THE IMPACT OF CLOTHING AND PROTECTIVE GEAR ON BIOPHYSICAL, PHYSIOLOGICAL, PERCEPTUAL AND PERFORMANCE RESPONSES OF RUGBY PLAYERS DURING A SIMULATED RUGBY PROTOCOL

BY

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THESIS

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ABSTRACT

Background: Clothing and protective gear worn during intermittent exercise has shown to increase physiological and perceptual responses, and negatively impact performance capacities, due to increased heat strain, suggested to hasten the onset of fatigue. However, the mechanisms of fatigue experienced in rugby remain unclear. **Objectives:** The aim of this study was establish whether clothing and protective gear worn during a simulated rugby protocol impacts players‘ biophysical, physiological, perceptual and performance responses. **Methods:** 15 registered university and school first XV rugby players with a mean (± SD) age of 20.9 years (± 1.9) volunteered to participate in the study. Testing was performed in a controlled laboratory setting, with temperatures having to be within the range of 16º C-22º C. The mean (± SD) ambient temperature was 17.6º C (± 1.6) for the control condition and 17.3º C (1.5) for the experimental condition. The mean (± SD) relative humidity was 65.2 % (± 9.5) for the control condition and 66.3 % (± 10.0) for the experimental condition. Player‘s performed two protocols of 80-minutes; a control condition (minimal clothing and protective gear) and an experimental condition (full clothing and protective gear). Physiological, perceptual and performance responses were measured at set intervals during the protocol, while biophysical responses were measured pre-, at half-time and post-protocol during a 3-minute EMG treadmill protocol. **Results:** Muscle activity significantly ($p < 0.05$) increased with increasing running speeds. There were no significant ($p > 0.05$) differences for muscle activity between conditions, except for the semitendinosus muscle, which was significantly ($p < 0.05$) higher during the control condition while running at high speeds. Players‘ heart rates, core temperatures and perceptual responses were significantly ($p < 0.05$) higher during the experimental condition, compared to the control condition. Performance responses were significantly ($p < 0.05$) lower during the experimental condition. **Conclusion:** The main driver of physiological and perceptual responses was the exercise itself. However, the additional clothing and protective gear exacerbated the responses, particularly towards the end stages of the protocol. This negatively impacted players‘ performance. Muscle activity appeared to be unaffected by increased body temperatures. However, core temperatures never reached critically high levels during either condition.

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CHAPTER I

INTRODUCTION

BACKGROUND TO THE STUDY

Rugby is a national sport in South Africa and is played at many levels: international level, national level, club level, tertiary level and school level. Since professionalism was adopted by the International Rugby Board (IRB) in 1995 (Garraway *et al*., 2000), many changes have been made to the game (McMillan, 2006; Mellalieu *et al*., 2008). Changes to the rules of rugby have made play more open, faster and more attractive for spectators (Duthie *et al.,* 2003), which has increased the physiological stress experienced by players. Rugby is described as a prolonged, intermittent and highly unpredictable sport with limited information regarding the factors contributing to the development of fatigue (Morris *et al*., 1998; Girard *et al*., 2008). This general lack of research is typical of many intermittent sports and is possibly be due to the current lack of experimental models available to measure such highly unpredictable patterns of activity (Reilly *et al*., 1990).

Despite the apparent evidence of fatigue experienced in rugby, little information exists regarding the mechanisms involved. Recent developments in technology have allowed for the physiological demands of rugby to be accurately quantified and players‘ activity profiles evaluated during match-play using a Global Positioning System (GPS) (Cunniffe *et al*., 2009). Cunniffe *et al*. (2009), using this GPS technology, showed that rugby players total running distances, peak running speeds, number of peak accelerations, average speed, and peak heart rates were all greater during the second half of a match due to players having less rest breaks and more continuous periods of play in the second half. Therefore, the second half of a rugby match is proposed as being more intense than the first (Cunniffe *et al*., 2009), suggesting that fatigue does not occur in a rugby match.

Fatigue is classified as being either central or peripheral in origin (Kay *et al*., 2000). Effects occurring within the motor unit are considered the domain of peripheral fatigue whereas events in the brain and spinal cord are described as central fatigue (Asmussen, 1979). During ongoing dynamic exercise, the differentiation between the two becomes more difficult (Nybo and Secher, 2004). Traditionally, the mechanisms of fatigue experienced during intermittent exercise are suggested to be peripheral in origin (Glaister, 2005), whereas during prolonged, continuous exercise, fatigue is proposed to be caused by central mechanisms (St Clair Gibson and Noakes, 2004), which down-regulates muscle activity as the duration of exercise continues (Gonzalez-Alonso *et al*., 2000; Mohr *et al*., 2003; Rahnama *et al*., 2003). This is based on the central governor theory proposed by Noakes (Noakes, 1996; Noakes, 1998; Noakes, 2000). This ‗central governor' is proposed to prevent any catastrophic failure to vital organs from occurring during exercise (Noakes *et al*., 2004; Lambert *et al*., 2005; St Clair Gibson *et al*., 2001; St Clair Gibson and Noakes, 2004; Tucker *et al*., 2004; Tucker *et al*., 2006). However, the central governor theory has been used to rather tentatively explain fatigue during continuous exercise, and may not be appropriate in describing the onset of fatigue during intermittent exercise, such as rugby. Therefore, gaining insight into the mechanisms of fatigue experienced during rugby is important, as fatigue is identified as one of the primary causes of muscle injuries during match-play (Roig Pull and Ranson, 2007).

During exercise-induced hyperthermia, central mechanisms are proposed to down-regulate the amount of skeletal muscle recruited during exercise, ultimately reducing athletes work rates (Bruck and Olschewski, 1987; Tucker *et al*., 2004). Two different models have been used to try and explain the reduction in performance capacities during exercise in the heat. The first model suggests that during exercise-induced hyperthermia, fatigue occurs when a critical core temperature of approximately 40º C is reached (Bruck and Olschewski, 1987; Cheung and McLellan, 1998; Gonzalez-Alonso *et al*., 1999; Nybo and Nielsen, 2001a). Once this critical temperature is reached, a decrease in central drive occurs, ultimately reducing muscle activity and athletic performance (Walters *et al*., 2000). However, this model is used to explain the decrements in performance during exercise, performed at a constant work rate, and not during self-regulated exercise. The second model suggests during self-

paced exercise, an athlete‘s work is regulated so that this critical temperature is prevented from being reached (Tucker *et al.,* 2004). Therefore, this model proposes that even during mild hyperthermia or heat strain, the central nervous system may be impacted without a critical core temperature of approximately 40º C being reached (Nybo and Nielson, 2001). During heat stress, the cardiovascular and thermoregulatory systems are stressed due to an increased need to pump blood to the exercising muscles and dissipate body heat (Todd *et al*., 2005). This causes the athlete‘s cardiovascular and thermoregulatory responses to become exacerbated and negatively impact performance capacities (Galloway and Maughan, 1997). However, this has largely been examined from an endurance exercise perspective. Many factors have been suggested to cause relative hyperthermia in rugby players (Cohen *et al*., 1981), however it is well known that movement generates the most heat (Parsons, 2000).

Rugby union involves full-body-contact between opponents, and as such, players wear clothing and protective, designed mostly and obviously, for protection from impacts (Finch *et al*., 1999), as well as strength and durability. However, often the designers of protective gear afford little consideration to the thermal load this kit imposes on players (King *et al*., 2002). This has often resulted in protective gear being counterproductive to heat dissipation (Goodman *et al*., 1995; Cheung *et al.*, 2003; Godek *et al*., 2005). When mechanisms of heat dissipation become hindered, body temperatures may become exacerbated during exercise, resulting in critically high levels. Players often do not have a choice as to the type of clothing worn during matches as these are selected and often regulated, by the laws of the game. However, players are able to select what protective gear to wear, as the use of these items has not been made compulsory by the IRB. Despite many scientific studies being conducted on the effectiveness of this gear, much controversy still exists. In fact, it has been suggested that wearing protective gear (mouth guards, shoulder pads and headgear) increases player‘s self-confidence and aggression when going into contact situations, which increases the risk of injuries (Sinclair, 2009). According to Marshall *et al*. (2005), all protective gear, except for mouth guards and headgear, are largely ineffective in preventing serious injuries, which raises questions surrounding whether or not these items should be worn at all. Therefore, the use of

protective gear may only contribute to increasing body temperatures during exercise and provide little protection against the more serious injuries.

Although it has been shown that the locomotor aspects of rugby cause progressive decrements in muscle activity and strength in the muscles of the lower limbs, particularly in the hamstrings (Brown, 2009), this has not yet been quantified relative to increases in body temperature. The additional rugby gear combined with the intermittent exercise may cause player‘s physiological and perceptual responses to become further exacerbated, due to increased heat strain, and negatively impact players‘ performance capacities (Kulka and Kenney, 2002; Cannon and Christie, 2010). However, during exercise-induced heat, the mechanisms behind these performance decrements remain unclear, especially during rugby.

STATEMENT OF THE PROBLEM

Although the importance of investigating the contact nature of the game is acknowledged, the main purpose of this study was to quantify the biophysical, physiological and perceptual changes associated with the intermittent profile of rugby and to determine whether the additional clothing and protective gear worn during an 80-minute (min) simulated rugby protocol causes these responses to become exacerbated and negatively impact players‘ overall performance capacities.

RESEARCH HYPOTHESIS

It was expected that during an intermittent activity profile, representative of rugby-union play, while wearing full rugby clothing and protective gear, the biophysical (muscle activity), physiological (heart rate and core temperature) and perceptual responses (central and local ratings of perceived exertion (RPE)) of players would be higher than when performing the same exercise bout in minimal clothing and no protective gear. Furthermore, the decrements in performance were also expected to be greater while performing the intermittent activity profile in full rugby gear. Lastly, it

was expected that player's biophysical, physiological, perceptual and performance responses would increase over time during both conditions simulated rugby protocols.

STATISTICAL HYPOTHESES

HYPOTHESIS 1: BIOPHYSICAL RESPONSES

Muscular Activity

I). The null hypotheses proposed that there will be no difference in muscle activity between conditions.

Ho: μMAconVM=μMAexpVM
Ha: μMAconVM≠μMAexpVM

Ho: μMAconVL=μMAexpVL
Ha: μMAconVL≠μMAexpVL

Ho: μMAconBF=μMAexpBF
Ha: μMAconBF≠μMAexpBF

Ho: μMAconST=μMAexpST
Ha: μMAconST≠μMAexpST

II). The null hypotheses proposed that there will be no change in muscle activity over time during both conditions.

Ho: $\mu MAconVM_0 = \mu MAexpVM_0 = \mu MAconVM_{40} = \mu MAexpVM_{40} = \mu MAconVM_{80} = \mu MAexpVM_{80}$
Ha: $\mu MAconVM_0 \neq \mu MAexpVM_0 \neq \mu MAconVM_{40} \neq \mu MAexpVM_{40} \neq \mu MAconVM_{80} \neq \mu MAexpVM_{80}$

Ho: $\mu MAconVL_0 = \mu MAexpVL_0 = \mu MAconVL_{40} = \mu MAexpVL_{40} = \mu MAconVL_{80} = \mu MAexpVL_{80}$
Ha: $\mu MAconVL_0 \neq \mu MAexpVL_0 \neq \mu MAconVL_{40} \neq \mu MAexpVL_{40} \neq \mu MAconVL_{80} \neq \mu MAexpVL_{80}$

Ho: $\mu MAconBF_0 = \mu MAexpBF_0 = \mu MAconBF_{40} = \mu MAexpBF_{40} = \mu MAconBF_{80} = \mu MAexpBF_{80}$
Ha: $\mu MAconBF_0 \neq \mu MAexpBF_0 \neq \mu MAconBF_{40} \neq \mu MAexpBF_{40} \neq \mu MAconBF_{80} \neq \mu MAexpBF_{80}$

Ho: $\mu MAconST_0 = \mu MAexpST_0 = \mu MAconST_{40} = \mu MAexpST_{40} = \mu MAconST_{80} = \mu MAexpST_{80}$
Ha: $\mu MAconST_0 \neq \mu MAexpST_0 \neq \mu MAconST_{40} \neq \mu MAexpST_{40} \neq \mu MAconST_{80} \neq \mu MAexpST_{80}$

HYPOTHESIS 2: PHYSIOLOGICAL PARAMETERS

A). Cardiovascular Responses

I). The null hypothesis proposed that there will be no difference in heart rate responses between conditions.

Ho: μHRcon0-80 =μHRexp0-80

Ha: μHRcon0-80 ≠μHRexp0-80

II). The null hypothesis proposed that there will be no change in heart rate responses over time during both conditions.

Ho: μHRcon0&HRexp0 = μHRcon10&HRexp10 = μHRcon20&HRexp20 = μHRcon30&HRexp30 = μHRcon40&HRexp40 = μHRcon50&HRexp50 = μHRcon60&HRexp60 = μHRcon70&HRexp70 = μHRcon80&HRexp80

Ha: μHRcon0&HRexp0 ≠μHRcon10&HRexp10≠ μHRcon20&HRexp20≠ μHRcon30&HRexp30≠ μHRcon40&HRexp40 ≠μHRcon50&HRexp50≠ μHRcon60&HRexp60 ≠ μHRcon70&HRexp70≠ μHRcon80&HRexp80

B). Core Temperature

I). The null hypothesis proposed that there will be no difference in core temperature responses between conditions.

Ho: μCTcon0-80 = μCTexp0-80

Ha: μCTcon0-80 ≠μCTexp0-80

II). The null hypothesis proposed that there will be no change in core temperature responses over time during both conditions.

Ho: μCTcon0&CTexp0 = μCTcon10&CTexp10 = μCTcon20&CTexp20 = μCTcon30&CTexp30 = μCTcon40&CTexp40 = μCTcon50&CTexp50 = μCTcon60&CTexp60 = μHRcon70&CTexp70 = μCTcon80&CTexp80

Ha: μCTcon0&CTexp0 ≠μCTcon10&CTexp10≠ μCTcon20&CTexp20≠ μCTcon30&CTexp30≠ μCTcon40&CTexp40 ≠μCTcon50&CTexp50≠ μCTcon60&CTexp60 ≠ μCTcon70&CTexp70≠ μCTcon80&CTexp80

HYPOTHESIS 3: PERCEPTUAL RESPONSES

A). Central Ratings of Perceived Exertion (RPE)

I). The null hypothesis proposed that there will be no difference in central RPE between conditions.

Ho: $\mu CenRPEcon_{0-80} = \mu CenRPEexp_{0-80}$

Ha: $\mu CenRPEcon_{0-80} \neq \mu CenRPEexp_{0-80}$

II). The null hypothesis proposed that there will be no change in central RPE over time during both test conditions.

Ho: $\mu CenRPEcon_{20} = \mu CenRPEexp_{20} = \mu CenRPEcon_{40} = \mu CenRPEexp_{40} = \mu CenRPEcon_{60} = CenRPEexp_{60} = \mu CenRPEcon_{80v} = \mu CenRPEexp_{80}$

Ha: $\mu CenRPEcon_{20} \neq \mu CenRPEexp_{20} \neq \mu CenRPEcon_{40} \neq \mu CenRPEexp_{40} \neq \mu CenRPEcon_{60} \neq \mu CenRPEexp_{60} \neq \mu CenRPEcon_{80} \neq \mu CenRPEexp_{80}$

B). Local RPE

I). The null hypothesis proposed that there will be no difference in local RPE between conditions.

Ho: $\mu LocRPEcon_{0-80} = \mu LocRPEexp_{0-80}$

Ha: $\mu LocRPEcon_{0-80} \neq \mu LocRPEexp_{0-80}$

II). The null hypothesis proposed that there will be no change in local RPE over time during both test conditions.

Ho: $\mu LocRPEcon_{20} = \mu LocRPEexp_{20} = \mu LocRPEcon_{40} = \mu LocRPEexp_{40} = \mu LocRPEcon_{60} = LocRPEexp_{60} = \mu LocRPEcon_{80} = \mu LocRPEexp_{80}$

Ha: $\mu LocRPEcon_{20} \neq \mu LocRPEexp_{20} \neq \mu LocRPEcon_{40} \neq \mu LocRPEexp_{40} \neq \mu LocRPEcon_{60} \neq \mu LocRPEexp_{60} \neq \mu LocRPEcon_{80} \neq \mu LocRPEexp_{80}$

HYPOTHESIS 4: PERFORMANCE RESPONSES

I). The null hypothesis proposed that there will be no difference in performance responses between conditions.

Ho: μPERFcon0-80 = μPERFexp0-80

Ha: μPERFcon0-80 ≠μPERFexp0-80

II). The null hypothesis proposed that there will be no change in performance responses over time during both test conditions.

Ho: $\mu PERFcon_{20} = \mu PERFexp_{20} = \mu PERFcon_{40} = \mu PERFexp_{40} = \mu PERFcon_{60} = PERFexp_{60} = \mu PERFcon_{80} = \mu PERFexp_{80}$

Ha: $\mu PERFcon_{20} \neq \mu PERFexp_{20} \neq \mu PERFcon_{40} \neq \mu PERFexp_{40} \neq \mu PERFcon_{60} \neq \mu PERFexp_{60} \neq \mu PERFcon_{80} \neq \mu PERFexp_{80}$

Where:

- HR = Heart rate responses
- CT = Core temperature responses
- MA = Muscle activity
- exp= Experimental condition
- con = Control conditions
- 0-80 = time intervals during the simulated rugby protocol
- I = Differences between conditions
- II = Differences over time
- BF = Biceps Femoris
- VL = Vastus Lateralis
- ST = Semitendinosus
- VM = Vastus Medialis
- CenRPE= Central ratings of perceived exertion
- LocRPE= Local ratings of perceived exertion
- PERF= Performance responses

DELIMITATIONS

This study was delimited to 15 male rugby players. All players were between the ages of 18-24 years. These players were selected from Rhodes University and Grahamstown schools‘ first team rugby squads. All testing was held in a laboratory

setting so that the environmental conditions could be controlled. The ambient temperature and relative humidity had to be maintained within a certain range. This study was not focussing on the contact activities of rugby, therefore, only the locomotor activities of rugby were included in this protocol. These activities included standing still, walking, jogging, striding and sprinting along a 20-meter (m) wooden walkway.

Players were required to attend three test sessions. During the first session, the test conditions, the protocol and procedures of the test were all thoroughly explained (both verbally and in writing) to the players. All players, regardless of experience were habituated to performing the activities of the simulated protocol, running on the treadmill, and how to properly use the Borg Scale for local and central ratings of perceived exertion (RPE). Following this habituation, player‘s demographic data (age, stature, playing position, and mass) as well as their injury history were recorded. Any players with recent musculoskeletal injuries of the lower limbs or recent illness were excluded from the study. Baseline measures were then taken, which included body fat percentage (using the skin-fold technique) and reference (resting) heart rates. Lastly, the order of testing was then randomly assigned to each player. The second and third test sessions involved players performing one of two test conditions; a control condition, during which players performed the protocol in minimal clothing and no protective gear; and an experimental condition, during which players performed the protocol in full rugby clothing and protective gear. Players performed the same protocol during each test condition, so that the effects of wearing the full rugby clothing and protective gear during a simulated rugby activity could be assessed.

The dependant variables measured included; physiological responses (heart rate and core temperature), perceptual responses (central and local RPE), which were all measured at set time intervals during the 80-min rugby protocol. Players‘ biophysical responses (muscle activity using electromyography), were measured during a 3-min EMG treadmill protocol, designed by Rahnama *et al*. (2006). Player‘s performance responses were measured, using an LED system that counted the total number of

20m shuttles players were able to complete during each of the 20-min work-bouts, making up the 80-min protocol. These data were collected in the same way and at the same time intervals during the control and experimental conditions. Testing occurred during the end stages of the 2011 South African rugby season (August-October).

LIMITATIONS

Although every effort was made to control the impinging variables, the following factors did pose limitations to the current study and should be taken into consideration when examining results:

The current study was limited, as it was focussed only on the locomotor aspects of rugby and was performed in a laboratory setting. Therefore, this study eliminated the impact that the physical contact nature of the game places on players, as well as the many different environmental factors (sun, wind, and rain) that affect players during real match-play. Furthermore, players performed the protocol on a wooden walkway, wearing running shoes, which eliminated the impact that running on grass in rugby boots places on players during match-play. Furthermore, the wooden walkway was made from plywood, which may have caused players to slip at either end of the walkway when turning around.

The players used in this study did not all wear full protective when they play rugby. Although all players wore some protective gear during play, they may not have been accustomed to using certain types of gear, such as thermal undergarments and scrumcaps. Therefore, there may have been differing levels of adaptation/acclimatisation to using these items, which may have impacted their responses. To control for this, the same protective gear was used for all the players. Furthermore, the gear itself may have hindered player‘s performance capacities due to the bulky nature of the gear. Players were permitted to use their own rugby

jerseys worn in rugby matches, as they both teams used the same brand of jersey. Therefore, the rugby jerseys were standardised.

This study used players from all positions, except for players from the front row, due to the high intensity of the protocol. The simulated rugby protocol was designed using time-motion data from Brown (2009) and Cunniffe *et al*. (2009). Therefore, some of the players, particularly some of the forwards, may have been unaccustomed to working at such high intensities during the protocol.

Despite all the players playing at a first team level, the training statuses of the players could not be controlled, as each team attended their own team‘s individual training sessions and also partook in their own training programmes. These differences in training status can be seen in the baseline fitness measures (resting heart rates).

All subjects were asked not to consume alcohol or perform any strenuous exercise 24 hours (hrs) prior to testing, not to eat 3 hrs prior to testing and drink 500 ml of water 1 hour before coming in for each test session. The researcher could not fully control for this. On arrival all players were asked if they had followed the researcher‘s instructions and if they had not done so, testing was either cancelled or postponed to a later date.

Lastly, although it is important to ensure that Electromyographic (EMG) electrodes are placed in the same location to ensure that reliable and valid measures are recorded, the electrodes had to be removed and replaced between trials. This may have affected the validity biophysical measures, as the electrodes could not be placed in exactly the same location as the previous condition.

CHAPTER II

REVIEW OF RELATED LITERATURE

DEVELPOMENT OF RUGBY UNION

The 2007 the Rugby World Cup in France was the third largest sporting event after the 2004 Athens Olympic Games and the 2006 FIFA Soccer World Cup, attracting over two million spectators and viewed by a worldwide audience of over three billion people (Mellalieu *et al*., 2008). In 1871, when the first international match was played between England and Scotland, Rugby Union Football became an international sport. After the second World Cup in South Africa in 1995, professionalism was adopted by the International Rugby Board (IRB) (Garraway *et al*., 2000) and has resulted in many changes being made to the game over the years (Mellalieu *et al*., 2008). These professional players have had to adapt to the increasing demands of the game and are now expected to show the skills, strength and fitness levels expected from full-time athletes (Garraway *et al*., 2000; Scott *et al*., 2002). Lucrative contracts are now being offered to elite players, which increases the pressure to perform at constantly high levels (Mellalieu *et al*., 2008). In a study of Five and Six Nations matches spanning the period 1988-2002, it was shown that rugby is a faster, ruck-dominated game, containing more phases of play (Eaves and Hughes, 2003). Since then, McMillan (2006) has shown that many changes being made to the game of rugby. Duthie *et al* (2003) found that these changes to the rules of the game have increased the intensity of rugby matches and ultimately increased the physiological stress experienced by players.

NATURE OF RUGBY

Rugby Union is a ball-carrying team sport, which involves full-body-contact activities and is played over two 40-minute (min) halves, separated by one 10-min break (half-time interval) (Duthie *et al*., 2003). There are no other stoppages during a match, except for injuries (Duthie *et al.*, 2003). Rugby is highly intermittent and varied in

nature, and involves repeated high-intensity sprints and a high frequency of physical contact between players (Duthie *et al*., 2007). Rugby has been described as being complex and chaotic, with conditions changing from game to game, and even from phase-to-phase (Vaz *et al*., 2010). Therefore, rugby produces a variety of physiological responses during match-play (Duthie *et al*., 2003).

According to Morton *et al*. (1978), player's heart rates range between 135-180 $bt.min^{-1}$ during a match with a mean heart rate of 161 $bt.min^{-1}$. Cunniffe *et al*. (2009) found that elite rugby players' mean heart rates were 172 $bt.min^{-1}$, which was higher than those described by Morton *et al*. (1978), possibly due to the intense and physical nature of elite rugby. During match-play, the ball is usually in play for 30-minutes, while the remaining time comprises of injury time, conversions, penalty kicks, or the ball being out of play (Duthie *et al*., 2003). Two teams of 15 players, excluding players being sent off for transgressions (Duthie *et al*., 2003) compete by attempting to carry the football across the opponent's goal line (Marshall *et al*., 2005). In general, more heavily built players play as 'forwards', while more slightly built, faster players tend to play as 'backs' (Marshall *et al*., 2005). Each player is assigned a specific position and number, and is grouped into either the forward pack numbered one to eight, or the backs numbered nine to fifteen (Duthie *et al*., 2003).

PHYSICAL NATURE OF THE GAME

Recent developments in technology have allowed the physiological demands of rugby to be accurately quantified using a Global Positioning System (GPS) device (Cunniffe *et al*., 2009). However, this technology has only recently been used in rugby, which still leaves the locomotor demands of the game under researched. GPS technology is now being used by researchers in sport, where the training loads and activity profiles of players can be evaluated during match-play (Cunniffe *et al*., 2009). Further, GPS devices also provides information on the distance covered, running speeds, heart rate, intensity of player impacts (g-force), and the energy expended during a match.

According to Cunniffe *et al*. (2009), backs and forwards experience many impacts during a match, with forwards experiencing significantly more impacts than backs (792 vs. 1274). An impact in rugby is an involvement in a tackle, ruck/maul, or collision with the ground. Players (forwards and backs) experience more impacts during the second half of match-play (Cunniffe *et al*., 2009; Bathgate *et al*., 2002), possibly due to this period of the match being more intense than the first half of match-play (Cunniffe *et al*., 2009).

In terms of running, players cover an average of 6953 meters (m) (6680 m for forwards and 7227 m for backs) during a match, depending on their playing positions (Cunniffe *et al*., 2009). Backs are shown to travel further distances (7227 m) than forwards (6680 m), which suggests that backs participate in a greater amount of higher intensity locomotor work than forwards (Cunniffe *et al*., 2009). Approximately 70% of the match is spent standing or walking, 25% running at moderate intensities (jogging and cruising), and 5% running at maximal intensities (high-intensity running and sprinting) (Cunniffe *et al*., 2009). This means that for every one minute of running, players will rest or move slowly for about five minutes (Cunniffe *et al*., 2009). During the second half of a match, the number of rest breaks decrease, and involves players performing more high-intensity running, which suggests that play is more continuous in the second half of a match (Cunniffe *et al*., 2009). Interestingly, Cunniffe *et al*. (2009), showed that rugby players total running distance, peak running speed, number of peak accelerations, average speed, total number of impacts and peak heart rates are all greater during the second 40-min of play. Therefore, the physiological load is suggested to be greater during the second half of a match. Further, cross-quarter comparisons show the third playing quarter, just after half-time, to be most intense, which suggests that fatigue is not a factor between halves (Cunniffe *et al*., 2009). During a match, players change speeds approximately 750 times, or once every three to four seconds (Cunniffe *et al*., 2009), due to the highly intermittent and unpredictable nature of the game. The average sprint is 15-20m long, and occurs approximately 30 times during a match, while fast jogging for 20m occurs 90 times per match (Cunniffe *et al*., 2009). Important to note, is the fact that the study by Cunniffe *et al*. (2009) only used two test subjects, which leaves the generalizability of these findings questionable. However, GPS technology has only

recently been used to measure the locomotor demands of rugby, which leaves this area of research under researched.

Forwards spend more time doing very high intensity movement, and less time walking or standing than backline players (66.5% of the match for forwards, compared to 77.8% for backs) (Cunniffe *et al*., 2009). This is explained by the forwards being involved in more periods of static exertion (scrums, rucks and mauls). Therefore, backs are considered to perform less work than forwards, despite being involved in more locomotor activities during match-play (Cunniffe *et al*., 2009).

