGLISH PROFESSIONAL FOOTBALL</b>	
4.1	Introduction.....	51
4.2	Methods.....	52
4.3	Results.....	53
4.4	Discussion.....	55
<b>Chapter 5</b>	<b>THE SENSITIVITY OF FITNESS TESTS, COMMONLY USED WITH PROFESSIONAL SOCCER, TO DETECT PERFORMANCE CHANGES THAT OCCUR AS A CONSEQUENCE OF TRAINING IN ELITE FEMALE SOCCER PLAYERS</b>	
5.1	Introduction.....	57
5.2	Methods.....	58
5.3	Results.....	61
5.4	Discussion.....	68
<b>Chapter 6</b>	<b>THE EFFECT OF HIGH CARBOHYDRATE MEALS WITH DIFFERENT GLYCEMIC INDICES ON RECOVERY OF PERFORMANCE DURING PROLONGED INTERMITTENT HIGH-INTENSITY SHUTTLE RUNNING</b>	
6.1	Introduction.....	72
6.2	Methods.....	74
6.3	Results.....	77
6.4	Discussion.....	82
<b>Chapter 7</b>	<b>THE INFLUENCE OF COLD-WATER IMMERSION ON THE INDICES OF MUSCLE DAMAGE FOLLOWING PROLONGED HIGH INTENSITY INTERMITTENT EXERCISE</b>	
7.1	Introduction.....	86
7.2	Methods.....	88
7.3	Results.....	92
7.4	Discussion.....	96
<b>Chapter 8</b>	<b>A FURTHER INVESTIGATION INTO THE INFLUENCE OF COLD-WATER IMMERSION ON THE INDICES OF MUSCLE DAMAGE FOLLOWING PROLONGED HIGH INTENSITY INTERMITTENT EXERCISE</b>	
8.1	Introduction.....	100
8.2	Methods.....	101
8.3	Results.....	104
8.4	Discussion.....	109
<b>Chapter 9</b>	<b>GENERAL DISCUSSION</b>	
9.1	Introduction.....	112
9.2	Fitness assessment within soccer.....	113
9.3	Recovery of performance.....	114

## LIST OF FIGURES

3.1	Diagrammatical representation of the LIST exercise protocol.....	47
5.1	Displays the results from the MSFT and the treadmill maximal oxygen uptake test pre- and post-training.....	65
5.2	Displays the blood lactate concentrations during the submaximal treadmill test pre- and post training.....	66
5.3	Heart rate responses during the submaximal treadmill test pre- and post- training.....	67
6.1	Plasma FFA concentrations ( $\text{mmol.l}^{-1}$ ) during R2 in the HGI and LGI trials.....	80
6.2	Plasma Glycerol concentrations ( $\text{mmol.l}^{-1}$ ) during R2 in the HGI and LGI trials.....	80
6.3	Plasma glucose concentrations ( $\text{mmol.l}^{-1}$ ) during R2 in the HGI and LGI trials.....	81
6.4	Serum insulin concentrations ( $\mu\text{IU.ml}^{-1}$ ) during R2 in the HGI and LGI trials.....	81
7.1	Perceived muscle soreness following exercise for cryotherapy (solid bars) and control (clear bars) groups.....	94
7.2	Serum creatine kinase activity following exercise for cryotherapy (solid line) and control (broken line) groups.....	94
7.3	Serum myoglobin concentration following exercise for cryotherapy (solid line) and control (broken line) groups.....	95
8.1	Perceived muscle soreness following exercise for cryotherapy (solid bars) and sham (clear bars) groups.....	106
8.2	Sprint performance (15m) 24h post exercise for cryotherapy (solid bars) and sham (clear bars) groups.....	107
8.3	Serum myoglobin concentration following exercise for cryotherapy (solid line) and sham (broken line) groups.....	107
8.5	Serum creatine kinase activity following exercise for cryotherapy (solid line) and sham (broken line) groups.....	108
9.1	Schematic representation of the thesis major studies and a flow summary of how each study was conceptualised.....	119

## LIST OF TABLES

2.1	A summary of investigations employing cryotherapy prior to, during or after exercise.....	39-40
3.1	Intra-assay variation for blood sampling data.....	49
4.1	The most frequently used fitness tests within professional football clubs.....	53
5.1	Results from all fitness tests pre- and post-training.....	63
6.1	Characteristics of test meals (for a 70kg subject).....	79
7.1	Physiological characteristics and physical activity status of groups.....	89
7.2	Isometric maximal voluntary contraction of the leg flexors and extensors following intermittent shuttle running and treatment.....	93
8.1	Physiological characteristics and physical activity status of groups....	102
8.2	Isometric maximal voluntary contraction of the knee and hip flexors and extensors following intermittent shuttle running and treatment..	105

## LIST OF ABBREVIATIONS

List of the abbreviations and acronyms contained in this thesis. Abbreviations are defined in the text in the first instance.

ANOVA	analysis of variance
BM	body mass
BMI	body mass index
CHO	carbohydrate
EDTA	potassium ethylenediaminetetraacetic acid
EIMD	exercise induced muscle damage
FFA	free fatty acids / non-esterified fatty acids
g	gram
h	hour
HR	heart rate
HR <sub>max</sub>	maximum heart rate
IMTG	intra-myocellular triacylglycerol
kJ	kilojoule
kg	kilogram
l	litre
LIST	Loughborough intermittent shuttle test
LOA	limit of agreement
mg	milligram
min	minute
MJ	megajoule
ml	millilitre
mmol	millimole
mOsmol	milliosmole
MSFT	multi stage fitness test
MVC	maximal voluntary contraction
NSAID	non-steroidal anti-inflammatory drugs
PV	plasma volume
$\dot{Q}$	cardiac output
RER	respiratory exchange ratio
RPE	subjective rating of perceived exertion
s	second
SEM	standard error of measurement
SD	standard deviation
STPD	standard temperature and pressure dry
T <sub>core</sub>	core temperature
T <sub>sk</sub>	skin temperature
$\bar{T}_{sk}$	weighted mean skin temperature
V <sub>O<sub>2</sub></sub>	oxygen uptake
$\dot{V}O_{2\max}$	maximum oxygen uptake
$\dot{V}_E$	expired gas volume

$\bar{x}$  mean  
°C degrees celsius



# CHAPTER I

## INTRODUCTION

Success or failure in soccer is dependant on the culmination of many factors that include the skills, experience and fitness of the players. Clearly, teams need players with a good mastery of basic techniques and a firm tactical understanding of how and when to implement their skills to best advantage. However, the physical demands of elite soccer are such that players cannot survive only on their skill, experience and tactical knowledge.

There is a growing body of available information that describes the physical demands of the game in great detail. For example, we now have information on distance covered during games and a good understanding of the types and intensities of movements of individual players as the result of video and computer assisted match analyses. It is becoming more evident that the physical fitness of players has a significant influence on the overall success of teams (Balsom et al., 1999; Mohr and Bangsbo, 2001; Mohr, Krstrup and Bangsbo et al., 2003). Therefore, one of the contributions of the physiological sciences to improving the preparation of players in elite level soccer is the development of a better understanding of the fitness requirements of the game.

Monitoring the fitness of players provides important information about the physical condition of individuals in terms of their strengths and weaknesses. Furthermore, this information provides an essential benchmark against which the success of rehabilitation from injury can be assessed. Clearly, the types of assessments that are employed will vary depending on the fitness components that are deemed to be functionally relevant. Therefore, it is surprising that there appears to be very little published information on whether or not professional football clubs use fitness testing routinely; what tests they use, if any, and how are the results used? Furthermore, if testing is routinely used as a measure of the effectiveness of training and recovery from injury, it is important to know whether or not these tests are sensitive enough to detect the training-induced adaptations that may occur. Reliable and relevant information on fitness can be of great

benefit to players, coaches and the team's medical staff because it can be used to inform decisions about the readiness of the individual for match play.

Therefore, one of the aims of this thesis was to find out whether or not Premier League football clubs in England use fitness testing routinely and determine the nature of the tests together with how the results of the tests are used. In this context the question of the sensitivity of fitness tests to training-adaptations in well-trained players was also addressed.

Elite players have very full match and training schedules that extend throughout the competitive season. The demands on players are often so great that training per se is reduced or even abandoned to ensure an adequate recovery between matches. Therefore, it is not surprising that recovery strategies have become the focus of a great amount of interest. Several strategies designed to increase recovery of functional fitness between periods of prolonged high intensity exercise have been examined (Barnett, 2006). Unfortunately, the number of well-controlled studies examining recovery strategies on subsequent performance within the area of intermittent exercise is limited. The two areas that are central to successful recovery of performance are the restoration of muscle and liver glycogen stores and the rapid reduction of muscle soreness.

Extensive research has examined the role certain foods play in the whole recovery process. In particular, we have a good understanding of the importance of carbohydrate feeding in the immediate hours following the completion of exercise. For example, it is now well accepted that in order to maximise the replenishment of muscle glycogen stores in 24 hours after prolonged heavy exercise a diet that provides the equivalent of 9-10 g·kg<sup>-1</sup> BM of carbohydrate is necessary (Burke et al., 2001). This amount of carbohydrate in the recovery diet restores endurance capacity during prolonged constant pace running (Fallowfield and Williams, 1997) as well as during prolonged intermittent brief high intensity shuttle running (Nicholas et al., 1997). However, some questions remain unanswered for example, does the type of carbohydrate in the recovery diet influence the restoration of functional fitness?

Historically carbohydrates were classified as being either 'simple' or 'complex' depending on whether or not they contained fibre. Those carbohydrates containing fibre

were automatically classified as 'complex'. However, the current classification is based on the glycaemic response following the ingestion of a standard amount of the food (usually 50g of available carbohydrate). Those carbohydrates that result in a slow rise and low peak in blood glucose concentrations are termed 'low glycaemic' carbohydrates whereas those that produce a rapid rise and high peak in blood glucose concentrations are 'high glycaemic' carbohydrates (Jenkins et al., 1984). The glycaemic index is a method of quantifying the glycaemic responses to a prescribed amount of carbohydrate in relation to the same amount of a standard carbohydrate, usually glucose but also white bread (Jenkins et al., 1981). Currently carbohydrate foods are classified as high (HGI) or low (LGI) glycaemic index that can be given numerical values in relation to reference value for glucose of 100. The glycaemic index of commonly available foods is assessed by determining the area under the blood glucose curve for 50g of available carbohydrate in relation to the area under the blood glucose curve for 50g of glucose measured over a two hour postprandial period (Henry et al., 2005).

Several studies have shown that consuming HGI carbohydrates immediately after prolonged exercise promotes a greater rate of muscle glycogen resynthesis than when carbohydrate intake is delayed (Ivy, 1998). A HGI carbohydrate recovery diet restores endurance running capacity (Fallowfield and Williams, 1997, Nicholas et al., 1997) whereas ingesting an isocaloric mixed diet with a normal amount of carbohydrate does not. Furthermore, it appears that a HGI carbohydrate recovery diet restocks muscle glycogen stores to a greater extent than does a LGI carbohydrate recovery diet (Burke et al., 1993). However, there is only a limited literature on the influences of HGI and LGI carbohydrate recovery diets on subsequent performance. In one recent study Stevenson and colleagues reported that endurance running capacity was greater 24 hours after prolonged constant paced treadmill running when their subjects consumed a LGI rather than a carbohydrate recovery diet (Stevenson et al., 2005). However, it cannot be assumed that the benefits of a LGI carbohydrate recovery diet occur following all forms of exercise. Therefore, another aim of this thesis was to examine the influences of HGI and LGI recovery diets on endurance capacity during prolonged intermittent high intensity exercise because this is the type of exercise that most closely resembles the activity patterns that occur in soccer (Nicholas et al., 2000).

The activity patterns in soccer are such that players sprint, jog, run and walk in almost a random fashion that is dictated by their playing position, who has possession of the ball, their fitness in relation to that of the opponents as well as how long they have played. Acceleration, deceleration and changing direction at speed are some of the most taxing of the non-contact activities demanded of soccer players during competition. These activities produce large stresses on the body as a whole and on the skeletal muscle, tendons, ligaments and joints- so much so that post-competition muscle soreness is a common experience for most soccer players. Although we are relatively knowledgeable about how to restock the body's carbohydrate stores and to rehydrate successfully during the recovery period there is little research-based information on modalities that reduce post-exercise muscle soreness (Barnett, 2006).

Any strategy that can limit the amount of muscle soreness may help restore performance during subsequent exercise more rapidly. Recently cold-water immersion has gained in popularity among elite sports persons because of anecdotal evidence to suggest that post-exercise 'ice baths' help accelerate recovery. However, there is only limited evidence available on the effectiveness of these techniques to reduce the severity of exercise induced muscle damage (EIMD). The studies that have been published tend to concentrate on isolated muscle groups. Nevertheless, the strongest support for the effectiveness of cold-water (ice baths) immersion as an aid to rapid recovery is provided by the anecdotal reports from players. Therefore, another aim of research described in this thesis was to investigate the effectiveness of ice baths in reducing the post-exercise muscle soreness following prolonged high intensity intermittent exercise.

In summary, the series of studies that are described in this thesis were designed to examine areas that are critical to the preparation and recovery of soccer players. They evolved after answering the original question about the assessment of fitness of players in Premier League Football Clubs in England i.e. are the fitness tests sensitive enough to detect training-induced adaptations and are there ways of improving the recovery of players following training and competition. Thus the aim of the first study was to provide an overview of the prevalence of fitness testing within English professional football (Chapter 4). In the second study the aim was to determine the sensitivity of the most commonly used fitness tests by applying them before and after a period of intense training of already well-trained soccer players (Chapter 5). In the third study the aim

was to compare the benefits of HGI and LGI 24 hour recovery diets on subsequent performance of prolonged intermittent high intensity shuttle running (Chapter 6). The aims of studies described in Chapters 7 and 8 were to examine the effectiveness of cryotherapy on reducing muscle soreness and restoration of performance following prolonged intermittent high intensity shuttle running. The final chapter attempts to summarise the findings of these studies in the context of the relevant literature and signpost directions for future studies. Therefore, the overall aim of this thesis was to further our understanding and contribute to the limited scientific evidence available within these specific areas affecting the preparation and recovery of elite soccer players.

## CHAPTER 2

### REVIEW OF LITERATURE

#### 2.1 Introduction

The purpose of this chapter is to critically examine the pertinent literature concerned with aspects of assessment, training and recovery strategies for soccer players. The chapter is divided into 5 main sections. The first section covers a number of studies that have attempted to establish testing protocols and procedures for the physiological assessment of players. The second section aims to highlight and critique a number of training methods used within football, namely concerning the areas of endurance and strength. Following this, the next section will consider carbohydrate intake during recovery from intermittent exercise with a particular focus on the effect of glycaemic index on exercise performance and substrate metabolism. The final two sections of this chapter will focus on the aetiology of exercise induced muscle damage and the effect of cryotherapy on indices of muscle damage.

#### 2.2 Fitness Testing soccer players

The physical demands of soccer are now well understood. Successful players not only need a mastery of the technical and tactical aspects of the game but also competencies in a number of key physical components including aerobic and anaerobic power, muscle strength, flexibility and agility (Reilly and Thomas, 1976; Ekblom, 1986; Reilly and Doran, 2003). It appears that the physical match demands have become more challenging in recent years (Strudwick and Reilly, 2001) and this has implications for the preparation, training and nutritional programmes of players (Reilly and Golbourne, 2003).

In order, for fitness staff, technical coaches and managers to have an objective understanding of their players physical condition it is necessary to profile players

through fitness assessments. There are a wide variety of fitness tests available that can be used in the laboratory or field to make objective assessments of individuals, many of these are discussed below.

### **2.2.1 Laboratory tests for endurance parameters**

Aerobic endurance performance in soccer can be determined by maximal oxygen uptake ( $\text{VO}_2 \text{ max}$ ), lactate threshold and running economy (Pate and Kriska, 1984). A maximal oxygen uptake test is one of the most commonly used measures to assess aerobic power and performance. Definitions of maximal aerobic power ( $\text{VO}_2 \text{ max}$ ) and protocols used for its assessment have been described previously in textbooks (Astrand & Rodahl, 1986) and are therefore not included within this literature review. However, Howley and co-workers (1995) have recently questioned some of the long established criteria used to determine whether an  $\text{O}_2$  uptake level is actually maximal (*see* Shepard, 1984). Howley and co-workers (1995) suggested that criteria such as achievement of a plateau in  $\text{VO}_2$ , a respiratory exchange ratio (RER) above 1 and the elevation of heart rate to age-predicted maximum used to determine  $\text{VO}_2 \text{ max}$  are somewhat dubious. Howley and colleagues (1995) suggest careful consideration should be taken when maximal testing including an orientation session to ensure reliable results, suitable warm-up procedures and relevant protocols (with associated gas collection procedures), and appropriate criteria for the verifications of the achievement of  $\text{VO}_2 \text{ max}$ . In a recent review article Svensson and Drust (2005) stated that  $\text{VO}_2 \text{ max}$  tests for soccer players should be performed on a treadmill as opposed to a cycle ergometer because a running test is more relevant to the activity patterns in soccer.

Assessment of blood lactate concentrations during sub-maximal treadmill running is often used as an assessment of endurance performance (Sjodin & Jacobs, 1981). Clearly, the practitioner is concerned with the onset of blood lactate accumulation. Davis (1985) describes this transitional phase as the change between the predominance of aerobic and anaerobic metabolism. Some investigators have used  $4 \text{ mmol}^{-1}$  onset of blood lactate accumulation as an objective reference point (Sjodin & Jacobs, 1981). The concept of an anaerobic threshold has always remained a contentious issue and it is not within the context of this literature review to examine it (For further information, see Brooks, 1985).

A recent study by McMillan and co-workers (2005) examined the lactate threshold responses during a season with professional British youth soccer. A total of 9 players completed all 6 lactate tests throughout different stages of the season. McMillan and colleagues (2005) found running velocity at two lactate thresholds increased from the start of pre-season training to the early weeks of the competitive season, however the following 4 testing periods (December, January, April and June) showed no changes. Others have used 'lactate-testing' with senior professional players throughout the season (Edwards et al., 2003). For example, a study examining the effects of 5 weeks of pre-season training found improvements in aerobic performance using this method (Bangsbo, 1994). Interestingly, Bangsbo and co-workers (1994) found that a sub-maximal lactate test was more sensitive to training changes compared to a maximal oxygen uptake test. However, Dunbar (1999) reported no changes in lactate response to sub-maximal treadmill running following the pre-season period in another group of English professional players. There are a number of threats to the reliability of lactate testing including diet and nutrition, training state (Gollnick et al., 1988) and the nature of the exercise prescribed (*intermittent* compared to *continuous*) (Williams, 1990).

An additional way to assess endurance performance during running activity in the laboratory is to assess running economy. Running economy has been defined as the energy demand for a given velocity of sub-maximal running and is determined by measuring the steady-state  $O_2$  consumption at a standardised running velocity (Saunders et al., 2004). Interestingly, several authors have reported that running economy and maximal oxygen uptake are poorly correlated (Conley and Krahenbuhl, 1980; Daniels and Daniels, 1992), suggesting that different physiological factors may affect these values. A recent study conducted on elite youth soccer players suggested that some of the endurance improvements recorded could be due to improvements in running economy (McMillan et al., 2005). Furthermore, Helgerud and co-workers (2001) found improvements in running economy following 8 weeks of two high intensity running interval sessions per week with soccer players. Moreover, Hoff and Helgerud (2003) reported a 5% improvement in running economy when using a combined training programme of both strength and interval running training.



## **2.2.2 Laboratory tests for strength and power parameters**

Force production during eccentric and concentric actions can be assessed with the use of isokinetic dynamometry (Seger et al., 1988). Several authors have reported that isokinetic dynamometry provides a controlled environment in which the neuromuscular performance of the joint system can be stressed (Baltzopoulos and Gleeson, 2001; Sapega et al., 1982). Iossifidou and Baltzopoulos (1998) state that one of the main advantages of isokinetic dynamometry is the accuracy in assessment provided by constant pre-selected velocity of movement. This method of strength testing has been used to test Swedish, Japanese and Greek players with all three studies demonstrating a difference in strength scores depending on playing standard (Oberger et al., 1986; Togari et al., 1988; Gillis et al., 2003). A common use of isokinetic dynamometry has been to measure the muscle strength of agonist and antagonist groups of muscles, particular the hamstrings (H) and quadriceps (Q) ratio (Aagaard et al., 1995; Aagaard et al., 1998). A functional assessment of H/Q ratio can be calculated from the maximal eccentric hamstring strength divided by maximal concentric quadriceps strength during extension, or vice versa in flexion (Aagaard et al., 1998). This method is considered more appropriate than the conventional calculation of dividing maximal knee flexion strength by maximal knee extension strength. However, Gleeson and Mercer (1992) have shown that several trials may be needed to familiarise the individual and to overcome any reliability problems during isokinetic measurements. A further drawback of this testing method is that the isolation of muscle groups will reduce the validity of the measurements to functional performance, as the multi-joint movements involved in most sports will not be recreated (Kannus, 1994), this is particularly pertinent when considering the testing of soccer players.

## **2.2.3 Field based physiological assessment**

### **2.2.3.1 Field based assessment of endurance performance**

Field assessments of soccer players often incorporate more function movement patterns, thus increasing the validity of these tests (Balsom, 1994). Field tests have the advantage that all players in a team can be tested frequently, easily and rapidly at low cost (Krustrup et al., 2005).

A number of researchers have attempted to establish protocols to examine aerobic endurance performance in the field. Ramsbottom and co-workers (1988) designed a multi-stage fitness test. This test involves participants running continuously between lines placed 20m apart, paced by an audio 'bleep' sound played from a tape recorder. The test is progressive in nature and the time allowed to cover 20m gets progressively shorter every minute. The overall aim for individuals is run for as long as possible. The test allows for the estimation of  $VO_2 \text{ max}$  from test results, which has been validated by Ramsbottom and co-workers (1988). In this study, a relatively strong correlation of 0.92 was reported between  $VO_2 \text{ max}$  and shuttle level achieved.  $VO_2 \text{ max}$  is estimated from a regression equation based on the relationship between  $VO_2 \text{ max}$  and the peak running speed achieved during the last stage. There are several examples of the multi-stage fitness test being used within English football (Davis et al., 1992; Strudwick et al., 2002).

Bangsbo and Linquist (1992) have proposed a soccer-specific endurance test. This protocol lasts 16.5 min, during which players alternate between 40 bouts of high intensity exercise lasting 15 seconds and 40 bouts of low intensity exercise each lasting 10 sec. The activity patterns involve a variety of soccer specific running movements around a circuit. Recently, Chamari and colleagues (2005) compared this field-based test to more traditional laboratory based testing with elite young soccer players. The Bangsbo test did not correlate significantly with  $VO_2 \text{ max}$  in young soccer players (Chamari et al., 2005).

The Yo-Yo intermittent endurance tests were designed by Bangsbo (1993) in an attempt to make field based assessments of players specific to football. Bangsbo (1993) established two types of Yo-Yo tests. The first test was developed to assess an individual's ability to perform bouts of repeated intense intermittent exercise (Yo-Yo *intermittent endurance test*) and the second was designed to assess an individual's ability to recover from intense exercise (Yo-Yo *intermittent recovery test*). These tests are similar to the multi-stage shuttle test in that players run repeatedly over a distance of 20m however following each pair of 20m runs both Yo-Yo tests incorporate a short period of active rest. Performance in the Yo-Yo tests is similar to that for the multi-stage fitness test with the level and number of shuttles completed being recorded (Svensson and Drust, 2005). A recent study by Krstrup and co-workers (2003) closely

examined the physiological response, reliability and validity of the Yo-Yo Intermittent Recovery Test. Eleven players were tested twice with a weeks separation between tests in order to determine the test-retest reliability. No differences were found between performance in the first and second Yo-Yo test performed within a week ( $1867 \pm 72$  vs  $1880 \pm 89$  m). Furthermore, a significant but moderate correlation was reported between the Yo-Yo test result and the amount of high intensity running during a soccer match ( $r = 0.71$ ,  $P < 0.05$ ). Other findings from using the Yo-Yo intermittent recovery test have included; ability to show seasonal variability in fitness levels (Krustrup et al., 2003), assess the soccer-specific endurance capacity of male and female players and referees (Krustrup and Bangsbo, 2001; Krustrup et al., 2003; Mohr et al., 2003; Krustrup et al., 2005) differentiate fitness levels between positions (Bangsbo and Michalsik, 2002), detect performance changes as a result of training (Krustrup and Bangsbo, 2001) and to differentiate between top-class and moderate ability soccer players (Mohr et al., 2003).

Following advancements in technology, a recent development of small portable metabolic measurement systems has made it possible to measure oxygen uptake directly during soccer-specific tests (Hoff, 2005). A dribble track course for training soccer players has been described in detail (Hoff et al., 2002) and will be discussed in the following section, however, Kemi and co-workers (2003) have since reported that this dribble track can be a reliable way to examine  $VO_{2 \max}$ , using a portable metabolic system. Interestingly, Chamari et al., (2005) further investigated the use of the Hoff dribble test and reported it to be a valid field based test of endurance performance correlating closely to a tradition  $VO_{2 \max}$  test performed on the treadmill. Furthermore, it appears to be sensitive to detect changes in endurance performance following a period of aerobic training (Chamari et al., 2005).

#### **2.2.3.2 Field based assessment of strength, power and repeated sprint performance**

Accurate assessment of sprint performance can be made relatively simply in the field with the use of portable light cell timing gates. Several authors have examined soccer players' acceleration speeds over various distances using this method (Strudwick et al., 2002; Helegrud et al., 2003; McMillan et al., 2005). Similarly, assessments of strength can be made very simply in the field. Typically, maximal strength is defined as 1-

repetition maximum (1-RM) in a standardised movement, as in the squat exercise (Hoff, 2005).

A further measure of power important to soccer players is jump height. This variable can be measured using a force platform that calculates centre of mass displacement, or a movement registration system that can follow the displacement of a fixed point (Hoff, 2005). Both the Seargent's test and a test mat, using time for calculation of jumping height, have a higher variation than desirable, but are useful for estimates of leg power (Hoff, 2005). Interestingly, Wisloff and co-workers (1998) reported a strong correlation between maximal muscle strength, vertical jump performance and 10-30m sprint times in well-trained male elite soccer players. Furthermore, jump height appears to be an important determine of success in football, Arnason and co-workers (2004) reported a significant relationship between team average jump height (countermovement jump and standing jump) and team success when studying Icelandic elite teams.

Clearly, there are several jumping methods that can be measured. It is well established that individuals will jump higher if countermovement is permitted rather than a squat jump (Bobbert et al., 1996). Bobbert and colleagues (1996) have gone on to suggest a plausible reason for this increase in jump height with the addition of countermovement is because the beneficial effects of the stretch shortening cycle. Harman and co-workers (1990) have reported excellent test-retest reliability in a number of jump test variables, for example, the use of arms, no arms, countermovement and no countermovement. Interestingly, the use of arms in jump performance has been shown to contribute a mean of 10% to take-off velocity (Harman et al., 1990). It is perhaps for these reasons that it is suggested that the design of tests to assess jumping ability should consider the specific jump type used in the sport or interest (Young et al., 1997). Other methods of assessing power within the field have included single leg horizontal hop tests (Ross et al., 2002), medicine ball explosive power tests (Stockbrugger et al., 2001) and single leg vertical jumps (Young et al., 2001).

Several researchers have focused their attention on fitness tests that involve repeated sprint activity (Bangsbo, 1994; Boddington et al., 2001; Wragg et al., 2000). The repeated sprint test proposed by Bangsbo (1994) consists of 7 x 35 m sprints punctuated by recovery periods of 25 s. The test results can be based on a number of

outcomes including quickest time for each effort, mean sprint time and a fatigue index (i.e. the rate of decline in sprint performance). Wragg and co-workers (2000) examined the reliability and validity of this testing protocol and reported high reliability with a coefficient of variation of 1.82% over 6 main trials. However, there was an initial learning effect after the first attempt at the protocol. Boddington and co-workers (2001) examined the reliability of a similar testing protocol that involved six repeated attempts at a multiple 5 m shuttle run. Briefly, six beacons were placed 5 m apart in a straight line to cover a total distance of 25m. Participants ran maximally back and forth between the cones rotating between 10 and 5 m attempting to cover as much distance as possible in each of the six 30 s exercise bouts. Boddington et al., (2001) found the highest reliability when measuring total distance covered during the 5 m multiple shuttle test compared to other physiological data recorded during the test.

## **2.3 Fitness training soccer players**

### **2.3.1 Endurance training interventions within soccer**

#### **2.3.1.1 The importance of aerobic endurance capabilities in soccer**

Several authors have used different ways to demonstrate the importance of aerobic endurance on soccer performance. Studies by Bangsbo (1994) and Smaros (1980) have illustrated a significant correlation between maximal oxygen uptake and distance covered in a game. Furthermore, there appears to be a similar correlation between  $\text{VO}_2_{\text{max}}$  values and the amount of high intensity running in a game, specifically the number of sprints attempted (Smaros, 1980). Two studies have attempted to establish a link between endurance fitness levels and team performance. Apor (1988) reported that the ranking of four teams in the Hungarian top soccer division reflected the ranking between  $\text{VO}_2_{\text{max}}$  of the teams. A similar study reported a higher squad average  $\text{VO}_2_{\text{max}}$  level in a team finishing at the top of the Norwegian elite league (Rosenberg;  $\text{VO}_2_{\text{max}}$  67.6  $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) compared to one much lower placed (Strindheim; 59.9  $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) (Wisloff et al., 1998). However, the findings of these latter studies are limited due to the small number of teams compared.

Several studies have highlighted the fact that the amount of sprinting, high intensity running and distance covered are lower in the second half than in the first of a game (Reilly and Thomas, 1976; Bangsbo, 1994; Bangsbo et al., 1991; Mohr et al., 2003a, b). A recent study conducted by Mohr and co-workers (2003a) that analysed physical performance data during match play of top class players found that the amount of high intensity running in the 5 min period immediately after the most intense 5 min interval recorded during the game was observed to be less than the average of the entire game. It is not within the discussion of this chapter to examine the potential causes of fatigue (see review; Mohr et al., 2005). However, it would seem logical that improved endurance performance could off-set or decrease the recovery time following this fatiguing exercise, due to the important role that aerobic metabolism plays in recovery from intense periods of intermittent exercise (See review; Tomlin and Wenger, 2001) and substrate metabolism during prolonged exercise.

### **2.3.1.2 Limitations of maximal oxygen uptake**

The four main physiological factors that can limit  $\text{VO}_2 \text{ max}$  are; 1) the pulmonary diffusing capacity, 2) maximal cardiac output ( $\text{Qmax}$ ), 3)  $\text{O}_2$  carrying capacity of the blood and 4) skeletal muscle utilisation of  $\text{O}_2$ . The interplay between these and the exact bottleneck/limiting factor has been widely debated (See, Bassett and Howley, 2000). It is not within the boundaries of this chapter to highlight the arguments and counterarguments for the precise limitations of maximal oxygen uptake, however, the weight of evidence continues to illustrate that even in well trained skeletal muscle the highest recorded metabolic rates can be increased by increasing  $\text{O}_2$  supply (Richardson, 2000). It is the  $\text{O}_2$  supply i.e. the heart's ability to pump blood that is continually cited as a limiting factor (Richardson, 2000; Wagner, 2000) and more specifically the stroke volume (SV) of the heart (Hoff et al., 2005).

Recent investigations have found that SV does not appear to plateau as often suggested and indeed, its volume will continue to increase with an increasing workload (Zhou et al., 2001). In this study Zhou and co-workers (2001) compared SV responses to a graded treadmill test between elite distance runners, university standard runners and untrained. The researchers found that SV responded differently to exercise as individuals went from moderate to maximal. The university level and untrained group

remained unchanged whilst the SV continued to rise sharply in the elite group. Zhou et al., (2001) suggest that a possible mechanism for this SV rise could be an increased left ventricle chamber size. However, it remains unclear if this continued rise in SV is an adaptation to training, a consequence of genetics or a combination of both influences (Zhou et al., 2001).

The increasing stroke volume up to the point of  $\text{VO}_2 \text{ max}$  in trained athletes has been the background for using high intensity training interventions in recent endurance interventions within elite soccer (Hoff et al., 2005).

### **2.3.1.3 Training interventions for aerobic endurance within soccer**

Recently, a number of studies have examined the effects of short-term endurance interventions with soccer teams. Helgerud and co-workers (2001) trained a group of male elite youth players twice a week in addition to their normal training for a period of 8 weeks. The squad were divided into two groups, one completed the additional training the other acted as a control. The exercise protocol consisted of high intensity aerobic interval training. Subjects performed 4 bouts of 4 min running at an intensity of 90-95% max HR interspersed with 3 min low intensity recovery between each interval. An 11% overall improvement in  $\text{VO}_2 \text{ max}$  was reported and this manifested a 20% increase in total distance covered during match-play, along with 23% increase of involvements with a ball and a 100% increase in the number of sprints performed by each player (Helgerud et al., 2001). Later, Hoff and colleagues (2002) examined whether dribbling a ball around a prescribed circuit and small-sided football practice games could be used to stimulate the same training intensities achieved in the previous study using treadmill running. Six elite male soccer players were used in this study and reported results demonstrated that the dribble track and small-sided games elicited values of 93.5% and 91.3% of maximum heart rate, respectively. Hoff et al., (2002) concluded that soccer specific exercise using a ball dribbling circuit or small group play might be performed as an aerobic interval training method. This has since been supported by further research (Sassi et al., 2003). An additional conclusion drawn from Hoff et al., (2002) study was that heart rate monitoring is a valid measure of actual exercise intensity in these types of training modes (Hoff et al., 2002). This conclusion was made following the comparison of correlation coefficients for  $\text{VO}_2$  and heart rate frequency during sub-

maximal treadmill running, the dribble circuit and the small-sided soccer play. This later finding has since been further supported by a study validating the use of heart rate as an indicator of aerobic demand during soccer activities in amateur soccer players (Esposito et al., 2004).

A further study from Helgrud and colleagues (2003) reported improvements in  $VO_2 \text{ max}$  of 8.1% following 8 weeks of the same interval treadmill running activities with elite senior male soccer players. However, it should be noted that players in this study completed additional strength training (discussed in next section). Furthermore, caution should be taken when interpreting these results because the training was carried out throughout the pre-season period (a time one would expect improvements in endurance capacity) and without the use of a control group. McMillan and co-workers (2005) recorded a 9% improvement in  $VO_2 \text{ max}$  with values rising from 63.4 to 69.8  $\text{ml.kg}^{-1}.\text{min}^{-1}$  following 10 weeks of specific soccer interval training (Hoff et al., 2002) 2 times per week with elite youth players. A similar study conducted by Chamari et al., (2005) trained 18 male junior players (Age category - Under 15s) over 8 weeks using the same protocol as McMillan et al., (2005). Chamari et al., (2005) reported  $VO_2 \text{ max}$  improvements of 12%.

Unfortunately, none of these training studies have attempted to establish the exact adaptations causing these apparent high rises in maximal oxygen uptake. However, Hoff and Helgerud (2004) suggest that continuous interval training for work period of 3-8 min at intensities of 90-95% HRmax should increase  $VO_2 \text{ max}$  by increasing maximal cardiac output via improvements in SV.

### **2.3.2 Strength training interventions within soccer**

Strength is defined as the maximal torque a muscle or group of muscles can generate (Komi, 1992), whereas power is the product of force and velocity, and refers to the ability of the neuromuscular system to produce the greatest possible impulse in a given time period (Schmidtbleicher, 1992). Both of these fitness components are extremely important with soccer and this section highlights some basic training methods/interventions that have been documented within the area. However, it is not



within the scope of this literature review to critique the complex morphological adaptations to strength training.

Few controlled studies have examined the effects of strength training on soccer performance. Hoff and Helgerud (2002) examined the effects of maximal strength training on running economy and aerobic endurance performance. Twenty-four elite male soccer players from 3 different teams formed the training group. The training group completed 4 x 5 reps of >85% 1RM half squats with emphasis on maximal mobilisation of force in the concentric action i.e. upward phase, 3 times a week for 8 weeks. The control group continued with normal soccer training. The training group increased 1 RM levels by 33.7% from 161.3kg to 215.6kg in half squats with no change in body mass. Further improvements were reported in rate of force development and running economy, however  $VO_2 \text{ max}$  levels remained unchanged. Hoff and Helgerud (2002) suggest that the adaptations were likely to be largely neural and this was supported by no change in body mass.

A similar study conducted by Helgerud and Hoff (2002) trained elite players twice a week with 4 x 4 half squats at an overload close to 4 RM. However, the players also performed high intensity aerobic training within this study. Half squat 1 RM scores increased from  $115.7 \pm 23.1$  to  $176 \pm 18.2$ kg. Furthermore, improvements were recorded in 10m sprint performance and vertical jump scores. A control group was not used during this study and the training intervention was conducted throughout pre-season therefore caution needs to be taken when interpreting these results.

A recent study examined the effects of 11 weeks of soccer training combined with a progressive, explosive-type strength training (including full squat, power clean, vertical jumps and sprinting exercises) with young players (Gorostiaga et al., 2004). The training resulted in significant improvements in the low load portions of the load-vertical jumping height curve (Gorostiaga et al., 2004). Gorostiaga and co-workers (2004) concluded that young, trained soccer players with low initial strength levels can increase explosive strength by adding a low-frequency, low-intensity, explosive-type strength training program.

### 2.3.3 Concurrent training studies

The physical demands of match activities in intermittent team sports like soccer result in the need for the physical conditioning of players to develop a number of fitness components. Players need to be aerobically fit as well as being strong and powerful. This can often result in individuals performing both endurance and strength training programmes concurrently. The effect of these forms of concurrent training programs has been examined in recent years. Some studies have reported that the strength gains made during concurrent strength and endurance training are inhibited compared to strength training *per se* (Hickson, 1980; Dudley et al., 1985; Hunter et al., 1987; Kraemer et al., 1995). This is often referred to as the 'interference effect' (Docherty et al., 2000). Other studies have found similar strength gains in the concurrent training group compared to individuals who completed the same strength training alone (Sale et al., 1990; McCarthy et al., 1995). It has also been reported that endurance performance may be reduced following training when combined with strength training compared to endurance training *pre se* (Nelson et al., 1990 cited in Leveritt et al, 1999), although other studies did not find this to be the case (Hickson et al., 1980; Kraemer et al., 1995). The variation among these findings is caused by the difficulty in making comparisons between studies. Researchers differ in a number of key training principles they choose to adopt and the initial fitness levels of the subjects recruited.

#### 2.3.3.1 No interference in strength and endurance performance

Sale and co-workers (1990) conducted a  $3 \text{ d}\cdot\text{wk}^{-1}$  training study with a duration of 22 weeks. Two groups consisting of 4 males and 4 females in each were used for this intervention. Group A trained both lower limbs for strength (S) and one for additional endurance (SE) and group B trained both legs for endurance (E) and one limb for additional strength (ES). Similar strength improvements were recorded for Group A (S vs. SE) following training. One repetition maximum (1 RM) for single leg press improved by 30% in the S compared to 20% in the SE training leg. No interaction was seen between training modes. The endurance improvements in Group B (E vs. ES) were similar following training. Maximal oxygen uptake improved by 8% in both groups. Sale and colleagues (1990) concluded that concurrent strength and endurance training did not interfere with strength or endurance development in comparison to strength and

endurance training alone. The authors of this study suggest that no antagonism occurred during concurrent training because of the hybrid nature of the S and E training involved and the moderate total volume of training (Sale et al., 1990).

