## The Effects of Training Stimulus on the Time Course of Recovery in Central and Peripheral Fatigue in a Group of Elite Youth Male Footballers

By

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#### **Buckinghamshire New University**

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Abstract

#### Abstract

**Purpose:** To discover the effect of different training stimuli on the time course of recovery of fatigue from competitive youth football matches and the role that the Central Nervous System plays in this. Method: Ten participants from a Premier League football academy were recruited. Each participant completed preliminary trial testing prior to six testing sessions completed for each of the two different training weeks. Transcranial magnetic stimulation, femoral nerve stimulation and electromyography were used to measure electrical activity within the vastus lateralis during maximal voluntary contractions of an isometric knee extension. The results were recorded to assess neuromuscular function pre-match, post-match and during prior to each training session in the following week. **Results:** There were significant differences dependent on the day of testing for MVC (p = <0.001), TMS %VA (p =0.020), PS deficit (p = 0.043) and PS %VA (p = 0.030). Central and peripheral fatigue were reduced in participants in comparison to previous studies. However, peripheral fatigue was significantly lower than central fatigue for the following values: Post-match deficit in TW-A (p = 0.008); Tuesday deficit in TW-A (p = 0.044); Post-match deficit in TW-AN (p = 0.043), Tuesday deficit in TW-AN (p = 0.047); Post-match %VA in TW-A (p = 0.026); Tuesday %VA in TW-AN (p = 0.031). There were no significant differences between the two training weeks for deficit or %VA for either TMS or PS, suggesting that both methods of training elicited similar central and peripheral recovery. **Conclusion:** These results agree with previous research that individuals recover from both central and peripheral fatigue as time passes from a previous match. This study finds that the effects of peripheral fatigue were less than central fatigue which opposes previous studies, possibly due to the elite level and the age of the athletes.

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#### Author's Declaration

I declare that this thesis and the work presented in it are my own and have been generated by me as the result of my own original research.

I confirm that:

1. This work was done wholly or mainly while in candidature for a research degree at this University.

2. Where any part of this thesis has previously been submitted for a degree or any other qualification at this University or any other institution, this has been clearly stated.

3. Where I have consulted the published work of others, this is always clearly attributed.

4. Where I have quoted from the work of others, the source is always given. With the exception of such quotations, this thesis is entirely my own work.

5. Where elements of this work have been published or submitted for publication prior to submission, this is identified and references given at the end of the thesis.

6. This thesis has been prepared in accordance with the Coventry University and Buckinghamshire New University.

7. I confirm that if the submission is based upon work that has been sponsored or supported by an agency or organisation that I have fulfilled any right of review or other obligations required by such contract or agreement.

Callum Sharpin

Introduction

#### **1.0 Introduction**

Football has been described by Gifford (2008) as a two team, invasion-based team game composing of two 45-minute halves. The winner of each match is dependent on the number of goals scored, meaning many high intensity efforts are required of both attacking and defending players (Buchheit et al. 2010). A study by Mohr et al. in 2003 found that during a football match, players change direction every 5 seconds, completing approximately 1,300 actions, including 200 at high intensity. Since then, the physiological demands of the game have increased, and a more recent study by Bangsbo (2014) stated that elite level football players cover 2.43 kilometres (km) at high intensity, with players in the Premier League covering 681 metres (m) at speeds greater than 19 km/h, with 248 m of that at a maximal sprint. In a standard 90-minute Premier League game, players cover on average 10.5 km, which is 34% more than the 7.8 km covered by amateur players (Playertek 2017).

Of the eleven players per team, each is placed under different physiological demands dependent on playing position (Buchheit et al. 2010). For example, an elite under 19 midfield player would expect to cover ~9732 m during a game in comparison to ~8910 m as a centre back, suggesting the requirement for a greater aerobic capacity (Rago et al. 2017). Midfielders would also expect to cover 1090.41  $\pm$  119.12 of high-speed running compared to a striker who would cover 533.76  $\pm$  166.90, suggesting that anaerobic capacity is a crucial component for a midfielder (Rago et al. 2017). Even considering the positional variation, the averages in elite players are higher than their youth counterparts (Bangsbo, 2014). It is, therefore, crucial that for players to rise from lower-league football to the international team, or from an academy to the first team, they are conditioned to withstand the demands of the game to make the step from the youth academy to the first team.

Introduction

#### 1.1 Fatigue

Goodall et al. (2017) define muscular fatigue as a failure to maintain force through isotonic, isometric and concentric contractions, therefore requiring an enhancement of voluntary effort to retain power output. Fatigue is considered as one of the major limitations in maintenance of high-performance endurance sport and can be divided into both central and peripheral components. However, Abiss and Laursen (2005) and Thomas et al. (2017) add that fatigue is an inevitable and negative consequence of physical activity. Fatigue develops through intramuscular metabolic alterations, such as the accumulation of blood lactate or depletion of phosphocreatine during high intensity training and matchplay, which inhibits the ability to produce forceful contraction.

In team sport, Waldron and Highton (2014) define fatigue as a reduction of maximal force or power associated with sustained exercise, resulting in a decline in performance. With increased in technology in football, including Global Positioning Systems (GPS) which for tracking players match-play physical performance, including high intensity running distance (km/h) and total distance (km). This allows fitness coaches to identify reductions in performance to be tracked from baseline values at the beginning of the match, to the end (Bradley and Noakes, 2013). Decreases in physical outputs such as contraction force and sprint performance can be used to identify physical decline of a player, suggesting acute fatigue in the periphery (Mohr et al. 2003), named by Noakes and St Clair Gibson (2004) as "Catastrophe Theory".

Performance in elite level football can be limited by numerous factors, both psychological and physical including issues such as motivation levels or a reduction in intramuscular energy stores (Jones and Vanhatalo 2017, Josefsson et al. 2017). However, it has been stated by Abbiss and Laursen (2005), Algahannam (2011) and

Introduction

Thomas et al. (2015) that stress placed upon the physiological and metabolic systems through the high intensity demands of the sport can induce high fatigue levels in athletes. This limits the ability of an individual to maintain maximal power output, distal control of muscle function and high-speed running capacity through a complex interaction of the central and peripheral nervous systems (Goodall et al., 2015; Noakes and St Clair Gibson 2004). However, whilst there have been numerous studies which have attempted to define fatigue in a generic and sporting setting, there are numerous differing definitions. Abbiss and Laursen (2005) suggest that these differing definitions are created by individual disciplines to suit their specialism. They go on to debate that a physiologist will view fatigue as the failing of a specific physiological system, while a psychologist will see it as a 'sensation of tiredness'.

With the numerous differing views on what fatigue is, it is crucial for sports scientists and exercise professionals to have a clear definition before attempting to investigate the cause and effects.

Therefore, for the purpose of this study, the working definition of fatigue will be "the inability of an individual to reproduce maximal force through an isometric Vastus Lateralis (VL) contraction". This can be identified through a reduction in the percentage of voluntary activation or an increased difference between voluntary contraction and simulated contraction.

Consequently, recovery from fatigue will be defined as the ability of the individuals to recreate baseline values for maximal voluntary contraction, percentage of voluntary activation and difference between voluntary contraction and simulated contraction.

#### 2.0 Literature Review

In this chapter there will be a breakdown of previous research into fatigue and it's causes. This will be followed by a review of current literature on fatigue in sport, football and youth sport and will conclude with the research aims and hypotheses.

#### 2.1 Fatigue: A Historical Account

Fatigue has been defined as a progressive exercise-induced reduction in voluntary activation (Andersen et al 2012). Goodall et al. (2017) expand to define muscular fatigue as a failure to maintain force through isotonic, isometric and concentric contractions, therefore requiring an enhancement of voluntary effort to retain power output. However, Abiss and Laursen (2005) and Thomas et al. (2017) add that fatigue is an inevitable and negative consequence of physical activity. The differing views on an overall definition of fatigue come from numerous disciplines and sports, with those professionals in team sports such as football having an alternative view to those in individual sports such as tennis (Reid and Duffield, 2017). For example, a tennis player may compete for up to five hours, with stroke outcome being an indicator of fatigue (Reid and Duffield, 2017), whilst a football player may show a reduction in high speed running regularity.

A study by Mathers and Grealy (2014) has stated that fatigue in golfers can be both physical (e.g. coordination and poor timing) and mental (e.g a shift in attentional focus and lack of concentration) due to the repeated mechanics of the swing and the golfer covering a distance over 8,000m over a four-hour period (Sell et al. 2008). Physical fatigue can be defined as a state of disturbed homeostasis which can be attributed to work and work environment, i.e. a stress on the body (Astrand et al. 2003). Taylor

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(2010, p.8507) continues to add that stress is defined as "a negative emotional experience accompanied by predictable biochemical, physiological, cognitive, and behavioural changes, directed either towards altering the stressful event or accommodating to its effects" (Mathers and Grealy, 2014, p.1). With competitive golf demanding high levels of cognitive and motor control, fatigue can therefore be a limiting factor of performance. Conversely, invasion sports such as football involve similar decision making to golf, albeit at a much greater speed which, combined with large distances covered at high-intensity, are predominating factors for cognitive and physiological fatigue (Bradley and Noakes, 2013).

In addition to decision making and greater match play duration, there is also central nervous system fatigue. Central factors at spinal and supraspinal levels also contribute to a decrease in force development through exercise-induced reduction in voluntary activation (Goodall et al. 2017). The inability of the central nervous system to produce suitable and rapid responses to certain stimuli diminishes as the duration and intensity of activity increases due to a reduced output from the motor cortex, resulting in decreased excitability of cells within the motor neurone pool (Ament and Verkeke, 2009). Therefore, individuals will react more slowly to changes in the environment, such as movement of an opponent or the ricochet of the ball.

#### 2.1.1 Causes of Fatigue

Noakes (2000) states five different models to explain fatigue, with different professionals favouring different models. For example, the cardiovascular/anaerobic model of fatigue will be more strongly advocated by cardiovascular and respiratory physiologists than any of the other models. Noakes (2000) continues to suggest that

it is improbable that only one physiological system or scientific discipline is entirely responsible for the deterioration of human exercise performance. Decorte et al. (2012) suggest that the type of contraction, duration of exercise, nature of the activity and the number of limbs involved can all affect the rate of fatigue development.

These five models mentioned previously fit in to two major forms of fatigue; central and peripheral (Ament and Verkeke, 2009; Rampinini et al. 2011; Boccia et al. 2017; Thomas et al, 2017). These typically occur in conjunction with each other, with central fatigue occurring substantially post-game, but with a marked improvement after 24 hours, whilst peripheral fatigue remained below baseline measurements up to 72 hours post-game (Thomas et al. 2017). By understanding which area of the body is fatigued, it is therefore possible to intervene with training programmes to limit the effects of this.

As previously suggested, there are two major systems that contribute to the overall fatiguing of an individual: central fatigue and peripheral fatigue (Ament and Verkeke, 2009; Rampinini et al. 2011; Boccia et al. 2017; Thomas et al, 2017). Each of these has numerous pathways in which fatigue can manifest, for example within the central system, fatigue is supraspinal, meaning physical burnout of an athlete can result from fatigue in the cerebral cortex resulting in a decrease in voluntary activation of muscles (Andersen et al 2012). Conversely, there are multiple peripheral systems that can tire individually or collaboratively, resulting in termination of exercise (Desai and Bottoms, 2016; Inzlicht and Marcoca, 2016). These include the cardiorespiratory system (McArdle et al. 2010; Fletcher et al. 2013) and depletion of energy reserves (Lovell et al. 2013).

In addition to the two physical causes of fatigue, there is a psychological, or perceptual element that must also be considered (Alain and Matran, 2012). The ability of the individual to perceive the threat of any physiological feedback, such as blood lactate concentrate or depletion of substrates, will be dependent on the cognitive development of the athlete (St Clair Gibson et al. 2017). Each of the physical and psychological causes of fatigue will be covered in the following sections.

#### 2.2 Central Fatigue

As previously stated, Central fatigue refers to inadequate activation of motor neurons and therefore reduced neural drive to the muscle (Abd-Elfattah et al. 2015). Ament and Verkeke (2009) expand to state that central fatigue is the loss of contraction force or power caused by processes proximal to the neuromuscular junction. It has been suggested via The Hill Model of Exercise Physiology (Figure 1) that these supraspinal factors also affect those proximal processes leading to fatigue (Maugan, 2009).

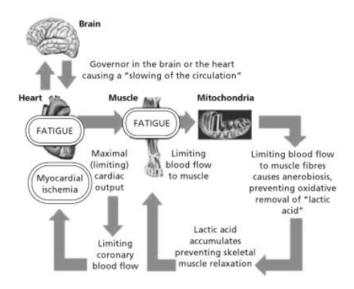


Figure 1 The Hill Model of Exercise Physiology (Maughan, R., 2009. The Olympic Textbook of Science in Sport)

In comparison to the vast literature on peripheral fatigue and the tests used to record this, there is a paucity of research on the importance of central fatigue. Extended periods of exercise initiate the production of lactate in working muscles, inhibiting their ability to produce effective and efficient movements (Hogarth et al. 2015). However, researchers are identifying the role that brain activity and the stimulation of the CNS play in the effectiveness of muscles at the latter stages of activity (Nybo 2003). This can be due to the speed at which signals are transmitted through and across neurones or the speed at which the cerebral cortex is able to initiate this impulse due to the concentration of neurotransmitters (Nybo 2003).

Measurement of the Hoffman's Reflex (H-Reflex) is a valuable tool to evaluate neurologic function in individuals (Palmieri et al. 2004). The H-Reflex is a reflectory reaction of muscles after an electrical stimulation of the sensory fibres, either centrally or peripherally, namely the la afferents in the muscle spindles, which differs from the spinal stretch reflex as it bypasses the muscle spindle (Palmieri et al. 2004). The stretch reflex occurs after a stretching of the muscle, whilst the H-Reflex only occurs post-electrical stimulation (Palmieri et al. 2004), though both action potentials travel along afferent neurones to  $\alpha$  motor neurones, resulting in a twitch response from the muscle, described by Sherwood (2011) as the result of a single presynaptic action potential briefly initiating a muscle contraction. When an external nerve stimulation is too low to elicit a response, no change is seen in an electromyography (EMG) graph. However, afferent neurones relay the impulse to the spinal column due to the high sensitivity of the CNS, which assumes a stretching of a nerve has taken place (Palekar, 2011; Grif Alspach, 2013). The CNS then relays an action potential to the muscle to protect it, which is of a long latency time (up to 35ms), but high voltage, known as the H-Reflex (Palmieri et al, 2004; Misra and Kalita, 2010).

As the intensity of the stimulation increases, more muscle fibres are innervated, eventually resulting in an artificial maximal contraction, removing the H-Reflex from the EMG but producing a large M wave, occurring 3-6ms after the onset of stimulation (Kamen and Gabriel, 2010). This occurs due to action potentials being propagated towards the spinal cord. If the activity in the la afferents causes the presynaptic terminal to depolarise, neurotransmitters are released into the pre-synaptic cleft at the la- $\alpha$  motor neuron synapse. This causes excitory postsynaptic potentials in the motor neurons, which if substantial enough generate an action potential and therefore the muscle contracts and displaces and H-Reflex trace on the EMG (Palmieri et al. 2004).

During voluntary contraction, when receiving a supramaximal external stimulation, the action potentials collide, producing both a large M wave and a response similar to the H-Reflex, known as the V-wave, as this only occurs during voluntary contraction (Bawa et al. 2015).

Whilst information is gathering in adult populations, central fatigue is still a relatively unexplored area in children, with papers explaining its safety and its use for disorders, but not its role in sport. It is crucial to understand the role of the CNS in children, especially in elite athletes, as the state of maturation both physically and psychologically can play a large role in the development of them as a future professional footballer (Malina et al. 2005).

#### 2.2.1 Fatigue in the Cerebral Cortex

During a sustained voluntary contraction, motor neurone firing rates decline, which is often confused with central fatigue. This has been described by Gandevia et al (1996) as a useful change which matches the muscle activation to the slower contractile

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properties of muscles during fatigue. When stimulating the motor cortex during maximal voluntary contractions, extra force occurs (Gandevia et al. 1996; Badawy et al. 2012). This suggests that no part of the motor pathway (from motor cortex to myofibril) is acting at maximum, in spite of maximal effort, suggesting that at least some degree of fatigue occurs 'upstream' of the motor cortex.

An increment in force during cortical stimulation during MVC shows submaximal cortical output. This, however, may be due to decreased cortical excitability, defined by Badawy et al. (2012) as a level of neuronal excitability in the motor cortex controlled by the flow of ions through ion channels or through a complex interaction through cells. As a MVC continues, the force generated diminishes, however the increment in force during cortical stimulation suggests that the failure of voluntary activation occurred as a failure of drive to corticospinal neurones as a consequence of the muscle remaining fatigued (Taylor and Gandevia, 2001). Therefore, when in a fatigued state, the effects of corticospinal output on motor neurone excitability are evident (Taylor and Gandevia, 2001; Jubeau et al. 2014).

During brief MVCs, cortical output is close to optimal, however during prolonged exercise, cortical output becomes suboptimal. EMG responses through cortical stimulation in fatigued muscle return to baseline quickly, whilst voluntary output remains limited, suggesting reduced cortical activation plays a role in central fatigue (Jubeau et al. 2014).

#### 2.2.2 Neuromuscular Fatigue

During repeated muscular activation, it is common to see a reduction in force production, defined by Enoka and Duchateau (2008) as neuromuscular fatigue.

Ayramo et al. (2017) add that excitation-contraction (E-C) coupling and cross-bridge cycling are the two major processes attributed to high intensity running or jumping exercise.

The reduction in E-C coupling has been attributed to an inadequate activation of the calcium (Ca2+) release channels, which may be caused by changes in the cytoplasmic environment, particularly due to low Adenosine Triphosphate (ATP) and high magnesium (Mg2+) levels (Lamb, 2002). A decreased efficiency of E-C coupling will result in a decrease of contractile force, therefore playing a crucial role in muscle fibre fatigues (Ament and Verkeke, 2009). High concentrations of hydrogen (H+) and Mg2+ blocks Ca2+ release. This in turn limits contractile ability due to a reduced capacity of nerves to propagate action potentials alongside the sarcolemma (Ament and Verkeke, 2009). Debold (2002) suggests that the actin-myosin cross-bridge is compromised during exercise, preventing efficient muscle contraction. This is due to both the decreased duration of the attachment of the actin and myosin filaments during the cross-bridge cycle and therefore, the duration of the cross-bridge cycle itself (see section 2.3.1.2).

The occurrence of fatigue, therefore, results in this impairment of force production, however, previous evidence suggests that contractile history can facilitate force production, known as post-activation potentiation (PAP) (Hancock et al. 2015). Consequently, whilst fatigue and PAP are direct opposites, it is possible for both to occur concurrently (Hodgson et al. 2005). There are two measures of neuromuscular output which calculate the effect that contractile history has on force production; i) muscle twitch force and ii) H-Reflex amplitude (Robertson et al. 2013). The muscle twitch is the result of a presynaptic action potential initiating a brief muscle contraction

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(Sherwood, 2011) with the H-Reflex being the consequent refectory reaction following electrical stimulation of the muscle (Palmieri et al. 2004) (see section 2.2).

Artificial stimulation, as explained in Sections 2.6.1 and 2.6.2, can therefore be used to measure the fatigue in working muscles. When an individual completes an MVC and receives an external stimulation, the two action potentials collide, creating an increase in power output from the muscle, similar to the H-Reflex (Kamen and Gabriel, 2010). This is known as the V Wave as it only occurs during voluntary contraction, with the magnitude of this referring to the extent at which the motor neuron pool in the working muscle has been utilised (Bawa et al. 2015). However, the use of the V-Wave in research is as common as the H-Reflex, due to the sensitivity of the V-Wave to supraspinal input, unlike the H-Reflex which is dependent on events at spinal level (Bawa et al. 2015)

#### 2.2.3 Psychological and Perceptual Fatigue

Whilst the supraspinal limiting factor of exercise can be physical due to blocking of action potentials and the stimulation of type III and type IV nerves, which induce a decrease in motor neuron firing rate and an inhibition of the output from the motor cortex (Ament and Verkeke, 2009), a psychologist will also argue the psychological impact on fatigue (Alain and Matran, 2012). The perception of effort - the intensity of subjective stress or discomfort felt during physical activity - can play a key role in the termination of exercise (Alain and Matran 2012). With multiple physiological systems, including the cardiovascular system being put under large amounts of stress, it has been argued by Ulmer (1996) that fatigue is a safety mechanism to prevent exhaustion. Through self-regulation, participants perceive afferent physiological

feedback from the periphery, such as blood lactate concentration, substrate depletion and respiratory rate in a process known as teleoanticipation (Ulmer 1996). The ability of the individual to accurately perceive the threat that each of these items of feedback has on the homeostasis is influenced by psychological factors such as experience and cognitive development (St Clair Gibson et al. 2017).

An individual's perception of effort has been described by Pageaux (2014) as a limiting factor of performance. This is commonly measured using a Rate of Perceived Exertion (RPE) scale (Borg, 1982), which allows individuals to quantify their feelings of effort through any given task. The scale has the same qualities as metrics in physics and physiology, with an absolute zero and the same difference between all measurement points (Borg, 1982). There are two scales, ranked from either 6-20 or 1-10 (Borg, 1982). The 6-20 scale can be used to denote heart rates of 60-200 beats per minute (BPM), with differences between the RPE and BPM being used to instruct the participant on the intensity of their work (Borg, 1982). However, this has been considered too confusing for the lay population, with the 1-10 scale being more commonly used as studies have shown that the general population has a greater understanding of rating situations of objects out of ten (FluidSurveys, 2014).

A negative consequence of using the RPE scale, however, is that it is crucial to explain what each number relates to in terms of intensity and therefore Borg (1982) argues that it is important to use both numbers and explanations for each metric. Alain and Matran (2012) found that individuals tend to increase their RPE in relation to duration of exercise remaining rather than the intensity of their effort, meaning that with five minutes gone RPE would be low, but with five minutes remaining it would be higher, regardless of the intensity. It can be difficult within a team environment to obtain accurate RPEs, as often individuals will copy the score given by teammates. This may

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be so the individual feels like they conform to the normative scores and therefore do not stand out as an individual who struggled with the session or found it much easier than others did (Husain, 2012).

A study by Christian et al. (2014) has found that the perceptions of peripheral discomfort, such as muscular pain, are independent of the sense of effort, however power output runs in parallel with this sense of effort. Therefore, individual effort appears to be consciously regulated alongside the sense of effort, which has been presented as being generated via efferent copies of a centrally-originating signal (Proske, 20015). This suggestion has been based on the premise that "motor areas in the brain would directly influence sensory areas, producing sensations independently of afferent sensory feedback" (Ross and Bischof, 1981). Christian et al. (2014) argue therefore that, whilst the Borg scale RPE is a comprehensively used tool when measuring perceptual effort, it would be beneficial to use two separate measures for the "sense of effort" and "perceptions of physical discomfort" in place of RPE which combines the two.

#### 2.3 Peripheral Fatigue

Ament and Verkeke (2009) have defined peripheral fatigue as "the loss of contraction force or power caused by processes distal to the neuromuscular junction". This has been reiterated by Froyd et al. (2016) who have defined peripheral fatigue as an impairment in muscle function rather than a reduction in the capacity of the central nervous system to activate these muscles. Peripheral fatigue is measured as a reduction in evoked force responses to electrical or magnetic supramaximal stimulations delivered to the motor nerve to relaxed muscles (Froyd et al. 2016).

As suggested previously, the peripheral causes of fatigue include the cardiorespiratory system (McArdle et al. 2010; Fletcher et al. 2013), depletion of energy reserves (Lovell et al. 2013) and hyperthermia (Piil et al. 2017). The ability, for example, of the cardiovascular system to transport blood to working muscles plays a key role in the intensity at which an individual can work (McArdle et al. 2010). This in turn results in a greater rate of phosphocreatine depletion and therefore a weaker muscle contraction (Ament and Verkeke, 2009; Thrietl 2011). The peripheral causes of fatigue will be discussed in detail in the following sections.

#### 2.3.1 Cardiorespiratory System and Fatigue

Endurance performance can be determined by the capacity of the athlete's heart to pump unusually large volumes of blood and oxygen (O<sub>2</sub>) to the working muscles (McArdle et al. 2010; Fletcher et al. 2013). The variation in blood pressure (BP) and heart rate (HR) responses to different training stimuli have been heavily investigated and are thought to be affected by both central and peripheral mechanisms (Rio-Rodriguez et al. 2016). The role these mechanisms play is dependent, however, on the configuration of the activity, for example the volume, intensity, number of repetitions and duration of rest periods (Rio-Rodriguez et al. 2016).

With the heart responsible for the transport of O<sub>2</sub> to working muscles, performance will therefore be impacted by the cardiac output (CO), defined by Vincent (2008) as the amount of blood that leaves the heart in one minute and is calculated as HR x Stroke Volume (SV) (McArdle et al. 2010; Fletcher et al. 2013). This is dependent on four variables (Vincent 2008):

1) Heart Rate – the number of times the heart beats per minute.

- 2) Contractility the force at which the heart contracts.
- 3) Preload the degree of myocardial distension prior to contraction. This is largely dependent on the amount of ventricular filling, allowing the walls of the cardiac muscle to stretch, producing a more forceful contraction. Preload must not be confused with venous return. Venous return must be equal to the cardiac output in the same period as there is no space for blood storage in the heart, whilst preload is one component of cardiac output.
- Afterload the force against which the ventricles must act to eject blood, dependent on diastolic arterial blood pressure.

Cardiac output will increase during exercise, due to an increased HR and SV, however eventually, the heart is unable to supply enough O<sub>2</sub> to working muscles, which will differ dependent on exercise intensity (Ward et al. 2015). This is described by the Hill Model of Exercise Physiology (Figure 1), which suggests that the development of skeletal muscle anaerobiosis caused by myocardial ischemia prevents the conversion of pyruvate to acetyl-coA to enter the Krebs Cycle. Lactic acid is produced to prevent a build-up of pyruvate which would slow down energy production, however extra demands are placed on the heart in an attempt to deliver more oxygen to working skeletal muscles which are working anaerobically (Maughan, 2009).

To ensure the protection of the heart during the ischemic conditions, a "governor" in the brain reduces cardiac function (Maughan, 2009). The ability of the cardiac muscle to stretch and recoil to produce a large cardiac output is compromised after prolonged effort (Rowland et al. 2002). A study by Douglas et al. (1987) found that left ventricular (LV) dimension was significantly decreased at the end of an Ironman race which would suggest that cardiac output would be compromised. Additionally, a study by Rowland et al. (2002) found that trained cyclists displayed an increased LV dimension

compared to untrained individuals at the beginning of exercise, however the dimensions of the LV in both populations gradually declined during the testing protocol, suggesting this is a common trait in all individuals. Therefore, whilst Lovic et al. (2017) argue that trained athletes will have an increased systolic and diastolic LV dimension, along with an increased wall thickness and stroke volume and a decreased resting heart rate, the decrease in LV dimension throughout exercise suggests this may contribute to the reduced O<sub>2</sub> in working muscles.

Acton (2013) argues that the lack of O<sub>2</sub> supply to working muscles may be caused by impaired respiratory muscle function, suggesting that not enough O<sub>2</sub> is inspired to meet the demands of the individual. Inspired air reaches the alveoli in the lungs and after gaseous exchange, the oxygenated blood is then transported to the left atrium of the heart through the pulmonary circulation system (Rogers, 2011). Carbon dioxide-rich blood will return to the lungs via the right ventricle which is then expired to be removed from the body (Rogers, 2011). This process is automatic and respiratory rate adjusts to internal and external stimuli through feedback via chemoreceptors which interpret changes such as oxygen and carbon dioxide levels in the blood (Ward et al. 2015). During exercise, the requirement of oxygen of the working muscles results in decreased oxygen and increased carbon dioxide levels which therefore requires a greater respiratory rate to address this balance (Rogers 2011; Ward et al. 2015).

The diaphragm and intercostal muscles facilitate breathing, changing the volume of the lungs to change the air pressure, which either forcefully removes air or inhales it (Rogers, 2011). These muscles however play a part in other functions such as talking and chewing and are therefore susceptible to fatigue in the same way as skeletal muscle (Rogers, 2011). The volume of air during a deeper breath, common during exercise, is greater than during regular inhalation at rest (Ward et al. 2015). The

respiratory rate can also increase, allowing up to 25 times more air to be expired per minute (Rogers 2011). As respiratory rate increases to meet the required oxygen uptake there comes a threshold at which the individual is unable to meet the oxygen demands and therefore the central governor will terminate the exercise in order to preserve the homeostasis of the body (Maughan, 2009). This threshold is affected by the VO<sub>2</sub> max of the individual, with those able to consume and use greater amounts of oxygen per minute being able to function at a greater capacity for longer (Trimmel et al. 2017).

## 2.3.2 Energy Systems and Fatigue

To produce energy for movement, the breakdown of Adenosine Triphosphate (ATP) into Adenine Diphosphate (ADP) and inorganic phosphate ions (iP) occurs in the working muscles during exercise. The human body utilises differing energy systems to produce energy from ATP, each working best under different conditions (Lovell et al. 2013).

Ament and Verkeke (2009) preceded Lovell et al. (2013), suggesting that there are three main energy systems responsible for force production. However, whichever system is predominant, there will always be an increased volume of ADP and iP, whilst during anaerobic glycolysis, an increase in hydrogen ions (H+) and pH levels is also evident (Ament and Verkeke, 2009). The increase in ADP, iP and H+ will consequently affect the duration of the actin-myosin crossbridge (Ament and Verkeke, 2009). The attachment of the myosin head to the actin filament is then facilitated by iP to produce a power stroke, shortening the muscle. ATP attaches to the myosin head to weaken the crossbridge, allowing reattachment to initiate new movement. The ADP, iP and H+

affect the duration of the attachment and detachment of the actin and myosin filaments during the cross-bridge cycle and therefore, the duration of the cross-bridge cycle itself (Ament and Verkeke, 2009).

An increase in ADP has been shown to slow down the period of actin and myosin attachment (Thiriet 2011), whilst an increase in iP depresses isometric contraction force and the turnover of the enzyme ATPase (Thiriet, 2011). Therefore, whilst the presence of ADP has been proven to increase force production in working muscles, it simultaneously reduces the velocity of the cross-bridge cycle, making contractions slower, which coincides with a reduction in force producing capacity caused by large concentrations of iP and H+ in the muscle fibres (Ament and Verkeke, 2009; Thrietl 2011). Within a period of extended exercise, such as a football match, the depletion of energy reserves can limit overall performance during the later stages of a match.

#### 2.3.2.1 Depletion of Anaerobic Energy Reserves

During the initial period of up to two minutes of activity, the body produces ATP anaerobically, through two different systems (Plowman and Smith, 2010). Initially, ATP stored in myosin cross-bridges is broken down, resulting in ADP and iP in a process known as phosphorylation (Greger and Windhorst, 1996; McArdle et al. 2009). Simultaneously, phosphocreatine (PC) is broken down by the enzyme creatine kinase, producing creatine (C) and iP (Rakhmetov et al. 2015). The energy from this reforms ADP and iP to create ATP which can be broken down once more to produce energy for exercise (Greger and Windhorst, 1996; McArdle et al. 2009). This system produces energy anaerobically for approximately ten seconds of activity before stores of PC are

depleted, resulting in the transition to energy production through anaerobic glycolysis (Greger and Windhorst, 1996; McArdle et al. 2009).

Anaerobic Glycolysis is the process of converting glycogen stored in the body to glucose, which is then broken down to synthesise ATP in the working muscles (Plowman and Smith, 2010) by several enzymes, including phosphofructokinase (PFK). The by-products (pyruvate and hydrogen ions) lower the pH and turn the muscle increasingly acidic. As exercise intensity increases, greater amounts are produced. Therefore, due to the anaerobic nature of the system, there is insufficient oxygen to break down the pyruvate and synthesise additional ATP (Milioni et al. 2017). A large amount of  $CO_2$  is then produced in the Krebs Cycle, which is exhaled. This acidity in the muscle, caused by anaerobic metabolism, may lead to an increased respiratory rate during the latter stage of exercise, due to the stimulation of respiratory chemoreceptors (Beachey 2012).

Lactate is formed when the availability of oxygen for respiring cells is inadequate, through the combination of one pyruvate molecule and two hydrogen ions by the enzyme lactate dehydrogenase, acting as a temporary buffering system to reduce acidosis (Ament and Verkeke, 2008). The lactate threshold is defined as the workload at which lactate production is exactly in equilibrium with the lactate consumption of the tissues (Sharkey and Gaskill, 2013), above which the blood lactate concentration increases. Chen et al. (2016) suggest that lactate is consumed by cells in two ways to increase energy supply. Firstly, the lactate may be metabolised to pyruvate by cytosolic lactate dehydrogenase (LDH), which can then be utilised by working muscles to produce ATP. Secondly, lactate may be imported into the mitochondria and metabolised by mitochondrial LDH.

The accumulation of lactate ultimately exceeds the ability of the body to process it and therefore limits the functioning capacity of working muscles and limits the force and velocity of muscle contraction (Plowman and Smith, 2010). This is known as the onset of blood lactate accumulation (OBLA) and is defined by Ament and Verkeke (2008) as the workload at which blood lactate concentration exceeds 4mmol/L. It is understood that workloads above the OBLA can only be maintained for a limited time before termination of exercise is enforced, meaning that fatigue sensations increase directly with the accumulation of exercise-associated metabolites such as lactate (Sharkey and Gaskill, 2013). The OBLA is individual for each athlete, meaning that some will have to terminate exercise earlier than others. This threshold can be found during laboratory or field-based testing, usually before the start of the football season.

The presence of nitric oxide (NO) aids with the uptake of blood glucose to aid muscle production once stores of muscle ATP have diminished (Ament and Verkeke, 2009). However, 1-2 hours after the commencement of exercise, the availability of blood glucose is also reduced (Ament and Verkeke, 2009), which can be delayed by the consumption of glucose-containing food or drinks.

### 2.3.2.2 Depletion of Aerobic Energy Reserves

As previously suggested (see section 2.3.2.1), the accumulation of waste products limits the ability of working muscles to maintain forceful output (Plowman and Smith, 2010). However, between the lactate threshold (LT) and OBLA, the active muscles work both anaerobically and aerobically, utilising ATP from both systems (Lovell et al. 2013). The point at which this occurs depends on the LT of the individual and their

ability to work under anaerobic conditions for extended periods of time (Plowman and Smith, 2010).

Once working under aerobic conditions, the ability of the individual's cardiorespiratory system to provide muscles with oxygenated blood becomes much more crucial (Sharkey and Gaskill, 2013). Therefore, there is a requirement to test for a person's LT alongside their VO<sub>2</sub> max, defined as the maximum volume of oxygen an individual can utilise during exercise, measured in millilitres of oxygen in one minute per kilogram of bodyweight (Trimmel et al. 2017). By identifying the LT and VO<sub>2</sub> max, it is possible to provide individualised aerobic training, targeted at increasing these values, resulting in a greater energy-producing efficiency for each of the energy systems (Plowman and Smith, 2010). As previously suggested, during pre-season testing, individual VO<sub>2</sub> max and LT scores can be identified, allowing fitness coaches to prescribe individualised running speeds, durations and distances to help increase base levels of aerobic and anaerobic fitness.

Through aerobic training, increases in tidal volume, minute ventilation and capillary density increase blood flow to working muscles (Coburn and Malek, 2012). It has therefore been argued that "aerobic fitness enhances recovery from high intensity intermittent exercise through increased aerobic response, improved lactate removal and enhance PCr re-generation" (Tomlin and Wenger, 2001). However, numerous studies oppose this belief, suggesting that a greater VO<sub>2</sub> max will not have any effect on reducing recovery times for high intensity, intermittent exercise (Cooke et al, 1997; Carey et al, 2007). Therefore, Hoffman (1997) has suggested a minimum baseline of aerobic capacity, above which there is no increase in removal of waste and replenishment of ATP and PCr levels, meaning that increased aerobic fitness will only improve recovery time up to a certain point.