During rugby, all players, regardless of position, perform deceleratory and acceleratory movements (stop/start nature of the game), with repeated concentric and eccentric movements (McLean, 1992; Duthie *et al*., 2005). Depending on whether the muscle shortens, lengthens or remains at a constant length, three types of muscle actions can be distinguished (concentric, eccentric or isometric actions) (Prilutsky, 2008). A muscle acts eccentrically if it is active and its length increases in response to an external force (e.g. force produced by other muscles, weight of external load) (Prilutsky, 2008). Conversely, a muscle acts concentrically if it is active and becomes shorter (Prilutsky, 2008). When the length of the muscle is prevented from shortening by external forces and remains constant, the muscle is isometrically active (Prilutsky, 2008). Eccentric actions occur in most sports and have been shown to be less metabolically demanding than concentric and isometric actions (Prilusky, 2008). Eccentric actions result in a different type of fatigue compared to other muscle actions (Prilustky, 2008). During moderate eccentric actions, it has been suggested that the developed muscle strength and power can be maintained for longer periods of time (Crenshaw *et al*., 1995), while subjective ratings of perceived exertion are considerably lower (Pandolf *et al*., 1978), when compared to concentric actions of the same intensity. This is possibly due to fewer muscle fibres being recruited during eccentric exercise, when compared to concentric and isometric actions of the same intensity (Prilutsky, 2008). Therefore it is important to understand the biomechanical and physiological consequences of eccentric muscle actions and what impact these

actions may have on performance (Prilutsky, 2008), as eccentric muscle actions have been associated with a greater risk of injury, compared to other muscle actions.

INJURIES IN RUGBY

Rugby Union is one of the most popular team sports in the world, but it also has one of the highest reported incidences of injuries (Brooks *et al*., 2005). An injury is defined as an event that forces a player to either leave the field or miss a subsequent match (Bathgate *et al*., 2002). The severity of an injury is defined by the number of days a player takes to return to full fitness (Brooks *et al*., 2005). According to Brooks *et al*. (2005), a team will suffer 70 match injuries (backs, 32 and forwards 38) per season. The average numbers of match injuries a player will sustain during a season is 1.8, and the average amount of playing/training time each player will lose per season due to injury is 33 days (Brooks *et al*., 2005). It has been shown that more injuries occur during match-play, compared to training sessions (Bathgate *et al*., 2002; Junge *et al*., 2004), with over two thirds of these injuries occurring during contact situations between players (Junge *et al*., 2004). Most injuries occur during the tackling phase (58 %), either tackling or being tackled, whereas open play, which occurs when the ball is no longer in a set piece, in a ruck/maul, or in the tackle, accounts for 19.6% of all injuries (Bathgate *et al*., 2002). The set pieces in rugby (scrums and lineout‘s) accounts for only 2.1 % of all rugby injuries (Bathgate *et al*., 2002).

Numerous studies have shown that more injuries occur during the second-half of a rugby match (Hughes and Fricker, 1994; Bathgate *et al*., 2002, Brooks *et al*., 2005), possibly due to players running more, covering greater distances, experiencing twice as many impacts, and having less rest breaks during the second half of a match (Cunniffe *et al.,* 2009). These findings are similar to those observed in soccer (Hawkins and Fuller, 1999). Interestingly, when subdivided into quarters, it is shown that the third quarter of a rugby match is the period of play when most injuries occur, and has been suggested to be due to incomplete warm-up, or reduced concentration after the half-time break (Bathgate *et al*., 2002). This emphasizes how vital training status, conditioning and player management are, since fatigue has been identified as

one of the main factors causing muscular injuries in the lower limbs, particularly in the hamstrings (Verral *et al*., 2001; Woods *et al*., 2004; Petersen and Hölmich, 2005). Although, these studies suggest that more injuries occur during the second half of rugby, possibly due to fatigue, these tests never actually measured fatigue, which leaves this issue unclear. Hamstring injuries are common in all sports that require rapid acceleration and maximum speed running (Hoskins and Pollard, 2005), as repeated bouts of sprinting has shown to cause significant changes in running technique over time (Pinniger *et al*., 2000), possibly due to muscle fatigue. The incidence of hamstring injuries is higher for backs than forwards (Brooks *et al*., 2005), possibly due to the higher running demands placed on the backs, as well as the greater number of acceleratory and deceleratory movements made by backs during a match (Deutsch *et al*., 1998).

PROTECTIVE GEAR WORN IN RUGBY

The development and physical nature of rugby predisposes players to a vast array of injuries (Sinclair, 2009) and has resulted in the establishment of many commercial companies becoming involved in the design and production of protective gear (Sinclair, 2009). Although the use of protective gear has not been made compulsory (Junge *et al*., 2004), the use of protective gear by players is increasing, but the information on the proper use of this equipment is still lacking (Sinclair, 2009). Despite many scientific studies being conducted on the effectiveness of protective gear, much controversy still exists (Sinclair, 2009). It has been suggested that wearing protective equipment, such as mouth guards, shoulder pads and headgear, may actually increase the risk of injuries, as players attitudes have been shown to be altered while wearing protective gear (Sinclair, 2009). For example, player‘s self-confidence and aggression have shown to increase when going into contact situations while wearing protective gear (Sinclair, 2009). According to Marshall *et al*. (2005), all protective gear, except for mouth guards and headgear, are largely ineffective in preventing serious injuries (Marshall *et al*., 2005), which raises questions surrounding whether or not these items should be worn at all.

Mouth Guards

Mouth guards have shown to reduce the incidence of fractured teeth and head accelerations during rugby matches (Bernhardt and Anderson, 2005; Finch *et al*., 2001; Knapik *et al*., 2007). However, it still remains inconclusive as to whether mouth guards are effective in the preventing concussions (Sinclair, 2009). In New Zealand, the use of mouth guards has been made compulsory in all matches played below international level by the New Zealand Rugby Union (Junge *et al*., 2004), but has yet to occur in South Africa.

Head-Gear

Head-gear has been recommended for use at all levels, as it provides a measure of protection against lacerations and abrasions (Jones *et al*., 2004; Wilson, 1998). However, the effectiveness of this equipment for the prevention of concussion has also yet to be proven (Kahanov *et al*., 2005; Wilson, 1998). Furthermore, significant heat loss has shown to occur from the head area (Cheung *et al*., 2000), so by covering the head with protective gear during exercise may cause increased heat gains.

Shoulder Pads

Shoulder pads are worn to absorb and disperse force from direct impacts between players and prevent direct impact injuries and soft-tissue bruising (Sinclair, 2009). However, there still remains no consensus regarding the effectiveness of this equipment in preventing more serious damage to the shoulder joint. According to Bathgate *et al*. (2002), shoulder padding can in no way prevent rotator cuff tears or glenohumeral instability injuries, as most of the shoulder injuries experienced by players involve rotational forces (Gerrard, 1998). However, shoulder padding is not designed for prevention against rotational forces (Gerrard, 1998).

Compression Gear

Compression garments are now being worn underneath normal playing apparel in team sports (Houghton *et al*., 2009). Manufacturers claim that this gear improves venous blood flow, which increases the delivery of blood to muscles and improves lactate removal and physiological performance (Houghton *et al*., 2009). Further, it has been shown that this gear reduces the risk of injury, with regard to sprains and strains, and prevents the recurrence of hamstring injuries (Upton *et al*., 1996; Doan *et al*., 2003; Bernhardt and Anderson, 2005; Marshall *et al*., 2005). Manufacturers also claim that wearing compression garments aids in cooling the body by improving blood flow to the skin, and increases the evaporation of sweat from clothing (Houghton *et al*., 2009). In contrast however, compression gear has shown to increase skin temperature (Doan *et al*., 2003) and muscle temperature (Sinclair, 2009) during exercise, as it actually hinders the mechanisms for heat loss (Houghton *et al*., 2009).

Base-Layer Garments

The use of base-layer garments in team sports has become very popular in recent years (Roberts *et al*., 2007). These garments are typically worn next to the skin and claim to afford distinct functional properties to the wearer (Roberts *et al*., 2007). Manufacturers claim that base-layer hot garments keep the wearer comfortably cool during exercise and are normally worn in hot conditions (Roberts *et al*., 2007). The base-layer cool garment keeps the wearer comfortably warm during exercise, and is normally worn in cold conditions (Roberts *et al*., 2007). Both of these garments are claimed to reduce moisture retention because of good wicking properties (Roberts *et al*., 2007). However, to date, the thermoregulatory effects and moisture management properties have yet to be studied (Roberts *et al*., 2007).

THERMOREGULATION

Human core temperature is maintained at approximately 37° C by thermoregulatory mechanisms that control heat production and heat dissipation (Brotherhood, 2008). If

these thermoregulatory mechanisms are unable to achieve adequate heat loss, core temperature may increase towards critically high levels where collapse or possible heat stroke may occur (Brotherhood, 2008).

Humans are homeotherm‘s, which means that core temperature is constantly being regulated so that it remains within an optimal range of approximately 37° C (Parsons, 2000). When the human body is subjected to thermal stress, the thermoregulatory system reacts by altering its present state (Parsons, 2000). However, this response to thermal stress may have negative consequences on the athlete‘s health, comfort and performance (Parsons, 2000). According to Parsons (2000), six important factors need to be considered when trying to determine how athletes will respond during exercise in different environments. These include air temperature, radiant temperature, air velocity, humidity, the exercise, and the clothing worn during exercise (Parsons, 2000).

Mechanisms of Heat Loss and Gain

Four mechanisms for heat loss exist to ensure that the human body is kept in thermal homeostasis (Gavin, 2003). These include evaporation, radiation, convection and conduction. Of the four mechanisms, radiation and evaporation transfer most of the heat from the human body (Grubenhoff *et al.*, 1998). Convection and evaporation are controlled by natural physiological responses, namely circulatory dynamics and sweating (Grubenhoff *et al*., 1998).

RADIATION

Radiative heat transfer is the transfer of heat between two surfaces, each with different temperatures (Brotherhood, 2008), without molecular contact (McArdle *et al*., 2006). Radiative heat transfer occurs when heat is lost to cooler surroundings, or when heat is gained from a warmer surrounding (Brotherhood, 2008). The human body is usually always warmer than the environment, which means that radiant heat energy moves through air to a solid, cooler object in the environment (McArdle *et al*.,

2006). Radiant heat is also absorbed by the body from the sun because the body‘s temperature is cooler than the heat given off by the sun‘s rays (Noakes, 1986). Many rugby games are played during the day, which means that players will absorb the heat given off from the sun‘s rays. The amount of radiant energy absorbed from the sun will be greatest when there is no cloud cover (Noakes, 1986).

CONDUCTION

Heat exchange via conduction involves direct heat transfer from one molecule to another through a liquid, solid or gas (McArdle *et al*., 2006). Most of the body‘s heat is transported by circulation towards the skin for heat dissipation to occur, however, a small amount of body heat continually moves via conduction directly through the deep tissues towards the cooler surface (McArdle *et al*., 2006). Therefore, heat loss via conduction involves warmer air molecules or cooler surfaces making direct contact with the skin (McArdle *et al*., 2006).

The rate of conductive heat loss depends on two factors, namely the temperature gradient between the skin and surrounding surfaces, and the skin and surrounding surfaces thermal qualities (McArdle *et al*., 2006). The effectiveness of heat loss via conduction depends on how quickly the air next to the body moves away once it is warmed (McArdle *et al*., 2006). If air movement or convection proceeds slowly, the air next to the skin warms and acts as an area of insulation that reduces further heat loss via conduction from occurring (McArdle *et al*., 2006). In air temperatures of 33º C, skin temperature becomes warmer than the temperature of the air and results in convective heat loss occurring. However, at higher air temperatures, skin temperature becomes lower than the airs temperature causing increased heat gains, which could affect athlete‘s performance capacities or increase the risk of developing heat-related illness (Brotherhood, 2008).

EVAPORATION

The evaporation of sweat normally provides a powerful physiological cooling mechanism for humans in warm conditions (Nunneley, 1989), with each vaporized litre of water extracting 580 kCal of heat from the body, transferring it to the environment (McArdle *et al*., 2006). During intense exercise, heat loss via convection and radiation are typically not enough to achieve thermal homeostasis so, additional cooling via the evaporation of sweat from the skin, is needed (Brotherhood, 2008). The evaporation of sweat from the skin exerts a cooling effect on the body and serves as the primary defence mechanism against overheating (McArdle *et al*., 2006).

The evaporative requirements are determined by the sum of metabolic heat production and the radiative and convective heat exchanges (Brotherhood, 2008). To achieve thermal homeostasis, the net load of heat production, radiative heat transfer and convection must be balanced by evaporative cooling (Brotherhood, 2008). Evaporative cooling occurs due to sweat, at the level of the skin, being transferred as water vapour to the environment in a physical process similar to that of convective heat transfer (Brotherhood, 2008). Further evaporative heat loss also lost occurs during ventilation (Burton *et al*., 2004). Evaporative cooling is the most common way to dissipate body heat in environments with high ambient temperatures (warmer than the body‘s temperature) (Yaqub and Al Deeb, 1998), as heat loss via conduction, convection and radiation become hindered at high ambient temperatures (McArdle *et al*., 2006).

Three factors influence the total amount of sweat vaporized from the skin, namely: the surface exposed to the environment, the temperature and relative humidity of the ambient air and the convective air currents surrounding the body (McArdle *et al*., 2006). Relative humidity is the most important factor determining the effectiveness of evaporative heat loss (McArdle *et al*., 2006). When the relative humidity of the surrounding air is high, the evaporation of sweat becomes hindered and sweat will accumulate without any heat loss occurring (Yaqub and Al Deeb, 1998), due to the

ambient vapour pressure approaching that of moist skin (about 40 mm Hg). This may increase the risk of dehydration and overheating occurring and cause an earlier onset of fatigue (McArdle *et al*., 2006). When the relative humidity of the air is low the water vapour pressure gradient between the sweat at the level of the skin and the surrounding air increases (Yaqub and Al Deeb, 1998). This increased water vapour pressure gradient between the sweat and air causes more sweat evaporating from the skin (Yaqub and Al Deeb, 1998).

The effectiveness of evaporative heat loss depends on whether enough physiological sweat can be produced, and whether the environment will allow for evaporation to occur (Brotherhood, 2009). Individual sweat rates vary and depend on factors such as ambient air temperature, humidity, air movement, exercise intensity, insulating clothing or equipment, and body size (Godek *et al*., 2005). In a study by Pyke and Hahn (1980), Australian Rules football players' sweat rates averaged 1.8 litres per hour ($L.hr^{-1}$) when playing in ambient temperatures of approximately 38° C. However, in American Football players, higher average sweat rates were observed (2.14 $L.hr^{-1}$), possibly due to the additional clothing and protective gear worn by football players. Clothing and protective gear worn during exercise reduces the ability for this sweat to evaporate, which may cause body temperatures to continue rising (Godek *et al*., 2005). In rugby, players are recommended to take in adequate amounts of fluid during matches in hot and humid conditions, and wear gear that combines both strength and heat-loss qualities (Brewer and Davis, 1995).

CONVECTION

High wind speeds cause a large amount of cooler air to cross the skin and allows for greater heat loss via convection to occur (Noakes, 1986). By simply running, an effective wind speed is produced which aids convective heat loss (Noakes, 1986). However, during exercise in hot environmental conditions, the air velocity created by running may not be enough for adequate heat loss to occur (Noakes, 1986). If there is little wind or a wind coming from behind the athlete at the same speed that the

athlete is moving forward, it will cause the runner to run in a totally windless environment, which hinders convective heat loss (Noakes, 1986).

Muscle Contractile Effect on Body Temperature

The muscles are the largest group of tissues in the body, making up 45 % of body mass (NIOSH, 1986). Even at rest, the muscles generate approximately 20-25 % of the body‘s total heat (NIOSH, 1986). The amount of metabolic heat produced at rest is quite similar for all individuals, however during exercise heat produced by a muscle can be much higher and it must be dissipated if the body‘s heat balance is to be maintained (NIOSH, 1986). The heat load from metabolism is, therefore, widely variable, and exercise in the heat, which imposes its own heat load or hinders heat dissipation, creates the greatest challenge to normal thermoregulation (NIOSH, 1986). Muscular activity is associated with increases in muscle temperature, which drives core temperature (NIOSH, 1986). During high intensity exercise, the greatest proportion of physiological heat strain is caused from increased muscular contraction (Parsons, 2000). Muscle work contributes not only towards heat production, but also towards heat dissipation (Shimizu *et al*., 1998). This places athletes at a disadvantage, as conflicting demands are placed on the cardiovascular system, because both the working muscles and cutaneous circulation require large portions of the cardiac output during exercise (Vigas *et al*., 2000).

Heat stress and strain in sport are a multifaceted mix of behaviours, physics and physiology (Brotherhood, 2008). During exercise, heat production is a function of maximal aerobic power and the relative exercise intensity (Brotherhood, 2008). Exercise increases metabolic activity, which produces energy; however the excess energy not converted to work, is released as heat in the muscles (Havenith, 2002). During exercise the conversion of chemical energy (stored in ATP) into mechanical energy is extremely inefficient (McArdle *et al*., 2005). As much as 70 % of the total chemical energy used during muscular contraction is released as heat instead of as athletic undertaking (Noakes, 1986), which could cause body temperatures to reach critical levels (McArdle *et al*., 2006). According to Noakes (1986), it is necessary for

the body to call upon a number of very effective heat-loss mechanisms to prevent dangerous overheating and heat stroke, and control the heat gains associated with exercise. During endurance running, which is continuous in nature, reaching core temperatures of approximately 40°- 41° C are considered ‗normal responses' to strenuous exercise (Brotherhood, 2008). However, during intermittent sports, such as rugby, reaching core temperatures of approximately 39.5° C is considered normal (Brotherhood, 2008). The possible reason for rugby players reaching considerably lower core temperatures may be due to the fact that intermittent sports involve short bursts of high-intensity exercise followed by periods of rest, which may allow players core temperatures to remain cooler than that of endurance runners.

As exercise starts, blood flow to the active muscles increases, not only because the heart pumps more blood, but also because blood is preferentially diverted away from the non-essential organs towards the active muscles and skin (Noakes, 1986). As it passes through the muscles, the blood is heated and distributes this additional heat throughout the body, especially towards the skin (Noakes, 1986), as the peripheral vessels dilate, allowing the warm blood to flow towards the cooler periphery (McArdle *et al*., 2006). It is essential that the body dissipates this additional heat to maintain thermal homeostasis (Noakes, 1973). Thermal homeostasis is only possible when the sum of the environmental and metabolic heat load placed on the body is matched by the outward heat dissipation to the environment (Nunneley, 1989).

During intense periods of exercise, the body must decide whether to pump more blood to the muscles to maintain their increased energy requirement, or increase the blood flow to the skin for adequate heat dissipation to occur (Noakes, 1986). During exercise, the body favours increased blood flow to the muscles, which causes body temperatures to increase, because the body's ability to dissipate this additional heat to the level of the skin is hindered (Noakes, 1986). Therefore, during exercise, the main driver of core temperature is the exercise itself, as heat loss mechanisms become hindered by the blood being diverted towards the active muscles. When core temperature reaches critically high levels, the body's cutaneous circulation increases and diverts blood away from the muscles and towards the body's vital

organs, so as to avoid any damage to these organs occurring. The increase in cutaneous circulation initiates the sweating response in an attempt to cool the body down. Thus, when homeostasis is disturbed during exercise-induced heat stress, the body diverts the circulation away from the active muscles in an attempt to reduce the athlete‘s work rate so that body temperature can return to normal levels.

Impact of Clothing and Protective Gear on Body Temperature

Many thermoregulatory studies have often investigated thermal responses without considering the influence that the clothing has on the athlete (Pascoe *et al*., 1994). In many circumstances, athletes have little choice in selecting the type or amount of clothing worn during competition, as these are often specified by the rules of the sport (Maughan and Shirreffs, 2004). Clothing and protective gear worn during exercise in the heat has shown to limit the benefits associated with increased aerobic fitness or heat acclimation, as water vapour permeability is limited by clothing (Nunneley, 1989). This results in large biophysical requirements for evaporative heat loss, which results in an increased sweat rate (Cheuvront *et al*., 2004). However, this may further increase the risk of dehydration if fluids are not replaced (Roberts *et al*., 2007). The threat is therefore increased core temperature coupled with rising levels of dehydration.

Wearing clothing and protective gear during exercise may result in a situation of incompensable heat stress, because mechanisms of heat loss become hindered (Godek *et al*., 2005). Research done on university rugby players (Cannon and Christie, 2010) and football players (Kulka and Kenney, 2002) showed that wearing clothing and protective gear increased physiological responses and body temperature, and caused significant reductions in performance, when compared to exercise in minimal clothing and no protective gear. This suggests that the additional heat generated by exercising in clothing and protective gear may be responsible for an earlier onset of fatigue, which could explain the decrements observed in performance over time.

During hyperthermia, the phenomena of heat loss and heat gain while wearing clothing may be divided into three stages (Kakitsuba, 2004). In the first stage, an individual‘s sweat rate increases, causing water vapour accumulating in the clothing‘s micro environment (Kakitsuba, 2004). In the second stage, the clothing‘s microenvironment becomes fully saturated with sweat, causing the evaporation of sweat to cease, while sweating continues (Kakitsuba, 2004). In the third stage, the sweat, which has gradually absorbed into the clothing, evaporates from the clothing surface instead of from the skin (Kakitsuba, 2004), which is known as the phenomenon called reverse evaporation (Nelbace and Herrington, 1942). When reverse evaporation occurs, the surface temperature of the clothing may decrease, whereas the skin temperature continues to rise (Kakitsuba, 2004). Therefore, clothing increases thermal insulation, which obstructs heat loss and reduces the transfer of water vapour from the skin to the environment (Roberts *et al*., 2007).

Rugby jerseys have been suggested to negatively affect player‘s abilities to dissipate heat during exercise in warm, humid conditions (Meir *et al*., 1994). Clothing and protective gear should be designed to allow for adequate heat loss to occur. In rugby, it has been suggested that different lightweight jerseys be worn that allow for greater heat dissipation to occur, and benefit players competing in hot and humid conditions (Brewer and Davis, 1995). However, the effect that rugby clothing and protective gear has on rugby players during training sessions or matches remains unknown. Many players unknowingly wear inappropriate clothing and protective gear, which may not only be ineffective in preventing serious injuries, but also counterproductive for heat dissipation. In a study by King *et al*. (2002), it was shown that the protective gear and clothing worn by cricket batsmen impedes the mechanisms of heat loss during exercise, and results in players sweat rates significantly increasing in an attempt to dispel this additional heat load and maintain thermal homeostasis (King *et al*., 2002). However, protective gear is largely resistant to moisture transfer, as these items create a humid microclimate, increases local humidity and slows evaporative heat loss, causing players‘ body temperatures to progressively increase while batting (King *et al*., 2002).

Other Contributors to Heat Gain

BODY COMPOSITION

Body mass and excess body fat negatively affects sporting performance, as it impacts an individuals' power to body mass ratio, thermoregulation, and aerobic capacity (Meir *et al*., 2001). Body mass is often used as a predictor for selection into teams, or as a forward or backline player in rugby. In general, more heavily built players play as 'forwards', while more slightly built, faster players tend to play as 'backs' (Marshall *et al*., 2005).

POOR TRAINING STATUS

Fitter individuals adapt more easily to heat stress, as training results in cardiovascular and thermoregulatory adaptations, which decreases physiological strain and increases heat tolerance during exercise in the heat (Fox *et al*., 1967; Garden *et al*., 1966). Adaptations after heat acclimation include increased sweating, lower heart rates, body temperatures and perceived exertion during exercise in the heat (Wyndham, 1973).

DEHYDRATION

Fluid loss, as minimal as a 1-2 % loss in body mass, can begin to hinder performance and thermoregulatory functioning (ACSM, 1985). Dehydration profoundly impairs physiologic function and compromises athlete's ability to train and compete (McArdle *et al*., 2005). In a study by Edwards *et al*. (2006), it was shown that soccer players performance capacities were negatively impacted by mild dehydration, however it remains unclear whether this was due to fluid loss, or the negative psychological associations that occur as a result of a greater perception of effort in that state. The symptoms of dehydration include: dry mouth, thirst, irritability, general discomfort, headache, apathy, weakness, dizziness, cramps, chills, vomiting, nausea, head or neck heat sensations, excessive fatigue and/or decreased performance (ACSM, 1985). According to Goodman (1985), fluid loss during exercise is one of the primary factors predisposing athletes to hyperthermia

(Goodman, 1985). To ensure and maintain optimal hydration levels during exercise, fluid intake should be approximately equal to fluid loss (ACSM, 1985).

LACK OF ACCLIMATIZATION

Heat acclimatization is the ability of man to maintain a relatively constant body temperature, even when external temperature or metabolic heat production change (Bass *et al*., 1955). Adaptations after heat acclimation include increased sweating responses, decreased heart rate, core temperature and skin temperature, and perceived exertion while exercising in the heat (Wyndham, 1973). Therefore, players not accustomed to training or playing matches in hot and humid conditions may not experience the benefits of heat acclimation.

FATIGUE

In general, fatigue has been described as a preventable and negative result of exercise, since the outcome almost always causes a reduction in performance capacities and overall function (Kirkendall, 1990). However, fatigue does not need to have such negative connotations, as fatigue has been described as a safety mechanism, regulated by central or peripheral input, to prevent the development of injury or death by continuously regulating exercise intensity (Noakes, 1998).

According to Pincivero *et al*. (2000), fatigue is a time-dependant process that encompasses numerous central, peripheral and psychological factors. Traditionally, fatigue is proposed to occur as a result of changes occurring anywhere between the brain and the muscle fibre (Weir *et al*., 2006) and is specific to the movement pattern of the exercise (Spendiff *et al*., 2002). However, it still remains unclear how central and/or peripheral factors contribute to the development of muscle fatigue during intermittent sports (Girard *et al*., 2008; Morris *et al*., 1998), because no obvious boundary between the nervous system and muscle exists (Barry and Enoka, 2007).

During exercise, fatigue causes a breakdown in the athlete‘s movement patterns (Bates *et al.*, 1977), which not only affects performance, but may also increase the risk of injury. Fatigue has been identified as one of the main factors causing muscular injuries in the lower limbs, particularly in the hamstrings (Verral *et al*., 2001; Woods *et al*., 2004; Petersen and Hölmich, 2005), which is the second most commonly occurring injury experienced by rugby players (Brooks *et al*., 2005). Therefore, by gaining insight into the possible mechanisms of fatigue experienced during competitive sports, possible intervention strategies can be used to try and delay the onset of fatigue, and therefore reduce the risk of suffering muscle injuries.

Fatigue has been defined as the inability to maintain a required force (Edwards, 1981). However, this definition did not include the exercise as a factor of fatigue (Barry and Enoka, 2007). Therefore, fatigue was redefined as a reduction in the force-generating capacity of the neuromuscular system that occurs during continued exercise (Bigland-Ritchie *et al*., 1983). However, this definition does not accommodate for changes that occur during movement nor the sensations that accompany fatiguing contractions (Jones, 1993; McComas *et al*., 1995). During fatigue, performance is proposed to become acutely impaired, with an increase in the perceived effort necessary to exert a desired force and an eventual inability to produce this force (Enoka and Stuart, 1992). Therefore, during exercise-induced fatigue, there is a reduction in the maximal force generating capacity of muscle (Gandevia, 1998).

According to the principle of task dependency however, there is no single cause of fatigue, and it is suggested that fatigue is dependent on the details of the task being performed (Barry and Enoka, 2007). This principle acknowledges that fatigue can be caused from multiple mechanisms and that the main causes vary from task-to-task (Barry and Enoka, 2007). The main factors affecting fatigue include subject motivation, patterns of muscle activation, intensity and duration of activity, and whether the exercise is continuous or intermittent in nature (Barry and Enoka, 2007).

