# Is Playerload related to fatigue following different types of repeated maximal sprint exercises in recreationally active young men?

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# Abstract

**Introduction:** GPS units are commonly used by sports clubs to estimate the "external load" [e.g. Catapult Playerload$^{TM}$ (PL)] experienced by the athlete during exercise to predict fatigue response, despite a lack of evidence for a relationship between PL and exercise fatigue. The aim of this study was to (i) compare PL and fatigue during and after two different maximal sprint protocols and (ii) examine relationships between PL and fatigue/recovery.

**Methods:** Eighteen healthy, recreationally active, males (age: 20.7 ± 3.0 yr; height: 168 ± 41 cm; body mass: 73.7 ± 11.0 kg) performed either 15 x 30m maximal sprints (SP, n=11) or 15 x (3 x 10m) maximal shuttle sprints (3T, n=7) with 90 s rest between repetitions and 180 s rest after repetition five and 10. PL, heart rate (HR) and rate of perceived exertion (RPE) were measured for the duration of the exercise. Maximum isometric voluntary contraction knee extensor/flexor torque (MVC), range of motion (ROM), quadriceps/hamstring muscle soreness (algometer and visual analogue scale, VAS), (in)voluntary rate of torque development (RTD) and *vastus lateralis/biceps femoris* long head EMG were measured during knee extension/flexion pre, immediately post and 48 h post exercise. Voluntary muscle activation (VA) and the torque-frequency relationship (TFR) were measured in the hamstring muscle group using electrical stimulation at the same time points. Blood samples were drawn from the antecubital vein pre, post and post 48 hr completion of the protocols

**Results**: PL (126.8 ± 13.8 *vs.* 107.6 ± 12.7 AU; P<0.05), HR (163.4 ± 5.2 *vs.* 149.6 ± 4.6 bpm; *P*=0.01) and RPE (13.0 ± 0.3 *vs.* 12.0 ± 0.6 AU; *P*=0.02) were all higher in SP vs. 3T. PL correlated positively with HR ($R^2$=0.497) and RPE ($R^2$=0.570) in both protocols combined. There was a main effect for time for hamstring MVC, quadriceps MVC, creatine kinase concentration, interleukin-6 concentration, lactate, range of motion, RTD and TFR (P≤0.04) but there were no group x time interactions for these variables. There was a group x time interaction for soreness, with SP exhibiting higher VAS values at post and

post 4h hr compared to 3T ($P \leq 0.04$). Moreover, PL did not correlate with changes in MVC, RTD, ROM, TFR or soreness in either protocol or in both protocols combined ($P > 0.05$).

**Conclusions:** Our data demonstrate that two different types of repeated maximal sprinting exercises lead to a similar impairment of quadriceps and hamstring muscle function. Post exercise measures of fatigue do not differ between repeated 30 m straight line sprinting and repeated 3 x 10 m shuttle sprinting, except for subjective measures of muscle soreness, which were higher after straight line sprinting compared to shuttle sprinting. PL was positively related with physiological load during the exercise but did not correlate with changes in indices of central or peripheral fatigue. Therefore, our data support the use of GPS units to predict physiological load during maximal sprinting but not to predict rate of recovery following these types of maximal exercise.

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**Declaration**

I declare that the work contained in this thesis is entirely my own.

**List of Abbreviations**

**ATP –** Adenosine triphosphate

**BF** – *biceps femoris*

**BFLH** – *biceps femoris long head*

**BM** – *Body mass*

**BMI** – Body mass index

**$Ca^{2+}$ -** Calcium

**CAF** – Central activation failure

**CK** – Creatine kinase

**CNS** – Central nervous system

**DOMS** – Delayed onset muscle soreness

**E-C** – Excitation contraction coupling

**EIMD** – Exercise induced muscle damage

**EMG** – Electromyography

**FMS** – Functional movement screen

**GI** – Glycaemic index

**GPS** – Global positioning system

**HR** – Heart rate

**IL-6** – Interleukin-6

**IKD** – Isokinetic dynamometer

**ITT** – Interpolated twitch technique

**MU** – Motor unit

**$M_{Max}$** –Maximum evoked muscle action potential

**MVC** – Maximum voluntary contraction

**PL** – PlayerLoad$^{TM}$

**RF** – *rectus femoris*

**RPE** – Rating of perceived exertion

**RTD** – Rate of torque development

**SP**- sprint protocol

**ST** - *Semitendinosus*

**TGS** – Total genotype score

**TH** – tryptophan hydroxylase

**VL** – *Vastus lateralis*

**VM** – *Vastus medialis*

**3T** – Shuttle run protocol

# Contents

# Chapter 1

## Aims and objectives

The overarching aim of the thesis is:

***To investigate if PlayerLoad™ is related to fatigue following soccer specific exercise in recreationally active young men.***

This aim has been achieved through a literature review and the completion of a study.

The objective of the literature review is

1) To provide an overview of fatigue and the models used to explain the phenomenon.
2) To explore the physical demands of soccer and the fatigue present following soccer performance
3) To examine the current methods used to monitor fatigue in soccer with special reference to global positioning system derived PlayerLoad™.

The objectives of the study are:

1) To compare fatigue responses and PlayerLoad™ between two repeated sprint exercise
2) To examine the relationship between measures of fatigue and PlayerLoad™

# Chapter 2

## A literature review on fatigue in soccer

### Introduction

During exercise, skeletal muscles produce force, which is transmitted via the tendons to the bones to generate movement. This cannot continue indefinitely, as the onset of neuromuscular fatigue reduces the effectiveness of the muscle to produce force, which in turn, reduces the capacity to continue exercising. During exercise, neuromuscular fatigue is best described as a reduction in maximal voluntary muscle force (Gandevia, 2001) in the presence of increased perception of effort (Enoka and Stuart, 1992), the effects of which can be reversed with rest. Fatigue manifest as a large number of symptoms depending on the mode, intensity and volume of exercise performed, as well as substrate availability, training status, hydration status and environmental conditions (Bergström et al., 1967, Fitts, 1994, Chin and Allen, 1997, Karelis et al., 2002, Shei and Mickleborough, 2013). While the exact mechanisms leading to fatigue remain largely undetermined (Shei and Mickleborough, 2013), it is generally accepted that neuromuscular fatigue can be classified as either “central” or “peripheral” in origin.

Central fatigue occurs proximal to the neuromuscular junction and encompasses factors such as mental exhaustion (Marcora et al., 2009, Kayser, 2003), reduced neural drive (Rampinini et al., 2011, Amann et al., 2008, Gandevia et al., 1996), internal motivation, neural transmission, motor unit recruitment (Bigland-Ritchie et al., 1978, De Luca et al., 2009, Garland and Miles, 1997, Pascoe et al., 2014, Johnson et al., 2004, Person and Kudina, 1972, Bigland‐Ritchie and Woods, 1984) and decreased psychomotor performance (Connell et al., 2016). Peripheral fatigue includes the factors that occur distal to the neuromuscular junction and related to disturbances to biochemical

homeostasis within the muscle**.** These include, but are not limited to, disturbances in electrolyte levels within the muscle (Fitts, 1994, Cairns and Lindinger, 2008, Maughan et al., 1994, Maughan and Leiper, 1995), reduced ATP concentration, increased inorganic phosphate concentration, reduced phosphocreatine (Edwards, 1981, Enoka and Stuart, 1992, Fitts, 1994), muscle glycogen depletion (Krustrup et al., 2006, Bergström and Hultman, 1966), dehydration (Armstrong et al., 1985), impaired $Ca^{2+}$ release from the sarcoplasmic reticulum (Lee et al., 1991, Allen et al., 1989) and changes in muscle pH (Gladden, 2004, Gleeson, 1996, Sahlin et al., 1976, Sahlin and Henriksson, 1984). As well as having an immediate effect on performance, these changes can impair physical and mental performance in the subsequent days following the fatiguing exercise.

Perhaps the most performance limiting factor in the days following exercise is the damage induced to the working skeletal muscle. Structural damage to the sarcomeres can occur as a result of prolonged exercise of sustained high intensity, unaccustomed movements, or exercise containing high amounts of eccentric contractions (Allen et al., 2008, Byrne et al., 2004, Proske and Allen, 2005). Exercise induced muscle damage (EIMD) is a post exercise component of fatigue that can result in muscle soreness (Talag, 1973), strength loss (McCartney et al., 1983), reduced range of motion around a joint (Cleak and Eston, 1992) and reduction in tendon compliance (Proske and Morgan, 2001). Further exercise prior to full recovery will result in a severe reduction in physical performance capabilities and may carry an increased risk of injury (Dupont et al., 2010).

Fatigue, and in particular EIMD, are common symptoms following competitive soccer match play due to its frequency of explosive actions and highly demanding nature (Bangsbo et al., 1996, Mohr et al., 2003, Varley and Aughey, 2013). This is particularly problematic for top-level soccer teams during the competitive season, as matches can occur every 3-4 days, leaving insufficient time to fully recover (Ispirlidis et al., 2008, Fatouros et al., 2010, Mohr et al., 2016). This is further complicated by the need to train in preparation for the next game, which will also hinder the recovery process. Moreover, there will be differences in post-match fatigue between players, likely due to a combination of the natural

variances in the individual response to exercise (Manzi et al., 2010, Erskine et al., 2010a) and the significant differences in physical actions performed in match play between playing positions (Bloomfield et al., 2007, Varley and Aughey, 2013). Understanding the differences in physical demands required to perform squad roles effectively, and the fatigue response of individuals is vital for the development of individualised training and recovery programmes that accurately replicate match demands (Reilly, 2005). This understanding will also help minimise injury prevalence, as underlying fatigue can be a risk factor for injury (Dupont et al., 2010, Small et al., 2010, Mair et al., 1996). Due to the impracticality of performing controlled laboratory tests in a professional environment, soccer clubs often have to employ simpler, possibly less reliable/valid methods of assessing fatigue in their squad. Recent studies have found that muscular injuries, and in particular hamstring injuries, are the most common type of injury in soccer (Ekstrand et al., 2009, Hägglund et al., 2005). Not only do they occur more frequently in match play but have gradually increased in frequency over the last 13 years as intensity of top level matches has increased (Croisier et al., 2008, Ekstrand et al., 2016, Bush et al., 2015). This suggests that current methods used to both assess and manage players' response to fatiguing exercise may be lacking/ineffective, leading players to experience insufficient recovery or preparation for match play, therefore leading to increased risk of muscle injury.

## Aims and scope of the review

The complexity of fatigue has led to conflicting evidence regarding the most effect way to measure and monitor fatigue, even more so in professional soccer club environments. This review will briefly cover some of the main models to explain fatigue, and discuss the physiological mechanisms underpinning the short and long-term effects of fatigue associated with soccer, as well as the methods currently used to monitor fatigue in soccer. As suggested by (Abbiss and Laursen, 2007), the term "fatigue" can have a different meaning across scientific disciplines and the correct context is important.

In this review, the term fatigue will be used to describe a physiological symptom caused by exercise, which contributes to reduced performance related to team sports. Therefore, themes relating to clinical fatigue and non-exercise related fatigue will be excluded from the scope of this review.

**Key themes**

Soccer, fatigue, central fatigue, peripheral fatigue, exercise-induced muscle damage, external load, monitoring fatigue, GPS, PlayerLoad$^{TM}$.

## Physical demands of soccer

Professional soccer is a highly demanding intermittent sport that has been shown to elicit high levels of fatigue, taxing both the aerobic and anaerobic energy systems (Rampinini et al., 2011). Elite players are required to cover 9-12 km, maintain an average $VO_2$max of 70%, and maintain a peak average heart rate of 86% during a match (Bangsbo et al., 1996, Bangsbo et al., 1991, Reilly and Thomas, 1976, Mohr et al., 2005, Castellano et al., 2015, Datson et al., 2017). Due to the intermittent nature of the game, players perform a purposeful action every 5 seconds (Mohr et al., 2003), which over the course of the match equates to over 1400 purposeful movements (Mohr et al., 2003, Reilly, 2003) over 200 high speed runs (Mohr et al., 2003), over 700 changes of direction (Bloomfield et al., 2007) and 19 maximal sprints (Rienzi et al., 2000) at an average distance of 20 m (Di Salvo et al., 2007). These actions also require sharp accelerations and decelerations, which have also been shown to be physically demanding tasks (Cavagna et al., 1971, Di Prampero et al., 2005, Magalhães et al., 2010). Players also perform jumps, tackles, contacts with opponents and ball striking actions, which further increases the physical demand of the match (Bangsbo, 1993). Many of these actions require eccentric contractions, which has the potential to elicit muscle damage (Byrne et al., 2004, Howatson and Milak, 2009, Rampinini et al., 2011). It should also be noted that there are numerous external factors that can affect the physical demand of a match, including playing standard, weather, level of opposition, match situation, playing style and even geographical location of league (Bloomfield et al., 2007, Lago-

Ballesteros and Lago-Peñas, 2010, Reilly and Thomas, 1976, Di Salvo et al., 2007, Lago-Peñas and Lago-Ballesteros, 2011) .

Perhaps the most important external factor influencing physical demand of a soccer match is playing position. Bloomfield et al. (2007) examined total purposeful movements during a game for each playing position and found that central defenders (2.5 ± 1.3 %) spend significantly less time sprinting in a match compared to both midfielders (6.4 ± 3.1% ) and attackers (5.5 ± 3.3%). Although, more specifically it was shown fullbacks (402 ± 165m), wingers (446 ± 161m) and forwards (404 ± 140m) covered significantly more distance at high speed than central defenders (215 ± 100m) and central midfielders (248 ± 116m). However central midfielders have been shown to cover significantly more total distance than both defenders and forwards (Di Salvo et al., 2007) and have less recovery time between intense activities (Bradley et al., 2009). Whilst all positions showed declines in high intensity efforts in the second half of the game, this was more evident for central defenders and forwards (Bradley et al., 2009). Wide midfielders were also shown to perform more dribbles with the ball in a match, which was shown to elicit higher blood lactate, perceived exertion and increased energy cost than running without the ball at the same speed (Reilly and Ball, 1984). Positional differences were also found for total number of turns and type of turns completed in match play, as well as vertical jumps and tackles made (Bloomfield et al., 2007, Bradley et al., 2009, Reilly and Thomas, 1976, Di Salvo et al., 2007, Barrett et al., 2015). Given such variance in actions performed during match play, it could hypothesised that post game, players will have different levels of fatigue not only due to the natural variance in response to exercise (Manzi et al., 2010), but also because of their different playing positions demands.

## Introduction to Exercise Fatigue

Exercise-induced fatigue has long been of interest to sports scientists yet remains only partially understood (Shei and Mickleborough, 2013). Since fatigue is a limit to physical performance understanding the mechanisms behind fatigue, the time course of fatigue symptoms following exercise, and the optimal recovery process is of huge interest to athletes and coaches alike. Despite the importance of improving our understanding of fatigue, identifying the exact process that leads to muscle failure has proven extremely difficult to do. While symptoms of fatigue have been generally accepted to fall into either central or peripheral factors, which will be discussed in more detail later on, the leading cause of fatigue which contributes to muscle failure remains a mystery. For over 50 years a plethora of studies have been undertaken to identify the main site of failure during exercise, with numerous models being proposed to explain the phenomenon.

The cardiovascular/anaerobic model has the capacity of the cardiovascular system to deliver blood and oxygen to the working skeletal muscle as the main rate limiting factor in maximal exercise (Hill et al., 1924). Once the muscles demand for oxygen cannot be met, the muscle will enter anaerobiosis and will soon hit a plateau before complete failure. Time to reach this stage of "catastrophe" can be prolonged by adaptations to the cardiovascular system attained from training such as increased blood flow and muscle oxygen consumption, allowing exercise to continue. However this model has limitations that have been debated at great length (Noakes, 1998, Bassett Jr and Howley, 1997). The main limitation stems from the premise that if pumping capacity of the heart ever exceeded that limit of oxygen consumption, then the heart would be the first muscle to be effected by the oxygen deficient (Noakes, 1998). This would begin a chain of events that would lead to progressive myocardial ischaemia (Noakes, 2000) which does not occur in healthy athletes (Raskoff et al., 1976). It was therefore proposed that while cardiac output certainly determines the $VO_2$ max (Rowell, 1993), the termination of exercise must occur long before full anaerobiosis of the skeletal and heart muscle and

there may by a control mechanism in the central nervous system to limit exercise in order to prevent this (Noakes, 2000).

This led to the proposal of the central governor theory as a limiting factor of exercise (Gibson and Noakes, 2004). This model suggests physical exhaustion is not an absolute event, but is relative based on subconscious mental calculations of the requirements of exercise and feedback from the skeletal muscle and through feelings of discomfort (Ulmer, 1996). Additionally, the brain may limit the exercise intensity by controlling factors such as the amount of motor units that activated and therefore amount of muscle fibres recruited and power output potential of the muscle. This restriction of power output may additionally act as an injury prevention mechanism. A study by Palmer et al. (1994) added support to this theory by demonstrating seemingly random fluctuations in heart rate amongst trained cyclists during a 104km race. These observed changes in heart rate were not associated with geographical changes nor increases in work intensity. Gibson and Noakes (2004) suggested this was an example of a non-linear dynamic process, with heart rate being altered due to feedback from other sites of a complex system of maintaining homeostasis. It should therefore be considered that attempting to define just one site of failure at the expense of others would suggest an over engineering effect within the system. For example, a neuromuscular system that is able to maintain maximal efficiency indefinitely would be redundant if the skeletal muscle fails after a short while. Therefore fatigue which leads to exercise failure is more in line with the Complex System theory (Gibson and Noakes, 2004, Kay et al., 2001) and should be viewed as a combination of factors and not dictated by a sole symptom.

While there exists debate on which model best describes the model of fatigue, two models that describe the origin of the symptoms are widely accepted. The central and peripheral models describe if the observed reduction in a muscles ability to generate force is determined by a bio chemical change within the muscle, or from a reduction in central motor drive.

## Central fatigue

Central fatigue occurs proximal to the neuromuscular junction and encompasses factors such as reduced internal motivation, alterations in the synthesis and metabolism of central monoamines, central nervous system (CNS) transmission, motor unit recruitment and firing frequency. The inability of the CNS to activate fully is known as central activation failure (CAF), the accumulation of which is known as central fatigue. If a muscle is receiving suboptimal impulses from the CNS, then maximal force production will be unachievable. Central fatigue has been suggested to be the main contributor to drops in MVC immediately post exercise (Rampinini et al., 2011) and is associated with lower intensity (Goodall et al., 2015), long duration exercise, becoming more pronounced as exercise duration increases (Millet and Lepers, 2004)

# Motor Cortex

The motor cortex is a region of the brain that begins the process of muscle contraction by generating the neural impulses that travel along the CNS, to the motor neurons, and ultimately to the muscle fibres. Isolated single muscle maximum force production can drop by as much as 50% following repeated maximal efforts (Gandevia et al., 1998). Rapid recovery of this force occurs within 30 seconds, however full recovery may only be possible after 4-5 minutes and may even remain sub optimal beyond that (Gandevia et al., 1996). Using involuntary electrical stimulation it was demonstrated that following maximal contractions, drops in force were partly explained by reduced output from the motor cortex (Gandevia et al., 1996, Hunter et al., 2006, Kennedy et al., 2014). Additionally, using intermittent contractions for the elbow flexor, Taylor et al. (2000) showed that reductions in maximal torque occurred, but could be increased when the motor cortex was stimulated directly. These data demonstrate sub optimal output from the motor cortex as a contributing factor to reduced voluntary muscle activation, and a component of central fatigue.