The presence of the both aerobic and anaerobic metabolism leads to an increase in body temperature, due to only 20-25% of all consumed metabolic energy being converted into mechanical work (Ament and Verkerke, 2009). Therefore, the role of the central governor (see section 2.4.2) is crucial in retaining homeostasis within working muscles (Desai and Bottoms, 2016).

### 2.3.3 The Mechanism of Thermoregulation and Fatigue

In order to maintain a stable body temperature, the extra heat generated during exercise must be dissipated (Ament and Verkeke, 2009). As suggested by Piil et al. (2017), hyperthermia reduces motor drive from the CNS, resulting in the termination of exercise. Neurons in the pre-optic area of the hypothalamus sense an increase in core temperature brought about by the by-product of heat from working muscles (Ament and Verkeke, 2009). A study by Gonzalez-Alonso (1999) found that the core temperate of the body can increase up to 40°C during exercise, at which point the subjects were unable to maintain the workload due to reduced central drive.

Studies by Todd et al. (2005), Gonzalez-Alonso (1999) and Nielsen et al. (2001) all found that there was an unknown inhibiting factor in the motor cortex, preventing participants continuing exercise above 40°C. It has been argued therefore, that progressive temperature increase stresses the cardiovascular system to a point where blood flow to the brain is inhibited. This reduces the heat loss from the brain, which Otani et al. (2017) argue introduces the sensations of fatigue and the sense of effort. This concept may relate to the "Catastrophic Failure Model" (Noakes and St Clair Gibson, 2004; Ament and Verkeke, 2009), which is discussed in section 2.4.1.

#### 2.4 Theories of Fatigue

In addition to the two major systems of fatigue (central and peripheral), there are also two main theories of fatigue, linear and non-linear (Noakes and St Clair Gibson, 2004; Desai and Bottoms, 2016; Inzlicht and Marcoca, 2016). These theories directly oppose each other, with linear theory suggesting that one of the prementioned systems, such as the cardiovascular or biomechanical, are stressed beyond its capacity, resulting in the termination of exercise (Noakes and St Clair Gibson, 2004). However Non-Linear theory suggests that the interaction of multiple peripheral physiological systems results in an accumulation of fatigue which prevents the athlete from continuing (Desai and Bottoms, 2016; Inzlicht and Marcoca, 2016).

In addition to these two theories, Waldron et al. (2017) argue that fatigue can be a temporary state, which can be recovered from during exercise or competition if the intensity of exercise drops for a period of around five minutes, allowing the athlete to partially recover and continue exercise. All theories of fatigue will be discussed in the following section.

## 2.4.1 Linear Theory of Fatigue

The linear theory of fatigue is outlined by the model of catastrophic failure, which suggests that activity ceases if at least one of the body systems are stressed beyond their capacity (Noakes and St Clair Gibson, 2004; Ament and Verkeke, 2009). This theory states that exercise will put such pressure on the cardiovascular system, to maintain the intensity of exercise. If the heart is no longer able to supply sufficient oxygen to working muscles, waste products including blood lactate accumulate, resulting in the termination of exercise due to the inability of the muscles to then

generate force, known as anaerobiosis (Noakes and St Clair Gibson, 2004). Consequently, the catastrophic failure model is explained by Noakes et al. (2005) as a cause and effect model proposing a steady decline in system function which ends in a "catastrophic event", a breakdown when metabolic demand overwhelms the capacity of peripheral tissues.

As a result of anaerobiosis, any increase in energy generation in the active muscles must come from anaerobic metabolism (Noakes and St Clair Gibson, 2004). This is because the intake of oxygen is inadequate, leading to the formation of lactic acid and therefore an increasing oxygen debt, resulting in exhaustion and fatigue in the working muscle (Yokoi et al. 2015; Ozimek et al. 2017). Whilst Noakes and Marino (2009) suggest that the catastrophic failure model prevents a casualty in the individual by protecting the heart, working muscles are, therefore, not the cardinal priority and will be the area of the body that suffers exhaustion. Noakes and Marino (2009) therefore argue that a governor in the brain would be activated by limited myocardial oxygen delivery, thereby limiting the activation of skeletal muscle by the motor cortex.

As previously discussed, teleoanticipation (Ulmer 1996) argues that metabolic changes maintain homeostasis within the peripheral systems. However the catastrophic failure model suggests that metabolic changes occur in response to maintain a specific exercise intensity (Noakes and St Clair Gibson, 2004).

### 2.4.2 Non-Linear Theory of Fatigue

In contrast to the Linear Theory of Fatigue, the Central Governor Model argues that it is not only the "catastrophic" event within one system in the body that induces fatigue, but the consequence of the complex interaction of multiple peripheral physiological

systems acting as different signallers to the brain in a dynamic and non-linear integrative manner (Desai and Bottoms, 2016; Inzlicht and Marcoca, 2016).

The central governor, a central nervous system mechanism (Inzlicht and Marcoca, 2016) receives feedback from any changes in metabolic activity, such as depletion of muscle glycogen, ATP stores, or an increase in blood lactate, and aims to restore these changes back to normal homeostasis (St Clair Gibson et al. 2017) and therefore prevents any system from reaching terminal fatigue. If this system was perfect, any changes during exercise would activate afferent signals and induce a response, reverting the system back to its baseline.

However, as metabolic and physiological variables continually oscillate during exercise and at rest, it is difficult for this central control centre to keep these constant (Inzlicht and Marcoca, 2016). Therefore, large changes in these levels sends feedback to the motor cortex through mechanoreceptors and chemoreceptors, which will limit force production in the attempt of preventing long term damage to the working muscles. However, multiple systems can send negative feedback at once. For example, a lack of muscle glycogen and an accumulation of lactate can occur simultaneously, sending multiple signals to the brain and creating the "fatigue" which will terminate exercise before exhaustion. Therefore, as suggested by Noakes (2000 p.124), "the complexity of the physiological and other factors determining human performance is emphasized when the limitations of each of these models is exposed".

Edwards and Mann (2011), argue that the central governor model (Figure 2) predicts that performance is continually regulated by the subconscious brain (via constant manipulations of motor unit recruitment) to allow the individual to complete the task as quickly and efficiently as possible, without excessively stressing physiological responses, thus ensuring homeostasis is maintained and preventing premature termination of exercise. It has been suggested by Alain and Matran (2012) that this is achieved better if the athlete is able to self-regulate in the knowledge of the duration and intensity. A football match, for example, will last 90 minutes, with extra time added very rarely in cup competitions. Therefore, there is set duration for players to perform at, allowing players to self-pace and balance the quality of performance across all bouts of exercise, i.e. the two halves, with the shortest possible recovery (Edwards and Mann, 2011).

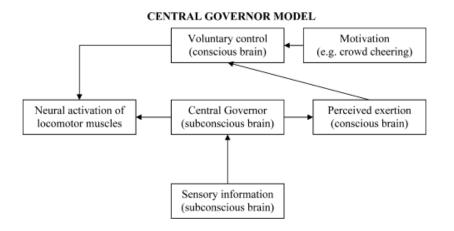


Figure 2 The Central Governor Model (Marcoca, S.M., 2008. Do We Really Need a Central Governor to Explain Brain Regulation of Exercise Performance? European Journal of Applied Physiology)

It has been suggested that whilst this non-linear fatigue can result in the termination of exercise, it can be temporary, with individuals showing a partial or full recovery, allowing an increase in physical output later in a training session (Waldron and Highton, 2014). This is known as transient fatigue (Waldron and Highton, 2014).

#### 2.4.3 Transient Fatigue

Players will increase their running performance at latter stages of a game or training session, which Waldron and Highton (2014) suggest reflects either full or partial recovery. This 'transient' or 'temporary' fatigue is described by Mohr et al (2003) and Waldron et al. (2017) as a period of around five minutes of running intensity below the mean match intensity, occurring immediately after the most intense period. Once this low-intensity period has finished, players are able to achieve a total recovery in running intensity (Waldron et al. 2013). Mohr et al. (2003) have argued that this five-minute period accounts for any situational changes within the match, such as goals or extended periods of possession, eliminating the identification of typical match fluctuations.

As discussed previously, non-linear theory suggests that multiple factors play a role in the development of fatigue (Desai and Bottoms, 2016; Inzlicht and Marcoca, 2016). However, whether the cause of fatigue be multi-faceted or due to one factor, progressive declines in running intensity, which can be measured by GPS (Bradley and Noakes, 2013), have been widely accepted as an indication of fatigue in sport (Waldron and Highton, 2014). It has been shown in a study by Lacome et al. (2016) that international rugby union players experience a greater decrease in physical (high speed running) and skill-related performance during the final 10 minutes of the game that they do after the five-minute period of the greatest intensity. Therefore, whilst the phenomenon of transient fatigue should not be ignored in research, the cumulative build-up of fatigue is currently more frequently studied. Post-match recovery is often more highly valued amongst sport and exercise scientists than in-game recovery (Alcaraz et al. 2017, Rowell et al. 2017).

### 2.5 Consequences of Fatigue

As previously argued by Noakes and Marino (2009), the sensations of fatigue can be viewed as a protective mechanism to prevent life-threatening conditions in the body. However, whilst systems such as the cardiovascular and respiratory systems are protected from terminal conditions, limitations of other bodily processes can lead to other issues.

Nybo (2003) argues that Central fatigue will reduce motor control and limit musclestabilisation, leading to a greater risk of joint injuries, with studies showing an increase in muscular injuries later in games. Ekstrand et al. (2011) and Espregueira-Mendes et al. (2017) found that the majority of muscle injuries occurred towards the end of each half in a match, with Rahnama et al. (2002) adding that mild and severe injuries were more likely to happen in the final 15 minutes of a game than any other period. Additionally, after 24 games in the Premier League in the 2017-2018 season, 22.6% of all goals were scored in the final 15 minutes of games (SoccerStats, 2018). The next highest scoring time was the final 15 minutes of the first half with 17.5% (SoccerStats, 2018), suggesting that as the duration of competition without rest increases, concentration levels drop leading to more goals scored.

Each of these issues are discussed further in the following sections.

# 2.5.1 Fatigue and Muscle activity

The central and peripheral mechanisms of fatigue are generally measured during maximal (MVC) or submaximal voluntary contractions, allowing measurement of muscle torque (Abd-Elfattah et al. 2015). In a sustained MVC, the torque produced is highest at the start of the contraction before progressively dropping, suggesting that

motor recruitment is highest at the start, but de-recruitment occurs and consequently, firing rates weaken (Abd-Elfattah et al. 2015).

During submaximal voluntary contractions, participants will hold a contraction at a specific pre-determined torque until they are unable to maintain it. The strength of the contraction determines the number of motor units recruited at the start, but this number increases during the contraction as the force produced by the initially recruited units fades (Abd-Elfattah et al. 2015). By creating an artificially stimulated motor evoked potential (MEP) through transcranial magnetic stimulation, an increase in muscle torque was generated, indicating an increase in corticospinal responsiveness, suggesting a level of central fatigue during submaximal contractions (Taylor and Gandevia, 2001; Jubeau et al. 2014). This superimposed twitch shows that central processes proximal to the site of motor axon stimulation contribute to a loss of force, with some central fatigue attributed to supraspinal mechanisms (Abd-Elfattah et al. 2015).

By using electromyography (EMG), it is possible to note an increase in motor unit recruitment (Kamen and Gabriel, 2010). The reduction in EMG amplitude during contractions has been attributed to a decrease in muscle activation. This reduced number of action potentials in the targeted muscle is due to many factors, including a decline in force twitch amplitude, and increase of inhibitory feedback and the inability of the CNS to produce the required output (Abd-Elfattah et al. 2015).

## 2.5.2 Fatigue and Cognition

The decisions made by players during a football match often must be made in splitseconds, with athletes responding to ever-changing stimuli, such as ball position,

location of opponents and verbal cues from teammates and staff members (Lee et al. 2017). Therefore, whilst the physical impairments caused by prolonged activity have been shown to limit performance, there are also arguments from researchers that cognitive function is also reduced following exercise (Tracey et al. 2007). However, other studies (Young et al. 2015, Nuechterlein et al. 2016) suggest that short periods of exercise can result in improved cognitive functioning in adults. Therefore, Kamijo et al. (2007) Abd-Elfattah et al. (2015) suggest that the relationship between acute physical activity and cognitive performance has an inverted U shape, dependent on the duration and intensity of the activity. A study by (Hogervorst 1996) found that submaximal activity, with a heart rate of around 120-130 beats per minute and a duration between 20 and 40 minutes led to an increased sensori-motor and cognitive performance. Conversely, prolonged exercise at the same intensity, resulting in dehydration results in a disruption in short term memory and psycho-motor abilities (Cian et al. 2001).

It is, therefore, crucial to understand whether fatigue is caused by dehydration (Cian et al. 2000) or changes in cortical activity in the brain alongside hypoxia, which is caused by exercise (Goodall et al. 2010). If the reduction in function is caused by dehydration, the athletes and their supporting team can then adapt their nutrition programmes (Holway and Spriet 2011). However, hypoxia and cortical activity changes present more of a challenge to adapt through training programmes.

# 2.5.3 Fatigue and Proprioception

Proprioception is defined by Abd-Elfattah et al. (2015) as the area of the body screened from the environment by surface cells (skin) which contain

mechanoreceptors for any changes in movement, including the regulation of postural equilibrium and joint stability. These mechanoreceptors monitor several subconscious peripheral sensations known as "muscle senses", including posture, passive and active movements and resistance to movement (Proske and Gandevia, 2012). Located primarily in muscle, ligament and tendon, these mechanoreceptors quantitatively transduce the mechanical events of movement and changes in joint position in to neural signals, allowing the individual to adapt quickly to these changes (Grigg 1994; Nyland et al. 2017).

As Abd-Elfattah et al. (2015) suggests that proprioception plays a large part in maintaining the functional stability of the joint, a deterioration in proprioception through mental or physical fatigue highlights a greater risk of ligament injury. Barber-Westin and Noyes (2017) adds that muscle fatigue causes a decrease in muscle activation patterns, which in turn affects the joint sense of position leading to disturbed balance and reduced neuromuscular control in the lower limb. A study by Miura et al (2004) found that with a 15% decrease in knee flexion and extension output, there was a statistically significant effect on knee proprioception.

Skinner et al. (1986) however found that even without a decrease in strength, a decrease in proprioceptive control was seen following exercise. This contradicts Miura et al. (2004) who argued that general fatigue load affected proprioception, by arguing that by maintaining strength post-exercise suggests a deficiency of central proprioceptive signals. Central fatigue, as previously stated, will reduce motor control and limit muscle-stabilisation, leading to a greater risk of joint injuries (Nybo, 2003). Studies into central and peripheral causes of proprioceptive decline are limited, however it is clear that an inability to control the joint position in a fatigued state will result in a heightened risk of injury. It could be argued that as an individual may be

performing sub-maximally in a fatigued state and therefore could be at a lower risk of injury due to reduced forces. However, reduced force production can lead to an increased risk of injury when interacting with external factors, such as landing on the ground or physical contact with opposition players (Abd-Elfattah et al. (2015).

## 2.6 Methods of Measuring Fatigue

Measurement of fatigue and the recovery from this is crucial when it comes to prescription of training stimulus to individuals and teams (Thorpe et al. 2016; Rowell et al. 2017). By understanding the aetiology of the impairment of muscle function, it is therefore possible to adapt training programmes and prescribed load on a daily, weekly and monthly basis (Thorpe et al. 2016). It is therefore important to measure levels of both peripheral and central fatigue to understand which physiological or psychological systems are inhibiting performance (Thomas et al. 2017) and these will be discussed below. Through supramaximal stimulation of the motor nerve during maximal contraction, it is identified the degree to which motor units are not recruited or firing at optimal rates as these will produce extra force (Gandevia et al. 1996).

### 2.6.1 Measurement of Central Fatigue

Transcranial Magnetic Stimulation (TMS) is a non-invasive process using magnetic fields to stimulate nerve cells in the brain (Goodall et al. 2014). This procedure is commonly used to treat depression if used regularly, with a study by Griffiths et al. (2019) finding 51.1% of UK participants with depression improved their Clinical Global Impression scores through TMS treatment. However, TMS has also proved to be a

valuable technique for investigating the mechanisms of central fatigue and neural adaptation in response to locomotor exercise.

A rapidly altering magnetic field is delivered via a concave double cone coil to the scalp of the participant, inducing weak electrical currents to excite cerebral neurons (Goodall et al. 2014), with the concave double cone coil able to reach deep into neural structures (Magstim 200, The Magstim Company Ltd., Whitland, UK). It is possible to deliver these deep stimulations to precise groups of neurons in relation to specific muscle groups, as the distinct regions of the muscular system and their connecting neurons are linked to their own geographical location across the motor cortex, known as the homunculus (EMB Consult, 2015) (see Figure 3).

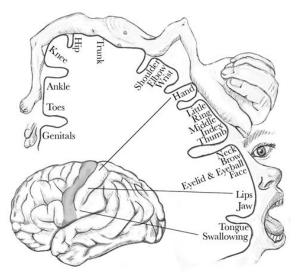


Figure 3 Motor Cortex Homunculus (EMB Consult, 2015)

Central fatigue can therefore be demonstrated when during an MVC, the cerebral cortex is stimulated, and the force produced is increased (Belanger and McComas, 1981). If an increase in force evoked can be produced via nerve stimulation during an MVC, it is clear that the full potential of the neuron pool was not utilised, motor units were not recruited, or the speed of firing was not enough to produce an action potential at the point of stimulation (Herbert and Gandevia, 1999). Bipolar surface

electromyography (EMG) can then be used to evaluate and record the electrical activity created by participants' skeletal muscles through placement of electrodes on the belly of the muscle undergoing stimulation (Nichols Larsen et al. 2015).

A study by Cavaleri et al. (2018) found that the use of TMS was a reliable method for brain mapping in healthy individuals. Derkele et al. (2019) proceeded this with a study into the reliability and validity of TMS in producing involuntary contractions of the knee extensors. The study found that absolute reliability was strong enough to depict a true detectable change in the sample's voluntary activation following the fatiguing exercise.

#### 2.6.2 Measurement of Peripheral Fatigue

The nerve most commonly used amongst sports practitioners to test for peripheral fatigue in the lower limb is the femoral nerve, due to its ease of location (Goodall et al. 2017, Stebler et al. 2017). The femoral nerve is the largest to originate in the lumbar plexus (L1-L4) (Tsui 2008). It passes through psoas major, the iliacus muscles, the iliac fascia and beneath the inguinal ligament before splitting into anterior and posterior nerves in the upper thigh (Tsui 2008). By placing a cathode superficially on the skin in the femoral triangle, it is possible to elicit an action potential from this nerve to the quadriceps muscles, most specifically the vastus lateralis (Thomas et al. 2017). By comparing the difference in force generated (N) between voluntary contractions and peripherally stimulated contractions, practitioners can identify the deficit (Thomas et al. 2017), with greater deficit in the voluntary contraction suggesting an inability of the muscle to produce maximal force (Thomas et al. 2017).

Issues can arise with peripheral stimulation, however these are rarely threatening to the individual. Hardware related complications such as extension lead failure or battery

depletion are much more common than biological complications such as infection, which only occurs if the skin is broken (Eldabe 2016). A more common biological issue is pain as the sensation of a simulated electrical impulse in the femoral triangle can be an uncomfortable sensation Eldabe, 2016). As the trial and error method to find the correct location often taking a number of attempts, participants may have to undertake a number of stimulations, even before testing begins.

## 2.7 Determining Fatigue in Sport

With elite football players competing in multiple games per week, the requirement for monitoring of training and match load is crucial to allow the appropriate recovery in time for the next fixture (Lundberg and Wekström, 2017). De Hoyo et al. (2016) and Lundberg and Wekström (2017) suggest it can take up to 72 hours for an individual to be fully recovered from a match, which may include another fixture, meaning the individual may still be feeling the effects of fatigue before kick-off. Therefore, it is crucial to prescribe effective recovery modalities or training stimuli to improve technical, tactical and physical attributes, whilst allowing the best possible chance of a player being physically fresh for their next matchday (McLean et al. 2016).

A study by de Hoyo et al. (2016) researched Countermovement Jump (CMJ) performance in football players, finding that concentric jump force and jump height were both reduced 48 hours post-match. Additionally, a study by (Thomas et al. 2017) found that the assessment of drop jumps to assess Reactive Strength Index (RSI) were a useful benchmarking tool for fatigue assessment. They also found that central fatigue recovers more quickly than peripheral fatigue, indicating that processes relating to the resolution of muscle function primarily occur proximal to the

neuromuscular junction. (Thomas et al. 2017). This study has its flaws however, primarily being the lack of physical training in the days following the matchday, leading to a quicker recovery which is unrealistic in an elite sporting setting.

An interesting difference between professional first team football players and those that are in youth academies involves periodisation and development. It is widely acknowledged that professional football academies in England place a large emphasis on development of football players, whilst the first team primarily focus on maintenance of fitness (Ronnestad et al. 2011; Lloyd et al, 2015). Players in undereighteen squads and below stick to a pre-planned coaching syllabus, including number of hours of training, with programmes having technical / tactical, psychological, physical and social topics (Football Association, 2018). This is known as the Elite Player Performance Plan (EPPP) which was introduced in 2012, with the mission of producing more and better home-grown players through the empowerment of each individual player with a player-led approach (Premier League, 2019).

Therefore, it can be argued that training prescription will differ between academy and first team squads to allow a greater learning to take place in the weekly programmes of academy teams. Consequently, it is then down to the coaching staff in these younger teams to balance the physical benefits of recovery sessions with the loss of technical and tactical development opportunities by not partaking in training sessions.

### 2.7.1 Fatigue in Elite Sport

At elite level, there are numerous differences between sports, with individual and team sports and invasion games and racquet sports having different physical outputs. Repeated maximal sprints however are common in most sports, requiring peak muscle

activation alongside limiting the ability of the individual to set a pacing strategy (Huerau et al. 2015), therefore limiting the effects of teleoanticipation on fatigue.

Pacing strategies involve the conscious and subconscious variation of workload over the duration of competition to limit premature fatigue (Billaut et al. 2011). This strategy is usually established at the beginning of the match, with participants having previous experience of the duration and intensity as well as arriving in a certain physiological (muscle glycogen levels, hydration) and psychological (motivation, confidence) state (Billaut et al. 2011). By understanding the requirements of the match, participants can adjust skeletal muscle recruitment and mechanical output to maintain performance (Huerau et al. 2015). However, repeated high-intensity bursts (<6s and >300% intensity at which VO<sub>2</sub> max is achieved) are repeated randomly through competition and it is therefore difficult to self-pace, as the external factors such as opposition and the pattern of the match do not allow for adequate recovery after every high-intensity action (Lander et al. 2009). Therefore, it is unclear at any given intensity, which mechanism is creating neuromuscular fatigue, either central or peripheral.

This point is reiterated through a study from Lander et al. (2009) who stated that externally-paced work presents a greater physical demand when compared to an internally-paced exercise of the same intensity. They added that either inadequate or excessive recovery between exercise blocks will compromise training outcomes, meaning the athlete must regulate when they feel prepared to recommence training or competition. This presents a difficulty in team sports training settings, with players reacting and recovering from a training or match stimulus at different rates (Varley et al. 2017), it will therefore be near-impossible to begin a group session with all players recovered to the same level.

#### 2.7.2 Fatigue in Elite Football

Rampinini et al. (2011) state that football players change activity on average every 5 seconds and complete approximately 1300 actions during a match, with almost 200 of these being complete at high intensity. These high intensity activities include direction changes, dribbling and tackling (Mohr et al. 2003)

During a simulated soccer match, measurements of central and peripheral fatigue were measured by Goodall et al. (2017). They found that maximum voluntary contractions (MVC) were reduced from baseline measurements at half time, full time and the end of extra time. Potentiated knee-extensor force was reduced by 15% at half time, with a with no further drop until the end of the simulation, suggesting a plateau in peripheral fatigue response which has been demonstrated in numerous sports across different studies (Decorte et al. 2012; Froyd et al. 2013; Goodall et al. 2015). This has been explained by Goodall et al. (2017) by players meeting the early exercise demand by exhausting the higher threshold motor units, which are more susceptible to fatigue before utilising the more fatigue-resistant motor units which exert a smaller response to peripheral stimulation (Harper et al. 2016). The use of TMS also identified a decrease in voluntary activation measured through motor cortex stimulation, showing a reduced capacity for the motor cortex to drive the knee-extensors immediately post-stimulation, suggesting a contribution of supraspinal factors to central fatigue in the participants (Goodall et al. 2017).

Thomas et al. (2017) followed up on the study by Goodall et al. (2017) by investigating the recovery of the individuals in the week succeeding the soccer simulation. They found that maximal voluntary contraction was significantly reduced post-exercise and was not recovered up to 72-hours after. Motor cortex stimulation with TMS was reduced immediately and 24-hours post-exercise but was recovered 48-hours post,

suggesting a recovery from central fatigue after 48-hours. Conversely, motor nerve stimulation displayed a decrease up to 72-hours post-exercise, with Thomas et al. (2017) arguing that the reduction in voluntary activation 3-days post-exercise is due to peripheral factors.

The limitations of the two studies (Goodall et al. 2017; Thomas et el al, 2017) leave a gap in the research for field-based study. Both studies use simulated soccer match play, allowing for the reliability in the physical outcomes such as distance covered (km), high intensity distance (km) and number of high intensity efforts. However, this does not allow for cognitive components of competitive matches, such as the motivational differences between players and stress levels which can fluctuate throughout a match (Jones and Vanhatalo 2017, Josefsson et al. 2017), as well as the ability of players to self-pace as they are aware of the exact protocol, which Eston et al. (2012) suggests elicits different physical outcomes. Additionally, participants in the study by Thomas et al. (2017) do not complete any prescribed physical activity following the trial. This is unrealistic in an elite sports setting, with teams and individuals completing recovery protocols such as weekly cold-water immersion (Nedelec et al, 2015), alongside gym-based and pitch-based physical or technical training to prepare for the subsequent match. These results, therefore, show the intimate recovery modalities of individuals over a 72-hour period, however, in elite competition, this 3-day period of relaxation is not common. It is crucial to understand the effect that training has on the recovery from fatigue.

### 2.7.3 Fatigue in Youth Football

It has been well-established that children experience a lower degree of physical fatigue than adults and consequently require less recovery time, especially during high intensity exercise (Armatas et al. 2010). Ayramo et al. (2017) have stated that these differences can be explained by maturation-related development of force development capability, anaerobic capacity and the glycolytic enzyme system, with children benefitting more from their oxidative capacity than adults as the anaerobic system is not sufficiently mature to provide the physical output associated with short duration exercise (Ratel and Blazevitch, 2017). This system becomes more sophisticated post-Peak Height Velocity (PHV), during adolescence, typically around the age of fourteen in males (Housh et al. 2017), with individuals beginning to create waste products such as hydrogen ions in the sarcolemma which impairs many physiological processes such as force production (Milioni et al. 2017).

It is reasonable to expect that as differences in anaerobic performance in adults and children are maturation-related, mechanisms of fatigue will also be dependent on the stage of physical development (Ayramo et al. 2017). For example, due to their lower anaerobic capacity, children are less affected by metabolites such as hydrogen ions, meaning that metabolic (peripheral) fatigue is lower than in adults. A study in maximal isometric and isokinetic tests in children also suggested they showed a lower degree of central fatigue than adults (Armatas et al. 2010), though they did show a lower level of voluntary activation.

Quicker recovery from maximal exercise in children has been attributed to a reduced production of hydrogen ions, due to a lower anaerobic capacity which results from this population having less-developed glycolytic fast-twitch muscle fibres and lower testosterone levels (Falgairette et al. 1991; Crone, 2017). Consequently, with less

hydrogen ions present, children can restore the muscle phosphocreatine levels twice as quickly as adults (Tonson et al. 2010), suggesting children experience less fatigue and can recover more quickly than adults, meaning their training methods will be significantly different.

When measuring the high-intensity output from children and adolescents, it is crucial to understand that typical speed bands (Bangsbo, 2014) will not apply to them as they are often unable to hit the speeds that adults will be able to (Catapult 2018). By individualising speed bands to each player, it is then possible to monitor both training load and changes in fitness levels throughout a season (Catapult, 2018). This is often done through mid-season testing, including sprint testing and aerobic endurance testing, to identify improvements in speed and  $VO_2$  max. Often the increases in total distance and high speed running distance in each match can be used as a measure of increasing fitness levels through a season, however these can vary dependent on opponent.

# 2.8 The Effect of Training Methods on Recovery

Training loads in elite football are usually manipulated through the total session or individual activity duration, rest duration, pitch size or number of bouts of work (Edwards et al. 2011). By training in this way, players are then able to cope with situational changes in competition, such as the need to score a goal due to a losing position, which changes the intensity at which players are working (Bangsbo et al. 2017). By adapting training to allow players to work at differing intensities, it assists the individual's ability to complete a range of different competitive situations within the capabilities and levels of their own tolerable fatigue (Bangsbo et al. 2017). Therefore,

by manipulating the training load over daily, weekly and monthly periods, it is possible to produce different physical outcomes, allowing differing rates of recovery (McArdle et al. 2010; Christopher et al. 2016; Bangsbo et al. 2017).

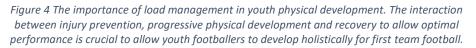
A challenge that coaches in elite youth football will encounter is the requirement to not only ensure players are in an optimum physical condition for competitive matches, but also to ensure that they are promoting progressive physical development through to adulthood (Ryan et al. 2018). The development of the previously discussed energy systems, as well as improvements in physical characteristics, such as speed and power, plays a crucial role in a young player's progression towards professional football. (Ryan et al. 2018).

The training load for youth players may therefore need to be greater than that of the elite, adult population in order to allow progressive physical development. This can result in a number of negative consequences. These can range from individuals experiencing the delayed onset of muscle soreness (DOMS) producing a reduction in performance, to overuse injuries, which can be caused through a high training load for an individual who is not fully through their maturation process (Leppänen et al. 2019).

It has been argued that the speed of recovery from prolonged physical exertion, such as football match-play, can be increased through physical activity (Nalbandian et al. 2018). This is due to the ability of increased blood flow to aid in the removal of waste products (Wiewelhove et al. 2018), with active recovery producing a hormonal environment which may favour lipolysis and oxidative metabolisms (Nalbandian et al. 2018). The intensity of the session needs to be managed as, although increased blood flow and testosterone can aid recovery, high power activities can cause further muscle damage (Raeder et al. 2017). With participants working over smaller distances, it is harder for them to hit their maximum velocities (Casamichana and Castellano, 2010) and therefore will keep their heart rate high whilst limiting the muscle damage caused by maximum intensity efforts (Casamichana and Castellano, 2010).

It is therefore crucial that the optimal training methods are identified in order to create a balance between the development of physical traits required for professional football, availability for training and matches and preparing the youth individuals to be in optimum condition for competitive matches (Figure 4).





#### 2.9 Conclusion and Study Rationale

It has been suggested by Ament and Verkeke (2009), Rampinini et al. (2011), Boccia et al. (2017) and Thomas et al, (2017) that there are two different routes of fatigue, each with multiple mechanisms. Central fatigue has been defined as inadequate activation of motor neurons and therefore reduced neural drive to the muscle (Abd-Elfattah et al. 2015) with the loss of contraction force caused by processes proximal to the neuromuscular junction (Ament and Verkeke, 2009). It is understood that peripheral fatigue is the loss of contraction force caused by processes distal to the neuromuscular junction (Ament and Verkeke, 2009).

Numerous previous studies have identified how each of these mechanisms of fatigue can be measured in multiple team and individual sports, with the use of the H-Reflex and V-Wave being utilised commonly through Peripheral Stimulation and Transcranial Magnetic Stimulation (Belanger and McComas, 1981; Thomas et al. 2017). By measuring the effects of artificial stimulation during maximal and sub-maximal contractions, it is possible to identify many facets of fatigue, such a motor neuron excitability, E-C coupling responses and fatigue in the neuromuscular junction (Palmieri et al. 2004).

Previous studies into simulated soccer match-play have identified an acute decrease in voluntary activation measured through TMS, showing a reduced capacity for the motor cortex to drive the knee-extensors immediately post-stimulation, suggesting a contribution of supraspinal factors to central fatigue in the participants (Goodall et al. 2017). It was also found that maximal voluntary contraction was significantly reduced post-exercise and was not recovered up to 72-hours after. Motor cortex stimulation with TMS was reduced immediately and 24-hours post-exercise but was recovered 48-hours post, suggesting a recovery from central fatigue after 48-hours. Conversely,

motor nerve stimulation displayed a decrease up to 72-hours post-exercise, with Thomas et al. (2017) arguing that the reduction in voluntary activation 3-days postexercise is due to peripheral factors.

The limitations of previous studies leave a gap in the research for field-based study. Both studies (Goodall et al. 2017; Thomas et el al, 2017) use simulated soccer match play, allowing for the reliability in the physical outcomes, however, this does not allow for cognitive components of competitive matches, such as the motivational differences between players (Jones and Vanhatalo 2017, Josefsson et al. 2017), as well as the ability of players to self-pace as they are aware of the exact protocol, which Eston et al. (2012) suggests elicits different physical outcomes.

A study by Brownstein et al. in 2017 used semi-professional adult footballers in a competitive fixture, assessing central nervous system function in the days following the match. They found that competitive match-play elicited significant post-match decline in MVC force (-14%) which took 72 hours to recover. Through the completion of peripheral stimulation, the percentage of voluntary activation was also reduced post-match but recovered 48 hours later. This reiterates points made in previous studies, yet focuses on non-elite athletes of adult stature, meaning that youth football is a relatively unexplored area.

Therefore, there is a current gap in the research for a field-based study on elite youth footballers to identify the effects that training protocols have on the time-course and aetiology of recovery from competitive soccer matches.

## 2.10 Research Aims and Hypotheses

Through the completion of the Literature Review, it has been possible to create a number of research aims and hypotheses.

## 2.10.1 Research aims

- To identify the deficit between voluntary, isometric quadricep contractions and involuntary, Transcranial Magnetic Stimulation-Induced and Peripheral Stimulation-induced isometric quadricep contractions in elite youth football players and the speed at which different training methods facilitate recovery.
- 2) To analyse the extent to which the deficit between voluntary, isometric quadricep contractions and involuntary, Transcranial Magnetic Stimulation-Induced and Peripheral Stimulation-induced isometric quadricep contractions differ in a fatigued state in elite youth football players.
- 3) To discover the effect that fatigue plays in force production and decipher the role that the both central and peripheral mechanisms play in this.

## 2.10.2 Research Hypotheses

- Elite youth football players at a Premier League Academy will present with a greater level of both central and peripheral fatigue following a competitive football match compared to prior to the match.
- 2) Following a competitive football match, a training week of lower intensity, including less high speed running and smaller training areas, will facilitate

accelerated recovery from both central and peripheral fatigue than a training week of higher intensity.

# 2.10.3 Null Hypotheses

- Elite youth football players at a Premier League Academy will not present with a greater level of both central and peripheral fatigue following a competitive football match compared to prior to the match.
- 2) Following a competitive football match, a training week of lower intensity, including less high speed running and smaller training areas, will not facilitate accelerated recovery from both central and peripheral fatigue than a training week of higher intensity.

Methodology

### 3.0 Methodology

In this chapter, the study design, experimental procedures and method of data analysis will be discussed.

### 3.1 Research Approach

The researcher completed the study through a quantitative approach, with an experimental design. This quantitative method was chosen to allow the researcher to test the previously stated hypotheses. Generally, quantitative methods provide summaries of data that suggest generalisations of the phenomenon under study, with fewer variables but more strictly adhered to procedures. This helps to allow validity and reliability in the study (Matveev, 2002), which allows the study to be replicated (Babbie 2017). Horn (2008) adds that to maintain validity and reliability, these studies must also be carried out in unnatural and artificial environments, resulting in laboratory results rather than real-world results.

There are also disadvantages of quantitative study. It has also been argued that researchers can lead participants towards results that reflect their views, rather than that of the participants, resulting in a 'structural' bias as often quantitative research is used to prove or disprove a theory rather than identify a new phenomenon (Nykiel, 2007).