During submaximal muscle activations (Faulkner, 2003), performance may be maintained at the target intensity for longer durations of time (Vollestad, 1997). However, during sustained maximal muscle activations, muscle force progressively decreases due to fatigue (Merton, 1954). This reduction in force generating capacity occurs from the start of the exercise because the target intensity can no longer be maintained during the progressive development of fatigue (Vollestad, 1997; Barry and Enoka, 2007). However, many studies have focused on fatigue experienced during isometric contractions, which may not be representative of muscle activity and the development of fatigue during human locomotion (Green, 1995).

In the beginning stages of exercise, muscular efficiency is high, but rapidly decreases as the duration of exercise increases (Gonzalez-Alonso *et al*., 2000). For example, during a football match, fatigue, although evident throughout the match, becomes more evident towards the end of the match (Rahnama *et al*., 2003; Mohr *et al*., 2003). Professional football players have shown to cover a 5 % greater distance during the first half of a match, compared to the second half (Bangsbo *et al*., 1991), confirming that a progressive reduction in athlete‘s exercise capacity occurs as the match continues (Asmussen, 1979). Therefore, fatigue decreases an athlete‘s exercise capacity, as a result of a brief reduction in the maximal force generating capacity of skeletal muscle while fatigued (Vollestad, 1997). In contrast, Cunniffe *et al*. (2009) showed that during the second half of a rugby match, players‘ performed more work compared to the first-half, which suggests that fatigue may not be a factor affecting players performance capacities as player‘s performance responses never showed decrements during the second half despite both the duration and intensity of exercise increasing over time.

Peripheral and Central Fatigue

Fatigue has been classified as either being central or peripheral in origin (Kay *et al*., 2000). Effects occurring within the motor unit are considered to be within the domain of peripheral fatigue, whereas events in the brain and spinal cord are described as central fatigue (Asmussen, 1979). However, during ongoing dynamic exercise, the

differentiation between central and peripheral factors contributing to fatigue becomes more difficult (Nybo and Secher, 2004). It is assumed that exercise ceases at a point known as exhaustion, where individuals can no longer voluntarily generate the required power output to perform the task (Marcora *et al*., 2009). Exercise has been suggested to be centrally regulated, based on the neural integration of afferent information from the periphery, and is influenced by factors such as previous experience and training (St Clair Gibson and Noakes, 2004).

It has been long been assumed that exercise is limited by central and/or peripheral muscle fatigue (Noakes and St Clair Gibson, 2004). Peripheral muscle fatigue has been defined as a reduced capacity of skeletal muscle to produce force due to action potential failure, excitation-contraction coupling failure, or impairment of cross bridge cycling, despite the neural drive remaining unchanged or increasing (Hakkinen, 1983). In contrast, central fatigue has been described as a reduction in the neural drive to muscle, causing a subsequent reduction in force production, regardless of changes in skeletal muscle contractility (Enoka and Stuart, 1992). During central fatigue, there is a reduction in muscle recruitment patterns (Girard *et al*. 2008; Girard *et al*., 2009), measured via electromyography (iEMG), which causes a reduction in force production (Woods *et al*., 1987). This central regulation of exercise intensity is proposed to occur before a catastrophic failure to vital organs can occur (Noakes *et al*., 2004; Lambert *et al*., 2005; St Clair Gibson *et al*., 2001; St Clair Gibson and Noakes, 2004; Tucker *et al*., 2004; Tucker *et al*., 2006). However, other studies have shown that muscle activity increases during fatigue (Mizrahi *et al*., 1997; Brown, 2009). This phenomenon is proposed to occur due to mechanisms of peripheral fatigue (Gerdle *et al*., 1997). This causes a reduction on the force generating capacity of muscles, despite there being an increase in neural drive, also measured via EMG, so that a constant work rate can be maintained and allow the exercise to continue (Gerdle *et al*., 1997). This occurs until the athlete reaches the point of exhaustion where the exercise has to be terminated (St Clair Gibson and Noakes, 2004).

Peripheral Fatigue

The peripheral fatigue model has proven to be the most popular explanation for fatigue for the past several decades (Noakes, 2000). This model suggests that system failure occurs because of either metabolite accumulation or substrate depletion, and can only be prevented by reducing the workload or completely terminating the exercise (Lambert *et al*., 2005). However, it has been shown that fatigue does not occur due to Adenosine Triphosphate (ATP) depletion (Noakes and Lambert, 2004), as this never occurs during exercise (Lambert *et al*., 2005). Alternatively, Hill *et al*. (1924b) proposed that maximal exercise performance is limited by cardiovascular functioning, due to dependent metabolic changes occurring in the active muscles, but only occurs after the onset of fatigue (Hill *et al*., 1924b). Therefore, during moderate to high intensity exercise, fatigue develops when the cardiorespiratory system can no longer provide enough oxygen to the exercising muscles, and induces the anaerobic metabolism (Noakes, 2000).

Metabolic changes during exercise causes system failure to either the peripheral muscles or the heart, and causes exercise to be terminated, despite an increase in the central drive from the brain to the peripheral muscles (Hakkinen, 1983). This increased neural drive is aimed at maintaining the current work rate and prolonging the exercise (Hakkinen, 1983; Taylor, 1997). However, continuing the exercise at the current work rate would surely worsen the metabolic crisis, which would lead to exhaustion, where exercise has to be terminated (St Clair Gibson and Noakes, 2004). At the point of exhaustion, the individual is required to rest for a period of time, so that the metabolic crisis can be reversed and the concentrations of inhibitory metabolites can return to normal levels and therefore, allow the athlete to eventually continue exercising (St Clair Gibson and Noakes, 2004). Therefore, the peripheral fatigue model predicts that exercise always terminates at an absolute, temporarily irreversible end point (St Clair Gibson and Noakes, 2004). However this model of fatigue has been criticized, as the heart would most probably be the first organ affected by anaerobiosis, during maximal exercise, and not skeletal muscles (Noakes, 2000).

Central Fatigue

Alternatively, the central fatigue model hypothesizes that the reduction in power output during prolonged exercise, which regulates exercise, is not caused by limiting physiological processes in the peripheral organs, but rather from central processes (St Clair Gibson and Noakes, 2004). During fatigue, the central nervous system (CNS) reduces muscle recruitment, which causes the athlete‘s exercise capacity to become impaired (Davis and Bailey, 1997). Central fatigue is proposed to occur as a result of conscious or subconscious mechanisms (James *et al*., 1995). When an individual decides that the sensations experienced during exercise are intolerable, exercise intensity is purposefully reduced at a conscious level (James *et al*., 1995). Alternatively, when central fatigue occurs subconsciously, an athlete‘s muscle power output is regulated by altered efferent commands being sent from the brain during all forms of exercise (James *et al*., 1995). The primary objective for this central down-regulation of muscle power, and therefore exercise intensity, is to maintain whole-body homeostasis (St Clair Gibson and Noakes, 2004).

Central Governor Hypothesis

The long-forgotten central governor hypothesis, as originally proposed by Hill and colleagues in the 1920s (Hill *et al*., 1924a; Hill *et al*., 1924b), was rediscovered by Noakes (Noakes 1998; Noakes, 2000; Noakes, 2001). According to Hill *et al.* (1924b), the limit to which the muscles may be driven, while breathing air, is limited by the athlete‘s inability to take in enough oxygen to supply the active muscles and vital organs to continue exercising at the exercise intensity. This has been shown by the existence of a plateau in oxygen consumption, despite an increase in exercise intensity (Hill and Lupton, 1923). The heart is suggested to be capable of regulating its output, based on its degree of saturation of arterial blood when there is inadequate oxygen available to the exercising muscles (Hill *et al*., 1924b), and decreases exercise intensity. It would be ineffective for the heart to work at excessively high intensities, if by doing so it resulted in arterial blood becoming less saturated with oxygen (Hill *et al*., 1924b). Therefore, some mechanism, either in the heart muscle or in the nervous system, has been suggested to decrease the rate of circulation when the level of oxygen becomes too low in the blood (Hill *et al*., 1924b).

This mechanism therefore, acts as a ‗governor', ensuring that a normal level of oxygen is maintained in the circulating blood (Hill *et al*., 1924b).

Another hypothesis has been proposed, in which the CNS controls central circulation during high intensity exercise (Noakes, 1996; Noakes, 1998; Noakes, 2000). This central governor controls physical activity to ensure that the human body can function normally as a complex system during exercise (Noakes *et al*., 2005). This is achieved by slowing blood circulation during exercise, which prevents the heart from becoming ischemic (Noakes, 1996; Noakes, 1998; Noakes, 2000). Therefore, exercise performance is regulated by the central nervous system specifically to ensure that catastrophic physiological failure does not occur during normal exercise in humans (Noakes *et al*., 2004; Lambert *et al*., 2005; St Clair Gibson *et al*., 2001; St Clair Gibson and Noakes, 2004; Tucker *et al*., 2004; Tucker *et al*., 2006). This regulation of exercise does not completely prevent homeostasis from being disturbed, but rather prevents these changes from reaching levels where exercise has to be terminated or damage to vital organs occurs (Tucker and Noakes, 2009). Therefore, the subconscious brain regulates the number of motor units recruited, based on a pacing strategy, to allow the athlete to continue exercising in the most efficient way, while ensuring that internal homeostasis and a metabolic and physiological reserve capacity is maintained (St Clair Gibson and Noakes, 2004).

Recently, it has been shown that pacing during self-paced exercise is influenced by the rate of heat storage and thus body temperature (Tatterson *et al*., 2000; Marino *et al*., 2004; Tucker *et al*., 2004). Athletes are proposed to select an exercise intensity that will prevent changes to the various physiological systems occurring that may limit performance (Tucker and Noakes, 2004). This reduction in muscle recruitment begins almost from the onset of exercise and progressively develops until the muscles can no longer perform the required exercise (Gandevia, 2001). However, this model does not properly explain the development of fatigue during certain types of exercises, for example intermittent exercise, which requires athletes to produce very high forces or power outputs for brief periods of time (Noakes and Tucker, 2008). In a study by Brown (2009), on rugby players performing an 80-min simulated

rugby protocol, the muscle activity of the quadriceps muscles (vastus lateralis and vastus medialis) was shown to increase during fatigue, while the muscle activity of the hamstring muscles (biceps femoris and semitendinosus) decreased over time (Brown, 2009). Furthermore, in a study by Girard *et al*. (2010), on squash players, it was shown that a progressive decrease in the normalized muscle activity (measured via surface electromyography (EMG)) of the knee extensor muscles (two vastii) and muscle strength occurred over time, suggesting that central fatigue mechanisms do exist during intermittent exercise.

Ratings of Perceived Exertion and Central Regulation

According to Marcora *et al*. (2009), central and/or peripheral muscle fatigue may not cause exhaustion during high-intensity aerobic exercise, as proposed by Noakes and St Clair Gibson (2004). Rather, it has been proposed that exercise tolerance in highly motivated individuals is limited by subjective perceptions of effort (RPE) (Marcora, 2008; Marcora *et al*., 2008; Marcora *et al*., 2009). Therefore, exercise is regulated via the selection of an appropriate pacing strategy, based on subjective perceptions of effort (Tucker and Noakes, 2009).This information produces a conscious perception of effort, which regulates the amount of skeletal muscle activated during the exercise (Noakes and Tucker, 2008). However, this is dependent on the athlete‘s previous experience and subjective perceptions of effort (Tucker and Noakes, 2009).

During exercise in the heat, at a constant rating of perceived exertion (RPE), the brain adopts different pacing strategies, based on the afferent feedback from the skin thermoreceptors (Tucker *et al*., 2006). Therefore, anticipatory mechanisms exist that regulate exercise intensity before the body temperature reaches critical levels (Tucker *et al*., 2006). Within a few minutes after the initiation of exercise, the rate of heat storage becomes regulated, so that it can remain constant throughout the exercise, regardless of environmental conditions (Tucker *et al*., 2006). At the beginning stages of exercise, muscle power output progressively decreases, so that athletes can finish the exercise with similar core temperatures and ratings of

perceived exertion (Tucker *et al*., 2004). However, this regulation of body temperature through the regulation of exercise may negatively impact the athlete‘s overall performance during competitive sports (Tucker *et al*., 2004). Therefore RPE is an important factor of the central governor theory, as it can be used to predict the duration and intensity of exercise, based on the rate of rise in RPE (Crewe *et al*., 2008), and provides a conscious perception of the possible effects of the many physiological changes that develop during exercise (Noakes and Tucker, 2008). The sensation of fatigue is suggested to be the sensory representation of the underlying neural integration processes, rather than a defined and measurable physical event, that is reduced skeletal muscle force output (St Clair Gibson and Noakes, 2004). Exercise activity is controlled as part of a pacing strategy, where active neural calculations are made in the governor region of the brain, which integrates internal sensory signals and information from the external environment to produce an acceptable exercise intensity that will continue to allow homeostasis to be preserved at a level of sensory effort that the individual finds to be within acceptable limits (St Clair Gibson and Noakes, 2004). Using the end point as a reference point, the central nervous system continuously modifies the work rate and metabolic demands of the various physiological systems by altering the number of motor units recruited during exercise (St Clair Gibson and Noakes, 2004).

During exercise, the subconscious brain determines the metabolic cost that is required to perform the exercise bout, taking into consideration the environmental conditions and present physical condition of the individual, and selects an appropriate pacing strategy (St Clair Gibson and Noakes, 2004). However, the regulation of the pacing strategy remains poorly understood, as much of the classical physiology studies have focused on the factors that ultimately limit, rather than regulate exercise performance (Tucker and Noakes, 2009). When exercise is self-paced and the work rate is free to change in response to external and internal physiological cues, then a complex system alters exercise intensity to prevent changes in physiological systems that may be limiting or detrimental to exercise performance (Tucker and Noakes, 2009). Therefore, a centrally mediated pacing strategy for self-paced exercise of all durations is proposed to balance the requirement for optimal performance with the requirement to complete the task

without causing chronic damage to muscles or vital organs occurring (Tucker and Noakes, 2009).

Central Fatigue During Heat Stress

Tucker *et al*. (2004) found that cycling power output and muscle activity (measured via electromyography) decreased in hot (35° C) environmental conditions compared to cool (15° C) conditions during a 20-kilometre (km) cycling time-trial. An interesting finding in this study was that core temperatures were similar in both the hot and cool environmental conditions and remained well below the proposed critical core temperature of 40° C, possibly due to mechanisms regulating exercise intensities to maintain thermal homeostasis (Tucker *et al*., 2004). Therefore, hot environmental conditions have shown to influence exercise capacities, as an appropriate pacing strategy is selected so that critically high body temperature are prevented from being reached (Tucker and Noakes, 2009). According to Tucker *et al*. (2006), body temperature and the rate of heat storage are suggested to be the main factors regulating exercise in all environmental conditions when the end point of the exercise is unknown at the start.

Central Fatigue During Exercise-Induced Hyperthermia

During exercise, some motor units are recruited while others remain de-activated, which delays the onset of muscular fatigue (Tortora and Derrickson, 2006). This allows the activation of an entire muscle to be sustained for an increased period of time. During exercise, the central governor has been proposed to reduce muscle force production and work rates, through the regulation of muscle activity, so that the exercise can be completed while maintaining thermal homeostasis (Tucker *et al*., 2004). However, this protective mechanism may not only decrease performance capacities (Nybo and Nielsen, 2001a), but also increase the risk of injuries. The central governor has been used to explain fatigue and decreased exercise capacity during continuous exercise; however, due to the intermittent nature of rugby, this model may not be appropriate for describing the development of fatigue in rugby as

rugby is predominantly dictated by its unpredictable nature, making it difficult for players to select an appropriate pacing strategy.

Central fatigue, during exercise-induced hyperthermia, has been proposed to occur at either a conscious or unconscious level (James *et al*., 1995). At the conscious level, the individual decides that the sensations experienced during the activity are unacceptable and voluntarily reduces exercise intensity (James *et al*., 1995). In contrast, at an unconscious level, the afferent information from the active muscles, joints or tendons inhibits muscle activity, causes he exercise intensity to be forcibly reduced. At an unconscious level, no amount of voluntary effort can overcome this regulatory process (James *et al*., 1995).

Two different models have been used to try and explain the reduction in performance capacities during exercise in the heat. The first model proposes that during exercise in the heat, performed at a constant work rate, fatigue begins when an excessively high core temperature is reached, which causes decrements in muscle recruitment and muscle force (Bruck and Olschewski, 1987; Nybo and Nielsen, 2001a). Previous studies (Nybo and Secher, 2004; Nybo and Nielsen, 2001a) suggest that reaching a critical temperature of approximately 40º C during exercise causes a reduction in muscle recruitment, which serves to regulate the athlete‘s work rate. In the second model, exercise is self-paced, and the exercise work rate (Tatterson *et al*., 2000; Tucker *et al*., 2004) or force production (Morrison *et al*., 2004) becomes reduced before excessive heat accumulation occurs. Therefore, this model proposes that the work rate does not decline due to the attainment of a critically high core temperature, but rather that the athletes work rate is regulated by central mechanisms to prevent excessive heat storage from occurring in the first place (Tucker *et al.,* 2004). Thus, even during mild hyperthermia or heat strain, the central nervous system may be impacted without a critical core temperature of approximately 40º C being reached (Nybo and Nielson, 2001).

In contrast, other studies found muscle activity to decrease during exercise, without reaching critically high body temperatures. In a study by Tucker *et al*. (2004) it was found that muscle activity of the vastus lateralis significantly decreased at 10- and 20-minutes of a cycling protocol, despite core temperatures being well below 40º C (Tucker *et al*., 2004). Therefore, reduced muscle recruitment during exercise in the heat cannot be explained by the direct effects of reaching a critical core temperature, as previously hypothesized (Bruck and Olschewski, 1987; Nielson *et al*., 1990; Cheung and Sleivert, 2004). Rather, the early decline in power output has been attributed to an anticipatory response in the brain, causing a reduction in muscle recruitment, ensuring that the rate of heat production is decreased (Tucker *et al*., 2004). This serves to prevent an early onset of fatigue or heat-related illness, even during exercise in the heat (Tucker *et al*., 2004).

Fatigue has been proposed to be part of an active neural process that functions to prevent the development of absolute fatigue and organ damage (Noakes and St Clair Gibson, 2004). Most researchers studying central fatigue measure the electromyographic (EMG) changes in active muscles during submaximal or maximal muscle activations, assuming that these changes reflect alterations in central neural drive (St Clair Gibson, 2004). Therefore, the central fatigue model predicts that muscle activity decreases during prolonged exercise due to a decrease in central drive to the muscles (St Clair Gibson and Noakes, 2004). The reduced muscle function is therefore, secondary to the reduction in central neural drive (St Clair Gibson and Noakes, 2004). However, it has been argued that central fatigue may not limit performance during dynamic muscular activations, as most individuals consciously decide to limit work rate and minimize the discomfort of exercise (James *et al*., 1995). Similarly, motivated subjects have shown to be able to override the desire to reduce activity (James *et al*., 1995).

PHYSIOLOGICAL RESPONSES DURING EXERCISE AND HEAT STRESS

From a physiological standpoint, athletes experience the most stress during exercise in the heat (Maughan and Shirreffs, 1997), especially while wearing clothing and

protective gear, which hinder mechanisms of heat dissipation (Givoni and Goldman, 1972).

Body Temperature

The ability to sense and control body temperature is a key feature of human survival (Lim *et al*., 2008). Mean body temperature can be calculated using skin temperature, which accounts for 40 % of the mean body temperature, and core temperature, which accounts for 60 % (Brown *et al*., 2006).

CORE TEMPERATURE

Body temperature measurement is fundamental to the study of human thermoregulation during exercise (Sawka and Wenger, 1988) because an excessively high core temperature have shown to negatively impact the performance capacities of athletes (Gonzalez-Alonso *et al*., 1999). The ideal site for core temperature measurement is one that is convenient, is not affected by changing environmental conditions, responds rapidly and shows small changes in core temperature (Sawka and Wenger, 1988). Core temperature is often measured at the oesophagus, rectum, mouth, or external auditory tympanic membrane (Sawka and Wenger, 1988).

An alternate method of core temperature measurement is the ingestible telemetric temperature pill, which is ingested and transmits a temperature signal, relative to the surrounding gastrointestinal temperature, by radio wave to an external receiver for data logging (Byrne and Lim, 2007). This method has become popular in field-based studies, as once the pill is swallowed, individuals are unaware of its presence, and has been described as an excellent alternative to more invasive methods of temperature measurement (Byrne and Lim, 2007). The ingestible telemetric temperature sensor has shown to be a convenient and valid index of core temperature (Byrne and Lim, 2007). Several other studies have used this method of core temperature measurement during various sports such as football (Godek *et al*.,

2004) and cross country (Godek *et al*., 2006), soccer (Edwards and Clark 2006; Byrne *et al*., 2006) and during an Ironman triathlon (Laursen *et al*., 2006).

Deviations of approximately 3.5° C from resting skin temperatures (approximately 37° C) can result in physiological impairments and even death (Moran and Mendal, 2002). During physical exercise, human‘s ability to maintain thermal homeostasis becomes critical for survival and the ability to continue work (Lim *et al*., 2008). According to Shapiro and Seidman (1990), body temperature can increase from resting (37° C) to temperatures greater than 42° C, where cell damage occurs and the central nervous system becomes impaired. During exercise, metabolic heat production can increase by as much as 10- to 20-fold (Sawka and Wenger, 1998). However, less than 30 % of this heat generated during exercise is converted into mechanical energy (Sawka and Wenger, 1998). More than 70 % of metabolic heat is transferred to the body‘s‘ periphery (skin) for heat dissipation (Lim *et al*., 2008). When the heat dissipatory mechanisms of the body are unable to cope with this metabolic heat production, body heat starts to accumulate in the body, causing core temperatures to rise (Lim *et al*., 2008).

Hyperthermia has been suggested to function as a trigger for central fatigue during exercise (Nielson and Nybo, 2003). When the body‘s threshold temperature is reached, central mechanisms inhibit exercise performance by limiting cardiac functions, to protect the body from overheating (Gonzalez-Alonso *et al*., 1999). This forces a reduction in exercise intensity, which prevents any further increases in body temperature occurring (Lim and Mackinnon, 2007). In a study by Gonzalez-Alonso *et al*. (1999), it was found that fatigue in trained athletes occurred at similar core temperatures (approximately 40° C), despite there being variability among the athlete‘s pre-exercise body temperatures or how quickly body temperatures rose. This indicates that a critical core temperature exists where decrements in performance occur due to mechanisms of central fatigue.

Hughson *et al*. (1980) suggests that the duration and intensity of exercise contribute significantly to the amount of heat accumulated in the body during exercise. The type of exercise has also been said to influence thermoregulatory responses (Smolander *et al*., 1991). Intermittent exercise is suggested to place increased physiologic strain on the human body during exercise, compared to a continuous exercise bout of the same energy expenditure (Kraning and Gonzalez, 1991; Edwards *et al*., 1973; Bangsbo, 1994). Intermittent exercise has been associated with increased body temperatures when compared to continuous exercise of a similar work-rate (Ekblom *et al*., 1971; Cable and Bullock, 1996, Drust *et al*. 1999). However, according to Drust *et al*. (2000), core temperatures do not increase more during intermittent exercise, due to the stop/start nature of the exercise. In this study soccer players performed a 45-min soccer-specific protocol, and a 45-min continuous treadmill protocol, performed at the same average speed (Drust *et al*., 2000). It was shown that player‘s core temperature responses were similar during both exercise protocols (Drust *et al*., 2000). This suggests that rates of metabolic heat production and heat dissipation were equivalent during both types of exercise, and is not affected by the stop/start nature of intermittent exercise (Drust *et al*., 2000).

In a study by Ozgunen *et al*. (2010), soccer player‘s core temperatures increased rapidly from kick-off during two matches played in moderate and hot environmental conditions, respectively, and did not show any significant differences during the first 15-20-min of play, despite significant differences between environmental conditions. However, in the second quarter of the match, core temperatures continued to rise in the hot environmental conditions, whereas in the moderate conditions, core temperatures remained fairly constant (Ozgunen *et al*., 2010). In both matches, core temperatures fell during the half-time break, and did not return to the peak first-half levels (39.1 ±0.4° C in the moderate environmental conditions and 39.6 ±0.3° C in the hot environmental conditions) during the second half of the match (Ozgunen *et al*., 2010).

In a study by Edwards and Clark (2006), it was found that recreational soccer players core temperatures continued to increase from half-time (45-min) to full-time

(80-min), whereas professional players‘ core temperatures reached a steady-state. This was suggested to be from the superior heat acclimatisation in the professional players (Edwards and Clark, 2006).

Cardiovascular Responses

Increases in heart rate during both intermittent and continuous exercise occur due to a progressive reduction in stroke volume throughout the exercise bout (Rowell, 1990). This is due to blood being redistributed towards the skin to aid in heat dissipation (Rowell, 1990). The effects of heat stress on thermal and cardiovascular strain are closely related, especially during high intensity exercise (Cheung and Sleivert, 2004). Muscle activation is known to be the greatest contributor to physiological responses (McArdle *et al*., 2006). From a performance perspective, cardiovascular strain accompanying hyperthermia could cause these physiological responses to become further exacerbated and possibly cause athletic performance to become hindered (McArdle *et al*., 2006). Therefore, muscle activity during intermittent exercise, combined with the clothing and protective gear worn in rugby, may increase the cardiac load on experienced by players (Cannon and Christie, 2010). This increased cardiac strain means that the cardiorespiratory system is no longer able to provide the exercising muscles with enough oxygen, which causes the anaerobic metabolism to be induced, which is associated with the development of fatigue (Noakes, 2000). During intermittent activity, cardiovascular strain is suggested to be greater than during steady-state exercise performed at the same exercise intensity (Drust *et al*., 2000). This may be due to intermittent exercise producing staggered increases in heart rate, while maintaining a constantly high heart rate during low-intensity recovery periods (Drust *et al*., 2000).

Heart rate responses increase in hot or humid conditions, due to increased skin temperatures (Christie and King, 2008). Even at rest, heart rates and cardiac output are shown to increase in the heat, while superficial arterial and venous blood vessels dilate to divert warm blood to the periphery (McArdle *et al*., 2006). During intense exercise in the heat, circulation must try and supply increased cardiac output to the

active muscles as well as to the skin for heat dissipation (Kenney and Johnson, 1992). This may increase the cardiovascular strain being placed on athletes during exercise in the heat. Heart rates, while wearing full rugby gear and minimal rugby gear, have been shown to be similar during a 40-min simulated rugby protocol, except towards the end stages, where heart rates were significantly higher in the full rugby gear. This suggests that the players heart rates were unaffected by the additional clothing and protective gear up until the latter stages of the protocol (Cannon and Christie, 2010). During the intermittent exercise bout in minimal clothing and no protective gear, heart rates stabilized at 20-minutes and then began to drop towards the end of the protocol. This suggests that players‘ heart rates reached a ‗steady-state‘, despite the intermittent nature of the protocol (Cannon and Christie, 2010). Therefore, towards the end of the protocol, player‘s heart rates continued to rise when wearing the full rugby gear and protective gear, due to the increased heat load, which exacerbated player‘s cardiovascular responses (Cannon and Christie, 2010).

PERFORMANCE RESPONSES

It has been previously shown that short duration, dynamic exercise performance is improved when the muscles are pre-warmed (Cheung and Sleivert, 2004). However, due to the exercise intensity or duration increasing, or factors that limit heat dissipation, the physiological strain placed on the body may increase due to higher body temperatures, which has shown to negatively impact athlete‘s exercise capacities (Cheung and Sleivert, 2004, Morris *et al*., 1998). This anticipatory reduction in work rate, as a result of excessive heat gains, was shown by Tucker *et al*. (2004), where exercise capacity was reduced in hot (35 ºC) environmental conditions, compared to the same exercise in cool (15 ºC) or normal (25 ºC) conditions. Therefore, athletes‘ performance capacities were shown to be significantly hindered by heat stress, because of the anticipatory reduction of muscle power (Tucker *et al*., 2004).