Similarly, McCarthy and co-workers (1995) found that 3 d·wk<sup>-1</sup> for 10 weeks of combined strength and endurance training induced substantial concurrent and compatible increases in VO<sub>2 max</sub> and strength performance. In this study the strength group (n=10) (S) conducted 8 classical strength exercises consisting of 3 sets of 5-7 reps (fatiguing between 5-7 reps), the endurance group (n=10) (E) cycled for 50 min at 70% heart rate reserve and the combined group (n=10) (SE) performed the S and E programs. The improvements in isotonic strength were 23% and 22% in the S and SE groups, respectively. The improvements in VO<sub>2 max</sub> were 18% and 16% in the E and the SE groups, respectively. It should be noted that subjects were sedentary prior to the inception of this training study, which helps to explain the relatively large improvements in strength and endurance.

#### **2.3.3.2 Interference in strength performance**

Interestingly, there have been several studies that have found smaller increases in strength performance following concurrent training regimes compared to the same strength training alone. Hickson et al. (1980) conducted 5 d·wk<sup>-1</sup> for 10 weeks training of combined heavy weight training (Multiple sets of 5 repetitions at > 80% 1 RM) with high intensity cycling and running. These authors found that the strength gains were greater in the S group compared to the CC. However, the E group improved VO<sub>2 max</sub> to a similar extent as the CC group. Another study conducted 3 d·wk<sup>-1</sup> of current strength training (2 sets of 30-second isokinetic knee extensions 4.19 rad<sup>-1</sup>) and cycle activity (5 x 5 min bouts of 40 – 100% VO<sub>2 max</sub>) for 7 weeks (Dudley et al., 1985). Although this intervention was for a shorter duration and the exact intensities and protocols of the training varied slightly from the previous authors the results followed the similar trend. The S group improved their peak torque up to and including the training speed, however the CC group increased their peak torque only at slower velocities. Interestingly, the E group showed similar improvements as the CC group.

### **2.3.3.3 Possible mechanisms causing interference in strength development**

Clearly, several studies have proposed some form of negative effect on strength development when performing concurrent strength and endurance training. However, very few investigators have carefully examined the cause of these interference effects. Those that have propose several different explanations to the phenomenon. In a recent review, Leveritt (1999) suggests that the proposed mechanisms associated with this inhibited strength development during concurrent training may be explained in one of the following ways; over-training, situation of conflict between strength and endurance adaptations within the skeletal muscle or a residual fatigue as a result of the endurance training impairing the quality of the strength training.

Very few studies have examined the possible effects of over-training during concurrent strength and endurance training programmes. Leveritt and co-workers (1999) explained that the experimental design of concurrent training studies invariably results in one group (strength and endurance) conducting double the volume of work as the other two groups (strength alone and endurance alone). Indeed, Dudley and co-workers (1987) have suggested that subjects involved in concurrent training may become over-trained in comparison to others who are performing strength and endurance training alone. However, Dudley and colleagues (1987) went on to suggest that if individuals in the concurrent training group were to become over-trained then a decrement in performance should be seen for both the strength and endurance performance.

Leveritt and co-workers (1999) have suggested that the case for over-training is unlikely to be the reason for performance decrements in certain studies. For example, in a study conducted by Dudley and colleagues (1985), the volume of endurance training was low ( $5 \times 5 \text{ min on } 3 \text{ d}\cdot\text{wk}^{-1}$ ) as was the volume of strength training ( $2 \times 30\text{sec sessions on } 3 \text{ d}\cdot\text{wk}^{-1}$ ). It was suggested that this volume of training is insufficient to induce an over-training effect.

### **2.3.3.4 Adaptations within the skeletal muscle during concurrent training**

Bell and co-workers (2000) examined the effects of concurrent strength and endurance training on skeletal muscle properties and hormone concentrations. Four groups were

used consisting of an endurance group (E), strength group (S), concurrent strength and endurance group (CC) and a control group (C). The S group completed 8 exercises of 2-6 sets and 4 – 12 repetitions (progressive overload) and the E group completed either a 30 – 42 min cycle at ventilation threshold or a high intensity interval session for 3 d·wk<sup>-1</sup> 12 weeks. The CC group combined both modes of training and completed these for 6 d·wk<sup>-1</sup> 12 weeks. Following training the strength group recorded an increase in type I and II fibre size whereas the CC group had only experienced an increase in type II. There appeared to be a higher concentration of myofibrillar ATPase in the type II fibres of the S group compared to the CC. Interestingly, the decline in myofibrillar ATPase seen in E group following training was not seen in the CC group. Bell and colleagues (2000) suggest that the concurrent training was able to counteract the possible decrease in this enzyme activity as a result of endurance training. The CC group increased their capillary to fibre ratio, however this was not seen in the S or the E groups. Finally, the urinary cortisol concentration at rest was higher in the women following CC training. This marker was used to examine any increase in catabolic state that is associated with protein degradation.

Kraemer and colleagues (1995) made a similar finding to Bell and co-workers (2002) in that, there appears to be an antagonist effect on muscle fibre cellular adaptations. In this study, type I fibres of subjects in the CC group did not experience hypertrophy unlike in the whole body strength group alone nor did they decrease in size similar to the response in the E group. These findings suggest that muscle fibre area adaptations in response to simultaneous training differ from single training mode adaptations. Interestingly, in this study Kraemer and colleagues (1995) observed a large increase in the exercise-induced and total cortisol (as described by the area under the response curve) responses in the CC group following training. This preceded a large increase in the exercise-induced and total testosterone (as described by the area under the response curve) at the end of training. Kraemer and co-workers (1995) suggests these large hormonal alterations only seen in the concurrent training group may suggest some form of over-training response.

However, a study conducted by McCarthy and co-workers (2002) found that following 10 weeks of high-intensity strength training (S) and cycle endurance training (E) 3 d·wk<sup>-1</sup>. The concurrent training group (CC) had very similar muscular adaptations at a

macro and micro level compared to the S group. Improvements of 12% and 14% in thigh extensor area was seen following training in the S and CC groups, respectively. Furthermore, very similar improvements occurred in the thigh flexor and adductor areas measured using the same CT scan method in all cases. Interestingly, at a 'micro level' McCarthy and colleagues (2002) also failed to find any differences in the improvement gains in muscle fibre distribution between the S and CC group following training. Finally, there were no changes seen in any of the groups in the level of neural activation measured for a given level of torque.

## **2.4 Carbohydrate intake during recovery from exercise**

An important goal of the athlete's everyday diet is to provide the muscle with substrates to fuel the training programme that will achieve optimal adaptations and performance enhancements (Burke et al., 2004). It is well established that the availability of carbohydrate as a substrate for the muscles and central nervous system can be a limiting factor to performance during heavy periods of training or when performing a prolonged session. Clearly, carbohydrate stores in the body are limited (See review; Coyle, 1995) and therefore careful consideration needs to be taken when considering the amount, timing and type of carbohydrate consumed prior, during and post exercise. The elite performer can often have very limited time to recover between training sessions and competition. Therefore, the need for an optimal recovery diet is paramount; the focus of the next section will be on carbohydrate recovery diets.

### **2.4.1 The amount of carbohydrate required**

Following the completion of prolonged exercise the metabolic priority is muscle glycogen repletion. This drive to resynthesise muscle glycogen appears to occur immediately post exercise even without the presence of food (Fournier et al., 2004) although the most important dietary factor affecting muscle glycogen storage is the amount of carbohydrate consumed (Burke et al., 2004).

Several studies have examined the effect of muscle glycogen repletion over 24 h following prolonged exercise. Careful examination of the data suggests that a positive

relationship exists between the amount of carbohydrate consumed post exercise and muscle glycogen storage. However, Burke et al., (2004) illustrate that there appears to be a storage capacity or threshold near the 8 to 10 g.kg<sup>-1</sup> BM. Interestingly, both Burke et al., (1995) and Costill et al., (1981) made direct comparisons between different volumes of carbohydrate consumed in the 24 h recovery period in trained individuals and the effect on glycogen storage. The findings from these studies support the notion that increasing carbohydrate intake in the recovery period will increase glycogen storage.

The figure of 7-10 g.kg<sup>-1</sup> BM has since been used in guideline recommendations for daily carbohydrate intake during periods of heavy training and or competition. However, there is a suggestion that these figures may underestimate requirements in instances when active rather than passive recovery is employed (Coyle et al., 1991). Furthermore, in activities that involve a large amount of eccentric muscle actions it could be advantageous to increase the carbohydrate requirements (Asp et al., 1998 and Costill et al., 1990). In this latter study, Costill and co-workers (1990) found that muscle glycogen resynthesis was impaired in the leg that completed eccentric exercise (10 sets of 10 eccentric contractions of the knee extensor muscles) compared to the control leg. Furthermore, subjects receiving 8.5 g.kg<sup>-1</sup> BM stored significantly more glycogen than those who were fed 4.3 g.kg<sup>-1</sup> BM (Costill et al., 1990).

It appears that not only the total quantity of carbohydrate during the recovery period is important but also the frequency of feeding intervals. A recent study by van Loon and colleagues (2000) reported that increasing the rate of carbohydrate ingestion from 0.8 to 1.2 g.kg<sup>-1</sup> BM resulted in higher muscle glycogen resynthesis rates. This finding was in contrast to the earlier studies of Blom et al. (1987) and Ivy et al. (1988). However, the major difference in van Loon's study design was that participants were fed every 30 min unlike the earlier studies that employed 2 h intervals between feeding. The net effect of the differences in frequency of feeding could have meant that the 2 h intervals were too long to sustain elevated blood glucose and insulin that would have promoted maximum rates of muscle glycogen resynthesis (Jentjens and Jeukendrup, 2003). Finally, a study by Jentjens and co-workers (2001) failed to find any increases in muscle glycogen storage from 1.2 g.kg<sup>-1</sup> BM when additional protein was consumed suggesting an upper limit exists to carbohydrate utilisation in the initial recovery phase.

## 2.4.2 The timing of carbohydrate

Muscle glycogen synthesis occurs in a biphasic manner following prolonged strenuous exercise (Jentjens and Jeukendrup, 2003). The initial rapid phase of glycogen synthesis can occur without the presence of insulin and lasts approximately 30-60 min. This phase is referred to as the insulin-independent phase and it appears that both low levels of muscle glycogen (Price et al., 1994) and the presence of exogenous CHO (Ivy et al., 1988) are needed for this phase to occur. The second phase of muscle glycogen synthesis can last for at least 2 hours in the presence of CHO availability and high insulin levels. This phase is slower and is referred to as the insulin-dependent phase (Jentjens and Jeukendrup, 2003).

It appears that the highest rate of muscle glycogen storage occurs during the first two hours after exercise (Ivy et al., 1988). Feeding carbohydrate during this initial period can capitalise on the increased availability of GLUT-4 transporter proteins and a heightened activation of the enzyme glycogen synthase (Wojtaszewski et al., 2002). Indeed, Ivy and co-workers (1988) who reported a rate of  $7.7 \text{ mmol.kg}^{-1}.\text{wwh}^{-1}$  during the first 2 h of recovery compared to a slower rate of  $4.3 \text{ mmol.kg}^{-1}.\text{wwh}^{-1}$  in the following 2 h. This highlights the importance of feeding immediately post-exercise when recovery times are very short. Interestingly, a further finding from this study was that muscle glycogen resynthesis was 45% lower if feeding was delayed by 2 h compared to immediately feeding subjects (Ivy et al., 1988). However, when the athlete has the luxury of longer recovery times it appears that the importance of initial feeding at the termination of exercise is of less importance. A study by Parkin and colleagues (1997) found that delaying feeding during the initial 2 h post exercise had no effect on muscle glycogen restoration when measured at 8 and 24 h post event.

The question of feeding schedules during a 24 h recovery period has been the topic of investigation. Costill and co-workers (1981) reported no differences in muscle glycogen restoration in subjects that were provided with equal amounts of carbohydrates in either 2 large meals or 7 smaller meals. These findings were supported by a further study that found no differences between administering 4 large meals or 16 one-hourly snacks when comparing muscle glycogen restoration over a 24 h period (Burke et al., 1996). However, when examining short-term recovery (4-6 h) there is a body of evidence to



support the feeding of carbohydrates in 15-30 min intervals in order to maximise glycogen synthesis, presumably as a consequence of elevated blood glucose and insulin levels (Doyle et al., 1993; van Hall et al., 2000; van Loon et al., 2000; Jentjens et al., 2001). Caution, should be taken with the findings from these latter studies because results are compared to other studies rather than employing a control group that consumed the same amount of carbohydrates at less frequent intervals.

### **2.4.3 The type of carbohydrate**

#### **2.4.3.1 The glycaemic index concept**

The term 'glycaemic index' (GI) has been used for a number of years within clinical nutrition as a classification system for carbohydrates. In 1981, Jenkins and co-workers published the glycaemic index concept to provide a numeric classification of carbohydrate foods according to their glycaemic responses (Jenkins et al., 1981). The term refers to the relative degree to which the concentration of glucose in the blood raises after the consumption of a food i.e. 'the glycaemic response' (Burke et al., 1999).

In order to test the GI of a carbohydrate it requires ingesting 50g of the test food and measuring the blood glucose response over 2 h. After the blood glucose concentration over the two hours is graphically represented – with glucose concentration on the vertical axis and time on the horizontal axis – the incremental area under the blood glucose curve (IAUC) is measured for the test food and compared to consumption of 50g of glucose as the reference/standard food (GI = 100) (Rankin, 1997). The glycaemic index is given as a percentage, i.e. the percentage area under the blood glucose curve for the test food compared to that for glucose (Burke et al., 1999).

Equation for calculating the GI of a food:

$$\text{GI} = (\text{Blood glucose IAUC of test food} / \text{Blood glucose IAUC of standard food}) \times 100$$

Following the calculation of IAUC outlined by Wolever and colleagues (1986) and the assignment of a GI value, foods can be classified accordingly as low (GI<55), moderate (GI 55-75) or high (GI>70) (Foster-Powell et al., 2002).

The Food and Agricultural Organisation and World Health Organisation Expert Consultation (FAO/WHO, 1998) have introduced a standardised protocol for the measurement of GI of foods in an attempt to minimise variability within measurements. The standardised procedures include; using at least six subjects when testing the foods, ensuring subjects are tested in the morning following a fast of 10-12 hours, analysing blood glucose samples from whole capillary blood rather than plasma. This standardised method has been shown to be reliable within various international laboratories with a 95% confidence interval of the GI value obtained in 10 subjects being  $\pm 15$  (Wolever et al., 2003a).

#### **2.4.3.2 Influences of the GI response to feeding**

Several factors can affect the GI response to a test food. As previously mentioned the method of blood sampling should be standardised. Wolever and Bolognesi (1996a) compared venous and capillary blood and found that glycaemic responses measured in capillary samples had less random variation. It is important that the 50g of carbohydrate used when testing the glycaemic response should not include carbohydrates that cannot be absorbed in the small intestine (Wolever, 2003b). Clearly, comparisons between different carbohydrate glycaemic responses cannot be made if a proportion of one of the 50g of test foods contains fibre, resistant starch and other unabsorbed carbohydrates. A further potential threat to the reliability of the GI results recorded for individual foods is that of the reference food that is given the arbitrary value GI value of 100. It was mentioned earlier that glucose is commonly used however white bread has been used as a reference food. The problem is glucose has a glycaemic response 40% greater than that of bread; therefore the GI values obtained from each reference food will differ by a factor of 1.4 (Wolever, 2003b). Other major influences on the GI responses to feeding include between-subject and within-subject variation, time of day, composition of the carbohydrate absorbed and the feeding pattern of the test meal (Wolever, 2006).

Different subjects vary in their glycaemic responses over a wide range (Wolever, 2003b). However, Wolever and co-workers (1990; 1992) have shown that when the glycaemic response of a food is expressed relative to that of the reference food taken by the same subject, the variability between subjects is reduced to the extent that is no

longer becomes statically significant. Glycaemic responses of the same subject after consuming standard test meals under standardised conditions vary from day to day (Wolever, 2003b).

A study conducted by Wolever and Bolognesi (1996b) reported a potential time of day effect on glycaemic responses to a test meal. They compared the glycaemic responses of two breakfast cereals in healthy subjects either in the morning after a 12 hour fast or at midday 4 hours after a standard breakfast. Absolute glycaemic responses (AUC) at lunchtime were significantly less than those at breakfast despite consuming exactly the same foods.

The digestible carbohydrate in starch is absorbed entirely as glucose (Wolever, 2003b). However, only approximately half the carbohydrate in sucrose, fruits and dairy products is as glucose, the other half being fructose or galactose, neither of which raise plasma glucose or insulin appreciably (Lee and Wolever, 1998). Fructose produces a lower glycaemic response than most other carbohydrates (Wolever, 2003b). A further study has examined the glycaemic response rate of feeding 50g of carbohydrate over different time periods (Jenkins et al., 1990). Participants were fed 50g of carbohydrate on two occasions; once they were encouraged to consume the carbohydrate beverage immediately (5-10min) and the other time subjects were asked to consume the drink slowly by sipping over a period of 3.5 h. Jenkins et al., (1990) findings included a reduced rise in insulin during the slower consumption trial and an increased rate of glucose disappearance following a glucose tolerance test.

Interestingly, several studies have reported that the exaggerated glycaemic and insulinaemic response to feeding immediately post exercise is independent of the glycaemic index of the carbohydrate consumed (Burke et al., 1993; Rose et al., 2001; Stevenson et al., 2005). Rose et al., (2001) suggest that this is the result of greater gut glucose output and greater hepatic glucose escape.

#### **2.4.3.3 The effect of different GI carbohydrates in recovery from exercise**

Very few studies have examined the effect of different GI carbohydrates in recovery from exercise. However, since glycogen storage is influenced by insulin and a rapid

supply of glucose substrate, it would seem logical that carbohydrate sources with a moderate to high glycaemic index (GI) would enhance post-exercise refuelling (Burke et al., 2004).

A study by Burke and co-workers (1993) examined the influence of the glycaemic index of isocaloric carbohydrate meals during a 24 h recovery period on muscle glycogen following prolonged continuous cycling. All subjects consumed  $10\text{g}\cdot\text{kg}^{-1}\text{BM}$  of either HGI or LGI CHO at controlled intervals during the recovery period. When the effects of the first post-exercise meal were excluded, the totals of the incremental glucose and insulin areas under the curve were greater following each meal with the HGI compared to the LGI CHO (Burke et al., 1993). The HGI recovery diet resulted in a higher concentration of muscle glycogen ( $106 \pm 11.7$  mmol/kg wet wt vs.  $71.5 \pm 6.5$  mmol/kg wet wt) however no subsequent performance indicators were examined.

A subsequent study by Stevenson et al., (2005) examined the influence of the glycaemic index of isocaloric CHO meals in the 24 h period following prolonged continuous running activity. In this study, subjects were required to consume  $8\text{g}\cdot\text{kg}^{-1}\text{BM}$  of either HGI or LGI CHO in controlled quantities and intervals. This study did not measure muscle glycogen however performance was assessed on the subsequent day following the recovery diets. Interestingly, endurance running capacity was improved following the LGI CHO recovery diet. Furthermore, fat oxidation rates were elevated during the second run during the LGI trials. It is possible that the increased fat utilisation resulted in a glycogen sparing effect and hence an improved performance. However, it is equally plausible that the elevated fat oxidation was a consequence of a reduced muscle glycogen availability thus making an explanation for improved running capacity harder to examine.

## **2.5 Exercise induced muscle damage**

Activity patterns in soccer are intermittent in nature and require a significant number of movements (including acceleration, deceleration, twisting, turning and jumping) that involve eccentric muscle actions. A negative consequence of these types of muscle contractions is that they are liable to induce an element of muscle soreness. Clearly, the

degree of soreness caused as a result of these eccentric muscle actions is dependant on a number of factors including how accustomed the muscle is with this activity, the intensity and duration of the muscle damaging exercise. It appears that novice participants in exercise are the most prone to severe muscle soreness although this sensation is certainly not exclusive to this group. Interestingly, any repeat of this type of exercise in the days following this period of soreness tends to result in a blunted subsequent response, suggesting some amount of muscle adaptation occurs very quickly. Therefore, this period of post exercise muscle soreness may represent a very important time during which some form of physiological adaptive process is taking place. Several interventions have been examined in attempt to find ways to lessen this initial muscle soreness and potentially reduce the amount of time taken to recover from a bout of exercise. Clearly, an intervention that may reduce the inhibitory effects of muscle damaging exercise and potentially reduce recovery times could be very beneficial to soccer players faced with compressed fixture lists and periods of intense training.

### **2.5.1 Aetiology of exercise induced muscle damage**

It is not within the scope of this literature review to critique all the studies available to gain an understanding of the precise mechanisms responsible for causing exercise induced muscle damage (EIMD) but rather outline the key factors to what is a very complex physiological process.

There is little doubt that a strong link exists between eccentric muscle contractions and delayed onset of muscle soreness (DOMS). Some studies have found this link from comparing eccentric and concentric work in the same subjects (Armstrong et al., 1983; Schwane et al., 1983), others have made subjects use one limb for concentric work and compared this to the other limb which has performed eccentric work (Newham et al., 1983a; Newham et al., 1983b; Newham et al., 1983c). It appears the degree of soreness produced during eccentric exercise is related to the magnitude of torque produced and therefore maximal soreness being generated by high-intensity, high velocity eccentric activity (Cleak and Eston, 1992).

A potential cause of DOMS following eccentric exercise is the result of the changes forced on the muscle structure during the contraction. Eccentric contractions are characterised by the muscle being forced to lengthen as an external force overpowers the internally produced torque (Lakomy, 1996). It is believed that following this action many of the overstretched sarcomeres will relax and return to previous state however some of the weaker sarcomeres can become permanently overstretched (Talbot & Morgan, 1996). The destructive results of this are commonly seen in the myofibril with several investigations citing the Z disk as being the most disturbed following eccentric exercise (Friden et al., 1981; Armstrong et al., 1983). This disruption to the Z disk area within the sarcomeres is often referred to as 'Z disk streaming or smearing' and is a common characteristic to EIMD (Ebbeling and Clarkson, 1989). It appears that type II fibres could be more susceptible to this form of contractile injury following eccentric exercise (Friden et al., 1983b).

It is very possible that if these morphological disruptions within the sarcomeres are great enough in severity or number that this may impact to some degree on muscle function in subsequent days. Interestingly, it appears that the muscle damage per se may not be responsible for DOMS and any deterioration in muscle function but rather it causes a metabolic reaction that brings about an inflammatory response and this could well contribute to the discomfort and dysfunction (Armstrong, 1984; Armstrong, 1990; Smith, 1991;).

At the onset of exercise it is well understood that a number of dynamic changes occur to our metabolic state. The magnitude of these changes and subsequent potential stress to our body are dependant on the nature of the activity. Metabolic stresses that occur during high intensity strenuous exercise like soccer include the accumulation of waste products (Armstrong, 1984), ATP turnover rate (Ebbeling and Clarkson, 1989), ion imbalances (Pyne, 1994; Allen, 2001), ischemia and hypoxia (Ebbeling and Clarkson, 1989; Pyne, 1994). As previously mentioned the initial mechanical injury may evoke several metabolic stresses further contributing to muscle damage and soreness (Armstrong, 1984). These stresses can potentially lead to a disruption in cellular homeostasis and cause an imbalance in calcium ( $\text{Ca}^{2+}$ ) concentrations. Clearly, any disruption to  $\text{Ca}^{2+}$  concentrations can be harmful due to the integral role it has in excitation-contraction coupling, failure of  $\text{Ca}^{2+}$  pumps to maintain the concentration

gradient through structural damage or inhibited ATP resynthesis may lead to a loss of  $\text{Ca}^{2+}$  homeostasis (Jackson et al., 1984; Armstrong et al., 1991). It is plausible that this disruption to  $\text{Ca}^{2+}$  homeostasis could be linked to the effect of the exercise induced damage to the sarcoplasmic reticulum (Byrd, 1992). This causes a rapid influx of  $\text{Ca}^{2+}$  from extracellular compartment into the intracellular compartment due to large concentration gradient and this activates a number of compounds active in muscle autolysis (Byrd, 1992), including calcium-dependent proteolytic and phospholipid degrading enzymes that lead directly to tissue damage (Pyne, 1994). These calcium-mediated changes have been referred to as the ' $\text{Ca}^{2+}$  overload phase' (Armstrong et al., 1991).

A potential consequence of this progressive deterioration of the sarcolemma during the post-exercise phase is that the diffusion of intracellular components into the interstitium and plasma attract monocytes that convert into macrophages (Armstrong, 1984). A further result of this  $\text{Ca}^{2+}$  overload phase is the activation of the respiratory burst in phagocytic cells (Rossi, 1986), this then initiates an inflammatory response causing secondary muscle damage that may, in part, contribute to DOMS (Smith, 1991).

It is not within the scope of this literature review to examine the extent to which these inflammatory processes have in the aetiology of EIMD. However, further evidence can be gained from careful consideration of studies examining the effects of non-steroidal anti-inflammatory drugs (NSAIDs) on muscle soreness following exercise.

### **2.5.2 Indices of muscle damage**

Any sedentary individual that decides to embark on a new exercise regime is only too aware of the sensation of DOMS and for the layperson this can be sufficient evidence that some degree of muscle damage has occurred. However, there are a number of other more objective measures used in the scientific community to determine the extent of this EIMD.

Direct histological evidence obtained from the examination of muscle biopsies has long been considered to be one of the most pertinent assessments of muscle damage although in recent years improvements in magnetic resonance imaging have created an non-invasive

approach to this assessment. However, both these methods have practical limitations for the investigator. Paradoxically, the process of obtaining a muscle biopsy in itself can cause further muscle damage to the sample about to be examined (Malm et al., 2000). Furthermore, it is unlikely that any particular muscle sample will be representative of 'global muscular damage' because the muscle damage is unlikely to be evenly distributed across the muscle. The expense and accessibility of magnetic resonance imaging for the assessment of muscle damage limits its use within this research field.

Two very common indirect measures of EIMD include measurement of certain myofibrillar proteins that leak from the muscle following structural disturbances and the post exercise assessment of muscular function. Both of these methods have been extensively used and despite some limitations appear to be consistent indicators of the extent of muscle damage that has occurred.

#### **2.5.2.1 Myofibrillar protein leakage**

Two myofibrillar proteins commonly used as indirect evidence for EIMD are creatine kinase (CK) and myoglobin (Mb). Both of these markers are exclusive to muscle and therefore only have an increased presence in the systemic circulation if the muscle cell membranes are damaged (Ebbeling and Clarkson 1989).

The time course of the efflux of myofibrillar proteins appearing in the systemic circulation is dependant on a number of factors. The nature of the exercise causing the damage will influence the rate of and overall rise in CK activity and Mb concentrations in the systemic circulation. It appears that prolonged exercise (Armstrong, 1986; Thompson et al., 1999) and exercise that is predominately eccentric in nature (Koller et al., 1998; Sorichter et al., 2001b) cause large alterations to the activity and concentrations of CK and Mb, respectively. Myoglobin is a smaller protein than CK and this explains why the time cause of Mb to peak in the systemic circulation is considerably shorter than that of CK (Byrnes et al., 1985). Mb tends to peak in the hours following muscle damaging exercise whilst CK is likely to peak in subsequent days.

Interestingly, the time-cause for peak levels of these myofibrillar proteins appearing in the systemic circulation does not consistently coincide with the sensation of DOMS



and therefore it is unlikely that either CK or Mb can be held responsible for this post exercise discomfort (Cleak and Eston, 1992).

One major potential drawback of using CK as a reliable marker of EIMD is that there are large inter-individual differences (Ebbeling and Clarkson, 1989). This effect has been referred to as 'responders' and 'non-responders' (Clarkson and Ebbeling, 1988). Suggested reasons for this phenomenon include training status, aging, possible CK inhibitors in certain individuals, and muscular disorders (Noakes, 1987; Kagen & Aram, 1987; Clarkson and Ebbeling, 1988).

Several studies have reported significant rises in CK and Mb in the hours and days following the LIST (Thompson et al., 1999; Bailey et al., 2001; 2003). Thompson and co-workers (1999) reported elevated activity levels of CK in the two days following this prolonged demanding intermittent protocol of free running. Similarly, Bailey et al., (2001) reported rises in Mb concentration in the hours following exercises and elevated activity levels of CK in the days following this intermittent exercise protocol designed to simulate the activity patterns experienced in soccer.

#### **2.5.2.2 Muscular dysfunction**

It is clear that following strenuous exercise there is an element of muscular soreness and pain. Arguably, one of the most relevant assessments of the extent of this EIMD is its effect on subsequent exercise performance. Several investigators have used measures of muscular dysfunction to quantify the impact of muscle damage, with maximal voluntary contractions of a particular muscle group being one of the most popular methods of assessment (Bryne et al., 2004). Several studies have used muscular performance decrements as a potential indicator of how effective certain preventative or therapeutic interventions are on EIMD. Clearly, any intervention that has the potential to limit the decline in muscle function in the days following performance could be beneficial to soccer players who are often faced with the challenge of attempting to compete following very short rest periods from the previous performance.

## 2.6 Effect of cryotherapy on muscle damage

Stretching, anti-inflammatory drugs, anti-oxidant supplementation, massage, ultra sound, exercise and cryotherapy have all been mooted as potential treatment strategies for DOMS and muscular dysfunction following strenuous exercise (Cleak and Eston, 1992). Cryotherapy administered through cold-water immersion ('ice baths') has become a popular post exercise treatment for elite athletes in a variety of sports.

Cryotherapy has been defined as the use of ice or cold application for therapeutic purposes that results in the withdrawal of heat from the body, thereby lowering tissue temperature (Knight, 1995). Cryotherapy has long been established as a treatment for soft tissue injuries in sport and can be administered in a number of different ways including ice, ice-packs, ice massage, cryokinetics, chemical spray, thermo-electric cooling and cold water immersion (Knight, 1995; Enwemeka et al., 2002).

Cryotherapy is believed to diminish the inflammatory reaction of trauma, to reduce oedema, haematoma formation and pain (Meeusen and Lievens, 1986). Other physiological actions such as vasoconstriction, decrease of blood flow, reduction of muscle spasm, decrease in nerve conduction velocity have been attributed to cryotherapy (Meeusen and Lievens, 1986).

One of the most prominent effects of cryotherapy is its analgesia (Swenson et al., 1996). The exact cause of this analgesic effect has been categorised by Knight (1995) as direct or indirect. The direct effects include the elevation of the pain threshold by decreasing temperature of nerve fibres blocking nervous transmissions of pain receptors or pain fibres (Knight, 1995). Furthermore, cryotherapy directly affects muscle spindles by decreasing the rate of discharge (Knight, 1995). The indirect analgesic effects of cooling may be the result of removing the source of the pain, like muscle spasms and the spasticity of muscle, by either sensory and motor nerve activity, a reflex mechanism or by breaking the pain-spasm cycle (Knight, 1995).

The cause of this analgesic effect is the application of cold onto the skin. However, it is interesting to consider how much skin and muscle temperature is actually altered during

and following cold application. Cold application inevitably reduces skin temperature (Kowal, 1983), however the extent of this decline is dependant on time, duration, method and temperature of the cold that is applied. Comparisons between studies examining the effects of cryotherapy on intramuscular temperature show some degree of variation, perhaps not least due to the different depths at which the thermistor probe is placed. The changes reported in muscle temperature are certainly smaller than that of the skin and subcutaneous fat as one might expect. It appears that the decrease of intramuscular temperature depends on the length of the cold treatment (Johnson et al., 1979) and correlates with the initial temperature of the application modality (Trnavsky et al., 1980 cited in Meeusen and Lievens, 1986). A review from Meeusen and Lievens (1986) presents inconclusive evidence for the initial effect of cooling on muscle temperature; some studies report initial increases, others report decreases and some no change. However, several studies have reported a prolonged cooling effect on the muscle even after the treatment has been withdrawn (Bing and Watts, 1962; Oliver and Johnson, 1976).

The application of cold appears to have an effect on haemodynamics. The cold causes constriction of capillaries around the area of application that in turn is believed to retard haematoma formation (Meeusen and Lievens, 1986). Unfortunately, studies examining the effects of temperature on muscle blood flow are limited by measurement errors and procedures.

Following a period of time that a tissue is cold it can actually start to re-warm independent of any external heating agent. This phenomenon is often referred to as the 'Hunting response' and essentially describes the cycle of cooling and re-warming following the application of cold. The initial application of cold causes an increase in the affinity of the post-junctional  $\alpha$ -adrenoceptors for noradrenaline, this results in a period of powerful vasoconstriction and reduction of blood flow around the site of cooling. However, as the tissue continues to cool, sympathetic nerve conduction is interrupted and vasodilatation occurs due to the cessation of noradrenaline release. The blood flow is restored to this area of tissue, which in turn, re-establishes nerve conduction and this combined with an increased affinity of the post-junctional  $\alpha$ -adrenoceptors for noradrenaline leads to further vasoconstriction (Shepherd et al., 1983

cited in Meeusen and Leivans, 1986). This creates an on-going cyclical mechanism of vasoconstriction and vasodilation (Swenson et al., 1996).

Cryotherapy has been used in sports injuries to control the level of inflammation and indeed dampen the inflammatory response (McLean, 1989). The degree of inflammation within a muscular tissue is influenced by the capillary permeability and cellular response both of which are directly influenced by temperature.

Despite, much debate over the precise mechanisms responsible for any possible benefits of cryotherapy in the recovery of tissue injury, it is clear that analgesia, vascular responses and metabolic alterations all make a contribution. However, the evidence is not completely clear when examining whether cryotherapy will actually alter recover times following EIMD. Cheung and co-workers (2003) inconclusively reviewed a number of studies in this area and reported evidence of a positive effect, a negative effect and some showing no effect at all. A number of key studies have been summarised in table 2.1 and some of the most relevant studies are discussed below.

Eston and Peters (1999) reported a greater relaxed joint line and lower CK response at 24 and 48hr post eccentric exercise in those subjects who received ice water immersion for 15 min at 15<sup>0</sup>C. However, no effect was seen on perception of soreness between groups. Eston and Peters (1999) reported that the reductions in CK activity were likely to be the result of cryotherapy either reducing permeability of lymph vessel walls reducing the efflux of from the muscle or reducing the amount of post exercise damage. The suggestion that cryotherapy somehow increased the clearance of CK was discounted to the requirement of an increased blood flow. Cryotherapy is likely to induce the oppose effect of vasoconstriction and reduced blood flow (Eston and Peters, 1999).

A study conducted by Howatson and Van Someren (2003) reported similar potential beneficial effects of cryotherapy. In this study they examined the effects of ice massage on the signs and symptoms associated with exercise induced muscle damage. Those participants who had ice massage treatment (15 min of ice massage immediately post exercise, 24hr and 48hr post exercise) following the completion of single arm bicep curls with an emphasis on the eccentric phase of the movement had lower CK response

72 hours post exercise. Interestingly, it appears that repeated bouts of cryotherapy in the immediate hours post exercise is no more beneficial than a single application (Padden-Jones and Quigley, 1997).

In contrast to these positive findings several studies have found little or no effect of cryotherapy treatments on EIMD (See table 2.1). One such study used eccentric elbow flexion as the method of inducing muscular damage in 22 subjects and reported no effect of ice massage on range of movement, DOMS and CK. These contrast to the findings of Howatson and van Someren (2003).

There are several possible reasons for a lack of consistency in findings between these studies. Variations in the method of cold application, temperatures, frequency of application have all been suggested as possible causes of discrepancies (Eston and Peters, 1999). Furthermore, variations will exist in the response to muscle damaging exercise in subjects depending on training status, training history and familiarity to eccentric exercise (Cheung et al., 2003).

## **2.7 Summary**

There appears to be a number of fitness assessments available to support staff wishing to gain a better understanding of their players physical condition, indeed many of these protocols have been described in the literature and tested for their reliability and validity. A good example of one such study is the Yo-Yo intermittent recovery shuttle running test, designed by Bangsbo and co-workers (2003). However, very few of these fitness tests protocols have been examined to determine their sensitivity to detect training-induced changes in fitness. Furthermore, despite an extensive search it seems that very little information is available when one wishes to know exactly which, if any, of these fitness assessments are used within professional football. For this reason the investigation into the extent and content of fitness assessment practices within professional soccer is warranted.

There has been an increase in the number of training studies being published concentrating specifically on interval training with elite soccer players. The evidence

from several investigators is consistent in that additional high intensity interval training performed twice a week in addition to normal training can have quite marked improvements in aerobic capacity of players within a relatively short timeframe. However, these studies have shown this when using male senior and junior elite players, no information is available on this training protocol on elite female soccer players.

There is convincing evidence demonstrating the importance of carbohydrate consumption during the recovery following prolonged exercise. Several reviews have successfully summarised a number of studies that show that the quantity, timing and type of carbohydrate within the recovery phase post exercise are all critical to the overall success of muscle glycogen resynthesis. However, there is very little research on the effects of different types of CHO recovery diets on subsequent performance. This warrants further investigation.

There are several studies highlighting the potential rate limiting effects of muscle damaging exercise (mostly eccentric exercise) on subsequent muscle glycogen resynthesis. For this reason the focus of the literature review shifts to examine research on a recovery modality (cryotherapy) that is associated with a potential beneficial effect on muscle soreness and function following eccentric actions. However, a summary of this research (included in table 2.1) highlights large variations in study design, damaging exercise, treatment protocols and overall conclusions. Furthermore, no literature was available on the effects of cryotherapy following intermittent running experienced in popular sports like rugby and soccer. Once more, this led our investigations towards this potentially important area for elite intermittent games players who are often faced with very busy training and playing schedules.

In summary, a search of the available literature on these key influences on performance show that there is a general lack of published information on this area. Therefore, the aim of this programme of research described in this thesis was to undertake research studies to help provide missing information in order to progress our understanding and facilitate best practise.

**Table 2.1** A summary of investigations employing cryotherapy prior to, during or after exercise

Reference	Sample	Exercise	Cryotherapy	Findings
Prentice (1982)	N=50 (males and females)	Strenuous exercise	20 min treatment 24 hr post exercise. 5 different groups: <ul style="list-style-type: none"> <li>- cold &amp; static stretch</li> <li>- cold &amp; PNF stretch</li> <li>- heat &amp; static stretch</li> <li>- heat &amp; PNF stretch</li> <li>- control</li> </ul>	Cold & static stretching superior treatment in reducing muscle pain. ----- Cold & stretching more effective than heat & stretching.
Yackzan et al. (1984)	N=30 (female)	Eccentric contractions of elbow flexors	15 min ice massage treatment either IMPE, 24hr, 48hr post exercise	No effects on DOMS or ROM. Those treated 24hr post reported greater soreness in treated arms compared to untreated.
Braun and Clarkson (1989)	N=7 (males)	Eccentric contractions of elbow flexors	Ice water immersion 25 min pre-exercise & during exercise	No effect on CK, isometric MVC & relaxed joint angle
Denegar and Perrin (1992)	N=40 (females)	Eccentric contraction of the elbow flexors	20 min treatment 48hr post exercise or either: <ul style="list-style-type: none"> <li>- Crushed ice applied to biceps</li> <li>- Transcutaneous electrical nerve stimulation (TENS)</li> <li>- Cold and TENS</li> <li>- Sham TENS</li> <li>- Control</li> </ul>	Cold, TENS & the combined treatment decreased DOMS ----- Cold treatments increased elbow extension ROM ----- Static stretching reduced perceived pain.