An experiment is described by Oxford Dictionary (2019) as a scientific procedure undertaken to make a discovery, test a hypothesis, or demonstrate a known fact. The design of an experiment is crucial as the information gained from properly planned,

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executed and analysed experiments can be used to improve functional performance (Anthony, 2014).

It has been identified that within field-study, it is difficult to complete experiments within these unnatural environments and therefore reliability and validity may be compromised. John (2017) states that experiments risk failure or compromised implementations due to the requirement for everything to be in the right place at the right time, often relying on the cooperation of third parties.

Even if an experiment is implemented effectively including randomisation of subjects, properly administered interventions and measurements of outcomes, there is a possibility of limitations which affect the result (John, 2017). For example, the control group may have been exposed to the intervention which can skew the outcome of the experiment, producing a false outcome (John, 2017). It is therefore crucial that the relationship between the researcher(s) and the participants, and organisations to which they belong, are very transparent to limit the chance of external factors influencing the procedure.

Within the current study, it could be argued that the testing location was unnatural in a sporting context, with participants completing the chosen testing protocol in the physio-room at their training ground. However, this was the closest location to the training pitch which allowed standardisation of the environment, including temperature, avoiding weather-based variations such as rain and wind which would have skewed the results. Due to these factors, it was therefore not possible to use this equipment beside the pitch.

In order to standardise results, it was crucial to gain numerical evidence of fatigue from both central and peripheral contributions. A mixed-methods approach was considered

by the researcher, however time constraints would limit the effect of interviews postexercise, as participants would begin their recovery process whilst other players were completing the physical testing protocol, leading to players giving a misrepresentation of their immediate post-match fatigue. Brannen (2017) states that it is a common misconception that mixed-methods is a superior research method, as the researcher must judge if any important aspects of the research problem would be ignored if a single research approach was employed. Therefore, a quantitative research method was chosen to use numerical data to compare central and peripheral contributions to fatigue within the different interventions.

#### 3.2 Study Experimental Design

The research variables were determined prior to the commencement of testing, based on information discovered through the literature review:

Dependent Variable – Recovery status of athletes, determined through voluntary activation testing protocols with artificial stimulation in the form of Transcranial Magnetic Stimulation and Peripheral Stimulation. The recovery status is measured throughout multiple testing protocols throughout the week.

Independent Variable – The nature of the training protocols over two separate weeks that were designed to elicit different physiological outcomes. The physical outcomes of sessions were measured through GPS tracking units to differentiate training load between the two interventions. The difference between the two separate training variations will be expanded upon later in this chapter.

The research was of a repeated measures design with each participant completing two separate weeks of testing. This testing involved two differing training programmes (Aerobic Training Week (TW-A) and Anaerobic Training Week (TW-AN), which were completed by different participants in a randomised order to prevent individuals preparing for each week differently. For example, all participants completed both TW-A and TW-AN, however not all completed TW-A first. The participants completed the two different training weeks in a random order to nullify both the practice and boredom effects which may alter their responses to the differing stimuli (Field, 2005). The protocol was completed over six separate weeks, with between two and four participants tested each week.

Repeated measures procedures are described by Field (2005) as a more reliable measure than exposing different participants to different scenarios as there are less random and uncontrollable differences, or "white noise" as the individuals are the same and therefore inter-personal differences are ignored. This systematic variation is due to the researcher changing the training stimulation for all players, rather than random factors that exist between two differing groups, such as base VO<sub>2</sub>max levels for example (Field, 2005).

Testing took place through the mid to late part of the season when games were played every Saturday, between the dates of 18<sup>th</sup> November 2017 and 2<sup>nd</sup> March 2018. The participants completed the testing protocol (described in sections 3.4 to 3.5.4) six times throughout each of their two, weekly protocols (pre-match, post-match, 48 hours post-match, 72 hours post-match, 120 hours post-match and 144 hours post-match). They completed their baseline testing on the Saturday morning before their match. Rowell et al. (2017) argue that prior to the match should be the point at which they are at their most physically ready to compete. This was then repeated immediately post-

game, with players returning to the testing room before receiving feedback from the coaches and showering. Rowell et al. (2017) argue that this point should be the participants' most fatigued state of the week.

These tests were then also completed in the mornings 48 hours, and 72 hours postgame as well as 48 hours and 24 hours prior to the following game. This allows the assessment of recovery from the previous game alongside the training protocol, whilst being able to identify the nature of any deficits in voluntary muscle contraction, deriving from either central or peripheral contributions (Boccia et al. 2017; Thomas et al, 2017).

## 3.3 Participants

Ten male academy football players (mean  $\pm$  SD; age: 18  $\pm$  1 years; stature: 179.1  $\pm$  6.7cm; body mass: 73.7  $\pm$  5.4kg; maximal aerobic speed [MAS], tested through an incremental treadmill protocol: 15.0  $\pm$  1.2 km/h) signed an informed consent form and volunteered to participate in the study. All participants had been competitively playing elite youth football for their current Premier League Academy or a previous club for a minimum of two years. The participants trained a minimum of four times per week (alongside gym-based conditioning sessions) and played in one competitive league or cup fixture each Saturday. The participants' competitive season ran from August to April where teams competed in 30 to 35 games per season. Prior to testing, written informed consent (Appendix A) was gained from all participants, in addition to the written informed consent from parents of all participants under the age of eighteen.

All participants completed a Physical Activity Readiness Questionnaire (PAR-Q) Form (Appendix B). As part of the Premier League rulings, all players registered on a fulltime scholarship were required to complete a cardiac screening from Cardio Direct

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(London), therefore the researcher was aware that all participants were medically safe to complete the protocol. This screening consisted of two parts which are both reviewed by a consultant cardiologist. The paperwork included a detailed health questionnaire and an extensive list of family medical history. The second part of the screening involved a physical examination consisting of a 12-lead electrocardiogram and an echocardiogram. This was carried out routinely at the age of sixteen unless further investigations and tests or more regular screening for monitoring of structural abnormalities are required. Ethical approval was gained from the Ethics Committee of Buckinghamshire New University prior to commencement of the project (Appendix C).

Participants were included in the study if they were currently enrolled in a full-time scholarship in a Premier League football club, fit between the ages of sixteen and eighteen, had completed both parental and player consent forms and were free of injury at the start of the protocol. As there were just 17 players in the squad, which included two goalkeepers, there was a small pool of players to choose from for the study on this club. As one player was injured and the remaining four did not volunteer to participate, there was a limited number of participants available to complete the testing protocol.

### **3.4 Preparation for Testing**

Prior to the commencement of testing, the researcher presented the study to the members of the Under Eighteens squad of a Premier League football team. The protocol and procedures involved were explained, allowing for any questions. Following this presentation, potential participants were given information sheets, PAR-

Q forms and consent forms and asked to discuss with parents before completing and returning the forms.

Participants had completed each of the training sessions which were to be completed in TW-A and TW-AN. The training interventions, which were to elicit different physical outcomes over the two-week protocol were also completed prior to testing to familiarise the participants. They were also habituated to the methods used to assess neuromuscular fatigue prior to and post training, such as Transcranial Magnetic Stimulation (TMS) and Peripheral Stimulation (PS) over a minimum of two practice sessions. Participants were given the chance to withdraw at any stage of the protocol, including post-practice trials once they fully understood what was involved in participating. Throughout the trials, the researcher explained the purpose of each test and the results that the participant would see on the laptop, answering any questions they may have had.

Prior to commencement of the trial procedures, all participants completed testing as part of their usual elite scholarship including; incrementally-graded treadmill running protocol to determine lactate threshold (Appendix D) (and 30m sprint tests to determine maximum velocity (km/h) (Appendix E) using Catapault GPS Catapult Sports, 2018). Through these tests, speed bands were set individually on the GPS units which allows for individualised monitoring of the participants' abilities to hit and maintain certain speeds (Hunter et al, 2015; Lovell & Abt, 2013) and therefore quantify training load, by determining values of high speed running and sprinting distance.

On each testing date over the two-week period participants consumed a controlled breakfast (two slices of brown toast with either butter and jam or scrambled eggs), which was the regular breakfast they would consume in the club canteen on a daily

basis. Participants were also asked to refrain from caffeine and alcohol consumption for the entire protocol, however this was not monitored when they were away from the club. Each participant had the option of apple, orange or cranberry juice and were allowed one 250ml glass of the juice of their choice. It was advised that individuals maintained a healthy, balanced diet throughout the protocol. Whilst at the club's training ground, players consumed meals cooked by the club chef (Appendix F) with participants and parents/guardians being advised on the optimal diet whilst at home, including the consumption of carbohydrates and proteins to aid recovery. Participants were told to refrain from any physical activity other than the training that was completed at their club.

## 3.5 Experimental Protocol

Participants completed the testing protocol on six occasions for each of the two separate weekly periods, as shown in Table 1. The first testing session (described in the following sections) was completed prior to kick off on matchday, at which point the participant should have been at their optimal physical condition, with the second test coming immediately post-match when they should be in their most fatigued state (Rowell et al.2017).

Table 1 Weekly Testing Schedule. Pre and post-match testing were completed on the Saturday (Matchday (MD)). Recovery status was then recorded prior to training each day through the following week. The training sessions were completed on Monday (MD +2), Tuesday (MD +3), Thursday (MD +5) and Friday (MD +6). Participants had a day off on the Sunday and reported to college on the Wednesday and therefore did not complete any physical activity.

Saturday Matchday (MD)	Sunday	Monday MD +2	Tuesday MD +3	Wednesday	Thursday MD +5	Friday MD +6
<u>08:00 – 09:00</u> Testing pre- match	OFF	08:00 – 09:00 Testing pre- training	<u>08:00 – 09:00</u> Testing pre- training	OFF	08:00 – 09:00 Testing pre- training	08:00 – 09:00 Testing pre- training
<u>09:50 – 10:25</u> Pre-match warm up		<u>10:30-11:45</u> Training - intervention	<u>10:30-11:55</u> Training - intervention		<u>10:30-11:50</u> Training - intervention	<u>10:30-11:35</u> Training - intervention
<u>10:30 – 12:15</u> Match						
<u>12:15 – 13:15</u> Testing post- match						

As the independent variable of this study was the nature of the training protocols over two separate weeks, sessions were designed to elicit different physiological outcomes. The aerobic training week (TW-A) was of an aerobic nature, with larger distances covered at a slower speed, whilst the anaerobic training week (TW-AN) was of high intensity and anaerobic nature, limiting distances covered and aiming for an internal loading with large numbers of accelerations and decelerations. The sessions completed over the two weekly protocol can be seen in Appendices G to N.

The weekly schedule of players rarely varied, due to commitments such as education on a Wednesday, therefore training occurred on the same days at the same times each week (see Table 1). To modify the independent variable and allow differing training stimuli over the two weeks, the squad completed standardised sessions on Tuesdays and Fridays, with the sessions on Mondays and Thursdays being different to elicit varied physical responses (Table 2). Whilst the session plans (Appendices G to N) were different on these days between weeks TW-A and TW-AN, they were designed to create similar physical outputs in terms of total distance and high-speed

running (metres >17km/h), however, the technical and tactical outputs of the session were required to be different due to the requirements of the technical coaches. During the testing protocol, the total training load was accrued through the pitch-based sessions seen in Table 1. Participants did not complete any extra sessions such as; gym-based conditioning, pitch-based running, recovery, Pilates, yoga.

The individual sessions throughout the week were monitored, with each of the participants wearing a Catapult Minimax S4 GPS unit (Melbourne, Australia) which works at a frequency of 10 Hertz (Catapult (1), 2018) and allows numerous metrics to be recorded, including total distance (m) and high speed distance, which was quantified as any distance a player covered at >17km/h, which was the threshold that had been used in previous seasons by the club, and therefore comparisons could be made with previous data when assessing the physical capabilities of players. This therefore helped the researcher to quantify intensities of sessions due to previous experience at using this threshold.

Participants arrived at the pre-match testing in a rested state, having completed a light training session 24 hours previously (See Appendices K and L) The Rate of Perceived Exertion (RPE) was taken from each player prior to this session to monitor their training load, ensuring their rested state for their protocol. Players would be excluded from testing if they presented an RPE of 8 or greater (described as a very hard session), however none were required to be removed (RPE 5  $\pm$  1).

## 3.5.1 Pre-Testing Warm Up

Participants completed a standardised warm up before each of their testing dates. The warm up consisted of two parts:

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- 1) A pulse raiser and muscle activation stage. Each participant completed a fiveminute spin at 100 revolutions per minute (rpm) on Gear 6 of a Keiser M3 spin bike, which was chosen as this was both reproducible by all individuals, as was regularly completed by all participants as the warm up for their lower body strength sessions during their regular training weeks. This was followed by ten bodyweight squats and ten bodyweight lunges on each leg. They finished this stage of the warm up with five countermovement jumps with their hands on their hips. Each individual then sat on the seat and was connected to the EMG and had their right ankle strapped into the non-compliant strap.
- 2) Once the equipment was set up, participants were instructed to complete a right leg extension at 50%, 75% and 100% of their maximal voluntary contraction (MVC), with a 20 second break between each contraction. The 100% MVC was recorded to prepare the Labchart software for individualised zones of 50%, 75% and 100% MVC for the testing protocol.

The only exception to this warm up process was the post-match testing when players completed stage two of the warm up, but not stage one, as they should already be physically prepared for the test having just come off the pitch. Stage two was required to set individual zones on the Labchart software.

## 3.5.2 Electromyography Testing

The evoked force and electromyographic (EMG) responses of the Vastus Lateralis of the right leg to two differing stimuli to discover the changes in CNS and muscle function due to fatigue (Thomas et al. 2016):

a) Transcranial Magnetic Stimulation (TMS) of the motor cortex

#### b) Peripheral Electrical Stimulation (PS) of the femoral nerve

A calibrated load cell (Maypole LTD., Birmingham) was attached to the participant's right leg via a non-compliant strap, superior to the ankle malleoli. This strap, used to record muscle force (N) during isometric knee-extensor contractions, was connected to a myometer (Digital Multi-Myometer, MIE Medical Research Ltd, Leeds, UK) which converted the force to Newtons (N). The positioning of the strap was altered for different participants to ensure consistency. It was ensured that during testing, participants were sat with hip and knee angles at 90°, in an upright position. The participants were advised to grip the seat for support during contractions, especially those at maximum effort (see Figure 5 (A)).

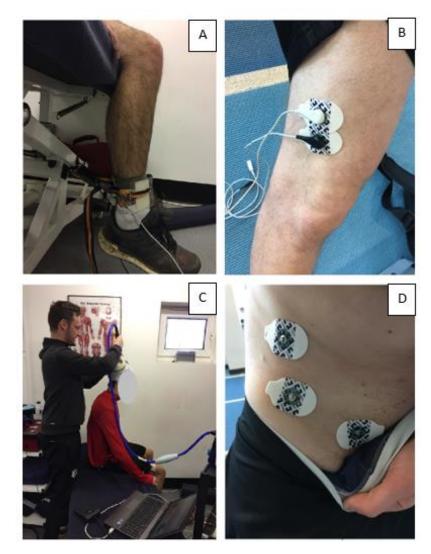


Figure 5 Electrode placement sites A) The location of the calibrated load cell attached to the participant's right leg via a non-compliant strap, superior to the ankle malleoli. B) The placement of two bipolar surface electrodes, placed 20mm apart over the muscle belly of the Vastus Lateralis. C) The position of the concave double coil used for Transcranial Magnetic Stimulation. D) Placement of electrodes in the femoral triangle.

Electromyography (EMG) was used as a method to evaluate and record the electrical activity produced by skeletal muscle. Two bipolar surface electrodes were placed 20mm apart over the muscle belly of the Vastus Lateralis (VL) of the individual (Thomas et al. 2017) to target the femoral nerve, which Rao (2015) states is responsible for extension of the knee, crucial for football (Figure 5 (B)). This electrode placement replicates that from the study on a simulated soccer match by Thomas et al. (2017). Before electrodes were placed, the skin was shaved and wiped with an alcohol wipe. The reference electrode for TMS was connected to the lateral malleoli

of the right ankle via a dry earth strap. The placement of electrodes was kept consistent throughout the study to ensure the reliability of the results (Figure 5 (D)). A myometer (Mecmesin, Slinfold) was interfaced with a Powerlab 26T (ADinstruments, Sydney, Australia) to measure the isometric muscle strength of the quadriceps during each of the increasingly intense contractions. The raw data from the Powerlab 26T was relayed into a laptop displaying EMG activity (mv).

When recording the data through the Labchart software, which was part of the Powerlab package, the low pass filter was turned on. This filter is described as an analogue anti-aliasing filter as it attenuates the higher frequencies, allowing lower frequencies to be recorded (National Instruments, 2018). The frequencies removed are those which are greater than the Nyquist frequency, defined by National Instruments (2018) as the minimum rate at which a signal can be sampled without error, generally considered to be at least twice the maximum frequency of the signal of interest. This filter therefore removes "noise" from other electronic or radio frequencies present at the time of testing and was set to cut off frequencies lower than 0.5 Hertz (Hz) and greater than 10Hz, which may cloud the results with unreliable data.

### 3.5.3 Transcranial Magnetic Stimulation Testing Protocol

Each participant had been familiarised to this protocol a minimum of once prior to the commencement of testing. Single pulse Transcranial Magnetic Stimulation (TMS) was delivered using a concave double coil (110mm diameter, maximum output 1.4T), powered by a mono-pulse magnetic stimulator (Magstim 200, The Magstim Company Ltd., Whitland, UK). The double coil has two large cup shaped windings positioned side by side, with a flat central section and angled sides to fit the participant's head

(Magstim, 2018). This coil allows for better magnetic coupling which in turn induces a significantly higher current in the central fissure (Magstim, 2018). This coil is utilised to stimulate the motor cortex areas which control the muscles of the lower body.

The coil was held over the vertex to stimulate the left hemisphere in the optimal position to evoke the greatest Motor Evoked Potential (MEP) in the Vastus Lateralis (VL) of the right leg, alongside a small MEP in the agonist; the biceps femoris. Each participant wore a white swimming cap to allow the researcher to mark the optimal coil location and ensuring reproducibility of the stimulation (see Figure 5 (C)).

The participant underwent 1ms stimulations of 55% intensity, with an eight second break between stimulations. Once the location that induced the greatest EMG response was found, the intensity percentage was manipulated to find the level at which an involuntary response was recorded from the VL at above 0.05 microvolts in five out of ten stimulations. Once the lowest possible intensity was recorded, this was increased by 20% to ensure a supramaximal stimulation (Thomas et al. 2016). Single pulse TMS was then delivered at this given intensity during brief (3-5 s) contractions at 50%, 75% and 100% MVC, separated by 10 s of rest to determine voluntary activation with TMS. It was ensured that participants were able to contract at the desired intensities as guidelines were shown on the screen in front of them at 45-55% and at 70-80% MVC, based on the MVC produced during the warm up. They were instructed to contract maximally for the third attempt so therefore did not require guidelines.

The percentage of voluntary activation (%VA) is a calculation of the percentage of the stimulated contraction that was produced voluntarily i.e. if the voluntary contraction was 90N and the stimulated contraction via TMS was 100N, the percentage of

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voluntary activation would be 90%. The use of this measurement removes the comparison of raw data (the force produced (N), and shows the percentage of muscle activation an individual could produce, ignoring whether this was at a high or low force.

#### **1.5.2 Peripheral Stimulation Testing Protocol**

Single, electrical stimuli (200µs pulse width) were delivered to the right femoral nerve through self-adhesive surface electrodes (CF3200, Nidd Valley Medical Ltd., North Yorkshire, UK) using a constant current stimulator (DS7AH, Digiteemer Ltd., Welwyn Garden City, Herfordshire, UK). The cathode was positioned on the nerve, in the femoral triangle, with the anode positioned midway between the greater trochanter and the iliac crest (Thomas et al. 2016). Trial and error was used to ensure the correct location was found, with the twitch amplitude on screen indicating when the nerve was located. Single stimuli were delivered to the relaxed muscles beginning at 40 mA, the intensity was increased by 20 mA until a plateau occurred in twitch amplitude and M-wave. To ensure a consistent supramaximal stimulus and account for any changes in axonal excitability caused by the development of fatigue, the stimulation intensity was then increased by 30%. Single electrical stimuli was then delivered at this given intensity during brief (3-5 s) contractions at 50%, 75% and 100% MVC, separated by 10 s of rest to determine voluntary activation with peripheral stimulation.

Each participant was familiarised to peripheral femoral nerve stimulation in two trials in the weeks prior to testing commencing. The complete testing protocol can be seen in Figure 6.

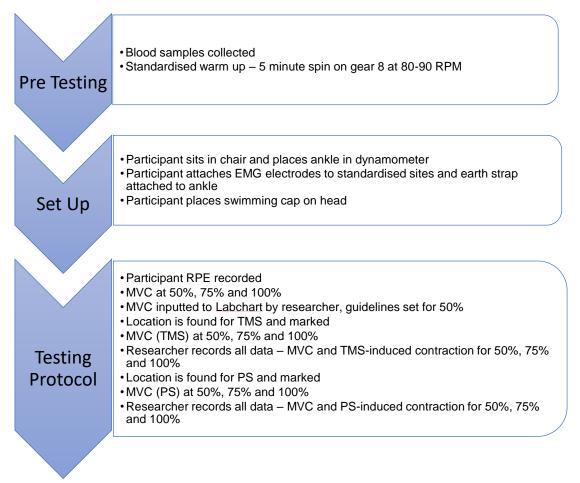


Figure 6 Testing Protocol. This diagram outlines the process from start to finish of each testing protocol, that the participants completed six times per week.

# 3.6 Physical Performance and Intensity Measurement

A number of metrics were measured as part of the experiment to monitor the training load that each individual completed throughout the testing process. These metrics will be described in the following sections.

# 3.6.1 Global Positioning System

The Global Positioning Software (GPS) units (Catapult Minimax S4) (Figure 7) were positioned between the shoulder blades of the individuals using a vest which was the

appropriate size to prevent excess movement during activity. The equipment used and placements were consistent throughout all trials of each individual.



Figure 7 Catapult Minimax s4 unit.

The GPS data was displayed in the software (Catapult Sprint 5.1.7) after download. This data was then converted to Microsoft Excel format to be analysed. The participants were acclimatised to the GPS units during their regular training sessions prior to both the trial and the testing. These units were then worn for the duration of all training sessions and matches during the testing protocol

The measures recorded were: Total Distance (TD, m), High Speed Running Distance (distance of speeds >17km/h) (HSR, m), Player Load (PL).

The reliability of 10Hz Catapult GPS units was tested by Johnston et al. (2014), who found that there was <1% error for total distance measures when comparing the actual distance travelled to that tracked by the unit. There was also <1.9% error between units for this measure. The study found that 10Hz units, as used in this study, were more reliable then 1Hz, 5Hz and 15Hz units.

However, it was found that error for speeds over 20km/h was >10%, which can have a significant effect on total high-speed running distance. However, with a lower threshold for high speed running in this study (>17km/h), this will be more reliable than previous studies that use higher thresholds.

#### 3.6.1.1 Total Distance

Each GPS unit calculates the position of the individual by timing the signals emitted by GPS satellites orbiting the earth. Each satellite continually transmits messages, including the time of transmission and the location of the satellite. By calculating the distance between the satellite and the GPS unit, it is possible to calculate the distance travelled as the athlete moves (Catapult, 2019 (4)). This metric was chosen to identify how far, on average, participants travelled in each match or training session. It would be expected that in TW-A, where sessions were designed to be in bigger areas, that participants would cover greater distances.

### 3.6.1.2 High Speed Running Distance

As described previously, by calculating the distance between the satellite and the GPS unit, it is possible to calculate the distance travelled as the athlete moves (Catapult, 2019). The High-Speed Running distance relies more greatly on the time recorded at each location, as it refers to any distance an individual travels at greater than 17km/h. This metric was chosen to contextualise the external load of each session, with running at high speeds putting strain on lower limb muscles, including hamstrings and quadriceps. It would be expected that in TW-A, where sessions were designed to be in bigger areas, that participants would cover greater distances of high-speed running, This is due to participants being unable to accelerate quickly enough in smaller areas to be able to hit higher speeds without leaving the training area.

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#### 3.6.1.3 Player Load

Player load is a metric calculated through the following algorithm, where  $a_y$  is forward (anterior-posterior) acceleration,  $a_x$  is sideways (medial-lateral) acceleration, and  $a_z$  is vertical acceleration (Nicollela et al. 2018).

$$PlayerLoad^{TM} \sqrt{\frac{(a_{y(t)} - a_{y(t-1)})^2 + (a_{x(t)} - a_{x(t-1)})^2 + (a_{z(t)} - a_{z(t-1)})^2}{100}},$$

Player Load measures work performed independent of distance, providing an objective view of an athlete's workload at any given time. The cumulative total throughout each session is calculated through an algorithm which includes changes of direction, distance (m) and speed (km/h), allowing a more detailed analysis and quantification of work completed by individuals when working in tight areas and producing high forces through high intensity efforts, such as during small-sided games (Catapult, 2019 (5)).

This metric was chosen to allow comparison between the two training weeks on the intensity of the session in its entirety, taking into account all metrics including total distance, high speed running distance, accelerations, decelerations and high intensity efforts.

#### 3.6.2 Blood Lactate Analysis

The blood lactate analyser (Biosen C-Line, EKF Diagnostics, Penarth) (figure 8) was prepared and calibrated approximately 30 minutes prior to testing. Blood samples were taken from participants both pre and post-game via an earlobe prick (Accu-Check Safe-T Pro, UK) on the participant's right ear and entered into the analyser,



Figure 8 Blood lactate analyser (Biosen C-Line, EKF Diagnostics, Penarth)

identifying blood lactate concentration (mmol/l). The researcher wore protective vinyl gloves and all equipment used was disposed of in a sharps bin. The Biosen C-Line is seen to be reliable, with a coefficient of variation of <1.5% (EKF Diagnotiscs, 2019).

An earlobe was chosen due the previous testing batteries conducted by the participants involved in the study who had been acclimatised to earlobe blood sampling, this making the testing procedure more efficient.

It has been noted that capillary earlobe sampling is suitable for collection of the 100-200µl of blood required for the Biosen blood lactate analyser (Coulson and Archer, 2015). Coulson and Archer (2015) continue to add that blood lactate concentrations were between 0.3 and 0.5 mmol/l<sup>-1</sup> lower in earlobe samples when compared to fingertip samples which is likely due to delays in the release in lactate into centralised circulation due to changes in localised blood flow in the forearms. This was discovered in a study of rock climbers (Draper et al. 2006) who rely on their upper limbs more greatly than football players so the difference in fingertip and earlobe blood lactate concentration could be expected to be more minimal in this study.

When taking a blood sample, the researcher was careful to avoid cross-contamination by changing gloves and washing hands between participants, having been trained and had previous experience in gaining earlobe blood samples. It was crucial that the blood samples obtained were full when transported into the capillary tube to increase the accuracy of the lactate measurement.

Pre-match blood samples were taken immediately prior to the commencement of the pre-testing protocol standardised warm-up, with post-match lactate analysis taking place pitch-side, immediately as the player came off the pitch.

#### 3.6.3 Rating of Perceived Exertion

Prior to testing, participants had been used to reciting their Rate of Perceived Exertion (RPE) to staff at their football club on a scale of 1-10 and had been educated on the meaning of each score during a presentation at the beginning of the current season. There is a scale on the wall of the changing room (See Appendix O) which players can revisit at any time to refresh their understanding. RPEs were given to the researcher verbally immediately following each training session and game to allow individuals to give their initial feelings on the session rather than allowing recovery time to alter their perceptions of the session difficulty. The RPE scale was shown to the participants to refresh their knowledge each time they gave a score. However, scores may have been affected by the public location of the data collection. With individuals hearing scores given by fellow participants, this may have affected the scores that they gave, reducing the validity of the data.

The researcher timed the duration of the session (minutes) from the beginning of the warm up until the end of the final drill, including rest breaks and transition between

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drills and this was multiplied by the given RPE to give and RPE training impulse (Trimp) as a measure of the total training load.

## 3.7 Reliability and Validity

To ensure the reliability and validity of the measurement, the equipment used, and placements of the electrodes on the hip and the vastus lateralis were consistent throughout all trials of each individual. These sites were found through palpation and marked on each individual position.

It was crucial that the methods for locating the hotspot in the region of the brain for the TMS testing was done in a consistent method. This was done through trial and error of coil placement whilst stimulating the motor cortex. Once the location which found the greatest involuntary response on the EMG trace was found, the researcher marked this on the white swimming cap by drawing around the coil with a marker and ensured that the coil was in the position during each test. This trial and error method was then replicated for locating the femoral nerve for peripheral stimulation testing.

To ensure the reliability of the testing procedures, all TMS and PS testing took place in the physio-room at the training ground to allow reproducibility. Generally, quantitative methods provide summaries of data that suggest generalisations of the phenomenon under study, with fewer variables but more strictly adhered to procedures, allowing validity and reliability in the study (Matveev, 2002), which allows the study to be replicated (Babbie 2017). Horn (2008) adds that to maintain validity and reliability, these studies must also be carried out in unnatural and artificial environments, resulting in laboratory results rather than real-world results. It has been identified that within field-study, it is difficult to complete experiments within these unnatural environments and therefore reliability and validity may be compromised. John (2017) states that experiments risk failure or compromised implementations due to the requirement for everything to be in the right place at the right time, often relying on the cooperation of third parties.

## 3.8 The Training Intervention

As previously discussed, the independent variable is the nature of the training protocols over two separate weeks that were designed to elicit different physiological outcomes. These will be described in the following sections.

## 3.8.1 Aerobic Training Week

The Aerobic Training Week (TW-A) was designed to allow players to cover higher distances during the intervention sessions on the Monday and Thursday, which would allow participants to hit greater distances at high speed (>17km/h) which allows for more a highly aerobic training session, therefore the durations of each section were of a greater duration and with less recovery time. The coaching staff at the academy were required to complete certain coaching topics throughout the week which were combined with the physical parameters to design the week. The session durations and areas can be seen in Table 2.

Day	Drill	Duration	Area size
	Warm up	15 minutes	15m x 10m
	Passing drill	4 x 3 minutes	20m x 30m
		30 seconds	
		recovery	
	Possession	4 x 3 minutes	20m x 25m
Monday		1 minute recovery	
MD +2	Possession	6 x 2 minutes	15m x 15m
		30 seconds	
		recovery	
	Small sided game	3 x 6 minutes	30m x 40m
		90 seconds	
		recovery	
	Warm up	18 minutes	20m x 20m
	Passing drill	4 x 3 minutes	20m x 30m
Tuesday		30 seconds	
Tuesday	14/	recovery	45 00
MD +3	Wave game	20 minutes	45m x 60m
	Small-sided game	2 x 7.5 minutes	30m x 60m
		90 seconds	
		recovery	Em x 40m
	Warm up	15 minutes	5m x 40m
Thursday	Passing Drill	4 x 3 minutes	20m x 30m
MD +5		30 seconds work 12 minutes	65m x 30m
	Wave game Match	2 x 12 minutes	
	Match		Full pitch 105m x 65m
	Individual work	2 minutes recovery 10 minutes	
	Warm up	12 minutes	10m x 15m
Friday	Possession	3 x 4 minutes	10m x 25m
MD +6	L 02262210[]	1 minute recovery	10111 X 2011
	Game	12 minutes	25m x 30m
	Set piece practice	12 minutes	

 Table 2 The Physiological breakdown of drills, durations and area sizes of each of the sessions in the Aerobic Training Week

## 3.8.2 Anaerobic Training Week

The Anaerobic Training Week (TW-AN) was designed to allow players to cover shorter distances during the intervention sessions on the Monday and Thursday, which would require participants to hit lower distances at high speed (>17km/h. Players were therefore in smaller areas, requiring a greater number of accelerations, decelerations

and changes of direction. Due to the strain on the participants during these more intense anaerobic drills, the duration of each section was shorter and with greater recovery time. The coaching staff at the academy were required to complete certain coaching topics throughout the week which were combined with the physical parameters to design the week. The session durations and areas can be seen in Table 3.

Day	Drill	Duration	Area size
	Warm up	15 minutes	15m x 10m
Monday	Passing drill	12 minutes	30m x 40m
MD +2	Attacking /	30 minutes	20m x 25m
	defending practice		
	Warm up	15 minutes	28m x 28m
	Transfer game	6 x 90 seconds	10m x 25 m
		30 seconds	
		recovery	
	Possession	2 x 4 minutes	30m x 30m
Tuesday		1 minute recovery	
MD +3	Running drill	3 x 35 seconds	25m x 30m
		70 seconds	
		recovery	
	Possession game	6 x 90 seconds	30m x 40m
		30 seconds	
		recovery	
	Small-sided game	8 minutes	30m x 40m
	Warm up	15 minutes	10m x 20m
	Passing drill	15 minutes	50m x 60m
Thursday	Pattern of play	12 minutes	50m x 60m
MD +5	Small-sided game	2 x 8 minutes	30m x 30m
		2 minutes recovery	
	Individual work	10 minutes	
	Warm up	12 minutes	10m x 15m
	Possession	6 x 1 minute	10m x 15m
Friday		30 seconds	
MD +6		recovery	
	Pattern of play	15 minutes	65m x 30m
	Game	6 x 3 minutes	30m x 30m
	Set pieces	10 minutes	

Table 3 The Physiological breakdown of drills, durations and area sizes of each of the sessions in the Anaerobic Training Week

### 3.9 Data Analysis

All data recorded for each participant was co-ordinated in Microsoft Excel and both the mean and standard deviation were calculated. Graphics such as tables, bar charts and line graphs were used to initially analyse the results. Normality testing was completed via a Shapiro-Wilk test on SPSS. This normality test was chosen as it is more applicable to sample sizes of <50 (Laerd Statistics 2019).

IBM SPSS Statistics (Version 23) was then used to conduct statistical analysis. Mixed between-within subjects', repeated measures analysis of variance (ANOVA) was completed for a number of different variables:

- Transcranial Magnetic Stimulation Deficit from stimulated contraction to maximal voluntary contraction (MVC). The deficit is the difference between the force produced in a voluntary contraction and the evoked force produced during a stimulated contraction via TMS, with a greater deficit in the voluntary contraction suggesting an inability of the muscle to produced maximal force (Thomas et al. 2017).
- 2) Transcranial Magnetic Stimulation Percentage of MVC
- 3) Peripheral Stimulation Deficit from stimulated contraction to MVC
- 4) Peripheral Magnetic Stimulation Percentage of MVC

A level of probability was set at p<0.05 for all statistical test to show a statistical difference between variables, such as training week (TW-A and TW-AN) and day of the week (Saturday, Monday, Tuesday, Thursday, Friday). Due to the small sample size, it was expected that a number of the Shapiro-Wilks tests would show a non-normal distribution of data. Whilst it could be argued that a non-parametric test would therefore be a more suitable test as it does not assume normality of data, there are

other issues that come with these tests (Glass et al. 1972, Harwell et al. 1992, Lix et al. 1996). As non-parametric tests assume that data in different groups have the same distribution as eachother, if they have different shaped distributions, such as left-skewness or right-skewness, a parametric test may be better (Glass et al. 1972, Harwell et al. 1992, Lix et al. 1996).

The Post-Hoc test utilised was the Wilks-Lambda. This was chosen as a direct measure of the proportion of variance which is unaccounted for by the independent variable (the training intervention) (Bartlett et al. 2000). Wilks' lambda statistic can be mathematically adjusted to a statistic which makes it easier to calculate the P-value (Bartlett et al. 2000).

Standard deviation was used to compare the distribution of the data. Arnold and Schilling (2017) argue that the standard deviation should be chosen over the standard error as most statistical analyses are examining the sample and not making mathematical inferences of the whole population. Partial etc squared was used to identify effect size.

Paired samples t-tests were also used to for the comparison of means for a number of different variables:

- All physical performance and intensity measurements, including Total Distance, High Speed Running distance, Player Load, RPE, RPE trimp and blood lactate concentrations.
- 2) Comparison of maximal voluntary contraction, deficit and percentage of voluntary activation results from TMS and PS for all testing dates.
- To identify where any differences occurred in the event of inconclusive posthoc tests.