In a study by Ozgunen *et al*. (2010) during a soccer-match played in moderate environmental conditions and another match played in hot conditions. During both matches, player‘s performance capacities decreased during the second half of the match, however the reductions were significantly greater in the match played in the hot environmental conditions (Ozgunen *et al*., 2010). This could be explained by the significantly higher body temperatures being reached in the match played in hot conditions compared to the match played at moderate temperatures (Ozgunen *et al*., 2010). This confirms the theory proposed by Noakes *et al*. (2005), which suggests that players‘ work rates decrease at elevated when body temperatures.

In a study by Cannon and Christie (2010), it was shown that rugby players‘ performance capacities (total number of 20m shuttles completed during protocol) were negatively impacted at high body temperatures, due to the additional clothing and protective gear worn during a simulated 40-min rugby protocol. In contrast, while performing the same exercise work-bout in minimal clothing and no protective gear (control condition), players performance capacities were significantly better, possibly due to significantly lower body temperatures being reached (Cannon and Christie, 2009).

CONCLUSION

Despite rugby being a very popular sport, it still remains very under researched. The clothing and protective gear worn in rugby has shown to exacerbate players physiological and perceptual responses, and negatively impact performance capacities due to increased body temperatures, which may have hastened the onset of fatigue. However, the mechanisms behind these performance decrements remain unclear. Therefore, the current research aimed to determine the effect that the clothing and protective gear has on players biophysical, physiological, perceptual and performance responses.

CHAPTER III

METHODS

PILOT TESTING

Prior to testing, extensive pre-pilot and pilot studies were performed in the laboratory. During these pilot studies, the physiological, perceptual and performance responses of university level rugby players were measured while wearing two different rugby kits (full rugby clothing and protective gear versus minimal rugby clothing and no protective gear) during a 40-minute (min) simulated rugby protocol designed and validated by Brown (2009). This protocol only incorporated the locomotor aspects of the game, including standing still, walking, jogging, striding and sprinting. During this study, it was discovered that player's physiological and perceptual responses were exacerbated while wearing the full rugby clothing and protective gear (Cannon and Christie, 2010). Based on this study, several questions arose specifically surrounding the performance decrements as a result of the additional heat strain caused from wearing the rugby clothing and additional protective gear during the simulated rugby protocol.

EXPERIMENTAL DESIGN

Rugby is intermittent and varied in nature and involves repeated high-intensity sprints which produce a variety of physiological responses during match-play (Duthie *et al*., 2007). It has been described as complex and chaotic, with conditions changing from game to game and even from phase-to-phase (Vaz *et al*., 2010). Due to the nature of rugby, along with most other intermittent sports, the mechanisms of fatigue remain largely unclear (Morris *et al*., 1998; Girard *et al*., 2008). According to Duthie *et al*., (2005), rugby is stop-start in nature with players rapidly changing direction, suddenly braking to stay on-sides and rapidly accelerating to sprint. These movements result in repeated eccentric demands being placed on the musculature of players lower limbs. In a study by Girard *et al*. (2008) on tennis players, it was

shown that prolonged tennis play induced significant reductions in muscle activity (vastus lateralis, vastus medialis and biceps femoris) from the start of exercise (0-min) to 150-min. Interestingly, at 180-min (end of the exercise bout) it was found that the muscle activity almost returned back to initial levels (Girard *et al*., 2008). Brown (2009) found that a rugby activity profile (locomotor only) caused no changes in the activity of the quadriceps musculature. However, the simulated rugby activity caused the muscle activity of the bicep femoris and semitendinosus (hamstrings) to increase, and the strength of the hamstrings to decrease. Interestingly, in a study by Cunniffe *et al*. (2009), it was found that hamstring injuries were the second most commonly occurring injuries in rugby players, and that the majority of these injuries occurred during the second half of rugby match-play, possibly due to fatigue. This suggests that the changes in muscle activity and the decrements in muscle power that accompany fatigue may be responsible for this increased risk of injury (Grieg *et al*., 2006).

According to Brotherhood (2008) after the initiation of exercise in moderate environments, body temperatures increase for approximately 20-minutes and then stabilize, which indicates that thermal balance has been achieved (Brotherhood, 2008). In a study on cricket players, it was suggested that stress over a short duration of 24-minutes or less does not negatively impact performance (Christie and King, 2008). However, because rugby matches are highly intermittent in nature, are 80-min in duration and often involve players wearing protective gear, their responses may be further exacerbated by the longer duration of exercise (80-min), varied intensities of exercise and the clothing and protective gear worn. Therefore, the simulated protocol comprised of four 20-min work bouts, with the exercise intensity progressively increasing over time.

Reaching a critical core temperature of approximately 40° C during continuous exercise has shown to negatively impact the performance capacities of athletes (Gonzalez-Alonso *et al*., 1999) although this has not been demonstrated during intermittent exercise. Intermittent and continuous exercise both require muscle activation, however the extent to which this occurs during intermittent activity has not

been fully elucidated. During soccer match-play, it has been shown that players‘ core temperatures increase significantly during the first quarter of match-play, and then shows small increases throughout the remainder of the match, so that peak core temperatures (39.3 +/-0.7° C) were achieved during the final quarter of the match (Duffield *et al*., 2009). Exercise intensities were high during the first quarter of the match, stabilized during the middle stages, and then increased slightly during the final quarter of the match (Duffield *et al*., 2009). Increases in heart rate and reductions in stroke volume occur when core temperatures rise towards 36-40 °C (Duffield *et al*., 2009). An excessively high core temperature during exercise may result in reduced central muscle activation and a subsequent decrease in exercise capacities (Tucker and Noakes, 2009). When core temperature reaches approximately 38° C, skin blood flow, and therefore heart rate responses have shown to plateau (Gonzalez-Alonso *et al*., 1999). This indicates that a high internal body temperature may be responsible for fatigue in athletes during prolonged exercise in the heat (Gonzalez-Alonso *et al*., 1999). Therefore, during competitive team sports of an intermittent nature, core temperature may be regulated, and a pacing strategy employed to ensure that the internal heat load of players can be regulated (Duffield *et al.*, 2009).

It remains unclear as to whether the clothing and protective gear worn during rugby causes players responses to become exacerbated. This may cause an earlier onset of fatigue, which could reduce player‘s performance capacities and increase their risk of injury (Roig Pull and Ranson, 2007). Previously, three major factors were suggested to cause relative hyperthermia in rugby players. These included the degree of dehydration, degree of acclimatization and the clothing worn (Cohen *et al*., 1981). In a study by Goodman *et al*. (1985), it was shown that the clothing worn in rugby has a greater influence on the thermoregulatory responses of players than was previously thought, largely eliminating dehydration and lack of acclimatization as main factors of hyperthermia (Goodman *et al*., 1985). It was found in a study by Mathews *et al*. (1969) that rectal temperature, skin temperature, heart rate and total fluid loss were lower during exercise in shorts, compared to the same exercise bout in full football uniform. Cannon and Christie (2010) found that the clothing and protective gear worn during a simulated rugby protocol exacerbated players‘

physiological responses, more so than when players performed the same exercise protocol in only shorts (Cannon and Christie, 2010). Furthermore, player‘s performance capacities were hindered while performing the rugby activity in full rugby gear (Cannon and Christie, 2010). Therefore, this suggests that the increased heat stress, caused by exercising in full rugby gear, may be responsible for hastening the onset of fatigue and hinder overall performance capacities.

Fatigue remains poorly understood, and has been classified as being either central or peripheral in origin (Kay *et al*., 2000). During ongoing dynamic exercise, the differentiation between central and peripheral factors contributing to fatigue becomes more difficult (Nybo and Secher, 2004). More recently, it has been proposed that central fatigue causes a reduction in muscle power during prolonged exercise, which results in the termination of exercise, and is not caused by limiting physiological processes in the peripheral organs, but rather from central processes (St Clair Gibson and Noakes, 2004). Fatigue, experienced during exercise-induced heat stress, has been suggested to be caused from central mechanisms, which down-regulates the amount of skeletal muscle recruited during exercise, and ultimately hinders athletic performance. This is based on the central governor theory, proposed by Noakes (Noakes, 1996; Noakes, 1998; Noakes, 2000), which suggests that during exercise-induced heat stress, athlete‘s work rates becomes reduced through the regulation of skeletal muscle recruited during exercise, thus ensuring that a potentially catastrophic failure of homeostasis does not occur (St Clair Gibson *et al*., 2001; Noakes *et al*., 2004; St Clair Gibson and Noakes, 2004; Tucker *et al*., 2004; Lambert *et al*., 2005; Tucker *et al*., 2006). However, this central governor has been used to explain fatigue during continuous exercise, and may not be appropriate for describing the onset of fatigue during intermittent activity, because of its unpredictable nature.

EXPERIMENTAL CONDITIONS

Two conditions were tested and were referred to as the experimental condition (EC) and control condition (CC). Players were required to complete the same work bout

for both test conditions, so that the impact of the clothing and protective gear could be observed. During the CC, players wore rugby shorts, underpants, short socks and shoes (minimal clothing and no protective gear), while during the EC, players wore full rugby gear, which included clothing (rugby jersey, shorts, socks, shoes and underpants), as well as protective gear (shoulder pads, scrumcap and thermal top). Player‘s never wore mouth guards during the EC. The kit worn during the CC (Figure 1a) was regarded as the best case scenario for thermoregulation during exercise, while the rugby kit worn during the EC (Figure 1b) was regarded as the worst case scenario. Therefore, although this was not ecologically valid, as players do not play rugby in the CC kit, it was done to demonstrate the physiological phenomenon of wearing full rugby gear during a simulated rugby protocol, rather than having application to the real world. Because the rugby clothing, produced by the same manufacturer, is relatively similar to each other, players were permitted to provide their own rugby clothing during the EC. In contrast, the protective gear worn during the EC was standardised. Therefore, all players wore the same protective gear during the EC, so as to avoid any variations in the thermoregulatory responses of players occurring, due to different items of protective gear being worn.

Figure 1: The control (a) and experimental (b) conditions kit

WORK-BOUT

The work-bout used in this study was modified from the protocol designed in our laboratory by Brown (2009) and from time-motion data from Cunniffe *et al*. (2009). The protocol was designed to match the locomotor demands of competitive match-play as closely as possible. Only the non-contact rugby activities were included, namely standing still, walking, jogging, striding, and sprinting. Of the 80-minutes, 62 % consisted of standing and walking, 22 % jogging, 8 % striding, and 7.6 % sprinting. The simulated rugby work-bout consisted of four 20-min exercise bouts (Appendix A), matching the actual duration of a match. The first and second work-bouts were identically matched, whereas the third and fourth quarters differed in intensity, according to the time-motion data obtained from the Cunniffe *et al*. (2009) study. Between the first and second quarters, as well as between the third- and fourth-quarter there was a 2-min rest interval. The half-time interval (between the second and third bout) matched that of an actual rugby game, being 10-minutes in duration. Table I summarizes the activities of the first and second quarters of the protocol.

Table I: The activities of the first and second quarter of the rugby work bout (taken from Brown, 2009).

Activity	**Duration (%)**	**Time (min:sec)**
Standing	47	9:24
Walking	18.25	3:39
Jogging	19.2	3:50
Striding	8.3	1:40
Sprinting	7.25	1:27
Total	**100**	**20:00**

Table II shows the activities involved in the third and fourth quarters of the simulated rugby protocol. According to Cunniffe *et al*. (2009), during the second half of a rugby match, players are involved in more high intensity activities than the first of a match. Furthermore, during the second half of a match, players were shown to have less rest breaks compared to the first half (Cunniffe *et al*., 2009). Thus, the first and second 40-min of the simulated protocol was modified to accommodate for these differences. Cross-quarter comparisons showed that the third quarter (after half-time) was more intense than the fourth (Cunniffe *et al*., 2009). All players performed the same protocol for both test conditions, so that the impact of wearing protective gear and clothing during a simulated rugby protocol could be determined. By having the players perform the same exercise protocol, the effect of the kit alone on player‘s responses could be ascertained, thus removing the differing exercise intensities as a variable impacting player‘s responses, if the exercise protocol differed between forwards and backs.

Table II: The modified activity profile for the third and fourth quarters of the simulated rugby protocol (Adapted from Brown, 2009)

	Third Quarter		**Fourth Quarter**	
Activity	**Duration (%)**	**Time (min:sec)**	**Duration (%)**	**Time (min:sec)**
Standing	40.5	08:10	37.6	07:31
Walking	18.6	03:43	19.4	03:53
Jogging	25.1	05:01	25.4	05:05
Striding	8.2	01:38	9.3	01:51
Sprinting	7.6	01:32	8.33	01:40
Total	**100**	**20:00**	**100**	**20:00**

EQUIPMENT AND MEASUREMENT PROTOCOL

DEMOGRAPHIC DATA

At the initial testing session, general demographic data was obtained for each player, including age, the number of years playing rugby, position played and any injuries or illnesses (current or past). A physical activity screening questionnaire was also completed by each player to determine injury risk and overall health status (Appendix A).

ANTHROPOMETRIC DATA

Body Mass

The body mass of each player was measured using a calibrated electronic Toledo® Scale (model 8142). All players were required to remove their shoes, accessories and heavy clothing before being weighed. Each player then stood still in the middle of the scale, in an upright position and their body mass was recorded.

Stature

Stature was measured using a Harpenden Stadiometer and was measured to the nearest millimetre (mm). Players were asked to remove their shoes, stand on the stadiometer in an upright position facing forwards, with their heels pressed against the base of the stadiometer and their heads facing forwards. Stature was measured from the floor to the vertex in the mid-sagittal plane.

Body Mass Index (BMI)

BMI was calculated by using measures of stature and body mass and using the following equation: BMI= Mass (kg)/Stature $(m)^2$

MORPHOLOGICAL DATA

Body Composition

Body composition was measured via the skinfold technique using Harpenden Skinfold callipers (Quinton Instrument, Seattle, Washington, USA). Seven skinfold sites were measured, namely the chest, triceps, subscapular, suprailiac, abdominal, thigh and medial calf. All measures were taken on the right hand side of the player's body. Skinfold callipers were placed 10 millimetre (mm) away from the thumb and index finger, perpendicular to the skinfold, and halfway between the crest and base of the anatomical site. Each site was measured three times, and if the re-test measures were not within a 3 % error margin, the measurement was repeated. Body density was then calculated using the following equation, devised by Jackson and Pollock (1978):

$$BD = 1.11200000 - 0.00043499 (\text{sum of 7 skinfolds}) + 0.00000055 (\text{sum of 7 skinfolds})^2 - 0.00028826 (\text{age})$$

This figure was then used in the Siri Equation (Siri, 1961) to calculate body fat percentage (%). This was done by using the following equation:

$$\text{Fat (\%)} = [(4.95/\text{density}) - 4.51] \times 100$$

MUSCULOSKELETAL PARAMETERS

Muscle Activity

A surface electromyography (EMG) device (Muscle Tester Mega ME6000P16, Mega Electronics Ltd, Finland) was used to measure muscle activation. Muscle activation was measured by attaching EMG electrodes to the surface of the skin and measuring the electrical activity of the muscle directly below them. Prior to testing, the skin areas on the players dominant leg had to be prepared, by shaving and then cleaning the area with alcohol swabs which were allowed to dry to reduce electrical impedance. To assess the changes in electrical activity, two disposable Silver-Silver electrodes (Kendall, Meditrace 200) were attached to the skin above the muscles.

The vastus medialis and vastus lateralis were assessed to determine muscle activation of the quadriceps. The biceps femoris and semitendinosus were measured to determine the muscle activation of the hamstrings. These muscles were selected due to the proximity to the surface of the skin. Electrodes were placed at a set inter-electrode distance of 20 mm. Electrode placement for the vastus medialis was 20 % of the distance from the medial joint line from the knee to the anterior superior iliac spine. The vastus lateralis placement area was at the midpoint between head of the greater trochanter and the lateral femoral epicondyle. Electrode placement for the bicep femoris and semitendinosus was 50 % of the distance from the ischial tuberosity to the medial and lateral femoral epicondyles, respectively. A third „neutral' electrode was than attached at least 100 mm away from the active muscle and positioned on an inactive muscle. The Megawin© Software was used together with the electromyography equipment and a wireless transmitter was connected to a laptop computer, which linked the equipment. These muscles were measured prior to the commencement of the rugby protocol, and then during the half-time interval (at 40-minutes), and then again after the protocol at 80-minutes.

Electromyographic raw data activity was amplified at 1000 Hz, and was recorded telemetrically (Muscle Tester „Mega ME6000P16", Mega Electronics Ltd, Finland). The raw Electromyographic signal was filtered using a „data processing filter system', followed by a 15-350 band pass filter. The amount of EMG activity was quantified by determining the area under the curve observed for EMG (µV) vs. time (sec), resulting in an integrated EMG (iEMG) measurement. The root mean square (RMS) of the EMG signal was obtained at each time interval (pre-, half-time and post-protocol) for both conditions to make comparisons. The units used to interpret the EMG data were arbitrary units (a.u).

This EMG protocol used in this study was a treadmill protocol designed to assess lower limb muscle fatigue after an intermittent, soccer-specific exercise protocol (Rahnama *et al*., 2006, Table III).

This protocol was validated by Brown (2009), for Rugby Union, and for use in this study. Players were required to perform the EMG protocol (Table III) on a treadmill. The speeds for the protocol were 6 km.h^{-1} (walking), 12 km.h^{-1} (jogging), 15 km.h^{-1} (cruising) and 21 km.h^{-1} (sprinting). The speed of 5km.h^{-1} was used as an interface between the measured speeds of 6, 12, 15 and 21 km.h^{-1} in order to mimic the intermittent activity profile. In order to assess muscle activation changes over time, the EMG protocol was performed three times during each condition (pre, half-time and post-protocol).

Table III: The EMG measurement protocol (Rahnama *et al*., 2006).

Time (min:s)	Speed (km.h^{-1})	Duration (min:s)
00:00	6	01:10
01:10	5	00:20
01:30	12	00:15
01:45	5	00:20
02:05	15	00:15
02:20	5	00:20
02:40	21	00:15
02:55	5	00:05
03:00	**STOP**	

PHYSIOLOGICAL PARAMETERS

Cardiovascular Responses

The CorTemp™ Data Recorder HT150016 (HQ Inc, Florida, USA) was used to measure all of the heart rate data. This device allowed the players to perform the protocol without having to wear a watch, as all of the data was downloaded from the belt worn around the player‘s chest and sent to a data recorder. On arrival, each player had the heart rate monitor belt attached over the chest. The belt strap was adjusted so that it was secure and made contact with the skin. Reference heart rate

was recorded prior to testing. Players were instructed to lie down and relax for a minimum of two minutes and allow heart rate values to reach as close to resting values as possible. During the two minutes, the lowest recorded heart rate was used as the player‘s reference (resting) heart rate to predict players‘ fitness levels. Heart rates were recorded at 5 second (s) intervals during both the experimental and control protocol. Heart rate was used as an indicator of the physiological effort required to perform the rugby protocol.

Core Temperature

Core temperature was measured using the CorTemp™ Data Recorder HT150016 (HQ Inc, Florida, USA). This required players to ingest a Core Body Temperature Sensor HT150002 (CorTemp HQ Inc, Florida, USA) and the data was transmitted to an external ambulatory CorTemp™ Data Recorder HT150016. The calibration number and serial number of each telemetric pill was placed in to the core temperature data recorder prior to testing. The telemetric pills signal was then tested by holding the data recorder at the base of the players back.

Players‘ had to ingest the core temperature three hours prior to testing in order for the sensor to gather an accurate core temperature measurement. On arrival to the laboratory, the data recorder was then switched on and held against the player‘s abdomen to ensure that the sensor was transmitting a signal to the data recorder. Core temperature was measured prior to-, at 5-min intervals during the protocol, during the half-time interval, and post-protocol. Data obtained was used to compare core temperature responses between conditions.

PSYCHOPHYSICAL PARAMETERS

Ratings of Perceived Exertion (RPE)

RPE was used to identify perceived physical effort during the test activities. There are two categories for RPE, namely: Central RPE, which focuses on perceptions of

central cardiorespiratory strain, and Local RPE which focus on effort perceptions of selected muscular groups, which in this case was for the lower limbs. The Borg scale (Appendix A) was used to provide the players RPE during the test conditions. This scale ranges from a value of six (almost no strain), to a maximal value of 20 (representing exhaustion), and is the most commonly used psychophysiological rating scales used to assess the strain experienced by players, as it provides a good indicator of the degree of strain players are experiencing during exercise (Borg, 1970). The Borg Scale was explained to all the players in detail before testing commenced. During the test, players were required to rate the work-bout on the numerical scale in order to indicate their level of exertion.

PERFORMANCE PARAMETERS

Player‘s performance responses were calculated by counting the total number of 20m shuttles completed during each 20-min work bout. This was done using an LED system, which was positioned in the middle of the 20m walkway. The walkway, which players were required to run along, was made from plywood. When player‘s crossed the LED beam, the counter would record each shuttle. At the end of each 20-min work-bout, the research assistant would record the total number of shuttles completed. Although the pace was set by the researcher, players were unable to keep up with the pace of the protocol, due to muscle fatigue. This would possibly result in player‘s being unable to perform as many shuttles during the EC, compared to the CC, due to additional heat load caused by exercising in full rugby clothing and protective gear. At the end of the 80-min protocol, the total number of shuttles was calculated, and comparisons were made between conditions to determine if any differences occurred.

CONTROLLABLE VARIABLES

The testing was performed in a controlled laboratory setting. This was done so that extraneous factors could be controlled in as far as possible so as not to affect the results. The temperature range in which testing was to be performed had to be within 16º-22º C. If conditions were either too hot or cold, testing was postponed. This was

done to isolate the effect that the clothing and protective gear had on players during the simulated protocol, and to ensure that the environmental conditions did not impact players body temperatures. The dry temperature and relative humidity was measured before the test session began for each condition, using an Electronic Thermo-Hygrometer. Fluid loss and dehydration has been shown to impact player‘s responses and performance capacities, therefore fluid intake was controlled throughout the 80-min protocol so that it did not impact the results. Players were required to take in adequate amounts of water the day prior to testing, and then 250 ml of water 20-minutes prior to testing, during the half-time interval, and again after the protocol (at 80-minutes).

PLAYERS

The sample consisted of 15 male rugby players from the Grahamstown area in the Eastern Cape Province of South Africa. These players were from the Rhodes University and Graeme College 1st XV rugby teams. Both forwards and backs were used except for front row forwards. All players volunteered to participate in this test, and ethics was granted by the Ethics Committee of the Department of Human Kinetics and Ergonomics, Rhodes University, South Africa (Appendix A). Prior to testing, players were advised that they were free to withdraw from participation at any time without any adverse consequences.

PLAYER SELECTION

There were 15 rugby players recruited from the Rhodes University and Graeme College 1st team rugby squads playing in the 2011 season. The selection criteria were as follows: All players had to have at least six years‘ experience in playing rugby, all players had to be between the ages of 18 and 25 years, and all players had to be actively taking part in the 2011 rugby season as a member of each team‘s rugby squad. Front row forwards were excluded from participation from this test. Any players with recent or recurring injuries to the lower limbs, or any previous surgery to the knee were excluded from participation, as this may have impacted the results of the study. Prior to inclusion in the sample, all prospective participants were required

to complete a physical activity-screening questionnaire (Appendix A). This questionnaire showed that all test players were in good health. Similarly, any players with recent illness, or any health problems that may have placed them at risk during testing were also excluded from participation in this study.

Player Characteristics

Table IV: Means (±SD) for player‘s characteristics

Measure	Mean	Standard Deviation (SD)
Age (years)	20.92	1.88
Rugby experience (years)	9.88	3.54
Mass (kg)	86.48	9.81
Stature (mm)	1816.67	89.84
Body fat (%)	9.42	2.28

ETHICAL CONSIDERATIONS

INFORMED CONSENT

All players were informed both verbally and in writing about the nature of the study (Appendix A). This study was approved by the Ethics committee of the Human Kinetics and Ergonomics Department of Rhodes University, Grahamstown, South Africa. Each player voluntarily gave informed consent to participate and was in no way pressured to do so by other team members, coaches or captains (Appendix A).

PRIVACY AND ANONYMITY OF RESULTS

To ensure the anonymity of players, a coding system was used so that all information gathered from the players could not be traced back to them. The player‘s names on each data collection sheet (Appendix A) were used only for recording data. Players were informed before experimentation that data would be held on file

only for statistical analyses. This data would be deleted after the study was completed, with only one copy being kept and stored in the department.

EXPERIMENTAL PROCEDURES

PHASE 1: INTRODUCTION AND COLLECTION OF BASELINE DATA

All testing was carried out in the Human Kinetics and Ergonomics (HKE) Department at Rhodes University, Grahamstown, South Africa. Players attended a preliminary information session in the HKE department, during which all aspects of the testing procedure and the background of the study was explained in writing (Appendix A) as well as verbally. Following agreement to participate and the signing of the informed consent form (Appendix A), the first stage commenced, which involved the collection of basic data (Appendix A), which included age, playing position and a full injury history of each player. Players were then required to fill out a Physical Activity Screening Questionnaire (Appendix A), so that it could be ascertained whether players were healthy enough to participate in this study.

PHASE 2: EXPERIMENTATION

Players were instructed not to engage in strenuous activity 24-hours (hrs) before being tested, to have avoided alcohol consumption within the 24-hrs before testing, to eat a small meal three hours prior to coming in, and to take in adequate fluids the day before testing, as well just before testing begins. According to The American College of Sports Medicine (1996), approximately 500 ml of fluid should be taken in about 2 hours before exercise, to ensure proper hydration. Prior to testing the researcher asked each player if the requirements had been fulfilled. If any of the requirements were not adhered to, testing was to be postponed to a later date. The players were required to urinate before testing began, to avoid any disruptions during testing.

Prior to the testing session, the order in which the two test conditions were administered was randomly assigned to each player. This served to reduce the possibility that differences found in the responses could be attributed to the different test conditions and not to the order of the test conditions. Players were then required to come to the laboratory for two further test sessions to complete the EC and CC in a random order. During these sessions physiological, biophysical, and perceptual and performance responses were measured.

Before testing could commence, players‘ anthropometric, demographic and morphological (Appendix A) measures were recorded (Appendix A). The, environmental conditions were then measured to ensure that conditions were suitable for testing (Appendix A). If the temperature was not in the desired range, or the humidity was too high, testing was postponed to a later date. Players were required to ingest a telemetric pill, which was used to continuously measure core temperature, three hours before each experimental session, to ensure sensor transit beyond the stomach but not expulsion before data collection (Byrne and Lim, 2007). These pills were given to each player, by the researcher, the day before either test session. The researcher was present at the time of ingestion to ensure that the pills were ingested at the correct time. Just before testing commenced, the researcher checked to see that the pill was transmitting a signal to the handheld data logger. EMG electrodes were attached to the relevant muscle groups and the EMG wires were then attached to these electrodes and secured to the player‘s skin with Fixamol Tape so that there was minimal movement during the normalized treadmill protocol. It was ensured that these wires were not crossing, so as to avoid any interruption in data collection. Players were then fitted with a heart rate monitor belt, placed around their chest.

Before the protocol commenced, player‘s resting heart rates were recorded, following lying on the floor in a supine position for two-minutes. The lowest value for heart rate taken after two-minutes of rest was used as a reference heart rate. Players resting core temperature was then recorded prior to testing, by having the players sit in a cool environment, dressed only in rugby shorts. The researcher held

the core temperature data recorder at the base of the player‘s spine until the core temperature measurement was obtained. Players were allowed to complete their own warm-up, which included stretching exercises performed before the commencement of a rugby- match or training session. Baseline muscle activity was also recorded prior to testing, by having players perform the 3-min EMG treadmill protocol, wearing only rugby shorts. Immediately after this was completed, test players performed either the EC or CC.

