THE EFFECTS OF LEG PRE-COOLING ON THE KINETICS OF HEAT EXCHANGE BETWEEN CORE AND PERIPHERAL BODY COMPARTMENTS PRIOR TO INTERMITTENT SPRINT EXERCISE IN THE HEAT

A thesis submitted in partial fulfilment of the requirements for the award of

MASTER OF PHILOSOPHY

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By

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Firstly, this thesis is dedicated to my Mum and Dad who have supported me throughout my postgraduate journey.

Dedication is also to my amazing girlfriend who has been there from the moment our own journey started together.

“el único límite en mi vida es eso de mi imaginación”

“Perhaps the greatest stress ever imposed on the human cardiovascular system is the combination of exercise and hyperthermia. Together these stresses can present life threatening challenges, especially in highly motivated athletes who drive themselves to extremes in hot environments.”

Loring Rowell, Cardiovascular Physiologist, 1986.
ABSTRACT

Pre-cooling of the thigh muscles is an ergogenic method that can alleviate systemic heat strain during intermittent sprint performance; nevertheless the physiological mechanisms that explain these benefits are not fully understood. This thesis aimed to evaluate the effects of upper leg cooling on the kinetics of heat exchange within the body and investigate how intermittent sprint exercise performance, that mimics the physiological demands of team sports, can be optimised, in a hot, humid environment (~ 30 °C, 50 % relative humidity).

Study 1 determined the reliability of the Cycling Intermittent Sprint Protocol (CISP) by comparing two CISPs completed on two separate days in temperate conditions. The typical error of measurement (TEM) and intra-class correlation (ICC) for peak power output (PPO) from all twenty sprints between CISPs was 2.9 % and 0.96, respectively. TEM and ICC for mean power output (MPO) for all twenty sprints between CISPs was 4.2 % and 0.9, respectively. This test-retest research design suggests PPO to provide a more reliable measurement than MPO data during the CISP and supports the CISP as a reliable tool for assessing intermittent sprint exercise performance changes.

Study 2 investigated the time course of heat exchange between the peripheral “shell”, local muscle tissue and the whole body “core” compartments as a result of upper leg cooling. In the absence of changes in core temperature, 25 min of leg cooling changed skin and muscle temperature by 24 °C and 15 °C, respectively, with these changes being strongly correlated ($r = 0.974, P < 0.01$). Changes in temperature within the body occurred in the early phases of cooling; skin temperature reduced by 76 % within the first 2 minutes at a rate of 0.98 °C per minute and muscle temperature by 53 % in the first 4 minutes, with no changes from 14 minutes thereafter. Individuals with a greater thigh circumference took longer to reach a muscle temperature nadir as a result of cooling.

Study 3 investigated the optimal duration of cooling (10, 15, 20 or 25 minutes) that maximises intermittent sprint exercise performance in the heat. As a result of 15 minutes of cooling, average PPO and total work improved compared to that of the control by 3.75 % and 6.7 %, respectively, whereas average PPO and total work were the poorest of all conditions following 25 minute of cooling. The slowest rate of core temperature increase was also observed in the 15 minute cooling condition compared to the control.
Study 4 utilised the data collected within this thesis to test the accuracy of a prediction of muscle temperature using skin temperature (de Ruiter et al, 1999) against measured muscle temperature. As skin temperature reduced from normothermic values, the error of the muscle temperature prediction against measured muscle temperature increased, with differences of over 11 °C observed at lower temperatures. The smallest variation between measured and predicted muscle temperature was post exercise in the heat, however skin and muscle temperature at these time points were not correlated ($r = 0.277, P > 0.05$), further questioning the accuracy of the equation currently in the literature under these conditions.

The data presented within this thesis demonstrates that the CISP is a reliable tool for examining changes in intermittent sprint exercise performance and permits the evaluation of cooling interventions. The greatest ergogenic benefit in intermittent sprint exercise performance was observed following 15 minutes of upper leg cooling and this coincided with a maximal capacity to cool the muscle, even when longer durations of cooling were compared. It is suggested that improved intermittent sprint performance from local leg cooling is not due to an immediate drop in core temperature that creates increased capacity for body heat storage, rather local changes to the thermal environment of the thigh area being cooled that results in an altered rate of core temperature increase.
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### LIST OF ABBREVIATIONS

~ “approximately”

△ “change in”

°C Degrees Celcius

°⁻¹ “per”

% Percent

ANOVA Analysis of Variance

ATP Adenosine Triphosphate

BHC Body Heat Content

BM Body Mass

CI Confidence Interval

CISP Cycling Intermittent Sprint Protocol

CIVD Cold-Induced Vasodilation

cm Centimetre

CV Coefficient of Variation

ES Effect Size

h Hour

HR Heart Rate

ICC Intra-class Correlation

IET Intermittent Exercise Test

ISEP Intermittent Sprint Exercise Protocol

ISSR Intermittent Sprint Shuttle Running represents

J Joules

kg Kilograms

kJ Kilojoules

km kilometres

LIST Loughborough Intermittent Shuttle Test

LOA Limits of Agreement

m Meter

min Minute

ml Millilitre

mm Millimetre

MPO Mean Power Output
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<td>Physiological Strain Index</td>
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<tr>
<td>r</td>
<td>Correlation Coefficient</td>
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<tr>
<td>RH</td>
<td>Relative Humidity</td>
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<td>RPE</td>
<td>Rating of Perceived Exertion</td>
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<td>RSA</td>
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<td>PPO</td>
<td>Peak Power Output</td>
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<td>s</td>
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Finally, to all those that may not have been mentioned; housemates, friends and colleagues alike, that may have had an influence on my time and studies at university. Cheers!
DECLARATION

The material contained within the writing of this thesis represents the original work of the author. All data that has been used to develop conclusions for each study, has been primarily collected and analysed by the author. The following published communications of work have been due to research contained within this thesis:

Journal Articles


Peer-Reviewed Conference Proceedings


I declare that the research contained in this thesis, unless otherwise formally indicated within the text, is the original work of the author. The thesis has not been previously submitted to this or any other university for a degree, and does not incorporate any material already submitted for a degree.

Signed:

Date:
CHAPTER ONE

Introduction
CHAPTER I: INTRODUCTION

In November 2000, at the Fourth World Communication Conference on Sport and Society, Wladimir Andreff questioned why the hosting of major sporting events should not be shared among developing countries around the world. Since then, South Africa, Brazil and Qatar have been named as host nations to the FIFA World Championships and/or the Summer Olympics. In these countries, the environmental conditions during the time of year of competition may reach up to 40 °C (Qatar) and over 70 % relative humidity (Brazil) (www.weatheronline.co.uk). From an athletes’ perspective, climate in these countries provides further demands on the body beyond that already associated with elite level sport. The effect of ambient temperature on exercise performance is documented in the literature (Galloway and Maughan, 1997), with a large body of research focusing on the effects of heat stress on endurance-based protocols. Nevertheless it is known that the effects of heat stress are exacerbated during intermittent sprint exercise (Nevill et al, 1995).

Homeotherms are organisms that maintain a constant body temperature almost independently of the environmental conditions surrounding them. As a species humans have learned how to tolerate some of the most challenging environmental conditions; from the hot, dry conditions in the desert through to the sub-zero temperatures of the poles. However, the motivational aspects to achieve success in athletic competition can sometimes test the limits of human thermoregulation. Body temperature is regulated at around 37 °C (Cheung, 2010), but as an individual increases their activity levels, body temperature will rise as a result of the heat produced from metabolic reactions. Rises in body temperature as a consequence of combined exogenous and endogenous heat sources trigger many thermoregulatory responses that aim to maintain homeostatic temperature (Armstrong, 2000); a level of heat stress compensability. Nonetheless, during periods of prolonged exercise, particularly in the heat, the required evaporative heat loss can exceed the rate of evaporative cooling and consequently heat stress becomes uncompensable (Taylor and Cotter, 2006). The shift from compensability to uncompensability is influenced by numerous factors, not least thermal gradients, exercise intensity and body composition (Taylor and Cotter, 2006) and so manipulation of such influences can be pivotal in sporting performance in hot, humid conditions. In thermal literature, it is common for the body to be divided into 2 parts; the core and the shell. This two-compartment model is used to calculate body temperature from core and skin temperature, with the contribution of each determined through specific weighting coefficients.
(Jay and Kenny, 2007). Nevertheless, as thermal research has developed, the weighting of each of these compartments has been questioned. The inclusion of a third compartment has been proposed that incorporates tissue temperature, in particular that of the working muscle. While the weighting coefficients of a three-compartment model are still being developed, the measurement of tissue temperature is not always possible and so predictive equations have been used (de Ruiter et al, 1999; Racinais et al, 2005). Nonetheless, this data is derived within a range of temperate conditions and from a small muscle group at rest, opening a debate of its applicability during exercise under conditions of heat or cold stress.

The study of intermittent sprint exercise is becoming an increasingly popular research domain due to the capability to replicate team sport activity patterns such as football, hockey and rugby. Although the intensity and duration of each phase may vary depending on the protocol used and the sport it aims to replicate, intermittent exercise consists of a phase of maximal intensity exercise, a recovery phase and a rest phase. Due to the nature of the exercise, cardiovascular and physiological strain are increased following intermittent sprint exercise. When comparing intermittent sprint exercise to that of endurance based protocols, increases in heart rate, core temperature and muscle temperature have all been observed (Nevill et al, 1995; Castle et al, 2005; Morris et al, 2005). Furthermore when performed in hot conditions compared to temperate conditions, these responses are augmented (Castle et al, 2005). Kranning and Gonzalez (1991) reported a 33% faster rise in core temperature during intermittent exercise patterns during uncompensable heat stress. Within the literature, there are multiple tests of intermittent or repeated sprint ability; for example the Loughborough Intermittent Shuttle Test (LIST; Sunderland and Nevill, 2005) and the Soccer-specific Intermittent Exercise Test (SSIET; Oliver et al, 2007). Likewise, the Cycling Intermittent Sprint Protocol (CISP; Castle et al, 2004) has been used during prescriptive trials for the alleviation of climatic heat stress (Castle et al, 2006; Castle et al, 2012). Yet, there has been no reported analysis on the repeatability of these tests, despite their continued use and so it is not known if the discussions of possible mechanisms within these studies are worthwhile or irrelevant.

While body cooling post performance is already frequently used by athletes, cooling before or during prolonged exercise in the heat is suggested to alleviate the debilitating effects by increasing the body’s capacity to store heat (White et al, 2002). Nonetheless, cooling prior to performance as a solitary ergogen is not as common on the athletic scene. This is surprising
given the wealth of research demonstrating its benefits on endurance performance compared to no cooling, control trials. Reduced physiological strain, faster time trials and longer time to exhaustion are all reported following pre-cooling (Arngrimsson et al, 2004; Webster et al, 2005; Uckert and Joch, 2007; Quod et al, 2008). However, the effects of pre-cooling on intermittent exercise are inconsistent. This may be due to the design of the protocols, the dose of cooling provided or the method of cooling employed. Water immersion appears to be favoured within pre-cooling literature nevertheless the selection of cooling method can also be determined by practicality and purpose (Quod et al, 2006). The use of an ice vest aims to reduce core temperature, perhaps as this carries the greatest weighting in body temperature calculations, but reductions in whole body skin temperature are minimal (Cotter et al, 2001; Cheung and Robinson, 2004; Hunter et al, 2006) and so sufficient changes in thermal gradients are low. Water immersion, whole and half body alike, and isolated leg cooling cause reductions in core and skin temperature, consequently reducing body heat content (Marino and Booth, 1998; Kay et al, 1999) and muscle temperature (Booth et al, 2001; Booth et al, 2004; Castle et al, 2006), but the magnitude of these reductions may become detrimental if not monitored carefully. Despite the suggestion that pre-cooling has the greatest influence on the sub-maximal phases during repeated sprint exercise (Duffield and Marino, 2007), studies have shown improvements in intermittent sprint performance following different pre-cooling protocols, adding continued debate as to the mechanisms behind the ergogen.

Therefore the aim of this thesis was to investigate the effects of pre-performance cooling on intermittent sprint exercise while under climatic heat stress. Primarily, the pathways of heat transfer will be examined and the dose response to cooling determined, providing further knowledge that will have real-world application.

This thesis is presented in the following chapters:

**Chapter II** aims to draw on relevant literature associated with the causes of fatigue, leading onto the effects of heat stress on the body and its subsequent effects on exercise, in particular intermittent sprint performance. This chapter concludes with a discussion into the variations and advancements of pre-cooling techniques used on the athletic scene in an attempt to combat exertional heat stress.
Chapter III provides common methodology used within more than one experimental chapter.

Chapter IV introduces the first study that assesses the reliability of an intermittent sprint cycling protocol that has been previously used within the literature without previous reliability measurement. This protocol will be the chosen protocol within this thesis.

Chapter V presents the investigation of the time course of heat exchange at various sites of measurement during a single cooling bout, from the outer surface of the limb, through deep tissue temperature to core temperature of the body to develop a basis for prescription of a cooling dose.

Chapter VI investigates the possibility of a duration-dependant response to leg cooling on intermittent sprint performance in hot humid conditions. This study aims to develop the understanding of the mechanisms associated with pre-cooling as an ergogen to sporting performance.

Chapter VII draws on the data obtained from chapters V and VI to test the accuracy of an equation for predicting muscle temperature proposed within the literature against measured temperature values. From the measured data, a possible alternative is suggested that covers a greater array of temperature measures allowing for greater functionality in environmental physiology research.

Chapter VIII brings together the principle findings of each study within this thesis, discussing the fundamentals associated with pathways of heat exchange during pre-cooling and how these may be affected in a duration-dependant manner, while offering future directions for research to follow. It is concluded with how the findings in this thesis may be applied in a practical manner in the athletic environment.
CHAPTER TWO

Review of Literature
CHAPTER II: REVIEW OF LITERATURE

2.1. Introduction to the Literature Review

The following chapter addresses relevant research leading to the development of a research question and overall direction for this thesis. The literature review aims to inform the reader of how the body copes with environmental stress, with particular reference to a hot environment, through the processes of thermoregulation and how different aspects of temperature are regulated. The structure of the muscle and associated kinetics of heat exchange will be considered before moving on to the advances in theories pertaining to fatigue, while exercising in a hot environment. This covers both central and peripheral mechanisms of fatigue, discussing the mechanisms behind fatigue during intermittent sprint exercise in the heat. Theories of heat stress alleviation, specifically the techniques of pre-cooling will be presented before posing a basis for the application of isolated peripheral cooling to the exercising muscles.

2.2. Thermoregulation

2.2.1. Introduction to Thermoregulation - Maintaining Thermal Balance

Thermal balance is a dynamic equilibrium between factors affecting heat gain and heat loss, and the processes that modulate body temperature. The body is a unique self-adaptive machine that, regardless of the environment you are in or the stresses you are subjected to is constantly making adaptations to try to resume a normal homeostatic balance. Homeotherms achieve this through various physiological adaptive responses targeting homeostatic parameters converted through the sensory innervations of internal organ pathways (Kovács et al, 2005). This may be short term, through immediate endocrinal or cardiovascular responses to changes, or long term through acclimation, allowing the body to cope better under the given stressor. It is the peripheral thermal receptors located near to the skin surface that are the initial responders to the stressor, relaying sensory information to the regions of the brain; the hypothalamus and motor cortex. In turn these regions induce an appropriate response; either to dissipate heat or to conserve it, maintaining body temperature as near to 37 °C as possible. In addition to peripheral thermal receptors, cells in the hypothalamus can detect changes in blood temperature initiating synchronised responses from other regions of the hypothalamus (Iwase et al, 2002). This leads to dilation or constriction of skin vessels. It is
this blood that passes through the hypothalamus that serves as the principal resource for monitoring body warmth.

2.2.2. Thermoregulation and Exercise while under Heat Stress

During exercise, the body produces metabolic heat through contraction of the muscles. Exercise in the heat is a frequent example where the body is under external heat stress from the environment in addition to the issue of heat gain produced through normal exercise. This heat needs to be removed from the central organs and deeper tissues of the core or overheating will occur due to a disruption to the thermal balance. Increases in core temperature and heart rate are common responses to exercise in the heat (Galloway and Maughan, 1997). Furthermore, shifts in metabolism have been observed following exercise in the heat. Galloway and Maughan (1997) observed slower fat oxidation rates following exercise in warm conditions compared to temperate conditions. In the presence of hypothermic conditions, metabolism is shifted towards anaerobic form, therefore using the carbohydrate stores in the skeletal muscles quicker, limiting aspects of performance (Hubbard and Armstrong, 1989). The prime purpose of the body’s thermoregulatory mechanisms is to protect the body from drastic temperature excitation which can lead to serious illness. Effective heat dissipation depends on the speed of heat exchange from the core to the periphery and then to the surrounding environment (Hadad et al, 2004). This heat exchange, or transfer of heat within the body is controlled through sympathetic vasoconstrictor and vasodilator systems (Charkoudian, 2003) associated with peripheral vascular tone, which is thought to be governed through either core temperature, peripheral sensory input or both (Gordon, 2009). Four physical processes are involved in the production and transfer of heat throughout the body (thermogenesis) and to the environment (thermolysis); radiation, conduction, convection and evaporation (Jay and Kenny, 2007). The sun provides the earth with radiant energy through solar rays. Radiation does not require molecular contact and thus the warming effect is caused through transfer of heat energy from air to solids, in an effort to maintain a heat equilibrium within the environment. The human body can remain warm in cooler environments by absorbing reflective rays from the surrounding environment. Conduction involves the direct transfer of heat from one molecule to another through any type of medium. Internal conduction can occur within the body through transfer of heat produced from metabolism through muscle tissue, moving excess heat from the core to the
periphery and dissipating this heat from the body to the surrounding air or solid touching the skin providing the thermal gradient between the two surfaces and the molecular properties of each object allow this to occur. Convection is closely linked to conduction and involves the passing of heat through air molecules, carrying it away from the warmer object such as the body (Thellier et al, 2009). If convection, or the displacement of warm air, is slow then the rate of conduction through the body to the air will be slow due to the limited gradient produced between the surfaces (McArdle et al, 2001). On the contrary, if the rate of convection is faster, then the warm air surrounding the body will rapidly pass to the cooler air and thus heat from the body will be transferred at a quicker rate, significantly reducing body temperature. In cases of slow heat dissipation through means of conduction and radiation when ambient temperature exceeds that of body temperature, evaporative processes are employed. Evaporation occurs through respiratory pathways and the sweat glands located on the skin's surface. Studies have shown that increases in body temperature either as a result of exercise or environmental heat stress increase sweat gland recruitment (Buono and Connolly, 1992; Buono and Maupin, 2003). An increased sweat production assists in limiting body temperature rises through cooling of surface skin blood which returns to the deeper tissues. Additionally, water vapour (or sweat) evaporates into the environment, although this rate of evaporation is dependent on the levels of humidity and wind velocity in the surrounding environment (Hadad et al, 2004). An increased sweat rate response to cool the body has been linked to fatigue through concepts of dehydration, with water loss associated with increased stress on the cardiovascular system (Gonzalez-Alonso et al, 1997), circulatory system through reduced blood volume (Nielsen, 1984) as well as reduced cognitive function (Cian et al, 2001). Responses to exercise in the heat and fatigue are further discussed in section 2.4.

2.2.3. Thermoregulation and Exercise while under Cold Stress

Under conditions of cold stress, thermoregulation aims to preserve heat loss through the reverse of many of the mechanisms associated with heat dissipation, as well as increase heat production within the body. It has been proposed that three types of thermoregulatory responses to exposure to the cold occur; cold habituation, metabolic responses or insulative responses (van Marken Lichtenbelt et al, 2001) with these responses occurring on an acute or chronic scale depending upon the length of exposure (Young, 1996). Exercise is the most consciously used technique to increase heat production, however the onset of fatigue induced
through exercise combined with non-shivering and shivering thermogenesis has been observed through increases in energy expenditure (Haman et al, 2002). Under conditions of cold stress, frequent muscular contractions in the form of shivering have been shown to increase heat production between 15 - 50% (Ivanov, 2006). Vasomotor responses occur upon cold exposure, causing vasoconstriction of peripheral blood vessels reducing the transfer of heat from the core to the periphery (Young and Castellani, 2003). Hiramatsu et al (2007) observed significant reductions in peripheral skin blood flow and elevated blood pressure and noradrenalin levels following immersion of the hand into cold water.

Regardless of whether it is heat or cold stress, transfer gradients need to be balanced to maintain homeostasis. Core and shell heat balance equations have been used to develop an overall heat transfer gradient equation for the whole body. This can be illustrated as follows:

**Equation 2.1. Core and Periphery Equations of Heat Balance**

\[
S_{\text{core}} = M - (W_k) - (H) - \text{REHL}
\]

\[
S_{\text{shell}} = H - (E) - (C) - (R) - (K)
\]

(Blatteis et al, 2001)

**Equation 2.2. Combined Equation of Heat Balance for the whole body.**

\[
S_{\text{total body}} = M - (\pm W_k) \pm E \pm R \pm C \pm K \quad [\text{W/m}^2]
\]

where \( S \) = heat storage (positive ‘\( S \)’ represents heat gain and negative ‘\( S \)’ represents heat loss), \( M \) = metabolic heat production, \( W_k \) = energy produced or lost through external work (concentric or eccentric), REHL = respiratory evaporative heat lost, \( H \) = heat transfer through conductive and convective heat flow, \( E \) = evaporation, \( R \) = radiation, \( C \) = convection and \( K \) = conduction

(Young and Castellani, 2001).
2.2.4. Body Temperature Measurements

2.2.4.1. Core Temperature

Core temperature is an important measure for both survival purposes and to aid in the understanding of the complex physiological responses to exercise and environmental stresses that can be used to influence performance. The brain is a complex structure that has multiple regions which serve differing purposes. Located outside of the cerebrum (the area of the brain that controls voluntary movement) is the hypothalamus. One function of the hypothalamus is detecting changes in body temperature and instigating changes in thermoregulation (Iwase et al, 2002). A group of specialised neurons act like an internal body thermostat, with warm and cold sensors within the preoptic region monitoring temperatures around their set point. Warm receptors stimulate heat loss mechanisms when their temperature exceeds 37 °C, whereas by contrast, cutaneous afferents stimulate heat retention and heat generating mechanisms such as vasoconstriction and shivering at temperatures below 33 °C (Paulev and Zubieta, 2004).

![Figure 2.1. Location of the hypothalamus within the brain, and more specifically the region responsible for thermoregulation.](image)
Within the literature there are numerous sites that have been frequently utilised for measurement of core temperature, the most common of which are rectal and oesophageal measurement (Moran and Mendal, 2002). Gastro-intestinal techniques through ingestion of electronic sensor capsules (Sparling et al, 1993; Chapon et al, 2012) and aural/tympanic temperature (Easton et al, 2007) have also been documented. Naturally, each site may provide slightly different readings with variation within the body (Chapon et al, 2012) and circadian rhythm (Reilly and Waterhouse, 2009) both influencing values. Chapon et al (2012) observed differences in time of day despite the measurement technique used, with asymptote values 0.1 °C higher in the morning compared to the evening session. Despite higher asymptote values, it was reported that rectal temperature values rose slower than capsule temperature during exercise. However, a large disadvantage of the ingestible pill is consistency of location along the tract due to gastro-intestinal motility (Auwerda et al, 2001), which may cause differences in values, particularly during higher intensity activity. Similar to Chapon et al (2012), Kolka et al (1993) had previously observed slower body temperature responses with rectal temperature measurement when compared to the telemetry pill. Conversely, Sparling et al (1993) noticed higher values of rectal temperature measurement when compared to the ingestible capsule in both resting and exercising trained cyclists. Although Kolka et al (1993) deemed the pill method clinically acceptable, mobility within the body was suggested to be a factor that influences temperature variations, supporting Auwerda et al’s (2001) concerns.

Easton et al (2007) investigated the differences between rectal, telemetry pill and infra-red tympanic membrane temperature measurement during exercise heat stress with results showing no significant differences between rectal temperature and pill temperature measurement throughout the duration of exercise. At rest and during the early stages of the protocol, tympanic temperature was not different from rectal temperature either, yet as exercise duration increased and core temperature surpassed 37.5 °C, tympanic temperature values were significantly lower than that of rectal temperature (Figure 2.2). These differences were suggested to be due to a possible selective brain cooling mechanism employed as a result of heat stress as well as increased sweat volumes in the head region as a result of exercise. Additionally problems with tympanic thermometer methodology, such as sufficient insulation were considered, which will be a limiting factor in technique selection.
Figure 2.2. Differences between three measurement techniques of core temperature at rest and during exercise (Easton et al, 2007). * signifies difference between tympanic temperature and rectal temperature.

Kolka et al (1993) stated that oesophageal measurement detected greater changes than both telemetry pills and rectal temperature measurement, with changes per minute of up to 5 times faster than rectal temperature measurement for intense exercise. However, increased participant discomfort, tracheal cooling through inspired gases (Robinson et al, 1998) and the possibility of disruptions to respiration must be considered before this technique is used. Pulmonary arterial blood temperature is seen to provide the most accurate reflection of hypothalamic temperature however the techniques required are somewhat invasive and impractical for exercising situations (Easton et al, 2007).

Despite the differences in recorded temperature measurement, each of the techniques has advantages in their use that are counteracted in various disadvantages. Although oesophageal temperature measurement is suggested to be closest to that of pulmonary artery temperature (Robinson et al, 1998) which provides the closest reflection of hypothalamic temperature (Easton et al, 2007; Holtzclaw, 1993), it may also be considered the least comfortable of all methods. Progressions in telemetry pill development are bringing its values closer to that of the other methods however there is high variation in its location in the gastro-intestinal tract that may influence reported values. Measurement of tympanic temperature is the easiest of
the methodologies but studies have shown it to be inaccurate at times, possibly through lack of insulation or through cooling mechanisms employed by the body. The psychological aspect behind rectal temperature measurement may be considered the greatest barrier with the methodology yet, besides the delayed response to changes in body temperature, seems the least problematic of all methodologies. Robinson et al (1998) state that an understanding and consideration of the factors that influence each technique is required, so providing this is the case any technique can be used with validity.

On a brief note, the discomfort caused by oesophageal thermistors during insertion and exercise has been less tolerated by participants and consequently can have its own deleterious effect on both participants’ performance and the validity of the data (Garrett et al, 2012). Therefore the use of rectal temperature in the studies in this thesis will been opted for, especially since comparative pre-cooling and intermittent sprint literature have used similar techniques.

**2.2.4.2. Skin Temperature Measurement and Mean Surface Temperature**

In order for body temperature regulation to occur successfully, the hypothalamus requires constant afferent signals, which come from both core and peripheral regions of the body. The peripheral tissues consisting of skin and subcutaneous fat mainly of the arms and legs tend to be 5 – 6 °C cooler than the deeper core region (Campbell, 2008). The skin provides a protective insulative layer around the body and located within the skin are hot and cold thermoreceptors that detect any changes in skin temperature, sending messages via the spinal cord to the hypothalamus. Comprehensively, primary afferent nerve fibre endings containing multiple transient receptor potential ion channels detect changes in cutaneous activity (Schepers and Ringkamp, 2010). Additionally, it allows for heat exchange with the environment and the control of sweating (Nadel et al, 1971). On a practical basis, skin thermistors are placed in contact with various locations around the body, sending measurements to a data logger for each site. Early work by Hardy et al (1938) suggested that optimal skin temperature measurement derived from seven areas of the body; face/head, trunk, arm, hand, thigh, leg and foot with the mean of ten observations on the anterior, posterior and either side of the body taken. With these measurements, it was proposed that mean surface/skin temperature (MST) could be calculated via the following equation:
Equation 2.3. Mean Skin Temperature Equation by Hardy et al (1938)

\[ MST = 0.35 \, t_{\text{trunk}} + 0.19 \, t_{\text{thigh}} + 0.14 \, t_{\text{arm}} + 0.13 \, t_{\text{leg}} + 0.07 \, t_{\text{head}} + 0.07 \, t_{\text{foot}} + 0.07 \, t_{\text{hand}} \]

where \( t \) = temperature of.

Previously, Burton (1934) proposed a more simple formula incorporating merely the trunk, leg and lower arm. Ramanathan (1964) tested both formulas on three males and proposed that the weighting system should be 30 % to the upper body and 20 % to the lower body. This weighting was derived from only four sites; the chest, arms, thighs and lower legs due to the influence of each sector on changes on body temperature and skin covering active muscles, and had little variation and smaller discrepancies than the Hardy et al (1938) method. The proposed formula that is still used today is:

Equation 2.4. Current Mean Skin Temperature Equation by Ramanathan (1964)

\[ MST = 0.3 \, t_{\text{chest}} + 0.3 \, t_{\text{arm}} + 0.2 \, t_{\text{thigh}} + 0.2 \, t_{\text{leg}} \]

or

\[ MST = 0.3(t_{\text{chest}} + t_{\text{arm}}) + 0.2(t_{\text{thigh}} + t_{\text{leg}}) \]

Alternatively, non-contact infra-red thermometry has been used to measure skin temperature. Buono et al (2007) compared the validity of infra-red thermometry to contact thermistors and the results showed strong correlation between methodologies for both rest \((r = 0.95)\) and exercise \((r = 0.98)\), suggesting that non-contact methodologies could be employed in the future.

Furthermore, skin temperature has been linked to estimations of thermal sensation in order to provide information regarding an individuals’ thermal state (Choi and Loftness, 2012). The possibility of various skin temperature locations correlating with overall-body thermal sensation has been considered, although extreme temperatures can cause greater variance in measurement (Wang et al, 2007) which may be reflective of the measurement site and the influence of vasoconstriction and dilation.
2.2.4.3. Mean Body Temperature and Body Heat Content (BHC)

It has been reported that between 80 – 95 % of energy produced during exercise is released as residual heat (Jay and Kenny, 2007). Blood that perfuses active musculature gains this excess heat, which is then distributed to other regions around the body (Sawka et al, 2011). Furthermore, temperatures of the active musculature can surpass that of core temperature and the resulting heat transferred to the core via different thermal pathways, consequently causing body temperature to rise (Kenny et al, 2003). The imbalance between heat production and heat dissipation consequently affects thermal gradients that cause disruption in overall body temperature, body heat storage and the resulting body heat content. The terms body heat storage and body heat content are somewhat similar and consequently sometimes misinterpreted. Body heat storage, as described in equation 2.2 refers to the movement (convection, conduction, evaporation, radiation) thus storage of heat within the body with reference to heat balance. Body heat content is a direct consequence of this movement of heat within the body, providing you with a resulting net value for body heat, with positive body heat storage resulting in increased body heat content and a negative body heat storage resulting in decreased body heat content. In the absence of indirect and direct calorimetry which is considered the most accurate method, models of heat thermometry are used to calculate body heat content which can be used to assess the thermal imbalance of the human body under different environmental exposures and maximal heat exposure (Jay et al, 2007). Body heat content is calculated via the following equation:

**Equation 2.5. Equation of Body Heat Content**

\[
\text{Body Heat Content (Kilojoules)} = C \times \text{body mass during exercise} \times \Delta T_{\text{body}}
\]

where ‘C’ is the specific heat capacity of muscle tissue, ‘Δ’ signifies ‘change in,’ \(T_{\text{body}}\) represents the change in body temperature from start to end of exercise. Heat capacity of the tissue has been reported to be 3.47 kJ.kg\(^{-1}\).K\(^{-1}\).

(Jay and Kenny, 2007)
With the specific heat capacity of the tissue and body mass being near to constant, changes in BHC are reflective of changes in body temperature. Commonly, estimation of body temperature is achieved through a two-compartment thermometry model (Burton, 1935). This model breaks the body down into two parts, calculating the core through changes in core temperature measurement (i.e. $T_{re}$) and changes in the periphery/shell through skin temperature ($T_{skin}$) measurement and calculated as follows:

**Equation 2.6. Two-compartment Model of Body Temperature Equation**

$$T_{body} = (0.65 \times T_{re}) + (T_{skin} \times 0.35)$$

(Burton, 1935)

The weighting of core and skin temperature measurements has been suggested to depend upon the surrounding environment (Snellen, 2000) and are to equal unity at the value of 1.0, however the accuracy of the above equation has been brought into question, with studies showing that it underestimates body temperature (Snellen, 2000) by up to 35% (Jay et al, 2007) and so providing an inaccurate body heat content value. It has been suggested that this is due to the assumption of muscle tissue being considered part of the ‘core’ component despite active muscles contributing to a large amount of heat generation and so a three-compartment model incorporating a mid-region-compartment has been suggested (Jay and Kenny, 2007).

**Equation 2.7. Three-compartment Model of Body Temperature Equation, in ambient (24 °C) conditions**

$$T_{body} = (T_{re} \times 0.63) + (T_{skin} \times 0.24) + (T_{VL} \times 0.13)$$

where $T_{VL}$ is the temperature of the vastus lateralis muscle

(Jay and Kenny, 2007)
Given that muscle mass makes up a larger percentage of total body mass than skin mass, the inclusion of a muscle temperature component improves the inaccuracy of the two-compartment model. Nevertheless, review work by Jay and Kenny, (2007) states that only a 50% of the variance between calorimetry and compartment-model calculation is accounted for and that the technique of measurement may be problematic due to the invasive procedures involved, while non-invasive three-compartment models were reported to be as inaccurate.

As an alternative, an adjusted two-compartment model has been proposed that does not require the invasive muscle temperature measure. This model accounts for the underestimation of the two-compartment model (equation 2.6) through the inclusion of a correction factor (Colin et al, 1971). More specifically, this correction factor allows for changes in core temperature representing deep tissue temperature.

**Equation 2.8. Adjusted Two-compartment Model of Body Temperature Equation**

\[ T_{\text{body}} = (T_{\text{re}} \times 0.8) + (T_{\text{skin}} \times 0.2) + 0.4 \]

(Colin et al, 1971)

However, the use of this equation has been questioned. Jay et al (2007) state that the correction factor may be influenced by ambient temperatures, particularly in a hot environment resulting in the possibility of inaccurate measures, and so future research is required to test its validity.

### 2.2.5. Thermoregulation Summary

Through complex physiological adaptations, the body is constantly regulating body temperature to maintain thermostasis. Environmental heat stress; whether hot or cold, and exertional heat stress disrupts the thermal balance within the body, effecting internal body temperature which in turn may lead to environmental heat illness. During exercise, metabolic heat is produced through muscular contraction which is transferred from the core to the periphery via the blood when it is then passed to the environment through means of
evaporation. This process is assisted through vasodilation of peripheral blood vessels, which on the contrary, constrict under times of cold stress to prevent heat loss. Measuring core temperature and skin temperatures enable the monitoring of an individuals’ health as well as providing vital physiological information to exercise heat stress. There are many varying methodologies to core and skin temperature measurement with rectal temperature and skin thermistors the most commonly used. Measurement of these enables the calculation of total body temperature which has been incorporated into an equation to calculate body heat content in the absence of calorimetry. This is useful in observing thermal imbalance and maximal heat exposures. A two-compartment model involving core and shell components is widely used, yet work by Jay et al (2007) suggest that this is inaccurate and that a developing three-compartment model that includes a mid-region, or muscle aspect as a separate component, should be used instead. However models of this nature are still in their infancy with current literature still debating the optimal method for calculation of total body temperature and subsequent body heat content due to the invasive procedures required for deep tissue temperature measurement. Therefore developing a non-invasive temperature method seems to be the next step in thermometry research to assist in developing the three-compartment model.