<b>Isobell et al. (1992)</b>	N=22 (males and females)	Eccentric contractions of elbow flexors	8 x 15 min treatments at 0, 2, 4, 6, 24, 48, 72 & 96 hr post exercise of either: <ul style="list-style-type: none"> <li>- Ice massage</li> <li>- Exercise</li> <li>- Ice massage &amp; exercise</li> <li>- Control</li> </ul>	No effect on ROM, CK, MVC and DOMS in all groups
<b>Paddon-Jones &amp; Quigley (1997)</b>	N=8 (males)	Eccentric contraction of elbow flexors	5 x 20 min of ice water immersion at 5°C (1hr between each immersion). Starting IMPE	No effect on DOMS, MVC or limb volume/swelling
<b>Kokkinidis et al. (1998)</b>	N=12 (males)	Eccentric contraction of knee flexors	20 min treatment IMPE & 24hr post of either: <ul style="list-style-type: none"> <li>- Ice-compression</li> <li>- Static stretching</li> <li>- control</li> </ul>	No effect of ice treatment on DOMS, MVC or CK Ice improved recovery of ROM Stretching reduced DOMS & improved ROM
<b>Eston and Peters (1999)</b>	N=15 (females)	Eccentric contraction of knee flexors	15 min IMPE of either: <ul style="list-style-type: none"> <li>- Cold water immersion (15°C)</li> <li>- Control</li> </ul>	Improvements for relaxed joint angle & CK. No effect on DOMS, swelling and strength loss.
<b>Comeau (2000)</b>	N=14 (males)	Eccentric contractions of the knee flexors	20 min cold water immersion 15°C. Repeated for 5 days	No effect on CK, MVC, ROM & jump performance
<b>Howatson and Van Someren (2003)</b>	N=9 (males)	Eccentric contraction of elbow flexors	IMPE, 24 & 48hr 15 min of ice massage vs. SHAM	Lower CK after 72hr No effect on DOMS, ROM & swelling.



## CHAPTER 3

### GENERAL METHODS

#### 3.1 Introduction

Methods that are common to multiple studies within this thesis have been included in this section together with specific details of the procedures used for the collection, storage and analysis of blood. Details of methods that were exclusive to a particular study can be found in the relevant chapter.

All the procedures outlined in this chapter and in the method sections of the subsequent experimental chapters have received approval from the Loughborough University Ethical Advisory committee. All procedures were undertaken in accordance with the 'Code of Practice for Workers having Contact with Body Fluids'. Furthermore, all personal data were treated in accordance with the data protection act.

Any potential participants were issued with an information booklet outlining the aims, procedures and demands. Furthermore, a full verbal explanation was given to individuals outlining the commitments, and possible discomforts associated with the protocol before informed written content was obtained (Appendix A). At this point participants were asked to complete a general health questionnaire (Appendix C) in order to ascertain any potential adverse medical condition, illness or injury. Any participants who reported any current or past medical conditions indicated on these forms were excluded from participations. On the day of each trial a daily health questionnaire was administered (Appendix A). Participants were asked to complete a physical activity history questionnaire (Appendix D). Finally, it was made clear to all participants that they could withdraw from a study at any stage without giving a reason.

## **3.2 Experimental Testing Procedure**

### **3.2.1 Testing Facilities**

All testing for Studies 2, 3, 4 and 5 (Chapters 5, 6, 7 and 8) was conducted in the School of Sport and Exercise Sciences laboratory areas including the Indoor Fitness Centre. This Fitness Centre is a large open plan sports hall with a wooden non-slip sprung floor. The flooring has line markings at 0m, 10m and 20m used for the Multistage Shuttle Run Test (Ramsbottom et al., 1988) and the Loughborough Intermittent Shuttle Test (LIST) (Nicholas et al., 2000).

### **3.2.2 Anthropometry**

Height was obtained to the nearest 0.1 cm using a stadiometer (Holtain Ltd., Crymch, UK). Nude body mass was recorded to the nearest 0.1 kg using beam balance scales (Model 3306ABV, Avery Ltd., Birmingham, UK). Body mass was taken prior to exercise after the subjects had void and again post exercise once the excess sweat had been removed.

In Studies 4 and 5 skinfold thickness measurements were obtained using pre-calibrated callipers (Holtain Ltd., Crymch, UK). Measurements were taken from 4 sites, from the right hand side of the body in accordance for the ACSM guidelines to exercise testing and prescription (ACSM, 1995). The four sites used were all measured and marked prior to assessment. The four sites were; i) biceps; a vertical fold taken on the anterior aspect of the arm over the biceps muscle 1 cm above the level taken for the triceps, ii) triceps; a vertical fold on the posterior midline of the upper arm, halfway between the acromion and olecranon process, with the arm held freely to the side of the body, iii) subscapular; a diagonal fold taken at 45° 2 cm below the inferior angle of the scapula and iv) superrailium; an oblique fold in line with the natural angle of the iliac crest taken in the anterior auxiliary line immediately superior to the iliac crest. Results were presented as the sum of four skinfold sites (mm) averaged from the 3 duplicate measurements.

### **3.2.3 Estimation of Maximal Oxygen Uptake**

The multi stage fitness test (MSFT) was used in all studies to establish an estimation of maximal oxygen uptake (Ramsbottom, et al., 1988). This test protocol involved individuals running continuously back and forth over a distance of 20 m. The speed of these runs, controlled via an audio signal that was generated via a compact disc (National Coaching Foundation, Leeds, UK). The speed of the runs increased every minute. The initial running speed was  $2.22 \text{ m}\cdot\text{s}^{-1}$  increasing by  $0.14 \text{ m}\cdot\text{s}^{-1}$  at each new level. Participants were verbally encouraged to run until they reached volitional fatigue or until they were unable to maintain the required intensity on two consecutive shuttles. The level an individual achieved was recorded and used to determine an estimated maximal oxygen uptake value using a table of predicted values (Ramsbottom et al., 1988). From these values running speeds equivalent to 55% and 95% of each individuals estimated  $\text{VO}_2 \text{ max}$  were calculated and used in studies 3, 4 and 5 during the LIST protocol (See section 3.2.6).

### **3.2.4 Expired Air Collection and Analysis**

Expired air was collected using a standard Douglas bag technique (Williams et al., 1990) during the treadmill testing in study 2 and during all non exercise measurements in the other studies. During the LIST expired air was collected using a modified Douglas bag attached to a lightweight rucksack.

#### **3.2.4.1 Standard Douglas Bag Method**

Participants inserted a mouthpiece (Harvard Apparatus Ltd., Edenbridge, UK) and noseclip (Harvard Apparatus, Cambridge) attached to a 200L Douglas bag (Harvard Apparatus, Cambridge) via a lightweight low resistance two-way valve, a 0.5m length of wide-bore (30mm) lightweight tubing (Falconia) and a two-way tap (Harvard Apparatus, Cambridge). The mouthpiece was inserted 30s prior to the one min collection in order to ensure all atmospheric air in the tubing was expelled.

### **3.2.4.2 Analysis of Expired Air**

Gas fractions for oxygen and carbon dioxide were measured by sampling through a combined paramagnetic O<sub>2</sub> transducer (Servomex 1440, Crowborough, UK) and infra-red carbon dioxide analyser (Servomex 1440, Crowborough, UK). The analyser was calibrated prior to each trial with nitrogen (BOC Gases, Surrey, UK) and a certified reference gas of a known concentration (16.0% O<sub>2</sub> and 4% CO<sub>2</sub>).

Douglas bags were fully evacuated via a digital, dry, gas meter in order to determine total gas volume. A thermistor probe (Edale type 2984, Model C, Cambridge, UK) monitored the temperature of the expired air. The digital, dry gas meter was checked regularly against a precision 3L calibration syringe (5530 Hans Rudolph Inc, Kansas, Massachusetts, USA).

Gas Volumes were corrected to Standard Temperature and Pressure of a Dry gas (STPD). Values for oxygen consumption (VO<sub>2</sub>), carbon dioxide production (VCO<sub>2</sub>) and the respiratory exchange ratio (RER) were calculated under the assumptions of the Haldane transformation.

All participants were familiarised with expired air collection methods prior to main trials to minimise the risk of any abnormal breathing patterns.

### **3.2.5 Heart Rate Monitoring and Rate of Perceived Exertion**

During all trials in studies 3, 4 and 5 subjects' heart rate was recorded via coded short-range telemetric transmitters and wristwatch receiver-loggers (Polar Sports Tester™, Polar Electro, Kempele, Finland). During the training aspects of study 2 subjects wore Polar Team System belts (Polar™, Electro, Finland). All devices were downloaded onto a PC that had been installed with the necessary software (Polar HR Analysis Software, Version 5.04). During all trials participants rate of perceived exertion was recorded at regular intervals during exercise (Appendix F).

### 3.2.6 Dietary Analysis

In all of the experimental studies (except Study I) participant's nutritional intakes were monitored and analysed (Appendix G). Subjects were asked to conduct a weighed food and fluid intake diary over a two-day period. These were subsequently analysed using a computer software package (COMP-EAT version 4.0, Nutritional systems, UK). During Study III, subjects were requested to complete the food diary on the two days prior to the first trial. Individuals were asked to repeat the exactly same food and fluid intakes in the days preceding the second trial of the cross-over design.

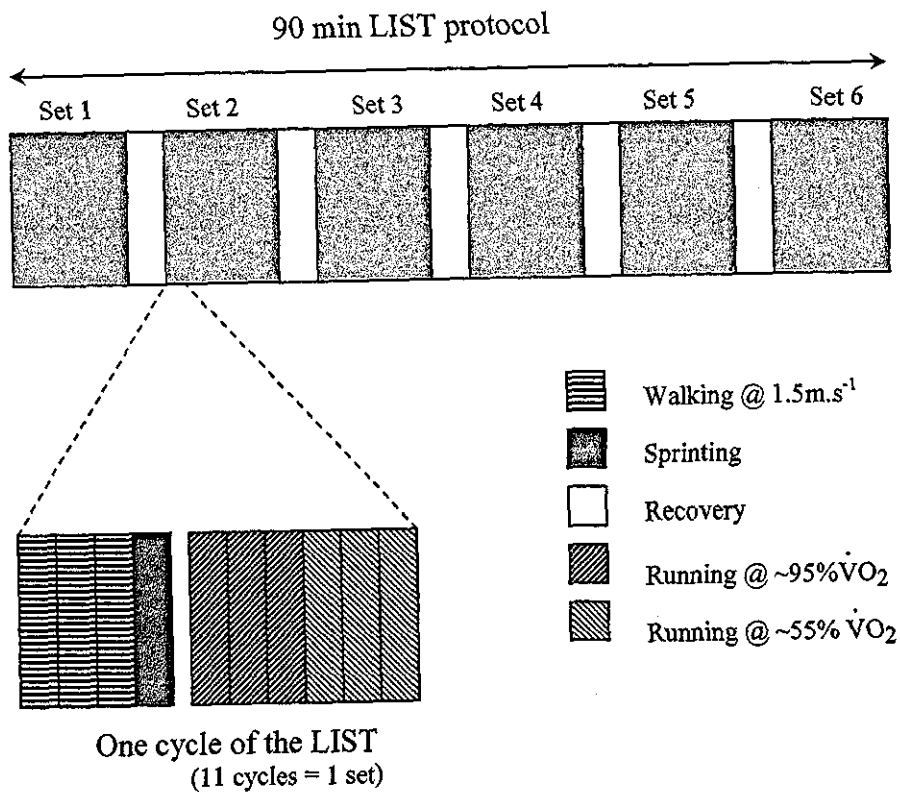
### 3.2.7 The Loughborough Intermittent Shuttle Test (LIST)

The exercise protocols for studies 3, 4, and 5 were based on the Loughborough Intermittent Shuttle Test (LIST), (Nicholas et al., 2000). This protocol involves shuttle-running exercise designed to stimulate the activity patterns of a game of soccer (figure 3.1). The overall duration of the protocol is 90 min at an average intensity of approximately 70%  $VO_{2\text{ max}}$ . Subjects cover a distance of approximately 12 km and make 624 changes of direction separated into 6 distinct blocks. Each block lasts approximately 15 min and is punctuated by rest periods of a 3 min duration. Within each block individuals are required to run and walk back and forth over a distance of 20 m. The cyclic activity pattern constitutes of;

- 3 x 20 m walking ( $1.54\text{ m}\cdot\text{s}^{-1}$ )
- 1 x 20 m maximal sprint
- 4s recovery
- 3 x 20 m at a running speed corresponding to 95% of participant's  $VO_{2\text{ max}}$
- 3 x 20 m at a running speed corresponding to 55% of participant's  $VO_{2\text{ max}}$

It should be noted that the order of the 'cruising' and jogging in this thesis differs from the sequence detailed by Nicholas and co-workers (2000). The reason for this slight alteration was an attempt to better control the speed of the cruise and jog components of each cycle. Unfortunately, subjects who ran slightly quicker on the jogging phase during the original sequence could then reduce the intensity of the cruising phase.

During these trials participants were prescribed a controlled fluid (water intake). The volume of fluid consumed equated to  $5\text{ml.kg}^{-1}$  pre-exercise and  $2\text{ml.kg}^{-1}$  during the 3 min rest period between 15 min blocks. This volume of fluid has previously been shown to maintain euhydration during the LIST running protocol (McGregor et al., 1999).



**Figure 3.1** Diagrammatical representation of the LIST exercise protocol

### 3.2.8 Blood Sampling

Blood sampling was included in all main trials during studies 3, 4 and 5. Participants were fitted with an indwelling cannula (Venflon, 18G, BOC, Ohmeda, Sweden) inserted into the antecubital vein. The cannula was attached to a 3-way stopcock (Connecta Ltd., Sweden) with a 10 cm extension tube. Each sample was taken while the subject remained standing and approximately 10 ml of blood was collected on each occasion.

Following each sample the cannula was flushed with sterile saline (0.9% Sodium Chloride, Steripak Ltd., UK) in order to keep it patent.

Following the collection of 10 ml of blood, it was immediately transferred from the syringe into collection tubes (Sarstedt Ltd., UK). Approximately 5 ml of blood was dispensed into an anticoagulant ethylenediaminetetraacetic (EDTA) tube, following which aliquots of blood were removed for the determination of haemoglobin, hematocrit and lactate. The tube was then placed into a centrifuge (Allegra X-22R Centrifuge, Beckman Coulter, Germany) where it was spun at 3000 rpm for 10 min at a temperature of 4°C. This allowed for the collection of plasma samples that were immediately frozen and stored at -85°C. The remaining 5 ml of whole blood collected was dispensed into a non-treated tube where it was left to clot for 45 min before being centrifuged at 3000rpm for 10 min at 4°C. This allowed for the collection of serum samples.

### **3.2.9 Blood Analysis**

All of these assays were carried out in the Loughborough University School of Sport and Exercise, Biochemistry Laboratory with the exception of the serum insulin and cortisol assays that were conducted in the Radiochemistry Laboratory situated on campus. All assays were conducted using commercially available kits.

#### **3.2.9.1 Plasma glucose, free fatty acids and glycerol**

An automatic photometric analyser (Cobas-Mira Plus, Roche Diagnostic Systems, Switzerland) was used to measure plasma glucose (GOD-PAP method, Randox, Ireland), free fatty acids (FFA) (ACS-ACOD method Wako NEFA C, Germany) and glycerol concentrations (GPO-PAP method, Randox, Ireland).

#### **3.2.9.2 Serum myoglobin and creatine kinase activity**

Serum myoglobin concentrations were measured using an immunoturbidimetric assay that is commercially available (Randox, UK). Serum creatine kinase activity was measured using spectrophotometric techniques and a commercially available kit

(Randox, UK). Both of these techniques were conducted using an automated system (Cobas-Mira Plus, Roche Diagnostic Systems, Switzerland).

### **3.2.9.3 Plasma volume**

Changes in plasma volume were determined using the haematocrit and haemoglobin values (Dill et al., 1974). Haematocrit values were measured using a microhaematocrit reader (Hawksley Ltd., Lancing, UK) following micro-centrifugation for 15 min. Haematocrit measurements were taken in triplicate. Haemoglobin was measured in duplicate using the cyanmethaemoglobin method (Boehringer Mannheim, GmbH Dianostica, Germany) with a spectrophotometer (UV mini 1240, Shimazu, Japan).

### **3.2.9.4 Serum insulin and cortisol**

In Study III radioimmunoassays were conducted to measure insulin (Coat-A-Count Insulin ICN Ltd, Eschwege, Germany) and cortisol (Corti-Cote ICN Ltd, Eschwege, Germany) concentrations using a gamma counter (Cobra 5000, Packard Ltd, Boston, MA, USA).

### **3.2.9.5 Blood lactate**

Whole blood (20 $\mu$ l in duplicate) was deproteinised in 200 $\mu$ l of 2.5% perchloric acid. Samples were then centrifuged for 3 min at 13000 rpm (Eppendorf centrifuge 5415D, Hamburg, Germany). Blood lactate concentrations were determined using a modified fluorometric method as previously described (Maughan, 1982).

### **3.2.9.6 Intra-assay variation**

The coefficient of variation (Standard Deviation/Mean\*100) of the blood, plasma and serum assays are shown in table 3.2. Each coefficient of variation was determined using at least 20 samples.



**Table 3.1** Intra-assay variation

Metabolite	Units	C.V (%)
Blood lactate	mmol.L <sup>-1</sup>	1.4
Plasma glucose	mmol.L <sup>-1</sup>	1.2
Plasma FFA	mmol.L <sup>-1</sup>	1.1
Plasma glycerol	μmol.L <sup>-1</sup>	2.3
Serum insulin	μIU.ml <sup>-1</sup>	6.2
Serum cortisol	μg.dL <sup>-1</sup>	4.4
Serum myoglobin	mmol.L <sup>-1</sup>	2.5
Serum creatine kinase	U.l <sup>-1</sup>	2.2

## CHAPTER 4

### **An overview of fitness testing practices within English professional football**

#### **4.1 Introduction**

The physiological demands of soccer are becoming increasingly well understood. Players must have physical competencies in several areas of fitness including endurance, speed, agility and flexibility to cope with the match demands. Several studies have shown a possible link between the success of the team and physical fitness.

It would seem logical for coaches and players to gain objective information on a player's performance or capacity within key areas of fitness. A number of different fitness tests have been established for use in both the laboratory and field settings. Many of these testing protocols are generic and used across a variety of sports. More recently, tests that may be more specific to the activity patterns of soccer have been developed (Kemi et al., 2003; Krstrup et al., 2003). Bangsbo (1994) suggests that the results from fitness testing can be used to plan short- and long-term training programmes, provide objective feedback and motivate players. Others suggest that testing can be used to study the effectiveness of a training programme (MacDougall and Wenger, 1991; Balsom, 1994).

Several review articles have focused sections of their papers on the area of fitness testing for soccer (Shepard, 1999; Hoff and Helgerud, 2004; Hoff, 2005; Svensson and Drust 2005). Interestingly, despite a relatively large number of publications into this area there appears to be nothing available in the public domain outlining the prevalence of fitness testing practices within English professional soccer. Therefore, the purpose of this study is to provide an overview of fitness testing practices within English professional football.

## 4.2 Methods

In order to establish the current working practises of fitness assessments within professional football clubs data was collected using a questionnaire. The questionnaire was designed to find out what fitness tests are most commonly employed, what time in the year were they conducted and how the results were used. The questionnaire contained a combination of multiple choice and short answer questions. Initially the questionnaire was piloted on two Premier League football clubs, after which amendments were made before establishing the final design.

All English Professional clubs were sent a letter that outlined the aims and objectives of the research. Each participating club that agreed to contribute to the study were asked to complete the questionnaire (Appendix J) in as much detail as possible between the months of May – September 2002 and return it by post. The Head Fitness Coach or the Head Physiotherapist was responsible for completing the questionnaires.

Any club not responding within two months of the deadline were sent one further copy of the questionnaire accompanied with a reminder letter.

### Statistical Analysis

The software package MedCalc<sup>®</sup> (version 4.16f – Windows 3.1) was used to analyse data. The  $X^2$  significance test was used to investigate any differences between Divisions. Statistical significance was accepted at  $P < 0.05$  level.

### 4.3 Results

The total number of professional football clubs that participated in this study was 69, a response rate of 75% (18 from the Premier League clubs [90%]; 18 from The Championship division [75%]; 20 from the first division [83%] and 13 from second division clubs [54%]. Sixty seven of the 69 clubs involved in this study used some form of fitness testing procedures with just one club from the first and one from the second division stating that no testing was carried out.

Overall, clubs employed a total of 21 different tests. A summary of the most frequently used fitness tests used by professional football clubs is shown in Table 1. The most commonly used being the multistage fitness test (MSFT) (Ramsbottom et al, 1988) and an acceleration sprint test over distances ranging from 5 to 40 metres. A greater proportion of Premier League clubs used vertical jump tests ( $P<0.05$ ), acceleration sprints ( $P<0.05$ ) and multiple sprint tests ( $P<0.01$ ) compared with the clubs from the Football Leagues.

Clubs used between 1 – 6 testing periods per season. The most frequent periods during the year to conduct tests on players were during the pre-season (85% of clubs test at this point), early season (63%) and mid-season (64%). There were no differences in the periods of the year during which testing was used by clubs. A total of 29% of all clubs reported that they had conducted reliability studies on the fitness tests they employed.

The most frequent use of fitness test results among clubs was to assess specific fitness levels of players (86%) and to assess a player's recovery from an injury (82%). A greater proportion of clubs in the Premier League, compared to the other league tables used the results to set training programmes (94% vs. 61%  $P<0.01$ ), establish baseline fitness scores (94% vs. 49%  $P<0.05$ ) and assess players before the club sign them (17% vs. 0%  $P<0.01$ ).

	Overall (%)	Premier League (%)	Other divisions (%)
MSFT	72%	50%	80%
Acceleration Sprints	60%	89%	49%*
Vertical Jumps	45%	78%	33%*
Agility tests	37%	39%	38%
1-5 RM Bench Press	37%	69%	29%
Yo-Yo Intermittent test	36%	33%	37%
Multiple sprint tests	33%	67%	20%**
1 – 5 RM Back Squat	25%	44%	18%
Cooper Run	19%	22%	18%
Laboratory based tests	7%	17%	4%

**Table 4.1:** Displays the most frequently used fitness tests within professional football clubs

Greater proportion of Premier League clubs used vertical jump (\* $P < 0.05$ ) and multiple sprint tests (\*\* $P < 0.01$ ) compared to clubs from other divisions

#### 4.4 Discussion

The main findings from this investigation were that the reported use of fitness testing within professional football was high. The most frequently used protocols were field-based tests including the MSFT, acceleration sprints and vertical jumps.

Field based fitness tests will have greater specificity and therefore increase the validity of the testing procedures (Balsom, 1994). The number of English clubs that reported the use of laboratory testing was extremely low. Laboratory based testing has a number of practical drawbacks that are likely to reduce its popularity including cost, facilities, time and the need for qualified test administrators. However, this does not mean that some of these types of tests will not be useful. A recent study reported the use of lactate threshold tests during the course of a season with elite junior soccer players (McMillan et al., 2005). In this study lactate testing was used throughout the course of the season and it proved a good measure of endurance training status.

It is perhaps no surprise that the most frequently used fitness test was a field-based assessment of aerobic power (MSFT), when one considers that during soccer match play, the majority of energy provision is derived from the aerobic energy system (Bangsbo, 1994). Furthermore, it is this energy system that dictates the players' ability to recover between short bouts of high-intensity exercise (Balsom, Ekblom and Sjodin 1994a; Balsom, Ekblom and Sjodin 1994b).

The MSFT has been used to estimate  $VO_{2 \max}$ . Ramsbottom and co-workers (1988) have validated this method and reported a strong relationship between predicting  $VO_{2 \max}$  following the completion of the field test compared to directly measured  $VO_{2 \max}$ . Svensson and Drust (2005) have criticised the MSFT based on the findings from two separate studies that state that it does not appear to be sensitive to training interventions or to differentiate between playing standards in soccer (Odetoyinbo and Ramsbottom, 1997; Edwards et al., 2003). An alternative fitness test used to measure the aerobic qualities of players is the Yo-Yo Intermittent Recovery Test (Krustrup et al., 2003). This test is intermittent and has been shown to correlate with the amount of high intensity running in matches. Furthermore, in a 12-week training study with soccer referees, Krustrup and Bangsbo (2001) reported that the test was sensitive to detect

changes resulting as a consequence of training. However, its use among English professional clubs appears to be very low. The exact reasons for the multi-stage fitness test being grossly more popular than other methods of assessing aerobic capacity was not determined within this study.

Our study found that a greater proportion of Premier League clubs used vertical jump tests ( $P < 0.05$ ), acceleration sprints ( $P < 0.05$ ) and multiple sprint tests ( $P < 0.01$ ) compared to the other football league clubs. The cost of equipment and the expertise needed to make accurate assessments of these measures may, in part, explain the differences between the divisions on these testing methods. Results from single sprint tests have been used to differentiate between different standards of play and different positional roles within the team (Kollath and Quade, 1993).

It appears that clubs from all divisions test their players at similar periods during the year. The pre-season period proved to be the most popular time to assess players' fitness status. This is a stage in the year when most clubs tend to have a greater focus on physical training following the close season break. Furthermore, without the presence of competitive matches during this period it is possible that clubs have more time to schedule in these fitness assessments.

In summary, it appears that the popularity of fitness testing in professional football is high. The period of the year when clubs administer these tests is similar between divisions and despite some variety in the types of tests employed, the majority of clubs use a similar core of fitness tests. However, the sensitivity of these fitness tests to detect physiological changes that result as a consequence of training is yet to be established.

## CHAPTER 5

### **The sensitivity of fitness tests, commonly used with Professional soccer, to detect performance changes that occur as a consequence of training in elite female soccer players**

#### **5.1 Introduction**

Our previous study (Chapter 3) reported that the MSFT, vertical jumps and single acceleration sprints were the most commonly used assessments of fitness in English professional football. Bangsbo (1994) suggests that fitness testing can be used to plan short- and long-term training programmes, provide objective feedback and motivate players. Others suggest that testing can be used to study the effectiveness of a training programme (MacDougall and Wenger, 1991; Balsom, 1994). Our previous study (Chapter 3) reported similar responses to the use of fitness test results within soccer.

In order to make judgements based on fitness testing data regarding the effectiveness of training it is essential that the administrator is aware of whether the test itself is sensitive enough to determine changes that occur as a consequence of training. It was stated in the previous chapter that the Yo-Yo endurance test has been shown to detect performance changes occurring as a result of training. However, this test is rarely used in English professional soccer. Therefore, it is necessary to determine the sensitivity of more commonly used tests within English professional soccer to detect changes in physical performances associated with training.

Therefore, the aim of this study was to determine if these fitness tests were sensitive enough to detect changes in performance associated with 6 weeks of additional training in already well-trained elite female soccer players.



## 5.2 Methods

### 5.2.1 Participants

The training group (TG) consisted of 16 elite female soccer players who represented either England Senior or England U19s teams. The players were based at The Football Association Women's Elite Player Development Centre at Loughborough University. The mean ( $\pm$  SEM) age, height and body mass for the TG was  $18 \pm 0.3$  years,  $164.1 \pm 1.8$  cm and  $61.4 \pm 1.9$  kg, respectively. The control group (CG) consisted of 7 females with a mean ( $\pm$  SEM) age, height and body mass of  $23 \pm 0.6$  years,  $170.9 \pm 1.8$  cm and  $66 \pm 2.7$  kg, respectively. The control group were not involved in any structured fitness regimes throughout this monitoring period.

### 5.2.2 Testing Procedures

Subjects reported to the laboratory one and half hours after a standardised breakfast. The body mass of subjects was recorded while in running kit (without footwear) using a beam balance (Avery Ltd., Model 3306 ABV). Height was measured using a stadiometer (Holtain Ltd.). Individuals performed a submaximal treadmill test followed by a  $\text{VO}_2$  max test (details below). These tests were separated by a 30 min rest period. Following these tests individuals were given a minimum of one and maximum of 3 days rest before performing all field-based tests in a sports hall (details below).

### 5.2.3 Laboratory tests

The Submaximal Treadmill Test involved subjects running on a level motorised treadmill (Runrace, HC 1200, Technogym, Italy) at a starting speed of 9 km/h. At the start of every 4<sup>th</sup> min the treadmill speed was increased by 1 km/h. Blood was taken from the right thumb during the last minute of each stage with minimal interruption (see blood analysis). This incremental test continued until subjects' heart rate reached at least 180 beats per min. and a RPE (Borg, 1973) score of 15 or greater. The test lasted approximately 16 min.

The method used to determine maximal oxygen uptake involved an incremental test of continuous uphill running to exhaustion, as previously described (Williams et al., 1990). The gradient of the treadmill began at 3° and was increased by 1.5° every 4<sup>th</sup> min until volitional fatigue. In order to ensure that the test was not terminated prematurely subjects had to achieve a RER value greater than 1, report a RPE value higher than 18 and a maximum heart rate (determined in previous testing periods). Expired air was collected during the 4<sup>th</sup> min of each block, using a Douglas bag (Harvard Apparatus, Edenbridge, Kent, UK), nose clip, respiratory tube and a mouthpiece (one-way valve). The composition of expired air was analysed using an infrared CO<sub>2</sub> analyser and paramagnetic O<sub>2</sub> analyser (Servomax 1440), and volume determined using gasmeter (Harvard Apparatus, Edenbridge, Kent, UK).

Heart rate was recorded during both tests using a short-range telemetry system (Sport Tester™, PE3000 Polar, Finland).

#### 5.2.4 Field-based tests

Subjects reported to a sports hall 1.5 hours after a standardised breakfast. The surface of the sports hall was wooden sprung flooring. Individuals were requested to wear indoor, non-slip trainers. Individuals performed a standardised 5 min warm-up consisting of different running activities before the testing commenced.

The first of the tests performed was the vertical jump. This test was conducted using a portable jump mat (Newtest, Full Power Testing System, Newtest Power Timers, Finland). Each subject had 3 attempts at the squat jump (no arms or countermovement) with 1 min rest between attempts. Following the total completion of the squat jump, they then had 3 attempts at the countermovement jump (without the use of the arms) and finished with the countermovement jump (with the use of the arms). All results were recorded and the best score for each jump method was used.

Following two preparation sprints, individuals were timed from a standing start, placed 1 metre behind the start gate, over a distance of 5m, 10m, 15 and 20m using photo-electric beams (Newtest, Full Power Testing System, Newtest Power Timers, Finland).

Subjects were given three attempts at this test with a minimum of 3 min recovery between attempts. The best time for each individual was used.

Finally, subjects performed the MSFT, as previously described (Ramsbottom et al., 1988). This test involved subjects shuttle running continuously between two lines 20m apart. The timing of each run was controlled by an audio signal (bleep) and running speeds increased each minute until the point of fatigue. The level the subject achieved was recorded as the last successfully completed shuttle before they reached volitional fatigue or failed to reach the requirements of the test. Heart rate was recorded during this test using Polar Team System™ heart rate monitors (Polar Ltd., London, UK).

The TG and the CG performed the tests as two separate groups. All tests were completed in the same location and order following the training period.

#### 5.2.5 Training Protocol

The training intervention lasted a total of six weeks. Each week consisted of two field based aerobic sessions and two gym based strength sessions. These sessions were completed in addition to the players' normal weekly training schedule consisting of 3 sets of 1.5 hours technical/tactical practice on separate days plus one competitive match (Appendix K).

The field based aerobic sessions consisted of interval training. The protocol employed involved 4 times 4 min of high intensity exercise at 90 – 95% of maximal heart rate, interspersed by 3 min of low intensity activity at 50-60% of maximal heart rate. These training intensities were adopted from Helgerud and co-workers (2003). The running activity during this training included dribbling a soccer ball around a marked track on a soccer pitch. This procedure has been validated and described in detail by Hoff and colleagues (2002). All subjects wore Polar Team System™ heart rate monitors (Polar Ltd., London, UK) throughout the aerobic interval training sessions and were verbally encouraged to maintain the prescribed exercise intensities. Heart rate traces were checked following each training session to ensure that individuals were training at the correct intensities.

The strength sessions involved 4 sets of 4 repetitions of half squats (90° angle of the knee joint) with 3 min rest between sets. Subjects were encouraged to lift as heavy weight as possible without failing and were asked to work slowly on the downward phase and explosively on the upward, concentric phase (Helgerud et al., 2003).

#### 5.2.6 Blood analysis

Duplicate 20µl aliquots of whole blood were taken by finger prick samples (Autoclix®, Mannheim, Boeringer) at every 4<sup>th</sup> min of each stage of the submaximal treadmill test. Blood samples were obtained while the subjects were running. Each 20 µl aliquot of whole blood was deproteinized with 200 µl 2.5% perchloric acid, centrifuged and frozen at -20 °C for subsequent analysis for lactate (Maughan 1982).

#### 5.2.7 Statistics

All results are presented as mean ± standard error of the mean (SEM). Delta differences were calculated from pre- to post-training values for body mass, squat jump, counter-movement jumps, acceleration sprints, distance covered, predicted and actual VO<sub>2 max</sub>. A one-tailed Independent t-test was used to examine the differences between the TG and CG delta scores due to the fact that we hypothesised an increase in performance following the training (Atkinson and Nevill, 2001). In an attempt to quantify the magnitude of the population effect (i.e. how worthwhile an effect was) the 95% confidence interval of the differences between treatments were stated. A three way analysis of variance (ANOVA) with repeated measures was used to assess the differences between groups, pre and post testing and 4 different time points during the Submaximal Treadmill Test. Statistical significance was accepted at P<0.05. All data was analysed using (SPSS for Windows Inc., Chicago, Illinois).

## 5.3 RESULTS

### 5.3.1 Subject Characteristics

The mean ( $\pm$  SEM) body mass of the TG increased by 1kg (pre-training body mass  $61.4 \pm 1.9$  vs. post-training  $62.4 \pm 2.0$  kg) ( $P < 0.01$ ). The body mass of the CG did not change over the training period (pre-mass  $66.2 \pm 2.7$  vs. post-mass  $66.9 \pm 2.6$  kg) (n.s).

### 5.3.2 Field Tests

The TG improved their maximal squat jump by 5.6% ( $P < 0.01$ ). Taking into account the control group there was a 3 cm improvement (95% CI = 0.88-5.12cm) in squat jump, 2cm improvement (95% CI = 0.14 – 4.24cm) in countermovement jump ( $P < 0.05$ ) and a 3cm improvement (95% CI = 0.25 – 5.25cm) in countermovement jump with the use of arms ( $P < 0.05$ ). Table 5.1 highlights the actual changes of the TG and CG following the intervention and the 95% confidence intervals.

There were no significant differences in the TG sprint times over 5, 10, 15 and 20m. However, there was a tendency for subjects to run quicker over 15 and 20m following the training period (Table 5.2). No differences were detected in the CG sprint and jump performances.

The TG improved their predicted  $VO_{2 \max}$  (Figure 5.1) and distance covered during the MSFT by 6% ( $P < 0.01$ ) and 12% ( $P < 0.01$ ), respectively after the training intervention (Table 5.3). These parameters remained unchanged in the control group.

### 5.3.3 Laboratory Tests

Subjects in the TG had a 4% improvement in their  $VO_{2 \max}$  values ( $P < 0.01$ ) following the training period (Figure 5.1). The post-training  $VO_{2 \max}$  values were  $51.2 \pm 1$  ml.kg.min<sup>-1</sup>. This improvement was 3.8 ml.kg<sup>-1</sup>.min<sup>-1</sup> greater than the  $VO_{2 \max}$  values of the control group (95% CI= 1.9 – 5.7). The TG managed to run for 74 seconds longer on their run time until exhaustion during the  $VO_{2 \max}$  test, following the training. This

equates to a 9.5% improvement in endurance capacity ( $P < 0.01$ ). No differences were detected in the CG from pre- to post- training period assessments for actual  $\text{VO}_2 \text{ max}$  ( $51 \pm 2.81$  vs.  $49 \pm 2.49 \text{ ml.kg}^{-1}.\text{min}^{-1}$ ) or predicted  $\text{VO}_2 \text{ max}$  ( $49 \pm 2.47$  vs.  $49 \pm 2.64 \text{ ml.kg}^{-1}.\text{min}^{-1}$ ).

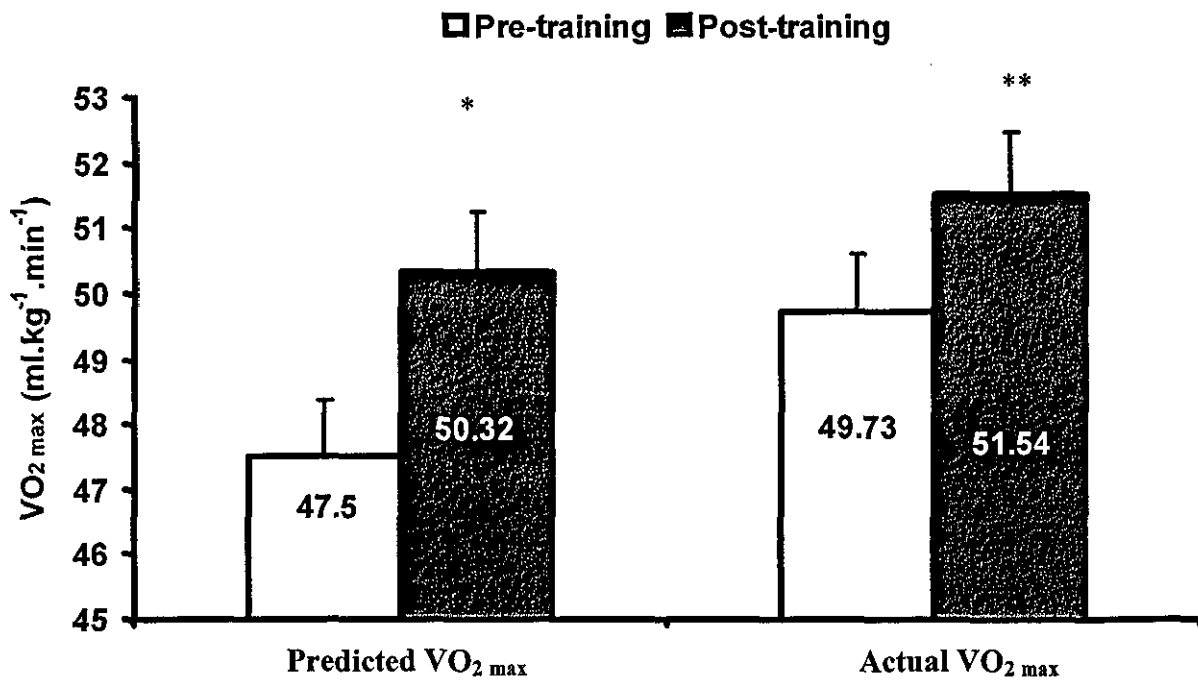
The TG had lower blood lactate concentrations ( $F_{1, 17} = 11.39$   $P < 0.003$ ) at each stage of the submaximal treadmill test following the training period (Figure 5.2). They also had lower heart rate responses ( $F_{1, 17} = 17.49$   $P < 0.001$ ) at each stage of the submaximal treadmill test following the training period (Figure 5.3). The rate of perceived exertion (RPE) scores (Borg 1973) during this test tended to be lower following the training period however, this was not found to be statistically significant (Final stage scores: TG pre-training  $16 \pm 0$  vs. post training  $15 \pm 0$ ) (n.s). No differences were detected in the CG for any of the parameters measured during the submaximal treadmill test.

#### 5.3.5 Control Variables

There were some minor changes in dry bulb temperature between the two testing periods (Pre:  $21.9 \pm 0.4$  vs. Post:  $19.8 \pm 0.6$ ,  $P < 0.01$ ). Furthermore, there were some minimal changes in relative humidity between the two testing periods ( $11 \pm 1$  vs.  $12 \pm 1$  %,  $P < 0.05$ ).