During either conditions protocol, players were required to follow the instructions from the researcher to either stand still, walk, jog, stride, and sprint at set time intervals. The researcher verbally instructed the player on what activity to perform, and used a whistle to signal the transition from each activity to the next. Players performed these activities along a 20 m long wooden walkway. Therefore, players were required to change direction once they reached either side of the walkway. Prior to the commencement of the protocol, players‘ were clearly instructed regarding the activities of the protocol. During maximal sprints, the researcher and research assistants provided motivation for the players, ensuring that the players were performing maximally. The biophysical, physiological, perceptual and performance responses obtained during the EC by the rugby players were compared to the same responses obtained during the CC.

During the control and experimental test conditions, core temperature and heart rate responses were measured at 10-min intervals throughout the 80-min protocol. Players central and local RPE and were measured at 10-, 20-, 30, 40- (half-time), 50-, 60-, 70-, and 80-min (post-test). This was done by having the research assistant showing the player the Bog Scale, and recording the players rating of perceived exertion. During the half-time interval (40-min) and post-protocol, players were required to perform the 3-min EMG treadmill protocol. Player‘s performance responses were measured using an LED system to count the total number of 20-metre (m) shuttles completed during each of the 20-min exercise bouts. Therefore, differences in the number of shuttles completed during each work bout were used to

determine whether there were changes in player‘s performance capacities during each test condition.

STATISTICAL ANALYSES

All statistical analyses were performed using the Statistica (Ver.8 SP1) software package. Basic descriptive statistics were conducted to accumulate general demographic and morphological data on the group. Descriptive measures of physiological, perceptual and performance data included means and standard deviations. A general linear model was used to interpret all results. A One-way ANOVA with repeated measures was used to detect differences over time and a two-way ANOVA to detect differences between conditions and over time. A three-way ANOVA was used to detect differences for biophysical responses over time, at different running speeds and between conditions.

CHAPTER IV

RESULTS

ENVIRONMENTAL CONDITIONS

Table V: Mean (± SD) environmental conditions for both conditions.

	CC	EC
Ambient Temperature (° C)	17.60 (± 1.60)	17.30 (± 1.52)
Relative Humidity (%)	65.20 (± 9.50)	66.26 (± 9.98)

Where CC represents the control condition and EC represents the experimental condition

As the environmental conditions were controlled, expectedly, there was no difference in ambient temperature and relative humidity between conditions (Table V).

PHYSIOLOGICAL RESPONSES

Heart Rate (HR)

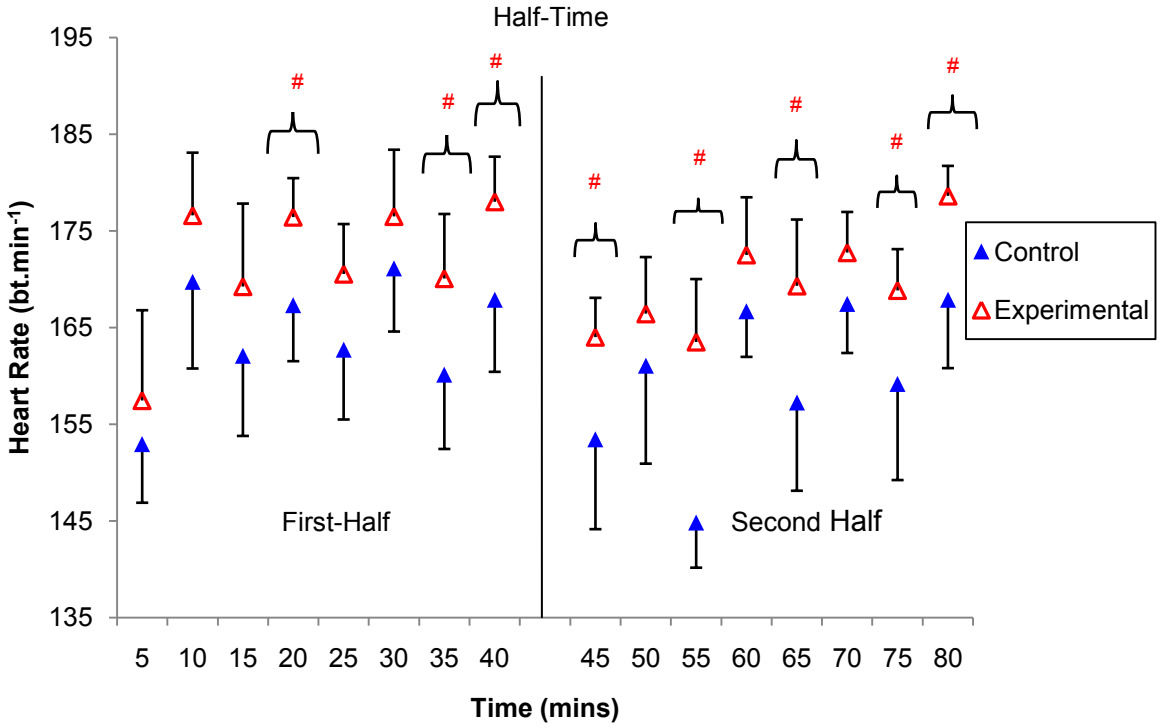
Table VI: Mean (± SD) heart rate responses over time during each condition.

	Heart Rate ($bt.min^{-1}$)	
Time (mins)	**Control Condition (CC)**	**Experimental Condition (EC)**
0	63 (± 3.67)]	64 (± 4.65)]
20	167 (± 11.59)	176 (± 4.03)
40	168 (± 7.45)	178 (± 4.67)
60	167 (± 4.73)	173(± 5.97)
80	168 (± 7.09)	179 (± 3.10)

Where] indicates a significant change in heart rate from rest to 20-min

Players' resting heart rates were similar prior to both conditions (63 $bt.min^{-1}$ for the CC and 64 $bt.min^{-1}$ for the EC). The only significant ($p < 0.05$) increase in heart rate

occurred in the transition from rest (0-min) to exercise (20-min) for both conditions (Table VI). For the remainder of both conditions (20-80 min), heart rate remained fairly constant.



Where # indicates a significant difference between conditions at comparable time intervals

Figure 2: Mean (± SD) heart rate responses comparing conditions over time.

Mean heart rates for the entire 80-min period were significantly ($p < 0.05$) higher during the EC (mean of 171 ±1.64 bt.min^{-1}, Figure 2) compared to the CC (156 ±7.2 bt.min^{-1}). Likewise, for the first-half, heart rates were higher during the EC (mean of 172 bt.min^{-1}), compared to the CC (mean of 164 bt.min^{-1}). Heart rates then decreased significantly ($p < 0.05$) after the half-time interval. As with the first-half of the EC, mean heart rate was significantly ($p < 0.05$) higher throughout the second half (170 bt.min^{-1}) of the protocol, compared to the CC (160 bt.min^{-1}), although the difference between conditions was smaller. At the end of the 80-min protocol, heart rates were significantly higher ($p < 0.05$) in the EC (179 bt.min^{-1}), compared to the CC (168 bt.min^{-1}).

Core Temperature

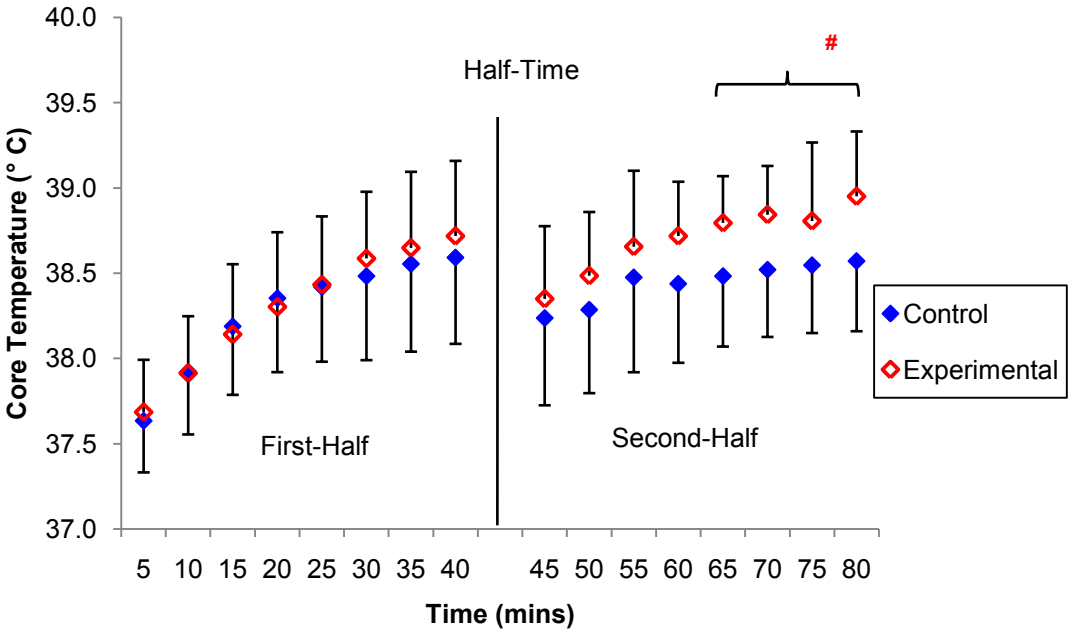
Table VII: Mean (± SD) core temperature responses over time during each condition.

	Core Temperature (° C)	
Time (mins)	**Control Condition (CC)**	**Experimental Condition (EC)**
0	37.29 (± 0.36)	37.31 (± 0.21)
20	38.35 (± 0.43)	38.30 (± 0.44)
40	38.59 (± 0.51)	38.72 (± 0.44)
60	38.44 (± 0.46)	38.72 (± 0.32)
80	38.57 (± 0.41)	38.95 (± 0.38)

Where ⌉ indicates a significant difference in core temperatures over time for both conditions

Player's resting core temperatures (Table VII) were similar prior to the commencement of both conditions (37.29 ±0.36° C for the CC and 37.31 ±0.21° C for the EC). During the EC, core temperature increased significantly ($p < 0.05$) throughout the first-half of the protocol (0-40 min), whereas during the CC, core temperatures increased significantly from rest to 20-min, and then remained fairly constant until 40-min. At 40-mins, core temperature was 38.59 ±0.51° C and 38.72 ±0.44° C for the CC and EC respectively (Table VII). After the half-time interval (Figure 3), core temperature decreased significantly ($p < 0.05$). Thereafter, during the second half, core temperature stabilised in both conditions.

During the first-half of the protocol, there were no differences in core temperature between conditions (Figure 3). From 65- to 80-min, core temperature was significantly ($p < 0.05$) higher during the EC, compared to the CC. At the end of the 80-min protocol, core temperature was significantly ($p < 0.05$) higher in the EC (38.57 ±0.41° C), compared to the CC (38.95 ±0.38° C).



Where # indicates a significant difference between conditions from 65-80 min

Figure 3: Mean (± SD) core temperature responses comparing conditions over time.

BIOPHYSICAL RESPONSES

For the purpose of this discussion, the biophysical responses of the quadriceps and hamstring muscles will be interpreted as follows: The muscle activity (arbitrary units) of each condition was first assessed at increasing running speeds at each time interval. The muscle activity of each muscle was then analysed over time and between conditions to the see the impact of the clothing and protective gear and the effect over time.

QUADRICEP MUSCULATURE

Vastus Medialis (VM)

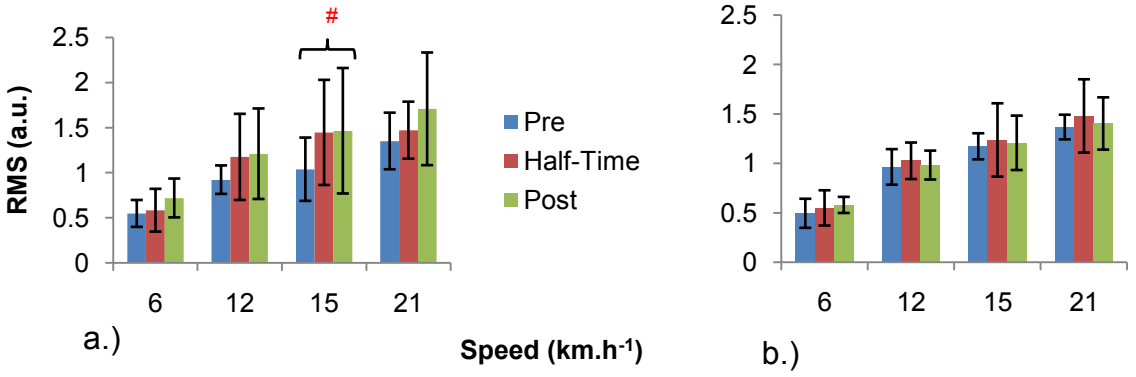
During both conditions, the muscle activity of the VM increased significantly ($p < 0.05$) with increasing speeds (Table VIII). At each speed, muscle activity remained similar for both conditions over time, except at 15 $km.h^{-1}$ for the CC (Figure 4a), where muscle activity increased significantly ($p < 0.05$) from baseline levels (pre-test) to levels at half-time and after the protocol. There were no significant differences in

muscle activity between conditions at comparable speeds and time intervals (Figure 4), however the trend was for higher muscle activity in the CC (Figure 4a).

Table VIII: Mean (± SD) changes in muscle activation (RMS) of the VM with increasing speeds for both conditions.

Where: ⌋ indicates a significant change in muscle activity with increasing speeds

	Muscle Activity (RMS)					
	Control Condition			Experimental Condition		
Speed ($km.h^{-1}$)	**Pre-**	**Half-Time**	**Post-**	**Pre-**	**Half-Time**	**Post-**
6	0.55 (0.2)	0.58 (0.2)	0.72 (0.2)	0.5 (0.1)	0.55 (0.2)	0.58 (0.1)
12	0.92 (0.2)	1.17 (0.5)	1.21 (0.5)	0.96 (0.2)	1.03 (0.2)	0.98 (0.1)
15	1.04 (0.4)	1.45 (0.6)	1.46 (0.7)	1.17 (0.1)	1.24 (0.4)	1.21 (0.3)
21	1.35 (0.3)	1.47 (0.3)	1.71 (0.6)	1.37 (0.1)	1.46 (0.4)	1.40 (0.3)



Where # signifies a significant difference between the measure prior to the protocol and at half-time during the CC

Figure 4: Mean (± SD) changes in activation of the VM over time for the CC (a) and EC (b).

Vastus Lateralis (VL)

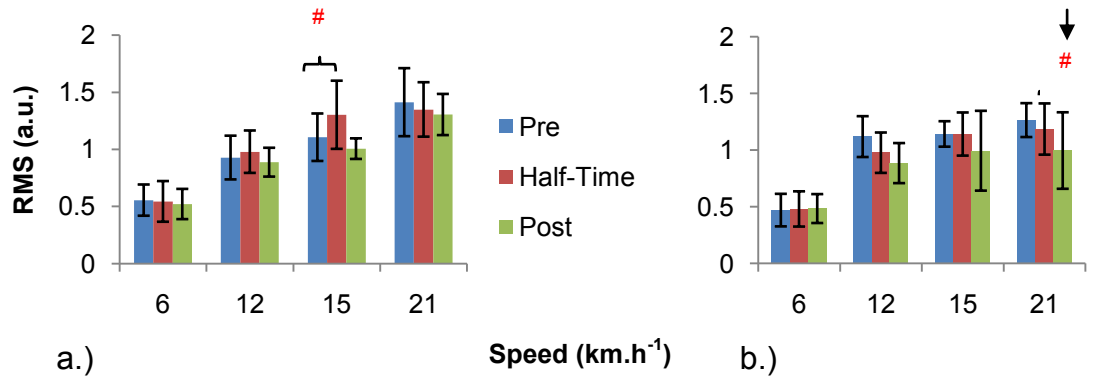
During the CC and EC, muscle activity increased significantly ($p< 0.05$) from 6 $km.h^{-1}$ to all the other speeds (12-, 15- and 21 $km.h^{-1}$) at all the time intervals (pre-protocol, half-time and post-protocol). For the CC, muscle activity increased significantly

($p < 0.05$) from 12- to 21 km.h^{-1} at all the time intervals (Table IX), as well as from 15- to 21 km.h^{-1} (pre-protocol) and from 12-15 km.h^{-1} (half-time). In contrast, during the EC, no further changes in muscle activity occurred.

Table IX: Mean (± SD) changes in muscle activation (RMS) of the VL with increasing speeds for both conditions.

	Muscle Activity (RMS)					
	Control Condition			Experimental Condition		
Speed (km.h^{-1})	**Pre-**	**Half-Time**	**Post-**	**Pre-**	**Half-Time**	**Post-**
6	0.56 (0.1)	0.58 (0.2)	0.72 (0.1)	0.50 (0.1)	0.55 (0.2)	0.58 (0.1)
12	0.93 (0.2)	1.17 (0.2)	1.21 (0.1)	0.96 (0.2)	1.03 (0.2)	0.98 (0.2)
15	1.11 (0.2)	1.45 (0.3)	1.46 (0.1)	1.17 (0.1)	1.24 (0.2)	1.21 (0.4)
21	1.41 (0.3)	1.47 (0.2)	1.71 (0.2)	1.37 (0.1)	1.46 (0.2)	1.40 (0.3)

Where: ⌋ indicates a significant change in muscle activity with increasing speeds



Where: # signifies a significant difference over time
↓ signifies are significant difference between conditions

Figure 5: Mean (± SD) changes in activation of the VL over time for the CC (a) and EC (b).

For both conditions, there were no significant changes in muscle activity over time, except at 15 km.h^{-1} in the CC, and 21 km.h^{-1} in the EC (Figure 5). At 15 km.h^{-1}, muscle activity increased significantly ($p < 0.05$) at half-time during the CC, and decreased significantly ($p < 0.05$) from half-time to the 80-min mark at a speed of 21 km.h^{-1} in the EC. When comparing conditions, the only significant difference

occurred at 21 km.h^{-1} (post-protocol) where the muscle activity during the CC was significantly ($p < 0.05$) higher than the EC.

HAMSTRING MUSCULATURE

Biceps Femoris (BF)

For the CC, the muscle activity (Table X) of the BF increased significantly ($p < 0.05$) from the start to the half-time measurements. At all other time intervals during the CC, there were no further increases in muscle activity with increasing speeds. During the EC, muscle activity significantly ($p < 0.05$) increased from pre-test levels at 6 km.h^{-1} to levels at 12-, 15- and 21 km.h^{-1}, and then again during the half-time interval from levels at 15 km.h^{-1} to levels at 21 km.h^{-1} , and from levels at 15 km.h^{-1} to levels at 21 km.h^{-1} (Table X). For the EC, there was no further increase in muscle activity with increasing running speeds.

Table X: Mean (± SD) changes in muscle activation of the BF with increasing speeds for both conditions over time.

	Muscle Activity (RMS)					
	Control Condition			**Experimental Condition**		
Speed (km.h^{-1})	**Pre-**	**Half-Time**	**Post-**	**Pre-**	**Half-Time**	**Post-**
6	0.67 (0.2)	0.51 (0.2)	0.58 (0.1)	0.55 (0.1)	0.55 (0.2)	0.58 (0.1)
12	0.98 (0.2)	0.70 (0.1)	0.70 (0.1)	1.00 (0.2)	1.03 (0.2)	0.98 (0.2)
15	1.08 (0.2)	0.81 (0.2)	0.69 (0.1)	1.20 (0.2)	1.23 (0.2)	1.21 (0.2)
21	1.27 (0.2)	0.83 (0.2)	0.94 (0.2)	1.25 (0.2)	1.46 (0.2)	1.40 (0.3)

Where: ⌝ indicates a significant change in muscle activity with increasing speeds

For both conditions, there were no significant changes in BF muscle activity over time (Figure 6). However, the trend showed muscle activity to progressively decrease over time. Furthermore, there were no significant differences in muscle activity between conditions (Figure 6).

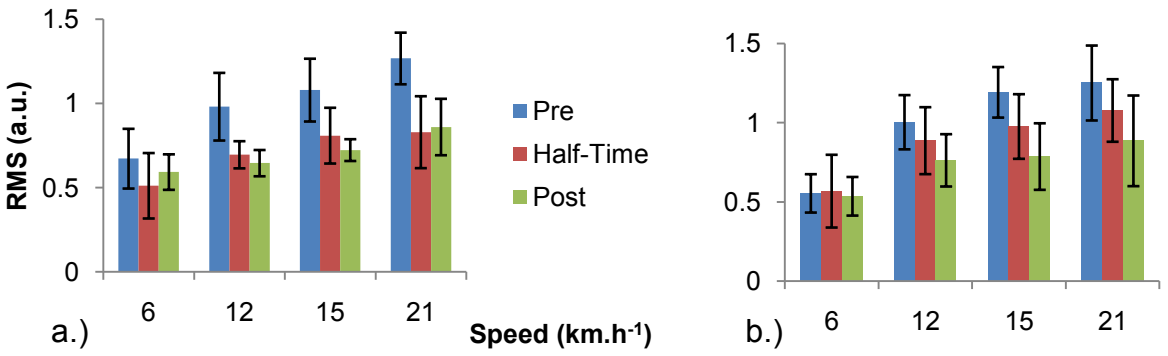


Figure 6: Mean (± SD) changes in activation of the BF for the CC (a) and EC (b) over time.

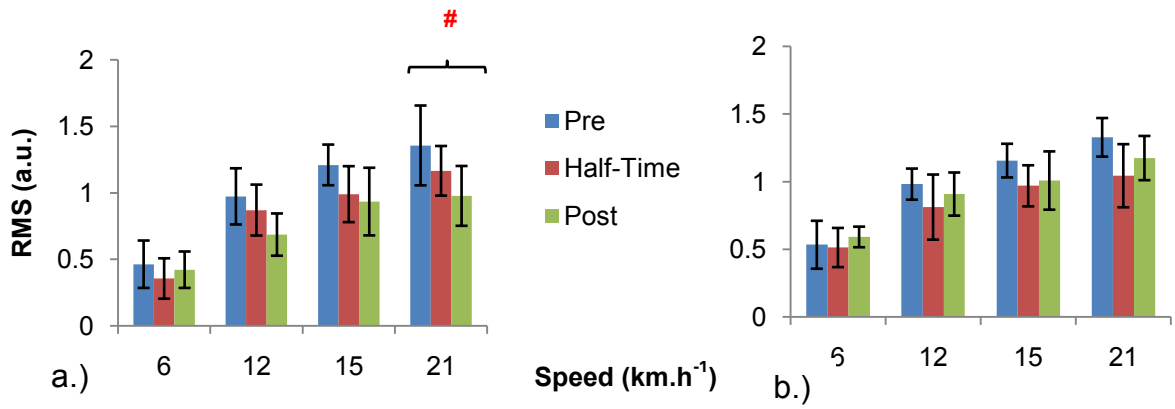
Semitendinosus (ST)

Table XI: Mean (± SD) changes in muscle activation of the ST with increasing speeds over time for both conditions.

	Muscle Activity (RMS)					
	Control Condition			Experimental Condition		
Speed ($km.h^{-1}$)	Pre-	Half-Time	Post-	Pre-	Half-Time	Post-
6	0.46 (0.2)	0.36 (0.2)	0.45 (0.1)	0.53 (0.2)	0.51 (0.1)	0.59 (0.1)
12	0.97 (0.2)	0.87 (0.2)	0.67 (0.2)	0.98 (0.1)	0.81 (0.2)	0.91 (0.2)
15	1.21 (0.2)	0.99 (0.2)	0.90 (0.3)	1.16 (0.1	0.97 (0.2)	1.01 (0.2)
21	1.36 (0.3)	1.17 (0.2)	0.99 (0.2)	1.33 (0.1)	1.04 (0.2)	1.18 (0.2)

Where: ⌋ indicates a significant change in muscle activity with increasing speeds

For both conditions, ST muscle activity increased significantly ($p > 0.05$) from levels at 6 $km.h^{-1}$ to all other running speeds at all the time intervals (Table XI). The only other significant ($p < 0.05$) increase in muscle activity occurred during the CC (pre-test) from levels at 12 $km.h^{-1}$ to levels at 21 $km.h^{-1}$ (Table XI). Muscle activity did not change significantly over time during the CC or the EC, except at 21 $km.h^{-1}$ in the CC (Figure 7a), where muscle activity decreased significantly ($p < 0.05$) from levels measured prior to testing to levels at the end of the protocol. There were no significant differences between conditions over time.



Where: # signifies a significant difference over time during the CC

Figure 7: Mean (± SD) changes in activation of the ST over time for the CC (a) and EC (b).

PERCEPTUAL RESPONSES

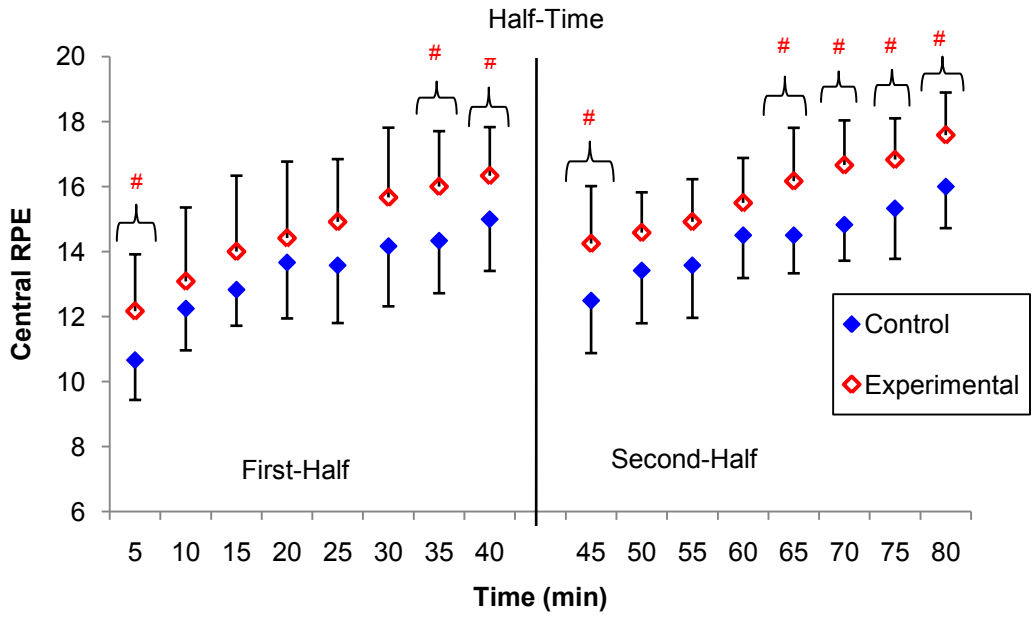
Central Ratings of Perceived Exertion (RPE)

Table XII: Mean (± SD) responses for Central RPE over time during each condition.

	Central RPE	
Time (mins)	**Control Condition (CC)**	**Experimental Condition (EC)**
5	10.7 (1.2)	12.2 (1.8)
20	13.7 (1.7)	14.4 (2.4)
40	15.0 (1.6)	16.3 (1.5)
60	14.5 (1.3)	15.5 (1.4)
80	16.0 (1.3)	17.6 (1.3)

Where: ⌉ indicates a significant changes in central RPE over time during each condition

Central RPE significantly ($p < 0.05$) increased over time for both conditions (Table XII). For both conditions, central RPE increased significantly ($p < 0.05$) from the start (5-min) to the end of the first half (40-min). With both conditions in the second half, central RPE remained fairly constant from 40- to 60-min and then significantly ($p < 0.05$) increased from 60-min to the end of the protocol (80-min).



Where # indicates a significant difference between conditions

Figure 8: Mean (± SD) central RPE comparing conditions over time.

Central RPE was significantly ($p < 0.05$) higher during the EC at 35- and 40-minutes (Figure 8). After the half-time interval, central RPE decreased significantly ($p < 0.05$) in both conditions. For both conditions, there was no difference in RPE during the mid-stage of the protocol. Thereafter, central RPE continued to gradually increase during the second half of the protocol in both conditions. Players' mean central RPE for the second half of the protocol was significantly ($p < 0.05$) higher for the EC (mean of 15.8 ±1.2), compared to the CC (mean of 14.3 ±1.1). During the second half, central RPE was significantly ($p < 0.05$) higher in the EC at 45- 65- 70-, 75 and 80-minutes. When comparing halves, central RPE was similar for both conditions. At the end of the protocol, central RPE was significantly higher in the EC (mean of 17.6 ±1.3), compared to the CC (mean of 16.0 ±1.3).

