EFFECT OF REPEATED ECCENTRIC DEMANDS PLACED ON THE LOWER LIMB MUSCULATURE DURING SIMULATED RUGBY UNION PLAY

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THESIS

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ABSTRACT

Epidemiological studies consistently report that muscular strains are a primary injury type in rugby union with the majority of the strains occurring to the quadricep and hamstring musculature. Recently it has been suggested that poor eccentric muscular strength is a precursor to hamstring and quadriceps strains during intermittent sports that require rapid acceleration and deceleration. Despite the high incidence of these muscle injuries in Rugby Union there has been little research into the possible mechanisms involved. Thus, the purpose of this study was to measure the physiological and perceptual responses during a simulated Rugby Union laboratory protocol and further, to identify changes in muscle recruitment patterns and muscle strength over time by comparing this protocol to a continuous, constant load protocol covering the same distance. The experimental condition (EXP) required university level players to perform 80 minutes of simulated rugby union play in a laboratory setting (on a walkway of 22m) which was compared to that of a control condition (CON) which involved subjects covering the same distance, at a constant speed of 4.2km.h^{-1} on a treadmill. Physiological, biophysical and perceptual responses were measured pre-, at half-time and post-protocol. Heart rate was significantly ($p<0.01$) greater as a result of EXP in comparison to the CON. Electromyography (EMG) of the vastus medialis was significantly ($p<0.01$) greater during the CON protocol. The EXP condition elicited higher iEMG activity in the hamstring musculature at all time intervals. In addition the iEMG of the semitendinosus decreased significantly ($p<0.01$) as a result of the EXP protocol. Peak eccentric knee extensors (EXT) (-13.19%) and flexors (FLEX) (-12.81%) torque decreased significantly during the experimental protocol. After passive half-time (236.67 $\pm$ 56.27Nm (EXT) and 173.89 $\pm$ 33.3NM (FLEX)) and at the end of the protocol (220.39 $\pm$ 55.16Nm and 162.89 $\pm$ 30.66Nm) reduced relative to pre protocol (253.89 $\pm$ 54.54Nm and 186.83 $\pm$ 33.3Nm). Peak eccentric knee extensors did not change during the control protocol. „Central" and "Local" Rating of Perceived Exertion values were significantly ($P<0.01$) greater during the EXP protocol with an increased incidence of hamstring discomfort and perceived pain (5 out of 10). The EXP protocol resulted in significantly ($p<0.01$) increased incidence of delayed onset muscle soreness (DOMS). In conclusion, a stop-start laboratory protocol elicited increased heart rate, negatively impacted on muscle activity of the hamstrings, decreased eccentric strength in the lower limb

musculature, resulted in increased ratings of „Central" and „Local" exertion and increased pain perception and increased incidence of DOMS. Thus, a stop-start rugby specific laboratory protocol has a negative impact on performance. Due to the specificity of the protocol being designed to match the demands of competitive match play it is expected that these changes in heart rate, muscle activity and strength, particularly eccentric strength, will impact negativity on performance during rugby match play and increase the likelihood of injury.

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CHAPTER I

INTRODUCTION

BACKGROUND TO THE STUDY

A vast amount of research has focussed on the demands placed on the body during steady-state activities, specifically those of an endurance-type nature, such as long distance running (Boobis *et al*., 1983; Cheetham and Williams, 1987; Coggan and Coyle, 1988). In contrast, the effects of intermittent sports, such as Rugby Union, cricket and hockey have not received as much attention, resulting in a lack of understanding of the demands placed on the body by these types of sports (Burke and Hawley, 1997; Drust *et al*., 2000).

Epidemiological studies consistently report on muscular strains being the main injury in sports of an intermittent nature (Mellalieu, 2008) and the most common of these strains are those that affect the quadriceps and hamstring musculature. Brooks *et al.* (2006) state that hamstring injuries alone account for 6-15% of all injuries incurred during Rugby Union. While there are many risk factors for musculoskeletal strain, scientists have hypothesized that poor eccentric muscle strength is a precursor to muscle strains during intermittent sports, including Rugby Union (McLean, 1992), which requires rapid acceleratory and decceleratory movements (Devlin 2000).However, to the author"s knowledge, no research has focussed on the changes, in specifically eccentric strength, but also in muscle recruitment patterns, over the time course of a rugby match. Therefore, it was hypothesized that poor eccentric strength per se may not be the only risk factor, but also changes in muscle activation and eccentric strength over time in a rugby game may be important influencing factors in the overall risk of injury particularly in the latter half of the game.

Rugby is played throughout the world with the International Rugby Board (IRB) encompassing 95 national unions (IRB). According to the IRB, rugby is played in over

100 countries; spanning 6 continents. Since the professionalization of Rugby Union in August 1995, the sport has undergone considerable changes both on and off the field. The recent Rugby World Cup in France was the world‟s third largest sporting event (Mellalieu, 2008). Such is the nature of the sport that lucrative contracts are now attainable for elite players, coaches and management staff. In professional team sports, the financial implications associated with success and failure place coaches under severe pressure to maximize their team‟s performance. One way of achieving this is through the development and implementation of effective training programmes. Training is designed to elicit improvements in strength, power, endurance, skill and tactical preparedness of players to maximize both individual and team performance (Brooks *et al.,* 2008). It is therefore imperative that the physical demands of competitive rugby be properly researched and understood, in order to optimise the training regimes of the players (Roberts *et al.,* 2008). Further, it is important to gain a more holistic understanding of the biomechanical, physiological and perceived demands placed on these players during all forms of rugby play. To date, research in this area has been sparse, largely due to the logistical constraints of assessing responses *in situ* and/or replicating rugby within a laboratory setting (Roberts *et al*., 2008; Quarrie *et al.,* 1995). Research is often undertaken within the confines of the environment of the team, organization or governing body (James *et al.,* 2005). Subsequently, these processes are rarely subjected to the scrutiny of methodological rigour required for use in academic publications (James *et al*., 2005). Nonetheless, it is imperative to endeavour to quantify the demands of competitive rugby in order to develop specific training regimes, which can subsequently mimic the physiological conditions imposed by the game. According to Brooks *et al.* (2008) effective conditioning and skills‟ training can help athletes meet the physical and technical demands of their sport and improve their performance while decreasing the likelihood of injury.

Rugby is a field-based team sport eliciting a variety of physiological responses resulting from low intensity aerobic bouts combined with repeated high intensity „all-out‟ sprints as well as many contact situations (Duthie *et al*., 2003). The activity profile is intermittent and complex in nature and varies depending on the position played (Mclean *et al*.,

1992). Furthermore, the physical and physiological demands of rugby depend not only on the rules of the game, but also on the characteristics of each particular game (Smith, 1998). Adding to the complexity, rugby is a physical game in nature with many contact situations requiring athletes to be fit, fast, muscular and skilful and prepared to engage in contact situations.

While understanding more of the contact impact is imperative, this study focussed specifically on the „stop-start" nature of the game with specific emphasis on the inside back players who spend the largest percentage of the game sprinting and who complete the most sprints compared to any other position (Duthie *et al*., 2005). There is a large focus on rapid acceleration in order to outpace the opposition and deceleration in order to stay „on-sides" and be in a position where one is able to receive the ball again. Therefore, despite all the different positions in Rugby Union, a common activity to all players is the repeated acceleratory and decceleratory movements and therefore repeated eccentric demands (McLean, 1992; Duthie *et al*., 2005). Repeated eccentric contractions have been shown to be associated with impaired muscle function and, more importantly, it has been suggested that eccentric damage to a particular muscle may pre-dispose it to injury (Fridén *et al*., 1981; Fridén *et al*., 1983). While the impact of this type of activity has received some research focus in other intermittent sports, rugby-specific research is lacking. This study therefore focussed specifically on the „stop-start" nature of the locomotion activities typically associated with inside back play in Rugby Union.

Charteris *et al*. (1976) recognised that there are many factors which influence performance and proposed the "Centre-M: Human Kinetic" model. The four broad areas of this model include the physical, biological and psychophysical parameters of human movement. The final domain proposed is a conceptual domain and highlights the importance of a holistic, interdisciplinary approach (Charteris *et al*., 1976). The current study therefore took a holistic stance by acknowledging that these factors impact directly and indirectly on performance during Rugby Union. Consequently, the current study assessed the physical demands using heart rate, muscle activation and strength

measures, while also assessing the psychophysical demands using body discomfort, rating of perceived exertion and muscle soreness.

STATEMENT OF THE PROBLEM

Despite the high incidence of hamstring and quadriceps muscle injuries in Rugby Union there has been little research into the possible mechanisms involved. Thus, the purpose of this study was to measure the physiological and perceptual responses during a simulated Rugby Union laboratory protocol and further, to identify changes in muscle recruitment patterns and muscle strength over time by comparing this protocol to a continuous, constant load protocol covering the same distance.

RESEARCH HYPOTHESIS

It was expected that the rugby specific protocol would elicit higher heart rate responses, muscle activity, perceptions of effort and body discomfort compared to the constant load protocol. It was further expected, that eccentric strength of the hamstring musculature would be compromised over time in the experimental protocol.

STATISTICAL HYPOTHESIS

1. **Hypothesis 1:** The first null hypothesis proposed is that there will be no change in the heart rate responses between protocols and over time.

   H_o: $\mu HRcont_{pre} = \mu HRexp_{pre} = \mu HRcont_{20} = \mu HRexp_{20} = \mu HRcont_{half} = \mu HRexp_{half} = \mu HRcont_{60} = \mu HRexp_{60} = \mu HRcont_{post} = \mu HRexp_{post}$

   H_a: $\mu HRcont_{pre} \neq \mu HRexp_{pre} \neq \mu HRcont_{20} \neq \mu HRexp_{20} \neq \mu HRcont_{half} \neq \mu HRexp_{half} \neq \mu HRcont_{60} \neq \mu HRexp_{60} \neq \mu HRcont_{post} \neq \mu HRexp_{post}$

2. **Hypothesis 2:** The second null hypothesis proposed is that there will be no difference in muscle activity between protocols and over time.

   H_o: $\mu MAcont_{pre} = \mu MAexp_{pre} = \mu MAcont_{half} = \mu MAexp_{half} = \mu MAcont_{post} = \mu MAexp_{post}$

H_a: $\mu MAcont_{pre} \neq \mu MAexp_{pre} \neq \mu MAcont_{half} \neq \mu MAexp_{half} \neq \mu MAcont_{post} \neq \mu MAexp_{post}$

3. **Hypothesis 3**

a) **Hypothesis 3a:** The third null hypothesis proposed is that there will be no difference in the following concentric strength parameters between protocols and over time.

i) Peak torque

H_o: $\mu PTcont_{pre} = \mu PTexp_{pre} = \mu PTcont_{half} = \mu PTexp_{half} = \mu PTcont_{post} = \mu PTexp_{post}$

H_a: $\mu PTcont_{pre} \neq \mu PTexp_{pre} \neq \mu PTcont_{half} \neq \mu PTexp_{half} \neq \mu PTcont_{post} \neq \mu PTexp_{post}$

ii) Work

H_o: $\mu Wcont_{pre} = \mu Wexp_{pre} = \mu Wcont_{half} = \mu Wexp_{half} = \mu Wcont_{post} = \mu Wexp_{post}$

H_a: $\mu Wcont_{pre} \neq \mu Wexp_{pre} \neq \mu Wcont_{half} \neq \mu Wexp_{half} \neq \mu Wcont_{post} \neq \mu Wexp_{post}$

iii) Power

H_o: $\mu Pcont_{pre} = \mu Pexp_{pre} = \mu Pcont_{half} = \mu Pexp_{half} = \mu Pcont_{post} = \mu Pexp_{post}$

H_a: $\mu Pcont_{pre} \neq \mu Pexp_{pre} \neq \mu Pcont_{half} \neq \mu Pexp_{half} \neq \mu Pcont_{post} \neq \mu Pexp_{post}$

b) **Hypothesis 3b:** The third null hypothesis proposed is that there will be no difference in the following eccentric strength parameters between protocols and over time.

i) Peak torque

H_o: $\mu PTcont_{pre} = \mu PTexp_{pre} = \mu PTcont_{half} = \mu PTexp_{half} = \mu PTcont_{post} = \mu PTexp_{post}$

H_a: $\mu PTcont_{pre} \neq \mu PTexp_{pre} \neq \mu PTcont_{half} \neq \mu PTexp_{half} \neq \mu PTcont_{post} \neq \mu PTexp_{post}$

ii) Work

H_o: $\mu Wcont_{pre} = \mu Wexp_{pre} = \mu Wcont_{half} = \mu Wexp_{half} = \mu Wcont_{post} = \mu Wexp_{post}$

H_a: $\mu Wcont_{pre} \neq \mu Wexp_{pre} \neq \mu Wcont_{half} \neq \mu Wexp_{half} \neq \mu Wcont_{post} \neq \mu Wexp_{post}$

iii) Power

H_o: $\mu Pcont_{pre} = \mu Pexp_{pre} = \mu Pcont_{half} = \mu Pexp_{half} = \mu Pcont_{post} = \mu Pexp_{post}$

H_a: $\mu Pcont_{pre} \neq \mu Pexp_{pre} \neq \mu Pcont_{half} \neq \mu Pexp_{half} \neq \mu Pcont_{post} \neq \mu Pexp_{post}$

4. **Hypothesis 4:** The fourth null hypothesis proposed is that there will be no difference in the ratings of perceived exertion (RPE) between protocols and over time.

   H_o: $\mu RPEcont_{pre} = \mu RPEexp_{pre} = \mu RPEcont_{20} = \mu RPEexp_{20} = \mu RPEcont_{half} = \mu RPEexp_{half} = \mu RPEcont_{60} = \mu RPEexp_{60} = \mu RPEcont_{post} = \mu RPEexp_{post}$

   H_a: $\mu RPEcont_{pre} \neq \mu RPEexp_{pre} \neq \mu RPEcont_{20} \neq \mu RPEexp_{20} \neq \mu RPEcont_{half} \neq \mu RPEexp_{half} \neq \mu RPEcont_{60} \neq \mu RPEexp_{60} \neq \mu RPEcont_{post} \neq \mu RPEexp_{post}$

5. **Hypothesis 5:** The fifth null hypothesis proposed is that there will be no difference in the ratings of muscle soreness during the 7 days post the experimental and control protocol.

   H_o: $\mu MScont_1 = \mu MSexp_1 = \mu MScont_2 = \mu MSexp_2 = \mu MScont_3 = \mu MSexp_3 = \mu MScont_4 = \mu MSexp_4 = \mu MScont_5 = \mu MSexp_5 = \mu MScont_6 = \mu MSexp_6 = \mu MScont_7 = \mu MSexp_7$

   H_a: $\mu MScont_1 \neq \mu MSexp_1 \neq \mu MScont_2 \neq \mu MSexp_2 \neq \mu MScont_3 \neq \mu MSexp_3 \neq \mu MScont_4 \neq \mu MSexp_4 \neq \mu MScont_5 \neq \mu MSexp_5 \neq \mu MScont_6 \neq \mu MSexp_6 \neq \mu MScont_7 \neq \mu MSexp_7$

Where:

- HR = Heart rate responses
- cont = Control protocol
- exp = Experimental protocol
- Pre = pre-protocol
- 20 = 20min into protocol
- half = half-way through protocol (i.e. 40 min)
- 60 = 60min into protocol
- post = post completion of 80 minutes protocol
- MA – Muscle activity
- PT – Peak Torque
- W – Work
- P- Power
- RPE – rating of perceived exertion
- MS – muscle soreness
- 1….7 – days post protocol

DELIMITATIONS

The study was delimited to 18 male rugby players, aged 18-23 years. All players were part of the Rhodes University, Grahamstown, South Africa first team rugby squad. The primary focus of this study was to identify changes in muscular activity and in eccentric and concentric force production changes of the quadriceps and hamstring musculature during a simulated rugby-specific laboratory protocol. Thus, each subject was required to perform two 80-minute laboratory protocols; a control protocol during which subjects walked at a constant speed of 4.2km.h^{-1}, and a rugby-specific protocol during which subjects completed an 80-minute intermittent protocol covering the same distance as the control protocol. This was to standardise the distance covered, but to negate the impact of rapid acceleration and deceleration. The rugby specific protocol comprised standing, walking, jogging, striding and sprinting, dispersed intermittently and designed to mimic the physiological demands of Rugby Union.

In the first session, the protocols were thoroughly explained to the subjects. All subjects, regardless of experience, were habituated to the CYBEX Trotter 900T treadmill and the treadmill on which the EMG normalisation protocol would take place. Subjects were also habituated to the CYBEX 6000 Isokinetic Dynamometer. In the second session baseline, demographic data (age, mass, stature) and injury history was recorded. Baseline measures included skinfolds, flexibility measures, vertical jump, agility tests, speed tests, strength tests and a multi-stage fitness test. Any player with a record of previous musculoskeletal injury of the lower limb was excluded from the study.

In the third and fourth sessions players executed one of either the two protocols, in which the order was randomized. Dependant variables examined included; heart rate responses (collected during competitive Rugby Union games and at set intervals during both laboratory protocols), muscle activity using electromyography, isokinetic data (peak torque, total work and average power), rating of perceived exertion (both „central" and „local") and body discomfort. These data were recorded in the same way and at the same time intervals for both the control and rugby-specific protocols. Perceived muscle

soreness was recorded for 7 days post both protocols. Testing sessions occurred during the 2009 Eastern Province Super League.

LIMITATIONS

Although every effort was made to rigorously control as many impinging variables as possible, the following factors did pose limitations to the current study and should be taken into consideration when examining the results:
This study was limited as the protocol was designed such that the physical demands of Rugby Union could be replicated in a laboratory setting. Therefore the study eliminated the physical impact of contact within a game and negated many environmental aspects such as wind, sun exposure and rain.

The study used players from all positions except the front row (numbers 1-3) and required these players to complete a protocol designed for inside back play. This may have impacted the results due to the unaccustomed demands placed upon the players.

Despite all players being part of the same team and therefore attending the same practices and completing the same fitness sessions, the training status of players could not be controlled as many players partook in their own exercise programmes. This difference in training status can be seen in the baseline test scores.

Although all subjects were instructed not to exercise or consume alcohol 24 hours prior, eat within 3 hours, or drink within an hour of both protocols and not to partake in strenuous exercise post exercise , the researcher could not control for this. On arrival all subjects were asked whether they had done any of the above mentioned activities and if they had, testing would not occur and the session would be re-scheduled.

Unfortunately due to the limited number in the Rhodes University 1st XV rugby squad (22) and the players excluded due to positions (front row) or previous injury the sample

size was limited to 18 players. The small sample size may impact the reliability of the study and applicability to other teams with different baseline data.

CHAPTER II

REVIEW OF RELATED LITERATURE

INTRODUCTION

During any aerobic activity the interplay between intensity and duration are the central influential factors determining the degree of muscular, physiological, biomechanical and perceptual stress experienced by the individual engaged in that activity. Extensive research has investigated the demands of exclusively high or low intensity activities (Boobis *et al*., 1982; Cheetham and Williams, 1987; Coggan and Coyle, 1988). In contrast, largely due to the complexity of the demands, limited studies have focussed on the impact of sports that elicit an intermittent activity profile (Reilly, 1997; Drust *et al*., 2000). The game of rugby is an intermittent, high-intensity sport characterized by rapid acceleratory and decceleratory movement (Deutsch *et al*., 2007). Because of this, many researchers in sport and exercise science have been discouraged and consequently attempts to study such sports are limited due to the lack of experimental models available (Drust *et al*., 2000). Thus the aim of this research was to gain a greater understanding of the muscle recruitment and strength changes as well as physiological and perceptual responses during simulated intermittent Rugby Union play.

RUGBY UNION

INTRODUCTION

Since the professionalisation of Rugby Union in 1995, the sport has undergone considerable changes both on, and off, the field. However, despite the rapid growth in the game itself, there has been slow growth in the scientific study of the game when compared to that of other sports (Mellalieu, 2008). More research is required to understand the requirements of the game and the characteristics of the players involved (Reilly, 1997; Nicholas, 1997; Deutsch *et al*., 2007; Duthie *et al*., 2003 and Mellalieu,

2008). Due to this, training regimes in rugby are based on an expert level rather than that of a scientific base. More research is required into the science of Rugby Union by examining the sport to meet the increased demand for knowledge regarding the requirements of the game, the risk of muscular injury and the characteristics of the players (Reilly, 1997; Nicholas, 1997; Duthie *et al*., 2003; Deutsch *et al*., 2007; Mellalieu, 2008). This will allow coaches to design training regimes on a scientific base rather than on expert base.

In rugby, the frequent bouts of physical contact make physiological data especially difficult to collect given the intrusive nature of blood sampling and the problems associated with players wearing instrumentation (Roberts *et al*., 2008). Despite this, physiological research has considered a range of factors relating to Rugby Union, including anthropometric characteristics of its players (Maud, 1983; Nicholas, 1997) and the various physical demands of the game (McLean, 1992; Deutsch *et al*., 1998). More recently, physiological research has focussed on the assessment of biochemical indices and markers of muscle damage as a result of tackle play (Takarada, 2003; Mashiko *et al*., 2004). Other studies have considered specific post-match strategies aimed at facilitating recovery and preventing overtraining (Gill *et al*., 2006).

It is important to note that a considerable amount of rugby-related injury literature exists, from both epidemiological and medical case-study sources, yet little research into understanding the physical requirements of the game is evident (Mellalieu, 2008). Despite a lack of understanding of the physical requirements of Rugby Union, performance analysis has received considerable attention within the Rugby Union specific literature. It is commonly used within the professional game as it is easy to quantify and measure post -match using video footage and therefore does not interfere with the game itself. These studies have covered aspects such as the analysis of patterns of play (Hughes and Williams, 1988), work rates and activity patterns of players using time-motion analysis (Duthie *et* al., 2005; Deutsch *et* al., 2007) and attempts to profile individual and team performances (James *et al*., 2003). These time-motion studies have been used to quantify the type, duration and frequency of discrete

movements making up the intermittent activity patterns in team sports (McLean, 1992, Deutsch *et al*., 2007, Roberts *et al*., 2008). Further, other studies have examined a range of concepts within the psychology of rugby. Topics have included motivation, burnout, overtraining stress, coping, emotions, attention, concentration and pre-performance routines (Mellalieu, 2008). Within the field of biomechanics, rugby-specific research appears to have been conducted sporadically. Limited literature exists considering aspects of running technique, which included comparisons to straight-line running; effect of different ball carrying methods on speed, agility and stepping (Sayers, 2003).

DESCRIPTION OF RUGBY UNION

Rugby Union is a self-paced activity characterized by a high-intensity, intermittent activity profile (Duthie *et al*., 2005). A rugby game consists of two forty-minute halves separated by a ten-minute half time. During an eighty-minute rugby game the ball is typically in play for thirty minutes (Duthie *et al*., 2005). The remainder of the time incorporates injury time, conversions, penalties, penalty goals or the ball is out of play (Deutsch *et al*., 2007). There are two teams on the field both with 15 players, with the exception of players being sent off for misconduct. Each player has a designated position and number outlined by the International Rugby Board (IRB). Due to the intermittent nature of the game, it requires a variety of different, intense bursts of activity as well as many static high intensity bouts and heavy contact situations (Roberts *et al*., 2008). The high intensity activities are interspersed by others of lower intensities and periods of recovery (Nicholas, 1997).

As a result of a varied, intermittent activity profile and different positional requirements, the physiological demands, are complex (Mclean *et al*., 1992). Due to the varied physical requirements of the frequent contact situations as well as being able to outpace the opposition, players are required to be fit, fast, muscular and skillful. Therefore Rugby Union is a field-based team sport eliciting a variety of physiological responses as a result of repeated high intensity sprints, static maximal exertions and contact situations

interspersed with low intensity recovery periods (Duthie *et al*., 2003). Thus when considering Rugby Union-specific fatigue it is important to account for the physiological and mechanical load (Greig and Siegler, 2009). In a comprehensive review of the demands on New Zealand rugby players, Quarrie and colleagues (1995) suggested that the physiological demands placed upon the players may also vary according to the environment, the level of play, the officiating style of the referee and the tactical course of the contest (Quarrie *et al*, 1995).

Positions in Rugby Union are outlined by the International Rugby Board (IRB): (1) loose head prop, (2) hooker, (3) tight head prop, (4) left lock, (5) right lock, (6) left flanker, (7) right flanker, (8) eighth man, (9) scrum half, (10) fly half, (11) left wing, (12) inside centre, (13) outside centre, (14) right wing and (15) full back (IRB, 2009). These positions are commonly grouped into two distinct categories referred to as the forwards, numbers1-8 (often referred to as „ball winners"), and the backs, numbers 9-15 (commonly referred to as „ball carriers"). Further distinctions are made; numbers 1-3 are the „front row", numbers 1-5 are the „'tight five" and the „'loose forwards" are numbers 6-8. Within the backs; numbers 9 and 10 are referred to as the „'half backs"; the "inside backs" are numbers 12 and 13, whilst numbers 11, 14 and 15 are referred to as the "outside backs" (Duthie *et al*., 2003; Reilly *et al*., 1990; Deutsch *et al*., 2007 and Welsh Rugby Union, 2009).

Figure 1: Rugby positions (source: www.flickr/photos.com).

In Rugby Union there are set phases of play which are used in order to restart the game. These are the scrum and the line-out, which include the forwards. The scrum is set up by numbers 1-8 and both teams compete for the ball by trying to hook the ball back with their feet whilst pushing maximally forwards against the opposition (IRB, 2009). The line out is formed by the forwards and involves throwing the ball back into the field of play whilst players jump into the air to compete for the ball (IRB, 2009). During open play a ruck or maul can be formed. These are typically used to retain possession of the ball. These phases of play are dominated by the forward players.

These four different set pieces are interspersed with open-play which comprises much more high-intensity running with the ball. Rugby is therefore an intermittent high-intensity sport, with activities that call for maximal strength and power (e.g. scrummaging and sprinting) combined with periods of lower-intensity aerobic activity and rest (Nicholas, 1997).

CHARACTERISTICS AND DEMANDS OF EACH POSITION

Insufficient data currently exists regarding the development and measurement of performance indicators in Rugby Union and, in particular, there is little research concerning position-specific performance indicators and their subsequent performance profiles (James *et al*., 2005). Positional groups have unique demands and due to this there is little homogeneity among these roles as each positional group has broad physical requirements, skills and tasks (Reilly *et al*., 1990).

The forwards

The chief responsibility of the forwards is to gain and retain possession of the ball, whether in open play or from set pieces such as the scrum and the line-out. These players are the most physically intimidating members of the team. Weight and power are important attributes, but as general athletic standards have increased in the modern

game, forwards are expected to display forms of speed and agility especially when carrying the ball (WRU, 2009). The front row (1-3) is vital to the correct functioning and movement of the scrum which is gained through correct scrummaging technique (Talk Rugby, 2009). The locks (4 and 5) are involved in jumping at the lineouts and tend to be taller individuals. They are extremely important in rucks and mauls as they need to be effective ball carriers. The loose forwards (6-8) are the only all-rounder position in Rugby Union. The loose forwards are strong and powerful as these players are required to gain, and retain, possession of the ball in loose and open play. The number 8 is a linking player between the backs and the forwards and therefore incorporates attributes of both broad positions.

The backs

The backline is expected to both create and convert point-scoring opportunities after the ball has been won by the forwards. Players are expected to be agile and dynamic, with speed being a major attribute for most positions. Kicking skills are a priority in certain positions. Just as forwards have been expected to become more agile in the modern game, so too have size and strength become important within the backline; this allows backs to contribute more effectively in defence and attack (Nicholas, 1997; WRU, 2009). The halfbacks control the possession and distribution of the ball obtained by the forwards and therefore endurance is pivotal. Correct positioning for offence and defence is paramount for these players (WRU, 2009). Outside backs (11, 14 and 15) require considerable speed to out-maneuver their opponents and provide support running, chasing down of kicks and cover defence (Nicholas 1997; Duthie *et al*., 2003).The inside backs (12 and 13), like the loose-forwards, are all-rounders. These players are involved in many contact situations as well as a larger proportion of time spent sprinting. Due to the rapid acceleratory and decceleratory movements required, these players were the focus of the current research. Inside backs typically cover 5530m during a competitive game and have been known to achieve similar sprint times to track sprinters over 15 to 35m (Deutsch *et al*., 1998; Duthie *et al*., 2003).

INJURIES DURING RUGBY UNION

Despite rugby being one of the most popular professional team sports in the world, it has one of the highest reported incidences of injury (Brooks *et al*., 2005). An epidemiological study of injuries in professional rugby quantified that the players suffered 91.41 injuries per 1000 hours of match play; this is over 1000 times higher than that for high-risk occupations (Brooks *et al*., 2005). Catastrophic injuries that occur in rugby particularly during scrummaging and contact situations, such as tackling, are obviously of serious concern (Bottini *et al*., 2000; Bathgate *et al*., 2001; Takarado, 2003). However, this research focussed specifically on inside backs who are not involved in scrummaging and who suffer mostly from muscle strains and sprains, particularly to the lower limb musculature during open play (Brooks *et al*., 2006). The importance of researching the impact of contact situations is vital and acknowledged, and the author recognises that this type of research is imperative to preventing catastrophic injuries in the modern game. However, the current study focussed on the impact of repeated eccentric contractions as a result of rapid acceleratory and decceleratory movements during open play.

Lower limb injuries account for 51.7% of all injuries reported in Rugby Union. Of all thigh injuries reported during Rugby Union, 53% were hamstring strains or tears, 37% contusions and 10% were quadricep tears (Bathgate *et al*., 2001). Although epidemiological studies indicate that hamstring and quadriceps muscle injuries comprise a large proportion of all injuries within Rugby Union, and are the most common injury site during open play, there is little or no research done in this area. Studies have quantified the occurrence of the injuries and the problem of recurrent hamstring injuries (Devlin, 2000), yet no study has explored the possible mechanism for such injuries or preventative strategies. Devlin (2000) proposes the lack of studies may be attributed to difficulties in establishing a specific pattern of rugby injuries; problems with collecting accurate injury statistics and also the meaningful interpretation of research findings in order to determine preventative measures that can be implemented practically. This shows that hamstring injuries are the most significant injury of the lower limb

musculature and therefore further research is needed into the possible mechanisms of injury.