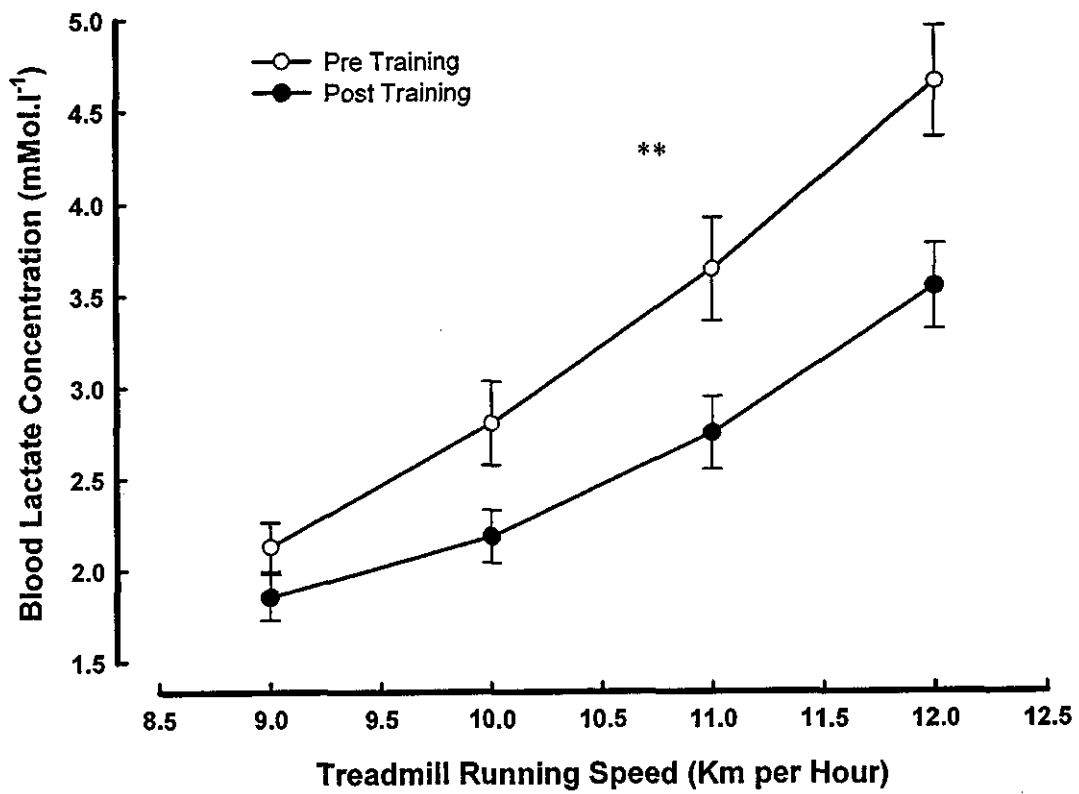
**Table 5.1** Displays the results from all fitness tests pre- and post-training. Improvements in the TG are marked as follows \*\*P<0.01 and \*P<0.05.

	<b>TG</b> Pre-training	<b>TG</b> Post-training	<b>CG</b> Pre-training	<b>CG</b> Post-training	<b>95% CI</b>
<b>Squat jump</b> (cm)	31 ± 1	33 ± 1	31 ± 1	29 ± 1	0.88 – 5.12
<b>CMJ</b> (cm)	32 ± 1	32 ± 1	32 ± 1	30 ± 1	0.14 – 4.24
<b>CMJ-arms</b> (cm)	36 ± 1	37 ± 1	35 ± 1	34 ± 1	0.25 – 5.25
<b>5m sprint</b> (s)	1.08 ± 0.02	1.13 ± 0.02	1.11 ± 0.03	1.12 ± 0.03	-0.05 – 0.13
<b>10m sprint</b> (s)	1.93 ± 0.02	1.94 ± 0.02	1.92 ± 0.02	1.93 ± 0.04	-0.7 – 0.75
<b>15m sprint</b> (s)	2.68 ± 0.02	2.67 ± 0.03	2.74 ± 0.04	2.79 ± 0.05	-0.61 – 0.1
<b>20m sprint</b> (s)	3.38 ± 0.02	3.36 ± 0.03	3.45 ± 0.05	3.44 ± 0.07	-0.11 – 0.84
<b>Predicted</b> <b>VO<sub>2</sub> max</b> (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	47.6 ± 1	50.63 ± 0.62	52.14 ± 1.6	52.69 ± 1.7	0.65 – 4.36
<b>Distance</b> <b>Covered</b> (m)	1647 ± 59	1843 ± 59	1936 ± 105	1966 ± 116	46 – 309

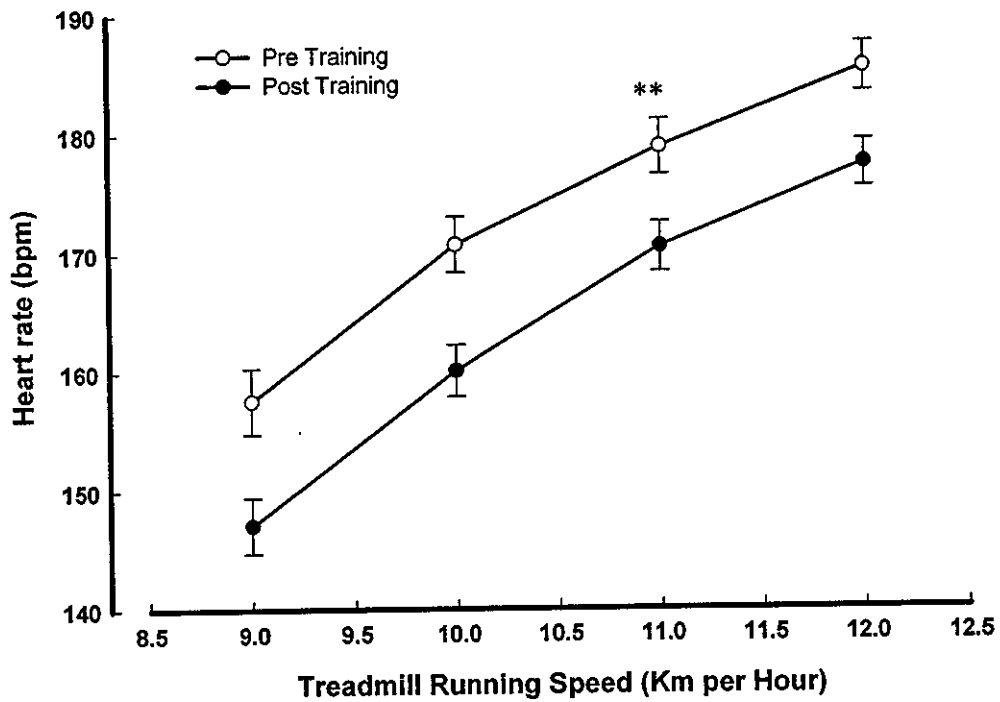


**Figure 5.1** Displays the results from the Progressive Multistage Shuttle Run and The Treadmill Maximal Oxygen Uptake Test. Improvements in the TG predicted VO<sub>2 max</sub> \*P<0.01 and actual VO<sub>2 max</sub> \*\*P<0.01.





**Figure 5.2** Displays the blood lactate concentrations during The Submaximal Treadmill Test pre and post training period. The TG had a lower blood lactate concentration at each stage of the test following training **\*\*P<0.01**.



**Figure 5.3** Heart rate responses during The Submaximal Treadmill Test pre and post training. The TG had a lower heart rate values at each stage of the test following training  
 \*\*P<0.01.

## 5.4 Discussion

The MSFT and vertical countermovement jump were sensitive enough to detect performance changes associated with a period of training in elite female players. Furthermore, high intensity aerobic interval training appears to be an effective method of improving the physical capabilities of already well-trained elite female soccer players.

The current study found a 6% and 4 % improvement in predicted  $VO_{2\text{ max}}$  and directly determined  $VO_{2\text{ max}}$  scores, respectively following the 6 weeks training period. This is consistent with similar training studies on male elite soccer players. Helgerud and colleagues (2002; 2001) reported improvements in  $VO_{2\text{ max}}$  of 8.1% and 10.8%, respectively ( $P < 0.01$ ) following the same training protocol over a period of 8 weeks. Furthermore, the improvements are similar to the 9% and 9.6% reported on male youth players by McMillan and co-workers (2005) and Chamari et al., (2005), respectively. McMillan and colleagues (2005) reported the training effect of each session of high intensity aerobic 4 min intervals being approximately 0.5%; this is consistent with our findings. The main reason for our results being lower than previous findings is likely to be due to the duration of the training period. Our study comprised of 6 weeks training compared to the 8 weeks in previous studies, the reason for adopting a slightly shorter training period in the present study was to control for menstrual cycle of the subjects. The players were tested on week one and then retested on week 8 exactly two months following the initial testing.

The improvements in predicted and actual  $VO_{2\text{ max}}$  following training were similar (6% vs. 4%). The slightly greater improvement in the MSFT may be due to the fact that the soccer specific training protocol incorporated a large proportion of turning, acceleration and deceleration. All of these activities are involved in the MSFT.

The Submaximal Treadmill Test results support the performance improvements in the  $VO_{2\text{ max}}$  test and the MSFT. Following the training period TG subjects had significantly lower lactate concentrations on each stage of the incremental treadmill running test. Furthermore, the TG also had significantly lower heart rates throughout the test. These

findings were expected considering the well-documented central and peripheral adaptations that result from aerobic interval training (See Maughan, et al. 1997). However, the exact mechanisms responsible for the changes seen in this study were not examined.

Interestingly, the TG experienced a 12% increase in the distance covered during the MSFT ( $p < 0.01$ ) and a 9.5% improvement in the run time to exhaustion on the treadmill  $VO_2 \text{ max}$  test ( $p < 0.003$ ). The benefits of these improvements in running capacity at very high intensities are likely to have a major positive impact on soccer match play. These improvements in endurance capacity are more pronounced than changes in the  $VO_2 \text{ max}$  values. This suggests that there are potentially greater performance improvements resulting from this type of training than the oxygen uptake values suggest. Therefore, limiting fitness assessment to  $VO_2 \text{ max}$  testing alone may under-estimate the impact of training per se.

The present study examined the validity of the MSFT (Ramsbottom, et al. 1988) for predicting  $VO_2 \text{ max}$  values in female subjects. Our post-training predicted  $VO_2 \text{ max}$  values and directly determined  $VO_2 \text{ max}$  values were  $50.3 \pm 0.6$  and  $51.5 \pm 0.9 \text{ ml.kg}^{-1}.\text{min}^{-1}$ , respectively. The limits of agreements between the two methods were  $\pm 5.0 \text{ ml.kg}^{-1}.\text{min}^{-1}$ . Therefore, the value an individual is assigned following the completion of the MSFT will fall within  $\pm 5.0 \text{ ml.kg}^{-1}.\text{min}^{-1}$  of their directly measured maximal oxygen uptake value. These findings support the work of Ramsbottom and co-workers (1988) who reported that maximal oxygen uptake values could be predicted from the progressive shuttle run test ( $r=0.92$ ;  $p < 0.01$ ) in men, with an estimated standard deviation about the regression line of  $3.5 \text{ ml.kg}^{-1}.\text{min}^{-1}$ . It appears that the MSFT is a reasonable predictor of  $VO_2 \text{ max}$ . Furthermore, it would appear from these findings that the MSFT test seems to be sensitive enough to detect changes in performance associated with soccer specific aerobic training.

This training resulted in significant improvements in vertical jump height. The TG improved their maximal squat jump by 5.6% ( $p < 0.01$ ). This equates to a 3 cm improvement (95% CI = 0.88-5.12). This jump test prohibits any countermovement and is commonly used to gain an indication of an individual's leg power (Sayers et al., 1999). Furthermore, the TG had improvements in the other two jump tests that involved

counter-movement. Interestingly, the 95% CI for this data suggests that this training protocol will almost certainly result in improvements to vertical jump performance and these could be as high as 5 cm in 6 weeks.

Helgerud et al (2003) showed similar improvements in jump performance (3cm or 5% improvement following 8 weeks training) using the same weight training protocol. Interestingly, our improvements occurred after only 6 weeks of strength training compared to the 8 weeks used by Helgerud and co-workers (2003).

The TG increased their body mass by 1 kg ( $p < 0.01$ ). This was the same relatively small increase as reported by Helgerud and colleagues (2003) using elite male soccer players. This finding further supports the suggestion that the strength and power gains achieved in the early stages of strength training are largely the result of neural adaptations rather than muscle hypertrophy (Sale, 1992; Hoff, 2001).

Despite, the improvement in jump performance following this training protocol no significant improvements were found in acceleration sprint performance over 5, 10, 15 and 20m. This finding differs from the results of Helgerud and co-workers (2002) who found a 3.3% and 1.6% significant improvement in 10m and 20m sprint times. It is possible that the physiological adaptations associated with 6 weeks of aerobic based interval training compromised the expected gains from the strength training. However, the TG subjects did improve vertical jump height, which suggests some noticeable physiological adaptations occurred that should benefit the production of force at speed. Another plausible explanation is that the specificity of the weight training techniques (i.e. heavy squat activity with maximum mobilisation during the upward phase) undoubtedly suited the vertical jump tests compared to acceleration sprints. The muscle actions required during the squat training closely mirrored the vertical jump performance test. Therefore, any neurological enhancements are likely to be more pronounced in a test that replicates the movement patterns of the training methods.

It should be noted that the performance improvements seen in this study have occurred even though the TG were already well trained. The elite players in the TG all completed three 1 and half hour technical and tactical training sessions plus a competitive match each week in the 6 months preceding the commencement of this training intervention.

Few studies have attempted to elicit changes in individuals already involved in frequent and well-structured training regimes (Billat et al., 2002; Helgerud et al., 2001; Helgerud et al., 2003; McMillan et al., 2005).

In summary, the two of the most commonly used fitness tests i.e. the MSFT (Ramsbottom et al, 1988) and the vertical jump tests, appear to be sensitive enough to detect performance changes that occur as a result of specific fitness training. Moreover, our results reconfirm that the MSFT can provide a valid predictor of  $VO_{2\text{ max}}$  in a field setting. It appears that six weeks of concurrent soccer specific aerobic endurance training and strength training can result in a number of major physical performance benefits to already well-trained elite female soccer players.

## CHAPTER 6

### **The effect of high carbohydrate meals with different glycaemic indices on recovery from high intensity intermittent shuttle running**

#### **6.1 Introduction**

The previous two investigations have highlighted the importance of fitness assessment within professional football. Whilst the task of improving players physical capabilities through regular training and assessment is of course an important one, this process is sometimes complicated by the presence of a number of matches in close succession. This is a scenario that is very common during the course of an professional football season in the UK and during this time the emphasis for the fitness practitioner becomes very much focused on the area of recovery.

One of the key factors in an effective recovery process is adequate nutrition. It is well established that consuming a high carbohydrate (CHO) diet during the recovery period following prolonged exercise increases the rate of muscle glycogen resynthesis compared to a normal mixed diet (Burke et al., 2004). Furthermore, endurance capacity during prolonged constant paced running (Fallowfield and Williams, 1997) and during prolonged intermittent high- intensity shuttle running is improved the day after prolonged exercise with high CHO recovery diets (9-10 g·kg<sup>-1</sup> BM) (Nicholas et al., 1997). Therefore it is common practice in many team sports to consume high CHO diets particularly during periods when training or competitive matches are being performed on consecutive days.

However, while the benefits of high CHO recovery diets are clearly recognised little attention has been given to the possible influences of the different types of carbohydrates on subsequent performance. Carbohydrates can be classified according to their postprandial glycemic response (Jenkins et al., 1981) and this criteria has been used to classify a large selection of foods (Foster-Powell et al., 2002). High glycemic CHO (HGI) foods are characterised by a rapid increase in blood glucose and accompanying rise in insulin concentrations. Glycogen storage is influenced both by insulin and an available supply of glucose as substrate. Therefore, CHO foods with

moderate to high glycemic index (GI) would be expected to enhance post-exercise refuelling of the body's limited CHO stores. Indeed, a study by Burke and colleagues reported *greater muscle glycogen concentrations 24 h after prolonged cycling when subjects consumed a HGI rather than a LGI CHO diet* (Burke et al., 1993). However, the authors did not appear to have assessed exercise performance following the HGI and LGI recovery diets.

Therefore, Stevenson and colleagues conducted a study to examine the influences of high and low GI diets on endurance running capacity after a recovery of 24 h (Stevenson et al., 2005). Their subjects ran longer on the LGI recovery diet than they did on the HGI recovery diet. Furthermore, the rate of fatty acid oxidation was also *greater during exercise after the LGI than after the HGI recovery diet*. This study used constant pace treadmill running and so contributes to the limited literature on carbohydrate diets and endurance running capacity. Nevertheless there is a dearth of literature on the possible influences of HGI and LGI recovery diets on subsequent exercise capacity during intermittent high-intensity running. Therefore, the purpose of the present study was to examine the effect of high CHO meals with different GI values on 22 h recovery from prolonged intermittent-high intensity shuttle running.



## 6.2 Methods

### 6.2.1 Participants

Seven male semi-professional soccer players participated in this study. Their mean ( $\pm$  SD) age, body mass (BM) and maximal oxygen uptake values were  $23 \pm 2$  years,  $73.7 \pm 9$  kg and  $58 \pm 0.8$  ml·kg<sup>-1</sup>·min<sup>-1</sup> respectively.

### 6.2.2 Preliminary measures

The subjects reported to the laboratory 7 - 10 days prior to their first main trial to complete a Progressive Multistage Shuttle Running Test (Ramsbottom et al., 1988) in order to estimate their maximal oxygen uptake ( $\text{VO}_{2 \text{ max}}$ ). Thereafter, the subjects completed a 45 min familiarisation with the LIST (Nicholas et al., 2000).

### 6.2.3 Experimental design

Each subject participated in two experimental trials separated by at least 7 -10 days. Each main trial was completed over a two-day period. During the two days immediately prior to the first main trial subjects recorded their food intake so that they could consume the same diet prior to the second main trial. On the morning of each main trial the subjects arrived at the laboratory in a fasted state where they provided a urine sample and then their nude body mass was obtained using a beam balance (3306 Avery, Birmingham, England). A cannula (Venflon 18G, Becton-Dickinson Ltd., Helsingborg, Sweden) was then inserted into an antecubital forearm vein and connected to a 3-way tap (Becton-Dickenson Ltd, Helsingborg, Sweden) with a 10cm extension tube for blood sampling. Heart rates were recorded using a short range telemetry system (Polar™ Electro, Kempele, Finland).

On Day 1 the subjects performed 90 min of an intermittent high-intensity shuttle running protocol (R1) (LIST) (Nicholas et al., 2000). This has been previously described (chapter 3). Following R1 they were given a diet that provided 8g CHO·kg BM<sup>-1</sup> for the 22 hr recovery. Meals and snacks were composed of either high (HGI) or low (LGI) GI carbohydrates in a randomised cross-over design (Table 1). On day 2,

subjects returned to the laboratory, again in a fasting state and performed 5 x 15 min of the LIST (R2 - Part A). This was followed by the following pattern of activity: two jogs (55%  $\text{VO}_2 \text{ max}$ ), one walk and one maximal sprint that were continued to the point of fatigue (R2 - Part B). Volitional fatigue was defined as the inability of the subjects to maintain the required pace or maintain consecutive sprints at times that were no less than 95% of their mean times in blocks 2 and 3 of the LIST during R2 - Part A.

On all occasions subjects were given  $5\text{ml}\cdot\text{kg}^{-1}$  BM of water before exercise and  $2\text{ml}\cdot\text{kg}^{-1}$  BM every 15 min during the 3 min rest periods.

#### **6.2.4 Test Meals**

Isocaloric recovery meals consisting of HGI or LGI CHO foods, as calculated by the method of Wolever et al (1986), were provided for each subject after R1 (Table 6.1). Breakfast was consumed 30 min following the completion of R1 and lunch was provided 3 h later. Both of these meals were prepared and consumed in the laboratory. Subjects were then provided with two snacks and an evening meal that was later consumed at home. Participants were asked to eat one snack between lunch and the evening meal (7pm) and were required to eat the final snack between 8-9pm. The diets were composed of predominantly HGI or LGI carbohydrates however, other foods (e.g. milk, cheese and lettuce) were included in both diets in order to make the meals more palatable. The amount of CHO in each of the two diets was calculated as available carbohydrates. Both diets consisted of 72% CHO, 11% fat and 17% protein. The nutritional content of each meal was calculated from the information provided by the manufacturer. The GI of the total mixed diets was calculated from the weighted means of the GI values for the component foods (Wolever and Jenkins, 1986). The calculated GI for the HGI and LGI diets was 70 and 35, respectively.

#### **6.2.5 Sample collection and analysis**

See general methods (chapter 3).

### 6.2.6 Statistical Procedures

An analysis of variance (ANOVA) with repeated measures on both factors (experimental treatment and time) was used to examine differences in the physiological and metabolic responses in R1 and R2 – Part A. However, due to variations in run times to exhaustion, a Student's paired T-test was used to analysis differences at the point of fatigue. The same analysis was carried out on all non-time dependant variables. Statistical significance was accepted at an alpha level of  $P < 0.05$ . All results are presented as mean  $\pm$  SEM. An effect size analyses based on a similar study (Stevenson et al., 2005), estimates that a sample size of 7 has 80% power to detect a difference in run times of 10 min.

## 6.3 Results

### 6.3.1 Run times

All subjects completed R1 and R2 but no differences were found in run-times to exhaustion between trials during Part B of R2 (HGI  $25.3 \pm 4.0$  min v. LGI  $22.9 \pm 5.6$  min  $P=0.649$ ). Neither were there differences between trials in the calculated fatigue index for sprint performance during Part B of R2. Furthermore, no differences were found in the number of sprints attempted or the total distance covered during Part B of R2 (HGI  $43 \pm 7$  v. LGI  $39 \pm 10$  and HGI  $3474 \pm 531$  m v. LGI  $3097 \pm 793$  m, respectively). A retrospective power test on our data showed that we can be 89% confident that any differences in run times between treatments would have been detected (if they existed) with the sample size used.

### 6.3.2 Plasma FFA and Glycerol

Plasma FFA and glycerol concentrations rose progressively during R1. Immediately post-exercise, FFA concentrations reached  $0.54 \pm 0.16$  and  $0.49 \pm 0.07$   $\text{mmol.l}^{-1}$  for HGI and LGI trials, respectively. During R2 there was a similar rise in plasma FFA (Fig. 6.1) and glycerol concentrations however there were no differences between HGI and LGI trials (Fig. 6.2).

### 6.3.3 Plasma Glucose and Serum Insulin Responses

Plasma glucose concentrations rose during first 30 min of exercise in both R1 and R2 for both conditions and no differences were observed between trials. Plasma glucose concentrations were maintained within a range of  $4 - 6$   $\text{mmol.l}^{-1}$  throughout all runs. At the point of fatigue during Part B of R2, plasma glucose concentrations were similar for both trials ( $5.01 \pm 0.56$  and  $4.97 \pm 0.57$   $\text{mmol.l}^{-1}$  in the HGI and LGI trials, respectively) (Fig. 6.3). Serum insulin concentrations decreased at the start of exercise from pre-exercise concentrations and continued to fall slowly throughout exercise in both trials (Fig. 6.4) ( $P<0.05$ ).

#### 6.3.4 Blood Lactate

During R1 and R2 there were no differences in blood lactate concentrations between trials (average concentrations during R1 were  $2.6 \pm 0.8$  mmol/L in the HGI trial and  $2.6 \pm 0.6$  mmol/L in the LGI trial and average concentrations during R2 were  $2.6 \pm 0.2$  mmol/L in the HGI trial and  $2.1 \pm 0.3$  mmol/L in the LGI trial). However, blood lactate concentrations were higher during R2 part B compared with R2 part A in both trials (average concentrations during R2 part A were  $2.6 \pm 0.7$  compared to R2 part B  $3.6 \pm 0.44$  mmol/L, ( $P < 0.05$ )).

#### 6.3.5 Heart Rate and Rating of Perceived Exertion (RPE)

Heart rate values were similar for both trials on Day 1 and Day 2 and were higher during Part B of R2 in both trials compared to earlier in exercise ( $P < 0.05$ ). Although there were no differences in RPE values between trials they were higher during Part B than Part A of the LIST ( $P < 0.05$ ).

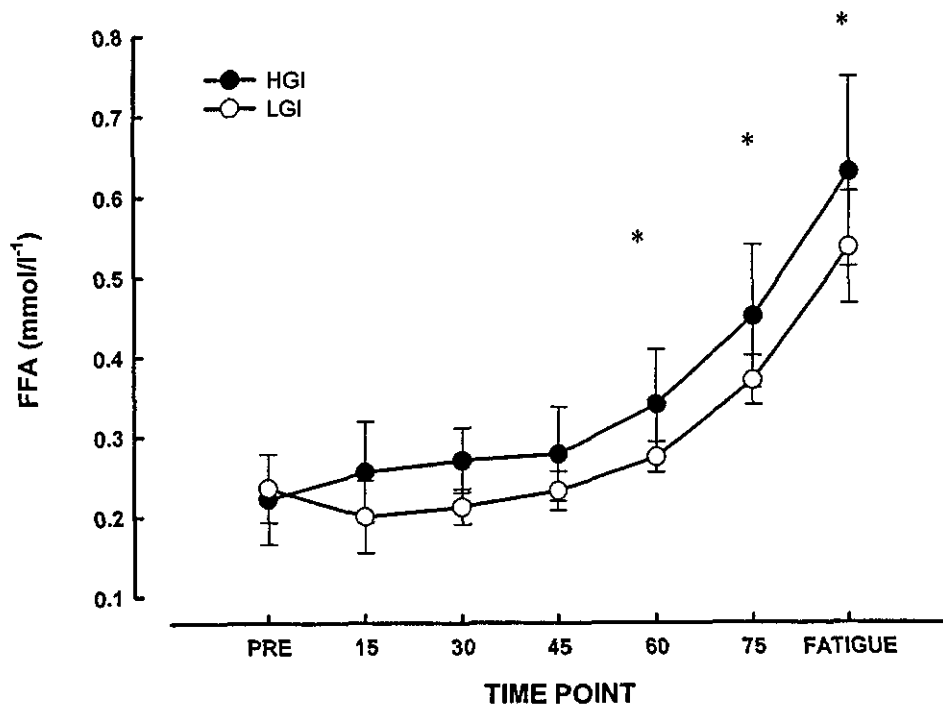
#### 6.3.6 Hydration Status

There was no difference in pre-exercise body mass before each trial. At the end of R1 in both trials subjects lost approximately 1% of their pre-exercise body mass and a similar percentage change in body mass occurred during R2. There were no significant differences in pre-exercise urine osmolality values between trials ( $640 \pm 243$  and  $684 \pm 231$  ml·Osmo·kg<sup>-1</sup> in the HGI and LGI trials, respectively).

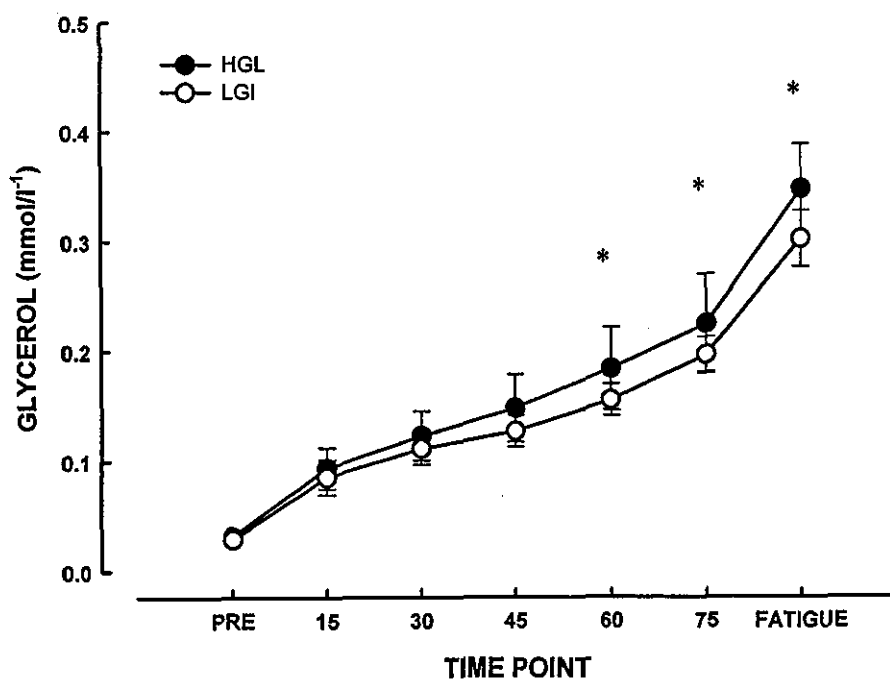
**Table 6.1 Characteristics of test meals (for a 70kg subject)**

(GI calculated by previously described method (Wolever et al., 1986) with GI values taken from Foster-Powell and co-workers (2002) Corn Flakes: Kellogg's (UK) Ltd. Manchester UK; Lucozade original drink UK)

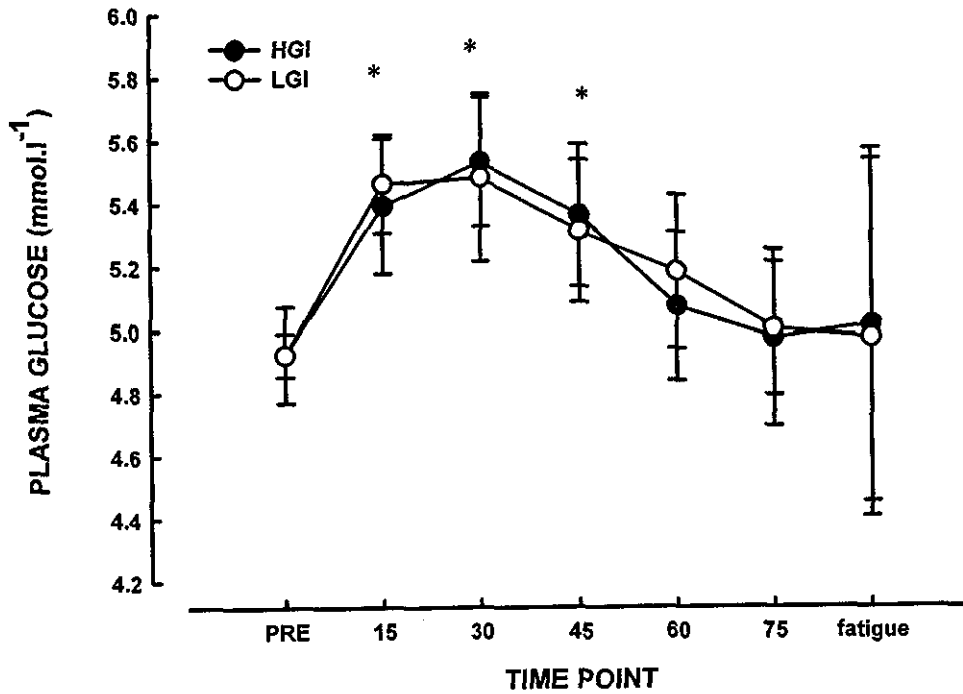
<b>Meal</b>	<b>Description</b>	<b>Macronutrient Content</b>
<b>HGI breakfast</b>	62g Corn Flakes# + 257ml skimmed milk 80g white bread + 10g flora + 20g jam 155ml Lucozade original	730 kcal 139g CHO, 9.9g fat, 20g protein
<b>LGI breakfast</b>	86g muesli + 257ml skimmed milk 67g apple, 103g tinned peaches, 128g yoghurt, 257 ml apple juice	732 kcal 139g CHO, 9g fat, 23g protein
<b>HGI lunch</b>	158g white bread, 154g turkey breast, 50g cheese, 40g lettuce, 180g banana, 200ml Lucozade original	1076 kcal 148g CHO, 24g fat, 63g protein
<b>LGI lunch</b>	154g whole wheat pasta, 150g turkey breast, 50g cheese, 40g lettuce, 185g pasta sauce, 150g pear, 150ml apple juice	1075 kcal 149g CHO, 25g fat, 60g protein
<b>HGI dinner</b>	255g baked potato, 410g tinned spaghetti 50g cheese, 40g lettuce, 67g mars bar, 170ml Lucozade original	1100 kcal 176g CHO, 31g fat, 28g protein
<b>LGI dinner</b>	360g chilli beans, 200g wheat tortilla, 50g cheese, 40g lettuce, 260ml orange juice	1100 kcal 176g CHO, 29g fat, 39g protein
<b>HGI snacks</b>	154g white bread, 40g jam, 20g flora	600 kcal 96g CHO, 17g fat, 15g protein
<b>LGI snacks</b>	170g yoghurt, 100g apple, 100g flapjack	625 kcal 97g CHO, 25g fat, 15g protein
<b>HGI totals</b>		3520 kcal. 560g CHO, 84g fat, 126g protein (72% CHO, 11% fat, 17% protein) <i>GI = 70*</i>
<b>LGI totals</b>		3600 kcal. 560g CHO, 88g fat, 135g protein (72% CHO, 11% fat, 17% protein) <i>GI = 35*</i>



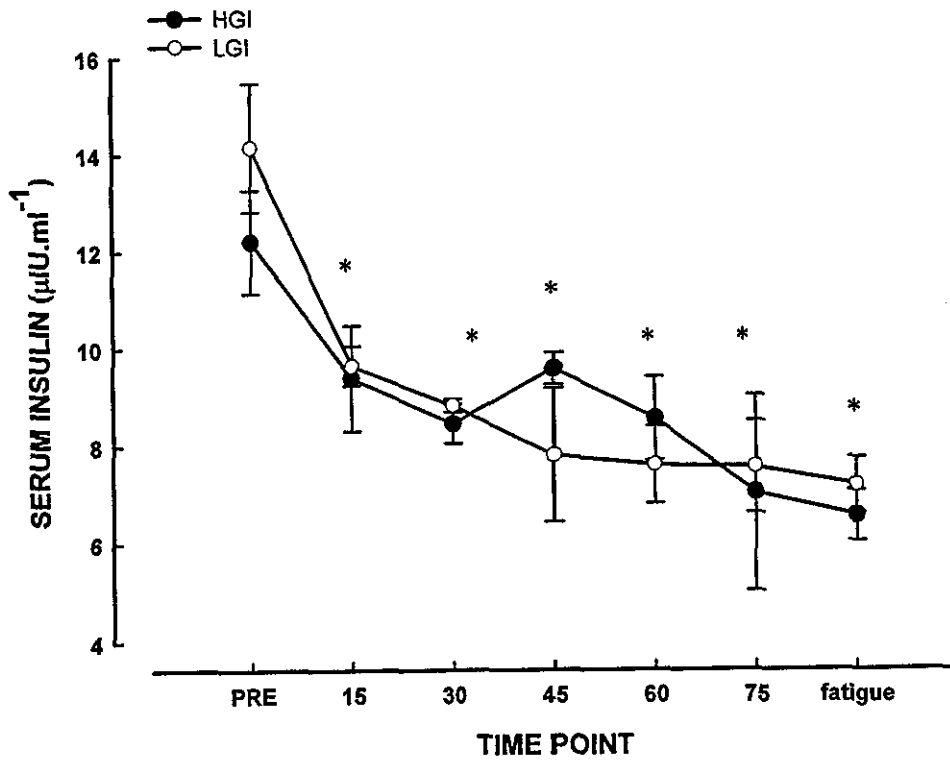
**Figure 6.1** Plasma FFA concentrations ( $\text{mmol}\cdot\text{l}^{-1}$ ) during R2 in the HGI and LGI trials (mean  $\pm$  SEM).  
 $P < 0.05$  \* different from pre-exercise for both groups.



**Figure 6.2** Plasma Glycerol concentrations ( $\text{mmol}\cdot\text{l}^{-1}$ ) during R2 in the HGI and LGI trials (mean  $\pm$  SEM).  
 $P < 0.05$  \* different from pre-exercise for both group



**Figure 6.3** Plasma glucose concentrations (mmol.l<sup>-1</sup>) during R2 in the HGI and LGI trials (mean ± SEM).  
 P<0.05 \* different from pre-exercise for both groups.



**Figure 6.4** Serum insulin concentrations (µU.ml<sup>-1</sup>) during R2 in the HGI and LGI trials (mean ± SEM).  
 P<0.05 \* different from pre-exercise for both groups.



## 6.4 Discussion

The main finding of the present study was that a HGI carbohydrate diet consumed during the 22hr recovery period following prolonged intermittent high-intensity shuttle running had no greater effect on sprint performance or endurance capacity the following day than a LGI carbohydrate recovery diet.

Prolonged intermittent high intensity exercise relies heavily on muscle glycogen as a substrate for the sustained high rate of ATP resynthesis (Balsom et al., 1999a; 1999b; Gaitanos et al., 1993; Nicholas et al., 1999; Parolin et al., 1999). During R2 Part A of the LIST consisted of five 15 min periods of exercise that includes 11 sprints. Therefore the subjects in the present study completed 55 maximal sprints during the first 75 min and then an additional 43 and 39 sprints during Part B of the LIST in the HGI and LGI trials, respectively. In an earlier study using the same intermittent exercise protocol we showed that when a high CHO recovery diet was consumed after performing the LIST to fatigue, endurance capacity was restored 22 h later whereas that was not the case after an isocaloric mixed diet (Nicholas et al., 1999). After the high CHO recovery diet the subjects ran for 3.3 min longer than they did on the mixed diet. In contrast, when the subjects consumed the isocaloric mixed diet that included their normal intake of CHO during the 22 h recovery, they ran for 2 min less than on the previous day (Nicholas et al., 1999). Therefore it is clear from this earlier study that a high CHO recovery diet improves recovery from prolonged intermittent high intensity exercise.

Burke and colleagues reported that a HGI recovery diet resulted in a 48% greater muscle glycogen concentration 24 h after prolonged cycling than was achieved when their subjects consumed a LGI diet (Burke et al., 1993). However, they did not assess the exercise performance of their subjects following the 24 h recovery. Nevertheless, it would be reasonable to speculate that their subjects would have produced better performances after the HGI than after the LGI recovery diet because they would have started exercise with higher muscle glycogen stores (Burke et al., 1993). Therefore, it was surprising to find that there was no difference in performance after the HGI and LGI recovery diets in the present study.

In a previous study we found a greater endurance capacity 22 h after prolonged treadmill running when runners consumed a LGI CHO recovery diet than when the diet was composed of HGI CHO (Stevenson et al., 2005). After completing a 90 min treadmill run at 70%  $\text{VO}_{2\text{max}}$  the runners were randomly assigned to either the HGI or LGI recovery diet. After an overnight fast they returned to the laboratory and ran to exhaustion at the same intensity as on the previous day. The mean run time to exhaustion after the LGI recovery diet was 12 min longer than after the HGI diet (108.9 vs 96.9 min) (Stevenson et al., 2005). Even though runners probably began the second run with higher muscle glycogen stores after the HGI recovery diet (Burke et al., 1993; Stevenson et al., 2005; Wee et al., 2005) it is likely that they would have used more glycogen during the run than when they had consumed the LGI recovery diet (MacConell et al., 1999). It is possible, though only speculation that after an hour or so of running the muscle glycogen stores may have been similar following the two dietary conditions even though the rates of glycogenolysis were initially different. However the greater rate of fat oxidation during the second run in the LGI trial may have been able to cover the energy production deficit as muscle glycogen concentrations decreased (Zderic et al., 2004) more completely than in the HGI trial. A compensatory up-regulation of fat oxidation in skeletal muscle may have reduced the need for an increased contribution from blood glucose to cover energy production in the LGI trial but not in the HGI trial. The differences in the demand for an increased turnover of blood glucose towards the end of exercise may also have contributed to the differences in the times to fatigue (Claassen et al., 2005).

A further speculation is that during intermittent high intensity exercise, in which the subjects perform 11 maximum sprints every 15 min block, the high rates of glycogenolysis (Gaitanos et al 1993) will have increased both the lactate and hydrogen ion concentrations. During high intensity exercise there is a decrease in fatty acid oxidation rates that appears to be a result of an inhibition of the transfer of fatty acids through the mitochondrial membrane possibly because of changes in the carnitine palmityl transferase (CPT1) (Achten and Jeuckendrup 2003). If this was the case then the potential benefits of increased fatty acid oxidation following the LGI diet may have been negated as a consequence of the repeated periods increased glycogenolysis-induced rise in intra-muscular hydrogen concentrations.

