Neuromuscular, endocrine and mood response to a single elite rugby union match.

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Neuromuscular, Endocrine and Mood Response to a single Elite Rugby Union Match

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Abstract

The recovery time-course of neuromuscular performance, hormone function, and mood disturbance was examined for 60 hours (h) following a competitive match in a group of professional rugby union players. Saliva samples (testosterone, cortisol and testosterone-cortisol [T/C] ratio), countermovement jumps (peak power output [PPO]), and mood (Brief Assessment of Mood Questionnaire) were collected 36 h pre-match (baseline), and repeated at 12, 36 and 60 h post-match. PPO decreased below baseline at 12 (Baseline 6100.4 ± 565.1 W vs. 12 h 5680 ± 589 vs. 5679.9 ± 588.6 W; $P < 0.05$) and 36 h (5760.5 ± 638.5 W; $P < 0.05$), but had recovered at 60 h (5949.7 ± 504.5 W; $P > 0.05$). Cortisol (C) concentrations increased from baseline at 12 (Baseline 0.39 ± 0.09 vs. 12 h 0.59 ± 0.17 $\mu g \cdot dl^{-1}$; $P < 0.05$) and 36 h (0.59 ± 0.20 $\mu g \cdot dl^{-1}$; $P < 0.05$), but were similar at 60 h post-match. Testosterone (T) concentrations decreased from baseline at 12 (Baseline 213.6 ± 84.2 vs. 12 h 150.7 ± 56.1 $pg \cdot ml^{-1}$; $P < 0.05$), but were similar at 36 h (172.60 ± 70.95 $pg \cdot ml^{-1}$; $P > 0.05$) and 60 h (184.17 ± 73.98 $pg \cdot ml^{-1}$; $P > 0.05$) post-match. The T/C ratio decreased from baseline at 12 (Baseline 551.2 ± 219.3 vs. 12 h 265.6 ± 123.0; $P < 0.05$) and 36 h (310.0 ± 148.0; $P < 0.05$), before returning to baseline at 60 h post-match (399.0 ± 210.2; $P < 0.05$). Mood disturbance increased at 12 h ($P < 0.05$), before returning to baseline at 36 and 60 h post-match. In conclusion, post-match changes in neuromuscular function, salivary hormones and mood disturbance were identified in professional rugby union players. Players and coaches can expect reduced neuromuscular function and hormonal disruption for 36 h before recovering at 60 h post-match; with mood recovered by 36 h post-match. Knowledge of these recovery time courses may prove useful for player training program design and post-match recovery strategies.

Key words: recovery, testosterone, cortisol, T/C ratio, power

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CHAPTER ONE

1.0 Introduction

1.0 Introduction

Rugby Union is characterised by repeated, high-intensity work periods of relatively short duration with varying recovery periods that differ according to player position (Duthie, Pyne & Hooper, 2003; Duthie, Pyne, Marsh & Hooper, 2006; Deutsch, Kearny & Rehrer, 2006). Participants are exposed to a high-frequency and magnitude of contacts (collisions and static exertion) (Takarada, 2003) and high-intensity stretch shorten cycle (SSC) movements (Elloumi, Maso, Michaux, Robert & Lac, 2003) which contribute to muscle damage (Takarada, 2003; Smart, Gill, Beavan, Cook & Blazevich, 2008), disruptions in hormone function (Elloumi et al., 2003; McLellan, Lovell & Gass, 2010), and potential reductions in neuromuscular performance in the days following the match (McLellan & Lovell., 2012; Twist, Waldron, Highton, Burt & Daniels, 2011). For example, Takarada et al. (2003) reported significant correlations between creatine kinase ([CK]) levels (an indicator of muscle damage) and the number of tackles made during an amateur rugby match. Similarly, Smart et al. (2008) reported that game time and defending were significantly correlated to changes in [CK]. In these studies, [CK] peaked at 24 hours (h) and showed increases of approximately 1000U/L compared with baseline values. Although these studies provide some information on the magnitude and time course of changes in muscle damage following a competitive rugby match, there is little coinciding data regarding concomitant changes in muscle function which may be a more relevant indicator of neuromuscular recovery (McLellan & Lovell, 2012; Komi, 2000). Rugby union players must train aggressively to consistently perform at a high level, but high-frequency competitive bouts combined with intense training (with insufficient recovery) may potentially lead to underperformance (Barnett, 2006; Montgomery, Pyne, Hopkins, Dorman, Cook & Minahan, 2008; Kramer, Spiering, Volek, Martin, Gerard & Howard et al., 2009; King & Duffield 2009), injury

(Bishop, Jones & Woods, 2008), and illness (Cunniffe, Griffiths, Proctor, Davies, Baker & Jones, 2011). Thus, understanding the recovery processes associated with competition-induced fatigue may help to understand and subsequently manage some of these issues.

Due to the high frequency of SSC movements within many sports, athletes are especially prone to developing low frequency fatigue (LFF) (Martin, Millet, Martin, Deley & Lattier, 2004; Jones 1996). LFF is defined as the preferential loss of force at low frequencies (20 Hz) of stimulation compared with high frequency stimulation (50 Hz) (Martin, Millet, Martin, Deley & Lattier, 2004). The reduction in neuromuscular performance, typically 24-48 hours (h) following intense SSC activity is characterised by swelling, inflammation, soreness and muscle remodelling processes, and is associated with factors such as intensity, duration and modality of exercise (Dousset, Avela, Ishikawa, Kallio, Kuitunen & Kyrolainen et al., 2007; Nicol, Komi, & Marconnet, 1991). Attenuated neuromuscular function following intense competitive bouts might also reflect impairments in excitation contraction coupling associated with LFF (Edwards, Hill, Jones & Merton., 1977; Allen, Lannergren & Westerblad, 1995, Allen, Lamb, & Westerblad, 2008).

Some studies have also investigated changes in the hormonal state, in particular testosterone (T) and cortisol (C), as measures of fatigue and recovery post-competition (McLellan, Lovell & Gass., 2010; 2011; Elloumi et al., 2003). These hormones are indicative of an athlete's anabolic/catabolic state (Crewther, Lowe, Weatherby, Gill & Keough, 2009), neuromuscular trainability (Crewther, Cook & Cardinale, 2011) and physiological stress (Hoffman, Maresh, Newton, Rubin, French & Volek, 2002). However, there are conflicting findings with regard to the competition responses. For example, there are reports of elevated T post-competition (Elias, 1985; Kraemer, Fry, Rubin, Triplett-McBride, Gordon & Koziris et al., 2001; Hakkinen & Pakarinen, 1993; Filaire, Maso, Sagnol, Ferrand & Lac., 2001),

decreases in T (Kraemer, 1992), or no changes in T (Hoffman et al., 2002). C, on the other hand, generally increases after competition (McLellan, Lovell & Gass, 2010; Hoffman et al., 2002; Elias, 1981; Haneishi, Fry & Moore, 2007; Fry, Schilling, Fleck & Kraemer, 2011; Gonzalez-Bono, Salvador, Serrano & Ricarte, 1998). These equivocal findings are likely to be a result of physical factors including duration, intensity and modality of physical exertion (Lac & Berthon, 2000; McGuigan, Egan & Foster, 2004), whether sports are contact, or collision based, and the use of pre and post competition training and recovery strategies (McLellan, Lovell & Gass, 2011). Additionally, post competition hormone fluctuations may also be a result of psychological factors including competition outcome (Fry, Schilling, Fleck & Kraemer, 2011; Filaire, Alix, Ferrand & Verger, 2009; Gonzalez-Bono, Moya-Albiol, Martinez-Sanchis & Salvador, 1999; Mazur & Lamb, 1980; Mehta & Josephs, 2006; Suay, Salvador, Gonzalez-Bono, Sanchis, Martinez, Martinez-Sanchis & Simon et al., 1999), match venue (Carre, 2009; Neave & Wolfson, 2003), competitive standard of the athletes involved (Crewther, Heke & Keough, 2010) and behavioural characteristics of individuals, such as aggression (Salvador, Suay, Gonzalez-Bono & Serrano, 2003; Schulthesis & Rodhe, 2002).

A limited number of studies have investigated the hormonal state of elite rugby union players in response to competition. For example, Elloumi et al. (2003) reported lower post-match concentrations of C and fluctuations in the T: C ratio for 6 days following the match. However, previous research has demonstrated fluctuations in endogenous hormone concentrations in a variety of field-based contact sports (Cormack, Newton & McGuigan, 2008; Argus, 2009; Hoffman et al., 2005) and it is likely that this study is limited by comparisons to baseline hormone samples obtained 2 months prior to the assessed games, and the use of non-elite subjects. Given the strong link between the hormonal and neuromuscular systems, both in terms of their recovery profiles and their contribution to neuromuscular

function (Crewther, Cook & Cardinale, 2011), it would be prudent to examine the interactions of T, C and neuromuscular function pre and post a competitive rugby union match. This concept has been previously reported following elite rugby league competition, whereby declines in neuromuscular performance variables such as peak rate of force development (PRFD) and peak power output (PPO) have been associated with substantial increases in C and decreases in T at 24 post-match (McLellan, Lovell & Gass., 2011). Furthermore, acute and longitudinal changes in post-match hormone concentrations have been demonstrated in rugby union players with increases in C and decreases in T, and fluctuations in T: C ratio for up to ~72 h post-match (Cunniffe, Hore, Whitcombe, Jones, Davies & Baker, 2010).

Fatigue may also manifest itself as changes in athlete behaviour (Gonzalez-Bono et al., 1999; Filaire et al., 2001; Elloumi et al., 2008), or psychologically including reduced motivation, disturbed mood, increased perceived soreness (Kellman & Gunther, 2000. Mood in particular has demonstrated a consistent dose-response relationship as a result of overtraining and has also been used successfully in relation to performance and training recovery. Only a small number of previous studies have investigated mood disruption over the post competition recovery period in rugby union (e.g. Suzuki, Umeda, Nakaji, Shimoyama, Mashiko & Sugawara, 2004; Nicolls, Backhouse, Polman & McKenna, 2009). One study by Suzuki et al. (2004) reported increases in fatigue and depression following a rugby union match and suggested that psychological stress resulting from a rugby match is a result of both exercise intensity and player's satisfaction with their own performances. Similarly, a study by Nicolls, Backhouse, Polman & McKenna (2009) revealed that players reported many stressors being "worse than normal" on the day following a single rugby union match compared to the day before the match, characterised by feelings of tiredness and fatigue. Additionally, there are some evidence suggesting that physiological measures of

fatigue/overtraining (including mood) are related to some objective markers of fatigue (Bouget, Rouveix, Michaux, Pequignot & Filaire, 2006; Maso, Lac, Filaire, Michaux & Robert, 2005; Odagiri, Shimomitsu, Iwane, & Katsumura, 1996).

Despite the high-intensity SSC movements and high frequency of contacts in elite rugby union matches, leading to high levels of muscle damage, there is no published literature on the recovery patterns of the neuromuscular and endocrine systems following an elite rugby union match. Furthermore, no studies have attempted to match neuromuscular, endocrine and mood data to establish their interrelationships (casual or causal) during the post-competition recovery period.

Therefore, the aim of this MPhil is to investigate the time course of recovery for various neuromuscular, endocrine and mood parameters following an elite rugby union match, and investigate whether the behaviour of these variables are similar to each other during the following recovery days. The results from this project will provide unique information on athlete fatigue (e.g. magnitude and time course) and recovery arising from a high level rugby union match.

CHAPTER TWO

2.0 Review of Literature

2.0 Review of Literature

2.1.0 Neuromuscular Fatigue

Neuromuscular fatigue can be briefly described as a negative change in force-generating capacity which occurs as a result of changes in brain, spinal and muscle fibre signalling (Boerio, Jubeau, Zory, & Maffiuletti, 2005; McIntosh & Rassier, 2002; Place, Lepers, Deley, & Millet, 2004). This concept can be divided into two categories; (1) central fatigue, referring to changes occurring in the spinal cord and brain, and (2) peripheral fatigue, referring to alterations within the muscle fibre (Weir et al., 2009). It has been suggested that the duration, volume, intensity and type of muscle contractions performed will determine the contributions of central and peripheral factors (Thorlund et al., 2009).

2.1.1. Central Fatigue

Central fatigue involves a progressive degradation of the muscle voluntary activation, induced by exercise and exercise induced muscle damage, and encompasses supraspinal and spinal factors capable of causing a decrement in motor neuron excitation (Gandevia et al., 1995). Causes and mechanisms underpinning central fatigue are poorly understood, but several possible mechanisms have been proposed. The Central Fatigue Hypothesis is based on the assumption that the synthesis and metabolism of central monoamines including serotonin, dopamine, and noradrenaline are altered. This is related to decreased excitation supplied via the motor cortex (Gandevia, 1998; Taylor Todd & Gandevia, 2006; Meeusen et

al., 2006; Abbiss & Laursen, 2005; Noakes, 2000), inducing a decline neural drive through corticospinal intracortical inhibition (Boyas & Guevel, 2011).

At the spinal level, exercise-induced muscle damage (EIMD) may activate group Ia, Ib, II and IV afferents (Dousset et al., 2007; Avela et al., 1990; Nicol et al., 2006; Weir et al., 2006; Verin, Ross & Demoule et al., 2006), leading to central fatigue. Pre-synaptic inhibition is considered to be the predominant underlying mechanism, leading to reduced force output by decreasing post synaptic excitation (Pettorossi, Della, Bortolami & Brunnetti, 1999). Furthermore, it has been demonstrated that group 1a afferents are mediated by group III and IV afferents (Avela et al., 1999; Ducateau & Hainaut, 1993), which affect muscle spindle and golgi tendon organ activity (Gandevia, 2001). As a result, observed decreases in central drive may occur to protect the muscle from injury. Other mechanisms potentially contributing to central fatigue include including stretch reflex disfacilitation and suboptimal cortical output (Babault et al., 2006).

2.1.2. Peripheral Fatigue

Peripheral fatigue is traditionally associated with a reduction in the ability of skeletal muscle to produce force (Nordlund, Thorstensson & Creswell, 2004) and may also occur within components of the neuromuscular junction and terminal branches of the motor axons (Babault et al., 2006). Muscle damage, usually as result of eccentric muscle contractions at longer muscle lengths is linked to impairments in excitation contraction coupling (e-c) mechanisms, and may also contribute to the delay in muscle force recovery (Edwards et al., 1977; Westerblad, Duty, & Allen, 1993; Allen et al., 1995; Allen & Westerblad, 2001).

Two further theories have been proposed to explain the occurrence of peripheral fatigue, the muscle power model and the neuromuscular propagation theory. The muscle

power model proposes that disruptions in Ca^{2+} release and re-uptake account for decrements in muscle function (Allen et al., 1995; Abbiss & Laursen, 2005). Furthermore, the lack of ATP and altered fibre recruitment as a result of muscle damage may contribute significantly to this process (Zehnder, Muelli, Buchli, Kuehne, & Boutellier, 2004). .

The neuromuscular propagation failure theory (Lepers, Maffiuletti, Rochette, Brugniaus, & Millet, 2002) involves increased concentrations of intercellular H+ and extracellular K+ inducing sub-optimal performance in action potential transmission at the neuromuscular junction and terminal branches of motor axons (Abbiss & Laursen, 2005). This can impair SR Ca^{2+} release and lead to a decrease in force production (Abbiss & Laursen, 2005; Noakes, 2000). As less Ca^{2+} is released per stimulus, higher central drive is required to maintain the same levels of force (Abbiss & Laursen, 2005). This has been demonstrated through the use of repeated low frequency stimulation, and has been termed Low Frequency Fatigue (LFF).

2.1.3. Low Frequency Fatigue

Low Frequency Fatigue (LFF) is defined as a proportionally greater loss of force when muscle is stimulated at low frequencies (10-30Hz) (Edwards et al (1977), usually observed via a reduction in the 20:80 Hz ratio (Martin et al., 2004). In practice, field sports involving a high frequency of SSC and eccentric type contractions create muscle damage and associated disruptions to e-c coupling (Martin et al., 2004; Jones, 1996; Keeton & Binder-MacLeod, 2006). Specifically, these disruptions involve lower SR Ca^{2+} release, causing a transient decline in the amount of Ca^{2+} available for attachment to binding sites on troponin. This causes limitations in cross bridge formation and subsequent force output per action

potential (Bigland-Ritchie, Cafarelli, & Vollestad, 1986; Strojnik & Komi, 2000; Keeton & Binder-MacLeod, 2006), and can take several days to recover from (Jones, 1996).

2.1.4. Neuromuscular Fatigue & Competition

Studies investigating fatigue responses to field sports over the days following competition have investigated both contact and non-contact team sport, and have indicated that substantial disruptions occur in neuromuscular performance variables as a result of competitive bouts. For example, a study by Rampinini, Bosio, Ferraresi, Petruolo, Morelli & Sassi (2011) examined various neuromuscular function including variables such as maximal voluntary contraction (MVC), 20 m sprint times, muscle soreness and maximal voluntary contraction (%VA), over 48 h following a professional soccer match (n = 20). Peak torque at 10 Hz and 40 m sprint performance were significantly reduced at 24 hours (h) post-match before returning to baseline values at 48 h with no change in any other variables. Also characterising neuromuscular fatigue arising from a high level soccer match was a recent study by Krustrup et al. (2011), where MVC force, SR function and muscle glycogen, soreness and damage levels were assessed during the 72 h post-competition recovery period. MVC force sustained over 1 s was significantly reduced at 0 and 24 h, by 11% and 10% respectively.

Similarly, Andersson et al. (2008) investigated the neuromuscular time course of recovery between two soccer matches separated by 69 h in a group of elite females. Significant decrements in CMJ height were apparent leading into the second match which was attributed to myofibrillar damage as supported by substantial elevations in [CK] 45 h post-match, suggesting impairments in e-c coupling mechanisms (Edwards et al., 1977; Allen et al., 1995; Allen, 2001). Increases in 20m sprint performance and [CK] and decreases in

CMJ height were also noted after two days following the match. Collectively, these studies in high level soccer indicate that substantial decrements in neuromuscular performance are still present in athletes immediately following competitive bouts, and for durations of approximately 69 h following competition.

Studies in other non-contact sports include that of Ronglan, Raastad & Borgesen (2006) where the extent of neuromuscular fatigue and recovery following a handball training camp and an international tournament in a group of elite females (n = 7). Significant declines in jump height (- 6.9 ± 1.3 %) and 20 m sprint times were reported following the second day of the training camp. Despite little activity on the third day of the training camp, followed by a rest day, values for jump height and 20 m sprint time remained depressed, indicating a prolonged depression in neuromuscular performance following intermittent sport. Subjects also participated in a handball tournament, which resulted in gradual declines in CMJ height (- 6.7 ± 1.3 %) and 20 m sprint times (3.7 ± 0.4 %). These findings were attributed to muscle damage and subsequent alterations in e-c coupling, resulting from high intensity SSC movements including running, jumping, changing direction and decelerating.

Specifically to contact sports, Cormack et al., (2008a), examined neuromuscular responses to an Australian Rules Football (ARF) match in 22 elite level male players. Testing included a single CMJ and 5 repeated CMJs prior to, then immediately, 24 h, 72 h, 96 h and 120 h following the match. All CMJ performance variables (flight time [s] = -3.5%; mean power [W] = -13%; relative mean power [W·kg] = -12.4%; relative mean force [N·kg] = -2.5%; flight time: contraction time ratio [s] = -17.1; CMJ5 flight time [s] = -3.6 %) were significantly depressed at 24 h post-match. Mean power (W), relative mean power (W·kg) and flight time (s) (FT) were also significantly depressed at 72 h post-match, and FT (s) remained depressed at 120 h post-match. All other variables had recovered to baseline values

by 96 h post-match. Declines in neuromuscular performance in this study were accounted for by changes in knee and hip angles (Augustsson, Thomee, Linden, et al., 2006), increased ground contact time (GCT) through decreased muscle tendon stiffness (Toumi, Poumarat, Best, Martin, Fairclough and Benjamin, 2006) and reduced central drive, related to LFF (Abbiss & Laursen, 2005).