MUSCLE INJURIES

Epidemiological studies have consistently shown that muscular strains are a primary injury type in professional Rugby Union with the majority of these strains occurring to the quadriceps and hamstring musculature (Bottini *et al*., 2000). Brooks *et al.* (2006) state that hamstring injuries alone account for 6-15% of all injuries incurred during Rugby Union. The majority of these injuries have occurred whilst the player is accelerating or decelerating (Devlin, 2000).

Many factors are associated with these types of strains such as previous history, fatigue, reduced flexibility, stiffness, strength imbalances, inherent weakness, player position and training error; some of these risk factors have been extensively studied (Jonhagen *et al*., 1994; Devlin, 2000; Small *et al*., 2008). More recently it has been suggested that poor eccentric muscular strength is a precursor to hamstring and quadriceps strains during intermittent sports that require rapid acceleration and deceleration (Greig and Siegler, 2009). An etiological risk factor which is commonly attributed to the high incidence of quadricep strains is poor eccentric muscular strength (Croisier *et al*., 2002). The temporal pattern of injury during match play indicates that fatigue might be a factor as the highest incidence of injuries has been observed in the fourth quarter of the game (Devlin, 2000). Muscle strength deficiency, due to fatigue, has been proposed to increase susceptibility to injury (Rahnama *et al*., 2003). Decreased hamstring force as a result of fatigue reduces the ability to absorb energy thereby increasing the risk of injury (Garrett, 1996). Therefore, according to Mair *et al.* (1996), injury risk may be greatest with muscle weakness during eccentric contractions, as fatigued muscles are more susceptible to stretch injury while eccentrically contracting.

However, evidence-based information on the risk factors and injury-prevention strategies are limited (Devlin, 2000). Assumptions regarding risk factors and

suggestions for preventing hamstring and quadriceps injuries in Rugby Union have therefore been based on studies undertaken in other sports. This approach may not be valid due to the different anthropometric characteristics of Rugby Union players and the specific demands of Rugby Union compared with other sports (Brooks *et al*, 2006). The fatigue associated with Rugby Union match play is specific to the characteristic activity profile. Sports involving stretch-shortening cycle activities such as: sprinting, high-intensity running, stopping, starting and quick changes in direction and kicking have been associated with high incidences of hamstring muscle strains (Brooks *et al*., 2006). Rugby Union incorporates all of the above -mentioned aspects, and this may therefore account for the high incidences of hamstring injuries. The intermittent nature of Rugby Union and the repeated eccentric demands of the modern game, places all players at risk of severe muscle damage and impaired muscle function, predisposing them to severe injuries (Friden *et al*., 1981). It is well documented that repeated eccentric muscle actions are related to a greater degree of muscle damage compared to concentric or isometric contractions (Golden and Dudley 1992). Thompson *et al.* (1999) reported that multiple-sprint sports, such as Rugby Union, have shown severe muscle damage which is associated with impaired muscle function due to the rapid acceleratory and decceleratory movements. Friden *et al*. (1981) suggest that damage to a particular muscle may predispose the muscle to more significant injury in the future. Therefore this research sought to assess the impact of rapid acceleratory and decceleratory activities and the role of repeated eccentric demands on muscle function.

CHARACTERSITICS OF ECCENTRIC MUSCLE ACTIONS

Rugby Union is stop- start in nature, with a large emphasis on the stopping action in order to stay „on-sides" and be in a position where one is able to receive the ball again. Consequently, despite all the differences in positional requirements of Rugby Union, a common activity to all positions is the repeated acceleratory and decceleratory movements (McLean, 1992; Duthie *et al*., 2005). This results in eccentric contractions being heavily involved due to the contracting muscles exerting a braking force in order to control the motion of the body (Whitehead *et al*., 1998). Repeated eccentric contractions

are associated with impaired muscle function and more importantly it has been suggested that eccentric damage to a particular muscle may predispose it to injury (Friden *et al*., 1981; Friden *et al*., 1983).

Eccentric contractions occur when a muscle develops tension as it lengthens. Human movement is rarely isolated to lengthening muscle contractions; alternatively the stretch-shortening cycle occurs during functional activities, and describes the sequence of an active muscle lengthening followed by an active shortening muscle action (Byrne *et al*., 2004). This integrated muscle activity is linked to increased performance enhancement compared to that of an isolated shortening muscle action (Komi, 1984; Komi 2000). This is achieved due to the enhanced final propulsive (concentric) action when compared with the performance of an isolated concentric action (Byrne *et* al., 2004). The mechanisms underlying performance enhancement during the stretch-shortening cycle and their relative contributions are highly debatable, but four mechanisms have been identified: (i) the time available to develop force; (ii) storage and reutilisation of elastic energy; (iii) restored potential of contractile machinery; (iv) contribution of reflexes (Byrne *et al*., 2004).

Lengthening muscle actions contribute to the stretch shortening cycle and therefore exercise-induced muscle damage is a common occurrence following such exercise, (Small *et al*., 2008). Repeated stretch-shortening cycles may lead to fatigue which has been proposed to occur in response to muscle contractile capacity failure. This failure may be attributed to muscle damage caused during the eccentric phase of the cycle (Abbiss and Laursen, 2005).

Mechanisms of fatigue in eccentric contractions have been shown to produce immediate and prolonged loss of strength caused by factors at, or distal to, the neuromuscular junction (Byrne *et al*, 2004). According to Noakes (2000) characteristics of this fatigue are a failure of contractile capacity of the exercised muscles with a reduced tolerance to muscle stretch and a delayed transfer from muscle stretch to muscle shortening during the stretch-shortening cycle. This results in increased durations of both the

decceleratory and acceleratory phases of running. The implications of this in Rugby Union are great as it reduces a player's ability to accelerate and decelerate; both integral components in the game. The ability to cope with the repeated eccentric muscle demands may require substantial eccentric muscle strength to reduce the extent of muscle damage and the ability to prevent injury.

It is theorized that when muscle fibres undergo lengthening during an eccentric contraction, actomyosin bonds undergo a mechanical detachment, rather than an ATP-dependant process (Flitney and Hirst, 1978). Due to the efficiency of eccentric contractions higher muscle strain is distributed over fewer fibres (Linnamo *et al*., 2003). Despite this, eccentric contractions are associated with improved force-generating abilities when compared to concentric or isometric contractions (Faulkner, 2003). In order to reach a required level of force generation, eccentric muscles require a reduced level of voluntary activation by the nervous system (Enoka, 1996). This suggests that eccentric muscle contractions require unique activation strategies by the nervous system. These differences in neural recruitment patterns between eccentric and concentric contractions may be related to the modulation of the relative excitability within the motorneurones innervating a muscle (Enoka, 1996). During a fatiguing squash-specific activity profile electromyography (EMG) maximal voluntary contraction (MVC) of the quadricep musculature was significantly decreased (-16%). According to Girard *et al*. (2009) this suggests that the type of exercise alters excitation-contraction coupling.

It has been hypothesized that the increased tension of eccentric contractions may disrupt the intermediate filaments that surround the Z-bridges, and stretch out the space between the pairs of intermediate filaments that surround the Z-lines of single sarcomeres, resulting in destruction and streaming of the Z-lines (Waterman-Storer, 1991). This may be heightened by the damage to the sarcolemma or sarcoplasmic reticulum, which would result in an increased intracellular calcium concentration. The increase in this concentration would activate proteolytic enzymes causing further structural protein degradation (Waterman-Storer, 1991). This is compounded by ultra-structural changes that occur in response to repeated eccentric contractions which

include sarcolemmal disruption, fragmentation of the sarcoplasmic reticulum, dilation of the transverse tubule system, disruption of myofibrilar contractile components and the cytoskeleton, changes in extracellular myofibre matrix and swollen mitochondria (St Clair Gibson *et al*., 1998; Fridén and Lieber,1992, Cleak and Eston, 1992).

Eccentric muscle actions lead to increases in passive and dynamic muscle stiffness (Enoka, 1996). These increases are generally attributed to either increased cross-bridge stiffness, or to increased tendon stiffness (Burgess, 2009). Fridén *et al*. (1983) proposed that the force per active muscle unit resulted in mechanical distribution of ultra- structural elements, such as the Z-line and contractile elements, within the muscle fibres. Coupled with this, eccentric contractions are associated with a reduction in muscle fibre recruitment resulting in a subsequent increase in loading of individual muscle fibres (Armstrong, 1990).

Eccentric contractions are associated with the overstretching of sarcomeres beyond their optimum length (Morgan and Allen, 1999). This is exacerbated by the lack of homogeneity in sarcomere length. Therefore, during active eccentric contractions the majority of the lengthening is accommodated by the weakest sarcomeres until a critical point is reached. After this the sarcomeres undergo rapid and uncontrolled alterations in length which results in no overlap between the actin and myosin filaments. On cessation of exercise, some sarcomeres return to resting length whilst others do not, which results in a loss of interdigitation of the filaments and subsequent disruption (Proske and Morgan, 2001). In addition, after repeated eccentric contractions, a series of events occur which include a loss of calcium homeostasis and an inflammatory response which results in the characteristic pathology associated with exercise-induced muscle damage (Armstrong, 1990).

The cytoskeleton of skeletal muscle is comprised of structural proteins which maintain the structural integrity of the myofibrillar lattice (Patel and Lieber, 1997). Changes that may occur as a result of exercise-induced muscle damage include primary or secondary sarcolemmal disruption, swelling, disruption of the sarcotubular system, disruption of the

contractile components of the myofibril, abnormalities of the extracellular matrix and cytoskeletal damage (Armstrong, 1990). The impact of exercise-induced muscle damage on the contractile proteins, such as actin and myosin, has been well documented. These include z-line streaming, focal disruptions of the A-band and mitochondrial disruptions (Morgan and Allen, 1999). Fridén *et al*. (1983) reported that cytoskeletal disturbances are a major contributing factor to the observed ultra- structural changes.

FUNCTIONAL CONSEQUENCES

It is well documented that skeletal muscle function, including force production, is significantly impaired during and following muscle-damaging exercise, particularly eccentric contractions (Clarkson *et al.,* 1992; Enoka, 1996; Fridén *et al*., 1992). These changes include force loss, shifts in optimal muscle length and the length-tension relationship, increase in passive tension and alterations in neuromuscular control (Byrne *et al*., 2004).

Force loss

Prolonged strength loss that occurs after eccentric loading is considered to be one of the most reliable indirect measures of exercise-induced muscle damage (Warren *et al*., 1999; Clarkson and Hubal, 2002). Therefore, it is important to determine what happens during the exercise bout in order to quantify the time course of effect. Concentric contractions result in decrements in force immediately after exercise, but do not produce damage that lasts more than a few hours (Warren *et al*., 1999). However, eccentric contractions result in force loss immediately after exercise and a recovery period that is longer (up to 24 hours post exercise) than that associated with a concentric protocol. The largest decrement in strength loss and the most prolonged recovery times are associated with high-force eccentric exercise (Clarkson and Hubal, 2002). High-force eccentric exercise (exercise consisting of maximal or near maximal eccentric contractions) can often generate up to 50-65% loss of force-generating capacity when compared to pre-exercise values (Saxon *et al*., 1995). This loss in force-generating

ability has major implications for rugby players as their ability to accelerate and decelerate will decrease and therefore players will not perform at required levels. Further, depending on the loss of force, players may be exposing themselves to injury, especially during sprinting which has been observed as the primary mechanism for injury (Devlin, 2000).

Immediate and prolonged reductions in isometric force have also been observed in the knee extensors following exercise-induced muscle damage. Byrne and Eston (2002) reported a 30% reduction in isometric strength of the knee extensors immediately post-maximal eccentric quadriceps exercise and incomplete recovery in muscle strength up to seven days post protocol. This reduction of quadricep strength may lead to injury of the quadricep musculature. Greig and Siegler (2009) found that eccentric peak hamstring torque deteriorated as a function of a soccer-specific protocol. It was found that the function of the hamstring musculature had deteriorated to the point that the fatigued soccer player may become more susceptible to both muscular strain and impaired joint stability.

Thus it is evident that activity that involves repetitive eccentric contractions of the lower limb musculature will result in immediate and prolonged reductions in muscle strength and a decreased rate of force development (Kellis and Baltzopoulos 1995; Byrne *et al*., 2004). The mechanism by which force is lost following repeated eccentric contractions has not been clearly established. Studies investigating the time course of recovery post exercise-induced muscle damage have reported a poor relationship between muscle soreness and force producing capabilities (Nosaka *et al*., 2002).

Length-tension relationship changes

Numerous studies reported a shift in optimum length following eccentric exercise (Enoka, 1996; Clarkson and Hubal, 2002; Byrne and Eston, 2002 and Nosaka *et al*., 2002). The optimum angle for torque production shifts to the right, indicating a shift in the length-tension relationship toward longer muscle lengths for maximal force

production (Whitehead *et al*., 1998). This shift appears to be a reliable and useful measure of exercise-induced muscle damage and the magnitude of shift appears to correlate to the extent of muscle damage (Jones *et al*., 1997). Increased exercise intensity and exercise volume are associated with greater shifts in optimum length (Bowers *et al*., 2004).

It has been proposed that after eccentric loading which causes muscle damage, there is an increase in series compliance due to overextended and non-contracting sarcomeres (Proske and Morgan, 2001). Therefore, a longer muscle length is required to achieve that same degree of myofilament overlap and thus force production. This predisposes players to muscle strains and tears as the muscle is lengthened often beyond its means.

Passive tension

Following a bout of eccentric exercise that causes muscle damage there is a resultant increase in passive tension (Proske and Allen, 2004). This rise in passive tension occurs immediately post an eccentric exercise bout and is accompanied by a shift in optimum length and an increase in active tension (Whitehead *et al*., 1998). This occurs as a result of sarcomere distribution and membrane damage at the level of the sarcoplasmic reticulum or the t-tubules following eccentric muscle actions (Enoka, 1996). The subsequent uninhibited release of calcium into the sarcoplasm results in activation of the contractile filaments which lead to the development of an injury contracture (Proske and Allen, 2004).

Following muscle damage, it is postulated that the contracture will be maintained whilst ATP levels remain elevated. Due to the muscle damage inflicted by eccentric exercise, further sarcomeres within the region of muscle damage will, too, shorten. These result in the added stressors being applied to the adjacent areas, subsequently extending the contracture, mounting passive tension and increasing the likelihood of injury (Proske and Allen, 2004).

Neuromuscular control

Exercise-induced muscle damage following intense repetitive stretch shortening exercise is associated with prolonged reductions in maximal force and electromyographic activity (EMG) (Avela *et al*., 1999). Nicol *et al* (1991) demonstrated changes in neuromuscular function after a marathon race which included reductions in maximal isometric knee extension torque and maximal integrated EMG (iEMG) of vastus lateralis and vastus medialis. Avela *et al*. (1999) determined the effects of a marathon on the neuromuscular function in endurance runners. There was a significant reduction in the ability to perform maximal stretch shortening exercise following the marathon. This was evident with significant reductions in average eccentric and concentric forces, take-off velocity, and EMG activity of the vastus medialis and soleus muscles. Similar studies have been done on sports which are characterised by intermittent bouts of high intensity, such as soccer, tennis and squash and have resulted in significant reductions in maximal stretch shortening cycles, concentric and eccentric forces and EMG activity of lower limb musculature (Rahnama *et*., 2003; Grieg, 2008; Girard *et* al., 2008; Grieg and Siegler 2009; Girard *et* al., 2009).

There has been little research into the effects of intermittent activity on neuromuscular control. A study of the impact of prolonged intermittent exercise on neuromuscular fatigue during tennis demonstrated that prolonged intermittent exercise (3 h) induced significant torque loss (10-13%), particularly toward the end of the activity, which was accompanied by significant reductions in normalized EMG activity (Girard *et al*., 2008). Despite this result being significant, the torque loss was moderate when compared to that of prolonged running, cycling or skiing activity. The intermittent nature in activity may be responsible for the differences in torque reduction. This assumption is supported by previous findings showing that high-intensity intermittent exercise recovery periods play an important role in limiting fatigue (Ratel *et al*., 2003).

Several mechanisms have been proposed to explain the reduction in muscle function following marathons. The reduction in neural input may be due to the occurrence of

central fatigue, supraspinal fatigue, peripheral inhibition, disfacilitation of the alpha-motorneuron pool or impairment of peripheral mechanisms (Burgess, 2009). In addition, the reduced stretch reflex sensitivity has been associated with decreased muscle stiffness. This may be attributed to the fact that the reduced muscle stiffness may be related to the reduction in muscle function, leading to impaired utilization of elastic energy (Avela, *et al.,* 1999).

The reduction in neuromuscular efficiency of the knee extensors exhibited as a decrease in the force and iEMG; has also been observed following eccentric exercise (Byrne *et al*., 2004). Further impairments in the proprioception have recently been observed as a result of exercise-induced damage (Saxon *et al*., 1995). These studies demonstrate that the force-generating capacity of muscle and motor control may be affected by a bout of eccentric exercise which causes muscle damage.

EXERCISE-ASSOCIATED MUSCLE SORENESS

Exercise-associated muscle damage or delayed onset muscle soreness (DOMS) describes the combined sensation of muscle pain, soreness and tenderness that develops after unaccustomed exercise and is particularly evident following eccentric muscle contractions or repetitive stretch shortening cycles (Cleak and Eston, 1992). DOMS is usually first evident within 8 to 24 hours after the exercise bout; peaks and typically dissipates within 7 to 10 days. It is the most commonly used indicator of altered muscular function in human studies (Byrne *et al*., 2004).

The underlying mechanisms responsible for the pain associated with DOMS are not well understood although the degree of discomfort and muscle disturbance depends largely on the intensity and duration of effort and type of exercise performed (McArdle *et* al., 2001; Cheung *et al.*, 2003). It has been suggested that the soreness may result from swelling and pressure in the muscle (Smith, 1991). This swelling and pressure has been attributed to muscle and connective tissue damage, and may be related to the inflammatory response that can be induced by eccentric muscle actions (Armstrong,

1984; McIntyre *et al*., 1995). In a study conducted by Proske and Morgan (2001), swelling peaked 24 hours post exercise, which coincided with maximal pain perception and the swelling began to subside by 4 days post exercise, when DOMS began to dissipate.

Symptoms associated with DOMS include reduced range of motion and force production, increases in limb volume, swelling and stiffness and leakage of myofibrillar proteins into the blood (Smith, 1991; Clarkson *et al*., 1992; MacIntyre *et al*., 1995; Chelbourn *et al*., 1998). However, despite the symptoms of DOMS being induced by eccentric muscle contractions or repetitive stretch-shortening cycle exercises, DOMS shares a poor temporal relationship with histological evidence of muscle damage and therefore is not a direct marker of muscle damage (MacIntyre *et al*., 1995, Byrne *et al*., 2004). Nonetheless, DOMS is still frequently being used as an indicator of muscle damage.

A number of theories have been proposed to explain the pain stimulus associated with DOMS including: lactic acid, muscle spasm, connective tissue damage, muscle damage, inflammation, enzyme efflux theories and other proposed models (Cheung *et al*., 2003). It has been suggested that the initial events following exercise-induced muscle damage may be related to mechanical or metabolic mechanisms. The mechanical theory includes adaptations at the levels of the whole muscle and muscle fibre, as well as at the myofibrillar level, specifically in the cytoskeleton. The metabolic theory involves alterations in calcium concentrations, muscle temperature and pH, insufficient mitochondrial respiration and oxygen free radical production (Kendall and Eston, 2002).

Exercise -induced muscle damage occurs frequently in athletes. Of greatest concern to the athlete is the loss of muscle function that accompanies muscle damage and results in under-performance and the inability to perform at required levels (Byrne *et al*., 2004). Thompson *et al.* (1998) examined the impact of prolonged high-intensity intermittent shuttle-running on muscle soreness and markers of muscle damage and concluded that muscle soreness was the greatest in the hamstring musculature and persisted for the

longest period. In a sport such as Rugby Union, which is characterised by repeated acceleratory and decceleratory movements and therefore repeated eccentric demands (McLean, 1992; Duthie *et al*., 2005), the likelihood of loss of function and predisposition to injury is high and therefore more research is needed to assess the impact of these movements in order to offer realistic preventative strategies.

PROPOSED MECHANISMS FOR ALTERED MUSCLE FUNCTION AFTER ECCENTRIC MUSCLE CONTRACTIONS

The reduction in force and tension producing capabilities following eccentric exercise may be associated with alterations in peripheral mechanisms such as disorganization of the contractile machinery and calcium regulation, excitation-contraction coupling failure, selective muscle fibre damage and the redistribution of sarcomere lengths (Morgan and Allen, 1999; Proske and Morgan, 2001). Nevertheless, the role of central fatigue and alterations in neuromuscular control should also be considered in the loss of muscle function following exercise-induced muscle damage (Morgan and Allen, 1999).

Central changes

The structural changes associated with eccentric exercise may be accompanied by alterations in neuromuscular performance (Sargeant and Dolan, 1986). This has been demonstrated in the knee extensors where there was a reduction in neuromuscular efficiency following eccentric exercise (Byrne *et al*., 2004). This reduction in neuromuscular activity is shown as a decrease in the force: iEMG activity, both during and after eccentric activity. Decreased force: iEMG ratio indicates that increased central activation is required for the generation of maximal and submaximal forces. In order to compensate for the changes in contractile function, alterations in the firing patterns of the damaged muscles, including the recruitment of additional motor units and increased firing frequencies may occur (Ebbeling and Clarkson, 1989).

Literature provides conflicting evidence for the changes in neuromuscular efficiency following exercise-induced muscle damage. Newman *et al*. (1983) demonstrated increased neural activation after a damaging activity bout, while other studies have shown that neural activation remained unchanged or was impaired after exercise (Nosaka *et al*., 2002). Further investigation is needed to determine the role of central fatigue following eccentric exercise that causes muscle damage.

REPEAT BOUT EFFECT

It is well established that a single bout of unaccustomed, predominantly eccentric exercise results in skeletal muscle damage (Morgan and Allen, 1999). However, a repeat bout of the same or similar eccentric exercise results in significantly reduced symptoms of muscle damage, such as muscle soreness, swelling, strength, range of movement and levels of muscle proteins circulating than the initial bout of exercise (Clarkson *et al*, 1992). This protective adaptation to a single bout of eccentric exercise is referred to as the repeat bout effect. This effect has been shown to last between several weeks and six months (Nosaka *et al*., 2001). This effect appears to be specific to the exercised muscle groups as there is currently no evidence of protective adaptations in contralateral muscle groups that have not been subjected to an initial bout of eccentric exercise (Clarkson *et al*., 1992).

Unfortunately there is little consensus in the literature regarding the mechanisms of the repeat bout effect. Several theories have been proposed to explain this phenomenon, but the underlying mechanisms remain unclear. The proposed theories can be categorised as neural, mechanical and cellular mechanisms (Nosaka *et al*., 2001). It is evident that one theory cannot explain the various observations of the repeat bout effect. It is therefore possible that the repeat bout effect may occur through the interaction of the neural, mechanical and cellular mechanisms (Burgess, 2009). The repeat bout effect is an important factor when considering the adaptations to intermittent activity profiles which are dominated by repeated eccentric demands.

OTHER FACTORS PREDISPOSING RUGBY PLAYERS TO HAMSTRING INJURIES

During running, the hamstring muscles become active in the last third of the swing phase undergoing eccentric contraction to decelerate knee extension and oppose the activity of the quadriceps musculature. When ground contact occurs, the hamstring muscles change from a maximal eccentric to concentric activity and develop the greatest force of any lower extremity muscles (Mann *et al.*, 1986). Thus the hamstrings are subjected to high forces making them vulnerable to injury (Bennell *et al*., 1998).

ANATOMY OF THE MUSCLE

The bicep femoris may be the most likely hamstring muscle to sustain injury due to the dual innervation of the muscle (Agre, 1985). Despite this, other biarticular muscle groups, such as the gastrocnemius and quadriceps do not exhibit incidences of injury as high as that for the hamstring group. This finding suggests that the forces generated and absorbed by the hamstring muscles are significantly higher than those in other muscle groups (Brooks *et al*., 2005). Alternatively it may be attributed to the fact that other predisposing risk factors are influential in causing muscle injuries. The dual innervation has been implicated as a cause of muscle strains because of poor coordination of the contraction of the two heads, especially with the onset of fatigue (Devlin, 2000). This allows separate parts of the muscle to be activated at separate times, thereby increasing the likelihood of injury.

PREVIOUS INJURY

Recurrent hamstring injuries are a common problem in Rugby Union, with 34% of hamstring injuries recurring (Devlin, 2000). In a study by Orchard (1998) previous injury to the hamstring musculature was the most significant risk factor for further hamstring injuries. This researcher also showed that history of a recent hamstring injury predisposed the player to a subsequent quadricep or hamstring injury. There is an assumption that the aetiological factors that contribute to an initial acute injury are the same as those involved in recurrent injury. However, there is little research to support

such an assumption (Devlin, 2000). One of the problems with recurrent hamstring strains is that there is no consensus for rehabilitation (Croisier *et al*., 2002, Petersen and Hölmich, 2005).

REDUCED FLEXIBILITY

The aetiological factors commonly listed for hamstring injuries include poor flexibility as a risk factor (Devlin, 2000; Croisier *et al*., 2002; Dadebo *et al*., 2003; Witvrouw *et al*., 2003; Petersen and Hölmich, 2005). A lack of flexibility may cause the musculotendinous unit to stretch beyond its ability to elongate in the latter part of the swing phase and may result in tearing, especially during sprinting (Agre, 1985). Witvrouw *et al*. (2003) reported that uninjured players had significantly more flexible hamstrings. They also found a significant association between pre-season hamstring tightness and the subsequent development of injury in football players. These findings are consistent with the suggestion of many experts in sports medicine who believe that muscle flexibility plays a role in the development of injuries (Garrett, 1996; Petersen and Hölmich, 2005). However, a study on provincial Rugby Union players found that there was no correlation between reduced flexibility and the incidence of hamstring injury (Naylor, 2007).

HAMSTRING TO QUADRICEPS RATIO

The hamstring:quadricep ratio (H:Q) is a functional ratio used to examine the similarity between hamstring and quadriceps moment-velocity patterns to assess knee functionality and muscle balance (Rosene *et* al., 2001; Petersen and Hölmich, 2005). The ratio has conventionally been expressed as concentric hamstring to quadricep strength and recently of eccentric hamstring strength to concentric quadricep strength as this has been suggested to be a better descriptor of function (Aagaard *et al*, 1998). The conventional H:Q ratio implies that concentric or eccentric contractions take place for the knee extensors and flexors simultaneously. However, in practice, knee joint movement only allows for eccentric hamstring contraction to be combined with concentric quadricep

contraction during extension and vice versa during flexion (Aagaard *et al*, 1998). Therefore, the conventional H:Q ratio has been suggested to merely indicate whether qualitative similarity exists between the moment-velocity patterns of the hamstrings and quadriceps (Rosene *et al*., 2001).

Given the function of the hamstrings during sprinting, it is feasible that reduced strength, inequality of strength between the left and right hamstring or strength imbalances between the hamstring and quadriceps musculature may predispose an individual to hamstring injuries (Bennell *et al*., 1998). It has been suggested that the H:Q ratio may reflect predisposition to injury (Rosene *et* al., 2001). This predisposition may result from decreased antagonistic hamstring coactivation during extension loads. The relationship between muscle imbalance and injury has always been a logical assumption, but one that is poorly illustrated by scientific arguments (Croisier *et al*., 2002). Liemohn (1978) found no differences in isometric hamstring/quadriceps ratios between hamstring injured and non-injured groups of track and field athletes. Subsequent studies have also reported no significant differences in the ratio of injured and non-injured players (Bennell *et al*., 2003). Rosene *et al*. (2001) found that athletes who did not regularly exercise their hamstrings had a significant decrease in hamstring activation compared to athletes who did regularly exercise their hamstrings during knee flexion and extension. It has been suggested that highly developed quadriceps musculature contributes to decreased antagonistic hamstring coactivation, thereby increasing the susceptibility to hamstring strains (Rosene *et al*., 2001).

Although it is difficult to generalize, the normal H:Q ratio is considered to be 50% to 80% as averaged through the full range of motion, with a higher ratio at increased speeds (Aagaard *et al*, 1998). As the ratio approaches 100%, the hamstrings have increased functional capacity for providing stability and increased strength (Rosene *et al*., 2001), thereby decreasing the likelihood of injury.

FATIGUE

Muscle fatigue has been described as a time-dependant process that encompasses numerous central, peripheral and psychological factors that affect force production (Pincivero *et al*., 2000). With a higher incidence of injuries being observed during the fourth quarter of Rugby Union, it seems that fatigue plays a large role in the occurrence of hamstring injuries (Wekesa *et al*., 1996; Devlin. 2000; Petersen and Hölmich, 2005; Holtzhausen *et al*., 2006). This impact of fatigue may therefore be partially addressed by changes in coaching and training. Alternatively, this may be attributed to changes within the physiology of fatigued muscle (Byrne *et al*., 2004). Findings suggest that immediate and prolonged loss of strength after repeated eccentric exercise is caused by factors at, or distal to, the neuromuscular junction (Byrne *et* al., 2004). It has been suggested that fatigue results in a decrease in iEMG activity (Girard *et al*., 2008; Girard *et al*., 2009). However, other research has found iEMG activity to increase as a product of fatigue (Mizrahi *et al*., 1997; Psek and Cafereli, 1993; Yeung *et al.*, 1999). Pinniger *et al*. (2000) observed a decrease in EMG activity due to altered kinematics especially during sprinting following a repeated dynamic hamstring fatiguing task. Therefore the impact of fatigue on the neural command is not well understood.

Due to these changes in fatigued muscle it has been proposed that the dual innervations of the bicep femoris are a factor in hamstring injury due to the poor coordination, during a fatigued state, allowing the separate parts of the hamstring muscle group to activate at different times (Brooks *et al*., 2005). This results in a decrease in strength of the hamstrings and a resultant decrease in the H:Q ratio, placing the hamstrings at risk of injury (Devlin, 2000). Despite these factors mentioned above, it has been shown that the most likely causes for hamstring strains are possibly quite complex, interactive and multi-factorial involving flexibility, strength, warm up and fatigue (Dadebo *et al*., 2003; Petersen and Hölmich, 2005).