## Motor Unit

The motor unit consists of a motor neuron and the muscle fibres it innervates. Motor units work in conjunction with one another to control muscle contractions; all of the motor units able to activate a single muscle are known as the collective motor pool. Motor units can control muscle force output via excitatory and inhibitory factors. This is achieved by the activation and deactivation of specific fibres and controlling the action potential at the muscle fibre (Pascoe et al., 2014). Surface electromyography is a common method used to measure the activation of motor units and an observed decreased can be interpreted as a surrogate measure of descending motor command (Billaut, 2011). A direct correlation between EMG amplitudes and power output during cycle sprints with minimal recovery has been observed (Billaut, 2011). A similar relationship exists when pace was altered during endurance exercise, highlighting changes in motor unit recruitment (St Clair Gibson et al., 2001).

Decreases in EMG were also present following repeated MVCs (Gandevia et al., 1996, Garland and McComas, 1990, Lepers et al., 2000). These decreases in motor firing rates were suggested to be due to a combination of reduced neural drive as well as any number of peripheral inhibitory feedback mechanisms such as the Renshaw cells (De Luca et al., 2009), afferent feedback from muscle spindles (Garland and Miles, 1997) and nonreciprocal inhibition of Golgi tendon organs (Heckman and Enoka, 2012). A decline in afferent excitatory input of the motor unit was the attributing factor in the observation of Johnson et al. (2004). They showed that when a contraction is held at a constant rate, a stronger excitatory input to the motor neuron was required to maintain it, which supported previous findings (Bigland‐Ritchie and Woods, 1984, Person and Kudina, 1972, Bigland-Ritchie et al., 1986, Garland et al., 1994). However, when examining a larger array of motor units (30+) more recent work has suggested that a fatigued muscle actually increases firing rate of motor units, as well as recruiting new ones (Contessa et al., 2016, De Luca et al., 2009, Adam and De Luca, 2003). When examining single motor units Contessa et al. (2016) observed a clear and consistent trend in firing adaptations, which would explain the observed apparent decrease in previous work. They demonstrated that

averaging of a smaller group of motor units firing rate would give a false indication that firing rate decreases during a contraction. It was therefore theorised that as a muscle contracts, a greater number of motor units are required to maintain force.

## Neural Drive

Evidence of suboptimal neural drive can also be assessed by direct muscle or nerve stimulation methods such as transcranial magnetic stimulation (Todd et al., 2003) or the interpolated twitch technique (Merton, 1954). These method excludes some of the peripheral factors which may influence EMG signal propagation and transmission (Farina, 2006). The observation of a superimposed twitch during a maximal contraction suggests that a voluntary activation was not maximal. This suggests the motor units are either not being recruited or are not firing enough to generate maximal force. This impairment of voluntary activation has been suggested to be the main contributor to early reductions in muscle force (Goodall et al., 2015, Marshall et al., 2014) and occur following prolonged exercise such as running or cycling (Gandevia, 2001, Millet and Lepers, 2004, Sidhu et al., 2009). More recently, it was observed following as little as 2 maximal sprints (Goodall et al., 2015) yet the exact mechanisms underpinning impairments to neural drive remain unclear (Carroll et al., 2016).

As previously mentioned, evidence of central fatigue is routinely observed following soccer performance. Using a protocol designed to match the internal and external load of soccer match play ($SAFT^{90}$) Marshall et al. (2014) demonstrated that reduced maximal knee flexor torque and rate of torque development were associated with central, and not peripheral fatigue factors. Maximal voluntary torque decreased towards the end of play periods in conjunction with reductions in surface EMG and peak to peak response to stimulation (M-Wave) in the *biceps femoris*. These data supported a study by Rampinini et al. (2011), who examined the contributions of central fatigue following match play. They demonstrated an 8 % reduction in voluntary muscle activation following simulated soccer match play. Additionally, reductions in voluntary activation were present for up to 48 h following a

competitive soccer match (Brownstein et al., 2017). These observed reductions in voluntary activation demonstrate suboptimal recruitment of motor units, and therefore demonstrate a central component of fatigue contributing to a decrease in muscle force production.

## Peripheral fatigue

### Lactate accumulation

To produce energy for muscle contractions during exercise, a process known as glycogenolysis is used to generate ATP and pyruvate. During low intensity exercise (in the presence of oxygen), pyruvate undergoes oxidation in the mitochondria of the muscle fibre to generate more ATP, however when exercise intensity is high, there is a build-up and subsequent conversion to lactate in the myoplasm (Gladden, 2004). It has previously been thought that this build-up of lactate leads to alterations in muscle pH levels and inhibits muscle performance (Sahlin et al., 1976, Spriet et al., 1987). However, more recent studies have challenged this hypothesis by identifying that muscle acidosis alone cannot cause severe fatigue (Westerblad et al., 2002, Bruton et al., 1998) with research suggesting it has little to no effect upon (Wiseman et al., 1996) muscle force and may even be beneficial (Nielsen et al., 2001). For a review see (Cairns, 2006).

The lactate threshold represents the point at which lactic acid concentration in the blood will begin to increase faster than the rate at which it can be removed. With appropriate training the blood lactate threshold becomes augmented and may be a better indicator of aerobic performance in aerobically demanding activities such as soccer than traditional $VO_{2max}$ (Helgerud, 1994). Its ability to reflect repeated high intensity activity therefore makes it a useful marker of adaptation and training status (Edwards et al., 2003, McMillan et al., 2005). Average blood lactate concentrations during soccer matches is reported between 2 to 12 mmol/L and peaks towards the end of match halves. Furthermore, values are lower in the second half compared to the first (Bangsbo et al., 1991, Ekblom,

1986, Krustrup et al., 2005, Ali and Farrally, 1991) possibly suggesting a decrease in time spent above the lactate threshold as players begin to tire. One interesting finding regarding lactate is that blood concentration is not correlated with muscle concentration suggesting it is not an accurate parameter for assessing localised lactate production (Bangsbo et al., 2007). While the theory of changes to muscle pH and lactate accumulation reducing muscle performance is an attractive prospect due to its simplicity, the reality is that numerous studies have shown accumulation of lactate does not cause significant drops in muscle performance (Bangsbo et al., 2008, Bangsbo et al., 1992, Lamb and Stephenson, 2006, Mohr et al., 2004b). Furthermore Krustrup et al. (2006) demonstrated only small changes in muscle pH that were not related with a reduction in performance. While blood lactate can certainly be used to control training prescription and recovery status, its role in the accumulation of fatigue remains unclear.

## Substrate depletion

During periods of high intensity exercise, glycogen is the main source of energy for muscle contractions (Hermansen et al., 1967). The duration and intensity of exercise will dictate which substrate is utilised for the resynthesis of ATP. Endogenous stores of substrates used to generate ATP (e.g. carbohydrate and fat) are therefore a key regulating factor of exercise capacity is substrate availability. Muscle glycogen is essential during exercise, and the availability of it can be a limiting factor during exercise. It has been shown that exercise capacity is severely reduced when muscle glycogen concentration is low, even in the presence of other substrates (Bergström et al., 1967).

The high frequency of intense actions performed during the course of a soccer match (Bloomfield et al., 2007, Krustrup et al., 2005, Bangsbo, 1993) mean that glycogen is a crucial substrate for ATP production during match play (van Loon et al., 2001, Bradley et al., 2009). Studies into muscle glycogen concentration after a soccer match demonstrate significant decreases (Krustrup et al., 2011, Krustrup et al., 2006). Krustrup et al. (2011) examined the time course over 72 hours of muscle glycogen content

in seven professional soccer players from the Danish first and second divisions. In post-match biopsies obtained from the *vastus lateralis* muscle, glycogen content was found to be (193±22 mmol kg $dw^{-1}$) which was only 43% of the pre-game concentration (449±34 mmol kg $dw^{-1}$). Levels had partially recovered at 24 hours but remained 27% lower than pre match values. Similar results were found in 31 players in the Danish fourth division: Krustrup et al. (2006) found 47% of all muscle fibres were either mostly or completely depleted of glycogen which was linked to observed decreases in sprint performance. When investigating the effects of differing pre match glycogen levels, Saltin (1972) observed that low pre match levels (<200 mmol kg $dw^{-1}$) were almost entirely depleted at half time. This will have a detrimental effect on performance as studies have linked low muscle glycogen to decreased sprint performance seen towards the end of soccer matches (Bangsbo et al., 2006, Jacobs et al., 1982, Krustrup et al., 2011).

Under normal conditions a carbohydrate rich diet would augment glycogen resynthesis, however it appears that this process is impaired following a soccer match (Krustrup et al., 2011, Bangsbo et al., 2006). This may be explained by the observations of Asp et al. (1998) that damaged fast twitch muscle fibres resynthesise glycogen slower than slow-twitch fibres. Moreover, glycogen resynthesis in muscles damaged via eccentric exercise was found to be supressed by 35% at 48 hours despite high carbohydrate consumption (Zehnder et al., 2004). The timing of ingestion can also influence the rate of replenishment, with ingestion immediately following exercise being optimal for absorption (Ivy et al., 1988). Glycogen resynthesis rate was 45% slower in the *vastus lateralis* when supplements were consumed following 2 hour delay post exercise, compared to immediate consumption (Ivy et al., 1988). As well as timing, type and amount of carbohydrate ingestion should also be considered. Amounts above usual habitual diet should be ingested to avoid decrements in future performance (Zehnder et al., 2001) and augment stores above their normal levels via a super compensation effect (Bergström and Hultman, 1966). The rate at which carbohydrates are absorbed are measured on the Glycaemic Index (GI) (Jenkins et al., 1981) with foods higher on the scale being absorbed at a faster rate (Blom et al., 1987). For optimal rates of glycogen synthesis, foods rich with high GI carbohydrates such as

glucose should be ingested in combination with fructose (low GI) (Jetjens, 2006). The time course of complete glycogen replenishment for soccer players following a match appears to be between 24 and 72 hours (Jacobs et al., 1982, Krustrup et al., 2006, Bangsbo et al., 2006).

## Exercise-induced muscle damage (EIMD)

The process of an action potential from the nervous system being converted into a mechanical response is known as excitation-contraction coupling (E-C). When $Ca^{2+}$ is released from the sarcoplasmic reticulum, cross-bridges are formed between proteins actin and myosin causing the muscle fibre to contract (Edwards et al., 1977). It was theorised that disruptions to this process occurs following eccentric exercise. Warren et al. (1993) found greater strength loss at lower frequencies of stimulation, suggesting $Ca^{2+}$ release was being suppressed. Balnave and Allen (1995) reached a similar conclusion when caffeine stimulated $Ca^{2+}$ release in eccentrically damaged single muscle fibres, highlighted failure of the E-C process, and could account for up to 43% of the reductions in observed tetanic force in mouse muscle. The reason for these observations may be alterations of the sarcoplasmic reticulum's ability to reuptake $Ca^{2+}$ (Lamb, 2002, Tupling, 2004, Allen et al., 2008). Reductions in tetanic $Ca^{2+}$ were shown immediately and up to 3 days following eccentrically damaged muscles (Allen et al., 2008). The amount of $Ca^{2+}$ stored within a fibre is essential to its function, therefore a suboptimal amount would limit exercise performance (Trinh and Lamb, 2006, Dutka et al., 2005). Further symptoms of EIMD include mechanically disrupted cell membranes, Z-disk streaming, myofibular tearing, and sarcomere damage (Raastad et al., 2010, Patel et al., 2004, Gregory et al., 2007, Koh and Escobedo, 2004, Goll et al., 2003).

Following damage to the muscle fibres there is an inflammatory response as pro-inflammatory cytokines rapidly amass to the sites of damage to begin the repair process (MacIntyre et al., 1995). Interleukin-6 precedes other cytokines in responding, highlighted by its higher production than any other, as suggested by a number of studies (Ispirlidis et al., 2008, Pedersen and Toft, 2000). This is

accompanied by swelling of the damaged muscle and the development of soreness, peaking around 48 hours post exercise, in a phenomenon known as delayed onset muscle soreness (DOMS). DOMS can be classified as a type 1 muscle strain (Gulick and Kimura, 1996), and is commonly present after eccentric muscle contractions and has therefore been regularly observed following soccer specific exercise. The magnitude of soreness is subjective and can range from slight muscle stiffness to severe movement restricting pain. The extent of soreness can be attenuated with repeated bouts of exercise (McHugh, 2003), nutritional intervention (Bryer and Goldfarb, 2006) massage therapy (Smith et al., 1994) and every effort should be taken to do so in order to optimise match day performance. Under the presence of DOMs the contractile properties of the muscle are reduced (Chen et al., 2011, Friden and Lieber, 1992), leading to the inability to perform motor skills at the desired intensity or accuracy. Hamill et al. (1991) showed that maximum ankle and knee kinematics were significantly altered during running under the influence of DOMS. These findings may be explained by the regular observation of reduced range of motion around exercise induced damaged muscles, which is caused by increased swelling associated with the acute inflammatory response to the injury (Francis and Hoobler, 1987, Francis and Hoobler, 1988, Saxton et al., 1995, Nosaka and Clarkson, 1996, Evans et al., 1986).

The structural damage to the muscle fibres is one of the most critical symptoms of fatigue following high intensity exercise. The explosive nature, body contacts, and demanding actions of soccer match play, studies have regularly demonstrated EIMD in players post-match, categorised by other markers of muscle damage. The presence of intramuscular proteins such as creatine kinase, myoglobin and lactate dehydrogenase in the blood can be markers of structural damage to muscle fibres. Creatine kinase (CK) is an enzyme found in large quantities within skeletal muscle and its presence in the bloodstream can be a marker of muscle damage, a common observation following intense activity including soccer matches (Brentano and Martins, 2011, Rampinini et al., 2015, Pohl et al., 1981, Lazarim et al., 2009). Blood CK concentration ranges from increases of 70% to 250% and peak between 24 and 72 hours post-match (Rampinini et al., 2011, Ascensão et al., 2008, Ispirlidis et al., 2008, Andersson et al., 2008). Return to baseline can take as long as 120 hours, however with professional

players regularly exercising due to training, they are found to have higher resting levels than normal (Nédélec et al., 2012).

## Hydration and temperature

Drinking fluids prior to and during in exercise is important to thermoregulate (Nielsen et al., 1971, Greenleaf and Castle, 1971), maintain plasma osmolality (Costill and Fink, 1974) and replenish electrolytes via isotonic drinks (Nielsen et al., 1986). The negative effects of both ambient temperatures and core body temperature on increased time to exhaustion have been well-documented (Febbraio et al., 1994, Galloway and Maughan, 1997, Kruk et al., 1990, Olschewski and Bruck, 1988). When exercising with body temperatures approaching 40$^{o}$C, regardless of muscle glycogen content, trained subjects time to exhaustion was significantly reduced (González-Alonso et al., 1999). Studies have also shown that in hyperthermic conditions, while under the effects of dehydration, there were increases in lactate production, reductions in cardiac output and blood flow (González‐Alonso et al., 1998, González‐Alonso et al., 1999). Dehydration also brings discomfort through the feeling of thirst (Armstrong et al., 1985, Cheuvront et al., 2003). These effects can appear after just a 2% reduction in body mass due to sweating, a common observation in soccer (McGregor et al., 1999, Edwards et al., 2007, Mohr et al., 2004a, Maughan et al., 2004), especially in hot conditions where as much as 5% body mass can be lost (Mustafa and Mahmoud, 1979). This level of dehydration has been shown to negatively influence soccer performance (Edwards et al., 2007). Post-match performance in a sport-specific fitness test showed 13-15% decrements in distance covered in participants where fluid ingestion had been prevented (Edwards et al., 2007). However, some studies have found moderate fluid loss to have no effect on anaerobic performance (Cheuvront et al., 2006, Hoffman et al., 1995) or technical ability (Hoffman et al., 1995). Nevertheless, ingestion of fluids immediately following exercise can prevent dehydration being a factor in subsequent bouts, as the time to rehydration can be as short as 6 hours (Shirreffs et al., 1996).

## Repeated Sprint Performance

Short repeated sprinting ability is an important factor in soccer and has been shown to be decisive in goal scoring situations, which influence the outcome of matches (Faude et al., 2012). Reductions in performance of sprint ability have been routinely observed not only following match play, but during later stages of halves. Using simulated soccer matches, Ascensão et al. (2008) reported significant reductions in peak torque in the quadriceps (10%) and hamstrings (15%), which remained below baseline values as long as 72 hours. This was found to impact sprint performance with 20m sprint time increasing by 7% at 30 minutes following the game, and remaining increased for up to 72 hours. Similar findings were observed in female players, with 20m sprint time and peak torque being reduced following a match, however the time course of recovery to baseline was on average only 5 hours (Andersson et al., 2008). Magalhães et al. (2010) observed 5% increases in 20m sprint times following a soccer specific exercise protocol in trained individuals. When a soccer match was used, the same group found 10% increases in sprint times which remained 5% increased 72 hours later. The discrepancy may be a result of a lack of maximal effort in a non-match play situation, participants may have been less motivated to exert maximal effort in non-competitive situations.

During competitive seasons, it is common for professional teams to be required to play three matches within 1 week (Anderson et al., 2016, Morgans et al., 2014b). This was shown to have a marked effect on repeated sprint performance following the second game (Mohr et al., 2016). Using simulated matches, performance and recovery markers were taken on each day following a 3 game week (Mohr et al., 2016). A reduction in repeated sprint performance of 2-9% was observed in the second and

third games. While this did not significantly impact average sprint speed, or frequency of rapid decelerations during the 3 matches, it did impact high intensity running distance in games 2 and 3.

## Strength loss

Reductions in lower limb maximal voluntary contraction (MVC) strength occurs due a combination of both central and peripheral fatigue factors. Reductions in MVC is common finding following soccer performance and will effect and individuals ability to perform powerful actions such as jumping, sprinting and ball striking which can influence the outcome of the match (Delextrat et al., 2010, Andersson et al., 2008, Magalhães et al., 2010).

Even amongst professional level players, lower limb MVC can decrease by as much as 17%, and can persist for up to 3 days following soccer matches (Silva et al., 2013). The repeated changes of direction, ball striking, accelerating and decelerating place significant demand on the knee flexors and extensors. Using the applied SAFT$^{90}$ protocol (a simulated soccer match protocol) Small et al. (2010) found a diminished capacity of the knee flexor muscles for generate eccentric force, decreased peak eccentric hamstring torque, and an impairment to the eccentric hamstring: quadriceps strength ratio which may be an injury risk factor (Aagaard et al., 1998). Some studies have reported greater strength loss in knee flexors compared to knee extensors (Delextrat et al., 2013, Small et al., 2010). This may explain the greater prevalence of muscle injuries in the knee flexors compared to knee extensors (Woods et al., 2004) particularly in the last 15 minutes of playing halves (Hawkins et al., 2001).

## Jump performance

Due to the frequency of jumping actions in soccer, the stretch-shortening cycle is heavily relied upon during soccer matches. Following prolonged exercise there have been observed increases in impact

force and prolonged ground contact time when jumping, due to disruptions to the stretch shortening cycle (Nicol et al., 1991, Avela and Komi, 1998). Being able to jump maximally to challenge for the ball in both offensive and defensive situations, can be the difference between a win and a loss.

Following soccer, decreases in counter movement, vertical, counter movement and drop jump performance have been observed with immediate decreases as much as 12% (Magalhães et al., 2010, Andersson et al., 2008, Krustrup et al., 2010). Jump performance following simulated or competitive soccer matches can remain ~10% decreased at 24 hrs (Magalhães et al., 2010, Andersson et al., 2008, Ispirlidis et al., 2008) and may remain ~10% decreased up to 72 hrs (Magalhães et al., 2010).

## Technical skills

Correctly performing technical actions such as passing or shooting, are required to score goals in soccer which influence the outcome of the match. Number of successful shots and passes have been linked to success (Lago-Ballesteros and Lago-Peñas, 2010). Being able to maintain the ability to perform these skills for as much of the match as possible will therefore increase a team's chances of winning, as a disproportionate number of goals are scored within the last 15 minutes of play (Reilly, 2003). It can therefore be hypothesised that teams who can maintain technical performance for the longest period of time will have a higher chance of scoring as the opposition begin to fatigue.