The lack of differences in sprint performance and endurance capacity in the present study after the LGI and HGI recovery diets is difficult to explain when it appears that the subjects on the HGI recovery diet probably began exercise with higher muscle glycogen concentrations. One explanation might be that following the HGI recovery diet the subjects began intermittent exercise using more CHO than fat because of its greater availability (Roepstorff et al., 2005), whereas after the LGI recovery diet they used more fat and less CHO. The run-walk-sprint nature of Part A of the LIST is conducive to fat metabolism during the low intensity phase of each 15 min block of activity. Although the rates of glycogenolysis during Part A of the LIST may have been different after the HGI and LGI recovery diets, the glycogen stores may have been reduced to similar values at the end of the 75 min of exercise. If this was the case then the subjects would have started Part B with similar muscle glycogen concentrations and as the results show, their run times to fatigue were also similar.

Another consideration is that this intermittent high intensity shuttle running protocol produces significant post-exercise muscle soreness (Thompson et al., 1999) because of the eccentric muscle contractions during the frequent changes in speed and direction. Extensive eccentric muscle contractions have been shown to decrease the rate of glycogen resynthesis (Doyle et al., 1993) and lead to an increased the rate of glycogen utilisation during subsequent exercise (Asp et al., 1998). If this were the case in the present study then it would be reasonable to expect poorer sprint performance and endurance capacity after the LGI recovery diet because the lower recovery rate of muscle glycogen resynthesis would have been exacerbated by the eccentric nature of the prior exercise i.e. R1. However, the absence of a difference in sprint performance and endurance capacity between the two trials suggests that fatigue during Part B may not have been entirely due to differences in pre-exercise glycogen concentrations.

Fatigue during Part B may have been in part due to low muscle glycogen stores and the inability to resynthesis phosphocreatine (PCr) rapidly enough to maintain ATP turnover rate at the required level (Bogdanis et al., 1996; Gaitanos et al., 1993). In addition, the accumulation of metabolites such as hydrogen ions, ADP, AMP, inorganic phosphate and magnesium may have created a cellular environment that contributed to the inability of the working muscles to sustain energy production (Sahlin., 1998).

In summary, the main finding of this study was that the type of CHO (high or low GI) provided as part of a recovery diet did not influence performance during subsequent prolonged high intensity intermittent shuttle running. Carbohydrate rather than fat would have been the main fuel during this high intensity running protocol and therefore the potential benefit of a recovery diet that promotes fat oxidation during continuous running would have had little impact during this type of exercise.

## CHAPTER 7

### **The Influence of cold-water immersion on the indices of muscle damage following prolonged high intensity intermittent exercise**

#### **7.1 Introduction**

Following prolonged high intensity intermittent exercise an individual may not only be faced with the problem of depleted energy stores but also the deleterious effects associated with muscle damage. These effects that often follow a bout of unaccustomed or eccentric based exercise are well documented. The time course and severity of muscle soreness, muscular dysfunction and appearance of markers of muscle damage in the systemic circulation may vary considerably depending of the duration, intensity and type of exercise performed (Clarkson et al., 1986; Eston et al., 1994; Thompson et al., 1999). Perhaps these factors in part explain why the precise aetiology of exercise-induced muscle damage remains elusive. Nevertheless, delayed-onset muscle soreness (DOMS) and associated decrements in muscular function are still one of the most commonly reported sport related injuries (Cheung et al., 2003).

Many investigations have attempted to alleviate or prevent exercise-induced muscle damage and its associated symptoms. Treatment strategies include stretching, ultrasound, massage, antioxidant supplementation and administration of non-steroidal anti-inflammatory drugs (for a review see Cheung et al., 2003). More recently attention has focused on the effect of cryotherapy in aiding recovery from muscle damaging exercise. The role of cryotherapy as a treatment of sports related injuries is well documented (Bleakley et al., 2004) although support for its specific application to exercise-induced muscle damage is predominantly anecdotal.

Cryotherapy is proposed to reduce the inflammatory response to injured tissue as well as decrease oedema, haematoma formation and pain (Swenson et al., 1996). Thus, cryotherapy may be considered a pertinent treatment modality as inflammation is integral in the aetiology of exercise-induced muscle damage and muscle soreness is the most commonly reported symptom of this exercise related injury (Smith, 1991). Additionally, inflammation has been shown to exacerbate existing disruption to skeletal muscle tissue as this immune response is coupled with secondary damage via transient

hypoxia as well as the non-specific cytotoxic actions of leukocytes (MacIntyre et al., 1996; Lapointe et al., 2002).

Prentice and co-workers (1982) were among the first to identify beneficial effects of cryotherapy, in combination with static stretching, on indices of muscle damage following exercise. Individuals that received cryotherapy and stretching had a reduction in EMG signal 24 hours following strenuous knee extension/flexion exercise. As EMG was the only dependant variable used to assess muscle damage it is difficult to clarify the precise mechanism responsible for the effects of this treatment strategy.

More recently, research has focused on the role of ice massage or ice water immersion on indices of muscle damage. Eston and Peters (1999) found ice water immersion (15 min at 15 °C) was effective in reducing plasma creatine kinase activity and muscle stiffness in the days following eccentric exercise. Howatson and van Someren (2003) reported similar positive effects on creatine kinase activity with repeated ice-massage immediately following, 24 and 48 h post eccentric exercise. Conversely, Isabell and co-workers (1992) observed no effect of ice massage on indices of muscle damage and suggested repeated cryotherapy maybe contra-indicatory over a period of 4 days.

As the physiological effects of cryotherapy are inversely related to the proposed aetiology of exercise-induced muscle damage there is an obvious rationale for the use of this prophylactic treatment. To this end the aim of the current investigation was to elucidate the potential beneficial role that cryotherapy may have in aiding recovery from a bout of strenuous, muscle damaging exercise.

## 7.2 Methods

### 7.2.1 Participants

Twenty healthy males volunteered to take part in this study, which had received approval from the University Ethical Advisory Committee. All participants were semi-professional soccer or rugby players.

### 7.2.2 Experimental Design

Having abstained from exercise for at least two days subjects arrived in the laboratory in the fasted state (10-12 h). A venous blood sample (~10 ml) was taken from a vein in the antecubital fossa after the subject had been supine for at least 10 min. Following this perceived muscle soreness was recorded and muscular function was assessed using an isokinetic dynamometer and a vertical jump height test described in detail below. Subsequently, subjects completed the Loughborough Intermittent Shuttle Test (LIST) as described previously. Deep body temperature was monitored using an ingestible thermometer pill (CorTemp™, HQI, Palmetto, USA) and a data recorder at regular intervals. Participants were given a specific health questionnaire (Appendix B) and information sheet (Appendix E) relating to the usage of this type of ingestible temperature pill. Subjects were required to ingest a predetermined volume of water during exercise equal to 5ml.kg<sup>-1</sup> pre-exercise, and 2ml.kg<sup>-1</sup> following each 15 min block. This has previously been shown to maintain euhydration ( $\pm$  1.5% body mass) during intermittent exercise (McGregor et al., 1999). Nude body mass was determined immediately before and after exercise. A venous blood sample was taken immediately post-exercise and additional samples were taken 1, 24 and 48 h after exercise. Subjects were instructed not to resume exercising until the conclusion of testing.

### 7.2.3 Cryotherapy treatment

Immediately following exercise subjects were randomly allocated to either the cryotherapy or control group matched for a number of physiological characteristics as well as physical activity status (Table 7.1). The cryotherapy group immersed their lower limbs (ensuring that the iliac crest was fully submerged) in an ice water bath for 10

minutes. The water was maintained at a temperature of  $10 \pm 1^\circ\text{C}$  by the addition of crushed ice and was repeatedly agitated to avoid the formation of a warmer boundary layer. During this period control subjects remained at rest in the same body position as cryotherapy subjects. Heart rate and deep body temperature were monitored at regular intervals throughout treatment and up to 15 min post-cryotherapy. Additionally, ratings of perceived coldness were assessed during treatment and recovery using a modified CR-10 scale (Borg, 1982) which ranged from 1 “not cold” to 10 “very, very cold” (Appendix I).

**Table 7.1:** Physiological characteristics and physical activity status of groups (mean  $\pm$  SD). \* sum of 4 skinfolds (*triceps, biceps, suprailiac, subscapular*).

	Treatment (n=10)	Control (n=10)	P-value
Age (years)	23.6 $\pm$ 4.1	21.7 $\pm$ 2.0	P = 0.123
Height (m)	1.80 $\pm$ 0.06	1.81 $\pm$ 0.05	P = 0.665
Mass (kg)	85.9 $\pm$ 12.8	81.5 $\pm$ 11.2	P = 0.517
BMI (kg.m <sup>-2</sup> )	26.3 $\pm$ 2.8	24.9 $\pm$ 2.7	P = 0.487
Sum Skinfolds* (mm)	35.3 $\pm$ 12.8	31.3 $\pm$ 6.3	P = 0.583
VO <sub>2</sub> max (ml.kg <sup>-1</sup> .min <sup>-1</sup> )	55.2 $\pm$ 4.8	56.2 $\pm$ 5.3	P = 0.676
Weekly exercise session	5 $\pm$ 2	4 $\pm$ 1	P = 0.265

#### 7.2.4 Assessment of muscle damage

Ratings of perceived soreness were assessed using a visual analog scale (Appendix H) ranging from 1 “not sore” to 10 “very, very, sore” (Thompson et al., 1999) pre-exercise, immediately-post-exercise and again at 1, 24, 48 and 168 h.

Maximal voluntary isometric contraction (MVC) was assessed using an isokinetic dynamometer (Cybex model 770, LUMEX Inc., USA). Subjects were familiarised with the apparatus and protocol on at least two occasions prior to participation in the LIST. Following a warm-up set of five sub-maximal repetitions of knee extension and flexion (1.05 rad.s<sup>-1</sup>) subjects completed two repetitions of isometric knee extension and flexion on their dominant limb for 5 s at 1.05 rad and 0.35 rad from an established reference point (determined from maximum knee extension in all subjects), respectively.



Contractions were separated by 60 s rest periods and subjects were verbally encouraged and received visual feedback during each repetition. The greatest peak torque achieved from both repetitions was recorded.

Vertical jump height was recorded as previously described (Byrne and Eston, 2002). Subjects performed the squat jump technique with no countermovement in order to minimise the effects of the stretch-shortening cycle. Jumps were performed on an electronic timing mat (Powertimer 1.0 Testing System, Newtest OY, Finland) which is triggered by the feet of the participant at take-off and again upon landing. The height of each jump is calculated using the flight time, vertical take-off velocity of the centre of mass and acceleration due to gravity. (Byrne and Eston, 2002) Subjects performed three consecutive jumps on each occasion. Jumps were separated by 60 s rest and the greatest jump was taken as the peak height.

Sprint performance was assessed both during the LIST and again 48 h post-exercise. Sprint times were measured using two infra-red photoelectric cells (RS Components Ltd, Switzerland) interfaced with the computer. Subjects were required to perform 11 x 15 m maximal sprints during each block of the LIST. The values recorded during the first block of the LIST were compared to a subsequent 15 min block performed 48 h following the initial exercise bout.

#### 7.2.5 Blood Analysis

Aliquots of blood were used to determine haemoglobin concentrations by the cyanomethemoglobin method (Boehringer Mannheim, GmbH Diagnostica, Germany) and haematocrit by micro-centrifugation (Hawksley Ltd, Lancing, UK). Changes in plasma volume were assessed using haematocrit and haemoglobin values. (Dill and Costill, 1974) The remaining blood was dispensed into a tube and left to clot and then centrifuged (4°C) at 4000 rev·min<sup>-1</sup> for 10 min to obtain serum. Serum creatine kinase activity (CK) was determined at 37°C using a commercially available spectrophotometric technique (Randox, UK) designed specifically for use on an automated system (COBAS Mira Plus, Roche Diagnostics Systems, Switzerland). Serum myoglobin concentrations (Mb) were measured with an immunoturbidimetric assay also specifically developed for the automated system (Randox, UK).

### 7.2.6 Statistical analysis

An independent two-way analysis of variance (ANOVA) with repeated measures was used to determine if any differences existed between treatment and time during exercise and recovery. The Greenhouse-Geisser correction was utilised for epsilon <0.75, while the Huynh-Feldt correction was adopted for less severe asphericity. When significant F values were found, the Holm-Bonferroni step-wise method was utilised to determine the location of the variance.(Atkinson, 2002) Values for creatine kinase activity and myoglobin were not normally distributed and therefore these values were log transformed prior to ANOVA. Log transformation always resulted in normal distribution and therefore these ANOVA results are reported. Pearson product moment correlations were used to examine the relationship between variables. Data analysis was conducted using SPSS version 11 and significance was accepted at the 5% level. Values are expressed as the mean  $\pm$  standard error of the mean (*SEM*) throughout.

## 7.3 Results

### 7.3.1 Response to intermittent exercise

Mean heart rate during the LIST was  $165 \pm 3$  b $\cdot$ min<sup>-1</sup> for both groups. Ratings of perceived exertion increased from  $14 \pm 1$  at the end of the first block to  $17 \pm 1$  for both groups ( $F_{3,47} = 3.99$   $P < 0.001$ ). Deep body temperature during exercise was available for 15 subjects (cryotherapy  $n = 8$ , control  $n = 7$ ). Temperature increased from  $37.49 \pm 0.10^\circ\text{C}$  to  $38.06 \pm 0.13^\circ\text{C}$  following 90 min ( $F_{1,17} = 48.7$   $P < 0.001$ ). During exercise participants drank  $1.26 \pm 0.06$  litres of water, losing  $1.14 \pm 0.25$  kg in body mass. Mean sprint time during the LIST was  $2.70 \pm 0.03$  s. Estimated changes in plasma volume did not differ during the testing period for either group.

### 7.3.2 Response to cryotherapy treatment

Heart rate decreased during treatment from  $107 \pm 4$  b $\cdot$ min<sup>-1</sup> to  $94 \pm 3$  b $\cdot$ min<sup>-1</sup> ( $F_{4,67} = 28.37$   $P < 0.001$ ) and continued to decline 15 min following cryotherapy to  $87 \pm 3$  b $\cdot$ min<sup>-1</sup> ( $F_{4,67} = 28.37$   $P < 0.001$ ). Cryotherapy had no effect on heart rate response compared to the control group ( $F_{2,40} = 2.62$   $P < 0.09$ ). Deep body temperature ( $n = 15$ ) decreased from  $37.94 \pm 0.14^\circ\text{C}$  to  $37.67 \pm 0.13^\circ\text{C}$  during the treatment period and continued to fall 15 min post-treatment  $37.41 \pm 0.11^\circ\text{C}$  ( $F_{1,19} = 45.31$   $P < 0.001$ ). Cryotherapy had no effect on deep body temperature compared to the control group. Perception of coldness was elevated during treatment in the cryotherapy group ( $6 \pm 1$ ) compared to the control group ( $1 \pm 1$ ) and remained elevated during recovery ( $F_{3,40} = 13.84$   $P < 0.001$ ).

### 7.3.3 Indices of muscle damage

Exercise resulted in severe muscle soreness that peaked immediately post-exercise and again 24 h later ( $F_{3,49} = 60.7$   $P < 0.001$ ). Cryotherapy treatment reduced ratings of perceived soreness 1, 24 and 48 h post-exercise ( $F_{3,49} = 2.8$   $P = 0.05$ ) (Figure 7.1).

Maximal isometric voluntary contraction for leg extension was unaffected following exercise and treatment (Table 7.2). However, MVC for leg flexion was reduced 24 and 48 h post-exercise ( $F_{3,54} = 3.89$   $P < 0.01$ ) and returned to pre-exercise values 168 h post-

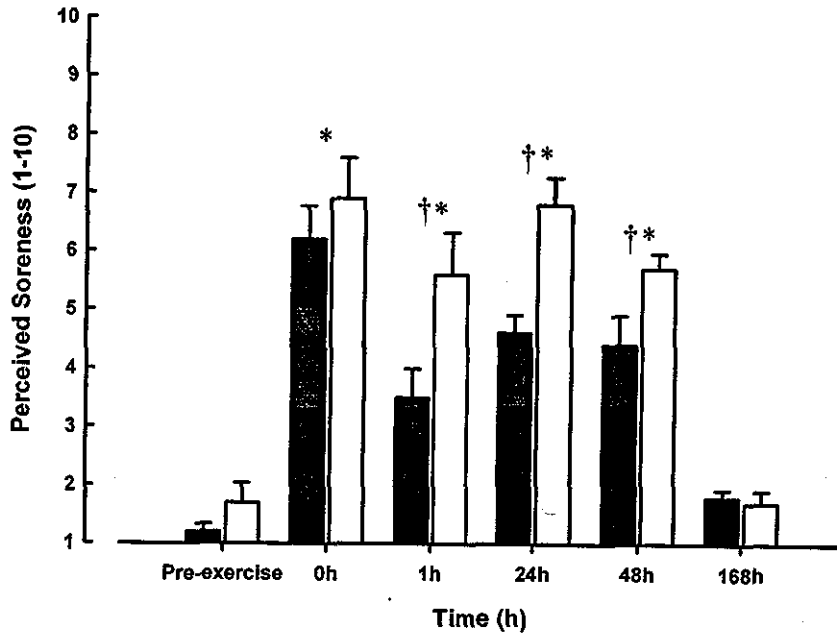
exercise ( $P \leq 0.05$ ). Cryotherapy reduced decrements in MVC at 24 and 48 h compared to the control condition ( $P \leq 0.05$ ) (Table 7.2).

**Table 7.2:** Isometric maximal voluntary contraction of the leg flexors and extensors following intermittent shuttle running and treatment (means  $\pm$  SEM). † values are different between groups ( $P \leq 0.05$ ). \* values are different from pre-exercise ( $P \leq 0.05$ ).

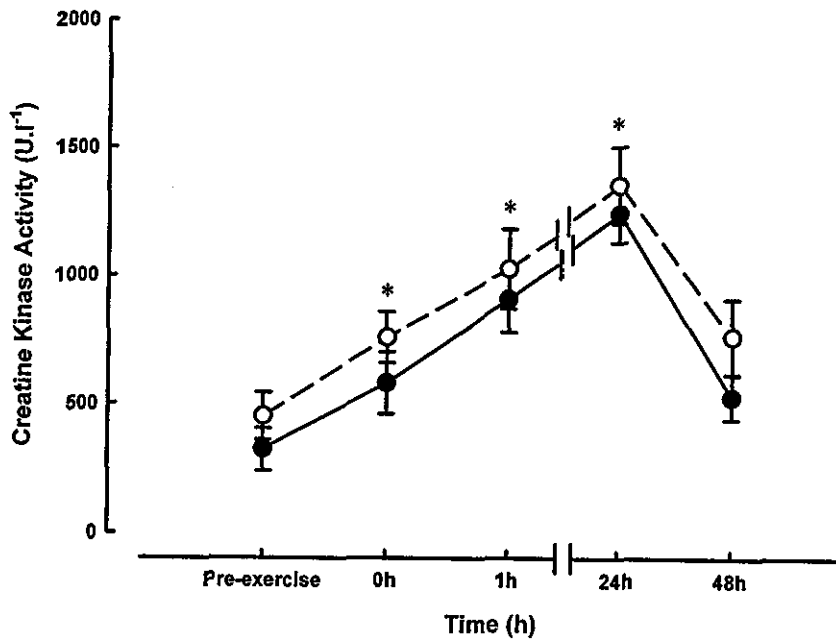
MVC (Nm.kg <sup>-1</sup> )	Pre-exercise	24h	48h	168h
<b>Flexion</b>				
<b>Cryotherapy</b>	2.80 $\pm$ 0.12	2.46 $\pm$ 0.13 *†	2.71 $\pm$ 0.15 *†	2.76 $\pm$ 0.09
(% pre-exercise)	-	(88 $\pm$ 4)	(97 $\pm$ 3)	(99 $\pm$ 3)
<b>Control</b>	2.53 $\pm$ 0.14	2.00 $\pm$ 0.15 *	2.15 $\pm$ 0.15 *	2.48 $\pm$ 0.15
(% pre-exercise)	-	(79 $\pm$ 5)	(86 $\pm$ 5)	(99 $\pm$ 2)
<b>Extension</b>				
<b>Cryotherapy</b>	4.97 $\pm$ 0.24	4.96 $\pm$ 0.26	4.68 $\pm$ 0.28	4.78 $\pm$ 0.23
(% pre-exercise)	-	(94 $\pm$ 3)	(94 $\pm$ 3)	(97 $\pm$ 3)
<b>Control</b>	4.23 $\pm$ 0.27	3.84 $\pm$ 0.37	4.29 $\pm$ 0.39	4.47 $\pm$ 0.31
(% pre-exercise)	-	(91 $\pm$ 7)	(101 $\pm$ 6)	(106 $\pm$ 4)

Peak vertical jump height was reduced from pre-exercise (0.36  $\pm$  0.01 m) at 24 (0.35  $\pm$  0.01 m) and 48 h (0.34  $\pm$  0.01 m) for both groups ( $F_{3,51} = 7.47$   $P < 0.001$ ). Vertical jump height was unaffected by treatment. Mean sprint times for 11 x 15 m sprints were unaffected 48 h following exercise and treatment.

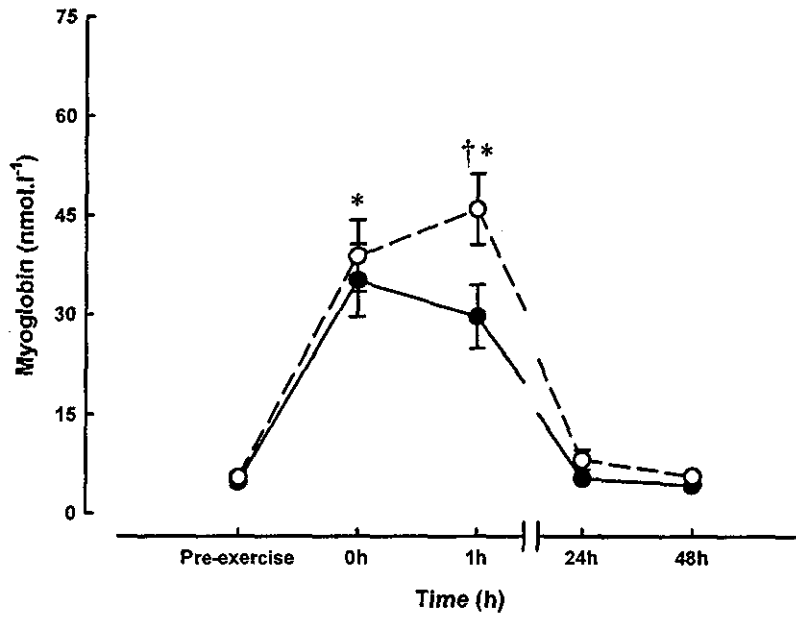
Creatine kinase activity was elevated immediately post-exercise ( $F_{2,38} = 26.21$   $P < 0.001$ ) peaking 24 h later but this response was not influenced by cryotherapy ( $F_{2,42} = 0.84$   $P = 0.45$ ) (Figure 2). Myoglobin concentration increased immediately post-exercise in both groups ( $P \leq 0.05$ ). Concentrations peaked 1 h post-exercise in the control group but were reduced at this time in the cryotherapy group ( $P \leq 0.05$ ) (Figure 7.3).



**Figure 7.1: Perceived muscle soreness following exercise for cryotherapy (solid bars) and control (clear bars) groups. Values are mean  $\pm$  S.E.M.**  
 \*  $P \leq 0.05$  different from pre-exercise for both groups, †  $P \leq 0.05$  different between groups.



**Figure 7.2: Serum creatine kinase activity following exercise for cryotherapy (solid line) and control (broken line) groups. Values are mean  $\pm$  S.E.M.**  
 \*  $P \leq 0.05$  different from pre-exercise for both groups.



**Figure 7.3: Serum myoglobin concentration following exercise for cryotherapy (solid line) and control (broken line) groups. Values are mean  $\pm$  S.E.M. \*  $P \leq 0.05$  different from pre-exercise for both groups,  $\dagger P \leq 0.05$  different between groups.**

## 7.4 Discussion

The objective of this investigation was to corroborate the efficacy of cryotherapy as a treatment to alleviate symptoms of exercise-induced muscle damage. The main findings provide support for this proposal as individuals that received cryotherapy treatment following exercise reported a reduced perception of muscle soreness, lower decrements in torque during MVC and a reduced serum myoglobin response post-exercise. These findings are inline with similar investigations employing cryotherapy as a modality to treat exercise-induced muscle damage (Eston and Peters, 1999; Howatson and Van Someren, 2003; Yanagisawa et al., 2003).

The intermittent shuttle-running protocol used to elicit muscle damage resulted in severe muscle soreness and an associated period of muscular dysfunction comparable to that previously documented (Thompson et al., 1999; Bailey et al., 2002). Additionally, myofibrillar protein appearance was increased in the hours and days following exercise to similar levels and over the same time course as observed in investigations employing both this exercise protocol (Thompson et al., 1999; Bailey et al., 2002) and other analogous eccentric based exercise models (Byrnes et al., 1985; Thompson et al., 2004). The variability in the efflux of these proteins into the systemic circulation has been attributed to their relative molecular mass ( $M_b = 17,800$  versus  $CK = 80,000$  Da) (Cairns et al., 1983) which may explain the more rapid appearance of myoglobin post-exercise. Soreness was most frequently reported in the musculature of the lower limbs and was greatest in the hamstrings, conceivably related to the eccentric actions of this muscle group during intermittent running exercise (Thompson et al., 1999). The moderate relationship ( $r = -0.58$ ,  $P \leq 0.05$ ) between decrements in MVC of the leg flexors and muscle soreness 48 h post-exercise provides some support for the proposed association between muscle injury, dysfunction and soreness that is not well documented (Warren et al. 1999; Nosaka et al., 2002).

The initial increase in muscle soreness observed immediately following exercise is often referred to as acute onset muscle soreness. This soreness is related to the accumulation of by-products that are either metabolic or contraction induced (Miles and Clarkson, 1994) rather than DOMS which is more commonly associated with muscle damage (Cheung et al., 2003). This may account for the biphasic increase in muscle soreness

observed following exercise and support the proposal that cryotherapy was effective in reducing muscle injury rather than facilitating removal of exercise-induced accumulation of by-products. The observed reductions in DOMS 24 and 48h post-exercise with cryotherapy is in line with similar investigations (Prentice, 1982; Denegar and Perrin, 1992; Yanagisawa et al., 2003). However, some attribute this reduced pain perception to the analgesic effects of cooling rather than inhibition of muscle damage (Meeusen and Lievens, 1986; Denegar and Perrin, 1992; Gulick, et al., 1996). The application of cold, sufficient to lower muscle tissue to temperatures around 10-15°C, reduces nerve conduction velocity, muscle spindle activity, the stretch-reflex response and spasticity thus inhibiting the pain-spasm cycle (Meeusen and Lievens, 1986). However, the duration of this analgesia is limited (1-3 h) (Meeusen and Lievens, 1986) so this mechanism may only account for the initial reductions in muscle soreness observed 1 h post-exercise. Denegar and Perrin (1992) observed similar beneficial effects of cryotherapy (ice packs) on DOMS. These authors documented a further reduction in perceived soreness when the treatment was supplemented with a period of stretching. They proposed that stretching results in stimulation of the Golgi tendon organ, motor inhibition and reduced muscular tension resulting in a concomitant reduced in the pain-spasm cycle (Denegar and Perrin, 1992). Although cooling, either independent or accompanied by passive stretching, has inhibitory influences on pain perception some investigations reporting prophylactic effects of cryotherapy on exercise-induced muscle damage have failed to observe a concomitant effect on muscle soreness (Eston and Peters 1999; Howatson and Van Someren 2003).

Cryotherapy also improved recovery of peak torque during MVC of the knee flexors 24-48h post-exercise. Exercise resulted in a reduction of knee flexion peak torque at 24 ( $12 \pm 4\%$ ) and 48 h ( $3 \pm 3\%$ ) in the cryotherapy group which was markedly less than that experienced by the control group at 24 ( $21 \pm 5\%$ ) and 48 h ( $14 \pm 5\%$ ). Values had returned to those achieved pre-exercise by 7 days post-exercise in both groups. This pattern of strength loss and recovery is similar to that previously reported following this exercise protocol (Thompson et al., 1999; Bailey et al., 2002; Thompson et al., 2003), although decrements were lower compared with previous findings (Bailey et al., 2002; Thompson et al., 2003). This variation could be attributed to the training status of subjects, as the individuals that participated in this investigation were predominantly intermittent games players and thus less susceptible to muscle damage due to the well



documented adaptation to repeated exposure to damaging exercise (Ebbeling and Clarkson, 1989). This may also account for the minimal effects of exercise on peak torque MVC of the knee extensors as well as sprint performance and vertical jump height. Nevertheless, the decrements that occurred in the knee flexors is more likely a result of the large forces exerted on this muscle group during deceleration and the critical antagonist action of this muscle group during maximal sprinting whilst performing this intermittent exercise protocol. In assessing muscle function as an index of exercise-induced muscle damage the recommendations of Warren and co-workers (1999) are further corroborated although their avocation of specificity when assesses muscle injury following running exercise is not supported as the more sensitive assessment of peak torque during MVC was more successful in identifying changes in muscle function compared to the more specific sprint and jump tests.

The effects of cryotherapy on the appearance of myofibrillar proteins are in agreement with previous investigations (Eston and Peters, 1999; Howatson and Van Someren, 2003). It is still unclear what mechanism is responsible for the difference in myoglobin concentration following cryotherapy treatment. Others have postulated that cryotherapy may reduce post-exercise muscle damage via a decreased permeability of blood and lymph vessels as a result of an attenuation of the inflammatory response. These investigations employed creatine kinase activity as the sole marker for myofibrillar protein release (Eston and Peters, 1999; Howatson and Van Someren, 2003). This particular marker is subject to large variability between individuals and caution is advised when interpreting the response of this particular myofibrillar protein (Clarkson and Ebbeling, 1988; Warren et al., 1999). This explanation may, in part, account for the lack of a treatment effect observed with creatine kinase activity. Also, as secondary damage to skeletal muscle resulting from inflammation may be more pronounced in the hours rather than days post-exercise it is possible that myoglobin is a more accurate indicator of subsequent injury (Lapointe et al., 2002). Although cryotherapy treatment had no effect on deep body temperature compared to the control group, where cooling rates were  $0.029 \pm 0.005$  and  $0.028 \pm 0.005$  °C.min<sup>-1</sup> for treatment and control groups respectively, previously investigation have reported reductions in subcutaneous and intramuscular temperatures during similar cryotherapy treatments (for a review see Meeusen and Lievens, 1986). Therefore, it is reasonable to assume that ice water immersion in this investigation was effective in lower intramuscular temperature. With

this in mind, it is possible that cryotherapy mediated a reduced inflammatory response and subsequent secondary muscle damage attenuating the efflux of myoglobin.

Some authors recommend more prolonged treatment in order to maximise any advantageous properties of cryotherapy and argue that a greater treatment time may have more profound effects on lowering intramuscular temperature (Meeusen and Lievens, 1986). Paddon-Jones and Quigley (1997) administered repeated cold water immersions (5 x 20 min) separated by 60 minute rest periods following eccentric elbow extension exercise. However, these authors do not observe any effect of cryotherapy on muscle soreness, isometric torque and limb volume. Yanagisawa and colleagues (2003) observed no additional effect of repeated cryotherapy compared to a single application following eccentric exercise. Although cold-water immersion immediately post eccentric exercise reduced muscle soreness and swelling there was no additive effect when the same treatment was repeated 24 h later (Yanagisawa et al., 2003). Interestingly, others have reported increased soreness following repeated cold treatments providing limited support for repeated application of cryotherapy post damaging exercise (Yackzan et al., 1984; Isabell et al., 1992).

Results from this investigation suggest that cryotherapy applied as ice water immersion is effective in reducing some of the deleterious symptoms associated with exercise-induced muscle damage. The precise mechanisms responsible for this required further clarification but perhaps highlight the multitude of factors involved in the aetiology of exercise-induced muscle damage.

## CHAPTER 8

### **A further investigation into the influence of cold-water immersion on the indices of muscle damage following prolonged high intensity intermittent exercise**

#### **8.1 Introduction**

In the previous Chapter we examined the effectiveness of cold-water immersion as a treatment for exercise induced muscle damage (EIMD). Several markers were used to assess recovery in the hours and days following exercise. The results provide some evidence for the benefits of this treatment modality as an aid to recovery. However, not all the markers of muscle damage and indices of muscle recovery reflected beneficial effects of cryotherapy. This inconsistency may have been a consequence of the timing of the collection of blood samples and measurements of muscle function that might be resolved with more frequent sampling.

In designing the previous study we were aware that we may have introduced some bias in favour of the treatment group because it was difficult to provide a plausible sham treatment for the control group. The use of non-steroidal anti-inflammatory drugs (NSAID) is sufficiently widely known for the treatment of muscle soreness that this offered a plausible method of providing a sham treatment for the control group.

Therefore, the aim of this study was to further examine the influence of cryotherapy on EIMD with more frequent pre- and post- treatment measurements together with the inclusion of a Sham treatment for the control group in the form of a placebo NSAID.

## 8.2 Methods

### 8.2.1 Subjects

Twenty healthy males volunteered to take part in this study, which had approval from the University Ethical Advisory Committee. All were all semi-professional soccer or rugby players, but were unfamiliar with the mode of exercise to be used in the present study (LIST). Subjects were asked to abstain from other proposed treatments of EIMD including massage and anti-inflammatory drugs for the duration of the investigation.

### 8.2.2 Experimental Design

Having abstained from exercise for at least two days subjects arrived in the laboratory in the fasted state (10-12 h). A venous blood sample (~10 ml) was taken from a vein in the antecubital fossa after the subject had been supine for at least 10 min. Thereafter, perceived muscle soreness was recorded and muscular function was assessed using an isokinetic dynamometer followed by a 15 m sprint test (described in detail below). Once these measurements had been made the subjects completed the LIST (Previously described in Chapter 3). Subjective ratings of perceived exertion were recorded every 15 min during the LIST, heart rate was monitored every 15 s by short-range telemetry (Polar 8810, Finland) and deep body temperature was monitored using an ingestible thermometer pill (CorTemp<sup>TM</sup>, HQI, Palmetto, USA) and a data logger. Subjects ingested the equivalent of 5ml.kg<sup>-1</sup> water pre-exercise, and 2ml.kg<sup>-1</sup> following each 15 min block. This drinking strategy has been shown to maintain euhydration ( $\pm$  1.5% body mass) during intermittent exercise (McGregor et al., 1999). Nude body mass was determined immediately before and after exercise. A venous blood sample was taken immediately post-exercise and additional samples were taken 1, 2, 24 and 48 h after exercise. Subjects were instructed not to resume any additional exercise until the conclusion of testing.

### 8.2.3 Cryotherapy treatment

Immediately following exercise subjects were informed of the treatment group they had been allocated. The cryotherapy or sham groups were matched for a number of

physiological characteristics as well as physical activity status (Table 8.1). The cryotherapy group immersed their lower limbs (ensuring that the iliac crest was fully submerged) in an ice water bath for 10 minutes. The water was maintained at a temperature of  $10 \pm 1^\circ\text{C}$  by the addition of crushed ice and was repeatedly agitated to avoid the formation of a warmer boundary layer. Subjects in the Sham group were asked to take a placebo tablet described as non-steroidal anti-inflammatory. During this time the Sham group remained at rest in the same body position as cryotherapy subjects. Heart rate and deep body temperature were monitored at regular intervals throughout treatment and up to 15 min post-cryotherapy.

**Table 8.1:** Physiological characteristics and physical activity status of groups (mean  $\pm$  SD).

	Treatment (n=10)	Control (n=10)	P-value
Age (years)	21.4 $\pm$ 3.1	21.5 $\pm$ 2.9	P = 0.913
Height (m)	1.77 $\pm$ 0.05	1.77 $\pm$ 0.07	P = 0.975
Mass (kg)	74.3 $\pm$ 6.7	75.1 $\pm$ 8.8	P = 0.805
BMI (kg.m <sup>-2</sup> )	23.7 $\pm$ 1.6	23.9 $\pm$ 2.2	P = 0.703
Sum Skinfolds* (mm)	27.1 $\pm$ 4.7	27.2 $\pm$ 4.5	P = 0.958
$\dot{V}\text{O}_2\text{max}$ (ml.kg.min <sup>-1</sup> )	56.0 $\pm$ 2.3	56.2 $\pm$ 2.4	P = 0.812
Weekly exercise session	4 $\pm$ 1	5 $\pm$ 2	P = 0.085

\* sum of 4 skinfolds (*triceps, biceps, suprailiac, subscapular*).

#### 8.2.4 Assessment of muscle damage

Ratings of perceived soreness were assessed using a visual analog scale ranging from 1 “not sore” to 10 “very, very, sore” (Thompson et al., 1999) pre-exercise, immediately-post-exercise and again at 1, 2, 24, 48 and 168 h.

Maximal voluntary isometric contraction (MVC) was assessed using an isokinetic dynamometer (Cybex model 770, LUMEX Inc., USA). Subjects were familiarised with the apparatus and protocol on at least two occasions prior to participation in the LIST. Following a warm-up set of five sub-maximal repetitions of knee extension and flexion (1.05 rad.s<sup>-1</sup>) subjects completed two repetitions of isometric knee extension and flexion on their dominant limb for 5 s at 1.05 rad and 0.35 rad from a reference position (determined from maximum knee extension in all subjects), respectively. Following this

subjects were asked to complete two measures of hip extension and flexion isometric contractions on their dominant limb. Each effort was separated by 60 s rest period; subjects were verbally encouraged and received visual feedback during each repetition. The greatest peak torque achieved from both repetitions was recorded.

Sprint performance was assessed prior to commencing the LIST (3 x 15 m maximal sprints separated by 3 x 20m walks ( $1.54 \text{ m}\cdot\text{s}^{-1}$ ) and again 24 h post-exercise. Sprint times were measured using two infra-red photoelectric cells (RS Components Ltd, Switzerland) interfaced with the computer. During the LIST all sprint times were recorded.

### 8.2.5 Statistical analysis

An independent two-way analysis of variance (ANOVA) with repeated measures was used to determine if any differences existed between treatment and time during exercise and recovery. The Greenhouse-Geisser correction was utilised for epsilon  $<0.75$ , while the Huynh-Feldt correction was adopted for less severe asphericity. When significant F values were found, the Holm-Bonferroni step-wise method was utilised to determine the location of the variance (Atkinson, 2002) Values for creatine kinase activity and myoglobin were not normally distributed and therefore these values were log transformed prior to ANOVA. Log transformation always resulted in normal distribution and therefore these ANOVA results are reported. Pearson product moment correlations were used to examine the relationship between variables. Data analysis was conducted using SPSS version 11 and significance was accepted at the 5% level. Values are expressed as the mean  $\pm$  standard error of the mean (*SEM*) throughout.

## 8.3 Results

### 8.3.1 Response to intermittent exercise

Mean heart rate during the LIST was  $170 \pm 3$  b $\cdot$ min<sup>-1</sup> for both groups. Ratings of perceived exertion increased from  $12 \pm 1$  at the end of the first block to  $16 \pm 1$  for both groups at the end of exercise ( $P \leq 0.05$ ). Deep body temperature during exercise was obtained for 15 subjects (cryotherapy  $n = 6$ , control  $n = 9$ ). Temperature (measured in both groups) increased from  $37.38 \pm 0.08^\circ\text{C}$  to  $38.68 \pm 0.12^\circ\text{C}$  following 90 min ( $P \leq 0.05$ ). During exercise participants lost  $0.46 \pm 0.07$  kg in body mass. Mean sprint time during the LIST was  $2.7 \pm 0.04$  s. The estimated exercise-induced changes in plasma volume did not differ between either groups.