These mechanisms and strength decrements may be of more importance than studies quantifying physiological fatigue, as there is limited applicability of physiological fatigue models in Rugby Union. The energy supply-energy depletion model of exercise is specific for exercise lasting more than 2-3 hours (Noakes, 2000). As rugby is an 80-

minute game, this model does not affect the performance of rugby players. Rugby Union is characterized by repeated high-intensity efforts, alternating with short, less than 20s, periods of recovery (Duthie *et al.,* 2005). Therefore rugby players are protected from the risk of developing hypoglycemia by the short duration of the game (80-minutes) and the 10-minute half time. Also, players are frequently provided with fluid and fuel during the match to counteract any possible fluid imbalances or glycaemic effects. As no rugby-playing nation is situated at high altitudes due to the scarcity of large flat playing surfaces at altitude, players are protected from hypoxia. As rugby is a winter sport, players are unlikely to be at risk of developing elevated body temperature; in fact players are often required to exercise in extremely cold conditions.

INJURY PREVENTION

In a study by Mjølsnes *et al.* (2004), two hamstring strength training programmes were compared. One programme included traditional hamstring curls (concentric training) whereas the other included Nordic hamstring curls (focussed on the eccentric phase). Results showed that it was possible to significantly increase the eccentric torque in well-trained soccer players during a 10- week programme focussing on eccentric strength. No significant increase was seen in the concentric group. Therefore there is a benefit to eccentric training and this may be advantageous in the reduction of hamstring injuries. Unfortunately the study did not focus on injuries acquired after the training programme.
The inclusion of correct dynamic movement weight training may decrease the incidence of injury as this training has been shown to show significant benefits in reduced incidence of injury (Brooks *et al*., 2005). The same study showed that using non-weight-bearing endurance training activities such as rowing and cycling, rather than running, decreased the incidence of lower limb overuse injuries.

Long seasons, and lack of a proper end- of -season break for many players, may be partly responsible for the high occurrence of overuse injuries (Holtzhausen *et al*., 2006). Adequate rest and recovery are vital components in ensuring peak physical conditioning (Bottini *et* al., 2000). Pre-season training programmes may also have had an influence,

as most of the chronic overuse injuries are recorded in the first third of the season (Holtzhausen *et al*., 2006). Therefore, it is important that training programmes are carefully planned, examining the work rates, recovery and training demands on the players so as not to elicit overuse injuries. Therefore, the patterns and prevalence of recurrent and chronic overuse injuries in professional rugby must be studied further in order to make appropriate recommendations (Brooks *et al*., 2006).

CONCLUSION

In conclusion, it is evident that repeated eccentric demands are highly prevalent in intermittent sports, such as Rugby Union, the focus of the current study. Unfortunately there is a lack of understanding and research in this area. Sports that require rapid acceleratory and decceleratory movements have shown an increased incidence of musculoskeletal injuries of the lower limbs, specifically the hamstring musculature. Eccentric contractions have been associated with impaired force production during and following damaging exercise (Clarkson *et al*., 1992; Enkoa, 1996; Fridén *et al*., 1992). Therefore the current research aimed at quantifying these functional changes over time during a simulated rugby-specific intermittent laboratory protocol.

CHAPTER III

METHODS

INTRODUCTION

Physical activity of variant intensity and duration, places the body under cardiovascular and musculoskeletal strain (Coggan and Coyle, 1988). A vast amount of research has focussed on the demands placed on the body during steady-state activities, specifically those of an endurance-type nature, such as long distance running (Boobis *et al*., 1983; Cheetham and Williams, 1987; Coggan and Coyle, 1988). Little research has investigated the effects of intermittent activities such as soccer, Rugby Union, hockey, tennis and batting during cricket (Drust *et al*., 2000). This is largely due to the difficulties in obtaining reliable and accurate *in situ* data, as collecting procedures tend to interfere with intermittent sports during match play (Reilly, 1997). In an attempt to establish the demands placed on the body during field games of a contact nature, researchers have devised similar laboratory-based protocols to establish the cardiovascular, musculoskeletal and perceptual responses to these types of activities (Drust *et al*., 2000). Most of the previous studies in Rugby Union have focussed on investigating the time-motion data as well as heart rate responses. There are few data that have looked at the patterns of muscle fatigue and changes in muscular strength, especially eccentric strength. It is well known that Rugby Union is characterized by stop-start activities placing considerable eccentric demands on the lower limb musculature. The high incidence of rapid acceleration and deceleration in Rugby Union involves rapid stretch-shortening cycles which increase the likelihood of injury (Brooks *et al*., 2006). Therefore, the main purpose of this study was to understand the demands placed on the lower limb musculature (muscle activity and strength changes) as well as the physiological and perceptual responses during a Rugby Union- simulated protocol in order better to understand the mechanism of fatigue and therefore, likely injury.

PILOT TESTING

Prior to testing, extensive pilot studies were conducted both in the field and the laboratory. The first pilot test involved the assessment of the physiological demands during competitive rugby and evaluated the suitability of the proposed equipment to be used. Heart rate monitors needing a watch to record the data were unsuitable as the receiver worn on the wrist hindered play. During the contact situations the buttons on the reviever were pressed and data recording was stopped. Therefore it was deemed necessary to use a system that did not require a watch to be worn, such as the Suuntu® t6 Memory Belt, as the data can be stored on the belt itself. Furthermore, additional pilot tests were conducted to explore different protocols of an intermittent and continuous nature in order to establish the intensity and stop-start nature of the rugby specific protocol that was devised for the experiment proper.

ETHICAL CONSIDERATIONS

INFORMED CONSENT

All subjects were informed both verbally and in writing of the nature of the study (see appendix A) which was approved by the ethics committee of the Human Kinetic and Ergonomics Department of Rhodes University, Grahamstown, South Africa. Voluntary, written consent was given by all players without any pressure from captains, coaches or other team members (appendix A).

PRIVACY AND ANONYMITY OF RESULTS

A coding system was used to ensure that any information obtained from the players could not be traced to them. The name on the data collection sheets (appendix B) was used only for record purposes, and the players were informed that data would be held

on file for statistical analyses only and be deleted after completion of the study with only one copy being stored in the department.

SUBJECT CHARACTERISTICS

Eighteen male university first team rugby players were recruited for the present study. All players represented Rhodes University in the Eastern Province, South Africa, Super League. The sample group included all positions of a rugby team, excluding the front row, positions 1-3. All subjects were between the ages of 19 and 26 years (Table I).

Table I: Basic demographic data.

Measure	Mean	SD	CV
Age (years)	20.91	1.69	8.10
Stature (mm)	1811	72	4
Body mass (kg)	87.2	10.9	12.5
BMI (kg.m^2)	26.63	3.36	12.62
Sum of skinfolds (mm)	66.20	27.78	42.01
Years of rugby experience	10.32	3.75	36.30

SD = standard deviation; CV = Coefficient of variation (%)

EXPERIMENTAL DESIGN

Heart rates recorded on the current cohort during match play as well as time-motion data measured during the 2001 and 2002 *Super 12* Rugby Union competition was used to inform the design of the rugby specific intermittent laboratory protocol (Duthie *et al.,* 2005). The same data was used by Roberts *et al.* (2008) to design a Rugby Union specific protocol to assess rugby-specific garments. The aim was to match as closely as possible, the demands of competition play. Duthie *et al.* (2005) analysed 16 games during the 2001 and 2002 season using three Australian provincial teams during the *Super 12*. The video recordings were analysed using an in-house game analysis system (Part-timer V1.1, Australian Sports Commission, Canberra) that identified each

movement (Table I). Movements included standing still, walking, jogging, striding, sprinting, static exertion, jumping, lifting and tackling. This analysis combined both absolute measurements (frequency, time(s), relative (%) spent in various activities) with individual work-to-rest ratio data. These codes are further explained by the operational definitions (Table II).

Table II: Categories of time-motion data.

"Low Intensity"	
Standing still	Standing or lying on the ground without being involved in pushing or any other game activities. This can include small movements which are not purposeful (e.g. stumbling back and forth, turning sideways, etc.).
Walking	Walking forwards or backwards slowly with purpose. One foot is in contact with the ground at all times (e.g. walking to a scrum following a breakdown).
Jogging	Running forwards slowly to change field position, but with no particular haste or arm drive (e.g. Jogging down-field to a line-out).
"High Intensity"	
Striding	Running with manifest purpose and effort, accelerating with long strides, yet not at maximal effort (3/4 pace) (e.g. running into a back line to receive the ball).
Sprinting	Running with maximal effort. This is discernible from cruising by the inclusion of head and arm movements.
Static exertion	This involved rucks, mauls and scrums.
Jumping	Jumping in a lineout or to catch a ball in play.
Lifting	Actively lifting another player into the air.
Tackling	The act of grabbing a player who has the ball and bringing him to the ground.

Shaded areas refer to "high intensity" activities.

Striding, sprinting, static exertion, jumping, lifting and tackling were regarded as high intensity work (shaded in Table II) whilst standing, walking and jogging were regarded as rest activities (Duthie *et al.,* 2005). The duration of each interval of high intensity work was divided by the duration of the following rest interval to give a work-to-rest ratio for that passage of play (Table III).

Table III: Percentage (%) of total time spent at work or rest.

	Forwards		Backs	
	Front row	Back row	Inside backs	Outside backs
Work	13	15	6	4
Rest	87	85	94	96

It is well known that backline players are more prone to lower limb injuries largely due to their high volume of sprinting which requires rapid acceleration and deceleration (Devlin, 2000; Bottini *et al*., 2000). According to Duthie *et al*. (2005) the position involved in the most sprint activities was the inside backs, who spend approximately 84(±30)s of a game sprinting with 29 sprints completed, more than any other position (Table IV).

Table IV: Maximum values relating to sprints recorded in various positions, over the matches investigated (adapted from Duthie *et al*., 2005).

Activity	**Front row**	**Back row**	**Inside backs**	**Outside backs**
Frequency of sprinting (n/game)	8	14	29	24
Total time sprinting (s)	19	34	84	71
Average sprint duration (s)	2	2.4	3	2.9
Percentage of time spent sprinting (%)	0.4	0.7	1.6	1.4

Shaded area refers to the players with the highest sprint volumes

However, although it is evident that all backline players sprint more and for longer than forwards (Table IV), the inside backs have the highest sprint volume. Based on this observation it was decided to use their specific demands to inform the current rugby-specific protocol.

Shown, in Table V, are the activity profiles of inside backs as described by Duthie *et al*. (2005). Although sprinting was highlighted, it is evident that inside backs are involved in numerous activities. Most notably they spend the majority of their time standing and the least amount of time sprinting. Despite this, injury data still suggest that the rapid acceleration and deceleration is responsible for the high injury occurrence (Brooks *et al*., 2005). Therefore the aim of this study was not only to focus on the sprinting aspect of the game, but to design a rugby-specific protocol which would mimic an actual 80 - minute game but without any contact situations (Tables V and VI). This rugby-specific protocol would be replicated as a floor- based laboratory protocol.

Table V: Activity profile of Inside backs (adapted from Duthie *et al.,* 2005).

Activity	**Total time (min:s)**	**Percentage of total time (%)**	**No. of movements per game**	**Average duration (s)**
Standing	38:17	43	200	11.9
Walking	30:04	34	195	9.9
Jogging	14:50	17	197	4.6
Striding	2:10	2.5	67	2
Sprinting	1:24	1.6	29	3
Static exertion	1:39	1.8	27	3.8
Work	5:33	6	85	4
Rest	83:07	94	86	61.5

The heart rate data collected on the current cohort during actual match play over five matches was categorised into different heart rate zones. This was a secondary control measure to ensure that the simulated experimental protocol was representative of match play responses of the current cohort. Maximal and average heart rates were recorded and used in the design of the Rugby Union-specific laboratory protocol. A comparison of the heart rate data of Deutsch *et al*. (1998) and the current study are shown in Table VI.

Table VI: Mean percent time spent in four different heart rate zones for inside backs.

Heart rate zone	Relative time (%)	
	Deutsch *et al*., 1998	**Current Cohort**
Low (<75%)	12.50	15.35
Moderate (75-84%)	35.50	23.15
High (85-95%)	40.50	47.30
Maximal (>95%)	11.50	14.20

Shaded area shows heart rates between 75-84%

The heart rate responses showed that players spent the highest percentage of time, 47.30%, in a high heart rate zone (Table V). Despite players only spending 6% of the game involved in high intensity activities and the other 94% engaged in low intensity activities or at rest, the heart rate data (Table VI) shows that players exert themselves physically during a competitive rugby game. This indicates that even with 94% of the time spent completing low intensity activities, players are not able to recover fully during those periods and therefore spend 76% of the competitive game in moderate to high intensity heart rate zones.

EXPERIMENTAL CONDITIONS

Experimental Protocol

To facilitate ease of data collection the 80 minute activity profile was divided into four twenty- minute cycles of identical demands. This cycle was then repeated four times.

Table VII: The data set upon which the rugby-specific intermittent laboratory protocol was based.

Activity	Total time (min:s)	Percentage of total time per set (%)	No. of movements per set	Average duration (s)
Standing	8:36	43	50	11.9
Walking (5.4km.hr^{-1})	6:48	34	48.75	9.9
Jogging (11.5km.hr^{-1})	3:24	17	49.25	4.6
Striding (18km.hr^{-1})	0:30	2.5	16.75	2
Sprinting (24km.hr^{-1})	0:19	1.6	7.25	3
Static exertion	0:21	1.8	6.75	3.8
Work	1:33	6	21.25	4
Rest	20.77	94	21.5	61.5

The data set in Table VI was arbitrarily distributed to provide a 20- minute activity profile replicating the activity of a competitive rugby match (Figure 1 and appendix B). The exact protocol can be found in Appendix B. During this condition, players were required to follow instruction by the researcher to stand, walks, jog, stride and sprint at set intervals. This was done on a wooden walkway which was 22m in length. Players therefore had to change direction repeatedly during the protocol depending on their cycle of activity. Players were clearly instructed regarding the activities prior the start of the protocol and were motivated during the „all-out" sprint blocks.

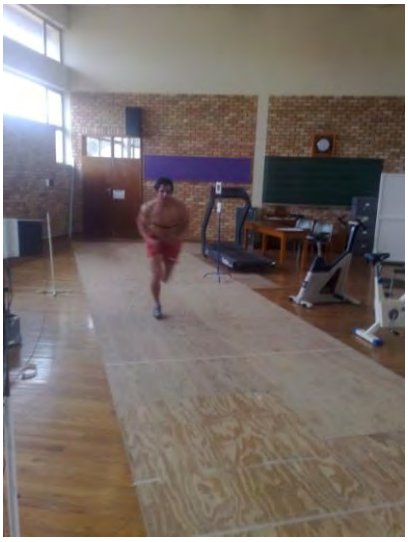


Figure 2: Player during an all out sprint phase running on the wooden walkway

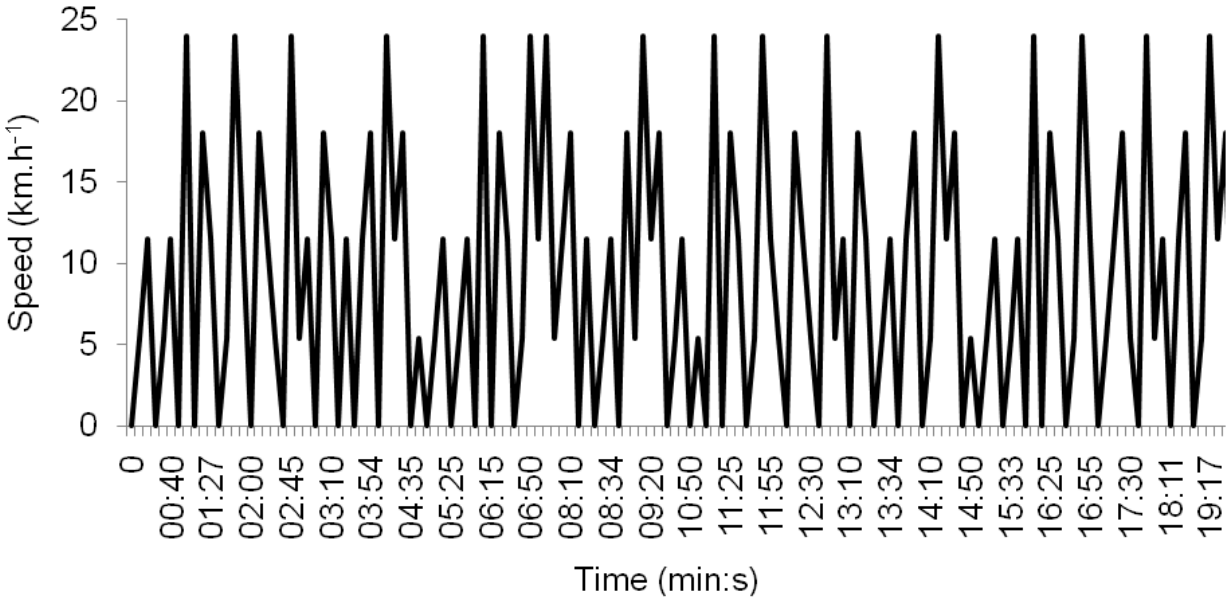


Figure 3: The Rugby Union-specific intermittent laboratory protocol.

Control Protocol

In order to replicate the distance travelled throughout a competitive Rugby Union game, but to negate the impact of rapid acceleration and deceleration, players were required to participate in a control condition. This involved walking at a continuous speed for the

duration of a rugby match on a motorized treadmill. According to Deutsch *et al*. (1998) and Duthie *et al*. (2003) inside backs covered, on average, a distance of 5530m during competitive rugby. This distance was used to calculate the constant speed the of the control protocol (s=d/t). Therefore, subjects were required to walk at a constant speed of 4.2km.h^{-1} for a full 80 minutes.

INSTRUMENTATION AND TREATMENT

Baseline testing, prior to the experiment proper, included measures of body mass, stature, body composition and various fitness measures. Measures during the experimental and control conditions included measures of changes in muscle activity, isokinetic strength and perceptual responses.

ANTHROPOMETRIC PARAMETERS

Body Mass

Body mass of each individual player was measured to the nearest 0.01kg using a calibrated electronic Toledo® Scale (model 8142). All subjects were required to remove their shoes, accessories and heavy clothing. Each subject was then required to stand still in the middle of the scale in an upright position.

Stature

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

Skinfold measures

Body fat percentage was measured using Harpenden Skinfold calipers (Quinton Instrument, Seattle, Washington, USA). Seven skinfold sites were recorded, namely: chest, triceps, subscapular, suprailliac, abdominal, thigh and medial calf. All measures were taken on the right hand side of the body with the skinfold caliper placed 10mm away from the thumb and finger, perpendicular to the skinfold, and halfway between the crest and the base of the anatomical site. Duplicate measures were taken and a retest was done if the duplicate measures were not within a 3% error margin. Body density was calculated using an equation devised by Jackson *et al*. (1978) as follows:

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

This figure was then used the Siri equation to work our body fat percentage.

PHYSICAL PARAMETERS

MUSCLE ACTIVITY

The muscles assessed in order to determine the quadriceps activation were vastus medialis (VM) and vastus lateralis (VL) whilst the biceps femoris (BF) and semitendinosus (ST) were used to determine hamstring activation. These muscles were chosen due to their proximity to the surface as muscle activity was assessed via the use of surface electrodes. Due to the activation pathways of the quadriceps and hamstrings it is possible to infer hamstring and quadriceps activity from the measurement of these four muscles (Pincivero *et al*., 2000).

A surface electromyography (EMG) device (Muscle Tester Mega ME6000P16, Mega Electronics Ltd, Finland) was used in the current research. Muscular activity was measured by attaching EMG electrodes to the surface of the skin and recording the

changes in electrical activity in the muscle directly below them. In order to assess these changes two disposable, pre-filled Silver-Silver Chloride electrodes (Kendall, Meditrace 200) were attached to each of the above mentioned muscles. Electrodes were placed at a fixed inter-electrode distance of 20mm. Prior to electrode placement, the skin areas were shaved and cleaned with alcohol swabs and allowed to dry in order to reduce electrical impedance. Electrode placement for the VM muscle was 20% of the distance from the medial joint line from the knee to the anterior superior iliac spine. Placement for the VL was the midpoint between the head of the greater trochanter and the lateral femoral epicondyle. Electrode placement for the BF and ST was 50% of the distance from the ischial tuberosity to the medial and lateral femoral epicondyles, respectively (Pincivero *et al*., 2000). A third „neutral" electrode was attached at least 100mm away from the active muscle and placed on an inactive muscle. The Megawin© Software was used in conjunction with the electromyography equipment and a wireless transmitter was connected to a laptop computer, linking the two pieces of equipment.

Electromyographic raw data activity was sampled at 1000Hz, and was amplified and recorded telemetrically (Muscle Tester "Mega ME6000P16", Mega Electronics Ltd, Finland). The raw EMG 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 for EMG (µV) vs. time (s), resulting in an integrated EMG (iEMG) measurement. The iEMG value of the half time and post-protocol EMG protocol were normalised to the iEMG value obtained from the pre-protocol EMG protocol. During running, the normalisation to the EMG obtained during a standardised submaximal run has been shown to be the most appropriate method of normalisation of iEMG (Burgess, 2009).

Muscle activity during both protocols was normalised using a dynamic baseline measure of the relevant muscle groups and changes in muscle activity were measured during a 3-minute EMG protocol designed by Rahnama *et al*. (2006). This EMG protocol was designed to assess lower limb muscular fatigue after a soccer-specific intermittent

exercise protocol, and in pilot investigations for this study, was validated for Rugby Union and in particular the protocol used in this study.

Table VIII: The EMG measurement protocol as per Rahnama *et al*. (2006).

Time (min:s)	Speed (km.h^{-1})	Duration (min:s)
00:00	5	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	00:20
03:00	STOP	

The EMG normalization protocol required players to walk at a speed of 6 km.h^{-1}, jog at a speed of 5 km.h^{-1}, 6 km.h^{-1}, 12 km.h^{-1}, stride at 15 km.h^{-1} and sprint at a speed of 21 km.h^{-1} for 15 seconds. 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 protocol was completed on a treadmill as it allowed for the speed to be controlled. Further, it was repeated three times during both protocols (pre-, half time and post-protocol).

ISOKINETIC TESTING

Isokinetic testing involves the assessment of maximal muscle tension throughout a range of joint motion set at a constant angular velocity (Franklin *et al*., 2000). Baseline measures of isokinetic data were collected using a CYBEX 6000 Isokinetic Dynamometer. The dynamometer was set up to suit each subject, following the manufacturer"s guidelines, with the setup maintained throughout the exercise protocol (CYBEX, Division of Lumex, Inc., Rononcoma, NY 11779). The crank axis was aligned

with the axis of rotation of the knee joint, and the cuff of the dynamometer"s lever arm secured around the ankle, proximal to the malleoli. Restraints were applied across the chest and across the test thigh, proximal to the knee joint, so as not to restrict any motion. Knee extension is produced by the contraction of the quadriceps femoris muscle (which consists of the rectus femoris and vasti; medialis, intermedius and lateralis). Knee flexion is the result of the contraction of the hamstring muscle group (consisting of the biceps femoris, semitendinosus and semimembranosus). In general the hamstring muscle group has been shown to produce about 60% of the torque developed by the quadriceps muscle group at slow isokinetic test velocities (Morris *et al*., 1983; Perrin *et al*., 1993 and Li *et al*., 1996). The range of motion assessed in the knee flexion/extension range was 90 degrees. In the case of knee extension and flexion testing, correction for gravity was an important consideration as the weight of the leg being tested has an influence on the quadriceps to hamstring (Q/H) ratio.

Determination of the dominant lower-extremity was based on kicking preference. Before testing commenced the subjects were familiarized with the test procedures on the isokinetic dynamometer and completed a number of trials at each of the testing speeds as part of the warm-up with the dynamometer in the manual mode. Two isokinetic protocols quantifying concentric knee extensor and knee flexor strength and the concentric and eccentric knee flexor strength were administered. The delivery of these tests was randomized. Both protocols were completed at a constant speed of 60 $deg.s^{-1}$. After the player had completed a warm up and stretch, familiarization trials on the dynamometer in each mode of exercise were performed. Testing included three repetitions, performed on each leg at 60 $deg.s^{-1}$ with a 30s rest period between each set. Participants were instructed that each repetition should be a maximal contraction throughout the entire range of motion. No visual feedback was provided with regard to performance, but encouragement from the researcher was permitted. This testing was completed prior to testing, at half time and at fulltime during the experimental and continuous protocols.

STRENGTH

One repetition maximum refers to the heaviest weight that can be lifted only once using good form and is the „gold standard" of dynamic strength testing (Franklin *et al*., 2000). Due to the safety concerns with performing one repetition maximum, two assistants were present at all times to ensure the safety of the subject. If the weight was too heavy the assistants would help remove the weight thereby removing the risk of the weight falling on the individual and decreasing the risk of muscular strains.

One repetition maximum was assessed using a bench press. This measure of strength was used as a baseline measurement for comparison against previously established strength norms. The players were instructed to lie supine on a bench with their feet flat on the floor, hips and shoulders in contact with the bench. Subjects were then instructed to grip the Olympic bar slightly wider than shoulder width. Subjects lowered the bar in a controlled manner to the centre of the chest, touching the chest lightly and then extending the arms upward until they were fully extended. Subjects were required to perform a light warm-up of 5 to 8 repetitions at 40 to 60% of their perceived maximum. After a minute"s rest with light stretching the subject was instructed to perform 3 to 5 repetitions at 60-80% of perceived maximum. At this stage the subject was close to their one repetition maximum and therefore a small amount of weight was added. If the lift was successful subjects were instructed to rest for 3 to 5 minutes before attempting a heavier weight. The one repetition maximum was recorded as the weight of the last bench press successfully completed (Franklin *et al*., 2000).

FLEXIBILITY

Flexibility is the ability to move a joint through its complete range of motion and is very important in athletic performance (Franklin *et al*., 2000). The sit and reach test was used to determine the flexibility of the lower back and hamstring musculature.

Prior to completion of the sit and reach test, subjects were instructed to perform a warm-up stretch in order to minimize the risk of injuring themselves. Subjects were instructed to sit with their feet against the sit and reach box with legs fully extended and knees relaxed. Subjects were then required to stretch forward with both hands as far as possible and to hold that position at the furthest point momentarily. The score is the most distant point reached by the fingertips. The results were then recorded. This was repeated 3 times with the best result being recorded (Franklin *et al*., 2000).

AGILITY

Agility refers to a player‟s ability to maintain or control body position while quickly changing direction during a series of movements (Twist and Benickly, 1995). It encompasses movements in non-linear directions e.g. running backwards, sideways etc. as opposed to just running straight. Therefore, agility is crucial in rugby as a large majority of the movements are multi-directional and the maintenance of body position is paramount. Agility is thus determined by the subjects‟ fast speed strength in all movement directions and the neuromuscular co-ordination of the muscles involved.

Subjects were required to complete an Illinois Agility Run, as shown in Figure 4, as quickly as possible with no errors. Subjects were instructed to start the test lying in the prone position on the starting line with their chin touching the floor. On the sound of the whistle subjects stood up and accelerated towards and around the cones as instructed. Individuals were required to complete the run twice whilst being timed and the faster of two trials was recorded.

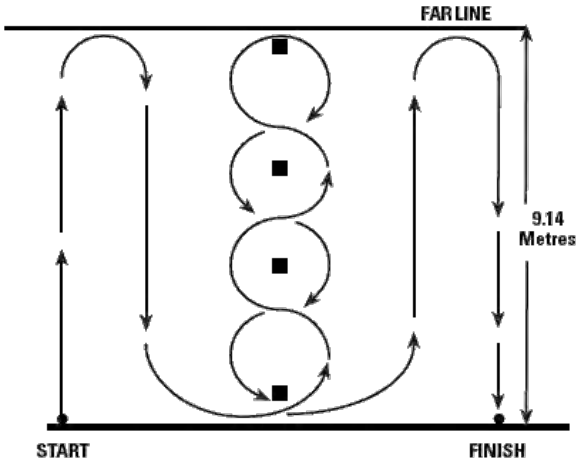


Figure 4: Diagram of Illinois Agility Run.

SPEED

Speed and acceleration are essential components of Rugby Union as players are often required to accelerate to move to other positions nearby or sprint over extended distances. Rugby players typically sprint over distances between 10-20m (Duthie *et al.,* 2003).

Speed was measured over 10m and 40m distances. Players were instructed to perform two 40m sprints with split times being taken at 10m and 40m. Two individuals timed both distances and the average of their two trials was recorded. After both trials had been completed the subject"s fastest time for each distance was recorded.

POWER

Power was assessed using the vertical jump. Subjects were required to stand sideways next to the vertical power touch pad and extend their arm up, pushing the lowest part of the touch pad up with their fingers, whilst remaining flat footed. Subjects were instructed to jump off both feet, from a crouched stance, and reach up as far as possible, touching the touch pad at their highest point. Once three vertical jump trials were completed the best result was recorded.

PHYSIOLOGICAL PARAMETERS

HEART RATE MONITORING

A Suuntu® T6 heart rate monitor was used to store all the data. These monitors were chosen as they do not require the individual to wear a watch as all data is stored directly onto the belt and one is able to download the data post the exercise bout. Heart rate recoding has been used as an indicator of work output due to its linear relationship with oxygen uptake at submaximal loads (Deutsch *et al.,* 1998). Therefore, recording heart rate during competitive Rugby Union would be a predictor of physiological strain and output from the subject.

Reference heart rate was recorded prior to match day, during baseline assessment. Subjects were instructed to lie down and relax for a minimum of two minutes to ensure heart rate values were as close to resting as possible. Heart rate responses are, however, affected by anticipation, movement, changes in breathing patterns, speech and many other factors making the recording of a resting heart rate difficult. During the 2 minutes the lowest recorded heart rate was used as the subject‟s reference heart rate. As a result of the normal variability in resting heart rate, Durant *et al*. (1992) suggests that three measures of resting heart rate are needed in order to achieve a reliability above 0.9 that this measure may be used as a reference figure. On the basis of this, three reference heart rate recordings were taken prior to match day; these were taken before match day as to avoid recording of anticipatory heart rate.

An anticipatory heart rate was recorded just prior to the competitive match. This only occurred once players had completed their warm up and just prior to kick off of the competitive match. Heart rate was then recorded during match play using the heart rate monitors that were fixed in place with tape in order to ensure that contact was maintained throughout the match. The beat-to-beat data was then downloaded from the belt and analysed. The data collected was then calculated into heart rate zones based on age-predicted maximum heart rate and then compared to the data of Deutsch *et al*. (1998).