2.3. Understanding theories of fatigue following exercise in a hot environment

2.3.1. Introduction to exercise tolerance in the heat and the possible causes of fatigue

Body temperature increase as a result of both high intensity and prolonged exercise causes a rapid rise in the body’s core temperature (von Duvillard et al, 2004), reaching a steady state when body heat production is equal to body heat loss. When an individual is subjected to hot conditions, the temperature gradient is unbalanced in favour of heat production and thus results in premature termination of the individuals’ performance (Haded et al, 2004). A classic example of this was observed by Galloway and Maughan (1997) who reported rectal temperatures of 0.7 °C higher following endurance cycle exercise in a hot (31 °C) environment compared to a temperate (21 °C) environment. Moreover, final rectal temperature in a cold (4 °C) environment produced rectal temperature values of less than 39 °C, approximately 1 °C lower than those observed in the temperate condition. However the direct causes of cessation of exercise are still widely debated. Furthermore, fatigue can be
considered a generic term and interpretation down to the researcher. Ultimately, fatigue has been defined as the inability to maintain force (Edwards, 1982) and so early research focused on muscular or peripheral fatigue. Progressions in research have seen many models of fatigue evolve, incorporating both peripheral and central mechanisms that are employed by the body in an attempt to prolong performance as much as possible. Yet some of these models are situational specific, directed at individual sporting events or specific environmental factors. A subconscious limiting or critical core body temperature has been proposed (Neilsen et al, 1993), where it has been shown that cessation of exercise at similar core temperatures may exist despite differences in climatic conditions, metabolite changes and performance improvements. Alternatively, a conscious self-pacing strategy has been suggested (Tucker, 2009) where exercise is centrally regulated. Participants will exercise at a comfortable and perhaps pre-determined pace in order to gain maximum performance outputs towards the end of performance and avoid catastrophe. This section aims to review these differing theories pertaining to fatigue.

2.3.2. Critical Core Temperature Hypothesis

Neilsen et al. (1993) first suggested that fatigue occurred at a critical limiting core temperature particularly as fatigue did not result from depletion in substrate stores or glucose breakdown, nor changes in blood flow through the muscle or skin, reductions in cardiac output or substantial lactate or potassium build up. Eight individuals exercising at 60 % maximal oxygen uptake increased time to exhaustion from 48 to 80 minutes following 9 - 12 days of heat acclimation (40 – 42 °C, 10 – 15 % RH), compared to a control group who trained for the same duration at the same predetermined intensities but in normothermic conditions (Figure 2.3). The extended exercise duration demonstrated the influence of acclimation occurring which, as evident in other literature (Patterson et al, 2004) improved sweat response and enhanced skin blood flow, thus reducing the rate of core temperature increase through increased heat loss. Nevertheless, exhaustion still occurred when participants reached the same core temperature, despite these performance improvements. Acclimation merely delayed this occurrence. Nielsen et al (1993) therefore concluded that fatigue was fundamentally caused through high core temperatures and not circulatory failure.
The following decade saw numerous studies on both animals and humans support this theory through manipulation of resting core temperatures as this was seen to be the best technique due to the small effect of on other factors associated with heat stress that may disrupt performance (Walter et al, 2000). Fuller et al. (1998) tested the effect of abdominal and brain temperatures in eight rats exercising in the heat to voluntary exhaustion. Rats were rested in a normothermic or hot environment before completing a run in a hot environment. The hot condition trials were 33 °C and 38 °C, the latter condition performed with and without pre-warming to manipulate initial core temperatures. Despite the significant higher brain and abdominal temperatures in the 38 °C pre-heating condition at resting levels, termination of exercise occurred at the same brain temperatures, even though exercise duration between trials varied (Figure 2.4). Additionally, brain temperatures were consistently higher than abdominal temperature suggesting that this was a larger determinant than abdominal temperature. Fuller et al (1998) exercised the rats to exhaustion as a result of critical levels of hyperthermia but did not induce heat illness, heat stroke and consequently death as other studies have previously done. This resulted in lower brain and abdominal temperatures than those studies and so the authors concluded that high body temperature cannot be stated as a direct cause of termination of exercise, rather a contributing factor, but did support the concept proposed by Nielsen et al (1993) that high core temperatures attenuate the ability of the central nervous system to drive the muscle.
Figure 2.4. Figure from Fuller et al (1998) showing termination of exercise at similar internal temperatures, despite varied starting temperatures. *Brain and intra-abdominal temperature of one rat plotted vs. time for exercise trials A (23 °C rest, 33 °C run), B (23 °C rest, 38 °C run), C (38 °C rest, 38 °C run). Temperatures at fatigue for each trial are indicated by the dashed line. The additional red arrows indicate the temperature at termination of exercise.*

Walters et al (2000) were bolder than Fuller et al (1998), simply stating that ‘exercise in the heat is limited by a critical internal temperature,’ although this could be because coercion was used to induce prolonged levels of exercise thus reaching a more definite critical level of fatigue. Fuller et al (1998) defined fatigue as the point at which the rats could no longer keep pace with the treadmill whereas Walters et al (2000) defined it as the ‘inability to avoid shock’ in a method to try to replicate the motivational drive of that of a highly trained endurance athlete. Although differences in methodology exist, both studies provide evidence for the critical limiting core temperature theory. Walters et al (2000) exercised eleven rats to the point of exercise termination despite shock administrant in a hot environment (35 °C). Rats started exercise with three varied initial hypothalamic and core temperatures, achieved through 2.06 GHz of microwave irradiation. Although run time after preheating was
significantly reduced, there were no differences in temperatures at exhaustion across any of the trials.

This supports that of Gonzalez-Alonso et al (1999) who conducted a study similar to that of Fuller et al. (1998) but with trained individuals, cycling at 60% maximal oxygen uptake. Participants had varying starting body temperatures (36°C, 37°C and 38°C), manipulated through water immersion at 17°C, 36°C and 40°C respectively for 30 minutes, and differing rates of body heat storage achieved through water-perfused jackets during exercise. They concluded that fatigue was caused by high internal temperatures ‘per se’ during prolonged exertional heat stress and that time to exhaustion was inversely related to initial core temperature and directly related to the rate of body heat storage.

![Simplified figure from Gonzalez-Alonso et al (1999) showing termination exercise at similar oesophageal temperature despite varied rates of heat storage](image)

**Figure 2.5.** Simplified figure from Gonzalez-Alonso et al (1999) showing of termination exercise at similar oesophageal temperature despite varied rates of heat storage

Nybo and Nielsen (2001) tested the effect of hyperthermia on central and peripheral mechanisms of neuromuscular fatigue on fourteen endurance trained cyclists. Participants performed sustained maximal voluntary contractions combined with superimposed electrical stimulation to the nervus femoralis after a hyperthermic (40°C) and a thermoneutral (18°C)
exercise trial at 60 % \( \dot{V}O_{2\text{max}} \). Reductions in central activation were lower in hyperthermia and the decline in maximal voluntary force attenuated under these conditions. Central activation failure was a significant contributor to reduced voluntary contraction, with a 28 % larger reduction as a result of hyperthermia compared to the control. The reduced force was noticeable in both the exercising and non-exercising muscle. This suggests that the activation of muscle fibres during maximal contraction is dictated more so by a high body temperature rather than changes in fatiguing muscles, especially as core temperature stabilised at ~38° during the control rather than ~ 40 °C under hyperthermia. Additionally, exhaustion was not evident in the participants in the control, demonstrating the effect of increasing body temperatures on central activation and subsequent performance. This was further confirmed in the data of two participants who were passively induced to hyperthermic levels, similar to that of Gonzalez et al (1999), yet still had the same response to maximal voluntary contraction in the non-exercising muscle.

Although methodology in the literature varies, all lead to the conclusion that termination of exercise occurs at a critical core temperature in one way or another, yet whether this is the direct cause or not is still contested. Hyperthermia induces the release of heat shock proteins as a protective mechanism for the body (Lee et al, 2006), while the release of neurotransmitters (Meeusen and Watson, 2007) such as serotonin, dopamine and noradrenalin (Meeusen and Roelands, 2010), increased ammonia levels and its effect on the central nervous system (Davis and Dailey, 1997) and dehydration have all been associated with the termination of exercise. Serotonin has been linked to fatigue because of its known role in feelings of lethargy, drowsiness and sleep, while dopamine and noradrenalin promotes arousal and motivation (Meeusen and Watson, 2007). Nevertheless, it is thought that these factors are associated with a critical core temperature and more the existence of a central governor; a centrally regulated process that uses a combination of inputs from peripheral and central afferents. Peripheral factors such as reduced efficiency of muscle metabolism and neuromuscular function impairment (Hargreaves and Febbraio, 1998), reduced substrate availability (Hasegawa et al, 2008) and changes in blood volume and skin blood flow (Yoshida et al, 1997) are all evident at fatigue in normothermic conditions yet are exacerbated in the presence of hyperthermia. However, what is still not clear is whether this is as a result of a critical core temperature or whether each one of the aforementioned conditions simply coexists, combining synergistically to cause a catastrophic effect.
2.3.3. Anticipatory Regulation, Central Governor and the Catastrophe Theory

The critical core temperature hypothesis has been linked to what is known as the catastrophe theory, where the body is in some way centrally regulated through a “governor” in order to reduce muscle function and exercise output to prevent fatality. Early research in the 1920’s first provided the suggestion of a central governor. Following self-experimentation, Archibald Hill stated that failure of exercise at higher intensities was due to increased muscle anaerobiosis and the inability to remove lactate. It was suggested that this inability was due to insufficient cardiac output and distribution of blood to the working muscles. He believed that exercise was limited by maximal cardiac output, and that the limit to maximal cardiac output was cardiac failure, which can be deemed catastrophic (Noakes, 2012). Notwithstanding, Hill et al (1924) stated that myocardial ischemia during maximal exercise was not common and so something must protect the heart from becoming ischemic during and post exercise. It was
here that the theory of a “governor” was proposed (Hill et al, 1924) where the heart or brain reduced the pumping capacity of the heart immediately upon myocardial ischemia.

Fig. 2.7. The complete A.V. Hill Catastrophic Model of Human Exercise Performance (1924), cited in Noakes (2012). The circled section highlights the first suggestion of a governor within exercise and fatigue literature.

Ulmer (1996) suggested that a “central” governor may exist within the central nervous system where a feedback system is in operation. It is thought that through brain regulation and anticipatory learning (Eston, 2012) combined with internal sensory feedback (Ulmer, 1996), the body regulates exercise intensity throughout the duration of performance to ensure that completion of exercise is achieved in the shortest possible time and at the highest possible output level without causing damage to homeostasis and physiological systems. The balance of inhibition and facilitation systems determines motor output (Tanaka and Watanabe, 2012) and exercise may be limited by adjustments to neurotransmitter
concentrations which may reduce central activation. The muscles are activated by the primary motor cortex in the brain, via neurons from the corticospinal tract that synapse with motor neurons, propagating the action potential to neuromuscular junctions for muscle fibre stimulation (Latesh, 2008). As the demands of exercise increase, increased activation of the muscle is required. However it is thought that afferent signals are transported back to the brain from the periphery initiating a down regulation in central motor output and thus muscle recruitment accordingly to prevent this homeostatic catastrophe (Gandevia, 2001; Abbiss and Laursen, 2005). This information can be conscious, such as perceived thermal sensation or muscular pain, and subconscious, such as baroreceptors, chemoreceptors and thermoreceptors within the body’s internal environment. The prefrontal cortex is associated with those tasks completed consciously, operating as a ‘perception-action interface’ (Fuster, 1995) providing the facility to dictate actions based on internal goals (Miller and Kohen, 2001) and conscious peripheral feedback. Rooks et al (2010) provided evidence for a region in the brain with results in a study showing that prefrontal cortex oxygenation increased in a curvilinear fashion as exercise intensity increased from moderate to hard intensity. However, at maximal intensity, oxygenation levels began to drop, resulting in reductions in cognitive function.

**Figure 2.8.** Central governor model showing the influence of afferent signals from the vital organs and peripheral and central senses on central drive, thus controlling the activation of skeletal muscle. (Taken from Abbiss and Laursen, 2005)
Both conscious and subconscious sensory feedback constitute to the theory of a central governor, yet the ability to push one’s self passed that of safe homeostatic levels, as seen in many elite athletes, provides evidence for the role of motivation to complete an exercise task. Individual experience must contribute somewhat to the cessation of exercise, with feelings of discomfort, tiredness and pain all contributing to motivational drive (Marcora, 2010). This definition of fatigue as a conscious sensation rather than a physiological process is referenced in the literature (Noteboom et al, 2001; St. Clair Gibson et al, 2003). Rather than physiological markers automatically reducing performance output on a subconscious level, the motivational model (Abbiss and Laursen, 2005) suggests that reductions in neuromuscular function are intentionally adjusted consequently diminishing the influence of motor control activation. The rating of perceived exertion (Borg, 1970) is a method of pertaining ones perceptual effort and provides a good indicator of the ability to continue or complete an exercise task (Nybo and Secher, 2004). Work by Marcora et al (2009) support the idea of a motivation model by concluding that mental fatigue through higher perceptions of effort, and not cardiorespiratory and muscle energetics, was the limiting factor for exercise cessation. Transmission blocking at the neuromuscular junction has been used to support this theory. Gallagher et al (2001) induce partial neuromuscular blockade to increase central command influence during three MVC’s and a seven minute dynamic exercise bout. RPE significantly increased by 6 points on the Borg scale following neuromuscular blocking even though the workload was the same, suggesting that it is efferent inputs from the brain rather than afferent returns that control exercise intensity. Based on findings such as these Marcora et al (2009) state that the increased demand to exercise at similar intensities, despite weakened muscular transmission, can be perceived as an increase in effort. This perception of effort therefore becomes a feed-forward signal that controls the cessation of exercise. This could be said of feelings of discomfort through thermal sensation which could affect motivational drive to continue, with high or lower perceptive thermal sensation making it uncomfortable to exercise at an optimum.

Notwithstanding, fatigue is an intricate concept, integrating the numerous central and peripheral phenomenon’s and so selecting one as the key modulator of exercise termination seems somewhat unjustified. With this in consideration, the central governor may operate based on both a conscious and subconscious level, with both levels regulating the level of fatigue or the cessation of exercise, but through different factors. A critical core temperature can be an excellent indicator of cessation of exercise, similar to that of rating of perceived


exertion, yet the ability to continue producing repeated maximal efforts is still witnessed. Castle et al (2006) noticed a progressive increase in core temperature in repeated maximal effort cycling bouts, while observing a gradual decline in peak power output. However, peak power output of the final sprint exceeded that of the penultimate sprint, despite the continual elevated core temperatures. If a critical core temperature was the direct cause, this final increase would be non-existent and so suggests that anticipatory regulation via a central governor was evident, with down regulation of muscle activity occurring to allow the completion of exercise. The argument against the critical core temperature being the lead modulator in exercise capacity is that if a critical core temperature initiated the down regulation of motor recruitment, then body temperature would have to reach critical levels before any reduction occurred which could be catastrophic. With this in mind, Marino (2004) suggests that the central nervous system reduces the efferent signals required for muscle recruitment prior to a critical limiting temperature. It could be that during times of high intensity exercise, subconscious afferent signals are sent to the central governor to make adjustments to the efferent signals required for changes in neurotransmitter concentrations in an attempt to reduce muscle fibre recruitment and the risk of homeostatic catastrophe.

Nevertheless, increased motivational drive sees repeated maximal muscular contractions continue alongside continual rises in perceived exertion and core temperature to a limiting end point, whereupon critical fatigue is reached and thus termination of exercise occurs. This idea then incorporates the critical core temperature theory coinciding with peripheral and central regulation through the means of a central governor. Marino (2004) proposed a theory that incorporates the above concepts as well as the contribution of muscle sympathetic nerve activity (MSNA), based on studies showing the stimulation of group III and group IV muscle afferents to increase the firing potential in response to heat stress (Ray and Gracey, 1997). This theory suggests that it is signals from both the periphery (afferent feedback from the skeletal muscle) and core (body temperature rises) that provide the central nervous system with the compulsory information required for down-regulation of muscle activity and consequently reductions in exercise intensity or even cessation or exercise. Using superimposed electrical stimulation on maximal voluntary contractions to determine central regulation of skeletal muscle, Nybo and Neilsen (2001) reported reductions in central drive of 7% following exercise induced hyperthermia, concluding that hyperthermia causes a down-regulation of motor command from the central nervous system.
Figure 2.9. Model showing the incorporation of both body temperature increase and muscle afferents in the down regulation of exercise and cessation of exercise as suggested by Marino (2004). CNS = central nervous system, MSNA = muscle sympathetic nerve activity

Although the concept of a central governor appears to be favoured in the literature (Swart et al, 2012; Missenard et al, 2009; Noakes, 2007; Noakes and St. Clair Gibson, 2004), a critical limiting temperature cannot be dismissed as a key factor in exercise cessation. Morrison et al (2004) concluded that hyperthermia induced reductions in voluntary activation percentage, providing an argument for anticipatory regulation, yet the restoration of this activation to normal functioning levels only occurred when core temperature returned to resting levels, solidifying that both theories work alongside one another to prevent fatality.

Millet (2011) proposed a model to explain fatigue, particularly for ultra-endurance based exercise. This model is based on the ‘flush toilet’ and is unique in that it emphasizes the importance of peripheral fatigue. Nonetheless, it supports the concept that exercise is regulated by a central governor to avoid catastrophic failure. It is made up of four components i) the buoy, ii) the filling rate, iii) the waste pipe and iv) the security reserve. A key component in this model is RPE, which is represented by the level of water in the tank. Therefore as the water increases, you would see an increase in RPE. The ‘filling’ component
supplies the tank with water which causes the fluctuations in RPE. The rate at which the water fills and leaves the tank is associated with feed-forward, feedback and teleoanticipatory messages, determined by peripheral changes and central inhibition. Furthermore, psychological and environmental influences can have an influence. The waste section can be considered the recovery period, however this does not necessarily mean passive rest, instead can be as a result of a down-regulation of exercise intensity. The fourth component is the security reserve, and ensures that catastrophic failure may never exist because down-regulation and, in the most fatigued state, exercise termination would occur once this reserve is reached. The size of this reserve is suggested to be influence highly by motivation, with highly motivated athletes exercising further into the security reserve before ‘a flush occurs.’

Figure 2.10. The Flush Model of Exercise Regulation (Millet, 2011)

A recent article by Noakes (2012) further developed the idea of central regulation, addressing peripheral and central influences, as well as the inclusion of afferent signals and efferent
commands. The “Central Governor Model of Exercise Regulation” suggests that exercise performance is continuously modified by the brain due to conscious and subconscious factors that exist prior to and during exercise. In a similar concept to Millet (2012), exercise intensity is pre-determined in a feed-forward anticipatory manner, with motivational, mental and emotional state contributing to an initial intensity that is deemed suitable to complete the duration of exercise. Throughout exercise, physiological afferents such as heat accumulation and depletion of energy stores, and sensory afferents such as distance covered or time to endpoint allow contraction-by-contraction modification allowing completion of the exercise task (Tucker et al, 2006a). The presence of an end-spurt supports the continuous regulation of exercise, even during repeated maximal efforts (Castle et al, 2006). The key difference in this model is that everything starts and ends with the brain, and peripheral factors are merely afferents that allow conscious regulation based on perception and sensation. Additionally, unlike the flush model that was designed with one particular mode of exercise in mind, the Central Governor Model of Exercise Regulation can be applied to various modes of exercise, whether it is maximal sprinting work or endurance based exercise, or whether it is open or closed loop activity.

Figure 2.11. The Central Governor Model of Exercise Regulation (Noakes, 2012)
Interestingly, under conditions of hyperthermia, the brain appears to regulate central drive irrespective of any exercise components. It has been observed that increases in core temperature via passive hyperthermia have caused reductions in cortical voluntary activation, maximal EMG and maximal volitional force (Morrison et al, 2004; Ross et al, 2012). Furthermore, changes in muscle temperature do not appear to have similar effects on voluntary activation of the muscles (Thomas et al, 2006). It can therefore be suggested that fatigue during conditions of heat stress may be, partly, a result of reduced central nervous system function and activation of the exercising muscles.
2.4. The Muscle

2.4.1. Muscle Structure

The process of thermoregulation occurs throughout all of the body, with both central and peripheral adaptations occurring at an autonomic level. There is wide debate as to the direct causes of fatigue, as discussed in section 2.3, and to understand this and the conception of thermoregulation further, a brief section on muscle physiology is outlined.

Fibrous connective tissue; the epimysium, surrounds the whole muscle which contains bundles of muscle fibres. These muscle fibre bundles, or fascicules, are surrounded by a connective tissue layer called the perimysium. The main network of blood vessels and nerve pathways run through the perimysium (McComas, 1996). Within the perimysium are individual muscle fibres that are protected by connective tissue called the endomysium (see Figures 2.12 and 2.13). Muscle fibres contain functional units called myofibrils which are made up of small protein filaments, mainly actin and myosin, as well as tropomyosin and troponin, which collectively have a key involvement in muscle contraction. Within the endomysium, arterioles, venules and capillaries run parallel to individual muscle fibres, forming an extensive network of vessels that provide each muscle fibre with sufficient blood supply.
Figure 2.12. Cross-sectional drawing of muscle tissue showing the three differing layers of connective tissue that house individual muscle fibres and their contents (McComas, 1996)

This blood supply carries oxygenated blood to the working muscles and removes carbon dioxide in a continuous flow motion (McArdle et al, 2001). As the intensity of exercise increases, the oxygen demand within the muscles increases; a demand that is met through increased dilation of the surrounding capillaries which allows for an increased blood flow. Additionally, a membrane known as the sarcolemma sits under the endomysium, covering the surface of the muscle fibre. The sarcolemma is an elastic membrane that provides the basis of structure of the individual muscle fibre’s contents. These include the aforementioned myofibrils and the sarcoplasm; a fluid-like protoplasm that is home to enzymes, mitochondria, fuel particles, the nucleus and the sarcoplasmic reticulum. The sarcoplasmic reticulum is a network of tubules and vesicles that run along each individual myofibril. The vesicles provide structure to the muscle fibre while the transverse tubules connect the surface of the fibre to the inner components of the fibre, allowing the wave of depolarisation from the action potential to stimulate muscle contraction (McComas, 1996; McArdle et al, 2001). This is achieved through the release of calcium ions into the sarcoplasm via calcium pumps (Mougios, 2006) with the calcium ions binding with the tropomyosin that is inhibiting the binding sites of actin and myosin. Mitochondria contained within the sarcoplasm are made up of a double membrane matrix, known as cristae, that is home to the enzymes required for anaerobic and aerobic metabolism (McComas, 1996).
2.4.2. Transfer of heat through a limb

There are a number of layers that are involved within transfer of heat throughout the muscle, with some layers having sub-layers that play different roles. For example, there are up to five layers within the epidermis of the hands or feet from the epithelia tissue to the basal lamina; the stratum germinativum, the stratum spinosum, the stratum granulosum, the stratum lucidum and the stratum corneum (Martini, 2004). The epidermis provides the external protective layer of the body that is in contact with the external environment as well as being the location of pores involved with sweating and peripheral thermal receptors in the forms of afferent nerve endings (McArdle et al, 2001). The layer underneath the epidermis is called the dermis; the tissue that supports the epidermis. Considerably thicker and vascularised, the dermis is home to nerve fibres and sweat glands as well as a capillary network that supplies blood to the multiple structures within the tissue layer (Martini, 2004). Many of these structures protrude into the epidermis and provide the sensory information vital for thermoregulation, yet the main body of them start in the dermal layer. Main blood supply
comes from the blood vessels running along the superficial region of the subcutaneous layer that is in contact with the base of the reticular dermal layer. The subcutaneous layer is not part of the skin; instead it provides stabilisation of the skin in relation to underlying muscles and organs, provides a thick insulative and protective layer and the supplies the dermis and epidermis with blood (Martini, 2004).

Under heat stress, vasodilation of blood vessels occurs, allowing warmer blood to be transported to the cooler peripheral regions and dissipated to the environment through convection. Heat produced as a result of muscle contraction moves through deeper tissues via conduction. This heat is passed from warmer molecules to surrounding cooler ones towards the limb surface, increasing skin temperature. Heat loss is then achieved through means of convection, radiation or evaporation (Prek, 2005). This process continues as required until there is a level of equilibrium in thermal balance (see section 2.2). Cold receptors exist in greater quantity than hot receptors and stimulation of these receptors reduces blood flow through vasoconstriction limiting heat loss. Under cold stress, the external stressor absorbs heat from the body. Cooling techniques, such as pre-cooling via ice vest or ice pack (to be discussed in section 2.6) provide the body with an immediate cold stress. Kenner et al (2007) reported that cooling agents absorb heat from the body through conduction, increasing the temperature of the ice to melting point. Consequently, temperature of the skin and subcutaneous layers will drop. This will result in cooler blood surrounding these regions, as well as cooler deeper tissues due to conduction. Studies have shown that skin and deep tissue temperatures are closely related (Yanagisawa et al, 2007). Interestingly, Janwantanakul (2009) reported that increasing the contact area of ice pack application does not directly increase the rate of cooling. This suggests that upon a stressor, heat distribution is evenly spread demonstrating the insulative properties of the skin, muscle and subcutaneous fat and the body’s ability to conserve heat. Nevertheless, the quantity of ice was shown to increase the magnitude of cooling. From four ice pack application conditions, heavier packs of crushed ice (weighing 0.6 and 0.8 kg) lead to significantly greater reductions in interface temperature than same sized packs weighing 0.3 kg following 20 minutes of cooling. This may be due to the thermal properties of the ice pack, with heavier ice taking longer to reach melting point. Interestingly, surface temperature took 8 minutes to reduce to the minimum recorded values, with no further significant reductions apparent thereafter. Further research into the pathways associated with the kinetics of heat exchange during a cooling bout would provide useful knowledge for application of a cooling bout.
2.5. High Intensity and Intermittent Sprint Exercise

2.5.1. The components of intermittent sprint exercise

Intermittent sprint exercise can be broken down into various phases of rest, sprinting and recovery bouts. Laboratory-based protocols tend to differ depending on the nature of the study and the population application. Given the randomness of exercise bursts during games play, it is difficult to isolate an individual protocol as a criterion that reflects intermittent sprint exercise. There can often be an agglomeration of sprints during one phase of play consequently reducing periods available for recovery, followed by a dispersion of sprints throughout another phase resulting in longer durations of recovery (Williams, 1990; Spencer et al, 2004). Nevertheless it is believed that during these periods, match outcomes can be decided (Wadley and Le Rossignol, 1998). It is for this reason that intermittent sprint protocols are becoming more prevalent in the literature. Intermittent sprint based exercise protocols pertaining to team games players such as football, hockey and rugby will be used throughout this thesis.

Commonly, literature assessing intermittent sprint activity employs solely one set of a varied number of sprints interspersed with short recovery periods to assess performance (Spencer et al, 2005). Serpiello et al (2011) critiqued methodology of this kind stating that it poorly reflects the demands of game play in team sports due to the short duration of the protocol. They therefore tested an alternative repeated sprint exercise test, using a lower number of sprints but longer recovery period (5 x 4 second sprints with 20 seconds passive recovery) broken into three sets (separated by 4.5 minutes) to try to emulate the durations of games play more effectively. Despite the changes and implementation of three sets, the adapted protocol still runs for only 15 minutes, and although recovery durations are shorter between sprints, passive recovery is used. While reductions in peak power output and acceleration ability were still observed, it could be considered that a protocol of this nature is not reflective of team games play. A football match lasts for a minimum of 90 minutes (two halves of 45 minutes), a rugby match for 80 minutes (two halves of 40 minutes) and a hockey match for 70 minutes (two halves of 35 minutes). Exercise durations of 15 minutes with significant periods of passive rest as tested by Serpiello et al (2011) tend to be more common in fast-paced explosive American style sport such as American football and basketball. Therefore, a protocol of this nature seems inappropriate for longer duration intermittent sports.
Alternatively, sport-specific protocols have been used to increase construct validity. The Bangsbo Soccer-specific endurance test (Bangsbo, 1994) consists of 40 running bouts of high and low intensity exercise of a fixed duration. Moreover, the inclusion of a running slalom phase allows for similarity to movement patterns observed in team games play. However, the duration of the protocol is still only 16.5 minutes long. The Soccer-Specific Intermittent Exercise Test (Oliver et al, 2007) is considerably longer in duration (42 minutes) and consists of three bouts inclusive of a walking, jogging and cruising phase, a 5 second sprint and a 15 second rest, while a three minute period separates each bout. Similarly, the Loughborough Intermittent Shuttle Test (Nicholas et al, 2007), is longer in duration and divided into two parts. Part A consisting of five 15 minute exercise bouts of pre-set alternative intensity (a walk, a jog at 50 % VO2max, sprint and run at 80 % VO2max over a 20 meter distance), with each set separated by three minutes of passive rest. Part B is then fatiguing in nature, with intermittent shuttle running at 55 % and 95 % VO2max until exhaustion. Although the phases in these protocols are more conducive of team games play activity patterns and the duration of these protocols suit that of long duration team sports, both contain long rest periods between phases which are uncommon in team sports. Furthermore, the nature of running based protocols does not necessarily allow accurate measurement of physiological and performance parameters. Cycling based protocols overcome the problem of inaccurate measurement. Castle et al (2004 & 2006) used a cycling based protocol of intermittent nature. This protocol lasted 40 minutes in duration and avoided long periods of passive rest. Additionally, maximal sprint efforts can be analysed through measurement of power output, while recovery periods are a fixed pre-determined intensity, allowing the experimenter control of every aspect of the protocol except the maximal sprint.

It has been well documented that active recovery is more advantageous than passive rest for repeated sprint ability in team sports (Bogdanis et al, 1996). Toubekis et al (2006) concluded that active recovery of 50 - 60 % is detrimental to repeated 25m sprint performance in swimming despite reductions in blood lactate concentration. In consideration, it must be noted that recovery intensities corresponding to 60 % that of 100m sprint performance are relatively high for a recovery period. Belcastro and Bonen (1975) state that recovery should be low enough not to maintain or reactivate anaerobic metabolism, thus diminishing performance, which may have been the case for Toubekis et al (2006). Equally, Signorile et al (1993), Bogdanis et al (1996), Dorado et al (2004), Spierer et al (2004) and Menzies et al (2010) all showed improvements in performance attributable to active recovery as result of
increased $\text{VO}_2$ kinetics, maintained elevated $\text{VO}_2\text{peak}$, reduced capillary blood lactate levels and reduced cardiovascular strain.

### 2.5.2. The Effects of Heat Stress on Intermittent Sprint Exercise

Considering the factors previously discussed that contribute to fatigue, it is not surprising that intermittent sprint exercise performance is limited under climatic heat stress. Submaximal exercise in a hyperthermic environment has been reported to increase the rate of muscle glycogen degradation and presence of adrenalin within the muscle (Febbraio et al, 1994), while an increase carbohydrate oxidation and exacerbated lactate accumulation are suggested to occur following sprint exercise due to shifts towards anaerobic metabolism (Hargreaves, 2008). Decreases in cardiac output, stroke volume and blood flow resulting in increased in cardiovascular strain (Crandall and Gonzalez-Alonso, 2012), as well as thermoregulatory strain are also reported following high intensity exercise in the heat (Drust et al, 2005). Drust et al (2005) saw increases in both core and muscle temperature correspond with reductions in peak power output and mean power output during a repeated sprint test following 40 minutes of intermittent submaximal cycling (15 seconds high resistance (306 ± 22 W) and 15 seconds unloaded cycling). Participants underwent 40 minutes of repeat cycling, constructed of 15 seconds cycling at 60 % $\text{VO}_2\text{peak}$ and 15 seconds ‘rest’ of unloaded cycling before completing 5 all-out sprints separated by 15 seconds temperate (20 °C, 23.6 % RH) and hot (40 °C, 17 % RH) conditions. Elevations in core temperature in the hot conditions (+1.3 ± 0.3 °C compared to the control) were concluded to be the primary governor in impairment in sprint performance despite initial sprints for both temperate and hot conditions being similar. This was linked to the suggestion that increased glycogen utilisation affected glycolytic flux and acid-based homeostasis. However, warmer muscle temperature is attributed to increases in performance. Ball et al (1999) stated that the ability to reach a higher pedal cadence saw a 25 % improvement in sprint peak power output and 15 % in mean power output due to warmer muscles as a result of exercise in a hot environment, although the onset of fatigue occurred at a quicker rate. Bishop and Maxwell (2009) reported that a prolonged warm up negatively affected repeated sprint performance, suggesting that impaired performance could be a result of larger core temperature increases. Therefore, the increases in core temperature and muscle temperature per se in the Drust et al (2005) study should not be concomitantly associated with impaired performance. It could be suggested that denaturing of muscle protein required
for muscle contraction could have occurred (Hargreaves and Febbraio, 1998), particularly as muscle temperature exceeded 40 °C, yet this occurred in all conditions despite differences in repeated sprint performance. Alternatively, the input of inhibitory feedback from metaboreceptors in the muscle (Kent-Braun, 1999) should be considered alongside rises in core temperature in the down-regulation of exercise similar to that proposed in section 2.4.4. In contrast, studies have shown that supramaximal exercise in the heat is not affected by increased muscle glycogenolysis. Maxwell et al (1999) ran eight participants to exhaustion in a 20 second progressive maximal anaerobic running test (MART) in hot (32.8 °C, 80.5 % relative humidity) and cool (21.3 °C and 48 % relative humidity) conditions and despite observations of declines performance and time to fatigue in the hot conditions, muscle glycogen concentrations did not differ between conditions. It was suggested that the contribution of aerobic metabolism during the early stages of the MART may have had an influence, with oxygen consumption having been shown to increase under conditions of heat stress (Finn et al, 2001). Similarly, in a two-part intermittent sprint exercise protocol Morris et al (1998) reported no differences in energy utilisation or lactate production between moderate and hot environmental conditions. Participants completed an 11-set mixed-intensity pattern, that included a 15m maximal sprint. Following this, participants were required to repeatedly run at 99 % of predicted VO2max for sixty seconds with one minute intervals until exhaustion. Performance decrements were apparent in the hot condition compared to the moderate condition (total distance completed, hot vs. moderate: 8,842 ± 790 vs. 11, 277 ± 214 m, P < 0.01), yet upon termination of the test in each condition, no differences were observed in blood glucose, blood lactate or plasma metabolites. It was concluded that performance was associated with a continued rise in core temperature in the hot condition, compared with the moderate condition (hot vs. moderate: 39.4 ± 0.1 vs. 38.0 ± 0.1 °C, P < 0.01), which became a limiting factor in performance.