Shooting is perhaps the most important skill to execute correctly in soccer as shot outcomes directly influence the result of the match (Hughes and Churchill, 2005). Studies have shown that the most successful Spanish top division teams had more shots than those lower down the table (Lago-Ballesteros and Lago-Peñas, 2010). It was also shown that across 3 World Cups, the winning teams in matches had significantly more shots than losing and drawing teams. Shot accuracy is highly variable, with studies showing a coefficient of variance of 20% (Ali et al., 2007a, Russell et al., 2010), making it difficult to isolate fatigue as a factor in shot success. Yet one aspect of shooting that can be attributed to fatigue is decreased shot power as a result of strength loss, which is vital to control when

performing this action. One consistent observation is that following exercise, shot power and speed is reduced following non-soccer-specific and soccer-specific exercise (Lees and Davies, 1988, Kellis et al., 2006).

It should be noted that many soccer skills are open skills, which have a huge variety of factors influencing the outcome, regardless of fatigue state. There is also high variability in accurately reproducing skill performance in non-elite players. This has therefore made measuring skill performance somewhat difficult, as no simulated situation can accurately replicate the psychological and environmental demands of match play. This may explain why many studies have found no direct effect of fatigue on skill performance (Ali et al., 2007b, Ali et al., 2011, Rampinini et al., 2011, Russell et al., 2012, Rampinini et al., 2008).

## Monitoring fatigue

It is of great importance for professional soccer clubs to have their players free from fatigue and at peak condition for match days. As shown above, under the influence of fatigue some physical performance capabilities are markedly reduced, which is likely to reduce the chance of match success. This was highlighted by (Ekstrand et al., 2004), who used international level coaches to identify players who had underperformed at the 2002 FIFA world cup, and found that they had played the most games in the lead up to the tournament. Ekstrand et al. (2004) suggested the reason for this underperformance was long term mental fatigue due to potential burnout from a long, congested season (Kenttä and Hassmén, 1998). Reductions in aerobic fitness have been shown towards the end of congested professional competitive seasons (Haritonidis et al., 2004). It has also been shown that starting players showed decrements in sprint speed and vertical jump height at the end of competitive seasons compared to non-starting players (Kraemer et al., 2004).

Congested periods where soccer teams are expected to play 3 games in a week, a common situation for teams playing on European or domestic cup competitions, can be particularly problematic. Such

little recovery time in between matches requires periodised training and nutritional intervention to maximise performance (Anderson et al., 2016). A number for studies have examined if physical performance is impaired in the later games of the week and have found no significant differences in physical performance (Anderson et al., 2016, Rey et al., 2010, Djaoui et al., 2014, Dupont et al., 2010, Folgado et al., 2015). However, Folgado et al. (2015) used players' movement dyadic synchronisation to assess tactical performance and found a drop in games toward the end of a 3 game week, suggesting a drop in motivation as the cause. They suggested the drop in motivation stemmed from feelings of discomfort caused by underlying muscle soreness attained from playing competitive matches with little recovery time. It has also been shown that fatigued players may be more likely to sustain an injury (Brooks et al., 2008) especially during congested fixture periods (Dupont et al., 2010). It should be noted that some studies have found no increases in injury rate, however the difference in playing standard ad country, which can influence the physical demands of the game, may explain discrepancies (Lago-Penas, 2009, Carling et al., 2012).

Small changes in movement patterns as a result of fatigue, such as reduced range of motion, joint stiffness or reduced neuromuscular activation, could increase the risk of ligament injuries as a result of poor technique execution (McLean and Samorezov, 2009). Neuromuscular fatigue, for example, promotes inadequate joint stabilization via suboptimal muscle activation (Borotikar et al., 2008, Kernozek et al., 2008). Changes in landing posture (Chappell et al., 2005), hip rotation (Borotikar et al., 2008) and knee motion loads (Kernozek et al., 2008) have been observed and are common risk factors for anterior cruciate ligament injuries (Gleeson et al., 1998).

The presence of fatigue during subsequent exercise can arise as a result of either over training or "under recovery" (Bishop et al., 2008). Over training is defined as an imbalance between amount of training or competition compared to recovery, which can cause mental burnout, apathy towards the sport and reduced performance capacity (Kuipers and Keizer, 1988). Similarly, "under recovery" as defined by Bishop et al. (2008) as an insufficient amount of recovery time between performances. Full

recovery is when a player can reach pre-exercise benchmark performance markers, some of which require 72 hours to recover (Nédélec et al., 2012), yet matches may occur just 48 hours apart. To reach this state of optimal recovery in a limited period, there must be a clear understanding of the type of fatigue elicited and its effect on future performance.

## Methods to measure fatigue

Professional sport provides a difficult environment to measure physical status of athletes in a way that does not interfere with day to day activities. While there are gold standard measures of the fatigue symptoms listed above, many of them would be too time consuming, expensive or impractical to perform in professional sporting environments. For example, isokinetic dynamometers provide measures of strength loss, but with full squads of 20+ players, a limited time schedule, expensive equipment and specialist training required to operate, it may not be a viable option. A further limitation are tests that require maximal effort or prolonged activity that may elicit further fatigue and prolong the recovery process (Nédélec et al., 2013). Instead, more functional movement tests can be used to assess performance, which can be performed easily and quickly across a squad of players. For these tests to be effective, they must be sensitive enough to detect day to day fluctuations in performance between training and match days and injury risk factors.

## Physical performance measures

One example of a functional test used in professional soccer is via the Functional Movement Screen (FMS) (McCall et al., 2015). This methods involves 7 fundamental movements that are performed to evaluate painful or dysfunctional movement patterns, however the reliability of this has been challenged (Shultz et al., 2013). While Shultz et al. (2013) found while the test-retest reliability was good, the between observer reliability was poor. It was concluded that sufficient training should be administered to coaches implementing the tests to reduce this problem. Despite this it was shown that as many as 66% of clubs in top divisions directly use this method (McCall et al., 2014) with a further 16% using a modified version. The FMS is a simple, cost effective test that can be sensitive enough to detect day to day changes in muscle soreness, joint stiffness and drops in coordination.

Jump protocols have been frequently used to assess recovery in professional soccer and are often included in benchmark testing. Squat jumps and counter movement jumps can represent the stretch-shortening cycle as well as surrogate measures of power production and may serve as valid measures of recovery (Bangsbo, 1993). They have been shown to display good reliability with co-efficient of variation values of 2.6-5% (Cormack et al., 2008, Moir et al., 2004). However, there is little research examining jump performance over long periods during a competitive season, therefore making its use of a daily measure of fatigue in need of further study.

Isokinetic muscle contractions are also commonly used amongst professional clubs during rehabilitation and medicals, mostly as a screening process to identify injury risk (McCall et al., 2014). This process aims to identify muscle imbalances between pairs of opposing muscles which may be present following an injury or to predict a future strain (Croisier et al., 2008). Some studies have found this screening process to be effective in not only predicting, but also preventing further injury in professional soccer players by successfully identifying muscle imbalances, which can then be corrected

with strength and conditioning programmes (Croisier et al., 2008, Croisier et al., 2002). Others have found it not able to discriminate between those at risk of an injury (Bennell et al., 1998).

Muscle soreness ratings can act as a subjective measure of muscle function and mobility (Cook et al., 1997). Soccer players can experience marked muscle soreness following match play or intense training sessions (Cheung et al., 2003). Soreness peaks 24-48 hours following exercise and the magnitude of which can depend on age, training status and type of exercise performed (McHugh, 2003). Some studies however, have questioned the link between muscle soreness and muscle function, suggesting an individual's perception played a role in recovery status (Cook and Beaven, 2013) and that delayed muscle soreness is a poor reflection of EIMD (Nosaka et al., 2002). Soreness is usually measured on a visual analogue scale using subjective sensations of pain, so it is imperative that each individual is familiar with the ratings to avoid inaccurate data.

## Wellness questionnaires

Due to its simplicity and cost effectiveness, wellness questionnaires have been extensively used amongst professional athletes and coaches to monitor fatigue status (Coutts et al., 2007, Hooper et al., 1995, Kellmann, 2010). The reliability of these have been supported with one review suggesting they are more sensitive to changes in day to day training load than objective measures, such as creatine kinase, cortisol and $VO_2$max (Saw et al., 2015). Hooper et al. (1995) demonstrated that wellness questionnaires could identify potential over-training in professional swimmers. However, little data exists for professional soccer teams, who have large day to day variance in training and match load. Wellness questionnaires relating to muscle soreness and sleep quality, two key areas of recovery (Kenttä and Hassmén, 1998) have been used within soccer. While no study has directly reported it, one problem with this method is potential social factors influencing results of questionnaires such as concerns over social standing or team selection based on high scores.

## Physiological Measures

Following intense exercise, a number of biochemical markers associated with fatigue are found within the blood. As previously mentioned, creatine kinase is a common marker of muscle damage and blood concentrations are elevated following soccer matches and peak between 24-72 hours reaching concentrations between 70 to 250% of baseline (Ispirlidis et al., 2008, Magalhães et al., 2010, Thorpe and Sunderland, 2012). There has been recent attention, and controversy, on the usefulness of blood CK as a predictor of recovery status (Warren et al., 1999). Due to the differences in blood CK concentration between gender, age, ethnicity, and training status (Baird et al., 2012), a reference interval for athletes was devised to avoid misinterpretation of CK concentration: 82 to 1083 units per litre (U/L) for males and 47 to 513 U/L for females. (Mougios, 2007). While CK concentrations will increase with muscle damage, they can remain elevated for more than 72 hours (Ispirlidis et al., 2008) making its usefulness during congested fixture periods of multiple matches in a short period of time questionable. Additionally, other studies have questioned the link between CK, soreness and magnitude of muscle damage (Nosaka et al., 2002, Walsh et al., 2001).

Markers of the inflammatory response following soccer have been used as methods to identify fatigue state. Interleukin-6 (IL-6) is a cytokine and common marker of inflammation (Pedersen and Toft, 2000). Cytokines facilitate repair and regeneration of damaged muscles to allow growth to occur (Tidball, 2005, Gleeson and Bishop, 2000). Following soccer performance, there is an immediate rise in IL-6, with return to baseline around 24 hours later (Ispirlidis et al., 2008, Andersson et al., 2008). Concentration of IL-6 can be dependent on the type of exercise with eccentric contractions having a delayed peak and slower return to baseline than running (Fischer, 2006). Similarly uric acid was also found to be a consistent representation of muscle inflammation (Ispirlidis et al., 2008) with increases being present up to 48 hours following match play (Fatouros et al., 2010). Uric acid is a marker of the breakdown of amino acids (Viru and Viru, 2001) and held a parallel increase to perceived muscle soreness following a soccer match (Andersson et al., 2008).

## Predicting physiological responses- Training and Match Load

In recent years there has been a lot of focus on developing monitoring tools than can serve as valid methods for estimating the physical demands of a training session or match. This allows the identification of activity profiles, which can be used to help prevent injury by ensuring that training is not only replicating match demands, but also periodised to prevent over training (Reilly, 2005, Anderson et al., 2016, Morgans et al., 2014a). It can also help develop individual nutritional programmes by estimating energy cost of sessions (Anderson et al., 2016, Gaudino et al., 2014), track inter squad variability in movement patterns (Aughey, 2011) and can be used to predict the physical response from a upcoming session to ensure the correct physical demands are met. One common method has been the use of "load" which can be calculated in a number of ways both, subjectively and objectively.

Internal load aims to quantify the physiological stress imposed on the athlete during training (Alexiou and Coutts, 2008). In one method physiological stress is quantified by heart rate during the session and rating of perceived exertion (Borg, 1982). Heart rate during incremental exercise is linearly and positively related to oxygen consumption ($VO_2$) and therefore represents the aerobic demands of exercise (Åstrand, 2003). However, it should be noted that heart rate may not be sensitive enough to detect explosive actions or powerful movements, and may therefore underestimate the physical requirement of such activities (Impellizzeri et al., 2004). Another method involves multiplying training duration with RPE, and has been shown to have a good level of agreement with heart rate in soccer activities (Impellizzeri et al., 2004, Alexiou and Coutts, 2008).

External load aims to quantify the physical forces placed upon the body as well as metrics, such as running speed and distance covered. The use of trunk mounted global positioning systems (GPS) have been adopted to provide more objective measures of speeds and distance achieved during sessions and competitive matches which can be used to measure frequency and magnitude of movements in

a given space (Portas et al., 2012, Varley et al., 2012, Gregson et al., 2010, Nedergaard et al., 2014). Moreover, the addition of accelerometers to systems in recent years allows quantification of gross fatiguing movements such as jumps, sprints and the accelerations and decelerations associated with performing sharp turns (Boyd et al., 2011). These actions increase the demands of the exercise and not accounting for them could lead to an under estimation of both external and internal load. To simply express this data, PlayerLoad™ (PL) was developed. PlayerLoad™ is an arbitrary output from GPS units that is derived from 3-D measures of instantaneous changes in acceleration, which aims to quantify external load experienced by the athlete during exercise. GPS data have been shown to be reliable in quantifying distance and speed (Boyd et al., 2011).

Recently there has been an influx of research into methods to attempt to estimate physiological load during either training or match play using these metrics (Gaudino et al., 2014, Gaudino et al., 2013, Barrett et al., 2015, Casamichana et al., 2013, Aughey, 2011). For this technology to be effective in measuring load of a session there must be links between metric data and physiological responses. Strong correlations between PL values and measures of perceived exertion and heart rate were shown during soccer specific exercises (Portas et al., 2012), yet some suggests units may not sensitive enough to detect mild fatigue (Nedergaard et al., 2015). Using GPS accelerometer data, Jones et al. (2014) found that CK levels, an indication of muscle damage, were highest in those who had performed the most collisions and high speed runs in rugby players. Additionally when measuring blood creatine Kinase (CK) concentration, Young et al. (2012) showed positive correlations between CK and GPS load in Australian Rules football players. Players who performed the most eccentric muscle loading and high intensity work, such as accelerations and decelerations, were found to have significantly higher blood CK concentration post exercise. As previously mentioned, accelerations and decelerations are highly demanding activities that cannot be accurately quantified without accelerometer data.

One study highlighted the usefulness of GPS unit data by showing a link between amount of high intensity efforts and increased injury risks in team sport players (Gabbett and Ullah, 2012). The risk of a lower limb soft tissue injury was 2.7 times higher in players who regularly performed sprints exceeding 9 metres in length during sessions. Conversely, players who performed the highest amount of moderate to low intensity distance, and low intensity accelerations had a significantly reduced risk of injury (Gabbett and Ullah, 2012). Furthermore, Ehrmann et al. (2016) identified two GPS variables that could potentially predict soft tissue injuries. They found average meters per minute, a measure of intensity, increased significantly by 9.6% above the season average prior to an injury. They also observed average weekly PlayerLoad™ was significantly decreased below the season average leading up to injury, suggesting training that did not replicate match demands, a known injury risk (Ehrmann et al., 2016, Di Salvo et al., 2009).

Other studies have used GPS data to estimate the metabolic and energy demands of a session. Using a novel method Gaudino et al. (2013) calculated energy cost from soccer training and attempted to prevent the underestimation that comes from using speed and distance alone. Using a method proposed by (Di Prampero et al., 2005) and later refined by (Osgnach et al., 2010), they provided the first attempt to quantify individual external load between daily activity amongst a squad of professional players. Differences were observed between playing position as well as proof of underestimation when using speed and distance alone to estimate demand. They concluded that this method provided a better understanding of the demands of soccer and highlighted the importance of individual periodisation of training.

## Summary

The process of muscle contraction involves a number of steps beginning with an electrical impulse sent from the motor cortex in the brain and ending with mechanical binding of actin to myosin. During and following exercise, almost every stage of this process can be disrupted to reduce a muscles ability

to maintain optimal performance in a phenomenon known as fatigue. Some of these symptoms of fatigue can persist up to and beyond 72 hours, with some even requiring interventions to fully recover. Symptoms that occur proximal and distal to the neuromuscular junction are split into central and peripheral fatigue respectively. The magnitude of these symptoms can be affected by the type, duration and intensity of the exercise. Other factors include climate of exercise environment and training status of the individual. Interventions can accelerate recovery and reduce the effects of fatigue on future performance. Prior to full recovery, fatigue can severely reduce exercise capacity in subsequent bouts and may pose an increased risk of injury.

Soccer is a highly demanding physical sport that elicits high levels of fatigue and muscle damage following performance (Bangsbo et al., 2007, Rampinini et al., 2011). The physiological demands of soccer can differ significantly depending on playing position, team tactics, and opposition amongst others (Gaudino et al., 2013, Bloomfield et al., 2007). This can dictate the frequency and intensity of fatiguing movements such as sprints, jumps and total distance covered. It is therefore logical to assume that proceeding match play there will be a varying physiological response between individuals and playing positions. In order to maximise match performance and reduce the risk of injury, players should be as free from the effects of fatigue as possible. However, the busy schedule of professional soccer environments and requirements to sometimes play two games within 48 hours place restrictions on the level of tests that can be performed to quantify fatigue. Therefore, soccer clubs often have to employ simpler, less reliable methods to assess fatigue within a squad, some of which have not been fully validated. Despite advances in sports science over the last 20 years, soft injury incidence in soccer has in fact increased, with over 50% of injuries occurring in match play (Ekstrand et al., 2016). With some research suggesting underlying fatigue is a risk factor for injury, it may suggest that the current methods to monitor squad fatigue may be inadequate, which may in part be due to the increasing demands of the game (Bush et al., 2015).

In recent years attempts have been made to quantify physical demands of training sessions using both subjective and objective measures of load. Internal load includes rating of perceived exertion (RPE), heart rate and lactate which provide both subjective feelings of fatigue and estimations of intensity of a session. While certainly useful, these values cannot quantify the content of a bout of exercise, only the response elicited. Via the use of GPS units and video tracking technology, external load allows quantification of distances covered, number of high intensity actions performed and the forces places upon the body during exercise. These metrics can be used to calculate metabolic demand (Gaudino et al., 2014) detect changes in daily, weekly and monthly loads (Anderson et al., 2016) and ensure training is reflective of the demands of match play (Borresen and Lambert, 2009). While GPS units have been shown to be accurate in measuring distances and speeds performed in sessions, little research exists examining the relationship between GPS metrics and physiological responses. For future advancements in quantifying the physiological demands of soccer, a better understanding is needed between the performance and type of fatigue elicited. In professional soccer, fully understanding of the demands of training and match play will allow individualised training strategies which can optimise the effectiveness of the training and minimise injury risk.

# Chapter 3:

## Is Playerload™ related to fatigue following different types of repeated maximal sprint exercises in recreationally active young men?

### Introduction

Soccer is a highly demanding sport due to its intermittent nature and frequency of explosive actions (Bangsbo et al., 1996, Mohr et al., 2003). Elite players are required to perform over 1,400 purposeful movements (Mohr et al., 2003, Reilly, 2003), which comprise an average of 200 high speed runs (Mohr et al., 2003), 700 changes of direction (Bloomfield et al., 2007) and 19 maximal effort sprints during match play (Rienzi et al., 2000). These movements place significant stress up on the hamstring and quadriceps, and lead to the development of fatigue, which can be categorised as either central, i.e. distal to the neuromuscular junction (Gandevia et al., 1995, Davis and Bailey, 1997, Bigland-Ritchie et al., 1978) or peripheral, i.e. within the muscle (Gandevia, 2001, Finsterer, 2012, Amann, 2011, Edwards, 1981, Fitts, 1994).