Heart rate was also recorded during the experimental and control protocols as an indicator of the physiological effort required from the players in order to ensure that the physiological demands of the current cohort was similar to that of competitive Rugby Union.

MULTI-STAGE 20M SHUTTLE RUN

The multi-stage fitness test was designed to determine an individual"s maximal aerobic power and is used to estimate an athlete"s maximal oxygen uptake (VO_{2max}). The test predicts VO_{2max} with reasonable accuracy in a heterogeneous sample group (r = 0.92). This test is especially useful for players who partake in sports of an intermittent nature as it mimics this activity profile. The subjects were instructed to run on a 20m course set out with lines and beacons (Ledger *et al.*, 1988). Subjects were required to run from one end to the other paced by a sound emitted from a pre-recorded CD. The starting speed was 8.5km.h^{-1} and subjects were then required to increase speed at 0.5km.h^{-1} per minute ensuring at the completion of each 20m, that their foot crossed the line coinciding with the sound signal. When the player could no longer keep up with the pre-determined pace, the last stage number announced was used to predict their maximal oxygen uptake (appendix A).

PSYCHOPHYSICAL PARAMETERS

RATINGS OF PERCEIVED EXERTION

Differential ratings of perceptual effort were used in the present study in order to identify perceived physical effort. In particular, “Central” Ratings of Perceived Exertion (RPE), which focus on perceptions of central cardiorespiratory strain, and “Local” ratings which focus on effort perceptions of selected muscular groups, in this case the lower limb musculature, were utilised.

Borg‟s scale of Ratings of Perceived Exertion (1970) is one of the most commonly used psychophysical rating scales to assess the strain experienced by subjects as it provides a good indicator of the degree of physical strain experienced. The popularity of the RPE scale is that the perception of central strain has a close association with physiological measures, particularly heart rate. The scale, which ranges from a value of 6, for almost no strain, to a maximal value of 20, which represents exhaustion, was presented and explained in detail to all subjects (appendix B). During the protocol subjects were required to rate „central‟ and „local‟ strain on the numerical scale in order to indicate their level of exertion.

BODY DISCOMFORT MAP AND SCALE

Discomfort can be regarded as an indicator of the degree of strain placed on the musculoskeletal system. The scale used was developed by Corlett and Bishop in 1976 and has been adapted to include an anterior and posterior view of the body with 28 regions (appendix B). Discomfort is rated on this map with a rating scale of 1 for “minimal discomfort” to 10 for “maximal discomfort”. The purpose and rating of the scale was explained in detail to all subjects. Sites and discomfort were obtained and recorded at half time and on completion of the game and both laboratory protocols.

PAIN PERCEPTION SCALE

The pain perception scale (Table IX) was used by the players to rate discomfort in the lower limb musculature in the 7 days following each protocol.

Table IX: Rating scale of muscle soreness post protocols

Pain Perception scale	
1	Slightly sore – sore to touch
3	Sore when contracting
5	Uncomfortable to walk
8	Constantly painful
10	Unbearable

EXPERIMENTAL PROCEDURES

Experimentation was divided into three main phases. The first involved obtaining anthropometric, „reference" cardiovascular and baseline „fitness" measures of all players. The second phase involved assessment of the physiological and perceptual responses to *in situ* and uncontrolled competitive rugby matches. The third phase involved the assessment of muscular, physiological and perceptual responses to two protocols: a rugby-specific intermittent protocol (experimental condition) and a control protocol. All experimentation was conducted in the Human Kinetics and Ergonomics (HKE) department at Rhodes University, Grahamstown, South Africa.

PHASE I: INTRODUCTION AND COLLECTION OF BASELINE DATA

Subjects were all required to attend an introduction session in the HKE department. Players were instructed not to engage in strenuous activity within 24 hours of being tested, not to have consumed alcohol within the past 24 hours, to have eaten sufficiently, but not within three hours of being tested and to inform the researcher if ill (appendix A).

This session involved explaining the procedures to the subjects verbally and in writing (appendix A). It also addressed any queries that the subjects may have had. Subjects were required to sign a consent form before any data was collected. A physical activity screening questionnaire was filled in to ensure no players were at risk to exercise (appendix A). Basic data were obtained and included age, playing position, number of years" experience and a full injury history of each subject. General data was then measured which included stature, mass and skinfold measurements. Baseline fitness tests included a one repetition maximum bench press, Illinois Agility Run, sit-and-reach flexibility, vertical jump power test and 10m and 40m sprint as well as a beep test.

Baseline isokinetic data was collected at 60deg.s^{-1}. Subjects were required to perform a warm up of 5 minute slow jog and then stretch before completing isokinetic testing. Subjects were instructed to exert a maximal force throughout the entire range of motion.

Subjects were then familiarized with the Memory Belts as they were required to wear them for three practice sessions, before any data were recorded. Habituation to the memory belts was carried out in order to ensure that player comfort was not neglected and to put the players at ease and minimize any responses brought about by anxiety or stress imposed by the belt, rather than the game itself.

On a separate occasion, players were required to attend a treadmill habituation session to ensure that all individuals were familiar and comfortable running on a treadmill especially at the fast (21km.h^{-1}) speeds.

PHASE II: COLLECTION OF PHYSIOLOGICAL AND PERCEPTUAL DATA

Heart rate was monitored during competitive games to assess the level of physical strain of the players (Deutsch *et al.,* 1998). A total of five games was assessed with heart rate data being analysed and subdivided into four categories expressed as a percentage of maximum heart rate namely: low (<75%), moderate (75-84%), high (85-95%) and maximal (>95%) as can be seen in table VI.

On arrival at the field all relevant testing and requirements were explained to the players. A Suuntu® memory belt was placed on each individual before the game and fixed in place using tape in order to minimize movement of the belt. Players were then urged to wear shoulder and chest protection in order to minimize the movement of the heart rate strap. Continuous beat-to-beat heart rate data were successfully recorded and stored on the Memory Belt for all subjects throughout each match. All belts were synchronized to a central clock and half time was noted.

Reference heart rate data were established from data collected beforehand during habituation sessions. At the end of the game all memory belts were removed and labelled. Data were then downloaded from the memory belts and the beat- to- beat data were stored and analysed. The results (excluding half time readings) were analysed and maximal and average heart rates were noted. The data collected during the 5 competitive games were then compared to existing literature (Deutsch *et al.*, 1998). Ratings of central and local RPE and body discomfort were taken during the 10- minute half time break and immediately after the completion of the match.

PHASE III: EXPERIMENTATION

Prior to the testing session, the order in which the two protocols would be administered was randomly assigned to each player. This guaranteed that any differences found in the responses could be attributed to the different protocols and not to the effects of familiarisation and/or fatigue. Subjects were required to perform both tests within six to eight days of each other at the same time to reduce the impact of circadian changes on heart rate.

Subjects were all tested individually during the experimental session. On arrival subjects were fitted with a heart rate monitor. The area where the electrodes were to be placed was shaved and cleaned with alcohol to ensure good conductivity. EMG electrodes were then attached to the skin according to placements used by Pincivero *et al*. (2000) in the direction of the muscle fibres. This was done by the player contracting the individual muscle groups, getting them to perform specific movements and/or by palpation of the muscles by the researcher. Finally, the wires were attached to the electrodes.

Players then completed a warm-up: this included some stretching exercises specific to Rugby Union and running at a slow speed for 5 minutes. Prior to commencing the protocol, each player completed isokinetic base-line measures on an isokinetic dynamometer, the CYBEX 6000. The protocols were randomized in administration and

the same testing procedures were followed for each protocol. Three repetitions were performed on each leg at 60deg.s^{-1} with a rest period of 30s between each set.

Subjects were then required to complete the EMG measurement protocol. Immediately after this was completed subjects performed either the 80 minute rugby-specific protocol or the continuous control protocol. During both protocols heart rate measures were collected on a beat-to-beat basis using the Suunto® t6 memory belt. Central and Local RPE and body discomfort was recorded at 20 minute intervals. During the half time break subjects were required to perform the EMG measurement protocol and the isokinetic dynamometer protocol in a time frame of 10-15 minutes. The subjects then performed the second 40 minute exercise protocol after which the EMG measurement and isokinetic dynamometer protocols were completed for the final time. Subjects were required to record any pain of discomfort (delayed onset muscle soreness) induced by either protocol, for the following seven days (Table IX).

STATISTICAL ANALYSES

Statistical analyses were performed using Statistics software (StatSoft, Inc. (2007). STATISTICA (data analysis software system). Verison 8.0.). Descriptive analyses and tests for normal distribution were carried out using STATISTICA, before any statistical tests were performed. Repeated measures analyses of variance (ANOVA) were used to determine statistically significant changes in the physiological, muscular and psychological measures between the two protocols. The majority of the null hypotheses were rejected at a probability of $p<0.01$, providing a confidence level of 99%. This only allowed 1% chance of rejecting a true hypothesis (Type I error). However, a significance level of $p<0.05$ was used (where stated); this provided a 95% confidence level and allowed a 5% chance of rejecting a true hypothesis. Tukey *post-hoc* multiple comparison tests were performed where significance occurred within one protocol over time.

Predictive variance was calculated using the following formula: $1 – (\sqrt{1-R^2} \times 100)$.

All data are presented as means $\pm$ standard deviation.

CHAPTER IV

RESULTS

INTRODUCTION

The purpose of this investigation was to compare muscle activity changes and changes in eccentric and concentric force production of the quadriceps and hamstring musculature during a simulated Rugby Union laboratory protocol, which excluded the contact nature of the game. The protocol was used to investigate the time history of the physiological, biophysical, mechanical and perceptual responses to rugby-specific activity. A group of trained first team rugby players (n=18) was used and the results compared to a control protocol which involved continuous walking at a set pace of equivalent distance.

SUBJECT RECRUITMENT

18 subjects were recruited from the Rhodes University first XV rugby team. Selection criteria were as follows: All players had to be actively taking part in the Eastern Province Super League. Front row forwards were excluded from the study. Subjects with any injury to the knee, quadriceps, hamstrings or any previous knee surgery were excluded as this may have impacted on the results recorded.

BASELINE MEASURES

Table X: Means (±SD) baseline measures recorded pre-testing.

Measure		Mean	SD	CV
Years of rugby experience (yrs)		10.32	3.75	36.30
Flexibility (mm)		88. 9	113.9	128.1
Power (mm)		535.8	59.20	11.05
Strength (kg)		101	17	17
Strength as a percent of body mass (%)		117.03	14.92	12.75
Speed (s)	**10m**	1.98	0.19	9.60
	40m	5.76	0.42	7.29
Agility (s)		17.38	0.82	4.72
Multi-stage fitness test		10.6	1.9	17.6

SD = standard deviation; CV = Coefficient of variation (%)

All players had a number of years (10.32) experience playing Rugby Union (Table X). While speed varied little between players, this can be expected as speed over short distances is advantageous for all positions in Rugby Union. There was also low variability in the aerobic fitness measures and high variability in the flexibility and strength of the group: the latter two measures being position specific and/or not a precursor for performance.

PHYSIOLOGICAL MEASURES

HEART RATE

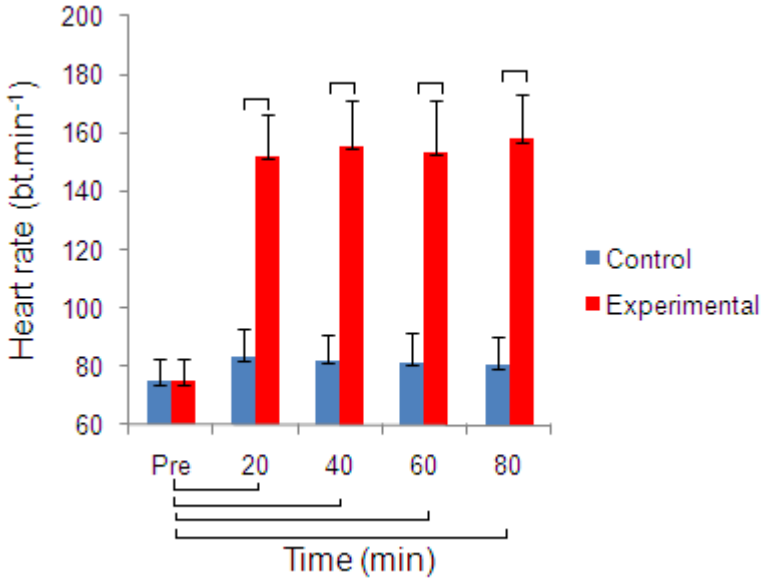


Figure 5: Comparison of heart rate responses for both protocols over time.

*Denotes significant differences between protocols

Significance bars between time intervals denotes a significant difference for both protocols

Mean anticipatory heart rates were not different prior to the both protocols indicating that players were not overly apprehensive prior to the more demanding experimental protocol (75±7 beat.min^{-1} for the control protocol and 75±8 beat.min^{-1} for the experimental protocol). Heart rate increased significantly with both protocols in the transition from rest to exercise with a significantly ($p<0.01$) greater increase in the experimental protocol compared to that of the control protocol. Heart rates did not change over time during either protocol although the experimental heart rates were significantly ($p<0.01$) higher than the control responses at all time intervals. During the control protocol heart rate ranged from 80 (±10) beat.min^{-1} to a maximum of 83 (±10) beat.min^{-1} (41% age predicted heart rate maximum). During the experimental protocol heart rate responses were similar over time and ranged from 152 (±14) beat.min^{-1} to 158 (±15) beat.min^{-1} (79% age predicted heart rate maximum).

BIOPHYSICAL MEASURES

ELECTROMYOGRAPHY

QUADRICEP MUSCULATURE

Vastus Medialis

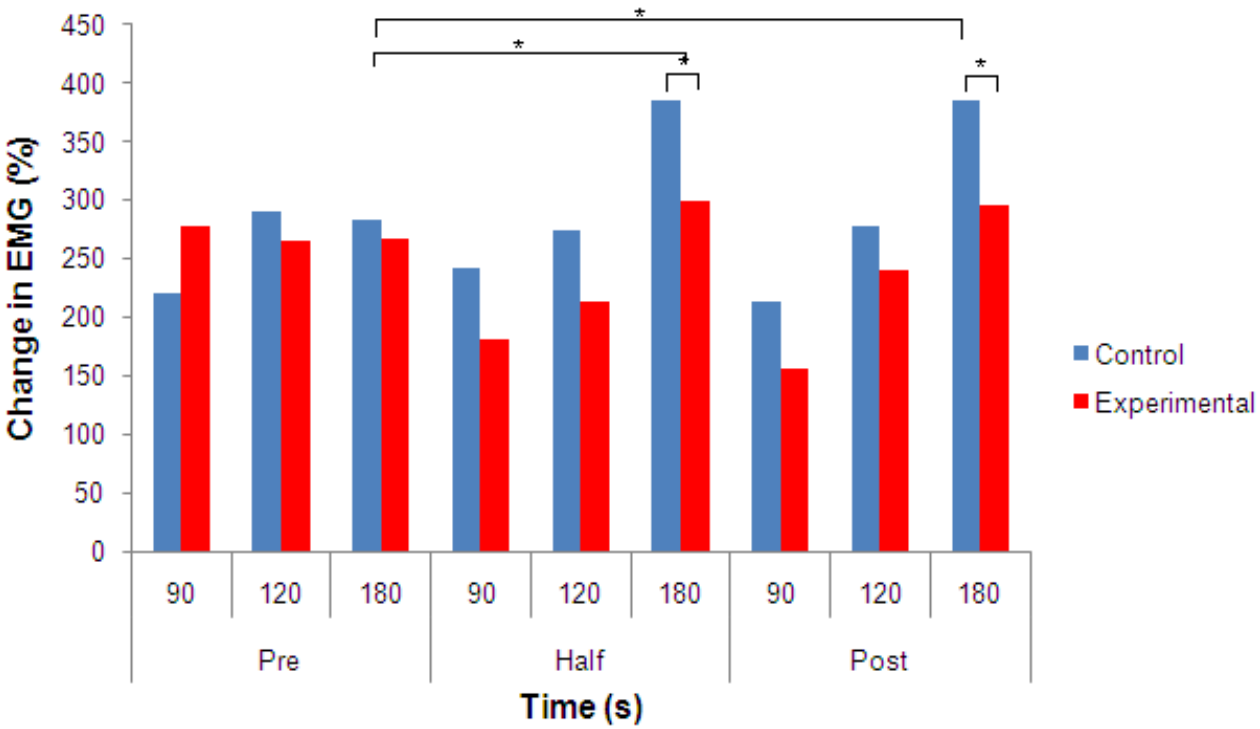


Figure 6: Comparison of percentage change in Vastus Medialis EMG for both protocols over time.

Significant increases with speeds not shown

There were no differences in the percentage change in EMG (from 0s) in the vastus medialis (VM) from pre-protocol to half time. Further, there was no difference between the two protocols at the 90s and 120s intervals. In contrast, there was a significant ($p<0.01$) increase in EMG activation in the VM at 180s in the control protocol at half and post condition intervals. The control protocol elicited significantly higher EMG activity levels at half time and on completion of the protocol than when compared to that of the starting measures.

Vastus Lateralis

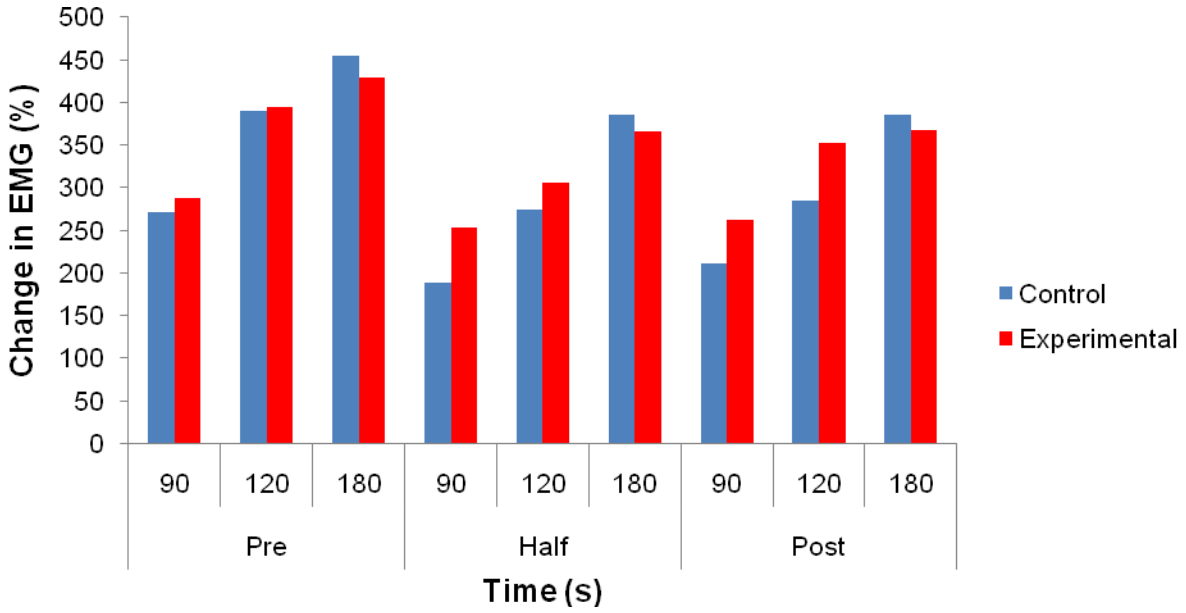


Figure 7: Comparison of percentage change in Vastus Lateralis EMG for both protocols over time.
Significant increases with speeds not shown

The percentage change in EMG of the vastus lateralis (VL) did not vary between the protocols and over time. At 90s and 120s the trend was for increased EMG activity with the experimental protocol at all time intervals. The trend was opposite at 180s when the control protocol showed higher EMG activity than the experimental protocol.

HAMSTRING MUSCULATURE

Bicep Femoris

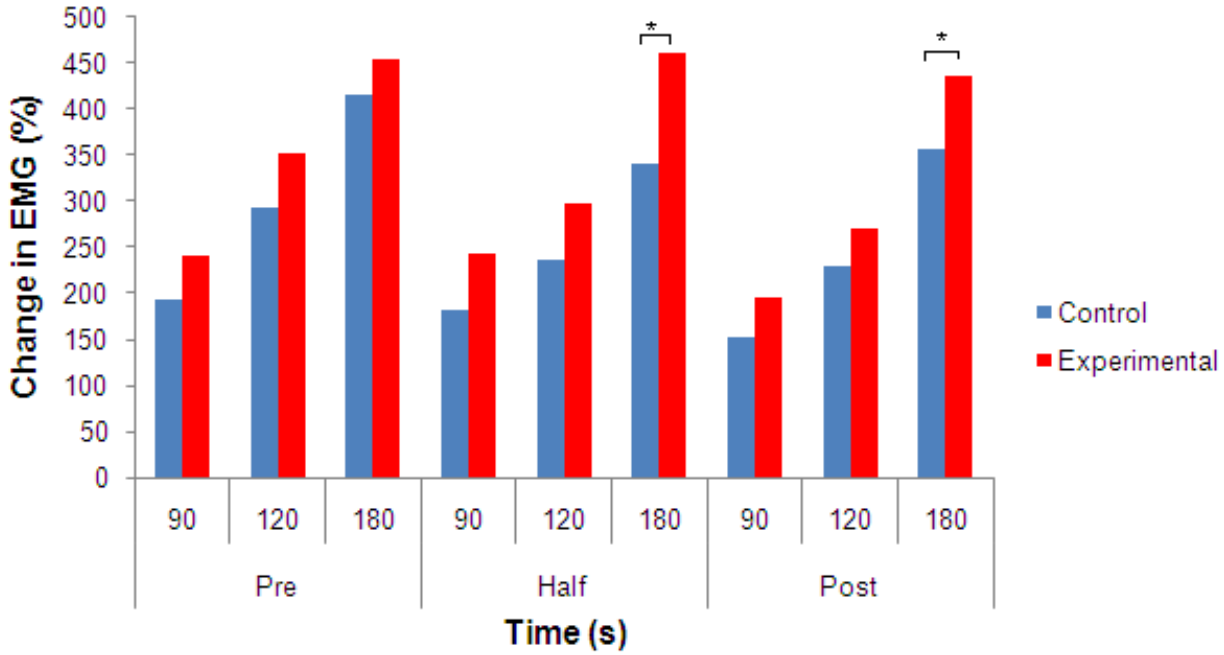


Figure 8: Comparison of percentage change in Bicep Femoris EMG for both protocols over time.

Significant increases with speeds not shown

While the trend was for higher muscle activity in the quadriceps musculature during the control protocol, the opposite occurred in the experimental condition when the EMG activity of the biceps femoris (BF) was higher at all 3 speeds and intervals. The percentage increase between the two protocols was not statistically significant at 90s or 120s. At 180s, the half time and final measure during the experimental protocol elicited significantly ($p<0.01$) higher EMG activity than the control protocol.

Semitendinosus

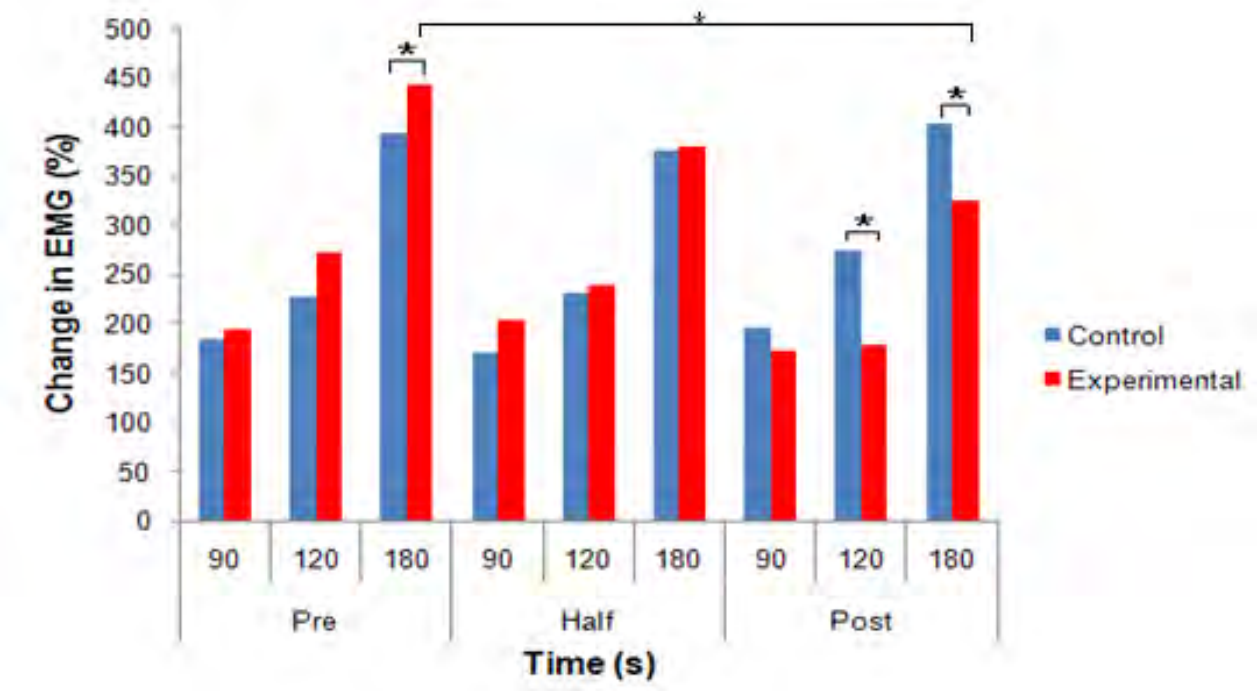


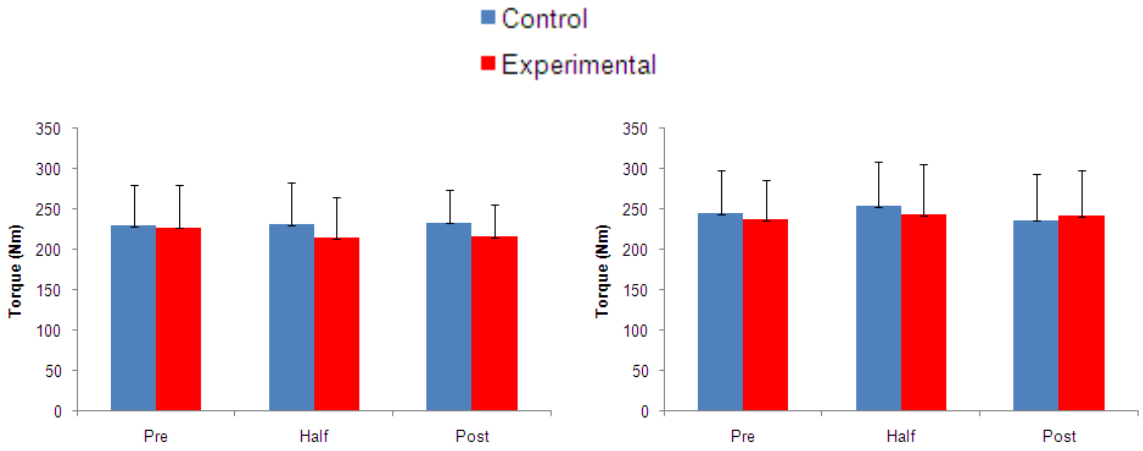
Figure 9: Comparison of percentage change in semitendinosus EMG for both protocols over time.
Significant increases with speeds not shown

There were no differences in EMG measurements at 90s or 120s pre-protocol. However, at 180s, the experimental protocol had significantly ($p<0.01$) higher EMG activity than the control protocol. In contrast the half time EMG results were similar. On completion of the protocol, the EMG results were similar at 90s, but at 120s and 180s, the control protocol elicited significantly ($p<0.01$) greater increases in EMG activity than the experimental protocol. There was a significant decrease in EMG activity from pre- to completion of the experimental protocol at 180s.

ISOKINETIC STRENGTH

QUADRICEPS – EXTENSORS

Peak torque

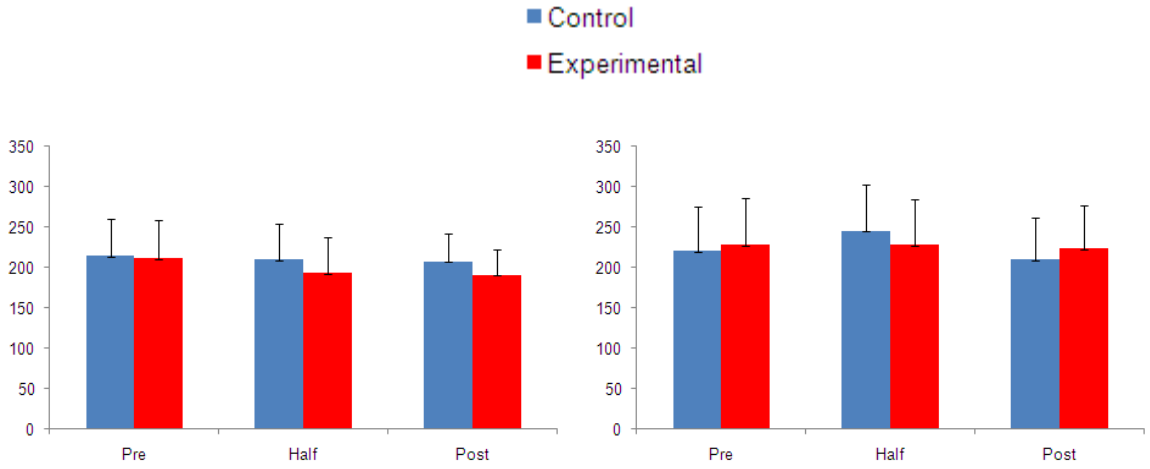


(A) **(B)**

Figure 10: Comparison of peak torque values of concentric extensors (A) and eccentric extensors (B) for both protocols over time.

Eccentric extensor strength was greater (254.69±57.86 Nm) than concentric extensor strength (221.24±47.32 Nm). There was no difference in concentric and eccentric extensor peak torque recorded for both protocols over time.