### 8.3.2 Response to cryotherapy treatment

Heart rate decreased during treatment from  $116 \pm 3$  b $\cdot$ min<sup>-1</sup> to  $94 \pm 2$  b $\cdot$ min<sup>-1</sup> ( $F_{3,8,234} = 47.927$   $P < 0.001$ ) and continued to decline 15 min following cryotherapy to  $85 \pm 2$  b $\cdot$ min<sup>-1</sup> ( $F_{3,8,234} = 47.927$   $P < 0.001$ ). Cryotherapy had no effect on heart rate response compared to the sham group. Deep body temperature ( $n = 15$ ) decreased from  $38.16 \pm 0.12^\circ\text{C}$  to  $37.75 \pm 0.08^\circ\text{C}$  during the treatment period ( $F_{13,169} = 29.214$   $P < 0.001$ ) and continued to fall 15 min post-treatment  $37.42 \pm 0.08^\circ\text{C}$  ( $P \leq 0.05$ ). There were no differences in deep body temperature between groups although during the cold water immersion, mean weighted mean skin temperature was significantly lower in the Cryotherapy group ( $F_{1,7,45} = 47.008$   $P < 0.001$ ).

### 8.3.3 Indices of muscle damage

Exercise resulted in severe muscle soreness, which was evident immediately after exercise and also 24 h later ( $P \leq 0.05$ ). The ratings of perceived muscle soreness were lower in the Cryotherapy compared to the Sham group at 1h and 2h post exercise ( $P < 0.01$ ), (Figure 1).

### 8.3.4 Muscle function

Maximal isometric voluntary contraction (MVC) for knee flexion and extension was unaffected following exercise and treatment (Table 8.2). However, MVC for all measures were reduced 24 and 48 h post-exercise ( $F_{3, 54} = 28.357$   $P < 0.001$ ) and returned to pre-exercise values 168 h post-exercise ( $F_{3, 54} = 28.357$   $P < 0.001$ ). Decrements in isometric MVC of hip extensors were less following cryotherapy at 24 h ( $12 \pm 3\%$  vs.  $14 \pm 2\%$  n.s) and 48 h ( $9 \pm 2\%$  vs.  $17 \pm 2\%$ ;  $F_{1, 18} = 8.547$   $P < 0.001$ ) compared to sham treatment (Table 8.2).

**Table 8.2:** Isometric maximal voluntary contraction of the knee and hip flexors and extensors following intermittent shuttle running and treatment (means  $\pm$  SEM).

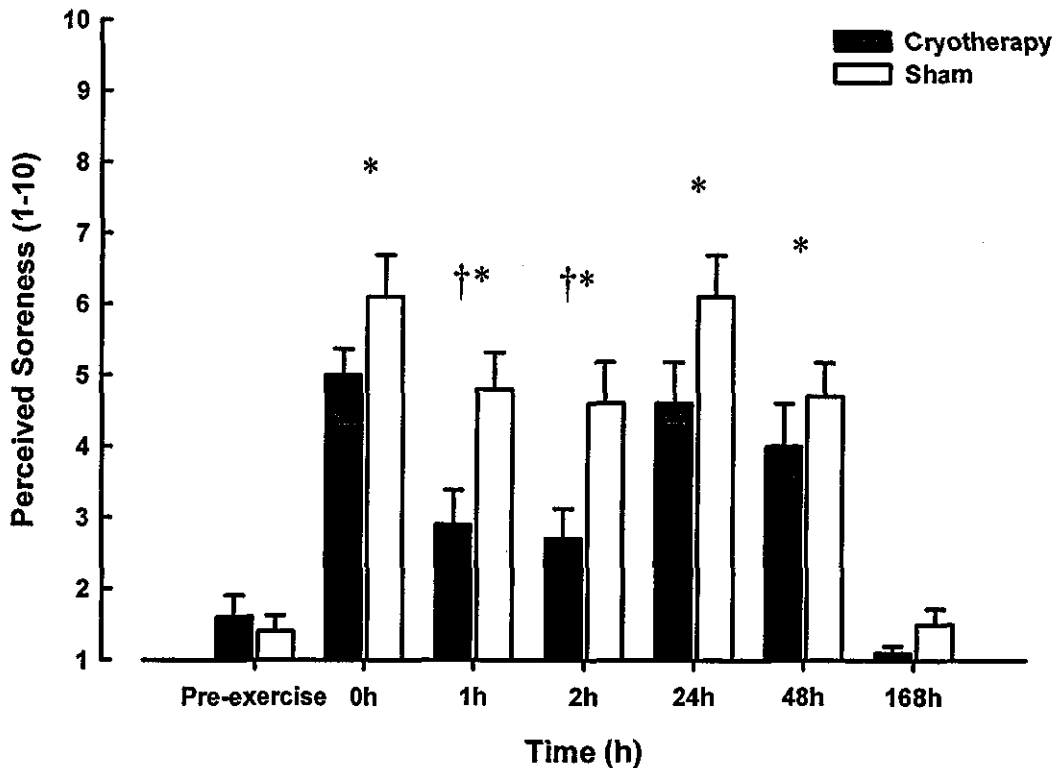
MVC (Nm.kg <sup>-1</sup> )	Pre-exercise	24h	48h	168h
<b>Knee Flexion</b>				
<b>Cryotherapy</b>	2.5 $\pm$ 0.1	2.0 $\pm$ 0.2 *†	2.0 $\pm$ 0.2 *†	2.4 $\pm$ 0.1
(% pre-exercise)	-	(79 $\pm$ 5)	(81 $\pm$ 3)	(98 $\pm$ 2)
<b>Sham</b>	2.6 $\pm$ 0.1	2.1 $\pm$ 0.2 *	2.1 $\pm$ 0.2 *	2.2 $\pm$ 0.2
(% pre-exercise)	-	(78 $\pm$ 5)	(78 $\pm$ 4)	(91 $\pm$ 2)
<b>Knee Extension</b>				
<b>Cryotherapy</b>	4.2 $\pm$ 0.2	3.6 $\pm$ 0.2 *†	3.7 $\pm$ 0.2 *†	3.9 $\pm$ 0.2
(% pre-exercise)	-	(84 $\pm$ 3)	(85 $\pm$ 3)	(94 $\pm$ 2)
<b>Sham</b>	4.1 $\pm$ 0.2	3.6 $\pm$ 0.2 *	3.6 $\pm$ 0.2 *	3.6 $\pm$ 0.2
(% pre-exercise)	-	(85 $\pm$ 3)	(88 $\pm$ 2)	(95 $\pm$ 2)
<b>Hip Flexion</b>				
<b>Cryotherapy</b>	3.0 $\pm$ 0.2	2.6 $\pm$ 0.2 *†	2.7 $\pm$ 0.2 *†	2.9 $\pm$ 0.2
(% pre-exercise)	-	(85 $\pm$ 4)	(87 $\pm$ 2)	(98 $\pm$ 3)
<b>Sham</b>	3.0 $\pm$ 0.1	2.5 $\pm$ 0.1 *	2.5 $\pm$ 0.1 *	2.7 $\pm$ 0.1
(% pre-exercise)	-	(84 $\pm$ 3)	(87 $\pm$ 3)	(96 $\pm$ 3)
<b>Hip Extension</b>				
<b>Cryotherapy</b>	5.8 $\pm$ 0.2	5.1 $\pm$ 0.2	5.3 $\pm$ 0.2	5.7 $\pm$ 0.2
(% pre-exercise)	-	(88 $\pm$ 3)	(91 $\pm$ 2)	(98 $\pm$ 2)
<b>Sham</b>	5.7 $\pm$ 0.3	4.7 $\pm$ 0.3	4.8 $\pm$ 0.2	4.8 $\pm$ 0.2
(% pre-exercise)	-	(82 $\pm$ 2)	(83 $\pm$ 2)	(90 $\pm$ 2)

† values are different between groups ( $P \leq 0.05$ ). \* values are different from pre-exercise ( $P \leq 0.05$ ).

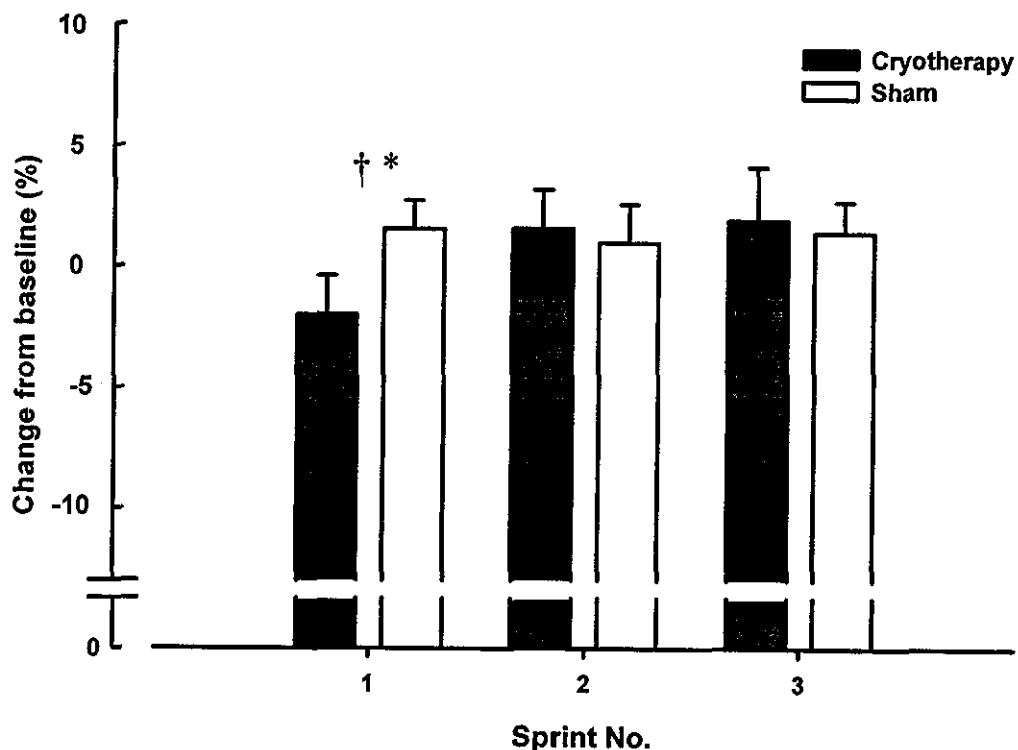


Sprint performance was better maintained in the Cryotherapy group compared to the Sham group in the first of the 3 x 15 m sprints 24hr post LIST ( $p < 0.05$ ) (Figure 8.2).

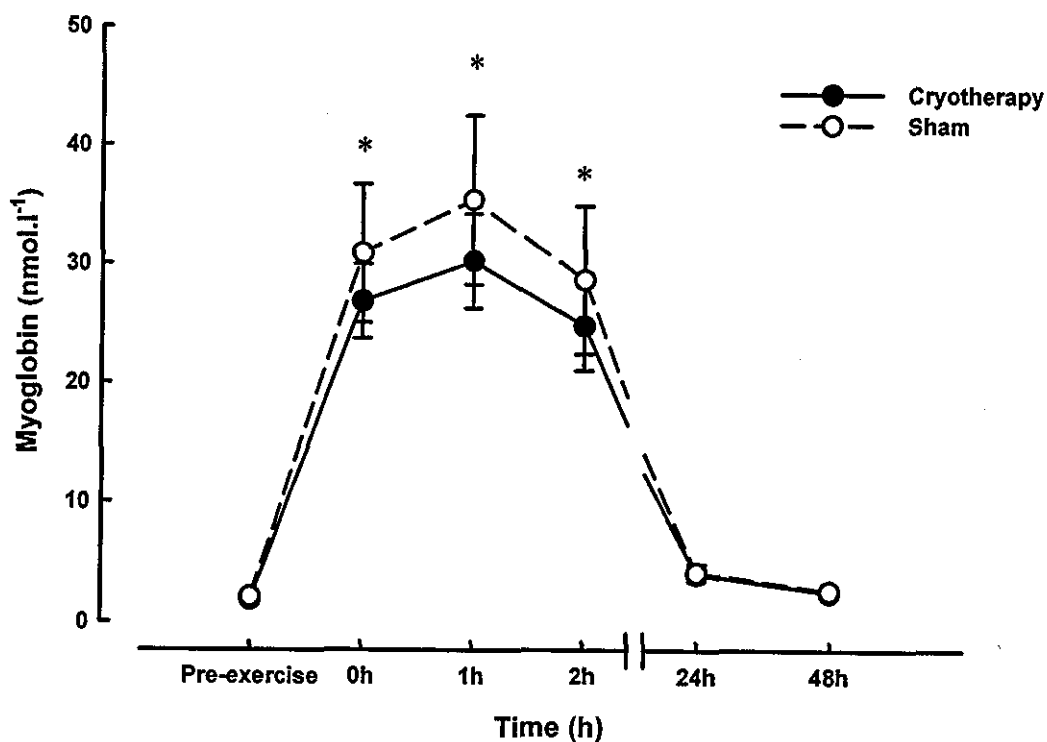
Creatine kinase activity was elevated immediately post-exercise ( $F_{1,7, 90} = 19.872$   $P < 0.001$ ) peaking 24 h later but this response was not influenced by cryotherapy (Figure 8.3). Myoglobin concentration increased immediately post-exercise in both groups ( $F_{13, 90} = 52.137$   $P < 0.001$ ). Concentrations peaked 1 h post-exercise in both groups but was unaffected by cryotherapy (Figure 8.4). There was a tendency for cryotherapy to reduce the rise in CK and Mb post-exercise however this was not statistically significant.



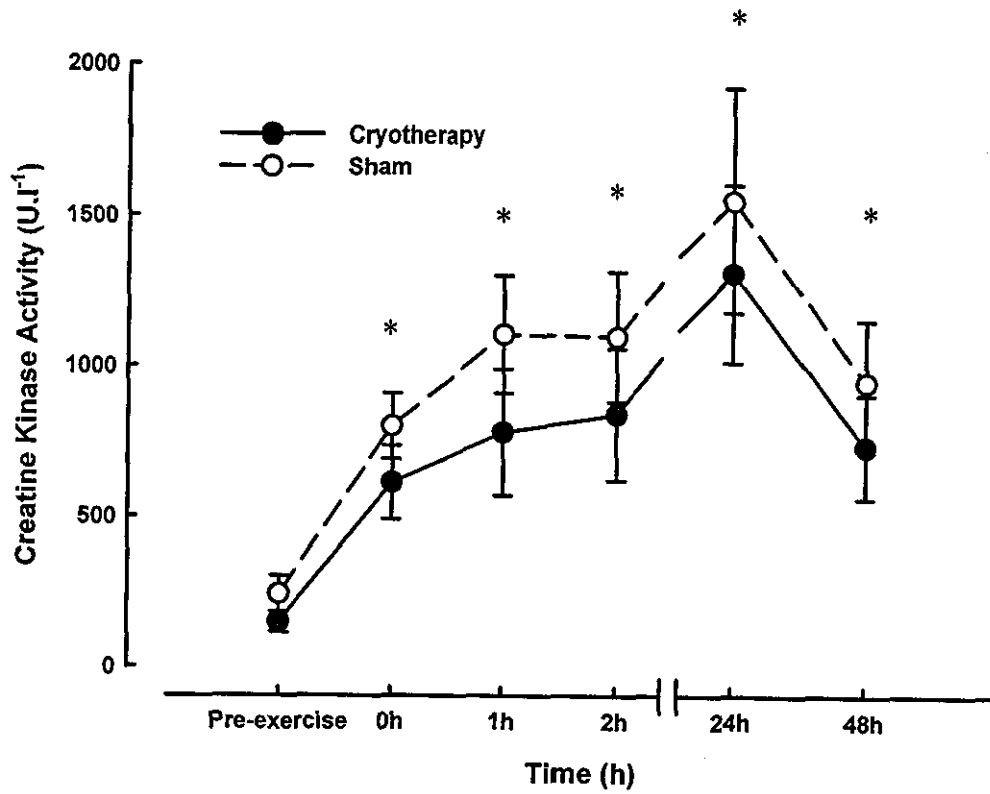
**Figure 8.1:** Perceived muscle soreness following exercise for cryotherapy (solid bars) and sham (clear bars) groups. Values are mean  $\pm$  S.E.M. \*  $P \leq 0.05$  different from pre-exercise for both groups, †  $P \leq 0.05$  different between groups.



**Figure 8.2:** Sprint performance (15m) 24h post exercise for cryotherapy (solid bars) and sham (clear bars) groups. Values are mean  $\pm$  S.E.M. \*  $P \leq 0.05$  different from pre-exercise for both groups, †  $P \leq 0.05$  different between groups.



**Figure 8.3:** Serum myoglobin concentration following exercise for cryotherapy (solid line) and sham (broken line) groups. Values are mean  $\pm$  S.E.M. \*  $P \leq 0.05$  different from pre-exercise for both groups, †  $P \leq 0.05$  different between groups.



**Figure 8.4:** Serum creatine kinase activity following exercise for cryotherapy (solid line) and sham (broken line) groups. Values are mean  $\pm$  S.E.M. \*  $P \leq 0.05$  different from pre-exercise for both groups.

## 8.4 Discussion

The main finding of this study was that the cryotherapy treatment caused a potent and almost immediate analgesic effect on muscle soreness and reduced the magnitude of decline in muscle function for some but not all of the performance measures on subsequent days.

The exercise protocol (LIST) used in this investigation induced severe muscle soreness, muscular dysfunction and increased myofibrillar protein appearance in the hours and days following its completion. The muscle damage effects caused as a result of the intermittent exercise was similar to previous studies including our last investigation (see Chapter 7). In this study subjects reported an initial increase in muscle soreness immediately post-exercise that peaked again at 24h and at 48h following exercise. This acute onset of muscle soreness was very similar to that reported in the previous chapter and is likely to be the result of the accumulation of metabolic by-products from the prolonged high intensity running. The secondary peak in muscle soreness in the days following exercise is a well-documented phenomenon, caused by secondary muscle damage and is often referred to as DOMS.

The immediate reduction in perceived soreness following treatment is likely to be due to the analgesic effects of cooling. The application of cold, reduces nerve conduction velocity, muscle spindle activity, the stretch-reflex response and spasticity thus inhibiting the pain-spasm cycle (Meeusen and Lievens, 1986). This analgesia can last up to 3 h (Meeusen and Lievens, 1986) and may explain the initial reductions in muscle soreness observed 1 and 2 h post-exercise. In this study the Cryotherapy group tended to report lower ratings of soreness 24h and 48h post-exercise however unlike our previous investigation this was not statistically significant. The reasons for the difference between the previous and current findings are hard to explain. Certainly, if cryotherapy were effective in reducing muscle injury it would be plausible for participants to experience less pain in the days following exercise. An observed reduction in DOMS 24 and 48h post-exercise with cryotherapy has been reported in similar investigations (Prentice, 1982; Denegar and Perrin, 2000; Yanagisawa et al., 2003).

There is further evidence that the treatment went some way to reduce the muscle injury effects of the LIST. Decrements in isometric MVC of hip extensors were less following cryotherapy at 24 h ( $12 \pm 3\%$  vs.  $14 \pm 2\%$  n.s) and 48 h ( $9 \pm 2\%$  vs.  $17 \pm 2\%$ ;  $P < 0.01$ ) compared to sham treatment. The hip extensors would have been heavily involved in the eccentric phase of deceleration following sprinting. Other muscle groups experienced similar pattern of dysfunction and recovery however there was no difference between treatments. The overall pattern of muscle dysfunction and recovery over a 7 day period has been reported in several other studies (Ebbeling and Clarkson, 1989; Thompson et al., 1999; Bailey et al., 2002) and described in the previous chapter. Interestingly, 15m sprint performance 24 h post LIST was less affected in the treatment group compared to the Sham group. In our previous investigation, sprint and jump performance on subsequent days were unaffected by cryotherapy. However, these measures were reassessed 48 h post LIST instead of 24 h post LIST, as in the current study. Warren and co-workers (1999) advocate this type of specificity when reassessing effects of muscle damaging exercise following running on subsequent muscle function. It is reasonable to speculate that the effects of cryotherapy went some way to reduce the magnitude of muscle damage and as such reduced any decrement in sprint performance 24 h post LIST. Alternatively, the trend for participants to feel less sore 24 h post LIST following cryotherapy may have meant that they simply felt more able to produce a maximal effort on this exercise test. The Sham treatment goes some way to addressing the concern over the possibility of biasing the results of the cryotherapy group as result of the control group over reporting because of their lack of treatment.

No differences were seen on the appearance of myofibrillar proteins with cryotherapy. This is in contrast to previous investigations (Eston and Peters, 1999; Howatson and Van Someren, 2003) including our last study. However, the pattern of myofibrillar protein appearance is similar to other investigations. Myoglobin concentrations peaked in the hours following exercise whilst creatine kinase activity was highest in the days following exercise. In this study there was a strong trend for cryotherapy to reduce the concentration of myoglobin in the blood in the hours following treatment, however this was not found to be statistically significant. Creatine kinase activity has been commonly used as a marker of EIMD. The reason for no differences in creatine kinase the cryotherapy group in this investigation could be due to a number of factors including insufficient reduction in EIMD, large inter-individual differences in responses. The

suggestion that cryotherapy may reduce post-exercise muscle damage via a vasoconstriction of blood and lymph vessels hence decreased leakage of plasma proteins into the interstitial spaces has not been supported in this investigation.

These findings suggest cold-water immersion immediately following prolonged intermittent shuttle-running reduces some indices of exercise-induced muscle soreness and dysfunction. Inconsistencies in the responses of all indices make it difficult to suggest the precise mechanisms responsible for these observations.

## CHAPTER 9

### General Discussion

#### 9.1 Introduction

The series of studies described in this thesis were designed to examine areas that are of central importance in the preparation for and recovery from competition and training of soccer players.

A brief summary of the main findings are as follows:

- Chapters 4 reported the results of a national survey that found that the prevalence of fitness testing in professional football is high. Field based fitness assessments appear to be used more than laboratory assessments. The most commonly used tests are the MSFT, acceleration sprints and vertical jumps. Furthermore, the findings reported in Chapter 5 demonstrate that the MSFT is sensitive enough to detect training-induced physiological changes in already well-trained female soccer players.
  
- Chapter 6 described the study that examined the influence of the type of carbohydrate on the recovery of performance. It appears that the GI of a high carbohydrate mixed diet consumed following 90 min of the LIST does not affect recovery of performance 24 h later. However, both a LGI and HGI high CHO diets were equally effective in restoring running performance and capacity in players within 24 h.
  
- Chapters 7 and 8 describe studies that examined the influences of cryotherapy on exercise-induced muscle damage and soreness. Incidentally these studies reinforce previous findings that the LIST induces severe muscle soreness and dysfunction in subsequent days. Cryotherapy administered in the form of cold-water immersion immediately post-intermittent exercise appears to be effective in alleviating some markers of EIMD and soreness, consequently improving the recovery of performance.

- The studies described in Chapters 7 and 8 also demonstrate that partial immersion (lower limbs up to the iliac crest) does not decrease deep body temperature at a greater rate than not being in cold water.

## 9.2 Fitness assessment within soccer

It is clear from our findings that the physical assessment of soccer players is a common practise in English professional football clubs. A number of reasons were given for using fitness testing and these included determining a player's strengths and weaknesses, examining the effectiveness of training regimes and providing a benchmark for players recovering from injury.

The most commonly used fitness tests within professional football included the MSFT, vertical jump and acceleration sprint performance. However, there are a number of key issues for sport science practitioners to consider when using these performance tests principally their reliability and validity. In terms of reliability, the practitioner must determine if any changes detected in performance are the result of actual physiological improvements rather than the result of variation between or within subjects (Atkinson and Nevill, 1988). Systematic and random error within each bout of testing has the potential to distort the magnitude of change recorded and therefore give a false impression of the effectiveness of a particular training period. This can be overcome by repeated performances of the same tests and statistical calculations to quantify the amount of systematic and random error that is present within a particular test (Atkinson and Nevill, 2001). The tests should have a high level of validity; for example, a player scoring highly on an aerobic endurance test should demonstrate these qualities in a match.

A major finding from our investigations, reported in Chapter 5, was that the MSFT was sensitive to detect changes that occur as a consequence of training in already well-trained players. Historically, studies on the physiological adaptations to training have used previously untrained or just recreationally active subjects and so reported large improvements in performance and metabolic indicators of cellular changes (Nevill et al., 1989; Helge et al., 1996). Therefore, had we used less well-trained players in this study then it is reasonable to predict that we would have recorded even larger changes



in the responses to the MSFT after a relatively short period of training. The advantages of the field tests chosen by a large number of football clubs are that they are relatively easier to administer than are laboratory fitness tests and also that they are perceived by players to be highly relevant to football. In our training study we found a 6% improvement in predicted  $\text{VO}_2 \text{ max}$  and a 12% improvement in distance run during the MSFT. In terms of the relevance to football the key finding was the 12% improvement in endurance capacity. Similarly when a performance test is part of the laboratory determination of  $\text{VO}_2 \text{ max}$  then information obtained is much more valuable than simply determining  $\text{VO}_2 \text{ max}$  per se. For example, if the laboratory test is designed only to determine  $\text{VO}_2 \text{ max}$  by adjusting the duration of the test to maintain consistency between the pre- and post training procedures then it is easy to miss the improvements in performance. For example, in our laboratory testing we recorded a modest improvement in the  $\text{VO}_2 \text{ max}$  values of the female soccer players i.e. 4% but an impressive improvement of 9.5% in run time to exhaustion. After training they were able to run for an additional 74 s at the higher exercise intensity than before training but if the only criteria of interest was  $\text{VO}_2 \text{ max}$  then this impressive improvement would have gone unrecorded. The evidence that these changes in  $\text{VO}_2 \text{ max}$  and in performance were the result of real physiological adaptations is provided by the observations of lower blood lactate concentrations and heart rate values during submaximal treadmill running. Therefore, whereas tests such as the MSFT provide a convenient and easy to administer method of routinely assessing the functional fitness of players, including periodic laboratory testing complements these tests and leads to greater understanding of the individual rates of adaptation to training programmes. This is important information for the practitioner because they must have the confidence to know that a 'real' training effect has occurred instead of just a variation in test scores.

### **9.3 Recovery of performance**

In chapters 6, 7 and 8 the recovery of performance following high intensity intermittent exercise was examined. A number of tests that were previously identified as being commonly used in professional football were used in conjunction with other measures to assess the recovery of performance in these latter studies. The whole subject of recovery is now of intense interest in elite soccer because of the unrelenting schedule of games clubs have to play each season. The problem facing the sports science

practitioner working within professional clubs is to determine which of the many recovery strategies that have been suggested to be beneficial are really worth implementing? Barnett and colleagues have recently reviewed a range of recovery modalities used by elite athletes and with the exception of nutritional interventions they were unable to recommend any recovery methods that were based on well controlled studies (Barnett, 2006)

The important role that nutrition plays in the recovery process is well established and understood (Burke et al., 2004). Successful refuelling of the body's carbohydrate stores is necessary for players to cope with daily training and frequent matches. Several studies have shown that HGI CHO enhance post-exercise refuelling due to the rapid rise in blood glucose and insulin. However, most of these studies have concentrated on single nutrients (Blom et al., 1987) or real foods that have been misleadingly categorised as 'simple' or 'complex' carbohydrates rather than by their effect on blood glucose (Costill et al., 1981; Roberts et al., 1988). Interestingly, Burke et al., (1993) were the first to report greater beneficial effects, in terms of muscle glycogen resynthesis, of HGI mixed diet compared to meals comprising of predominately LGI over a 24 hr period. However, in the only study to examine the effects on subsequent performance, Stevenson and co-workers (2005) reported an improved recovery of endurance running capacity in individuals who had consumed LGI high CHO diet. Our findings, reported in Chapter 6, used exactly the same composition of foods within our recovery diets but failed to find any difference between HGI or LGI diets on performance 24hr following the initial 90 min LIST. Possible reasons for differences between these two studies have already been discussed (See chapter 6). The demands of the activity patterns of the exercise protocol used in the present study were quite different from those in constant paced treadmill running to fatigue (Stevenson et al 2005). In submaximal running, endurance capacity is generally improved when the contribution of fat oxidation to substrate metabolism is increased. Using LGI carbohydrate recovery diets (Stevenson et al 2005) or LGI carbohydrate pre-exercise meals (Wu et al 2006) increase fat metabolism, sparing the limited glycogen stores and hence delay the onset of fatigue. However, during prolonged intermittent high intensity shuttle running the brief periods of sprinting combined with high intensity running demand large contributions from glycogen metabolism. Therefore, under these conditions potential benefits of a diet that promotes high rates of fat metabolism and glycogen sparing are generally overridden

because of the glycogen-demanding intensity of the exercise protocol (Nicholas et al 1999).

Furthermore the LIST protocol used in the studies described in Chapters 6 has been shown to cause severe muscle soreness and muscle damage (Thompson et al., 1999). It is possible that the rate of muscle glycogen resynthesis following this muscle-damaging exercise could be reduced. Eccentric muscle contractions have been shown to impair the process of muscle glycogen resynthesis (Doyle et al., 1993). The impairment of muscle glycogen resynthesis appears to occur despite high concentrations of circulating glucose and insulin. Insulin receptor binding and glucose transport are both plasma membrane-mediated events. A significant disruption of the muscle cell membrane by eccentric exercise might reduce insulin-stimulated glucose transport or the activation of glucose synthase (Doyle et al., 1993). However the available evidence does not seem to implicate a disruption of the GLUT 4 transporter proteins in the delayed resynthesis of muscle glycogen (Asp et al 1998). Nevertheless, one suggestion is that eccentric exercise may disrupt the activation of GLUT 4 proteins but the reduced muscle glycogen that occurs as a response to prolonged exercise stimulates the activity of these proteins with a resultant no net change (Asp et al 1998). Therefore, although the role of nutrition in the recovery period will always remain central to the restoration of performance, it is plausible that other recovery modalities that have the potential to reduce the severity of muscle damage may also contribute to an accelerated recovery process.

The results from the studies described in Chapters 7 and 8 showed that cryotherapy reduced some but not all markers of muscle soreness and damage. Furthermore, cryotherapy appeared to reduce the decrement in muscle function in the days following intermittent exercise in some but not all measures. It is these inconsistencies in some of the markers that make it hard to draw any firm conclusions as to the effectiveness of cryotherapy as a recovery modality. The effect of cryotherapy on the severity and pattern of muscle soreness was very similar in the studies reported in Chapters 7 and 8. The potential reasons for this analgesic effect have been discussed within the respective chapters. There is some evidence that cryotherapy reduces the severity of secondary muscle damage and helps better maintain muscle cell membrane integrity in the hours and days following exercise but this is far from conclusive. Myofibrillar protein leakage

followed the same pattern in both studies (Chapters 7 and 8), with a reduction in the rise in myoglobin present in venous blood samples in the hours post exercise following the cryotherapy treatment (see figure 9.1 and 9.2, below). The concentration of CK peaked 24h post- exercise in both studies but appeared to be unaffected by cold water immersion. It was not within the scope of these investigations to monitor the restoration of muscle glycogen in the days following the LIST but one may speculate that if cryotherapy reduced muscle damage and protected muscle membrane integrity then there is a possibility that glucose transporters i.e. GLUT-4 would help to restore muscle glycogen to a better extent.

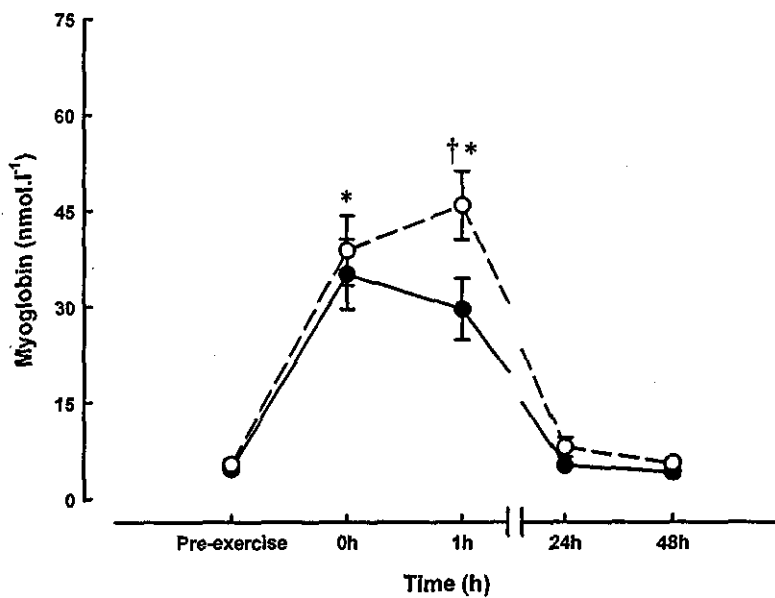


Figure 9.1 Myoglobin results from chapter 7

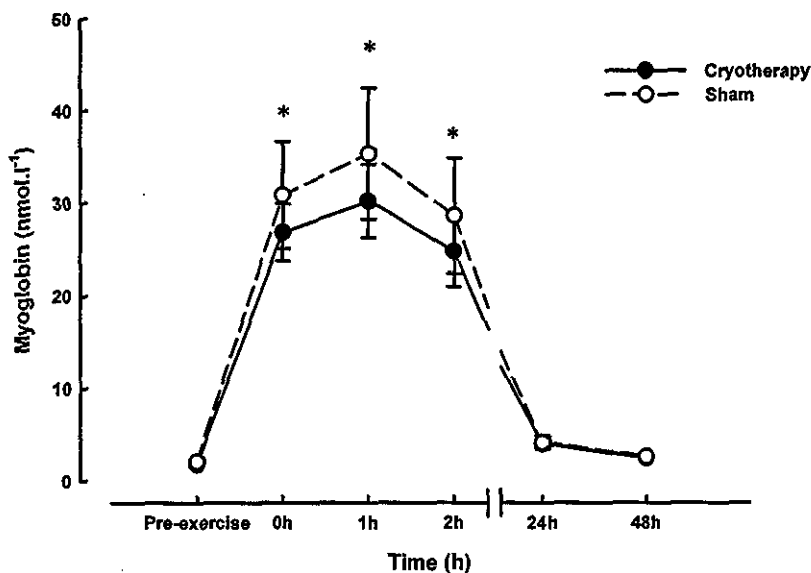


Figure 9.2 Myoglobin results from chapter 8

A variety of different muscle function tests were used in the studies described in Chapters 7 and 8 to examine the effect of cryotherapy on consequent performance in the days following damaging exercise. This is a particularly pertinent area to examine because soccer players can be faced with very short recovery periods between matches. Externally valid field based assessments that included vertical jumps and acceleration sprint tests were used together with more controlled isometric dynamometry to determine differences in performance following cryotherapy. All measures of muscle function indicated degrees of impairment in the days following the LIST suggesting these could act as a robust easy to administer method of assessing recovery from intense training periods or match play. The threat of any possible placebo effects masking the integrity of this data was a concern. Clearly, it was impossible to blind the treatment group from the cold-water immersion, therefore the group receiving no treatment may have considered their chances of recover impaired. However, this concern is unlikely to have been a major issue because in the subsequent study (Chapter 8) when a Sham treatment was introduced for the control group similar patterns of muscle dysfunction occurred. Of course, it has to be recognised that the cooling procedure used in these two studies may not have reduced tissue temperature low enough to produce more pronounced effects. Equally so the duration of the immersion in ice cold water may not have been long enough to reduce tissue temperature without causing damage. Although not ideal we had to trade-off duration against the water temperature in order to retain the loyalty of our subjects when designing these experiments. Therefore, there is clearly more work to be undertaken in defining the optimum conditions that will obtain the benefits of this recovery modality.

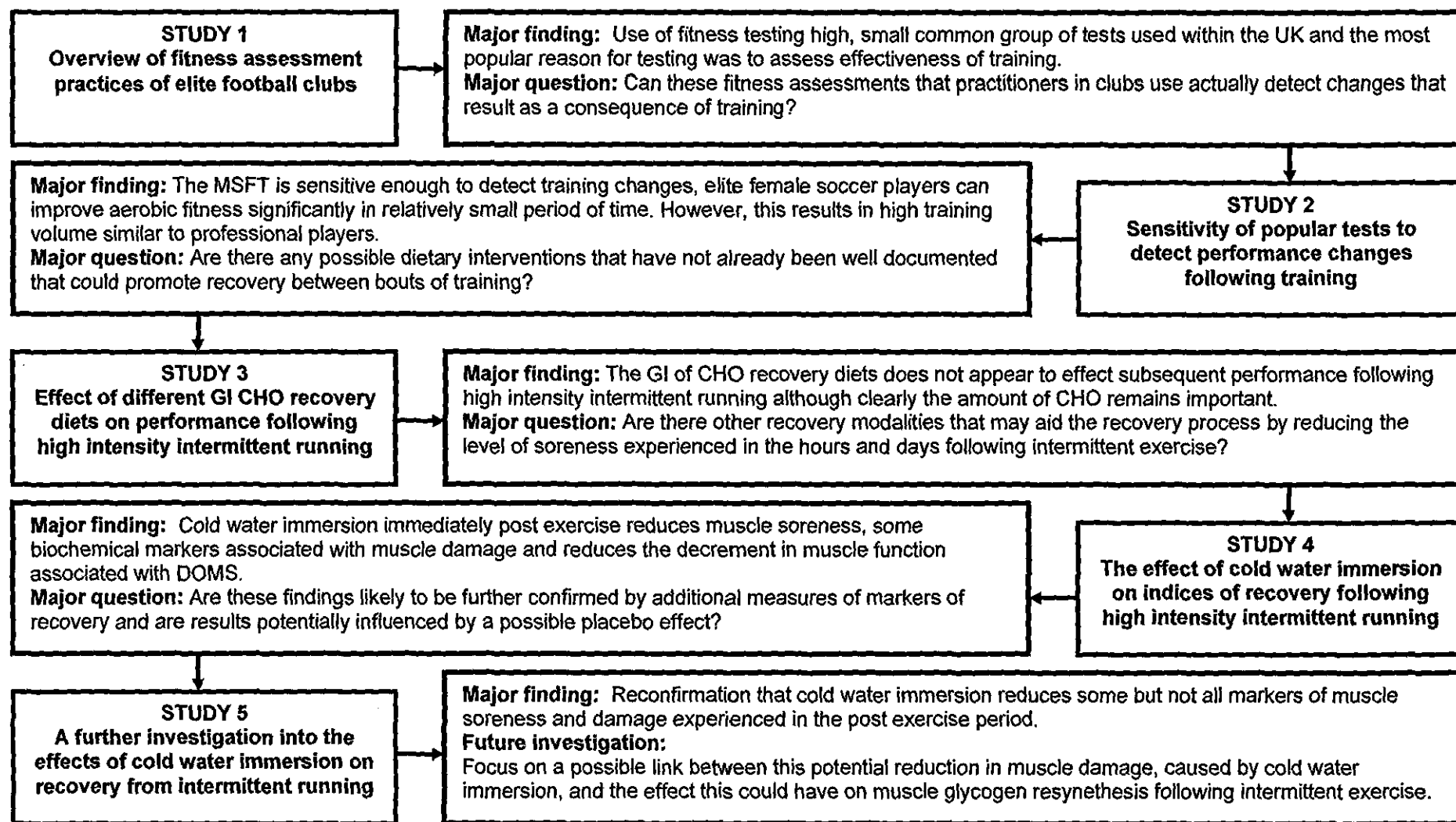


Figure 9.1 Schematic representation of the thesis major studies and a flow summary of how each study was conceptualised

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## Appendix A

### Statement of Informed Consent

I have read the subject information sheet, detailing the procedure and requirements which are involved with this study and I fully understand what is required of me. I have had an opportunity to ask for further information and clarification of the demands of each of the procedures. **I am aware that I have the right to withdraw at any time with no obligation to give reasons for my decision. I agree to take part in the study.**

Signed \_\_\_\_\_

Witnessed by \_\_\_\_\_

Date     /     /

### Daily Health Questionnaire

Please complete the following brief questions to confirm your fitness to participate in Today's experiment:

At present do you have any health problems for which you are:

1) On medication, prescribed or otherwise     Yes  No

2) Attending your general practitioner     Yes  No

Have you any symptoms of ill health, such as those associated with a cold or other common infection?