Central fatigue involves factors such as internal motivation, central nervous system transmission, motor unit recruitment and firing frequency. While central fatigue has been routinely observed following soccer specific exercise, focus has primarily been on how this affects *quadriceps femoris* function (Rampinini et al., 2011, Thorlund et al., 2009). Few studies have investigated how central fatigue might affect the hamstring muscle group (Marshall et al., 2014, Robineau et al., 2012). While peripheral fatigue has routinely been observed, evidence of central factors contribution to this fatigue have been observed by some (Marshall et al., 2014) but not all (Robineau et al., 2012) following simulated soccer matches.

Peripheral fatigue is thought to be associated with (but not limited to) permeability of the sarcolemma and the increase in muscle-specific proteins in the blood (e.g. creatine kinase), reduced ATP concentration, increased inorganic phosphate concentration, reduced phosphocreatine (Edwards, 1981, Enoka and Stuart, 1992, Fitts, 1994) and structural damage to the sarcomeres (Allen et al., 2008, Byrne et al., 2004, Proske and Allen, 2005). Structural damage to muscle fibres occurs following eccentric contractions (Byrne et al., 2004, Proske and Allen, 2005). Actions, such as sprinting and decelerating, place high eccentric load on the hamstrings and quadriceps and has been shown to cause a change in biomarkers of exercise-induced muscle damage (Rampinini et al., 2011, Howatson and Milak, 2009). Damaged muscles exhibit reduced range of motion, reductions in maximum strength (Clarkson and Tremblay, 1988, Goff et al., 1998, Saxton et al., 1995, Paddon-Jones and Quigley, 1998) and may also have altered movement patterns during running and landing (Borotikar et al., 2008, Chappell et al., 2005, Kernozek et al., 2008), placing individuals at a greater risk of injury (McLean and Samorezov, 2009).

Some peripheral symptoms can persist in the subsequent days and can limit performance for up to 72 hours (Nédélec et al., 2012, Andersson et al., 2008, Brink et al., 2010). This is especially problematic in elite soccer, as matches can occur just three days apart, leaving little time for recovery ahead of preparation for the next match. This is further complicated by differences in post-match fatigue experienced between squad members due to not only the natural variance in response to exercise (Manzi et al., 2010) and match demand between playing position (Bloomfield et al., 2007).

Accurately quantifying fatigue, however, is problematic, as no gold standard measurement exists. This is a particular challenge in elite sporting environments, where precise, yet invasive and time-consuming, techniques are impractical. In recent years the use of global positioning systems (GPS) have become common practices amongst top level football clubs (Portas et al., 2012) to measure training load, and competitive match load and attempt to predict physical fatigue from training or match play (Andrzejewski and Chmura, 2008). PlayerLoad™ is an arbitrary output from GPS units that

is derived from 3-D measures of instantaneous changes in acceleration, which aims to quantify external load experienced by the athlete. While GPS data have been shown to be reliable in quantifying distance covered and speed (Boyd et al., 2011) little research exists on comparing markers of fatigue with GPS unit data. Blood creatine kinase (CK) concentration (a recognised marker of muscle damage (Nédélec et al., 2012)) has been positively correlated with GPS unit outputs in Australian Rules football players (Young et al. (2012). However, peak blood CK concentration may not manifest until >72 hours post strenuous exercise and there exists a large inter-person variability (Nosaka and Clarkson, 1996, McNeil and Khakee, 1992), which can persist for up to 7 days (Ehlers et al., 2002) making its use in day to day monitoring unreliable.

Additionally, soccer players have significantly different movement demands during match play, depending on their playing position (Bloomfield et al., 2007). Numerous studies have attempted to quantify the physical response to different types of soccer-related exercise to mixed results. It has been shown that sub maximal repeated changing of direction elicits higher heart rate, blood lactate and energy cost than straight line running when matched for speed (Hatamoto et al., 2014, Dellal et al., 2010). However, during maximal sprints, straight line showed higher heart rate and PlayerLoad$^{TM}$ than changing direction (Taylor et al., 2016) but these were not matched for distance. To our knowledge, no studies have compared exercise induced fatigue in the hamstrings and quadriceps between repeated straight line sprinting versus repeated shuttle sprinting, common movement patterns in soccer matches (Bloomfield et al., 2007). Moreover, it is not known whether the GPS "load" measurement differs between distance-matched exercise, and whether this is related to physiological measures of fatigue allowing its use a tool to predict physical response to exercise.

The aims of this study were to (1) compare the central and peripheral (quadriceps and hamstring muscle) fatigue responses between two repeated sprint exercises matched for distance and add to the limited data on hamstring fatigue; and (2) examine the relationship between trunk mounted accelerometer data and physiological measures of fatigue. We hypothesised that the higher velocity

achieved would cause PlayerLoad$^{TM}$ values to be higher during straight line sprinting than during shuttle running. Furthermore, we hypothesised that straight line sprinting would elicit greater fatigue compared to shuttle running, and that PlayerLoad$^{TM}$ would be related to physiological measures of fatigue.

# Methods

## Participants

Eighteen healthy young men (age: 21 ± 3 yrs; height: 1.78 ± 0.1 m; body mass: 73.7 ± 11.1 kg) volunteered to take part in this study, which complied with the Declaration of Helsinki and was approved by Liverpool John Moores University Ethics Committee. Participants took part in recreational physical activity but had not completed lower limb resistance training for at least 6 months and were free from lower limb injury for at least 12 months. Physical activity levels were determined to general health and habitual physical activity questionnaire prior to participation (Baecke et al., 1982) and habitual physical activity levels are shown in Table 1. Habitual physical activity level was assessed from 16 questions, using a scale from 1 to 5, where 1 was the least active and 5 was extremely active. Participants were randomly assigned to one of two groups: (i) straight-line sprinting (SP, n = 11) and (ii) shuttle sprints (3T, n = 7). Baseline physical characteristics of both groups are shown in Table 1.

***Table 1:*** Baseline physiological information; BMI = body mass index. SP = sprint protocol group. 3T = shuttle protocol group.

| | *Sprint Protocol (SP)* | *Shuttle run protocol (3T)* |
|---|---|---|
| Age (yrs) | 19.5 ± 1.0 | 22 ± 4.0 |
| Height (m) | 1.8 ± 0.1 | 1.8 ± 0.1 |
| Weight (kg) | 73.4 ± 7.0 | 73.6 ± 15.2 |
| BMI (%) | 23.1 ± 2.5 | 23.32 ± 4.5 |
| Baseline Hamstring Strength (N·m*)* | 142.7 ± 26.7 | 153.1 ± 19.0 |
| Baseline Quadriceps Strength (N·m) | 266.9 ± 55.9 | 277.4 ± 41.8 |
| Physical activity score (1 = low; 3 = medium; 5 = high) | 3.2 ± 0.3 | 3.2 ± 0.4 |

## Experimental design

Participants were randomly assigned to one of two groups: (i) straight-line sprinting (SP, n = 11) and (ii) shuttle sprints (3T, n = 7). Three lab visits were required, once for a familiarisation session, once to complete the protocol and pre/post assessments (e.g SP or 3T), and once to complete the post 48 h assessment. Familiarisation took place one week before testing. Pre and 48 h post-exercise assessments were conducted at the same time of day for each participant. Participants were instructed not to partake in any strenuous physical activity, consume and alcohol, drugs or purported recovery supplementation in the 48 h before assessment, or during the study itself. Participants were instructed to follow their habitual diet during the study. Fatigue assessments were always performed in the same order on the right leg of each participant.

## Familiarisation

Participants were familiarised at least 1 week before testing to avoid any fatigue being present on the pre-tests. Upon arrival participants were familiarised with all of the procedures, under the conditions they would be completed on the testing day. Tests that required input from the participant (maximum isometric voluntary contraction, interpolated twitch technique, muscle soreness, exercise protocol) were performed multiple times until testers were content the participant could competently perform them.

## Repeated Sprint Protocol

A between groups design was used to avoid any repeated bout effect between testing conditions. Participants completed one of two protocols on an indoor synthetic running track. The first protocol was 15 x 30 m straight-line maximal sprints (SP) with a 12 m deceleration zone. The second was 15 x (3 x 10) maximal shuttle runs (3T) with an 8 m deceleration zone. Each protocol included 90 seconds rest between each repetition, with 180 s rest on repetition 5 and 10. In both protocols participants were asked to begin 1m behind the start line to avoid interfering with timing gates upon initial acceleration. Participants were instructed to sit on a chair between repetitions to standardise recovery. When performing the 3T protocol, participants were instructed to alternate the planted leg when changing direction in an attempt to spread the eccentric load across both legs. Participants were asked to perform each repetition in both protocols at maximum effort and strong verbal encouragement was provided throughout. Before the protocol, a five minute standardised warm up consisting of jogging, dynamic stretching and self-paced 20 m runs at 60%, 80%, 100% of perceived top speed, were performed. After each repetition, the following measures were taken, heart rate using (Polar Oy, Kempele, Finland), repetition time using timing gates (Brower Timing Systems, Draper, UT, USA) and three ratings of perceived exertion (RPE) adapted from the CR100 model (Borg and Borg, 2002). Participants were asked to provide ratings of (i) respiratory exhaustion such as breathlessness, (ii) lower limb exhaustion, such as muscle or joint pain and (iii) overall demand of the exercise, which were recorded as Central, Leg and Overall RPE, respectively. Water was available *ad libitum*. Protocols were completed with electrical stimulation pads and electromyography (EMG) electrodes (see below)

securely fitted with strapping attached on the right leg. Participants were advised to wear the same footwear for each testing day. Performance decrement during the protocol was assessed using the following formula (Fitzsimons et al., 1993, Glaister et al., 2008), where total sprint time = sum of time from all 15 sprints; and ideal sprint time = total number of sprints (15) x fastest repetition sprint time.

$$Performance\ decrement = \left(100\ X\ (total\ sprint\ time\ \div ideal\ sprint\ time)\right) - 100$$

### PlayerLoad™

During the protocol participants wore trunk mounted accelerometers (Catapult Optimeye S5, Catapult., Melbourne, Australia) fitted securely between the scapula, as per manufacturer recommendations in a Catapult branded specific GPS vest. PlayerLoad™ (PL) is quantified via micro sensor data trough integration of 100-Hz 2-16g triaxial accelerometry, with triaxial gyroscope data to measure body angular motion (sampling rate of 200-2000° per second) and 100 Hz triaxial magnometers for body direction and orientation. PL is equal to quotient of the square root of the sum of the squared instantaneous rates of changes in all acceleration planes over 100 (Boyd et al., 2011, Van Iterson et al., 2017). The equation used in calculating PL is described below (Wik et al., 2017). A cumulative PlayerLoad™ score was taken from between the start of the first repetition and the completion of the final repetition. Data were extracted using Catapult Openfield software (Catapult Optimeye S5, Catapult, Melbourne, Australia). PlayerLoad™ has demonstrated moderate (CV = 10 – 20%) to strong (CV = 0-10%) test-retest reliability in both lab (Barrett et al., 2014) and field based environments (Boyd et al., 2011, Van Iterson et al., 2017).

$$PlayerLoad^{TM} = \frac{\sqrt{(a_{y1} - a_{y-1})^2 + (a_{x1} - a_{x-1})^2 + (a_{z1} - a_{z-1})^2}}{100}$$

$a_y = forward\ acceleration$

$a_x = sideways\ acceleration$

$a_z = verticle\ acceleration$

## Assessment Protocol

### *Maximal Isometric Voluntary Contractions and Range of Motion*

An isokinetic dynamometer (HUMAC NORM, CSMI Solutions, Massachusetts, USA) was used to measure maximum voluntary contraction (MVC) isometric knee extension and flexion torque in the right leg of each participant in the seated position. Participants were firmly strapped at the hip, distal thigh and chest with inextensible straps to minimise movement. The seat length was adjusted for each participant to align the rotational axis of the dynamometer with the lateral femoral epicondyle. The lever arm of the dynamometer was firmly attached to the lower leg with inextensible straps. Participants were then asked to fully extend and flex their knee in the seated position with the hip joint at $90^{o}$, to allow range of motion around the knee joint to be taken ($0^{o}$ = full extension). Participants then performed a standardised warm-up, comprising 10 isokinetic knee extension and 10 flexion contractions at $60^{o} \cdot s^{-1}$, starting at 10% of participant's perceived maximum effort, and increasing by 10% with each repetition to 100% effort with the last repetition. Participants were then asked to perform a minimum of three extensions and three flexion MVCs (alternating every 30 s) at $30^{o}$ knee flexion. If the highest of the three attempts was >5% higher than the next highest, a fourth attempt was made to ensure a true MVC was achieved. Each MVC lasted 2–3 s and there was a rest-interval of 30 s between each alternating contraction. This was repeated and $80^{o}$ knee flexion. Hip angle was maintained at $90^{o}$ for all contractions. Biofeedback in the form of the torque-time information (which was projected on the wall in front of the participant in real time) and verbal encouragement, was provided throughout to help ensure participants performed maximum efforts. The range of joint angles used was based on the results of pilot work, which identified $30^{o}$ and $80^{o}$ as the optimum knee joint angles for isometric force production during knee flexion and extension respectively.

### *Voluntary muscle activation*

The interpolated twitch technique (ITT) was used to measure voluntary muscle activation capacity of the hamstring muscle group at 30 deg knee flexion during a seated position, the general procedure for which has been described elsewhere (Erskine et al., 2009, Erskine et al., 2010b, Marshall et al.,

2014, Merton, 1954). Electrical stimulation was administered via two 12.5 mm x 7.5 mm self-adhesive electrodes (DJO Global, California, USA), placed distally (anode, proximal to the popliteal crease) and proximally (cathode, distal to the gluteal fold) over the hamstrings. The relaxed hamstrings were stimulated with a 50 mA 100 Hz doublet of 200 $\mu$s pulse width, which was increased in 20 mA increments every 10 s (DS7AH; Digitimer Ltd., Welwyn Garden City, United Kingdom) until no further increase in doublet torque was observed. When using the quadriceps other studies have suggested increases of 20% to ensure supramaximal stimulation during the ITT. However, during pilot studies we found participants were uncomfortable at such values in the hamstrings, therefore the amplitude was increased by 10%. The supramaximal doublet stimulation was used two minutes later to elicit resting supramaximal doublet torque in the resting state (control doublet), followed 5 s later by a second doublet (superimposed doublet) during an isometric knee flexion MVC. Voluntary activation was calculated using the formula:

100 x [1- (superimposed doublet torque/ control doublet torque)]

### *Voluntary Peak Rate of Torque Development (pvRTD)*

The peak rate of voluntary force development (pvRTD) was defined as peak $\Delta$torque/$\Delta$time, i.e. the steepest point in the rise in torque from (manually determined) torque onset (Erskine et al., 2014) to isometric knee flexion/extension MVC, and the highest pvRTD from three attempts with no countermovement (identified increasing the resolution of the baseline torque trace) was used for further analysis. The time to pvRTD was also recorded from torque onset to pvRTD, and these values are displayed in Table 2.

### *Involuntary peak rate of torque development (piRTD)*

The highest piRTD was generally determined in the last three involuntary contractions of the maximum compound muscle action potential of the hamstrings ($M_{max}$) protocol (see below). As with pvRTD, piRTD was defined as the steepest rise in torque from torque onset.

### *Surface electromyography (EMG)*

Surface EMG was recorded from the biceps femoris long head (BF) and the *vastus lateralis* (VL) muscles. After preparing the skin (shaving, lightly abrading, and cleansing with 70% ethanol), two bipolar Ag-AgCl surface electrodes (Noraxon duel EMG electrode, Noraxon, Scottsdale, USA) were placed 20 mm apart on the skin, parallel to the presumed orientation of the muscle fibres, and one reference electrode (Ambu Blue, Ambu, Copenhagen, Denmark) was positioned over the medial tibial condyle. The exact location of the electrodes was marked on the participant's skin and recorded on an acetate sheet with a permanent marker to ensure precise electrode repositioning in the 48 h post exercise trial. BF and VL electrodes were placed mid belly at 33% of the distal end of the respective muscle, according to SENIAM guidelines (Hermens et al., 1999). Both muscles were identified using ultrasonography (Epiq 7, Phillips, Amsterdam, Netherlands), with the participant in the prone (BF, 0 deg knee flexion) or seated position (VL, 80 deg knee flexion) for the respective muscles.

Surface EMG signals were sampled at 2000 Hz (Biopac Systems, Santa Barbara, USA) before being band-pass filtered (10–500 Hz, AcqKnowledge, Biopac Systems, Santa Barbara, USA). The root mean square of the EMG signal of a 500-ms epoch around peak MVC was used to assess the activation of agonist and antagonist muscles. To minimize the variability in absolute BF EMG (Burden, 2010), BF EMG recorded at 30 deg knee flexion MVC was normalized to the evoked compound muscle action potential ($M_{max}$) of the BF (see below). Antagonist muscle co-activation (e.g. quadriceps activation during MVC flexion at 30 deg knee flexion, or hamstring activation during MVC extension at 80 deg knee flexion) was calculated as follows (where $EMG_{max}$ is the maximum EMG of the antagonist muscle when acting as an agonist at the same knee joint angle):

$$Antagonist\ muscle\ co-activation = \frac{EMGantagonist}{EMGmax} \times 100$$

Torque signals, electrical stimuli, and EMG activity were displayed on a computer screen, interfaced with an acquisition system (AcqKnowledge, Biopac Systems, Santa Barbara, USA) used for analogue-

to-digital conversion, at a sampling frequency of 2 kHz. Co-activation data were available for n = 13 (SP = 8, 3T = 5).

### *Biceps Femoris Maximal compound muscle action potential ($M_{max}$)*

Single square wave twitch pulses (200 µs duration) were applied to the hamstring muscle group (DS7AH; Digitimer Ltd., Welwyn Garden City, United Kingdom) at 10 to 20 mA incremental amplitudes, evoking M-waves (measured via surface EMG, as detailed above) until a maximal M-wave was produced (this was generally achieved at ~180 mA). The maximal compound muscle action potential ($M_{max}$) was defined as the mean peak-to-peak EMG response from the 3 highest observed M-waves. $M_{max}$ was used for normalisation of hamstring EMG during knee flexion MVC. Due to technical issues, biceps femoris EMG data normalised to $M_{max}$ data was available for n = 9 (SP = 4, 3T = 5).

### *Torque-frequency relationship*

The stimulation intensity used to determine the torque-frequency relationship of the hamstring muscle group was adjusted so that a 1 s 100 Hz tetanus produced approximately 20% isometric knee flexion MVC torque. Five minutes after the last knee flexion MVC at 30 deg knee flexion, the hamstring muscle was stimulated with 1s 1, 10, 15, 20, 30, 50 and 100 Hz trains in a random order, each separated by 1 min, to assess the torque-frequency relationship.

### *Perceived muscle soreness*

Participants were instructed to perform three unloaded squat repetitions to a standardised chair height (45 cm; knee flexion angle ≈ 90 deg), returning to full extension, and keeping their back as straight as possible. Three unilateral forward leg lunge were also performed on each leg. Using a visual scale, a soreness rating between 0 and 10 was taken, with 0 being no pain at all and 10 being unbearable pain.

Muscle soreness ratings were also taken from 9 points of the quadriceps and 6 points of the hamstrings (proximal end, belly of muscle and distal end sites of the *rectus femoris* (RF), *vastus lateralis* (VL), *vastus medialis* (VM), *biceps femoris* long head (BFLH) and *semitendinosus* (ST) using an algometer (FPK/FPN Mechanical Algometer, Wagner Instruments, Greenwich, USA). Participants were

asked to indicate when the applied pressure resulted in pain and were blinded to the results throughout the study.

### *Blood lactate concentration*

Capillary blood samples were taken on testing day at rest and immediately after performing the exercise protocol. Blood samples were drawn from a finger-tip using a Safety-Lancet Extra 18G needle (Sarstedt; Nümbrecht, Germany) and were analysed within 60 seconds of collection using a portable blood lactate analyser (Arkray Lactate Pro; Kyoto, Japan). All lactate probes were cleaned and calibrated in accordance with the manufacturer's instructions prior to use.