Rises in core temperature are commonly reported as detriments to intermittent sprint exercise in the heat, however the ability to perform at high levels throughout the duration of the exercise task is still observed. Duffield et al (2009) observed the largest increases in core temperature occurring during the first quarter (20 minutes) of Australian Rules football preseason games (80 minutes in duration), with the peak core temperature occurring during the last quarter. Reductions in exercise intensity were observed during the second and third quarters with the highest exercise intensity observed in the final quarter, with the implementation of a subconscious pacing strategy being the suggested reason behind the
ability to perform at the greatest possible output towards the end of the game. This increase in performance towards the end of an intermittent sprint exercise test was also observed by Castle et al (2006). The cycling intermittent sprint protocol (CISP) has been commonly employed as a repeated sprint exercise test, particularly in the heat, in a means to replicate that of team-game activity. The CISP is 40 minutes of intermittent sprint exercise consisting of 10 seconds of passive rest, a 5 second all out sprint from a stationary start followed by 105 seconds of active recovery at 35 % $\text{VO}_{2\text{peak}}$. The 35 % was chosen based on Belcastro and Bonen’s (1975) and Bonen and Belcastro’s (1976) studies looking at levels of recovery in removing lactic acid stating 32 % $\text{VO}_{2\text{max}}$ was the predicted optimal recovery level, with 55 % $\text{VO}_{2\text{max}}$ for ergometer based activity. Castle et al (2006) also took into account the work by Choi et al (1994) who stated that active recovery above that of 40 % $\text{VO}_{2\text{max}}$ reduced the amount of muscle glycogen re-synthesis, thus arriving at the 35 % $\text{VO}_{2\text{max}}$ level. 50 % recovery intensity when compared to 35 % recovery has since been shown to be detrimental to CISP performance in hot conditions (Maxwell et al, 2008). Castle et al (2006) saw the greatest decrements in peak power output across the CISP in the control condition (13.5 % from the first and highest sprint to the lowest sprint), as well as the highest core temperature and fastest rate of physiological strain. Despite this, peak power output for the final sprint of the CISP during the control, and all intervention conditions, surpassed that of the penultimate sprint. The authors stated that this demonstrated a lack of peripheral fatigue inhibiting performance supporting the case of a central governor, in particular during the vest cooling intervention where the greatest increase in final sprint occurred. It is thought that the continuous sensory feedback provided to the brain during vest cooling was less than other methods of cooling thus allowing the central governor to recalculate the available motor unit supplies for firing the muscle towards the end of the CISP subsequently improving performance. Data from this study rebut theory of Marcora et al (2009), who suggested that perceptual feedback acts as a key modulator to exercise, as there were no differences in RPE values at the end of the CISP when compared to the control. All conclusions aside, reliability of the CISP has not been formally documented and so full interpretations of the data should be considered assumptions until this is clarified.

Hyperammonemia has been suggested as a contributing factor to intermittent exercise fatigue, with the affects exacerbated in the heat (Mohr et al, 2006). Mohr et al (2006) had eight individuals complete 40 minutes of intermittent exercise followed by five all-out sprints in a control and hot environment. Similar to previous literature, higher core and muscle
temperatures were observed following exercise in the heat trial as well as declines in mean power output. Muscle glycogen, ATP and creatine phosphate levels were similar across conditions, with levels of plasma ammonia increasing by 34% in the heat compared to the control, although plasma potassium and muscle hydrogen accumulation was lower in the heat, leading the authors to suggest that, under heat stress, an enhanced ‘systemic ammonia response’ may be a factor that influences cessation of exercise. Ammonia is known to have a negative impact on cerebral function (Nybo et al, 2004) and so this could be a result of an enhanced purine nucleotide cycle within the muscle, elevating the levels of plasma ammonia (Wilkinson et al, 2012) reaching the cerebral cortex, thus limiting cognitive function and concomitantly affecting intermittent sprint performance. Interestingly, Sunderland and Nevill (2005) found increases in heart rate, rating of perceived exertion, core temperature and glucose concentration as a result of environmental heat stress following completion of the LIST, but not elevations in serum hormones, or plasma ammonia and blood lactate concentrations, agreeing with the findings of Morris et al (1998). Nine well-trained female hockey players completed the Loughborough Intermittent Shuttle Test (LIST) interspersed with field hockey skill tests in a temperate (19 °C, 50% RH) and a hot (30 °C, 37% RH) environment. A ten minute rest period at the half way stage was employed to mimic a half time interval. The skill test was performed before the LIST and after the second and fourth sets, with a decision-making time recorded during the skill tests. Declines in field hockey skill performance were observed in both conditions after 30 minutes, continuing further after 60 minutes of exercise. A 6% poorer performance in the second skills test was observed in the hot condition after the 30 minute period compared to the temperate condition, but no differences were found in the decision-making, leading the authors to conclude that the decline in skill performance was not related to muscle acidosis or ammonia accumulation. It is likely that the increased heart rate and elevated core temperature resulting in greater cardiovascular and heat strain, consequently increasing physiological strain, contributed to the cause of fatigue in the heat. This may be reflected in the increased feelings of thirst and perceived exertion observed, and thus a simultaneous increase in psychological strain.

A new angle on intermittent sprint exercise research is the concept of a free-paced exercise period during the protocol. Skein and Duffield (2010) and Skein et al (2012) use a fifty minute protocol that requires a 15m maximal sprint every minute followed by a self-paced submaximal mode lasting fifty seconds. This self-paced mode was one of the following: a walk, jog or hard run, with only one mode completed during each one minute recovery
phased and each mode rotated throughout the fifty minutes. The protocol was designed to replicate the physiological, perceptual and physical demands of team game activity, as suggested by (Spencer et al, 2005). While the construct validity of this protocol is apparent given that recovery during competitive sports is dependent on the individual, the performance measures of distance covered in each phase, given that they are participant dependent, may not provide the most robust measures for analysis and so further work may be required determining the reliability of such a protocol.

2.5.3. The Effects of Temperature on Muscle Function

As previously discussed, body temperature increases as a result of exercise, nonetheless, the origin of this heat accumulation has not been mentioned. Muscle contraction causes heat generation, whether it is through the process of muscle metabolism and mitochondrial oxidative phosphorylation (Crow and Kushmerick, 1982) or as a result of the thermoelastic properties of the muscle during tension-relaxation (Gilbert and Mutsumoto, 1976). Continual contraction will result in continual heat production and lead to continuous rises in peripheral and core temperature. Therefore, understanding the effects of changes in muscle temperature could be vital in achieving maximal levels of sporting performance.

Many studies have reported muscle temperature changes throughout exercise protocols in various environmental conditions (Febbraio et al, 1994; Sleivert et al, 2001; Drust et al, 2005; Castle et al, 2006; Duffield et al, 2009), however muscle temperature was not the lead variable in these investigations. Early research measured maximal peak power during 20 seconds constant load cycling following cooling or warming of the quadriceps muscles (Sargeant, 1987). Results showed that warming the legs for 45 minutes in a 44 °C water bath improved peak power by 11 % compared to control. On the contrary, cooling the legs for the same duration in an 18 °C and a 12 °C bath caused peak power to reduce by 12 % and 21 % respectively when compared to the control. Furthermore, a reported 4 % increase and 3 % decrease in power per 1 °C rise or fall in muscle temperature occurred, respectively. A temperature-velocity relationship, as previously suggested but not found by Bergh and Ekblom (1979a), was observed, with the greater improvements in power output evident at higher fixed intensities. Nevertheless, the increased performance resulted in a quicker onset of fatigue. Warmer muscles have been shown to produce greater force variability than cooler
muscles (Brazaitis et al, 2010), possibly due to faster relaxation that is associated with motor firing rates (Todd et al, 2005). Despite this, it is still common practice to warm the muscles prior to performance to achieve possible improvements in nerve conduction velocity, joint and muscle stiffness, metabolic reactions and to increase blood flow to the working muscles (for more benefits see review by Bishop, 2003). Conversely, Sargeant (1987) saw muscle temperature reductions of 21% (36.6 to 29 °C) following cooling in a water bath and suggested this induces reduced neural transmission. Fibre type differences were also brought into question, with the smallest cross-sectional area of type I slow twitch fibres yielding the smallest temperature related change in power output. These results are supported by Rademaker (1996) who reported significant power reductions in individuals with a higher percentage of type I muscle fibres at the same muscle temperatures. Eight participants, (four with 73 ± 8 % cross-sectional muscle fibre type I and four with 45 ± 7 % cross-sectional muscle fibre type I) completed maximal sprints for 5 seconds at three constant pedalling rates, in three muscle temperature conditions (cold, neutral and warm). Muscle temperature was manipulated via immersing the legs in water for 45 minutes. Results displayed significantly higher peak power in individuals with lower cross-sectional muscle fibre type I. Furthermore, temperature sensitivity was three times greater in those with a higher percentage of type I fibres. Edwards et al (1972) proposed the theory that an increased rate of cross-bridge cycling resulted from increases in muscle temperature, however this cannot be the case due to limited effect increases in muscle temperature have been shown to have on ATP and phosphocreatine stores (Febbraio et al, 1994; Starkie et al, 1999). Febbraio et al (1994) did observe an increase in ATP utilisation as a result of 40 minutes of continuous cycling in the heat when compared to a neutral environment, however stated that this was matched with increased anaerobic glycolysis and creatine phosphate hydrolysis. Instead, Starkie et al (1999) state that a warmer muscle has a direct effect on net glycogen utilisation, increasing with maintained higher muscle temperatures due to the influence warmer temperatures have on enzyme activity. Muscle glycogen is an essential fuel for muscle contraction with fatigue attributed to glycogen depletion (Holloszy et al, 1998). Increased muscle metabolism has been seen to increase lactate concentrations in the heat when compared to a control (Hunter et al, 2011), however this was not the case for Starkie et al (1999) who observed similar post lactate concentrations in hot and cold leg conditions. It was suggested that this was due to limited lactate removal in the cold leg probably due to vasoconstriction of blood vessels as a results of the stressor. Short-term cold stressors have been shown to increase peripheral blood flow, with Maekawa et al (1998) showing that a
dose-response relationship is evident with cold stress temperature and muscle blood volume. Cooler muscles have also been reported to reduce conduction velocity (Ce et al, 2012) with reduced EMG amplitude (Petrofsky and Lind, 1980) occurring below muscle temperatures of 22 °C, but not between 37 – 25 °C (de Ruiter et al, 1999). This is suggested to be as a result of a slowed motor unit action potential distribution (Ce et al, 2012). Reductions in power output have also been shown to be as a result of agonist-antagonist conflict; that is exercise contractions that occur during shivering cause co-contractions of the agonist and antagonist muscle pairs resulting in them working against each other rather than in unison (Oksa et al, 1997).

2.5.4. The Effects of Intermittent Sprint Exercise on Muscle Function

Explosive high intensity cyclical movements have been reported to cause a decrease in maximal muscle power production (Billaut et al, 2005). During higher intensity exercise, intracellular pressure is increased (Goto et al, 2007) due to occlusion of the blood vessels resulting in reduced blood flow and oxygen transport to the working muscles and possibly increasing oxidative stress which may be detrimental to performance (Goto et al, 2007; Powers and Jackson, 2008). Energy supply to these muscles is therefore derived from anaerobic glycolysis (Wells et al, 2009; Ohlendieck, 2012) through the hydrolysis of phosphates, as demonstrated in the reactions below (Wells et al, 2009):

\[
\text{ATP breakdown: } \text{ATP} + \text{H}_2\text{O} \xrightarrow{\text{ATP-ase}} \text{ADP} + \text{H}^+ \text{P}_i + \text{Energy} \\
\text{ATP resynthesis: } \text{ADP} + \text{P}_i + \text{Energy} \xrightarrow{\text{ATP-ase}} \text{ATP} \\
\text{ATP resynthesis PCr + ADP + Energy} \xrightarrow{\text{creatine kinase}} \text{ATP} + \text{Cr}
\]

where \(\text{ATP} = \text{adenosine triphosphate, } \text{H}_2\text{O} = \text{water, } \text{ADP} = \text{adenosine diphosphate, } \text{H}^+ = \text{hydrogen ions, } \text{P}_i = \text{inorganic phosphate, PCr} = \text{phosphocreatine}\)

Some parts of the literature suggest that muscle fatigue and reduced muscle metabolism is not the direct cause of termination of exercise (Cairns, 2006), potentially conflicting with that of
Drust et al (2005). Nevertheless an exhausted working muscle must be a contributing factor to termination, especially under heat stress. Bishop et al (2011) state that fatigue in repeated sprint ability is attributed to a variety of factors; whether they are neural factors and the inability to generate sufficient motor command in the motor cortex, or muscular factors and accumulation of metabolites. Muscle acidosis, caused through the increase in lactic acid concentration and flux of hydrogen ions as a result of exercise, was originally thought to be the major cause of fatigue. However, studies showed that high intensity contraction can still occur during acidic conditions and that the highest blood lactate concentrations are measured after termination of exercise (Systrom et al., 1990). Furthermore, animal studies showed that acidosis only really effected contraction in cooler conditions but, at physiological conditions more common to the muscle, acidosis had limited effect (Pate et al., 1995; Westerblad et al., 1997). It is now commonly regarded that it is not lactic acid that causes fatigue, rather an increase in potassium ions within the musculature has been proposed as a contributor to declines in performance. This is thought to be a result of reductions in sodium-potassium-adenosine-triphosphatase (Na\(^+\)-K\(^+\)-ATPase) activity (Fraser et al., 2001), resulting in the sodium-potassium pump activity being reduced (Clausen, 2003), consequently reducing muscle excitability and thus be deleterious on power output. This occurs in parallel with muscle energy store usage (Allen and Westerblad, 2001). As muscles repeatedly contract at high intensity workloads, depletion of muscle energy stores is evident, with the force-fatigability relationship suggesting that individuals with greater initial power output suffer a greater level of fatigue (Mendez-Villanueva et al., 2008). This may potentially be through the increased contribution of anaerobic metabolism, although it has been proven that training status plays a pivotal role in the amount of metabolism and so other mechanisms must be involved (Bishop and Edge, 2006). Sprint performance is frequently reported to decline after the first sprint, possibly due to a large depletion of phosphocreatine during that sprint and therefore, reduced levels available for further sprints (Bogdanis et al., 1996). Some researchers have suggested that neural drive influences afferent command in muscle activation. St. Clair-Gibson et al (2001) hypothesises that the first sprint at maximal volitional control sets a level as to the limits of muscle activation, and that repeated activation at this level would cause intracellular damage and damage to the muscle tissues. Therefore, reductions in power would in turn cause reductions in metabolites required for further maximal efforts preventing the risk of damage. This theory is in keeping with that of Ulmer’s (1996) regulation theory of teleoanticipation upon where constant feedback mechanisms cause adjustments in power output allowing exercise to continue rather than termination.
occur. In the last decade, increases in organic phosphate; products of energy metabolism as phosphocreatine is broken down, have also been linked to the impairment of excitation-contraction coupling (Bishop et al, 2011). Inorganic phosphate has negative effects on the sarcoplasmic reticulum release of calcium required by myofibrils reducing the strength of the binding of the cross bridges (Allen and Westerblad, 2001). If this is the case, calcium ion binding sites on troponin may become inhibited, reducing activation of muscle contraction (Baylor and Hollingworth, 2011) and performances decrements will become evident.

Figure 2.14. A schematic from Baylor and Hollingworth (2011) showing the movement of calcium ions during excitation-contraction coupling in frogs' skeletal muscle fibres. Calcium ions are released from the sarcoplasmic reticulum at the triadic junction, increasing concentrations for binding to the myoplasmic buffers troponin (CaTrop), ATP (CaATP), parvalbumin (CaParv) and sarcoplasmic reticulum calcium pump (CaPump) before reuptake into the sarcoplasmic reticulum. Inorganic phosphate would reduce the calcium release, limiting the free calcium required for binding.
Kent-Braun (1999) set out to determine the cause of muscle fatigue, investigating both central and peripheral contributions. Nine participants completed a 4 minute sustained isometric maximal voluntary contraction (MVC) using the dorsiflexor muscles, with an electrical stimulus being administered at the end of the 4 minutes. Force was recorded before and continuously during MVC, while changes in central activation ratio and $^{31}$-phosphorus magnetic resonance spectroscopy ($^{31}$P MRS) were used to measure central activation and metabolites. It was concluded that 20 % of muscle fatigue is due to central factors not associated with altered peripheral excitability with the remaining 80 % due to intramuscular metabolic factors. Decreases in central activation ratio and significant declines in MVC showed that during exercise there was a level of central activation failure. Yet it was the strong relationship between pH and fatigue that lead the authors to associate 80 % with intracellular factors, especially as the twitch interpolation after the 4 minutes showed no reductions in neuromuscular transmission. Although they suggested this change in pH was as a result of increased hydrogen ion concentration that has since been thought not to be the cause, the continual breakdown and re-synthesis of phosphocreatine during and after the MVC would continue to produce both hydrogen and inorganic phosphate that would negate activation required for maximal contraction, hence the reduced force production. The interesting finding was the link between changes in force and integral EMG, combined with changes in pH, which were associated with a feedback mechanism between intracellular metabolism and central drive. Although interpretation of integral EMG has been questioned in relation to central fatigue (Arabadzhiev et al, 2009) and the more recent research suggests that muscular acidosis through the production of hydrogen ions may not be the direct cause of fatigue, Kent-Braun (1999) state that it was primarily an increase in hydrogen ion concentration that had a large relationship with force decrements.
2.6. Combatting the effects of heat stress on intermittent sprint performance

Literature states that it can take up to 14 days for athletes to become fully acclimatised to hot conditions (Armstrong, 1998), yet differs as to whether trained or untrained individuals benefit more from it. Taylor and Cotter (2006) suggest that highly trained athletes have a lower adaptive potential to heat acclimation than that of the moderately trained or untrained athlete yet Garrett et al (2012) concluded that a short term heat acclimation protocol for highly trained athletes improves cardiovascular stability, reductions in core temperature and reduced end-exercise heart rate, resulting increasing endurance capacity. The benefits of heat acclimation have been around for decades (Mitchell et al, 1976; Sawka et al, 1983), and acclimation is still an integral factor in preparation to sporting performance however acclimation is only the first step to tackling continual exposure to hot conditions during the competitive environment. The technique of pre-cooling offers an acute ergogenic aid to combat the negative effects of prolonged exposure in a hot environment during competition, yet the arguments for and against it are varied.
2.6.1. Pre-cooling – an acute method to alleviating heat stress

2.6.1.1. Introduction to Pre-cooling

Despite the exact mechanisms behind fatigue discussed in previous sections still being widely debated, the detrimental effects of hyperthermia on the body and subsequent sporting performance is collectively acknowledged and thus techniques into negating these effects has been extensively researched. Individuals with lower core temperatures may have the opportunity to exercise for a longer duration, or to potentially increase exercise intensity throughout performance in the heat due to an increase time to a critical core temperature or a central drive to exercise is reached (Nielsen et al, 1993). Marino (2002) suggests that if a critical temperature is the limiting factor for duration of exercise, then commencing performance with a core temperature as low as possible would prove to be most beneficial.

2.6.1.2. The Pre-cooling Technique

Pre-cooling is a method for lowering initial core temperature that increases body heat storage; of which there are various methods of application (Quod et al. 2006). Although literature regarding its methodology appears only to be recent, the technique of pre-cooling has been researched since the late 1930’s, albeit for attempts to reduce thermal strain and investigating the dynamics of oxygen consumption at varied body temperatures (Bazett et al, 1937). It was not until 30 years later that pre-cooling was considered as a potential tool for improving physiological variables to aid performance, and not until 1979 that it was considered as an ergogenic aid for exercise performance (Bergh and Ekblom, 1979b). In this study, although reductions in oesophageal temperature were evident, the authors reported a decline in work time, thus poorer performance as a result of cooling, concluding this was due to a decrease in oxygen uptake. However, both the exercise protocol and method of cooling have to be questioned. To achieve desired cooling levels, participants were asked to exercise in cold water for up to 20 minutes which will have depleted energy stores to some extent and so reducing optimal performance potential. The cooling method also excessively reduced oesophageal temperature, so much so that multiple participants were in a state of hypothermia, rather than simply inducing mild cold stress, which has since been shown to cause vasoconstriction of the periphery and therefore altering the body’s natural
cardiovascular pattern (Therminarias, 1992) and hormonal responses (Therminarias et al. 1992).

2.6.2. The evolution and development of Pre-cooling – Modern techniques and their effects on exercise in a hot environment

Since the study of Bergh and Ekblom (1979b), many different cooling techniques have been developed. Initial experimentation simply used cold air exposure with a wind fan (Hessemer et al, 1984) whereas pre-cooling techniques of the present day include isolated locational cooling such as cold towels to the neck (Tyler and Sunderland, 2011) and ice packs to the thighs (Castle et al, 2006).

Lee and Haymes (1995) studied the effect of cold air exposure on fourteen male runners completing two high intensity (82 % maximal aerobic capacity) running tests to exhaustion having been subjected to a cooling condition and a control condition. For the control, participants sat in normothermic conditions (24 °C) for 30 minutes followed by 10 – 16 minutes of further rest. The cooling consisted of 30 minutes cold air exposure at 5 °C followed by 10 – 16 minutes of rest in normal conditions (24 °C) before then completing the exercise. It was concluded that exercise duration after pre-cooling was longer by 121 %. Rectal temperature was lower prior to exercise in the cooling condition compared to that of the control, and remained lower throughout the test. Although post-test analysis showed little differences in physiological variables (skin temperature, rectal temperature, heart rate, blood lactate) between conditions, body heat storage was greater throughout exercise as a result of the cooling condition with reduced stress on metabolic and cardiovascular systems. The ability to take on more heat without dramatic increases in cardiovascular and thermoregulatory strain as a result of the lower core temperatures is seen as the developing point for the progress of pre-cooling research.

2.6.2.1. Whole body immersion – the development of whole and half body immersion

The rate of heat transfer in water has been reported to be up to 25 times greater than in air of the same temperature (Noakes, 1986) and in severe cases of hyperthermia in athletes reduced
core temperature occurs up to twice as a fast as a result of whole-body cooling (Armstrong et al, 1996). Body heat content (BHC) as previously discussed in section 2.2.4.3 incorporates changes in overall body temperature and so reducing skin or core temperature individually or together will reduce BHC, with the magnitude of change in body temperature related to the change in BHC. With the belief of a high or critical core temperatures perhaps being one cause of fatigue, reductions in core temperature would appear the more obvious dependant factor to try to manipulate. Whole body immersion is a method employed to decrease both skin and core temperature. This was evident in a study by Booth et al (1997) who cooled eight participants via whole body cold water immersion before subjecting them to a self-paced time trial, with participants completing the furthest possible distance in 30 minutes. Reductions in core (rectal) and skin temperature before exercise were observed, consequently reducing mean body temperature which remained lower throughout the duration of exercise, although final rectal and skin temperatures were similar to that of the control.
Reductions in initial heart rate, and a lower heart rate for the first 10 minutes of exercise in conjunction with the reduced rectal temperature would have also improved physiological strain. As a result, total distance covered was improved. Similar improvements in self-paced time trial cycling were observed by Kay et al (1999) following whole-body water immersion, however the purpose was not to reduce core temperature, rather reduce skin temperature to manipulate a different variable associated with increasing the level of heat storage. Water temperature started at 29.7 °C and, after 10 minutes familiarisation, was continuously changed with cold water (8 – 11 °C), but ensuring the temperature did not drop at a faster rate.
than that of 2 °C per 10 minutes. Participants were immersed for up to 60 minutes with water temperature reducing by no more than 12 °C and at a slow enough a rate not to induce a drastic change in core temperature. Based on the equation for calculation of rate of heat storage presented in section 2.2.4.3, increased body heat storage should be apparent as result of the methodology. Nevertheless the contribution of core vs. periphery, particularly with the opinion that a critical limiting core temperature is an inhibitor to performance thus reduced core temperatures are the ergogen to delaying cessation, should be taken into consideration. Skin temperature was lower as a result of pre-cooling, consequently reducing total body temperature, and maintained lower than control throughout the duration of exercise. This significantly increased the rate of available heat storage by 69 W.m\(^{-2}\) with both skin and rectal temperatures remaining lower throughout the duration of exercise as a result of cooling, even though rectal temperature at the start of exercise was similar to that of the control. Rectal temperature at the termination of exercise was not different between conditions, yet distance covered in the 30 minute time trial was ~ 1 km further and this was ascribed to the increased body heat storage and reduced thermal and cardiovascular strain throughout the performance task.

It has been reported that core temperatures reduce as a result of peripheral cooling, likely to be as a result of subconscious vasodilation of the periphery. Under severe cold stress, the body employs a defensive mechanism to protect the vital organs located within the core region (Armstrong, 2000). Blood shunting through vasoconstriction of blood vessels within peripheral tissues causes a shift of warmer blood to the central organs and limits the distribution of heat from the core, in affect sacrificing the periphery to postpone core cooling (Moran et al, 2004). It would be logical to suggest that on removal of the stressor, rewarming of the periphery would be necessary to prevent chronic tissue damage with the heat coming from the core. Booth et al (1997) and Cotter et al (2001) reported reductions in rectal temperature post-cooling via whole body immersion prior to the performance task. Despite similarities in the protocol, Kay et al (1999) reported mild increases in skin temperature post cooling, however there was no reduction in rectal temperature. This may be as a result of the gradual process of cooling used as well as the higher water temperatures thus allowing greater familiarisation to the cold stress rather than a ‘shock-like’ effect to the body, instigating the need for peripheral vasoconstriction to ensure the core temperature is maintained. Nevertheless, core temperature was significantly lower after 15 minutes of exercise after cooling compared to the control. This may imply a reverse in theory, with
cooler peripheral blood returning to the core and the heat then being transferred away resulting in delayed lower core temperatures. Participant demographics may have contributed to the reduce rate of cooling too in the Kay et al (1999) study, with large body mass (Oksa et al, 1993) but not necessarily muscle mass (Oksa et al, 2007) reported to slow the rate of cooling. Duffield and Marino (2007) suggest that the benefits of pre-cooling may be affected by participant differences with body composition a limiting factor in both pre-cooling ability and heat tolerance.

Towards the end of the 1990’s, the theory became more understandable and scientists began developing pre-cooling aids more accessible to athletes and teams in the form of ice vests. The vests are normally applied prior to performance to reduce the body’s core temperature to create a heat sink, thus allowing more heat to be produced by the body before a critical temperature is reached.

2.6.2.2. The cooling vest – endurance exercise

The ice vest has become one of the most popular cooling methods with high profile sportsmen, being used in both normothermic conditions during match intervals to reduce increased thermal strain as a result of exercise and in hot humid conditions to reduce body temperature as a result of environment heat stress. Its popularity has more than likely grown due to its easy application as well as its lightweight storage, thus being able to be transported easily from location to location. Unlike ice baths, the ice vest also allows the individual to remain mobile and partake in any warm up activity, therefore achieving two goals during the same space of time. Research shows that wearing an ice vest prior to prolonged continuous activity improves overall time trial performance and increases time to exhaustion. Arngrimsson et al (2004) studied seventeen competitive distance runners’ time trial performance over 5km following a 38 minute active warm up during which they either wore a t-shirt for control or an ice filled vest for the cooling condition. Wearing the ice filled vest significantly blunted increases in body temperature, heart rate and increased perceptions of thermal discomfort. These differences were reported to have been eliminated during the first 3.2 km of the trial, however, a faster pace by participants was apparent in the last two-thirds of the race suggesting that the initial responses to the ice filled vest allowed a stronger finish to the trial and as a result, significantly reduce trial time by 13 seconds (1.1 % improvement).
This was calculated as a fifty seven meter improvement over 5000 m. It must be noted that since this study, Laursen et al (2007) have reported the typical error in 5 km running time trial protocols to be 2.0%, questioning how effective the intervention was.

Table 2.1. Mean total 5km running times for a control and following cooling via ice vest (Arngrimsson et al, 2004).

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>1.6 km</th>
<th>3.2 km</th>
<th>5 km</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vest</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>355 ± 35</td>
<td>355 ± 36</td>
<td>1,147 ± 130³</td>
</tr>
<tr>
<td>Control</td>
<td>724 ± 76�</td>
<td>717 ± 76</td>
<td>1,134 ± 132</td>
</tr>
<tr>
<td>Vest</td>
<td>1,147 ± 130³</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Nevertheless, reduced heart rate, skin and tympanic temperature were all reduced following pre-cooling, which have been shown to improve time to exhaustion on an incremental running test in the heat when compared to the effect of a warm up or a control (Uckert and Joch, 2007). The duration and perhaps intensity of the warm up may be questioned; 20 minutes of running at 70% maximal heart rate would induce some form of mild fatigue through depletion of energy stores, and completing this in hot conditions will have induced a level of hyperthermia prior to the performance task with elevated core temperatures of 1 °C higher than the control. Given that final core temperatures were similar between warm up and control, and lower for the cooling condition, reduced body heat storage can be suggested to be the limiting factor for running performance. Cooling both the core and periphery can be considered a means to achieve greater reductions in total body temperature and thus increase body heat storage, although this may possibly negate the effects of cooling. Cotter et al. (2001) cooled nine participants with an ice vest and cold air with the addition of leg cooling or warming via water-perfused cuff and concluded that pre-cooling via ice vest and leg cooling resulted in decreased thermal comfort compared to just vest cooling, although improved thermal comfort when compared to the control. Furthermore, additional leg cooling provided no extra benefit to the cooling of solely an ice vest despite greater reductions in mean body temperature being observed throughout the duration of the performance task. All cooling conditions reduced cardiovascular and physiological strain, as reflected in lower body temperatures and heart rates. The reduced muscle temperature and so reduced physical and biochemical processes involved in muscle contraction being inhibited as a result of addition
Leg cooling was suggested to explain the differences in performance between pre-cooling treatments. It was also suggested that a ‘double-exposure response’ may have inhibited thermoregulatory defence mechanisms through alteration in body temperature change thresholds resulting in reduced vasodilatory response and thus reduced cutaneous perfusion. Combined ice vest cooling and leg warming achieved an increased relative power output throughout the self-paced phase of the protocol of 17.5%, compared to a 16% improvement via leg cooling. This suggests that warmer muscles yet reduced core temperature is the optimal method to be employed.

2.6.2.3. The cooling vest – high intensity and intermittent sprint exercise

High intensity exercise and sprint exercise have not seen as much attention in the pre-cooling research, perhaps due to the nature of the exercise and the short durations involved, leaving limited time for the effects of pre-cooling to have an influence. Nonetheless, a large number of team sports involve repeated bouts of high intensity interspersed with recovery periods varying in length and duration, with match time being similar to some endurance events. Furthermore, some of the research relating to high intensity and sprint based exercise and pre-cooling appears to be less conclusive than that pertaining to performance of a longer duration. With the benefits of cooling on prolonged exercise evident within the literature, and the occurrence of bouts of steady state continuous exercise prevalent in intermittent exercise, it could be assumed that these benefits can be transferrable to high intensity repeated sprint exercise. Conversely, although the protocol was not specifically designed for games play, Cheung and Robinson (2004) found this assumption not to be the case. No differences were observed in peak and mean power output between cooling garment and control conditions in repeated-sprint exercise lasting a duration of 30 minutes. Mean skin temperature throughout exercise and initial rectal temperature were lower as a result of cooling, and peak heart rate during the sprints was higher in the control suggesting higher cardiovascular strain, however no differences in performance were observed. Similar observations were seen in a previous study (Drust et al, 2000) despite a different cooling technique being used. Six university standard soccer players completed a soccer-specific running test on a treadmill following a control or cold shower for 60 minutes. Cooling reduced rectal temperature response to exercise, but there were no differences in heart rate, metabolite utilisation or perceptive variables compared to a control. A third condition, mimicking a warmer environment (26 °C),
was also studied but only rectal temperature was different. Although observations in both studies show limited effects on performance despite reduced cardiovascular strain and lower core temperatures, both protocols were performed in moderate ambient temperatures and so individuals were unlikely to have experienced any exertional heat stress that would drastically disrupt homeostatic thermoregulation (Smith, 2005) suggesting that cooling would be far more beneficial on sprint performance when performed in the heat.

However, with a similar protocol to Cotter et al (2001), Sleivert et al (2001) had studied the effect of a combination of cooling methods on high-intensity exercise in the heat. In this study, body heat storage would in theory have been substantially reduced due to the cooling methods employed. Individuals underwent three trials; a control and two cooling conditions before completing a 45 second maximal output cycle test. Participants were cooled via ice vest on the torso in both cooling trials, with additional leg cooling or leg warming being the difference between trials. Results showed that both torso and leg cooling combined cause detrimental effects on 45 seconds sprint performance with peak power output and mean power output both limited. Torso-only cooling did not reduce peak power output compared to the control, nor did it provide the ergogenic aid expected. It may be the case that too large a heat sink was caused through both vest and leg cooling resulting in the body being too cold for optimal performance and so power outputs being reduced. A sub-study to this was the influence of warm-up on performance, with no warm-up after cooling displaying the poorest power outputs (Sleivert et al, 2001). Warm-up and leg warming both increased muscle temperature to similar to that of the control prior to the exercise task. It is obvious from this study that cold muscles are not fully functional which will be a hindrance to performance tasks, especially those of high intensity and short duration.

With the easy application of the ice vest, researchers have tried to utilise the technique for intermittent performance. Duffield et al (2003) looked to test the theory of multiple cooling periods on seven trained male hockey players. Individuals completed two conditions, one control and one containing multiple cooling periods whilst completing 80 minutes of intermittent repeated sprint cycling in 30 °C heat and 60 % relative humidity. Cooling periods consisted of wearing an ice vest five minutes before the protocol and during the recovery periods, twice for 5 minutes and once for 10 minutes. Naturally, torso skin temperature and rating of thirst significantly reduced while participants reported an increase in thermal comfort, however there were no significant differences in power output and work done, or
core body temperatures compared to the control and thus the use of an ice jacket in this manner proving to be non-beneficial. The duration of exposure to the ice vest during this study suggests that short bouts of exposure do not induce large enough changes in core temperature to produce the body heat sink required to aid in increased body heat storage and consequently improve intermittent sprint performance.

Luomala et al (2012) studied the effects of cooling midway through sub-maximal cycling performance in the heat. A cooling vest was added 30 minutes into a repeated protocol to exhaustion where participants completed nine minutes of 60 % \( \dot{V}O_{2\text{max}} \) followed by one minute at 80 % \( \dot{V}O_{2\text{max}} \). Time to exhaustion was improved, with the authors stating that this was due to reduced neuromuscular fatigue reflected in maintained mean power frequencies at towards exhaustion. Duffield and Marino (2007) further investigated the effect of half time cooling using two cooling techniques; an ice vest or an ice vest-immersion combination, on repeated sprint performance. Individuals completed 15 minutes of each cooling technique prior to any exercise, with an additional 10 minutes or cooling at a half time interval. The protocol consisted of a 15 meter all out sprint separated by a walk, jog and run at the participants own pace. There was no difference between conditions for sprint times or the distance covered during recovery. However, the combination of bath and vest showed an apparent improvement in the hard running phase of recovery when compared to the control. Additionally, reduced thermoregulatory strain and improved thermal comfort were evident as a result of combination cooling. This lead the authors to conclude that it may not be the maximal intensity phases that are influenced by the benefits of cooling, instead sub-maximal intensities are greater effected, which may influence outcome of overall performance in team sports involving both maximal and sub-maximal intensities. This was also observed by Skein et al (2012) who saw higher self-selected exercise intensities during a 50 minute repeated sprint test as a result of whole body immersion, however initial sprint performance was lower than when passive heating was employed. The controversy is that although cooling negates thermal strain on the body, initial sprint performance may be poorer, while warming the body may improve early sprint performance through warmer muscles and thus improved blood flow and increased conduction velocity (Bishop, 2003). However being warmer at the start of intermittent exercise in a warm environment will induce faster cessation due to lower body heat storage available (Jay et al, 2007). Skein et al (2012) state increasing physiological and thermal load may result in lower self-selected exercise intensities towards the latter stages of
performance which in turn may have negative consequences on match outcome (Mohr et al, 2003).