To minimise the risk of type-1 errors, the significance level was set to 95%. Additionally, the post-Hoc Bonferroni test was used, as Andrade (2019) suggests that when conducting multiple analyses on the same dependent variable, the chance of committing a Type I error increases, increasing the likelihood of coming about a significant result by chance. However, to correct for this, or protect from Type I error, a Bonferroni correction is conducted.

The next chapter deals with the results of the research findings and their discussions thereof.

## 4.0 Results

The purpose of this chapter is to analyse and present the empirical information gained through the study. The chapter is divided into seven sections focussing on each of the independent and dependent variables, concluding with a summary of the findings. The sections are divided as follows:

- 1) Maximal Voluntary Contraction
- 2) Transcranial Magnetic Stimulation (TMS)
- 3) Peripheral Stimulation (PS)
- 4) Global Positioning System (GPS) metrics
- 5) Rate of Perceived Exertion (RPE)
- 6) RPE Trimp
- 7) Blood Lactate

Throughout this section, all significance values are rounded to three significant figures.

## 4.1 Maximal Voluntary Contraction

As part of the standardised pre-testing warm up, each participant was required to complete a three-second isometric quadricep contraction at what they perceived to be 50%, 75% and 100% intensity. This maximal voluntary contraction (MVC) at 100% was recorded and used to set the intensity required through the procedure.

A Shapiro-Wilk test of normality was conducted prior to the statistical analysis (p = <0.039). Whilst this shows the data was not normally distributed, a mixed betweenwithin subjects' analysis of variance was used to analyse the data, as Kozak and Piepho (2018) argue that this is still an effective tool with the sample size. Mean force  $\pm$  standard deviation produced by the ten participants dropped from prematch to post-match by 8.08% in TW-A from 574.40  $\pm$  109.72N to 528.00  $\pm$  123.41N, however this was not significant (p = 0.337).

Mean force produced by the ten participants dropped from pre-match to post-match by 10.23% in TW-AN from 615.10  $\pm$  133.94N to 552.20  $\pm$  150.52N, however this was not significant (p = 0.386).

Mean MVCs returned to greater than the Pre-Match MVC by MD+2 (TW-A – 105.95% and TW-AN – 105.46%). TW-A saw a progressive increase in MVC force throughout the week, with a maximum mean of 702.10  $\pm$  136.13N on MD+6.

TW-AN however peaked on MD+3 at 701.60  $\pm$  158.54N with mean MVCs remaining greater than the pre-match baseline for the remainder of the testing week.

A mixed between-within subjects' analysis of variance was conducted to assess the impact of two different interventions (TW-A and TW-AN) on participants' maximal voluntary quadriceps contractions (MVC) across 6 different testing dates throughout the week (Pre-match, Post-match, Monday – MD+2, Tuesday – MD+3, Thursday – MD+5, Friday – MD+6). There was no significant interaction between testing day and training week, Wilks' Lambda = 0.315, F (5, 14) = 1.285, p = 0.324, partial eta squared = 0.315. There was a significant effect for testing day, Wilks' Lambda = 0.821, F (5, 14) = 12.869, p = 0.000. Through post-Hoc Bonferroni tests, it was found that there were significant differences on between the pre-match testing and MD+3 (p= 0.006), MD+5 (p= 0.010) and MD+6 (p = 0.000). There were also significant differences between the post-match testing and testing on MD+3 (p= 0.000), MD+5 (p= 0.000) and MD+6 (p = 0.821, suggesting that the change in MVC

throughout the week was substantial (see Figure 9). Data can be seen in Appendix P (1).

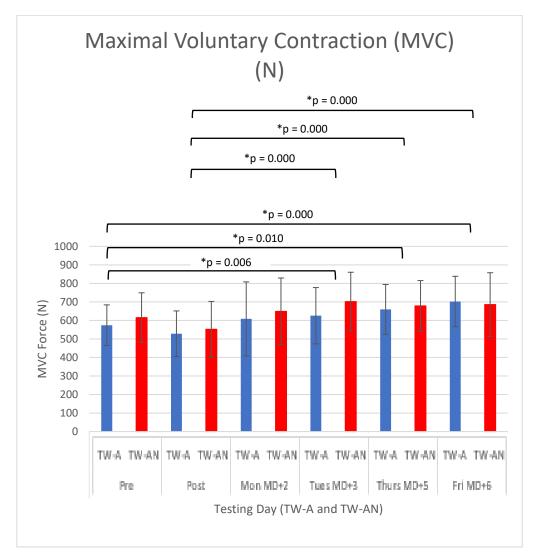


Figure 9 Mean force (N) for the Maximal Voluntary Contraction for both Training Weeks. Pre-match – MD+3 \*p = 0.006, pre-match to MD+5 \*p = 0.010, pre-match to MD+6 \*p = 0.000, post-match to MD+3 \*p = 0.000, post-match to MD+5 \*p = 0.000, post-match to MD+6 \*p = 0.000.

The between subjects' effect was not significant, F (1, 18) = 0.247, p = 0.625, partial eta squared = 0.014, suggesting no difference in the effectiveness of the two training weeks. Paired samples t-tests were used to more greatly compare the MVC on each testing day (Table 4).

	Aerobic Training			Anaer	robic Trair	ing	Comparison	
	Week				Week	٦	TW-A v TW-AN	
Testing Date	n	М	SD	n	М	SD	Р	
Pre-Match	10	574.40	109.72	10	615.10	133.94	0.121	
Post-Match	10	528.00	123.41	10	552.20	150.52	0.555	
Monday (MD+2)	10	608.60	199.68	10	648.70	180.58	0.478	
Tuesday (MD+3)	10	625.50	152.01	10	701.60	158.54	0.228	
Thursday (MD+5)	10	659.40	134.57	10	678.40	136.91	0.511	
Friday (MD+6)	10	702.10	136.13	10	685.60	171.96	0.636	

Table 4 Comparison of MVC over the two training weeks.

As seen in Figure 10, mean MVC for the centre forwards (CF) (n = 2) was the highest for all testing days and the only position in which MVC increased post-match. There were significant differences in TW-AN between CF and centre backs (CB) on both MD+2, p = 0.011 and MD+3, p = 0.043. There were no other significant differences between positions.

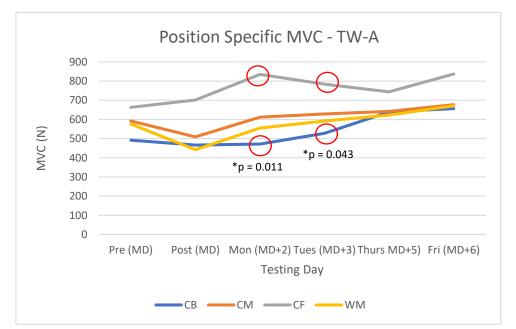


Figure 10 Mean force (N) for the Position Specific Maximal Voluntary Contraction TW-A. MD+2 TW-A (CB-CF \*p = 0.011), MD+3 (CB-CF \*p = 0.043)

As seen in Figure 11, mean MVC for the centre forwards (CF) (n = 2) was the highest for all testing days and the only position in which MVC increased post-match in TW-A. There were significant differences between CF and centre backs (CB) on both prematch testing, p = 0.008 and MD+6, p = 0.029. There were no other significant differences between positions.

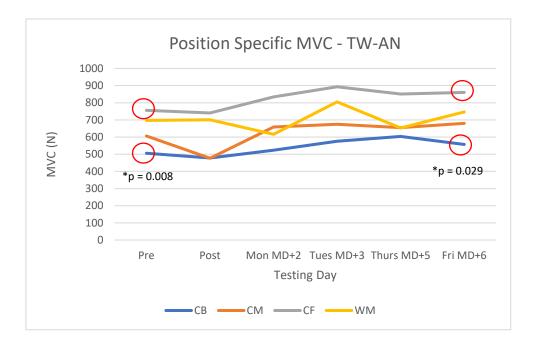


Figure 11 Mean force (N) for the Position Specific Maximal Voluntary Contraction TW-AN. Pre-match TW-AN (CB-CF \*p = 0.008), MD+6 (CB-CF \*p = 0.029)

## 4.2 Transcranial Magnetic Stimulation

Transcranial Magnetic Stimulation (TMS) was completed on each of the testing dates to measure the contribution of the central nervous system to any force production reductions. Both the deficit and the percentage of voluntary activation were analysed to allow comparison between both central and peripheral nervous systems.

#### 4.2.1 TMS Deficit

A Shapiro-Wilk test of normality was conducted prior to the statistical analysis (p = <0.001). Whilst this shows the data was not normally distributed, a mixed betweenwithin subjects' analysis of variance was used to analyse the data, as Kozak and Piepho (2018) argue that this is still an effective tool with the sample size.

As seen in Figure 13 the mean deficit between MVC and TMS-induced contraction increased from 25.84  $\pm$  20.13N to 38.46  $\pm$  19.86N in TW-A from pre-match to post-match, however was not a statistically significant difference (p = 0.175).

The mean deficit between MVC and TMS-induced contraction increased from  $25.02 \pm 13.85$ N to  $38.64 \pm 21.00$ N in TW-AN from pre-match to post-match, however this was not a statistically significant difference (p = 0.104).

Mean TMS deficit returned to the Pre-Match baseline by MD+2 in TW-A (25.57  $\pm$  20.32N), however remained elevated in TW-AN (37.55  $\pm$  25.96N). TW-A saw baseline deficit scores throughout the week, excluding a maximum deficit on MD+3 of 35.92  $\pm$  32.31N (see Figure 15). These changes were not statistically significant (p = 0.100).

Whilst TMS deficit peaked on MD+2 in TW-AN, mean TMS deficit returned to baseline values by MD+3 and continued to decrease by the end of the week. These changes were not statistically significant (p = 0.100).

A mixed between-within subjects' analysis of variance was conducted to assess the impact of two different interventions (TW-A and TW-AN) on participants' deficits from evoked force during TMS to maximal voluntary quadriceps contraction across 6 different testing dates throughout the week (Pre-match, Post-match, Monday – MD+2, Tuesday – MD+3, Thursday – MD+5, Friday – MD+6). There was no significant interaction between testing day and training week, Wilks' Lambda = 0.831, F (5, 14) =

0.571, p = 0.722, partial eta squared = 0.169. There was also no significant effect for testing day, Wilks' Lambda = 0.538, F (5, 14) = 2.307, p = 0.100, partial eta squared = 0.452. (Figure 12). Data can be seen in Appendix P (2).

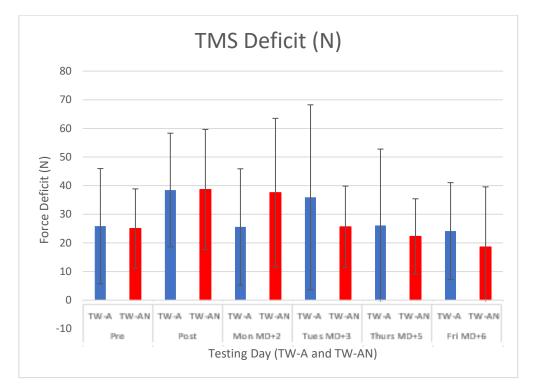


Figure 12 Mean TMS Deficit (N) for both Training Weeks.

The between subjects' effect was not significant, F (1, 18) = 0.04, p = 0.840, partial eta squared = 0.002, suggesting no difference in the effectiveness of the two training weeks. Paired samples t-tests were used to more greatly compare the TMS deficit on each testing day. As seen in Table 5, there were no significant differences found between the two weeks for the TMS deficit.

	Aerobic Training			Anaerobic Training			Comparison
	Week		Week			TW-A v TW-AN	
Testing Date	n	М	SD	п	М	SD	Р
Pre-Match	10	25.84	20.13	10	25.02	13.85	0.886
Post-Match	10	38.46	19.86	10	38.64	21.00	0.976
Monday (MD+2)	10	25.57	20.32	10	37.55	25.96	0.100
Tuesday (MD+3)	10	35.92	32.31	10	25.61	14.23	0.208
Thursday (MD+5)	10	26.08	26.69	10	22.27	13.13	0.687
Friday (MD+6)	10	24.15	16.90	10	18.57	21.00	0.560

Table 4 Comparison of TMS deficit over the two training weeks.

As seen in Figure 13, mean TMS deficit fluctuated greatly for all positions in TW-A, however there were no significant differences between positions.

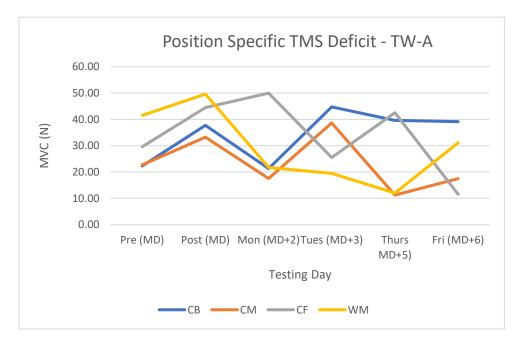


Figure 13 Mean force (N) for the Position Specific TMS deficit TW-A. MD+2 TW-A.

As seen in Figure 14, mean TMS deficit fluctuated greatly for all positions in TW-AN. There was a significant difference between CF and wide midfielders (WM) on MD+5, p = 0.015. There were no other differences between positions.

Results

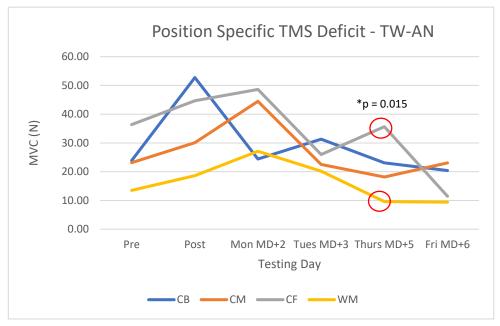


Figure 14 Mean force (N) for the Position Specific TMS deficit TW-AN. MD+5 TW-A (CB-WM \*p = 0.015).

## 4.2.2 TMS Percentage of Voluntary Activation

A Shapiro-Wilk test of normality was conducted prior to the statistical analysis (p = <0.001). Whilst this shows the data was not normally distributed, a mixed betweenwithin subjects' analysis of variance was used to analyse the data, as Kozak and Piepho (2018) argue that this is still an effective tool with the sample size.

The mean %VA during TMS-induced contraction decreased from 95.09  $\pm$  3.87% to 92.44  $\pm$  4.30% in TW-A from pre-match to post-match, however this was not statistically significant (p = 0.165).

The mean %VA during TMS-induced contraction decreased from 95.48  $\pm$  2.63% to 92.57  $\pm$  4.15% in TW-AN from pre-match to post-match, however this was not statistically significant (p = 0.077).

Mean TMS %VA returned to greater than the Pre-Match baseline by MD+2 in TW-A (95.70  $\pm$  2.87%), however remained reduced in TW-AN (94.35  $\pm$  3.25%). TW-A saw baseline deficit scores throughout the week, excluding a minimum %VA on MD+3 of

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 $93.43 \pm 6.18\%$ , suggesting a decreased central activation of the Vastus Lateralis following the training session on MD+2.

Whilst TMS deficit remained reduced on MD+2 in TW-AN, mean TMS %VA returned to baseline values by MD+3 and continued to increase by the end of the week (Figure 21), before slightly dropping from MD+5 (96.66  $\pm$  2.19%) to Friday (96.26  $\pm$  5.30%).

A mixed between-within subjects' analysis of variance was conducted to assess the impact of two different interventions (TW-A and TW-AN) on participants' percentage of voluntary activation (%) during TMS to voluntary maximal voluntary quadriceps contraction across 6 different testing dates throughout the week (Pre-match, Post-match, Monday – MD+2, Tuesday – MD+3, Thursday – MD+5, Friday – MD+6). There was no significant interaction between testing day and training week, Wilks' Lambda = 0.786, F (5, 14) = 0.762, p = 0.592, partial eta squared = 0.214. There was a significant effect for testing day, Wilks' Lambda = 0.419, F (5, 14) = 3.888, p = 0.020, partial eta squared = 0.581, suggesting a substantial change in percentage voluntary activation dependent on the day of testing throughout the protocol. Post-Hoc Bonferroni tests found a significant difference between pre-match and post-match TMS %VA (p = 0.012) and between post-match and MD+5 (p = 0.004) (Figure 15). Data can be seen in Appendix P (3).

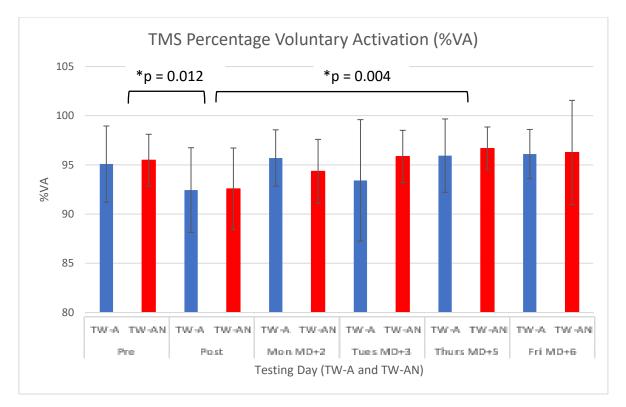


Figure 15 Mean TMS % Voluntary Activation (%) for the both Training Weeks. Pre-match to post-match \*p = 0.012, Post-match to MD+5 \*p = 0.004

The between subjects' effect was not significant, F (1, 18) = 0.11, p = 0.750, partial eta squared = 0.006, suggesting no difference in the effectiveness of the two training weeks. Paired samples t-tests were used to more greatly compare the TMS %VA on each testing day. As seen in Table 6, there were no significant differences found between the two weeks for the TMS %VA.

	Aerobic Training			Anaero	bic Trainii	ng C	Comparison
		Week		١	Week	TV	V-A v TW-AN
Testing Date	n	М	SD	п	М	SD	Р
Pre-Match	10	95.09	3.87	10	95.48	2.63	0.735
Post-Match	10	92.44	4.30	10	92.57	4.15	0.910
Monday (MD+2)	10	95.70	2.87	10	94.35	3.25	0.240
Tuesday (MD+3)	10	93.43	6.18	10	95.85	2.66	0.110
Thursday (MD+5)	10	95.94	3.73	10	96.66	2.19	0.598
Friday (MD+6)	10	96.10	2.50	10	96.26	5.30	0.928

Table 6 Comparison of TMS %VA over the two training weeks.

As seen in Figure 16, mean TMS %VA fluctuated greatly for all positions in TW-A. There was a significant difference between CB and centre midfielders (CM) on MD+6, p = 0.029. There were no other differences between positions.

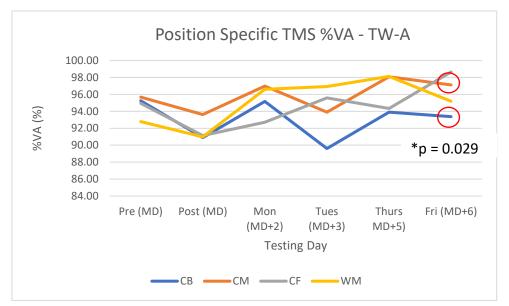


Figure 16 Mean percentage (%) for the Position Specific TMS %VA TW-A. MD+6 TW-A (CB-CM \*p = 0.029).

As seen in Figure 17, mean TMS %VA fluctuated greatly for all positions in TW-AN, however there were no significant differences between positions.

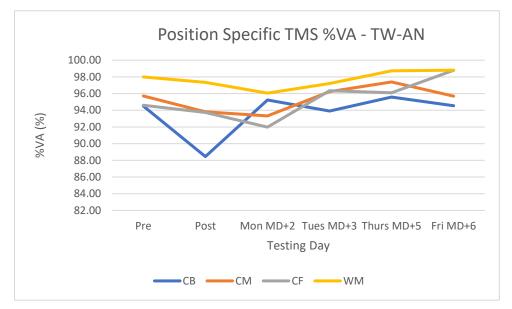


Figure 17 Mean percentage (%) for the Position Specific TMS %VA TW-AN.

## 4.3 Peripheral Stimulation

Peripheral Stimulation (PS) was completed on each of the testing dates to measure the contribution of the central nervous system to any force production reductions. Both the deficit and the percentage of voluntary activation were analysed to allow comparison between both central and peripheral nervous systems.

## 4.3.1 PS Deficit

A Shapiro-Wilk test of normality was conducted prior to the statistical analysis (p = <0.001). Whilst this shows the data was not normally distributed, a mixed betweenwithin subjects' analysis of variance was used to analyse the data, as Kozak and Piepho (2018) argue that this is still an effective tool with the sample size. The mean deficit between MVC and PS-induced contraction increased from  $13.84 \pm 15.86$ N to  $15.88 \pm 13.33$ N in TW-A from pre-match to post-match which was not statistically significant (p = 0.759).

The mean deficit between MVC and PS-induced contraction decreased from 24.04  $\pm$  25.74N to 21.09  $\pm$  14.38N in TW-AN from pre-match to post-match which was not statistically significant (p = 0.755).

Mean PS deficit increased from post-match (15.88  $\pm$  13.33N) to MD+2 in TW-A (17.46  $\pm$  23.20N). The mean PS deficit of TW-A dropped to below baseline deficit scores on MD+3 and remained there until the end of the week throughout the week.

Mean PS deficit increased from  $21.09 \pm 14.38N$  post-match to  $30.12 \pm 23.62N$  on MD+2 in TW-AN. The mean PS deficit of TW -AN dropped to below baseline deficit scores on MD+3 and remained there until the end 0f the week throughout the week.

A mixed between-within subjects' analysis of variance was conducted to assess the impact of two different interventions (TW-A and TW-AN) on participants' deficits from evoked force during PS to voluntary maximal voluntary quadriceps contraction across 6 different testing dates throughout the week (Pre-match, Post-match, Monday – MD+2, Tuesday – MD+3, Thursday – MD+5, Friday – MD+6). There was no significant interaction between testing day and training week, Wilks' Lambda = 0.638, F (5, 14) = 1.592, p = 0.226, partial eta squared = 0.362. There was a significant effect for testing day, Wilks' Lambda = 0.473, F (5, 14) = 3.115, p = 0.043, partial eta squared = 0.527, suggesting a substantial change in deficit dependent on the day of testing throughout the protocol, however post-Hoc Bonferroni tests did not find any significant difference between days. Paired samples t-tests were used to identify where these differences were, with only the MD+2 to MD+6 comparison finding a significant difference (p =

0.036), suggesting the greatest peripheral fatigue on MD+2, with the least on MD+6 (Figure 18). Data can be seen in Appendix P (4).

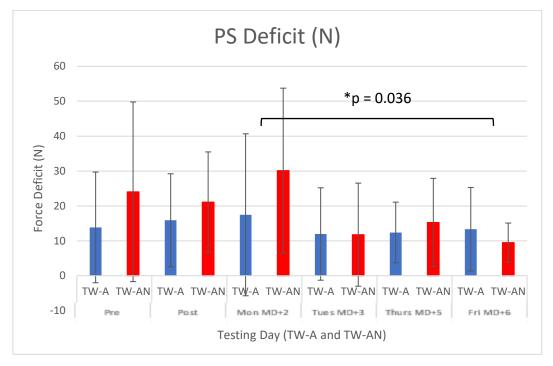


Figure 18 Mean PS Deficit (N) for both Training Weeks. MD+2 to MD+6 \*p = .036

The between subjects' effect was not significant, F (1, 18) = 1.192, p = 0.289, partial eta squared = 0.062, suggesting no difference in the effectiveness of the two training weeks. Paired samples t-tests were used to more greatly compare the PS deficit on each testing day. As seen in Table 7, there were no significant differences found between the two weeks for the PS deficit.

	Aerobic Training			Anaerob	ic Trainin	g Co	Comparison		
	Week			W	/eek	TW-	A v TW-AN		
Testing Date	n	М	SD	п	М	SD	Р		
Pre-Match	10	13.84	15.86	10	24.04	25.74	0.333		
Post-Match	10	15.88	13.33	10	21.09	14.38	0.385		
Monday (MD+2)	10	17.46	23.20	10	30.12	23.62	0.286		
Tuesday (MD+3)	10	11.99	13.24	10	11.76	14.77	0.975		
Thursday (MD+5)	10	12.39	8.70	10	15.26	12.64	0.433		
Friday (MD+6)	10	13.32	11.97	10	9.49	5.61	0.409		

Table 7 Comparison of PS deficit over the two training weeks.

As seen in Figure 19, mean PS deficit fluctuated greatly for all positions in TW-A. There was a significant difference between CF and WM on post-match testing, p = 0.038 and MD+2, p = 0.050. There was also a significant difference between CM and CF on post-match testing, p = 0.048 There were no other differences between positions.

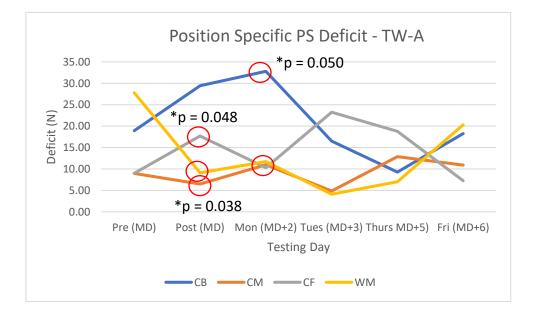


Figure 19 Mean force (N) for the Position Specific PS deficit TW-A. Post-match (CF-WM \*p = 0.038, CM-CF \*p = 0.048), MD+2 (CF-WM \*p=0.050).

As seen in Figure 20, mean PS deficit fluctuated greatly for all positions in TW-AN. There was a significant difference on pre-match testing, between CB and CM, p = 0.027 and between CM and CF, p = 0.025. There were also significant differences between CF and WM on MD+2, p = 0.019 and between CM and WM on MD+3, p = 0.024. There were no other differences between positions

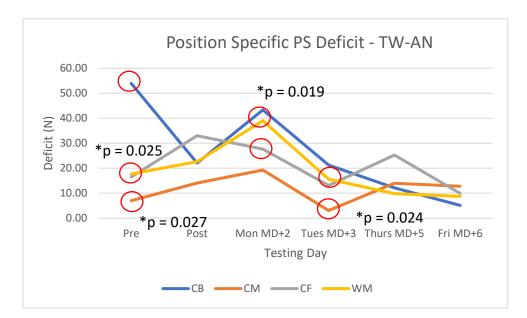


Figure 20 Mean force (N) for the Position Specific PS deficit TW-AN. Pre-match (CB-CM \*p = 0.027, CM-CF \*p = 0.025), MD+2 (CF-WM \*p=0.019), MD+3 (CM-WM \*p = 0.024).

## 4.3.2 PS Percentage of Voluntary Activation

A Shapiro-Wilk test of normality was conducted prior to the statistical analysis (p = <0.001). Whilst this shows the data was not normally distributed, a mixed betweenwithin subjects' analysis of variance was used to analyse the data, as Kozak and Piepho (2018) argue that this is still an effective tool with the sample size.

The percentage of voluntary activation is a calculation of the percentage of the stimulated contraction that was produced voluntarily i.e. if the voluntary contraction was 90N and the stimulated contraction via TMS was 100N, the percentage of voluntary activation would be 90%. The use of this measurement removes the

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comparison of raw data (the force produced (N), and shows the percentage of muscle activation an individual could produce, ignoring whether this was at a high or low force. The mean %VA during PS-induced contraction decreased from 97.52  $\pm$  2.96% to 96.63  $\pm$  3.33% in TW-A, however this was not statistically significant (p = 0.534).

The mean %VA during PS-induced contraction increased from  $94.84 \pm 6.56\%$  to  $95.59 \pm 3.32\%$  in TW-AN from pre-match to post-match, however this was not statistically significant (p = 0.755).

Mean PS %VA remained reduced on MD+2 in TW-A (96.68  $\pm$  4.52%), suggesting a level of peripheral fatigue 48 hours post-match. The mean PS %VA of TW-A rose above baseline %VA scores on MD+3 and remained there until the end of the week.

Mean PS %VA remained reduced on MD+2 in TW-AN (95.15  $\pm$  4.15%), suggesting a level of peripheral fatigue 48 hours post-match. The mean PS %VA of TW-AN rose above baseline %VA scores on MD+3 and remained there until the end of the week.

A mixed between-within subjects' analysis of variance was conducted to assess the impact of two different interventions (TW-A and TW-AN) on participants' percentage of voluntary activation (%) during PS to voluntary maximal voluntary quadriceps contraction across 6 different testing dates throughout the week (Pre-match, Postmatch, Monday – MD+2, Tuesday – MD+3, Thursday – MD+5, Friday – MD+6). There was no significant interaction between testing day and training week, Wilks' Lambda = 0.648, F (5, 14) = 1.520, p = 0.246, partial eta squared = 0.352. There was a significant effect for testing day, Wilks' Lambda = 0.450, F (5, 14) = 3.428, p = 0.031, partial eta squared = 0.550, suggesting a substantial change in percentage voluntary activation dependent on the day of testing throughout the protocol, however post-Hoc

Bonferroni tests did not find any significant difference between days. Paired samples t-tests were used to identify where these differences were, with significant differences found from Post-Match to MD+3 (p = 0.038), Post-match to MD+5 (P = 0.046), Post-Match to MD+6 (p = 0.014) and MD+2 to MD+6 (p = 0.037) (Figure 21). Data can be seen in Appendix P (5).

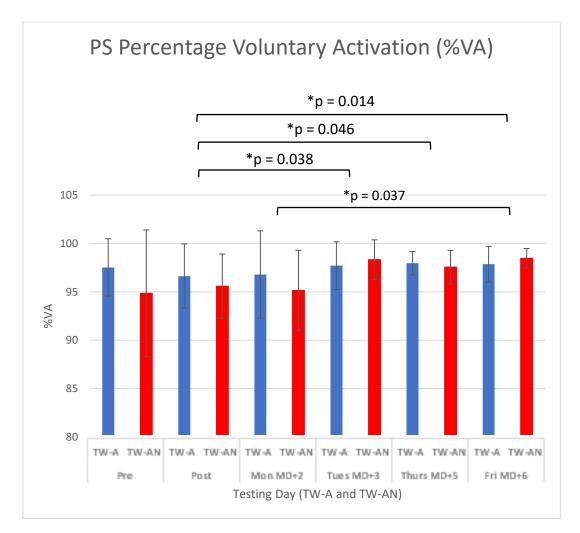


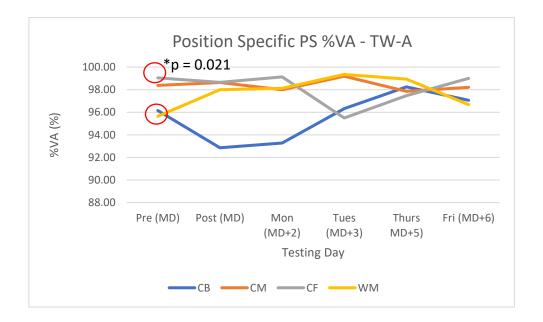
Figure 21 Mean PS % Voluntary Activation (%) for both Training Weeks. Post-match to MD+3 \*p = 0.038, Post-match to MD+5 \*p = 0.046, Post-match to MD+6 \*p = 0.014, MD+2 to MD+6 \*p = 0.037

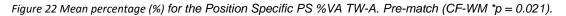
The between subjects' effect was not significant, F (1, 18) = 0.779, p = 0.389, partial eta squared = 0.041, suggesting no difference in the effectiveness of the two training weeks. Paired samples t-tests were used to more greatly compare the PS %VA on each testing day. As seen in Table 8, there were no significant differences found between the two weeks for the PS %VA.

	Aerobic Training			Anaerob	oic Training		Comparison		
		Week		W	/eek		TW-A v TW-AN		
Testing Date	n	М	SD	п	М	SD	Р		
Pre-Match	10	97.52	2.96	10	94.84	6.56	0.286		
Post-Match	10	96.63	3.33	10	95.59	3.32	0.465		
Monday (MD+2)	10	96.68	4.52	10	95.15	4.15	0.427		
Tuesday (MD+3)	10	97.71	2.47	10	98.33	2.05	0.489		
Thursday (MD+5)	10	97.97	1.21	10	97.56	1.73	0.483		
Friday (MD+6)	10	97.86	1.84	10	98.47	1.01	0.398		

Table 8 Comparison of PS %VA over the two training weeks.

As seen in Figure 22, mean PS %VA fluctuated greatly for all positions in TW-A. There was a significant difference between CF and WM on pre-match testing, p = 0.021. There were no other differences between positions.





As seen in Figure 23, mean PS %VA fluctuated greatly for all positions in TW-AN, however there were no significant differences between positions.

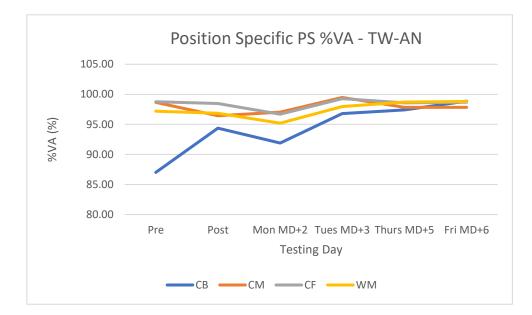


Figure 23 Mean percentage (%) for the Position Specific PS %VA TW-AN.

#### 4.4 Comparison of Central and Peripheral Fatigue

Paired samples t-tests were used to compare the means of the TMS and PS deficits and %VA for all testing dates. It can be seen that there were significant differences in the levels of central and peripheral fatigue post-match for TW-A (Deficit post-match – p = 0.010; %VA post-match – p = 0.008) and on MD+3 of both weeks (Deficit MD+3 TW-A – p = 0.046; Deficit MD+3 TW-AN – p = 0.049; %VA MD+3 TW-A – p = 0.049; %VA MD+3 TW-AN – p = 0.045). This suggests that there were significantly greater levels of central fatigue than peripheral fatigue, both post-match and 72 hours postmatch for both training weeks. There is also a significant difference on the MD+5 of TW-AN (p = 0.038). However, the values for TMS deficit are higher than those for PS deficit, and the values for TMS %VA are always lower than those for PS %VA (excluding pre-match in TW-AN). This could show a chronic elevation in central fatigue throughout the week, which can be increased through physical activity. The data can be seen in Table 9.