Similarly, McLellan et al. (2011) investigated neuromuscular responses to an elite Rugby League match using PF, PP and PRFD in a group of 17 elite players. Baseline measurements were obtained 24 h and 30 min prior to, and were repeated at 30 min, 24 h, 48 h, 72 h, 96 h and 120 h following the match. Significant reductions in PRFD and PP were reported 24 h post game compared to baseline values (30 min pre-match); with no significant changes in PF. Significant correlations were also reported between very heavy and severe impacts (as measured by GPS) and PRFD at 24 h post-match. It was suggested that these reductions in neuromuscular performance were a reflection of impairments in e-c coupling. As there was no change in PF, it was also suggested that the velocity component of the CMJ is a more sensitive component to fatigue than force, and supports previous literature which suggest power measures are useful in monitoring LFF (Fowles, 2006).

Furthermore, McLean et al. (2010) investigated fluctuations in neuromuscular fatigue variables following elite Rugby League matches in different length micro-cycles (5 d, 7 d, 9 d). Significant reductions in power and FT were observed up to 48 h following matches in all micro-cycles and CMJ relative power was consistently lower in the 7 d (where training loads were higher) compared to the 9 d micro-cycle. CMJ variables returned to baseline values 4 d following the match. Similarly, Coutts, McLean, Kelly, Cormack & McGuigan (2009) reported a significant decline in force and power measures during a CMJ 24 h after a rugby league match, which remained reduced for 48 h post-match.

Collectively, these studies indicate competitive bouts in contact team sport result in substantial disruptions in neuromuscular performance to the athletes involved. This highlights the importance of monitoring these variables in order to effectively assess when an athlete is ready to train and perform. Furthermore, it was also suggested that long term research into neuromuscular fatigue should involve power variables due to the sensitivity of the velocity component to neuromuscular fatigue (McLellan et al., 2010).

2.1.5. Potential role of muscle damage in neuromuscular fatigue

Field sports such as Rugby Union involve a high frequency of eccentric and SSC type movements (Takarada et al., 2003) which create disruptions in e-c coupling (Allen, 2001), and may account for the expression of LFF occurring through muscle damage (Allen, 2001; Clarkson & Hubal, 2002; Skurvydas, Sipavicience, Krutulyte, Gailiuniene, Stasiulis & Mamkus et al., 2005). Symptoms of muscle damage include disruption of intracellular muscle structure, sarcolemma and extracellular matrix, prolonged impairment of muscle function, delayed onset of muscle soreness (DOMS), stiffness and swelling (Newham, Mills & Quigley et al., 1983; Clarkson, Kazunori & Braun, 1992; Cleak & Eston, 1992; Rodenburg, Bar & De Boer 1993; Pyne, 1994).

The immediate loss of strength after eccentric exercise has been explained by the overstretching of sarcomeres, which results in non-optimal overlap between actin and myosin filaments, as well as disruptions in e-c coupling (Frieden & Lieber 2001). This "popping of sarcomeres theory" proposes that when stretch velocity exceeds the maximum stretch capabilities of the contractile elements, weaker sarcomeres will elongate. Following a small number of eccentric contractions, sarcomeres usually reinterdigitate on relaxation, however repeated and prolonged eccentric contractions cause sarcomeres to remain lengthened. The

force-generating ability of muscle is then compromised due to an alteration in the length-tension relationship (Jones, 1996). Falkner et al. (1993) suggested that some sarcomeres are stretched beyond overlap, and thus injured during eccentric contractions, whereas others maintain their length.

The recruitment of high threshold motor units during high intensity SSC activities also causes muscle damage through increased stress on a lower number of active fibres (Moritani, Muramatsu, & Muro, 1988; McHugh, Connolly, Eston, & Gleim, 1999). The alteration in the length tension relationship is of primary importance as these changes determine joint angles for optimal torque production (Morgan 1990). Mechanical injury to the sarcomeres is also affected by cytoskeletal proteins including titin and desmin, as these proteins function as Z-disk stabilisers (Morgan, 1999). Morphological studies indicate that the Z disk is the most vulnerable structure to eccentric exercise induced muscle injury. Disruption can also occur in the sarcolemma, T-tubules, myofibrils and the cytoskeletal system. In the immediate decline in force producing capabilities following exercise, the sarcomeres may demonstrate Z-disk streaming, and in more severe cases, smearing (dispersion of Z-disk material into neighbouring sarcomeres), focal loss and displacement of Z-disk material may occur.

Leakage of extracellular Ca^{2+} into the intracellular space is another consequence of sarcomere stretching. This is due to disruption of the sarcolemma, SR and transverse tubules (T-tubules), which are positioned in a perpendicular and parallel fashion to the long axis of the fibre affected (Allen, 2001; Armstrong, Warren & Warren, 1991). The activation of stretch activated channels and Ca-ATPase facilitates in the removal of intracellular Ca^{2+}, but if this cannot be achieved at a sufficient rate, Ca^{2+} dependent proteolytic and phospholypolytic processes are activated which degrade structural and contractile proteins, and membrane phospholipids (Armstrong et al., 1991), with subsequent increases in

relaxation time (Westerblad & Allen, 1994). This process occurs before and during the inflammatory phase whereby phagocytic cells such as macrophages are released (Armstrong et al., 1991).

Cheung, Hume & Maxwell (2003) also suggested that cellular respiration within the mitochondria may be inhibited through the slower rate of ATP resynthesis due to Ca^{2+} accumulation in injured muscle. Lower force capabilities may also result during the repair process of t-tubules and SR, as the Ca^{2+} release and re-uptake is inhibited by a seal between the two (Allen, 2001). Alterations in membrane potential is another factor contributing to the disruption in SR Ca^{2+} release case (McBride, Stockert, Gorin, & Carlsen, 2000), resulting in central nervous system inhibition (Rome, Morgan, & Julian, 1985) and ultimately a fatigued state.

2.1.6. Delayed recovery of neuromuscular function following muscle damage

It has been demonstrated that force production capabilities decline significantly following exhaustive SSC protocols, and recover over a period of up to 8 days (Komi, 2000; Dousset et al., 2007). Although force production capabilities have shown a return to baseline 2 h following exercise, a secondary longer lasting reduction in performance has also been demonstrated (Drinkwater et al., 2009). For example, Dousset et al. (2007) demonstrated a quick recovery of force following an exhaustive bout of drop jumps, with a secondary decline after 2 h, showing a bimodal recovery pattern (Figure 1.0).

This bimodal response has been observed following intermittent field sports including ARF (Cormack et al., 2008) and soccer (Andersson et al., 2008). The study by Andersson et al. (2008) demonstrated initial declines in peak torque, sprint performance and jump height,

before recovery of these parameters to near baseline after 5 hours. Secondary declines in CMJ performance and peak torque were reported, whereas sprint performance was maintained. Peak torque demonstrated full recovery after 27 h, but CMJ performance was still not fully recovery 72 h.

The decline in neuromuscular performance 1-3 days following fatiguing activity can be accounted for by the acute inflammatory response, and phagocytic activity (Faulkner et al., 1993). This is characterised by muscle swelling and inflammation, increases in biomarkers such as C-reactive protein (CRP) and neurotransmitters such as substance P (Inoue, Tokuyama, Nakayamada & Ueda, 1998), which activate group III and IV afferents resulting in further force decrements. Following this, force recovery may take up to 8-9 days to return to baseline levels (Dousset et al., 2007; Nicol et al., 2006). The second phase is also characterised by delayed onset of muscle soreness (DOMS) (Armstrong, 1984; Cheung et al., 2003) and is related to both muscle damage (Clarkson & Hubal, 2002) and the inflammatory processes (Faulkner et al., 1993, Armstrong, 1984; Smith, 1991). The bimodal recovery pattern and associated soreness also contribute to alterations in muscle fibre recruitment (Edgerton et al., 1996), compromised strength and power performance (Smith, 1991), changes in agonist-antagonist muscle ratios (Orchard, Marsden, Lord, & Garlick, 1997), and changes in the coordination of body segments (Edgerton, Wolf, Levendowski, & Roy, 1996).

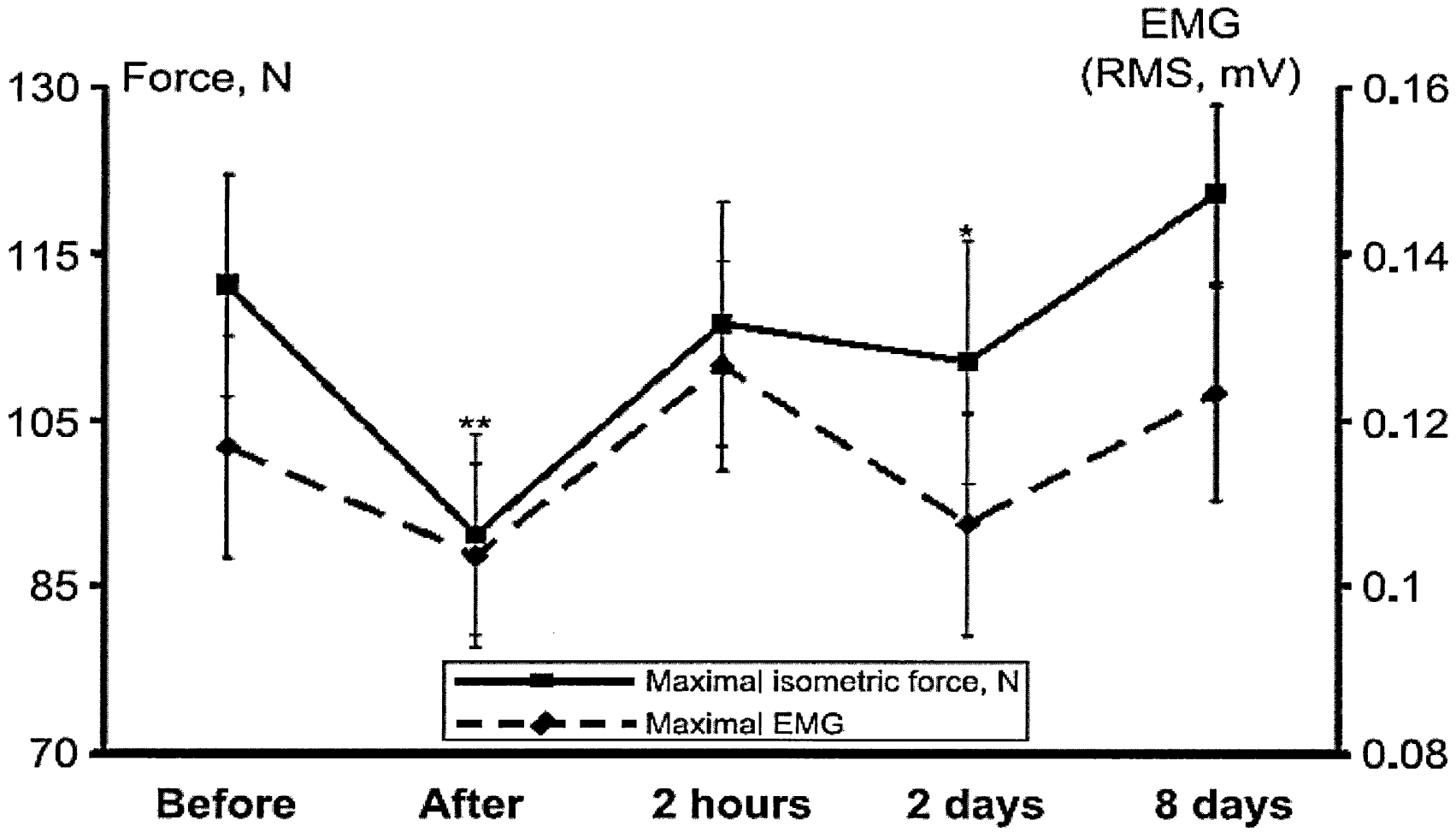

Figure 1.0. Bimodal changes in maximal isometric voluntary force and EMG values prior to fatiguing trials and during the recovery period. * indicates significant differences in force values (* = $P < 0.05$; ** = $P < 0.01$) (Dousset et al., 2007).

2.1.7. Dynamic assessment of neuromuscular fatigue

Several studies have used the SSC model to assess neuromuscular fatigue in athletic populations through the use of squat jumps (SJ), countermovemement jumps (CMJ) and repeated CMJ, each of which assess slightly different aspects of fatigue (Hoffman et al., 2002; Girard, Lattier, Maffiuletti, Micallef, & Millet, 2007; Cormack, et al., 2008a; McLean et al., 2010; Bosco, Luthanen & Komi (1983) identified limitations in the use of repeated jump tests, Wingates and the Margaria tests in assessing neuromuscular fatigue. These limitations include the inhibition of mechanical power output through metabolic factors, suggesting that more prolonged testing procedures should be avoided in assessing neuromuscular fatigue.

For example, McLellan et al. (2010) suggested that explosive type exercises may be most effective in detecting neuromuscular fatigue, given that some performance variables are maintained, whereas the recovery duration of others are more prolonged. Additionally, Cormack et al. (2008a) also suggested that a single CMJ was superior to 5 repeated CMJ due to inconsistencies in jump height reliability in the repeated trials. In support of this, Horita et al. (2003) assessed SJ and DJ performance before and 10 min, 20 min, 2 d and 4 d following an exhaustive bout of SSC exercise on a sledge ergometer. SJ performance showed an immediate decline following the exercise, but the later measurements showed recovery to baseline levels. However, DJ did not recover until 2 days later which further highlights different recovery patterns for different types of jumps.

Evidence from the aforementioned literature indicates that explosive, SSC type exercises are sensitive enough in detecting neuromuscular status, from which aspects of readiness to train can be determined. From a practical perspective, testing and monitoring of athletes requires the ability to show a valid and reliable reflection of sport specific

movements in as timely manner as possible, the single CMJ seems the most appropriate method to fulfil this purpose (McLellan et al. 2010). Additionally, high reliability of the CMJ with explosive power has been demonstrated with strong correlations of r = 0.87 (Markovic, Dizdar, Jikic, & Cardinale, 2004; Moir, Button, Glaister, & Stone 2004). However, several factors must be adhered in order to produce valid and reliable results.

For example, consistent warm up procedures must be undertaken before CMJ testing, as differences have been demonstrated between static and dynamic stretching, with improved performance arising from dynamic stretching (Hough, Ross, & Howatson, 2009). It has also been reported that the use of arms during a CMJ may add approximately 10% to jump height (Khalid, Amin, & Bober, 1989; Luhtanan & Komi, 1978); suggesting arms must be kept akimbo for the entire movement to ensure a valid assessment of lower body power.

Valid and reliable data calculation techniques are also an important consideration, as errors of up to 26% can be made if calculation rules are not adhered to (Street et al., 2001). This includes ≥ 580 Hz low pass filters (for both analogue and digital), and sampling frequencies of > 1080 Hz (Kieble, 1998), a minimum threshold for identifying take-off (< 2 N) and body weight measurement period of at least 2 seconds. Owen (2008) suggested that the use of a Kistler force plate (type 92866AA, Kistler Instruments Ltd, Farnborough, UK) requires a minimum of 5 s for an accurate assessment of body weight and minimal error in CMJ variables such as peak power output and jump height.

2.1.8. CMJ variables

Several variables indicative of muscle function can be derived from a single CMJ, including peak power output (PPO) (Hoffman et al., 2002; Cormack et al., 2008a; McLellan et al., 2010), mean power output (MPO) (Cormack et al., 2008a; Thorlund et al., 2009), peak

force (PF) (Hoffman et al., 2002, Cormack et al., 2008a), RFD (Thorlund et al., 2008; Thorlund et al., 2009) and peak RFD (PRFD) (McLellan et al., 2011), jump height (JH) (Hoffman et al. 2002; Girard et al., 2008; Thorlund et al., 2008; Cormack et al., 2008a; McLellan et al., 2010) and flight time: contraction time ratio (FT:CT) (Thorlund et al., 2008; Cormack et al., 2008a; Thorlund et al., 2009).

The response of JH seems to show inconsistencies following fatiguing exercise protocols. Some studies have reported decreases (Ronglan et al., 2006; Thorlund et al., 2008; Girard et al., 2008) or no change (Thorlund et al., 2009) in JH. It has been suggested that subjects may alter CMJ technique and in attempt to compensate for fatigue and in doing so manage to maintain similar JH scores (Thorlund et al., 2008; Rodacki et al., 2002). In support of this, Salles, Baltzopoulos & Rittweger (2011) reported peak JH at a 90° knee angle prior to take off, in comparison to 50° or 70° in maximal effort CMJ. This suggests that JH may not be a sensitive enough measure to detect neuromuscular fatigue. Thorland et al. (2008) also suggested that the change in jump strategy may be due to an attempt to compensate for reduced RFD in the early stages of the CMJ.

In the studies by Thorland (2008; 2009), average RFD was defined as time of transition between deceleration to acceleration in the eccentric phase (Tdec) to Tdec + 50ms and also Tdec to Tdec + 100ms. However, McLellan (2011) calculated RFD from the maximum force that occurred over the first derivative of the force time curve, which highlights further inconsistencies in determining this parameter, and creates difficulty in comparing studies. Furthermore, Viitasalo, 1982 reported that the variability of RFD values within subjects is also a result of the unlikelihood of subjects producing exactly the same tension leading into a CMJ. Furthermore, very low intraclass correlation coefficients (ICC) of PRFD have been reported (Hori, Newton, Kawamori, et al., 2009). In the same study, it was recognised that well trained subjects who are well familiarised with jumping may produce

rapid, high forces throughout the eccentric and concentric phases of the jump, with PRFD possibly occurring in either phase. The stage at which the PRFD occurs is dependent upon speed and depth of the eccentric and concentric phases as well as the magnitude of the force produced, suggesting that RFD is not a reliable or valid enough method of assessing neuromuscular function.

Studies by Cormack et al (2008a; 2008b) reported decreases in flight time contraction time ratio (FT: CT) following an Australian Rules Football (ARF) match, potentially reflecting alterations in neuromuscular function. However, it is unclear what these decreases are mediated by, creating difficulty in gaining an insight into the mechanisms of this change. Furthermore, force platform data collected in this study was recorded at a sampling frequency of only 200 Hz, and start time was defined as "the time from the initiation of the countermovement until the subject leaves the force plate." As ± 5 SD – 10 ms from body weight (N) is accepted as the most reliable method to attain start time (Owen, 2008), the validity and reliability of force platform data collected by Cormack is questionable.

Thorlund et al., (2008) reported decreases in FT: CT following a handball match, where as a later study by the same group observed no changes in FT: CT after a soccer match. FT: CT ratio must be considered on an individual level as team averages may introduce misdiagnoses of neuromuscular status due to individual responses to fatiguing exercise. Power variables derived from a CMJ are useful in determining an athlete's neuromuscular function (Komi, 2000), however care must be taken during calculation to ensure valid and reliable results are obtained. Studies by Cormack et al. (2008a; 2008b) did not correctly identify eccentric and concentric phases, as the minimum vertical ground reaction force prior to values increasing again served as the marker for the start of the concentric phase. However, the end of the eccentric and beginning of the concentric phase is

the point where velocity begins to change from negative to positive (Linthorne, 2001; Owen, 2008), suggesting that the power data in these studies is also questionable.

It appears that there are inconsistencies in the literature with regard to the responses of PF and PPO data to fatiguing exercise. For example, a study by Hoffman, Nusse & Kang (2003) reported decreases in PF and PPO during an intercollegiate American Football match, where these variables had returned to baseline values by the end of the match. In contrast to this, Thorland et al. (2008; 2009) did not show decreases in PF or PPO following elite handball or soccer matches. These inconsistencies are likely due to differences in requirements between sports, varying testing protocols and methods determining the data obtained.