### *Serum creatine kinase (CK) activity and interleukin-6 (IL-6) concentration*

A 5 mL blood sample was drawn from an antecubital vein into a serum collection tube (BD Vacutainer, Becton Dickinson, East Rutherford, USA) in the resting state at baseline. A further 5 mL blood sample was collected into a serum collection tube immediately post exercise and at 48 h post exercise. Each serum collection tube was kept on ice for 30 min before being centrifuged at 1200 *g* for 15 min. at 4°C. The serum was then aliquotted into 1.5 mL microcentrifuge tubes and stored at -80 deg until subsequent analysis. Samples were assayed using commercially available CK (Catachem Inc., Connecticut, NE, USA) and human IL-6 enzyme linked immunosorbent assay (ELISA) (Quantikine®, R&D systems, Minneapolis, MN, USA) kits according to the manufacturers' instructions. Serum IL-6 concentration was calculated by generating a four parametric logistic standard curve, with a sensitivity of 3.12 to 300 pg/mL. The minimum detectable IL-6 concentration was 0.70 pg/mL. Some participants declined having their blood taken, therefore IL-6 data was available for n = 16 (SP = 9, 3T = 7) CK data was available for n =15 (SP = 9, 3T = 6).

### *Reliability*

The test-retest reproducibility for isometric knee extensor MVC is high, with a coefficient of variation (CV) of 3.9% (Erskine et al., 2009). Likewise, the test-retest reproducibility for assessing voluntary activation via ITT is also high, with a CV of 2.4% (Marshall et al., 2014). The test-retest reproducibility for antagonist muscle (hamstring) co-activation (assessed via surface EMG activity in the biceps

femoris long head) during an isometric knee extension MVC, on the other hand, has been reported to be quite low, with a CV of 14.39% (Erskine et al., 2009).

### *Statistical Analysis*

All statistical analyses were performed using SPSS statistical analysis software (IBM, SPSS statistics, version 22). Two-way mixed ANOVAs determined whether there was a significant main effect for exercise protocol (between subject factor) or time (within subject factor), or an interaction between protocol and time for MVC torque, torque-frequency relationship, CK, IL-6, muscle soreness, RPE, voluntary muscle activation, antagonist muscle co-activation, and peak rate of torque development. In the presence of a significant main effect, Tukeys post-hoc pairwise comparisons were used. Unless otherwise stated, data were expressed as mean ± SD. To identify relationships between PlayerLoad™ and markers of physiological load and fatigue/recovery, Pearson's coefficient of determination was used. Statistical significance was accepted at $P<0.05$.

# Results

### *Sprint times*

There was a significant difference in average repetition time between SP (4.62 ± 0.23 s) and 3T (7.22 ± 0.38 s); $t_{16}$ = -17.901, *P* <0.001 (Table 2). However, there was no significant difference between fatigue decrement between SP (3.03 ± 1.10 %) and 3T (3.58 ± 1.10 %); $t_{16}$ = -1.00, *P* = 0.332

**Table 2.** Repetition times during repeated sprint exercises (seconds). SP = straight-line sprint run protocol 3T = shuttle run protocol. *significant difference.

| | *SP* | *3T* |
|---|---|---|
| ***Average repetition time (s)*** | 4.62 ± 0.24 * | 7.22 ± 0.38* |
| ***Performance decrement (%)*** | 3.03 ± 1.10 | 3.58 ± 1.10 |

*Knee extensor (KE) isometric MVC torque*

There was no main effect for group ($F_{1,16}$=0.764, P=0.395, Fig 1) but there was a main effect for time ($F_{2,32}$=17.754, P<0.001). SP quadriceps torque decreased by 21% from pre (266 ± 55.9 N·m) to post (210 ± 52.5 N·m) and remained decreased by 18% of baseline at post 48 h (211.4 ± 73 N·m). 3T decreased by 17% from pre (277.4 ± 42 N·m) to post (227.4 ± 41 N·m) but recovered to only 7% less than baseline at post 48 h (258.1 ± 43 N·m). There was no significant interaction between group and time ($F_{2,32}$=1.175, P=0.322).

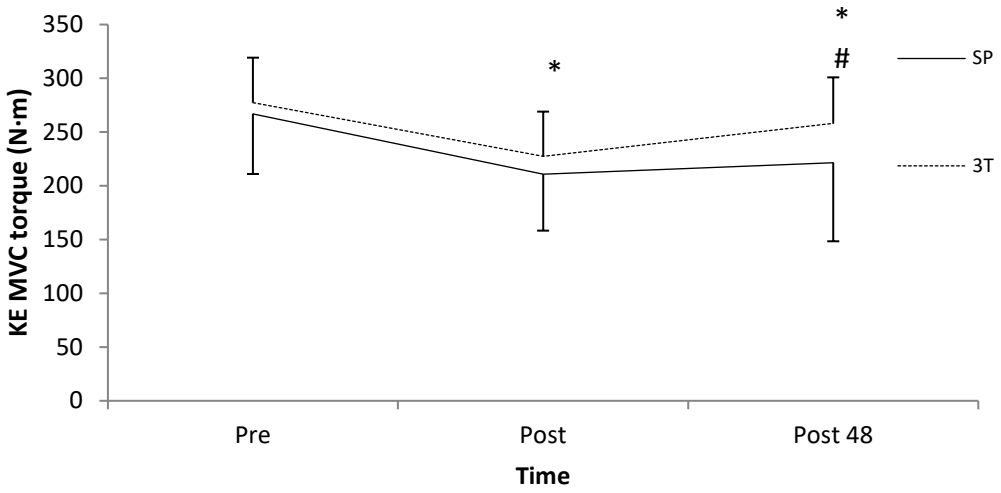


***Figure 1:*** Changes in knee extensor (KE) isometric MVC (maximum voluntary contraction) torque at 80 deg knee flexion after repeated sprint exercise. SP = sprint protocol group; 3T = shuttle protocol group. *significantly different (*P*<0.05) from Pre; #significantly different from Post.

*Knee flexor (KF) isometric MVC torque*

There was no main effect for group ($F_{1,16}$=2.026, P=0.174, Fig 2) but there was a main effect for time ($F_{1.597,29.71}$=19.539, P=<0.001). SP quadriceps torque decreased by 17% from pre (142.7 ± 7.0 N·m) to post (119.0 ± 8.0 N·m) and remained decreased by 18% of baseline at post 48 (116.4 ± 8.0 N·m). 3T torque decreased by 7% from pre (153.1 ± 9.0 N·m) to post (142.0 ± 9.0 N·m) and remained decreased by 14% at post 48 (131.0 ± 10.0 N·m). There was no significant interaction between group and time ($F_{1.857,29.71}$=1.255, P=0.295).

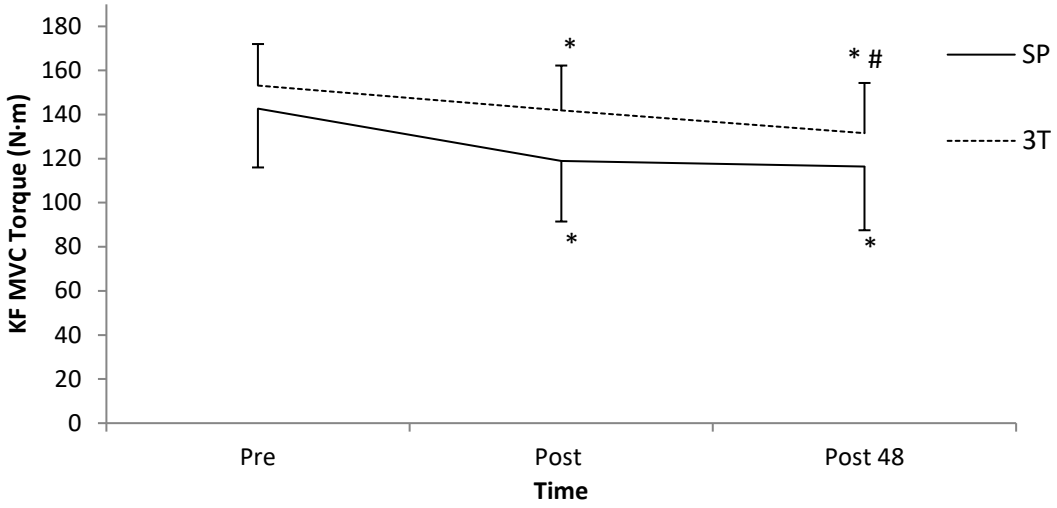


***Figure 2:*** Changes in knee flexor (KF) isometric MVC (maximum voluntary contraction) torque at 30 deg knee flexion after repeated sprint exercise. SP = sprint protocol group. 3T = shuttle protocol group. * = statistical significance (*P*<0.05) from Pre. # = statistical significance from Post.

### *Capillary blood lactate*

There was no main effect for group ($F_{1,17}$ = 0.44, *P* = 0.516, Figure 3), but there was a main effect for time ($F_{1,17}$ = 89.05, *P* = <0.001). SP blood lactate increased from baseline (1.6 ± 0.5 mmol/L) to post (8.9 ± 3.5 mmol/L), while 3T blood lactate increased from pre (1.5 ± 0.5 mmol/L) to (9.9 ± 3.4 mmol/L). There was no interaction between group and time ($F_{1,17}$ = 0.464, *P* = 0.5, Fig 5)

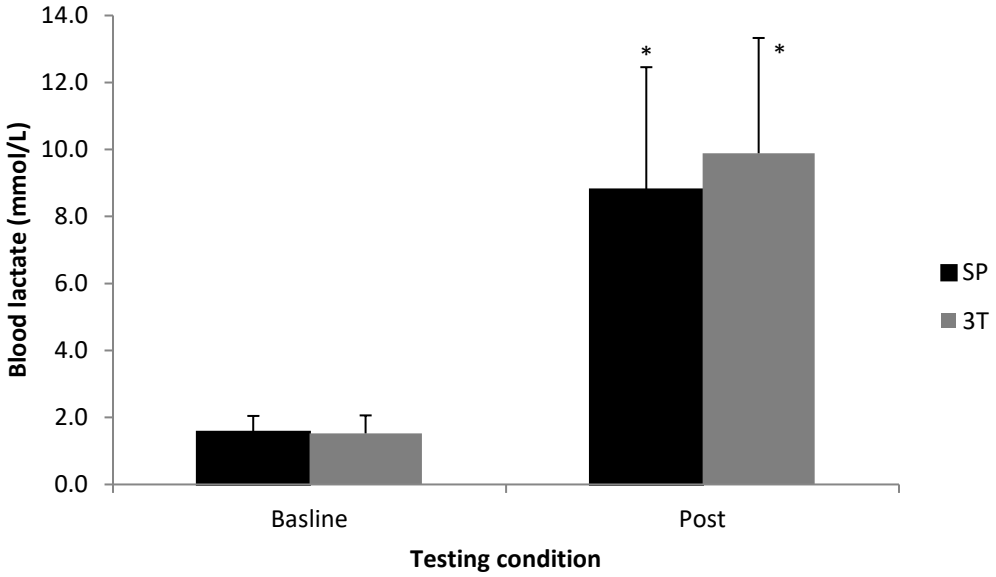


***Figure 3:*** Increases in blood lactate concentrations taken pre and 30 seconds post repeated sprint exercise. SP = sprint protocol group; 3T = the shuttle run protocol group. *significantly different to Pre (*P* <0.05).

### *Range of Motion*

There was no significant difference between group ($F_{1,17}$ = 0.177, *P*=0.679, Figure 4) but there was a main effect for time on ROM ($F_{1,17}$ = 14.382, *P*= <0.001). There was a significant difference between baseline and post (*P*=<0.001, 5.9 ± 0.8) and post to post 48 (*P*=0.008, 3.9 ± 1.1) in both groups. There was no group time interaction ($F_{1.643, 27.938}$ = 8.167, *P*=0.538).

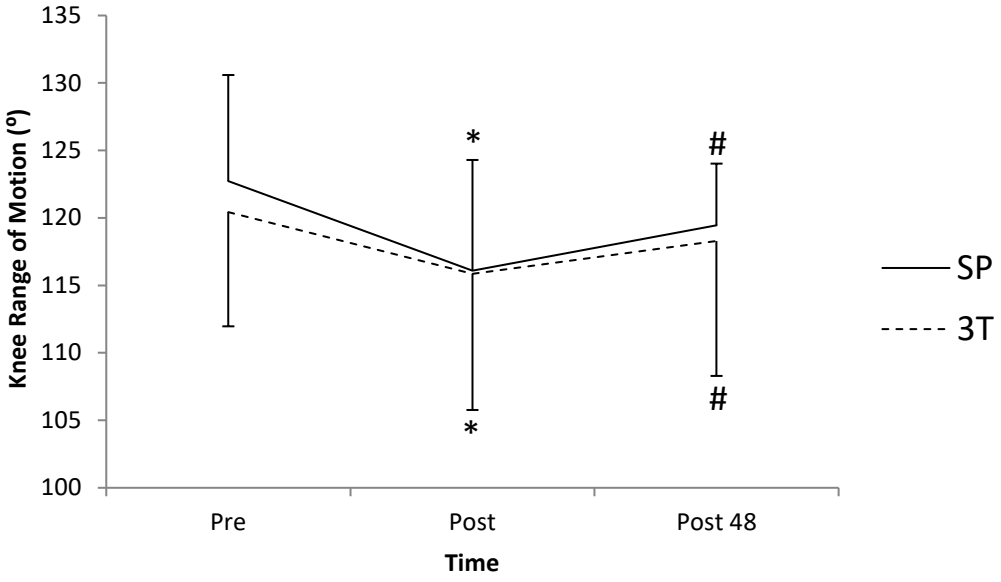


***Figure 4:*** Changes in range of motion around the knee joint following a repeated sprint protocol (°). SP = sprint protocol group. 3T = shuttle protocol group. *statistical significance (*P* = <0.05) from Pre. #statistical significance from Post.

*Voluntary muscle activation*

There was no effect for group ($F_{1,17}$ = 1.780, *P*=0.200) and there was no main effect for time on voluntary activation ($F_{1.57, 26.684}$ = 1.023, *P*=0.356, Fig 5). There was no interaction between group and time ($F_{1.394, 23.706}$ = 0.010, P= 0.966).

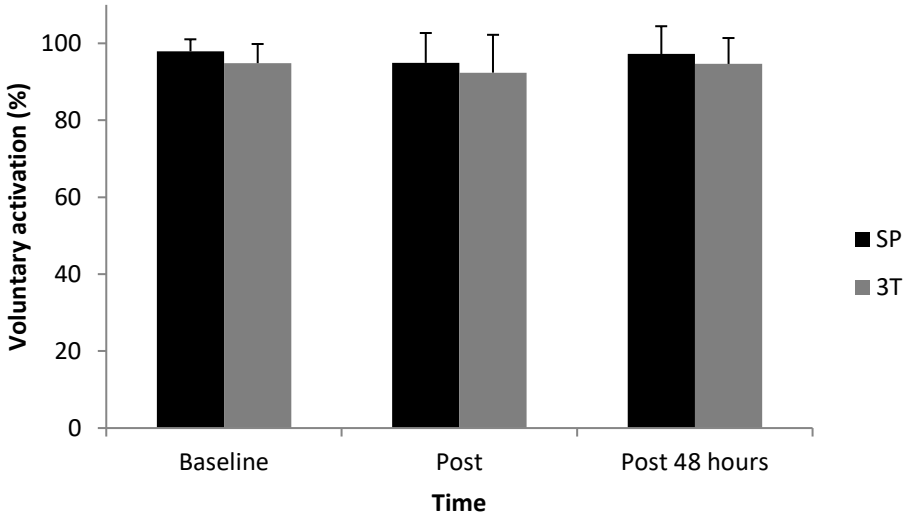


***Figure 5:*** Voluntary activation of the hamstrings determined via the interpolated twitch technique (ITT) at each testing day. SP = sprint protocol group. 3T = shuttle run protocol group.

### *PlayerLoad™*

There was a significant difference between PlayerLoad™ between groups SP (126.9 ± 13.8 AU) and 3T (107.67 ± 12.7 AU) protocols; t (25) = 3.243, *P* = 0.003. See figure 8.

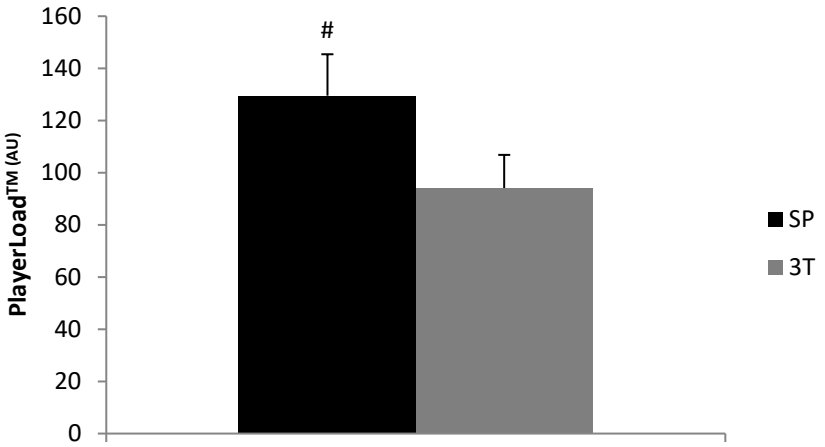


***Figure 6:*** Differences in cumulative PlayerLoad™ (AU) taken during each protocol. SP = sprint protocol group. 3T = shuttle run protocol. #significantly different to 3T.

*Algometer muscle soreness Pressure Pain Threshold (PPT)*

There was no main effect for group, ($F_{1,11}$ = 0.913, P= 0.36, Fig 7) and there was no main effect for time ($F_{1.169, 12.858}$ = 0.205, P = 0.205). There was also no interaction effect ($F_{1.169, 12.858}$ = 1.112, P = 0.33).

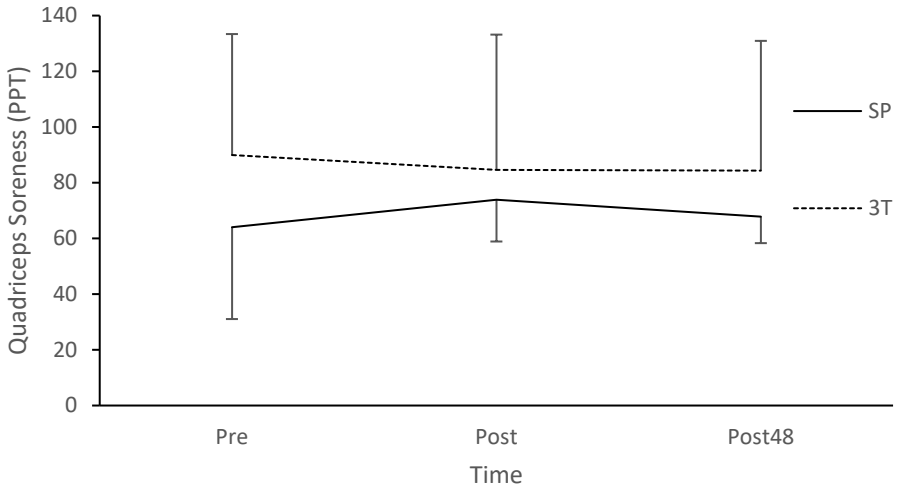


***Figure 7:*** Quadriceps muscle soreness determined at three different time points via algometer. SP = sprint protocol group. 3T = shuttle run protocol group.

There was no main effect for group ($F_{1, 11}$ = 0.507, P=0.419, Fig 8) and there was no main effect for time ($F_{1.861, 20.466}$=3.042, P=0.73). There was also no interaction effect ($F_{1.861,20.466}$ = 1.553, P=0.263).