Table 95 Comparison of TMS and PS Deficit and %VA over the two training weeks. This table includes the significant	
differences between the TMS and PS for each metric.	

ic Tra		al Pe	eriphe	eral Stim	Sig.		
Ν							
Stimulation							
Ν	М	SD	Ν	М	SD		
10	25.84	20.13	10	13.84	15.86	0.225	
10	38.46	19.86	10	15.88	13.33	0.010*	
10	25.57	20.32	10	17.46	23.20	0.475	
10	35.92	32.31	10	11.99	13.24	0.046*	
10	26.08	26.69	10	12.39	8.70	0.123	
10	24.15	16.90	10	13.32	11.97	0.097	
10	25.02	13.85	10	24.04	25.74	0.905	
10	38.64	21.00	10	21.09	14.38	0.063	
10	37.55	25.96	10	30.12	23.62	0.514	
10	25.61	14.23	10	11.76	14.77	0.049*	
10	22.27	13.13	10	15.26	12.64	0.038*	
10	18.57	21.00	10	9.49	5.61	0.200	
10	95.09	3.87	10	97.52	2.96	0.200	
10	92.44	4.30	10	96.63	3.33	0.008*	
10	95.70	2.87	10	96.68	4.52	0.561	
10	93.43	6.18	10	97.71	2.47	0.049*	
10	95.94	3.73	10	97.97	1.21	0.098	
10	96.10	2.50	10	97.86	1.84	0.070	
10	95.48	2.63	10	94.84	6.56	0.722	
10	92.57	4.15	10	95.59	3.32	0.098	
	N St N 10 10 10 10 10 10 10 10 10 10 10 10 10	Magnetic         Stimulation         N       M         10       25.84         10       38.46         10       25.57         10       35.92         10       26.08         10       24.15         10       25.02         10       25.61         10       25.61         10       25.61         10       25.02         10       25.61         10       25.61         10       25.02         10       95.09         10       95.09         10       95.09         10       95.70         10       95.70         10       95.70         10       95.70         10       95.70         10       95.43         10       95.43	HagneticSUUILATIONNMSD1025.8420.131038.4619.861025.5720.321025.6132.311026.0826.691024.1516.901025.0213.851038.6421.001037.5525.961025.6114.231025.6114.231025.6113.131025.6113.131095.093.871095.702.871095.744.301095.943.731095.482.63	Nagnetic           N         SD         N           N         SD         N           10         25.84         20.13         10           10         38.46         19.86         10           10         25.57         20.32         10           10         25.57         20.32         10           10         25.57         20.32         10           10         25.61         32.31         10           10         26.08         26.69         10           10         25.02         13.85         10           10         25.02         13.85         10           10         37.55         25.96         10           10         37.55         25.96         10           10         25.61         14.23         10           10         25.61         14.23         10           10         25.61         3.87         10           10         95.09         3.87         10           10         95.09         3.87         10           10         95.70         2.87         10           10         95.94	NagneticSUBURIATIONNSDNM1025.8420.131013.841038.4619.861015.881025.5720.321017.461035.9232.311011.991026.0826.691012.391026.0213.851024.041025.0213.851024.041038.6421.001021.091037.5525.961030.121025.6114.231011.761025.6114.231015.261025.7021.00109.491095.093.87109.491095.702.871096.631095.702.871097.711095.943.731097.861095.482.631097.861095.482.631097.86	NagneticSimulationNMSDNMSD1025.8420.131013.8415.861038.4619.861015.8813.331025.5720.321017.4623.201035.9232.311011.9913.241026.0826.691012.398.701024.1516.901013.3211.971025.0213.851024.0425.741038.6421.001021.0914.381037.5525.961030.1223.621037.5525.961030.1223.621022.2713.131015.2612.641085.7021.00109.495.611095.093.871094.945.611095.702.871096.633.331095.702.871096.634.521095.943.731097.971.211095.943.731097.971.241095.482.631097.861.841095.482.631097.861.84	

%VA Monday (MD+2) TW-AN	10	94.35	3.25	10	95.15	4.15	0.625
%VA Tuesday (MD+3) TW-AN	10	95.85	2.66	10	98.33	2.05	0.045*
%VA Thursday (MD+5) TW-AN	10	96.66	2.19	10	97.56	1.73	0.121
%VA Friday (MD+6) TW-AN	10	96.26	5.30	10	98.47	1.01	0.203

## 4.5 Global Positioning System Metrics

The Global Positioning Software (GPS) units (Catapult Minimax S4) were positioned between the shoulder blades of the individuals using a vest which was the appropriate size to prevent excess movement during activity. These units were worn for the duration of all training sessions and matches during the testing protocol. The metrics recorded were: Total Distance (TD, m), High Speed Running Distance (distance of speeds >17km/h) (HSR, m), Player Load (PL). These metrics will be explained in the following sections.

#### 4.5.1 Total Distance

The total distances of the training sessions and matches were recorded through the GPS units. Participants covered an average of  $9710.00 \pm 2158.64$ m in the match in TW-A. The total distances remained consistent between MD+2 and MD+5 before dropping on MD+6, which is the day before the next match.

Participants covered an average of 8696.30  $\pm$  3136.25m in the match in TW-AN. The mean total distances dropped to 4420.40  $\pm$  456.82m on MD+2 and rose to 5431.80  $\pm$  462.38m on MD+3 and 5376.90  $\pm$  588.19m on MD+5 before dropping on MD+6, which is the day before the next match.

Independent samples T-Tests were completed in SPSS to determine differences in Total Distance (m) between TW-A and TW-AN. As seen in in Figure 24, significant differences in TD between both training weeks were seen on both MD+2 (p = <0.001) and MD+6 (p = 0.001). Data can be seen in Appendix P (6).

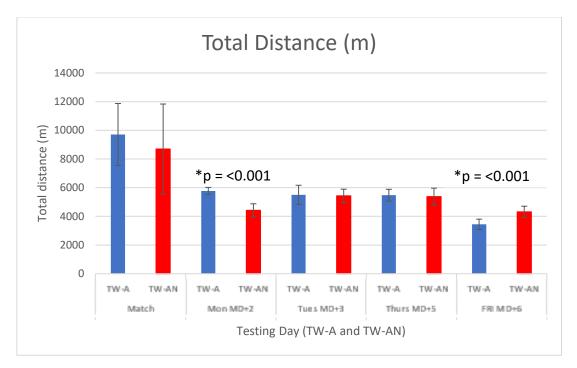


Figure 24 Mean Total Distance (m) for both Training Weeks. MD+2 \*p = <0.001, MD+6 \*p = <0.001

As seen in Figure 25, mean TD remained consistent through the training week for all positions in TW-A and there were no significant differences between positions.

Results

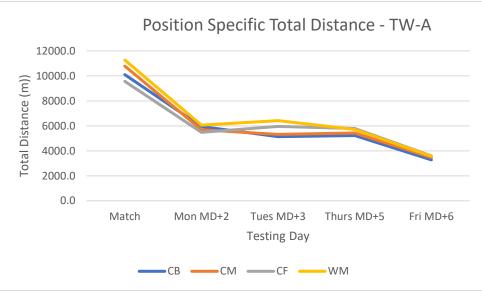


Figure 25 Mean Position Specific TD for TW-A.

As seen in Figure 26, mean TD remained consistent through the training week for all positions in TW-AN, however there was a significant difference between CM and CB, p = 0.025 and between CM and CF, p = 0.009 on MD+2. There were no other differences between positions.

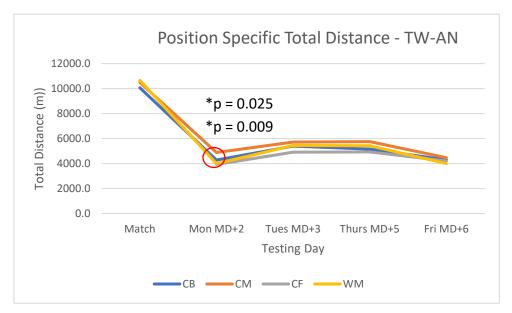


Figure 26 Mean Position Specific TD for TW-AN. MD+2 (CM-CB \*p = 0.025, CM-CF \*p = 0.009).

## 4.5.2 High Speed Running Distance

The High-Speed Running distances of the training sessions and matches were recorded through the GPS units. Participants covered an average of 1915.40  $\pm$  766.07m of High-Speed Running in the match in TW-A. The total distances were very low on the MD+2 and MD+3 but peaked at 1040.20  $\pm$  275.93m on MD+4 before dropping on MD+6, which is the day before the next match.

Participants covered an average of 19985.40  $\pm$  874.02m of High-Speed Running in the match in TW-AN. The mean High-Speed Running distances dropped to 536.40  $\pm$  252.56m on MD+2 and rose to 713.50m on MD+3 and 719.20m on MD+5 before dropping on MD+6, which is the day before the next match.

Independent samples T-Tests were completed in SPSS to determine differences in High Speed Running Distance (m) between TW-A and TW-AN. As seen in in Figure 27, significant differences in HSR between both training weeks were seen on Monday (p = 0.004), Thursday (p = 0.039) and Friday (p = 0.031). Data can be seen in Appendix P (7).

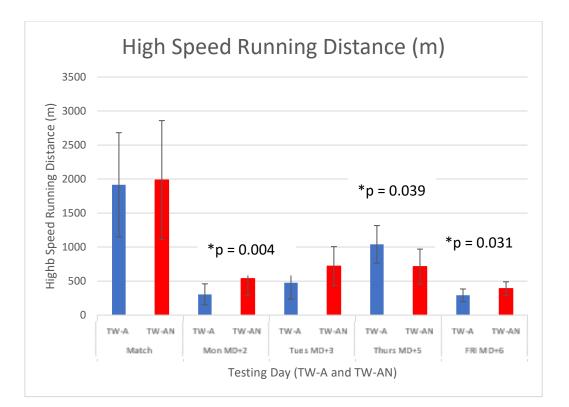


Figure 27 Mean High Speed Running Distance (m) for both Training Weeks. MD+2 - \*p = 0.004, MD+5 - \*p = 0.039, MD+6 - \*p = 0.031

As seen in Figure 28, mean HSR remained consistent through the training week for all positions in TW-A, however there were significant differences between WM and CB, p = 0.011 and between WM and CM, p = 0.008 on MD+3. There were no other differences between positions.

Results

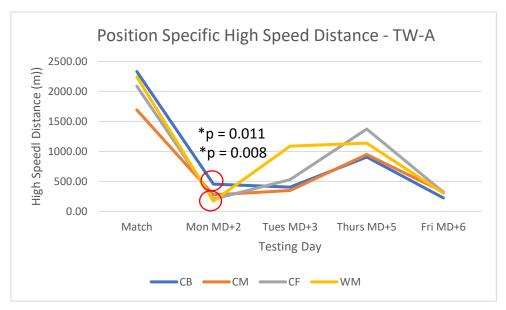


Figure 28 Mean Position Specific HSR for TW-A. MD+3 (WM-CB \*p = 0.011, WM-CM \*p = 0.008).

As seen in Figure 29, mean HSR remained consistent through the training week for all positions in TW-AN, however there was a significant difference between CM and CB, p = 0.034 on MD+3. There were no other differences between positions.

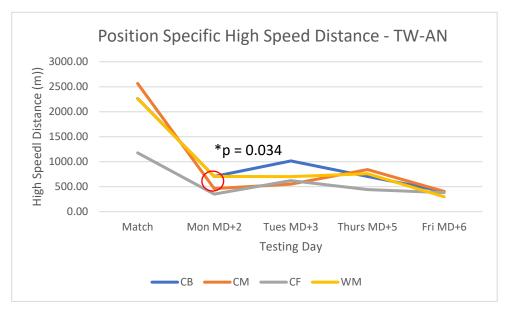


Figure 29 Mean Position Specific HSR for TW-AN. MD+3 (CM-CB \*p = 0.034).

#### 4.5.3 Player Load

Player load is a metric calculated through an algorithm through the GPS units. The mean match player load was  $952.65 \pm 227.91$  in TW-A. The player load reduced dramatically from the game to the training days on MD+2 ( $651.62 \pm 81.20$ ), MD+3 ( $650.91 \pm 111.67$ ) and MD+5 ( $606.71 \pm 606.71$ ) where it remained consistent before dropping on MD+6, which is the day before the next match.

Average match player load was 886.97  $\pm$  316.70 in TW-AN. The mean player load dropped to 503.73  $\pm$  59.31 on MD+2 and rose to 586.67  $\pm$  41.67 on MD+3 and 593.09  $\pm$  593.09 on MD+5 before dropping on MD+6, which is the day before the next match. Independent samples T-Tests were completed in SPSS to determine differences in Player Load between TW-A and TW-AN. As seen in in Figure 30, significant differences in HSR between both training weeks were seen on both MD+2 (p = 0.001) and MD+6 (p = 0.015). Data can be seen in Appendix P (8).

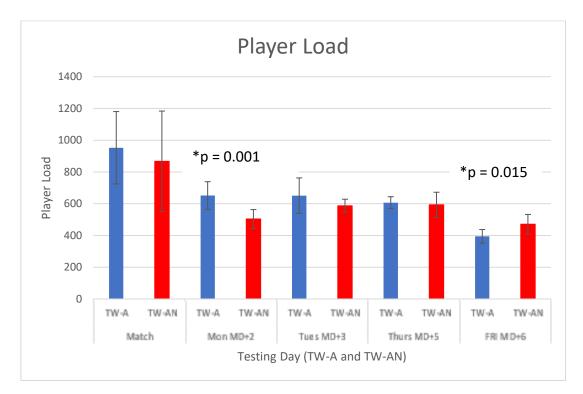


Figure 30 Mean Player Load for both Training Weeks. MD+2 - \*p = <0.001, MD+6 - \*p = 0.015

As seen in Figure 31, mean PL remained consistent through the training week for all positions in TW-A and there were no significant differences between positions.

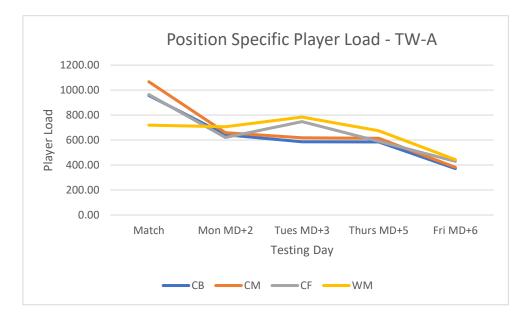


Figure 31 Mean Position Specific PL for TW-A.

As seen in Figure 32, mean PL remained consistent through the training week for all positions in TW-AN, however there was a significant difference between CM and CF in the match, p = 0.005. There were no other differences between positions.

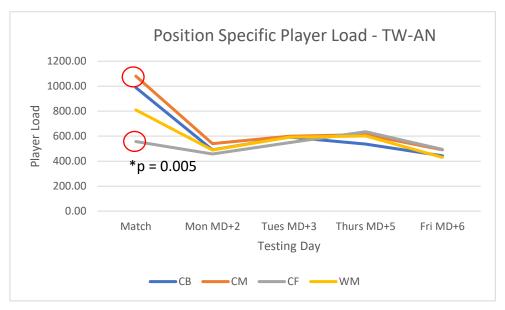


Figure 32 Mean Position Specific PL for TW-AN. Match (CM-CF \*p = 0.005).

# 4.5.4 Comparison of Training Weeks

Whilst the physical outcomes of sessions on each week varied in total distance, high speed running and player load depending on the day of each week, the total mean of each of these metrics for each training week was very similar (Table 10).

Paired samples t-tests t-tests were used to compare the collective training data for all sessions each week. There were no significant differences between the two weeks for TD (p = 0.344), HSR (p = 0.367) or PL (p = 0.095).

							Sig.	
GPS Metric	Ae	robic Trainir	ng Week	Ana	Anaerobic Training Week			
	Ν	Μ	SD	Ν	М	SD		
Total Distance (m)	10	20183.00	1238.42	10	19543.10	1456.01	0.344	
HSR (m)	10	2112.40	390.95	10	2358.70	621.83	0.367	
Player Load	10	2303.88	238.66	10	2154.31	156.65	0.095	
-								

Table 10 Mean total training GPS metrics for TW-A and TW-AN

# 4.6 Rating of Perceived Exertion

The RPE scale of 1-10 was used to determine perceived intensity of the training session. The average RPE for each match and session through the two-week testing protocol can be seen in Figure 33.

It can be seen that the peak RPE for the Aerobic training week was on MD+5, but was on MD+3 for the Anaerobic training week. Paired samples T-Tests were used to identify difference between the two weeks, with significant differences between the two training weeks on both MD+3 (p = 0.040) and MD+5 (p = 0.001). Data can be seen in Appendix P (9).

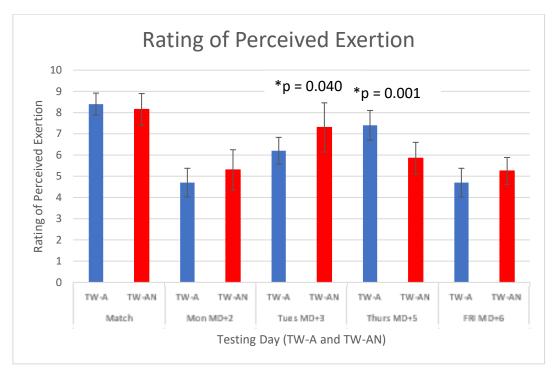


Figure 33 Mean Rating of Perceived Exertion for both Training Weeks. MD+3 - \*p = 0.040, MD+5 - \*p = 0.001

As seen in Figure 34, mean RPE remained consistent through the training week for all positions in TW-A and there were no significant differences between positions.

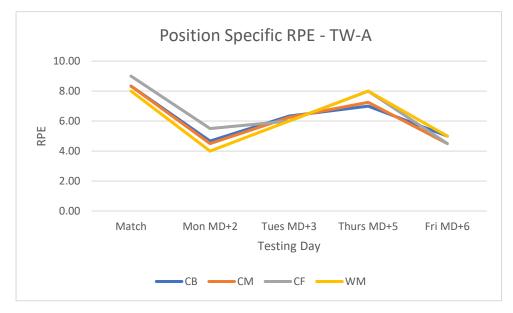


Figure 34 Mean Position Specific Rating of Perceived Exertion for TW-A.

As seen in Figure 35, mean RPE remained consistent through the training week for all positions in TW-A and there were no significant differences between positions.

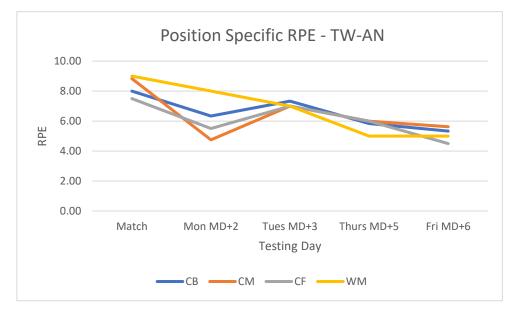


Figure 35 Mean Position Specific Rating of Perceived Exertion for TW-AN.

## 4.6.1 RPE Trimp

The researcher timed the duration of the session (minutes) from the beginning of the warm up until the end of the final drill, including rest breaks and transition between drills and this was multiplied by the given RPE to give and RPE Trimp as a measure of the total training load. This was utilised as a perceived total load. The weekly RPE Trimps can be seen in Figure 36.

It can be seen that the highest RPE Trimp was on MD+5 in the Aerobic training week and MD+3 in the Anaerobic training week, with both the lowest RPE Trimps on the MD+6.

Paired samples T-Tests were used to identify difference between the two weeks, with significant differences between the two training weeks on MD+3 (p = 0.043) and MD+5 (p = <0.001) (Figure 18). Data can be seen in Appendix P (10).

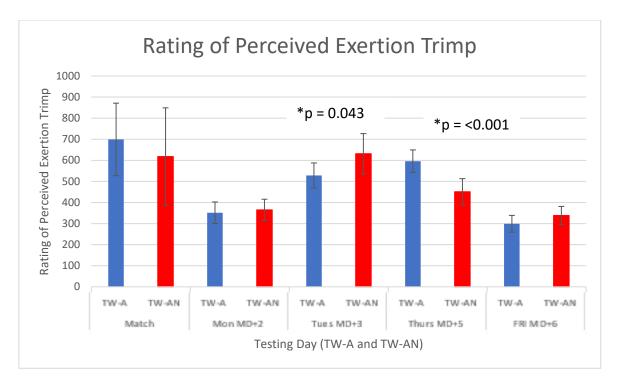


Figure 36 Mean Rating of Perceived Exertion Trimp for both Training Weeks. MD+3 - \*p = 0.043, MD+5 - \*p = <0.001As seen in Figure 37, mean RPE Trimp remained consistent through the training week between positions in TW-A, however there were significant difference on MD+5 between CB and CF, p = 0.002 and between CB and WM, p = 0.017. There was also a significant difference for the match between CM and CF, p = 0.002 There were no other differences between positions.

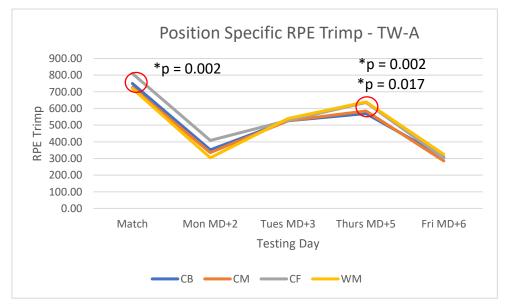


Figure 37 Mean Position Specific Rating of Perceived Exertion Trimp for TW-A. Match (CM-CF \*p = 0.002), MD+5 (CB-CF \*p = 0.002, CM-WM \*p = 0.017.

As seen in Figure 38, mean RPE remained consistent through the training week for all positions in TW-AN and there were no significant differences between positions.

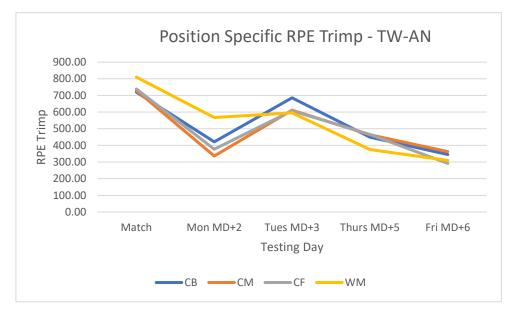


Figure 38 Mean Position Specific Rating of Perceived Exertion Trimp for TW-AN.

#### 4.7 Blood Lactate

Blood lactate results were only recorded pre-match and post-match as a measure of the intensity of the match. The mean blood lactate concentrations (mmol/l) for TW-A can be seen in Figure 39. A paired samples T-Test was used to compare the mean blood lactate concentrations from pre-match to post-match, showing a significant difference (p =.<0.001) for TW-A and for TW-AN (p =.<0.001).

Paired samples T-Tests were used to compare the mean blood lactate concentrations (mmol/l) between the two weeks with no significant differences in the pre-match testing (p = 0.994) or post-match (p = 0.308).

Blood lactate increased significantly from pre-match to post-match, showing completion of the match elicits a build-up of lactic acid, however there was no

significant difference between the two weeks, showing each match results in the same levels of anaerobic by-products. Data can be seen in Appendix P (11).

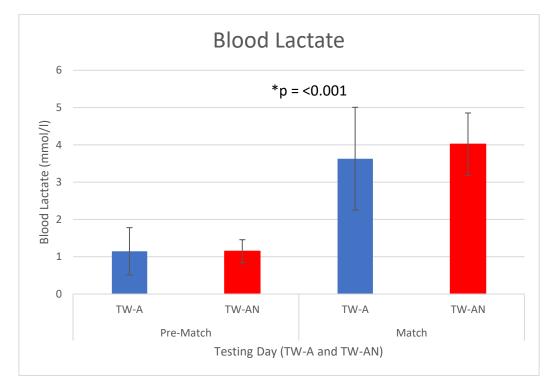


Figure 39 Mean Blood Lactate concentration - Aerobic and Anaerobic Training Weeks (Pre and Post-Match) mmol/l

As seen in Figure 40, mean blood lactate increased more for the two attacking positions (CF and WM) than for the defensive positions from pre-match to post-match. There were significant differences on post-match testing between CB and CF, p = 0.033, CM and WM, p = 0.050 and CM and CF, p = 0.013. There were no other differences between positions.

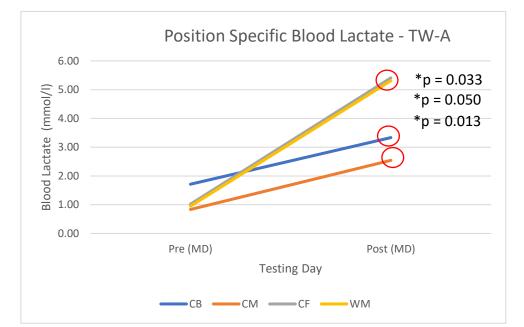


Figure 40 Mean Position Specific blood lactate for TW-A. Post-Match (CB-CF \*p = 0.033, CM-WM \*p = 0.050, CM-CF \*p = 0.013).

As seen in Figure 41, mean RPE remained consistent through the training week for all

positions in TW-AN and there were no significant differences between positions.

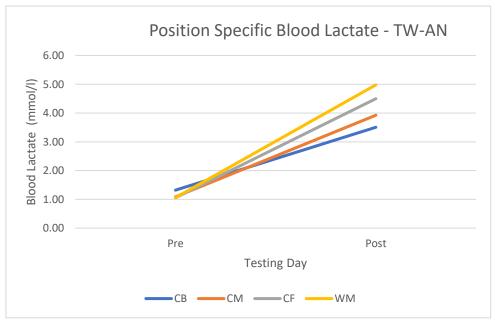


Figure 41 Mean Position Specific blood lactate for TW-AN.

Discussion

### 5.0 Discussion

This study aimed to discover the effect that fatigue plays in force production and decipher the role that the both central and peripheral mechanisms play in this. Through analysing the extent to which the deficit between voluntary, isometric quadricep contractions and involuntary, Transcranial Magnetic Stimulation-Induced and Peripheral Stimulation-induced isometric quadricep contractions differ in a fatigued state in elite youth football players, it was possible to investigate the speed at which different training methods facilitate recovery.

A number of significant findings were identified through this study. Firstly, on all but one of the testing days, both the deficit and %VA showed greater fatigue levels through Transcranial Magnetic Stimulation (TMS) than through Peripheral Stimulation (PS), suggesting individuals were displaying greater central fatigue than peripheral fatigue. However, both Brownstein et al. (2017) and Rampinini et al. (2011) found that TMSinducted involuntary contractions showed that participants had recovered from a drop in CNS voluntary activation 48 hours post-match. Thomas et al. (2017), Brownstein et al. (2017) and Rampinini et al. (2011) all found that PS-induced involuntary contractions took up to 72 hours to return to baseline measurements, suggesting that those processes distal to the neuromuscular junction took longer to recover from than those that were proximal.

The second significant finding from the study was that the MVC, deficits and %VA all vary dependent on the day of testing. Whilst the two training weeks did not elicit significant differences in each variable, the day in the week in which MVC, TMS %VA, PS deficit and PS %VA were tested produced a statistically significant difference. The greatest levels of peripheral and central fatigue were seen post-match and increased

Discussion

throughout the weeks for most metrics, in either a linear or non-linear fashion. This compares to those studies by Rampinini et al. (2011), Brownstein et al. (2017) and Thomas et al. (2017), who all found that central and peripheral fatigue levels will vary dependent on the distance in time from the previous match.

#### 5.1 Maximal Voluntary Contraction

The mean MVC produced by participants varied dependent on the day of testing. Whilst the two training weeks did not elicit a significant difference (p = 0.625), the day in the week in which MVC was tested produced a statistically significant difference (p = 0.000). Through post-Hoc Bonferroni tests, it was found that there were significant differences on between the pre-match testing and MD+3 (p= 0.006), MD+5 (p= 0.010) and MD+6 (p = 0.000). There were also significant differences between the post-match testing and testing on MD+3 (p= 0.000), MD+5 (p= 0.000) and MD+6 (p = 0.000). The partial eta squared was 0.821, suggesting there were substantial differences dependent on the day of testing. With MVCs being significantly higher towards the end of the week than both pre-match and post-match values, it can be argued that both training protocols provided the platform for effective recovery from the match.

This reflects findings from previous studies, in which MVC was recovered to pre-match values within 72 hours after a competitive match (Brownstein et al. 2017) and after a simulated match (Thomas et al. 2017). The findings in this study also show that by the MD+5 and MD+6, MVC values eclipse those from prior to the match. Thomas et al. (2017) found that MVC returned to baseline values when participants remained inactive in the days following the match, whilst the participants in this study returned

to baseline values quicker and then eclipsed those values whilst training. These findings will be explained in the following sections.

#### 5.1.1 Pre-Match to Post-Match

It can be seen that Vastus Lateralis MVC, which is globally utilised as a measure of central and peripheral fatigue (Goodall et al. 2017) was decreased from pre-match to post-match in both TW-A and TW-AN. This reduction in MVC of  $7.96 \pm 13.44$  % in TW-A and 10.04  $\pm$  14.55% in TW-AN is much lower than the decrease from the simulated soccer match by Goodall et al. (2017) of 20% and Clarke et al. (2015) of 15%. It is also much lower than the study by Brownstein et al. (2017) which found a drop of 14  $\pm$  9% from pre-match to post-match. The decrease pre to post-exercise can be considered moderate when compared with those (20%–35%) observed after prolonged running, cycling, or skiing exercises (Rampinini et al. 2011).

Due to the intermittent nature of football, with portions of the game being at walking pace, it would be expected that the decrease in MVC would be less significant than endurance events such as cycling. This may be because the muscle strain is more localised to the smaller number of muscle fibres utilised at a constant high intensity (Achten et al. 2003). However, the smaller percentage reduction of MVC in comparison to previous studies in football may be attributed to a number of factors. Firstly, the participants in this study were of an elite level. Therefore, it would be expected that through full time training with high-level teammates and individualised training programmes, that the individuals would be better conditioned to withstand the demands of competition and would therefore recover guicker.

Discussion

Secondly the total distance covered in the matches in TW-A (9710  $\pm$  2158.64m) and TW-AN (8696  $\pm$  3136.25m) is lower than that in studies by Brownstein et al (2017) (10041  $\pm$  626m) and Goodall et al. (2017) (14,400m). The study by Goodall et al. (2017) included a 30-minute period of extra time, which would explain the increase in fatigue levels.

The post-match values of 528  $\pm$  123.41N (TW-A) and 552.2  $\pm$  150.52N (TW-AN) compare to those of 527  $\pm$  54N found by Thomas et al. (2017) in their simulated protocol study. Therefore, whilst the mean pre-match MVCs are lower than previous studies (TW-A - 574.4  $\pm$  109.72N; TW-AN - 615.1  $\pm$  133.94N; Goodall et al. (2017) – 682.92N; Brownstein et al. (2017) – 726N) the reduction post-match is much less. This may be attributed to physical maturation, meaning that as the participants in this study are younger (age: 17.5  $\pm$  0.5 years) and have not reached their peak physical strength, they are unable to produce a high force in their non-fatigued state. However, as Milioni et al. (2017) state, due to their lower anaerobic capacity, children are less affected by metabolites such as hydrogen ions, meaning that metabolic (peripheral) fatigue is lower than in adults.

### 5.1.2 Aerobic Training Week

During TW-A, the MVC increases to greater than baseline values 48 hours post-match and increases through the week. This is 24 hours sooner than studies by Brownstein et al. (2017) and Thomas et al. (2017) who found that MVC was still slightly less, yet returned to baseline 72 hours post-match. As previously discussed, the elite level and age of the athletes may have had an effect on the recovery time. Nédélec et al. (2012) suggest that training status, age, gender and muscle fibre typology can all have an effect on the speed at which athletes recover.

The effect of active recovery cannot be discussed prior to MD+2, as there were no training sessions until after testing on MD+2 morning. Therefore, whilst the increase in MVC to greater than pre-match MVC may be due to active recovery, the values at 48 hours post-match must be attributed to other factors.

The greatest differences were seen between the centre forwards (CF) and the centre backs (CB), with significant findings on MD+2 (p = 0.011) and MD+3 (p = 0.043). This suggests that the CF were stronger than the CB. A study by Sporis et al. (2009), found that CF produced the greatest scores for power-based testing such as 10m sprint, 20m sprint and countermovement jump (CMJ), which replicates the MVC scores in this study.

#### 5.1.3 Anaerobic Training Week

TW-AN MVC is also above the pre-match value 48 hours post-match. During TW-AN, the MVC peaks on MD+3, possibly due to the reduction in total distance (TW-A – 9710.00  $\pm$  2158.64m; TW-AN – 8696.30  $\pm$  3136.25m) and player load (TW-A – 952.65  $\pm$  227.91; TW-AN – 886.97  $\pm$  316.70) in the match compared to TW-A. This reduction in total load in the match, combined with a less physically-demanding session on MD+2 of TW-AN compared to TW-A may have facilitated greater physical recovery by Tuesday. The GPS metrics will be discussed in following sections. The standard deviations (SD) for each testing date over each week are large, which will account for difference in physical strength between individuals, suggesting that the sample size may have been too small.

As with TW-A, there were only significant differences between the CF and CB which were on pre-match testing (p = 0.008) and MD+6 (p = 0.029).

Throughout both TW-A and TW-AN, the MVC for centre forwards was the highest on all testing dates. However, whilst this is an interesting finding, the small number of participants (n = 2) means that future study would be required with a large sample size to validate this.

#### 5.2 Transcranial Magnetic Stimulation / Central Fatigue

As previously mentioned, the TMS %VA (p = 0.020) produced a statistically significant difference dependent on the day of testing. Post-hoc Bonferroni tests identified these differences as pre-match to post-match (p = 0.012) and post-match to MD+5 (p = 0.004) There was also a significant difference from pre-match to post-match for TMS deficit (p = 0.039). There was no difference between training weeks for deficit or %VA. In both the TMS deficit and the TMS %VA, values returned to pre-match baseline measures after 48 hours in TW-A, whilst it took 72 hours in TW-AN. However, these differences were not statistically significant.

#### 5.2.1 Pre-Match to Post-Match

In both TW-A and TW-AN, the mean %VA dropped over 2% from pre-match to post match (2.48% and 2.75% respectively), showing a significant difference between the two training dates. This drop is relatively insignificant when comparing to previous studies, where CNS %VA drops by 15% (Goodall et al. 2017), 8.9% (Rampinini et al. 2011) and 5.3% (Brownstein et al. 2017).

Discussion

Interestingly, the study by Brownstein et al. (2017), which elicited the lowest reduction in CNS activation was the only one to be tested following a competitive match. These reductions in %VA, whilst greater than the current study, are more similar than those found following simulated matches Brownstein et al. (2017). This contradicts research into self-pacing, which suggests repeated high-intensity bursts are repeated randomly through competition and it is therefore difficult to self-pace, as the external factors such as opposition and the pattern of the match do not allow for adequate recovery after every high-intensity action (Lander et al. 2009). Therefore, it would be expected that greater levels of fatigue would be elicited from competitive matches due to the randomised nature of the workloads and decision-making in comparison to simulated matches.

### 5.2.2 Aerobic Training Week

The recovery of central fatigue in TW-A follows the time course as that in previous research as both the %VA and deficits returned to pre-match baseline measurements on MD+2, 48 hours post-match. This confirms the argument made by Thomas et al. (2017), that central fatigue returns to baseline measures 48 hours post-exercise when no extra training takes place. However, both the deficit and %VA show signs of fatigue following the training session on MD+2, in which participants on average covered 5764  $\pm$  252m with a player load on average of 651.62  $\pm$  81.20N. Both measures are significantly higher than those on MD+2 of TW-AN (TD – p = 0.030; PL – p = 0.000), inferring that increased values elicit increased levels of central fatigue.

Discussion

Both values return to baseline on MD+5 and MD+6, suggesting the session on MD+3 and the day off on MD+4 allow sufficient recovery. Comparison of the results is not feasible due to lack of previous data on participants 120 hours post-match.

The TMS deficit and %VA varied greatly through the week for all positions, with no obvious trend for any of them. There was a significant difference in %VA on MD+6 between the CB and centre midfielders (CM) (p = 0.029), with the CB showing a greater fatigue level following the training week than the CM.

## 5.2.3 Anaerobic Training Week

In TW-AN, deficit and %VA were not recovered to baseline values on MD+2, but were recovered by MD+3, 72 hours post-match. This finding contradicts the above findings as for the match in TW-AN, participants covered 11% less total distance, and had a player load of 7% less than for the match. Therefore, whilst the physical outputs of the game were higher in TW-A, the CNS was more sufficiently recovered 48 hours post-match. What is not taken into account, is the match itself. As Bradley and Noakes (2013) suggest, decision-making can cause central fatigue. Therefore, matches against tougher opposition may be more mentally draining than physically which may affect the ability to produce force during the recovery phase.

Whilst the metrics in TW-AN were not recovered to baseline 48 hours post-game, participants still produced a greater %VA than previous studies (94.35  $\pm$  3.25% compared to 90% in the study by Thomas et al. 2017). This may be attributed to the demographic of participants in the study, who were not yet fully matured and

participating at an elite level. When comparing elite to non-elite players, values such as rate of force development and peak force relative to body mass were found to be significantly higher in elite, youth athletes (Gissis et al. 2016). When combining this information with the knowledge that with children show a lower degree of central fatigue than adults (Armatas et al. 2010), it can be argued that elite youth athletes would be able to produce greater force at a greater level of central activation than those in previous studies.

The TMS deficit and %VA varied greatly through the week for all positions, with no obvious trend for any of them. There was a significant difference in deficit on MD+5 between the CB and wide midfielders (WM) (p = 0.015), with the CF showing a greater fatigue level following the training week than the WM.

### 5.2.4 Additional Findings

After the MD+3, both TW-A and TW-AN followed a linear recovery until MD+6 (1 day prior to the following match), suggesting that the training weeks did not differ in the effect on the CNS activation. Whilst there are differences in the timeframe in which players return to their baseline of voluntary activation from TW-A and TW-AN, this was not significant (p = 0.750), partial eta squared = 0.006 and therefore hypothesis 2 was rejected.