A large disadvantage with long duration cooling such as that by Sleivert et al (2001) is the thawing or warming of the cooling aid, therefore reducing the benefits associated with cooling through gradual rewarming of the body or through potential shivering thermogenesis. In addition to this, long durations of cold exposure may result in increased participant discomfort reducing motivational drive and the desire for future use accordingly, as described by Abbiss and Laursen (2005).

2.6.2.4. Isolated leg cooling through ice pack application – high intensity and intermittent sprint exercise

Early research looked at Wingate performance, albeit in temperate conditions, following leg cooling and saw reductions in power output although fatigue index was reduced (Crowley et al, 1991). This is in contrast to Marsh and Sleivert (1999) who saw improvements in 70 seconds high intensity cycling performance compared to a control, with reductions in core and skin temperature and heart rate. The reduced peripheral temperature was linked with possible increased blood availability to the working muscles through vasoconstriction of those muscles not required for performance. Crowley et al (1991) stated that despite reduced power output, there must be an optimal temperature range within which the greatest outcome of muscle performance is achieved and that peripheral hyperthermia is as much of a cause of muscle function impairment as other central causes. With evidence from Kay et al (1999) that improvements in cycling time can be achieved through reductions in shell temperature without reduced core temperatures, as well as the inhibitory effects of heat on the muscle discussed in section 2.5.4, it could be logical to assume that local cooling via direct application will yield similar results to systemic cooling methods. Although the number of studies involving cooling and intermittent exercise appear limited, with some literature suggesting local cooling negates performance (Sleivert et al, 2001), Castle et al (2006) tested this hypothesis, employing three differing pre-cooling techniques to alleviate heat stress on intermittent sprint performance for twelve male team sports players.
Participants completed four CISP’s following a 20 minute cooling technique; systemic cooling via ice vest or whole-body water immersion, local muscular cooling through application of ice packs to the quadriceps and hamstring muscle groups, or a control. All three techniques reduced skin temperatures compared to the control, however the vest did not reduced muscle temperature whereas the other techniques did. Unlike Kay et al (1999), rectal temperature was reduced as a result of water immersion as well as vest and ice packs. This was likely to be due to the differences in protocol with water temperature in Castle et al’s (2006) study approximately 8 °C lower. Rectal temperature remained lower than the control during the CISP with skin temperature remaining lower until sprint 4 for packs and vest and throughout the protocol for water. Castle et al (2006) reported thermal sensation differences between techniques, which based on theories by Marcora et al (2009), can be a key indicator for cessation of exercise. All techniques reduced thermal sensation by the end of pre-cooling, however these feelings were maintained until the end of the warm up for water immersion. This may suggest that the reduced core temperatures contribute to the improved feelings of
thermal sensation. Only ice packs and water immersion reduced muscle temperature significantly compared to the control during cooling, with muscle temperature remaining lower until sprint 8 for packs and through the duration of the CISP as a result of water immersion. However, these reductions in muscle, skin and rectal temperature as a result of cooling were not reflective in performance in all conditions. Packs produced the main effect, improving peak power output by 4% compared to the control with no other differences between conditions. The ice vest was only beneficial during the final stages of the CISP (Figure 2.16). Additionally, lower peak power output was observed as a result of water immersion at the start of the CISP suggesting that the body was too cold to perform high intensity exercise, contrasting that of Vaile et al (2008) who found that cold water immersion was affective in maintaining high-intensity cycling performance in the heat. Castle et al (2006) suggested this response to be as a result of a $Q_{10}$ effect on ATP hydrolysis. Interestingly, there was not a similar reduction in peak power output following ice pack cooling, despite significant reductions in skin and muscle temperature. It was proposed that this method of cooling offset the sensory afferent feedback to a central regulation centre through a paradoxical effect, resulting in an incorrect but beneficial motor recruitment response, consequently improving power output. Given the greatest improvements in PPO across a duration of a 40 minute CISP, leg cooling will be adopted as the chosen method of cooling within this thesis.

2.6.2.5. Mixed method cooling and duration dependant responses – high intensity and intermittent sprint exercise

Following on from Castle et al (2006), Duffield et al (2010) used ice pack application to the quadriceps in a mixed-method cooling protocol to limit the effects of heat on a self-paced intermittent-sprint exercise protocol involving lacrosse players. Players completed a 30 minute conditioning session incorporating self-paced sprints and self-paced recovery. The local cooling of the quadriceps was used in conjunction with ice vest and cold towels to the neck. Neck cooling has previously been shown to improve running performance (Minniti et al, 2011) through potential improvements in cerebral blood flow (Nybo et al, 2002) although possibly reducing feelings of comfort and increasing perceived exertion (Minniti et al, 2011). The mixed-method cooling saw improvements in total distance covered, although maximal sprint speeds were not different from the control. Instead, distance covered was increased in
the moderate-intensity phases. Cooling prevented a rapid rise in core temperature, which was lower throughout exercise. As with Skein et al (2012) and Duffield and Marino (2007), it was concluded that pre-cooling allowed for selection of higher sub-maximal intensities, possibly due to the delay in down-regulation of muscle recruitment or suboptimal net motor unit activity (Mendez-Villanueva et al, 2008; Racinais et al, 2008) associated with protecting against thermal stress.

The mixed-method pre-cooling technique has since been used (Minett et al, 2012) in an aim to determine a duration-dependant response to pre-cooling on intermittent-sprint exercise in hot conditions. Individuals were cooled via the same procedure as Duffield et al (2010) before completing a 10 set intermittent sprint protocol consisting of repeated-sprints simulating that of a cricket over and five minutes of self-paced running similar to Duffield and Marino (2007). Cooling treatments were given prior to performance for either 10 or 20 minutes as well as for the final five minutes of a 15 minute recovery interval at the half-time point. Cooling for 20 minutes improved peak sprint times post recovery interval as well as reduced percentage decline in sprint times when compared to the control. Additionally, the 20 minute condition allowed for greater distances to be covered when compared to the 10 minute condition. The authors attributed this to a greater body heat reserve however a lack of difference in core temperature values between cooling conditions suggests that other variables must have an influence. The authors concluded that a dose-response is evident within pre-cooling with longer durations providing an ergogenic benefit as a result of sustained neuromuscular function and greater endogenous thermoregulation, which seem more feasible in explaining the improved performance.
2.6.2.6. Pre-cooling summary

It may be plausible to suggest that manipulation of body temperature should be a carefully selected process with consideration of the exercise task involved. Pre-cooling has been shown to improve endurance performance in a variety of settings. Environmental heat stress poses additional problems to performance in the heat, with pre-cooling literature contradictory as to the influences of pre-cooling on performance. Short duration high intensity exercise may be hindered as a result of cooling particularly when body heat storage is substantially reduced (Sleivert et al, 2001) and this may be due to the limited time available for i) heat stress to influence performance and ii) for the benefits of cooling to take place. This notion is supported by Marino (2002) who states that pre-cooling might only be beneficial for endurance exercise of more than 30 minutes rather than short-duration higher intensity exercise. However, more recent research shows that intermittent sprint performance of longer durations can be improved as a result of cooling, with both systemic and local cooling both improving power output (Castle et al, 2006, Vaile et al, 2008) yet local cooling being the most effective (Castle et al, 2006). It has been suggested that pre-cooling influences recovery periods and intensities of sub-maximal level more than that of the maximal sprint efforts themselves and that it is this that allows for prolonged performance in intermittent sprint performance. Furthermore, a duration-dependant response to cooling may appear to exist although further research is required in this area.
2.7. Summary of Literature Review

Despite continual subconscious thermoregulation of the body, uncompensable heat stress negatively effects performance, in particular high intensity intermittent sprint exercise. Many theories of fatigue have been proposed with heat per se raising body temperature to that of a limiting temperature, although this theory has been questioned within the literature. Alternatively, some authors believe that the body has a central regulation system that influences performance intensity and dictates cessation of exercise. There is wide debate as to whether this occurs through core or peripheral afferents, such as skin and blood temperature or the production of metabolites within the muscle, or whether it is mediated on a psychological level.

Irrespective of the causes of fatigue and the possible fatal risks of heat illness, highly motivated athletes continuously push themselves to exhaustion and so techniques to alleviate heat stress have been devised. Acclimation has long been used as preparation to competition in a hot environment, due to adaptations within the body to the environmental stressor, although it can be deemed as a time consuming and therefore potentially expensive method. Acute pre-cooling techniques, which are a lot more time efficient, can be applied immediately prior to performance, with Castle et al (2011) referring to pre-cooling as a short-term intervention as the benefits can last up to 40 minutes. Advances in science have developed the technique from whole-body immersion tanks that are perhaps somewhat impractical to the lightweight and more practical ice vest. Recently, studies have shown that isolated leg cooling is an effective method of pre-cooling, however little is known as to why this may be the case.

With this in consideration, the question of which cooling technique is more beneficial still remains, especially when pre-cooling via ice pack application has been shown to improve peak power output in intermittent sprint performance by 4 % (Castle et al, 2006). Despite the cooling technique used to alleviate heat stress, the underlying mechanisms behind pre-cooling still appear equivocal. This would suggest that an increased understanding of pre-cooling and refining the technique used could help improve physiological benefits and sporting performance further, reducing the need for sports teams and athletes alike to opt for an ‘either or’ case scenario, and choose a heat stress alleviation technique depending on the nature of the competition one is partaking.
2.8. LIST OF HYPOTHESES

With the growing prevalence of intermittent sprint research becoming apparent in the literature, as well as the known effect heat has on performance, developing the understanding of tools that aim to negate these effects would be beneficial to sports teams and athletes alike. The intention of this thesis aimed to achieve this by testing the following hypotheses:

1a. The Cycling Intermittent Sprint Protocol (CISP), as used by Castle et al (2004; 2006; 2012) will demonstrate reliability following a test-retest design in temperate conditions.

1b. Completing a five-sprint familiarisation period will be sufficient enough to overcome a learning effect for the main CISP.

2a. Thigh cooling for 25 minutes using ice packs will cause reductions in skin and muscle temperature, consequently reducing core temperature compared to a 25 minute period of no cooling.

2b. Reductions in skin temperature, muscle temperature and core temperature will demonstrate strong interacting relationships with one another during 25 minutes of thigh cooling.

3a. A duration-dependant response to leg cooling using ice packs will be evident, with longer cooling periods eliciting the largest changes in core and muscle temperature.

3b. All leg cooling conditions will cause improvements in intermittent sprint exercise in hot humid conditions compared to a non-cooled control, with the larger physiological responses to leg cooling resulting in the greater improvements.

4. The equation proposed by de Ruiter et al (1999) that enables the prediction of muscle temperature from a given skin temperature will provide inaccurate values in comparison to a measured muscle temperature.
CHAPTER THREE

General Methods
CHAPTER III: GENERAL METHODS

The following chapter will describe methods, procedures and materials that are frequently used in two or more study chapters of this thesis. Where this was not the case, specific additional details are provided in the relevant study chapter.

3.1. Health and Safety

All experimentation took place in a BASES accredited environmental physiology laboratory at the University of Brighton, School of Sport and Service Management, formally the Chelsea School of Sport and Research Centre.

Ethical approval was granted by the Faculty Research Ethics and Governance Committee and the standard procedure of two or more experimenters to be present during any experimental testing was followed.

3.2. Participants

All participants were volunteers from University or county standard level sports and all competing in regular team sport activity three or more times per week. A team sport was defined as any activity involving more than one player where those players are required to complete both repeated short duration high intensity exercise combined with longer durations of low intensity exercise over a period of 30 – 120 minutes (e.g. rugby, football and hockey) (Sirotic and Coutts, 2008). All participants were provided with a written information sheet explaining the purpose, procedures and risks of participating in the relevant studies and were provided with the opportunity to ask any questions. An informed consent and medical questionnaire were completed prior to any testing at each visit to the laboratory, with strict criteria for eligibility to participate (See Appendix A). All participants were informed that they could withdraw from the studies at any time without having to provide a reason. Participants were anonymised during all studies so that personal details and data were kept confidential.
3.2.1. Participant Criteria

Participants were requested to follow a similar dietary pattern in the 24 hours prior to testing and were told to maintain any usual exercise routines during testing timeframes (to be discussed later). Participants abstained from alcohol, caffeine and vigorous exercise in the 48 hours before commencing each testing session. All participants were free from any previous heat or cold exposure for at least 6 weeks prior to the start of each study.

3.3. Experimental Design

In chapters IV and VI where the cycling intermittent sprint protocol (CISP) was employed, the first two visits to the laboratory involved preliminary measures and a familiarisation. Experimentation for visits 1 and 2 took place in a laboratory at room temperature (18 – 20 °C, ~ 40 % relative humidity). During subsequent visits, the CISP was completed, with the number of times detailed in each relevant study chapter. Where exercise testing took place, a within subject factors design was applied, with participants acting as their own control.

3.3.1. Visit 1: Preliminary Measures and VO\textsubscript{2Max} Test

On arrival to the laboratory for visit 1, participants were measured for height (cm) and nude body mass (kg) on a stadiometer (Harpenden, UK). Skinfold callipers (Idass, England) were used to determine the sum of skinfolds from four sites as described by the method of Durnin and Womersley (1974). Participants then completed an incremental maximal oxygen uptake test on a modified cycling ergometer (Monark 620 Ergomedic, Varberg, Sweden) fitted with power cranks (SRM: Scientific Model, Julick, Germany). Saddle height was set at a level parallel to the participants’ iliac crest, ensuring there was a slight bend in the leg at the lowest point of the pedal revolution. This height was recorded and used throughout each trial. Workload started at 96 W and increased incrementally by 24 W every stage until volitional exhaustion. Oxygen uptake was recorded using open air spirometry with expired air collected during the last minute of each stage for approximately 45 seconds. Oxygen, carbon dioxide, gas temperature and expired volume were all analysed (Servomex, Crowborough, England) to determine oxygen uptake, providing the author with a cycling VO\textsubscript{2peak} rather than VO\textsubscript{2max}. 
due to the abrupt termination of the test after the addition of the catastrophic workload/resistance.

3.3.2. Familiarisation to the Cycling Intermittent Sprint Protocol (Visit 2)

All participants were familiar with the technique of stationary cycling and were explained the protocol fully before commencing any testing. Familiarisation of the CISP comprised of one quarter of the full CISP (17 minutes) including a standardised warm up. Warm up consisted of 5 minutes steady cycling at 80 revs.min\(^{-1}\) against 95 W followed by two periods of 30 seconds passive rest and 30 seconds at 120 W, achieved by increasing revolutions to 100 revs.min\(^{-1}\).

3.3.3. The Cycling Intermittent Sprint Protocol (CISP)

The CISP comprised of a standardised warm up as described in section 3.3.2. followed by twenty, two minute bouts consisting of a ten second passive rest, a five seconds maximal sprint from a stationary start against a resistance of 7.5 % body weight (kg), 105 seconds active recovery against a pre-determined workload (35 % \(\dot{V}O_{2\text{peak}}\)) (Figure 3.1). The total length of the CISP protocol was 47 minutes inclusive of warm up. The CISP was performed on a modified cycle ergometer as described in section 3.3.1. in either an environmental chamber (Model TDL 500, Thermal Dynamics Limited, Swindon, UK) or experimental laboratory, details of which are provided in relevant chapters. A photograph of a participant completing the CISP in the environmental chamber can be seen in Figure 3.2.
Figure 3.1. Schematic representation of a standardised warm up (left of dotted line) comprising of 5 minutes active cycling at a constant load, two 30 second periods of passive recovery (R) separated by two 30 seconds bouts at 120 W, and one 2 minute block of the CISP comprising of 10 seconds passive rest, 5 seconds maximal sprinting and 105 seconds active recovery.
Figure 3.2. Participant completing the 5 second maximal sprint phase of one 2 minute block of the CISP.

3.4. Additional Measures, Procedures and Apparatus

The following section lists all measures and apparatus that were used in two or more studies, occurrences of which will be mentioned in relevant chapters.

3.4.1. Thermoregulatory Measures

Core temperature (Tre) was measured using rectal thermistor probes (Henley Medical, UK) measured at a depth of 10 cm passed the anal sphincter. Zinc oxide tape marked the relevant depth in order for participants to know they had inserted the probe to the correct depth and to prevent the probe from coming out during exercise bouts. The probe cable was then carried out of the top of the participants’ shorts, across their hip region and attached with a clip to prevent wires catching. The thermistor was plugged into a data logger (Libra Medical, Minnesota, USA) and temperature measured to an accuracy of ± 0.1 °C. Resting Tre was documented post participant preparation and set up to be used as a baseline for the criteria.
stated in section 3.1. Tre was recorded during all cooling trials, durations of which are stated in the relevant chapters. Skin thermistors (Squirrel Meter Logger, Grant Instruments 1000 series, Cambridge, UK) were used to measure skin temperature (Tsk) throughout experimentation, with data reported to an accuracy of ± 0.05 °C. Thermistors were placed on 4 sites; pectoris major, the muscle belly of the triceps brachii, over the central point of the anterior aspect of the thigh (skin covering the muscle belly of the rectus femoris) and the centre point of the muscle belly of the gastrocnemius located at the posterior aspect of the calf. Thermistors were attached using 3 pieces of zinc oxide tape to ensure each was attached securely. Where required, mean skin temperature was calculated using equation 2.4 (Ramanathan, 1964) (chapter II, section 2.2.4.3).

Muscle Temperature (Tmu) was measured to a pre-determined depth via ultrasound measurement in the vastus lateralis, using an 18 gauge 1.2 x 40 mm needle and data logger (Ellab Medical precision thermometer, DM852, Denmark). Ultrasound measurements were taken prior to any testing to gauge depth of the needle into the centre of the muscle belly and to ensure that there was no risk of striking the femur with too deep insertion of the indwelling muscle probe (Figure 3.3). Participants were in a relaxed seated position with their knee flexed at an angle of approximately 90°. All participants volunteered to have Tmu taken in all studies and were all offered anaesthetic cream (Elma, UK) prior to any readings to be taken place. Any individual that used the cream applied a generous layer to the designated site of Tmu measurement, as located by a physician, 1.5 – 2 hours before Tmu measurement. Where cream had been applied, it was removed with a damp towel immediately before Tmu measurement. The measurement site was sterilised with an alcowipe and dried with tissue before the measurement was taken. Tmu was always taken from the left leg for consistency purposes. Pilot work indicated that this provided easier measurement of Tmu for a right handed experimenter whilst providing minimal discomfort to the participants. The needle was inserted at 45° angle following the striation of the muscle fibres. The muscle thermistor was then fed through the needle and left for 10 seconds in order for a stable temperature to be reached. Temperature was recorded to an accuracy of ± 0.1 °C (Figure 3.4). Specific details of muscle temperature measurement (i.e. time points and frequency) are described in relevant study chapters.
Figure 3.3. Ultrasound image showing depth to the femur (A), depth of the vastus lateralis muscle (B) and the depth to the belly of the vastus lateralis muscle (C) for muscle temperature measurement.
Figure 3.4. The technique of muscle temperature measurement in the vastus lateralis muscle of the left leg, inserted to a predetermined depth via ultrasound measurement, with the participant in a seated relaxed position.

3.4.2. Heart Rate

Heart rate (HR) was recorded during every visit to the laboratory. The HR monitor (Polar Instruments FS2c, Polar Electro Oy, Professorinite 5, Kempele, Finland) was placed just below the sternum, running horizontally across the distal third of the pectoris major. The sensors were lubricated prior to use to ensure a strong pulse was detected. HR data was recorded every minute during the VO₂max test. During the CISP, HR was recorded one minute
into each 2 minute block for the duration of the protocol. HR was also recorded during all cooling procedures, details of durations are provided in relevant chapters.

3.4.3. Subjective Measures

Participants were asked to provide a perceptual feeling of effort during physical exercise. This was determined using Borg’s rating of perceived exertion (RPE) scale (Borg, 1970), which ranges from 6 (very very light) through to 20 (very very hard) with each number signifying a feeling of work rate. RPE was recorded during every minute until exhaustion during the \( \dot{V}O_{2\text{max}} \) test. During CISP trials, RPE was recorded at the minute stage after a sprint of each two minute block. RPE was also recorded at rest during cooling trials to evaluate exertion as a result of shivering thermogenesis. Perceptual feeling of body temperature was also recorded at the same intervals as RPE during CISP trials. This was achieved using Toner et al.’s (1986) thermal sensation scale (TS), which ranged from 0.0 (unbearably cold) to 8.0 (unbearably hot) at 0.5 intervals. During cooling periods, TS was also recorded specific to the cooled area (the thigh). For perceptive scales see Appendix B.

3.4.4. Performance Variables

All performance data was logged onto an SRM data box (SRM Powercontrol Meter, Julick, Germany) which was calibrated before each use. Calibration was completed against unloaded backwards cycling following that of manufacturer’s instructions. The slope of the Powermeter was set at 16.2 Hz/Nm with a sampling rate for interval storage of 0.5 seconds. The SRM box records markers for HR (beats.min\(^{-1}\)), power output (PO) (W), speed (km.h\(^{-1}\)), cadence (revs.min\(^{-1}\)), altitude (m) and temperature (\(^{\circ}\)C). PO, HR and cadence were the markers left visible for the \( \dot{V}O_{2\text{max}} \) test. During CISP protocols, HR and PO were the only two visible markers. PO was broken down into peak power output (PPO), mean power output (MPO) and work done (WD) (kJ). PPO (W) was taken as the highest single recorded value measured throughout the five second sprint.
3.5. Pre-cooling Technique

Cooling was achieved using gel ice packs (Hot and Cold Gel Pack, Kool Pak, UK), targeting the quadriceps (rectus femoris, vastus lateralis, vastus medialis) (Martini, 2004) and the hamstrings (biceps femoris, semimembranosus, semitendinosus) (Martini, 2004). Application was achieved through a two layer tubigrip bandage formulating a make-shift leg section of a pair of shorts (Figure 3.5) covering the hamstrings and quadriceps. Gel packs were slid in between cloth layers so to ensure secure application and to prevent possible ice burn through direct contact with the skin. Participants were in a seated position during cooling depending on their level of comfort with the upper portion of the legs resting on the hard surface of a bench, table or chair. The duration of cooling was determined from the point at when the last ice pack was secured in the tubigrip. Ice packs were assumed to have a surface temperature of -16 ± 5.6 °C on removal from a -20 °C freezer following the procedure of Castle et al (2006). All cooling took place in an ambient environment, simulating that of an air-conditioned changing room.

Figure 3.5. The pre-cooling technique used (prior to the legs being outstretched), with a tubigrip bandage fashioning a double layer pair of shorts with Kool Pak gel packs to cool the hamstrings and quadriceps muscles.
3.6. Testing Timeframes and Recovery

The following section details the duration of each study with rest day intervals between certain visits to allow for suitable recovery. It also details any techniques used to aid in returning participants to baseline levels after each visit.

3.6.1. Testing Timeframes

In each study, participants were provided with details of how many visits they would make to the laboratories and how long the study should last. This duration included days off between testing to ensure that participants were recovered from previous trials. Participants arrived at the laboratory at approximately the same time of day on each visit through the duration of the study to limit the effect of circadian rhythms on physiological variables such as blood pressure (Smolensky and Haus, 2001) and core temperature (Portaluppi et al, 2012) and thus consequently performance (Atkinson and Reilly, 1996). Participants were given a minimum of three days in between trials to allow any exercise fatigue or heat strain to subside with a maximum of two trials in any given week. All participants were informed to not eat in the 2 hours leading up to any exercise and to arrive at the laboratory hydrated. A urine sample was collected, and hydration status determined via urine refractometer measurement (Uronic NE, Atago Co. LTD, Tokyo, Japan) and dip test strips (Combur 10-test, Roche Diagnostics, Mannheim, Germany). Acceptable hydration for commencement of exercise was considered as a USG < 1.015 (Armstrong et al, 1994b, Armstrong, 2005). Where participants were not acceptably hydrated, additional fluid was consumed and a retest conducted or testing was postponed to another date.

3.6.2. Recovery techniques

Recovery techniques were prepared prior to testing. Fluid intake during any trial was not permitted, nevertheless fluid was provided immediately after termination of any test. A recovery bed was present in all laboratories. To cool participants post exercise, individuals were provided with a cold drink, cooled in a water bath with mild-cold water, with a large fan, wet towelling at the top of the spine and with water buckets for hands and feet, with
these techniques having been shown to rapidly reduce core body temperature (Harker and Gibson, 1995) during heat stress. Participants were not permitted to leave the laboratory until core temperature had returned to within 0.5 °C of baseline starting values. Food was provided post exercise to elevate energy levels sufficiently.

3.7. Statistical Analyses

All data analysis was completed on the statistical programme SPSS (statistical package for social sciences), versions 16.0 to 20.0. All data was checked for normality, and corrected using Huynh-Feldt or Greenhouse Geisser method accordingly where relevant depending on the level of sphericity. Data across all performance trials was analysed using a two-way repeated measures analysis of variance (ANOVA), conditions versus time and significant differences determined using Tukey’s honestly significant difference post hoc analysis test (Vincent, 1999; Field, 2009). All data was reported as mean ± SD and significance was accepted at the $P < 0.05$ level. Where differences were not found but $P$ values were approaching significance, exact values will be reported. Where relevant, Line of Equality plots have been conducted. Line of Equality plots demonstrates the level of distribution and diversity between two sets of data measuring the same outcome. Equality between data sets is shown by data points lying along the 45° ‘line of equality’ and variances will lie to the left and right of the line. Any other data analyses conducted for individual studies will be discussed in the relevant chapters.
CHAPTER FOUR

Study One

Peak Power Output provides the most reliable measure of performance in prolonged intermittent sprint cycling
CHAPTER IV: PEAK POWER OUTPUT PROVIDES THE MOST RELIABLE MEASURE OF PERFORMANCE IN PROLONGED INTERMITTENT SPRINT CYCLING

4.1. INTRODUCTION

Team sports such as rugby union, hockey and football are characterised by periods of intermittent high intensity activity, interspersed with longer spells of recovery and lower intensity activity (Williams, 1990). In such sports players typically complete twenty to sixty sprints per game with mean sprint durations of 1.6 – 3.2 seconds and mean recovery times between sprints of ~ 2 minutes (Spencer et al, 2004; Spencer et al, 2005; Roberts et al, 2008). The contribution of sprinting to the total activity profile in field-based sports is therefore small. Nevertheless, sprinting frequently precedes significant moments in play and as such, may be considered critical to the outcome of a game (Reilly, 1997; Spencer et al, 2004). Consequently a number of performance based protocols have been devised to examine the physiological and metabolic demands of sprinting in field based sports, including the Bangsbo Sprint Test (Bangsbo, 1994), the Loughborough Intermittent Shuttle Test (LIST) (Nicholas et al, 2000) and the Soccer Specific Test of Prolonged Repeated Sprint Ability (SSIET) (Oliver et al, 2007).

The majority of the aforementioned protocols are running based and use durations, intensities, and work-rest ratios developed from time motion analysis of team sports. Typically, in running based protocols, athletes are required to complete 6 – 21 sprints of 20 – 40 meter interspersed with 15 – 100 seconds of recovery (Nicholas et al, 2000; Oliver et al, 2007; Gabbett, 2010). Such protocols have demonstrated good reproducibility, logical and construct validity and specificity. However, they are not conducive to sophisticated measurement of complex physiological and metabolic variables necessary to develop understanding of the demands of high intensity activity (McGawley and Bishop, 2006). Cycling based protocols focussing on assessment of repeated sprint ability (RSA), an important determinant of successful performance in team sports (Spencer et al, 2005; McGawley and Bishop, 2006; Mendez-Villanueva et al, 2008), have been shown as a valid measure of sprint activity in games play (Bishop et al, 2001). However, such protocols, typically comprising 5 – 10, 6 seconds sprints with 24 – 30 seconds recovery (McGawley and Bishop, 2006; Mendez-Villanueva et al, 2008; Bishop et al, 2001), due to the truncated
duration of test the design, do not replicate intermittent sprint activity across durations consistent with many team games.

The accurate assessment of reliability should consider systematic measures such as a learning effect due to a lack of familiarisation, given that in multiple sprint-cycling based activity, learning effects have previously been observed (Phillips et al, 2004). In contrast to existing literature that identifies the need for two familiarisation sessions when repeated sprint exercise is performed on a cycle ergometer (McGawley and Bishop, 2006; Capriotti et al, 1999), practice or learning effects are minimised during the CISP by completion of one familiarisation session comprising a quarter of the protocol prior to the first main trial. The CISP permits examination of the physiological responses to intermittent sprint exercise, however, whether a single familiarisation is sufficient to negate learning is currently unknown.

Previously the CISP has been used to examine the effect of pre-cooling (Castle et al, 2006) on intermittent sprint performance and has demonstrated a significant 4 % improvement in PPO compared to a no cooling control trial. However, the reliability of the protocol has not been extensively determined (Castle, 2006). Running based tests designed to assess prolonged repeated sprint ability have reported TEM values for PPO of up to 7.9 % (95 % CI 5.8 – 14.4 %) (Oliver et al, 2007). As such it is difficult to determine if changes reported in previous studies using the CISP represent worthwhile interventional change or variation inherent in the test.

Therefore, the primary purpose of this study was to determine the reliability of the CISP using a group of well-trained games players. A secondary purpose was to assess the effectiveness of a five sprint familiarisation session prior to the main protocol.
4.2. METHODOLOGY

4.2.1 Participants

Eleven male participants (mean ± S.D: age, 23 ± 2.4 years; height, 178.5 ± 5.9 cm; body mass, 82.3 ± 8.4 kg; sum of skinfolds, 36.7 ± 11.2 mm; peak oxygen consumption, 42.6 ± 4.75 ml.kg⁻¹.min⁻¹) participated in this study, which was approved by the Institutional Ethics Committee.

4.2.2. Experimental Design

During the first visit to the laboratory, preliminary measures and the \( \text{V}O_{2\text{max}} \) test were completed as described in chapter III (section 3.3.1). After a 25 ± 5 minute self-paced recovery period, a familiarisation to the cycling intermittent sprint protocol (CISP) was undertaken. On the second and third visits participants completed the CISP (CISP1 and CISP2 respectively).

4.2.3. Data Analyses

Peak power output (PPO) was determined as the highest recorded power output during each sprint. Mean power output (MPO) was determined from the highest 3 second power output from the 5 second sprint to overcome frictional factors experienced due to starting sprints from a stationary position (Winter and Fowler, 2009). To determine the effectiveness of one familiarisation session before main trials to overcome learning effects, data from the five practice sprints were compared with those from the first five sprints of CISP1 and CISP2.

4.2.4. Statistical Analysis

Data for PPO and MPO was analysed using a two-way repeated measures ANOVA (condition vs. time). Data was assessed for heteroscedasticity using plots of the log transformed data and reliability measures. Typical error of the measurement (TEM), calculated from the standard deviation of the mean difference for each pair of trials using the
formula $TEM = SD_{diff}/\sqrt{2}$ and expressed as a mean coefficient of variation (CV), and intra-class correlation (ICC) were calculated from Log transformed data. ICC was determined using a reliability spreadsheet (newstats.org/xrely.xls) as used by Laursen et al (2007). Confidence limits of 95 % were determined for TEM and ICC using the methods described by Hopkins (2007). Limits of agreement were also calculated to determine the bias from the mean difference and standard deviation of differences. Sprints were also grouped into four phases comprising of five sprints each, for better analytical clarification. Significance was accepted at the level of $P < 0.05$. Effect sizes (ES) were estimated using partial Eta squared where 0.2 represented a ‘small’ effect size, 0.5 a ‘medium’ effect size and 0.8 a ‘large’ effect size (Nakagawa and Cuthill, 2007).
4.3. RESULTS

4.3.1. Familiarisation vs. CISP 1 vs. CISP 2

Mean PPO and MPO for the familiarisation period were not different from the first five sprints of CISP1 or CISP2 ($P > 0.05$, $ES < 0.058$). TEM and ICC for familiarisation vs. CISP1 and CISP2 were 2.6 % (1.8 - 4.7 %), 0.94 and 2.5 % (1.7 – 4.7 %), 0.95 respectively.

4.3.2. CISP 1 vs. CISP 2

There were no differences between CISP1 and CISP2 for mean PPO across all 20 sprints ($P > 0.05$, $ES = 0.073$). No differences between CISP1 and CISP2 were observed when grouping the sprints into four phases ($P > 0.05$, $ES = 0.070$). Mean MPO across all 20 sprints was not different between CISP1 and CISP2 ($P > 0.05$, $ES = 0.005$). Grouping the sprints into four phases also showed no difference between CISP1 and CISP2 ($P > 0.05$, $ES = 0.005$). Mean PPO demonstrated less variability than mean MPO throughout the CISP (mean of sprints 1 – 20), despite both showing strong correlations (Table 4.1).

Table 4.1. Mean (± SD) peak power output (PPO) (W), three second mean power output (MPO) (W), work done (WD) (J) TEM and ICC scores for 20 sprints, CISP trial 1 (CISP1) and CISP trial 2 (CISP2).

<table>
<thead>
<tr>
<th></th>
<th>PPO (W)</th>
<th>MPO (W)</th>
<th>WD (J)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CISP 1</td>
<td>1174 ± 132</td>
<td>1104 ± 129</td>
<td>3312 ± 387</td>
</tr>
<tr>
<td>CISP 2</td>
<td>1187 ± 145</td>
<td>1100 ± 112</td>
<td>3300 ± 336</td>
</tr>
<tr>
<td>Difference</td>
<td>-13 ± 13</td>
<td>4 ± 17</td>
<td>12 ± 51</td>
</tr>
<tr>
<td>TEM (CV 95 %)</td>
<td>2.9 (2.0 - 5.0)</td>
<td>4.2 (2.9 - 7.4)</td>
<td>4.2 (3.1 - 7)</td>
</tr>
<tr>
<td>ICC (r value)</td>
<td>0.96 (0.85 - 0.99)</td>
<td>0.90 (0.66 - 0.97)</td>
<td>0.90 (0.72 - 0.96)</td>
</tr>
</tbody>
</table>

N.B. TEM: typical error of the measure. CV: coefficient of variation, at a 95 % confidence interval (CI). ICC: intra-class correlation.
4.3.3. Differences in CISP Phases

With respect to grouped sprints for PPO, phases 1 and 4 showed the smallest TEM (3.4 ± 10.9 %, 3.3 ± 14.1 % respectively), whereas phases 2 and 3 showed higher TEM, (3.5 ± 12.8 %, 3.9 ± 14.1 % respectively) (Table 4.2). This was also evident for MPO. However, MPO TEM was greater for each phase when compared to PPO except for phase 1 (Table 4.2). ICC for PPO between CISP1 and CISP2 remained above $r = 0.92$ throughout each phase, however this was not the case in phase 2 and phase 3 for MPO, where ICC values dropped to below 0.90 ($r = 0.85, 0.85$, respectively).
Table 4.2. Mean (± SD) peak power output (PPO) (W), mean three second power output (MPO) (W), Work done (WD) (J), TEM and ICC scores for each phase (Phase 1 = sprints blocks 1 - 5, Phase 2 = 6 - 10, Phase 3 = 11 - 15 and Phase 4 = 16 - 20), CISP trial 1 and CISP trial 2.