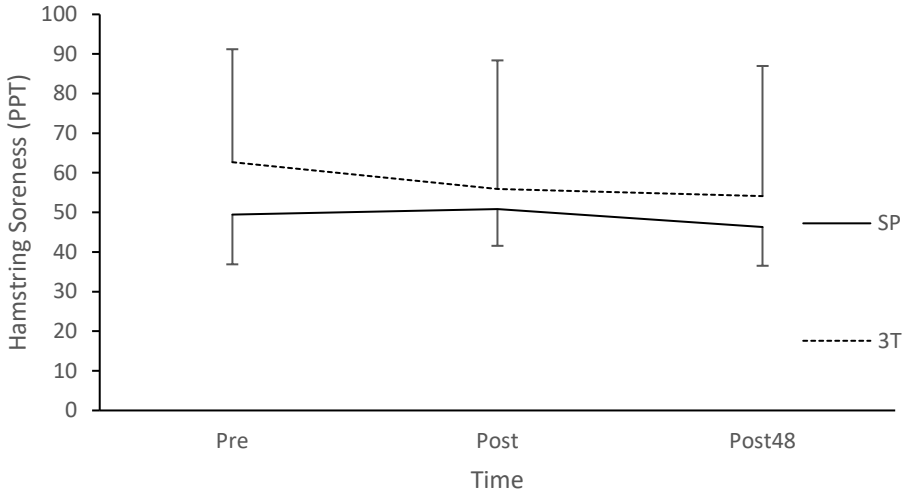


***Figure 8:*** Hamstring muscle soreness at three different time points determined via algometer. SP = sprint protocol group. 3T = shuttle run protocol group. There was no significant effect for time or group.

There was a main effect for group regarding squat soreness ($F_{1,16}$ = 4.773, *P* = 0.044). SP squat soreness was consistently higher for SP than 3T at both post and post 48 h time points. There was a main effect for time with soreness increasing significantly at each time point for both groups ($F_{1.21, 19.361}$ = 25.768, *P* < 0.001, Fig 9) but there was no interaction between group and time ($F_{1.210, 19.361}$ = 6.167, *P* = 0.106).

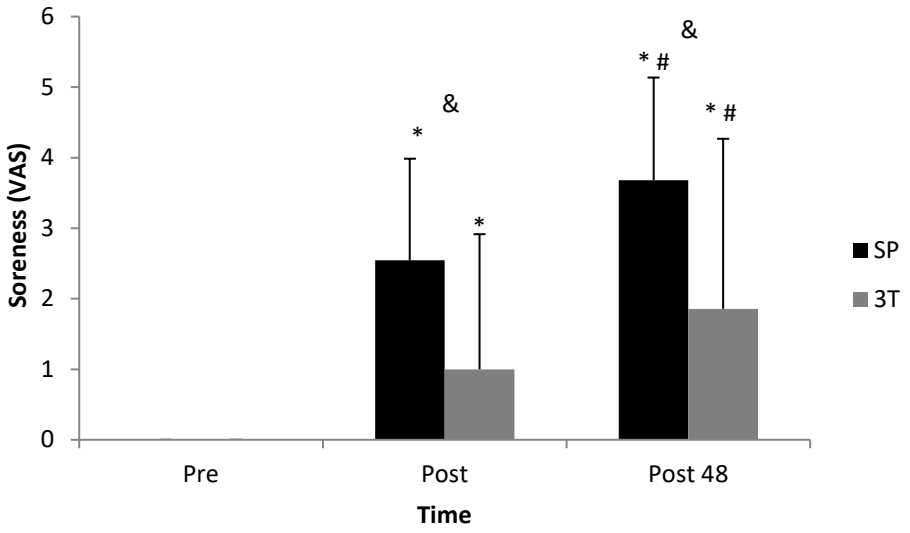


***Figure 9:*** Muscle soreness determined via visual analogue scale (VAS) in cm between groups after completing three unloaded squats. SP = sprint protocol group; 3T = shuttle run protocol group. *significantly different (*P*<0.05) from pre; #significantly different from post. &significant difference between groups.

There was a significant main effect for group ($F_{1,16}$ = 4.576, *P* = 0.048, Figure 10). SP group had significantly higher soreness at post and post 48 than the 3T group. There was a main effect for time with lunge soreness increasing significantly at each time point in both groups ($F_{2,32}$ = 16.362, *P* <0.001, Fig 12) but there was no interaction between group and time ($F_{4.523, 54.329}$ = 1.332, *P* = 0.278).

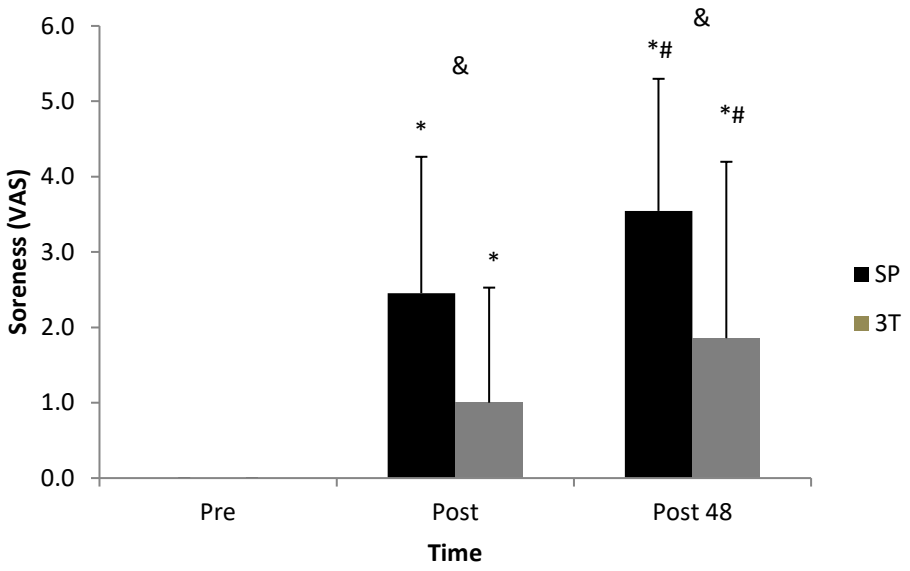


***Figure 10:*** Muscle soreness determined via visual analogue scale (VAS) between groups after completing three unloaded lunges on each leg. SP = sprint protocol group; 3T = shuttle run protocol group. * significant difference (*P*<0.05) from pre; # significant difference from post; & significant difference between groups.

*The torque-frequency relationship*

The absolute torque at each frequency for each participant was normalised to the torque at 100 Hz for that particular individual at that specific time point (Pre, Post or 48 h Post). There was no main effect for group ($F_{1,16}$=0.055, P=0.817) but there was a main effect for time on the torque-frequency relationship ($F_{2,32}$=29.411, P<0.001). As expected, there was a main effect for frequency ($F_{2.2.269,36.31}$=889.36, P=<0.001) but, interestingly, there was a time x frequency interaction ($F_{5.717,9147}$=9.382, P<0.001; Fig. 11a; Fig. 11b), with the torque-frequency curve moving to the right from Pre to Post, and remaining shifted to the right at 48 h Post.

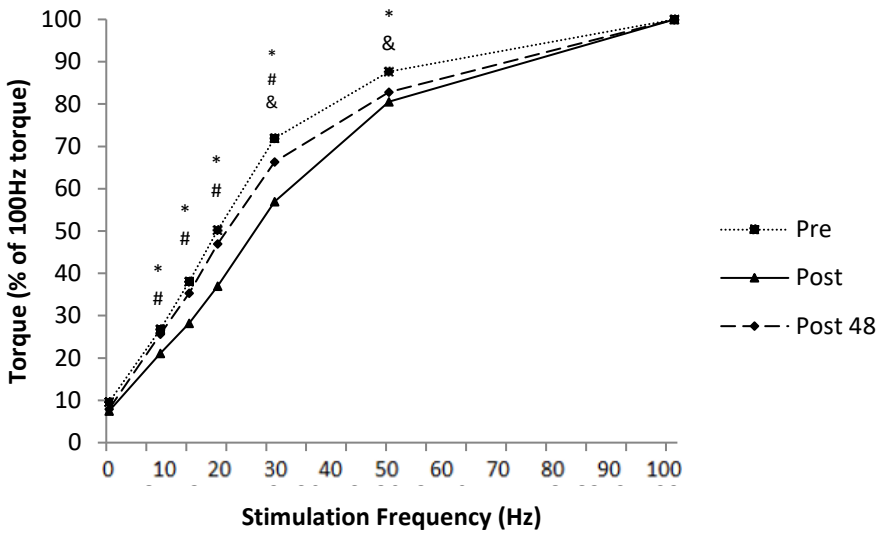


***Figure 11 a:*** The torque-frequency relationship for the hamstrings before (Pre), after (Post) and 48 h after (48 h Post) the sprint protocol (SP). Torque values are expressed as % of the torque produced in response to 100 Hz stimulation that produced torque equivalent to ~20% of MVC. *significant change in the force frequency relationship from pre to post; #significant change from post to post 48 h; &significant change from pre to post 48 h.

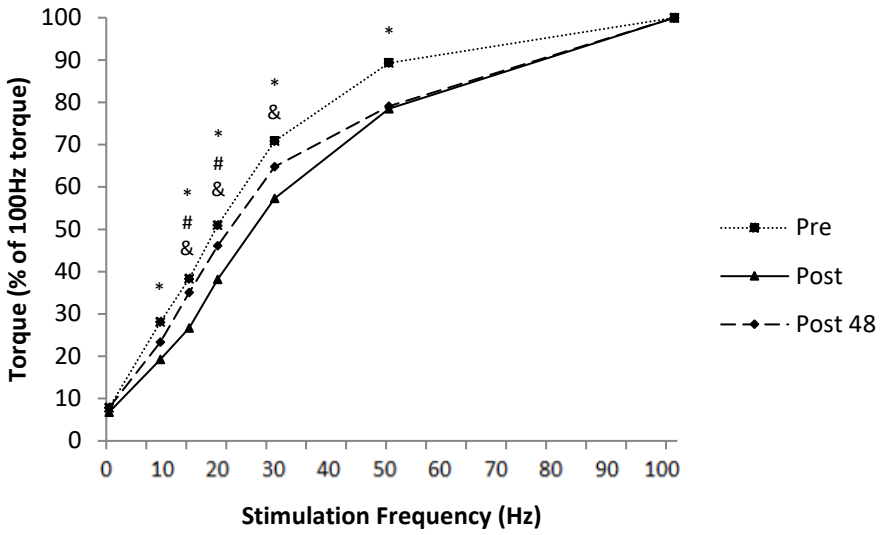


***Figure 11 b:*** The torque frequency relationship for the hamstrings before (Pre), after (Post) and 48 h after (Post 48) the shuttle protocol (3T). Torque values are expressed as % of torque produced in response to 100 Hz stimulation that produced torque equivalent to ~20% of knee flexion MVC. * indicates a significant change in the force frequency relationship from pre to post. # indicates a significant change from post to post 48 h; & indicates a significant change from pre to post 48 h.

### *Heart Rate*

There was a main effect for group ($F_{1,14}$ =7.286, P=0.01), with SP having a consistently higher HR than 3T (Fig. 12). There was an effect for time ($F_{3.876, 54.266}$ = 3.547, *P* = 0.018), but no interaction between group and time ($F_{3.876, 54.266}$=0.717, P=0.58).

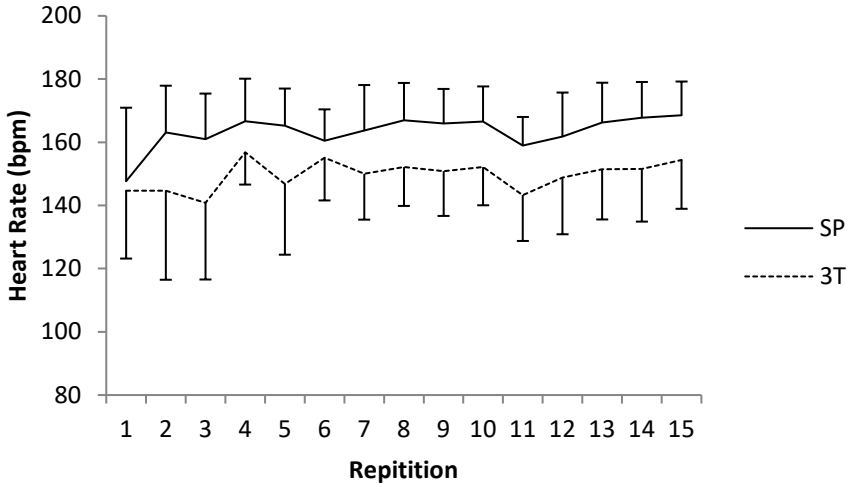


***Figure 12:*** Heart rate values taken at rest and 30 s after each repetition of SP or 3T. SP = sprint protocol; 3T =shuttle run protocol.

*Rating of Perceived Exertion:*

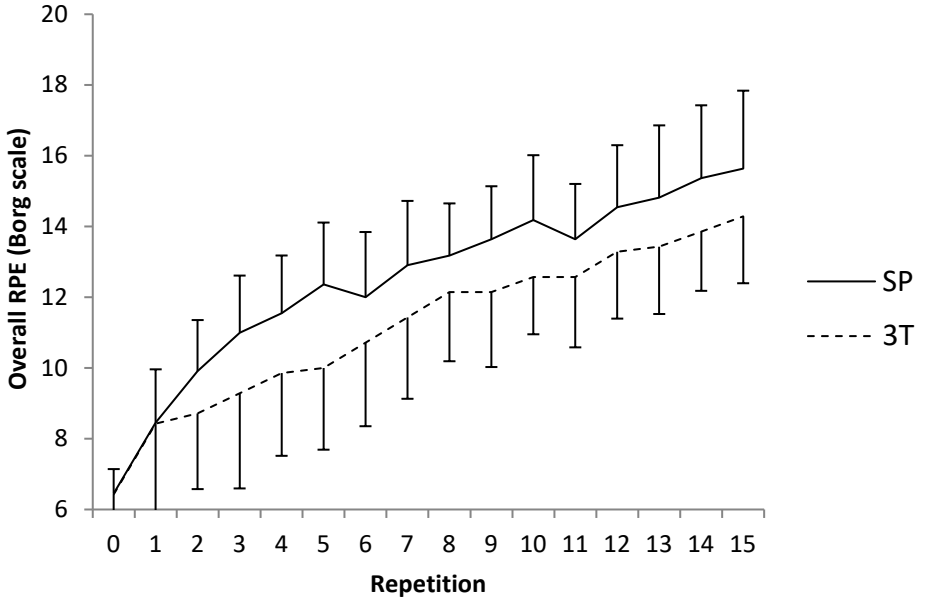


***Figure 13:*** Perceived overall RPE recorded immediately after each repetition according to the Borg scale (Borg, 1982). SP = sprint protocol. 3T = shuttle run protocol.

*Rate of torque development (RTD)*

There was no main effect for group for delta time between force onset and peak torque development during voluntary hamstring MVC ($F_{1,16}$ = 2.624, *P* = 0.125, Table 2). There was also no main effect for time ($F_{2,32}$ = 0.542, *P* = 0.587), and no interaction between group and time ($F_{2,32}$ = 1.533, *P* = 0.231).

There was no main effect for group for delta time between force onset and peak torque development during involuntary hamstring contraction ($F_{1,16}$ = 0.10, *P* = 0.923, Table 2). There was also no effect for time ($F_{1.545, 24.716}$ = 0.176, *P* = 0.784), and no interaction between group and time ($F_{1.785, 28.563}$ = 0.043, *P* = 0.924).

There was no main effect for group for delta time between force onset and peak torque development during voluntary quadriceps MVC ($F_{1,15}$ = 1.218, *P* = 0.287, Table 2). There was also no main effect for time ($F_{1.934, 24.571}$ = 0.465, *P* = 0.596), and no interaction between group and time ($F_{1.638, 24.}$571 = 0.930, *P* = 0.391).

**Table 3.** Changes in for delta time (seconds) between onset of force and peak torque development. Values taken during (i) Maximal isometric voluntary contraction of the hamstrings at 30 deg of knee flexion (ii) propagated single twitch electrical stimulation at rest (iii) Maximal isometric voluntary contraction of the quadriceps at 80 deg of knee flexion. Data presented as mean ± SD.

| | **(i) Voluntary Flexion** | | | **(ii) Involuntary flexion** | | | **(iii) Voluntary extension** | | |
|---|---|---|---|---|---|---|---|---|---|
| | Pre | Post | Post 48 | Pre | Post | Post 48 | Pre | Post | Post 48 |
| **SP** | 0.171 ± 0.042 | 0.179 ± 0.071 | 0.184 ± 0.06 | 0.058 ± 0.020 | 0.057 ± 0.022 | 0.054 ± 0.021 | 0.108 ± 0.061 | 0.093 ± 0.042 | 0.092 ± 0.043 |
| **3T** | 0.173 ± 0.062 | 0.157 ± 0.051 | 0.123 ± 0.200 | 0.056 ± 0.020 | 0.059 ± 0.021 | 0.055 ± 0.020 | 0.093 ± 0.030 | 0.102 ± 0.030 | 0.092 ± 0.022 |

There was no main effect for group for voluntary peak rate of torque development (vpRTD) in the hamstrings ($F_{1,16}$ = 0.296, *P* = 0.594, Table 3). There was a main effect for time ($F_{1.817, 29.072}$ = 3.603, *P*= 0.044) with vpRTD decreasing from pre to post and further decreasing to post 48. However, there was no interaction between group and time ($F_{1.817, 29.072}$ = 0.167, *P* = 0.827).

There was no main effect for group for involuntary peak rate of torque development (ipRTD) in the hamstrings ($F_{1,16}$ = 1.167, *P* = 0.296, Table 3). There was a main effect for time ($F_{2, 32}$ = 7.537, *P* = 0.002), with ipRTD decreasing from pre to post but there was no interaction between group and time ($F_{2,32}$ = 2.370, *P* = 0.11).

There was no main effect for group for vpRTD of the quadriceps ($F_{1,15}$ = 0.53, *P* = 0.478) see table 3. There was no main effect for time ($F_{2,30}$ = 2.029, *P* = 0.149), and no interaction between group and time ($F_{2,30}$ = 3.131, P = 0.062).

**Table 5**. Changes in absolute values of peak rate of torque development (pRTD) at respective time intervals, following completion of the SP or 3T sprint protocol. Values taken from (i) Highest maximal isometric voluntary contraction of the hamstrings at 30 deg knee flexion (ii) propagated single twitch electrical stimulation at rest (iii) Highest maximal isometric voluntary contraction of the quadriceps at 80 deg knee flexion. * Indicates a significant difference to pre. Data presented as mean ± standard deviation.

| | **Flexion pRTD ($N \cdot m \cdot s^{-1}$)** | | | **Involuntary flexion pRTD ($N \cdot m \cdot s^{-1}$)** | | | **Extension pRTD ($N \cdot m \cdot s^{-1}$)** | | |
|---|---|---|---|---|---|---|---|---|---|
| | Pre | Post | Post 48 | Pre | Post | Post 48 | Pre | Post | Post 48 |
| **SP** | 887 ± 101 | 833 ± 92* | 749 ± 86* | 201 ± 17 | 131 ± 20* | 174 ± 20* | 1514 ± 251 | 1598 ± 225 | 1895 ± 233 |
| **3T** | 989 ± 126 | 871 ± 115* | 827 ± 108* | 204 ± 21 | 185 ± 24* | 203 ± 25 | 2263 ± 300 | 1532 ± 268 | 1870 ± 279 |

*Correlations*

PlayerLoad™ correlated positively with HR (Fig. 14.1) and RPE (14.2) during both protocols (individually and combined) but did not correlate with any other measure of physiological fatigue, either in absolute terms or percentage changes over time.