### 5.3 Peripheral Stimulation / Peripheral Fatigue

The key findings from the study regarding peripheral fatigue were that there were significant differences on both the deficit (p = 0.043) and the %VA (p = 0.030) on the

day of testing. The main significant differences for testing day occurred between the MD+2 and the MD+6 for both PS deficit and PS %VA, which would be expected. 48 hours post-match is when the effects of delayed onset of muscle soreness are at their greatest (Pearcey et al. 2015) MD+6 is the day prior to the following match, when participants would expect to be fully recovered.

There were significant differences for the PS %VA from post-match to MD+3, MD+5 and MD+6, which reflects previous studies where the %VA was lowest post-match but recovered after 72 hours (Brownstein et al. 2017; Thomas et al. 2017). However, the difference was not significant between training weeks for the deficit (p = 0.289) or the %VA (p = 0.389).

As previously discussed, it is interesting to note that the PS deficit was always lower and %VA was always higher than the TMS. This suggests that the peripheral systems developed less fatigue throughout both training weeks in all testing sessions (except pre-match in TW-AN – p = 0.156). As stated, this may be due to both maturational differences and cognitive-related fatigue in comparison to other studies. With previous studies exploring the effects of central and peripheral components of fatigue following match-play or simulated match-play with no training stimulus, the effects of the training programme through the following days in this study cannot be ignored. Chronic peripheral fatigue throughout the time-period of this study may have had an effect on the ability of the skeletal muscle to produce force, especially in younger athletes (Thorpe et al. 2016).

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### 5.3.1 Pre-Match to Post-Match

The %VA drop of 0.84  $\pm$  4.44 % in TW-A and increase of 1.10  $\pm$  5.69% in TW-AN from pre-match to post-match is much less than that of previous studies of 7.1% (Brownstein t al. 2017), 7.7% (Rampinini et al. 2011) and 9.1% (Thomas et al. 2017). As the MVC reduced by 7.96  $\pm$  13.44 % (TW-A) and 10.04  $\pm$  14.55% (TW-AN), from pre-match to post-match, it is clear that the capacity of the muscle to produce force has depressed following the match. However, as the participants were able to produce similar percentages of their maximum force voluntarily, the activation of the PNS is not compromised. This suggests another peripheral factor has caused the reduction in MVC force post-match.

As stated in the following chapter, the blood lactate was significantly higher from prematch to post-match in both TW-A (p = 0.000) and TW-AN (p = 0.000). Once the accumulation of lactate exceeds the ability of the body to process it, the functioning capacity of working muscles is limited, which reduces the force and velocity of muscle contraction (Plowman and Smith, 2010). As the workloads of the match was above the OBLA, it would be expected that fatigue sensations would increase directly with the accumulation of lactate (Sharkey and Gaskill, 2013). This would account for the drop in MVC. Whilst the participants were producing a reduced force voluntarily, they were still able to produce a similar percentage of their maximum, albeit at a lesser force than pre-match.

It is possible that reductions in MVC were caused by muscle damage induced by the match. Such muscle damage could affect the muscle sense organs (Oliver et al. 2007), or the intrafusal fibres themselves (Komi, 2000). A study in 1997 by Brockett et al. established that eccentric exercise caused changes in the sense of joint position and

force production. These changes were attributed to muscle damage and disturbance of muscle receptors. It is possible that repeated sprinting, accelerating and decelerating during the match may have led to some muscle damage in this study, particularly within repeatedly recruited type II muscle fibres, which are more susceptible to muscle damage than type I fibres (Eston et al., 2003). Any such damage could then be directly related to the reduced muscle activity and performance observed in the MVC when fatigued.

### 5.3.2 Aerobic Training Week

PS deficit was highest on MD+2 of TW-A. This is 48 hours post-game, which is when Pearcey et al. (2015) argue the effects of delayed onset of muscle soreness (DOMS) peak. When combining this information with that which shows that the CNS has recovered to baseline at this point, it can be suggested that a muscle damage is causing any reduction in performance 48 hours post-match. However, the PS deficit dropped 32% on MD+3, following the session on MD+2, showing the effects of active recovery on muscle function.

As with the TMS positional differences, there were no obvious trends for the PS deficit of %VA for any positions, although there were a number of significant differences dependent on testing day. Post-match testing showed differences between CF and WM (p = 0.038) and CM and CF (p = 0.048). On MD+2, there was a significant difference between CF and WM (p=0.050). These findings show differences between the CF and all other positions, as with the TMS findings, suggesting a different recovery trajectory for CF. As previously discussed, the small number of participants in the CF category (n = 2), means that these results may not be reflective of the general population.

### 5.3.3 Anaerobic Training Week

PS deficit was also highest on MD+2 of TW-AN. However, similarly to TW-A, the PS deficit also drops dramatically ( $63.83 \pm 26.95\%$ ) on MD+3, following the session on MD+2. This may show that active recovery, including football specific work, has a positive effect on muscular performance (Nalbandian et al. 2018). This is due to the increased blood flow which aids in the removal of lactate (Wiewelhove et al. 2018), with active recovery producing a hormonal environment which may favour lipolysis and oxidative metabolism (Nalbandian et al. 2018). Whilst high power activities can cause further muscle damage, increased blood flow and testosterone can aid recovery (Raeder et al. 2017).

The above finding reflects that of central fatigue, that the session on MD+2 of both weeks allowed great improvements in peripheral fatigue (PS deficit). The improvement was much greater in TW-AN where both TD and PL are significantly lower than those on MD+2 of TW-A (TD – p = 0.030; PL – p = 0.000), inferring that increased values elicit increased time of recovery from peripheral fatigue.

There were no obvious trends in TW-AN for each position, although there were some significant differences on training day, particularly for CM who showed the lowest deficit through the week until MD+5. This opposes the finding in the TMS-induced stimulations, which suggests that a CM is more susceptible to central fatigue than peripheral. CM are generally expected to cover greater distances in games and previous studies show them having a greater aerobic capacity (VO<sub>2</sub> max) (Sporis et al. 2009). It could thus be argued that the effects of match-play would have a lesser

Both training weeks show significant improvements from 48 hours post-game to 72hours post-match, which is typical of previous studies (Brownstein et al. 2017; Thomas et al. 2017). This signifies that physical activity aids the recovery of peripheral fatigue however a balance must be found to prevent more damage being caused.

## 5.3.4 Additional Findings

The reduction in peripheral fatigue in comparison to previous studies may be affected by the age and maturational status of the participants in the current study. Previous research into adult populations found that the peripheral systems of fatigue generally recovered 72 hours post game in comparison to 48 hours post game for the central systems. However, Armatas et al. (2010) and Ayramo et al. (2017) state that children experience a lower degree of physical fatigue than adults and consequently require less recovery time, especially during high intensity exercise. Ayramo et al. (2017) argue that this can be explained by maturation-related development of force development capability, anaerobic capacity and the glycolytic enzyme system, with children benefitting more from their oxidative capacity than adults as the anaerobic system is not sufficiently mature to provide the physical output associated with short duration exercise (Ratel and Blazevitch, 2017).

Individuals begin to create waste products such as hydrogen ions in the sarcolemma which impairs many physiological processes such as force production (Milioni et al. 2017). Due to their lower anaerobic capacity, children are less affected by metabolites such as hydrogen ions, meaning that metabolic (peripheral) fatigue is lower than in adults. A study in maximal isometric and isokinetic tests in children also suggested they showed a lower degree of central fatigue than adults (Armatas et al. 2010), though

they did show a lower level of voluntary activation. This is confirmed in this study as, at the time of participation, the mean age of individuals was  $17.5 \pm 0.5$  years, which is short of the early twenties which Lilienfeld et al. (2015) argue is the point at which males are fully physically developed.

## 5.4 Comparison of Central Fatigue and Peripheral Fatigue

Previous studies into central and peripheral contributions to fatigue in football found that, whilst both systems were fatigued post-match or post-simulated match, the recovery through the week followed a linear path. Both Brownstein et al. (2017) and Rampinini et al. (2011) found that TMS-inducted involuntary contractions showed that participants had recovered from a drop in CNS voluntary activation 48 hours post-match. Thomas et al. (2017), Brownstein et al. (2017) and Rampinini et al. (2011), Brownstein et al. (2017) and Rampinini et al. (2011) all found that PS-induced involuntary contractions took up to 72 hours to return to baseline measurements, suggesting that those processes distal to the neuromuscular junction took longer to recover from than those that were proximal.

In this study however, both the deficit and the %VA testing showed greater levels of central fatigue throughout the entirety of the testing schedule, with the exception of the pre-match testing in TW-AN. However, it can be argued that the peripheral testing data was much more varied than any other point of the testing schedule for both the deficit (SD = 25.74) and the %VA (SD = 6.56). There were significant differences between the centrally-caused fatigue and peripherally-caused fatigue on both post-match and Tuesday testing for both training weeks, with significantly lower %VA and significantly higher deficits during TMS testing than PS testing (Deficit post-match TW-A – p = 0.008; Deficit post-match TW-AN – p = 0.043; %VA post-match TW-A – p = 0.026)

and on MD+3 of both weeks (Deficit MD+3 TW-A - p = 0.044; Deficit MD+3 TW-AN - p = 0.047; %VA MD+3 TW-AN - p = 0.031).

These differences could be attributed to a number of causes. Firstly, the high level of central-fatigue could be attributed to the cognitive components of competitive matches, such as the motivational differences between players which can fluctuate throughout a match (Jones and Vanhatalo 2017, Josefsson et al. 2017), however these were not assessed in this protocol. Participants were unable to self-pace as each event in the match was dependent on external factors, such as tactics and the order of goals scored, which Eston et al. (2012) suggests elicits different physical outcomes. Secondly, the reduction in peripheral fatigue in comparison to previous studies may be affected by the age and maturational status of the participants in the current study, as previously discussed.

#### 5.5 The Learning Effect

One argument that must be explored to explain the rate at which participants returned and exceeded their baseline pre-match measures through both training weeks is that of the learning effect. The learning effect is described by Goldberg et al. (2015) as changes in test performance attributed to increasing familiarity with and exposure to test instruments, paradigms, and items. Whilst participants were acclimatised to the testing procedures of both TMS and PS over a minimum of two practice sessions prior to the commencement of the study, it could be suggested that through completing the protocol more often they were able to produce greater forces and complete the MVC more efficiently due to the protocol becoming more familiar (Goldberg et al. 2015). The use of both PS and in particular, TMS, whilst non-invasive, can still be an

uncomfortable experience and the anticipation of the stimulation could in fact prevent the participant from completing a MVC. This would then lead to a greater deficit and a lower %VA. However, as participants acclimatised to the testing protocol, they would then feel more comfortable and able to focus more on producing an MVC and therefore produce results suggesting that they are less fatigued later in the training week.

It cannot be ignored that participants may have learned the test and therefore the results may not truly reflect the changes in the bodies of participants at supraspinal and peripheral levels. It can be seen that participants appear "less fatigued" at the end of each training week. It would be expected that if the learning effect was present, the difference between the two weeks would be greater, reflecting one week of a "learning phase" and one week of maximal voluntary contractions. However, as the order in which participants completed TW-A and TW-AN was random, this cannot be seen and mean scores for both deficit and %VA increase to greater than baseline by the end of the week, regardless of whether this week was the first or second one completed by each participant.

This suggests that, whilst the learning effect cannot be ignored in this study, it is not solely responsible for the results found.

# 5.6 Physical Performance and Intensity

In the following sections, the physical performance monitoring data will be discussed, including GPS metrics, RPE and blood lactate.

# 5.6.1 Match-Play GPS Metrics

During the matches, Catapult Minimax S4 GPS units (Melbourne, Australia) were used to assess total distance (TD), high speed running (HSR, distance covered at greater than 17km/h) and player load (PL). The mean TD for all players was substantially lower than in previous studies. The totals of  $9,710 \pm 2,158.64m$  (TW-A) and  $8,696 \pm 3,136.25m$  (TW-AN) are reduced from the scores of  $10,041 \pm 626m$  (Brownstein et al. 2017) and  $11,764 \pm 1,044m$  (Rampinini et al. 2011). However, a number of participants (five in TW-A and four in TW-AN), ran TD of greater than those mean distances in the matches and when taking out any participants in this study who did not complete the full 90 minutes, mean TD was much closer to previous studies ( $10338 \pm 895m$  in TW-A,  $9328 \pm 2563m$ ). It can be seen that there were not significant differences between the TD of each match (p = 0.411).

The HSR distances were less comparable to previous studies as the threshold at which running speed was considered to be at high speed differed between studies. For example, Mohr et al. (2003) chose >18km/h, whilst Rampinini et al. classified HSR as >15km/h. This study classified HSR as any distance covered >17km/h, which was the threshold used by the participants' club during their regular training sessions and matches as this was the limit used by the first team at the time. As such, whilst HSR can be compared between the two weeks, it cannot be compared to previous studies. The mean HSR distances of 1915  $\pm$  766m (TW-A) and 1985  $\pm$  874m (TW-AN) were not significantly different (p = 0.851) and therefore it can be assumed that the HSR output between the two matches would elicit similar physical responses.

As with the HSR distance, the PL was a largely unexplored area. This was utilised as a measure of physical output to compare the individuals between the two training weeks and not as a comparison to other studies. By utilising a GPS metric to calculate total workload through an algorithm, it was possible to compare the intensity of both

matches in their entirety, taking into account all metrics. The cumulative total throughout each session is calculated through an algorithm which includes changes of direction, distance (m) and speed (km/h), allowing a more detailed analysis and quantification of work completed by individuals when working in tight areas and producing high forces through high intensity efforts, such as during small-sided games (Catapult, 2019 (5)).

It can be seen that the player load was not significantly different between the matches in TW-A (952  $\pm$  228) and TW-AN (866.97  $\pm$  317) (p = 0.496). Therefore, it can be assumed that the physical output from both matches would elicit similar physical responses in the days following.

There were very few variances in GPS metrics for the matches, with only one significant difference found for player load in TW-AN between CF and CM (p = 0.005). This interestingly opposes previous findings which show large differences in distance and high-speed distance between positions (Buchheit et al. 2010). This may be due to matches not eliciting the outcomes expected from participants, which is an uncontrollable effect of field research.

## 5.6.2 Training GPS Metrics

The physical demands of each training session were crucial in this study, as the difference between the two weeks was the independent variable. Therefore, sessions were designed in a way to elicit different physical outputs between the two weeks in order to stress different energy systems.

As previously mentioned, the differences in the total physical output between the two weeks were not significantly different (TD p = 0.304; HSR p = 0.303; PL p = 0.115). As such, it may be argued that the lack of difference between the central and peripheral causes of fatigue between the two weeks would be expected. With both deficits and %VA recovering to baseline values or greater by MD+6 of each week, it can be seen that the physical demands of both training weeks allowed sufficient recovery by the following match.

It is crucial to understand that there were significant differences between metrics for certain days, meaning that whilst TD, HSR and PL may have been similar for the whole week, the way in which they were divided between sessions varied which elicit different rates of recovery. Firstly, there were significant differences between total distance on MD+2 (p = 0.030) and MD+6 (p = 0.000). When combining the increase in total distance and significant decrease in HSR (p = 0.024) on M MD+2 of TW-A compared to TW-AN, it can be argued that the increase of more low intensity running may account for the slightly reduced %VA (TMS and PS) on MD+3 in TW-A.

The HSR distance is significantly different on the MD+2 (p = 0.024), MD+5 (p = 0.013) and MD+6 (p=.035). With HSR being significantly higher on the Monday in TW-AN, it would be expected that more muscle damage would be caused, resulting in increased peripheral fatigue on the MD+3 when compared to TW-A. However, this was not found in this study. Participants were showing much greater signs of peripheral fatigue on MD+2 of TW-AN in both the PS deficit and PS %VA compared to TW-AN. By completing the session on MD+2 of TW-AN, participants made a much greater recovery from peripheral deficit from MD+2 to MD+3 (TW-A – 5.47N; TW-AN – 18.36N), suggesting shorter total distances, but with greater HSR distances may facilitate quicker peripheral recovery.

The player load was also significantly lower on MD+2 of TW-AN (p = 0.000), suggesting this session design elicited a lower total workload which may help explain the difference in recovery from MD+2 to MD+3 in both weeks.

Interestingly, the most significantly different session between TW-A and TW-AN was MD+6. TW-AN had significantly higher TD (p = 0.000), HSR (p = 0.035) and PL (p = 0.005) than TW-A. This may be due to the pattern of play drill in TW-AN which utilised a large area, eliciting large distances of running and high-speed running. This drill is not a typically anaerobic drill, but coaches at the participants' club wanted to work on tactical themes which conflicted the researcher's physical output. The randomised nature of this study and the fixture schedule meant that players would not be tested on the morning following this session and therefore the effect this session had on fatigue was not measured.

As previously suggested, there were no significant differences between the two training weeks for MVC (p = 0.625), TMS deficit (p = 0.840), TMS %VA (p = 0.750), PS deficit (p = 0.290) or PS %VA (0 = 0.39). It can be seen that each of these physical metrics were recovered to above the pre-match values by MD+6 of TW-A and TW-AN, suggesting that both training methods were effective forms of recovery for the following match. This could be due to a similarity in the overall physical outcomes of the two interventions. Whilst the physical outcomes of sessions on each week varied in total distance, high speed running and player load depending on the day of each week, the total mean of each of these metrics for each training week was very similar.

There were not great variances in GPS metrics between positions for the training sessions. MD+2 sessions on TW-A and TW-AN produced significant differences in total distance and high-speed distance for all positions, possibly due to small sided

games and passing drills which involved players to be playing in their specific positions. However, there were similar drills on other days which did not produce position-specific differences. This could also be attributed to the small sample size in each position, which may not give an accurate reflection of the population

## 5.6.3 RPE and RPE Trimp

The rate of perceived exertion (RPE) was taken from participants following each match and training session as a perceptual measure of the intensity of each session. Significant differences between the two weeks were seen on MD+3 (p = 0.017) and MD+5 (p = 0.000). However, there were no significant differences for any deficit or %VA metrics on any of the sessions following the MD+3 or MD+5, suggesting that whilst the participants had perceived differences on intensity, there were no physical effects. This may be because there were no statistical differences between GPS physical metrics on these two training days, other than HSR on MD+5 (p = 0.013). Therefore, whilst players viewed sessions as more physically demanding, there were limited differences in physical output.

The RPE trimp, the RPE multiplied by the duration of the session, was also recorded as a load measure. This is understood to be a more quantitative measure of load than RPE (Thatcher and Curtis, 2016). A short session of a high RPE may be an easier session than one of longer duration with a lower RPE, however it would be assumed that the session of a higher RPE would be a more physically demanding session.

The RPE trimp was significantly higher on MD+3 (p = 0.010) and MD+6 (p = 0.050) of TW-AN and the MD+5 of TW-A (p=.000). However total mean training RPE trimp for the two weeks was not statistically different (p=.061; TW-A – 1775 ± 114; TW-AN –

1785  $\pm$  183), suggesting that both training weeks returned to pre-match testing measures due to the similar perceived training loads. The RPE trimp for the matches in TW-A (699.20  $\pm$  172.02) and TW-AN (617.90  $\pm$  231.20) was higher than that from a study by Fullagar (2015) of 522  $\pm$  180, however this study was on professional players who would be of a more elite level than those in the current study.

There were no positional differences in RPEs in either week or RPE Trimps in TW-AN. There were differences between RPE Trimps in the match on TW-AN between CM and CF, which reflects the intensity of the match. MD+5 also elicited significant differences between CB and CF and between CM and WM. This does not reflect physical outputs from the GPS between any positions, suggesting that maybe the high intensity actions such as accelerations and decelerations impacted the participants' perceptions of the session intensity. It is not possible to prove this theory as this data was not recorded for this study.

### 5.6.4 Blood Lactate

The blood lactate was measured from pre-match to post-match to identify the blood acidity level as a result of prolonged match-play. Blood lactate was not measured as part of the pre-training testing sessions throughout the week as it would be expected that lactate levels would be at baseline values prior to training due to sufficient oxygen supply and would therefore add no value to the study (Ament and Verkeke, 2008). Due to insufficient supplies of equipment, post-training lactate measures were also not recorded and therefore, the use of blood lactate measures was to quantify the intensity of the matches.

As previously discussed, the blood lactate was significantly higher from pre-match to post-match in both TW-A (p = 0.000) and TW-AN (p = 0.000). This reflects studies by Draganidis et al. (2015) and Fransson et al. (2018) who found that lactate increased from pre-match to post match from 2.1 ± 0.3mmol/L to 6.0 ± 0.7mmol/L and from 1.14 ± 0.2mmol/L to 5.02 ± 1.0mmol/L respectively.

These values are slightly higher than those recorded post-match of  $3.63 \pm 1.38$ mmol/L (TW-A) and  $4.02 \pm 0.84$ mmol/L (TW-AN), which may be explained by the athletes tested. Those tested by Draganidis et al. (2015), were semi-professional and those tested by Fransson et al. (2018) were members of a third division Swedish team, who completed a simulated protocol for the test. The pre-match values from this study of  $1.15 \pm 0.63$ mmol/L (TW-A) and  $1.15 \pm 0.31$ mmol/L (TW-AN) were similar to those previous studies.

There were no significant differences between position in TW-AN but in TW-AN, the more explosive positions of CF and WM produced significantly higher post-match lactate than the CM and CB. As suggested with the RPE trimp, this is not reflective of the GPS outputs, suggesting that physical movement that was not recorded could be the cause of this increase in blood lactate, such as accelerations and decelerations, which Buchheit et al. (2010), state are increased in CM and WM.

Interestingly, the blood lactate values recorded by Fransson et al. (2018) after every 15 minutes of the simulated protocol were higher in the first half than the second. This is something that was not explored in this study as it would not have been possible to stop the competitive fixture every 15 minutes to record lactate, but could be explored in future research.

### 5.7 Study Limitations

As this study was the first to intimately research fatigue modalities in elite, youth, male footballers, there were some major limitations that would not have been expected from previous studies. Due to this study being completed on competitive matches, the location of the TMS and PS testing away from the pitch was necessary for reliability and validity reasons. However, this was not directly opposite the pitch, meaning a slight delay from the finish of the match to the testing beginning which may explain the reduced fatigue levels in TMS and PS testing when compared to previous studies. With the testing area being approximately 400m from the match pitch, it would take five minutes for participants to return there. With up to four participants being tested, there were occasional delays of up to an additional five minutes whilst participants waited for others to be tested. Ostojic (2016) argues that whilst this is a minimal delay, the recovery process will have already begun (reduction in heart rate) and therefore may have affected the results. Due to the field-based nature of the study, all participants finished the match at the same time, unless they were substituted, and therefore this slight delay was unavoidable. It was ensured that players were tested in the same order from pre-match to post-match.

Whilst there were significant differences between the different GPS metrics and RPE Trimps on different days between the two training weeks, the totals for the two weeks were very similar. With this study being based on real training sessions for elite competition and participants training to earn professional contracts, the physical parameters for training sessions were difficult to manipulate, especially as the testing interventions took place over six weeks. Participants needed to recover and complete physical development work to allow them the best opportunity to earn future employment. This meant that whilst the study was accommodated and sessions were

altered accordingly, there were not huge differences between the two weeks as each participant needed to hit certain physical benchmarks through each week. Although there were significant differences for all metrics on numerous days, there were no differences throughout the week for TD (p = 0.304), HSR (p = 0.303) or PL (p = 0.115). With this study researching the effect of fatigue over the week, the difference in workload was not significant to cause a large effect.

### **5.8 Directions for Future Study**

The current study has identified differing time courses of recovery dependent on the physical design of sessions, with both training weeks facilitating sufficient recovery to baseline values before the following match. However, with participants not being tested 24 hours post-match, when previous studies found the most intimate detail about recovery from central and peripheral factors (Rampinini et al. 2011; Brownstein et al. 2017; Goodall et al. 2017), a lot of information was lost. If this study were to be replicated, it would be important to add in the testing session 24 hours post-match to greater show recovery methods, especially those from central factors as previous studies have found that most individuals have recovered centrally by 48 hours (Rampinini et al. 2011; Brownstein et al. 2017; Goodall et al. 2011; Brownstein et al. 2017; Goodall et al. 2011; Brownstein at a most individuals have recovered centrally by 48 hours (Rampinini et al. 2011; Brownstein et al. 2017; Goodall et al. 2011; Brownstein et al. 2017; Goodall et al. 2017).

As previously discussed, it was difficult to hugely manipulate training sessions, however future study should have greater differences between the training weeks for all GPS metrics and RPEs. This may show a different response to training in both central and peripheral fatigue, allowing greater understanding of the effect of TD, HSR and the intensity of training on the time-course of recovery from competitive match-play.

Whilst this study included all pitch-based sessions through the weeks, gym-based conditioning and recovery modalities were removed from the participants schedules. Ihsan et al. (2016) argue that recovery strategies such as cold-water immersion and lymphatic drainage can reduce recovery time, whilst an increase in load from strength sessions may increase recovery time (Miranda et al. 2018). Therefore, to give a complete representation of the effects of an elite youth football training week, a full schedule needs to be completed in future studies.

The study would have benefited from more metrics measuring the intensity of sessions. Firstly, the use of heart rate monitors would have allowed the researcher to monitor internal training load. An increase in heart rate can facilitate removal of waste product from the muscles (Wiewelhove et al. 2018). However, by monitoring individual heart rate zones, it would be possible to keep a balance between an increased heart rate to aid removal of waste products and working at a near maximum heart rate for extended periods of time, which may increase chronic fatigue through the week (Kampshoff et al. 2015).

Secondly, Eston et al. (2003) argue that repeated high intensity actions such as sprinting, accelerating and decelerating may lead to muscle damage, which can affect the MVC. The thresholds for accelerations and decelerations on the GPS units used for the currently study were not sensitive enough to identify the number of high intensity actions and were therefore ignored. However, by identifying the number of high intensity actions, it would be possible to assess the external load of participants in more detail, showing the effects that these muscle damage-causing actions have on recovery.

Whilst RPE scales were used to measure the perceptual intensity of sessions, the perceptual effect of these sessions was not measured. The physical effects of training were recorded through TMS and PS, however the feelings of the participants were not considered, and whilst they may have presented as physically recovered, they may not have felt ready to train. Therefore, a mixed-methods approach including well-being questionnaires would add another layer of depth to the recovery status of athletes.

Finally, research into the training methods which are most effective for each position would allow more individualised training programmes to be written. As central midfielders and wingers will have different physical outputs in matches such as TD and HSR, the fatigue for each post-match may differ. This study identified differences between playing position in numerous metrics, however due to the small sample sizes of each position, this would need to be explored in more depth to gain a reliable understanding of the overall population.

## 6.0 Conclusion

With elite football players often competing in multiple games per week, the requirement for monitoring of training and match load is crucial to allow the greatest recovery in time for the next fixture (Lundberg and Wekström, 2017). De Hoyo et al. (2016) and Lundberg and Wekström (2017) suggest it can take up to 72 hours for an individual to be fully recovered from a match, which may involve another fixture, meaning the individual may still be feeling the effects of fatigue before kick-off. Therefore, it is crucial to prescribe effective recovery modalities or training stimuli to improve technical, tactical and physical attributes, whilst allowing the best possible chance of a player being in the optimum physical condition for their next matchday (McLean et al. 2016).

Reflecting on previous study provided an insight into how recovery time may vary between central and peripheral methods. The use of TMS identified a decrease in voluntary activation measured through motor cortex stimulation, showing a reduced capacity for the motor cortex to drive the knee-extensors immediately post-stimulation, suggesting a contribution of supraspinal factors to central fatigue in the participants (Goodall et al. 2017). Conversely, motor nerve stimulation displayed a decrease up to 72-hours post-exercise, with Thomas et al. (2017) arguing that the reduction in voluntary activation 3-days post-exercise is due to peripheral factors.

The primary aim of this study was to identify the deficit between voluntary, isometric quadricep contractions and involuntary, Transcranial Magnetic Stimulation-Induced and Peripheral Stimulation-induced isometric quadricep contractions in elite youth football players and the effect that different training methods have on recovery. The data demonstrates that a competitive youth fixture elicits reductions in the force-

generating capabilities of the Vastus Lateralis, with this fatigue being made up of both central and peripheral factors. The main findings are identified below:

- Maximal Voluntary Contraction: The mean MVC produced by participants varied dependent on the day of testing. Although the two training weeks did not elicit a significant difference (p = 0.625), the day in the week in which MVC was tested produced a statistically significant difference (p = 0.000). With MVCs being significantly higher towards the end of the week than both pre-match and post-match values, it can be argued that both training protocols provided the platform for effective recovery from the match.
- Central Fatigue: TMS %VA (p = 0.020) produced a statistically significant difference dependent on the day of testing. There was also a significant difference from pre-match to post-match for TMS deficit. However, there were no differences between training weeks for deficit or %VA, suggesting that the physical outputs of the two training weeks were similar. In both the TMS deficit and the TMS %VA, values returned to pre-match baseline measures after 48 hours in TW-A, whilst it took 72 hours in TW-AN, which may be due to mental fatigue. However, these differences were not statistically significant.
- Peripheral Fatigue: The key finding from the study regarding peripheral fatigue was that there were significant differences on both the deficit (p = 0.043) and the %VA (p = 0.030) on the day of testing. The main significant differences for testing day occurred between the Monday and the Friday for both PS deficit and PS %VA. However, the difference was not significant between training weeks for the deficit (p = 0.289) or the %VA (p = 0.389). It is interesting to note that the PS deficit was always lower and %VA was always higher than the TMS. This suggests that the peripheral systems developed less fatigue throughout

both training weeks in all testing sessions (except pre-match in TW-AN - p = 0.156). This may be due to both maturational differences and cognitive-related fatigue in comparison to other studies.

Physical performance and intensity: Whilst the physical outcomes of sessions on each week varied in total distance, high speed running and player load depending on the day of each week, the total mean of each of these metrics for each training week was very similar (TD p = 0.304; HSR p = 0.303; PL p = 0.115). RPE trimps were higher than in previous studies for matches, which may be because individuals in the previous research were professional, adult players, who may find a match less physically demanding. The blood lactate values recorded post-match were lower than those in previous studies, which may be due to the level of the athletes in this study.

The first hypothesis of the study was that an elite, youth football player will present with a greater level of both central and peripheral fatigue following a competitive football match. In all MVC and %VA scores for both TMS and PS for TW-A and TW-AN, scores were lower post-match. The deficit scores for all the above metrics were greater post-match than pre-match. As none of these were statistically significant, hypothesis one was proved to be incorrect.

The second hypothesis was that following a competitive football match, a training week of lower intensity, including less high speed running and smaller training areas, will facilitate accelerated recovery from both central and peripheral fatigue than a training week of higher intensity. As there were no statistical differences in MVC, deficit or %VA for either TMS or PS between the two training weeks, the null hypothesis must be accepted.

# **6.1 Practical Applications**

Collectively, these data add to growing evidence that prolonged impairments in the capacity of the peripheral and central nervous systems to activate the quadriceps muscles are implicated in fatigue following intermittent high intensity exercise (Rampinini et al., 2011; Thomas et al., 2017). Therefore, this may have important implications for the optimisation of the weekly training process and the implementation of appropriate and effective recovery interventions to allow optimal physical preparation for competitive youth football matches.

The results of this study can have a number of practical applications. Firstly, by understanding the time-course of recovery, coaches and sports scientists will be able to make more informed decisions when planning training schedules. As the young participants from this study recover from peripheral fatigue at a quicker rate than adults from other studies, the training sessions for this club can vary from those at first team level because the youth players are above their baseline measures of deficit and %VA quicker. The ability of the individuals in this study to produce their MVC and their prematch %VA at an accelerated rate will allow them to begin a more intense training schedule quicker than they currently do. As discussed, it is crucial for youth footballers to balance the risk of injury, recovery and progressive physical development. However, with a quicker recovery, there is a greater window in the week for physical sessions to promote development into athletes that are able to withstand the demands of first team football.

Alongside these training prescriptions in a one match training week, it is also possible to understand recovery status of athletes when they have multiple matches in one week. By understanding the aetiology of fatigue, it is possible to decide on effective

recovery strategies to accelerate recovery in congested fixture schedules to improve performance and reduce injury risk. Thus, it will be possible for the club involved in this study and other academies across the country to research the effect that recovery strategies, such as cryotherapy, have on central and peripheral divisions of fatigue. As previously discussed, the introduction of well-being questionnaires at the club in this study will help to identify subjective fatigue alongside the objective measures that are already in place to provide a more comprehensive understanding of readiness to train.

Whilst the study investigated numerous different variables and identified some interesting findings, the conflict between researcher and practitioner produced some difficulties. Study limitations were mostly attributed to completing this study via field-based research, which was a major strength but also a weakness. The inability to greatly alter training prescription due to EPPP rulings lead to very similar physical outputs from TW-A to TW-AN. This is a realistic overview of a training week in an elite academy, however by producing opposing physical outputs in each training week, it would be expected that greater differences would have been found which may have been more informative for future training prescription.

References

#### References

Abd-Elfattah, H,M., Abdelazeim, F.H. and Elshennawy, S., 2015. Physical and Cognitive Consequences of Fatigue: A Review. *Journal of Advanced Research.* 6(3). Pages 351-358.

Abiss, C. and Laursen P., 2005. Models to Explain Fatigue During Prolonged Endurance Cycling. *Sports Medicine*. 35(10). Pages 865-898.

Achten, J., Venables, M.C. and Jeukendrup, A.E., 2003. Fat Oxidation Rates are Higher During Running Compared with Cycling Over a Wide Range of Intensities. *Metabolism.* 52(6). Pages 747-752.

Acton, A., 2013. *Thiadiazoles – Advances in Research and Application: 2013 Edition: Scholarly Paper*. Scholarly Editions. USA: Atlanta.

Alain, G. and Matran, R., 2012. Relationships Between Pscyhological Factors, RPE and Time Limit Estimated by Teleoanticipation. *Sports Psychologist.* 26. Pages 359-374.

Alcaraz, P.E., Matinlauri, A., Abedin-Maghanagi, A., Freitas, T.T., Martinez-Ruiz, E., Castillo, A., Mendiguchia, J. and Cohen, D.D., 2017. Comparison of the Isometric Force Fatigue-Recovery Profile in Two Posterior Chain Lower Limb Tests Following Simulated Football. *British Journal of Sports Medicine.* 51(4). Page 285. Alghannam, A., 2012. Metabolic Limitations of Performance and Fatigue in Football. *Asian Journal of Sports Medicine.* 3(2). Pages 65-73.

Ament, W. and Verkerke, G.J., 2009. Exercise and Fatigue. *Sports Medicine*. 39(5). Pages 389-422.

Andersen, G., Christenen, D., Kirkevold, M. and., Johnsen, S.P. 2012. Post-Stroke Fatigue and Return to Work: A 2-Year Follow-Up. Acta, Neurologica Scandanavica. 125(4). Pages 248-253

Anthony, J. 2014. *Design of Experiments for Engineers and Scientists.* (Second Edition). Elsevier. United Kingdom: London.

Armatas, V., Bassa, E., Patikas, Kitsas, I., Zangelidis, G. and Kotzamanidis., 2010. Neuromuscular Differences Between Men and Pre-Pubescent Boys During a Peak Isometric Knee-Extension Intermittent Fatigue Test. *Pediatric Exercise Science*. 22(5). Pages 205-217.

Arnold, B.L. and Schilling, B.K., 2017. *Evidence-Based Practice in Sport and Exercise: A Guide to Using Research.* F.A. Davis Company. USA: Philadelphia.

Andrade, C., 2019. Multiple Testing and Protection Against a Type 1 (False Positive) Error Using the Bonferroni and Hochberg Corrections. Indian Journal of Psychological Medicine. 41(9). Pages 99-100. Astrand, P.O., Rodahl, K., Dahl, H.A., and Stromme, S.E. 2003. *Textbook of Work Physiology: Physiological Bases of Exercise.* (Fourth Edition). Human Kinetics. USA: Champaign.

Ayramo, S., Vilmi, S., Antero Mero, A., Piirainen, J., Nummela, A., Pullinen, T., Avela, J. and Linnamo, S., 2017. Maturation-Related Differences in Neuromuscular Fatigue After a Short-Term Maximal Run. *Human Movement.* 18(3). Pages 17-25.