Furthermore, Salles et al. (2011) reported variations in JH measurements with respect to different pre-jump depths; however, PPO was unaffected suggesting that PPO is a more robust method of assessing neuromuscular function than JH. A further study in support of PP rather than JH is that of Sanchez-Medina & Gonzalez-Badillo (2011), where acute mechanical and metabolic responses to resistance exercise protocols differing in the number of repetitions performed in each set with respect to the maximum predicted number were analysed. Strong validity was reported in using velocity loss as an indicator of neuromuscular fatigue. Moreover, the CMJ is a useful monitoring tool for elite athletes in the assessment of neuromuscular function.

2.2.0 Endocrine response to competition

Since an imbalance between training/competition stress and recovery may lead to a state of over-reaching or over-training, timely hormonal measures may assist in the monitoring of the responses to stressful rugby-related situations, particularly competition (Elloumi, 2003). Higher glucocorticoids, including cortisol (C), are usually associated with higher physiological and psychological stress, and may be indicative of disruptions in homeostatic balance following physical exertion (Crewther et al., 2009). However anabolic hormones such as testosterone (T) are associated improved performance (Crewther et al., 2011). For example, Kraemer & Ratamess (2005) reported that endogenous T may play a pivotal role in power production through its effects on the nervous system. The interaction of T with receptors on neurons amplifies levels of neurotransmitters, increases cell body size and dendrite length/diameter, and contributes to the regeneration of nerves (Kraemer & Ratamess, 2005).

Some studies have suggested that event outcome may contribute to fluctuations in steroid hormones (Salvador, 2004; Filaire, 2001; Edwards, 2006; Fry et al., 2011), with higher T associated with winning immediately following the event, and lower T associated with losing (Kivlighan,, Granger & Booth, 2005; Booth et al ., 1989; Elias, 1981; Mazur & Lamb, 1980). Further factors which may influence the degree to which these hormones fluctuate are playing time (starters versus non-starters) (Hoffmann, et al ., 2002), circadian rhythms (Salvador, 2004), competition importance (Salvador 2004; Hoffmann et al., 2002), muscle damage (McLellan et al., 2011) and the resulting inflammatory response (Borer, 2003).

2.2.1 Endocrine responses to team sport competition

The majority of studies investigating hormones and competition have focussed on acute responses of T and C in a variety of different sports (Haneishi et al., 2007; Gonzalez-Bono et al., 1999; Thorpe & Sunderland 2011; Edwards, Wetzel & Wyner; Filaire et al., 2001; Salvador, Gonzalez-Bono & Serrano, 2001; Salvador et al., 1999; Suay et al., 1999; Kraemer et al., 2001, 2011; Moreira et al., 2010). Few studies have investigated fluctuations in T and C over the days following competition, with some of these examining the responses of T and C to intermittent contact sports including rugby league (McLellan et al., 2010; 2011), American football (Hoffman et al., 2002; 2005; Kraemer et al., 2009) and Australian rules football (ARF) (Cormack et al., 2005; 2008). For example, McLellan et al. 2010 examined the salivary T and C responses to elite Rugby League match play in a group of elite males. Saliva samples were obtained 24 h pre, within 30 min of the game conclusion, and 24, 48 h, 72 h, 96 h and 120 h post-match. C concentrations were significantly elevated from 24-30 min pre match, and up to 24 h post-match compared with baseline values. Additionally, the authors made an interesting link between the fluctuations in C and neuromuscular performance in that the players with the largest decrements in PF also produced a higher salivary C post-match.

The same group also examined pre, during and post-match endocrine responses to a further rugby league match (McLellan et al., 2011). Saliva samples were obtained 24 h and 30 min pre-match, then 30 min, 24 h, 48 h, 72 h, 90 h and 120 h post-match. A significant increase in C was observed 24 h post game with a simultaneous decrement in T:C ratio, which returned to baseline values within 24 h post-match. The study also accounted for increases in C to be a reflection of the duration, intensity, and combative nature of rugby league and psychological influences of anxiety and perceived stress of the competition

environment. Additionally, decrements in neuromuscular performance via a match induced decline in central drive and failure of the excitation contraction mechanism may indicate that players experienced greater psychological stress, or competed to a higher intensity or longer duration, resulting in greater concentrations of post-match C. The response of C was also examined with respect to collision in rugby league match play and reported consistent post-match increases in C. The gradual decline over the recovery days following the match represents the removal of match related psychological and physiological stressors over this period. Interestingly, this study also reported no relationship between C concentrations and collisions (measured through the use of GPS), and suggested that this indicates that rugby league match play generates sufficient psychological and physiological stress to cause an elevation in C with a return to baseline within 48 h. Furthermore, Cormack et al. (2005) reported a significant increase in C, which induced a substantial decrease in T:C ratio up to 48 h following an ARF match, suggesting a catabolic state for this period of time following competition.

Specific to Rugby Union, Elloumi, Maso, Michaux, Robert & Lac, (2003) examined the time course of recovery following a rugby union match for six days by comparing pre and post-match salivary T and C fluctuations to baseline concentrations. Higher post-match concentrations of C were reported, but T:C ratio was increased above basal levels for 5 days post-match. The exact reasons behind the conflicting findings are unclear, but may be attributed to the level of competition/match intensity, the opposition players and overall outcome, and post-match recovery protocols. It was noted that C concentrations increase as a result of exercise intensities > 60% maximal power for durations of >30 min (Kirschbaum & Hellhammer 1989; Lutoslawaska et al., 1991; Snegovskaya & Viru, 1993), and a greater duration and intensity produces responses of a larger magnitude (Lac & Berthon, 2000; Port,

1991; Urhausen et al. 1987), with the psychological constraint associated with the competition environment reinforcing the stress response.

The substantial increases in C post-match demonstrate dramatically different results from protocols obtained in a laboratory setting, where no changes in C are reported over the recovery days (Snegovskaya & Viru, 1993; Hooper et al., 1995; Vuorimaa et al., 1990). This further highlights the importance of collecting data from a competitive situation given that results obtained under laboratory conditions, even if they are exhausting, are not comparable to competitive situations which create a greater physiological and psychological demand. However the mechanisms for the C fluctuations over the days following the match are not provided in the study.

The cells in the dorsal and ventral parts of the PVN control the activity of sympathetic (S) nerves and release adrenomedullary catecholamines, but these processes depend on the presence and action of CRF (Borer, 2003). CRF is the primary stimulus for ACTH and β-endorphin out of the POMC precursor in the anterior pituitary corticotrophs (Borer, 2003). The rise in C secretion follows ACTH release after a 15-30 min delay, and secretion of C and adrenomedullary A is elicited at exercise intensities between 80-90% $VO2_{max}$ (Borer, 2003). At these intensities, rising plasma concentrations of C reach the adrenal medulla from the surrounding cortex and stimulate the expression of the PNMT gene and biosynthesis of A. A then stimulates ATCH secretion from the anterior pituitary, resulting in further C secretion (Borer, 2003), which may be a mechanism for the release of C in the athletes in this study.

As for T, the match induced a 20% drop in T, and the authors accounted for this by the inhibition of the gonadotrophic axis at a hypothalamic level via CRH. However, T then increased above baseline values during the recovery period, however the mechanisms for these changes were unexplained and the authors did not establish a link between the changes

of T and C following the match. However, baseline samples were collected approximately two months prior to the match measured, which did not account for fluctuations in baseline samples over time, and with training (Viru & Viru, 1995).

Collectively, these studies indicate that the physiological and psychological stress of competition is associated with consistent increases in C during the days following competition, with more inter-individual differences in the response of T. Steptoe (1990) classified psychophysiological response to stress into characteristics of the stressor, psychological and personality characteristics, and biological and constitutional factors of the individual. These competition related increases in C are most likely related to muscle damage and subsequent inflammation arising from the activities involved in team sports, particularly those involving contact (McLellan et al., 2010; 2011). The response of T is likely down to more psychophysiological stimuli, behavioural traits and individual perceptions (Filaire et al., 2001).

Table 2.0: Studies examining the responses of T and C to competition

Author	Aim	Subjects	Methods	T	C	T:C
Aizawa et al. 2006	C responses to a soccer match	9 elite females	Blood samples obtained pre competition, 3 days during competition and 3 days following competition		↑ on days 2 and 3 of competition	
Elloumi et al. (2008)	Monitor recovery period following a rugby match by measuring changes in T and C over a six day period	n = 20 males of Tunisian Rugby Team	Saliva for baseline (2 months pre), day of competition (8am, 4pm, 6pm and 8pm) and 6 days post competition (8am and 8 pm)	↓ post-match and during recovery period. ↑ during 5 days recovery	148% ↑ post-match. Return to baseline within 4 h	↓ at 4pm on match day vs. baseline.↑ T:C for 5 days following match
Cormack et al. 2005	Acute short term response of endocrine variables and relationships with NM performance	n = 22 elite male ARF players	Saliva samples 48 h pre-match, then 24, 72, 96 & 120 h post. Performance measures were CMJ and CMJ5.	Unclear results	↑immediately post, 24 & 24, ↓ at 96 and 120	↓ immediately and 24 h post, then unclear results thereafter.
Cormack et al. 2008	NM and hormone status through competitive season	n = 15 elite male ARF players	Saliva samples obtained through 22 match season. Single CMJ followed by repeated CMJ5.		↓ at all time points apart from trivial response in mid 1-2 (144 h post game 1).	
McLellan et al. 2010	Salivary C response to elite RL match	17 elite males	Saliva samples at 24 h pre, then 30 min, 24, 48, 72, 96 & 120 h post-match.		↑ from 24-36 h	
McLellan et al. 2011	Salivary hormone changes during in season RL period	12 elite RL players	Saliva obtained during varying microcycle lengths following matches	No change	No change	No change
McLellan et al. 2011b	Examine pre and post-match endocrine response to elite RL	17 elite males	Saliva samples at 24 h pre, then 30 min, 24, 48, 72, 96 and 120 h		↑ 24 h post-match	Return to baseline within 24 h

	match		post-match			
Passerlergue & Lac, 1995	T, C & T:C fluctuations during and following wrestling competition	15 males (national & international standard)	Saliva obtained at baseline (3 weeks prior to comp), and post competition samples obtained daily for 8 days	↑ throughout competition, not significantly different during recovery period	↑ following competition, and during recovery period	↑ during recovery period
Salvador, Gonzalez-Bono & Serrano, 2001	Hormone fluctuations in response to a judo competition	17 male judo athletes	Saliva analysed for T, C & T:C 1.5 h pre, then during 8 days following competition	↑in some athletes prior to competition	↑ prior to competition	
Kraemer et al. 2011	T response to competitive judo	12 male NCAA division I males	Serum collected pre and immediately post for T and C, from 5 matches over 2 days	↑ for winners and losers but greater ↑ for winners. Related to change in adrenaline	Similar ↑ for winners and losers.	
Cunniffe et al. (20110	Immunoendocrine responses over a 3 week international rugby union series	8 elite rugby union players	Venous bloods obtained on camp entry, then before, and 0, 14, and 38 h after 2 games over 21 days	37% ↓ after both games	40% ↑ after both games	Gradual ↑ throughout tournament (35% 45% ↑ on days 19 and 21 compared to camp entry)
Argus et al. (2009)	Assess changes in T & C over 13 weeks of elite rugby	32 professional rugby players	Saliva obtained before strength testing (5 times during competitive season)	Moderate ↑ over competitive season (54%)	Moderate ↑ over competitive season (9.7%)	Small ↓ (22%)

2.2.2. Relationship between neuromuscular and endocrine variables of fatigue

Previous work has alluded to a relationship between neuromuscular and endocrine variables of fatigue (McLellan et al., 2011). For example, an inverse relationship was reported between C and CMJ performance; however this relationship existed only in a group of soccer players and non-starters (Kraemer et al., 2004), indicating reduced neuromuscular performance in a fatigued state following a bout of competition. This study also reported significant correlations between T:C and CMJ performance in starters at the end of a season. However, one study reported no significant correlation between endocrine measures of performance in a group of rugby league players, although it was suggested that players were experiencing a catabolic state prior to endurance testing, which influenced results in a negative manner (Coutts et al., 2007). Crewther et al., (2009) reported a significant positive correlation between T and 10m sprint velocities, as well as strong correlations between C and SJ power output in a group of elite male rugby players. Furthermore, McLellan et al. (2011) reported a significant correlation between C and reductions in PF 30 min following an elite rugby league match in a group of elite males.

As the endocrine system shares its signalling and coordinating function with the nervous system, the two have evolved to integrate physiological functions (Borer, 2003). Neurotransmitters are released into the synaptic cleft to activate receptors on adjacent neurons, and are characterised by the speed of transmission (ms) and discrete paths of delivery (Borer, 2003). Specific to sport and the control of muscular activity, acetylcholine (Ach) is released from motor nerve endings to interact with nicotinic cholinergic receptors of skeletal muscle (Borer, 2003). Delivery of endocrine messages to specific targets is

established by the specificity of receptors on target cells. When chemical messengers are secreted into the circulation, they are called neuroendocrine secretions, or neurohormones. For example, hypothalamic corticotrophin releasing hormone (CRH) is discharged into the hypothalamo-hypopheseal portal capillaries during exercise and other forms of stress. Additionally, some cells also disseminate chemical messengers by a combination of endocrine and neural transmission. For example, sympathetic nerves communicate largely by neurotransmission of noradrenaline (NA), but some NA also has neuroendocrine effects resulting from spill-over into general circulation, as is the case during exercise (Borer, 2003).

2.2.3. Relationship between endocrine and psychological variables of fatigue

Similarly to exercise, psychological stressors that elicit anxiety, anger, aggression, or fear can induce secretion of A and C, and other hormones including GH, PRL and TSH (Borer, 2003). Psychological stress of competition in addition to heavy training loads may also predispose athletes to a greater risk of upper respiratory tract infections, particularly in elite athletes engaged in contact sports (Cunniffe et al., 2010). Some studies have also reported the effects of psychological stress on hormone responses, with chronic stress depressing T levels (Aakvaag et al., 1978; Matsumoto et al., 1970).

Chemical messengers are secreted prior to and during exercise which are psychologically and physiologically arousing (Borer, 2003). In elite sport, this is demonstrated by consistent anticipatory increases in levels of C prior to competition, which appears to be a prerequisite for peak performance (Crewther et al., 2011). Additionally, studies in combat sports such as judo have reported that T concentrations are related to

behavioural aspects of fighting style, including number of offensive, or attacking manoeuvres (Salvador et al., 1999; Suay et al., 1999; Salvador, Gonzalaez-Bono & & Serrano., 2001). Furthermore, the responses of T and C were related to success in sports such as weightlifting (Passerlergue, Roberts & Lac, 1995; Passerlergue & Lac, 1999), wrestling (Kraemer et al., 2011) and tennis (Mazur & Lamb, 1980; Booth et al., 1989). Similarly, studies suggesting that home competition venues show differential sex steroid profiles compared to away venues (Carre et al. 2006; 2009; Neave & Wolfson, 2003) adds another dimension to understanding the influence of pre-competition hormones on subsequent performance in elite sport.

Mood score data has been used to quantify the behavioural responses to the competition outcomes. For example, Gonzalez-Bono et al., (1999) reported that negative mood is significantly enhanced in losers, while winners showed a better appraisal of team performance and higher internal attribution. Additionally, the T response correlated negatively with external attribution in winners and positively in losers, and suggested that T fluctuations are related to individual contribution and to the causes attributed. Interestingly, Booth et al. (1989) reported that the most positive pre-competition mood was related to the highest pre-competition T levels, which was further associated with competition success in tennis. Collectively, these studies indicate that psychological cues including game venue, outcome and individual performance provide a strong stimulus for the acute release of steroid hormones in response to competition.

However, few studies have been carried out in mood response during the days following competition. Of those that have, McLean et al., (2010) reported that perception of fatigue, overall well-being and soreness were significantly reduced in the 48 h period following elite rugby league matches during 3 microcyles of varying lengths. Similarly, Elloumi et al. (2008) reported the relative state of tiredness at the end of the season was greater than at the start, with decreases in T and T:C during training. However, consistent

increases in C were observed in response to competition but the response of T was largely determined by match stake.

Collectively, it appears that many physiological and psychological stimuli are involved in the secretion of the steroid hormones in response to competition, particularly in those situations with higher match importance. Underpinning these findings may be the stimulation of catecholamines, CRF, and ACTH which function to widen the focus of attention and establish sensory readiness to external cues in preparation for competition (Borer, 2003).

2.2.4. Hormone measurement

In the last few years saliva analysis has become a useful method of choice for steroid hormone analysis (Gatti, 2010). Serum constituents, which are not part of the normal salivary constituents (i.e. steroid hormones), can reach the saliva via passive diffusion. These molecules must diffuse passively across five barriers including the capillary wall, interstitial space, the basal cell membrane of the acinus cell or duct cell, the cytoplasm of the acinus or duct cell and the luminal cell membrane (Gatti, 2010; Kaufman & Lamster, 2002; Groschl, 2008). This mechanism is directly applied to lipid-soluble compounds such as unconjugated steroid hormones including T and C.

The use of saliva offers many benefits for assessing steroid hormones in athletes, because saliva collection is non-invasive, stress-free and enables real-time repeated sampling where blood collection is often undesirable or difficult (Crewther & Cook, 2010). Moreover, the salivary T and C concentration measures have correlated (r = 0.6-0.97) with blood

hormones, especially the free hormone that initiates the biological response at target tissue (Raff, Homar & Skoner, 2002; Turkes et al., 1980; Westermann, Demir & Herbst, 2004) and the bio-available hormone that is potentially available to tissue (Crewther & Cook, 2010). Given the correlation between serum and salivary hormonal values, studies have been carried out to evaluate the influence of physical exertion on salivary hormone levels. In response to exercise the T and C changes are often greater than blood-derived (total) measures, possibly reflecting the free and bioavailable hormones.

Radioimmunoassay (RIA) and enzyme-immunoassay (EIA) are two of the most common methods of salivary hormone analysis. The EIA measurement of salivary T and C concentrations have also correlated strongly ($r = 0.96 - 0.99$) with RIA in non-athletic populations (Raff, Homar & Skoner, 2002; Turkes et al., 1980; Westermann, Demir & Herbst, 2004). A study by Crewther & Cook (2010) examined the T and C concentrations of weightlifters before and after 4 workouts with the aim to validate a commercially available EIA kit against the criterion RIA method. Moderate to strong relationships between EIA and RIA were revealed for both T ($r = 0.72$) and C ($r = 0.96$), and correlations between pre and post workout samples for T ($r = 0.76 - 0.78$) and C (0.96 - 0.97) were also reported which is in agreement with previous research in non-athletic populations (Raff, Homar & Skoner, 2002, 2003; Turkes et al., 1980; Westermann, Demir & Herbst, 2004). The authors attributed the stronger correlation for C (versus T) to differences in absolute concentrations, the assay specifications (e.g. sensitivity, specificity) for each hormone, as well as hormone pharmacokinetics (e.g. diffusion rate, metabolism) within the salivary glands. Nevertheless, it was concluded that a commercially available EIA kit provided valid measures of salivary T and C concentrations in athletic groups. Moreover, different analysis methods provide different absolute values, but exhibit high relative validity which is useful in tracking relative changes in hormonal status (Crewther & Cook, 2010).

2.3.0. Responses to real vs. simulated competition

Previous research has highlighted the fact that the responses to exercise protocols carried out in controlled laboratory environments can vary significantly from those achieved in actual elite competitions (Elloumi et al., 2003) in some key physiological variables (e.g. CK).

2.3.1. Muscle damage

Particular attention has been paid to the [CK] responses in laboratory and field-based environments. For example, studies examining responses to rugby union competition by Smart et al. (2008) and Takarada (2003) produced mean [CK] of 926.8 ± 183.7 IU and 1081 ± 159 U/L respectively. Similarly, concentrations of approximately 1000 IU/L have been demonstrated in response to competitive soccer, whereas American football has shown post-match concentrations of approximately 330 $U \cdot L^{-1}$ (Kraemer et al., 2009). However, responses to twice-daily practices in American football have been shown to exceed 5000 ($U \cdot L^{-1}$) (Ehlers et al., 2002). In addition, two studies by McLellan et al. (2010; 2011) reported peak [CK] at 24 h following rugby leagues matches with values of 889.23 ± 238.27 $U \cdot L^{-1}$ and 941 ± 392 $U \cdot L^{-1}$, where [CK] remained elevated for up to 120 h post-match. Additionally, a study by Takahashi et al. (2007) observed peak [CK] of 508.8 ± 204.2 IU/L following two matches with a four hour break between games. Similarly, Ehlers et al. (2002) demonstrated extremely high levels as a result of large volumes of blunt trauma and high intensity SSC movements undertaken by players in a training camp.