Work

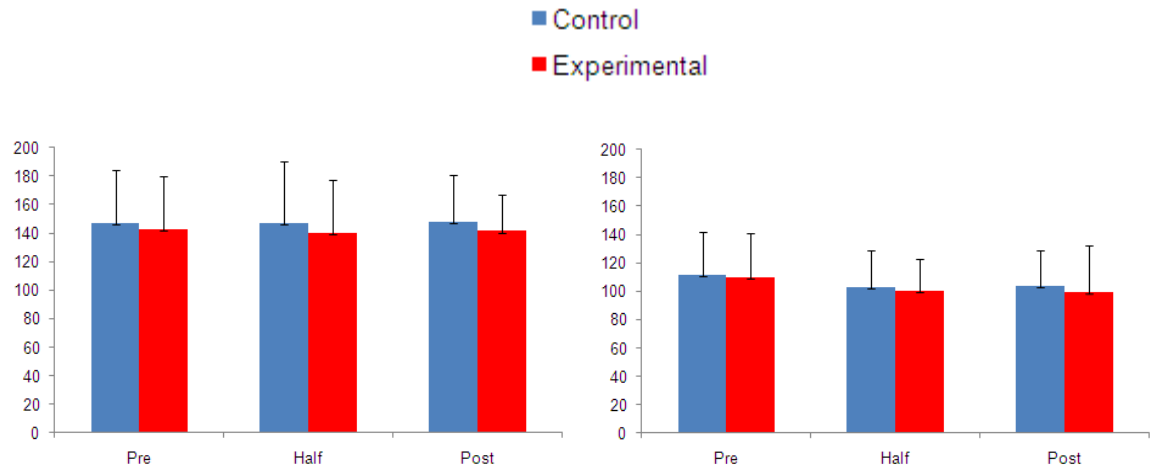


(A) **(B)**

Figure 11: Comparison of work done by the concentric extensors (A) and eccentric extensors (B) for both protocols over time.

Work done by the concentric and eccentric extensors showed no difference between the protocols and over time. Overall, the extensors were stronger eccentrically (242.15±58.29 J) than concentrically (204.59±50.48 J). Work done by the eccentric extensors was, on average, 277.57% of body mass, whilst the concentric extensors were 234.51% of body mass.

Power



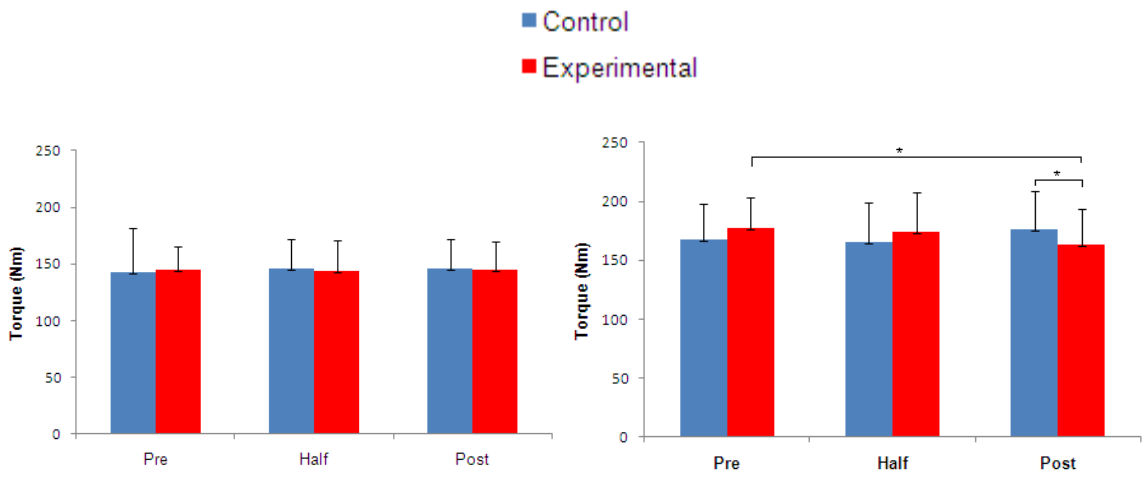
(A) **(B)**

Figure 12: Comparison of power produced by the concentric extensors (A) and eccentric extensors (B) for both protocols over time.

The power produced by the concentric extensors was similar over time and for both protocols with a maximum value recorded post-control protocol (142.56±28.74 W) and a minimum recorded at half time (136.90±30.13 W). Power produced by the eccentric extensors did not vary during the experimental protocol. Maximum values were recorded at the start (111.20 ±32.15 W) and minimum values were recorded at half time (100.58±24.86 W).

HAMSTRINGS – FLEXORS

Peak torque

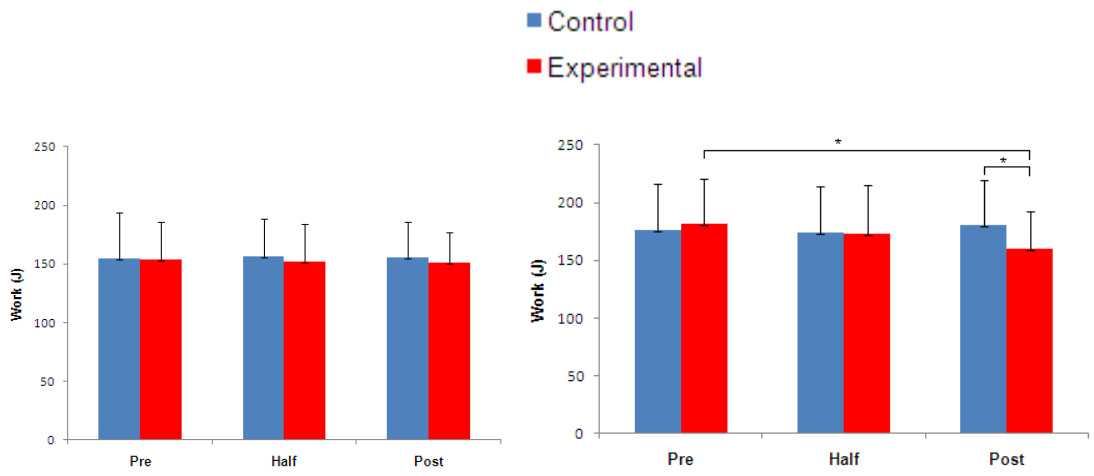


(A) **(B)**

Figure 13: Comparison of peak torque produced by the concentric flexors (A) and eccentric flexors (B) for both protocols over time.

Overall the eccentric flexors produced greater peak torque values (all values < 145Nm) than the concentric flexors (all values >145Nm). Further, the eccentric flexor values were significantly ($p<0.05$) different as a product of time and protocol. There was no difference in peak torque produced by the concentric flexors over time during the control protocol. During this protocol, values ranged from a minimum value of 142.56 (±34.70) Nm at the beginning to a maximum of 146.11 (±27.52) Nm. The peak torque produced by the eccentric flexors during the experimental protocol decreased significantly ($p<0.05$) over time from a pre-protocol value of 176.83 (±26.79) Nm to 162.89 (±30.66) Nm at the end. Thus there was a 7.88% decrease in peak torque during the duration of the experimental protocol. In contrast, eccentric peak torque increased by 5.11% over the duration of the control protocol. On completion of both protocols, the peak torque of concentric flexors (175.83±32.69 Nm) was significantly ($p<0.05$) higher than those obtained on completion of the experimental protocol (162.89±30.67 Nm). Peak torque recorded post the control protocol was 7.90% greater than the values recorded following the experimental protocol.

Work

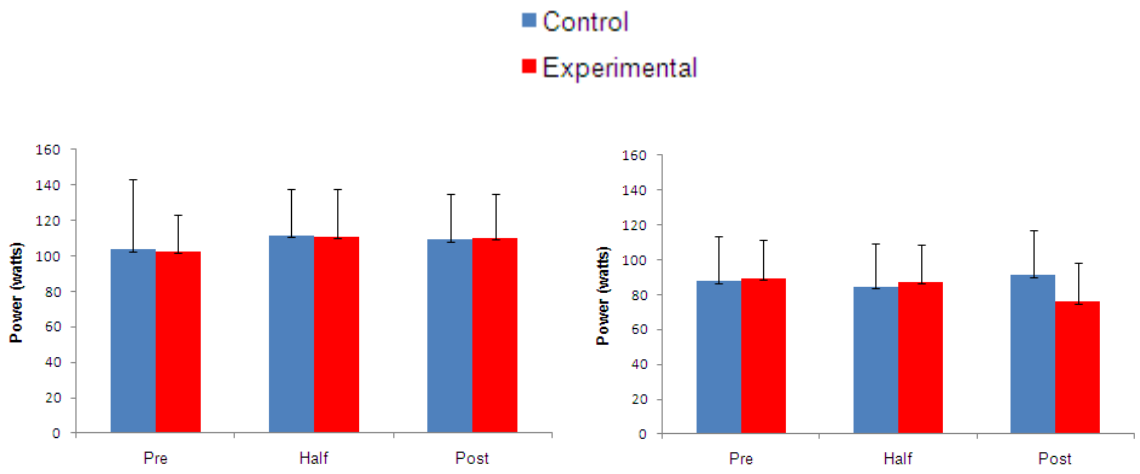


(A) **(B)**

Figure 14: Comparison of work done by the concentric flexors (A) and eccentric flexors (B) for both protocols over time.

There was no difference in work done by the concentric flexors during either protocol and over time. In contrast work done by the eccentric flexors decreased significantly ($p<0.05$) over time during the experimental protocol. More specifically, work done by the eccentric flexors decreased by 12.09% over time. In contrast work done increased by 2.60% during the control protocol. On completion, the control protocol (183.35±48.51 J) showed significantly ($p<0.05$) higher values than the experimental protocol (156.85±34.98 J). Post-control values were 17.04% greater than post-experimental values.

Power



(A) **(B)**

Figure 15: Comparison of power produced by the concentric flexors (A) and eccentric flexors (B) for both protocols over time.

There was no difference in the power produced as a result of protocol or over time. However, the concentric flexors produced more power (all values >100W) than the eccentric flexors (all values <100W).

QUADRICEPS TO HAMSTRING RATIO

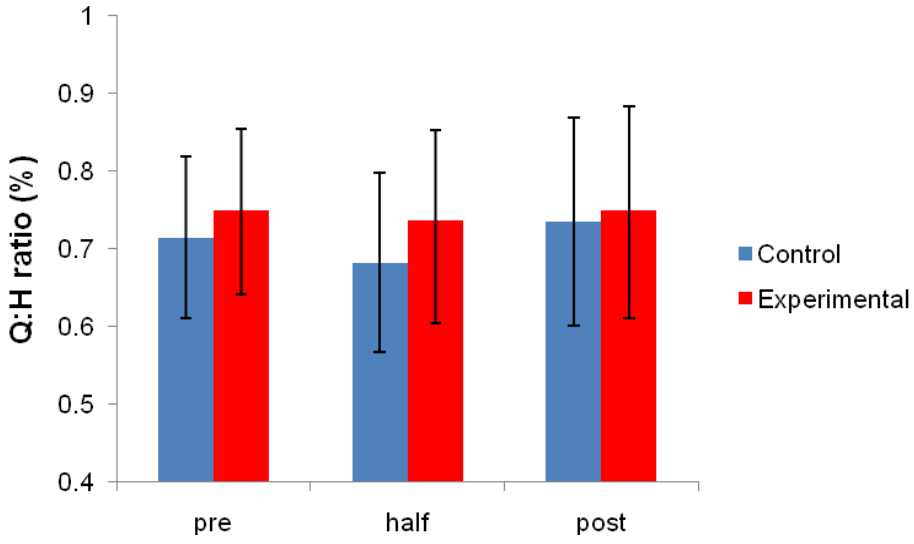


Figure 16: Changes in the quadriceps to hamstring ratio (Q:H) over time during both the control and experimental protocols.

Quadriceps:hamstrings (Q:H) ratio ranged from 0.72 to 0.75. These ratios did not change in either protocol over time.

PERCEPTUAL RESPONSES

CENTRAL AND LOCAL RPE

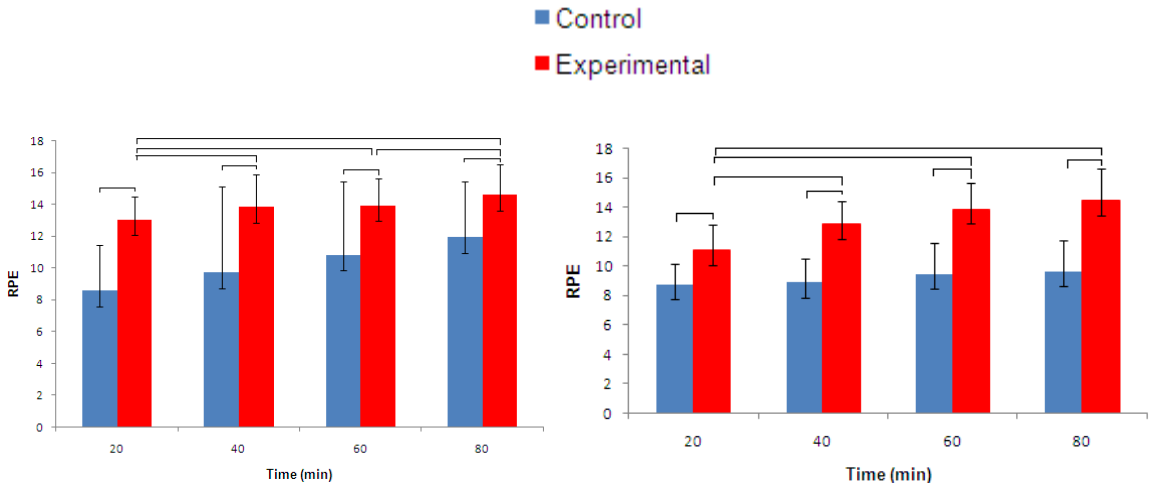


Figure 17: Comparison of "Central" RPE (A) and "Local" RPE (B) values recorded for both protocols over time.
Significance bars between time intervals denotes a significant difference for both protocols

There were no significant changes in "Central" or "Local" RPE over time during the control protocol. "Central" and "Local" RPE values were significantly ($p<0.01$) higher at all measurement intervals for the experimental protocol when compared to control values. Perceived "Central" ($p<0.05$) and "Local" exertion ($p<0.01$) increased significantly over time (20, 40, 60 and 80 minutes) during the experimental protocol. During this protocol "Central" and "Local" RPE values were similar, while in the control protocol "Central" RPE values were higher at 20, 40 and 60 min (9; 10; 11) than "Local" RPE (8; 9; 9). Therefore, during the control protocol, local discomfort was less. Further, there was greater variability in the "Central" PRE responses compared to the "Local" responses during both protocols.

BODY DISCOMFORT

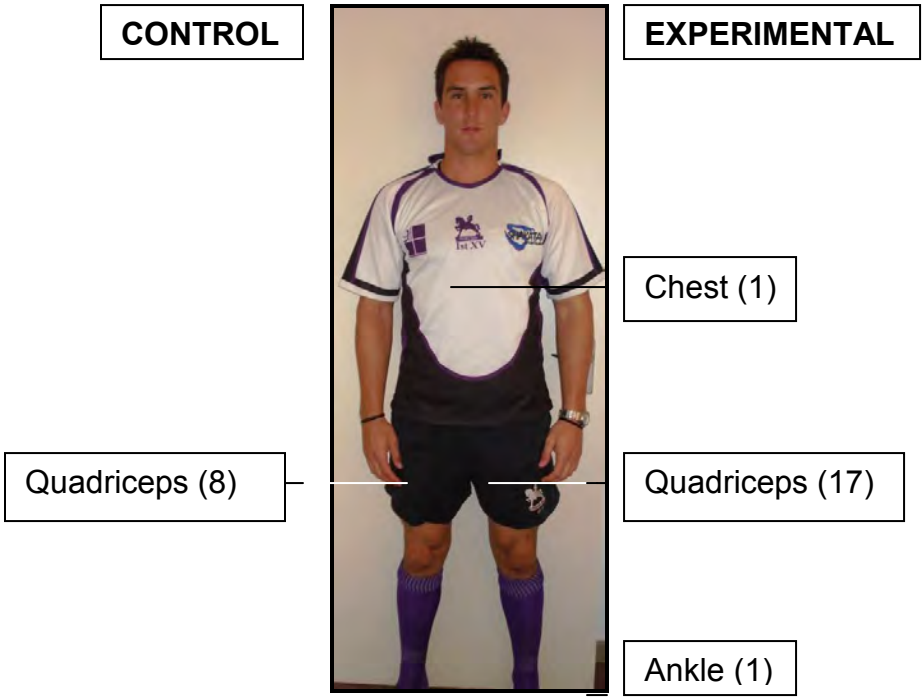


Figure 18: Perceived anterior body discomfort during the control and experimental protocols.

() indicates the area of discomfort and the number of subjects who reported discomfort in that area

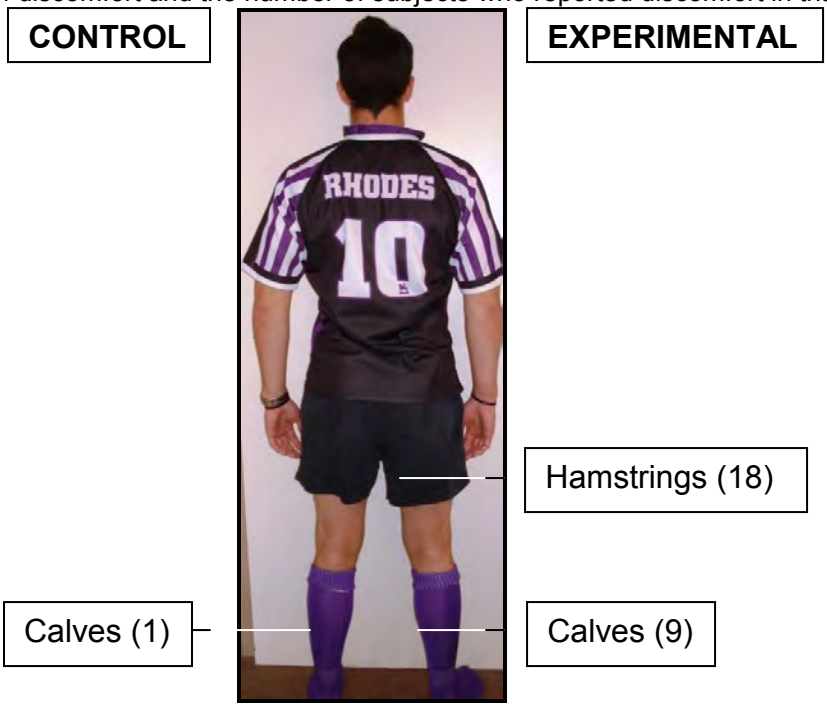


Figure 19: Perceived posterior body discomfort during the control and experimental protocols.

() indicates the area of discomfort and the number of subjects who reported discomfort in that area

During the experimental protocol there were many more reports of body discomfort, 46 in total, in comparison to the nine reported during the control protocol. Quadriceps discomfort was indicated during both protocols with 8 reports in the control and 17 during the experimental. The highest rating of discomfort in the experimental condition was to the hamstring musculature (18 reports – total sample). During the control protocol the ratings of perceived discomfort were low (Figure 20); and during the experimental protocol they were higher.

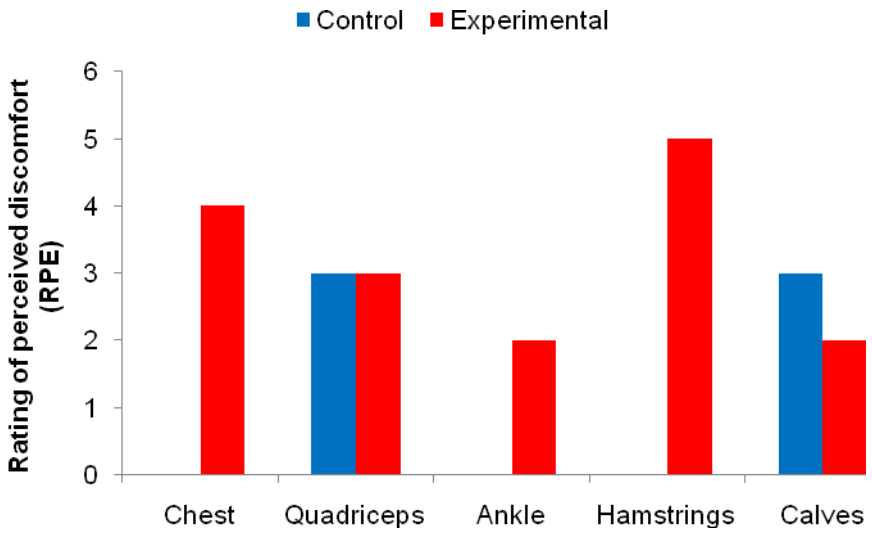


Figure 20: Perceived body discomfort rating, out of a possible ten, during the control and experimental protocol.

The gastrocnemius region and quadriceps were reported in both protocols and had similar intensity ratings. There were many other ratings in the experimental protocol, most notably the hamstrings with the most frequency reported and the most intense (5) ratings.

EXERCISE INDUCED MUSCLE SORENESS

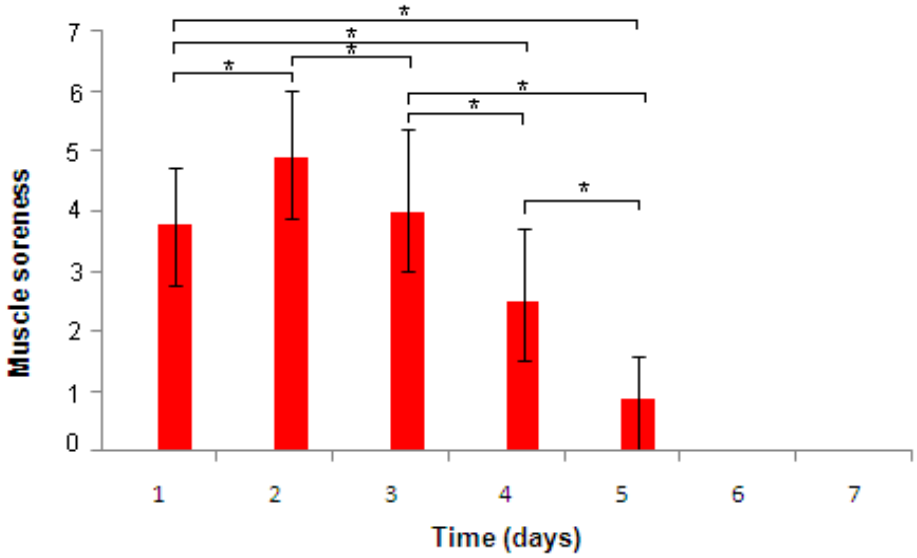


Figure 21: Comparison of rating of exercise induced muscle soreness for seven days post-experimental protocol.

There were no ratings of delayed onset muscle soreness following the control protocol. The experimental protocol elicited muscle soreness for five days following the activity (Figure 21). Day two resulted in significantly ($p<0.01$) greater muscle soreness than the other four days. There was a significant ($p<0.01$) increase in muscle soreness from day one to day two; thereafter there was a significant ($p<0.01$) decline in muscle soreness until days six and seven when soreness had completely dissipated.

MULTIVARIATE ANALYSES

ISOKINETIC STRENGTH AND PERCEIVED DISCOMFORT

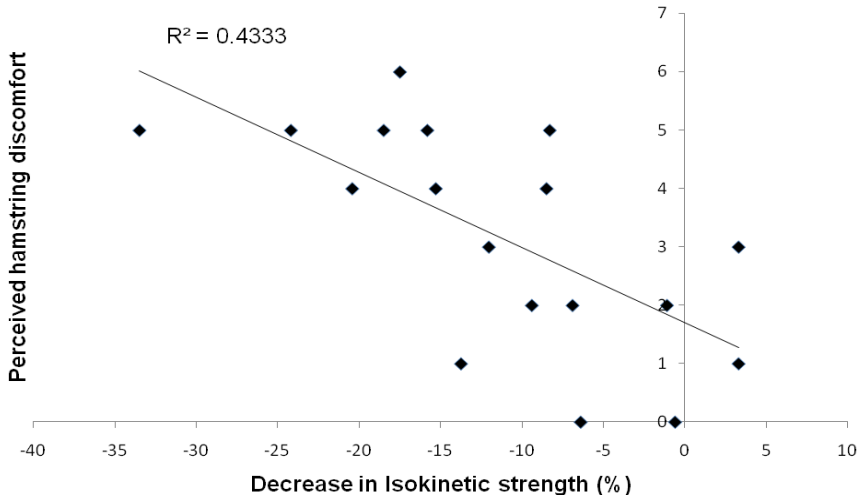


Figure 22: The relationship between the decrease in eccentric flexor isokinetic strength and perceived hamstring discomfort during the experimental protocol.

There was a weak relationship between perceived hamstring discomfort post the experimental protocol and a decline in eccentric flexor strength, (R^2=0.43). Therefore, 9.70% of the time, perceived hamstring discomfort can provide an indication of comprised eccentric strength of the hamstrings.

HEART RATE AND "CENTRAL" RATING OF PERCEIVED EXERTION

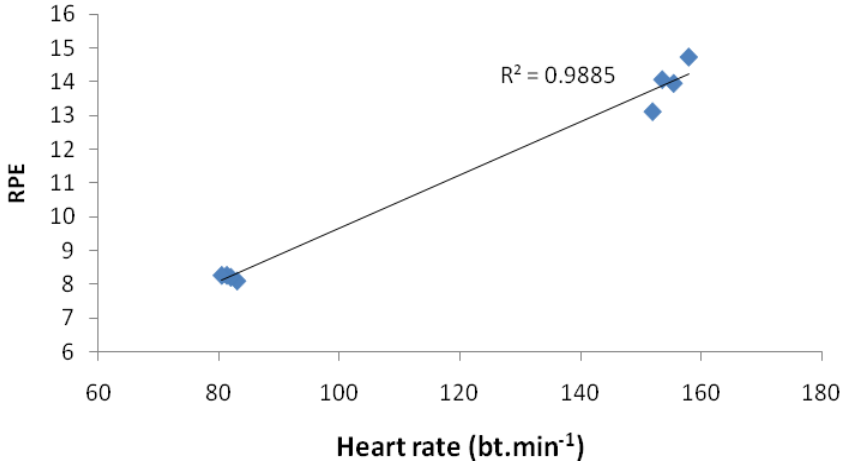


Figure 23: Relationship between Central RPE and heart rate.

There was a very strong relationship between „Central" RPE and heart rate (R^2=0.99) during the experimental protocol. Therefore „players" accurately perceived the demands on their central cardiovascular system during the intermittent activity profile.

ELECTROMYOGRAPHY AND ISOKINETIC STRENGTH

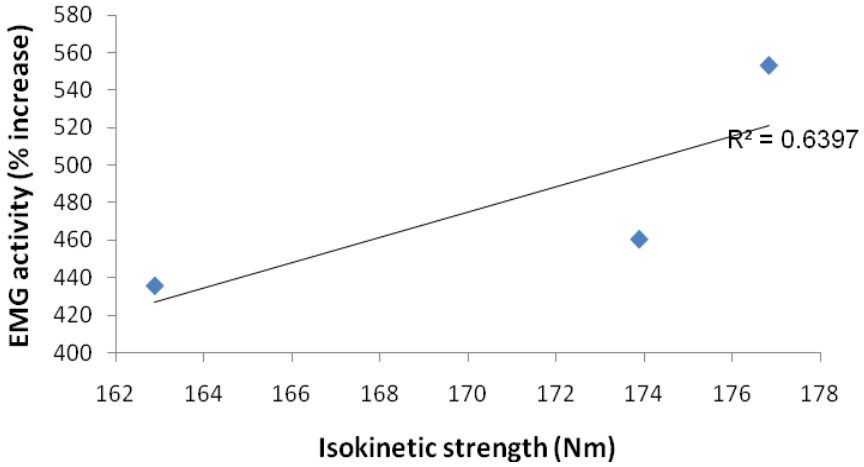


Figure 24: Relationship between EMG recorded for the bicep femoris and isokinetic strength of the hamstring musculature.

There was a moderately strong relationship (R^2=0.64) between percentage change of iEMG activity (from baseline) of the bicep femoris and isokinetic strength of the eccentric flexors during the experimental condition. Therefore in 23.16% cases, a decrease in iEMG activity will lead to a decrease in isokinetic strength.

CHAPTER V

DISCUSSION

INTRODUCTION

The aim of this study was to investigate the time history of the physiological, biophysical, mechanical and perceptual responses to rugby-specific activity. Direct comparisons with previous studies are difficult as fatigue effect is specific to the exercise protocol and previous authors have failed to represent the intermittent activity profile of rugby match play (St Clair Gibson *et al*., 2001). The protocol used considered the mechanical demands of the intermittent running characteristic specific to Rugby Union, replicating the short duration of exercise bouts and subsequently providing acceleratory and declaratory demands typical of match play. It should be noted that the participants used in the present study were competing at a university level, but were expected to complete a laboratory intermittent protocol based on professional activity profiles (Duthie *et al.,* 2005).

BASELINE MEASURES

The average number of years playing rugby was 10.32 (±3.75) as can be seen in Table X, pg 61). This shows that all players had a number of years‟ experience and skill. There was a large amount of variation in the flexibility measures (88.9 ±113.9 mm) recorded during the sit and reach test. This can be considered „average‟ when compared to normative data (ACSM guidelines, 2006). The importance of good hamstring flexibility during rugby is debatable although recently Naylor (2007) found that flexibility, measured by the sit and reach test, was not a predictor for rugby injury or lower limb injury. In contrast, Witrouw *et al*., (2003) showed a significant association between pre-season hamstring muscle tightness, measured in the supine position, and the development of a hamstring injury in soccer. Agre (1985) suggests that a lack of flexibility may cause the musculotendinous unit to stretch beyond its ability to elongate in

the latter part of the swing phase and a tear may result. This is most likely to happen during sprinting because the requirement for flexibility is greatest and the forces involved are high. This is in accordance with injury statistics of Rugby Union as large quantities of sprinting have been shown to predispose players to hamstring injuries (Devlin, 2000; Bottini *et al*., 2000). It is interesting to note that despite the lack of flexibility in the current sample group, none of the players assessed developed hamstring injuries during the duration of the 2009 season. Therefore, these results are in accordance with Naylor (2007) who used a similar sample group to the current research.