Babbie, E.R., 2017. *The Basics of Social Research.* (Seventh Edition). Cengage Learning. USA: Boston.

Badawy, R.A.B., Loetscher, T., Macdonell, R.A.L. and Brodtmann, A., 2012. Cortical Excitability and Neurology: Insights Into the Pathophysiology. *Functional Neurology*. *27*(3). Pages 131-145.

Bangsbo, J., 2014. Physiological Demands of Football. *Sports Science Exchange*. 27(315). Pages 1-6.

Bangsbo, J., Krustup, P., Hansen, P.R., Ottesen, L., Pfister, G. and Elbe, A.M., 2017. Science and Football VIII: The Proceedings of the Eighth World Congress on Science and Football. Routledge. United Kingdom: Abingdon.

Barber-Westing, S.D. and Noyes, F.R., 2017. Effect of Fatigue Protocols on Lower Limb Neuromuscular Function and Implications for Anterior Cruciate Ligament Injury Prevention Training: A Systematic Review. *American Journal of Sports Medicine*. 45(14). Pages 3388-3396.

Bartlett, H.P., Simonite, V., Westcott, E. and Taylor, H.R., 2000. A Comparison of the Nursing Competence of Graduates and Diplomates from UK Nursing Programmes. Journal of Clinical Nursing. 9. Pages 369-381.

Bawa, P.N.S., Piotrkiewicz, M. and Schmied, A., 2015. Mechanisms Underlying Firing in Healthy and Sick Human Motoneurons. *Fronteirs in Human Neuroscience*. 9. Page 174.

Beachey, W., 2012. *Respiratory Care Anatomy and Physiology: Foundations for Clinical Practice* (Third Edition). Elsevier Health. United Kingdom: Kidlington.

Belanger, A.Y. and McComas, A.Y. 1981. Extent of Motor Unit Activation During Effort. *Journal of Applied Physiology.* 51(5). Pages 1131-1135.

Billaut, F., Bishop, D.J., Schaerz, S. and Noakes, T.D., 2011. Influence of Knowledge of Sprint Number on Pacing During Repeated-Sprint Exercise. *Medicine and Science in Sports and Exercise*. 43(4). Pages 665-672.

Boccia, G., Dardanello, D., Zoppirolli, C., Bortolan, L., Cescon, C., Schneebeli, A., Vernillo, G., Schena, F., Rainoldi, A. and Pellegrini. 2017. Central and Peripheral Fatigue in Knee and Elbow Extensor Muscles After a Long-Distance Cross-Country Ski Race. Scandinavian Journal of Medicine and Science in Sports. 27(9). Pages 945-955.

Borg, G.A.V., 1982. Psychophysical Bases of Perceived Exertion. *Medicine in Sports Science and Exercise*. 14(5). Pages 377-381.

Bradley, P.S. and Noakes, T.D. 2013., Match Running Performance Fluctuations in Elite Soccer: Indicative of Fatigue, Pacing or Situational Influence? *Journal of Sports Sciences*. 31(15). Pages 1627-1638.

Brannen, J., 2017. *Mixing Methods: Qualitative and Quantitative Research.* (Second Edition). Routledge. United Kingdom: Abingdon.

Brockett, C., Warren, N., Gregory, J.E., Morgan, D.L. and Proske, U., 1997. A Comparison of the Effects of Concentric Versus Eccentric Exercise on Force and Position Sense in the Human Elbow Joint. *Brain Research.* 771. Pages 251-258.

Brownstein, C.G., Dent, J.P., Parker, P., Hicks, K.M., Howatson, G., Goodall S. and Thomas, K., 2017. Etiology and Recovery of Neuromuscular Fatigue Following Competitive Soccer Match-Play. *Frontiers in Physiology.* 25(8). Pages 831- 843.

Buchheit, M., Mendez-Villanueva, A., Simpson, B.M. and Bourdon, P.C., 2010. Match Running Performance and Fitness in Youth Soccer. *International Journal of Sports Medicine.* 31. Pages 818-825.

References

Carey, D.G., Drake, M.M., Pliego, G.J. and Raymond, R.L., 2007. Do Hockey Players Need Aerobic Fitness? Relation Between VO<sub>2</sub> Max and Fatigue During High Intensity Intermittent Ice Skating. *Journal of Strength and Conditioning Research.* 21(3). Pages 963-966.

Carroll, T.J., Taylor, J.L. and Gandevia, S.C., 2017. Recovery of Central and Peripheral Neuromuscular Fatigue After Exercise. *Journal of Applied Physiology.* 122(5), Pages 1068-1076.

Casamichana, D. and Castellano, J., 2010. Time-Motion, Heart Rate, Perceptual and Motor Behaviour Demands in Small-Sides Soccer Games: Effects of Pitch size. *Journal of Sports Sciences*. 28(14). Pages 1615-1623.

Catapult (1)., 2018. *Catapult Minimax S4 Brochure.* Available from: https://www.scribd.com/document/204037166/Catapult-Minimax-S4-Brochure (Accessed on 12/04/2018)

Catapult (2)., 2018. *Individualisation of GPS Speed Thresholds: Challenges and Complexities.* Available from: https://www.catapultsports.com/blog/individualisation-gps-speed-thresholds-challenges-complexities (Accessed on 28/02/2018).

Catapult (3)., 2018. *Our Products.* Available from: https://www.catapultsports.com/products (Accessed on 14/11/2018).

Catapult (4)., 2019. *Catapult Fundamentals: Why Use GPS Tracking Technology?* Available from: https://www.catapultsports.com/blog/catapult-fundamentals-gps-tracking-technology (Accessed on 27/02/2019).

Catapult (5)., 2019. *Catapult Fundamentals: What Can Playerload Tell Me About Athlete Work?* Available from: https://www.catapultsports.com/blog/fundamentals-playerload-athlete-work (accessed on 27/02/2019).

Cavaleri, R., Schabrun, S.M. and Chipchase, L.S., 2018. The Reliability and Validity of Rapid Transcranial Magnetic Stimulation Mapping. *Brain Stimulation.* 11(6). Pages 1291-1295.

Chen, Y.J., Mahieu, N.G., Huang, X., Singh, M., Crawford, P.A., Johnson, S.L., Gross, R.W., Schaefer, J. and Patti, G.J. 2016. *Nature Chemical Biology.* 12. Pages 937-943.

Christian, R.J., Bishop, D.J., Billaut, F. and Girard, O., 2015. The Role of Sense of Effort on Self-Selected Cycling Power Output. *Frontiers in Physiology.* 31(5). Page 115.

Christopher, J., Beato, M. and Hulton, A.T., 2016. Manipulation of Exercise to Rest Ratio Within Set Duration on Physical and Technical Outcomes During Small-Sided Games in Elite youth Soccer Players. *Human Movement Science*. 48. Pages 1-6. Cian, C., Barraud, P.A., Melin, B. and Raphel, C., 2001. Effects of Fluid Ingestion on Cognitive Function After Heat Stress or Exercise-Induced Dehydration. *International Journal of Psychophysiology.* 42. Pages 243-251.

Cian, C., Koulmann, N., Barraud, P.A., Raphel, C., Jiminez, C. and Melin, B., 2000. Influence of Variations in Body Hydration on Cognitive Function: Effects of Hyperhydration, Heat-Stress and Exercise-Induced Dehydration. *Journal of Psychophysiology.* 14. Pages 29-36.

Clarke, N., Farthing, J.P., Lanovaz, J.L. and Krentz, J.R., 2015. Direct and Indirect Measurement of Neuromuscular Fatigue in Canadian Football Players. Applied Physiology, Nutrition and Metabolism. 40. Pages 464-473.

Coburn, J.W. and Malek, M.H., 2012. *NSCA's Essentials of Personal Training* (Second Edition). Human Kinetics. USA: Champaign.

Cooke, S.R., Petersen, S.R. and Quinney, H.A., 1997. The Influence of Maximal Aerobic Power on Recovery of Skeletal Muscle Following Anaerobic Exercise. *European Journal of Applied Physiology and Occupational Physiology.* 75(6). Pages 512-519.

Coulson, M. and Archer, D., 2015. *Physical Fitness Testing: Analysis in Exercise and Sport.* A&C Black. United Kingdom: London.

Crone, E., 2017. *The Adolescent Brain: Changes in Learning, Decision-Making and Social Relations*. Routledge. United Kingdom: Abingdon.

Debold, E.P., 2012. Recent Insights into Muscle Fatigue at the Cross-Bridge Level. *Frontiers in Physiology.* 3. Page 151.

Decorte, N., Lafaix, P.A., Millett, G.Y., Wuyam, B. and Verges, S., 2012. Central and Peripheral Fatigue Kinetics During Exhaustive Constant-Load Cycling. *Scandinavian Journal of Medicine and Science in Sport.* 22(3). Pages 381-391.

De Hoyo, Cohen, D.D., Sanudo, B., Carrasco, L., Alvarez-Mesa, A., del Ojo, J.J., Dominiquez-Cobo, S., Manas, V. and Otero, Esquina. 2016. Influence of Football Match Time-Motion Parameters on Recovery Time Course of Muscle Damage and Jump Ability. *Science and Medicine in Football.* 34(14). Pages 1363-1370.

Dekerle, J., Greenhouse-Tucknott, A., Wrightson, J.G., Schäfer, L. and Ansdell, P., 2019. Improving the Measurement of TMS-Assessed Voluntary Activation in the Knee Extensors. *PLoS One.* 14(6).

Desai, T. and Bottoms, L., 2016. Neck Cooling Improves Table Tennis Performance Amongst Young National Level Players. *Sports.* 5(1). Pages 1-8.

Douglas, P.S., O'Toole, M.L., Hiller, W.D., Hackney, K. and Reichek, N., 1987. Cardiac Fatigue After Prolonger Exercise. *Circulation.* 79. Pages 1206-1213.

References

Draganidis, D., Chatzinikolaou, A., Avlonitj, A., Barbero-Alvarez., J.C., Mohr, M., Malliou, P., Gourgoulis, V., Deli, C.K., Douroudos, I.I., Margonis, K., Gioftsidou, A., Fouris, A.D., Jamurtas, A.Z., Koutedakis, Y. and Fatouris, I.G., 2015. Recovery Kinetics of Knee Flexor and Extensor Strength after a Football Match. *PLoS One.* 10(6).

Draper, N., Brent, S., Hale, B. and Coleman, I., 2006. The Influence of Sampling Site and Assay Method on Lactate Concentration in Response to Rock Climbing. European Journal of Applied Physiology. 98(4). Pages 363-372.

Edwards, A.M., Bentley, M.B., Mann, M.E. and Seaholme, T.S., 2011. Self-Pacing in Interval Training: A Teleoanticipatory Approach. *Psychophysiology.* 48(1). Pages 136-141.

EKF Diagnostics., 2019. Biosen C-Line – Glucose and Lactate Analysers. Available from: https://www.ekfdiagnostics.com/biosen-analyzer.html (Accessed on 31 July 2019)

Ekstrand, J., Hägglund, M. and Walden, M. 2011. Injury Incidence and Injury Patterns in Professional Football – The UEFA Injury Study. *British Journal of Sports Medicine*. 45(7). Pages 553-558.

Eldabe, S., Buchser, E. and V. Duarte, R., 2016. Complications of Spinal Cord Stimulation and Peripheral Nerve Stimulation Techniques: A Review of the Literature. *Pain Medicine.* 17(2). Pages 325-336.

References

EMB Consult., 2015. *Homunculus: Somatosensory and Somatomotor Cortex.* Available at: https://www.ebmconsult.com/articles/homunculus-sensory-motor-cortex (Accessed on 14 February 2018)

Enoka, R.M. and Duchateau, J., 2008. Muscle Fatigue: What, Why and How it Influences Muscle Function. *Journal of Physiology*. 586. Pages 11-23.

Espregueira-Mendes, J., van Dijk, C.N., Nevret, P., Cohen, M., Della, Villa, S., Pereira, H. and Oliveira, J.M. 2017. *Injuries and Health Problems in Football: What Everyone Should Know.* Springer. Germany: Berlin.

Eston, R., Byrne, C. and Twist, C., 2003. Muscle Function After Exercise-Induced Muscle Damage: Considerations for Athletic Performance in Children and Adults. *Journal of Exercise Science and Fitness.* 1. Pages 85-96.

Falgairette, G., Bedu, M., Fellmann, N., Van-Praagh, E. and Coudert, J., 1991. Bio-Energetic Profile in 144 Boys Aged from 6 to 15 Years with Special Reference to Sexual Maturation. *European Journal of Applied Physiology and Occupational Therapy.* 62(3). Pages 151-156.

Field, A., 2005. *Discovering Statistics Using SPSS.* (Second Edition). Sage Productions. UK: London.

Fletcher, G.F., Ades, P.A., Kligfield, P., Arena, R., Balady, G.J., Bittner, V.A., Coke, L.A., Fleg, J.L., Forman, D.E., Gerber, T.C., Gulati, M., Madan, K., Rhodes, J., Thompson, P.D. and Williams, M.A., 2013. Exercise Standard for Testing and Training: A Scientific Statement from the American Heart Association. *Circulation.* 128. Pages 873-934.

FluidSurveys., 2014. *Presenting Your Rating Scales – Numbered Versus Worded Lists.* Available at: http://fluidsurveys.com/university/number-versus-word-rating-scales/ (Accessed: 31 January 2018).

Fransson, D., Vigh-Larsen, J.P., Fatouros, I.G., Krustup, P. and Mohr, M., 2018. Fatigue Responses in Various Muscle Groups in Well-Trained Competitive Male Players After a Simulated Soccer Game. *Journal of Human Kinetics*. 61(1). Pages 85-97.

Froyd, C., Beltrami, F.G., Millet, G.Y. Noakes, T.D. 2016. No Critical Peripheral Fatigue Threshold During Intermittent Isometric Time to Task Failure Test with the Knee Extensors. *Frontiers in Physiology.* **7**. Page 627.

Froyd, C., Millet, G.Y. and Noakes, T.D., 2013. The Development of Peripheral Fatigue and Short-Term Recovery During Self-Paced High-Intensity Exercise. *The Journal of Physiology.* 591(5). Pages 1339-1346.

Fullagar, H.H.K., 2015. Impaired Sleep and Recovery After Night Matches in Elite Football Players. *Science and Medicine in Football.* 34(14). Pages 1333-1339.

References

Gandevia, S.C., Allen, G.M., Butler, J.E. and Taylor, J.L., 1996. Supraspinal Factors in Human Muscle Fatigue: Evidence for Suboptimal Output from the Motor Cortex. *Journal of Physiology.* 490(2). Pages 529-536.

Gifford, C. 2008., Soccer. The Rosen Publishing Group. USA: New York.

Glass, G.V., Peckham, P.D. and Sanders, J.R., 1972. Consequences of failure to meet assumptions underlying fixed effects analyses of variance and covariance. *Review of Education in Research.* 42(3). Pages 237-288.

Goldberg, T.E., Harvey, P.D., Wesnes, K.A., Snyder, P.J. and Schneider, L.S., 2015. Practice Effects Due to Serial Cognitive Assessment: Implications for Preclinical Alzheimer's Disease Randomized Controlled Trials. *Alzheimer's and Dementia: Diagnosis, Assessment and Disease Monitoring.* 1(1). Pages 13-111.

Gonzalez-Alonso, J., Teller, C., Andersen, S.L., Jensen, F.B., Hyldig, T. and Nielsen, B., 1999. Influence of Body Temperature on the Development of Fatigue During Prolonged Exercise in the Heat. Journal of Applied Physiology. 86. Pages 1032-1039.

Goodall, S., Charlton K., Howatson, G., and Thomas K., 2015. Neuromuscular Fatigability During Repeated-Sprint Exercise in Male Athletes. *Medicine and Science in Sports and Exercise*. 47(3). Pages 528-536.

Goodall, S., Howatson, G., Romer, L., and Ross, E., 2014. Transcranial Magnetic Stimulation in Sport Science: A Commentary. *European Journal of Sport Science*. 14(1). Pages 332-340.

Goodall, S., Ross, E.Z. and Romer, L.M., 2010. Effect of Graded Hypoxia on Supraspinal Contributions to Fatigue with Unilateral Knee Extensor Contractions. *Journal of Applied Physiology.* 109(6). Pages 1842-1851.

Goodall, S., Thomas, K., Harper, L.D., Hunter, R., Parker, P., Stevenson, E., West, D., Russell, M. and Howatson, G., 2017. The Assessment of Neuromuscular Fatigue During 120 Min of Simulated Soccer Exercise. *European Journal of Applied Physiology.* 117(4). Pages 687-697.

Greger, R. and Windhorst, U., 1996. *Comprehensive Human Physiology: From Cellular Mechanisms to Integration.* Springer. Switzerland: Cham.

Grif Alspach, J., 2013. *Core Curriculum for Critical Care Nursing – E-Book*. Elsevier. United Kingdom: Kiddlington.

Griffiths, C., O'Neill-Kerr, A., Millward, T. and da Silva, K. 2018. Repetitive Transcranial Magnetic Stimulation (rTMS) for Depression: Outcomes in a United Kingdom (UK) Clinical Practice. *International Journal of Psychiatry in Clinical Practice*. 23(2). Pages 122-127. Grigg, P., 1994. Peripheral Neural Mechanisms in Proprioception. *Journal of Sports Rehabilitation.* 3. Pages 2-17.

Hancock, A.P., Sprks, K.E. and Kullman, E.L., 2015. Postactivation Potentiation Enhances Swim Performance in Collegiate Swimmers. *Journal of Strength and Conditioning Research.* 29(4). Pages 912-917.

Harper, L.D., Hunter, R., Parker, P., Goodall, S., Thomas, K., Howatson, G., West, D.J., Stevenson, E. and Russell, M., 2016. Test-Retest Reliability of Physiological and Performance Responses to 120 Minutes of Simulated Soccer Match-Play. *Journal of Strength and Conditioning Research.* 30. Pages 3178-3186.

Harwell, M.R., Rubinstein, E.N., Hayes, W.S. and Olds, C.C., 1992. Summarizing Monte Carlo results in methodological research: the one- and two-factor fixed effects ANOVA cases. *Journal of Educational and Behavioural Statistics.* 17(4). Pages 315-339.

Herbert, R.D. and Gandevia, S.C., 1999. Twitch Interpolation in Human Muscles: Mechanisms and Implications for measurement of Voluntary Activation. *Journal of Neurophysiology*. 82(5). Pages 2271-2283.

Hodgson, M., Docherty, D. and Robbins, D., 2005. Post-Activation Potential. *Sports Medicine.* 35(7). Pages 585-595.

Hoffman, J.R., 1997. The Relationship Between Aerobic Fitness and Recovery from High-Intensity Exercise in Infantry Soldiers. *Military Medicine*. 162(7). Pages 484-488.

Hogarth, L.W., Burkett, B.J., and McKen, M.R., 2015. Influence of Yo-Yo IR2 Scores in Internal and External Workloads and Fatigue Responses of Tag Football Players During Tournament Competition. *PLoS ONE.* 10(10).

Hogervorst, E., Riedel, W., Jeukendrup, A. and Jolles, J., 1996. Cognitive Performance After Strenuous Physical Exercise. *Percept Motor Skills*. 83. Pages 479-488.

Holway, F.E. and Spriet, L.L., 2011. Sport-Specific Nutrition: Practical Strategies for Team Sports. *Journal of Sports Sciences*. 21(1). Pages 115-125.

Horn, T.S., 2008. *Advances in Sport Psychology.* (Third Edition). Human Kinetics. USA: Illinois.

Housh, T.J., Housh, D.J. and DeVries, H.A., 2017. *Applied Exercise and Sport Physiology, With Labs.* (Fourth Edition. Routledge. United Kingdom: Abingdon.

Miranda, H., Maia, M.F., Paz, G.A., de Souza, J., Simao, R., Farias, D.A. and Willardson, J.M., 2018. Repetition Performance and Blood Lactate Responses Adopting Different Recovery Protocols Between Training Sessions in Trained Men. *The Journal of Strength and Conditioning Research*. 32(12). Pages 3340-3347.

References

Hunter, F., Bray, J., Towlson, C., Smith, M., Barrett, S., Madden, J., Abt, G. and Lovell, R. 2015. Individualistion of Time-Motion Analysis: A Method Comparison and Case Report Series. International Journal of Sports Medicine. 36(1). Pages 41-48.

Hureau, T.J., Ducrocq, G.P., and Blain G.M., 2009. Peripheral and Central Fatigue Development During All-Out Repeated Cycling Sprints. *Medicine and Science in Sports and Exercise.* 48(3). Pages 391-401.

Husain, A., 2012. Social Psychology. Dorling Kindersley. India: New Dehli.

Ihsan, M., Watson, G. and Abbiss, C.R., 2016. What are the Physiological Mechanisms for Post-Exercise Cold Water Immersion in the Recovery from Prolonged Endurance and Intermittent Exercise? *Sports Medicine.* 46(8). Pages 1095-109.

Inzlicht, M. and Marcoca, S.M., 2016. The Central Governor Model of Exercise Regulation Teaches us Precious Little about the Nature of Mental Fatigue and Self-Control Failure. *Frontiers in Psychology*. 7. Pages 1-6.

John, P., 2017. Field Experiments in Political Science and Public Policy: Practical Lessons in Design and Delivery. Routledge. USA: New York.

Johnston, R.J., Watsford, M.L., Kelly, S.J., Pine, M.J. and Spurrs, R.W., 2019. Validity and Interunit Reliability of 10 Hz and 15 Hz GPS Units for Assessing Athlete Movement Demands. Journal of Strength and Conditioning Research. 28(6). Pages 1649-1655. Jones, A.M. and Vanhatalo, A., 2017. The 'Critical Power' Concept: Applications to Sports Performance with a Focus on Intermittent High-Intensity Exercise. *Sports Med.* 47(1). Pages 65-78.

Josefsson, T., Ivarsson, A., Lindwall, M., Gustafsson, H., Stenling, A., Böröy., Mattsson, E., Carnebratt, J., Sevholt, S. and Falkevik, E., 2017. Mindfulness Mechanisms in Sports: Mediating Effects of Rumination and Emotion Regulation on Sport-Specific Coping. *Mindfulness.* 8(5). Pages 1354-1363.

Jubeau, M., Rupp, T., Perrey, S., Temesi, J., Wuyam, B., Levy, P., Verges, S. and Millet, G.Y., 2014. Changes in Voluntary Activation Assessed by Transcranial Magnetic Stimulation during Prolonged Cycling Exercise. *PLoS One.* 9(2).

Kamen, G. and Gabriel, D.A., 2010. *Essentials of Electromyography.* Human Kinetics. USA: Champaign.

Kamijo, K., Nishihira, Y., Higashiura, T. and Kuroiwa, K., 2007. The Interactive Effect of Exercise Intensity and Task Difficulty on Human Cognitive Processing. *International Journal of Psychophysiology.* 65. Pages 114-121.

Kampshoff, C.S., Chinapaw, M.J.M., Brug, J., Twisk, J.W.R., Schep, G., Nijziel, M.R., van Mechelen, W. and Buffart, L.M., 2015. Randomised Controlled Trial of the Effects of High Intensity and Low-to-Moderta Intensity Exercise on Physical Fintness and Fatigue in Cancer Survivors: Results of the Resistance and Endurance Exercise After ChemoTherapy (REACT) Study. *BMC Medicine.* 13. Page 275.

Khanna, A.K. and Tiwary, S.K., 2016. *Ulcers of the Lower Extremity.* Springer. Switzerland: Cham.

Komi, P.V., 200. Stretch-Shortening Cycle: A Powerful Model to Study Normal and Fatigued Muscle. *Journal of Biomechanics.* 33. Pages 1197-1206.

Kozak, M., and Piepho, H.P. 2018. What's normal anyway? Residual plots are more telling than significance tests when checking ANOVA assumptions. *Journal of Agronomy and Crop Science*. 204(1). Pages 86-98.

Lacome, M., Piscione, J., Hager, JP. And Carling, C., 2017. Fluctuations in Running and Skill-Related Performance in Elite Rugby Union Match-Play. *European Journal of Sports Science*. 17(2). Pages 132-143.

Laerd Statistics., 2019. Testing for Normality Using SPSS Statistics. Available from: https://statistics.laerd.com/spss-tutorials/testing-for-normality-using-spss-statistics.php (Accessed on 31 July 2019).

Lamb, G.D., 2002. Excitation-Contraction Coupling and Fatigue Mechanisms in Skeletal Muscle: Studies with Mechanically Skinned Fibres. *Journal of Muscle Research and Cell Motility.* 23(1). Pages 81-91.

Lander, P.J., Butterly, R.J., and Edwards, A.M. 2009., Self-paced Exercise is Less Physically Challenging Than Enforced Constant Pace Exercise of the Same Intensity: Influence of Complex Central Metabolic Control. *British Journal of Sports Medicine*. 43. p789-795.

Lee, M.J.C., Lloyd, D.G., Lay, B.S., Bourke, P.D. and Alderson, J.A., 2017. Different Visual Stimuli Affect Body Reorientation Strategies During Sidestepping. *Scandinavian Journal of Medicine and Science in Sports.* 27(5). Pages 492-500.

Leppänen, M., Pasanen, K., Clarsen, B., Kannus, P., Bahr, P., Parkkari, J., Haapasalo, H. and Vasankari., 2019. Overuse Injuries are Prevalent in Children's Competitive Football: A Prospective Study Using the OSTRC Overuse Injury Questionnaire. *British Journal of Sporte Medicine.* 53(3). Pages 165-171.

Lilienfeld, S.O., Lynn, S.J., Namy, L.L., Woolf, N.J., Jamieson, G., Marks, A. and Slaughter, V., 2015. *Psychology: From Enquiry to Understanding* (Second Edition). Pearson. Australia: Melbourne.

Lix, L.M., Keselman, J.C. and Keselam, H.J., 1996. Consequences of assumption violations revisited: A quantitative review of alternatives to the one-way analysis of variance F test. *Review of Education in Research*. 66(4). Pages 579-619.

Lloyd, R.S., Oliver, J.L., Faigenbaum, A.D., Howard, R., De Ste Croix, M.B., Williams, C.A., Best, T.M., Alvar, B.A., Micheli, L.J., Thomas, D.P., Hatfield, D.L., Cronin, J.B. and Myer, G.D., 2015. Long-Term Athletic Development – Part 1: A Pathway for All Youth. *The Journal of Strength and Conditioning Research.* 29(5). Pages 1439-1450.

Lovell, D., Kerr, A., Wiegand, A., Soloman, C., Harvey, L. and McLellan., 2013. The Contribution of Energy Systems During the Upper Body Wingate Anaerobic Test. *Applied Physiology, Nutrition and Metabolism.* 38(2). Pages 216-219.

Lovell, R. and Abt, G. 2013. Individulisation of Time-Motion Analysis: A Case Cohort Example. International Journal of Sports Physiology and Performance. 8(4). Pages 456-458.

Lovic, D., Narayan, P., Pittaras, A., Faselis, C., Doumas, M. and Kokkinos, P., 2017. Left Ventricular Hypertrophy in Athletes and Hypertensive Patients. *Journal of Clinical Hypertension.* 19(4). Pages 413-417.

Lundberg, T.R. and Wekström, K., 2017. Fixtrue Congestion Modulate Post-Match Recovery Kinetics in Professional Soccer Players. *Research in Sports Medicine*. 25(4). Pages 408-420.

Magstim., 2018. *110mm Double Cone Coil.* Available from: https://www.magstim.com/product/16/110mm-double-cone-coil (Accessed on 12/04/2018).

Malina, R.M., Cumming, S.P., Moran, P.J., Barron, M., and Miller, S.J., 2005. Maturity Status of Youth Football Players: A Non-Invasive Estimate. *Medicine and Science in Sports and Exercise.* 37(6). Pages 1044-1052. Marcoca, S.M., 2008. Do We Really Need a Central Governor to Explain Brain Regulation of Exercise Performance? *European Journal of Applied Physiology.* 104. Pages 929-931.

Mathers, J.F., and Grealy, M.A., 2012. Motor control strategies and the effects of fatigue on golf putting performance. *Frontiers in Psychology.* 13(4). Pages 1005-1022.

Matveev, A.V., 2002. The Advantage of Employing Quantitative and Qualitative Methods in Intercultural Research: Practical Implications from the Study of Perceptions of Intercultural Communication Competence by American and Russian Managers. *Russian Journal of Communication.* 168. Pages 59-67.

Maughan, R., 2009. *The Olympic Textbook of Science in Sport*. Wiley. United Kingdom: Chichester

McArdle, W.D., Katch, F.L. and Katch, V.L., 2009. *Exercise Physiology: Nutrition, Energy and Human Performance* (Seventh Edition). Lippincott Williams and Wilkins. USA: New York.

McArdle, W.D., Katch, F.L. and Katch, V.L., 2010. *Essentials of Exercise Physiology* (Fourth Edition). Lippincott Williams and Wilkins. USA: New York.

McLean, S., 2016. Kerherve, H., Naughton, M., Lovell, G.P., Gorman A.D. and Solomon, C., The Effect of Recovery Duration on Technical Proficiency During Small Sided Games of Football. *Sports.* 4(3). Pages 39-48.

References

Milioni, F., Zagatto, A.M., Barbieri, R.A., Andrade, V.L., Dos Santos, J.W., Gobatto, C.A., da Silva, A.S., Santiago, P.R. anf Papoti, M., 2017. Energy Systems Contribution in the Running-Based Anaerobic Sprint Test. *International Journal of Sports Medicine*. 38(3). Pages 226-232.

Misra, U.K. and Kalita, J., 2010. *Clinical Neurophysiology* (Second Edition). Elsevier. India.

Miura, K., Ishibashi, Y., Tsuda, E., Okamura, Y., Otsuka, H. and Toh, S., 2004. The Effect of Local and General Fatigue on Knee Proprioception. *The Journal of Arthroscopic and Related Surgery.* 20(4). Pages 414-418.

Mohr, M., Krustup, P., ND Bangson, J. 2003. Match Performance of High Standard Soccer Players with Special Reference to Development of Fatigue. *Journal of Sports Science*. 21(7). Pages 219-228.

Nalbandian, H.M., Radak, Z. and Takeda, M., 2017. Effects of Active Recovery During Interval Training on Plasma Catecholamines and Insulin. The Journal of Sports Medicine and Physical Fitness. 58(6). Pages 917-922.

National Instruments., 2018. *Anti-Aliasing Filters and Their Usage Explained.* Available from: http://www.ni.com/white-paper/54448/en/ (Accessed on 12/04/2018). Nédélec, M., McCall, A., Carling, C., Legall, F., Betholin, S. and Dupont, G., 2012. Recovery in Soccer – Part 1: Post-Match Fatigue and Time Course of Recovery. *Sports Medicine*. 42(12). Pages 997-1015.

Nedelec, M., Halson, S., Delecroix, B., Abd-Elbasset, A., Ahmaidi, S., and Dupont, G., 2015. Sleep Hygiene and Recovery Strategies in Elite Soccer Players. *Sports Medicine.* 45(11). Pages 1547-1559.

Nichols Larsen, D.S., Kegelmeyer, D.K., Buford, J.A., Kloos, A.D., Heathcock, J.C. and Basso, D.M., 2015. Neurologic Rehabilitation: Neuroscience and Neuroplasticity in Physical Therapy Practice. USA. McGraw-Hill Education: Ohio.

Nicolella, D.P., Torres-Ronda, L., Saylor, K.J. and Schelling, X., 2018. Validity and Reliability of an Accelerometer Based Player Tracking Device. PLoS One. 13(2).

Nielsen, B., Hyldig, T, Bidstrup, F., Gonzalez-Alonso, J. and Christoffersen, G.R., 2001. Brain Activity and Fatigue During Prolonged Activity in Fatigue. *European Journal of Physiology.* 442. Pages 41-48.

Noakes, T.D. and Marion, F.E. 2009. Maximal Oxygen Uptake is/is not Limited by a Central Nervous System Governor. *Journal of Applied Physiology.* 106(1). Pages 338-339.

Noakes, T., and St Clair Gibson, A. 2004. Logical Limitations to the "Catastrophe" Models of Fatigue During Exercise in Humans. *British Journal of Sports Medicine*. 38(5). Pages 648-649.

Noakes, T.D., St Clair Gibson, A. and Lambert, E.V., 2005. From Catastrophe to Complexity: A Novel Model of Integrative Central Neural Regulation of Effort and Fatigue During Exercise in Humans. *British Journal of Sports Medicine.* 39. Pages 120-124.

Nuechterlein, K.H., Ventura, J., McEwen, S.C., Gretchen-Doorly, D., Vinogradov, S. and Subotnik, K.L., 2016. Enhancing Cognitive Training Through Aerobic Exercise After a First Schizophrenia Episode: Theoretical Conception and Pilot Study. *Schizophrenia Bulletin.* 42(1). Pages 44-52.

Nybo, L., 2003. CNS Fatigue and Prolonged Exercise: Effect of Glucose Supplementation. *Medicine and Science in Sports and Exercise*. 35(4). Pages 589-594.

Nykiel, R.A., 2007. *Handbook of Marketing Research Methodologies for Hospitality and Tourism.* The Haworth Hospitality and Tourism Press. USA: Binghamton.

Nyland, J., Gamble, C., Franklin, T. and Caborn, D.N.M., 2017. Permanent Knee Sensorimotor System Changes Following ACL Injury and Surgery. *Knee Surgery, Sports Traumatology, Arthroscopy.* 25(5). Pages 1461-1474. Oliver, J., Armstrong, N. and Williams, C., 2007. Changes in Jump Performance and Muscle Activity Following Soccer-Specific Exercise. *Journal of Sports Sciences*. 26(2). Pages 141-148.

Ostojic, S.M., 2016. Post-Exercise Recovery: Fundamental and Interventional Physiology. *Frontiers in Physiology.* 7(3).

Otani, H., Kaya, M., Tamaki, A. and Watson, P., 2017. Separate and Combined Effects of Exposure to Heat Stress and Mental Fatigue on Endurance Capacity in the Heat. *European Journal of Applied Physiology.* 117(1). Pages 119-129.

OxfordDictionary.2019.Experiment.Availablefrom:https://en.oxforddictionaries.com/definition/experiment (Accessed on 09/01/2019)

Ozimek, M., Szmatlan-Gabrys, U., Gabrys, T., Stanula, A., Stanisz, L., Graback-Pietruszka., Majka, J. and Eliasz-Radzikowski, W. 2017. Control in Competitive Sport as Exemplified by a Women Basketball Team in the Polish First League. *Studia Sportiva.* 11(1). Pages 37-43.

Pageaux, B., 2014. The Psychobiological Model of Endurance Performance: An Effort-Based Decision-Making Theory to Explain Self-Paced Endurance Performance. *Sports Medicine.* 44(9). Pages 1319-1320.

Palekar, S., 2011. *Clinical Diagnosis for Medical Undergraduates.* Popular Prashakan Ltd. India: Bombay.

References

Palmieri, R.M., Ingersoll, C.D. and Hoffman, M.A., 2004. The Hoffman Reflex: Methodologic Considerations and Applications for Use in Sports Medicine and Athletic Training Research. *Journal of Athletic Training.* 39(3). Pages 268-277.

Pearcey, G.E.P., Bradbury-Squires, D.J., Kawamoto, J.E., Drinkwater, E.J., Behm., D.G. and Button. D.C., 2015. Foam Rolling for Delayed-Onset Muscle Soreness and Recovery of Dynamic Performance Measures. *Journal of Athletic Training.* 50(1). Pages 5-13.

Piil, J.F., Lundbye-Jensen, J., Trangmar, S.J. and Nybo, L., 2017. Performance in Complex Motor Tasks Deteriorates in Hyperthermic Humans. *Temperature.* 4(4). Pages 420-428.

Playertek., 2017. So You Think You're a Pro? Playertek Data Uncovers the Real Difference Between Amateur and Professional Footballers. Available from: https://www.playertek.com/blog/playertek-data-amateur-professional-footballers/ (Accessed on 17th July 2019)

Plowman, S.A. and Smith, D.L., 2010. *Exercise Physiology for Health, Fitness and Performance* (Third Edition). Lippincott Williams and Wilkins. USA: Philadelphia.