Local RPE

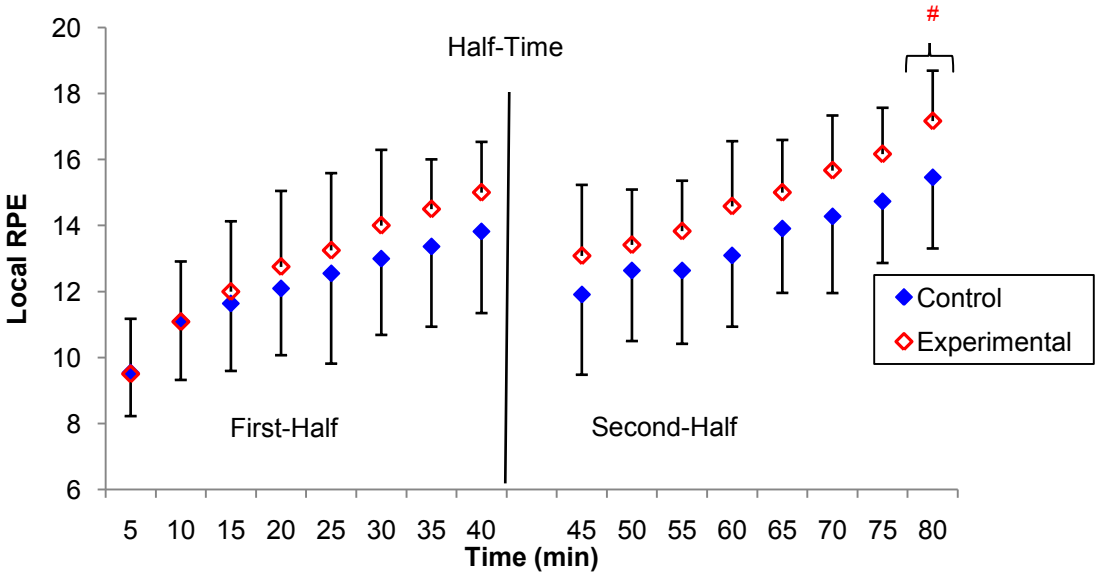
Near the start of the protocol (5-min), local RPE was the same (Table XIII) for both conditions (9.5). In the first-half, mean local RPE significantly increased ($p < 0.05$) to 13.8 (±2.5) in the CC, and to 15 (±1.5) in the EC at 40-min. After the half-time

interval (at 45-min), local RPE significantly ($p < 0.05$) decreased for both conditions (Figure 9). In the second half, the trend was for local RPE to increase with time. Noteworthy however, is that the ratings were higher at the commencement of the second half, compared to the first half (Figure 9).

Table XIII: Mean (± SD) responses for Local RPE comparing conditions over time.

	Local RPE	
Time (mins)	**Control Condition (CC)**	**Experimental Condition (EC)**
5	9.5 (1.3)	9.5 (1.7)
20	12.1 (2.0)	12.8 (2.3)
40	13.8 (2.5)	15.0 (1.5)
60	13.1 (2.2)	14.6 (2.0)
80	15.5 (2.2)	17.2 (1.5)

Where: ⌋ indicates a significant changes in local RPE over time during each condition



Where # indicates a significant difference between conditions

Figure 9: Mean (± SD) local RPE comparing conditions over time.

Local RPE was consistently higher for the EC at all comparable time intervals in the second half compared to the first-half. For the CC, local RPE was a mean of

12.1 (±1.4) and 13.6 (±1.2) in the first and second half respectively, while for the EC it was 12.8 (±1.8) and 14.9 (±1.4). During the second half, local RPE continued to increase significantly ($p < 0.05$) for both conditions (Table XIII). At the end of the protocol, local RPE was significantly ($p < 0.05$) higher for the EC (17.2 ±1.5), compared to the CC (15.5 ±2.2). However, there was no significant difference for local RPE between conditions (overall mean of 13.1 ±2.1 for the CC and 13.81 ±1.82 for the EC).

PERFORMANCE RESPONSES

During the CC, performance (total number of 20-m shuttles completed) significantly ($p < 0.05$) increased over time (Table XIV) with the most number of shuttles being covered in the last quarter (mean of 106 ±10). For both conditions, there were no changes in performance in the first and second quarter. In the second half (third- and fourth quarters) of the CC, performance increased significantly from first-half levels (first and second quarters), while in the EC, performance remained constant.

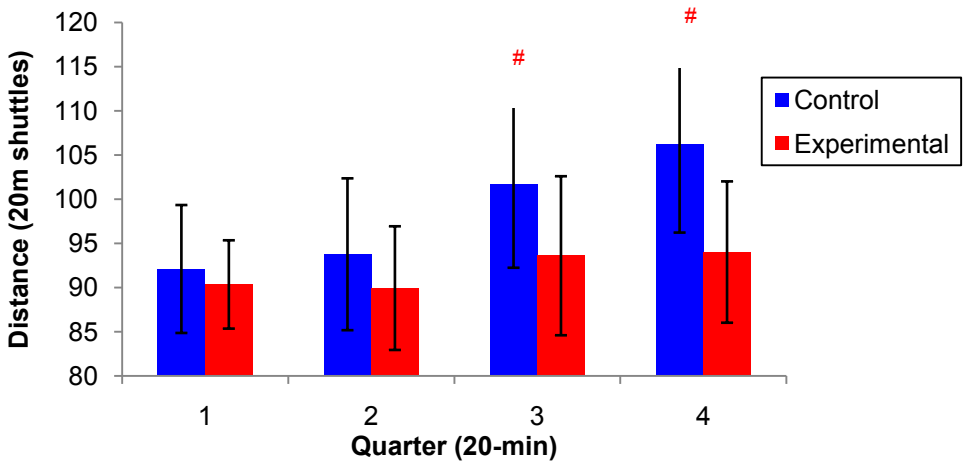
Table XIV: Mean (± SD) performance responses comparing conditions over time.

	Performance (20-metre shuttles)	
Quarter	**Control Condition (CC)**	**Experimental Condition (EC)**
1	92 (± 7)	90 (± 5)
2	94 (± 9)	90 (± 7)
3	102 (± 9)	94 (± 9)
4	106 (± 10)	94 (± 8)
Total	**394 (± 32.7)**	**368 (± 26.7)**

Where: ⌋ indicates a significant change in performance over time for each condition

In the first-half of both conditions, player's performance responses were similar, with completing a mean of 186. Player's completed a mean of 186 (±7.8) shuttles for the CC and 180 (±6.3) shuttles for the EC (Figure 10). For the second half of the protocol, player's completed significantly ($p < 0.05$) more shuttles in the CC (mean of

208 ±9.8), compared to the EC (mean of 188 ±8.4). Therefore, players performed significantly ($p < 0.05$) more shuttles overall in the CC (394 ±32.7), compared to the EC (368 ±26.7).



Where # indicates a significant difference between conditions

Figure 10: Mean (± SD) performance responses comparing conditions over time.

MULTIVARIATE ANALYSIS

Heart Rate and Central RPE

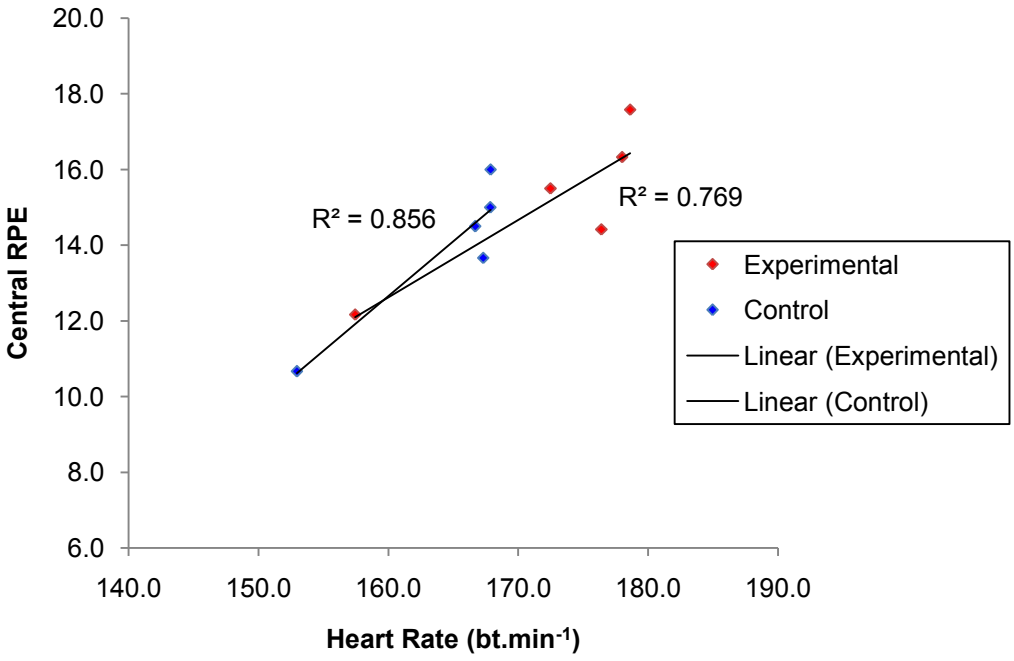


Figure 11: Relationship between heart rate and central RPE during both conditions.

There was a strong relationship between players heart rates and central RPE during the CC (R^2=0.86) and EC (R^2=0.77). Therefore, players accurately perceived the cardiovascular demands during both conditions simulated protocols.

CHAPTER V

DISCUSSION

PHYSIOLOGICAL RESPONSES

Cardiovascular Responses

Player's heart rates ranged between 145-169 bt.min^{-1} (mean of 162 bt.min^{-1}) during the control condition (CC), and 157-179 bt.min^{-1} (mean of 171 bt.min^{-1}) during the experimental condition (EC). During both conditions, heart rates were within the range of 135-180 bt.min^{-1}, which is reportedly the range representative of actual match-play (Morton *et al*., 1978). Therefore, the simulated 80-minute (min) rugby protocol is deemed to be representative of actual match-play, despite excluding all of the contact activities of the game.

Mean resting heart rates (Table VI, page 66) were similar for both conditions (63 bt.min^{-1} for the CC and 64 bt.min^{-1} for the EC). The transition from rest (0-min) to the initiation of exercise showed significant ($p < 0.05$) increases in heart rate for both conditions (Figure 2, page 67), which is a natural response to exercise (McArdle *et al*., 2006). Heart rates increased to a mean of 167 bt.min^{-1} (±5.8) at 20-min for the CC, and to 176 bt.min^{-1} (± 4.03) for the EC. Therefore, at 40-min, heart rates were significantly ($p < 0.05$) higher in the EC, compared to the CC. For both conditions, heart rates were significantly ($p < 0.05$) higher during the second half of the protocol. However, heart rates were significantly higher during the second half of the EC (mean of 170 ±1.36 bt.min^{-1}), compared to the CC (mean of 160 ±2.40 bt.min^{-1}). Players' heart rate responses, experienced in both test conditions, are consistent with those observed in previous rugby (Cunniffe *et al*., 2006; Brown, 2009) and soccer (Greig, 2006) studies. However, during the EC, players reached higher heart rate, when compared to the CC, likely due to the clothing and protective gear worn during the exercise protocol.

Despite the increased exercise intensity of the second half of the protocol, heart rates remained fairly comparable to first-half measures for both conditions (Figure 2, page 67). In contrast, previous studies found rugby players heart rates to be significantly ($p< 0.05$) higher during the second half of match-play (Cunniffe *et al*., 2009). However, the study by Cunniffe *et al*. (2009) was conducted during actual match-play, where environmental conditions were uncontrolled and all contact activities of the game were included. In this study, testing was conducted in a controlled laboratory setting, where environmental conditions were controlled, and excluded all of the contact activities involved in rugby.

The overall mean heart rate for the CC was 162 (±7.2) $bt.min^{-1}$ and 171 (±5.9) $bt.min^{-1}$ for the EC, a finding which was significant ($p< 0.05$). The increased heart rates experienced during the EC could be explained by the conflicting demands being placed on the cardiovascular system, as a result of exercising in the additional rugby kit, as both the working muscles and cutaneous circulation require large portions of the cardiac output to maintain homeostasis (Vigas *et al*., 2000; Todd *et al*., 2005). Mean heart rate experienced during the EC was similar to those experienced during actual rugby match-play (Cunniffe *et al*., 2009), despite the simulated protocol used in this study excluding all of the contact activities of the game. Therefore, the additional 9 $bt.min^{-1}$ experienced during the EC, compared to the CC, could tentatively be explained by the additional rugby gear worn during the EC, as players performed the same protocol for both conditions.

Core Temperature

Players mean resting core temperatures (Table VII, page 68) were similar for both conditions (37.29 ±0.36° C for the CC and 37.31 ±0.21° C for the EC), and is considered to be within the normal range for individuals at rest (Parsons, 2000; Brotherhood, 2008). Core temperatures significantly ($p< 0.05$) increased during the first 20-min of both conditions (Figure 3, page 69). At 20-min, core temperatures reached 38.35 ±0.43° C for the CC and 38.30 ±0.44° C for the EC. These core temperature responses are similar to those experienced by soccer

players during match-play (Duffield *et al*., 2009; Ozgunen *et al*., 2010). This is considered to be a normal response to exercise, as muscular activity is proposed to be the main driver of core temperature (NIOSH, 1986; Parsons, 2000). From 20-40 minutes, player‘s core temperatures continued to significantly ($p < 0.05$) increase in the EC, reaching a mean of 38.72 ±0.44°C at 40-minutes (Table VII, page 68). From the 20-min time interval of the CC, core temperatures remained stable throughout the remainder of the protocol, which suggests that thermal homeostasis had been achieved.

After the half-time interval (measured at 45-min), core temperatures had significantly ($p < 0.05$) decreased from peak first-half levels in both conditions (mean of 38.59 ±0.51 to 38.24 ±0.51° C for the CC and 38.72 ±0.44 to 38.35 ±0.43° C for the EC). These core temperature responses were similar to those experienced during soccer match-play (Ozgunen *et al*., 2010) which is also an intermittent sport. For the remainder of both conditions (45- to 80-min), core temperatures remained constant (Figure 3, page 69). However, at the end of the protocol, player‘s core temperatures were significantly higher in the EC (38.95° C), compared to the CC (38.57° C). These significantly ($p < 0.05$) higher core temperatures caused by wearing clothing and protective gear are similar to those observed in previous cricket (King, 2002), football (Kulka and Kenney, 2002) and rugby (Cannon and Christie, 2009) studies where athlete‘s core temperatures were significantly higher while exercising in full clothing and protective gear, compared to when performing the same exercise bout in minimal clothing and no protective gear. Therefore, although exercise is identified as the main driver of core temperature responses, the additional clothing and protective gear worn during the EC caused players core temperatures to reach significantly ($p < 0.05$) higher levels towards the latter stages of the protocol (38.95 ±0.38° C for the EC compared to 38.57 ±0.41° C for the CC). This is because clothing and protective gear have been shown to hinder the mechanisms of heat loss during exercise (Godek *et al*., 2005; Goodman *et al*., 1995).

When comparing halves, players mean core temperatures were higher during the second half of both conditions (Figure 3, page 69). During the CC, mean core

temperature was 38.27 ±0.34° C for the first-half and 38.44 ±0.12° C for the second half. During the EC, mean core temperature was 38.30 ±0.37° C for the first-half and 38.70 ±0.20° C for the second half. This was due to the second half of the protocol being more intense than the first, as heat production during exercise is a function of relative exercise intensity (Brotherhood, 2008), which explains the higher core temperatures in the second half of both conditions. According to Brotherhood (2008), during intermittent exercise, reaching core temperatures of approximately 39.50° C is considered to be normal; levels which were not reached in this investigation. The mean peak core temperature for the CC was 38.59 ±0.51° C (at 40-min) and 38.95 ±0.38° C (at 80-min) for the EC. These lower core temperatures observed for both conditions may have been due to testing being conducted in a laboratory setting, where environmental conditions were strictly controlled and relatively mild. Alternatively, players could have paced themselves knowing the end point of both conditions and preventing them from reaching a critical core temperature (Tucker *et al*., 2004) aimed at preventing excessively high core temperatures from being reached, thus maintaining homeostasis (Bruck and Olschewski, 1987; Cheung and McLellan, 1998; Gonzalez-Alonso *et al*., 1999; Nybo and Nielsen, 2001a).

BIOPHYSICAL RESPONSES

Quadricep Musculature

During the EMG treadmill protocol for both conditions, the muscle activity of the VM increased significantly ($p < 0.05$) with increasing running speeds (Table VIII, page 70), which is similar to the findings of previous soccer (Rahnama *et al*., 2006) and rugby studies (Brown, 2009). This is due the muscles becoming more active for a greater percentage of the gait cycle when changing from slower to faster running speeds (Nilsson *et al*., 1985). Therefore, this indicates that the VM were required to produce greater muscular output to cope with the increasing demands of the protocol.

In both conditions, the muscle activity of the VM were unaffected over time, except at 15 $km.h^{-1}$ during the CC, where muscle activity increased significantly ($p < 0.05$) from

baseline levels (pre-protocol) at half-time and after the protocol (Figure 4a, page 70). Previous studies found VM muscle activity to decrease during tennis (Girard *et al*., 2008) and squash (Girard et al., 2009) matches, which are also highly intermittent in nature. Muscular fatigue has been suggested to cause a progressive reduction in muscle activity over time (Gonzalez-Alonso *et al*., 2000; Mohr *et al*., 2003; Rahnama *et al*., 2003; Girard *et al*., 2009), while others suggest that muscle fatigue causes muscle activity to increase (Brown, 2009). Therefore, it appears that the muscle activities of the VM muscles were unaffected by fatigue during an 80-min rugby protocol, except at the higher running speeds (15 $km.h^{-1}$) during the CC, where muscle activity significantly ($p< 0.05$) increased over time. This may have been due to the players selecting an appropriate pacing strategy that would allow for homeostasis to maintained, and may have prolonged the onset of muscle fatigue during both conditions. According to de Koning *et al*. (2011), athlete's change the intensity of exercise, based on their momentary RPE and the amount of time remaining in the exercise work-bout. In this study, players were aware that the simulated rugby protocol was 80-min in duration, and were thus able to select an appropriate strategy to maintain homeostasis and complete the exercise work-bout with an adequate physiological reserve.

For the VL, muscle activity also increased significantly ($p< 0.05$) at higher running speeds (Table IX, page 71). Therefore, the VL muscle activity became more active while running at higher running speeds (12-, 15- and 21 $km.h^{-1}$), compared to muscle activity while walking at 6 $km.h^{-1}$. During the CC (Figure 5a, page 71), VL muscle activity increased significantly ($p< 0.05$) from levels at 12- to 21- (pre-, half-time and post-protocol) and 15 $km.h^{-1}$ (half-time), as well as from 15- to 21 $km.h^{-1}$ (pre-protocol), which indicates the importance of the VL in controlling the locomotor activities associated with running at high speeds.

When comparing the VL muscle activity over time, no significant changes occurred for both conditions, except at 15 $km.h^{-1}$ in the CC (Figure 5a, page 71), and 21 $km.h^{-1}$ in the EC (Figure 5b, page 71), where muscle activity increased significantly ($p< 0.05$) from levels measured prior to the commencement of the

protocol to levels at half-time (CC) and from half-time levels to levels measured after the protocol (EC).This increase in muscle activity indicates that the muscle was required to produce a greater muscular output in order to achieve the same workload (Greig *et al*., 2006).Therefore, the muscle activity of the VL appeared to be unaffected by fatigue experienced during the protocol.

During both conditions, the muscle activity of the quadriceps (VM and VL) were unaffected by increasing core temperatures, as muscle activity was similar throughout both conditions, despite players reaching significantly ($p < 0.05$) higher core temperatures during the EC (Figure 3, page 69). Previous studies (Bruck and Olschewski, 1987; Cheung and McLellan, 1998; Gonzalez-Alonso *et al*., 1999; Nybo and Nielsen, 2001a) suggest that reaching a core temperature of approximately 40° C during exercise causes a reduction in muscle activity, ultimately reducing the athletes work rate and ensuring that a potentially catastrophic failure of homeostasis does not occur (Noakes *et al*., 2004).This is based on the central governor theory proposed by Noakes (Noakes, 1996; Noakes, 1998; Noakes, 2000). However, throughout both conditions, player's never reached these critically high core temperatures. Players reached peak core temperatures of 38.95 ±0.38° C during the EC and 38.59 ±0.51° C during the CC (Figure 3, page 69). Therefore, the roles of the VM and VL (quadriceps muscles) in controlling the locomotor activities of both conditions seem similar, despite players reaching significantly higher core temperatures in the EC. Therefore, it can be concluded that the main driver of quadriceps muscle activity was the simulated rugby exercise protocol and not the clothing and protective gear.

Hamstring Musculature

During both conditions, the muscle activity of the BF increased significantly ($p < 0.05$) with increasing speeds (Table X, page 72). However, these changes in muscle activity only occurred from levels measured before the commencement of the simulated rugby protocol (pre-test) from 6km.h^{-1} to the higher speeds (12-, 15- and

21 km.h^{-1}). Therefore, no further changes in BF muscle activity occurred at the higher running speeds.

For both conditions, the muscle activity of the BF showed marginal reductions over time (Figure 6a and 6b, page 73). These findings are similar to those of previous studies, where the muscle activity of the BF was shown to decrease over time during soccer (Rahnama *et al.,* 2006; Greig *et al*., 2006) and rugby-specific (Brown, 2009) protocols. The reduction in the muscle activity of the BF may be attributed to the redistribution of muscular work, due to altered kinematics of movement during fatigue, which increases the risk of injury, particularly in the hamstring musculature (Greig *et al*., 2006). During sprinting, Pinniger *et al*. (2000) found that there was altered kinematics of movement and muscle activity as a result of fatigue, which causes a breakdown in the athlete‘s movement patterns (Bates *et al*., 1977). Therefore, sprinting has been identified as the main mechanism of injury in rugby, due to this altered kinematics experienced during fatigue (Brooks *et al*., 2005). This reduction in muscle activity of the BF is supported by epidemiological data, which states that there is an increased incidence of hamstring injuries during the second half of rugby match-play (Brooks *et al*., 2009).

During both conditions, the muscle activity of the ST increased at higher running speeds (Table XI, page 73), which was similar to the biophysical responses of the BF (Table X, page 72). From 6km.h^{-1} to the higher running speeds of 12-, 15- and 21 km.h^{-1}, it was observed that for both conditions, the muscle activity of the ST increased significantly ($p<0.05$). There were no significant changes in ST muscle activity over time, except for the EC at 21 km.h^{-1}where muscle activity decreased significantly ($p< 0.05$) from levels measured before the protocol and at half-time (Figure 7b, page 74). However, much like the BF, the ST muscle activity showed marginal reductions ($p> 0.05$) over time at comparable speeds. However, these changes in muscle activity for both conditions were not significant.

Therefore, the muscle activity of the BF and ST remained similar during both conditions. Thus, it can be suggested that the greater heat load caused by wearing the additional rugby kit during a simulated rugby protocol did not affect the muscle activity of the hamstrings, even during the EC, where players experienced significantly higher core temperatures at the end of the protocol, compared to the CC. Therefore, it can be concluded that the effect of the exercise work-bout on the muscle activity of the quadriceps and hamstrings was greater than the effect of the clothing and protective gear. Furthermore, it can be suggested that during an 80-min simulated rugby protocol, the activation (units are arbitrary) of the quadricep (VM and VL) were unaffected by central mechanisms of fatigue. In contrast, the muscle activity of the hamstring muscles (ST and BF) decreased over time, which suggests that these muscles were affected by central mechanisms of fatigue, or possibly due to high eccentric demands of the protocol.

PERCEPTUAL RESPONSES

Central Ratings of Perceived Exertion (RPE)

For both conditions, central RPE significantly ($p < 0.05$) increased from levels measured at the beginning of the protocol (5-min) to levels at 20-, 40-, 60- and 80-min. These findings were similar to those of previous rugby (Brown, 2009) and soccer studies (Drust *et al*., 2000). From 20- to 40-min of the CC protocol, central RPE remained relatively constant. In contrast, for the EC, central RPE increased significantly ($p < 0.05$) during this time interval. For both conditions central RPE was significantly ($p < 0.05$) lower after the half-time interval (at 45-min), and then remained fairly constant up until 60-min (Figure 8, page 75). From 60- to 80-min, central RPE significantly ($p < 0.05$) increased in both conditions, possibly due to the increasing demands of the simulated rugby protocol in the second-half. The mean post-protocol central RPE was significantly ($p < 0.05$) higher for the EC (mean of 17.6 ±1.3), compared to the CC (mean of 16 ±1.3), despite player‘s performing the same exercise protocol. The post-protocol RPE values of both conditions were significantly ($p < 0.05$) higher than those observed in a previous rugby study by Brown (2009). In this study (Brown, 2009) mean post-protocol central RPE was only 14.8 at the end of an 80-min simulated rugby protocol. However, the

protocol used in the study by Brown (2009) consisted of four identically matched 20-min work-bouts and did not match the increasing locomotor demands of actual rugby match-play.

Mean central RPE was significantly ($p < 0.05$) higher in the EC (15.2 ±1.8), compared to the CC (13.8 ±1.6). For the EC, central RPE was significantly ($p < 0.05$) higher at the beginning and end stages of each half (Figure 8, page 75), which was similar to player‘s cardiovascular responses (Figure 2, page 67). Therefore, it can be concluded that the main driver of players central RPE was the exercise work-bout itself, with the clothing and protective gear worn in the EC exacerbating player‘s perceptual responses, particularly at the beginning and latter stages of each half.

Local RPE

For both conditions, local RPE significantly ($p < 0.05$) increased over time (Table XIII, page 76). During both conditions, local RPE increased significantly ($p < 0.05$) from the beginning (5-min) to the end of the protocol (80-min), due to the increased muscular demands placed on the muscles of the lower limbs to accelerate and decelerate the body during such a highly intermittent exercise protocol (McLean, 1992; Duthie *et al*., 2005). During both conditions, local RPE was significantly ($p < 0.05$) lower after the half-time interval (Figure 9, page 76). In the second half, the trend for local RPE to increase with time was evident again. Noteworthy however, is that the ratings were significantly ($p < 0.05$) higher at the beginning of the second half (45-min), in comparison to the early stages of the first-half (5-min). As such, local RPE was consistently higher at all comparable time intervals in the second half compared to the first-half.

Players‘ local RPE was similar for both conditions, except at 80-min, where perceptual responses were significantly ($p < 0.05$) higher in the EC (mean of 17.2 ±1.5), compared to the CC (mean of 15.5 ±2.2). Therefore, it can be suggested that the clothing and protective gear worn during the protocol caused

players to perceive the protocol to be more taxing on the lower limb muscles, as players performed exactly the same protocols during both conditions.

Central RPE was significantly ($p < 0.05$) higher throughout the EC, compared to the CC, due to the additional clothing and protective gear worn during the simulated rugby protocol. These significantly ($p < 0.05$) higher perceptual responses, experienced during the EC, were similar to players cardiovascular responses in the EC, which suggests that players were accurately able to reflect effort required to perform the exercise protocol while wearing the additional clothing and protective gear. These findings are similar to those observed in previous football (Kulka and Kenney, 2002), cricket (King, 2002) and rugby (Cannon and Christie, 2009) studies. Based on these findings, it can be suggested that the significantly ($p < 0.05$) higher central RPE experienced throughout the EC may have been responsible for the decreased performance capacities observed during this study, as exercise tolerance in highly motivated individuals is suggested to be limited by subjective perceptions of effort (RPE) (Marcora, 2008; Marcora *et al*., 2008; Marcora *et al*., 2009), which regulates exercise via the selection of an appropriate pacing strategy (Tucker and Noakes, 2009). In contrast, local RPE responses were similar for both conditions, except at the end of the EC (80-min), where local RPE was significantly ($p < 0.05$) higher than the CC. Therefore, it can be suggested that players perceived the simulated rugby protocol to be more taxing on the cardiovascular system while wearing the full rugby clothing and protective gear. However, it appears that for both the EC and CC, the player‘s perceived the demands of the rugby protocol to be equally taxing on the muscular system. However, the higher local RPE at the end of the EC protocol suggests fatigue of the lower limb muscular. There were no significant changes in muscle activity over time during the protocol however, the trend for the hamstring muscle activity was to decrease over time, suggesting that muscle activity of the BF and ST were impacted by the intermittent nature of the protocol.