The average power achieved during the vertical jump (535.8±113.9 mm) can be categorized as „average" when compared to norms (ACSM guidelines, 2006), and lower than those previously measured on US national rugby players (Carlson *et al*., 1994) yet similar (558.4mm) to university athletes measured by Duthie *et al*. (2003). This difference between data collected by Carlson *et al*. (1994) was to be expected due to the differences in level of player between these studies. The one repetition maximum bench press findings are considered „average" (101±71 kg) and higher than those measured on junior South African rugby players who had a mean of 95 kg (Durandt *et al*., 2006). Speed over 10m was 1.9 (±0.19) s and 5.8 (±0.42) s over 40m. Durandt *et al*. (2006) recorded speeds of 1.9 (±0.1) s over 10m for 18 year old elite South African players and 5.6 (±0.1) s for 40m. Although similar, the slower times in this cohort may be due to the slightly higher body mass recordings (87.24±10.89 kg) compared to that of 84.9 (±8.3) kg reported on in the Durandt *et al.* (2006) study.

The Illinois Agility Run took the subjects, on average, 17.38 (±0.82) s to complete which is considered „average" when compared to ACSM guidelines (2006). Eighteen- year -old junior elite athletes took 15.1 (±0.8) s to complete the same run (Durandt *et al*., 2006). This large time difference may be attributed to the fact that the players in the current research had a larger body mass than the subjects measured by Durandt *et al*. (2006) or that the current study did not use elite players, but rather players of a first team university level. Subjects achieved level 10.7 (±1.9) in the multi-stage fitness test, which was lower than the US national team who achieved 13.7 (±0.9) and reported on by

Carlson *et al*. (1994). It was also lower than the average level reached by the Springbok rugby team prior to winning the 2007 world cup (13.1).

PHYSIOLOGICAL MEASURES

HEART RATE

Anticipatory heart rates were similar for both the control (75±7 beat.min^{-1}) and experimental protocol (75±8 beat.min^{-1}) indicating that players were not overly apprehensive prior to the more demanding experimental protocol. Heart rate then increased significantly in the transition from rest to exercise which was to be expected (McArdle *et al*., 2001). This is due to the greater demands placed on the body during physical exertion, necessitating increases in the supply of oxygen and removal of waste products. From Figure 5 (pg 61) it was clear that heart rate was significantly greater during the experimental protocol than the control protocol for each of the 20-minute intervals which were in accordance with soccer specific research (Grieg *et al*., 2006).

There were no differences in heart rates over time during both protocols. Heart rate increased in the experimental protocol from 152 (±14) beat.min^{-1} at the 20- minute interval to 156 (±16) beat.min^{-1} at half time and 158 (±15) beat.min^{-1}on completion of the protocol. This increase over time was consistent with soccer specific findings (Greig *et al*., 2006).

During the experimental protocol, players spent the majority of their time at 76 – 79% of age predicted heart rate maximum. This was marginally lower than that measured during competitive rugby play when the players spent the majority of their time at 85-95% of age predicted heart rate maximum. Therefore there was a 13% decrease in heart rates recorded in the laboratory. These findings are similar to Greig *et al*. (2006) who also replicated the demands of competitive soccer in a laboratory setting, and obtained responses which were consistently lower than those observed during competitive play. These differences may be explained, in part, by situational factors. Reilly *et al*. (1990) recorded heart rate data during friendly soccer matches and found the data to be lower than that of competitive heart rate data. This was even though the

distances covered were within 2% of competitive match-play. Just as a friendly game may place reduced stress on a player, completing the same activity profile in a laboratory setting may reduce the emotional stress inherent to competitive play. Additional stressors such as interactions with other players, match situation and the importance of the outcome of the game may be responsible for this increase in competitive heart rate (Greig *et al*., 2006; Bangsbo, 1994). In addition to the psychological stressors that would be evident in match-play, the nature of the laboratory protocol is such that many activities inherent in competitive play are not included. Utility movements such as running with the ball and contact situations have been suggested to increase the physiological load (Takarado, 2003). Furthermore, according to Willford *et al*. (1998), the irregular intermittent profile of match play is further complicated by its multidirectional nature. “Unorthodox” modes of running such as backward or lateral running have an increased physiological cost and this load increases disproportionately as a function of movement speed (Willford *et al.*, 1998).

BIOPHYSICAL MEASURES

ELECTROMYOGRAPHY

QUADRICEPS MUSCULATURE

For the purpose of this discussion the results of the vastus medialis and vastus lateralis will be discussed together. Expectedly, the percentage change in iEMG from baseline measures increased significantly with an increase in speed (Mizrahi *et al,* 1997; Pincivero *et al*., 2000).

The control protocol elicited higher EMG activity levels in both the vastus medialis and vastus lateralis. Results recorded at 180s, for the vastus medialis (Fig 6, pg 63) showed that the control protocol elicited significantly higher iEMG activity at half time and on completion of the protocol. The increase in iEMG activity was an indication that the muscle was required to produce a greater muscular output in order to achieve the same workload (Greig *et al*., 2006). There was no significant change in the iEMG activity of the

vastus lateralis at all measurement intervals (Fig 7, pg 64). Girard *et al.* (2009) found larger decrements in iEMG activity of the vastus medialis (-12%) than the vastus lateralis (-5%) as a result of a squash specific activity; the decrements during tennis specific activity have also been shown to elicit greater fatigue in the vastus medialis compared to the vastus lateralis (Girarad *et al*., 2008). This suggests that the role of the vastus lateralis in controlling walking and an intermittent activity is similar.

The gait cycle during walking has been shown to require a great contribution from the quadriceps musculature, particularly during the loading and mid-stance phases (Perry, 1992). Therefore the increase in vastus medialis iEMG activation may be attributed to the increased contribution of the quadriceps musculature in controlling the gait cycle during the control condition. Numerous previous publications have suggested that fatigue results in decreased frequency of iEMG activity (Girarad *et al*., 2008; Girarad *et al*., 2009). In contrast, other researchers have found EMG activity to increase as a product of fatigue (Psek and Cafereli, 1993; Mizrahi *et al*., 1997; Yeung *et al.*, 1999). Data recorded in the present study suggest that iEMG activity of the quadricep musculature increased as a result of fatigue.

HAMSTRING MUSCULATURE

Bicep Femoris

Expectedly, muscle activity increased as a function of speed (Mizrahi *et al,* 1997; Pincivero *et al*., 2000). Additionally, the iEMG activity of the biceps femoris was higher for the experimental condition at all time intervals (Fig 8, pg 65). This suggests higher activation of the bicep femoris as a consequence of the intermittent protocol (Psek and Cafereli, 1993; Mizrahi *et al*., 1997; Yeung *et al.*, 1999). However, during both protocols, there were no changes in BF muscle activity over time.

At 180s during half time and at the end of the experimental protocol iEMG BF activity levels were significantly higher than during the control protocol. This suggests that the

bicep femoris was required to produce greater muscular output to achieve the same standardised workload following an intermittent activity profile (Greig *et al*., 2006). The variation in iEMG activity of the bicep femoris observed during the experimental condition is a direct result of the load placed upon the hamstring group by the frequency of acceleration (Greig *et al.,* 2006). This increase in iEMG activity was particularly prevalent when sprinting (180s) due to the hamstring complex being required to work harder to produce the same running speed (Greig *et al*., 2006). Due to the repeated eccentric demands of acceleration and deceleration in the intermittent protocol, the increase in iEMG activity may be attributed to the unique activation strategies of eccentric contractions (Enoka, 1996). These differences in neural recruitment patterns between eccentric and concentric contractions may be related to the modulation of the relative excitability within the motorneurones innervating a muscle (Enoka, 1996). Therefore the differences in excitability as a product of fatigue may have resulted in large increases in iEMG activity during the experimental protocol. This may predispose the hamstrings to injury as the increased activity may create the apparent inhibition of the quadriceps group (Grieg *et al*., 2006). Consequently, mechanical fatigue may be more important than physiological fatigue with respect to the incidence of injury during the latter stages of a rugby game.

iEMG activity post the experimental protocol was marginally lower than the pre- and half time values. Rahnama *et al*. (2006) and Greig *et al*. (2006) have previously reported that iEMG activity of the bicep femoris decreased as a function of time. This reduction in iEMG activity toward the latter stages of the game supports epidemiological data reporting an increased incidence of hamstring injuries recorded in the latter stages of match play (Brooks *et al*., 2005). The reduction on iEMG activity may be attributed to the fact that there is a redistribution of muscular work and this may lead to an increase in the likelihood of muscular strain due to altered kinematics of movement (Greig *et al*., 2006). Pinniger *et al*. (2000) observed altered kinematics and iEMG activity in sprinting following a repeated dynamic hamstring fatiguing task. Sprinting has been identified as a primary mechanism of rugby injury (Brooks *et al*., 2005) and therefore the altered kinematics as a result of fatigue highlight the mechanical implications of injury incidence.

Semitendinosus

EMG activity of the semitendinosus (Fig 9, pg 66) at 180s pre-protocol was significantly higher for the experimental condition than the control condition. However, post the intermittent condition the control protocol elicited significantly higher iEMG activity levels at 120s and 180s. This is in contrast to the findings of the bicep femoris, despite bicep femoris and semitendinosus both belonging to the same muscle complex. This lack of uniformity of iEMG activity changes in the hamstring musculature has been highlighted before (Pincivero *et al.,* 2000). This difference may be a direct consequence of semitendinosus being primarily active during the terminal swing and the bicep femoris being active during the loading phase, early swing and terminal swing during a normal gait cycle. It can be suggested that due to these very different activity levels during walking, similar differences are found during running.

During the experimental protocol iEMG activity during the sprinting phase decreased significantly ($p<0.05$) as a function of time; this decrease may be an indicator of fatigue (Rahnama *et al*., 2006; Grieg *et al*., 2006) and was similar to the decrease in bicep femoris activity over time, albeit greater. In contrast, the iEMG activity of the continuous protocol stayed fairly constant over time. This may indicate that fatigue of the semitendinosus occurred as a result of the intermittent activity profile. Therefore, rapid acceleratory and decceleratory movements may cause fatigue in the semitendinosus muscles in rugby players and may predispose them to injure this muscle.

ISOKINETIC STRENGTH

QUADRICEPS – EXTENSORS

Peak torque

The quadriceps were stronger eccentrically than concentrically which was to be expected considering it is well documented that muscles are stronger eccentrically than

concentrically (Enoka, 1996; Faulkner, 2003). Prior to the start of testing, peak concentric and eccentric torque (221.24±47.32 Nm and 254.69±57.86 Nm) were similar to those recorded in university soccer players (Rahnama *et al*., 2003). Rahnama *et al.* (2003) measured values of 216 (±36) Nm and 223 (±40) Nm for concentric and eccentric torque respectively. In contrast, in another soccer-specific investigation by Greig (2008) lower values for peak concentric strength (182±31Nm) of the extensors were found compared to the values recorded on the current sample. This large difference (39.24 Nm) may be attributed to the small sample size used or the differences in body mass. Grieg (2008) used players with an average body mass of 77.1 (±8.3) kg; whereas the current cohort was approximately 10 kg heavier with a mean body mass of 87.2 (±10.9) kg. Increased body mass has been shown to have a positive effect on force production (Bennell *et al*., 1998).

In the present study, concentric and eccentric quadriceps strength was maintained, within statistical limits, throughout both protocols (Fig 10, pg 67). The experimental findings are similar to those of Greig (2008) who found that concentric and eccentric quadriceps strength did not decrease due to soccer-specific fatigue. In contrast, a 10-13% decrease in torque production of the knee extensors has been associated with a 2 - hour tennis- specific activity (Girard *et al*., 2008). This difference may be attributed to the increased duration of the activity bout, or the different tennis- specific activity profile.

Work

Work done by the concentric and eccentric quadriceps was similar over time (Fig 11, pg 67) for both protocols with the eccentric extensors eliciting higher peak values (242.15±58.29 J) compared to the concentric extensors (204.59±50.48 J). Therefore, on average, concentric work was 234.51% of body mass while eccentric work was 277.57%. In contrast, Siqueira *et al*. (2002) found that work done as a percentage of body weight was 310.5% for the concentric extensors in runners and 344.2 % for jumper athletes; unfortunately eccentric values were not recorded. These higher values may be attributed to the fact that the runners and jumpers perform different activities for training

and the athletes were professional athletes, whereas players used in the current study were university- level athletes.

Work capacity of a muscle is determined by calculating the total area under the torque-position curve. Total work is an indicator of fatigue and therefore a predictor of injury (Eston and Reilly, 2001). As the work done by the concentric and eccentric extensors did not change over time throughout both protocols it is an indicator that fatigue of the quadriceps was not heavily induced by the stop-start nature of the protocol.

Power

In the current study, power produced by the concentric and eccentric extensors did not vary as a product of time for both protocols (Fig 12, pg 68). This implies that neither protocol impaired the rate at which the quadriceps musculature could produce force. Importantly, Siqueira *et al*. (2002) suggests that power should be measured at high velocities ($240^{o}.s^{-1}$) so it is probable that had this study used higher speeds, power may have been shown to decrease.

HAMSTRINGS – FLEXORS

Peak torque

Baseline measures for the concentric flexors were similar for both protocols (142.56$\pm$34.70 Nm for the control and 144.67$\pm$26.24 Nm for the experimental). In contrast the pre-protocol values for the eccentric flexors varied slightly between protocols (167.28$\pm$30.21 Nm and 176.83$\pm$26.79 Nm for the control and experimental protocol respectively). Eccentric flexor strength was greater than concentric strength which was an expected finding (Enoka, 1996; Faulkner, 2003; Rahnama *et al*., 2003).

Concentric hamstring strength was maintained throughout both protocols with similar results recorded for the control and experimental conditions (Fig 13, pg 69). Again, this

has been shown to occur during soccer activity (Grieg, 2008). In contrast to the concentric response, eccentric hamstring strength decreased significantly ($p<0.05$) during the experimental protocol (Fig 13, pg 69) with post values (162.89±30.66 Nm) being significantly lower than the pre-protocol measures (176.83±26.79 Nm). Soccer-specific research has shown that eccentric hamstring strength decreased as a function of exercise duration (Greig, 2008; Grieg and Siegler, 2009). The time history of eccentric hamstring strength decrements supports epidemiological data, with hamstring injuries more likely to occur within the latter stages of a rugby game (Wekesa *et al*., 1996; Devlin, 2000; Petersen and Hölmich, 2005). It is proposed that this greater incidence of injuries may be attributed to altered coordination, technique or concentration. Alternatively, these injuries may be due to physiological changes within the fatigued muscle (Woods *et al*., 2004). The dual innervations of the biceps femoris are a risk factor for injury due to the poor coordination allowing separate parts of the muscle to be activated at separate times (Woods *et al*., 2004). This asynchrony may be a result of fatigue or due to irritation or damage along the path of the muscle supplying the force (Devlin, 2000). This induced fatigue and increased susceptibility to injury may be a result of inefficiencies produced by a poorly coordinated running style leading to a lack of muscular control of the pelvic girdle which can contribute to overuse and repetitive strain of the hamstrings (Devlin, 2000).

Eccentric hamstring strength was not restored to pre-protocol levels during the half time interval. For example, at 40 minutes, peak eccentric torque of the hamstrings was 173.89 (±33.30) Nm compared to the start of 176.83 (±26.79) Nm. This is in accordance with Greig (2008). In contrast, concentric levels at the half time interval (144.05±30.55 Nm) were restored to starting levels (144.67±26.24 Nm). Eccentric contractions show force loss immediately after exercise and a recovery period that is longer (up to 24 hours post -exercise) than that associated with a concentric protocol (Clarkson and Hubal, 2002). The largest decrement in strength loss and the most prolonged recovery times are associated with high-force eccentric exercise (Saxon *et al*., 1995). Kellis and Baltzopoulos (1995) found that recovery was delayed after eccentric training in comparison to that of concentric training. This suggests that the muscle function that

accompanies muscle damage results in under-performance and the inability to perform at the required levels (Byrne *et al*., 2004). Eccentric muscle contractions have unique neural activation patterns which may result in delayed recovery (Enoka, 1996). This increased recovery time may lead to further decrements in strength loss during the second half of the intermittent protocol or a rugby game and thereby places the individual at greater risk of musculature injury at this time.

The decline in eccentric hamstring strength following the experimental protocol may be attributed to the greater contribution of the hamstrings in controlling the intermittent activity profile (Greig and Siegler, 2009; Greig *et al.,* 2006). The hamstrings function eccentrically to slow hip flexion and knee extension during acceleration and deceleration (Williams, 1985). Thompson *et al.* (1999) reported that multiple-sprint sports, such as Rugby Union, have shown severe eccentric muscle damage which is associated with impaired muscle function due to the rapid acceleratory and decceleratory movements. Sprinting has previously been observed as the primary mechanism of injury as the frequency of speed changes places greater emphasis on the acceleration and deceleration phases of running, thereby increasing the contribution of the hamstring musculature (Woods *et* al., 2004; Mann *et al.*, 1986). Thus, the stop-start nature of the experimental protocol, with a large emphasis placed on the sprinting demands, probably resulted in eccentric muscle damage which was evident in the perceived pain ratings (Fig 19, pg 73 and Fig 20, pg 74) and therefore impaired the ability of the hamstrings to produce force. The increased muscle contribution of the bicep femoris in maintaining running mechanisms during the intermittent condition, in parallel with the decreased eccentric strength, may further increase the risk of injury.

Work

Work done by the concentric flexors was 176.52 % of body mass which is less than that recorded for non-athletes (187%), jumper athletes (226.2%) and runners (206.5%) (Siqueria *et al*., 2002). This may be attributed to the fact that the players used in this study were not professional athletes like the jumper athletes and runners used by

Siqueria *et al*. (2002). Interestingly, the subjects used in the current study had a similar value to non-athletes. This similarity to non-athletes may be attributed to greater eccentric demands during Rugby Union as a result of the stop-start nature and therefore concentric strength may not be as important as the demands have shown not to result in changes in concentric strength (Fig 14, pg 70). Unfortunately eccentric values were not measured by Siqueria *et al*. (2002).

Work done by the eccentric flexors decreased significantly as a result of time during the experimental protocol (181.56±38.60 J pre-protocol to 159.61±32.44 J post-protocol) (Fig14, pg 67). In contrast, the work done during the control protocol increased slightly from 176.16 (±40.63) J to 180.78 (±38.78) J. Further, the post-protocol value of the experimental condition was significantly lower than that of the control. As a decrease in work done can be used as an indicator of fatigue (Eston and Reilly, 2001), it is evident that the stop-start nature of the protocol induced eccentric fatigue of the flexors which may predispose these muscles to injury (Wekesa *et al*., 1996; Petersen and Hölmich, 2005). Both fatigue and poor eccentric strength have been shown to be precursors of muscular injuries (Brooks *et al*., 2005 and Devlin, 2000).

Power

The power produced by the concentric and eccentric flexors was similar in both conditions and did not vary over time (Fig 15, pg 71). This is in contrast to the results of Glaister (2005) who reported power loss as a result of multiple sprint work. The fact that the results of the current study were not significant may be attributed to the fact that the speed used ($60^{o}.s^{-1}$) was more suitable to record accurate strength measurements rather than power output.

QUADRICEP TO HAMSTRING RATIO

Baseline quadriceps to hamstring ratio (Q:H) measured for each condition was 0.71 and 0.75 (Fig 16, pg 71). This is considered to be within the normal range (Aagaard *et al.*,

1998). These values are greater than those reported on by Greig (2009) in professional soccer players where baseline values of 0.57 were recorded. Rosene *et al*. (2001) collected data from university soccer and volleyball players and found the average Q:H ratio to be 0.49 (±0.11). The higher ratios in this study may indicate that the current sample group had stronger hamstring strength due to the added emphasis on strength training in Rugby Union and/or due to the added emphasis of eccentric demands during the rugby activity profile and therefore included in their training stimulus (Nicholas, 1997). The strength indices of concentric strength were used to investigate strength imbalances; these have previously been reported as a risk factor for hamstring injuries (Woods *et al*., 2004; Rosene *et al*., 2001). As concentric strength of both the knee flexors and extensors was not significantly impaired during both protocols, the strength ratio was maintained. These findings are in accordance with soccer-specific literature where the Q:H did not vary over time as a result of a soccer-specific activity profile (Greig, 2009). It has been suggested that players are at substantially greater risk of hamstring strains when the Q:H is less than 0.61 (Bernell *et al*., 1998). The current study recorded Q:H ratios all greater than 0.68 and therefore neither protocol placed players at a greater risk of hamstring injuries. Despite a functional Q:H ratio being suggested to be a better indicator of functionality (Rosene *et al*., 2001) , the traditional ratio was used as there is little research on the functional ratio.

PERCEPTUAL RESPONSES

CENTRAL AND LOCAL RPE

"Central" ratings of perceived exertion were higher than the "Local" ratings throughout both protocols (Fig 17, pg 72). Post-protocol ratings of "Central" and „Local" RPE during the experimental condition were similar with the "Central" being rated as 14.6 and "Local" as 14.4. Therefore subjects found the intermittent condition to be equally taxing on the cardiovascular and muscular systems. However, during the control protocol subjects rated "Central" RPE as 11.9 and "Local" as 9.6 post-protocol. Consequently

subjects perceived the continuous condition to be more taxing on the cardiovascular system rather than the muscular system.

"Central" RPE values were significantly ($p<0.01$) higher during the experimental condition (mean of 14) than the control condition (mean of 10) at all time intervals. Therefore, subjects perceived the intermittent experimental protocol to be more taxing on the cardiovascular system than the steady-state control protocol. During the control protocol "Central" RPE increased slightly as a result of the exercise duration. A post-protocol value of 11.9 was recorded. However, during the experimental condition, the "Central" RPE rating increased significantly to 14.6 as a result of the exercise. Doing soccer-specific research, Drust *et al*. (2000) reported similar "Central" RPE ratings on completion of the experimental protocol (15) and which were significantly different to their control protocol (12) which was continuous in nature.

There was a larger variation in the perceived effort placed on the cardiovascular system. This may be attributed to a greater variation in cardiovascular fitness levels amongst the team as some individuals participated in other sports whilst others did not. This is supported by the large variation in the baseline multi-stage fitness test results (10.5 ± 2.1 levels).

"Local" RPE stayed similar at all time intervals during the control protocol, however, ratings increased significantly over time during the experimental condition (from 11.11 to 14.44). The intermittent condition elicited significantly higher "Local" RPE ratings at all time intervals compared to the control condition. Local RPE of the hamstring musculature specifically was significantly higher during the experimental condition (13) than the control condition (0). This may be attributed to the increased muscular demands, particularly eccentric demands, required to accelerate and decelerate the body during an intermittent activity profile (McLean, 1992; Duthie *et al*., 2005). Epidemiological data state that muscular injuries are more likely to occur during the latter stages of a rugby game (Brooks *et al*., 2005).

The post-protocol “Central” (14.6) and “Local” (14.4) RPE values recorded during the experimental condition were similar to those recorded after the three competitive rugby matches assessed prior to the experimental set up. During actual competition the mean “Central” RPE was 14.8 and “Local” was 15.1. Therefore, subjects perceived the experimental protocol to be as taxing on the cardiovascular system as a competitive Rugby Union match. The “Local” RPE values recorded during the protocol were slightly lower than the values recorded after a competitive match. This may be attributed to the contact nature of the competitive match. Subjects were asked to try and distinguish between bumps and bruises from the contact and just rate muscular pain, but the perceived pain from the contact situations may have influenced the results.

BODY DISCOMFORT

The control protocol induced most discomfort in the quadriceps which may be attributed to the active role of the quadriceps during the gait cycle while walking (Perry, 1992). Overall however, the experimental condition elicited a greater number of reports of body discomfort. These were predominantly to the quadriceps (17) and the hamstrings (18) (Fig 18 and 19, pg 73). The quadriceps musculature discomfort was rated the same: three out of ten, for both the control and experimental condition (fig 20, pg 73). Therefore, despite the increase in intensity during the experimental protocol, subjects recorded the same discomfort in the quadriceps. This may be attributed to the fact that the intermittent protocol did not place any added strain on the quadriceps musculature as the hamstrings were more heavily involved in controlling the intermittent activity profile (Williams, 1985; Greig *et al.,* 2006; Greig and Siegler, 2009).

Of main interest is the incidence of hamstring discomfort during the intermittent protocol (100%) and the intensity of rating: five out of a maximal ten (Fig 20, pg 74). Therefore the hamstrings were perceived to be the most taxed muscle during the intermittent activity profile. As hamstrings were not rated during the control protocol it can be assumed that the discomfort felt can be attributed to the intermittent activity profile and not due to the distance covered, as this was the same for both protocols.

EXERCISE INDUCED MUSCLE SORENESS

There was no evidence of DOMS following the control protocol. In contrast, the experimental protocol elicited DOMS in all 18 subjects. DOMS was evident within 24 hours post the experimental condition which is a well -established time-course of effect (Byrne *et al*., 2004). The maximum rating on day 2 was 5 and was associated with the verbal anchor of “uncomfortable to walk”. After day 3 muscle soreness started to subside and had dissipated 6 days post the experimental protocol (Fig 21, pg 75). These findings support those of Armstrong (1984) and Nosaka (2002) who suggest that DOMS peaks within 24 to 72 hours after the exercise bout, and typically dissipates within 7 to 10 days after the exercise bout. The hamstring musculature was the muscle which experienced the most muscle soreness highlighting the role of this musculature in the activity profile of rugby.

The presence of DOMS suggests structural damage to the muscle and connective tissue induced during eccentric activity (Cheung *et al*., 2003). This was to be expected, particularly in the hamstring musculature, due to the presence of repeated eccentric contractions as a result of the rapid acceleratory and decceleratory movements, specifically that of the biceps femoris (Greig *et al.,* 2006; Greig and Siegler, 2009).

Exercising with the presence of DOMS may place athletes at risk of injury as athletes might compensate by making significant alterations to and reductions in performance and often a less than optimal training intensity (Cheung *et al*., 2003). Unfortunately due to the schedule of a rugby season players are required to play weekly competitive games as well as training practices, and therefore players are often required to partake in exercise whilst experiencing some form of muscle soreness which places them at risk of injury.

MULTIVARIATE ANALYSES

ISOKINETIC STRENGTH AND PERCEIVED DISCOMFORT

There was a weak relationship (R^2=0.43) between perceived hamstring discomfort post the experimental protocol and decreased eccentric flexor strength. Therefore, in 9.7% of cases, perceived hamstring discomfort will indicate a reduction in eccentric hamstring strength. Hence, the use of hamstring discomfort as an indicator of reduced eccentric hamstring strength is limited. Unfortunately, this relationship has not been explored by existing literature and therefore due to the weak correlation, no conclusions can be drawn. However, further exploration into this area may be of interest as it would be a useful tool to use within a game setting in order to prevent injury.

HEART RATE AND "CENTRAL" RATING OF PERCEIVED EXERTION

Early studies have demonstrated strong correlations between central ratings of perceived exertion and heart rate during dynamic continuous exercise (Nicholas *et al*., 2000). The intermittent protocol in this study also demonstrated a strong relationship between "Central" RPE and heart rate, (R^2=0.99) (Fig 22, pg 76). This shows that the subjects‟ ratings of central perceived exertion accurately reflected their physiological responses to intermittent exercise. Further, the players were accurate in their rating of perceived exertion and were aware of the physiological strain due to the activity profiles. Despite this, it has been suggested that ratings of perceived exertion be used carefully (Noakes, 1992).

ELECTROMYOGRAPHY AND ISOKINETIC STRENGTH

It has been suggested that fatigue, typical of intermittent sports, such as Rugby Union, leads to a reduction in muscle activity due to the duration, magnitude and type of contraction. It is likely that a decrease in muscle activity will cause a reduced muscle torque during repeated isokinetic efforts (Rahnama *et al*., 2003; Rahnama *et al*., 2006).

Thus, it would be expected that there will be a reduced muscle torque as muscle activity decreases due to fatigue. The current study found a moderately strong (R^2= 0.64) relationship between changes in iEMG activity and isokinetic strength of the eccentric flexors. This is in accordance with the existing literature assessing soccer-specific fatigue (Rahnama *et al*., 2003; Rahnama *et al*., 2006). This suggests that as the muscles fatigue in response to an intermittent activity profile, iEMG activity decreases which leads to decreased isokinetic strength. Therefore, the significant ($p<0.01$) decrease in muscle activity of the semitendinosus and the progressive decline in muscle activity for the bicep femoris, in comparison with quadricep iEMG, would imply an earlier and more pronounced weakening of the knee flexor strength. This is supported by the isokinetic data collected in the current study.

CONCLUSION

The control protocol did not impact significantly on the physiological, biophysical and perceptual responses. However, in contrast, the rugby-specific intermittent activity profile elicited significant changes in all three areas of responses measured.

Heart rate increased significantly and iEMG activity of the semitendinosus decreased significantly as a response to the intermittent condition. iEMG of the bicep femoris was significantly higher in the experimental condition at all time intervals but also demonstrated a trend towards decline over time in the intermittent protocol. These results indicate that physiological and muscular fatigue was induced by the stop-start nature of the protocol. There was a significant reduction in eccentric strength and work done by the flexors following the intermittent condition providing further evidence of fatigue.

Players perceived the intermittent condition to be significantly more taxing on the cardiovascular and musculoskeletal systems of the body than the control protocol. During the experimental protocol players perceived the most body discomfort in the hamstrings, which was closely followed by the quadriceps. The values recorded during

the experimental condition were similar to those recorded as a result of a competitive Rugby Union match; this suggests that the laboratory protocol closely matched the demands of competitive play. Muscle soreness was evident only after the experimental condition where subjects reported soreness up to 5 days following the protocol.

CHAPTER VI

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

INTRODUCTION

Whilst Rugby Union is an intermittent game which involves many physical contact situations, this study focussed particularly on the „stop-start" nature of the game. The study also focussed specifically on the demands placed on the inside back players who spend the largest percentage of the game sprinting and who complete the most sprints compared to any other position (Duthie *et al*., 2005). Rugby Union is characterised by "stop-start" movements - rapid acceleration, used in sprinting, and deceleration, in order to brake the movement of the body and stay „on-sides", which results in repeated eccentric demands being placed on the lower limb musculature(McLean, 1992; Duthie *et al*., 2005). Eccentric contractions and the associated muscle damage have been linked to impaired muscle function and a predisposition to injury (Friden *et al*., 1983; Friden *et al*., 1981). The primary objective of this study was to identify changes in muscle activity, muscle strength as well as physiological and perceptual responses during a simulated rugby-specific intermittent laboratory protocol and to compare this to a continuous protocol of consistent intensity.