Premier League., 2019. *Long-Term Strategy Designed to Advance Premier League Youth Development.* Available from: https://www.premierleague.com/youth/EPPP (Accessed on: 17th July 2019). Proske, U., 2005. What is the Role of Muscle Receptors in Proprioception? *Muscle Nerve.* 31. Pages 780-787.

Proske, U. and Gandevia, S.C., 2012. The Proprioceptive Senses: Their Roles in Signalling Body Shape, Body Position and Movement, and Muscle Force. *Physiological Reviews.* 92(4). Pages 1651-1697.

Raeder, C., Wiewelhove, T., Schneider, C., Doweling, A., Kellman, M., Meyer, T., Pfeiffer, M. and Ferrauti, A. 2017. Effects of Active Recovery on Muscle Function Following High-Intensity Training Sessions in Elite Olympic Weightlifters. Advances in Skeletal Muscle Function Assessment. 1(1). Pages 3-12.

Rao, S.D., 2015. Clinical Manual of Surgery. Elsevier. India: New Delhi

Rago, V., Pizzuto, F. and Raiola, G., 2017. Relationship Between Intermittent Endurance Capacity and Match Performance According to the Playing Position in Sub-19 Professional Male Football Players: Preliminary Results. *Journal of Physical Education and Sport.* 17(2). Pages 688-691.

Rahnama, N., Reilly, T. and Lees, A. 2002. Injury Risk Associated with Playing Actions During Competitive Soccer. *British Journal of Sports Medicine*. 36. Pages 354-359. Rakhmetov, A.D., Lee, S.P., Ostapchenko, L.I. and Chae, H.Z., 2015. Analysis of Creatine Kinase Activity with Evaluation of Protein Expression Under the Effect of Heat and Hydrogen Peroxide. *The Ukrainian Biochemical Journal.* 87(1). Pages 75-82.

Rampinini, E., Bosio, A., Ferraresi, I., Petruolo, A., Morelli, A., and Sassi, A. 2011. Match-Related Fatigue in Soccer Players. *Medicine and Science in Sport and Exercise*. 43(11). Pages 2161-2170.

Ratel, S. and Blazevitch, A.J., 2017. Are Pre-Pubertal Children Metabolically Comparable to Well-Trained Adult Endurance Athletes. *Sports Medicine*. 47(8). Pages 1477-1485.

Reid, M. and Duffield, R. 2014. The Development of Fatigue During Match-Play Tennis. *British Journal of Sports Medicine*. 48(1) Pages 7-11.

Rio-Rodriquez, D., Inglesias-Soler, E. and del Olmo, M.F., 2016. Set Configuration in Resistance Exercise: Muscle Fatigue and Cardiovascular Effects. *PLoS One.* 11(3).

Robertson, G., Caldwell, G., Hamill, J., Kamen, G. and Whittlesey, S., 2013. Research Methods in Biomechanics (Second Edition). Human Kinetics. USA: Champaign.

Rogers, K., 2011. The Respiratory System. Britannica Publishing. USA: New York.

Ronnestad, B.R., Nymark, B.S. and Raastad, T., 2011. Effects of In-Season Maintenance Training Frequency in Professional Soccer Players. *The Journal of Strength and Conditioning Research.* 25(10). Pages 2653-2660.

Ross, H.E. and Bischof, K., 1981. Wundt's Views on Sensations of Innervation: A Re-Evaluation. *Perception.* 10. Pages 319-329.

Rowell, A.E., Aughey, R.J., Hopkins, W.G., Stewart, A.M. and Cormak, S.J., 2017. Identification of Sensitive Measures of Recovery After External Load from Football Match Play. *International Journal of Sports Physiology and Performance*. 12(7). Pages 969-976.

Rowland, T., Unnithan, V., Fernhall, B., Baynard, T. and Lange, C., 2002. Left Ventricular Response to Dynamic Exercise in Young Cyclists. *Medicine and Science in Sports and Exercise.* 34(4). Pages 637-642.

Ryan, D., Lewin, C. Forsythe, S., 2018. Developing World-Class Soccer Players: An Example of the Academy Physical Development from an English Premier League Team. *Strength and Conditioning Journal.* 40(3). Pages 2-11.

Saward, C., Morris, J.G., Nevill, M.E., Nevill, A.M., and Sunderland, C. 2016. Longitudinal Development of Match-Running Performance in Elite Male Youth Soccer Players. *Scandinavian Journal of Medicine and Science in Sports*. 26(8). Pages 933-942. Sell, T.C., Abt, J.P., and Lephart, S.M. 2008. Physical Activity-Related Benefits of Walking During Golf. *Science and Golf V: Proceedings of the World Scientific Congress of Golf.* Pages 128-132.

Sharkey, B.J. and Gaskill, S.E., 2013. *Fitness Cycling.* Human Kinetics. United Kingdom: Leeds.

Sherwood, L., 2011. *Fundamentals of Human Physiology.* (Fourth Edition). Cengage Learning. USA: Boston.

SoccerStats. 2018. England – Premier League. Available from: http://www.soccerstats.com/timing.asp?league=england (Accessed on 31 January 2018).

Sporis, G., Jukic, J., Ostojic, S.M. and Milanovic, D., 2009. Fitness Profiling in Soccer: Physical and Physiological Characteristics of Elite Player. *Journal of Strength and Conditioning Research.* 23(7). Pages 1947-1953.

St Clair Gibson, A., Swart, J. and Tucker, R., 2017. The Interaction of Psychological and Physiological Homeostatic Drives and Role of General Control Principles in the Regulation of Physiological Systems, Exercise and the Fatigue Process – The Interactive Governor Theory. *European Journal of Sport Science*. 18(1). Pages 25-36.

Stebler, K., Martin, R., Kirkham, K.R., Küntzer, T., Bathory, I. and Albrecht. E., 2017. Electrophysiological Study of Femoral Nerve Function After a Continuous Femoral Nerve Block for Anterior Cruciate Ligament Reconstruction: A Randomized, Controlled Single-Blind Trial. *The American Journal of Sports Medicine*. 45(3). Pages 578-583.

Taylor, S.E., 2010. Mechanisms Linking Early Life Stress to Adult Health Outcomes. *Proceedings of the National Academy of Sciences Of the United Sates of America.* 107(19). Pages 8507-8512.

Taylor, J.L. and Gandevia, S.C., 2001. Transcranial Magnetic Stimulation and Human Muscle Fatigue. *Muscle & Nerve.* 24(1). Pages 18-29.

Thatcher, R. and Curtis, F., 2016. Agreement Between Session RPE and Heart Rate Derived TRIMP Across a Range of Running Speeds. *Gazetta Medica Italiana*. 175(7). Pages 308-3123.

The Football Association. 2018. *The Future England Player*. Available from: https://community.thefa.com/england\_dna/p/future\_england\_player (Accessed on 07/02/2018).

Thomas, K., Dent, J., Howatson, G., and Goodall, S. 2017. Etiology and Recovery of Neuromuscular Fatigue Following Simulated Soccer Match-Play. *Medicine and Science in Sports and Exercise.* 49 (5). Pages 955-964.

Thomas, K., Goodall, S., Stone, M., Howatson, G., St Clair Gibson, A., and Ansley, L. 2015. Central and Peripheral Fatigue in Male Cyclists after 4-, 20- and 40-km Time Trials. *Medicine and Science in Sports and Exercise*. 47(3). Pages 537-546.

References

Thorpe, R.T., Strudwick, A.J., Buchheit, M., Atkinson, G., Drust, B. and Gregson, W., 2016. Tracking Morning Fatigue Status Across in-Season Training Weeks in Elite Soccer Players. *International Journal of Sports Physiology and Performance.* 11(7). Pages 947-952.

Thiriet, M., 2011. *Cell and Tissue Organization in the Circulatory and Ventilatory Systems.* Springer-Verlag. USA: New York.

Todd, G., Butler, G.E., Taylor, J.L. and Gandevia, S.C., 2005. Hyperthermia: A Failure of the Motor Cortex and the Muscle. Journal of Physiology. 563. Pages 621-631.

Tomlin, D.L. and Wenger, H.A., 2001. The Relationship Between Aerobic Fitness and Recovery from High Intensity Intermittent Exercise. *Sports Medicine.* 31(1). Pages 1-11.

Tonson, A., Ratel, S., Le Fur, Y., Vilmen, C., Cozzone, P.J. and Bendahan, D., 2010. Muscle Energetics Changes Throughout Maturation: A Quantitative 31P-MRS Analysis. *Journal of Applied Physiology.* 109(6). Pages 1769-1778.

Tracey, C., Leigh, W., John, P. and Christopher, P., 2007. Effects of a Maximal Exercise Test on Neurocognitive Function. *Journal of Sports Medicine*. 41. Pages 370-374.

Trimmel, K., Bacha, J. and Huikuri, H.V., 2017. *Heart Rate Variability: Clinical Applications and Interaction Between HRV and Heart Rate.* Frontiers. Switzerland: Lausanne.

Tsui, B.C.H., 2008. Atlas of Ultrasound- and Nerve Simulation-Guided Regional Anaesthesia. Springer. Switzerland: Cham.

Varley, M.C., Di Salvio, V., Modonutti, M., Gregson, W. and Mendez-Villanueva., 2017. The Influence of Successive Matches on Match-Running Performance During an Under-23 International Soccer Tournament: The Necessity of Individual Analysis. *Journal of Sports Sciences.* 36(5). Pages 585-591.

Vincent, J.L., 2008. Understanding Cardiac Output. Critical Care. 12(4). Page 174.

Waldron, M., and Highton, J. 2014. Fatigue and Pacing in High-Intensity Intermittent Team Sport. *Sports Med.* 44. Pages 1645-1658.

Waldron, M., Thomson, E., Highton, J. and Twist, C., 2017. Transient Fatigue is not Influenced by Ball-in-Play Time During Elite Rugby League Matches. *Journal of Strength and Conditioning Research.* 33(1). Pages 146-151.

Ward, J.P.T,. Ward, J. and Leach, R.M., *The Respiratory System: At a Glance* (Fourth Edition). Wiley. United Kingdom: Chichester.

Yokoi, Y., Yanagihashi, R., Morishita, K. and Fujiwara, T., 2015. Repeated Recovery Effects of Exposure to Normobaric Hyperoxia: Blood Lactate Levels and Tissue Oxygenation in Local Muscle Fatigue. *Physiotherapy Journal*. 101(1). Pages 1697-1721.

Young, J., Angevaren, M., Rusted, J. and Tabet, N., 2015. Aerobic Exercise to Improve Cognitive Function in Older People Without Known Cognitive Impairment. *The Cochrane Database of Systematic Reviews.* 22(4). Appendices

## **Appendix A: Informed Consent Form**



# The Effects of Microcycle Planning on the Time Course of Recovery in Central and Peripheral Fatigue in Elite Youth Male Footballers Participant Consent Form

Please tick the appropriate boxes

I have read and understood the project information sheet
I have been given the opportunity to ask questions about the project
I agree to take part in the project. Taking part in the project will include partaking in Transcranial Magnetic Stimulation and Peripheral stimulation of the femoral nerve during 50%, 75% and 100% of maximal voluntary contractions. These will be completed over twelve separate testing occasions over two separate weekly periods
I understand that my taking part is voluntary; I can withdraw from the study at any time up until 1 <sup>st</sup> April 2018 and I will not be asked questions about why I no longer want to take part
Select only one of the next two options:
I would like my name used where I have said or written as part of this study will be used in reports, publications and other research outputs so that anything I have contributed to this project can be recognised
I do not want my name used in this project

#### Appendices

I understand my personal details such as phone number or address will not be revealed to people outside of this project	
I understand that my data may be quoted in publications, reports, web pages, and other research outputs but my name will not be used unless I requested it above	
I agree for the data I provided to be archived and made available to other researchers on request	
I understand that other researchers will have access to these data only if they agree to prese the confidentiality of these data	rve

I understand that other researchers may use my data in publications, reports, web pages and other research outputs.....

#### **Transcranial Magnetic Stimulation Contraindications Checklist:**

Please read the following statements. If you are able to answer 'Yes' to any, you may be at higher risk for the study and will need to discuss your participation with the lead researcher.

- 1. Do you have epilepsy or have you ever had a convulsion or a seizure?
- 2. Have you ever had a fainting spell or syncope?
- 3. Have you ever had severe (i.e., followed by loss of consciousness) head trauma?
- 4. Do you have any hearing problems or ringing in your ears?
- 5. Do you have metal in the brain/skull (except titanium)? (e.g., splinters, fragments, clips, etc.)
- 6. Do you have cochlear implants?
- 7. Do you have an implanted neurostimulator? (e.g., DBS, epidural/subdural, VNS)
- 8. Do you have a cardiac pacemaker or intracardiac lines or metal in your body?
- 9. Do you have a medication infusion device?
- 10. Are you taking any medications? (Please list)
- 11. Did you ever have a surgical procedure to your spinal cord?
- 12. Do you have spinal or ventricular derivations?
- 13. Did you ever undergo TMS in the past?
- 14. Did you ever undergo MRI in the past?

Select only one of the next two options:

I answered 'Yes' to one or more of the above statements. I will therefore need to discus	SS
my participation in this study with the lead researcher	
I answered 'No' to all of the above statements	

On this basis I am happy to participate in the "The Effects of Microcycle Planning on the Time Course of Recovery in Central and Peripheral Fatigue in Elite Youth Male Footballers" study.

Name of Participant	Signature Date
Name of Parent	Signature Date
Name of Researcher	Signature Date

If you have any queries or concerns, please contact Callum Sharpin on any of the following formats: Email: callum.sharpin@watfordfc.com Email: callum.sharpin@bucks.ac.uk Telephone: 07896061823 Email: mark.stone@bucks.ac.uk

One copy to be kept by the participant, one to be kept by the researcher

# **Appendix B: Physical Activity Readiness Questionnaire**



# The Effects of Microcycle Planning on the Time Course of Recovery in Central and Peripheral Fatigue in Elite Youth Male Footballers PAR-Q Form

Please answer the following questions

- 1. Has your doctor ever said that you have a bone or joint problem, such as arthritis that has been aggravated by exercise or might be made worse with exercise? YES / NO
- 2. Do you have high blood pressure? YES / NO
- 3. Do you have low blood pressure? YES / NO
- 4. Do you have Diabetes Mellitus or any other metabolic disease? YES / NO
- Has your doctor ever said you have raised cholesterol (serum level above 6.2mmol/L)? YES / NO
- 6. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor? YES / NO
- 7. Have you ever felt pain in your chest when you do physical exercise? YES / NO
- 8. Is your doctor currently prescribing you drugs or medication? YES / NO
- Have you ever suffered from unusual shortness of breath at rest or with mild exertion? YES / NO
- 10. Is there any history of Coronary Heart Disease in your family? YES / NO
- 11. Do you often feel faint, have spells of severe dizziness or have lost consciousness? YES / NO
- 12. Do you currently drink more than the average amount of alcohol per week (21 units for men and 14 units for women)? YES / NO
- 13. Do you currently smoke? YES / NO
- 14. Do you know of any other reason why you should not participate in the study? YES / NO

If you answered YES to any of the questions above please give details:

.....

If you answered YES to one or more questions: If you have not recently done so, consult with your doctor by telephone or in person partaking in the study. Tell your doctor what questions you answered 'yes' to on PAR-Q or present your PAR-Q copy. After medical evaluation, seek advice from your doctor as to your suitability for participation.

If you answered NO to all questions: If you answered PAR-Q accurately, you have reasonable assurance of your present suitability for participation in the study.

On this basis, I am happy to participate in the "The Effects of Microcycle Planning on the Time Course of Recovery in Central and Peripheral Fatigue in Elite Youth Male Footballers" study.

Name of Participant	Signature Date
Name of Parent	Signature Date
Name of Researcher	Signature Date

If you have any queries or concerns, please contact Callum Sharpin on any of the following formats: Email: callum.sharpin@watfordfc.com Email: callum.sharpin@bucks.ac.uk Telephone: 07896061823 Email: mark.stone@bucks.ac.uk

One copy to be kept by the participant, one to be kept by the researcher

## **Appendix C: Ethical Approval**

Dear Callum

#### Ethical approval: Ref UEP2017May02

I am writing to confirm that ethical approval was granted by the University Research Ethics Panel of Buckinghamshire New University on 14 June 2017 for your project:

"The Effects of Microcycle Planning on the Time Course of Recovery in Central and Peripheral Fatigue in Elite Youth Male Footballers"

This approval is valid for data collection between 10 July and 10 November 2017.

Please ensure that you quote the above reference number as evidence of ethical approval and in all materials used to recruit participants.

The Research and Enterprise Development Unit must be notified of any amendments to the proposed research or any extension to the period of data collection.

I hope that your research project goes well.

Yours sincerely,

Monch.

Dr M. Nakisa

Secretary to the University Research Ethics Panel Research and Enterprise Development Unit

Dear Callum

#### Ref UEP2017May02 – Extension to data collection period

I am writing to confirm that on 24 November 2017 the University Research Ethics Panel of Buckinghamshire New University approved your request to extend data collection for your project:

"The Effects of Microcycle Planning on the Time Course of Recovery in Central and Peripheral Fatigue in Elite Youth Male Footballers"

This approval is valid for data collection between 24 November 2017 and 23 March 2018.

Please ensure that you quote the above reference number as evidence of ethical approval and in all materials used to recruit participants.

The Research and Enterprise Development Unit must be notified of any amendments to the proposed research or any extension to the period of data collection.

I hope that your research project goes well.

Yours sincerely,

Mart.

Dr M. Nakisa

Secretary to the University Research Ethics Panel Research and Enterprise Development Unit

# **Appendix D: Incremental Treadmill Testing Protocol**

### Aim of Test

- To ascertain players Maximum Aerobic Speed (MAS)
- To ascertain players maximum heart rate

#### Equipment required

- Treadmill
- Stopwatch
- Blood testing equipment
  - Ear prickers
  - Collection tubes
  - Sharps bin
  - Tissues
- Recording sheet

#### Location/Surface

- Gym

#### **Procedures**

- All player must complete the test in trainers
- Introduction to the procedure and test purpose.
- Set up the treadmill
  - Set to 1% incline
- Collect an ear blood sample prior to the warm up

- Perform a warm-up. Player will complete 6 minutes at 7km/h
- Player comes off the treadmill. Collect an ear blood sample.
- The player then completes 3 minutes at each of the following levels, coming off the treadmill after each level for an earlobe blood sample: 9km/h, 11km/h, 13km/h, 15km/h, 17km/h.
- Player returns to treadmill after final blood sample and completes one minute at each of the following levels without leaving the treadmill: 18km/h, 19km/h, 20km/h, 21km/h, 22km/h, 23km/h, 24km/h, 25km/h.
  - End heart rate is collected and the level which the player managed to complete the full minute.

# Appendix E: 30m Sprint Test Protocol

### Aim of Test

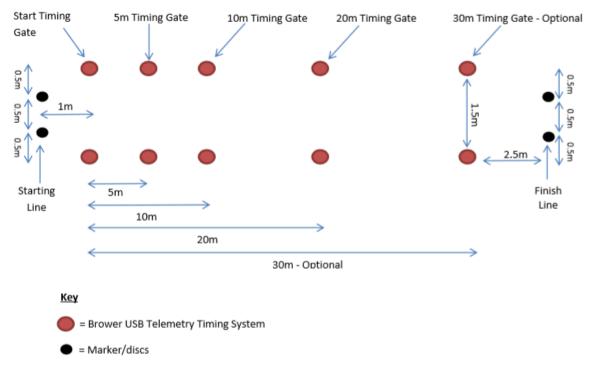
- To ascertain players ability to accelerate
- To ascertain players speed over various distances

### Equipment required

- Brower USB Telemetry Timing System
  - 5 gates
  - Gate Height 95cm
- Measuring tape
- Marker/discs
- Recording sheet
- GPS/Heart rate optional

## Location/Surface

- Indoor artificial surface



#### Layout - (not to scale)

#### **Procedures**

- All player must complete the sprints in their match/training boots
- Introduction to the procedure and test purpose.
- Demonstration of the test Ensure perfect technique and explanation of key technical components.
- Breakdown the specific requirements of the sprints and the importance of adherence to them.
- Perform a specific group warm-up: This should be a maximum of 10 minutes and the contents should be dictated by the club staff based upon the understanding of the player's needs.
- Players are called over to the sprint test area in a pre-selected order (e.g alphabetical, age, position).
- The player stands with their preferred foot forward with toes behind the starting line (1m back from starting timing gate).

Appendices

- The player gets into a 2 point athletic starting stance Body position this will not be coached by the FTO's, they will only monitor players backward/forward swaying.
- Players start accelerating in their own time from the starting line and are instructed not to stop sprinting until they pass the finishing line (2.5m past final timing gate). It is essential that the player does not sway or move back then forward when initiating the acceleration from the standing start.
- Once completed the player recovers and the next player is called to complete the sprint.

## Notes

- All players must complete a total of 3 valid sprints
- If any of the technical points are not adhered to then the sprint is void and the player should complete another attempt. To prevent potential injury risk the total amount of sprints that each player completes must not exceed 4.
- All players must have a minimum of 2 minutes rest between each valid sprint.
- All 3 valid sprints should be recorded for 5m, 10m, 20m and 30m (optional) to the nearest 100th/sec with the best sprint for each distance inputted within the final results.
- 5m, 10m and 20m distances are all mandatory for collection however, clubs can choose if they wish to continue to collect data for 30m

# Appendix F: Standardised Breakfast Menu

## Breakfast Menu

## 8:30 am

Healthy cereals (Weetabix, Healthy Granola Type Cereal)

Semi skimmed milk and soya milk

## Toaster

Sliced White and Granary Bread

Jams - Strawberry, Raspberry, Marmite, Honey, Low Fat butter

## Cold Buffet

Fruit basket (oranges, bananas, apples, pears)

Hot Buffet

Porridge (Soya Milk/Dairy Milk)

Baked Beans, Scrambled Eggs

## <u>Drinks</u>

Tea and Coffee

Still mineral water

Apple juice, Fresh Orange Juice, Cranberry Juice

# Appendix G: Aerobic Training Week – Monday (MD+2) Session Plan

## <u>Warm up – 15 minutes</u>

Hip mobility hurdles x10 Circle with stretches (all dynamic) Small hurdles (plyometrics with sprints x10)

#### Passing Drill

4 x 3 minutes 30 seconds recovery 20m x 30m

### Possession – Group 1

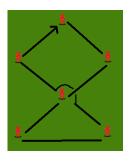
4 x 3 minutes 1 minute recovery Points for passing through the gate 25m x 20m 5 players v 5 players

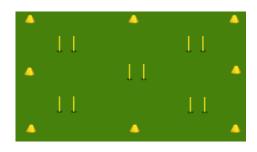
#### Possession – Group 2

6 x 2 minutes
30 seconds recovery
6 players v 3 players
Each player is in the middle for 2 blocks
Small Sided Game
3 x 6 minutes
90 seconds recovery
8 players v 8 players

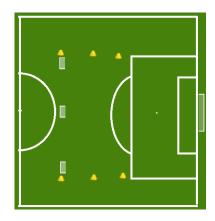
Players to attack both big and small goals in games











# Appendix H: Aerobic Training Week – Tuesday (MD+3) Session

# Plan

### Warm up – 18 minutes

Cross pulse raiser, with dynamic stretches, followed by change of direction sprints to poles

Small hurdles (plyometrics with sprints x6)

#### Passing Drill

4 x 3 minutes 30 seconds recovery 20m x 30m Variation in passing patterns

#### Wave Game

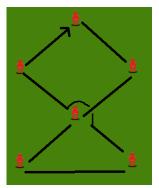
20 minutes Passing and moving through poles 3 defenders to prevent crosses / goals

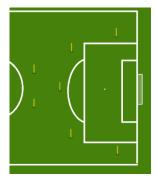
#### Small Sided Game

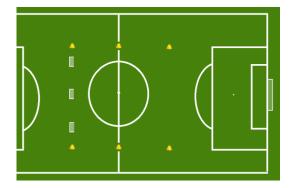
- 2 x 7.5 minutes
- 90 seconds recovery
- 8 players v 8 players

Players to attack both big and small goals in games









# Appendix I: Aerobic Training Week – Thursday (MD+5) Session

# Plan

### <u>Warm up – 15 minutes</u>

Pulse raiser through small hurdles with passing drill, followed by hip mobility through smart hurdles

Circle with dynamic stretches

Sprints / plyo through small hurdles and smart hurdles



#### Individual work

#### 10 minutes

Allows players to work on individual weaknesses such as finishing, heading, long rang passing etc

# Appendix J: Aerobic Training Week – Friday (MD+6) Session Plan

## <u>Warm up – 12 minutes</u>

Pulse raiser and dynamic stretches through channels

Linear sprints (5 / 10m) – reacting to stimulus such as ball dropping etc

#### **Possession**

3 x 4 minutes

Transfer game, 3 teams. Once they lose the ball or can't transfer to other team, they go into the middle.

1 minute rest

#### <u>Game</u>

3 teams

12 minutes

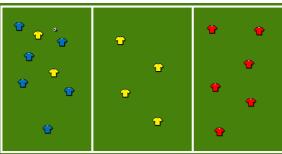
Once a team concedes, they are off and go on the outside

Maximum 2 minute games

#### Set pieces

12 minutes

Players to practice set pieces before following days game.











# Appendix K: Anaerobic Training Week – Monday (MD+2) Session

# Plan

## <u>Warm up – 12 minutes</u>

Hip mobility hurdles x10 Circle with stretches (all dynamic) Small hurdles (plyometrics with sprints x10)



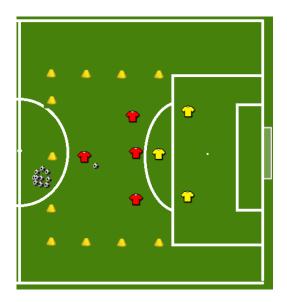
## Passing Drill - 12 minutes

Variation in passing patterns Players finish in a small goal 30m x 40m



## Attacking and Defending Practice – 30 minutes

4 attackers v 3 defenders 25m x 20m Points awarded for goals scored or stopped Game was stopped regularly for coaching points



# Appendix L: Anaerobic Training Week – Tuesday (MD+3) Session

# Plan

#### <u>Warm up – 15 minutes</u>

Passing pulse raiser in the shape of a cross, with dynamic stretches

28m x 28m

Z / N drills x6

#### Transfer game

6 x 90 seconds

30 seconds recovery

10m x 25m

Each team works for 90 seconds with 2 players working

to close down the ball and 2 players resting. Points awarded for a transfer or winning the ball.

#### **Possession**

2 x 4 minutes

30m x 30m

Points awarded for completing five passes

#### Running Drill

3 x 35 seconds work, 70 seconds recovery

#### **Possession Game**

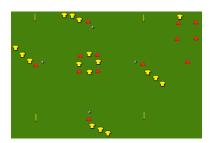
3 teams: 2 teams keep the ball, one team tries to win it back and score in the goal

6 x 90 seconds

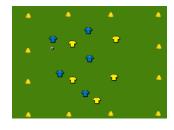
30 seconds recovery

30m x 40m

Points awarded for goals scored by defending team











# Small Sided Game

- 1 x 8 minutes
- 4 players v 4 players
- 30m v 40m
- 2 players from the third team by each goal as bounce players
- If you concede you come off and next team come on



# Appendix M: Anaerobic Training Week – Thursday (MD+5) Session

# Plan

## <u>Warm up – 15 minutes</u>

Pulse raiser through small hurdles Circle with dynamic stretches 3 change of direction drills

## Passing Drill

15 minutes 60m x 50m Variation in passing patterns Stopped regularly to be coached

#### Pattern of Play

12 minutes 60m x 50m Passing drill repeated with defenders

## Small sided game

8 v 6

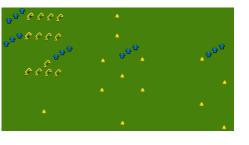
2 x 8 minutes

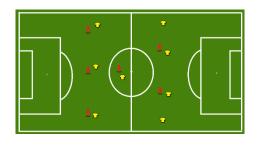
Team defending the big goal have to press and the team defending the small goals have to shuffle and slide across.

## Individual work

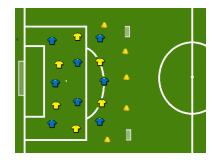
10 minutes

Allows players to work on individual weaknesses such as finishing, heading, long rang passing etc









# Appendix N: Anaerobic Training Week – Friday (MD+6) Session

# Plan

### <u>Warm up – 12 minutes</u>

Pulse raiser and dynamic stretches through channels

Reactive agility (10m) – reacting to coach calling out each colour then a sprint on the coach's clap

#### **Possession**

6 x 1 minute

2 teas of 8 players, each player numbered 1-8. Each rep, two numbers are called and they try to win the ball from the opposition team as many times in a minute. Points are scored for winning the ball.

30 seconds rest

#### Pattern of play – 15 minutes

Stopped and coached over the period – tactical work for the following day's game.

#### <u>Game</u>

3 teams

6 x 3 minutes

Each team plays 2 games and has one game on the outside

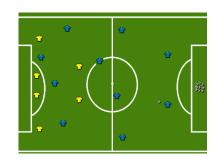
#### Set pieces

10 minutes

Players to practice set pieces before following days game.











# **Appendix O: Rating of Perceived Exertion Scale**



#### Rate of Percieved Exertion (RPE)

	0	Rest
1946	1	Very, Very Easy
	2	Easy/Light
	3	Light
	4	
	5	Moderate
	6	
	7	Hard
	8	Very Hard
	9	
	10	Maximal

This is how hard YOU found the session out of 10!!! Not how hard you think it was supposed to be, it is how hard you feel you worked!!!

# Appendix P: Raw Mean Data

	Aer	obic Training We	eek	Anaerobic Training Week			
Testing Date	п	М	SD	n	М	SD	
Pre-Match	10	574.40	109.72	10	615.10	133.94	
Post-Match	10	528.00	123.41	10	552.20	150.52	
Monday	10	608.60	199.68	10	648.70	180.58	
Tuesday	10	625.50	152.01	10	701.60	158.54	
Thursday	10	659.40	134.57	10	678.40	136.91	
Friday	10	702.10	136.13	10	685.60	171.96	

#### **Maximal Voluntary Contraction**

Appendix N(1) – MVC scores for TW-A and TW-AN across the six testing dates

#### TMS Deficit

	Aerobic Training Week			Anaerobic Training Week			
Testing	n	М	SD	n	М	SD	
Date							
Pre-Match	10	25.84	20.13	10	25.02	13.85	
Post-Match	10	38.46	19.86	10	38.64	21.00	
Monday	10	25.57	20.32	10	37.55	25.96	
Tuesday	10	35.92	32.31	10	25.61	14.23	
Thursday	10	26.08	26.69	10	22.27	13.13	
Friday	10	24.15	16.90	10	18.57	21.00	

Appendix N(2) – TMS deficit scores for TW-A and TW-AN across the six testing dates

#### <u>TMS %VA</u>

	Aeı	robic Training W	/eek	Anaerobic Training Week				
Testing	n	М	SD	N	М	SD		
Date								
Pre-Match	10	95.09	3.87	10	95.48	2.63		
Post-Match	10	92.44	4.30	10	92.57	4.15		
Monday	10	95.70	2.87	10	94.35	3.25		
Tuesday	10	93.43	6.18	10	95.85	2.66		
Thursday	10	95.94	3.73	10	96.66	2.19		
Friday	10	96.10	2.50	10	96.26	5.30		
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Appendix N(3) – TMS %VA scores for TW-A and TW-AN across the six testing dates

## PS Deficit

	Aeı	Aerobic Training Week			Anaerobic Training Week			
Testing	n	М	SD	N	М	SD		
Date								
Pre-Match	10	13.84	15.86	10	24.04	25.74		
Post-Match	10	15.88	13.33	10	21.09	14.38		
Monday	10	17.46	23.20	10	30.12	23.62		
Tuesday	10	11.99	13.24	10	11.76	14.77		
Thursday	10	12.39	8.70	10	15.26	12.64		
Friday	10	13.32	11.97	10	9.49	5.61		

Appendix N(4) – PS deficit scores for TW-A and TW-AN across the six testing dates

# <u>PS %VA</u>

	Aeı	robic Training W	/eek	Anaerobic Training Week		
Testing	n	М	SD	п	М	SD
Date						
Pre-Match	10	97.52	2.96	10	94.84	6.56
Post-Match	10	96.63	3.33	10	95.59	3.32
Monday	10	96.68	4.52	10	95.15	4.15
Tuesday	10	97.71	2.47	10	98.33	2.05
Thursday	10	97.97	1.21	10	97.56	1.73
Friday	10	97.86	1.84	10	98.47	1.01

Appendix N(5) – PS %VA scores for TW-A and TW-AN across the six testing dates

## Total Distance

	Ae	Aerobic Training Week			Anaerobic Training Week		
Testing Date	п	М	SD	п	М	SD	
Match	10	9710.00	2158.64	10	8696.30	3136.25	
Monday	10	5764.20	252.02	10	4420.40	456.82	
Tuesday	10	5499.60	667.63	10	5431.80	462.38	
Thursday	10	5472.00	414.18	10	5376.90	588.19	
Friday	10	3447.20	358.33	10	4314.00	392.76	

Appendix N(6) – Total Distance scores for TW-A and TW-AN across the testing dates

# <u>HSR</u>

	Ae	Aerobic Training Week			Anaerobic Training Week			
Testing Date	n	М	SD	п	М	SD		
Match	10	1915.40	766.07	10	1985.40	874.02		
Monday	10	304.70	154.47	10	536.40	252.56		
Tuesday	10	475.60	238.73	10	719.50	286.91		
Thursday	10	1040.20	275.93	10	713.20	256.84		
Friday	10	291.90	91.80	10	389.60	99.67		
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Appendix N(7) – HSR Distance scores for TW-A and TW-AN across the testing dates

## Player load

-	Ae	robic Training V	Veek	Anaerobic Training Week		
Testing	n	М	SD	п	М	SD
<i>Date</i> Match	10	952.65	227.91	10	886.97	316.70
Monday	10	651.62	81.20	10	503.73	59.31
Tuesday	10	650.91	111.67	10	586.67	41.67
Thursday	10	606.71	37.47	10	593.09	79.27
Friday	10	394.65	42.78	10	470.83	61.48

Appendix N(8) – Player Load scores for TW-A and TW-AN across the testing dates

# <u>RPE</u>

Aer	obic Training W	/eek	Anaerobic Training Week		
п	М	SD	п	М	SD
10	8.40	0.52	10	8.15	0.75
10	4.70	0.67	10	5.30	0.95
10	6.20	0.63	10	7.30	1.16
10	7.40	0.70	10	5.85	0.75
10	4.70	0.67	10	5.25	0.63
	n 10 10 10 10	n M 10 8.40 10 4.70 10 6.20 10 7.40	108.400.52104.700.67106.200.63107.400.70	n         M         SD         n           10         8.40         0.52         10           10         4.70         0.67         10           10         6.20         0.63         10           10         7.40         0.70         10	n         M         SD         n         M           10         8.40         0.52         10         8.15           10         4.70         0.67         10         5.30           10         6.20         0.63         10         7.30           10         7.40         0.70         10         5.85

Appendix N(9) – RPE scores for TW-A and TW-AN across the testing dates

# <u>RPE Trimp</u>

	Ae	robic Training W	/eek	Anaerobic Training Week		
Testing	n	М	SD	n	М	SD
Date						
Match	10	699.20	172.02	10	617.90	231.20
Monday	10	351.50	51.05	10	364.90	50.58
Tuesday	10	528.20	59.50	10	631.40	95.47
Thursday	10	596.10	53.37	10	450.95	62.22
Friday	10	299.10	39.78	10	338.20	43.20
	1 1/		( <b>T</b> ) ( )		NI (I (	

Appendix N(10) – Load (RPE Trimp) scores for TW-A and TW-AN across the testing dates

# Blood Lactate

	Aer	obic Training W	/eek	Anaerobic Training Week		
Testing	n	М	SD	n	М	SD
Date						
Pre-Match	10	1.15	0.63	10	1.15	0.31
Post-Match	10	3.63	1.38	10	4.02	0.84

Appendix N(11) – Blood Lactate scores for TW-A and TW-AN from pre-match to post-match