<table>
<thead>
<tr>
<th></th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Phase 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PPO (W)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CISP 1</td>
<td>1203 ± 111</td>
<td>1178 ± 129</td>
<td>1160 ± 153</td>
<td>1152 ± 140</td>
</tr>
<tr>
<td>CISP 2</td>
<td>1221 ± 138</td>
<td>1189 ± 148</td>
<td>1166 ± 147</td>
<td>1170 ± 157</td>
</tr>
<tr>
<td>TEM (CV 95 %)</td>
<td>3.4 (2.3 - 6.0)</td>
<td>3.5 (2.4 - 6.2)</td>
<td>3.9 (2.7 - 6.9)</td>
<td>3.3 (2.3 - 5.8)</td>
</tr>
<tr>
<td>ICC (r value)</td>
<td>0.92 (0.73-0.98)</td>
<td>0.94 (0.78-0.98)</td>
<td>0.94(0.78-0.98)</td>
<td>0.95 (0.84-0.99)</td>
</tr>
<tr>
<td><strong>MPO (W)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CISP 1</td>
<td>1134 ± 105</td>
<td>1104 ± 133</td>
<td>1093 ± 151</td>
<td>1086 ± 135</td>
</tr>
<tr>
<td>CISP 2</td>
<td>1138 ± 99</td>
<td>1101 ± 115</td>
<td>1081 ± 119</td>
<td>1080 ± 124</td>
</tr>
<tr>
<td>TEM (CV 95 %)</td>
<td>3.0 (2.1 - 5.4)</td>
<td>5.1 (3.6 - 9.2)</td>
<td>5.7 (3.9 - 10.2)</td>
<td>4.3 (3.0 - 7.6)</td>
</tr>
<tr>
<td>ICC (r value)</td>
<td>0.92 (0.73-0.98)</td>
<td>0.85(0.54-0.96)</td>
<td>0.85(0.53-0.96)</td>
<td>0.91 (0.7-0.97)</td>
</tr>
<tr>
<td><strong>WD (J)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CISP 1</td>
<td>3402 ± 315</td>
<td>3312 ± 399</td>
<td>3279 ± 453</td>
<td>3258 ± 405</td>
</tr>
<tr>
<td>CISP 2</td>
<td>3416 ± 297</td>
<td>3303 ± 345</td>
<td>3243 ± 357</td>
<td>3240 ± 372</td>
</tr>
<tr>
<td>TEM (CV 95 %)</td>
<td>2.9 (2.2 - 4.7)</td>
<td>5.1 (3.8 - 8.3)</td>
<td>5.8 (4.3 - 9.4)</td>
<td>4.2 (3.1 - 6.8)</td>
</tr>
<tr>
<td>ICC (r value)</td>
<td>0.92 (0.79-0.97)</td>
<td>0.85 (0.61-0.95)</td>
<td>0.84 (0.59-0.94)</td>
<td>0.91 (0.76-0.97)</td>
</tr>
</tbody>
</table>

TEM: typical error of the measure. CV: coefficient of variation, at a 95 % confidence interval (CI). ICC: intra-class correlation.

4.3.4. Agreement and Equality

PPO showed an even distribution within the 95 % limits of agreement. These data showed systematic bias with wide limits of agreement (Bias ± 1.96 SD = -14 ± 147 W; LOA: -162 – 133 W) (Figure 4.1, A). MPO showed greater variation between CISP1 and CISP2 as MPO increased (Figure 4.1, B). Systematic bias was lower for MPO compared to PPO.
(demonstrated in Figure 4.2), however the limits of agreement were still high (Bias ± 1.96 SD = 4.6 ± 158.5W; LOA: -153.9 – 163.1 W).

**Figure 4.1.** Limits of Agreement plots for peak power output (PPO) (W) and mean power output (MPO) (W) (A and B respectively). The differences between CISP’s are plotted against the individual means for the two tests. Bias lines and 95 % limits of agreement are also presented on the plot.
**Figure 4.2.** Relationship in peak power output (PPO) for CISP1 vs. CISP2 (A) and relationship in mean three second power output (MPO) for sprints in CISP1 vs. CISP2 (B). Line of Equality is shown by the black line.
4.4. DISCUSSION

The purpose of this study was to determine the reliability of the cycling intermittent sprint protocol (CISP). The main finding of this study was that the CISP can be used as a reliable test of high intensity intermittent exercise in games players. Additionally, one familiarisation trial is sufficient to minimise any potential for a learning effect on PPO and MPO when employing the CISP.

The reliability of the CISP was assessed from two trials separated by three days. The typical error of measurement scores across all 20 sprints for PPO and MPO for the two trials of the CISP [2.9 % (95 % CI 2.0 - 5.0) and 4.2 % (95 % CI 2.9 - 7.4) respectively] suggested a low level of within subject variability. The same conclusions can be drawn for PPO when sprints were grouped into 4 phases. For MPO the variation between each typical error of measurement for each phase suggested greater within subject variability in the middle two phases.

No other studies to date have examined the reliability of the CISP. When compared to running based protocols examining PPO and MPO in repeated sprint activity on non-motorised treadmills, the TEM values (expressed as a CV) reported in this study are lower than those observed in other studies. Oliver et al (2007) reported a typical error of 7.9 % (95 % CI 5.8 – 14.4) for peak power and 5.9 % (95 % CI 4.3 - 10.2) for mean power in the soccer specific intermittent sprint test (SSIET) when performed with youth soccer players. Such discrepancy between TEM values for PPO and MPO in our study and that of Oliver et al (2007) may be explained by the mode of assessment of repeated sprint ability as the reliability of power output from non-motorised treadmill running is lower (Oliver et al, 2007; Tong et al, 2001). In addition, compared with the random error reported in repeated trials for cycle ergometers (Paton and Hopkins, 2001) the values reported for mean and peak power in this study compared favourably. Therefore, where PPO and MPO are the important dependent variables chosen to assess worthwhile interventional change, protocols employing cycle ergometer exercise may enable more accurate assessment of performance. However, researchers must also consider that, for team-sport athletes whose primary means of locomotion is running, cycle exercise lacks specificity and so contributes to greater variability and reduced sensitivity when monitoring interventional change. Thus, careful consideration must be given to the mode of exercise used.
A commonly employed cycling test to assess repeated sprint ability in games players is the 5 x 6 second maximal sprint test (McGawley & Bishop, 2006; Bishop et al, 2004). McGawley & Bishop (2006) reported typical error scores for PPO in the first sprint between trial 1 and 2 of 25 W (95% CI: 17 – 48 W). In the current study the typical error in the first sprint between trial 1 and 2 was approximately 48 W (33 – 84 W). Expressed as a percentage of maximal PPO, TEM is similar in both studies (McGawley sprint 1: ~ 2.8 %, present study sprint 1: 2.5 %) suggesting the CISP compares favourably. Similarly, ICC values reported in our study are similar with those observed in other studies of intermittent sprint exercise involving treadmill running and overland sprinting (Gabbett, 2010; Laursen et al, 2007; Lemmink et al, 2004).

Limits of agreement were smaller for PPO than MPO suggesting that this is the more reliable measured variable. PPO showed an even distribution across individual sprints throughout the CISPs which coincides with the low TEM scores. Conversely, MPO was more varied, with this variation increasing as power output increased. This may be explained through individual subject differences and the diversity of muscle fibre type found within team games players causing differences in participant fatigability (Brutsaert and Parra, 2006). Bland and Altman (1986) suggest that unless the data is clinically important, for example the variability between measures does not affect participant prescription, two measures can be used interchangeably providing that the differences lie within the limits of agreement, even if these limits are wide. This assumption can be applied to the present study, with previous power based studies reporting SD of up to ± 147 W or more (Castle et al, 2006), which is similar to the CISP reliability data. Previous research (Tong et al, 2001; Nevill and Atkinson, 1997) states this to be acceptable range and so it may be proposed large limits can still allow good methodological reliability. Nevertheless, in future, researchers should use their own knowledge of the protocol and any interventions to interpret data accurately.

The present study showed greater within subject variability for MPO in phases 2 and 3. Laursen et al (2007) suggest greater variability in time to exhaustion tests may be related to participant boredom or lack of motivation. Although not a time to exhaustion test, the CISP’s duration and frequent need for participants to perform maximal efforts make it conceivable that such variables contributed to the greater variability found in phase 2 and 3 for MPO, despite consistent verbal encouragement throughout. The fixed paced portion of the CISP does not allow self-selection of exercise intensity as participants are required to maintain
predetermined recovery intensity. However, during the 5 second sprint, although instructed to be all out, participants may have self-paced. Consequently, participants may be adopting a pacing strategy to allow completion of the protocol thus reflecting greater variability in MPO in the middle portion of the CISP, particularly as research shows the occurrence of pacing in anticipation of the number of sprints within a trial (Billaut et al, 2011).

A practice or learning effect is defined as any systematic change in the performance scores during the performance of a novel exercise task, distinct from any experimental intervention (Mendez-Villanueva et al, 2008; Watt et al, 2002). In a meta-analysis of 30 tests of power, Hopkins et al (2001) identified a practice effect between first and second experimental trials and recommended at least one practice trial should precede formal testing. In previous studies using the CISP, only one familiarisation of five sprints prior to main trials is involved. An important finding of the current study was that no difference ($P > 0.05$) existed between the five-sprint familiarisation and the first five sprints in either CISP trial 1 or CISP trial 2. Although the large SD values may have prevented detection of any difference, previous investigations examining repeated sprint ability with the CISP have reported similar SD values and identified significant differences between trials (Castle et al, 2006; Castle et al, 2011). Similarly, when percentage change is considered, peak power output declined by 0.69 % in CISP trial 1 (95 % CI: -2.05 - 0.65 %) and increased in trial 2 (95 % CI: -0.35 - 2.1 %). There was a decline of 0.96 % (-2.57 - 0.64 %) for mean power output in CISP trial 1 and an increase of 0.36 % (-1.09 - 1.81 %) in CISP trial 2. This lack of substantial change between trials indicates minimal learning effect between practice and subsequent trials (Hopkins et al, 2001). Therefore it is plausible to suggest that one familiarisation trial consisting of 5 sprints is sufficient to obtain a reliable measure of intermittent sprint exercise performance. This finding contradicts other investigations examining reliability of intermittent high intensity sprint exercise. Capriotti et al (1999) reported two familiarisation sessions identical to the tests were required for satisfactory reliability when participants unfamiliar with multiple sprint exercise performed 10 x 7 second sprints with 30 second recovery. With a homogenous sample of trained female soccer players, McGawley and Bishop (2006) reported one familiarisation trial was sufficient to obtain a reliable measure of repeated sprint ability, but recommended two as reproducibility of measures improved significantly with a second familiarisation session. Hence, completion of three full trials of the current protocol could have improved reliability for CISP trial 2 compared with trial 3. In addition, if a complete 20-sprint familiarisation trial of the CISP was completed in a rested state, further insight into the
efficacy of one practice trial would have been gained. However, the suggestion of extending
the familiarisation process for the CISP to two sessions is not recommended due to the
prolonged, repetitive nature of the protocol and the possible negative effect on subject
motivation if too many sessions are required (Hopkins, 2000). In addition, the current
investigation analysis of PPO and MPO for sprints 1 – 5 in the familiarisation vs. CISP1
compared to familiarisation vs. CISP2 revealed no differences in TEM, ICC and 95 %
confidence limits suggesting no improvement in reproducibility of measures when further
trials were included. Consequently it can be suggested, for trained games players’ familiar
with intermittent sprint exercise, one familiarisation session comprising five sprints is
sufficient to obtain reliable measures of PPO and MPO in the CISP.

The typical error of measurement expressed as a CV may be used to estimate sample size for
future studies using the CISP when a smallest worthwhile change is known using the formula
\[ N = \frac{8s^2}{d^2} \] (Hopkins, 2000). Previously, Castle et al (2006) have stated the application of pre-
cooling treatment to intermittent sprint exercise in the heat using the CISP results in a 4 %
significant improvement in power output. Accordingly, in the current study to detect a 4 %
change a sample size of four participants may be sufficient when PPO is the variable of
concern. When MPO is considered the sample size required to detect a worthwhile change
increases to nine assuming similar variability amongst the participants recruited.

4.4.1. Conclusion

This study was the first to examine the reliability of the CISP, a test previously utilised to
reproduce activity patterns in games play. The protocol is unique in terms of cycling based
tests of intermittent sprint exercise due to its duration. Consequently, it permits the
examination of the effect of differing interventions and training both performance in
intermittent sprint exercise over durations typical of one half of a team game while also
permitting investigation of physiological responses to such exercises. Results demonstrate
that the CISP as a reliable measure, but typical error of measurement and ICC scores show
that interpretation of PPO data rather than MPO data is more reliable for assessing the
reliability of the protocol. A familiarisation period comprising of a quarter of the CISP is a
sufficient length to remove any learning effect influence on the main protocol.
Pre-cooling the legs using ice packs reduces peripheral skin and muscle temperatures without concomitant reductions in core temperature
CHAPTER V: PRE-COOlING THE LEGS USING ICE PACKS REDUCEs PERIPHERAL SKIN AND MUSCLE TEMPERATURES WITHOUT CONCOMITANT REDUCTIONS IN CORE TEMPERATURE

5.1. INTRODUCTION

Pre-cooling is one method employed by athletes in an attempt to alleviate the debilitating effects of increased heat gain on sporting performance. Previously, a limiting core temperature has been proposed, with cessation of exercise attributed to high internal body temperatures (Gonzalez-Alonso et al, 1999). However, it is now thought that this may only be one of many factors of fatigue (Noakes, 2012), particularly given that exercise can be maintained beyond proposed core temperature thresholds (Ely et al, 2009). Ultimately, the purpose of pre-cooling is to lower body temperature to create a heat sink (Quod et al, 2006) so that individuals will be able to exercise for longer or at higher intensities before critical levels of exertional heat stress cause cessation of exercise (Neilsen et al, 1993). Following 20 minutes of thigh only cooling compared to a no cooling control, Castle et al (2006) reported 4% improvements in peak power output during repeated sprint-cycling when exercising in a hot humid environment. Chapter IV showed these improvements to be meaningful given the observed reliability of the protocol, and it was suggested that cooling of the legs creates a larger heat sink through additional reductions in deep tissue temperature for the absorption of metabolic and environmental heat, consequently slowing the rate of physiological strain (Castle et al, 2006).

Despite the method of pre-cooling, reduced core temperatures are frequently reported, with subsequent improvements in time to exhaustion and power output profiles (Lee and Haymes, 1995; Booth et al, 1997; Marsh and Sleivert, 1999; Castle et al, 2006). Conversely, improved power output and time trial results have been reported following lower skin temperatures rather than reduced core temperatures (Kay et al, 1999; Schlader et al, 2011a). Schlader et al (2011a) observed improvements in mean power output despite no change in core temperatures and suggested this was due to higher selected initial exercise intensities. Furthermore, Kay et al (1999) attributed improved time trial performance to reduced thermal and cardiovascular strain and improved body heat storage following manipulation of skin temperature while keeping core temperature constant.
The passive thermal system of the body incorporates the conductive and convective processes of muscle tissue and blood flow (Fiala et al, 1999; Ferreira and Yanagihara, 2009). Transition of thermal energy from one mass to another occurs when there are differences in temperature between objects and their surroundings, with heat exchange attempting to reach a state of temperature equilibrium. During cooling, reductions in peripheral (skin) temperature are suggested to increase the thermal gradient of the core to the shell (Kay et al, 1999). This increased gradient will therefore cause conductive and convective transfer of heat away from the warmer core reducing core temperature in an attempt to warm the periphery, potentially delaying the onset of heat gain.

The growing interest in pre-cooling to combat the negative effects of exertional heat stress is prevalent in the literature yet the durations used within the applied world vary. Knowledge regarding the durational responses of thermoregulatory variables would be useful.

Therefore, the purpose of this study was to determine the time course of heat exchange between the peripheral shell, local muscle tissue and whole body core compartments during leg cooling.
5.2. METHODOLOGY

5.2.1. Participants

Six participants, all male (mean ± SD; age: 23 ± 2 years, height: 176 ± 2 cm, body weight: 77.5 ± 5.9 kg, leg circumference: 54.3 ± 3.1 cm, sum of four skinfolds: 47.5 ± 10 mm, body fat percentage: 18.96 ± 2.51 %) participated in this study, which was approved by the Institutional Ethics Committee.

5.2.2. Experimental Design

All participants were free from any form of muscular or joint injury and abstained from any exhaustive activity within the 24 hours prior to any testing. During their first visit to the laboratory, participants had all preliminary measurements taken as described in chapter III (section 3.3.1). During the second and third visits, participants completed a pre-cooling period as described in chapter III (section 3.5) for 25 minutes and a no cooling control trial, in a randomised order. Ice packs had a surface temperature of -12.1 ± 1.3 °C on removal from a -20 °C freezer.

5.2.3. Physiological Measures and Experimental Procedures

On arrival to the laboratory, nude body mass (kg) was recorded prior to any preparation and core temperature (Tre) and skin (Tsk) temperature of the vastus lateralis were monitored every 2 minutes throughout the protocol. The skin thermistor was attached to the vastus lateralis muscle rather than the rectus femoris due to vastus lateralis muscle being the chosen site for muscle temperature measurement. Thermal sensation (TS) of the leg was also recorded every two minutes throughout the protocol.
5.2.3.1. Twenty Five Minute Cooling Period

Once in a comfortable supine position and stated that they were comfortable, each participant had a measurement of resting muscle temperature (Tmu) twice to ensure accuracy of the reading of the muscle temperature probe, taken at a 30 second interval through the same 18 gauge needle. The skin thermistor was then positioned on the relevant site and a resting Tskin value was recorded, in addition to resting Tre and TS. In visit 2 the initial temperature of the ice packs immediately after removal from the freezer was recorded before being positioned, leaving enough room between two packs for Tmu to be recorded safely and accurately, at which point the cooling period commenced. All measures were recorded every two minutes for 25 minutes, other than Tmu which was taken every two minutes from four minutes into the protocol, as pilot work showed this to be more comfortable for the participant once the sensation of having ice packs on the legs had become familiar.

5.2.4. Statistical Analysis

Data for Tsk, Tmu, Tre and TS were analysed using two-way repeated measures analysis of variance (condition vs. time). Change in temperature for Tpack, Tsk, Tmu, Tre and TS during the cooling condition was analysed using one-way analysis of variance. Post hoc analysis was conducted as described in chapter III (section 3.7). The strength of relationship between Tpack vs. Tskin and Tmu, Tsk vs. Tmu and TS and Tmu vs. Tre and TS were determined using Pearson Product Momentum Correlation. All data was reported with means and standard deviations. \( P < 0.05 \) was the level at which significance was accepted.
5.3. RESULTS

25 minutes of leg cooling significantly reduced muscle temperature (Tmu), skin temperature (Tsk) and thermal sensation (TS) of the legs when compared to the control \( (P < 0.01) \). There were no significant changes in core temperature (Tre) between cooling and the control \( (P = 0.494) \).

5.3.1. Rate of Change Pre to Post Cooling

Table 5.1. displays pre and post cooling values for ice pack, skin, muscle and core temperature and perceptive thermal sensation of the legs during 25 minutes of isolated leg cooling. Rate of change of each variable has been calculated. From pre to post cooling, Tsk observed the greatest decline in temperature of all variables, reducing by \( 24.41 \pm 2.3 \degree C \) at a rate of \( 0.98 \pm 0.09 \degree C . \min ^{-1} \) \( (P < 0.01) \), whereas Tmu significantly decreased by \( 15.4 \pm 0.8 \degree C \) at a rate of \( 0.6 \pm 0.0 \degree C . \min ^{-1} \) \( (P < 0.01) \). Tpack significantly increased by \( 6.2 \pm 2.50 \degree C \) at a rate of \( 0.25 \pm 0.10 \degree C . \min ^{-1} \) \( (P < 0.01) \). Tre rose by \( 0.1 \pm 0.1 \degree C \) throughout the leg cooling, although this was not statistically significant \( (P > 0.05) \).

Table 5.1. Mean (± SD) temperature and rate of change for packs (Tpack), skin surface of the thigh (Tskin), vastus lateralis muscle (Tmu) and core temperature (Tre), as well as thermal sensation (TS) of the legs following 25 minutes of cooling.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Temperature (°C)</th>
<th>Rate of change (°C.min(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre cooling</td>
<td>Post cooling</td>
</tr>
<tr>
<td>Tpack</td>
<td>-12.1 ± 1.3</td>
<td>-5.9 ± 3.8 *</td>
</tr>
<tr>
<td>Tskin</td>
<td>31.70 ± 1.03</td>
<td>7.30 ± 3.33 *</td>
</tr>
<tr>
<td>Tmu</td>
<td>32.6 ± 2.2</td>
<td>17.2 ± 3.1 *</td>
</tr>
<tr>
<td>Tre</td>
<td>37.3 ± 0.3</td>
<td>37.4 ± 0.4</td>
</tr>
<tr>
<td>TS</td>
<td>4.3 ± 0.6</td>
<td>3.2 ± 0.5 *</td>
</tr>
</tbody>
</table>

*denotes significant change from pre-cooling values, at the \( P < 0.05 \) level.
5.3.2. Thermal Sensation

TS reduced from resting values by 1.1 ± 0.1 at the end of leg cooling ($P < 0.05$), however larger reductions from resting values occurred during the first 2 minutes of cooling upon application of ice packs (Figure 5.1). TS decreased by 3.3 ± 0.0 ($P < 0.01$) to its lowest point from rest to two minutes into cooling (4.3 ± 0.6 to 1.0 ± 0.6). From two minutes, TS steadily increased and was significantly greater than the lowest value after a further 10 minutes of cooling ($P < 0.05$). TS from the lowest point after two minutes to the end of cooling increased by 2.2 ± 0.1, at a rate of 0.095 ± 0.005 per minute ($P < 0.01$) (Figure 5.1) nevertheless was still significantly lower than at rest ($P < 0.01$).

![Figure 5.1. Mean (± SD) changes in perceptive thermal sensation of the legs from rest (0 minutes) throughout 25 minutes of leg cooling.](image)

5.3.3. Thermoregulatory responses throughout cooling

A 76 % reduction in Tsk occurred within the first two minutes of cooling ($P < 0.01$), at a rate of 9.30 ± 0.93 °C.min$^{-1}$. Further reductions, at a rate of 0.56 ± 0.03 °C.min$^{-1}$ ($P < 0.05$) were observed up until 12 minutes, yet after this point Tsk plateaued with no further reductions apparent ($P > 0.05$). Tsk was at its lowest after 18 minutes, however this was not significantly lower than after 12 minutes ($P > 0.05$) (Figure 5.2).
Reductions in Tmu were slower than that of Tsk with 53% of reductions occurring within the first 4 minutes of cooling ($P < 0.01$), at a rate of $2.05 \pm 0.7 \, ^\circ\text{C}.\text{min}^{-1}$. A further 46% reduction was seen up until 14 minutes of cooling ($P < 0.05$) at a rate of $0.71 \pm 0.28 \, ^\circ\text{C}.\text{min}^{-1}$ with no further differences observed hereafter ($P > 0.05$). Rate of change in Tmu from minute 14 until end of cooling was $0.009 \pm 0.08 \, ^\circ\text{C}.\text{min}^{-1}$ (Figure 5.2)

Tpack increased in temperature by $6.15 \, ^\circ\text{C}$ at a rate of $0.24 \pm 0.1 \, ^\circ\text{C}.\text{min}^{-1}$ throughout 25 minutes of cooling ($P < 0.01$), however a significant increase in temperature from the start of cooling was only apparent after 18 minutes ($P < 0.05$) (Figure 5.2).

**Figure 5.2.** Mean (± SD) temperature change for pack temperature (Tpack), skin temperature (Tsk), muscle temperature (Tmu) and core temperature (Tre) during 25 minutes of leg cooling. Error bars for Tre were excluded for visual clarity. N.B. Tmu was taken at rest and then at 4 minutes, the 2 minute point is a moving average.
5.3.4. The relationship between sites of temperature measurement

A strong negative correlation was observed between increases in Tpack and decreases in Tsk throughout cooling ($r = 0.789$, $P < 0.01$, Figure 5.3 A). The decrease in Tsk was strongly correlated with decreases in Tmu ($r = 0.974$, $P < 0.01$, Figure 5.3 B). A negative correlation was observed between reductions in Tmu and Tpack ($r = 0.971$, high effect, $P < 0.01$, Figure 5.3 C). A strong correlation was observed between increases in TS and decreases in both Tsk and Tmu ($r = 0.879$, $P < 0.01$, Figure 5.3 D, and $r = 0.932$, $P < 0.01$ respectively) throughout 25 minutes of leg cooling.
Figure 5.3. Correlation analysis describing the path of heat transfer from the periphery to the body core as a result of cooling. Analysis for pack temperature vs. skin temperature (A), skin temperature vs. muscle temperature (B), pack temperature vs. muscle temperature (C) and skin temperature vs. thermal sensation (D). R values were significant at the $P < 0.01$ level.
5.3.5. Individual Responses to Cooling

Pre and post cooling Tmu ranged by 6.4 °C and 7.6 °C respectively, while the temperature-duration response varied greatly among participants (Table 5.2). Tmu decreased by 15.4 ± 3.7 °C although participant 1 only reduced by 8.9 °C whereas participants 4 and 6 reduced by ~18 °C. Over 90% of this temperature change occurred after 16 minutes in all participants other than participant 3. In two participants, Tmu increased after 16 minutes to the end of cooling.

Table 5.2. Participant demographics and muscle temperature (Tmu) data at rest and post cooling, with calculated rate of change during 25 minutes of cooling and percentage change in muscle temperature after 16 minutes of cooling

<table>
<thead>
<tr>
<th>Participant</th>
<th>Body Mass (kg)</th>
<th>Thigh Circumference (cm)</th>
<th>Sum of Skinfolds</th>
<th>Tmu at rest (°C)</th>
<th>Tmu post cooling (°C)</th>
<th>Rate of change in Tmu (°C.min⁻¹)</th>
<th>Change in Tmu after 16 minutes (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>68.8</td>
<td>52.0</td>
<td>39.1</td>
<td>29.4</td>
<td>20.5</td>
<td>0.36</td>
<td>98.88</td>
</tr>
<tr>
<td>2</td>
<td>74.4</td>
<td>52.5</td>
<td>45.1</td>
<td>34.2</td>
<td>19.3</td>
<td>0.60</td>
<td>102.68</td>
</tr>
<tr>
<td>3</td>
<td>86.0</td>
<td>60.5</td>
<td>63.7</td>
<td>32.0</td>
<td>17.6</td>
<td>0.58</td>
<td>75.69</td>
</tr>
<tr>
<td>4</td>
<td>78.6</td>
<td>54.0</td>
<td>52.4</td>
<td>31.3</td>
<td>12.9</td>
<td>0.74</td>
<td>101.09</td>
</tr>
<tr>
<td>5</td>
<td>81.0</td>
<td>54.0</td>
<td>36.0</td>
<td>35.8</td>
<td>18.8</td>
<td>0.68</td>
<td>89.91</td>
</tr>
<tr>
<td>6</td>
<td>76.8</td>
<td>53.0</td>
<td>48.4</td>
<td>32.7</td>
<td>13.9</td>
<td>0.75</td>
<td>98.94</td>
</tr>
</tbody>
</table>

N.B. a change in Tmu of > 100% after 16 minutes displays an increase in temperature between 16 minutes and end of cooling.
Figure 5.4. Individual data displaying the muscle temperature-duration response during 25 minutes of leg cooling. Error bars included for ‘average’ only to aid in visual clarity.

Figure 5.5. Individual data displaying the core temperature-duration response during 25 minutes of leg cooling. Error bars included for ‘average’ only to aid in visual clarity.
Tre did not change from resting values in two participants throughout cooling. Reductions in Tre of up to 0.2 °C were observed in three participants within the first two minutes however Tre remained constant hereafter (Figure 5.5). Tre increased by 0.4 °C at a rate of 0.016 °C.min⁻¹ for one participant only.
5.4. DISCUSSION

Many studies pertaining to the transition of thermal energy within the body are associated with changes as a result of exercise and the development of heat strain. Within pre-cooling literature, studies are uncommon when discussing thermal gradients and transition of thermal energy, with skin and core temperature the most frequently reported thermoregulatory variables. Furthermore, there is some difficulty in determining relationships between thermal stressors and all of the thermoregulatory responses that may contribute to exercise regulation (Schlader et al, 2011b). Therefore the aim of this study was to determine the time course of heat exchange between the periphery and the core, providing evidence for the optimal duration of cooling needed for application of this intervention in the sporting environment. It was hypothesised that cooling using ice packs would induce reductions in skin and muscle (peripheral) temperature, consequently causing core temperature to decline. This study demonstrated that local leg cooling using ice packs causes significant reductions in peripheral temperatures without concomitant reductions in core temperature. Changes in pack, skin and muscle temperature were all strongly correlated, yet after 18 minutes, there were no further changes in these variables despite continued cooling.

The second law of thermodynamics states that heat transfer only occurs from a hot body to a cold body to reach a state of equilibrium between masses (Serrin, 1979). One would therefore assume that the convective and conductive paths of heat transfer during a cooling bout would be from the warm core to the cooler shell due to heat loss of the periphery to the cold stressor. In turn this would reduce core temperature prior to exercise. However, in the present study core temperature was not reduced at any point during cooling. Reduced core temperatures have not always been reported as a consequence to cooling. Duffield et al (2011) saw no changes in core temperature following 20 minutes of mixed-method cooling. Additionally, Kay et al (1999) observed improvements in cycling time trial performance following reduced skin temperature without reductions in core temperature. In the present study, skin temperature significantly reduced during the first 12 minutes of cooling, with the majority of this reduction occurring during the first two minutes. Although correlation does not prove causation, the strong relationships between reductions in muscle temperature and reductions in skin temperature, suggest the transfer of heat from the deep tissues to the shell. Furthermore, the relationship between reductions in skin temperature and increases in pack temperature, suggest the transfer of heat away from the body, or in this case to the ice pack.
Pre-cooling is used to lower core and, excluding ice vest cooling, muscle temperature prior to exercise in hot environments in order to prolong or improve performance. Gonzalez-Alonso et al (1999) and Drust et al (2005) both concluded that elevated core and muscle temperatures are primary factors associated with fatigue. Booth et al (2004) suggest that fatigue and rises in muscle temperature occur concomitantly, and that central factors must govern fatigue, highlighting the benefits of a cooling ergogen. Castle et al (2006) reduced core and muscle temperatures through local leg cooling and saw improvements in peak power output and work done. Without changes in core temperature, as in the present study, it may be that the observed reductions in skin and muscle temperature following local cooling allow for greater metabolic and environmental heat load without affecting cardiovascular responses (Kay et al, 1999; Castle et al, 2006).

Kay et al (1999) state reductions in mean skin temperature of 4 °C allow for an increased ability of the body to store heat. In the present study, skin temperature of the thigh reduced by 24.4 °C. Given the factors used to calculate body heat content, large reductions such as this will account for reductions in total body heat content, despite no changes in other skin or core temperature measures. Reductions in skin temperature increase the thermal gradient of the core to the shell (Kay et al, 1999) creating the desired heat sink associated with the pre-cooling ergogen. Consequently, this may delay the rise of core temperature therefore prolonging exercise performance. Nevertheless, if achieving lower body heat content following a pre-cooling technique allows for increased heat storage and thus improved exercise performance (Kay et al, 1999), the data from the current study could suggest that thigh cooling for two minutes would induce performance improvements due to significant temperature reductions after this duration. Although differences in technique, it has been shown that cooling using an ice vest for five minute durations does not elicit the required benefits to performance (Duffield et al, 2003) despite reductions in mean skin temperature compared to no cooling. Data from this study therefore questions the validity of body heat content calculations following local changes in temperature lending support to Jay et al (2007) for a three-compartment thermometry model to improve estimation of body heat content. Furthermore, it demonstrates the accuracy required by the experimenter in the methodology of basic thermoregulatory measures, while drawing upon the necessary caution required when using derivative equations from these thermoregulatory measures, given the possible error in calculation or of the multiple variables involved.
Frequently, vasomotor responses are attributed to cooling (Lee and Haymes, 1995; Marsh and Sleviert, 1999; Hornery et al, 2005; Castle et al, 2006; Vaile et al, 2008; Luomala et al, 2012). It is suggested that decreased peripheral blood flow enhances central blood volume and blood delivery to the working muscles (Lee and Haymes, 1995; Marsh and Sleviert, 1999; Vaile et al, 2008). Initial increases in core temperature have been reported during a cooling bout due to vasoconstriction of the periphery re-directing warmer blood to the core (Schmidt and Bruck, 1981; Hessemer et al, 1984; Lee and Haymes, 1995). Notwithstanding, it has been suggested that reductions in core temperature occur post cooling (Booth et al, 1997; Cotter et al, 2001). Webb (1986) and Romett (1988) suggest that it is during a warming phase that the majority of core temperature decrease occurs. This is due to vasodilation of the periphery on removal of the stressor thus allowing cold blood back to the core. This cold blood causes reductions in core temperature consequently creating the desired heat sink. Booth et al (1997) reported improved running performance despite rapid rewarming of the skin during early stages of exercise (0-15mins) following whole body cooling due to an “afterdrop” in core temperature and so enhanced body heat storage. It is likely that an aggressive cooling technique such as the use of ice packs induced a sympathetic stimulus of rapid vasoconstriction of the periphery (Marino and Booth, 1998). This could have prevented redistribution of heat to or from the core, hence the lack of change in core temperature. Castle et al (2006) reported reductions in core temperature following 20 minutes of thigh cooling using ice packs with further reductions occurring during the warm up. The lack of a warm up or exercise component in the present study meant that temperature variables were not measured post cooling.