Previous studies have also investigated the responses of simulations aiming to replicate the physical demands of competitive settings, and compared them to real competition settings. For example, Magalhaes, Rebelo, Oliveira, et al. (2010) compared physiological measures between the Loughborough Intermittent Sprint Test (LIST) and a competitive soccer match. Significant increases in [CK] were reported after the competitive and LIST trials, both peaking at 24 h. Although not significant, higher [CK] levels were observed following the soccer match (~ 1200 $U \cdot L^{-1}$) compared to the LIST (~ 1000 $U \cdot L^{-1}$) protocol, which may indicate a higher degree of muscle damage as a result of competition. Similar physiological [CK] ranges have also been reported in other studies, such as Ascensao, Leite, Rebelo, Magalhaes, & Magalhaes (2010), where recovery interventions were investigated following a soccer match. It is also interesting to note that [CK] values are similar between activities combining blunt trauma and high intensity SSC movements (such as rugby union) combined, and activities involving high intensity SSC type alone. Furthermore, the study noted that some key aspects of soccer specific high intensity SSC activities and blunt trauma, such as jumping, running backwards and tackling were omitted from the protocol. In contrast to this, the LIST protocol included deceleration type movements utilising eccentric muscle contractions which increase susceptibility to muscle damage.

Similarly, Twist & Sykes (2011) examined [CK] responses to a simulated rugby league match with values reaching ~ 400 $IU \cdot L^{-1}$. The simulation in this study attempted to include all aspects of rugby league match play, including contact (via the use of "down-up" movements). The authors noted the lower values in comparison to competitive match play which was accounted for by the absence of true contact in this simulation, therefore not replicating the physical collisions experienced by players during competition. More recently, Bendiksen, Bischoff & Randers et al. (2012) aimed to evaluate the use of a multi-faceted simulated soccer game called the Copenhagen Soccer Test (CST) elicited similarities in

physiological loading as a competitive game. This test consists of 2 x 45 min field tests including repeated 5 min bouts representing 152, 171, 69, 41, 55, 40, 30 and 23 m of walking (6 km.h-1), jogging (8 km.h-1), low (~12 km.h-1), moderate (~ 15 km.h-1), high speed running (18 km.h-1), sprinting (>25 km.h-1), backwards running (~10 km.h-1) and backwards/sideways sliding (~8 km.h-1), respectively with bouts split into high, medium or low intensity bouts with technical skill tests included. Although there were no significant differences in physiological responses between conditions, the subjects used in this protocol were only second and third division players, which could suggest that game intensities at this level are not comparable to that of the elite game. Additionally, circadian fluctuations were not accounted for, as the CST was carried out in the morning, whereas the competitive match was performed in the afternoon.

Howatson & Milak (2009) used a repeated sprint protocol of 15 x 30 m with a 10 m deceleration zone with a 60 s recovery between sprints. Significant increases in [CK] were observed compared to baseline values, with a peak at 24 h (776 ± 312 $IU \cdot L^{-1}$). [CK] was also significantly elevated at 48 and 72 h. However, these [CK] levels are substantially lower than those observed in the competition based studies (Smart et al., 2008; McLellan et al., 2010; McLellan et al., 2011; Takahashi et al., 2007). Furthermore, the repeated sprint protocol fails to replicate the frequent occurrence of contact and collisions involved in rugby matches through tackling, which plays a considerable role in the [CK]response (Hoffman et al., 2002; Takarada, 2003). Therefore, it is possible that these protocols may under predict the levels of muscle damage in comparison to field sports such as soccer and rugby.

Furthermore, some studies have investigated the [CK] response to laboratory based protocols, usually involving restricted jumping activities on a sled ergometer. [CK] values that are notably lower than those obtained from competitive settings have been reported from these studies (Twist & Eston, 2005; Dousset et al., 2007; Chatzinikolaou, Fatouros,

Gourgoulis, et al., 2010). However, an exception to this is a study by Skurvydas, Sipaviciene, Krutulyte, et al., (2006). This is possibly a result of localisation of muscle damage and exercise to the lower limbs through a jumping protocol that has minimal involvement on the upper limbs or the effects of blunt trauma. Collectively, these studies indicate substantial differences in muscle damage responses between simulated and real competition.

2.3.2. Metabolic

Metabolic markers such as lactate have also been investigated during laboratory based (Dousset et al., 2007; Chatzinikolaou et al., 2010), rugby simulation protocols (Roberts et al., 2010) and field based rugby matches (Docherty, Wenger, & Neary, 1998; McLean, 1992; Deutsch et al., 1998; Alvear-Ordenes, Garcia-Lopez, Paz, & Gonzales-Gallego, 2005). For example, the Bath University Rugby Shuttle Test (BURST) (Roberts et al., 2010) produced lactate levels of 4.5 $mmol \cdot L^{-1}$, which are lower compared to competition studies. For example, McLean (1992) reported lactate values of 6.7 ± 1.6 mmol·L and 5.9 ± 1.1 mmol·L for a number 8 and fly half respectively, throughout the course of a rugby match. Similarly, Deutsch et al. (1998) reported rugby match lactate concentrations of 6.6 mmol·L for forwards and 5.1 mmol·L for backs in a group of elite under-19 players. In contrast to this, Takarada, 2003 reported lower lactate levels of 3.3 mmol·L immediately post-match, which may be accounted for by a low intensity passage of play in the closing stages and subsequent lactate metabolism prior to sampling. The lower exercise intensity in the BURST protocol, as indicated via lower lactate levels (Roberts et al., 2010), may suggested poor ecological validity, and fails to replicate the high intensity and unpredictable nature of elite contact competition environments.

2.3.4. Endocrine

Marked differences in hormonal responses are also apparent between simulations, laboratory based protocols and field based competition. In particular, a consistent and acute pre-competition, anticipatory rise in C is observed, likely stimulated by additional psychological pressure, with higher external stress in comparison to simulations, training or laboratory based testing (Suay et al., 1999; Passelergue & Lac, 1999; Filaire et al., 1997; Filaire et al., 2001; McLellan et al., 2010; McLellan et al., 2011). It has been demonstrated that C demonstrates consistent fluctuations in response to competition stress, whereas the response of T tends to show higher variability (Cormack et al., 2008a; McLean et al., 2010; Kraemer et al., 2009). For example, Haneishi et al., (2007) examined the response of T and C in a group of NCAA division I female soccer players. A significant post-match increase in C (250 % for starters and 140 % for non-starters) was observed compared to no significant change in C before and after a training session (Figure 3.0).

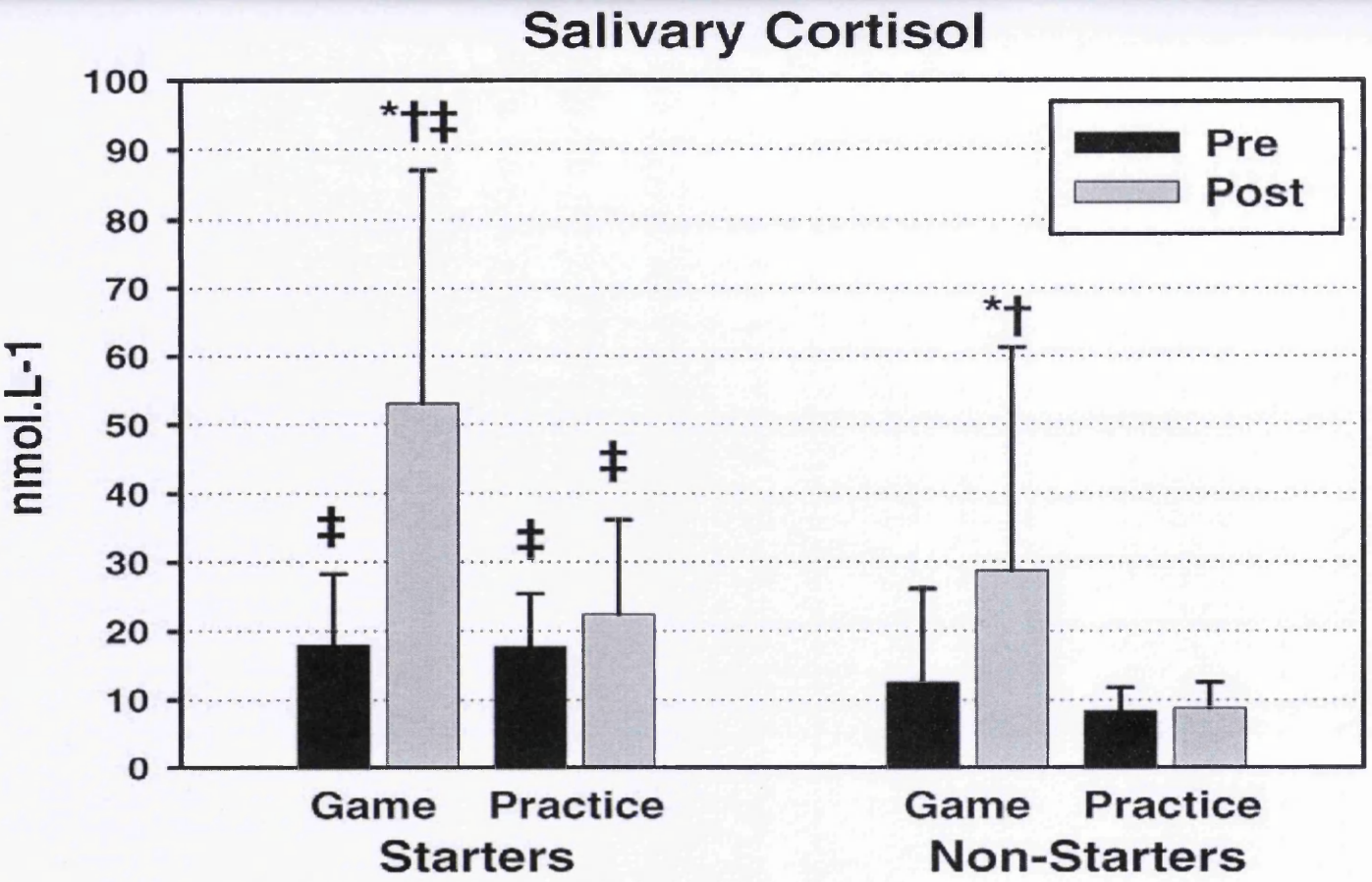

Figure 3.0: Salivary cortisol concentrations (Mean ± *SD*; nmol·L^{-1}) in response to a competitive game and a typical practice for both starters (n = 10) and nonstarters (n = 8) on a female collegiate soccer team. * Different from pregame, † Different from post practice, ‡ Different from nonstarters ($p < 0.05$) (Haneishi et al., 2007).

Similarly, a study by Passelergue & Lac (1999) and later replicated by Crewther et al. (2011) both reported significantly higher C concentrations (> 100%) in elite weightlifters at all time points on the day of a competition when compared to a simulated weightlifting competition. Moreover, the performance outputs of these athletes during the real competitions were found to be superior to that achieved during the simulated weightlifting events. Collectively, these studies highlight substantial differences in hormone responses, which are possibly related to performance level (Crewther et al., 2011), so physiological responses with subsequent effects appear quite difference under each condition. This further highlights the importance of collecting data from a competitive situation given that results obtained under laboratory conditions, even if they are exhausting, are not comparable to competitive situations which create a greater physiological and psychological demand (McLellan et al., 2011).

Overall, it appears that simulations, training or laboratory settings are unable to produce the hormonal responses of real competition due to lower pressures on the athletes, and other external factors involved in competitive atmospheres, such as the crowd and venue location (Carre, 2006; 2009). In support of this, Forster et al. (1993) stated that although intermittent tests leading to volitional exhaustion may induce a degree of physiological strain, it is questionable that this is comparable to actual competition.

To summarise, there are differences in the data collected in laboratory settings that use exhaustive exercise protocols, simulated competition and training situations, compared to elite competitive field-based settings. Parameters showing these differences include markers of neuromuscular, endocrine, muscle damage, biochemical and inflammatory function. Thus, methods attempting to simulate elite competition, or where the aim is to gather surrogate information on elite competition, will produce data with poor ecological validity.

2.4.0. Mood disturbance as a marker of fatigue

The majority of research in psychological markers of fatigue has predominately focussed on over-training and under-recovery, with links to staleness, burnout and the over-training syndrome (Hooper, Mackinnon & Hanrahan, 1997; Kellman & Gunther 2000; Raedeke, 1997). This research generally assesses an athlete's recovery based on parameters such as perceived stress, behavioural symptoms associated with fatigue (e.g. insomnia), current mood, or a combination of the three. For perceived stress and mood there are specific validated self-report measures/inventories such as the Recovery-Stress Questionnaire for Athletes (RestQ-Sport; Kellman & Kallus, 2001) which is the most commonly used self-report measure of perceived stress. For mood, the two most commonly used questionnaires are the Profile of Mood States (POMS; McNair, Lorr & Droppleman, 1971) and the Brunel Mood Scale (BRUMS; Terry, Lane & Fogarty, 2003) which show a dose-response relationship with overtraining (e.g. Berger et al., 1999; Kellman & Gunther, 2000).

A few studies have been performed assessing mood in varying contexts using the Brunel Mood Scale. For example, Martinnen et al. (2011) reported significantly higher values for confusion in a group of collegiate wrestlers following self-selected weight loss in preparation for competition. Selwell et al. (2006) also used this questionnaire to assess anger, calmness, confusion, depression, fatigue, tension and vigour before 4 home and 4 away matches with the aim to assess the effects of game location on pre-competition mood score. Additionally, Lane, Lane & Firth (2002) used the BRUMS questionnaire to examine post competition depressed mood in a group of 195 distance runner following 10 mile race, and reported that depressed mood is related to the intensity of other mood dimensions assessed by POMS, and moderates relationships between scores and performance satisfaction. Overall,

these studies suggest that the BRUMS questionnaire may be utilised as a tool to assess mood disturbance in various groups of athletes where data collection must be quick, as well as valid and reliable.

The POMS requires athletes to rate their current feelings or mood against a list of 65 words, and generally shows reduced vigour and increased tension, depression, anger, fatigue and confusion during periods of heavy training. For example, Schmiki et al. (2011) reported increased anger and depression in athletes showing prolonged decrements in performance as a result of non-functional over-reaching. In a similar fashion to the POMS, Main & Grove, 2009 developed a 22-item Training Distress Assessment Model (TDAM) which combines measures of perceived stress, mood and behavioural symptoms, and has since been used successfully to monitor training overload in elite rowers (Main et al., 2010).

One study by Suzuki et al. (2004) used POMS to assess the psychological impact of a rugby match in a group of college rugby players, with players split into low intensity and active rest (normal daily activities) recovery groups. Depression and fatigue scores were significantly higher immediately post-match in both groups, with no change in tension at this point. However tension was significantly lower in the active rest group two days following the match, with no changes in anger or vigour scores in either group. Another study investigated mood state as a function of an American Football season (Newby & Simpson, 2003), with scores analysed to assess whether mood varied in relation to the outcome of the previous week's game. It was suggested that although subject's overall mood state was not influenced by game outcome, scores on tension, vigour, fatigue and confusion were related to game outcome. A study by Nicolls, Backhouse, Polman & McKenna (2009) examined fluctuations in a number of psychological constructs over a 3 day period in response to a single rugby union game. Interestingly, players reported many stressors being "worse than normal" on the day following a match, compared to the day before the match, characterised

by feelings of tiredness and fatigue, which support the findings of the present study. However, the study by Nicholls further reported an "unpleasant, low activation state across" the three analysis days and suggested these subjects were in an overtrained state (as measured by Daily Analysis of Life Demands Questionnaire, and the Activation Deactivation Adjective Check List).

Although other psychological factors such as cognition and emotion are involved player's moderating performance and recovery, and it is generally agreed that mood and emotion are separate constructs. However,, these studies suggest that exposure to stressful environments; particularly competition (Suzuki et al., 2004) may substantially affect various aspects of athlete's mood state, which is often also dependent on game outcome (Newby & Simpson, 2003).

Although these measures are valid and reliable from a research perspective, they are extremely long and time consuming for athletes to complete, and applied practitioners indicate that athletes generally resent completing long psychological inventories on a regular basis. This can result in coaches and additional support staff seeking methods of monitoring fatigue with bespoke short-form measures. To address this, questionnaires such as the Recovery-Cue (Kellmann, Botteril & Wilson, 1999) and the Brief Assessment of Mood (BAM) (Dean, Whelan & Meyers, 1990) have been developed as shortened versions of the RESTQ-Sport and POMS. The Recovery-Cue is a seven item questionnaire that measures perceived exertion, physical recovery, sleep quality, social recovery and self-regulation and is based on the RESTQ-Sport. Although research using this questionnaire is scarce, the authors have provided a description of two case studies where it was used successfully in an applied setting (Kellmann et al., 2002). The BAM is shortened version of the POMS, which instead of using multiple items per factors simply asks the participants to rate their mood based on six factors of the POMS (i.e. anger, tension, depression, vigour, fatigue, and confusion).

Although there are issues with concurrent validity using single item/factor questionnaires, in applied settings this needs to be balanced against time constraints of the coaches and athletes. Moreover, the literature appears to demonstrate that abbreviated questionnaires correlate well with full versions, and that these shortened versions are more suited to situations in which time constraints are an issue (LeUnes & Burger, 2000). Mood disruption, as measured via the BAM questionnaire has been assessed more frequently and successfully in relation to performance and training recovery (Morgan, Costill, Flynn, Raglin, O'Connor, 1988; Raglin, Eksten & Garl, 1995), compared to the Recovery- Cue questionnaire. Additionally, compared to the items on the Recovery-Cue that consists of multi-faceted constructs such as recovery, exertion, and self-regulation, mood is not a multi-faceted construct. For example, whereas recovery can be subdivided into physical and mental aspects, each of the mood states measured by the BAM is already the lowest denominator. Therefore, it could be argued that using multiple items to ensure content validity and reliability are redundant. For example, if the question is only asked once (e.g. "how angry do you feel?") it is more likely to generate a true response than multiple versions of the same question, as per the POMS (Robins, Hendin & Trzensiewski, 2001). Therefore single item questionnaires such as the BAMS may be superior for applied practice. However, no previous studies have used this questionnaire to assess mood disruption in a group of elite or recreational athletes (Table 2.0).

Table 2.1: Studies examining mood response to intense training and competition

Name	Subjects	Aim	Methods	Results
Morgan et al., 1987	200 F 200M swimmers	Summarise a 10 year monitoring period of mood states in competitive swimmers	Monitoring via POMS questionnaire at intervals ranging from 2-4 weeks during individual seasons from 1975-1986	Mood state disturbances ↑in a dose response manner as training stimulus↑. Mood disturbance returned to baseline levels with ↓ in training load
Fry et al., 1994	5 well trained men	Examine immune function and psychological reactions to acute overtraining	Two intensive interval training sessions per day followed by 5 days active recovery. Venous blood obtained and abbreviated POMS questionnaire completed on days 1, 6, 11 and 16 of study. Performance assessed on days 1, 11 and 16 via treadmill test to exhaustion at 18km/h at 1% grade.	Overtrained state accompanied by severe fatigue, immune system deficits, mood disturbance, physical complaints, sleep difficulties and ↓appetite. Mood state changed with respect to significant ↑ in fatigue after first 5 and upon conclusion of training. Significant ↓ in vigour and significant ↑ in total mood disturbance on conclusion of training. Mood returned to baseline during recovery but feelings of fatigue and immune system deficits persisted throughout.
Nix et al., 2012	25 recreationally active men	Examine differences between 3 recovery methods following muscle damaging activity relative to maximal isokinetic contractions, perceived muscle soreness and psychological mood states	Subjects assigned to anti-gravity treadmill, conventional treadmill or static stretching recovery methods, following downhill running protocol. Isokinetic muscle strength, muscle soreness and POMS assessed at baseline, 15 min, then 3, 24, 48, 72 and 96 h post exercise.	Muscle soreness significantly ↑at 15 min, 3, 24 and 48 h, with significant ↓ thereafter. Significant ↑ in mood disturbance from baseline to 48 h, with significant ↓ thereafter. Significantly ↑ mood disturbance in treadmill recovery group vs. anti-gravity treadmill group.