PERFORMANCE RESPONSES

Player‘s completed significantly ($p < 0.05$) more 20-metre shuttles (Table XIV, page 77) during the CC (overall mean of 393.4 ± 34.3 shuttles), compared to the EC (overall mean of 366.6 ± 28.9 shuttles). Similar findings were observed in previous rugby (Cannon and Christie, 2010) and football studies (Kulka and Kenney, 2002), where players performance capacities were significantly ($p < 0.05$) hindered while exercising in full kit, compared to when performing the same exercise work-bout wearing minimal clothing and no protective gear. This is due to the physiological and perceptual responses becoming exacerbated while exercising in the additional clothing and protective gear, and ultimately hinders athlete‘s performance capacities (Kulka and Kenney, 2002; Cannon and Christie, 2009).

For both conditions, players complete a similar number of shuttles in the first-half of the protocol (Figure 10, page 78). However, during the second half of the protocol, player‘s completed significantly ($p < 0.05$) more shuttles in the second half of the CC protocol, compared to the EC, despite players performing the same protocol in both conditions. It was expected that players would complete significantly more shuttles during the second half of both protocols, as the intensity of the second half was more intense than the first-half, which incurs a greater physiological load on player‘s during the second half (Cunniffe *et al*., 2009). However, results indicate that player‘s performance responses did not increase during the second half of the EC protocol, despite the increasing intensity of the protocol. This is possibly due to regulatory mechanisms, which down-regulate exercise in an attempt to prevent critically high body temperatures from being reached, thus maintaining homeostasis (Tucker *et al.,* 2004). Therefore, with increased body temperatures experienced during the EC (wearing full rugby kit), performance responses remained fairly stable throughout the protocol, and did not progressively increase like those observed in the CC. This suggests that fatigue does occur during a rugby activity, and is worsened by wearing full clothing and protective gear. These findings contradict those of Cunniffe *et al.* (2009), which suggest that fatigue does not occur in rugby, as players‘ performance

responses (total running distances) did not increase during the EC, but remained fairly stable .

Player‘s central (Figure 8, page 75) and local RPE (Figure 9, page 76) were consistently higher in the EC, compared to the CC, which indicates that player‘s perceived the simulated rugby protocol to be more taxing on the cardiovascular and muscular systems while wearing the full rugby kit, despite having performed the same exercise protocol in both conditions. Exercise tolerance has been suggested to be limited by subjective perceptions of effort (Marcora, 2008; Marcora *et al*., 2008; Marcora *et al*., 2009), rather than from central and/or peripheral muscle fatigue, as proposed by Noakes and St Clair Gibson (2004). During self-paced exercise, an athlete‘s work rate is regulated via the selection of an appropriate pacing strategy, based on subjective perceptions of effort and the amount of time left during the exercise bout (St Clair Gibson *et al*., 2006; Tucker and Noakes, 2009). In this study, players were aware that the simulated rugby protocol was 80-min in duration. Therefore, it can be suggested that players selected an appropriate pacing strategy, based on their momentary RPE and the amount of time left in the protocol.

Player‘s covered significantly more shuttles during the CC, compared to the EC, possibly due to a greater perception of effort experienced during the EC. Due to these increased perceptual responses observed in the EC, players may have decreased their exercise work-rates in an attempt to maintain thermal homeostasis, and thus would explain the significant decline in performance. In contrast, during the second half of the CC, player‘s performance capacities increased significantly during the third- and fourth quarters of the protocol (Table XIV, page 77), possibly due to lower central and local RPE. This confirms the hypothesis proposed by de Koning *et al*. (2011), which states that when athlete‘s RPE is less than expected at any given moment of time during an exercise work-bout, an athlete‘s work rate will increase. Therefore, during the second half of the CC, heart rates (Figure 2, page 67), core temperatures (Figure 3, page 69), and central and local RPE (Figure 8, page 75 and Figure 9, page 76) was lower. Therefore, this lower RPE

experienced during the second half of the CC protocol could explain the significant improvement in performance capacities.

During the EC, heart rate, core temperature and RPE were exacerbated, due to the additional heat load. These exacerbated responses, experienced during the EC, caused players RPE to be greater than expected, particularly in the second half of the protocol, which may have caused a conscious decision to decrease exercise work rates by reducing running speed. This resulted in significantly fewer shuttles during the third- and fourth quarters of the protocol, despite the intensity of the second half being greater than the first. This finding confirms the hypothesis, proposed by de Koning *et al*. (2011), which suggests that when RPE is greater than expected during exercise, athlete‘s work-rates (running speed) become reduced via the selection of an appropriate pacing strategy. Therefore, player‘s consciously decided to reduce their exercise work rates during the EC, based on their increased subjective perceptions of effort. This conscious down-regulation of exercise intensity is suggested to occur in an attempt to maintain homeostasis and prevent critically high core temperatures from being reached (Tucker *et al.,* 2004).

MULTIVARIATE ANALYSIS

Heart Rate and Central RPE

Previous studies have shown a strong relationship between heart rate and central RPE during dynamic exercise of a continuous nature (Nicholas *et al*., 2000). Similarly, there were strong correlations between heart rate and central RPE (Figure 11, page 78) for both conditions (R^2=0.86 for the CC and R^2=0.77 for the EC), which indicates that players were accurately able to reflect their cardiovascular responses during both conditions protocol. Therefore, due to these high correlations, RPE can be used to accurately predict player‘s heart rate responses during an 80-min simulated rugby protocol.

CONCLUSION

Therefore, the additional heat load, caused by wearing full rugby gear during the EC, caused players physiological and perceptual responses to become exacerbated, particularly during the second half of the EC protocol. These exacerbated responses caused players to perceive the simulated rugby protocol to be more taxing on the cardiovascular and muscular systems, which caused players to select an appropriate pacing strategy that would allow them to complete the exercise without reaching excessively high core temperatures and RPE. Therefore, the increased body temperatures, caused by wearing the full rugby kit in the EC, resulted in players‘ performance responses being hindered, possibly due to an earlier onset of fatigue.

CHAPTER VI

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

INTRODUCTION

Whilst rugby is an international sport, and is popular in many countries around the world, there still remains little scientific literature regarding the mechanisms of fatigue experienced during the game. Wearing clothing and protective gear has shown to increase athlete‘s physiological and perceptual responses, and negatively impact overall performance capacities (Kulka and Kenney, 2002; Cannon and Christie, 2009). Wearing clothing and protective gear has shown to hinder the mechanisms of heat loss (Goodman *et al*., 1995; Godek *et al*., 2005), causing athlete‘s body temperatures to continue rising towards critically high levels. Although rugby players have little choice in selecting the clothing to wear during match-play, the use of protective gear is dependent on the players personal preferences, as the use of these items have not yet been made compulsory in South Africa. According to Marshall *et al*. (2005), all protective gear, except for mouth guards and headgear, are largely ineffective in preventing serious injuries (Marshall *et al*., 2005), which raises questions surrounding whether or not these items should be worn at all. Therefore, the use of protective gear may only contribute to increasing body temperatures during exercise and provide little protection against the more serious injuries experienced in rugby. During exercise-induced hyperthermia, central mechanisms are proposed to down-regulate the amount of skeletal muscle recruited during exercise, ultimately reducing athletes work rates in an attempt to maintain homeostasis and prevent any catastrophic failure to vital organs occurring (Noakes *et al*., 2004; Lambert *et al*., 2005; St Clair Gibson *et al*., 2001; St Clair Gibson and Noakes, 2004; Tucker *et al*., 2004; Tucker *et al*., 2006). However, the mechanisms of fatigue experienced during exercise-induced hyperthermia remain unclear. Furthermore, it remains unclear whether wearing rugby clothing and protective gear worn during exercise causes body temperatures to be driven closer towards critical levels, thus hastening the onset of muscular fatigue. Therefore,

gaining insight into the mechanisms of fatigue experienced during rugby is important, as fatigue is identified as one of the primary causes of muscle injuries during match-play (Roig Pull and Ranson, 2007).

SUMMARY OF PROCEDURES

The 80-min simulated rugby protocol was designed to match the actual demands of match-play. The protocol used in this study was modified using existing literature (Cunniffe *et al*., 2006) to adapt the rugby protocol used by Brown (2009), and included only the non-contact activities of the game. The activities included standing, walking, jogging, striding and sprinting. Therefore, all the contact activities of rugby were excluded from the protocol. The protocol consisted of four quarters (20-min each). The first and second quarters (first-half) were identically matched, however the third- and fourth quarters (second half) were more intense and involved less time for recovery. Basic, demographic, anthropometrical and morphological data were obtained prior to experimentation at a preliminary testing session. At this preliminary session the experimental procedures were explained to the players, players were habituated to running on the treadmill and performing the activities of the simulated rugby protocol. Players were then required to fill out a physical health screening questionnaire and were then finally afforded the opportunity to ask any questions regarding the test. For the experimental phase, players were required to attend two testing sessions within a week (approximately 7 days) of each other. These were randomly assigned to each player. During the control condition (CC), players were required to perform the test wearing minimal (rugby shorts, underwear, short socks and running shoes) and during the experimental condition (EC), players wore full rugby clothing (rugby jersey, rugby shorts, long socks, running shoes, shoulder pads, scrumcap and thermal undergarment). Players were each given a telemetric core temperature pill (CorTemp HQ Inc, Florida, USA) to be taken three hours before testing commenced.

On arrival, the tester ensured that all the requirements had been fulfilled by the player and measured the environmental conditions (ambient temperature and

relative humidity) to ensure that testing could commence. Players were fitted with a heart rate monitor, the dominant leg was cleaned with alcohol and shaved in order to ensure good conductivity and then the electrodes were attached and all positions measured from an anatomical marker to ensure that future placement was identical. Players were instructed to lie down and relax for a minimum of two minutes and allow heart rate values to reach as close to resting values as possible. This measurement was used as the players resting heart rate. At the same time, players resting core temperatures were measured. Players were then required to perform a light warm-up (slow running) and perform their own stretching exercises.

Following this, baseline muscle activity was recorded prior to testing. During this protocol, player's performed a 3-min EMG treadmill protocol, designed by Rahnama *et al*. (2006), wearing only rugby shorts, short sock s and running shoes.The speeds of the activity categories in this protocol were 6 $km.h^{-1}$ (walking), 12 $km.h^{-1}$ (jogging), 15 $km.h^{-1}$ (cruising) and 21 $km.h^{-1}$ (sprinting). The speed of 5$km.h^{-1}$ was used as an interface between the measured speeds of 6, 12, 15 and 21 $km.h^{-1}$ in order to mimic the intermittent activity profile. This EMG protocol was performed three times during each condition (pre, half-time and post-protocol).

Immediately after this was completed, players got dressed into either conditions kit and performed the simulated rugby protocol. During either condition, players were required to follow the verbal instructions from the researcher to either stand still, walk, jog, stride, and sprint at set time intervals. A whistle was used as a signal for the transition between activities. Players performed these activities along a 20m long wooden walkway. Players were required to change direction once they reached either side of the walkway. During the maximal sprints, the researcher and research assistants provided motivation for the players, ensuring that the players were performing maximally. The physiological, biophysical, perceptual and performance responses were measured at set time intervals during the EC and were then compared to the same responses measured at the same time intervals during the CC.

As this investigation took a holistic approach, the following dependant variables were used in the analyses:

Physiological responses:	Heart rate
	Core temperature
Biophysical responses:	Electromyography
Perceptual responses:	‗Central' and ‗local' RPE
Performance response:	Total number of 20 metre shuttles completed during each 20-min work-bout

SUMMARY OF RESULTS

Players mean resting heart rates were similar prior to the commencement of both protocols. For both conditions, heart rate increased significantly in the transition from rest to exercise. For the EC, mean heart rate was significantly ($p < 0.05$) higher throughout the remainder of the 80-min simulated rugby protocol, compared to the CC.

Mean resting core temperature was similar for both conditions prior to testing. For both conditions, core temperature increased significantly over time during the rugby protocol. Core temperature responses were similar for both conditions during first-half of the protocol, as well as in the beginning stages of the second half. However, from 65- to 80-min, core temperature was significantly ($p < 0.05$) higher for the EC, compared to the CC. For both conditions, core temperature never reached the critically high levels of approximately 40° C, where exercise becomes regulated by central mechanisms of muscle fatigue.

For the vastus medialis (VM) and vastus lateralis (VL) muscles, recruitment increased significantly ($p < 0.05$) with increasing running speeds during the electromyographic (EMG) treadmill protocol. There were no significant changes in the activation of these muscles over time. However, the trend for the quadricep (VM and VL) muscle activity was to increase over time. For the VM muscle activation, there were no significant differences between conditions at all speeds and time intervals. However, for the VL, muscle activity was significantly ($p < 0.05$) lower for EC while running at 21 $km.h^{-1}$, compared to the CC. The muscle activation of the bicep femoris (BF) and semitendinosus (ST) increased significantly ($p < 0.05$) with increasing running speeds during the treadmill protocol. There were no significant changes in the muscle activity over time, except during the CC, where the muscle activity of the ST decreased significantly ($p < 0.05$) over time at 21 $km.h^{-1}$. Interestingly, the trend for the hamstring (BF and ST) muscle activity was to decrease over time. Similar to the results of the quadricep muscles, there were no significant differences in muscle activity for the hamstring muscles between conditions.

Central RPE increased significantly over time for both conditions. For the EC, central RPE was constantly higher than ratings during the CC throughout the protocol. Towards the end stages of the first- and second half of the EC protocol, central RPE was significantly ($p < 0.05$) higher than the CC. Local RPE also increased significantly over time during the protocols. Local RPE was similar for both conditions, however at the end of the EC protocol (80-min), local RPE was significantly ($p < 0.05$) higher than those reported during the CC.

During the first-half of both conditions, players completed a similar number of 20m shuttles. In contrast, during the second half of the CC protocol, players completed significantly ($p < 0.05$) more shuttles than the EC. Therefore, during the second half of the EC protocol, performance capacities were compromised.

STATISTICAL HYPOTHESES

HYPOTHESIS 1: BIOPHYSICAL RESPONSES

Muscular Activity

I). The null hypotheses proposed that there will no difference in muscle activity between conditions. The results force the acceptance of the null hypothesis for the vastus medialis (VM), bicep femoris (BF) and semitendinosus (ST) muscles, as the muscle activity was similar for both conditions. However, the results for the vastus lateralis muscle force the rejection of the null hypothesis, as muscle activity was significantly lower in the experimental condition, compared to the control condition.

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II).The null hypotheses proposed that there will no difference in muscle activity over time during both conditions. Results force the rejection of the null hypothesis for the vastus medialis during the control condition, vastus lateralis during both conditions and the semitendinosus muscle during the control condition, as muscle activity showed significant changes over time. However, there we no significant changes in muscle activity over time for the vastus medialis, during the experimental condition, the biceps femoris, during both conditions, and the semitendinosus during the experimental condition. Therefore, the null hypothesis was accepted for these muscles during the relevant conditions.

HYPOTHESIS 2: PHYSIOLOGICAL PARAMETERS

A). Cardiovascular Responses

I).The null hypothesis proposed that there will be no difference in heart rate responses between conditions. The results force the rejection of the null hypothesis, as heart rate was significantly higher for the experimental condition, compared to the control condition.

II). The null hypothesis proposed that there will be no change in heart rate responses over time during both conditions. Results forced the rejection of the null hypothesis, as heart rate showed significant increases in the first 20-min of both protocols. From 20- to 80-min of the protocol, heart rate remained fairly stable during both conditions, which forces the acceptance of the null hypothesis.

B). Core Temperature

I). The null hypothesis proposed that there will be no difference in core temperature responses between conditions. The null hypothesis was rejected, as core temperature was significantly higher for the experimental condition, compared to the control condition.

II). The null hypothesis proposed that there will be no change in core temperature responses over time during both conditions. Results forced the rejection of the null hypothesis, as core temperatures significantly increased from resting levels to measures recorded at 20-min during both protocols. During the control condition, core temperature remained fairly constant throughout the remainder of the protocol, which forces the acceptance of the null hypothesis. In contrast, during the experimental condition, core temperature increased significantly from 20- to 40-min, forcing the rejection of the null hypothesis for this time interval. For the remainder of both conditions, core temperature remained stable, which forces the acceptance of the null hypothesis.

HYPOTHESIS 3: PERCEPTUAL RESPONSES

A). Central Ratings of Perceived Exertion (RPE)

I). The null hypothesis proposed that there will be no difference in central RPE between conditions. The null hypothesis was rejected, as central RPE was significantly higher for the experimental condition, compared to the control condition.

II).The null hypothesis proposed that there will be no change in central RPE over time during both test conditions. Central RPE increased significantly from 5- to 20-min during both protocols, thus forcing the rejection of the null hypothesis. Central RPE remained fairly stable from 40- to 60-min for both conditions, which forces the acceptance of the null hypothesis for these time intervals. However, from 60- to 80-min, central RPE significantly increased for both conditions, which forces the rejection of the null hypothesis.

B). Local RPE

I). The null hypothesis proposed that there will be no difference in local RPE between conditions. Similar to the results of central RPE, players‘ local RPE was significantly higher towards the end stages of the experimental condition. Therefore, results forced the rejection of the null hypothesis.

II). The null hypothesis proposed that there will be no change in local RPE over time during both test conditions. Results force the rejection of the null hypothesis, as local RPE significantly increased from 5- to 20-min, and from 20- to 40-min during both protocols. Local RPE then remained fairly constant from 40- to 60-min of both protocol, thus forcing the acceptance of the null hypothesis for these time intervals. However, from 60- to 80-min, local RPE once again significantly increased, which forces the rejection of the null hypothesis.

HYPOTHESIS 4: PERFORMANCE RESPONSES

I). The null hypothesis proposed that there will be no difference in performance responses between conditions. Results indicate that performance capacities were significantly better for the control condition, compared to the experimental condition. Therefore, results force the rejection of the null hypothesis.

II). The null hypothesis proposed that there will be no change in performance responses over time during both test conditions. Results force the rejection of the null hypothesis for the control condition, as performance significantly improved in the second half of the protocol, compared to the first-half. However, for the experimental condition, performance responses remained stable throughout the protocol, thus forcing the acceptance of the null hypothesis.

CONCLUSION

In this study, players‘ biophysical responses of the quadricep (vastus medialis and vastus lateralis) and hamstring (bicep femoris and semitendinosus) musculature increased significantly at increased running speeds for both conditions. Furthermore, the muscle activity of the vastus medialis (VM), vastus lateralis (VL) and semitendinosus (ST) showed significant changes over time at certain time intervals for both conditions. However, for the biceps femoris (BF), there were no changes in muscle activity over time. The trend for the quadriceps muscles were to increase over time, whereas the trend for the hamstrings muscle activity (BF and ST) was to decrease over time, which suggests that fatigue, was greater in the hamstring musculature. The decreased muscle activity for the hamstrings over time, compared to the quadriceps could be explained by the high eccentric demands being placed o the hamstring muscles during the intermittent rugby protocol. This may increase the risk of injury as fatigue has been identified as one of the main factors causing hamstring injury.

Players‘ physiological (heart rate and core temperature) and perceptual (central and local RPE) responses were significantly higher during the experimental condition (EC), compared to the control condition (CC), due to the additional heat load caused by wearing the full rugby kit during a simulated rugby protocol. Furthermore, the physiological and perceptual responses significantly increased over time, as the intensity of the second half was more intense than the first-half. Therefore, this suggests that the exercise was the primary driver of physiological and perceptual responses, and the additional clothing and protective gear, worn during the rugby

protocol, caused these responses to become exacerbated, particularly in the second half of the EC protocol. Finally, player‘s performance capacities were significantly worsened during the experimental conditions protocol, due to the additional clothing and protective gear worn during the 80-min simulated rugby protocol. The additional heat load, caused by wearing this additional kit during exercise, resulted in players‘ physiological and perceptual responses becoming further exacerbated, particularly in the second half of the protocol. However, results indicate that players muscle activity was similar for both conditions, despite players reaching significantly higher body temperatures during the experimental condition, compared to the control condition. This may have been due to players never reaching excessively high core temperatures during both conditions, due to the intermittent nature of the protocol or from conscious mechanisms regulating exercise. Therefore, the decrements in performance experienced during the experimental conditions were not caused by central mechanisms, which are suggested to unconsciously down-regulate muscle activity, and therefore performance. Rather, it appears that the decrements in performance occurred as result of conscious perceptions of effort, which cause players to select an appropriate pacing strategy during the protocol that would allow them to complete the exercise, while maintaining homeostasis.

RECOMMENDATIONS

It is acknowledged that there are difficulties associated with conducting field-based studies, especially those in a sporting and a rugby environment, as rugby is very unpredictable in nature, involves many contact situations between players, and is played in many different environmental conditions. More research *in situ* is required in order to accurately assess the biophysical, physiological and perceptual demands placed on players while wearing full rugby kit. Furthermore, the impact of this additional kit, worn during actual match-play, needs to be conducted in order to determine whether this additional gear causes player‘s responses to become further exacerbated, which may result in an earlier onset of muscle fatigue. This will aid in understanding the mechanisms of muscle fatigue involved in rugby, whether increased body temperatures caused by wearing the additional rugby gear hastens the onset of fatigue, negatively impacts players overall performance capacities and increases the risk of injury. Due to the limitations of conducting field-based studies, it

may be more realistic to incorporate contact activities into the protocol, by using tackle bags in a laboratory study and have players perform more of the contact activities associated with rugby. Furthermore, it may be a more accurate reflection of actual rugby match-play if the protocol was performed on a field, where environmental conditions can play a role in impacting player‘s responses.

It is advisable to use a bigger sample size in order to allow for a greater generality of the findings and a better statistical significance. As the simulated rugby protocol was based on the time-motion data of inside backs, further studies should focus on the inside back position in order to lessen the variance created by performing an unaccustomed playing profile.

Players were required to perform the rugby protocol, which required players to rapidly accelerate and decelerate, as found in a rugby match. Therefore, players performed rapid eccentric contractions, which have been associated to muscle damage and swelling. Furthermore, player‘s body temperatures were significantly higher while performing the protocol in full rugby gear, which may have caused players to sweat more during the experimental conditions, compared to during the control conditions. Therefore, this muscle swelling and increased sweating may have influenced the conductivity of the EMG surface electrodes. Surface EMG is known to be influenced by many factors, which may influence the conductivity of the electrode activity and impact the accuracy of measurement. In order to be more accurate, alternative methods of measuring muscle activity should be used, such as needle EMG. However, this method has also been suggested to be an impractical method of measuring muscle activity during exercise.

Although players were asked to drink 500 ml of water 1-hour before the test, this could not be assured. A better approach for future studies would be to ask all players to consume 500 ml of water upon arrival at the laboratory, thus standardising their fluid levels.

The differentiation between the rugby clothing and protective gears‘ impact on players‘ responses during a rugby activity could not be ascertained in this study, as only two test conditions were used. Future studies should use three test conditions. In the first condition, players should perform the rugby protocol wearing minimal clothing (shorts, short socks and running shoes). In the second condition, players should wear protective gear with no rugby jersey (shorts, long socks, running shoes, shoulder pads, thermal top and scrumcap), and in the third condition, players should perform the rugby protocol while wearing both a jersey and protective gear (shorts, long socks, running shoes, standard rugby jersey, shoulder pads, thermal top and scrumcap).

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APPENDIX A:

WRITTEN INFORMATION TO PLAYERS

SIMULATED RUGBY PROTOCOL

BORG SCALE

PHYSICAL ACTIVITY SCREENING QUESTIONNAIRE

DATA COLLECTION SHEET

ETHICS APPROVAL

PLAYERS' CONSENT FORM

WRITTEN INFORMATION TO PLAYERS

Department of Human Kinetics and Ergonomics

Rhodes University

Written Information Sheet

Title of Study

Selected musculoskeletal, physiological, perceptual and performance changes in response to a simulated rugby activity while wearing different kits

General Aims of the Study

Presently, there is limited information regarding how rugby clothing and protective gear affects players‘ responses and performance due to increased heat stress. It has been shown that a rising body temperature may be the main factor causing fatigue during exercise. Therefore, the clothing and protective gear worn in rugby may cause body temperatures to reach excessively high temperatures to such an extent that performance may start to decline because this kit hinders heat loss. Therefore, the aim of this study is to investigate the impact that rugby clothing and protective gear has on player responses during an 80-minute (min) simulated rugby activity. Players‘ physiological, biophysical, perceptual and performance measures will be measured during this test.

Players

Only the non-contact activities of rugby are being used in this study. Therefore, only backs are going to be tested, as forwards perform more contact activities than backs.

Testing Sessions

There will be three testing sessions held in the Human Kinetics and Ergonomics (HKE) Department, Rhodes University. During the first testing session, all the procedures and objectives of the study will be explained to you, and give you an opportunity to ask any questions regarding the study. You will then be required to sign an informed consent if you wish to voluntarily participate in this study. All players may pull out of the experiment at any time without any negative consequences. You will then fill out a health screening questionnaire to ensure your safety during testing and ensure that you are a suitable candidate for this study. You will then be required to provide some basic data, and then the research assistants will take various anthropometric (stature, body weight and body mass index) and morphological measurements (Body Fat Percentage- using the skinfold technique).

The rugby protocol will then be explained and each activity will be demonstrated to you. You will be required to perform a 5-min trial run of the protocol as well as the treadmill protocol so that you can become accustomed to performing each activity of the protocol and running on a treadmill. The perceptual rating scales will then be explained to you, so that you understand how to use them appropriately. Finally, you will be randomly assigned to a test group. The duration of the first data collection session will be approximately 1.5 hours.

During test sessions 2 and 3, you will be asked to perform one of two conditions once only, while the researchers monitor certain responses. These work-bouts involve 2 clothing conditions during which you will wear either 1) full rugby clothing and protective gear (experimental kit) or 2) minimal clothing and no protective gear (control kit). Each test session will last approximately 100-mins. You will not perform both test conditions on the same day, so as to allow you enough time to fully recover. Therefore each of you will be given approximately one week to recover before performing the second test condition.

You will be required to swallow a sensor, which is approximately 20mm x 14mm in size (the size of a large medicinal pill). You will be offered water to swallow it with, and, unless you have particular difficulty swallowing pills, this will not be difficult. Within the pill, there is a telemetry system, micro battery and a quartz crystal temperature sensor.This pill will be used to accurately measure core temperature during testing. The pill transmits a signal via a magnetic current sent to the CorTemp^{TM} Data Recorder (HQ Inc., Florida, USA). The pill is designed to _lodge' itself in the gastrointestinal tract (The tube that extends from the mouth to the anus), and cannot be palpated from the surface. The pill is designed to remain in your system for approximately three days, during which, you will not consciously feel its presence. While the sensor is in your system there is very little danger of it falling apart, or of any of its parts becoming dispersed within your system. Should this occur, which is unlikely, these small pieces will simply be excreted along with natural waste products. While the sensor is inside you, you can eat and drink as normal and without experiencing any gastro-intestinal discomfort.

There is a risk that after the three days, the pill will not be naturally excreted from your system, but if by some chance you feel that the pill has not left your body, please do not worry, as the pill transmits a signal, which can be detected by me or a member of the research team. The pill will continue to transmit a signal as long as it remains in your system, and once excreted the signal will not be detectable anymore. The signal transmitted from the sensor pill represents very little danger to you, as is transmitted via magnetic currents to the data receiver. There are no documented risks of radiation, or any risk of developing cancer as a result of having this pill inside you.