An interesting observation is that skin and muscle temperature both reached a plateau after 12 and 14 minute of cooling, respectively. To the authors’ knowledge, there have been no precooling studies specifically looking at the time course of skin and muscle temperature change on such an acute level. Janwantanakul (2009) did observe a plateau in surface temperature at 8 minutes, however the study was assessing the differences in contact area and quantity of ice on surface temperature. In studies that have also measured muscle temperature, variations in temperature reductions following cooling is evident (Sleivert et al, 2001; Booth et al, 2004; Castle et al, 2006). However, the techniques and durations of cooling differ between these studies and it is not known at what point muscle temperature was at its lowest as only pre and post measures are taken. This study therefore suggests that muscle temperature measurement can be an additional important measure given that skin and
muscle temperatures appear to plateau at some stage during one bout of cooling. With this the case, it can be questioned as to why excessive cooling durations are used, with the possibility of no further thermoregulatory benefits. Furthermore, extreme muscle temperature reductions, and skin temperatures of 12.5 °C have been shown to decrease conduction velocity (McMeeken et al, 1984; Ce et al, 2012) and EMG amplitude (Petrofsky and Lind, 1980) consequently effecting peak power output and work done (Crowley et al, 1991). Sargeant (1987) reported a 3 % decline in peak power output per 1 °C fall in muscle temperature compared to a control in 20 second sprint cycling bouts. Therefore, monitoring muscle temperature during cooling will allow for the control of the pre-cooling response ensuring negative responses to pre-cooling are not observed during intermittent sprint performance. In many cooling studies, one continuous bout of cooling is used, yet consideration should be made for re-application of the technique to maintain a constant temperature supply. In the present study, pack temperature began to rise immediately on application and thus the magnitude of cooling would not have been constant. It may be that plateaus in skin and muscle temperature reduction occur concomitantly with reduced cooling effectiveness of the pack due to a reduced temperature gradient over time. Future leg cooling studies using ice packs should investigate the effect of cooling following re-application of the cold stressor to determine the thermal responses of the skin and muscle and their effect on consequent intermittent sprint performance. Additionally, Booth et al (2004) reported a common asymptote following rises in muscle temperature. It may be that there is a range in the thermal capacity of the muscle and it is the optimal manipulation of this temperature range that should be targeted during pre-cooling.

Responses to cooling are reported as means and standard deviations in the literature with durations frequently reported as a generic prescription. However, individual responses to cooling are apparent in the present study. Despite variations in the reductions, muscle temperature reduced in all participants with a temperature plateau occurring near to the same time point. However, core temperature response was different among participants, with thigh cooling providing small reductions in core temperature in three participants, a large increase in one participant while remaining unchanged in two participants. These results have practical implications given that the desired outcome of cooling is to achieve a heat sink through reduced skin, muscle or core temperatures. Yet individual variation shows that the magnitude of heat sink response may not be the same across a population. Prescribing pre-cooling via temperature reductions may not be feasible in the field due to differences in
individual temperature-duration response and the limited time before competition. Results from this study therefore demonstrate the necessity of an accurate duration that can be used as a generic prescription for optimal subsequent performance.

5.4.1. Conclusion

Isolated leg cooling causes reductions in skin and muscle temperature without concomitant reductions in core temperature. Reductions were apparent until 14 minutes at which point a plateau in temperature change was observed. It is suggested that this is due to rapid vasomotor responses to the aggressive cooling technique used. However, this method of precooling may allow for increased metabolic and environmental heat load due to increased body heat content. This has practical application for use of leg cooling in the field demonstrating that excessive cooling durations appear unnecessary, while further work is required assessing the effect of a constant temperature supply during thigh cooling on thermoregulatory responses.
CHAPTER SIX

Study Three

A duration-dependent response to isolated leg cooling exists for intermittent-sprint exercise in hot, humid conditions
CHAPTER VI: A DURATION-DEPENDENT RESPONSE TO ISOLATED LEG COOLING EXISTS FOR INTERMITTENT-SPRINT EXERCISE IN HOT, HUMID CONDITIONS

6.1. INTRODUCTION

Hot humid conditions cause alterations in cardiovascular (Gonzalez-Alonso et al, 2008; Gonzalez-Alonso, 2012) and neuromuscular function (Hargreaves, 2008) and shifts in energy metabolism (Galloway and Maughan, 1997) which may affect thermoregulatory responses resulting in increased core temperatures (Drust et al, 2005). Prolonged exercise in the heat can result in numerous heat related illnesses, including heat cramps (Stofan et al, 2005), heat stroke or heat exhaustion (Noakes, 2003). Furthermore, reduced time to exhaustion (Gonzalez-Alonso et al, 1999) and slower time trial results (Tatterson et al, 2000) have also been reported from following exercise in a hot humid environment.

The demands of prolonged intermittent sprint exercise places the body under further cardiovascular and physiological strain. The debilitating effects of heat stress are exacerbated during intermittent sprint performance (Kranning and Gonzalez, 1991; Nevill et al, 1995). Increased cardiovascular strain (Morris et al, 2005) and elevations in core temperature (Drust et al, 2005) have been reported while slower sprint times (Morris et al, 2000) and declines in peak power output (Castle et al, 2006) have also been observed.

Within the last 10 years, pre-cooling techniques have been used to alleviate the problems of exertional heat stress associated with intermittent sprint exercise. Cooling decreased physiological strain (Cotter et al, 2001), reduced core temperature (Webster et al, 2005) and significantly improved peak power output (Castle et al, 2006) when carried out prior to exercise. Morris et al (2005) and Drust et al (2005) both state that excessive rises in muscle temperature impair power output and relate this associated rises in core temperatures and potential reduction in neuromuscular transmission to the active musculature. This notion is supported by Castle et al (2006) who concluded that cooling the thigh muscles maintained a lower muscle temperature and improved peak power output by ~ 4% compared to non-cooled control. Thigh cooling had the greatest ergogenic effect, better than water immersion or ice vest. This may have been due to reduced structural and functional alterations in muscle proteins normally caused by heat stress (Hargreaves and Febbraio, 1998).
Although improvements in performance and thermal comfort are frequently measured, the optimal duration of pre-cooling is still unclear. Cooling durations have varied from repeated bouts of short duration (2 x 5 minutes and 1 x 10 minute bout) (Duffield et al, 2003) to much longer continuous cooling periods (60 - 75 minutes) (Cheung and Robinson, 2004; Drust et al, 2000a), with many studies opting for an estimated duration, long enough for what the researchers deemed sufficient for ergogenic benefit, but short enough not to compromise thermal comfort. Between 10 and 45 minutes is the range most frequently used (Castle et al, 2006; Duffield and Marino, 2007), with shorter durations having little benefit. Duffield et al (2003) found that cooling for 5 minutes, albeit during rest periods, had no effect on work done, power output, heart rate, blood lactate concentration, core or mean skin temperature, perceived exertion, sweat loss, or ratings of fatigue. This might suggest that longer periods of cooling are necessary for ergogenic benefits to be evident. However, there are no criterion based cooling durations for optimal effect on physiological measures and performance in intermittent sprint performance in the heat. Minett et al (2012) investigated the effect of two durations (10 and 20 minutes) of mixed-method cooling on an intermittent sprint protocol, consisting of repeated maximal sprints interspersed with phases of self-paced recovery. Cooling for 20 minutes resulted in greater distances covered, attributed to larger reductions in body heat storage, despite no differences in core and skin temperature between cooling conditions.

To the authors’ knowledge, Minnett et al (2012) is the first study to measure a duration-dependant response to cooling. The lack of difference in thermoregulatory variables between conditions suggests the mechanisms behind the effects of cooling are difficult to ascertain. Therefore, the purpose of this study was to determine the optimal duration of a single bout of pre- cooling on repeated sprint ability in hot humid conditions.

It was hypothesised that cooling will improve intermittent sprint performance in hot, humid conditions. It was also hypothesised that the sprint performance response to cooling would occur in a duration-dependant manner.
6.2. METHODOLOGY

6.2.1. Participants

Twelve participants, all male (mean ± SD; age: 22 ± 2 years, height: 178.1 ± 3.9 cm, body weight: 76.7 ± 7.5 kg, sum of four skinfolds: 40.4 ± 3.9 mm, peak oxygen consumption: 44.94 ± 6.1 ml.kg\(^{-1}\).min\(^{-1}\)) participated in this study, which was approved by the Institutional Ethics Committee.

6.2.2. Experimental Design

The first two visits to the laboratory consisted of preliminary measures and a maximal ventilatory test, and a familiarisation as described in chapter III (sections 3.3.1 and 3.3.2). The subsequent five visits involved the participants completing the CISP in an environmental chamber (30 ± 2 °C and 50 ± 6 % relative humidity), aiming to simulate the hot and humid conditions. Each visit was preceded by a cooling period that was assigned in a random order to each participant. Cooling periods were of 10, 15, 20 and 25 minutes as well as a control session involving no cooling. Following cooling, participants completed the standardised warm up and CISP as described in chapter III (sections 3.3.2 and 3.3.3).

6.2.3. Physiological Measures and Experimental Procedures

Nude body mass was recorded prior to any preparation and then again after the exercise protocol was completed. Muscle temperature (Tmu) was taken three times during each visit; one before cooling, one immediately after cooling and one on completion of the CISP. Core (Tre) and skin temperature (Tsk), heart rate (HR), rating of perceived exertion (RPE) and thermal sensation (TS) were measured every five minutes during each cooling phase. All measures were recorded at one minute during the active recovery phase of each two minute bout of the CISP to account for any change in rises in temperature (Armstrong et al, 1994a).
6.2.4. Statistical Analysis

Data for peak power output (PPO), work done (WD) and core temperature (Tre) were analysed using two-way repeated measures analysis of variance (ANOVA) (condition vs. time). Muscle temperature (Tmu) was analysed using a one-way repeated measures ANOVA. Post hoc analysis was conducted as described in chapter III (section 3.7). All data was reported with means and standard deviations. $P < 0.05$ was the level at which significance was accepted. Where significance was not found, data will be reported as trends to demonstrate direction of interventions.
6.3. RESULTS

6.3.1. Performance Variables

6.3.1.1. Peak Power Output and Work Done

Participants completed the CISP following each cooling duration. Table 6.1. displays mean peak power output (PPO), work done (WD) and percentage change from the control for each pre-cooling condition during the CISP. Only the 15 minute cooling condition was significantly higher from the control for mean PPO during the CISP with an overall improvement of 3.75 % \((P < 0.01)\), in addition to being higher than all other conditions \((P < 0.01)\). The 25 minute condition was the lowest of all conditions for mean PPO and was significantly lower than the 15 and 20 minute condition \((P < 0.01)\).

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mean PPO (W)</th>
<th>Change in mean PPO vs. control (%)</th>
<th>Mean WD (kJ)</th>
<th>Change in mean WD vs. control (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>1006 ± 104 °C</td>
<td>-</td>
<td>4.0 ± 0.5 °C</td>
<td>-</td>
</tr>
<tr>
<td>10 minutes</td>
<td>1005 ± 101 °C</td>
<td>-0.064</td>
<td>4.0 ± 0.5 °C</td>
<td>1.06</td>
</tr>
<tr>
<td>15 minutes</td>
<td>1044 ± 105 °A</td>
<td>3.75 °A</td>
<td>4.3 ± 0.5 °A</td>
<td>6.7 °A</td>
</tr>
<tr>
<td>20 minutes</td>
<td>1013 ± 107 °C</td>
<td>0.74</td>
<td>4.1 ± 0.4 °AC</td>
<td>3.0 °A</td>
</tr>
<tr>
<td>25 minutes</td>
<td>998 ± 98 °CD</td>
<td>-0.782</td>
<td>4.0 ± 0.5 °C</td>
<td>-0.40</td>
</tr>
</tbody>
</table>

\(^{A}\) denotes significant difference from the control. \(^{C}\) denotes significant difference from 15 minute condition. \(^{D}\) denotes significant difference from the 20 minute condition. Significance was deemed at the \(P < 0.05\) level.

The 15 and 20 minute cooling conditions were the only conditions that were significantly higher than the control for mean WD, with the 15 minute cooling condition improving the greatest by 6.7 % \((P < 0.01)\) (Table 6.1).
The 15 minute cooling condition gave rise to a greater total WD across the CISP than all other conditions ($P < 0.01$) (Figure 6.1). Total WD in the CISP was $5.3 \pm 0.3 \text{ kJ}$ and $2.4 \pm 1.0 \text{ kJ}$ higher in the 15 and 20 minute conditions, respectively, when compared to the control ($P < 0.01$). The 25 minute cooling condition was the lowest of all conditions, although this was not significantly different to the control ($P > 0.05$).

![Figure 6.1](image)

**Figure 6.1.** Total (± SD) work done (kJ) at the end of 20 sprints in the cycling intermittent sprint protocol after a control, 10 minutes, 15 minutes, 20 minutes and 25 minutes of leg cooling. $^A$ denotes significant difference from the control. $^C$ denotes significant difference from 15 minute condition. Significance was accepted at the $P < 0.05$ level.

Figure 6.2 displays mean PPO for individual sprints throughout the CISP for each condition and the control. From sprint 8, the 15 minute cooling condition maintained the highest PPO for the remainder of the CISP when compared to the other conditions, however only sprints 10, 13, 15 and 20 (minute 20, 26, 30 and 40) were significantly higher than the control condition ($P < 0.01$). All conditions were lower than the control for the first four sprints however, none of these differences were statistically significant ($P > 0.05$). In all conditions PPO for the final sprint surpassed that achieved in the penultimate sprint. However, only the 15 and 20 minute conditions were significant ($P > 0.05$) (Table 6.2).
Figure 6.2. Mean peak power output throughout the cycling intermittent sprint protocol after a control, 10 minutes, 15 minutes, 20 minutes and 25 minutes of leg cooling using ice packs. \( c \) denotes significant improvement in the 15 minute condition compared to the control. Significance was deemed at the \( P < 0.05 \) level. Error bars have been included for the 15 minute condition only to aid in visual clarity.

Table 6.2. Peak power output (PPO) and difference between sprints 19 and 20 of the cycling intermittent sprint protocol for each of the four pre cooling conditions and the control. Values are reported as mean ± SD.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Penultimate Sprint (sprint 19) (W)</th>
<th>Final Sprint (sprint 20) (W)</th>
<th>Difference (W)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>980 ± 90</td>
<td>1024 ± 97</td>
<td>44 ± 7</td>
<td>0.08</td>
</tr>
<tr>
<td>10 minutes</td>
<td>1015 ± 127</td>
<td>1049 ± 169</td>
<td>33 ± 42</td>
<td>0.08</td>
</tr>
<tr>
<td>15 minutes</td>
<td>1052 ± 117</td>
<td>1111 ± 108</td>
<td>59 ± 9</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>20 minutes</td>
<td>985 ± 138</td>
<td>1052 ± 95</td>
<td>70 ± 48</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>25 minutes</td>
<td>995 ± 112</td>
<td>1009 ± 92</td>
<td>14 ± 20</td>
<td>0.5</td>
</tr>
</tbody>
</table>
Individual PPO plots (condition vs. control) show direction towards the 15 minute condition, to the left of the line of equality, with 75% of participants having improved PPO compared to the control (Figure 6.3). This was not the case for the 10 or 25 minute condition where the direction of PPO was to the right of the line of equality, with 33% of participants showing a decline in PPO compared to the control. The 20 minute condition was similar to that of the control condition, sitting along the line of equality with only 50% of participants improving.

![Graphs showing PPO plots](image)

**Figure 6.3.** Individual participant plots for mean peak power output vs. the control for the 10 (A), 15 (B), 20 (C) and 25 (D) minute cooling conditions, with lines of equality to display the direction of bias between conditions.

### 6.3.2. Physiological Variables

#### 6.3.2.1. Core Temperature

Table 6.3. displays core temperature (Tre) throughout each phase of the CISP, including change from rest to post warm up, for each condition of pre-cooling and the control condition. Tre in the control condition remained unchanged throughout the ‘no cooling’ phase and by the end of the warm up ($P > 0.05$). Tre for each of the cooling conditions
remained unchanged during the pre-cooling period ($P > 0.05$). Post warm up, there were no changes were observed in Tre for any cooling conditions ($P > 0.05$).

**Table 6.3.** Core temperature (Tre) at three time points (rest, post warm up and post cycling intermittent sprint protocol), calculated rate of change in core temperature for each of the pre-cooling conditions and control in hot humid conditions and time significant increase from the first sprint. Values are reported as mean ± SD.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Tre rest (°C)</th>
<th>Tre post warm up (°C)</th>
<th>Tre post CISP (°C)</th>
<th>Δ Tre during CISP (°C)</th>
<th>Rate of Tre increase (°C.sprint⁻¹)</th>
<th>Time to sig. increase in Tre from sprint 1 (mins)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>37.3 ± 0.2</td>
<td>37.4 ± 0.1</td>
<td>38.7 ±0.3</td>
<td>1.3 ± 0.2</td>
<td>0.07 ± 0.01</td>
<td>14</td>
</tr>
<tr>
<td>10 minutes</td>
<td>37.4 ± 0.3</td>
<td>37.3 ± 0.3</td>
<td>38.5 ± 0.3</td>
<td>1.2 ± 0.0</td>
<td>0.06 ± 0.0 A</td>
<td>20</td>
</tr>
<tr>
<td>15 minutes</td>
<td>37.4 ± 0.2</td>
<td>37.3 ± 0.3</td>
<td>38.4 ± 0.3</td>
<td>1.1 ± 0.1</td>
<td>0.05 ± 0.0 A</td>
<td>16</td>
</tr>
<tr>
<td>20 minutes</td>
<td>37.2 ± 0.2</td>
<td>37.2 ± 0.2</td>
<td>38.6 ± 0.3</td>
<td>1.4 ± 0.1</td>
<td>0.07 ± 0.0</td>
<td>16</td>
</tr>
<tr>
<td>25 minutes</td>
<td>37.3 ± 0.2</td>
<td>37.2 ± 0.3</td>
<td>38.4 ± 0.3</td>
<td>1.2 ± 0.0</td>
<td>0.06 ± 0.0 A</td>
<td>16</td>
</tr>
</tbody>
</table>

*A denotes significantly slower increase in core temperature compared to the control condition. Significance was deemed at the $P < 0.05$ level.

Figure 6.4. shows change in Tre from resting values, throughout each pre-cooling condition, warm up and each sprint of the CISP. Tre was lower in all conditions when compared to the control from sprint 1 of the CISP, however none of these were significant ($P > 0.05$). Tre increased at a significantly slower rate throughout the CISP in the 10, 15 and 25 minute conditions compared to the control ($P < 0.01$) (also shown in Table 6.3).
6.3.2.2. Muscle Temperature

Muscle temperature (Tmu) reduced from resting values in all cooling conditions, with the 15 minute condition showing the greatest reductions, however none of these were significant within conditions, nor when compared to the control \((P > 0.05)\). The 15, 20 and 25 minute cooling conditions had a significantly greater difference between post cooling Tmu and post CISP Tmu \((P < 0.05)\). Post CISP Tmu was significantly lower than control in only the 15 minute cooling condition \((P < 0.05)\) (Figure 6.5). Only the 15 minute condition displayed a positive linear relationship between change in muscle temperature and mean PPO (Figure 6.6), however this was only a low-moderate effect \((r = 0.374, P > 0.05)\). The 10, 20 and 25 minute conditions displayed a low relationship in change in muscle temperature and mean PPO \((r = -0.11, r = -0.19\) and \(r = -0.08\), respectively, \(P > 0.05\)) (Figure 6.6).
Figure 6.5. Mean (± SD) change in muscle temperature from rest, to post cooling and then post cycling intermittent for control, 10 minutes, 15 minute, 20 minutes and 25 minutes of cooling. *denotes significant difference in muscle temperature from post cooling to post CISP ($P < 0.05$). $^A$ denotes significant difference in muscle temperature compared to the control post cooling ($P < 0.05$).
Figure 6.6. Individual comparisons between 12 participants for 10 minutes (A), 15 minutes (B), 20 minutes (C) and 25 minutes (D) of cooling, assessing relationships between change in muscle temperature and mean peak power output during the cycling intermittent sprint protocol.

6.3.2.3. Body Heat Content

There was no difference between mean body heat content (BHC) at rest ($P > 0.05$). BHC for the control, 10 minute, 15 minute, 20 minute and 25 minute cooling conditions at rest showed variation of only 45 kilojoules between conditions. BHC significantly reduced compared to the control following 10, 15, 20 and 25 minutes of cooling ($P < 0.05$) with the 15 and 20 minute conditions reducing the greatest ($P < 0.01$). No significant difference between cooling conditions was found at the end of cooling ($P > 0.05$). At the end of the warm up, BHC was lower in all conditions compared to the control ($P < 0.05$), with the 15 minute condition remaining $20 \pm 48$ kJ lower than at rest. There were significant increases in BHC for the 10 and 20 minute cooling conditions from the end of cooling to the end of the warm up ($P < 0.05$) (Table 6.4).
Table 6.4. Body heat content (BHC) (kJ) at three time points (rest, post cooling and post warm up) and change between time points for each of the pre-cooling conditions and the control. Values are reported as mean ± SD.

<table>
<thead>
<tr>
<th>Condition</th>
<th>BHC at rest (kJ)</th>
<th>BHC post cooling (kJ)</th>
<th>Δ BHC rest – post cooling (kJ)</th>
<th>BHC post warm up (kJ)</th>
<th>Δ BHC post cooling to post warm up (kJ)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>9177 ± 878</td>
<td>9177 ± 878</td>
<td>-</td>
<td>9479 ± 803</td>
<td>302 ± 75</td>
</tr>
<tr>
<td>10 minutes</td>
<td>9221 ± 663</td>
<td>8815 ± 683 ^A</td>
<td>-406 ± 20</td>
<td>9317 ± 612 ^A</td>
<td>502 ± 71 *</td>
</tr>
<tr>
<td>15 minutes</td>
<td>9176 ± 715</td>
<td>8741 ± 688 ^A</td>
<td>-435 ± 27</td>
<td>9156 ± 763 ^A</td>
<td>415 ± 75</td>
</tr>
<tr>
<td>20 minutes</td>
<td>9176 ± 752</td>
<td>8692 ± 617 ^A</td>
<td>-484 ± 135</td>
<td>9206 ± 639 ^A</td>
<td>515 ± 22 *</td>
</tr>
<tr>
<td>25 minutes</td>
<td>9180 ± 794</td>
<td>8821 ± 687 ^A</td>
<td>-359 ± 106</td>
<td>9258 ± 632 ^A</td>
<td>437 ± 55</td>
</tr>
</tbody>
</table>

^A denotes significant difference from the control. * denotes significant increase post cooling – post warm up. Significance was accepted at the $P < 0.05$ level.

In all conditions body heat content remained lower than the control during the first stage of the CISP (sprints 1 – 4) ($P < 0.05$). The 15 minute cooling condition remained lower than the control ($P < 0.05$) until sprint 7, yet was not different to any of the other cooling conditions ($P > 0.05$). Trends showed that BHC in the 15 and 20 minute cooling conditions remained lower than the control throughout the remainder of the CISP although this was not significant ($P > 0.05$) (Figure 6.7).
Figure 6.7. Mean (± SD) body heat content at rest, during each leg cooling condition, the warm up and during the cycling intermittent sprint protocol. -30 minutes signifies rest, left of dotted lines are the cooling periods, 0 minutes signifies warm up, to the right of the dotted lines signifies BHC during the CISP. * denotes significant difference from the control in all conditions. C denotes significant difference from the control in the 15 minute condition. Significance was deemed at the $P < 0.05$ level. Error bars have been removed for visual clarity.

6.3.2.4. Heart Rate

Mean heart rate (HR) remained unchanged from rest to end of cooling for the control and all four cooling conditions ($P > 0.05$). HR in all conditions significantly increased from post cooling to post warm up of the CISP ($P < 0.05$). Although there were no differences in HR between conditions after sprint 1, HR remained lower ($P < 0.05$) in the warm up compared to sprint 1 in only the 15 and 20 minute cooling conditions (Figure 6.8). HR in the 10 minute condition (158 ± 13 beats.min$^{-1}$) was significantly lower than that of the control (163 ± 13 beats.min$^{-1}$) ($P < 0.01$), but the 15 minute (161 ± 12 beats.min$^{-1}$), 20 minute (160 ± 13 beats.min$^{-1}$) and 25 minute (158 ± 20 beats.min$^{-1}$) conditions were similar. There was no significant difference in HR between cooling conditions throughout the CISP (Figure 6.9).
all conditions, only sprints 1 and 2 were significantly lower than that of the final sprint ($P < 0.05$).

**Figure 6.8.** Mean heart rate (± SD) throughout control, 10, 15, 20 and 25 minutes of cooling, at the end of the warm up and after the first sprint during the cycling intermittent sprint protocol. -30 minutes signifies rest (prior to cooling), 0 minutes signifies end of standardised warm up, signified by the dotted line. Error bars have been removed for visual clarity.
**Figure 6.9.** Mean heart rate (± SD) throughout the cycling intermittent sprint protocol after a control, 10 minutes, 15 minutes, 20 minutes and 25 minutes of leg cooling. Error bars have only been included for the control condition to aid in visual clarity.

### 6.3.3. Perceptual Variables

#### 6.3.3.1. Thermal Sensation

Figure 6.10. displays measures of thermal sensation (TS) from rest, during cooling and during the warm up. TS did not differ between conditions at any point during the cooling ($P > 0.05$). TS reduced from rest to end of cooling by $1.4 ± 0.2$, $1.6 ± 0.3$, $1.5 ± 0.1$ and $1.6 ± 0.9$ for the 10, 15, 20 and 25 minute conditions respectively, however none of these were significant ($P > 0.05$). Only the 10 minute cooling condition significantly increased from the end of cooling to the end of the warm up ($P < 0.05$). There were significant reductions ($P < 0.05$) in TS from rest in the 15, 20 and 25 minute cooling conditions, however this was during the first 5-10 minutes of cooling only. TS significantly increased from the lowest point of cooling to the end of the warm up in all conditions ($P < 0.05$).
Figure 6.10. Mean (± SD) thermal sensation scores during control, 10 minute, 15 minutes, 20 minute and 25 minute cooling conditions and warm up. 0 minutes signifies TS resting values (prior to cooling), 30 minutes signifies TS scores during the warm up (post cooling). The dotted line signifies the warm up. Error bars have only been included for the control to aid in visual clarity.

In all cooling conditions, TS remained significantly lower ($P < 0.05$) than the control at the end of the warm up (Figure 6.11), and this remained the case until sprint 9 where only the 15 minute condition was lower. TS did not differ between cooling conditions throughout the CISP ($P > 0.05$). All cooling conditions were significantly lower ($P < 0.05$) than the control after the final sprint in the CISP. In the 15 minute cooling condition, TS after sprint 19 was the only sprint during the CISP not to be significantly lower than the control.
Figure 6.11. Mean (± SD) thermal sensation scores during the warm up and throughout the cycling intermittent sprint protocol after a control, 10 minutes, 15 minutes, 20 minutes and 25 minutes of leg cooling. -1 (left of dotted line) signifies TS during the warm up, to the right of the dotted line signifies TS during the CISP. * denotes significant difference from the control in all conditions. C denotes significant difference from the control in only the 15 minute condition. Significance was deemed at the P < 0.05 level. Error bars have only been included for the control condition to aid in visual clarity.
6.3.3.2. Rating of Perceived Exertion

At the end of the warm up, mean rating of perceived exertion (RPE) was lower than the control following 10, 15, 20 and 25 minutes of cooling, however this was not significant ($P = 0.908$, $P = 0.664$, $P = 0.908$ and $P = 0.908$ respectively). There was no difference between conditions or compared to the control for RPE throughout the CISP ($P > 0.05$). RPE was not difference between conditions after the final sprint of the CISP ($P = 0.873$, $P = 0.575$, $P = 0.825$, $P = 0.914$ respectively) (Figure 6.12).

**Figure 6.12.** Mean (± SD) rating of perceived exertion during warm up and throughout the cycling intermittent sprint protocol after a control, 10 minutes, 15 minutes, 20 minutes and 25 minutes of leg cooling. -1 (left of dotted line) signifies RPE during the warm up, to the right of the dotted line signifies RPE during the CISP. Error bars have only been included for the control condition to aid in visual clarity.
6.4. DISCUSSION

The purpose of this study was to determine whether intermittent sprint exercise in the heat was affected by cooling in duration-dependant manner. The aim was to establish the optimal duration of leg cooling as an ergogenic aid to intermittent sprint cycling performance in hot humid conditions. Cooling durations were determined from existing pre-cooling literature, with durations of 10 – 25 minutes as used by Duffield et al (2003), Castle et al (2006), Duffield and Marino (2007) and Minett et al (2012) included. Less than 10 minutes was considered too short a time to elicit any major effects on performance (Duffield et al, 2003). A range of times were chosen to span those already shown to have some effect on physiological or performance measures (chapter V, Castle et al, 2006; Minett et al, 2012). It was firstly hypothesised that leg cooling would aid in improving intermittent sprint cycling performance. Secondly, it was hypothesised that improved performance would be a consequence of longer durations of cooling. The main finding was that cooling for 15 minutes elicited the greatest improvement in performance, with a 3.75 % improvement in peak power output and a 6.7 % improvement in work done when compared to the control. However, longer periods of cooling impaired performance; after 25 minutes of cooling performance was reduced when compared with all other times, including the non-cooled control condition. Muscle temperature was only significantly lower than the control after 15 minutes of cooling and further periods of time did not significantly reduce muscle temperature further. When perceived thermal strain was measured it was lower in all cooling conditions post cooling and remained so until sprint 9 (18 minutes) from which point only 15 minutes of cooling maintained a significantly lower thermal strain.

Peak power output throughout the CISP was greatest in the 15 cooling minute condition. It is difficult to compare this with any previous research, due to the lack of literature on duration-dependant responses to pre-cooling. Duffield and Marino (2007) cooled for 15 minutes prior to a field-based sprint protocol that incorporated phases of self-paced recovery however the purpose of this study was to look at a half-time cooling intervention involving mixed-method cooling, with results directed towards the cooling technique rather than the duration applied. Minett et al (2012) is the only study where intermittent sprint performance was investigated following different durations of cooling (10 and 20 minutes of mixed-method cooling). Their study reported no differences in sprint performance between the control and the 10 minute cooling condition. Improvements in sprint times were observed in only the second bout of
exercise (50 – 85 minutes of exercise) after 20 minutes of cooling. Castle et al (2011) suggest cooling as a short term intervention with thermoregulatory benefits such as reduced thermal strain and subsequent improved performance lasting up to 40 minutes, differing to the results of Minett et al (2012). In the present study, improvements in peak power output occurred in the 15 minute condition, but not in the 20 minute cooling condition, with significant differences in individual sprints compared to the control occurring between 20 and 40 minutes. Despite the differences in duration, this agrees with Castle et al’s (2006) study where improvements in peak power output following 20 minute pack cooling occurred during the latter part of the CISP. However, it was 15 minutes of cooling that displayed optimal performance improvements of all conditions, rather than 20 minutes, as described by Castle et al (2006). Prolonging cooling to 25 minutes caused reductions in both peak power output and work done. These findings indicate that a duration-dependant response in performance exists with isolated leg cooling and that there is a threshold beyond which further cooling is counterproductive.

Normally exercise performance in the heat reduces power, endurance and recovery. In this study such negative effects of heat on performance were not as great as has been reported in previous intermittent sprint and heat-based research (Sunderland and Nevill, 2004; Castle et al, 2006; Skein et al, 2012) particularly when cardiovascular strain was measured. Part of this may be due to the low body mass of participants in this study compared with other research studies. Individuals with a higher body mass have been shown to produce and store more heat at the same running speeds of lighter individuals, with these effects accentuated when exercising in the heat (Marino et al, 2000). Participants in the present study were on average 7 kg lighter than those of Castle et al (2006) and 8 kg lighter than those of Skein et al (2012). Marino et al (2004) suggest that under heat stress, heavier athletes select lower intensities than lighter athletes in order to maintain exercise duration to complete a task. As with chapter five, reductions in core temperature were not observed as a result of any of the cooling periods, which conflicts with the results of Castle et al (2006). Reductions in core temperature of 0.5 °C have been observed upon removal of the cold stressor (Booth et al, 1997; Cotter et al, 2001). This core temperature reduction has been referred to as an “afterdrop” and has been linked to perfusion of cold tissue and re-distribution of heat from the core to the periphery or the cooling of the blood returning to the core (Giesbrecht and Bristow, 1992). Additionally, the conductive and convective transfer of heat through deep tissues has also been suggested to be a factor (Webb, 1986; Romett, 1988). However, an
afterdrop is not always present (Kay et al, 1999). Kay et al (1999) manipulated the cooling procedure so that a rapid change in core temperature did not occur over 60 minutes and a slower rate of cooling has been shown to limit core temperature changes (Webb, 1986). Furthermore, Cotter et al (2001) included a mid – cooling warm up that may have increased blood circulation during the cooling period which was maintained into the exercise trial allowing for faster rewarming of the periphery from the core (Webb, 1986). Rectal temperature did show a trend towards lowering in the cooling trials after the pre-test warm up and during the CISP. This was reflected in the significantly slower rise in temperature (0.05 - 0.06 °C.sprint⁻¹ vs. 0.07 °C.sprint⁻¹) throughout the CISP suggesting that the use of peripheral cooling delays the onset of heat gain within the core, rather than causing an immediate drop in core temperature. Increasing core temperatures have been linked to the down regulation of muscle recruitment through reductions in central activation (Thomas et al, 2006; Abbiss et al, 2010) and so maintaining a lower core temperature may aid in achieving greater performance outcomes in warm environmental conditions. This may explain the performance differences observed, particularly in the 15 minute cooling condition, however does not explain why the 25 minute cooling condition yielded lower performance measures despite similar rates of change in core temperature. Differences in performance have been more obvious in studies eliciting greater hyperthermic stress. Nybo and Neilsen (2001) reported core temperatures of 40 °C during a hyperthermic trial, 1.3 °C higher than the control condition in the present study. These lower levels of hyperthermia may explain why the differences between the no cooling control and the cooling interventions appear less obvious.

The effect of muscle temperature on sprint ability still appears equivocal. Drust et al (2005) saw reductions in power output in repeated sprinting in the heat and associated this with increases in muscle temperature. Conversely, exercise-induced hyperthermia has been shown to have no effect on isokinetic maximal contractions (Ftaiti et al, 2001) while warming of the muscles has been show to improve performance (Sargeant, 1987; Ball et al, 1999). Furthermore, the effect of muscle cooling also appears to be ambivalent. Improvements in peripheral blood flow have been observed as a result of extreme short-term cold stressors (Maekawa et al, 1998). However, cold muscle sees an increase in the duration of muscle fibre action potentials, which indirectly decreases conduction velocity (Farina et al, 2005; Ce et al, 2012) and reduces EMG amplitude (Petrofsky and Lind, 1980). During peripheral pre-cooling using ice pack or cold-water immersion, muscle temperature declines (chapter V; Gonzalez-Alonso et al, 1999; Sleivert et al, 2001; Booth et al, 2004; Castle et al, 2006). In all
of these studies expect the one in chapter 5, muscle temperature reduced by up to 6 °C, whereas, as was the case in chapter 5, the present study measured reductions of up to 10 °C, with some participants showing reductions of up to 16 °C. Variations in pre-cooling technique and position of the temperature measurement probe may be able to explain these differences. Castle et al (2006) showed that packs reduce muscle temperature at a faster rate than ice vests or whole body immersion in cold water. Cooling for 15, 20 and 25 minutes reduced muscle temperature the greatest yet the relationship between muscle temperature and performance is not simple. There were no significant differences between the reductions in muscle temperature, with all lowering to similar values, supporting the possibility of a temperature range as suggested in chapter 5, despite longer cooling in some conditions. De Ruiter et al (1999) reported reductions in maximal isometric force below muscle temperatures of 22 °C which could explain reduced performance for some participants. However this does not explain the larger peak power output produced during the 15 minute condition despite similar reductions in temperature when compared to other conditions suggesting other responses may be a factor. Impairment of muscle contractibility has been observed below muscle temperatures of 31 °C (Clarke et al, 1958) with neuromuscular transmission and specifically the release of acetylcholine and calcium ions reduced (Vincent and Tipton, 1988). Improved performance has been observed following reductions in peripheral temperatures without changes in core temperature (Kay et al, 1999). Furthermore, cooling of the skin has been shown to increase the number of motor units (Yona, 1997) required for sustained activity, through enhanced afferent feedback via the spinal cord (Oksa et al, 2002). It could be that certain levels of reduction in muscle temperature may contribute to enhanced afferent feedback and thus be the cause for increased performance, as seen for the 15 minute condition. This may be due to an additional delaying response of central regulation in protecting against hyperthermia (Castle et al, 2006).