Slivka et al., 2010	8 M trained cyclists	Impose a period of intensified training to determine if commonly used markers of overtraining parallel changes in physical performance	21 days of intensified training with salivary IgA, T and C, 1 hour time trial performance HR response and POMS analysed over 21 days	POMS category vigour significantly ↓ from day 1 to 4 and remained lower throughout. No other changes in any parameters.
Deilan et al., 2012	15 elite adolescent wrestlers	Effects of intensive training on salivary T, C, α-amylase, and mood	Saliva sampling and Brahms mood questionnaire completed over 3 week period	No changes in mood or saliva hormones
Suzuki et al., 2011	129 young elite athletes, 77 soccer players, 52 middle distance runners, control athletes.	Investigate whether field performance tests can distinguish between non-functional over-reaching (NFO) and controls	POMS, ATCH and C measured in laboratory	↑ anger and depression in athletes showing prolonged decrements in performance as a result of non-functional over-reaching
Suzuki et al. 2004	15 college rugby players	Compare difference in recovery between rest and active recovery following rugby union match	7 performed daily activities, 8 performed low intensity exercise following match. Blood sampling and POMS completed before, immediately after, then 24 and 48 h post-match.	↑ muscle damage, ↓neutrophils and ↑ mental fatigue immediately after match. ↑ depression and fatigue in both groups immediately post-match, no change in tension. Tension significantly ↓ in active rest group at 48 h post-match. No change in anger or vigour throughout.
Newby & Simpson, 2003	University American Football Players	Examine whether mood varies as a function of season	POMS collected from players each week prior to Saturday match, with scores analysed to assess whether mood varied as a function of the previous week's outcome.	Tension, vigour, fatigue and confusion related to game outcome
Mashiko et al. 2004	37 university rugby players	Relationship between physical and mental fatigue following a rugby match with respect to	POMS, blood samples and serum collected before and after a match	Positive correlations between blood markers of muscle damage and change in TMD score in forwards. Positive correlations between lipid based parameters and changes in POMS score.

		position played.		
Elloumi et al. 2008	Elite male rugby players	Hormonal, physical and psychological parameters following a season.	Fitness and relative tiredness evaluated at the end of the season	Psychological conditions remained stable throughout season.
Coutts & Reaburn, 2008	20 semi professional rugby league players	Assess whether RESTQ-Sport can monitor perceived stress and recovery during intensified training	Players divided into normal training and intensified training groups for 6 weeks followed by 7 day taper. Players assessed by multi-stage fitness test and RESTQ-Sport before, and every 2 weeks and upon completion of taper.	RESTQ-Sport positively correlated with scores in fatigue, disturbed breaks, and general stress.

2.4.1 Relationship between psychological and endocrine variables of fatigue

Similar to exercise, psychological stressors that elicit anxiety, anger, aggression, or fear can induce secretion of A and C, and other hormones including GH, PRL and TSH (Borer, 2003). Psychological stress of competition in addition to heavy training loads may also reduce production of salivary IgA, suppress NK cell activity and increase the incidence of upper respiratory tract infections, particularly in elite athletes engaged in contact sports (Cunniffe et al., 2010). Some studies have also reported the effects of psychological stress on hormone responses, with chronic stress depressing T levels (Aakvaag et al., 1978; Matsumoto et al., 1970).

In environments which cause significant psychological and physical stress, such as competition, interactions between mind and body are reciprocal. Chemical messengers are secreted prior to and during exercise which are psychologically and physiologically arousing (Borer, 2003). More specifically to elite sport, this is demonstrated by consistent anticipatory increases in levels of C prior to competition (Salvador et al., 1999; Suay et al., 1999; Salvador, Gonzalaez-Bono & & Serrano., 2001). Additionally, studies in combat sports such as judo have reported that T concentrations are related to behavioural aspects of fighting style, including number of offensive, or attacking manoeuvres (Salvador et al., 1999; Suay et al., 1999; Salvador, Gonzalaez-Bono & & Serrano., 2001). Furthermore, the responses of T and C have also been related to success in sports such as weightlifting (Passerlergue, Roberts & Lac, 1995; Passerlergue & Lac, 1999), wrestling (Kraemer et al., 2011) and tennis (Mazur & Lamb, 1980; Booth et al., 1989).

Mood score data has predominantly been collated on immediate responses to the competition outcome, rather than the following days of the post competition recovery period.

For example, Gonzalez-Bono et al., (1999) reported that negative mood is significantly enhanced in losers, while winners showed a better appraisal of team performance and higher internal attribution. In addition, T responses correlated negatively with external attribution in winners and positively in losers, and suggested that T fluctuations are related to individual contribution and to the causes the success is attributed to. Interestingly, Booth et al. (1989) reported that the most positive pre-competition mood was related to the highest pre-competition T levels, which was further associated with competition success in tennis. Collectively, these studies indicate that psychological cues including game venue, outcome and individual performance provide a strong stimulus for the acute release steroid hormones in response to competition.

Few studies have been carried out in mood response during the days following competition. Of those that have, McLellan et al. (2010) reported that perception of fatigue, overall well-being and soreness were significantly reduced in the 48 h period following elite rugby league matches during 3 microcyles of varying lengths. Similarly, Elloumi et al. (2008) reported the relative state of tiredness at the end of the season was greater than at the start, with decreases in T and T:C during training. However, consistent increases in C were observed in response to competition but the response of T was largely determined by match stake.

Collectively, it appears that many psychophysiological stimuli are involved in the secretion of the steroid hormones in response to competition. Underpinning these findings may be the stimulation of catecholamines, CRF, and ACTH which function to widen the focus of attention and establish sensory readiness to external cues in preparation for competition, with post competition soreness and fatigue, and possibly disruptions in mood associated with the acute phase inflammatory response resulting from muscle damage (Borer, 2003; McLellan et al., 2011).

CHAPTER THREE

3.0 Methods

3.0. Methods

3.1.0. Experimental approach to the problem

The present study examined the time-course and relationships of various neuromuscular, endocrine and mood parameters in the recovery period following a single elite rugby union match. Baseline saliva samples, countermovement jump (CMJ) and mood disturbance (Brief Assessment of Mood Questionnaire) were collected 36 hours (h) before a professional rugby union match, with these measures repeated at 12, 36 and 60 h post-match. Testing was completed at the same time of day to avoid the effects of the circadian rhythm on hormonal concentrations (Beaven, Gill, & Cook, 2008) and performance (Cappert, 1999). Subjects completed a standardised warm up, before performing 3 maximal effort CMJs on a portable force platform (Kistler Instruments Ltd, Farnborough, UK), from which variables including peak power output (PPO), jump height (JH) and Flight Time:Contraction Time (FT:CT) were derived and utilised as markers of neuromuscular function. Saliva samples were assayed for T and C, and the T:C ratio, to represent endocrine markers of anabolic/catabolic state pre and post-match.

3.1.1. Subjects

Fourteen elite male Rugby Union players were recruited for this study ([mean ± *SD*]) (24.91 ± 4.35 yrs, 1.85 ± 0.10 m, 105.18 ± 12.31 kg. All players were full-time members of a professional squad and engaged in a full-time training and competition schedule. Participants played for a team in the South of Wales, UK, competing in the professional Celtic League

and European Cup competitions. Written informed consent was provided by all participants in the study, which was approved by the University Ethics Committee. Participants were considered healthy and injury-free at the time of this study. Prior to any data being collected, subjects attended a session outlining the purpose of the study and the procedures involved.

3.1.2. Testing schedule

Testing was completed over a period of 96 h with baseline data collected 36 h prior to, and post-game data collected at 12, 36 and 60 h following the match (McLellan, Lovell & Gass, 2011; Cormack et al., 2008). Time points of data collection were kept consistent with all procedures completed within one hour, and the game measured was scheduled for an evening kick-off. Upon arrival, participants provided a 2 ml saliva sample via passive drool, followed by completion of the Brief Assessment of Mood (BAM) questionnaire. Participants then performed a 5 min standardized warm up consisting of jogging and dynamic stretching, before completing 3 maximal effort CMJs on a portable force platform. Having stood in an initial stationary phase of 5s (for an accurate determination of body mass), participants dipped to a self-selected depth and exploded in an upward direction in attempt to gain maximal height. Participants landed back on the force platform, and arms were kept akimbo throughout the entire movement in order to isolate the lower limbs Aragon-Vargas & Gross, 1997; Hatze, 1998). This protocol was replicated at the post-match data collection time points, and subjects rested throughout the 60 h post-match period with no training, or recovery interventions taking place during this time.

3.2.0. Hormone sampling and analysis

Participants were instructed to avoid eating, drinking and gum chewing for 30 min as well as brushing teeth and using dental floss 15 min prior to giving saliva samples. These restrictions were implemented to minimise any risk of blood contamination in saliva. Saliva samples were collected via passive drool into 15 mL containers (Alpha Laboratories, Eastleigh, Hampshire) and stored at -80°C until assay. Following 10 min of centrifugation at 3000 G, samples were analysed in duplicate for T and C concentrations using an enzyme immunoassay kit (IBL International, Hamburg, Germany). Standard curves were constructed per the manufacturer's instructions and additional internal standards were included. The minimum detection limit for the T assay was 2.0 $pg \cdot mL^{-1}$, with intra- and inter-assay coefficients of variation of 2.0 - 9.8%. The C assay had a detection limit of 0.3 $ng \cdot mL^{-1}$ with intra- and inter-assay coefficients of variation of 3.5 - 8.7%. Assay plates were read using a micro-plate reader (Biohit BT800, Helsinki, Finland). Salivary hormone concentrations represent the unbound, biologically active fraction of steroid hormones (Groschl, 2008; Kauffman & Lamster, 2002). Salivary concentrations of T and C have shown very high correlations with serum steroid levels ($r > 0.97$) (Peters, Walker, Riad-Fahmy & Hall, 1982; Vining, McGinely, Maksvytis, & Ho, 1983; Vittek, L'Hommedieu, Gordon, Rappaport, & Southern 1985).

3.3.0. Force platform and CMJ assessments

For the measurement of lower body power, the ground reaction force (GRF) time history of a CMJ was completed on a Kistler portable force platform with built-in charge amplifier (type 928666AA, Kistler Instruments Ltd, Farnborough, UK). Arms were kept akimbo throughout the movement to ensure the isolation of the lower limbs (Aragon-Vargas & Gross, 1997; Hatze, 1998). Having completed a standardised warm up, 3 CMJs were then completed each subject, by bending the knees to a self selected depth before exploding in the upward direction with the aim of achieving maximum height, before landing back on the force platform. Bodyweight (BW) was determined during an initial stationary stance phase for at least 5 s, and a sample rate of 1000 Hz was used for all jumps, with confirmation of the platform's calibration confirmed pre and post testing. The stretch shorten cycle (SSC) has been described as a valid and reliable method in the assessment of neuromuscular function (Marcovik, Dizdar, Jukic & Cardinale 2004; Moir, Button, Glaister & Stone, 2004). The SSC is utilised through a CMJ and may detect neuromuscular fatigue through neural, metabolic and mechanical elements collectively with the stretch-reflex activation (Komi, 2000).

3.3.1. Force platform data analysis

The vertical component of the GRF as the subject performed the CMJ was used in conjunction with the subject's BW to determine the instantaneous velocity and displacement of the subject's centre of gravity (CG) (Hatze, 1998). Instantaneous power was determined using the following standard equation:

$$\text{Power (W)} = \text{vertical GRF (N)} \times \text{vertical velocity of CG } (m \cdot s^{-1})$$

To determine the velocity of the subject's CG numerical integration was performed using Simpson's rule with intervals equal to the sample width. Prior to the calculation of the strip area, the subject's BW (as measured in the stationary phase) was subtracted from the GRF values. The area of the strip, of width equal to the sample rate, then represented the impulse for that time interval. Using the relationship that impulse equals change in momentum; the strip area was then divided by the subject's mass to produce a value for the change in velocity for the CG (it was assumed that the subjects mass remained constant throughout the jump). This change in velocity was then added to the CG's previous velocity to produce a new velocity at a time equal to that particular intervals end time. This process was continued throughout the jump. As this method can only determine the change in velocity, it was necessary to know the CG's velocity at some point in time. For this purpose, the velocity of the CG was taken to be zero prior to the initiation of the jump (during the period of body weight measurement) and specifically at the point identified as the start of the jump. The start point was defined as the time when the subject's GRF exceeded the mean ± 5 *SD* from the values obtained in the second (of the stationary body weight measuring phase) immediately prior to the command to jump, in a fashion similar to Vanrenterghem, DeClercq & Van Cleven (2001). Integration started from this point.

FT, defined as the duration for which the subject is airborne was calculated by subtracting the time of toes down (ttd) onto the force platform from the time of take off (tto).

$$FT = ttd - tto$$

Vertical displacement was determined by a second integration. The instantaneous velocity time history was numerically integrated (in the same way as described above) from the start point of the jump. The height (vertical displacement) of the CG at the start point of the jump was defined as zero. Jump height was then defined as the difference in the vertical

displacement of the CG, between take off (toes leave the force plate) and maximum vertical displacement achieved. Test-retest reliabilities (ICC's) for PO, and maximum jump height were; 0.979 and 0.890 respectively (Kilduff, et al ., 2008).

3.4.0 BAM Questionnaire and Scoring

Mood state was assessed using the modified BAM Questionnaire. This 6 item measure of mood is designed as a brief version of the Profile of Mood State (McNair et al ., 1971), and consists of a 5-point scale where players describe how they feel at that particular instant in 6 mood adjectives (anger, tension, vigour, confusion, fatigue and depression). This is often used in applied settings where quick, accurate measures of mood are required. This information was provided at the same time of day on each testing occasion. Total mood disturbance scores were calculated from subtracting the vigour score from the sum of the remaining five negative items with higher scores indicating elevated mood disturbance. BAM Mood Disturbance Scores have been highly correlated with POMS Total Mood Disturbance Scores ($r = 0.88$, $p < 0.01$; Dean et al ., 1990). Mood score data was collected from twelve of the fourteen individuals participating in this study.

3.5.0 Data analysis techniques

Following data screening for normality and homogeneity of variance, a one-way repeated measures analysis of variance (ANOVA) was applied to determine differences

between force platform variables, hormonal concentrations, and mood scores over time following the match. Where significant main effects of time were identified, data were further explored with Bonferroni corrected pairwise comparisons. Percentage changes from baseline at each time point are also presented. Statistical analysis was performed using SPSS software (version 16; SPSS Inc., Chicago, IL), with significance set at $P \leq 0.05$. Where significant differences have been identified, 95% confidence intervals are presented for an estimation of the population mean difference. Data are presented as mean ± SD.

CHAPTER FOUR

4.0 Results

4.0. Results

4.1.0. Neuromuscular Function

Repeated measures analysis of variance (ANOVA) revealed no significant time effects for BW [F (1.992, 24.984) = 3.233, $P \geq 0.05$, $ES = 0.204$] or jump height [F (1.716, 22.303) = 5.588, $P \geq 0.05$, $ES = 0.301$] post-match compared to baseline values. However, a significant time effect was observed for concentric peak power output (PPO) (W) [F (2.21, 28.68) = 13.454, $P \leq 0.05$, $ES = 0.509$]. Post hoc analyses revealed significantly lower values at 12 h (6100.4 ± 565.1 W vs. 5679.9 ± 588.6 W) ($P \leq 0.05$) and 36 h post-match (6100.4 ± 565.1 W vs. 5760.5 ± 638.5 W) ($P \leq 0.05$) compared with baseline values, with an average reduction of 6.82 ± 5.79 % and 5.67 ± 3.77 % respectively. Although values were not significantly different from baseline at 60 h, PPO was still depressed by 2.35 ± 3.65% at this time (6100.5 ± 565.1 W vs. 5949.7 ± 504.5 W) ($P \geq 0.05$).

No other significant time effects were observed between any other force platform variables. Force platform variables are summarized in Table 3.0, and pre and post-match force platform data are presented in Figure 3.1.

Table 3.0: Force platform variables obtained from CMJs at baseline, 12, 36 and 60 h post game.

	Baseline	12 h	36 h	60 h
Body Weight (kg)	105.2 ± 12.7	104.43 ± 12.0	105.6 ± 12.6	105.1 ± 12.3
Concentric Power (W)	6100.4 ± 565.1	5679.9 ± 588.6*	5760.5 ± 638.5*	5949.7 ± 504.5
Jump height (m)	0.39 ± 0.06	0.36 ± 0.07	0.37 ± 0.07	0.39 ± 0.07

Note: * indicates significant difference from baseline values ($P \leq 0.05$).

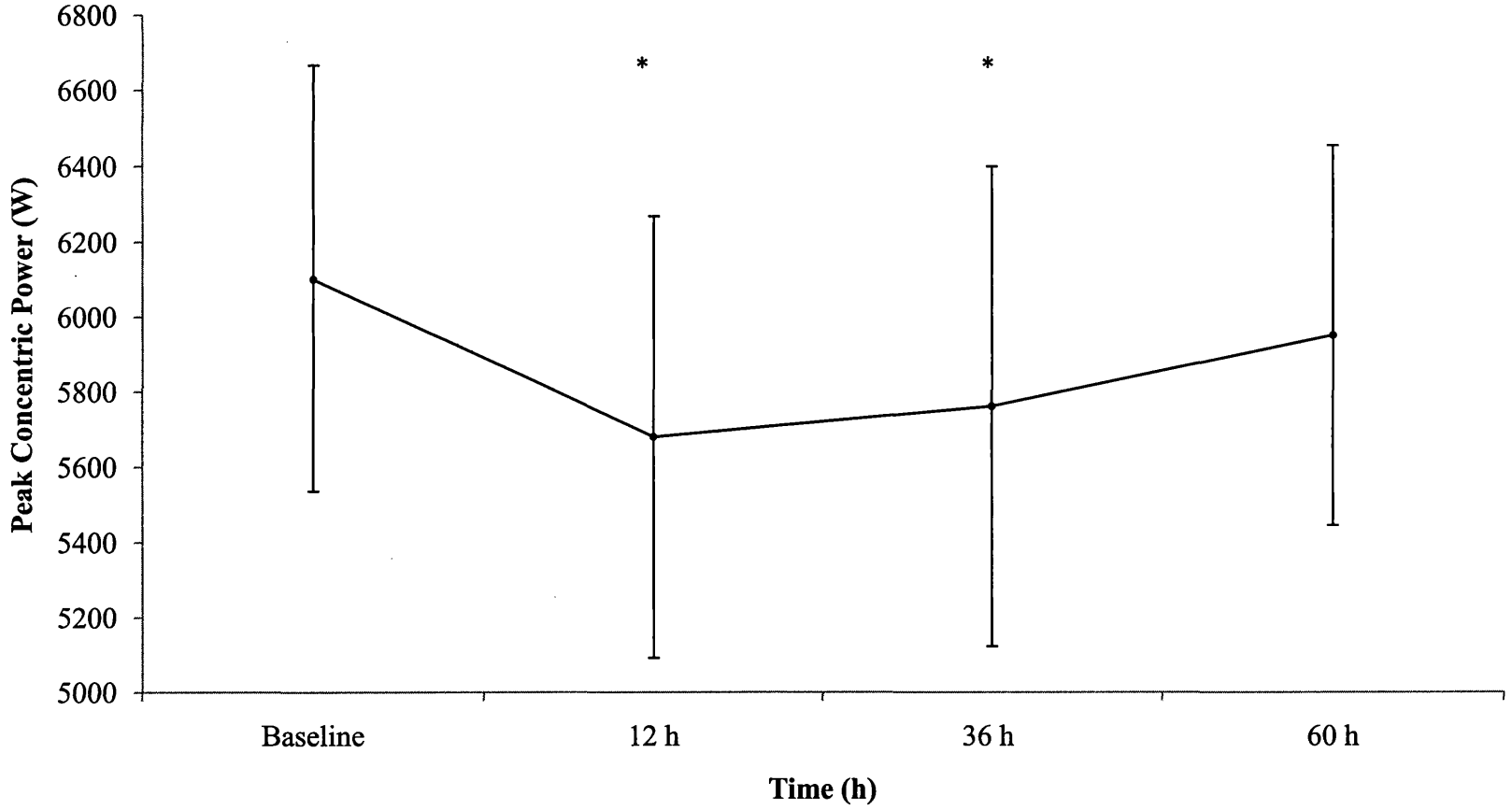

Figure 3.1: Peak concentric power at baseline, 12, 36 and 60 h post-match.