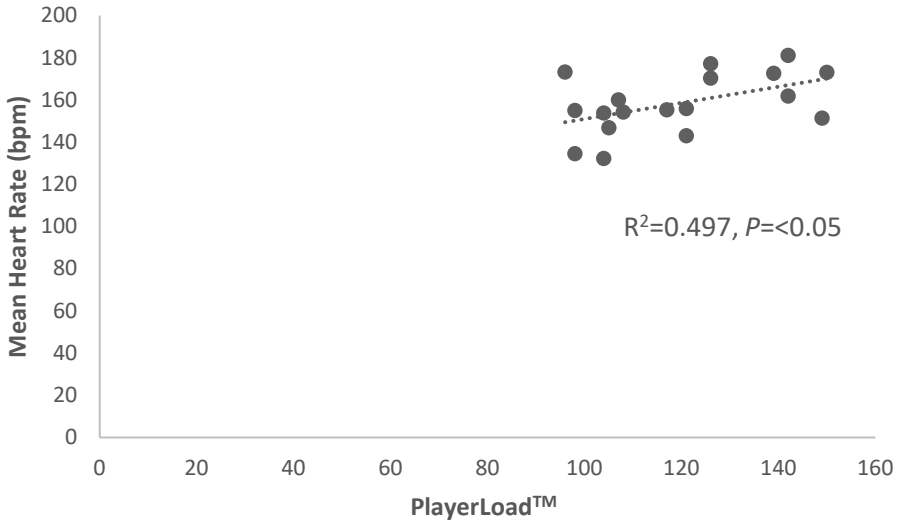


**Figure 14.1:** Relationship between mean heart rate (bpm) and PlayerLoad™ following repeated sprinting (SP and 3T combined). $R^2$ = 0.497, *P* = <0.05.

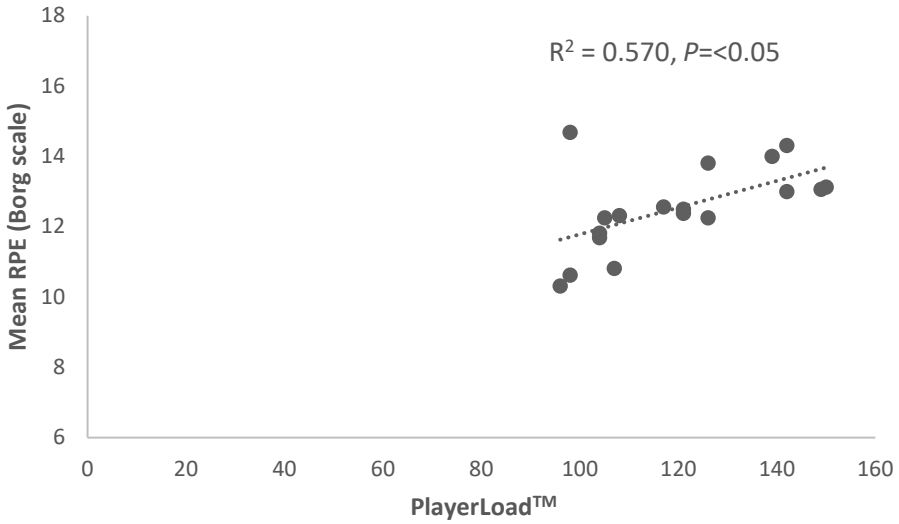


**Figure 14.2:** Relationship between rating of perceived exertion (Borg scale) and PlayerLoad™ following repeated sprinting (SP and 3T combined). $R^2$ = 0.570, *P* = <0.05.

### *Vastus lateralis and biceps femoris EMG*

There was no significant effect for group for BF EMG $M_{max}$ ($F_{1,9}$ = 3.060, *P* = 0.114), and there was no effect for time (F = $_{1.226, 11.035}$ = 0.370, *P* = 0.598). There was also no interaction between group and time (F $_{1.226, 11.035}$ = 0.115, *P* = 0.791).

There was no main effect for group for *vastus lateralis* $EMG_{max}$ (during knee extension MVC) ($F_{1,11}$ = 0.012, *P* = 0.916), and no effect for time ($F_{1.359, 14.944}$ = 2.551, *P* = 0.124). There was also no interaction between group and time ($F_{1.359, 14.499}$ = 2.776, *P* = 0.108, Table 5).

There was no main effect for group for co-activation of level of the *biceps femoris* muscle during knee extension MVC. (F 1,10 = 2.242, *P* = 0.151). There was a non-significant trend for co-activation to increase over time (F $_{2,20}$ = 3.324, *P* = 0.057) but there was no interaction between group and time ($F_{2,20}$ = 0.423, *P* = 0.655).

There was no main effect for group for co-activation level of the *vastus lateralis* muscle during knee flexion MVC ($F_{1,10}$ = 0.372, P = 0.555). There was no effect for time ($F_{1.511, 43.441}$ = 0.035, *P* = 0.926) and no interaction between group and time ($F_{1.450, 14.496}$, *P* = 0.496).

**Table 7**. Surface EMG data for each protocol (3T and SP). (i) represents surface EMG from the biceps femoris (volts) during a maximal voluntary flextion at 30 degree of knee angle, normalised to $M_{max}$(n = 11). (ii) represents surface EMG from vastus lateralis (volts)during a maximal voluntary extension at 80 degree of knee angle (n = 13). (iii) represents co-activation EMG of vastus lateralis during maximum voluntary flexion at 30 degree of knee angle, expressed as % of agonist muscle (n = 13). (iv) represents co-activation EMG of biceps femoris during maximum voluntary extension at 80 degree of knee angle, expressed as % of agonist (n = 13). Data presented as mean ± standard devation.

**(i) BF Flexion $EMG_{max}/M_{max}$** **(ii) VL Extension $EMG_{max}$ (V)**

| | Pre | Post | Post 48 | Pre | Post | Post 48 |
|---|---|---|---|---|---|---|
| **SP** | 0.0320 ± 0.0076 | 0.0247 ± 0.0067 | 0.0335 ± 0.0086 | 0.489 ± 0.358 | 0.542 ± 0.356 | 0.470 ± 0.349 |
| **3T** | 0.0525 ± 0.0301 | 0.0486 ± 0.0456 | 0.0503 ± 0.0249 | 0.607 ± 0.409 | 0.437 ± 0.365 | 0.349 ± 0.442 |

**(iii) Flexion CoA %** **(iv) Extension CoA %**

| | Pre | Post | Post 48 | Pre | Post | Post 48 |
|---|---|---|---|---|---|---|
| **SP** | 4.73 ± 7.79 | 2.60 ± 7.61 | 1.88 ± 7.35 | 4.55 ± 3.25 | 4.63 ± 2.67 | 7.74 ± 4.00 |
| **3T** | 5.43 ± 4.13 | 9.02 ± 8.41 | 6.86 ± 6.42 | 5.25 ± 2.41 | 3.18 ± 7.79 | 5.18 ± 3.99 |

## Blood biomarkers of muscle damage

### *Serum interleukin-6 (IL-6) concentration*

There was no main effect for group ($F_{1,15}$ = 2.262, *P*=0.153, Fig 15) but there was a main effect for time ($F_{1.299,\ 19.483}$ = 4.3, *P*=0.043), with serum IL-6 concentration increasing post exercise in both groups (*P*<0.05). However, there was no interaction between group and time ($F_{1.299,\ 19.483}$ = 0.532, *P*=0.520).

*significant difference from pre; &significant difference from 48 h post.

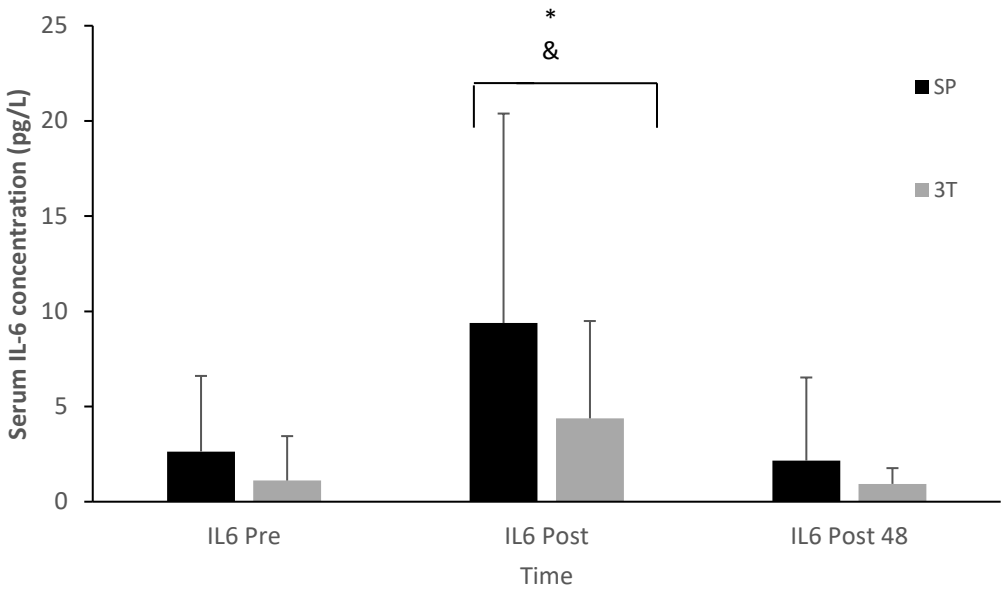


**Figure 15:** Serum interleukin-6 (IL-6) concentration measured pre, post and 48 h post repeated sprint exercise. SP = sprint protocol group; 3T = shuttle run protocol group.

### *Serum creatine kinase (CK) activity*

There was no main effect for group ($F_{1,14}$ = 1.799, *P*=0.201, Fig 16) but there was a significant effect for time ($F_{1.015, 14.203}$ = 5.544, *P*=0.033), with serum CK activity increasing in both groups from pre to post (*P*=0.004) and from post to 48 h post (*P*=0.045). However, there was no interaction between group and time ($F_{1.015,14.203}$ = 0.880, *P*=0.366).

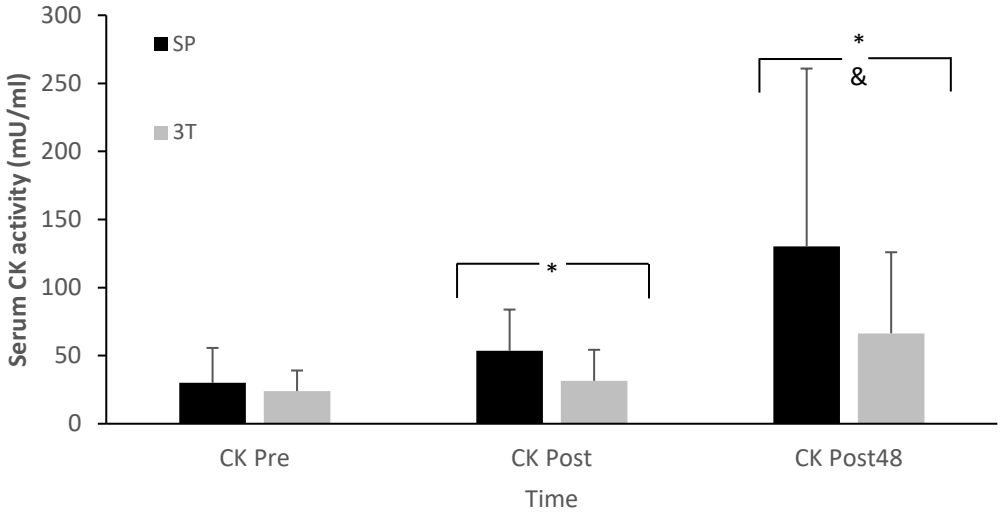


**Figure 16**: Serum creatine kinase (CK) activity measured pre, post and 48 h post repeated sprint exercise. SP = sprint protocol group; 3T = shuttle run protocol group. *significantly different from pre; &significantly different from post.

## Discussion

The aims of this study were to (1) compare the exercise induced fatigue in the hamstring and quadriceps muscle groups following two different repeated sprint protocols matched for distance; and (2) examine the relationship between trunk-mounted accelerometer "PlayerLoad™" data and measures of fatigue. This study found evidence for peripheral fatigue in the hamstring and quadriceps muscle groups following two different repeated sprint protocols, with some symptoms of fatigue persisting for up to 48 hours post exercise. Muscle soreness was the only factor that was higher in the straight-line sprinting group *vs.* the shuttle sprint group, and this difference remained elevated 48 h post exercise. Although both quadriceps and hamstring muscle strength decreased over time following both exercise protocols, these changes were similar between groups. Peak rate of torque development (pRTD) in the hamstrings (both voluntary and involuntary) were impaired following both exercise protocols, while voluntary pRTD of the quadriceps remained unaffected. The torque-frequency relationship (an indicator of low-frequency fatigue) shifted to the right following both protocols, with no difference between protocols. Interestingly, PlayerLoad™ was higher following straight line sprinting than shuttle runs, and was moderately correlated with heart rate and RPE, which were also higher in straight line *vs.* shuttle sprinting. Using the performance decrement formula developed by Fitzsimons et al. (1993), the decrease in sprint performance was very small (3%) and similar between sprint protocols, suggesting sufficient rest had been provided between repetitions in both protocols.

### Peripheral fatigue following repeated sprinting

The strength loss observed in the present study is broadly consistent with other studies that have shown evidence of fatigue in the quadriceps (Howatson and Milak, 2009, Goodall et al., 2015, Keane et al., 2015) and hamstring (Thompson et al., 1999, Timmins et al., 2014) following repeated sprinting and/or shuttle running. Our results showed that isometric maximal strength decreased following both types of sprinting protocols, and remained decreased at post 48 hours in both the hamstring and quadriceps muscle groups (Fig. 1-4). Decreases in hamstring strength from pre to post were 16% and

6% in the SP and 3T protocols respectively. Further decreases at post 48 in both groups were observed, with SP decreasing by a further 2% and the 3T by 7%. Quadriceps strength loss was more pronounced in both groups from pre to post, with a 21% loss in SP group and 17% in 3T. Although we hypothesised that SP would elicit greater hamstring fatigue than 3T (due to the eccentric hamstring contractions during the late swing phase of a high velocity sprint (Yu et al., 2008)), the large eccentric forces generated by the *quadriceps femoris* during the deceleration phase following the sprint probably elicited considerable muscle damage, reflected in the reduction in knee extensor MVC following the straight-line sprinting. Although the average repetition time, and therefore speed was lower (see table 6) during the 3T protocol, the increased number of breaking and turning movements probably elicited as much *quadriceps femoris* fatigue as the deceleration during SP.

Prolonged strength loss is commonly associated with delayed onset muscle soreness (DOMS) and a reduction in range of motion are common findings following high intensity eccentric exercise (Cleak and Eston, 1992, Clarkson and Hubal, 2002, Nosaka et al., 2002). These symptoms are indicative of exercise-induced muscle damage, peaking at 24-72 hours following exercise (Appell et al., 1992, Allen, 2001, Morgan and Allen, 1999, Clarkson and Tremblay, 1988). We observed no significant effect for time or group DOMS measured with the algometer in either muscle group (Fig. 9 and 10). This is somewhat surprising, as performing exercise with eccentric components, such as sprinting and rapid deceleration, would be expected to elicit significant muscle soreness especially in untrained subjects. However, it should be noted that we observed very large variation between participants when using pressure pain threshold (PPT) via an algometer to measure subjective soreness, which may explain our lack of any significant findings. Conversely, and more in line with other research (Jönhagen, 2005, Pearcey et al., 2015, Cleak and Eston, 1992, Thompson et al., 1999, Howatson and Milak, 2009), significant differences across all time points were observed when DOMS was measured using VAS in conjunction with functional movements (Fig. 11 and 12). This was accompanied by a significant reduction in range of motion around the knee joint in both groups immediately after, and at post 48

h. Additionally, the SP group demonstrated significantly higher soreness in the functional movements than the 3T group.

Further evidence of peripheral fatigue/muscle damage was found in the observed changes in CK activity and IL-6 concentration. Serum CK activity showed a significant increase over time in both groups, peaking at 48 hours, which is in agreement with previous work (Andersson et al., 2008, Ascensão et al., 2008, Ispirlidis et al., 2008, Rampinini et al., 2015). Elevated serum CK activity/concentration is a common observation following eccentric or high intensity exercise comprising maximum sprinting (Pohl et al., 1981, Ispirlidis et al., 2008, Rampinini et al., 2015) and is evidence of damage to the muscle fibres, possibly due to increased permeability of the sarcolemma (Manfredi et al., 1991)

Similarly, the increase in serum IL-6 concentration observed immediately following repeated sprints and return to baseline at 48 hours is in line with previous findings (Castell et al., 1996, Gadient and Patterson, 1999, Pedersen et al., 2003, Steensberg et al., 2000, Keller et al., 2003, Hellsten et al., 1997). Observed increases in IL-6 activity post exercise may be due to 2 different factors. IL-6 acts as both a pro-inflammatory and anti-inflammatory myokine that responds rapidly to damage within the muscle (Pedersen and Toft, 2000) and may be evidence of muscle damage. However, evidence also suggests IL-6 plays a role in the signalling process which spares glycogen usage by favouring fat and protein metabolism during exercise (Simith and Miles, 2000). Studies have demonstrated significant muscle glycogen depletion after only a small number of reps when performing repeated maximal exercise (Bogdanis et al., 1996, Gaitanos et al., 1993) which makes it a potential factor in the observed increase post exercise in the present study.

We found no between group differences regarding serum IL-6 concentration or CK activity, suggesting the level of ultrastructural damage to the muscle fibres was similar between protocols. This is somewhat surprising considering the higher muscle soreness observed within the SP group. However, previous studies have highlighted both large inter-person variability in CK values post exercise (Nosaka

and Clarkson, 1996) and a discrepancy between time of peak CK and peak muscle soreness (Newham et al., 1987, Walsh et al., 2001, Evans et al., 1986). Moreover, CK activity has been shown to positively correlate with magnitude of muscle damage (Nosaka and Clarkson, 1996), while muscle soreness was only weakly correlated with biomarkers of EIMD (Nosaka et al., 2002). Additionally, cytokine activity held only a weak relationship with both muscle damage (Bruunsgaard et al., 1997) and creatine kinase activity (Cannon et al., 1986). These findings may explain the lack of group effect regarding CK activity and IL-6 concentration in the present study, despite the between group effect for muscle soreness.

Further disruptions to contractile properties were observed by reductions in voluntary and involuntary pRTD during hamstring MVC at both post and post 48 h following both sprinting exercises. Marshall et al. (2014) reported similar changes in hamstring pRTD following completion of a simulated soccer match. Interestingly, these reductions were not observed in the quadriceps, which may suggest a greater disruption to the hamstrings muscle function following straight line sprinting compared to shuttle running, probably due to the more frequent eccentric hamstring contractions achieved during high-velocity sprinting, as discussed earlier.