Yes  No

If you have answered yes to any of the above questions please give more details below:

.....  
.....

Do you want to take part in today's experiments?     Yes  No

Signature: \_\_\_\_\_

Date     /     /

## Appendix B

### Health Screen for Ingestible Temperature Sensors

It is important that volunteers participating in research studies involving the core temperature pill are currently in good health and have had no significant medical problems in the past. This is to ensure (i) their own continuing well-being and (ii) to avoid the possibility of individual health issues confounding study outcomes.

**Please complete this brief questionnaire to confirm fitness to participate:**

1. **At present, or in the past** have you had any of the following health problems:

- (a) Any known or suspected obstructive disease of the gastrointestinal tract, including but not limited to diverticulitis and inflammatory bowel disease.

Yes  No

- (b) Any inflammatory bowel disease.

Yes  No

- (c) A history of disorders or impairment of the gag reflex.

Yes  No

- (d) Any previous gastrointestinal surgery.

Yes  No

- (e) Are you or might you undergo Nuclear Magnetic Resonance (NMR) scanning during the period that the disposable temperature sensor is within the body.

Yes  No

- (f) Any hypomotility disorders of the gastrointestinal tract including but not limited to Illus.

Yes  No

## Appendix C

### Health Screen for Study Volunteers

It is important that volunteers participating in research studies are currently in good health and have had no significant medical problems in the past. This is to ensure (i) their own continuing well-being and (ii) to avoid the possibility of individual health issues confounding study outcomes.

Please complete this brief questionnaire to confirm fitness to participate:

1. At present, do you have any health problem for which you are:

- (a) on medication, prescribed or otherwise..... Yes  No   
(b) attending your general practitioner..... Yes  No   
(c) on a hospital waiting list..... Yes  No

2. In the past two years, have you had any illness which require you to:

- (a) consult your GP ..... Yes  No   
(b) attend a hospital outpatient department..... Yes  No   
(c) be admitted to hospital ..... Yes  No

3. Have you ever had any of the following:

- (a) Convulsions/epilepsy..... Yes  No   
(b) Asthma..... Yes  No   
(c) Eczema ..... Yes  No   
(d) Diabetes ..... Yes  No   
(e) A blood disorder ..... Yes  No   
(f) Head injury ..... Yes  No   
(g) Digestive problems..... Yes  No   
(h) Heart problems ..... Yes  No   
(i) Problems with bones or joints ..... Yes  No   
(j) Disturbance of balance/coordination..... Yes  No   
(k) Numbness in hands or feet ..... Yes  No   
(l) Disturbance of vision..... Yes  No   
(m) Ear / hearing problems..... Yes  No   
(n) Thyroid problems ..... Yes  No   
(o) Kidney or liver problems..... Yes  No   
(p) Allergy to nuts ..... Yes  No

4. Has any, otherwise healthy, member of your family under the

- age of 35 died suddenly during or soon after exercise? ..... Yes  No

If YES to any question, please describe briefly if you wish (eg to confirm problem was/is short-lived, insignificant or well controlled.) .....

## Appendix D

### Physical Activity Questionnaire for Soccer Players

**Current Level**

\_\_\_\_\_ (e.g. university 1<sup>st</sup> team, recreational, national etc.)

**Current Fitness/Training Status**

\_\_\_\_\_ (e.g. fully/half match fit, recovering etc.)

**How many training sessions a week do you usually participate in?**

\_\_\_\_\_

**Do you practice Skills Training?**

Yes / No

If yes, how many sessions per week?

\_\_\_\_\_ per week

How many minutes does each session last?

\_\_\_\_\_ min

**Do you practice Interval Training?**

Yes / No

If yes, how many sessions per week?

\_\_\_\_\_ per week

How many minutes does each session last?

\_\_\_\_\_ min

**Do you practice Resistance Exercise (weights)?**

Yes / No

If yes, how many sessions per week?

\_\_\_\_\_ per week

How many minutes does each session last?

\_\_\_\_\_ min

**Do you practice Endurance Running?**

Yes / No

If yes, how many runs per week?

\_\_\_\_\_ per week

How many minutes does each run last?

\_\_\_\_\_ min

What is your weekly running mileage?

\_\_\_\_\_ miles per week

## Appendix E

### Body Temperature Measurement Pill

#### PLEASE READ BEFORE SWALLOWING THE PILL

You will have already completed a health screen questionnaire confirming your suitability to use the temperature sensor pill. You must not swallow the pill if any of the following applies to you:

- Any presence of any known or suspected obstructive disease of the gastrointestinal tract, including but not limited to diverticulitis and inflammatory bowel disease.
- Have or suspected to have any inflammatory bowel disease.
- Exhibit or having a history of disorders or impairment of the gag reflex.
- Previous gastrointestinal surgery.
- Will be undergoing Nuclear Magnetic Resonance (NMR) scanning during the period that the disposable temperature sensor is within the body (up to 72 hours).
- Hypomotility disorders of the gastrointestinal tract including but not limited to IBS.

#### Instructions

Before swallowing the pill please handle with care, replacements for lost or damaged pills are costly and must be imported from the US.

Your temperature sensor pill has been placed within a screw-top plastic tube to prevent accidental damage. Remove the sensor from the tube and take it out of the plastic bag it is sealed within. The pill is wrapped in an orange label to indicate it is clean, has not been tampered with and is calibrated correctly. Attached to the outside of this wrapper is a small magnet. This magnet holds a magnetic switch closed within the pill that stops the battery and hence the pill from working. Remove this magnet and the label before swallowing the pill. This will start the pill operating; from then on it has a battery life of 72 hours. Please place the magnet and orange label back inside the plastic bag and bring them along with you on the morning of the trial, so that we can confirm you have done this.

Swallow the sensor pill before you go to bed or before midnight (whichever is first) on the evening before the experimental trial. You should find no difficulty in swallowing the pill as it is coated in silicone which becomes very slippery when wet. Swallow in the same way as you would swallow a normal pill whilst standing or sitting, you may wish to swallow it with a glass of water. If you experience difficulty swallowing the pill, place it on the back of your tongue, tilt your head back and swallow with water. If you experience nausea and/or vomiting then stop immediately. If you have problems or queries contact Nick Gant on ##### or ##### (24 hour number).

On arrival to the laboratory the pill will be located with a receiver to check that it is functioning correctly and to ascertain its position in the body. After you finish the main trial, the pill will pass through your gastrointestinal tract at a rate dependent on your motility and appear in your faeces. The pill is designed to safely flush down the toilet with your faeces and cause no harm to the environment.

Appendix F

**Rating of Perceived Exertion**

A vertical scale for Rating of Perceived Exertion. The scale consists of a central vertical line with horizontal tick marks extending to the left and right. The numbers 6 through 20 are listed on the left side of the scale. Descriptive labels are placed to the right of the scale at specific intervals: 'Very Very Light' at 7, 'Very Light' at 9, 'Fairly Light' at 11, 'Fairly Hard' at 13, 'Hard' at 15, 'Very Hard' at 17, 'Very Very Hard' at 19, and 'Maximal' at 20.

6		
7		Very Very Light
8		
9		Very Light
10		
11		Fairly Light
12		
13		Fairly Hard
14		
15		Hard
16		
17		Very Hard
18		
19		Very Very Hard
20		Maximal

# Appendix G

## Diet and Physical Activity Diary

Day 1

Date

Time	Brand Name of item (except fresh food etc.)	Full description of each item*	Weight served (g)	Weight leftover (g)

**General Comments:**

\* Include - fresh, frozen, dried, canned etc. - cooked, boiled, grilled, fried (including type of fat), roasted.

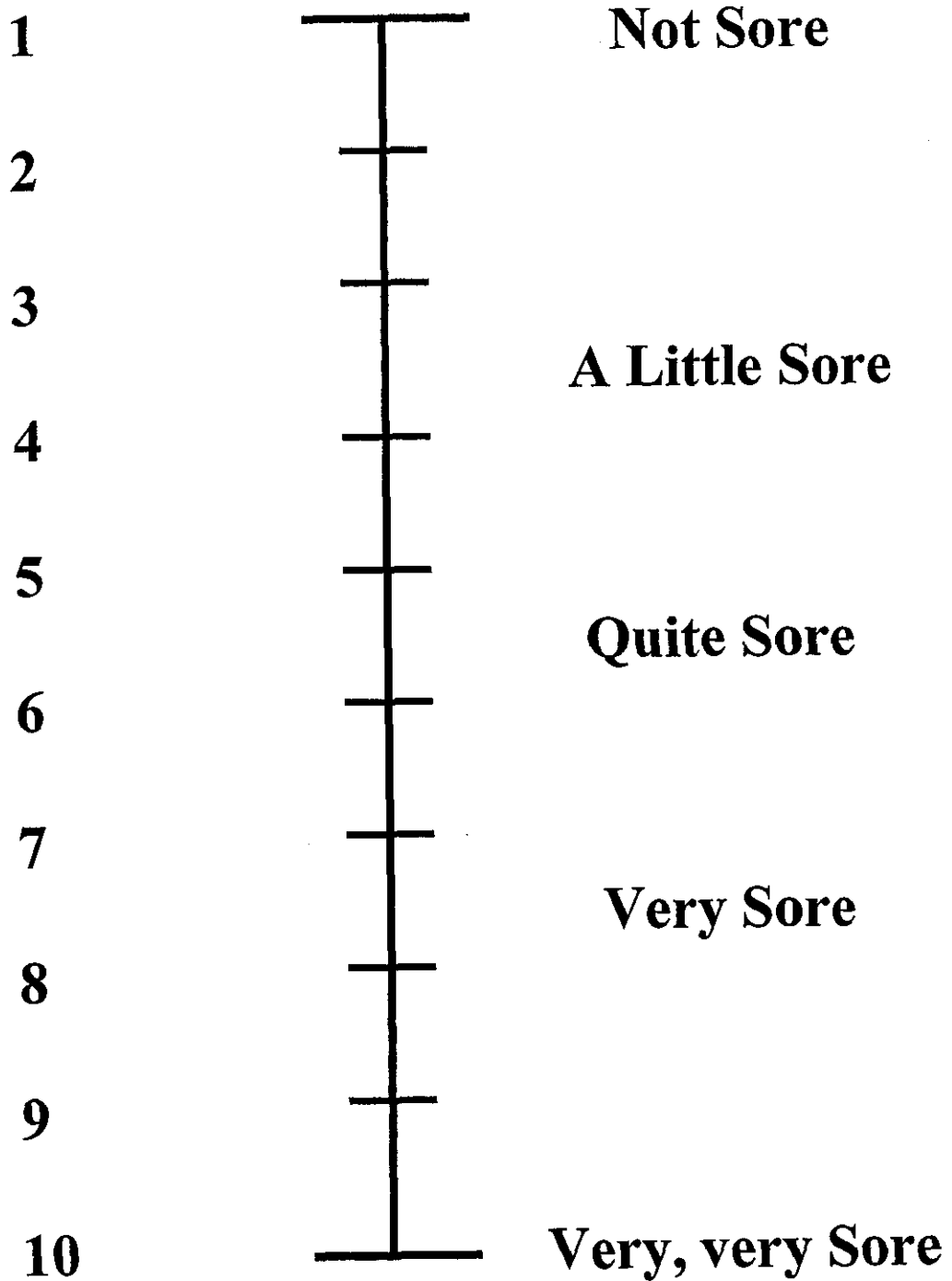
Time	Activity (Irregular to your normal daily routine)	Duration (min)	General Comments:

Wake-Up Time	Bed Time	General Comments:



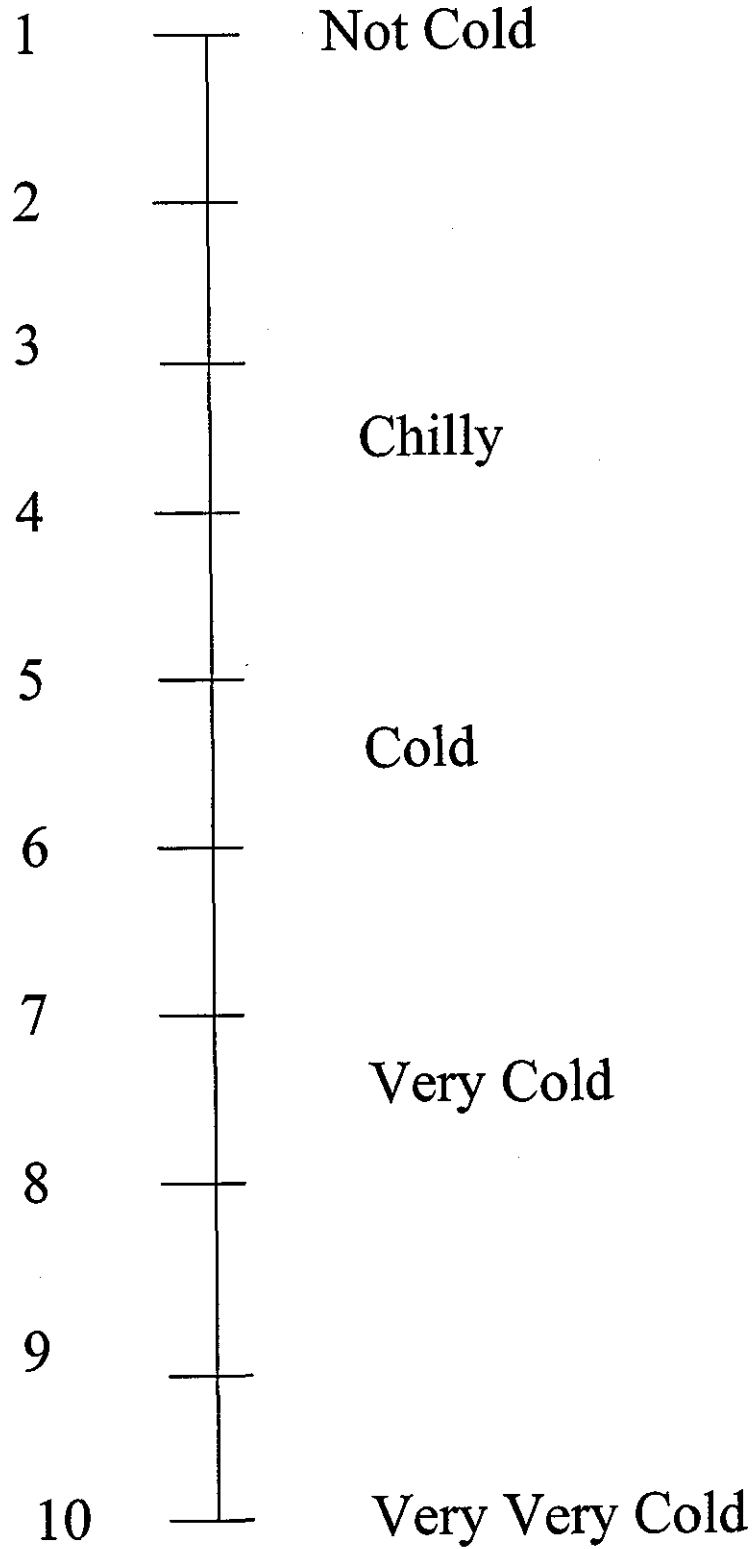
Appendix H

RATING OF PERCEIVED SORENESS



Appendix I

**RATING OF PERCEIVED COLDNESS**





THE FOOTBALL ASSOCIATION

An Audit of Fitness Tests in Professional football

Section A: Personal Details

1. Club no: [ ] 2. Staff member: [ ] 3. Position: [ ] 4. Years employed: [ ]

Section B: Fitness Tests

1. Do you use any form of fitness test? Yes [ ] No [ ]

If so, 2. Which of the following fitness components do you test for?

Power [ ] Speed [ ] Agility [ ] Strength [ ] Aerobic Capacity [ ] VO2max [ ]

Proprioception [ ] Speed Endurance [ ] Anthropometric Measurements [ ]

Other (please specify): [ ]

3. Which of the following tests do you use (please tick the first white box)?

4. How many times a year do you test players using this specific test (please put figure in second white box)?

'Bleep test' [ ] [ ] Acceleration Sprint tests (please specify distance) [ ] [ ]

1/3RM Bench Press [ ] [ ] 1/3RM Squat [ ] [ ] Yo-yo endurance test [ ] [ ]

Vertical CMJ with arms [ ] [ ] Vertical CMJ without arms [ ] [ ] Maximal Velocity Sprint [ ] [ ]

Cooper Run [ ] [ ] Agility T test [ ] [ ] Multi sprints tests (please describe below) [ ] [ ]

Heart rate profiling (please describe below) [ ] [ ] Illinois agility test [ ] [ ] VO2max test [ ] [ ]

Others (Please specify)

[ ]

Section C: Fitness Test Procedures

1. At what stage in the season do you test players?

End of season [ ] Close season [ ] Pre-season [ ] Early season (Aug - Nov) [ ]

Mid season (Dec - Feb) [ ] Late season (March - May) [ ] Monthly [ ] Weekly [ ]

If all tests are not performed at the same stages of the season (please specify)

[ ]

2. Do you test the entire squad at the same time? Yes [ ] No [ ]

If no please elaborate

[ ]

3. At what time of day do you test players?

AM  PM

4. Do your players undergo a standardised warm-up?

Yes  No

5. Have you found that players get similar scores if the test is done repeatedly within a short period of time?

Yes  No  Not tried

6. Who conducts the fitness tests (full-time or p/time)?

### Section D: Use of Results

1. Who is responsible for analysing and providing feedback from the results (full-time or p/time)?

2. What are the results used for?

Motivation  Set programmes  Assess programmes  Player selection

Position selection  Monitor over training  Assess specific fitness levels

Create player databases

Other (please specify)

3. Do you ever use these results to assess a player's recovery from a bout of injury?

Yes  No

4. At what stage during rehabilitation is the test/s employed?

5. Are there any other tests used for this purpose (please specify)

6. Do you have a set score that individuals have to achieve on these tests to be declared fit to play a competitive match?

Yes  No

7. Who makes the final decision whether a player is fit for selection?

**Section E: Your opinion on Fitness Tests**

In this section please tick the description that most closely matches your opinion of the statement:

	Strongly agree	agree	neither agree or disagree	disagree	strongly disagree
1. The fitness tests available are adequate to monitor the fitness levels of players	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
2. There is a need to design fitness tests that are more representative of match demands	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
3. The use of fitness test results are limited because of their relevance to football	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
4. There is a need to create player profiles to use for comparisons following injury	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

**Section F: Nutrition & Training Issues**

1. How many meals do players have at the club on a training day?

None  Breakfast  Lunch  Pre-training snack  Post-training snack

2. Do players eat a pre-match meal together on match days (please circle one answer or two if different for home & away [please specify])?

always 100%      very often ~75%      often ~50%      sometimes ~25%      never 0%

3. Do players eat together following a competitive match (please circle one answer or two if different for home & away [please specify])?

always 100%      very often ~75%      often ~50%      sometimes ~25%      never 0%

4. Who decides on the food and drink choices available to players?

5. Do you employ any strength and power components into your training programme (please tick first white box)?

6. How many times a week is this work done (please indicate a figure in second white box)?

Basic strength machines       Free-weights       Olympic bars – power work

Specific eccentric muscle training       Plyometrics

Please provide any comments on this training below:

**For enquires / clarification please contact:**

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Thank you for your assistance in the completion of this questionnaire.

Please return in the pre-paid envelope provided.



THE FOOTBALL ASSOCIATION

## APPENDIX K

### WEEKLY TRAINING TIMETABLE FOR ENGLAND FEMALE PLAYERS

DAY	AM	PM
MONDAY	High intensity aerobic interval session 1	RECOVERY WORK
TUESDAY	Strength Training session 1	Technical and Tactical session 1
WEDNESDAY	High intensity aerobic interval session 2	Technical and Tactical session 2
THURSDAY	Strength Training session 2	
FRIDAY		Technical and Tactical session 3
SATURDAY	REST	REST
SUNDAY		MATCH

## The Effect of High Carbohydrate Meals with Different Glycemic Indices on Recovery of Performance During Prolonged Intermittent High-Intensity Shuttle Running

Samuel Erith, Clyde Williams, Emma Stevenson, Siobhan Chamberlain, Pippa Crews, and Ian Rushbury

This study examined the effect of high carbohydrate meals with different glycemic indices (GI) on recovery of performance during prolonged intermittent high-intensity shuttle running. Seven male semi-professional soccer players (age  $23 \pm 2$  y, body mass [BM]  $73.7 \pm 9.0$  kg and maximal oxygen uptake  $58 \pm 1.0$  mL · kg<sup>-1</sup> · min<sup>-1</sup>) participated in two trials in a randomized cross-over design. On day 1, the subjects performed 90 min of an intermittent high-intensity shuttle running protocol [Loughborough Intermittent Shuttle Test (LIST)]. They then consumed a mixed high carbohydrate recovery diet (8 g/kg BM) consisting of either high (HGI) (GI: 70) or low (LGI) (GI: 35) GI foods. Twenty-two hours later (day 2) the subjects completed 75 min of the LIST (part A) followed by alternate sprinting and jogging to fatigue (part B). No differences were found between trials in time to fatigue (HGI  $25.3 \pm 4.0$  min vs. LGI  $22.9 \pm 5.6$  min,  $P = 0.649$ ). Similarly, no differences were found between trials for sprint performance and distance covered during part B of the LIST. In conclusion, the GI of the diet during the 22 h recovery did not affect sprint and endurance performance the following day.

*Key Words:* glycemic index, recovery, intermittent exercise, fatigue

It is well established that consuming a high carbohydrate (CHO) diet during the recovery period following prolonged exercise increases the rate of muscle glycogen resynthesis compared to a normal mixed diet (6). Furthermore, endurance capacity during prolonged constant paced running (10) and during prolonged intermittent high-intensity shuttle running is improved the day after prolonged exercise with high CHO recovery diets (9 to 10 g/kg BM) (16). Therefore it is common practice

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in many team sports to consume high CHO diets particularly during periods when training or competitive matches are being performed on consecutive days.

However, while the benefits of high CHO recovery diets are clearly recognized little attention has been given to the possible influences of the different types of carbohydrates on subsequent performance. Carbohydrates can be classified according to their postprandial glycemic response (13) and this criteria has been used to classify a large selection of foods (11). High glycemic CHO (HGI) foods are characterized by a rapid increase in blood glucose and accompanying rise in insulin concentrations. Glycogen storage is influenced both by insulin and an available supply of glucose as substrate. Therefore, CHO foods with moderate-to-high glycemic index (GI) would be expected to enhance post-exercise refueling of the body's limited CHO stores. Indeed, a study by Burke and colleagues reported greater muscle glycogen concentrations 24 h after prolonged cycling when subjects consumed a HGI rather than a LGI CHO diet (5). However, the authors did not appear to have assessed exercise performance following the HGI and LGI recovery diets.

Therefore, Stevenson and colleagues conducted a study to examine the influences of high and low GI diets on endurance running capacity after a recovery of 24 h (23). Their subjects ran longer on the LGI recovery diet than they did on the HGI recovery diet. Furthermore, the rate of fatty acid oxidation was also greater during exercise after the LGI than after the HGI recovery diet. This study used constant pace treadmill running and so contributes to the limited literature on carbohydrate diets and endurance running capacity. Nevertheless, there is a dearth of literature on the possible influences of HGI and LGI recovery diets on subsequent exercise capacity during intermittent high-intensity running. Therefore, the purpose of the present study was to examine the effect of high CHO meals with different GI values on 22 h recovery from prolonged intermittent high-intensity shuttle running.

## Methods

### Participants

Seven male semi-professional soccer players participated in this study. Their mean ( $\pm$  standard error of the mean) age, body mass (BM), and maximal oxygen uptake values were  $23 \pm 2$  y,  $73.7 \pm 9$  kg, and  $58 \pm 0.8$  mL  $\cdot$  kg<sup>-1</sup>  $\cdot$  min<sup>-1</sup> respectively. The study had approval from the Loughborough University Ethical Advisory Committee and all subjects gave informed written consent before the study began.

### Preliminary Measures

The subjects reported to the laboratory 7 to 10 d prior to their first main trial to complete a Progressive Multistage Shuttle Running Test (20) to estimate their maximal oxygen uptake ( $VO_{2max}$ ). Thereafter, the subjects completed a 45 min familiarization with the Loughborough Intermittent Shuttle Test (LIST) (17).

### Experimental Design

Each subject participated in two experimental trials separated by at least 7 to 10 d. Each main trial was completed over a 2-d period. During the 2 d immediately



prior to the first main trial subjects recorded their food intake so that they could consume the same diet prior to the second main trial. Furthermore, they were asked not to drink alcohol or caffeine-containing beverages nor perform strenuous exercise during the 2 d before the main trials. On the morning of each main trial the subjects arrived at the laboratory in a fasted state where they provided a urine sample and then their nude body mass was obtained using a beam balance (model 3306, Avery, Birmingham, UK). A cannula (Venflon 18G, Becton-Dickinson Ltd., Helsingborg, Sweden) was then inserted into an antecubital forearm vein and connected to a 3-way tap (Becton-Dickinson) with a 10 cm extension tube for blood sampling. Heart rates were recorded using a short-range telemetry system (Polar Electro, Kempele, Finland).

On day 1 the subjects performed 90 min of an intermittent high-intensity shuttle running protocol (R1) (LIST) (17). The LIST protocol involves shuttle running between two lines 20 m apart at speeds that are based on the estimated  $VO_{2max}$  of the subjects, i.e. 55% and 95%  $VO_{2max}$ . The running speed between the two lines was dictated by a computer-generated audio signal. The 90 min LIST was divided into 6 × 15 min blocks of activity that were separated by 3 min rest periods. Each 15 min block involved periods of walking (1.5 m/s) jogging (55%  $VO_{2max}$ ) and running (95%  $VO_{2max}$ ) as well as maximal sprints. The time taken to complete each 15 m sprint was measured using infra-red photo-electric cells (R.S. Components, Switzerland) interfaced with computer software. Each activity was repeated approximately 11 times in each 15 min block of the LIST (17). Following R1 they were given a diet that provided 8 g CHO/kg BM for the 22 h recovery. Meals and snacks were composed of either high (HGI) or low (LGI) GI carbohydrates in a randomized cross-over design (Table 1). On day 2, subjects returned to the laboratory, again in a fasting state and performed 5 × 15 min of the LIST (R2 -part A). This was followed by the following pattern of activity: two jogs (55%  $VO_{2max}$ ), one walk and one maximal sprint that were continued to the point of fatigue (R2-part B). Volitional fatigue was defined as the inability of the subjects to maintain the required pace or maintain consecutive sprints at times that were no less than 95% of their mean times in blocks 2 and 3 of the LIST during R2-part A.

On all occasions subjects were given 5 mL/kg BM of water before exercise and 2 mL/kg BM every 15 min during the 3 min rest periods.

### Test Meals

Iso-caloric recovery meals consisting of HGI or LGI CHO foods, as calculated by the method of Wolever et al. (26), were provided for each subject after R1 (Table 1). Breakfast was consumed 30 min following the completion of R1 and lunch was provided 3 h later. Both of these meals were prepared and consumed in the laboratory. Subjects were then provided with two snacks and an evening meal that was later consumed at home. Participants were asked to eat one snack between lunch and the evening meal (7 PM) and were required to eat the final snack between 8 and 9 PM. The diets were composed of predominantly HGI or LGI carbohydrates; however, other foods (e.g., milk, cheese, and lettuce) were included in both diets to make the meals more palatable. The amount of CHO in each of the two diets was calculated as available carbohydrates. Both diets consisted of 72% CHO, 11% fat, and 17% protein. The nutritional content of each meal was calculated from

**Table 1 Characteristics of Test Meals (for a 70 kg subject)**

Meal	Description	Macronutrient content
HGI breakfast	62 g corn flakes <sup>a</sup> + 257 mL skim milk	730 kcal
	80 g white bread + 10 g flora + 20 g jam	139 g CHO, 9.9 g fat
	155 mL Lucozade Original <sup>a</sup>	20 g protein
LGI breakfast	86 g muesli + 257 mL skim milk	732 kcal
	67 g apple, 103 g canned peaches	139 g CHO, 9g fat
	128 g yogurt, 257 mL apple juice	23 g protein
HGI lunch	158 g white bread, 154 g turkey breast	1076 kcal
	50 g cheese, 40 g lettuce, 180 g banana	148 g CHO, 24g fat
	200 mL Lucozade Original <sup>a</sup>	63 g protein
LGI lunch	154 g whole wheat pasta, 150 g turkey breast,	1075 kcal
	50 g cheese, 40 g lettuce	149 g CHO, 25 g fat
	185 g pasta sauce, 150 g pear	60 g protein
	150 mL apple juice	
GI dinner	255 g baked potato	1100 kcal
	410 g canned spaghetti	176 g CHO, 31 g fat
	50 g cheese, 40 g lettuce, 67 g Mars bar	28 g protein
	170 mL Lucozade Original <sup>a</sup>	
LGI dinner	360 g chili beans, 200 g wheat tortilla	1100 kcal
	50 g cheese, 40 g lettuce	176 g CHO, 29 g fat
	260 mL orange juice	39 g protein
HGI snacks	154 g white bread, 40 g jam, 20 g flora	600 kcal
		96 g CHO, 17 g fat
		15 g protein
LGI snacks	170 g yogurt, 100 g apple, 100 g flapjack	625 kcal
		97 g CHO, 25 g fat
		15 g protein
HGI total		3520 kcal. 560 g CHO
		84 g fat, 126 g protein
		(72% CHO, 11% fat, 17% protein)
		<b>GI = 70<sup>b</sup></b>
LGI total		3600 kcal. 560 g CHO
		88 g fat, 135 g protein
		(72% CHO, 11% fat 17% protein)
		<b>GI = 35<sup>b</sup></b>

Note. <sup>a</sup>Corn Flakes: Kellogg's (UK) Ltd. Manchester UK; Lucozade Original drink: GlaxoSmithKline (UK). <sup>b</sup>GI calculated by previously described method (Wolever et al., 1986) with GI values taken from Foster-Powell and co-workers (2002).

the information provided by the manufacturer. The GI of the total mixed diets was calculated from the weighted means of the GI values for the component foods (26). The calculated GI for the HGI and LGI diets was 70 and 35, respectively.

### Sample Collection and Analysis

Participants were asked to remain standing for all blood samples. At each sampling point, 11 mL of blood was collected and 5 mL of whole blood was immediately dispensed into an EDTA tube. Hemoglobin (Hb) concentration was determined on duplicate 20  $\mu$ l samples of whole blood using the cyanmethemoglobin method (Boehringer Mannheim, Mannheim, Germany). Hematocrit (Hct) values were determined in triplicate on samples of whole blood following microcentrifugation using a sliding micro-hematocrit reader (Gelman-Hawksley Ltd., Lancing, Sussex, UK). Changes in plasma volume were estimated using the method of Dill and Costill (8). Blood lactate concentration was determined fluorometrically using an enzymatic method (model 8-9, Locarte Co., London, UK) (15). Plasma samples were obtained by centrifugation of the remaining blood for a period of 10 min at 1700  $\times$  G and 4  $^{\circ}$ C. The aliquoted plasma was then stored at  $-80^{\circ}$  C for later analysis of free fatty acids (FFA) (ASC-ACOD method, Wako NEFA C, Wako Chemicals, Richmond, VA), glucose (GOD-PAP method, Randox, Co. Antrim, UK), and glycerol (Randox, Co. Antrim, UK) using an automatic photometric analyzer (Cobas-Mira plus, Roche, Basel, Switzerland). The remaining whole blood sample was dispensed into a tube containing a clotting activator and left to clot for 45 min. Serum samples were then obtained after centrifugation at 1700  $\times$  G for 10 min at 4  $^{\circ}$ C. The aliquoted serum was stored at  $-80^{\circ}$  C and later analyzed for insulin (Coat-A-count Diagnostica Products Corp., Caernavon, UK) by radioimmunoassay (RIA) using an automated gamma counter (Cobra 5000, Packard, Pangbourne, UK).

### Statistical Procedures

An analysis of variance (ANOVA) with repeated measures on both factors (experimental treatment and time) was used to examine differences in the physiological and metabolic responses in R1 and R2—part A. However, due to variations in run times to exhaustion, a Student's paired *t*-test was used to analyze differences at the point of fatigue. The same analysis was carried out on all non-time dependant variables. Statistical significance was accepted at an alpha level of  $P < 0.05$ . All results are presented as means  $\pm$  standard error of the mean.

## Results

### Run Times

All subjects completed R1 and R2 but no differences were found in run times to exhaustion between trials during part B of R2 (HGI 25.3  $\pm$  4.0 min vs. LGI 22.9  $\pm$  5.6 min,  $P = 0.649$ ). Neither were there differences between trials in the calculated fatigue index for sprint performance during part B of R2. Furthermore, no differences were found in the number of sprints attempted or the total distance covered during part B of R2 (HGI 43  $\pm$  7 vs. LGI 39  $\pm$  10 and HGI 3474  $\pm$  531 m vs. LGI 3097  $\pm$  793 m, respectively).

### Plasma FFA and Glycerol

Plasma FFA and glycerol concentrations rose progressively during R1 ( $P \leq 0.05$ ). Immediately post-exercise, FFA concentrations reached  $0.54 \pm 0.16$  and  $0.49 \pm 0.07$  mmol/L for HGI and LGI trials, respectively. During R2 there was a similar rise in plasma FFA (Figure 1) and glycerol concentrations; however, there were no differences between HGI and LGI trials (Figure 2).

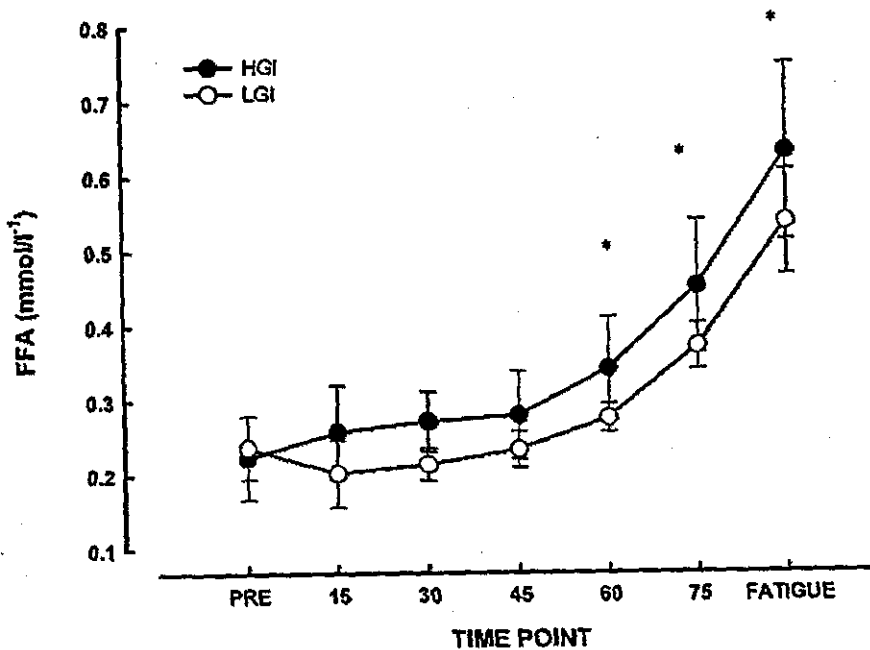


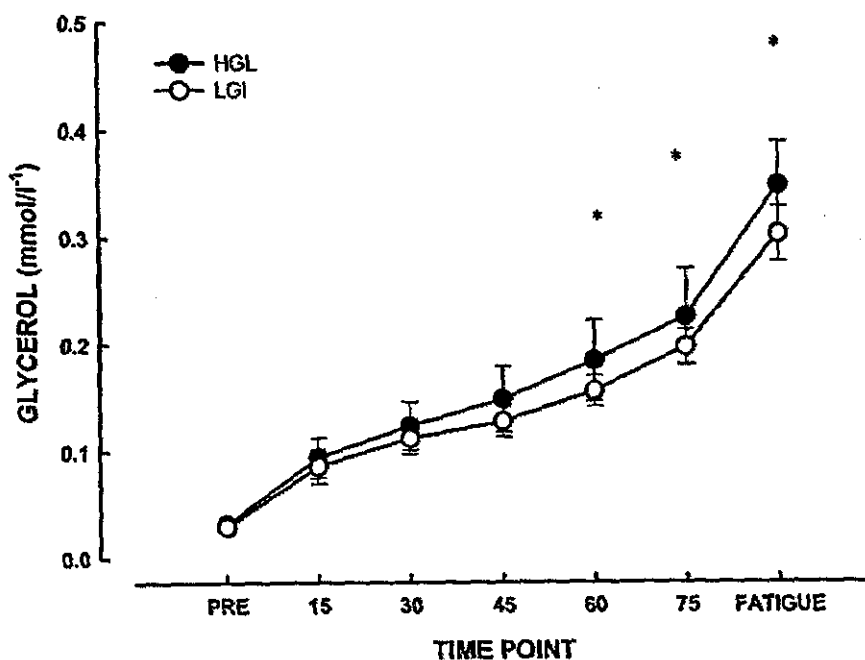
Figure 1 — Plasma FFA concentrations (mmol/L) during R2 in the HGI and LGI trials (mean  $\pm$  standard error of the mean).  $P \leq 0.05$  \* different from pre-exercise for both groups.

### Plasma Glucose and Serum Insulin Responses

Plasma glucose concentrations rose during first 30 min of exercise in both R1 and R2 for both conditions and no differences were observed between trials. Plasma glucose concentrations were maintained within a range of 4 to 6 mmol/L throughout all runs. At the point of fatigue during part B of R2, plasma glucose concentrations were similar for both trials ( $5.01 \pm 0.56$  and  $4.97 \pm 0.57$  mmol/L in the HGI and LGI trials, respectively) (Figure 3). Serum insulin concentrations decreased at the start of exercise from pre-exercise concentrations and continued to fall slowly throughout exercise in both trials (Figure 4) ( $P \leq 0.05$ ).

### Blood Lactate

During R1 and R2 there were no differences in blood lactate concentrations between trials (average concentrations during R1 were  $2.6 \pm 0.8$  mmol/L in the HGI trial



**Figure 2** — Plasma glycerol concentrations (mmol/L) during R2 in the HGI and LGI trials (mean  $\pm$  standard error of the mean).  $P \leq 0.05$  \* different from pre-exercise for both groups.