SUMMARY OF PROCEDURES

The physical load imposed on rugby players" was assessed within competitive games and using existing literature (Duthie *et al*., 2005). This data was used to inform the experimental activity profile. Demographic, anthropometric, previous injury history and resting cardiovascular data were obtained prior to experimentation. Subjects were required to attend a baseline testing session in order to quantify initial levels of fitness. These initial sessions provided an opportunity to explain the experimental procedures and habituate all players to the treadmill, isokinetic dynamometer and the laboratory set.

For the experimental phase, subjects were required to attend two randomized testing sessions within six to eight days of each other. On arrival subjects were fitted with a heart rate monitor. The non-dominant leg was cleaned with alcohol and shaved in order to ensure good conductivity. EMG electrodes were then attached and all positions measured from an anatomical marker to ensure future placement was identical.

Subjects were then required to complete a warm-up which included some stretching and running at slow speeds. Baseline isokinetic data was then recorded using a CYBEX 6000 isokinetic dynamometer. The differing protocols were randomised in administration. Three repetitions were recorded on both legs at 60deg.s^{-1} with a rest period of 30s between each set.

Following this, baseline EMG activity was measured during a normalising treadmill protocol at three speeds (12, 15 and 21km.h^{-1}). This data was processed in order to provide integrated EMG (iEMG) which was then used as reference data with which to compare the half time and post-protocol iEMG. Immediately after this was completed, players completed either the 80- minute rugby-specific intermittent laboratory exercise protocol or the continuous control protocol. During both protocols heart rate was recorded throughout and „Central" and „Local" Ratings of Perceived Exertion (RPE) and body discomfort were recorded at 20 -minute intervals. After 40 minutes, during the „half time break", subjects performed the iEMG measurement protocol and the isokinetic dynamometer protocol. The subjects then performed the second 40- minute exercise protocol after which the iEMG measurement and isokinetic dynamometer protocols were completed for the final time. Subjects were required to complete a delayed- onset muscle soreness (DOMS) diary for the following seven days following both conditions.

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

Physiological variables:	Heart rate
Biophysical variables:	Electromyography and isokinetic strength measures

Perceptual variables:	„Central" and „Local" RPE, body discomfort and perceived muscle soreness

SUMMARY OF RESULTS

Similar anticipatory heart rates were recorded prior to both protocols. Heart rate increased with exercise and was significantly ($p<0.01$) higher at each time interval for the experimental protocol than the control condition.

There were no changes in iEMG activity of the vastus medialis or vastus lateralis over time in either protocol or between protocols. In contrast, iEMG activity of the bicep femoris was significantly ($p<0.05$) greater during the half time interval and at the end of the experimental protocol compared to the continuous activity profile. The semitendinosus elicited significantly ($p<0.05$) greater iEMG muscle activity at the end of the control condition. Both the biceps femoris and semitendinosus activity decline over time in the experimental protocol although only the changes in semitendinosus was significant.

Isokinetic peak torque, work and power of the concentric and eccentric quadricep musculature did not vary over time during either protocol. Likewise, concentric hamstring strength, work and power were unchanged throughout both protocols. In contrast, eccentric hamstring strength decreased significantly ($p<0.05$) as a result of the intermittent activity profile and in comparison on the control protocol. The work done by the eccentric flexors did not vary during the continuous condition: however, work done post the experimental protocol was significantly ($p<0.05$) decreased over time and less than the post-control levels. Power produced by the eccentric hamstrings remained unchanged over time and as a result of condition. The Quadriceps:Hamstring ratio remained constant during both conditions.

"Central" and "Local" RPE remained unchanged during the course of the control condition. In contrast , perceived "Central" exertion ($p<0.05$) and "Local" exertion

($p<0.01$) increased significantly over time during the experimental protocol. There was a strong relationship between "Central" RPE and heart rate ($R^2=0.99$) during the experimental protocol. Therefore, as heart rate increased, there was an almost linear increase in "Central" RPE ratings of perceived exertion accurately reflecting physiological responses to exercise. During the experimental condition there were many more reports of body discomfort: 46 in total, in comparison to the nine reported during the control condition. The highest rating of discomfort in the experimental condition was the hamstring musculature. There were no cases of hamstring discomfort reported during the control protocol. The level of perceived discomfort was lower in the control condition than the experimental protocol.

Following the control condition there were no reports of delayed- onset muscle soreness (DOMS). In contrast, all players reported some form of pain and discomfort following the intermittent activity profile. Pain peaked at day two and had subsided six days post the experimental protocol.

STATISTICAL HYPOTHESES

The first hypothesis, 1, focussed on the physiological response, heart rate; and the results force the rejection of the null hypothesis as heart rate responses increased significantly from rest to exercise and there was a significant difference in heart rate recorded for the experimental condition and the control condition. However, it is tentatively retained over time in the control protocol, as heart rate remained constant once a „steady-state" had been achieved.

The second hypothesis, 2, focussed on muscle activity. Muscle activity of the hamstring musculature are significantly different between the experimental and control protocol. The activity levels of the semitendinosus were significantly reduced over time, as a result of the intermittent condition. These results force the null hypothesis to be rejected as there were significant differences between the protocols. However, it is tentatively accepted, as the control protocol did not elicit changes in muscle activity. In addition the

muscle activity of the quadricep musculature was not altered as a result of condition or over time in both protocols.

Hypothesis 3 (a) proposed that there would be no difference in concentric peak torque, work and power between protocols and over time. As there was no change in the mentioned strength parameters the null hypothesis is accepted.

In contrast, hypothesis 3 (b) proposed that there would be no difference in eccentric peak torque, work and power between protocols and over time. As eccentric strength and work of the flexors decreased significantly over time and was significantly different between protocols, the null hypothesis is rejected for the hamstring musculature. However, as eccentric strength, work and power of the extensors did not vary over time or as a result of protocol, the hypothesis is tentatively accepted for the quadricep musculature.

Hypothesis 4 is rejected as there are significant differences in “Central” and “Local” RPE between the two conditions and there are significant changes over time during the experimental protocol. And, therefore, the null hypothesis is rejected. However, it is tentatively accepted as there is no change during the control condition.

Hypothesis 5 is rejected as there are significant differences in muscle soreness post the experimental and control protocol. However, there was no difference on day 6 and 7 post protocol; therefore the hypothesis is tentatively accepted in those instances.

CONCLUSION

Unfortunately there is no existing literature that has quantified time-motion analysis on university-level rugby players. In order to improve on the applicability of the study it is important that the time-motion data used to inform the experimental protocol is the same playing level as the subjects used.

The continuous condition led to no changes in the physiological or biophysical measures over time. In contrast, the intermittent condition resulted in significantly increased physiological cost and changes in the biophysical responses. The rugby-specific intermittent exercise profile used elicited no changes in the biophysical variables measured of the quadriceps musculature. However, the protocol educed a significant decrease in eccentric hamstring peak torque and work whilst altering EMG activity of the bicep femoris and semitendinosus. This change in eccentric hamstring strength and muscle activity places the hamstring musculature at greater risk of musculoskeletal strain and injury (Grieg *et al*., 2006).

The intermittent protocol was perceived to be significantly more taxing of the cardiovascular and musculoskeletal systems with subjects rating „Central" and „Local" RPE much higher during the intermittent condition. Body discomfort of the hamstrings was the more reported area of discomfort and rated the highest during the experimental condition. This emphasises the increased demands placed upon the hamstring musculature during an activity profile which is intermittent in nature compared to a continuous protocol.

The large load placed on the eccentric hamstring musculature in response to a rugby-specific intermittent protocol, which is characterised by rapid acceleratory and decceleratory demands, is a risk factor for all players. In order to decrease the risk of injury it may be suggested that players spend more time training their hamstrings eccentrically in order to develop strength and improve fatigue ratios in order to become accustomed to the demands experienced during match play.

RECOMMENDATIONS

In order to decrease the prevalence of hamstring injuries within Rugby Union, further research needs to be done to understand the implications of repeated eccentric contractions within a Rugby Union context so that more conclusive answers can be found. This will facilitate the understanding of hamstring injuries and allow preventative strategies and rehabilitative procedures to be developed which will hopefully decrease the occurrence and recurrence of injuries within Rugby Union.

The intermittent activity profile required subjects to perform rapid acceleration and deceleration, as found in a rugby game. Therefore, subjects were required to perform rapid eccentric contractions. Eccentric contractions have been linked to muscle damage and swelling. This swelling of the muscle as a result of the eccentric demands may have influenced the conductivity of the surface electrodes. Surface EMG is known to be influenced by many factors which can interfere with conductivity of the electric activity. Therefore, it may be suggested that a more accurate method of measuring EMG be used, such as needle EMG, although this, too, may not be practical. Hence, one must take into account the limitations of EMG recordings.

Acknowledging the difficulties associated with field-based studies, especially those in a sporting and Rugby Union environment, more research *in situ* is required in order accurately to assess the physiological, biophysical and perceptual demands placed on the players. This will aid in the understanding of musculoskeletal injuries which occur within the game. Due to the limitations of field- based studies it may be more realistic to incorporate contact situations, by using tackle bags, and multi-directional running into a laboratory protocol. It may be a more accurate reflection of the simulated protocol if it is completed outside on a field where environmental factors will play a role in the demands placed on the player.

It is advisable to study a larger sample size in order to allow for a greater generality of the findings and a better statistical significance. A more homogenous group of subjects

as regards playing position should be recruited in order to strengthen the validity of these findings. Thus, as the activity profile was based on time-motion data of the inside backs, further studies should focus just on the inside back position in order to lessen variance due to the effects of an unaccustomed playing profile.

Players were acutely aware of the cardiovascular strain placed on their bodies and this was reflected in the very strong correlation (R^2= 0.99) of heart rate and RPE. Unfortunately, players were not aware of the musculoskeletal strain imposed on the body as a result of an intermittent activity profile which is shown in the weak relationship (R^2= 0.43) between perceived hamstring discomfort and eccentric strength decrements. Therefore it may be important to educate players on musculoskeletal strains and the pain and perceptions associated with them in order to decrease the likelihood of injury.

REFERENCES

Note: Asterisked citations * are secondary sources. These were not directly consulted and are referenced as fully primary sources, indicated in brackets, permit.

Aagaard P, Simonsen EB, Magnusson P, Larsson B and Dyhre-Poulsen P (1998). A new concept for isokinetic hamstring:quadriceps Muscle strength ratio. **American Journal of Sports Science and Medicine**, 26(2): 231-237.

Agre JC (1985). Hamstring Injuries, proposed aetiological factors, prevention and treatment. **Sports medicine**, 2: 21-33.

Abbiss CR and Laursen PB (2005). Models to explain fatigue during prolonged endurance cycling. **Sports Medicine**, 35(10): 865-898.

American College of Sports Medicine (ACSM‟s) Guidelines for exercise testing and prescription (2006). **Lippincott Williams and Wilkins**.

Armstrong RB (1984). Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. **Medicine and Science in Sports and Exercise**, 16: 529-538.

Avela J, Kryöläinen H, Komi PV and Rama D (1999). Reduced reflex sensitivity persists several days after long-lasting stretch-shortening cycle exercise. **Journal of Applied Physiology**, 86: 1292-1300.

Bangsbo J (2004). Physiology of soccer – with special reference to intense intermittent activity. **Acta Physiology of Scandinavia (supplement 619)**, 151: 1-155.

Bathgate A, Best JP, Craig G and Jamieson M (2002). A prospective study of injuries to elite Australian Rugby Union players. **British Journal of Sports Medicine**, 36: 265-269.

Bennell K, Wajswelner H, Lew P, Schall-Riaucour A, Plant D and Cirone J (1998). Isokinetic strength testing does not predict hamstring injuries in Australian Rules footballers. **British Journal of Sports Medicine**, 32: 309-314.

Boobis LH, Williams C and Wootton SA (1983). Human muscle metabolism during brief maximal exercise. **Journal of Physiology**, 42: 471-475.

Borg GAV (1970). Perceived exertion as an indicator of somatic stress. **Scandanavian Journal of Rehabilitative Medicine,** 2: 92-98. (See Borg, 1982).

Borg GAV (1982). Psychophysical bases of perceived exertion. **Medicine and Science in Sports and Exercise,** 14 (5): 377-381.

Bottini E, Poggi EJT, Luzuriaga F and Secin FP (2000). Incidence and nature of the most common rugby injuries sustained in Argentina (1991-1997). **British Journal of Sports Science**, 34:94-97.

Bowers EJ, Morgan DI and Proske U (2004). Damage to the human quadriceps muscle from eccentric exercise and the training effect. **Journal of Sports Sciences**, 22:1005-1014.

Brooks LHM, Fuller CW, Kemp SPT and Reddin DB (2005). Epidemiology of injuries in English professional Rugby Union: part 2 training injuries. **British Journal of Sports Sciences**, 39: 767-775.

Brooks JHM, Fuller CW, Simon PT, Kemp MD and Reddin DB (2006). Incidence, risk, and prevention of hamstring injuries in professional Rugby Union. **The American Journal of Sports Medicine**, 34 (8): 1297 – 1306.

Burgess TL (2009). Cardiorespiratory, Kinematic, Neuromuscular and metabolic characteristics during the recovery period after an ultramarathon race. **PhD Thesis**. MRC/UCT Unit for Exercise Science and Sports Medicine, University of Cape Town.

Burke LM and Hawley JA (1997). Fluid balance in team sports: guidelines for optimal practices. **Sports Medicine**, 24(1): 38-54.

Byrne C and Eston R (2002). Maximal intensity isometric and dynamic exercise performance following eccentric muscle actions. **Journal of Sports Sciences**, 24: 531-536.

Byrne C, Twist C and Eston R (2004). Neuromuscular Function after exercise-induced muscle damage. **Sports Medicine,** 34: 49-69.

Carlson RB, Carter JEL, Patterson P, Petti K, Orfanos SM and Noffal GJ (1994). Physique and motor performance characteristics of US national rugby players. **Journal of Sports Science**, 12: 403-412.

Charteris J, Cooper LA and Bruce JR (1976). Human Kinetics: A Conceptual Model for Studying Human Movement. **Journal of Human Movement Studies,** 2: 233-238.

Cheetham ME and Williams C (1987). High intensity training and treadmill sprint performance. **British Journal of Sports Medicine**, 21: 14-17.

Cheung K, Hume PA and Maxwell L (2003). Delayed onset muscle soreness: Treatment strategies and Performance. **Sports Medicine**, 33 (2): 145-164.

Chlebourn GS, Howell JN, Conatser RR and Giesey JJ (1998). Relationship between muscle swelling and stiffness after eccentric exercise. **Medicine and Science in Sports and Exercise**, 30: 529-535.

Clarkson PM, Nosaka K and Braun B (1992). Muscle function after exercise-induced muscle damage and rapid adaptation. **Medicine and Science in Sports and Exercise**, 24: 512-520.

Clarkson PM and Hubal MJ (2002). Exercise-induced muscle damage in humans. **American Journal of Physical Medicine and Rehabilitation**, 81: S52-S69.

Cleak MJ and Eston RG (1992). Delayed onset muscle soreness: mechanisms and management. **Journal of Sports Science**, 10: 325-341.

Coggan AR and Coyle EF (1988). Effect of carbohydrate feedings during high-intensity exercise. **Journal of Applied Physiology**, 65: 1703-1709.

Croisier L, Farthomme B, Naurois MH, Vanderthommen M and Crielaard JM (2002). Hamstring muscle strain recurrence and strength performance disorders. **American Journal of Sports Medicine**, 30(2); 199-203.

Dadebo B, White J and George KP (2003). A survey of flexibility protocols and hamstring strains in professional football clubs in England. **British Journal of Sports Medicine**, 38: 388-394.

Deutsch MU, Kearney GA and Rehrer NJ, (2007). Time-motion analysis of professional Rugby Union players during match-play. **Journal of Sports Sciences**, 25:4, 461-472.

Deutsch MU, Maw GJ, Jenkins D and Reaburn P, (1998). Heart rate, blood lactate and kinematic data of elite colts (under-19) Rugby Union players during competition. **Journal of Sports Sciences**, 16: 561-570.

Devlin L (2000). Recurrent posterior thigh symptoms detrimental to performance in Rugby Union. **Sports Medicine**, 29(4): 273-277.

Drust B, Reilly T and Cable NT (2000). Physiological responses to laboratory-based soccer-specific intermittent and continuous exercise. **Journal of Sports Sciences**, (18): 885-892.

Durandt J, du Toit S, Borresen J, Hew-Butler T, Masimla H , Jakoet I and Lambert M (2006). Fitness and body composition profiling of elite junior South African rugby players. **South African Journal of Sports Medicine**, 18 (2): 38-45.

Duthie G, Pyne D and Hooper S (2005). Time motion analysis of 2001 and 2002 super 12 rugby. **Journal of Sports Sciences**; 23(5): 523-530.

Duthie G, Pyne D and Hopper S, (2003). Applied physiology and game analysis of Rugby Union. **Sports Medicine**, 33 (13): 973-991.

Ebbeling BB and Clarkson PM (1989). Exercise-induced muscle damage and adaptation. **Sports Medicine**, 7: 207-234.

Enoka RM (1996). Eccentric contractions require unique activation strategies by the nervous system. **Journal of applied Physiology**; 2339-2346.

Faulkner JA (203) Terminology for contractions of muscle during shortening, while isometric, and during lengthening. **Journal of Applied Physiology**, 95: 455-459.

Flitney FW and Hirst DG (1978). Cross-bridge detachment and sarcomere „give" during stretch of active frog"s muscle. **Journal of Physiology**, 53: 201-216.

Fridén J and Lieber RL (1992). Structural and mechanical basis of exercise-induced muscle injury. **Medicine and science in Sports and Exercise**, 24: 521-53.

Friden J, Sjöström M and Ekblom B (1981). A morphological study of delayed muscle soreness. **Experientia** , **37**: 506-507.

Friden J, Sjöström M. and Ekblom B (1983). Myofibrillar damage following intense eccentric exercise in man. **International Journal of Sports Medicine**, 4: 170-176.

Garrett Jr WE (1996). Muscle strain injuries. **American Journal of Sports Medicine**, 24: s2-8.

Gerdle B and Karisson S (1994). The mean frequency of the EMG of the knee extensors is torque dependant both in unfatigued and fatigued states. **Clinical Physiology**, 14: 419-432.

Gill ND, Beaven CM and Cook C (2006). Effectiveness of post-match recovery strategies in rugby players. **British Journal of Sports Medicine**, 40: 260-263.

Girard O, Lattier G, Maffiuletti NA, Micallef J and Millet GP (2008). Neuromuscular fatigue during prolonged intermittent exercise: Application to tennis. **Journal of Electromyography and Kinesiology**, 18: 1038-1046.

Girard O, Micallef J, Noual J and Millet GP (2009). Alternation of neuromuscular function in squash. **Journal of Science and Medicine in Sport**, in press.

Glaister M (2005). Multiple sprint work: physiological responses, mechanisms of fatigue and the influence of aerobic fitness. **Sports Medicine**, 35 (9): 757-777.

Golden CL and Dudley GA (1992). Strength after bouts of eccentric and concentric actions. **Medicine and Science in Sports and exercise**, 24: 512-520.

Greig M (2006). Physiological and mechanical response to soccer-specific intermittent activity and steady-state activity. **Research in Sports Medicine**, 14: 29-52.

Greig M (2008). The influence of soccer-specific fatigue on peak isokinetic torque production of the knee flexors and extensors. **The American Journal of Sports Medicine**, 36(7): 1403-1409.

Greig M and Siegler JC (2009). Soccer- specific fatigue and eccentric hamstrings‟ muscle strength. **Journal of Athletic Performance**, 44(2): 180-184.

Holtzhausen LJ, Schwellnus MP, Jakoet I and Pretorius AL (2006). The incidence and nature of injuries in South African rugby players in the rugby Super 12 competition. **South African Medical Journal**, 96 (12): 1060-1265.

Hughes MD and Williams D (1988). The development and application of a computerized Rugby Union notational system. **Journal of Sports Sciences**, 6: 254-255.

James N, Mellalieu SD and Jones NMP (2005). The development of position-specific performance indicators in professional rugby union. **Journal of Sports Science**, 23: 63-72.

Jones C, Allen T, Talbot JA, Morgan DL and Proske U (1997). Changes in the mechanical properties of human and amphibian muscle after eccentric exercise. **European Journal of Applied Physiology**, 76: 21-32.

Jonhagen S, Daneels L and Asselman M (1994). Hamstring injuries in sprinters: the role of concentric and eccentric hamstring muscle strength and flexibility. **American Journal of Sports Medicine**, 22: 262-265.

Kellis E and Baltzopoulos V (1995). Isokinetic eccentric exercise. **Sports Medicine**, 19 (3): 202-222.

Kendall B and Eston RG (2002). Exercise-induced muscle damage and the potential protective role of estrogen. **Sports Medicine**, 32: 103-123.

Komi PV (1984). Biomechanics and neuromuscular performance. **Medicine and Science in Sports and Exercise**, 16:26-28.

Komi PV (2000). Stretch-shortening cycle exercise: a powerful model to study normal and fatigued muscle. **Journal of Biomechanics**, 33: 1197-1206.
Kuipers H (1994). Exercise-induced muscle damage. **International Journal of Sports Medicine**, 15:132-135.

Lieber RL and Friden J (1999). Mechanisms of muscle injury after eccentric contraction. **Journal of Science and Medicine and Sports,** 2 (3): 253-256.

Liemohn W (1978). Factors related to hamstring strains. **Journal of Sports Medicine**, 18: 71-76.

Lindstedt SL, LaStayo PC and Reich TE (2001). When active muscles lengthen: properties and consequences of eccentric contractions. **News in physiology and science**, 16:256-261.

Linnamo V, Häkkinen K and Komi PV (2003). Motor unit force levels. **Journal of Electromyography and Kinesiology**, 13: 93-101.

Linnamo V, Bottas B ad Komi PV (2000). Force and EMG power spectrum during and after eccentric and concentric fatigue. **Journal of Electromyography and Kinesiology**, 10: 293-300.

Mair SD, Seaber AV, Glisson RR and Garrett Jr WE. The role of fatigue in susceptibility to acute muscle strain injury. **American Journal of Sports Medicine**, 24(2): 137-143.

Mann RA, Moran GT and Dougherty SE (1986). Comparative electromyography of the lower limb extremity in jogging, running and sprinting. **American Journal of Sports Medicine**, 14: 501-510.

Mashiko T, Umeda T, Nakaji S and Sugawara K (2004). Position- related analysis of the appearance of and relationship between post-match physical and mental fatigue in university rugby football players. **British Journal of Sports Medicine**, 38: 617-621.

Maud PJ, (1983). Physiological and anthropometric parameters that describe a Rugby Union team. **British Journal of Sports Medicine**, 17 (1): 16-23.

McArdle WD, Katch FI and Katch VL (1996). Exercise Physiology: Energy, Nutrition and Human Performance. Fourth Edition. Baltimore: Williams and Wilkins.

McIntyre DL, Reid WD and Mckenzie DC (1995). Delayed muscle soreness: the inflammatory response to muscle injury and its clinical implications. **Sports Medicine**, 20:24-40.

McLean DA, (1992). Analysis of the physical demands of international Rugby Union. **Journal of Sports Sciences**, 10: 285-296.

Mellalieu S (2008). Science and rugby, an Editorial. **Journal of Sports Sciences**, 26 (8): 791-794.

Meyer T, Welter JP, Scharhag J and Kinderman W, (2003). Maximal oxygen uptake during field running does not exceed that measure during treadmill exercise. **European Journal of Applied Physiology**, 88: 387-389.

Mizrahi J, Voloshin A, Russek D, Verbitsky O and Isakov E (1997). The influence of fatigue on EMG and impact acceleration in running. **Basic Applied Myol.**, 7(2): 111-118.

Mjølsnes R, Arnason A and Østhagen T (2004). A 10-week randomized trial comparing eccentric vs. concentric hamstring strength training in well-trained soccer players. **Scandinavian Journal of Medicine and Science in Sports**, 14: 311–317.

Morgan DL and Allen DG (1999). Early events in stretch-induced muscle damage. **Journal of Applied Physiology**, 87: 2007.2015.

Morris A, Lussier L, Bell G and Dooley J (1983). Hamstring/quadriceps strength ratios in collegiate middle-distance and distance runners. **The Physician and Sports Medicine**, 11 (10): 71-77.

Naylor R (2007). Hamstring injuries in Provincial South African rugby players. Unpublished literature. University of Cape Town.

Nicholas CW (1997). Anthropometric and physiological characteristics of rugby union football players. **Sports Medicine**, 23 (6): 375-396.

Nicholas CW and Baker JS (1995). Anthropometric and physiological characteristics of first- and second-class Rugby Union players. **Journal of Sports Science**, 13: 15

Nicholas CW, Nuttall FE and Williams C, (2000). The Loughborough intermittent shuttle test: A field test that simulates the activity pattern of soccer. **Journal of Sports Sciences**, 18:2, 97-104.

*Nicol C, Komi PV and Marconnet P (1991). Fatigue effects of marathon running on neuromuscular performance. I. Changes in muscle force and stiffness characteristics. **Scandinavian Journal of Medicine and Science in Sports**, 1: 10-17.

Newham DJ, Mills KR, Quigley BM and Edwards RHT (1983). Pain and fatigue after concentric and eccentric muscle contractions. **Clinical sciences**, 64:55-62.

Noakes TD (1992). Lore of running. 2nd edition. Cape Town, **Oxford University Press**.

Noakes TD (2000). Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. **Scandinavian Journal of Medicine and Science in Sports**, 10:123-145.

Nosaka K, Newton M and Sacco P (2002). Delayed-onset muscle soreness does not reflect the magnitude of eccentric exercise-induced muscle damage. **Scandinavian Journal of Medicine and Science in Sports**, 12:337-346.

Nosaka K, Salamoto K, Newton M and Sacco P (2001). How long does the protective effect of eccentric muscle damage last? **Medicine and Science in Sports and Exercise**, 33: 1490-1495.

Olds T (2001). The evolution of physique in male Rugby Union players in the twentieth century. **Journal of sports sciences**, 19: 253-262.

Orchard J (1998). Recurrent Hamstring Injuries in Australian football. **Medicine and Science in Sports and Exercise**, 30; S52.

Pandolf KB (1975). Psychological and Physiological factors influencing perceived exertion. **Perceptual and Motor Skills**, 40: 563-574.

Patel TJ and Lieber RL (1997). Force transmission in skeletal muscle: from actomyosin to external tendons. **Exercise and Sports Science reviews**, 25: 322-363.

Perrin DH, Lephart SM and Weltman A (1989). Specificity of training on computer obtained isokinetic measures. **The Journal of Orthopaedic and Sports Physical Therapy**, 11 (6): 495-498.

Perry J (1992). Gait analysis: Normal and pathological functioning. **Slack Incorporated**.

Petersen J and Hölmich P (2005). Evidence based prevention of hamstring injuries in sport. **British Journal of Sports Medicine**, 39: 319-323.

Pincivero DM, Aldworth C, Dickerson T, Petry C and Shultz T (2000). Quadriceps-hamstring EMG activity during functional, closed kinetic chain exercise to fatigue. **European Journal of Applied Physiology**, 81: 504-509.

Pinniger GJ, Steele JR and Grobller H (2000). Does fatigue induced by repeated dynamic efforts affect hamstring muscle function? **Medicine and Science in Sports and Exercise**, 32 (3): 647-653.

Proske U and Allen TJ (2004). Damage to skeletal muscle from eccentric exercise. **Exercise and Sports Science Reviews**, 33: 98-104.

Proske U and Morgan DL (2001). Topical review: Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. **Journal of Physiology,** 2: 333-345.

Psek JA and Cafareli E (1993). Behavior of coactive muscles during fatigue. **Journal of Applied Physiology**, 74: 170-175.

Quarrie KL and Hopkins WG (2007). Changes in player characteristics and match activities in Bledisloe Cup Rugby Union from 1972 to 2004. **Journal of Sports Sciences**, 25(8): 895-903.

Quarrie KL, Hancock P, Waller AE, Chalmers DJ, Toomey MJ and Wilson BD (1995). The New Zealand rugby injury and performance project. III. Anthropometric and physical performance characteristics of players. **British Journal of Sports Medicine**, 29(4): 263-270.

Rahnama N, Reilly T, Lees A and Graham-Smith P (2003). Muscle fatigue induced by exercise simulating the work rate of competitive soccer. **Journal of Sport Sciences**, 21: 933-942.

Rahnama N, Lees A and Reilly T (2006). Electromyography of selected lower-limb muscles fatigued by exercise at the intensity of soccer match-play. **Journal of electromyography and kinesiology**, 16: 257-263.

Ramsbottom R, Brewer J and Williams C, (1988). A progressive shuttle run test to estimate maximal oxygen uptake. **British Journal of Sports Medicine**, 44(4): 141-144.

Ratel S, Lanzaar N, Williams CA, Beau M and Duche P (2003). Age differences in human skeletal muscle fatigue during high-intensity intermittent exercise. **Acta Pediatrics** 11: 1248-1254.

Reilly T and Brooks GA (1986). Exercise and the circadian variation in body temperature measurements. **International Journal of Sports Medicine**, 7: 358-368.

Reilly T, Secher N, Snell P and Williams C (1990). Physiology of Sports. **Spoon Press:Oxon.**

Reilly T and Borrie A (1992). Physiology of field hockey. **Sports Medicine**, 14 (1): 10-26.

Reilly T, (1997). Energetics of high intensity exercise (soccer) with particular reference to fatigue. **Journal of Sports Sciences**, 15 (3):257-263.

Roberts SP, Trewartha G, Higgitt RJ, El-Abd J and Stokes KA (2008). The physical demands of elite English Rugby Union. **Journal of Sports Sciences**, 26 (8): 825–833.

Rosene JM, Fogarthy TD and Mahaffey BL (2001). Isokinetic Hamstring:Quadriceps ratios in Intercollegiate Athletes. **Journal of Athletic Training**, 36(4): 378-383.

Sargeant AJ and Dolan P (1986). Human muscle function following prolonged eccentric exercise. **European Journal of Applied Physiology and Occupational Physiology**, 56: 704-711.