Further evidence of peripheral fatigue was shown with the main effect for time observed in the torque frequency relationship, however no between group effect was present (Fig. 13). A more fatigued muscle might be expected to elicit a shift in the torque-frequency relationship to the left (Bigland - Ritchie et al., 1983), due to a reduced rate of relaxation in the fatigued muscle fibres leading to higher forces being elicited at low frequency muscle stimulation (Jones, 1996). However, we found that the hamstring torque-frequency relationship shifted to the right following both sprinting protocols, and this change remained significant at 48 h post exercise. Although a shift to the right is contrary to what might be expected following low-frequency stimulation of the fatigued human adductor pollicis muscle (Bigland-Ritchie et al., 1983), this change is in line with what is commonly found *in vivo* in human muscle following strenuous exercise (Jones, 1996, Dundon et al., 2008, Binder-Macleod et al., 1998). Rather than simply fatiguing the muscle (and therefore having a greater influence on rate of

relaxation at low frequencies than force production *per se*), the strenuous exercise employed in the present study and by others (Westerblad et al., 1991, Binder-Macleod et al., 1998, Dundon et al., 2008) probably caused significant ultrastructural damage that prevented the muscle fibres from producing as much force at low frequency stimulation, thus shifting the torque-frequency to the right rather than to the left, and therefore explaining the discrepancy between the present study and the study by Bigland‐Ritchie et al. (1983)

### Central fatigue following repeated sprinting

As well as peripheral fatigue, a reduction in motivation (e.g. due to the feeling of exhaustion following repeated maximal sprints) may lead to reduced motor unit recruitment and/or firing frequency (Grimby et al., 1981, Gandevia et al., 1996). This would be reflected in reduced muscle activation, measured via surface EMG (Farina et al., 2004) or the interpolated twitch technique (ITT) (Gandevia et al., 1998).The present study showed no significant changes over time or differences between groups regarding *vastus lateralis* or *biceps femoris* surface EMG during MVC. Similarly, no significant changes in voluntary muscle activation (using the ITT) was observed either between groups or over time. Thus, there was no evidence of a reduction in either central activation or recruitment of motor units affecting muscle function in either group, so the observed changes in MVC torque, DOMS, pRTD and the torque-frequency relationship were likely due to peripheral factors rather than suboptimal neural drive. The absence of any decrease in muscle activation in the present study may be explained by the length of the protocol, at only 21 minutes (including sufficient rest time between repetitions), it may not have being long enough to significantly alter neuromuscular activation as central fatigue becomes more prevalent as exercise duration increases (Millet and Lepers, 2004). Despite Goodall et al. (2015) reporting evidence of central fatigue following just two repetitions of 30 m sprints, these measures were taken immediately following the repetition. More in line with the methods in the present study Marshall et al. (2014) reported no change in voluntary activation in the hamstrings following a simulated soccer match. The present study did not record maximum quadriceps voluntary

activation, however reductions of 8% were observed by Rampinini et al. (2011) following a 90 minute simulated soccer match in professional soccer players.

### Exercise intensity

Heart rate is related to rate of perceived exertion (RPE) (Impellizzeri et al., 2004, Alexiou and Coutts, 2008), a subjective rating of exercise intensity that increases with anaerobic demand (Drust et al., 2000). The mean heart rate of the SP group (163 ± 3 bpm) was higher than 3T (146 ± 4 bpm), and was similar to that reported during professional soccer matches (Krustrup et al., 2006). In the present study, RPE was divided into three categories: breathlessness, lower limb and overall (Borg and Borg, 2002). It was demonstrated that performing straight-line sprints elicited higher RPE values in all three RPE measures, which when combined with HR data, suggests a higher perceived physical demand compared to shuttle running. This is somewhat surprising, as other studies have demonstrated that the repeated accelerations and decelerations associated with changing directions place a higher demand upon the body than straight line sprinting (Hatamoto et al., 2014, Stevens et al., 2015). Stevens et al. (2015) showed higher energy demands when performing repeated accelerations and decelerations than straight line running at the same speed. Similarly Hatamoto et al. (2014) concluded that even at low running velocities, the addition of repeated changes of direction drastically increase the physical demands. In the present study, although both protocols were performed at maximal effort, the speed generated during straight line sprinting was much greater, demonstrated by the significant difference in average repetition time (see table 6), than during shuttle running, thus explaining the discrepancy between our findings and those of other studies.

Blood lactate concentration recorded immediately after completing the protocols significantly increased over time in both groups but no between group differences was observed. Other studies have shown a significant increase following repeated sprinting and shuttle running (Buchheit et al., 2010), who reported values higher than our own of (12.2 ± 2.2 $mmol.L^{-1}$). The shorter amount of recovery time used between reps by Buchheit et al. (2010) may explain the higher values observed. In contrast to the present study, shuttle running showed significantly higher lactate values (Dellal et

al., 2010) compared to straight line sprinting when intensity was matched. However, very short rest times between reps, (15 and 30 seconds) compared to the 90 seconds used in the present study, may explain the discrepancy between studies. It may be the rest time in the present study prevented any between group differences in blood lactate.

### PlayerLoad™

Our study showed higher PlayerLoad™ in straight line sprinting compared to shuttle running matched for distance covered. To our knowledge, this is the first study to document differences in GPS external load data from different sprinting exercises. PlayerLoad™ is an arbitrary value that is derived from 3-D measures of instantaneous changes in acceleration divided by the scaling factor, which is then used as an estimation of external load placed upon the body over a period of time. These data suggest that that there was a greater load placed upon the body during maximal 30 m sprinting than 3 x 10m shuttle running. Other studies have highlighted the higher external load placed upon the body when performing rapid accelerations and decelerations associated with changing direction (Varley and Aughey, 2013, Osgnach et al., 2010). The higher PlayerLoad™ values during straight line sprinting were in line with concomitant higher heart rate and RPE values in straight line *vs.* shuttle sprinting. Furthermore, PlayerLoad™ correlated with HR and RPE, supporting previous work by Portas et al. (2012) and suggesting that PlayerLoad™ can predict (to a moderate degree) physiological load during sprinting.

In attempting to explain the higher PlayerLoad™ during straight line sprinting compared to shuttle running, it should be considered that the greater speed achieved during the straight line sprinting *vs.* shuttle running would have led to considerable eccentric force exerted by the quadriceps (and subsequent higher ground reaction forces) during the rapid deceleration phase. These higher forces in SP would have been reflected in higher PlayerLoad™ values, which would have been expected to be reflected in a greater loss of quadriceps femoris MVC. However, knee extensor MVC decreased to a similar degree following both exercise protocols. This may be explained by a higher frequency of quadriceps eccentric contractions during 3T (increased number of decelerations and turns) compared

to SP. Thus, the overall volume of eccentric work experienced by the quadriceps may have been similar between protocols, leading to similar loss of strength and recovery following the different exercise protocols. In contrast, however, muscle soreness (identified via two different methods, i.e. body weight squats and body weight lunges) was greater in SP compared to 3T. This suggests that the deceleration phase during SP did actually elicit greater eccentric damage in the quadriceps than the 3T did. Furthermore, the late swing phase during high velocity sprinting is thought to elicit an eccentric contraction, and is thought to be the main cause for biceps femoris muscle strain associated with sprinting (Yu et al., 2008, Chumanov et al., 2012, Chumanov et al., 2007). This may explain the higher muscle soreness values with body weight squats following SP *vs.* 3T.

The second aim of this study was to investigate the relationship between PlayerLoad™ and physiological measures of fatigue following different sprinting exercises. Despite greater DOMS (both quadriceps and hamstring) in SP vs. 3T (suggesting greater fatigue in SP), PlayerLoad™ did not correlate with any physiological measure of fatigue/biomarker of muscle damage (including maximum voluntary strength, blood lactate concentration, IL-6, CK, muscle soreness, voluntary muscle activation, (in)voluntary rate of torque development, or the torque-frequency relationship) following either or both exercise protocols. Other studies in contact sports, such as rugby union (Jones et al., 2014) and Australian rules football (Young et al., 2012) found GPS unit were related to blood CK concentration. The present study showed no correlation between PlayerLoad™ and CK activity but the nature of high impact collisions and whole body movements occurring in rugby and Australian Rules football would elicit much higher CK concentrations than sprinting alone, which may explain the discrepancy in results.

Although PlayerLoad™ may have reflected greater absolute eccentric forces during SP than 3T, the higher frequency of lower eccentric forces experienced by the muscles during 3T may not have been reflected in PlayerLoad™ values. Thus, a similar volume of eccentric contraction-induced fatigue/damage may have led to similar strength loss between groups but this was not related to PlayerLoad™. Furthermore, the physiological mechanisms underpinning neuromuscular fatigue are

complex and there is no single factor that is considered more important than any other (Jones, 1996). Therefore, although a trunk-worn device utilising acceleration data and a specific algorithm appears to predict physiological load during a maximum sprinting task, it may not be sensitive enough to predict actual damage or fatigue experienced by the neuromuscular system. It should also be considered that while PlayerLoad™ may be used to indicate individual loading from an athlete, caution should be taken when comparing between individuals. Barrett et al. (2014) found high variance in the observed values between individuals and determined due to the positioning of the GPS unit, between the scapulae, the influence of upper body movements during running make comparisons between athletes difficult.

In the present study, while evidence of peripheral fatigue was present following both protocols, the only between group effect present was in DOMS. This is partly in agreement with our hypothesis that the straight line sprinting would elicit higher levels of fatigue, more specifically DOMS, in the hamstrings than repeated shuttle running. This hypothesis was based on the findings that sprinting contains high velocity eccentric contractions of the hamstring muscle group, which causes muscle damage (Proske and Allen, 2005) soreness (Talag, 1973, Hamill et al., 1991) and disruptions to contractile properties (Newham et al., 1987, Saxton et al., 1995, Allen, 2001). Our data show that while shuttle running is highly demanding and fatiguing, it did not elicit as much muscle soreness as maximum repeated sprinting, which can be the most restrictive symptom of fatigue on future exercise bouts prior to full recovery.

The majority of studies investigating EIMD and fatigue have focussed on the quadriceps muscle (Erskine et al., 2009, Erskine et al., 2010a), probably due to its superficial (easy access) location and, because it is the largest of the four heads, it is commonly considered representative of the quadriceps muscle group as a whole (Erskine et al., 2009). To our knowledge, only one previous study had used electrical stimulation to assess hamstring muscle activation before and after exercise (Marshall et al.,

2014), therefore our findings add to the limited research of hamstring specific fatigue following repeated sprints.

### Limitations of the present study

The present study found no reduction in surface EMG in either the *biceps femoris* or *vastus lateralis* during MVC despite observed reductions in maximal torque. In addition to the electrodes being positioned under the limb at a seated position, the greater amount of subcutaneous fat around the hamstrings compared to the quadriceps may have affected the EMG signal during transmission to the skin (Farina et al., 2004). When using EMG to measure muscle activation, it is vital that the re-application of surface EMG electrodes post and 48 h post exercise are as accurate as possible. While every care was taken to mark out with permanent marker and clean locations for the electrodes, sometimes post exercise reapplication proved problematic due to participant's sweat, which may have reduced the surface contact of the electrodes.

While participants were instructed to eat two hrs before arrival, and refrain from exercise for 48 hours prior to testing days and to not change their habitual diet before or during the study, no additional measures were taken to control diet, therefore muscle/liver glycogen concentration was not accounted for. It has been shown that muscle glycogen content can be a critical factor in time to fatigue (Bergström et al., 1967) and resynthesis can be supressed under effects of EIMD (Krustrup et al., 2011). A glycogen depletion protocol, controlled diet in the 24 hours prior to testing and in the 48 h duration of the study, plus muscle biopsies to determine the time course of glycogen re-synthesis may have been of interest in the present study, however, these additional requirements would have been impractical considering the timing and number of physiological measurements included in the present study. Furthermore, it is possible repeated biopsies may have influenced other markers of muscle damage.

No evidence of central fatigue was observed in the present study, however, voluntary activation was measured in the hamstrings and not the quadriceps muscle group. Evidence of decreases in voluntary activation of the quadriceps has been demonstrated following repeated sprints (Perrey et al., 2010) and simulated soccer matches (Rampinini et al., 2011). However, these data were unavailable in the present study due to impracticality of using the ITT in both the hamstrings and quadriceps muscle groups. Additionally, other studies which reported evidence of reduced central output have measured voluntary activation either immediately after (Rampinini et al., 2011, Brownstein et al., 2017) or between repetition (Goodall et al., 2015). In the present study, due to methodological restraints, and requirements to take blood and move rooms, there was a time delay between end of exercise and VA measurement of 30 minutes. This may have allowed sufficient recovery time for the CNS to prevent any observed decreases in VA post exercise.

No change in rate of torque development was observed in the quadriceps. However, it should be considered that rate of torque development was taken during isometric contractions on an isokinetic dynamometer, which required the limb to be fixed with secure strapping and a shin pad. The pad, which was positioned on the anterior side of the leg, would absorb the very early stages of movement during leg extension, making RTD in the quadriceps potentially unreliable.

## Summary and Practical Applications

Understanding the fatigue response from soccer-specific exercise is vital in advancing the knowledge of the demands of soccer match-play. This study highlights the peripheral fatigue induced from two different sprinting protocols, and adds to the limited data on post exercise fatigue of the hamstring muscle group, and relationship between GPS external load and physiological response to exercise. Our data suggest that performing repeated changes of direction at a lower speed may be as fatiguing as maximum sprinting, yet may not elicit muscle soreness post exercise. We found that PlayerLoad™ was moderately correlated with RPE and heart rate, thus potentially supporting its use a practical measure of exercise intensity.

## Conclusion

This study demonstrated (i) peripheral fatigue in the quadriceps and hamstrings after performing repeated straight line sprinting and shuttle running; (ii) straight line sprinting elicited greater muscle soreness than shuttle running; and (iii) PlayerLoad™ is moderately correlated with heart rate and RPE during repeated sprinting but not with central or peripheral fatigue measured immediately after and 48 h after these types of exercise.

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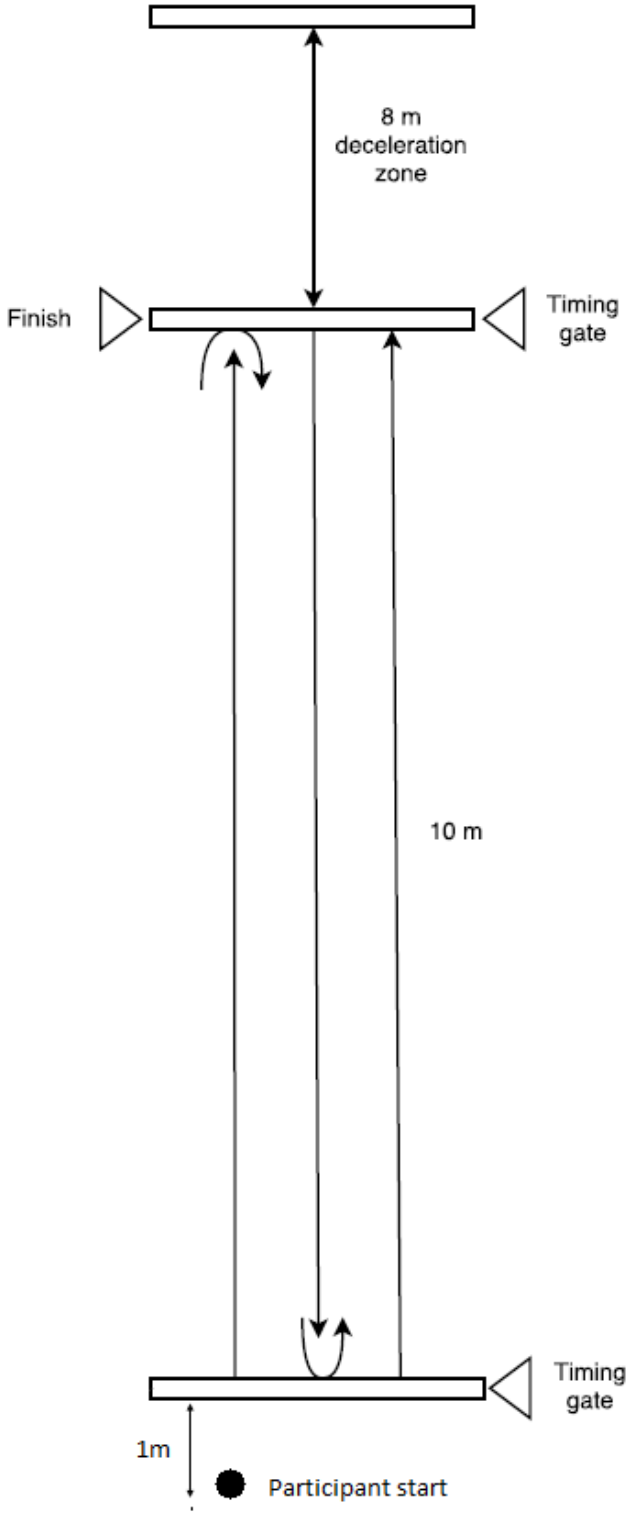
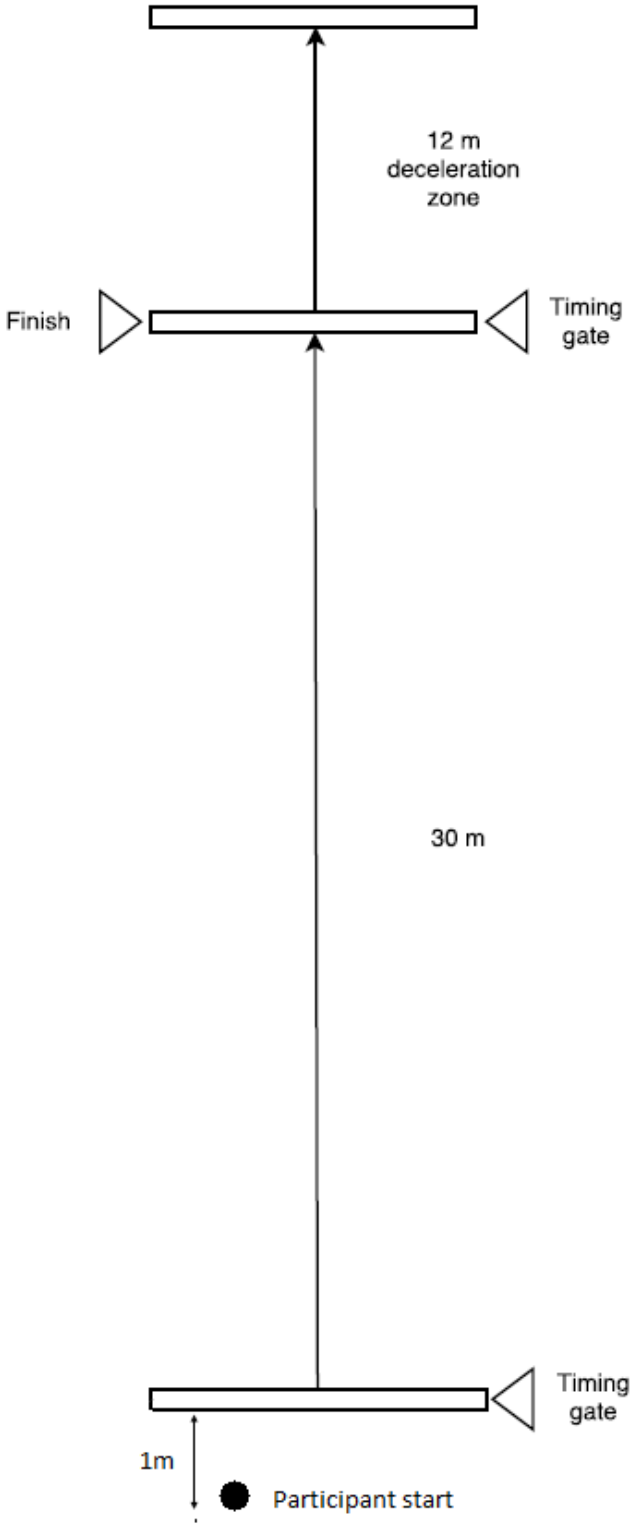
**Appendix:**

## *Schematic of 3T protocol*



## *Schematic of SP protocol*

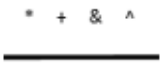


## *Schematic of experimental procedure*

Schematic representation of experimental procedure.

Testing day


Post 48 testing day




Key:

• Blood sample

# blood lactate

& MVC

^ ITT and LFF test

+ Functional soreness

Exercise protocol

Warm up

Biomechanical measure (in collaboration with another study. Data not presented)