Muscle temperature increase, post CISP, was greatest in the 15, 20 and 25 minute conditions. However, this is explainable given the greater drop in muscle temperature as a result of cooling and so lower starting temperature. Muscle temperature reached similar values in all conditions upon completion of the CISP (~ 38 °C) however muscle temperature at the end of the 15 minute cooling condition was 1 °C lower than that of the control, with only the control and the 25 minute condition exceeding 38 °C. Both the control and 25 minute cooling conditions produced the lowest peak power output and work done and it can be suggested that this is the result of increased force variability due to the higher muscle temperatures.
(Brazaitis et al, 2010). The detrimental effect of higher muscle temperature on mitochondrial function and the possible damage to the inner properties of membrane should also be considered due to potential proton leakage whereby protons re-enter the mitochondrial matrix without contributing to ATP synthesis (Willis et al, 1994) thus encumbering performance. Furthermore, the difference in participant body mass and sum of skin folds may again be accountable for the discrepancies in the level of cooling seen between the present study and Castle et al (2006) as it has been suggested that the rate of cooling is affected by larger body mass but not necessarily larger muscle mass (Oksa et al, 1993; Oksa et al, 1997). Lewis (1930) proposed a paradoxical vasodilatory protective response; “the hunting reaction” (also referred to as “Cold-Induced Vasodilation” or “CIVD,” Flouris and Cheung, 2008), whereby under acute cold stress the vasoconstricted blood vessels experience a period of vasodilation, increasing blood flow and thus temperature of the periphery before vasoconstriction occurs again. This could be occurring on a local level in the legs given that core temperature changes were not evident yet skin and muscle temperature differences occurred after longer cooling durations. Temperature changes have been shown to occur in the hand after 5 – 10 minutes of cold exposure however, CIVD has been suggested to occur after 10 – 15 minutes of cold exposure as a protective mechanism against cold injury (Wilson and Goldman (1970). The hands contain a greater number of thermoreceptors compared to the legs and it may be that the higher sensitivity of these thermoreceptors explains the quicker temperature changes and thus a delayed vasodilatory response in the legs (Cotter et al, 1996). Furthermore, prolonged cooling may induce a second phase of vasoconstriction. This could partially explain why greater muscle temperature changes in the 15 minute condition caused greater power output for only this condition. Laser Doppler Flowmetry tests during pre-cooling could be conducted to clarify CIVD responses following acute aggressive local cooling and intermittent sprint exercise.

Although reductions in core temperature are seen as the greatest ergogen in the application of pre-cooling, manipulation of skin temperature has been shown to yield similar benefits (Kay et al, 1999). The factor that seems most important for this effect is the rate of body heat storage and increased body heat content. In the present study, reductions in core temperature did not occur but direct cooling of the leg muscles did cause reductions in skin temperature in all cooling conditions, thus significantly lowering body heat content compared to the control. However, the size of the reductions varied between cooling conditions in a similar fashion to that of muscle temperature. 10 minutes of cooling caused reductions in skin temperature yet
these were not as large as observed after 15 or 20 minutes of cooling ($r = -0.69$, $P < 0.01$). Skin temperature and body heat content were higher at the end of cooling in the 25 minute condition than that of the 15 or 20 minute conditions further suggesting the possibility of a local ‘hunting reaction’. Body heat content at the end of the warm up was lower than at rest in the 15 minute condition, which partially supports Marino (2002) in that commencing performance with lower thermoregulatory variables will allow for prolonged performance. Kay et al (1999) state that improved performance via reduced skin temperatures allowed for enhanced rate of heat storage, which may have been the case for the present study. Although there were no differences in performance in the early stages of the CISP, it could be suggested that reduced body heat content allows similar work intensities to occur yet with reduced effect on neuromuscular or cardiovascular function, resulting in prolonging of the fatiguing state. Throughout the CISP, there were no differences in body heat content between cooling conditions, shown by the similar rates of increase, which were all faster than that of the control, yet reaching similar final values. Nevertheless, the 15 minute cooling condition produced the greatest performance benefits. Thermoregulatory responses to heat stress, in particular sweat gland recruitment and sweat response (Buono and Connolly, 1992) can be associated with vasomotor responses. It may be that the correct duration-dependant dose of cooling delayed these sudomotor responses through vasoconstriction of the arteries and arterioles contributing to inhibitory feedback from peripheral thermoreceptors through reduced peripheral blood flow. The occurrence of vasodilatory responses in the longer duration may have limited the inhibitory feedback and in turn permitted normal thermoregulatory responses to heat gain, such as reduced activation of the musculature, to occur. Neither sweat response nor skin blood flow were measured in the present study and although an increased sweat response is usually observed with rising core temperature (Buono and Maupin, 2003) it could be surmised that skin temperature has a role the thermoregulatory responses to heat stress through the contribution of information from thermoreceptors to central regulation. The rate of heat gain during the CISP was slower in the cooling conditions compared to the control. This suggests that the physiological benefits of cooling may be observed during early performance yet perhaps allow for maintained performance in the latter stages of exercise when the physiological effects are beginning to subside. This has been observed previously (Booth et al, 1997; Kay et al, 1999; Cotter et al, 2001) and lead Arngrimmson et al (2004) to conclude that the magnitude of thermal strain is not proportional to the physiological ergogenic effects.
During cooling trials, systemic thermal sensation did not provide a reflective measure of how participants’ felt, probably due to the lack of change in upper body skin temperature. The large decreases in muscle temperature and thigh skin temperature did cause significant reductions in local perceptual measures of thermal sensation of the legs. From rest to immediately post cooling saw no differences in local thermal sensation between conditions however the longer durations of cooling saw significant local reductions within the first 10 minutes of cooling. Warming of the ice packs throughout the cooling period may explain the lack of rest to post cooling differences. Secondly, the extreme temperature drops of the skin may have caused a local habituation response of the thigh. Vasoconstriction responses and skin thermoreceptors may have initially been blunted locally by the central nervous system resulting in participants feeling warmer than they actually were (Klement and Arndt, 1992; Young, 1996). Additionally, some participants reported slight feelings of discomfort upon initial ice pack application however this began to subside as cooling continued. On commencement of exercise, particularly after longer cooling durations, more than half of the participants reported their legs to be feeling heavy, aching or numb. Nociceptors are usually active when skin temperature falls below 18 °C although innocuous feelings of cold have been reported at mild temperatures (25 °C) (Green, 2002) suggesting the influence of hyperalgesia and increased nociceptor activation effects on motivation and consequent performance (Acevedo and Ekkekakis, 2001). Nonetheless, whole body thermal sensation remained lower than the control in all conditions at the end of the warm up and for the first half of the CISP demonstrating that cooling via ice pack application reduces perceived thermal strain during intermittent sprint activity during hot humid conditions. Cooling for 15 minutes provided the best increase in thermal comfort for the duration of exercise, which may account for some of the improved performance measured in this group. Perceptual variables have recently been considered as factors in fatigue (Marcora et al, 2009) through input to a motivational model (Abbiss and Laursen, 2005) whereby fatigue is a predetermined phenomenon and central motor drive is reduced through previous experience, current feeling and mood state. However, the present data does not fully support this theory as although trends in rating of perceived exertion appeared lower after cooling, no differences were observed at the end of performance or indeed any point during the CISP, indicating that physiological factors may be more responsible for reduced intermittent sprint performance in the heat. This is further demonstrated by the improvements in the final sprint compared to that of the preceding sprints as was observed by Castle et al (2006). If ratings of perceived exertion and thermal strain were main determinants of fatigue, then it could be assumed that
as these rise, performance would decrease until cessation of exercise. This notwithstanding, the usefulness of ratings of perceive (RPE) as a dependent variable in association with physiological measures is susceptible to a number of other factors (see review by Chen et al, 2002) and test-retest data has shown variability in RPE scores widens as exercise intensity increases (Lamb et al, 1999). Furthermore, intra-individual differences have been observed during RPE increase during intermittent sprint exercise (Laurent et al, 2012) with the suggestion that increases in perceived exertion correspond better with physiological strain than with anticipatory feed-forward mechanisms. It may be that the nature of the protocol where individuals are repeatedly exercising at maximal capacity does not allow for an accurate detection of perceived exertion and that time-based increases are instead being observed (Faulkner et al, 2008). It is more plausible that during the CISP, physiological variables contribute to central motor drive through afferent feedback from the working muscles. This may cause a down-regulation of exercise intensity and duration through reduced central activation limiting motor unit recruitment, while self-motivation allows for athletes to continually produce high levels of peak power output during the sprints. This would explain why no cooling or longer cooling durations, such as have been used in other intermittent exercise cooling studies (Drust et al, 2000a; Sleivert et al, 2001; Cheung and Robinson, 2004) may have elicited poorer performance at the start or throughout the protocols; individuals felt they were too cold or too warm to produce maximal outputs. A cycle incorporating anticipatory feed-forward and feedback components has been proposed (Tucker, 2009). An initial expectation of work rate and exercise duration is pre-set before exercise commences and then continually modified as exercise progresses (Tucker et al, 2006). This adjustment in exercise intensity from physiological feedback prevents catastrophic homeostatic failure (Marino, 2004). During exercise, the anticipatory end point may be reset in a feed-forward manner consequently allowing for recruitment from power reserves resulting in final improvements at the end of the protocol.
6.4.1. Conclusion

In summary, this investigation confirmed previous evidence of the benefits of pre-cooling via ice pack application to the legs on subsequent intermittent sprint performance in the heat. These ergogenic benefits are displayed via suppression of physiological and perceptual responses to exertional heat stress. The most optimal duration of cooling was 15 minutes, eliciting improvements of 3.75% and 6.7% in peak power output and work done respectively, while cooling for longer durations becomes detrimental physiologically and on performance. Isolated leg cooling for 15 minutes, which is more feasible than longer cooling durations previously used, can yield improved performance for team game athletes competing in hot humid environments. However, before field-based application individuals should be aware of the duration-dependent responses to cooling and that individual difference may contribute to the specificity of cooling duration. Further research should tackle the effect of pre-cooling at a neuromuscular level, investigating the influence of isolated leg cooling and consequent reduced muscle temperatures on nerve conduction velocity, central activation ratios and motor unit recruitment and supra-spinal motor function.
CHAPTER SEVEN

Study Four

Predictions of muscle temperature based on skin temperature are not accurate following intermittent sprint exercise in hot humid conditions
CHAPTER VII: PREDICTIONS OF MUSCLE TEMPERATURE BASED ON SKIN TEMPERATURE ARE NOT ACCURATE FOLLOWING INTERMITTENT SPRINT EXERCISE IN HOT HUMID CONDITIONS

7.1. INTRODUCTION

Body temperature is regularly used as a key component in calculations of heat strain, with the body being divided into two compartments; the core and the shell (Jay and Kenny, 2007). In a two compartment model, muscle temperature is assumed to be part of the core component, despite the association between the working muscles and periphery. However, it has been suggested that this two-compartment model is not reflective of the kinetics of heat exchange within the body during exercise (Jay et al, 2007). Subsequently, Jay et al (2007) proposed that a three-compartment model that incorporates a deep tissue component, such muscle temperature should be considered, given that ~40 % of total body mass is muscle mass (Wang et al, 1992).

This discrepancy is further demonstrated under conditions of heat stress away from homeostatic values, particularly following pre-cooling where changes in local tissue temperatures have been observed without similar changes in core temperature (Cotter et al, 2001; Sleviert et al, 2001; Castle et al, 2006). Moreover, reductions in muscle temperature have been attributed to reduced muscle blood flow (Thorsson et al, 1985), possibly reducing lactate efflux from the working muscle (Starkie et al, 1999), reduced glycogenolysis (Starkie et al, 1999) and consequent reductions in sprint performance (Sargeant, 1987; Crowley et al, 1991). Leg cooling has been shown to improve intermittent sprint performance in the heat (Castle et al, 2006), but given the adverse effects of cold muscle on performance, the margin of temperature reduction before cooling negates the ergogenic benefits may be small. Therefore, muscle temperature measurement could be an important tool in optimising leg cooling strategies.

Nevertheless, assumptions arise where muscle temperature cannot be taken. A prediction of muscle temperature from skin temperature has previously been made using data from a submersed forearm in water of different temperatures, ranging from 17 – 45 °C (y = 1.02x + 0.89) (de Ruiter et al, 1999). Calculated values have then been used to draw conclusions such as the effect of temperature on the rates of force development (de Ruiter et al, 1999) and the diurnal effects of muscle temperature on power output (Racinais et al, 2005). However, the
equation does not account for temperatures outside of ambient conditions, nor has its accuracy been tested during exercise.

The purpose of this study was to test the accuracy of the muscle temperature prediction estimated from skin temperature as proposed by de Ruiter et al (1999).
7.2. METHODOLOGY

7.2.1. Participants

Data for nineteen participants was obtained from two different experimental studies (chapters V and VI). All participants fitted criteria as described in chapter III (section 3.2.1).

7.2.2. Data Collection

Data from chapters V and VI were used to gather skin (Tskin) and muscle (Tmu) temperature measures. Tskin and Tmu measures were taken at the same time point. In chapter V, Tskin ranged from of 31.7 ± 1.03 °C at rest, to 7.3 ± 3.33 °C following 25 minutes of leg cooling as described in chapter III (section 3.5). In chapter VI, Tskin was measured following four different durations of leg cooling and following 40 minutes of intermittent sprint cycling exercise in hot conditions. For a full breakdown of protocols, see relevant chapters.

7.2.3. Muscle Temperature Prediction Equation

De Ruiter et al (1999) measured temperature of the lower arm following 20 minutes of immersion at four different temperatures; 17.0, 22.5, 30.5 and 45.0 °C. Tskin and Tmu data were plotted (Figure 7.1) and the following regression equation produced from the data:

**Equation 7.1.** Prediction of muscle temperature as proposed by de Ruiter et al (1999)

\[ y = 1.02x + 0.89 \quad (r^2 = 0.98) \]

where \( y \) = muscle temperature and \( x \) = skin temperature

To determine the accuracy of this equation (de Ruiter et al, 1999), measured Tskin data was used to calculate a predicted muscle temperature, and compared to a measure Tmu.
Figure 7.1. The relationship between skin and muscle temperature (°C) obtained following lower arm immersion (de Ruiter et al, 1999).

7.2.4. Statistical Analysis

All data was tested for normality with missing temperature variables excluded on a pairwise (analysis-by-analysis) basis. Data for measured vs. predicted Tmu was analysed using a student’s paired sample t-test. In the case where data was not normally distributed, a non-parametric related-samples Wilcoxon signed rank test was conducted. Pearson’s product moment correlation coefficient used for relationships between measured Tskin and Tmu variables. All data was reported with means and standard deviations. Where regression equations were made, standard error of the estimate was reported. $P < 0.05$ was the level at which significance was accepted.
7.3. RESULTS

Across both chapters, two hundred and eighty-four temperature measurements have been used. Tskin data was separated into four groups, ranging from 0 – 9, 10 – 19, 20 – 29 and > 30 °C for resting measures and > 30 °C post exercise to assist in analysis. Mean measured Tmu at a range of mean Tskin measures at rest and following 40 minutes of intermittent sprint exercise in hot, humid conditions are displayed in Table 7.1. Additionally, a predicted Tmu has been calculated from the measured Tskin with the difference between measured Tmu and predicted Tmu reported.

<table>
<thead>
<tr>
<th>Skin Temperature (°C)</th>
<th>Tmu measured (°C)</th>
<th>Tmu predicted (°C)</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>REST</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 - 9</td>
<td>18.8 ± 3.8</td>
<td>7.7 ± 2.3 *</td>
<td>11.1 ± 1.5</td>
</tr>
<tr>
<td>10 - 19</td>
<td>23.6 ± 3.9</td>
<td>14.3 ± 2.7 *</td>
<td>9.3 ± 1.2</td>
</tr>
<tr>
<td>20 - 29</td>
<td>33.7 ± 2.0</td>
<td>30.5 ± 2.0 *</td>
<td>3.2 ± 0.0</td>
</tr>
<tr>
<td>&gt; 30</td>
<td>34.0 ± 1.3</td>
<td>32.2 ± 0.8 *</td>
<td>1.8 ± 0.5</td>
</tr>
<tr>
<td>POST EX.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 35</td>
<td>37.7 ± 1.7</td>
<td>36.7 ± 1.3 *</td>
<td>1.0 ± 0.4</td>
</tr>
</tbody>
</table>

*denotes a significant difference to measured muscle temperature (P < 0.01).

7.3.1. Muscle temperature at rest

Two hundred and thirty eight temperature measures were made at rest. Measured Tskin was strongly correlated with measured Tmu at rest (r = 0.942, P < 0.01). Differences were observed between measured Tmu and predicted Tmu at every measured skin temperature at rest and after exercise (P < 0.05). In all cases, predicted Tmu was lower than measured Tmu. The colder Tskin the larger the differences between measured and predicted Tmu (Figure 7.2). Tmu was underestimated by 11.1 ± 1.5 °C when skin temperature fell below 9 °C.
7.3.2. Muscle Temperature post exercise

Forty six temperature measures were made post intermittent sprint exercise in hot humid conditions. Tmu was underestimated by 1.0 ± 0.4 °C after 40 minutes of intermittent sprint exercise in the heat ($P < 0.05$). However, measured Tskin and Tmu were not correlated ($r = 0.277, P > 0.05$) (Figure 7.3).
Figure 7.3. Mean (± SD) skin and muscle temperature plots for measured and predicted data following 40 minutes of intermittent sprint exercise in hot, humid conditions.
7.4. DISCUSSION

The purpose of this study was to test the muscle temperature estimation proposed by de Ruiter et al (1999) against simultaneous muscle and skin temperature measures. The main finding was that predicted muscle temperature from the equation of de Ruiter et al (1999) significantly underestimated measured muscle temperature at rest, with the difference exacerbated as skin temperature was reduced. Furthermore, although the variation between measured and predicted muscle temperature was smallest post exercise (i.e. at the highest Tmu values), predicted muscle temperature was still underestimated.

To the authors’ knowledge, there is only one study that has attempted to calculate muscle temperature from other measured temperature variables. In a pilot study, de Ruiter et al (1999) measured skin and muscle temperature of the forearm following 20 minutes of water immersion at temperatures of 17, 22.5, 30.5 and 45 °C and produced a regression equation based on the data recorded. However, this pilot study consisted of only three participants and from these three participants, one participant was measured twice in two conditions and one participant not measured at all at one temperature. Furthermore, forearm contractions were made between measures and, while the authors state that muscle temperature returned to baseline on each occasion, fluctuations of up to 0.7 °C in muscle temperatures were reported and skin and muscle temperature were described as “relatively” stable, suggesting some variation. Therefore the reliability and validity of this equation should be questioned.

Despite these issues of validity, Racinais et al (2005) used the same equation to aid in conclusions of the effect of a warm up on muscular power during a sprint cycling protocol. In their conclusions they stated that circadian rhythm was a limiting factor in muscle temperature calculation (Racinais et al, 2005) despite de Ruiter et al (1999) reporting the predictive equation as reliable. However, the muscle groups involved may have also influenced the conclusion. The lower arm muscle group is considerably smaller than that of other areas of the body, such as the legs, and may not afford transferability of the equation. Furthermore, de Ruiter et al (1999) measured muscle temperature in the pilot study at rest whereas Racinais et al (2005) calculated muscle temperature of the legs at four points during exercise. In the present study, skin and muscle temperature were not correlated post exercise ($r^2 = 0.08, P > 0.05$). While the level of heat gain in the muscle may be exacerbated under hot conditions, it demonstrates the limited applicability of calculated muscle temperature from a linear regression equation produce from resting data. This variation may, in part, be due to
the difference in participants’ perception of maximal effort. Although instructed to sprint at
the best of their ability in Racinais et al (2005) and for the data used in the present study,
participant variability has been shown to affect exercise performance (Laursen et al, 2007).
Additionally, it has been reported that a sub-conscious pacing strategy is evident in
supramaximal exercise (Ansley et al, 2004) and both of these would consequently affect the
level of exertional heat gain.

Measured and predicted muscle temperature was significantly different at rest. However, at
skin temperatures more closely associated with normothermic conditions, the variation was
considerably smaller. This is likely to be due to the similarities in skin temperatures in the
present study compared to those recorded by de Ruiter et al (1999). Notwithstanding, when
skin temperature fell below 20 °C, the difference between predicted and measured muscle
temperature increased three-fold. Skin temperatures below 9 °C resulted in an
underestimation of muscle temperature of up to 11 °C. Besides variables associated with
subject difference, this inaccuracy may be associated with the plateau observed in muscle
temperature at ~ 20 °C (see chapter V) which is not incorporated in the linear regression
equation proposed by de Ruiter et al (1999). Therefore, the use of this equation should only
be considered for skin temperature ranges around those of ambient conditions while at rest.

7.4.1. Development of an improved muscle temperature equation

In light of these incongruences between our data and that of de Ruiter et al (1999), data from
the present study was used to produce a regression equation, but with a range in skin
temperature twice as large (present study vs. de Ruiter et al (1999): 1.8 - 34.0 °C vs. 20 - 36
°C, respectively). This equation, based on the relationship between measured skin and muscle
temperature, was:

Equation 7.2. Proposed muscle temperature prediction equation derived from data in this
thesis

\[ y = m x + c \pm SE_E \]  therefore, \[ \text{Predicted } y \ (°C) = 0.6328x + 14.788 \pm 2.48 \ (r^2 = 0.89) \]

where \( y \) = muscle temperature and \( x \) = skin temperature
Despite a lower $r^2$ value, it is proposed that this equation be used as a replacement of de Ruiter et al (1999) due to a number of reasons; i) a larger number of data points increasing statistical power and the precision of the prediction, while decreasing size of the confidence intervals associated with the relationship (Biau et al, 2008), ii) the greater range in skin temperature measures it is derived from, allowing its use outside of normothermic conditions, such as in pre-cooling studies, iii) the use of the leg muscles as the site of measurement given that the majority of exercise protocols within the literature relate to these muscle groups iv) a larger number of participants was used increasing ecological validity of the equation. Notwithstanding, results from this study suggest that the fluctuations in skin and muscle temperature due to individual variability do not allow for accurate prediction of muscle temperature post exercise in hot, humid conditions. Additionally, linear regression predictions neglect demographic differences; muscle mass to fat mass ratios, body mass to surface mass ratios, skin fold thickness of the limb, sex differences and depth of the muscle below the limb surface (Cheung et al, 2000; Havenith, 2001) and so any conclusions drawn from the prediction of muscle temperature should consider the possible contributing effects of these variables. Future research should look to produce an equation based on multiple regression data as this can reduce the size of the standard error of the estimate as well as allow the inclusion of other independent variables that may influence the prediction (Vincent, 1999), thus increasing its accuracy.

7.4.2. Conclusion

In conclusion, muscle temperature can be a useful measure in determining levels of heat strain as well as for the prescription of a pre-cooling dose prior to exercise in the heat. Where an actual measure cannot be made, predictions of temperature have been calculated. However, the equation proposed by de Ruiter el al (1999) underestimates measured muscle temperature by up to 11 °C as skin temperature reduces. Furthermore, the accuracy of the equation during or after exercise has been questioned following the lack of a significant relationship between skin and muscle temperature in these conditions. The equation $y = 0.6328x + 14.788 \pm 2.48$ has been proposed to be a more robust alternative given the larger sample size and greater range of temperatures used. The author suggests that further development of the equation is required using multiple regression analysis, incorporating
individual characteristics, such as thigh circumference and sum of skinfolds, to gain increased accuracy of the prediction.
CHAPTER EIGHT

General Discussion
CHAPTER VIII: GENERAL DISCUSSION

This chapter will be presented in three sections; firstly, an overview of the principle findings from studies 1 – 4 will be conducted, with a summary of the hypotheses reviewed (for full hypotheses see chapter II, section 2.8, page 67). Secondly, the common themes that have been discussed will be summarised and directions for future investigations that can be derived from this thesis proposed. Finally the practical applications of the research presented in this thesis will be discussed.

8.1. Principle findings from the studies conducted within this thesis

Study 1; Chapter IV The reliability of the cycling intermittent sprint protocol (CISP) was tested in the first study. Furthermore, the efficacy of a five-sprint familiarisation was also tested. One familiarisation period and two full CISP’s were completed in temperate conditions. No differences were observed between CISP’s. PPO provided a more reliable measure throughout the CISP with lower TEM compared to MPO, however strong reliability was observed for both PPO and MPO (ICC: 0.96 and 0.90, respectively). A low TEM (2.6 %) was reported for the familiarisation period compared to both CISP’s with a strong relationship observed between FAM vs. CISP 1 and CISP 2 (ICC: 0.95 and 0.96, respectively).

Hypothesis 1(a): The CISP would demonstrate reliability

Hypothesis 1(b): A five-sprint familiarisation overcomes a CISP learning effect

Based on data from this study, hypotheses one (a) and (b) were both accepted and the CISP was used for the remainder of this thesis.

Study 2; Chapter V The experiment reported in chapter V aimed to discover the time course of heat exchange between the peripheral shell through to whole body core compartments during a single bout of leg cooling. Thigh cooling for 25 minutes caused reductions in Tskin and Tmu compared to a no cooling control ($P < 0.01$). However, Tre remained unchanged following the cooling bout. Tskin and Tmu remained unchanged after
14 minutes of cooling. Reductions in Tskin were larger and occurred at a faster rate than Tmu, however significant relationships between the two were still observed ($r = 0.974, P < 0.01$).

Hypothesis 2 (a): Cooling would cause reductions in Tskin, Tmu and Tre

Hypothesis 2 (b): Interacting relationships would be observed between the temperature measurement locations

Data from this study demonstrated that peripheral cooling did not cause consequent reductions in core temperature. Without a change in core temperature no significant relationships were possible between Tre and Tskin or Tmu. Therefore hypotheses 2 (a) and (b) could only be accepted in part.

**Study 3; Chapter VI** Following on from chapter V, the study in chapter VI aimed to determine if the thermal responses to cooling transferred to a positive ergogen on intermittent sprint performance. Participants were cooled for durations of 10, 15, 20 and 25 minutes as well as a no cooling control prior to the completion of the CISP in hot, humid conditions. Tre remained unchanged throughout all cooling periods, yet increased at a slower rate in three of the cooling conditions compared to the no cooling control. Tmu was significantly lower in only the 15 minute cooling condition ($P < 0.05$). Cooling for 15 minutes produced the largest improvements in PPO and WD, by 3.75 % and 6.7 % respectively and it was suggested that this was due to a greater suppressive response of physiological and perceived measures to exertional heat stress. However, decrements in performance were observed after 25 minutes of cooling, while no differences were observed following 10 minutes of cooling.

Hypothesis 3 (a): Longer durations of cooling will cause the largest changes in Tre and Tmu

Hypothesis 3 (b): Longer durations of cooling will display a positive duration-dependant response in performance

Data from this study demonstrated that longer durations did not elicit the greatest changes in thermoregulatory measures, nor in performance improvements. Without these progressive duration-dependent responses, hypothesis 3 (a) and (b) were rejected.
Study 4; Chapter VII  

It was suggested in chapter VI that a certain level of reduction in Tmu may enhance afferent feedback and delay the central regulation response to hyperthermia, consequently improving performance, yet the difference between cooling being ergogenic and ergolytic may be small. Measurements of muscle temperature are not always possible to determine this and so the aim of the study reported in chapter VII was to test the accuracy of an equation to predict Tmu from Tskin, used within the literature. Tskin data from chapters V and VI were put into the equation and showed that predicted Tmu was underestimated compared to measured Tmu. This effect was exacerbated under cold conditions, with a difference of up to 11 °C being observed.

Hypothesis 4: *The Tmu prediction equation (de Ruiter et al, 1999) would be inaccurate*

Data from this study demonstrated the inaccuracy of the equation proposed by de Ruiter et al (1999), particularly when the muscle is subjected to the cold and therefore hypothesis 4 was accepted. A new equation based on more data points, spanning a larger range of temperatures, and from a larger sample size, was proposed.
8.2. Mechanisms associated with intermittent sprint cycling in a hot environment

8.2.1. The CISP

The prevalence of intermittent sprint exercise within the literature has increased within the last decade due to its ability to replicate activity patterns similar to that of team games. Typically, intermittent sprint exercise consists of a maximal sprinting bout, a low intensity recovery bout and a rest period. Nevertheless due to the unpredictable nature of team games, the durations of each phase and the intensity of the workload performed are hard to define. It has been reported that the number of sprints in a game can be between 20 and 40, each lasting an average of three, but a maximum of five seconds, with recovery periods lasting up to 300 seconds (Serpiello et al, 2011). Therefore the design of intermittent sprint protocols should consider this. Many of the protocols to date are running based; the Bangsbo Sprint Test, the Loughborough Intermittent Shuttle Test, the Soccer Specific Test Exercise Test (Bangsbo, 1994; Nicholas et al, 2000, Oliver et al, 2007). As ecological validity is high it is difficult for accurate physiological and performance based measures to be recorded. The importance of these measures is exemplified when trying to determine the mechanisms associated with heat strain and fatigue in intermittent exercise, particularly as the development of ergogens to combat the negative effects of heat stress rely on, and in some cases assume, the reliability and validity to be high. Cycling based protocols allow for control of physiological measurement as well as accurate data collection and the ability to set a specific workload during each phase. The sprint and recovery phases of the CISP are similar to that suggested by Serpiello et al (2011), while its duration replicates that of one half of a team game. Castle et al (2006) investigated the effects of different methods of pre-cooling on intermittent sprint performance in hot humid conditions using the CISP. Each method of pre-cooling elicited different performance outcomes; ice vest resulted in greater PPO than the control for the last 8 minutes of the CISP, cooling via water immersion resulted in lower PPO than the control for the first two sprints and cooling via ice packs on the thighs resulted in an overall improvement in PPO of ~ 4 % compared to the control. Previously, McGawley and Bishop (2006) reported TEM scores for sprint cycling PPO to be 2.8 % while TEM scores of nearly 8 % have been reported following running based protocols assessing repeated sprint ability (Oliver et al, 2007), presenting the question as to whether the performance improvements reported by Castle et al (2006) were meaningful. Despite the slight bias of 14 W towards a second trial and variation of 147 W seen within the 95 % limits of agreement, there were no
differences between CISP trials and TEM scores for PPO were only 2.9 % across 20 sprints (chapter IV). The benefit of using the CISP is the greater association to games play, particularly reflective in its duration, unlike other repeated sprint protocols with durations less than 5 minutes. To the authors’ knowledge, there is no other cycling based protocol of similar nature to the CISP within the literature that has been tested for reliability. Furthermore, the low TEM scores in comparison to cycling protocols of shorter duration reiterate its value. Watt et al (2002) reported TEM scores of 2.5 % between trials however a 30 seconds sprint is not reflective of games play activity (Serpiello et al, 2011) and so ecological validity is low. Running based protocols have been shown to be highly reliable when total sprint time has been observed (Table 8.1), however decrements in sprint time as a performance measure vastly reduce this reliability due to the lack of control of exercise intensity during each phase of the protocol. The CISP allows for the control of 96 % of exercise intensity, with only the 20 x 5 second maximal sprint efforts determined by the participant across 40 minutes of exercise. Therefore the use of the CISP for interventions in intermittent sprint activity is reliable and so it can be stated that the outcomes seen by Castle et al (2006) will have been due to the pre-cooling interventions rather than protocol test-retest variation. This can also be said of the duration-response to cooling observed in chapter VI. While the level of heat strain may be determined by individual difference (Marino et al, 2004), the same has only been hypothesised for pre-cooling response (Castle et al, 2011). However, the reliability of the CISP protocol now allows for a more accurate investigation of these hypotheses as any differences can be attributed to experimental design and not the protocol used.
Table 8.1. A comparison of studies assessing the reliability of repeated-sprint cycling using various protocols, with peak power output (PPO), mean power output (MPO) and work done (WD) outcomes documented.

<table>
<thead>
<tr>
<th>AUTHOR</th>
<th>YEAR</th>
<th>PERFORMANCE MEASURE</th>
<th>PROTOCOL</th>
<th>RELIABILITY SCORE (%)</th>
</tr>
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<tr>
<td>Spencer et al.</td>
<td>2006</td>
<td>Total sprint time</td>
<td>Running: 6 x 30m sprints</td>
<td>0.7 (TEM)</td>
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<tr>
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<td>2010</td>
<td>Total sprint time</td>
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<td>2013</td>
<td>PPO (sprint 1 only)</td>
<td>Cycling: Repeated CISP</td>
<td>2.5 (TEM)</td>
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<tr>
<td>Watt et al.</td>
<td>2002</td>
<td>PPO (between trials)</td>
<td>Cycling: 2 x 30s Wingate</td>
<td>2.5 (TEM)</td>
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<td>McGawley &amp; Bishop</td>
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<td>Mendez-Villanueva et al.</td>
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<td>WD (between trials)</td>
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<td>Running: 6 x 20m sprints</td>
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*TEM: typical error of the measure. CV: coefficient of variation*
Nevertheless, the CISP only replicates that of one half of a team game. Edwards (1982) defines fatigue as the failure to maintain the required or expected force. Mohr et al (2003) reported up to 45% less high-intensity running during the last 15 minutes of match performance compared to the first 15 minutes with the majority of fatigue occurring at the end of the match. Research within our laboratories has assessed the effect of interventions prior to completion of the CISP replicating that of a full match rather than only one half, in addition to using the CISP either side of a half-time intervention. However, the reliability of the CISP for this duration is unknown. It may be that the demands of the CISP across 80 minutes, particularly under climatic heat stress, causes greater variations in PPO or MPO due to an earlier onset and thus increased influence of fatigue. Therefore, future research should test the reliability of the CISP over the duration of two halves of a team game before half-time interventions are employed.