- Indicates a significant difference from baseline values ($P \leq 0.05$).

4.2.0 Salivary hormones

A significant time effect was observed for salivary T [F (2.7, 35.103) = 4.252, $P \leq$ 0.05, ES = 0.246*]*. Post Hoc analyses demonstrated a significant decline in salivary T concentrations 12 h post game (Baseline 213.6 ± 84.2 vs. 12 h 150.7 ± 56.1 pg·ml^{-1}; P < 0.05), but were similar at 36 h (172.60 ± 70.95 pg·ml^{-1}; P > 0.05) and 60 h (184.17 ± 73.98 pg·ml^{-1}; P > 0.05) post-match, with no significant differences in salivary T concentrations at these time points. Although not significantly different, salivary T concentrations were still depressed by 14.6 ± 34.0 % and 8.4 ± 8.4 % at 36 and 60 h respectively (Figure 4.1).

A significant time effect was also observed for salivary C concentrations [F (2.719, 35.348), = 5.507, $P \leq 0.01$, ES = 0.298]. Post hoc analyses indicated that salivary C increased from baseline at 12 h (Baseline 0.39 ± 0.09 vs. 12 h 0.59 ± 0.17 µg·dl^{-1}; P < 0.05) and 36 h (0.59 ± 0.20 µg·dl^{-1}; P < 0.05), but were similar at 60 h post-match (0.52 ± 0.21 µg·dl^{-1}; P > 0.05) compared to baseline values. Although not significant, C concentrations were still elevated by 34.6 ± 51.0 % at 60 h following the match (Figure 4.2).

Furthermore, a significant time effect was observed for T:C ratio [F (2.793, 36.305) = 11.367, $P \leq 0.01$, ES = 0.466]. Post Hoc analyses demonstrated significant decreases from baseline values at 12 h (Baseline 551.2 ± 219.3 vs. 12 h 265.6 ± 123.0; P < 0.05) and 36 h (310.0 ± 148.0; P < 0.05), before returning to baseline at 60 h post-match (399.0 ± 210.2; P < 0.05) (Figure 3.2). Salivary hormone results are summarized in Table 4.0.

Table 4.0: Salivary T ($pg \cdot mL^{-1}$) and C ($\mu g \cdot mL^{-1}$) concentrations and T/C ratio at baseline, 12, 36 and 60 h post game

	Baseline	12 h	36 h	60 h
T($pg \cdot mL^{-1}$)	213.6 ± 84.2	150.7± 56.1*	172.6 ± 70.9	184.2 ± 73.9
C ($\mu g \cdot dL^{-1}$)	0.39 ± 0.09	0.59 ± 0.17*	0.59 ± 0.20*	0.52 ± 0.21
T:C Ratio	551.2 ± 219.3	265.6 ± 123.0*	310.0 ± 148.0*	398.5 ± 210.2

Note: * indicates significant difference from baseline values ($P \leq 0.05$).

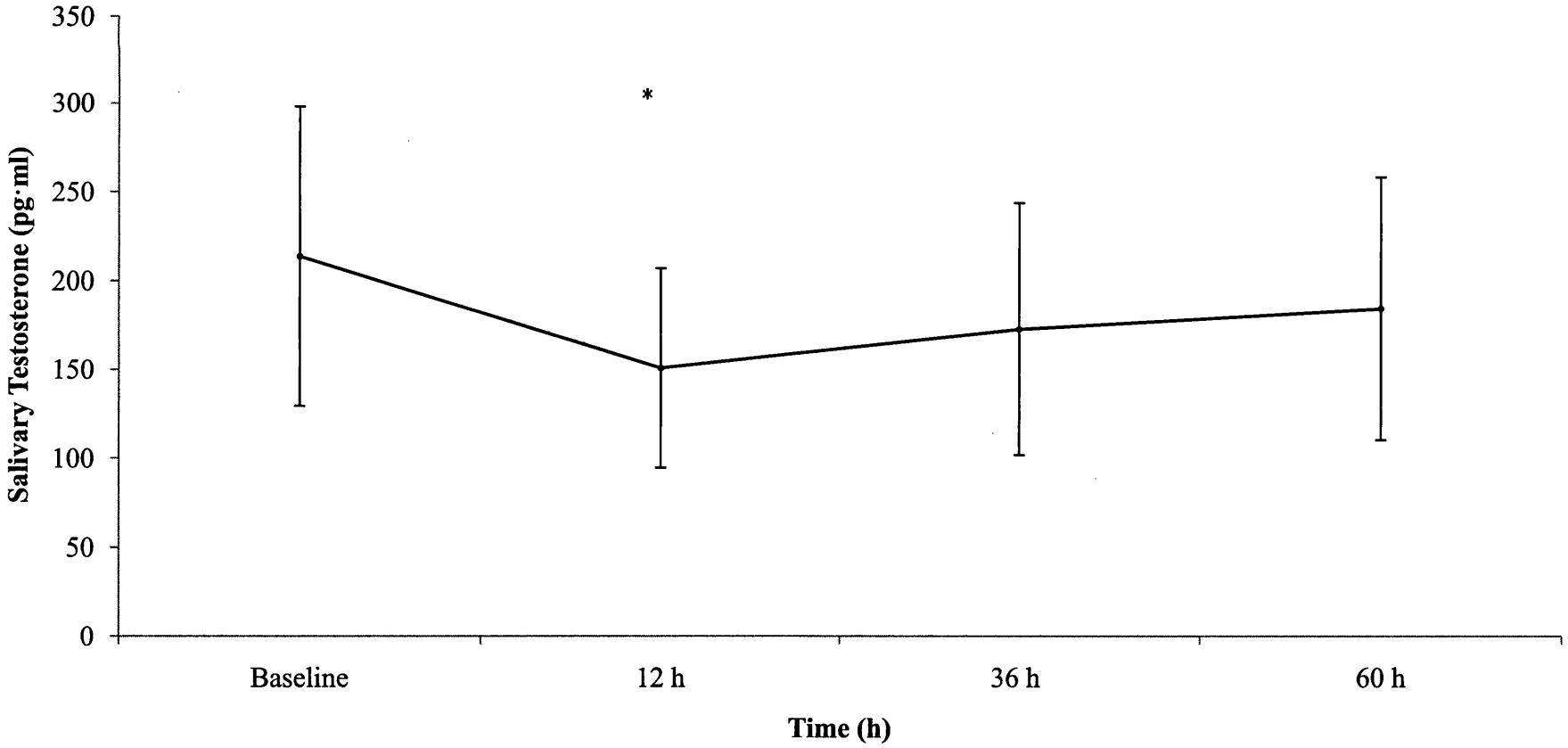

Figure 4.1: Salivary testosterone concentrations at baseline, 12, 36 and 60 h post-match ($pg \cdot mL^{-1}$)

- Indicates a significant different from baseline values ($P \leq 0.05$).

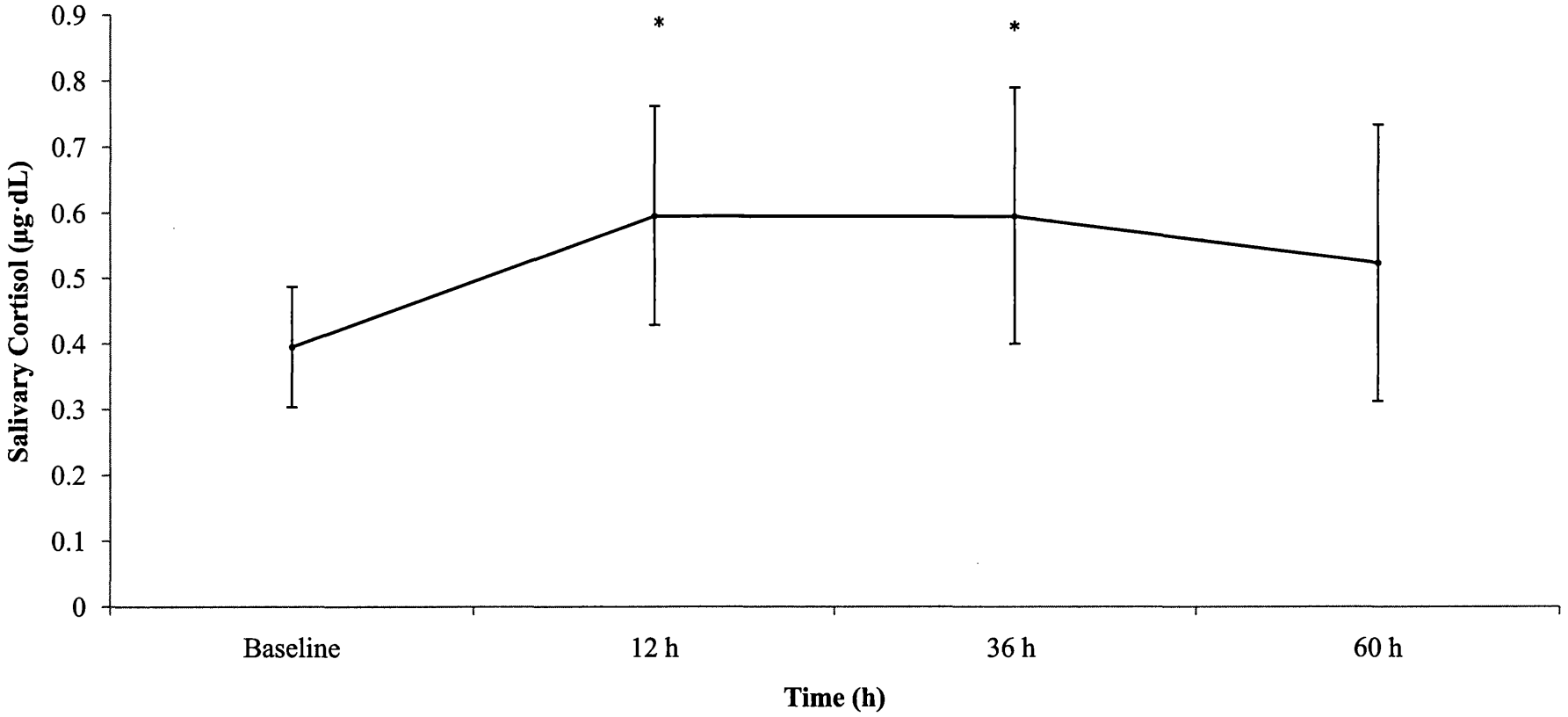

Figure 4.2: Salivary cortisol concentrations at baseline, 12, 36 and 60 h post-match ($\mu g \cdot dL^{-1}$)

- Indicates a significant different from baseline values ($P \leq 0.05$).

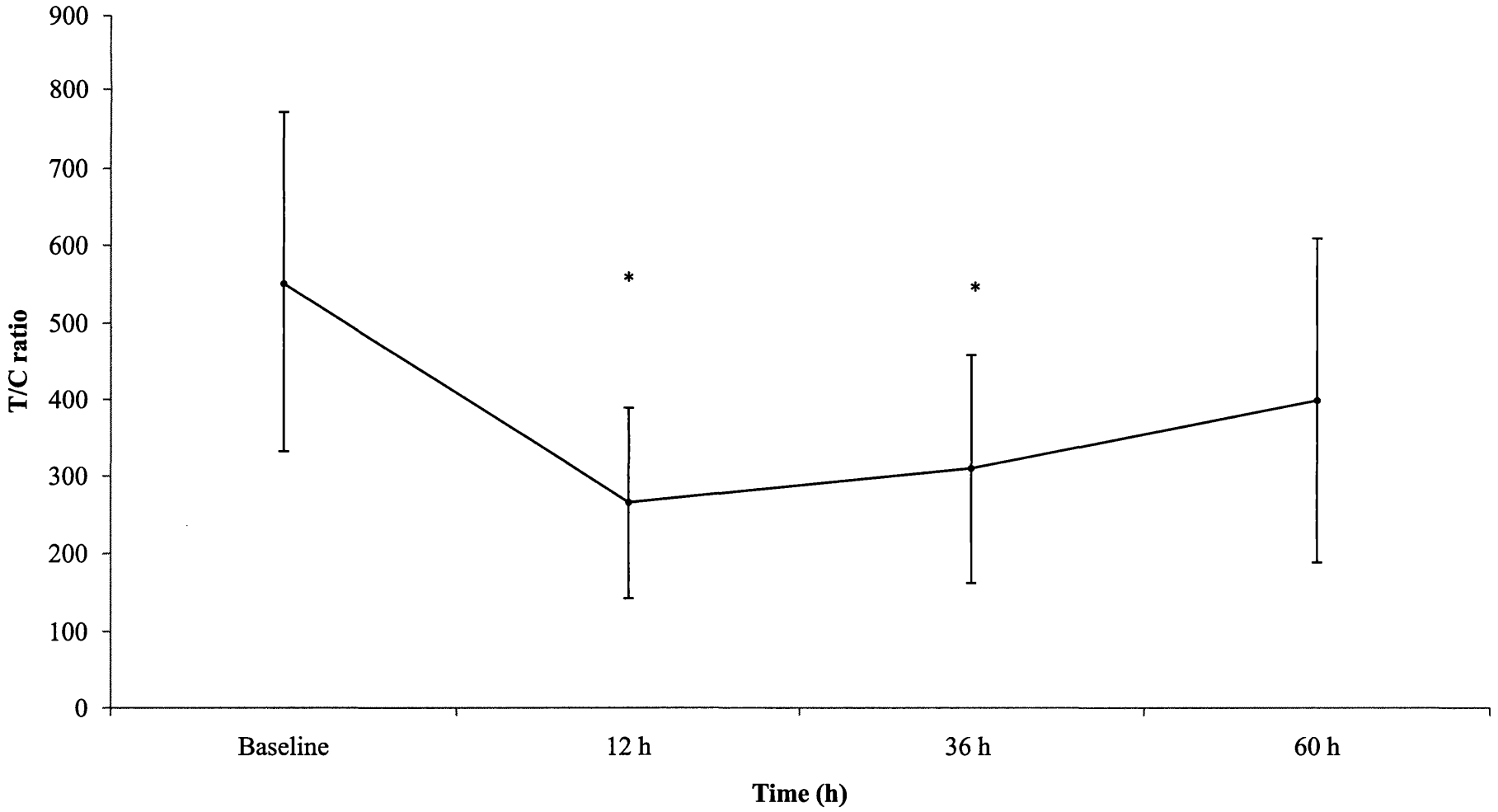

Figure 4.3: T:C ratio at baseline, 12, 36 and 60 h post-match.

* Indicates a significant different from baseline values ($P \leq 0.05$).

4.3.0. Mood Disturbance Scores

A significant time effect was also observed in mean mood disturbance scores [F (1.920, 21.115) = 4.152, $P \leq 0.05$, ES = 0.274]. Post hoc analyses revealed a significant increase in mood disturbance at 12 h compared with baseline scores (8 ± 4 vs. 5 ± 2 $P < 0.05$). However mood disturbance scores demonstrated recovery to those at baseline, with no significant differences observed at any other time points (Figure 5.0).

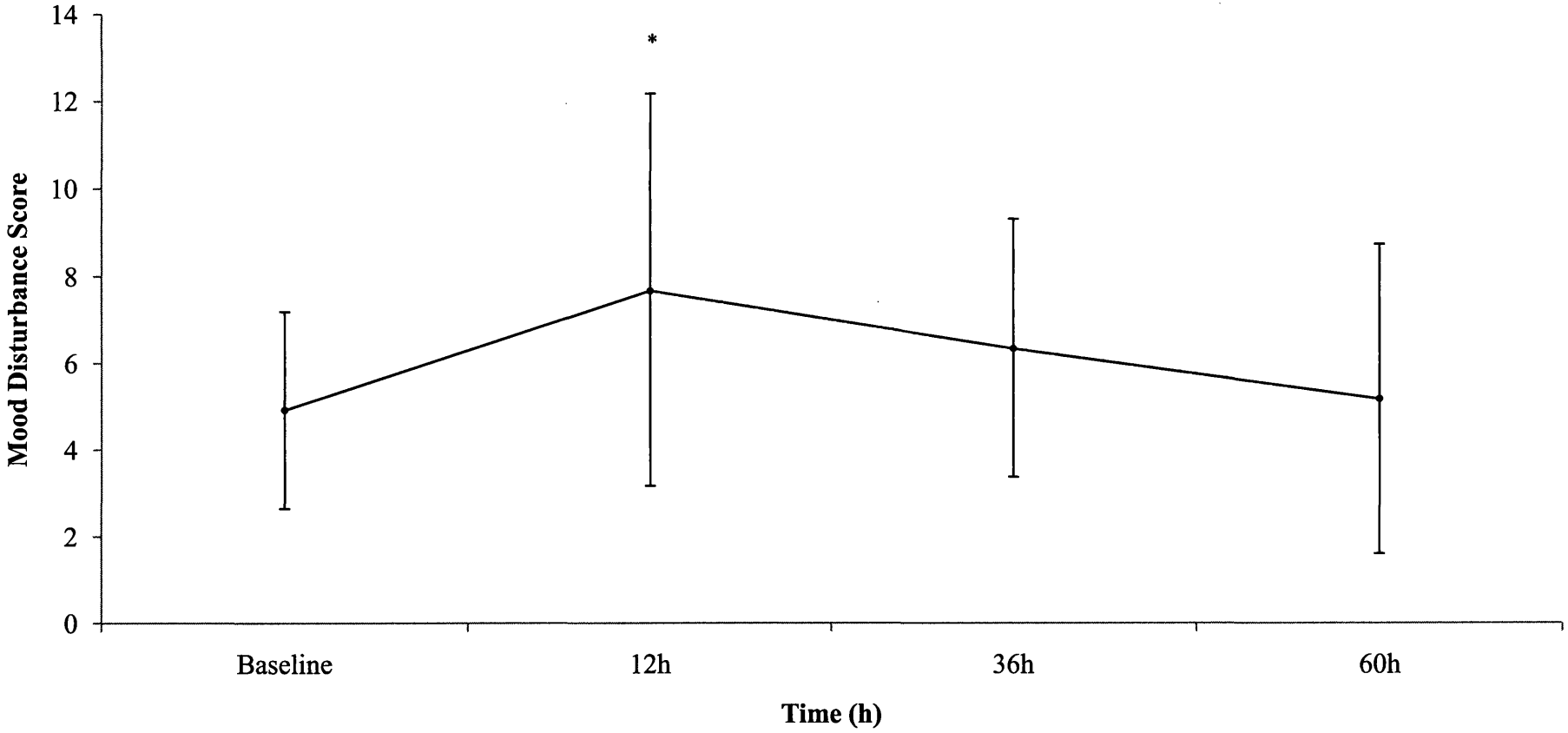

Figure 5.0: Mood disruption scores at baseline, 12, 36 and 60 h post-match.

* Indicates a significant different from baseline values ($P \leq 0.05$).

CHAPTER FIVE

5.0 Discussion

5.0. Discussion

The aim of the present study was to investigate the effects of an elite rugby union match on neuromuscular performance, endocrine function and mood disturbance. The primary findings were that neuromuscular and endocrine function remained significantly depressed until 36 h, and mood was significantly disturbed until 12 h following the match.