Saxton JM, Clarkson PM, James R, Miles M, Westerfer M, Clark S and Donnelly AE (1995). Neuromuscular dysfunction following eccentric exercise. **Medicine and Science in Sport and Exercise**, 27: 1185-1193.

Sayers M (2003). Running mechanisms of elite rugby players. **In proceedings of the international conference on the science and practice of Rugby**, Brisbane, Queensland.

Scott AC, Roe N, Coats AJS and Piepoli MF (2003). Aerobic exercise physiology in a professional Rugby Union team. **International Journal of Cardiology**, 87:173- 177.

Siqueira CM, Pelegrini FR, Fontana MF and Greve JM (2002). Isokinetic dynamometry of knee flexors and extensors: Comparative study among non-athletes, Jumper athletes and Runner athletes. **Rev. Hosp. Clinical Faculty of Medicine, University of Săo Paulo**, 57 (1): 19-24.

Siri WE (1961). Body composition from fluid space and density. In J. Brozek & A. Hanschel (Eds.). Techniques for measuring body composition (pp. 223-244). Washington, DC: **National Academy of Science.**

Small K, McNaughton L, Greig M and Lovell R (2008). The effects of multidirectional soccer-specific fatigue on markers of hamstring injury risk. **Journal of Sports Science and Medicine in Sport**, 13 (1): 120-125.

Smith LL (1991). Acute inflammation: the underlying mechanism in muscle soreness? **Medicine and Science in Sport and Exercise**, 23:542-551.

South African Rugby Union (2009). URL: www.saru.co.za. Last accessed 1 November 2009.

St Clair Gibson A, Broomhead S, Lambert MI and Hawley JA, (1998). Prediction of maximal oxygen uptake from a 20-m shuttle run as measured directly in runners and squash players. **Journal of Sports Sciences**, 16 (4): 331-335.

St Clair Gibson A, Lambert MI, Weston AR, Myburgh KH, Emms M and Kirby P (1998). Exercise-induced mitochondrial dysfunction in an elite athlete. **Clinical journal of Sports Medicine**,8:52-55.

St Clair Gibson A, Schabort EJ and Noakes TD (2001). Reduced neuromuscular activity and force generation during prolonged cycling. **American Journal of Physiology – Regulatory, integrative and comparative physiology**, 281: R187-R196.

Takarado Y (2003). Evaluation of muscle damage after a rugby match with special reference to tackle plays. **British Journal of Sports Medicine**, 37: 416-419.

Tesch PA, Dudley GA, Duvoisin MR, Hather BM and Harris RT (1990). Force and EMG signal patterns during repeated bout of concentric and eccentric muscle actions. **Acta Physiology of Scandinavia**, 138: 263-271.

Thompson D, Nicholas CW and Williams C (1999). Muscular soreness following prolonged intermittent high-intensity shuttle running. **Journal of Sports Sciences**, 17: 387-395.

Warren GL, Lowe DA and Armstrong RB (1999). Measurement tools used in the study of eccentric contraction-induced injury. **Sports Medicine**, 27: 43-59.

Waterman-Storer CM (1991). The cytoskeleton of skeletal muscle: is it affected by exercise? A brief review. **Medicine and Science in Sports and Exercise**, 23(11): 1240-1249.

Wekesa M, Asembo JM and Njororai WWS (1996). Injury surveillance in a rugby tournament. **British Journal of Sports Medicine**, 30: 61-63.

Welsh Rugby Union (2009). URL: www.WRU.co.uk. Last accessed 1 November 2009.

Whitehead NP, Allen TJ, Morgan DL and Proske U (1998). Damage to human muscle from eccentric exercise after training with concentric exercise. **Journal of Physiology**, 512: 615-620.

Wilhite MR, Cohen ER and Wilhite SC (1992). Reliability of concentric and eccentric measurements of quadriceps performance using KIN-COM dynamometer: the effect of testing order for three different speeds. **Journal of Orthopaedic and Sports Physical Therapy**, 15: 175-182.

Williams K (1985). Biomechanics of running. **Exercise and Sports Science Reviews**, 13: 389-441.

Williford HN, Olson MS, Gauger S, Duey WJ and Blessing DL (1998). Cardiovascular and metabolic costs of forward, backward and lateral motion. **Medicine and Science in Sports and Exercise**, 30 (9): 1419-1423.

Witvrouw E, Danneels L, Asselman P, D'Have T and Cambier D (2003). Muscle flexibility as a risk factor for developing muscle injuries in male professional soccer players: A prospective study. **The American Journal of Sports Medicine**, 31(1): 41-46.

Woods C, Hawkins RD, Maltby S, Hulse M, Thomas A and Hodson A (2004). The football association Medical Research Programme: an audit of injuries in professional football – analysis of hamstring injuries. **British Journal of Sports Medicine**, 38: 36-41.

Yeung SS, Au AL and Chow CC (1999). Effects of fatigue on the temporal neuromuscular control of the vastus medialis muscle in humans. **European Journal of Applied Physiology and Occupational Physiology**, 80: 379-385.

Yu B, Queen RM, Abbey AN, Liu Y, Moorman CT and Garrett WE (2008). Hamstring muscle kinematics and activation during overground sprinting. **Journal of Biomechanics**; 41: 3121-3126.

BIBLIOGRAPHY

Note: The following sources were consulted by the author during the conceptual growth of this dissertation. While not specifically cited, these works did play an important role in establishing the basis upon which this research was developed.

Ali A and Farrally M, (1991). A computer-video aided time motion analysis technique for match analysis. **Journal of Sports Medicine and Physical Fitness**, 31 (1): 82-88.

Ali A and Farrally M, (1991). Recording soccer players‟ heart rates during matches. **Journal of Sports Sciences**, 9: 183-189.

Aziz AR, Chia MY and Teh KC, (2005). Measured maximal oxygen uptake in a multi-stage shuttle test and treadmill-run test in trained athletes. **Journal of sports medicine and physical fitness,**45(3): 306-314.

Babault N, Pousson M, Ballay Y and van Hoecke J (2001). Activation of human quadriceps femoris during isometric, concentric, and eccentric contractions. **Journal of applied Physiology**, 91: 2628-2634.

Bales DW, Craig BN, Congleton JJ, Kerk CJ, Amendola AA, Gaines WG and Jenkins OC (2001). The influence of supporting the Oxylog instrument during a step test and heart rate in a lifting test. **Applied Ergonomics**, 367-377.

Bangsbo J, Mohr M and Krustrup P (2006). Physical and metabolic demands of training and match-play in the elite football player. **Journal of Sports Sciences**; 27(7): 665-674.

Bergh ULF, Ekblom B and Åstrand P (2000). Maximal oxygen uptake “classical” versus “contemporary” viewpoints. **Medicine and Science in Sports and Exercise**, 32(1): 85.

Brooks GA (1985). Anaerobic threshold: review of the concept and directions for further research. **Medicine and Science in Sports and Exercise**, 17(1): 22-31.

Coutts A, Reaburn P and Abt G, (2003), Heart rate, blood lactate concentration and estimated energy expenditure in a semi-professional rugby league team during a match: a case study. **Journal of Sports Sciences**, 21 (2): 97-103.

Doutreloux JP, Tepe P, Demont M, Passelergue P and Artigot A, (2002). Energy demands of playing positions in Rugby Union. **Science and Sports**, 17 (4): 189-197.

Eaton C and George K (2006). Position specific rehabilitation for Rugby Union players. Part 1: Empirical movement analysis data. **Physical Therapy in Sport** 7:22-29.

Esposito F, Impellizzeri FM, Margonato V, Vanni R, Pizzini G and Veicsteinas A, (2004). Validity of heart rate as an indicator of aerobic demand during soccer activities in amateur soccer players. **European Journal of Applied Physiology**, 93: 167-172.

Eston R and Reilly T (2001). Kinanthropometry and exercise physiology laboratory manual: Tests, procedures and data. Second edition. **Routledge**, London and New York.

Fitts RH (2004). Cellular mechanism of muscular fatigue. **Physiological Review**, 24:49-94.

Gabbett TJ (2005). A comparison of physiological and anthropometric characteristics among playing positions in junior rugby league players. **British Journal of Sports Medicine**, 39: 675-680.

Gabbett TJ, (2002). Influence of physiological characteristics on selection in a semi-professional first grade rugby league team: a case study. **Journal of Sports Sciences**, 20:399-405.

Gabbett TJ, (2004). Influence of training and match intensity on injuries in rugby league. **Journal of Sports Sciences**, 22 (5): 409-417.

Gabbett TJ, (2006). A comparison of physiological and anthropometric characteristics among playing positions in sub-elite rugby league players. **Journal of Sports Sciences,** 24(12): 1273-1280.

Gastin PB (2001). Energy system interaction and relative contribution during maximal exercise. **Sports Medicine**, 31 (10): 725-741.

Green HJ (1996). Mechanisms of muscle fatigue in intense exercise. **Journal of Sports Science**, 15: 247-256.

Hakkinen K and Komi PV (1983). Electromyography and mechanical characteristics of human muscle fatigue under voluntary and reflex conditions. **Elecroencephalogr. Clinical Neurophysiology**, 55: 436-444.

Hodge K, Lonsdale C and Ng JYY (2008). Burnout in elite rugby: Relationships with basic psychological needs fulfilment. **Journal of Sports Sciences**, 26(8): 835-844.

Jackson AS, Pollock ML (1978). Generalised equations for predicting body density of men. **British Journal of Nutrition**, 40: 497-504.

Jones Am and Doust JH (1996). A 1% treadmill grade most accurately reflects the energetic cost of outdoor training. **Journal of Sports Sciences**, 14: 321-327.

Jones DA, Newham DJ, Torgan C (1989). Mechanical influences on long lasting muscle fatigue and delayed onset pain. **Journal of Physiology**, 412:553-557.

Kemi OJ, Hoff J, Engen LC, Helgerud J and Wisloff U, (2003). Soccer- specific testing of maximal oxygen uptake. **Journal of sports medicine and physical fitness**,43: 139-144.

Knuttgen HG and Kramer WJ (1987). Terminology and measurement in exercise performance. **Journal of Applied Sports Science**, 1: 1-10.

Leger LA, Mercier D, Gadoury C and Lambert J (1988). The multi-stage 20-meter shuttle run test for aerobic fitness. **Journal of Sports Science**, 6: 93-101.

Li RCT, Wu Y, Maffuli N, Chan KM and Chan JLC (1996). Eccentric and concentric knee flexion and extension: a reliability study using the Cybex 6000 dynamometer. **British Journal of Sports Medicine**, 30: 156-160.

Meyers PT (1980). Injuries presenting from Rugby Union football. **Medicine Journal Aust.**, 12(2): 17-20.

Muller E, Benko U, Raschner C and Schwameder H (1998). Specific fitness training and testing in competitive sports. **Medicine and Science in Sports and Exercise**, 216 – 220.

Noakes TD (1997). Challenging beliefs: ex Africa semper aliquid novi. **Medicine and Science in Sports and Exercise**, 29:571-590.

Noakes TD and Durandt JJ (2000). Physiological requirements of cricket. **Journal of Sport Sciences**, 18: 919-929.

Robergs RA (2001). An Exercise Physiologist‟s “contemporary” interpretations of the “ugly and creaking edifices” of the VO_{2max} concept. **Journal of Exercise Physiology online**, 4(1).

Robinson PD and Mills SH (2000). Relationship between scrummaging strength and standard field tests for power in rugby. In: Hong Y, editor. Proceedings of XVIII International Symposium on Biomechanics in Sports; May 25-30; Hong Kong. Hong Kong: The Chinese University of Hong Kong, 980-981.

Sahlin K (1992). Metabolic aspects of fatigue in human skeletal muscle. **Medicine in Sports Science**, 34: 54-68.

Strath SJ, Bassett DR, Swartz AM and Thompson DL (2001). Simultaneous heart rate-motion sensor technique to estimate energy expenditure. **Medicine and Science in Sports and Exercise**, 2118 – 2123.

Turmilty D (1993). Physiological characteristics of elite soccer players. **Sports Medicine**, 16:80-96.

Vanderfield G (1975). Fitness testing of rugby players (abstract). **Australian Journal of Sports Medicine**, 14:14.

APPENDIX A: GENERAL INFORMATION

Letter to Subject

Subject Consent form

Injury profile

Physical activity questionnaire

Pre-test instructions

Equipment checklist

Multi-stage Fitness Test tables

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HUMAN KINETICS & ERGONOMICS
Cell: 084 704 7080 • Fax: (046) 603 8934 • E-mail: g04b1758@campus.ru.ac.za

Dear Subject

INVOLVEMENT IN MASTERS' RESEARCH:

Effect of repeated eccentric demands placed on the lower limb musculature during simulated Rugby Union play.

Firstly, I must thank you for showing interest in my research. The aim of this study is to identify changes in force production and muscular fatigue of the lower limb musculature for both eccentric and concentric contractions during a Rugby Union intermittent laboratory protocol and a control protocol, which involves continuous activity.

The testing will involve 4 sessions. During the first session I will be taking anthropometric measures, namely stature, mass and skinfolds. I will also take reference heart rate and collect baseline fitness measures.

In the second session I will ensure that you are comfortable using a treadmill, particularly at high speeds. I will familiarise you with the dynamometer and the EMG. Once you are comfortable with the protocols and equipment, we will arrange a date and time for session 3 and 4.

The third and fourth sessions will require subjects to complete both protocols, the order of which will be randomly assigned. You will do a warm-up and some stretches before commencing the protocols. Baseline isokinetic data and maximal voluntary contractions

will be performed on the dynamometer. You will be required to complete the isokinetic and EMG protocol before beginning the assigned protocol. You will complete a 40-minute protocol before doing the isokinetic and EMG protocol again. After the final 40-minute protocol you will b required to complete the final isokinetic and EMG protocol. During the 40- minute sessions you will be required to give a rating of „local" and „central" perceived exhaustion and body discomfort.

You will be required to complete the second protocol within seven to ten days of completing the first protocol.

Thank you for your interest shown and for agreeing to participate in this research protocol. If you have any questions please feel free to ask me at any time.

Yours faithfully
Lisa Brown
BSc Hons (HKE)
MSc Student

SUBJECT CONSENT FORM

I,_____ having been fully informed of the research project assessing the physiological demands of competitive rugby and the design of a treadmill training protocol to elicit these same responses,

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

I am fully aware of the procedures involved as well as the potential risks and benefits associated with my participation as explained to me verbally and in writing. In agreeing to participate in this research I waive any legal recourse against the researchers of Rhodes University, from any and all claims resulting from personal injuries sustained whilst partaking in the investigation. This waiver shall be binding upon my heirs and personal representatives. I realise that it is necessary for me promptly to report to the researchers any signs or symptoms indicating any abnormality or distress. I am aware that I may withdraw my consent and may withdraw from participation in the research at any time. I am aware that my anonymity will be protected at all times, and agree that all the information collected may be used and published for statistical or scientific purposes.

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

SUBJECT (OR LEGAL REPRESENTATIVE);

_____	_____	_____
(Print name)	(Signed)	(Date)

PERSON ADMINISTERING INFORMED CONSENT:

_____	_____	_____
(Print name)	(Signed)	(Date)

WITNESS:

_____	_____	_____
(Print name)	(Signed)	(Date)

PLAYERS' HISTORY

Name	
Position	
Age	
Years playing rugby	
Injuries: (any lower limb injuries i.e.: ankle injury, calf strain, knee injuries or quadricep or hamstring injury)	

PHYSICAL ACTIVITY SCREENING QUESTIONNAIRE

Name: _____

Subject Code: _____

MEDICAL HISTORY

Tick 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	☐ Eye Problems
☐ Peripheral vascular disorders	☐ Asthma	☐ Hypoglycemia
☐ High/low blood pressure	☐ Emphysema	☐ Diabetes
☐ Epilepsy	☐ Migraine	☐ Hyperthyroidism

☐ Other (specify) _____

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

_____

_____

_____

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

_____

_____

_____

Are there any other concerns medical or otherwise that you feel are worth mentioning?

_____

_____

_____

Please indicate any prescribed or over-the-counter medication you are currently taking or have taken in the last 6 months.

_____

_____

_____

OTHER HABITS

Please tick the 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. Tick the appropriate block (s) and fill in the number of sessions.

☐ Jogging ____ ☐ Gym_____ ☐ Touch_____ ☐ Rugby_____

☐ Fitness ____ ☐ Swimming_____ ☐ Other _____

PRE-TEST INSTRUCTIONS

Please inform the researcher of any factors that you think may influence your results on the day of testing, for example if you are taking prescription medication, are asthmatic, or are ill. Note that if you are injured or have any quadriceps, hamstring, knee or any lower limb problems, it is advised that you do not participate in this study. In order for my results to be accurate, we require you to follow certain instructions before completing the test:

FOR 24 HOURS PRIOR TO TESTING:

- **Do not drink alcohol.**
- **Do not participate in strenuous exercise.**
- **Do not take medication (such as painkillers, aspirin, flu tablets etc) unless absolutely necessary.**
- **Try to get at least 8 hours of sleep the night before the test.**

ON THE DAY OF TESTING:

- **Eat a good meal about 2 hours before the test.**
- **Do not eat anything for 1.5 hours prior to testing.**

Please try to comply with these rules as it will help me greatly in my data collection. I appreciate your cooperation.

EQUIPMENT CHECKLIST

1. Cybex on and working
2. EMG batteries charged
3. Razor
4. Electrodes
5. 2 stopwatches
6. Treadmill working
7. RPE chart
8. Body discomfort chart
9. Fiximul tape
10. Data sheets
11. Heart Rate monitor
12. Tape measure

MULTI-STAGE FITNESS TEST VO_{2MAX} TABLES

In the table below locate the Level and Shuttle you achieved in the test to find your predicted VO_{2max} value.

Level	Shuttle	VO2 Max
4	2	26.8
4	4	27.6
4	6	28.3
4	9	29.5
Level	**Shuttle**	**VO2 Max**
6	2	33.6
6	4	34.3
6	6	35.0
6	8	35.7
6	10	36.4
Level	**Shuttle**	**VO2 Max**
8	2	40.5
8	4	41.1
8	6	41.8
8	8	42.4
8	11	43.3
Level	**Shuttle**	**VO2 Max**
10	2	47.4
10	4	48.0
10	6	48.7
10	8	49.3
10	11	50.2
Level	**Shuttle**	**VO2 Max**

Level	Shuttle	VO2 Max
5	2	30.2
5	4	31.0
5	6	31.8
5	9	32.9
Level	**Shuttle**	**VO2 Max**
7	2	37.1
7	4	37.8
7	6	38.5
7	8	39.2
7	10	39.9
Level	**Shuttle**	**VO2 Max**
9	2	43.9
9	4	44.5
9	6	45.2
9	8	45.8
9	11	46.8
Level	**Shuttle**	**VO2 Max**
11	2	50.8
11	4	51.4
11	6	51.9
11	8	52.5
11	10	53.1
11	12	53.7
Level	**Shuttle**	**VO2 Max**

12	2	54.3		13	2	57.6
12	4	54.8		13	4	58.2
12	6	55.4		13	6	58.7
12	8	56.0		13	8	59.3
12	10	56.5		13	10	59.8
12	12	57.1		13	13	60.6
Level	**Shuttle**	**VO2 Max**		**Level**	**Shuttle**	**VO2 Max**
14	2	61.1		15	2	64.6
14	4	61.7		15	4	65.1
14	6	62.2		15	6	65.6
14	8	62.7		15	8	66.2
14	10	63.2		15	10	66.7
14	13	64.0		15	13	67.5
Level	**Shuttle**	**VO2 Max**		**Level**	**Shuttle**	**VO2 Max**
16	2	68.0		17	2	71.4
16	4	68.5		17	4	71.9
16	6	69.0		17	6	72.4
16	8	69.5		17	8	72.9
16	10	69.9		17	10	73.4
16	12	70.5		17	12	73.9
16	14	70.9		17	14	74.4
Level	**Shuttle**	**VO2 Max**		**Level**	**Shuttle**	**VO2 Max**
18	2	74.8		19	2	78.3
18	4	75.3		19	4	78.8
18	6	75.8		19	6	79.2
18	8	76.2		19	8	79.7
18	10	76.7		19	10	80.2
18	12	77.2		19	12	80.6
18	15	77.9		19	15	81.3

Level	Shuttle	VO2 Max	Level	Shuttle	VO2 Max
20	2	81.8	21	2	85.2
20	4	82.2	21	4	85.6
20	6	82.6	21	6	86.1
20	8	83.0	21	8	86.5
20	10	83.5	21	10	86.9
20	12	83.9	21	12	87.4
20	14	84.3	21	14	87.8
20	16	84.8	21	16	88.2

APPENDIX B: DATA COLLECTION

Order of procedure

Experimental Protocol

Rating of Perceived Exertion Scale

Rating of Perceived Exertion Explanation Sheet

Body discomfort Map and Scale

Data collection sheets

ORDER OF PROCEDURE:

1. Welcome subject
2. Fill in injury history
3. Fit heart rate belt
4. Explain protocol and equipment
5. Fit electrodes – measure and take down values
6. Turn on EMG
7. EMG baseline protocol
8. Isokinetic baseline measures
9. 40-min protocol
    a. 20-min HR, RPE and BD.
    b. 40-min HR, RPE and BD.
10. EMG protocol
11. Isokinetic protocol
12. 40-min protocol
    a. 20-min HR, RPE and BD.
    b. 40-min HR, RPE and BD.
13. EMG protocol
14. Isokinetic Protocol

EXPERIMENTAL PROTOCOL

time (s)	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
04:50	Walk
05:00	Stand
05:11	Walk
05:21	Jog
05:25	Stand

time (s)	Activity
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
10:50	Stand
10:59	Walk
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

time (s)	Activity
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
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

time (s)	Activity
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
20:20	Stand

RATING OF PERCEIVED EXERTION

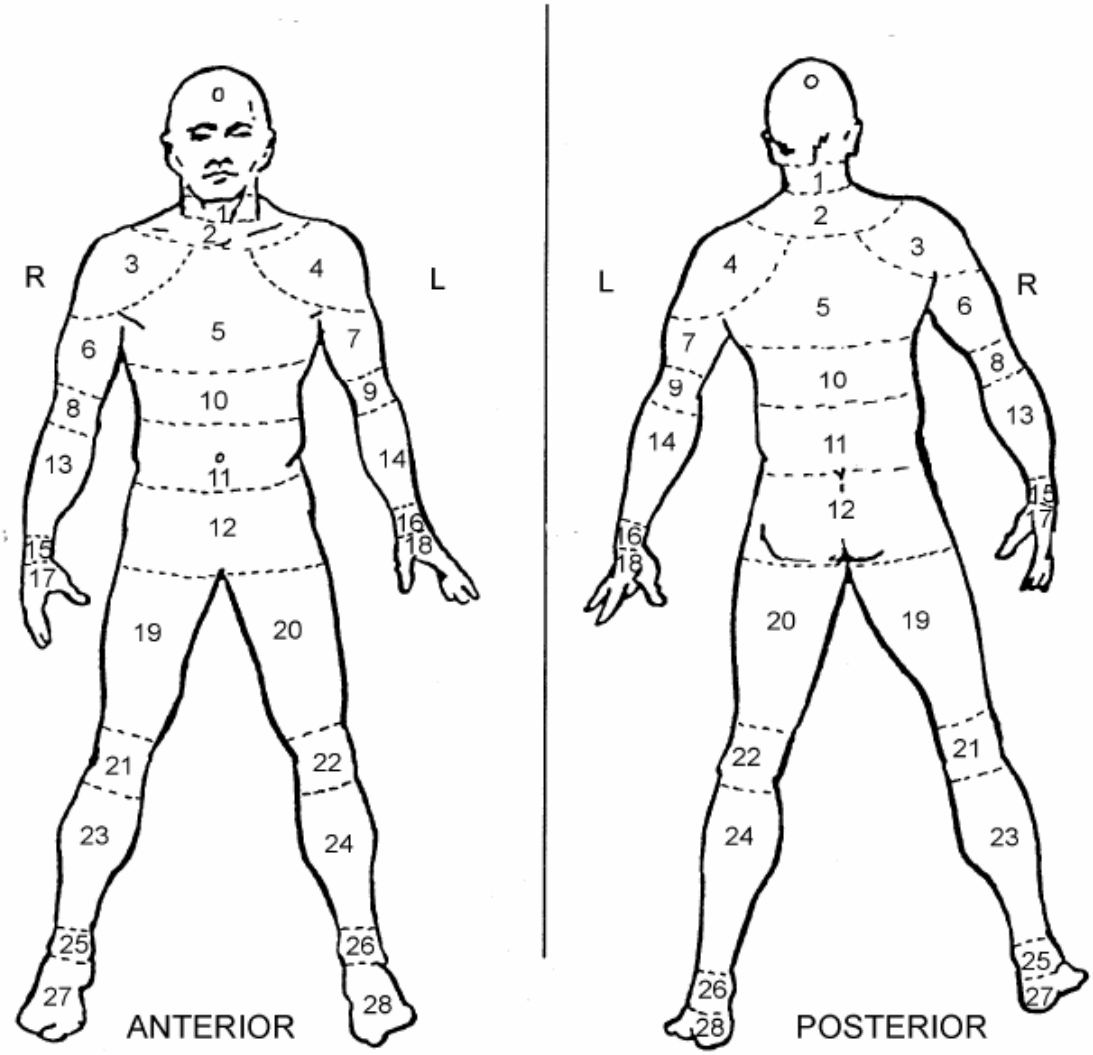
Numerical	Verbal
6	
7	Very Very Light
8	
9	Very Light
10	
11	Light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very Hard
18	
19	Maximal

INSTRUCTIONS TO SUBJECT FOR RPE

You will be required to complete 2 different protocols. During these tests you will be asked to estimate how hard you feel you are working; therefore a degree of perceived exertion will be recorded. After every 20 minutes you will be asked to point to a number on the scale presented which corresponds to your rating of perceived exertion. You will be required to give a „central" rating of perceived exertion; this should correspond to how your cardio-respiratory system is feeling. You will also be required to give a „local" rating of perceived exertion; this will correspond to how your lower limb musculature is feeling. The scale is graded from a rating of 6 to 20, and should closely correspond to your heart rate at that time through a factor of ten. A rating of 6 corresponds with one"s feeling of sitting quietly, whilst 20 reflects absolute maximum exertion when you can not continue exercising. When asked to rate your level of exertion, point to the numerical value which indicates your perceived exertion.

It is important that you try to estimate honestly and as objectively as possible. Do not underestimate the degree of exertion you experience, but do not overestimate it either. Try to be as accurate as possible.

BODY DISCOMFORT MAP AND RATING SCALE



MINIMUM INTENSITY **MAXIMUM INTENSITY**

(Adapted from: Corlett EN and Bishops RP (1976). A technique for assessing postural discomfort. **Ergonomics**, 19 (2): 175-182.)

DATA COLLECTION SHEETS

Name: _____
Subject Number: _____
Dominant leg: _____

RESPONSES

Time	Heart rate	RPE		Body Discomfort
		Local	Central	
20 min				
40 min				
60 min				
80 min				

PLACEMENT OF ELECTRODES

- Electrodes 20mm between them therefore 10mm on either side of midpoint

Muscle 1: Vastus Medialis
- Distance from patella to ASIS: _____
- 20% of distance: _____
- Electrode 1: _____
- Electrode 2: _____

Muscle 2: Vastus Lateralis
- Midpoint of head of trochanter and lateral femoral epicondyle _____
- Electrode 1: _____
- Electrode 2: _____

Muscle 3: Bicep femoris
- Midpoint of ischial tuberosity to the lateral epicondyle: _____
- Electrode 1: _____
- Electrode 2: _____

Muscle 4: Semitendinosus
- Midpoint of ischial tuberosity to the medial epicondyle:_____
- Electrode 1: _____
- Electrode 2: _____

MUSCLE SORENESS

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
Muscle soreness						
Rating of soreness						
Muscles affected						

Pain Perception scale	
1	Slightly sore – sore to touch
3	Sore when contracting
5	Uncomfortable to walk
8	Constantly painful
10	Unbearable

APPENDIX C: SUMMARY REPORTS

Statistical Table

STATISTICAL TABLES

Repeated measures analysis of variance (ANOVA) of the responses of the subject during the control and experimental protocol.

Measure		Effect		p
Heart rate		Between		0.00020
		Within	Control	0.68448
			Experimental	0.00012
iEMG – Vastus Medialis		Between		0.53668
		Within	Control	0.27495
			Experimental	0.16743
iEMG – Vastus Lateralis		Between		0.74632
		Within	Control	0.06328
			Experimental	0.27131
iEMG – Biceps Femoris		Between		0.01325
		Within	Control	0.11288
			Experimental	0.18091
IEMG - Semitendinosus		Between		0.19744
		Within	Control	0.90895
			Experimental	0.01243
Quadriceps Concentric	Peak Torque	Between		0.20038
		Within	Control	0.23283
			Experimental	0.13162
	Work	Between		0.16305
		Within	Control	0.30929
			Experimental	0.06722
	Power	Between		0.93642
		Within	Control	0.79367
			Experimental	0.92375
Quadriceps Eccentric	Peak Torque	Between		0.51269
		Within	Control	0.42889
			Experimental	0.47980
	Work	Between		0.24668
		Within	Control	0.08415
			Experimental	0.76777
	Power	Between		0.08456
		Within	Control	0.15782
			Experimental	0.20353
Hamstrings	Peak Torque	Between		0.94261
		Within	Control	0.54735
			Experimental	0.97932
	Work	Between		0.88620
		Within	Control	0.79275

Concentric			Experimental	0.75480
	Power	Between		0.96272
		Within	Control	0.37567
			Experimental	0.26174
Hamstrings Eccentric	Peak Torque	Between		0.00057
		Within	Control	0.135541
			Experimental	0.00001
	Work	Between		0.00987
		Within	Control	0.42988
			Experimental	0.00047
	Power	Between		0.11157
		Within	Control	0.25189
			Experimental	0.20353
Quadriceps:hamstring ratio		Between		0.47821
		Within	Control	0.65842
			Experimental	0.87592
„Central" RPE		Between		0.00349
		Within	Control	0.53361
			Experimental	0.00285
„Local" RPE		Between		0.0003
		Within	Control	0.5196
			Experimental	0.0001
Muscle Soreness		Between		0.0000
		Within	Control	0.9875
			Experimental	0.0001

Note: Significant differences found over the duration of the study were discussed in the Chapter V.