The pill has been used successfully as a research tool since the late 1980's. While there remains very little risk to the participant, adverse effects cannot be ruled out despite every precaution being taken. Should any adverse effects be detected, please rest assured that the research assistants and I will immediately seek the advice of medical professionals and will ensure that you are properly taken care of, at no cost to yourself or your medical aid.

You will also have electrodes attached to your right leg, so that muscle activity can be measured, using a surface electromyographic (EMG) device (Muscle Tester Mega ME6000P16, Mega Electronics Ltd, Finland). To ensure proper electrode conduction, you will be asked to have small areas of your upper leg shaved with a new razor and swabbed with alcohol. Muscle activity will be measured during a 3-min (pre-test, half-time, and post-test) EMG treadmill protocol, performed three times during the 80-min rugby protocol (0-min, half-time (40-min) and at 80-min). A physiological strain rating will be calculated determined by using core temperature and heart rate measurements. Perceptual responses will be determined by using a rating scale which you will answer subjectively (RPE scale). A performance measure will be determined by calculating the total distance covered in each 20-min exercise-bout. This will be measured by counting how many 20m shuttles you can complete in every 20-min work bout.

Risks

Players may participate in this study only if they feel free of any illness (e.g. colds and flu etc) or injury. The risk factors associated with the movements made during the rugby protocol and simulated protocols are similar to those experienced during actual rugby match play. For example, making sudden turns or changing your running speed during the protocol could result in you twisting your ankles, pulling muscles or other slip, trip and fall injuries. Another risk associated with this test involves you working within high heart rate zones, which could place the cardiovascular system under a considerable amount of strain during testing. There are however, longer rest periods (where you stand completely still) during the adapted rugby-specific protocol, as well as between 20-min work bouts (5-min) which will allow you to recover. Before testing, you will be asked to take in adequate amount of water the day before testing, as well as one hour before (500 ml). During the protocol, you will be allowed to take in a controlled amount of water (100 ml) at set intervals (20-, 40- and 60-min). However, if you feel that you cannot continue without taking in additional water, there will be additional water available for you.

Information obtained will be handled in the strictest confidence, as only the researcher will handle this information during data analysis. You will be required to sign a consent form before participating in this test. By filling out the physical activity screening questionnaire (to be given out at the initial testing session) accurately, those of you at risk of injury or in danger while performing this test can be excluded from performing this test and ensure your safety during testing. Physiological responses will be monitored throughout and the test will be terminated by the research assistants (HKE honours students) if these are considered to be excessive. These physiological responses would include excessively high heart rates (greater than each player‘s age predicted maximum heart rate (220 – age), body temperature (above 39.5ºC), and extremely high ratings of exertion (RPE).

The electromyographic electrodes and Fixamol tape may also pose a risk, as the adhesive on the back of the electrodes may cause an allergic reaction to your skin. To ensure that this does not occur, you will be asked whether they have any skin allergies in the physical activity screening questionnaire. If you are unsure, a small piece of tape and an electrode will be placed on your skin to test whether a reaction occurs.

The ingestible telemetric pills you will be required to swallow before testing may also pose a risk to your health if you have had certain problems with swallowing or have intestinal problems. However, these risks will be dealt with in the physical activity screening questionnaire, and if you indicate that you may not be a suitable candidate for using this core temperature measurement equipment you will be excluded from participating in this study. This pill will remain in your system for several days (approximately 3 days), however you will be unaware of its presence in your system, you will not feel it, and will not cause you any harm while in your system. There is also no risk of the pill exploding while inside you. While the pill is inside your system you can continue eating and drinking as normal.

Benefits

You will be required to come in for a pre-test session where anthropometric, morphological and demographic data will be collected. This data will be made available to you at any time after, or during the test and will serve to educate you about your body and your physical fitness levels, which you can then use to work on areas that can be improved upon, and possibly improve your rugby performance.

After testing, the overall findings/summative results of this study will be made available to you in writing, to provide a greater understanding about how your physiological, perceptual and performance responses are affected by rugby kit during a rugby activity, as well as provide more information regarding the mechanisms of fatigue during rugby. The experience of participating in the experiment will hopefully provide you with information so that you can work on problem areas reduce the risk of developing musculoskeletal injuries. Furthermore, these data are likely to prove useful in reducing the risk of heat-related disorders, identifying optimal fluid replacement practices and rest periods during matches, and help to determine whether the design of protective gear and rugby clothing needs to be reassessed.

I would like to thank you for showing interest in my research and look forward to hopefully working with you.

Michael-John Cannon

Masters (MSc) student

SIMULATED RUGBY PROTOCOL

1st and 2nd quarter

Time (min:sec)	Activity
0	Stand
00:11	Walk
00:21	Jog
00:25	Stand
00:34	Walk
00:40	Jog
00:50	Stand
01:13	Sprint
01:15	Stand
01:25	Stride
01:27	Jog
01:32	Stand
01:40	Walk
01:50	Sprint
01:55	Jog
02:00	Stand
02:14	Stride
02:20	Jog
02:30	Walk
02:41	Stand
02:45	Sprint
02:55	Walk
03:00	Jog
03:19	Stand
03:34	Jog
03:45	Stand
03:50	Jog
03:54	Stride
04:00	Stand
04:15	Sprint
04:20	Jog
04:25	Stride
04:35	Stand

Time (min:sec)	Activity
04:50	Walk
05:00	Stand
05:11	Walk
05:21	Jog
05:25	Stand
05:34	Walk
05:40	Jog
05:50	Stand
06:13	Sprint
06:15	Stand
06:25	Stride
06:27	Jog
06:32	Stand
06:40	Walk
06:50	Sprint
06:55	Stand
07:45	Sprint
07:55	Walk
08:00	Jog
08:10	Stride
08:19	Stand
08:30	Walk
08:34	Jog
08:45	Stand
09:00	Stride
09:10	Walk
09:15	Sprint
09:20	Jog
09:25	Stride
09:35	Stand
10:34	Walk
10:40	Jog
10:59	Walk

Time (min:sec)	Activity
11:10	Stand
11:13	Sprint
11:15	Stand
11:25	Stride
11:27	Jog
11:32	Stand
11:40	Walk
11:50	Sprint
11:55	Jog
12:00	Walk
12:11	Stand
12:14	Stride
12:20	Jog
12:30	Walk
12:41	Stand
12:45	Sprint
12:55	Walk
13:00	Jog
13:10	Stand
13:30	Walk
13:34	Jog
13:45	Stand
13:50	Jog
13:54	Stride
14:00	Stand
14:10	Walk
14:15	Sprint
14:20	Jog
14:25	Stride
14:35	Stand
14:50	Walk
15:00	Stand
15:11	Walk

Time (min:sec)	Activity
15:21	Jog
15:24	Stand
15:33	Walk
15:40	Jog
15:50	Stand
16:13	Sprint
16:15	Stand
16:25	Stride
16:27	Jog
16:32	Stand
16:40	Walk
16:50	Sprint
16:55	Jog
17:00	Stand
17:10	Walk
17:14	Jog
17:24	Stride
17:30	Walk
17:42	Stand
17:48	Sprint
17:55	Walk
18:00	Jog
18:11	Stand
18:50	Jog
18:54	Stride
19:00	Stand
19:11	Walk
19:17	Sprint
19:24	Jog
19:28	Stride
19:35	Stand
20:00	Stand

Third Quarter

Time (min:sec)	Activity
0	Jog
00:11	Walk
00:21	Jog
00:25	Stand
00:34	Walk
00:40	Jog
00:50	Stand
01:13	Sprint
01:23	Stride
01:27	Jog
01:32	Stand
01:40	Walk
01:50	Sprint
01:55	Jog
02:00	Stand
02:14	Stride
02:20	Jog
02:30	Walk
02:41	Stand
02:45	Sprint
02:55	Walk
03:00	Jog
03:19	Stand
03:34	Jog
03:45	Stand
03:50	Jog
03:54	Stride
04:00	Stand
04:15	Sprint
04:20	Jog

Time (min:sec)	Activity
04:25	Stride
04:35	Stand
04:50	Walk
05:00	Stand
05:11	Walk
05:21	Jog
05:25	Stand
05:34	Walk
05:40	Jog
05:50	Stand
06:13	Sprint
06:20	Jog
06:32	Stand
06:40	Walk
06:50	Sprint
06:55	Stand
07:25	Sprint
07:35	Walk
07:50	Jog
08:10	Stride
08:19	Stand
08:30	Walk
08:34	Jog
08:45	Stand
09:00	Stride
09:10	Walk
09:20	Jog
09:25	Stride
09:35	Stand
10:34	Walk

Time (min:sec)	Activity
10:40	Jog
10:59	Walk
11:10	Stand
11:25	Stride
11:27	Jog
11:32	Stand
11:40	Walk
11:50	Sprint
11:55	Jog
12:00	Walk
12:11	Stand
12:20	Jog
12:30	Walk
12:41	Jog
12:45	Sprint
12:55	Walk
13:00	Jog
13:10	Stand
13:30	Walk
13:34	Jog
13:45	Stand
13:50	Jog
13:54	Stride
14:00	Stand
14:10	Walk
14:20	Jog
14:25	Stride
14:35	Stand
14:50	Walk
15:00	Stand
15:11	Walk

Time (min:sec)	Activity
15:21	Jog
15:24	Stand
15:33	Walk
15:40	Jog
15:50	Stand
16:13	Sprint
16:15	Stand
16:23	Stride
16:27	Jog
16:50	Sprint
16:55	Jog
17:00	Stand
17:10	Walk
17:14	Jog
17:24	Stride
17:30	Walk
17:42	Stand
17:48	Sprint
17:55	Walk
18:00	Jog
18:11	Stand
18:21	Jog
18:50	Walk
18:54	Stride
19:00	Stand
19:11	Walk
19:17	Sprint
19:28	Stride
19:35	Stand
20:00	Stand

Fourth Quarter

Time (min:sec)	Activity
0	Jog
00:11	Walk
00:21	Jog
00:25	Stand
00:34	Walk
00:40	Jog
00:50	Stand
01:13	Sprint
01:23	Stride
01:27	Jog
01:32	Stand
01:40	Walk
01:50	Sprint
01:55	Jog
02:00	Stand
02:07	Stride
02:20	Jog
02:30	Walk
02:41	Stand
02:45	Sprint
02:55	Walk
03:00	Jog
03:19	Stand
03:34	Jog
03:45	Stand
03:50	Jog
03:54	Stride
04:00	Stand
04:15	Sprint
04:20	Jog
04:25	Stride
04:35	Stand

Time (min:sec)	Activity
04:50	Walk
05:00	Stand
05:11	Walk
05:21	Jog
05:25	Stand
05:34	Walk
05:40	Jog
05:50	Stand
06:13	Sprint
06:20	Jog
06:32	Stand
06:40	Walk
06:50	Sprint
06:55	Stand
07:25	Sprint
07:35	Walk
07:50	Jog
08:10	Stride
08:19	Stand
08:30	Walk
08:34	Jog
08:45	Stand
09:00	Stride
09:10	Walk
09:15	Sprint
09:20	Jog
09:25	Stride
09:35	Stand
10:34	Walk
10:40	Jog
10:59	Walk

Time (min:sec)	Activity
11:10	Stand
11:13	Sprint
11:15	Stand
11:25	Stride
11:27	Jog
11:32	Stand
11:40	Walk
11:50	Sprint
11:55	Jog
12:00	Walk
12:11	Stand
12:14	Stride
12:20	Jog
12:30	Walk
12:41	Jog
12:45	Sprint
12:55	Walk
13:00	Jog
13:10	Stand
13:30	Walk
13:34	Jog
13:45	Stand
13:50	Jog
13:54	Stride
14:00	Stand
14:10	Walk
14:15	Sprint
14:20	Jog
14:25	Stride
14:35	Stand
14:50	Walk
15:00	Stand

Time (min:sec)	Activity
15:11	Walk
15:21	Jog
15:24	Stand
15:33	Walk
15:40	Jog
15:50	Stand
16:13	Sprint
16:15	Stand
16:25	Stride
16:31	Jog
16:40	Walk
16:50	Sprint
16:55	Jog
17:05	Stand
17:10	Walk
17:14	Jog
17:24	Stride
17:30	Walk
17:42	Stand
17:48	Sprint
17:55	Walk
18:00	Jog
18:11	Stand
18:16	Jog
18:50	Walk
18:54	Stride
19:00	Stand
19:11	Walk
19:17	Sprint
19:28	Stride
19:35	Stand
20:00	Stand

BORG SCALE

RATING OF PERCEIVED EXERTION (BORG)
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

PHYSICAL ACTIVITY SCREENING QUESTIONNAIRE

PHYSICAL ACTIVITY SCREENING QUESTIONNAIRE
Please fill out as fully and honestly as possible
Name: _____
Subject Code: _____
Body Mass: _____

MEDICAL HISTORY
Circle any of the following conditions, diseases or disorders that you have had in the past or are presently being treated for, by a physician or health care professional.

Heart problems
Anemia (a decreased in number of red blood cells or hemoglobin in the blood)
Eye Problems
Peripheral vascular disorders (obstruction of large arteries in the body)
Asthma
Hypoglycemia (lower than normal levels of blood sugar)
High/low blood pressure
Emphysema
Diabetes (high blood sugar)
Epilepsy
Migraine
Hyperthyroidism (overactive thyroid gland causing an overproduction of thyroid hormones)
Obstructive diseases of the gastrointestinal tract

Other (specify) _____

Do you have a history of disorders or impairments of the gag reflex? If so briefly describe the problem.

_____
_____
_____

Have you had previous gastrointestinal surgery? If so briefly describe.

_____
_____
_____

Are you going to undergo Nuclear Magnetic Resonance (NMR)/ Magnetic Resonance Imaging (MRI) scanning during the time that the telemetric sensor is within your body?_____

Do you have any digestive disorders? If so, briefly describe

_____
_____
_____

Do you have a cardiac pacemaker or other implanted electromedical device in your body? _____

Have you recently experienced feelings of nausea or have you vomited recently (past 48-hrs)? If so, please provide full details

_____
_____
_____

Do you have any skin allergies/ Are you allergic to any adhesives used on the back of tape/strapping (e.g. Elastoplast)? If so, please describe what causes the allergic reaction?

_____
_____
_____

Have you had any recent medical problems? If so give details below.

_____
_____
_____

Are you currently suffering from any orthopaedic (bone) disorder problems? If so briefly describe the problem.

_____
_____
_____

Do you have any other medical conditions not mentioned in this questionnaire?

_____
_____
_____

Please indicate any prescribed or over-the-counter medication you are currently taking or have taken in the last 6 months (e.g. aspirin, anti-depressants, Ritalin etc).

_____
_____
_____

OTHER HABITS

Please circle appropriate box.
Do you smoke? Yes No
If yes, how many cigarettes per day? >40 20-40 10-19 1-9

EXERCISE HISTORY

Do you exercise regularly?
Yes No

How many days per week do you normally spend performing at least 30 minutes of moderate to strenuous exercise? 0 1 2 3 4 5 6 7

Do you experience shortness of breath or chest discomfort with exercise? Yes No
Can you jog 5km continuously at a moderate pace without discomfort?
Yes No

Provide a rough average of the number of organized/scheduled physical activity sessions you participate in during the week. Circle the appropriate choice(s) and fill in the number of sessions.

Jogging ____ Gym_____ Touch Rugby_____ Rugby _____
Fitness ____ Swimming_____ Other _____

DATA COLLECTION SHEET

PRE-TEST

Name: _____Code: _____

Position: _____ Test Condition: _____

Humidity _____% Ambient Temperature_____°C

Mass: _____kg

Age:___.

Stature: _____mm

Resting HR:_____

Resting Core Temperature:_____

Muscle Activity: Start time:_____ End time:_____

Body Fat %: Chest:_____

Subscapularis:_____

Tricep:_____

Suprailiac Crest:_____

Abdominal:_____

Thigh:_____

Calf:_____

5-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE: _____**
- **Central RPE:_____**

10-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE_____**
- **Central RPE_____**

15-MIN

- **Core Temperature: _____**
- **Heart rate: _____**
- **Local RPE: _____**
- **Central RPE: _____**

20-MIN

- **Core Temperature:_____**
- **Local RPE_____**
- **Central RPE_____**
- **Heart Rate**
- **Number of Shuttles :_____**

25-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE:_____**
- **Central RPE:_____**

30-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE_____**
- **Central RPE_____**

35-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE:_____**
- **Central RPE:_____**

40-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE_____**
- **Central RPE_____**
- **Number of Shuttles :_____**
- **EMG: Start Time- _____**
  End Time: _____

45-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE:_____**
- **Central RPE:_____**

50-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE:_____**
- **Central RPE:_____**
-

55-Min Core Temperature:_____

- **Heart Rate: _____**
- **Local RPE:_____**
- **Central RPE:_____**

60-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE_____**
- **Central RPE_____**
- **Number of Shuttles :_____**

65-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE:_____**
- **Central RPE:_____**

70-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE:_____**
- **Central RPE:_____**

75-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE:_____**
- **Central RPE:_____**

80-MIN

- **Core Temperature:_____**
- **Heart Rate: _____**
- **Local RPE_____**
- **Central RPE_____**
- **Number of Shuttles :_____**
- **Muscle Activity:**
  Start Time: _____
  End Time: _____

ETHICS APPROVAL

Human Kinetics and Ergonomics Ethics Committee Report

RHODES UNIVERSITY

Student Name:	Mike Cairns
Type of Research:	Masters Research Project
Project Title:	Selected musculoskeletal, physiological, perceptual and performance changes in response to a simulated rugby activity while wearing two different kits
Supervisor:	Dr Candice Christie
Report compiled:	14 June 2011

Approved	✓	Approved, on condition that suggestions have been effected	Request for rework and resubmission / Rejected

The requested changes have been effected and your application now has ethical approval.

Best of luck with your research

Signed

Dr CJ Christie
Chair: Human Kinetics and Ergonomics Ethics Committee

Confidential
HKE Ethical Committee Review Form
June 14, 2011

PLAYERS CONSENT FORM

I, _____ having been fully informed of the research entitled:

Selected musculoskeletal and physiological changes in response to simulated rugby activity comparing two different kits

Do hereby give my consent to act as a participant in the above named research.

I am fully aware of the procedures involved as well as the potential risks and benefits attendant to my participation as explained to me verbally and in writing. In agreeing to participate in this research, I waiver any legal recourse against the researchers of Rhodes University, from any and all injuries sustained. This waiver shall be binding upon my heirs and personal representatives. I realise that it is necessary for me to promptly report to the researchers any signs or symptoms indicating any abnormality or distress. I am aware that I may withdraw from participation, without consequences, at any time during the research. I was not pressured into participating in this research test, and did so voluntarily. I am aware that my anonymity will be protected at all times, and that all the information collected, including photographs taken, may be used and published for statistical or scientific purposes.

I have read the information sheet accompanying this form and understand it. Any questions that may have occurred to me have been answered to my satisfaction.

PLAYERS (OR LEGAL REPRESENTATIVE)

_____ _____

(Print name) (Signed) (Date)

PERSON ADMINISTERING INFORMED CONSENT

_____ _____

(Print name) (Signed) (Date)

WITNESS

_____ _____

(Print name) (Signed) (Date)

APPENDIX B:

STATISTICAL TABLES

BIOPHYSICAL RESPONSES

PHYSIOLOGICAL RESPONSES

PERCEPTUAL RESPONSES

PERFORMANCE RESPONSES

BIOPHYSICAL RESPONSES

Vastus Medialis

Effect	Repeated Measures Analysis of Variance (Spreadsheet1) Sigma-restricted parameterization Effective hypothesis decomposition; Std. Error of Estimate: .5728403				
	SS	Degr. of Freedom	MS	F	p
Intercept	*280.1252*	*1*	*280.1252*	*853.6602*	*0.000000*
Error	2.9533	9	0.3281		
CONDS	0.4115	1	0.4115	1.3528	0.274706
Error	2.7379	9	0.3042		
TIME	1.1793	2	0.5897	2.5054	0.109664
Error	4.2364	18	0.2354		
SPEEDS	*24.5769*	*3*	*8.1923*	*88.2674*	*0.000000*
Error	2.5059	27	0.0928		
CONDS*TIME	0.5181	2	0.2591	1.3992	0.272377
Error	3.3328	18	0.1852		
CONDS*SPEEDS	0.0201	3	0.0067	0.0637	0.978559
Error	2.8394	27	0.1052		
TIME*SPEEDS	0.2256	6	0.0376	0.5801	0.744564
Error	3.5006	54	0.0648		
CONDS*TIME*SPEEDS	0.2279	6	0.0380	0.7240	0.632116
Error	2.8330	54	0.0525		

Vastus Lateralis

Effect	Repeated Measures Analysis of Variance (Spreadsheet10) Sigma-restricted parameterization Effective hypothesis decomposition; Std. Error of Estimate: .3474965				
	SS	Degr. of Freedom	MS	F	p
Intercept	*219.7016*	*1*	*219.7016*	*1819.418*	*0.000000*
Error	1.0868	9	0.1208		
CONDS	0.1809	1	0.1809	2.857	0.125262
Error	0.5700	9	0.0633		
SPEEDS	*0.5148*	*3*	*0.1716*	*2.963*	*0.049878*
Error	1.5639	27	0.0579		
TIME	0.1451	2	0.0726	2.314	0.127547
Error	0.5645	18	0.0314		
CONDS*SPEEDS	0.2264	3	0.0755	1.275	0.302974
Error	1.5984	27	0.0592		
CONDS*TIME	0.0740	2	0.0370	2.263	0.132842
Error	0.2943	18	0.0164		
SPEEDS*TIME	*19.3063*	*6*	*3.2177*	*91.199*	*0.000000*
Error	1.9052	54	0.0353		
CONDS*SPEEDS*TIME	*0.4918*	*6*	*0.0820*	*3.368*	*0.006812*
Error	1.3142	54	0.0243		

Biceps Femoris

Effect	Repeated Measures Analysis of Variance (Spreadsheet20) Sigma-restricted parameterization Effective hypothesis decomposition; Std. Error of Estimate: .5058000				
	SS	Degr. of Freedom	MS	F	p
Intercept	*170.6500*	*1*	*170.6500*	*667.0349*	*0.000000*
Error	2.3025	9	0.2558		
CONDS	0.2153	1	0.2153	2.3493	0.159701
Error	0.8247	9	0.0916		
SPEEDS	0.5675	3	0.1892	0.9875	0.413438
Error	5.1722	27	0.1916		
TIME	0.1952	2	0.0976	1.5922	0.230834
Error	1.1033	18	0.0613		
CONDS*SPEEDS	0.1203	3	0.0401	0.3378	0.798140
Error	3.2059	27	0.1187		
CONDS*TIME	0.0724	2	0.0362	0.6387	0.539556
Error	1.0202	18	0.0567		
SPEEDS*TIME	*10.3035*	*6*	*1.7172*	*30.4679*	*0.000000*
Error	3.0436	54	0.0564		
CONDS*SPEEDS*TIME	0.4673	6	0.0779	1.1432	0.350325
Error	3.6789	54	0.0681		

Semitendinosus

Effect	Repeated Measures Analysis of Variance (Spreadsheet30) Sigma-restricted parameterization Effective hypothesis decomposition; Std. Error of Estimate: .3144352				
	SS	Degr. of Freedom	MS	F	p
Intercept	*190.9427*	*1*	*190.9427*	*1931.259*	*0.000000*
Error	0.8898	9	0.0989		
CONDS	0.1698	1	0.1698	2.361	0.158765
Error	0.6472	9	0.0719		
SPEEDS	0.2100	3	0.0700	1.008	0.404580
Error	1.8759	27	0.0695		
TIME	0.0706	2	0.0353	0.671	0.523485
Error	0.9466	18	0.0526		
CONDS*SPEEDS	*0.3139*	*3*	*0.1046*	*3.134*	*0.041852*
Error	0.9015	27	0.0334		
CONDS*TIME	0.0073	2	0.0037	0.120	0.887408
Error	0.5476	18	0.0304		
SPEEDS*TIME	*17.7875*	*6*	*2.9646*	*61.136*	*0.000000*
Error	2.6186	54	0.0485		
CONDS*SPEEDS*TIME	0.3910	6	0.0652	1.532	0.185304
Error	2.2962	54	0.0425		

PHYSIOLOGICAL RESPONSES

Heart Rate

Effect	Repeated Measures Analysis of Variance (Spreadsheet1) Sigma-restricted parameterization Effective hypothesis decomposition; Std. Error of Estimate: 28.82364				
	SS	Degr. of Freedom	MS	F	p
Intercept	*10485383*	*1*	*10485383*	*12620.79*	*0.000000*
Error	9139	11	831		
COND	*6886*	*1*	*6886*	*28.85*	*0.000226*
Error	2626	11	239		
TIME	*254637*	*16*	*15915*	*305.30*	*0.000000*
Error	9175	176	52		
COND*TIME	*1467*	*16*	*92*	*3.25*	*0.000060*
Error	4959	176	28		

Core Temperature

Effect	Repeated Measures Analysis of Variance (Spreadsheet3_(Recovered)) Sigma-restricted parameterization Effective hypothesis decomposition; Std. Error of Estimate: 1.823898				
	SS	Degr. of Freedom	MS	F	p
Intercept	*600558.8*	*1*	*600558.8*	*180532.1*	*0.000000*
Error	36.6	11	3.3		
CONDITIO	2.1	1	2.1	2.1	0.176106
Error	11.2	11	1.0		
TIME	*59.9*	*16*	*3.7*	*69.9*	*0.000000*
Error	9.4	176	0.1		
CONDITIO*TIME	*1.8*	*16*	*0.1*	*3.1*	*0.000112*
Error	6.3	176	0.0		

PERCEPTUAL RESPONSES

Central RPE

Effect	Repeated Measures Analysis of Variance (Spreadsheet21) Sigma-restricted parameterization Effective hypothesis decomposition; Std. Error of Estimate: 4.319699				
	SS	Degr. of Freedom	MS	F	p
Intercept	*80823.02*	*1*	*80823.02*	*4331.398*	*0.000000*
Error	205.26	11	18.66		
CONDITIO	*180.13*	*1*	*180.13*	*7.185*	*0.021391*
Error	275.78	11	25.07		
TIME	*675.27*	*15*	*45.02*	*25.099*	*0.000000*
Error	295.95	165	1.79		
CONDITIO*TIME	9.16	15	0.61	0.670	0.811054
Error	150.43	165	0.91		

Local RPE

Effect	Repeated Measures Analysis of Variance (Spreadsheet16) Sigma-restricted parameterization Effective hypothesis decomposition; Std. Error of Estimate: 8.738737				
	SS	Degr. of Freedom	MS	F	p
Intercept	*70200.17*	*1*	*70200.17*	*919.2651*	*0.000000*
Error	840.02	11	76.37		
CONDITIO	60.17	1	60.17	3.3380	0.094928
Error	198.27	11	18.02		
TIME	*1066.75*	*15*	*71.12*	*49.0844*	*0.000000*
Error	239.06	165	1.45		
CONDITIO*TIME	19.58	15	1.31	1.5172	0.104088
Error	141.98	165	0.86		

PERFORMANCE RESPONSES

Effect	Repeated Measures Analysis of Variance (Spreadsheet20) Sigma-restricted parameterization Effective hypothesis decomposition; Std. Error of Estimate: 19.92115 SS	Degr. of Freedom	MS	F	p
Intercept	869823.4	1	869823.4	2191.806	0.000000
Error	4365.4	11	396.9		
CONDITIO	1001.0	1	1001.0	20.651	0.000838
Error	533.2	11	48.5		
TIME	1367.0	3	456.0	27.191	0.000000
Error	553.4	33	16.8		
CONDITIO*TIME	385.7	3	128.6	7.630	0.000523
Error	556.0	33	16.8		