8.2.2. The kinetics of heat exchange following pre-cooling and subsequent intermittent sprint performance

The effect of heat stress on performance has been shown to be exacerbated during intermittent sprint exercise (Kranning and Gonzalez, 1991). Castle et al (2004) correlated reduced PPO with increased physiological strain (PSI) during a CISP. Originally, it was thought that by reducing core temperature prior to performance allowed for a greater amount of work prior to critical body temperature being reached (Marino, 2002; Quod, 2006). Given the complex evolution of humans and the ideology that there is not just one key variable to fatigue, the critical core temperature hypothesis (see chapter II) is now less supported within the literature. Despite this pre-cooling is still used. It is suggested that pre-cooling causes a heat sink for absorption of environmental and metabolic heat without changing the rate of heat gain (White et al, 2002) although the exact mechanisms behind this are still unknown. Reductions in core temperature are frequently reported following cooling and attributed to improved performance through protection of neuromuscular pathways and possible central regulation (Castle et al, 2006; Skein et al, 2012). Castle et al (2006) suggest that pre-cooling offsets the down-regulation of motor units associated with rises in core temperature and consequent reductions in central activation (Noakes et al, 2001). In their study, they reported reductions of 0.24 °C in core temperature following 20 minutes of cooling using ice packs. The study reported in chapter five observed the thermoregulatory responses to 25 minutes of
leg cooling at rest and there were no changes in core temperature evident. It has been suggested that the reductions in core temperature can occur upon removal of the stressor during a warming phase (Webb, 1986; Romett, 1988). This was also the case with Castle et al (2006) where further reductions were observed. However, no changes in core temperature were observed during cooling, the warm up or at any point during the CISP in chapter VI either, yet PPO and MPO improvements of up to 3.75 % and 6.7 %, respectively, were still observed. Nevertheless, the rate of core temperature increase during the CISP was slower in three of the cooling conditions compared to the control contradicting that of White et al (2002). The use of ice packs could be considered an aggressive cooling method, demonstrated in the rapid temperature reductions in skin and muscle temperature in chapter V. This may have induced severe vasoconstriction of the periphery preventing any changes in core temperature. Blood flow was not measured at any point during this thesis, but would provide a good insight as to the peripheral and core vasomotor responses to cooling. Likewise, it could be speculated that upon the removal of the stressor the rate of metabolic heat production from the warm up and CISP is equal to the rate of cold blood distribution from the periphery back to the core. This paradoxical vasodilation may therefore cause a delay in the rate of core temperature rise throughout the CISP, prolonging the time to a limiting body temperature and central activation failure associated with hyperthermia (Noakes et al, 2001; Todd et al, 2005). Peripheral blood flow measures, using Laser Doppler Flowmetry (Nilsson, 1980; Hamada et al, 2006), would provide a further insight into the thermal pathways associated with kinetics of heat exchange in pre-cooling research and identify if the occurrence of a hunting reaction or cold-induced vasodilatory response, as discussed in chapter VI, truly exists in intermittent sprint activity.

Alternatively, leg cooling could create a heat sink for increased metabolic and environmental heat absorption through reduced peripheral temperatures rather than through reduced core temperature. Improved performance has been demonstrated following cooling without concomitant reductions in core temperature (Kay et al, 1999; Schlader et al, 2011). Kay et al (1999) state that reduced skin temperature increases the body’s capacity to store heat due to an increased thermal gradient between the core and the shell. The heat capacity of muscle and the skin have been reported to be very similar (Geddes and Baker, 1967) and, although they did not measure muscle temperature, it could be the reduction in muscle tissue temperature that allows for a heat sink to occur. Given that 40 % of body mass is muscle mass in comparison to 5 % skin mass (Wang et al, 1992), the muscle tissue can be considered an
important factor in the pre-cooling mechanism. Similarly, Bogerd et al (2010) reported improved performance despite no change in core temperature following two intensities of cooling. Cooling intensity was controlled using an evaporative cooling shirt or ice vest, providing mild or strong cooling, respectively. Although the technique of cooling was different to that used within this thesis, strong cooling caused a severe vasoconstriction response compared to mild cooling. Skin blood flow was similar between cooling conditions, however reductions in body heat content were twice as large following strong cooling and attributed to improved performance, supporting the use of an aggressive cooling technique for increased heat sink, as observed in chapter VI.

In the experiment reported in chapter V both skin and muscle temperature reduced significantly from resting values. Furthermore, although not significant, large reductions in muscle temperature were apparent in chapter VI, with improved performance reported. Elevations in muscle temperature have been associated with reduced neuromuscular drive to the active musculature (Drust et al, 2005) and so a lower initial muscle temperature may also contribute to sustained central activation.

Body heat content has been suggested to mediate exercise intensity during fixed rate cycling (Tucker et al, 2006). Tucker et al (2006) noted that higher skin temperatures during the early phases of performance resulted in a quicker decline in power output even though core temperature was not at critical levels, suggesting the influence of anticipatory regulation. Castle et al (2006) suggest this may have been present during the CISP due to the rise in PPO towards the end of the protocol, a trend also seen in chapter VI. Tucker et al (2006) stated that higher levels of body heat content at the start of exercise led to a down-regulation of exercise intensity through increases in feelings of physical exertion so that the onset of hyperthermia was unlikely to occur. This idea presents fatigue as a psychological component, or emotion, rather than a depletion of physiological mechanisms, a concept heavily supported by Noakes (2012). It is also suggested that initial exercise intensity is pre-selected in a feed-forward manner, with all exercise, including maximal sprint exercise, proposed to be sub-maximal (Noakes, 2012). St. Clair-Gibson et al (2001) suggest that a muscle reserve must exist and is maintained through intrinsic efferent inhibitory pathways. Tucker et al (2006) state that exercise intensity is continuously regulated, with feed-forward input determining initial exercise intensity and afferent signals from thermoreceptors providing feedback to modify intensity accordingly. Noakes (2012) proposes The Central Governor Model of
Exercise Regulation, a development of Ulmer’s (1996) model of anticipatory regulation to try to explain performance outcomes. Within this model, numerous variables contribute to exercise regulation, with psychological and physiological components providing afferent feedback to the brain. During prolonged repeated sprint exercise, such as the CISP in hot conditions with no intervention present, heart rate, core temperature, physiological strain and perceived exertion all increase as exercise progresses, while reductions in PPO are observed (Castle et al, 2006). Pre-cooling can therefore be suggested to manipulate the physiological components of the model through inhibiting this afferent feedback, delaying the influence of central governor regulation allowing for improved performance throughout the protocol. Castle et al (2006) suggest that pre-cooling may, subconsciously, offset the down-regulation of motor units associated with pacing in a preventative mechanism against hyperthermia, therefore maintaining exercise intensity and prolonging performance.

The effect of pre-cooling on body heat content was discussed in chapter VI, with 15 minutes of cooling eliciting the greatest reductions in body heat content and greatest improvements on subsequent performance. There is much debate about the accuracy of body heat content as a tool to represent the level heat stress, as it is not conducive to deep tissue temperatures (Jay and Kenny, 2007). The incorporation of skin and core temperature (two-compartment model), as well as body mass and heat capacity of tissue gives an image of the thermal status of the whole body. However, Vallerand et al (1992) states that the use of body heat content is difficult to justify until the inclusion of varying depths of body temperature are measured. A three-compartment model, including a muscle temperature component, has been seen to estimate body heat content significantly better than that of the two-compartment model (Jay et al, 2007), however it was concluded that “the invasive measurement of intramuscular temperature is not a practical option for researchers” (Jay et al, 2007). Therefore, the equation proposed in chapter VII could be used in this instance, although continued development to improve the accuracy of a three-compartment model is still required.

Interestingly, the thermoregulatory responses to cooling seen in chapter V transposed to performance benefits (observed in chapter VI). Tskin and Tmu lowered to a plateau after 12 and 14 minutes of cooling respectively, with no other observed responses despite continued cooling. In chapter VI, 15 minutes of cooling elicited the greatest performance improvements in both PPO and WD alike, with further cooling detrimental. Pack temperature continued to rise after 15 minutes without continued reductions in peripheral temperature, demonstrating
that skin and muscle temperature may have a unique temperature range. Skin blood flow has been shown to reduce to minimal levels following local cooling of the skin (Charkoudian, 2003) with prolonged cold exposure inducing responses to prevent the onset of hypothermia (Stocks et al, 2004), perhaps causing cooling to become ergolytic. Chapter VI briefly discussed the possible influence of hyperalgesia and nociception to the cold and within the limb. Cooling of the skin has been shown to reduce muscle oxygen levels and muscle acidosis (Hom et al, 2004). Muscle acidosis can increase following reductions in muscle temperature, consequently reducing isometric force production in the cooler muscles (Westerblad et al, 1997). Although not measured, this may help explain some of the participants’ feelings of heavy or achy limbs and thus lower initial power outputs in all cooling conditions compared to the control. Furthermore, this could explain the lower power output and earlier onset of fatigue in the 25 minute cooling condition. Therefore, 15 minutes of cooling could evoke the optimal pre-cooling response, with reduced skin and muscle temperature providing the greatest heat sink and largest thermal gradient between the core and the shell. Moreover, this demonstrates the necessity for blood markers and blood flow measures in understanding the mechanisms behind pre-cooling, as well as presenting the questionable effect of pre-cooling on muscle pH, and its influence on intermittent sprint performance.

Of particular interest is the reduction in thermal sensation during cooling despite no changes in core temperature and so the concept of local thermal sensation was discussed in chapter VI. During cold acclimation, habituation, or numbing of the stressor, has been seen to differ between individuals (Armstrong, 2000). Similar thermal sensation responses to cooling were observed in chapters V and VI with initial application displaying the lowest thermal sensation scores. Combined with reduced skin and muscle temperature, lower thermal sensation scores could contribute to the inhibition of afferent feedback as suggested by Noakes (2012). Notwithstanding, an interesting argument is presented when performance measures are considered, as reduced performance was observed in the earlier stages of the CISP. Further research is required assessing the influence of thermal sensation as a regulator of exercise intensity and the possible effect of nociception on selection of initial intensity. Additionally, the validity of the thermal sensation scale in a systemic manner during local cooling should be reviewed given that it is not reflected of local changes in temperature.
Reduced performance has been attributed to both limited and excessive cooling (Figure 8.1). Cheung and Robinson (2003) cooled participants until core temperature reduced by 0.5 °C, or for a maximum duration of 75 minutes, whichever occurred first and reported a negative effect on both PPO and MPO. Sleivert et al (2001) cooled the torso and the legs and suggested that power generation in sprint performance may have been effected by metabolic mechanisms given that metabolism is altered in cold muscle. Muscle metabolism is not affected in heat stress (Maxwell et al, 1996; Marino et al, 2004; Castle et al, 2006) and so the decline in PPO and WD in longer cooling durations may be as a result of this, however further research is required in this area.
Figure 8.1. Calculated percentage change in performance in studies where pre-cooling prior to intermittent sprint exercise has been used. *denotes that a significance was found compared to a control. The top section signifies studies where performance is represented by distance covered (dotted bars). The bottom section signifies studies where performance is represented power output (PPO solid bars, WD striped bars). ISSR: intermittent sprint shuttle test; ISP: intermittent sprint protocol; ISEP: intermittent sprint exercise test; CISP: cycling intermittent sprint protocol; IET: intermittent exercise test. Error bars represent TEM determined in chapter IV for the CISP for PPO and WD.

Conversely, as discussed in chapter VI, cooling for shorter durations does not appear long enough to provide any ergogenic benefit. It appears that cooling the legs using ice packs (Castle et al, 2006) for 15 minutes (chapter VI) is the most ergogenic method for significant improvements in peak power output and work done (Figure 8.1). A significant improvement
of ~4% in PPO and ~7% in WD was observed following 15 minutes of cooling, respectively. When the percentage error of the protocol is considered (PPO: 2.9%, WD: 4.2%), an improvement of 1% and 2.5% is still apparent. At elite level the margins of difference are minute (Hopkins, 2005). In the context of the field of research, where skill level and participant variability can be considered constant, and the error of the protocol (incorporating the nature of the protocol and the equipment used) considered, an overall improvement as small as 1% in PPO or 2.5% in WD can be considered meaningful as it can be the difference between winning and losing. From the contained data within this thesis, the peak scores across controlled CISP trials were ranked, with the mean difference between ranking calculated as 26 W. This value can be considered an absolute score of meaningful change following an intervention. An ~4% improvement in PPO within the 15 minute condition is the equivalent of 40 W. This is similar to the improvements in the CISP observed by Castle et al (2006) following pre-cooling, where a 49 W difference between intervention and control was observed. However, body mass has been shown to affect the level of heat strain experienced (Marino et al, 2000; Marino et al, 2004) and thus may influence the pre-cooling response. Marino et al (2004) reported that individuals with a larger body mass produced heat at a faster rate consequently causing an earlier onset of reduced exercise intensity. Castle et al (2011) noted different responses to an identical cooling method used in their 2006 study and suggested body mass to be a factor, with cooling potentially more beneficial to heavier athletes. The effect of body mass on core temperature response during exercise was discussed in chapter VI, however it may be plausible that a pre-cooling response may also be influenced by body mass. Despite body mass and sum of skinfolds differences of participants used in chapters V and VI being 1.2 ± 2.4 kg and 7.1 ± 6.1 mm, respectively, muscle temperature reductions differed by 6.9 ± 0.4 °C, despite the same method of cooling used. This suggests that limb composition rather than body mass may affect individual response to cooling. The thermal conductivity of fat is slower than that of muscle (Herman, 2007) and so it would be expected that muscle temperature would be lower in the presence of a lower body fat following cooling. It may be that leaner individuals have a greater extent of capillarisation within the limb allowing for better transfer of heat away from the muscle, limiting the amount of temperature reduction. However this is speculative and would require body composition analysis to be completed before conclusions drawn. Nonetheless, the individual response to cooling should be researched given the differences in temperature response seen in chapter V, allowing for the development of individual prescription of pre-cooling. Furthermore, the concept of a dose response is now being researched (Janwantanakul, 2009; Bogerd et al, 2011).
2010; Minett et al, 2012), with this thesis providing evidence for a more specific duration-dependent response to cooling for practical application, but it could be hypothesised that the responses to cooling are dependent on the individual rather than the dose itself. Moreover, the effect of cold acclimation status may have a strong influence on those using pre-cooling on a frequent basis given the metabolic and insulative responses to repeated cold stress (Young, 1986; Armstrong, 2000) and possible cold-induced vasodilatory responses that may exist.

8.2.3. Progressions in Pre-Cooling Research - The Synergy of Pre-cooling and Acclimation/Acclimatisation

Acclimation or acclimatisation is another technique that has been used for centuries and involves complex adaptations to a particular stressor. Although similar, the terms are not inter-changeable due to the conditions upon which each is achieved; the former within laboratory conditions simulating an environment and the latter in the natural environment (Armstrong, 2000). To achieve the greatest level of adaptive response, a period of up to 14 days may be required and in the sporting environment athletes commonly travel to the environment they are competing prior to the start of competition to allow for some of these responses to occur. It has been shown that acclimation/acclimatisation improves performance (Castle et al, 2011) with a number of possible reasons having been proposed. Increased sweat response (Nielsen et al, 1997; Marshall et al, 2007; Buono et al, 2009), increased plasma volume (Armstrong and Maresh, 1991; Nielsen et al, 1997) and a reduced cardiovascular strain (Armstrong and Maresh, 1991; Nielsen et al, 1997) have all been reported as a result of acclimation. However whether acclimation and pre-cooling can coincide effectively can be questioned. Castle et al (2011) investigated the combined effect of pre-cooling and 10 days of heat acclimation on intermittent sprint exercise performance in the heat. Eight moderately trained male team sport players of county or university first team standard completed three intermittent sprint protocols (as described by Castle et al, 2006); one in a temperate environment (control) and two in a hot environment of which one had 20 minutes of leg cooling, before undergoing 10 days of heat acclimation; cycling at 80 revs.min⁻¹ for 1 hour against a resistance eliciting 50 % \( \dot{V}O_{2\text{peak}} \). The negative effects of heat on performance were observed during the pre-acclimation trials, with reductions in peak power output in the hot condition without cooling compared to the temperate and cooling pre-acclimation visits. Prior to acclimation pre-cooling improved the negative correlation between peak power output and
physiological strain seen in the pre-hot condition. However, acclimation achieved the same result and improved peak power output by 2% in the hot condition without any pre-cooling supplement, and the addition of pre-cooling did not yield any addition physiological, perceptual or performance related benefits. Data from this study demonstrate the benefits of pre-cooling as an acute ergogen compared to chronic adaptations that may be observed following acclimation. As observed in chapter VI, the time required for this ergogen to elicit similar benefits as observed by Castle et al (2011) is considerably less. It may be that pre-cooling is a more cost effective technique that can be used by sports teams and athletes alike.
8.2.4. Summary of future directions within this thesis

During the discussion of all study chapters, a number of questions have been raised that could be answered with future research, the most relevant of which that could contribute to the findings obtained within this thesis are listed below:

- To measure blood flow of the upper leg musculature using Laser Doppler Flowmetry during a single pre-cooling bout, warm up and CISP providing further explanation of the CIVD responses.
- To measure the influence of cooling on central motor drive, muscle activation and muscle metabolism and the possibility of reduced nerve conduction velocity, motor recruitment and the onset of muscle acidosis.
- To measure the effect of cooling on catecholamine response and whether this can be associated with improvements in intermittent sprint performance.
- To try to clarify the individual responses to cooling, specifically to determine the importance of Tcore, Tskin and Tmu in responders and non-responders and their relationship with more specific demographic characteristics such as body fat percentage or muscle mass to fat mass ratios.
- To further develop the Tmu prediction presented in chapter VII via multiple regression analysis, inclusive of key variables such as skinfold thickness and body mass.
- To measure the effects of repeated cold exposure on vasodilation and vasoconstriction response and subsequent intermittent sprint performance.
- Surprisingly, very little pre-cooling research focuses on the female population despite the number of female athletes competing at elite level and so it would be interesting to a similar pre-cooling response occurs.
8.2.5 Limitations to procedures used within this thesis

Within this thesis, methodology and procedures have followed that of previous related literature. Where it was felt necessary, a justification has been given providing the reasons why certain methodology was used. Below outlines any limitations that may have arisen from such methodology, with suggestions as to how these may be overcome in future research within the area:

- Sum of Skinfolds has been calculated using a 4-site method (Durnin and Womersley, 1974). However, to improve the accuracy of body composition analysis, particularly given the focus on the lower musculature in individual study chapters, a 7-site method would provide a better picture of overall body demographic.

- Measurement of thigh circumference was only recorded in chapter V (study 2) however, in a similar instance as sum of skinfold, measuring thigh circumference in other studies, in particular chapter VI (study 3), would have allowed for further support when explaining individual responses to cooling.

- Muscle temperature in chapter VI (study 3) was measured at three time points; at rest, post-cooling and post-performance. It would have been useful to record muscle temperature at time points during performance, although this may be somewhat impractical due continuous nature of the protocol. Nevertheless, it is an aspect that should be considered in future studies, with protocol revised to incorporate time periods for these measures. This would allow for additional assessment of the pathways involved in heat exchange between the periphery and core following pre-cooling during subsequent exercise. Furthermore, it would allow for improved practical application in the negation of heat stress during exercise, particularly if there existed any specific time periods of muscle temperature rise during exercise.

- Muscle temperature could have been measured post-warm up (chapter V) at the same time as other physiological and perceptual measures. This would have offered additional information regarding the influence of cooling on performance, and provided a more accurate measure of Tmu prior to the performance task than the measure taken post-cooling/pre-warm up. Future studies should incorporate this into their protocols.
8.3. Practical Implications

The results reported in chapter IV are more beneficial for laboratory rather than field-based application. The proven reliability of the CISP now allows for accurate conclusions to be drawn following the examination of different interventions on intermittent sprint exercise, without the protocol being considered a limiting variable. Furthermore, the duration and mixture of intensities in the CISP replicates activity patterns typical of one half of a team sport, allowing for practical application, following interventions, to be made with confidence.

Chapter V demonstrated the possible existence of a temperature range response within the skin, and more so, the muscle, with a plateau occurring following reductions due to cooling. This may be of practical importance, particularly as the intensity of a cooling bout has been suggested as a possible explanation to the differing performance outcomes within the literature (Bogerd et al, 2010). The knowledge of skin and muscle temperature responses can allow for future researchers, coaches and athletes alike, to prescribe a specific duration of cooling depending on the desired thermoregulatory outcomes. Furthermore, the results from this study have demonstrated that not all pre-cooling methods elicit reductions in core temperature and it may be peripheral changes that contribute to the heat sink associated with the ergogen.

Similarly, the study in chapter VI confirmed that a core temperature reduction is not required for improvements in performance. Leg cooling for 15 minutes elicited the greatest PPO and WD improvements without concomitant reductions in core temperature. Therefore, reductions in skin and deep tissue temperature of the periphery should be considered as practical ways to reduce body heat content thus increasing the heat sink required for sustained intermittent sprint performance in the heat. Furthermore, cooling for long durations becomes detrimental. This has practical application for use of leg cooling in the field demonstrating that excessive cooling durations that may reduce thermal comfort, not be particularly cost effective and are incredibly time consuming, appear unnecessary. Prescribing pre-cooling via temperature reductions may not be feasible in the field due to differences in individual temperature-duration response and the limited time immediately prior to competition. An accurate duration of pre-cooling, using ice packs to the legs, can be used as a generic prescription for sports teams competing in hot humid environments until further knowledge about individual pre-cooling responses is known.
Finally, the practical applications of the experiment in *chapter VII* allow researchers and athletes to monitor muscle temperature without the need of invasive procedures. This can be used for pre-cooling dose when peripheral temperature change is used as the desired heat sink, although more research is required assessing the effect of absolute muscle temperature change on intermittent sprint performance. Additionally, the advantages of calculating muscle temperature without invasive procedures allows for the continued development in thermometry of a three compartment model and calculations of body heat content inclusive of deep tissue temperatures.
8.4. Conclusion

Fatigue is a multi-dimensional concept and it is unlikely that cessation of exercise is a result of only one phenomenon. It is well documented that physiological strain is exacerbated following intermittent sprint exercise in the heat, with many researchers’ continually developing ways to alleviate heat stress. Traditionally, it was thought that reductions in core temperature were required for a “heat sink” to occur to maintain performance and delay the negative effects of heat strain. The concept of reduced core temperature derived from the critical core temperature hypothesis, which has since been seen to be inaccurate, yet core temperature has remained the primary variable to be manipulated. With new ideas about central regulation and the causes of fatigue constantly being proposed, it may be difficult to pinpoint the main mechanisms that influence cooling.

What has been illustrated in this thesis is that reductions in core temperature are not vital when employing the pre-cooling ergogen as a method to limit heat strain. Manipulation of peripheral temperatures, such as reduced skin and muscle temperature, can provide different thermoregulatory responses whilst yielding similar performance improvements. The required heat sink prior to exercise can therefore be associated with reductions in body heat content rather than just core temperature, as it incorporates both core and peripheral temperatures. Considering this, pre-cooling techniques should look to manipulate multiple components of thermometry rather than just one.

This thesis has proven the reliability of an intermittent sprint protocol with a duration reflective of one half of a team game. Furthermore, this thesis has supported that pre-cooling, specifically using ice packs on the legs, can improve intermittent sprint performance in hot humid conditions. The novel aspect of this thesis is that a duration-dependant response to leg cooling exists, and this should be considered by British sports teams using the ergogen when competing in hot, humid conditions. Yet the ideal dose; that is the volume of the limb covered, the temperature of the cooling ergogen and perhaps the quantity of ice packs used, should still be researched to allow for an optimal duration-dependent and dose response to cooling to be discovered. Finally, a non-invasive technique for calculating muscle temperature has been suggested that can be used by sport scientists interested in peripheral thermo-physiology and heat stress. The next steps in pre-cooling research are to test the ergogen in field-based settings to determine if the laboratory-based performance benefits that have been observed transfer into the competitive environment (Duffield et al, 2013).
CHAPTER NINE

References
CHAPTER IX: REFERENCES


Noakes, T.D. (2012). Fatigue is a brain-derived emotion that regulates the exercise behaviour to ensure the protection of whole body homeostasis. *Frontiers in Physiology, 3*: article 82.


CHAPTER TEN

Appendices
CHAPTER X: APPENDICES

Appendix A

A1. Further Health and Safety Information

A2. Example of a participant information sheet

A3. Informed Consent and Medical Questionnaire

A4. Participant Declaration

Appendix B

B1. Rating of Perceived Exertion

B2. Thermal Sensation Scale
APPENDIX A1.  HEALTH AND SAFETY INFORMATION

All laboratories used had prior risk assessments conducted and all electrical equipment checked for safe use. All equipment was the property of the University of Brighton and reusable equipment was thoroughly cleaned prior to each use. Mouthpieces, nose clips, and Falconian tubing used for ventilatory data collection were cleaned using Virkon Disinfectant. Heart rate monitors (Polar Instruments, Polar Electro, Finland) and thermal measuring equipment was washed in warm water prior to and after each use. Rectal thermistors were used to measure core body temperature and protective gloves used where necessary. Equipment used that had risk of cross contamination (needles and sharps, blood taking equipment and gloves) were disposed of in the relevant biohazard waste bin. Any reusable invasive thermistors were cleaned immediately after use in an autoclave (Astell, Swiftlock Secure-Touch +, benchtop, UK).

Before any testing was conducted, experimenters washed their hands with antibacterial gel and where invasive procedures were conducted, prior inoculations were administered.

Termination of any testing occurred if:

- the participants completed the exercise protocol
- requested to due to physiological or psychological discomfort
- the experimenter deemed the participant was showing signs of heat illness, discomfort or sickness
- the experimenter saw physiological reasons for termination; rectal temperature reductions of greater than 1.5 °C or increases beyond the ethically approved temperatures of 39.7 °C.
APPENDIX A2.

UNIVERSITY OF BRIGHTON
CHELSEA SCHOOL

PARTICIPANT INFORMATION SHEET

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Dr. Nick Webborn (Sportswise Ltd. The Sussex Centre for Sports Medicine)
Dr. Paul Castle (University of Bedfordshire)

PROPOSED RESEARCH QUESTION

Is there a duration-dependent response to isolated leg cooling for intermittent-sprint exercise in hot, humid conditions?

BACKGROUND TO THE PROJECT:
Hot and humid environmental conditions are an on-going concern for sports competitors. Previous research indicates the detrimental effects of heat on an individuals’ performance (Hargreaves and Febbraio, 1998), especially if acclimation has not previously occurred. Alleviating heat strain and the positive effects this may have on athletic performance has been a continuous topic of interest for scientists and with the demands of success continuously growing in the athletic scene, many athletes are being pushed to repeatedly perform short yet maximal sprint bouts with longer submaximal recovery, a type of activity characterised as prolonged, high intensity intermittent exercise (Sirotic & Coutts, 2007). Due to the mentality of athletes striving for success, techniques have long been used to reduce the strain on the body and prolong optimal performance. Acclimation has been used for over 50 years (Bean and Eichna, 1943; Robinson et al, 1943) yet more recent research has shown a technique known as pre-cooling to be effective (Cotter et al, 2001), of which there are many differing types (Quod et al, 2006). Castle et al (2006) investigated the most effective method of cooling prior to intermittent sprint exercise and found that localised leg cooling increased
peak power output (PPO) by the 4%. However with this method and all the other techniques, the duration of cooling has just been assumed. 20 minutes is frequently used (Castle et al, 2006; Duffield et al, 2003; Mitchell et al, 2003; Cotter et al, 2001) and thought to suffice to gain the ergogenic benefit yet studies still differ in opinion with some studies cooling for as long as 60 or 75 minutes (Booth et al, 1997; Cheung and Robinson, 2004, respectively).

AIMS OF THE STUDY:
The aim of this study is to determine whether there is an optimal time of cooling which can then be used in future pre-cooling studies.

OUTLINE OF THE PROJECT
Prior to testing each participant will complete a maximal oxygen uptake test on a cycle ergometer, plus a familiarisation test. Data from this will be used to determine resistance during the active recovery phase. The subsequent visits will involve the participants completing the cycling intermittent sprint protocol (CISP) following one of four cooling periods using ice packs, placed on the quadriceps (rectus femoris, vastus lateralis, vastus medialis) (Martini, 2004) and the hamstrings (bicep femoris, semimembranosus, semitendinosus) (Martini, 2004), in hot humid conditions. The CISP involves 20 sets of 2 minute periods, consisting of 10 seconds passive rest, 5 seconds maximal sprint effort from a stationary start against a resistance of 7.5 % body weight (BW) followed by 105 seconds of active recovery (35 % VO2 peak), determined from the maximal oxygen uptake test.

This process will be completed by each participant four times; each visit with a differing duration of cooling. Cooling durations will be 10, 15, 20 and 25 minutes, selected in a random order for each participant, as well as completion of a control (without cooling).

Throughout the study, heart rate (HR), rate of perceived exertion (RPE), thermal sensation (TS), core temperature via a rectal thermometer (Tre) and skin temperature (Tsk) via thermistors will be monitored, and muscle temperature (Tmu) will be taken three times (before cooling, after cooling and after the CISP).

PLEASE ENSURE YOU ARE WELL HYDRATED BEFORE PERFORMING ANY TESTING IN THE HOT ENVIRONMENT. Consume plenty of water during the 2 hour period prior to entry to the chamber.
ALCOHOL IS PROHIBITED 24 HOURS PRIOR TO COMPLETING EXERCISE.

POSSIBLE RISKS AND DISCOMFORTS

During pre-testing (visit 1) participants will be required to perform exercise at maximal effort. This will place their body under considerable stress, possibly causing blood pooling to the legs, resulting in possible nausea and feelings of dyspnea. During exercise in the heat, participants will experience increased thirst (Noakes, 2003), sweat production and an increased heart and breathing rate (Wilmore and Costill, 2004). A rectal thermometer will be used (inserted 10cm passed the anal sphincter) so that core temperature can measured at all times. This is both for physiological data purposes and as a safety procedure. Initial insertion may cause some discomfort, however no real discomfort should be felt thereafter. If core temperature rises by more than 2 °C or passes 39.7 °C the participant will be removed from the chamber and testing terminated. There will be equipment designed for rapid body cooling readily available adjacent to the chamber in the unlikely event this should occur. Skin thermistors will be placed on four sites over the body using zinc oxide tape. To prevent ice burn to the legs, the specific regions will be wrapped in a thin cotton cloth. Small discomfort may occur on initial placement but this will be due to unfamiliarity, and will tend only be for a short duration. Muscle temperature will be taken using a muscle temperature probe inserted at a predetermined depth from an ultrasound scan, and will be carried out under local anaesthetic (Emla cream placed on the skin for a minimum of 10 minutes before the procedure is carried out) if required. Some discomfort is expected, with initial insertion feeling like a small scratch to the surface yet once inserted only a feeling pressure on the leg will be felt (similar to pushing your thigh with your fingertip).

I HAVE READ AND UNDERSTOOD THE REQUIREMENTS OF THE STUDY, AND WHERE THERE ARE DOUBTS OR QUESTIONS, I HAVE ASKED THE LEAD EXPERIMENTER THE RELEVANT QUESTIONS TO SEEK THE ANSWERS.

Signed: ____________________  Printed: ____________________  Date: ____________

Experimenter Witness:

Signed: ____________________  Printed: ____________________  Date: ____________

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Physiology Informed Consent and Medical Questionnaire

Name……………………………….
Age…………………………………

Are you in good health?  Yes  No
Please Explain:

How often do you currently participate in vigorous activity?
(e.g. running, cycling, swimming, team sports)
< once per month
  once per month
  1 time per week
  2-3 times per week
  4-5 times per week
  > 5 times per week

Have you suffered from a serious illness or accident?  Yes  No
If yes, please give particulars:

Do you suffer, or have you ever suffered from:

Respiratory problems (e.g. asthma, bronchitis)?  Yes  No
Diabetes?  Yes  No
Epilepsy?  Yes  No
High or low blood pressure?  Yes  No
Cardiovascular problems?  Yes  No

Are you currently taking medication or dietary supplements?  Yes  No
If yes, please give particulars:

In the last 3 months, have you consulted your GP for any condition?  Yes  No

Are you currently taking part (or have recently taken part) in any other laboratory experiment?  Yes  No
PLEASE READ THE FOLLOWING CAREFULLY

Persons will be considered unfit to do the experiment / exercise task if they:

- Have fever, suffer from fainting spells or dizziness;
- Have suspended training due to a joint or muscle injury;
- Have a known history of medical disorders, i.e. high blood pressure, heart or lung disorder;

And, if appropriate to the study design:

- Have had hyper/hypothermia, heat exhaustion, or any other heat or cold disorder;
- Have anaphylactic shock symptoms to needles, probes or other medical-type equipment;
- Have chronic or acute symptoms of gastrointestinal infections (i.e. dysentery, salmonella);
- Have a history of infectious diseases (i.e. HIV, Hepatitis B);
- Have a known history of rectal bleeding, anal fissures, haemorrhoids, or any other condition of the rectum;

DECLARATION

I……………………………………………………………………………………………………………………..hereby volunteer to be a participant in experiments/investigations during the period of:

(month/s) ………………………………………… (year)…………………………

My replies to the above questions are correct to the best of my knowledge and I understand they will be treated with the strictest of confidence. The experimenter has fully informed me of, and I have understood, the purposes of the experiment and the possible risks involved.

I understand that I may withdraw from the experiment at any time and that I am under no obligation to give reasons for withdrawal or to attend again for experimentation.

Furthermore, if I am a student, I am aware that taking part or not taking part will neither be detrimental to, nor further my position as a full time student.

I undertake to obey the laboratory/study regulations and the instructions of the experimenter regarding safety, subject only to my right to withdraw as stated above.

Signature of participant………………………………………………………………………………

Date……………………………………………………………………………………………………

Signature of experimenter………………………………………………………………………………

Date…………………………………………………………………………………………………………………………
APPENDIX A4. PARTICIPANT DECLARATION

NAME:

EXPERIMENTER (S):

1. I have not had any kind of illness or infection in the last 2 weeks
2. I am not taking any form of medication
3. I am not carrying any form of injury
4. I have not eaten within the last 2 hours
5. I have not consumed alcohol in the last 48 hours
6. I have not performed exhaustive exercise in the last 48 hours

I will notify the experimenters if any of the above is in doubt before undergoing any exercise test. Signature for each visit (below) takes into account the above:

Visit 1:
Participant……………………………………………… (date)………………………
Experimenter……………………………………………… (date)………………………

Visit 2:
Participant …………………………………………… (date)…………………………
Experimenter……………………………………………… (date)………………………

Visit 3:
Participant …………………………………………… (date)…………………………
Experimenter……………………………………………… (date)………………………

Visit 4:
Participant …………………………………………… (date)…………………………
Experimenter……………………………………………… (date)………………………

Visit 5:
Participant …………………………………………… (date)…………………………
Experimenter……………………………………………… (date)………………………

Visit 6:
Participant …………………………………………… (date)…………………………
Experimenter……………………………………………… (date)………………………
APPENDIX B1.

Borg’s Rating of Perceived Exertion Scale

6
7 VERY VERY LIGHT
8
9 VERY LIGHT
10
11 FAIRLY LIGHT
12
13 SOMEWHAT HARD
14
15 HARD
16
17 VERY HARD
18
19 VERY VERY HARD
20

(Borg, 1970)
### Thermal Sensation Scale

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<tr>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>1.0</td>
<td>VERY COLD</td>
</tr>
<tr>
<td>1.5</td>
<td></td>
</tr>
<tr>
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<td>COLD</td>
</tr>
<tr>
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</tr>
<tr>
<td>3.0</td>
<td>COOL</td>
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<td></td>
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(Toner et al., 1986)