5.1.0 Neuromuscular Function

In the present study, peak concentric power (Fig. 2.1) was significantly lower at 12 h following the match (6.82 ± 5.79 %) and remained significantly depressed at 36 h (5.67 ± 3.77 %) compared to baseline. Although not significant, PP was still lower at 60 h compared to baseline values (2.35 ± 5.79 %). These findings are consistent with previous research examining the effects of neuromuscular function in the days following contact team sports (e.g. McLean et al., 2010; McLellan, Lovell & Gass, 2011), as well as non-contact team sport (Andersson et al., 2008). It is likely that the reduction in neuromuscular performance in the present study is primarily driven by muscle damage as a result of high intensity SSC movements such as high intensity running and SSC activity (e.g. changing direction), as well as collisions and blunt trauma involved in contact field sports (McLellan, Lovell & Gass, 2010; Smart et al., 2008; Takarada, 2003). The extent of muscle damage has been traditionally related to the duration, intensity and type of exercise (changing direction,

acceleration and deceleration). For example, Young et al. (2012) reported that running (between 4.0-6.0 $m \cdot s^{-1}$) and acceleration (3-15 $m \cdot s^{-2}$) are strongly related to muscle damage in Australian Rules football, with r values of 0.598 and 0.749 respectively. Additionally, Thorpe & Sunderland (2012) reported football players covering 617 ± 330m whilst sprinting (> $5m \cdot s^{-1}$), and % increases in [CK] was related to the number of sprints (39 ± 18) (r = 0.646), average sprint distance (15.2 ± 2.3 m) (r = 0.92) and high intensity (> 4 m·s-1) distance covered (~1200m) (r = 0.92). Furthermore, McLellan et al., (2010) reported 290 ± 69 m of sprinting in backs and 149 ± 32m in forwards in rugby league, with a greater number of sprints occurring in backs, but was not related to total distance covered. Only one previous study has attempted to evaluate the physiological demands of an elite rugby union match through the use of GPS (Cunniffe et al., 2009). It was reported that backs complete distances of approximately 500m and 300m for sprinting (> 7 $m \cdot s^{-1}$) and high intensity running (5 - 7 $m \cdot s^{-1}$) respectively, whereas forwards completed approximately 300 m for both sprinting and high intensity running in this study.

During the landing phase of running, the lower body is engaged in eccentric contractions which produce high tension per cross-sectional area of active muscle mass (Thorpe & Sunderland, 2012). This results in significant structural muscle damage, involving disruption of structural proteins including Z-lines, troponin (Thorpe & Sunderland, 2012), titin and desmin (Bongiovanni & Hagbarth, 1990; Komi, 2000), which is a key contributor to muscle damage after exercise (Thorpe & Sunderland, 2012). Previous literature has also suggested that strong relationships exist between contact activities and muscle damage arising from both rugby union and rugby league matches. For example, Takarada (2003) suggested that rugby union matches induce substantial muscle damage with peak [CK] occurring 24 h following the match, and is related to the number of tackles experienced during the match. In support of this, Smart et al. (2008) suggested that a large proportion of Δ

[CK] can be explained by physical impact, and also increased in a position specific manner. A study by McLellan, Lovell & Gass (2010) also alluded to positional dependent responses in rugby league, with muscle damage responses from backs arising predominantly from repeated high-intensity accelerations and decelerations in sprinting efforts, with the responses of forwards primarily from the exposure to repetitive high intensity collisions. More recently, McLellan et al. (2012) reported significant correlations between very heavy and severe impacts (as assessed by GPS) and decrements in PRFD 24 h post-match.

Collectively, these studies indicate that the direct impact of tackles and collisions, in addition to repeated high intensity SSC activity of rugby union results in substantial muscle damage. Previous research has suggested that the resulting muscle damage through high intensity SSC movements and blunt trauma from collision sport also accounts for reduced neuromuscular performance (Skurvydas et al., 2002; McLean et al., 2010; McLellan et al., 2011). Therefore it is likely that the high intensity and physical nature of rugby union account for the reductions in neuromuscular performance at 12 h and 36 h following the match in this study.

The reduction in neuromuscular performance beyond 24 h following field sport competition may reflect LFF via e-c coupling impairments, which can take between hours or days to recovery from (Edwards et al., 1977; Allen et al., 1995; Allen, 2001; McLellan et al., 2011). The mechanisms which underpin LFF are mainly attributed to Ca^{2+} release from the SR, with damage to t-tubules and cell membranes as a result of eccentric muscle contractions and blunt trauma (Morgan, 1990; Westerblad & Allen, 1991; Allen, 2001). Disruption to e-c coupling decreases the amount of Ca^{2+} released from the SR, which results in less Ca^{2+} binding to troponin C by the actin filaments, exposing less myosin binding sites and resulting in less cross bridge formation and ultimately, lower force production (McLellan, et al., 2010; 2011). Further mechanisms which may account for the decline in

neuromuscular performance at 12 and 36 h post-match may also relate to aforementioned mechanisms related to impairment of reflex sensitivity following muscle damage, (Avela et al., 1999a), decreased facilitation of the α-motoneuron pool and progressive withdrawal of spindle mediated fusimotor support (Avela et al., 1999a). Additionally, deteriorations in structural proteins such as titin and desmin intrafusal fibres may also reduce Ia afferent responsiveness, and may also contribute to decrements in neuromuscular function following muscle damage (Bongiovanni & Hagbarth, 1990).

A further symptom of this decline in neuromuscular function is the delayed onset of muscle soreness (DOMS) and is related to both muscle damage (Clarkson & Hubal, 2002) and the inflammatory process (Faulkner et al., 1993, Armstrong, 1984; Smith, 1991), and may interact with the aforementioned mechanisms (Skurvydas et al., 2005). A study by Skurvydas et al. (2005) reported a strong relationship between muscle soreness and decreased neuromuscular performance 24 - 48 h following SSC activity. DOMS may contribute to alterations in muscle fibre or motor unit recruitment (Edgerton et al., 1996), changes in agonist-antagonist ratios (Orchard, Marsden, Lord & Garlick, 1997) and changes in coordination of body segments (Edgerton, Wolf, Levendowski & Roy, 1996) which may lead to the changes in neuromuscular performance observed in the present study.

Interestingly, there were no significant differences in jump height reported at any time point throughout this study, and is in contrast to some of the previous literature in this area. For example, Andersson et al. (2008) demonstrated declines in CMJ height following a soccer match up to 72 h. Similarly, Ronglan, Raastad & Borgesen (2006) reported significant declines in jump height following a handball tournament, which resulted in gradual declines in CMJ height (- 6.7 ± 1.3 %). However, the results of this study are in agreement with Cormack et al. (2008a) who suggested jump height may be a less sensitive marker of neuromuscular fatigue compared to other measures. As fatigue measures were only observed

through PP, it could be hypothesised that variations in jump strategy occurred in attempt to maintain jump height (Thorlund et al., 2008; Salles et al., 2011). For example, downward and upward displacement of centre of mass in the CMJ is reduced, while the duration of that modified process is unchanged (i.e. CT), resulting in altered velocity of movement and therefore jump strategy (Thorlund et al., 2008). This alteration in jump strategy reduces the velocity in order to maintain time to create adequate work-output (and thereby kinetic impulse) so that the CMJ height reduction minimised (Thorlund et al., 2008).

5.2.0 Endocrine Response

In the present study, C was significantly increased at 12 h (56.0 ± 48.7%) and 36 h (59.2 ± 48.7%), and although not significant, C concentrations were still higher than baseline at 60 h (34.6 ± 51.0%). In the present study, increases in C during the 36 h following competition are likely to be related to muscle damage resulting from high intensity SSC movements, as well as collisions and blunt trauma, with the additional psychological constraint associated with the competition environment (Takarada. 2003; Smart et al., 2008; McLellan, Lovell & Gass., 2010). Additionally, protocols which stimulate the greatest metabolic acidosis demands and circulating [CK] 24 h post exercise also result in the greatest C responses and coinciding decrements in neuromuscular performance (Fragala et al., 2011). It is well understood that rugby union is predominantly an anaerobic sport producing elevated lactate responses (4.67 $mmol \cdot L^{-1}$ – 7.22 $mmol \cdot L^{-1}$) (Takarada, 2003; Deutsch et al., 2010), which may also account for elevations in C in the present study.

The present study also demonstrated a significant decrease in T at 12 h (-26.0 ± 29.8%) following the match compared to baseline concentrations. Although not significant, T

levels were still lower at both 36 (-14.6 ± 34.0%) and 60 h (-8.4 ± 27.8%). As a result of the fluctuations in T and C, significant decreases in T:C ratio were demonstrated at 12 h (-45.8 ± 23.2%) and 36 h (-35.8±32.9%) compared with baseline concentrations (-16.1±46.7) and although not significant, T:C was still depressed at 60 h (54.2 ± 23.2%). The observed pattern in T is likely down to more psychophysiological stimuli, behavioural traits and individual perceptions (McLellan et al 2010). The post-competition decreases in T may also be accounted for by the inhibiting effects of elevated glucocorticoid concentrations on the HPG axis (McLellan, Lovell & Gass, 2010).

These findings are also partially in agreement with those of Elloumi et al., 2003, where the match induced a drop of approximately 20% in T, compared to a 26.0 ± 29.8 % decrease in the present study. This response could be explained by the inhibition of the gonadotrophic axis at a hypothalamic level via CRH, most likely due to the stress response to muscle damage resulting from high intensity SSC movements and blunt trauma from the match (McLellan, Lovell & Gass, 2010). However, the authors also demonstrated increases above baseline concentrations during the recovery period, however the mechanisms for these changes were unexplained and the authors did not establish a link between the changes of T and C following the match. However, baseline samples were collected approximately two months prior to the match measured, which did not account for fluctuations in baseline samples over time, and with training (Viru & Viru, 1995).

The fluctuations in T and C are also in agreement with McLellan, Lovell & Gass (2010) where salivary T and C were examined in response to an elite Rugby League match. C concentrations were significantly elevated up to 24 h post-match compared with baseline concentrations. The same group also examined pre, during and post-match endocrine responses to a further rugby league match, with another significant increase in C observed 24 h post game with a simultaneous decrement in T:C ratio, which returned to baseline

concentrations within 48 h post-match (McLellan, Lovell & Gass, 2011). These fluctuations were attributed to a reflection of the duration, intensity, and combative nature of rugby league and psychological influences of anxiety and perceived stress of the competition environment. Additionally, the authors made an interesting link between the fluctuations in C and neuromuscular performance, in that the players with the largest decrements in PF also produced a higher salivary C post-match, however post-match samples were not obtained in the present study. The same group also examined the response of C with respect to collision in rugby league match play and reported consistent post-match increases in C. The gradual decline over the recovery days following the match represents the removal of match related psychological and physiological stressors over this period, and the re-establishment of homeostasis (McLellan, Lovell & Gass, 2010). Interestingly, this study also reported no relationship between C concentrations and collisions (measured through the use of GPS) experienced, and suggested that this indicates that rugby league match play generates sufficient psychological and physiological stress to cause an elevation in C with a return to baseline within 48 h.

5.3.0. Mood disturbance

The present study also demonstrated a significant disturbance in mood score at 12 h following the match, compared to baseline values. Although not significant, mood disturbance scores were still slightly higher at 36 h and 60 h following the match, and the most significant mood disturbance scores coincided with the greatest increase in C concentrations, which occurred at 12 h post-match (Figure 4.0). Only a small number of previous studies have investigated mood disruption over the post competition recovery period

in rugby union (e.g. Suzuki et al., 2004; Nicolls, Backhouse, Polman & McKenna, 2009). However, one study by Suzuki et al. (2004) examined fluctuations in mood following a rugby union match and reported increases in fatigue and depression following the match, This study also suggested that psychological stress (as measured by POMS) resulting from a rugby match is probably due to the combination of exercise intensity and player's satisfaction with their own performances, with the team winning on this occasion, as did the team in the current study. However, the study by Suzuki et al. (2004) did not measure the steroid hormones, or include any performance measures indicative of recovery following the match, creating difficulty in comparing the results of the present study. A study by Nicolls, Backhouse, Polman & McKenna (2009) examined fluctuations in a number of psychological constructs over a 3 day period in response to a single rugby union game. Interestingly, players reported many stressors being "worse than normal" on the day following a match, compared to the day before the match, characterised by feelings of tiredness and fatigue, which support the findings of the present study. However, the study by Nicholls further reported an "unpleasant, low activation state across" the three analysis days and suggested these subjects were in an overtrained state (as measured by Daily Analysis of Life Demands Questionnaire, and the Activation Deactivation Adjective Check List).

The vast majority of studies examining steroid hormones and mood disturbance in rugby and other athletic populations have primarily focused on longer term mood responses in relation to overtraining or over reaching, rather than the immediate recovery from competition. Maso et al. (2004) reported that T concentrations were negatively associated with overtraining items such as tiredness, whereas C concentrations were not related to overtraining in this group. These findings relate somewhat to the present study, in that the most significant disturbance in mood coincided with the largest decline in T concentrations at

12 h following the match. Additionally, Morgan et al. (1984) reported training performance improvements with positive mood and deteriorations in performance with greater mood disturbance. Furthermore, Flynn et al. (1994) reported significantly higher C levels in a group of overtrained swimmers, which correlated with depressed mood states. The results of the present study are in agreement with previous literature which suggests intense periods of competition or heavy training loads result in higher physiological and psychological stress and ultimately negative mood states; however the mechanisms of stress and negative mood are complex.

Subjective mood is a reflection of psychological well-being and may interact with HPA activity (Larsen, 2000), which may influence mood by regulating metabolic processes and aspects of immune function (Sheridan et al., 1994). Studies in mental health have reported higher levels of glucocorticoids resulting from HPA dysregualtion have been associated with affective disorders and alterations in mood (Goodyer et al., 1996), and psychological stress (Seyle, 1980). Additionally, elevated C has been related to the increases in negative mood and lower C with more positive psychological attributes such as higher self efficacy and positive mood (Lai et al., 2005; Rudolph & McAuley, 1995). These effects result from cortisols direct action on brain cells and/or by altering the availability of brain neurotransmitters (Stokes, 1995).

The HPA axis is also activated by pro-inflammatory cytokines such as IL-6 which frequently arise as a result of exercise induced muscle damage (Borer, 2002). The HPA system is strongly activated in depressed mood, via the activation of CRH neurons by CRH and C. There is a close functional interaction between the HPA-axis and the HPG-axis, with hypothalamic CRH neurons inhibiting hypothalamic control of the gonadal axis (Dudas & Merchenthaler, 2002a; Rivest & Rivier, 1995). Studies from clinical depression in males have

reported decreased T levels due to increased HPA activity parallel to a diminished HPG-axis. (Swaab et al., 2005).

There are few studies investigating the neurobiology of mood disturbance in elite rugby and other athletic populations, and this certainly highlights an area for further research, as previous studies have suggested that mood state in elite athletes may be a predictor of performance (Nicolls et al., 2009). Post competition mood state data in rugby players is scarce, and the data on mood disturbance in the present study, and from previous investigations suggest that rugby players experience more symptoms of stress and negative mood following competition which are of a physiological and psychological source (Nicolls et al., 2009; Maso et al., 2004; Suzuki et al., 2004). A novel finding of this study with regard to mood disturbance is that the fluctuations in the steroid hormones and neuromuscular performance follow similar patterns in time course of recovery following an elite rugby match as player's subjective feelings of fatigue. Limitations of this study include little information on match data, muscle damage and inflammatory variables. Additionally, if jump height is to be used as a sensitive marker of muscle fatigue, the depth of the countermovement should be kept consistent for all trials, or joint angles could be measured during self-selected countermovement depths to monitor how this fluctuates with muscle fatigue through video analysis (Salles et al., 2011). Furthermore, hydration status may be taken into account as some studies suggest differential effects of hydration status on concentrations of salivary steroid hormones (Crewther et al., 2008). This may be accomplished the use of urinary measures of hydration which is commonly completed in team sport environments (Oppliger & Bartock, 2002). Another limitation in this study is that only a single game was measured. Some previous research has suggested that some physiological responses (e.g. muscle damage) may differ from game to game across a season according to variables such as match importance, length of microcycle prior to the match and

through contact adaptation (Hoffman et al., 2005). Future research should examine match work-loads, intensities and collisions through the use of GPS technology to establish the origins of muscle damage (through stretch shorten cycle activities such as high intensity running, and/or contact and collisions) (McLellan et al., 2012), blood proteins such as CK as a marker of muscle damage (Takarada 2003) as well as inflammatory markers such as IgA (Cunniffe et al., 2010) following a rugby match, highlighting areas of future research for understanding competition induced disruptions in performance and physiological functions in elite rugby players, in light of the limitations highlighted in this study. However, measuring these parameters in athletes may enable coaches to make informed decisions in optimising individual players' recovery strategies during intense competition periods.

CHAPTER SIX

6.0 Conclusion

6.0. Conclusion

The results from the present study suggest that neuromuscular performance remains slightly depressed (although non-significant) until 60 h post game, with peak power output (PPO) representing the most sensitive measure. Homeostatic disruption driven by muscle damage following high intensity stretch shortening cycle (SSC) movements, as well as collision was characterised by elevations in salivary cortisol (C) and depressions in testosterone (T) during the days following the match. This data provides novel findings in similar patterns of player's subjective feelings of fatigue following similar patterns of recovery as neuromuscular and endocrine function. As each variable follows similar time courses of recovery, coaches may use PPO as a sensitive measure of neuromuscular performance, fluctuations in stress hormones and mood disturbance scores as markers of recovery. In conclusion, the profiling of a professional rugby union match revealed post-match changes in CMJ power, salivary hormones and mood disturbance. The directions of the changes in these measures varied up to 36 h, but had returned to baseline by 60 h post-match. Thus, a professional rugby union match elicits transient changes in important markers of recovery, with a large degree of individual variation.

CHAPTER SEVEN

7.0 References

7.0. References

Aakvaag, A., Bentdal, Ø., Quigstad, K., Walstad, P., Rønnigen, H. & Fonnum, F. (2008). Testosterone and Testosterone Binding Globulin (TeBG) in Young Men during Prolonged Stress. *International Journal of Andrology*, 1, 1-6.

Abbiss, C., R. & Laursen, P., B. (2005). Models to Explain Fatigue during Prolonged Endurance Cycling. *Sports Medicine*, 35, 865-898.

Allen, D,. G. & Westerblad, H. (2001). Role of phosphate and calcium stores in muscle fatigue. *Journal of Physiology*, 536, 657-665.

Allen, D., G. (2001). Eccentric muscle damage: mechanisms of early reduction of force. *Acta Physiologica Scandinavia*, 171, 311-319.

Allen, D., G., Lannergren, J. & Westerblad, H. (1995). Muscle cell function during prolonged activity: Cellular mechanism of fatigue. *Experimental Physiology,* 80, 497-527.

Alvear-Ordenes, I., García-López, D., De Paz, J., A. & González-Gallego, J. (2005). Sweat Lactate, Ammonia, and Urea in Rugby Players. *International Journal of Sports Medicine*, 26, 632–637.

Andersson, H., Raastad, T., Nilsson, J., Paulsen, G., Garthe, I. & Kadi, F. (2008). Neuromuscular Fatigue and Recovery in Elite Female Soccer: Effects of Active Recovery. *Medicine & Science in Sports & Exercise*, 40, 372-380.

Aragon-Vargas, L., F. & Gross, M., M. (1997). Kinesiology factors in vertical jump performance: differences among individuals. *Journal of Applied Biomechanics*, 13, 24-44.

Archer, J., (1988). The Behavioural Biology of Aggression. Cambridge University Press, Cambridge. In: Suay, F., Salvador, A., Gonzalez-Bono, E., Sanchis, C., Martinez, M., Martinez-Sanchis, S., V., M. & Montoro, J., B. (1999). Effects of competition and its outcome on serum testosterone, cortisol and prolactin. *Psychoneuroendocrinology*, 24, 551–566.