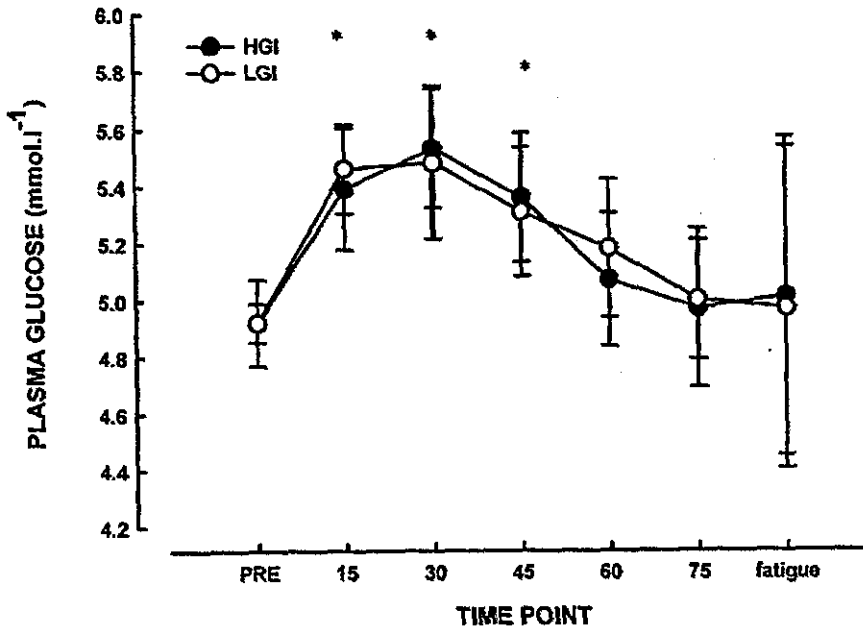
and  $2.6 \pm 0.6$  mmol/L in the LGI trial and average concentrations during R2 were  $2.6 \pm 0.2$  mmol/L in the HGI trial and  $2.1 \pm 0.3$  mmol/L in the LGI trial). However, blood lactate concentrations were higher during R2 part B compared with R2 part A in both trials (average concentrations during R2 part A were  $2.6 \pm 0.7$  compared to R2 part B  $3.6 \pm 0.44$  mmol/L,  $P \leq 0.05$ ).

### Heart Rate and Rating of Perceived Exertion (RPE)

Heart rate values were similar for both trials on day 1 and day 2 and were higher during part B of R2 in both trials compared to earlier in exercise ( $P \leq 0.05$ ). Although there were no differences in RPE values between trials they were higher during part B than part A of the LIST ( $P \leq 0.05$ ).

### Hydration Status

There was no difference in pre-exercise body mass before each trial. At the end of R1 in both trials subjects lost approximately 1% of their pre-exercise body mass and a similar percentage change in body mass occurred during R2. There were no significant differences in pre-exercise urine osmolality values between trials ( $640 \pm 243$  and  $684 \pm 231$  mL  $\cdot$  Osmol  $\cdot$  kg $^{-1}$  in the HGI and LGI trials, respectively).



**Figure 3** — Plasma glucose concentrations (mmol/L) during R2 in the HGI and LGI trials (mean  $\pm$  standard error of the mean).  $P \leq 0.05$ . \* different from pre-exercise for both groups.

### Environmental Conditions

There were no differences in ambient temperatures or relative humidity during each trial (HGI-day 1,  $14.9 \pm 2.2$  °C, relative humidity  $64.6 \pm 7.2\%$ ; HGI-day 2,  $15.6 \pm 1.0$  °C, relative humidity  $59.3 \pm 6.9\%$  compared to LGI-day 1,  $14.8 \pm 2.7$  °C, relative humidity  $62 \pm 8.4\%$ , LGI-day 2,  $14.6 \pm 2.4$  °C, relative humidity  $59.9 \pm 7.8\%$ ).

### Discussion

The main finding of the present study was that a HGI carbohydrate diet consumed during the 22 h recovery period following prolonged intermittent high-intensity shuttle running had no greater effect on sprint performance or endurance capacity the following day than a LGI carbohydrate recovery diet.

Prolonged intermittent high-intensity exercise relies heavily on muscle glycogen as a substrate for the sustained high rate of ATP resynthesis (2, 3, 12, 18, 19). During R2 part A of the LIST consisted of five 15 min periods of exercise that includes 11 sprints. Therefore the subjects in the present study completed 55 maximal sprints during the first 75 min and then an additional 43 and 39 sprints during part B of the LIST in the HGI and LGI trials, respectively. In an earlier study using the same intermittent exercise protocol we showed that when a high CHO recovery diet was consumed after performing the LIST to fatigue, endurance

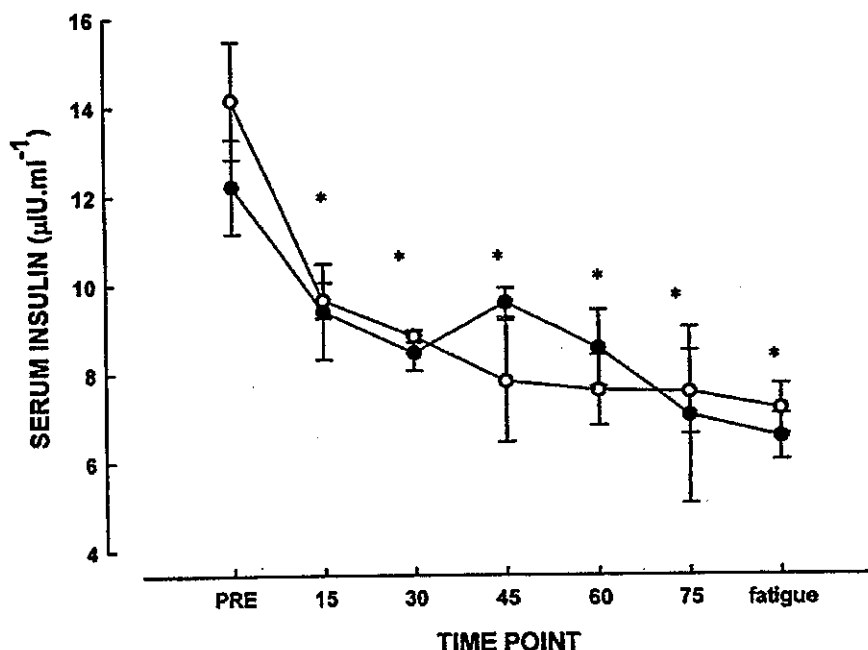


Figure 4 — Serum insulin concentrations ( $\mu\text{IU}/\text{mL}$ ) during R2 in the HGI and LGI trials (mean  $\pm$  standard error of the mean).  $P \leq 0.05$ . \* different from pre-exercise for both groups.

capacity was restored 22 h later whereas that was not the case after an isocaloric mixed diet (16). After the high CHO recovery diet the subjects ran for 3.3 min longer than they did on the mixed diet. In contrast, when the subjects consumed the isocaloric mixed diet that included their normal intake of CHO during the 22 h recovery, they ran for 2 min less than on the previous day (16). Therefore it is clear from this earlier study that a high CHO recovery diet improves recovery from prolonged intermittent high-intensity exercise.

Burke and colleagues reported that a HGI recovery diet resulted in a 48% greater muscle glycogen concentration 24 h after prolonged cycling than was achieved when their subjects consumed a LGI diet (5). However, they did not assess the exercise performance of their subjects following the 24 h recovery. Nevertheless, it would be reasonable to speculate that their subjects would have produced better performances after the HGI than after the LGI recovery diet because they would have started exercise with higher muscle glycogen stores (5). Therefore, it was surprising to find that there was no difference in performance after the HGI and LGI recovery diets in the present study.

In a previous study, we found a greater endurance capacity 22 h after prolonged treadmill running when runners consumed a LGI CHO recovery diet than when the diet was composed of HGI CHO (23). After completing a 90 min treadmill run at 70%  $\text{VO}_{2\text{max}}$  the runners were randomly assigned to either the HGI or LGI recovery diet. After an overnight fast they returned to the laboratory and ran to exhaustion

at the same intensity as on the previous day. The mean run time to exhaustion after the LGI recovery diet was 12 min longer than after the HGI diet (108.9 vs. 96.9 min) (23). Even though runners probably began the second run with higher muscle glycogen stores after the HGI recovery diet (5, 23, 25) it is likely that they would have used more glycogen during the run than when they had consumed the LGI recovery diet (14). It is possible, though only speculation, that after an hour or so of running the muscle glycogen stores may have been similar following the two dietary conditions even though the rates of glycogenolysis were initially different. However, the greater rate of fat oxidation during the second run in the LGI trial may have been able to cover the energy production deficit as muscle glycogen concentrations decreased (27) more completely than in the HGI trial. A compensatory up-regulation of fat oxidation in skeletal muscle may have reduced the need for an increased contribution from blood glucose to cover energy production in the LGI trial but not in the HGI trial. The differences in the demand for an increased turnover of blood glucose towards the end of exercise may have contributed to the differences in the times to fatigue (7).

The lack of differences in sprint performance and endurance capacity in the present study after the LGI and HGI recovery diets is difficult to explain when it appears that the subjects on the HGI recovery diet probably began exercise with higher muscle glycogen concentrations. One explanation might be that following the HGI recovery diet the subjects began intermittent exercise using more CHO than fat because of its greater availability (21), whereas after the LGI recovery diet they used more fat and less CHO. The run-walk-sprint nature of part A of the LIST is conducive to fat metabolism during the low-intensity phase of each 15 min block of activity. Although the rates of glycogenolysis during part A of the LIST may have been different after the HGI and LGI recovery diets, the glycogen stores may have been reduced to similar values at the end of the 75 min of exercise. If this was the case then the subjects would have started part B with similar muscle glycogen concentrations and, as the results show, their run times to fatigue were also similar.

Another consideration is that this intermittent high-intensity shuttle running protocol produces significant post-exercise muscle soreness (24) because of the eccentric muscle contractions during the frequent changes in speed and direction. Extensive eccentric muscle contractions have been shown to decrease the rate of glycogen resynthesis (9) and lead to an increased rate of glycogen utilization during subsequent exercise (1). If this were the case in the present study then it would be reasonable to expect poorer sprint performance and endurance capacity after the LGI recovery diet because the lower recovery rate of muscle glycogen resynthesis would have been exacerbated by the eccentric nature of the prior exercise, i.e., R1. However, the absence of a difference in sprint performance and endurance capacity between the two trials suggests that fatigue during part B may not have been entirely due to differences in pre-exercise glycogen concentrations.

Fatigue during part B may have been due in part to low muscle glycogen stores and the inability to resynthesize phosphocreatine (PCr) rapidly enough to maintain ATP turnover rate at the required level (4, 12). In addition, the accumulation of metabolites such as hydrogen ions, ADP, AMP, inorganic phosphate, and magnesium may have created a cellular environment that contributed to the inability of the working muscles to sustain energy production (22).



In summary, the main finding of this study was that the type of CHO (high or low GI) provided as part of a recovery diet did not influence performance during subsequent prolonged high-intensity intermittent shuttle running. Carbohydrate rather than fat would have been the main fuel during this high-intensity running protocol and therefore the potential benefit of a recovery diet that promotes fat oxidation during continuous running would have had little impact during this type of exercise.

### Acknowledgments

The authors would like to thank the Medical and Exercise Science Department of the English Football Association for funding this research.

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## Influence of cold-water immersion on indices of muscle damage following prolonged intermittent shuttle running

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(Accepted 21 August 2006)

### Abstract

The aim of this study was to assess the effects of cold-water immersion (cryotherapy) on indices of muscle damage following a bout of prolonged intermittent exercise. Twenty males (mean age 22.3 years,  $s = 3.3$ ; height 1.80 m,  $s = 0.05$ ; body mass 83.7 kg,  $s = 11.9$ ) completed a 90-min intermittent shuttle run previously shown to result in marked muscle damage and soreness. After exercise, participants were randomly assigned to either 10 min cold-water immersion (mean 10°C,  $s = 0.5$ ) or a non-immersion control group. Ratings of perceived soreness, changes in muscular function and efflux of intracellular proteins were monitored before exercise, during treatment, and at regular intervals up to 7 days post-exercise. Exercise resulted in severe muscle soreness, temporary muscular dysfunction, and elevated serum markers of muscle damage, all peaking within 48 h after exercise. Cryotherapy administered immediately after exercise reduced muscle soreness at 1, 24, and 48 h ( $P < 0.05$ ). Decrements in isometric maximal voluntary contraction of the knee flexors were reduced after cryotherapy treatment at 24 (mean 12%,  $s_x = 4$ ) and 48 h (mean 3%,  $s_x = 3$ ) compared with the control group (mean 21%,  $s_x = 5$  and mean 14%,  $s_x = 5$  respectively;  $P < 0.05$ ). Exercise-induced increases in serum myoglobin concentration and creatine kinase activity peaked at 1 and 24 h, respectively ( $P < 0.05$ ). Cryotherapy had no effect on the creatine kinase response, but reduced myoglobin 1 h after exercise ( $P < 0.05$ ). The results suggest that cold-water immersion immediately after prolonged intermittent shuttle running reduces some indices of exercise-induced muscle damage.

**Keywords:** Cryotherapy, intermittent exercise, muscle soreness, muscular dysfunction

### Introduction

The deleterious effects associated with muscle damage following a bout of unaccustomed or eccentric-based exercise are well documented (Armstrong, 1984; Clarkson & Sayers, 1999; Proske & Allen, 2005). The time course and severity of muscle soreness, muscular dysfunction, and appearance of markers of muscle damage in the systemic circulation can vary considerably depending on the duration, intensity, and type of exercise performed (Clarkson, Byrnes, McCormick, Turcotte, & White, 1986; Eston, Critchley, & Baltzopoulos, 1994; Thompson, Nicholas, & Williams, 1999). These factors may partially explain why the precise aetiology of exercise-induced muscle damage remains elusive. Nevertheless, delayed-onset muscle soreness (DOMS) and associated decrements in muscular function are one of the most commonly reported sport-related injuries (Byrne, Twist, & Eston, 2004).

Many investigations have attempted to alleviate or prevent exercise-induced muscle damage and its associated symptoms. Treatment strategies include stretching, ultrasound, massage, antioxidant supplementation, and administration of non-steroidal anti-inflammatory drugs (for a review, see Cheung, Hume, & Maxwell, 2003). More recently, attention has focused on the effect of cryotherapy in aiding recovery from muscle-damaging exercise (Eston & Peters, 1999; Howatson and van Someren, 2003; Yanagisawa *et al.*, 2003a,b). The role of cryotherapy as a treatment of sport-related injuries is well documented (Bleakley, McDonough, & MacAuley, 2004), although support for its specific application to exercise-induced muscle damage remains predominantly anecdotal.

Cryotherapy is proposed to reduce the inflammatory response to injured tissue as well as decrease oedema, haematoma formation, and pain (Swenson,

Sward, & Karlsson, 1996). Thus, cryotherapy may be considered a pertinent treatment modality because inflammation is integral in the aetiology of exercise-induced muscle damage (Smith, 1991) and muscle soreness is the most commonly reported symptom of this exercise-related injury (Armstrong, 1984). Additionally, inflammation has been shown to exacerbate existing disruptions to skeletal muscle tissue, as this immune response is coupled with secondary damage via transient hypoxia as well as the non-specific cytotoxic actions of leukocytes (Lapointe, Frenette, & Cote, 2002; MacIntyre, Reid, Lyster, Szasz, & McKenzie, 1996; Merrick, Rankin, Andres, & Hinman, 1999).

Recent research has focused on the role of cryotherapy on indices of muscle damage following eccentric exercise of isolated muscle groups. Eston and Peters (1999) observed that repeated cold-water immersion (15 min at 15°C every 12 h) was effective in reducing plasma creatine kinase activity and muscle stiffness, indirectly assessed as relaxed arm angle, in the days after repeated eccentric elbow flexion. Using a comparable muscle-damaging exercise protocol, Yanagisawa and co-workers (2003a,b) also reported some beneficial effects of cold-water immersion (15 min at 5°C) on exercise-induced muscle oedema as well as a tendency for reduced muscle soreness and creatine kinase activity. Conversely, Isabell, Durrant, Myrer, and Anderson (1992) observed no effect of cryotherapy (ice-massage) on indices of muscle damage and suggested repeated cryotherapy may be contra-indicatory over a prolonged period.

There is limited evidence to support cryotherapy following more dynamic whole-body exercise, which may be considered more ecologically valid when providing recommendations in a sports performance environment. The aim of this study was to assess the effects of a single administration of cryotherapy on the recovery from a bout of strenuous intermittent shuttle-running exercise.

## Methods

### Participants

Twenty healthy men (mean age 22.3 years,  $s = 3.3$ ; height 1.80 m,  $s = 0.05$ ; body mass 83.7 kg,  $s = 11.9$ ) volunteered to take part in the study, which had received approval from the university ethics committee. Participants completed a mandatory health questionnaire and provided written informed consent. All participants were habitually active in a variety of sports, but were unfamiliar with the exercise to be performed. Participants were required to abstain from therapeutic treatments including

massage and anti-inflammatory drugs for the duration of the investigation.

### Experimental design

Having refrained from exercise for at least 2 days, participants arrived at the laboratory in a fasted state (~10 h). A venous blood sample (~10 ml) was taken from a vein in the antecubital fossa after participants had been supine for at least 10 min. Next, perceived muscle soreness was recorded and muscular function was assessed using isokinetic dynamometry and a vertical jump test (described in detail below). Subsequently, participants completed the Loughborough Intermittent Shuttle Test (LIST) as described previously (Thompson *et al.*, 1999). Briefly, the LIST is a field test specifically designed to replicate the demands associated with intermittent activity such as soccer (Nicholas, Nuttall, & Williams, 2000). Participants were required to exercise at varying intensities for 90 min, with average exercise intensity equal to 75% maximal oxygen uptake ( $\dot{V}O_{2max}$ ) determined from a progressive shuttle-run test (Ramsbottom, Brewer, & Williams, 1988). Subjective ratings of perceived exertion were recorded every 15 min during the LIST (Borg, 1998), heart rate was monitored every 15 s by short-range telemetry (Polar 8810, Vantaa, Finland), and core body temperature was monitored at regular intervals using an ingestible thermometer pill (CorTemp<sup>TM</sup>, HQI, Palmetto, USA). Nude body mass was determined immediately before and after exercise. Participants were required to ingest water in a bolus equal to 5 ml · kg<sup>-1</sup> immediately before exercise and 2 ml · kg<sup>-1</sup> every 15 min during exercise. A venous blood sample was taken immediately after exercise and additional samples were taken 1, 24, and 48 h after exercise. Participants were instructed not to resume exercising until the conclusion of testing.

### Cryotherapy treatment

Before exercise, participants were matched for several anthropometric and physiological characteristics and randomly allocated to either a cryotherapy or control group (Table I). Immediately after exercise, the cryotherapy group immersed their lower limbs (ensuring that the iliac crest was fully submerged) in a cold-water bath for 10 min. The water was maintained at a mean temperature of 10°C ( $s = 0.5$ ) by the addition of crushed ice and was repeatedly agitated to avoid the formation of a warmer boundary layer. This single bout of cryotherapy was similar to that used in previous investigations (Yanagisawa *et al.*, 2003a,b) and has been shown to lower subcutaneous and intramuscular temperature

Table I. Physiological characteristics and physical activity status of groups (mean  $\pm$  s).

	Cryotherapy (n = 10)	Control (n = 10)	P-value
Age (years)	23.6 $\pm$ 4.1	21.7 $\pm$ 2.0	0.123
Height (m)	1.80 $\pm$ 0.06	1.81 $\pm$ 0.05	0.665
Body mass (kg)	85.9 $\pm$ 12.8	81.5 $\pm$ 11.2	0.517
Body mass index (kg $\cdot$ m <sup>-2</sup> )	26.3 $\pm$ 2.8	24.9 $\pm$ 2.7	0.487
Sum of 4 skinfolds (mm) <sup>a</sup>	35.3 $\pm$ 12.8	31.3 $\pm$ 6.3	0.583
$\dot{V}O_{2max}$ (ml $\cdot$ kg <sup>-1</sup> $\cdot$ min <sup>-1</sup> )	55.2 $\pm$ 4.8	56.2 $\pm$ 5.3	0.676
Weekly exercise sessions (n)	5 $\pm$ 2	4 $\pm$ 1	0.265

<sup>a</sup>Sum of four skinfolds (triceps, biceps, suprailiac, subscapular).

by 7–10°C (Meeusen & Lievens, 1986). During this time, control participants remained at rest in the same long seated position as the experimental participants. Heart rate and core body temperature were monitored at regular intervals throughout and for 15 min following the treatment period. Additionally, ratings of perceived coldness were assessed during treatment and recovery using a visual analog scale that ranged from 1 ("not cold") to 10 ("very, very cold").

#### Assessment of muscle damage

Ratings of perceived soreness were assessed using a visual analog scale (Thompson *et al.*, 1999) ranging from 1 ("not sore") to 10 ("very, very, sore") before, immediately after ( $\pm$  5 min), and 1, 24, 48, and 168 h after exercise. Participants rated general whole-body soreness while standing in the relaxed state and were encouraged to palpate major muscle groups during assessment.

Maximal voluntary isometric contraction (MVC) of the knee extensors and flexors was assessed while seated using an isokinetic dynamometer (Cybex model 770, LUMEX Inc., Ronkonkoma, USA). Participants were familiarized with the apparatus and protocol on at least two occasions before performing the LIST. Before assessment on the dynamometer, positional adjustments for knee extension and flexion were made to ensure movement was restricted to the sagittal plane and that the axis of rotation passed through the femoral condyles. Following a warm-up set of five sub-maximal repetitions of knee extension and flexion (1.05 rad  $\cdot$  s<sup>-1</sup>), participants completed two maximal isometric repetitions of the dominant limb for 5 s for extension at 1.05 rad and flexion at 0.35 rad, where full knee extension was 0 rad. These angles have previously been identified as optimal for peak force generation during isometric knee flexion and extension (Westing & Seger, 1989).

Contractions were separated by 60-s rest periods. Participants were verbally encouraged and received visual feedback during each repetition. The greatest peak torque achieved from both repetitions was recorded.

Vertical jump height was recorded as previously described (Byrne & Eston, 2002). Participants performed the squat jump technique with no countermovement to minimize the effects of the stretch-shortening cycle. Participants performed three consecutive jumps on an electronic timing mat (Powertimer 1.0 Testing System, Newtest Oy, Kiviharjuntie, Finland) on each occasion. Jumps were separated by 60 s rest and the highest jump was recorded as the peak height.

Sprint performance was assessed during the LIST and again 48 h after exercise. Sprint times were measured using two infrared photoelectric cells (RS Components Ltd., Zurich, Switzerland) interfaced with a computer. Participants were required to perform 11  $\times$  15-m maximal sprints during each 15-min exercise block of the LIST. The values recorded during the first 15-min block of the LIST were compared with a subsequent 15-min block performed 48 h after the initial exercise bout.

#### Blood analysis

Aliquots of blood were used to determine haemoglobin concentration by the cyanomethaemoglobin method (Boehringer Mannheim, GmbH Diagnostica, Mannheim, Germany) and haematocrit by microcentrifugation (Hawksley Ltd., Lancing, UK). Changes in plasma volume were assessed using these haematocrit and haemoglobin values (Dill & Costill, 1974). The remaining blood was dispensed into a tube, left to clot, and then centrifuged (4°C) at 4000 rev  $\cdot$  min<sup>-1</sup> for 10 min to obtain serum. Serum creatine kinase activity and myoglobin concentration were determined at 37°C using commercially available techniques (Randox, Crumlin, UK) designed specifically for use on an automated system (COBAS Mira Plus, Roche Diagnostics Systems, Rotkreuz, Switzerland).

#### Statistical analysis

A two-way analysis of variance (ANOVA) with repeated measures on time was used to determine if differences existed between treatment conditions. When significant *F* values were observed, the Holm-Bonferroni step-wise method was used to determine the location of the differences (Atkinson, 2002). Values for creatine kinase activity and myoglobin were not normally distributed and therefore these values were log transformed before ANOVA. Pearson product-moment correlations were used to

examine the relationship between variables. Data analysis was conducted using SPSS version 12.0 and statistical significance was set at  $P < 0.05$ . Values are expressed as means and standard errors of the mean ( $s_x$ ) unless otherwise stated.

## Results

### Response to intermittent exercise

Mean heart rate during the LIST was 165 beats  $\cdot$  min $^{-1}$  ( $s_x=3$ ) for both groups. Mean rating of perceived exertion increased from 14 ( $s_x=1$ ) at 15 min into exercise to 17 ( $s_x=1$ ) at the end of exercise for both groups ( $P < 0.05$ ). Core body temperature during exercise was available for 15 participants (cryotherapy,  $n=8$ ; control,  $n=7$ ). Temperature increased from 37.5°C ( $s_x=0.10$ ) to 38.1°C ( $s_x=0.13$ ) after exercise ( $P < 0.05$ ). During exercise, participants drank 1.3 litres ( $s_x=0.1$ ) of water and lost 1.2 kg ( $s_x=0.3$ ) of body mass. Mean sprint time during the LIST was 2.70 s ( $s_x=0.03$ ). Estimated changes in plasma volume did not differ during the testing period for either group.

### Response to cryotherapy treatment

Heart rate decreased during the treatment period from 107 beats  $\cdot$  min $^{-1}$  ( $s_x=4$ ) to 94 beats  $\cdot$  min $^{-1}$  ( $s_x=3$ ) ( $P < 0.05$ ) and continued to decline (87 beats  $\cdot$  min $^{-1}$ ,  $s_x=3$ ) 15 min after treatment ( $P < 0.05$ ) in both groups. Cryotherapy had no effect on heart rate response when compared with the control group. Core body temperature ( $n=15$ ) decreased from 37.9°C ( $s_x=0.14$ ) to 37.7°C

( $s_x=0.13$ ) during the treatment period and continued to fall 15 min post-treatment (37.4°C,  $s_x=0.11$ ) ( $P < 0.05$ ) but was not different between groups. Perception of coldness was elevated during cryotherapy (mean 6,  $s_x=1$ ) compared with the control group (mean 1,  $s_x=1$ ) and remained elevated during recovery ( $P < 0.05$ ).

### Indices of muscle damage

Exercise resulted in severe muscle soreness that peaked immediately after exercise and again 24 h later ( $P < 0.05$ ). Cryotherapy reduced ratings of perceived soreness at 1, 24, and 48 h post-exercise ( $P < 0.05$ ) (Figure 1).

Maximal isometric voluntary contraction for knee extension was unaffected after exercise and treatment. However, MVC for knee flexion was reduced at 24 and 48 h post-exercise ( $P < 0.05$ ) and returned to pre-exercise values at 168 h post-exercise ( $P < 0.05$ ). Cryotherapy reduced decrements in MVC at 24 and 48 h compared with the control group ( $P < 0.05$ ) (Figure 2).

Peak vertical jump height was reduced from pre-exercise values (0.36 m,  $s_x=0.01$ ) at 24 (0.35 m,  $s_x=0.01$ ) and 48 h (0.34 m,  $s_x=0.01$ ) for both groups ( $P < 0.05$ ). Vertical jump height was unaffected by cryotherapy. Mean sprint time during the first 15-min block of the LIST (2.67 s,  $s_x=0.03$ ) was unaffected 48 h (2.70 s,  $s_x=0.04$ ) after exercise and treatment.

Creatine kinase activity was elevated immediately after exercise ( $P < 0.05$ ), peaking 24 h later but this response was not influenced by cryotherapy (Figure 3). Myoglobin concentration increased

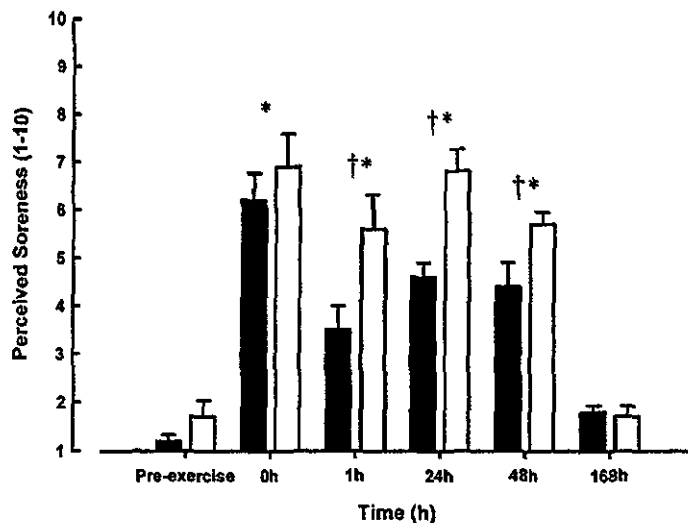


Figure 1. Perceived muscle soreness following exercise for cryotherapy (solid bars) and control (open bars) groups. Values are mean and standard errors. \*Different from pre-exercise for both groups ( $P \leq 0.05$ ); †Different between groups ( $P \leq 0.05$ ).

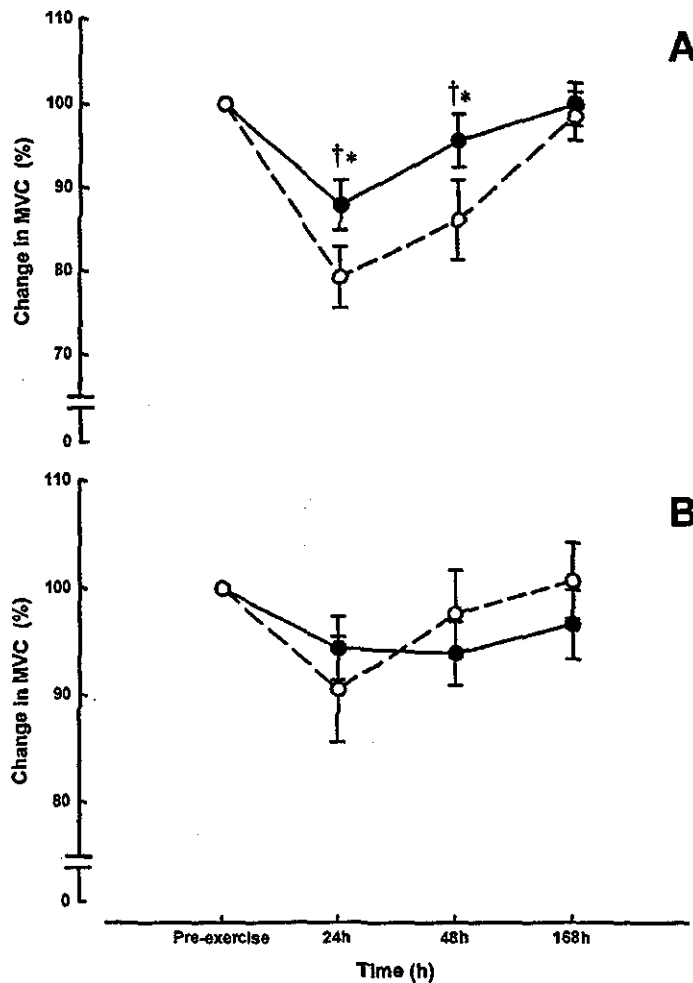


Figure 2. Isometric maximal voluntary contraction of the knee flexors (A) and extensors (B) following exercise for cryotherapy (solid line) and control (broken line) groups. Values are mean and standard errors. \*Different from pre-exercise for both groups ( $P \leq 0.05$ ); †Different between groups ( $P \leq 0.05$ ).

immediately after exercise in both groups ( $P < 0.05$ ). Concentrations peaked 1 h after exercise in the control group but were reduced at this time in the cryotherapy group ( $P < 0.05$ ) (Figure 3).

### Discussion

The main findings of this study were that individuals who received cryotherapy treatment after exercise reported a diminished perception of muscle soreness up to 48 h later, a lower decrement in MVC at both 24 h and 48 h post-exercise, and a reduced serum myoglobin response 1 h after exercise. These findings are consistent with those of similar investigations using cryotherapy as a modality to treat exercise-induced muscle damage (Eston & Peters, 1999; Howatson & van Someren, 2003; Yanagisawa *et al.*, 2003b).

The intermittent shuttle-running protocol used to elicit muscle damage resulted in severe muscle

soreness and an associated period of muscular dysfunction comparable to that previously documented (Bailey *et al.*, 2002; Bailey, Williams, Hurst, & Powell, 2003; Thompson *et al.*, 1999). Additionally, the increase in intracellular proteins was similar and over the same time course as observed in previous investigations using both this exercise protocol (Bailey *et al.*, 2002; Thompson *et al.*, 1999) and other analogous eccentric-based exercise models (Byrnes *et al.*, 1985; Thompson *et al.*, 2004). The greatest soreness was generally reported in the weight-bearing musculature of the lower limbs, specifically the hamstrings (Bailey *et al.*, 2003; Thompson *et al.*, 1999), conceivably related to the eccentric actions of this muscle group during intermittent running. The moderate relationship ( $r = -0.58$ ;  $P < 0.05$ ) between decrements in MVC of the knee flexors and muscle soreness at 48 h post-exercise provides some support for the proposed association between muscle injury, dysfunction, and soreness that is not well

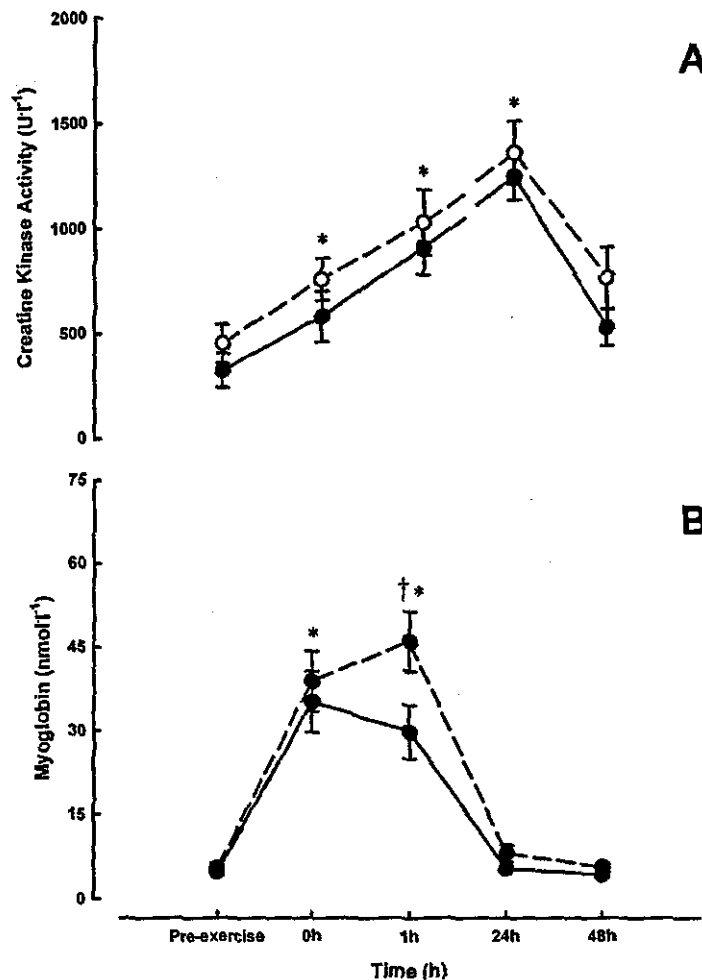


Figure 3. Serum creatine kinase activity (A) and myoglobin concentration (B) following exercise for cryotherapy (solid line) and control (broken line) groups. Values are mean and standard errors. \*Different from pre-exercise for both groups ( $P \leq 0.05$ ); †Different between groups ( $P \leq 0.05$ ).

documented (Nosaka, Newton, & Sacco, 2002; Warren, Lowe, & Armstrong, 1999).

The acute onset of muscle soreness observed immediately after exercise is related to the accumulation of by-products that are either metabolic or contraction induced (Miles & Clarkson, 1994) rather than DOMS, which is more commonly associated with muscle damage (Cheung *et al.*, 2003). This could account for the biphasic increase in muscle soreness observed following exercise and support the proposal that cryotherapy was effective in reducing muscle injury rather than facilitating removal of exercise-induced accumulation of by-products. The observed reductions in DOMS at 24 and 48 h post-exercise with cryotherapy is consistent with similar previous investigations (Denegar & Perrin, 1992; Prentice, 1982; Yanagisawa *et al.*, 2003b). Some authors attribute this reduced pain perception to the analgesic effects of cooling rather than inhibition of

muscle damage (Denegar & Perrin, 1992; Gulick, Kimura, Sittler, Paolone, & Kelly, 1996; Meeusen & Lievens, 1986). The application of cold, sufficient to lower muscle tissue to temperatures around 10–15°C, reduces nerve conduction velocity, muscle spindle activity, the stretch-reflex response, and spasticity, thus inhibiting the pain-spasm cycle (Meeusen & Lievens, 1986). However, the duration of this analgesia is limited to 1–3 h (Meeusen & Lievens, 1986), so this mechanism might only account for the initial reductions in muscle soreness observed 1 h after exercise. Denegar and Perrin (1992) observed similar beneficial effects of cryotherapy (ice packs) on DOMS. These authors documented a further reduction in perceived soreness when the treatment was supplemented with a period of stretching. They proposed that stretching results in stimulation of the Golgi tendon organ, motor inhibition, and reduced muscular tension resulting in



a concurrent reduction in the pain-spasm cycle (Denegar & Perrin, 1992). Although cooling, either alone or accompanied by passive stretching, has inhibitory influences on pain perception, some researchers reporting beneficial effects of cryotherapy on exercise-induced muscle damage have not observed a concomitant effect on muscle soreness (Eston & Peters, 1999; Howatson & van Someren, 2003).

Cryotherapy improved recovery of MVC of the knee flexors 24–48 h after exercise. Exercise resulted in a reduction of knee flexion peak torque at 24 (12%,  $s_x=4$ ) and 48 h (3%,  $s_x=3$ ) in the cryotherapy group, which was markedly less than that experienced by the control group at 24 (21%,  $s_x=5$ ) and 48 h (14%,  $s_x=5$ ). Values had returned to pre-exercise values 7 days after exercise in both groups. This pattern of strength loss and recovery is similar to that previously reported following this exercise protocol (Bailey *et al.*, 2002; Thompson *et al.*, 1999), although decrements were lower compared with previous studies (Bailey *et al.*, 2002; Thompson *et al.*, 2003). Additionally, these findings provide further support for the use of muscle function as an applicable and reliable measurement tool for quantifying exercise-induced muscle damage (Warren *et al.*, 1999). However, Warren and co-workers' (1999) endorsement of specificity when measuring muscle function was not supported, as assessment of isometric maximal voluntary contraction was more sensitive to decrements in muscular function than sprint and vertical jump assessments.

The effects of cryotherapy on the appearance of intracellular proteins are similar to those reported previously (Eston & Peters, 1999; Howatson & van Someren, 2003). It is still unclear what mechanism is responsible for the difference in myoglobin concentration following cryotherapy treatment. Others have postulated that cryotherapy might reduce post-exercise muscle damage via a decreased permeability of blood and lymph vessels due to an attenuated inflammatory response. These investigations employed creatine kinase activity as the sole marker for intracellular protein release (Eston & Peters, 1999; Howatson & van Someren, 2003). This particular marker is subject to large variability between individuals and caution is advised when interpreting the response of this intracellular protein (Clarkson & Ebbeling, 1988; Warren *et al.*, 1999). This explanation could, in part, account for the lack of a treatment effect observed with creatine kinase activity. Also, as secondary damage to skeletal muscle resulting from inflammation may be more pronounced in the hours rather than days after exercise (Lapointe *et al.*, 2002; Merrick *et al.*, 1999), it is possible that myoglobin is a more accurate indicator of subsequent injury. Although cryotherapy

treatment had no effect on core body temperature compared with the control group, as cooling rates were  $0.03^{\circ}\text{C} \cdot \text{min}^{-1}$  ( $s_x=0.01$ ) for both groups, previous investigations have reported reductions in subcutaneous and intramuscular temperatures during similar cryotherapy treatments (for a review, see Meeusen & Lievens, 1986). Therefore, it is reasonable to assume that cold-water immersion was effective in lowering intramuscular temperature. With this in mind, it is possible that cryotherapy mediated a reduced inflammatory response and subsequent secondary muscle damage attenuating the efflux of myoglobin. However, it is also conceivable that cold-water immersion elicited profound haemodynamic changes (Stocks, Taylor, Tipton, & Greenleaf, 2004) that could provide an alternative explanation for the differing appearance in this systemic marker of muscle damage.

The results of this study suggest that cryotherapy applied as a single bout of cold-water immersion immediately after exercise is effective in reducing some of the deleterious symptoms associated with exercise-induced muscle damage. The precise mechanisms responsible for this benefit requires further clarification but perhaps highlights the multitude of factors involved in the aetiology of exercise-induced muscle damage.

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