Armstrong, R., B. (1984). Mechanisms of exercise-induced delayed onset muscle soreness: a brief review. *Medicine and Science in Sports and Exercise*, 16, 529-38.

Armstrong, R., B., Warren, G., L. & Warren, J., A. (1991). Mechanisms of exercise induced muscle fibre injury. *Sports Medicine*, 12, 184-207.

Ascensão, A., Leite, M., Rebelo, A., N., Magalhães, S. & Magalhães, J. (2010). Effects of cold water immersion on the recovery of physical performance and muscle damage following a one-off soccer match. *Journal of Sports Sciences*, 29, 217-25.

Augustsson, J., Thomeé, R., Lindén, C., Folkesson, M., Tranberg, R. & Karlsson, J. (2006). Single-leg hop testing following fatiguing exercise: reliability and biomechanical analysis. *Scandinavian Journal of Medicine & Science in Sports*, 16, 111-120.

Avela, J., & Komi, P.V. (1998). Interaction between muscle stiffness and stretch reflex sensitivity after long term stretch shortening cycle exercise. *Muscle & Nerve*, 21, 1224-1227.

Avela, J., Jyrolainen, H., Komi, P., V. & Rama, D. (1999). Reduced reflex sensitivity persists several days after long-lasting stretch shortening cycle exercise. *Journal of Applied Physiology*, 86, 1292–1300.

Avela, J., Kyrolainen, H. & Komi, P., V. (1999) Altered reflex sensitivity after repeated and prolonged passive muscle stretching. *Journal of Applied Physiology,* 86, 1283–1291.

Baker, D. & Newton, R., U. (2008). Comparison of lower body strength, power, acceleration, speed, agility, and sprint momentum to describe and compare playing rank among Professional Rugby League. *Journal of Strength and Conditioning Research*, 22, 153–158.

Bangsbo, J., Madsen, K., Kiens, B. & Richter, E.A. (1996). Effect of muscle metabolism and fatigue during intense exercise in man. *Journal of Physiology*, 495, 587–596.

Barbas, I., Fatouros, I., G., Douroudos, I., I., Chatzinikolaou, A., Michailidis, Y., Draganidis, D., Jamurtas, A., Z.,Nikolaidis, M., G., Parotsidis, C., Theodorou, A., A., Katrabasas, I., Margonis, K., Papassotiriou, I. & Taxildaris, K. (2011). Physiological and performance

adaptations of elite Greco-Roman wrestlers during a one-day tournament. *European Journal of Applied Physiology*, 111, 1421-1436.

Beedie, C. J., Terry, P. C. & Lane, A. M. (2000). The profile of mood states and athletic performance: Two meta-analyses. *Journal of Applied Sport Psychology*, 12, 49-68.

Berger, B. G., Motl, R. W., Butki, B. D., Martin, D. T., Wilkinson, J. G. Owen, D. R. (1999). Mood and cycling performance in response to three weeks of high-intensity short-duration overtraining and a two week taper. *Sport Psychologist*, 13, 444-457.

Bigland-Ritchie, B., Cafarelli, E. &Vollestad, N., K. (1986). Fatigue of submaximal static contractions. *Acta physiologica Scandinavia. Supplementum,* 556, 137–148.

Bigland-Ritchie, B., R., Dawson, N., J., Johansson, R., S. & Lippold, O., C. (1986). Reflex origin for the slowing of motoneuron firing rates in fatigue of human voluntary contractions. *Journal of Physiology*, 379, 451–459.

Bishop, D. (2003). Warm Up I: Potential Mechanisms and the Effects of Passive Warm Up on Exercise Performance. *Journal of Strength and Conditioning Research*, 16, 439-454.

Boerio, D., Jubeau, M., Zory, R. & Maffiuletti, N., A. (2005). Central and Peripheral Fatigue after Electrostimulation-Induced Resistance Exercise. *Medicine & Science in Sports & Exercise*, 37, 973-978.

Bongiovanni, L., G. & Hagbarth, K., E. (1990). Tonic vibration reflexes elicited during Fatigue from maximal voluntary contractions in man. *Journal of Physiology*, 423, 1-14.

Bonitch-Dominguez, J., Bonitch-Gongora, J., Padical, P. & Feriche, B. (2010). Changes in peak leg power induced by successive judo bouts and their relationship to lactate production. *Journal of Sports Sciences*, 28, 1527-1534(8)

Booth, A., Shelley, G., Mazur, A., Tharp, G. & Kittok, R. (1989). Testosterone, and winning and losing in human competition. *Hormones and Behaviour*, 23, 556–571.

Bosco, C., Luhtanen, P. & Komi, P., V. (1983). A simple method for measurement of mechanical power in jumping. *European Journal of Applied Physiology,* 50, 273-282

Brain, P. F., (1990). Stress in agonistic contexts in rodents. In: Dantzer, R., Zayanm, R. (Eds.), Stress in Domestic Animals. Kluwer, Dordrecht, 73–85. In: Suay, F., Salvador, A., Gonzalez-Bono, E., Sanchis, C., Martinez, M., Martinez-Sanchis, S., V., M. & Montoro, J., B. (1999). Effects of competition and its outcome on serum testosterone, cortisol and prolactin. *Psychoneuroendocrinology*, 24, 551–566.

Budgett, R. (1998). Fatigue and underperformance in athletes: the overtraining syndrome. *British Journal of Sports Medicine*, 32, 107-110.

Byrne, C. & Eston, R. (2002). Maximal-intensity isometric and dynamic exercise performance after eccentric muscle actions. *Journal of Sports Sciences*, 20, 951–959.

Carre, J., Miur, C., Belanger, J. & Putnam, S. K. (2006). Pre-competition hormonal and psychological levels of elite hockey players: Relationship to the "home advantage." *Physiology & Behaviour*, 89, 392-398.

Chatzinikolaou, A., Fatouros, I., G., Gourgoulis, V., Avloniti, A., Jamurtas, A., Z., Nikolaidis, M., G., Douroudos, I., Michailidis, Y., Beneka, A., Malliou, P., Tofas, T., Georgiadis, I., Mandalidis, D. & Taxildaris, K. (2010). Time course of changes in performance and inflammatory responses after acute plyometric exercise. *Journal of Strength and Conditioning Research,* 24, 1389-1398.

Clarkson, P. & Hubal, M. (2002). Exercise-induced muscle damage in humans. *American Journal of Physical Medicine & Rehabilitation,* 81(Supplement): S52–S69.

Clarkson, P., M. & Tremblay, I. (1988). Exercise-induced muscle damage, repair and adaptation in humans. *Journal of Applied Physiology*, 65, 1-6.

Clarkson, P., M., Nosaka, K. & Braun, B. (1992). Muscle function after exercise-induced muscle damage and rapid adaptation. *Medicine and Science in Sports and Exercise*, 24, 512-20.

Cormack, S., J., Newton, R., U. & McGuigan, M., R. (2008a). Neuromuscular and endocrine responses of elite players to an Australian rules football match. *International Journal of Physiology and Performance*, 3, 358-374.

Cormack, S. J., Newton, R. U., McGuigan, & M. R. & Cormie (2008b). Neuromuscular and Endocrine Responses of Elite Players During an Australian Rules Football Season. *International Journal of Sports Physiology and Performance*, 3, 439-453.

Crewther, B. T., Heke, T. & Keogh, J. W. L. (2011). The effects of training volume and competition on the salivary cortisol concentrations of Olympic weightlifters. *Journal of Strength and Conditioning Research*, 25, 10-15.

Crewther, B. T. & Cook, C. (2010). Measuring the Salivary Testosterone and Cortisol Concentrations of Weightlifters Using an Enzyme-Immunoassay Kit. *International Journal of Sports Medicine*, 31, 486-489.

Cunniffe, B., Procter, W., Baker, J. & Davies, B. (2009). An Evaluation of the Physiological Demands of Elite Rugby Union Using Global Positioning System Tracking Software. *Journal of Strength and Conditioning Research*, 23, 1195-1203.

Cunniffe, B., Hore, A. J., Whitcombe, D. M., Jones, K. P., Baker, J. S. & Davies, B. Time course of changes in immunoendocrine markers following an international rugby game. *European Journal of Applied Physiology*, 108, 113-22.

Cunniffe, B., Griffiths, H., Proctor, W., Davies, B., Baker, J. S. & Jones, K. P. (2011). Mucosal immunity and illness incidence in elite rugby union players across a season. *Medicine and Science in Sports and Exercise*, 43, 388-397.

Curren, S. L., Andrykowski, M. A., Studts, J. L. (1995). Short Form of the Profile of Mood States (POMS-SF). *Psychological Assessment*, 7, 80-83.

Daly, W., Seegers, A., Rubin, D.A., Dobridge, J.D. & Hackney, A.C, (2005). Relationship between stress hormones and testosterone with prolonged endurance exercise. *European Journal of Applied Physiology*, 93, 375-380.

Davies, C., T., M., Mecrow, I., K. & White, M., J. (1982). Contractile properties of the human triceps surae with some observations on the effects of temperature and exercise. *European Journal of Applied Physiology*, 49, 255-269.

Dean, J. E., Whelan, J. P. & Meyers, A. W. (1990). An incredibly quick way to assess mood states: The Incredibly Short POMS. Presented to Association for the Advancement of Applied Sport Psychology, San Antonio, TX.

Deutsch, M., U., Kearney, G., A. & Rehrer, N., J. (2007). Time – motion analysis of professional rugby union players during match-play. *Journal of sports sciences*, 15, 461-472.

Deutsch, M., U., Maw, G., J., Jenkins, D. & Reaburn, P. (1998). Heart rate blood lactate and kinematic data of elite colts (under-19) rugby union players during competition. *Journal of Sports Sciences*, 16, 561–570.

Docherty, D., Wenger, H., A. & Neary, P. (1998). Time motion analysis related to the physiological demands of rugby. *Journal of Human Movement Studies*, 14, 269–277.

Dousset, E., Avela, M., Ishikawa, J., Kallio, S., Knuitunen, S., Kryolainen, H., & Linnamo, V. & Komi, P., V. (2007). Bimodal Recovery Pattern in Human Skeletal Muscle Induced by Exhaustive Stretch-Shortening Cycle Exercise. *Medicine & Science in Sports & Exercise*, 39, 453-460.

Dressendorfer, R., H. & Wade. C., E. (1991). Effects of a 15-d race on plasma steroid levels and leg muscle stiffness in runners. *Medicine & Science in Sports & Exercise*, 23, 954-958.

Duchateau, J. & Hainaut, K. (1993). Behaviour of short and long latency reflexes in fatigued human muscles. *Journal of Physiology*, 471, 787–799.

Duke, A., M. & Steele, D., S. (2000). Characteristics of phosphate-induced Ca^{2+} efflux from the SR in mechanically skinned rat skeletal muscle fibers. *American Journal of Physiology - Cell Physiology*, 278, C126-C135.

Edgerton, V., R., Wolf, S., L., Levendowski, D., J. & Roy, R, R. (1996). Theoretical basis for patterning EMG amplitudes to assess muscle dysfunction. *Medicine and science in sports and exercise,* 28, 744-51.

Edwards, R., R., T., Hill, D., K., Jones, D., A. & Merton, P., A. (1977). Fatigue of long duration in human skeletal muscle after exercise. *Journal of Physiology*, 272, 769-778.

Ehlers, G., G., Ball,T., E. & Liston, L. (2002). Creatine Kinase Levels are Elevated During 2-A-Day Practices in Collegiate Football Players. *Journal of Athletic Training*, 37(2), 151–156.

Elias, M., (1981). Serum cortisol, testosterone and testosterone-binding globulin responses to competitive fighting in human males. *Aggressive Behaviour*, 7, 215–224.

Elloumi, F., Maso, F., Michaux, O., Robert A. & Lac, G. (2003). Behaviour of saliva cortisol [C], testosterone [T] and the T/C ratio during a rugby match and during the post-competition recovery days. *European Journal of Applied Physiology*, 90, 23-28.

Elloumi, M., Maso, F., Michaux, O., Robert, A. & Lac, G. (2003). Behaviour of saliva cortisol [C], testosterone [T] and the T/C ratio during a rugby match and during the post-competition recovery days. *European Journal of Applied Physiology*, 90, 23-28.

Eston, R., Byrne, C. & 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, 85-96.

Eston, R., G., Finney, S., Baker, S. & Baltzopoulos, V. (1996). Muscle tenderness and peak torque changes after downhill running following a prior bout of isokinetic eccentric exercise. *Journal of Sports Science*, 14, 291-299.

Filaire, E., Bernain, X., Sagnol, M. & Lac, G. (2001). Preliminary results on mood state, salivary testosterone:cortisol ratio and team performance in a professional soccer team. *European Journal of Applied Physiology*, 86, 179-184.

Filaire, E., Duche, P. & Lac, G. (1997). Influences of an official competition and a training session on the salivary cortisol. *Science and Sports*, 12, 66-71.

Filaire, E., Maso, F., Sagnol, M., Ferrand, C. & Lac, G. (2001). Anxiety, Hormonal Responses, and Coping during a Judo Competition. *Aggressive Behaviour*, 27, 55-63.

Finni, T., Kyröläinen, H., Avela, J. & Komi, P., V. (2003). Maximal but not submaximal performance is reduced by constant-speed 10-km run. *Journal of Sports Medicine and Physical Fitness*, 43, 411-7.

Fitts, R., H. (2004). Mechanisms of muscular fatigue. *Principles of Exercise Biochemistry*, 46, 279-300.

Flynn, M. G., Pizza, F. X., Boone, J. B., Andres, F. F., Michaud, T. A. & Rodrequez-Zavas, J. R. (1994). Indices of training stress during competitive running and swimming seasons. *International Journal of Sports Medicine*, 15, 21-26.

Foster, C., Green, M., A., Snyder, A., C. & Thompson, N., N. (1993). Physiological responses during simulated competition. *Medicine and Science in Sports and Exercise*, 25, 877-882.

Fowles, J., R. (2006). Technical Issues in Quantifying Low-Frequency Fatigue in Athletes. *International Journal of Sports Physiology and Performance*, 1, 169-171.

French, D., N, Kraemer, W., J., Volek, J., S., Spiering, B., A., Judelson., D., A., Hoffman, J., R. & Maresh, C., M. (2007). Anticipatory responses of catecholamines on muscle force production. *Journal of Applied Physiology*, 102, 94-102.

Fridén, J. (1984). Changes in human skeletal muscle induced by long-term eccentric exercise. *Cell and Tissue Research*, 236, 365-72.

Friden, J., Sjostrom, M. & Ekblom, B. (1983). Myofibrillar damage following intense eccentric exercise in man. *International journal of Sports Medicine*, 4, 170-176.

Fryer, M., W., Owen., V., J., Lamb, G. D. & Stephenson, D., G. (1995). Effects of creatine phosphate and Pi on Ca^{2+} movements and tension development in rat skinned skeletal muscle fibres. *Journal of Physiology (Lond)*, 482, 123 140.

Gandevia, S., C. (2001). Spinal and Supraspinal Factors in Human Muscle Fatigue. *Physiological Reviews*, 81, 1725-1789.

Gatti, R., Antonelli, G., Prearo, M., Spinella, P., Cappellin, E. & De Palo, E. F. (2009). Cortisol assays and diagnostic laboratory procedures in human biological fluids. Clinical *Biochemistry*, 42, 1205-1217.

Gatti, R. & De Palo, E. F. (2010). An update: salivary hormones and physical exercise. *Scandinavian Journal of Medicine & Science in Sports*, 21, 157-169.

Gill, N., D., Beaven, C., M. & Cook, C. (2006). Effectiveness of post-match recovery strategies in rugby players. *British Journal of Sports Medicine*, 40, 260-3.

Girard, O., Lattier, G., Maffiuletti, N., A., Micallef, J., P. & Millet, G., P. (2008). Neuromuscular fatigue during a prolonged intermittent exercise: Application to tennis. *Journal of Electromyography and Kinesiology*, 18, 1038-46.

Gollhofer, A., Komi, P.V., Fujitsuka, N. & Miyashita, M. (1987). Fatigue during stretch-shortening cycle exercises: II. Changes in neuromuscular activation patterns of human skeletal muscle. *International Journal of Sports Medicine*, 8, 38-47.

Gollhofer, A., Komi, P.V., Miyashita, M., & Aura, O. (1987). Fatigue during stretch-shortening cycle exercises: Changes in mechanical performance of human skeletal muscle. *International Journal of Sports Medicine*, 8, 71-78.

Gonzalez-Bono, E., Salvador, A., Serrano, M., A. & Ricarte, J. (1999). Testosterone, cortisol and mood in a sports team competition. *Hormones and Behaviour*, 26, 235–240.

Gordon, A., M., Homsher, E. & Regnier. M. (2000). Regulation of Contraction in Striated Muscle. *Physiological Reviews*, 80, 853–924.

Groschl, M. & Rauh, M. (2006). Influence of commercial collection devices for saliva on the reliability of salivary steroids analysis. *Steroids*, 71, 1097-1100.

Grove, R. J. & Prappavesisis, H. (1992). Preliminary evidence for the reliability and validity of an abbreviated Profile of Mood States. *International Journal of Sports Psychology*, 23, 93-109.

Haff, G. G., Stone, M., O'Bryant, H., S., Harman, E., Dinan, C., Johnson, R. & Han, K., H. (1997). Force-time dependent characteristics of dynamic and isometric muscle actions. *Journal of Strength and Conditioning Research*, 11, 269-272.

Häkkinen, K. & Pakarinen, A. (1991). Serum hormones in male strength athletes during intensive short term strength training. *European Journal of Applied Physiology*, 63, 3-4.

Haneishi, K., Fry, A., C., Moore, C., A., Schilling, B., K., Li, Y. & Fry, M., D. (2007). Cortisol and stress responses during a game and practice in female collegiate soccer players. *Journal of Strength and Conditioning Research*, 21,583–588.

Harman, E., A., Rosenstein, M., T., Frykman, P., N. & Rosenstein, R., M. (1990). The effects of arms and countermovement on vertical jumping. *Medicine and Science in Sports and Exercise*, 22, 825-833.

Hatze, H. (1998). Validity and Reliability of Methods for Testing Vertical Jumping Performance, *Journal of Applied Biomechanics*, 14, 127-140

Hoffman, J., R., Kang, J., Ratamess, N., A. & Faigenbaum, A., D. (2005). Biochemical and Hormonal Responses during an Intercollegiate Football Season. *Medicine & Science in Sports & Exercise*, 37, 1237-1241

Hoffman, J. R., Maresh, C. M., Newton, R. U., Rubin, M, R., French, D, N. & Volek, J. S. (2002). Performance, biochemical and endocrine changes during a competitive football game. *Medicine & Science in Sports & Exercise*, 34, 1845-1853.

Hoffman, J., R., Nusse V. & Kang, J. (2003). The Effect of an Intercollegiate Soccer Game on maximal power performance. *Canadian Journal of Applied Physiology,* 28, 807–817.

Hooper, S., L., Mackinnon, L., T., Haward, A., Gordon, R., D. & Bachmann, A., W. (1995). Markers for monitoring overtraining and recovery. *Medicine and Science in Sports and Exercise*, 27, 106–112.

Hori, N., Newton, R., U., Kawamori, N., McGuigan, M., R., Kraemer, W., J. & Nosaka, K. (2009). Reliability of Performance Measurements Derived from Ground Reaction Force Data

During Countermovement Jump and the Influence of Sampling Frequency. *Journal of Strength and Conditioning Research,* 23, 874-882.

Horita, T., Komi, P., Hämäläinen, I. & Avela, J. (2003). Exhausting stretch-shortening cycle (SSC) exercise causes greater impairment in SSC performance than in pure concentric performance. *European Journal of Applied Physiology*, 88, 527-534.

Hough, P., Ross, E. & Howatson, G. (2009). Effects of Dynamic and Static Stretching on Vertical Jump Performance and Electromyographic Activity. *Journal of Strength and Conditioning Research*, 23, 507-512

Inoue, M., Tokuyaa, S., Nakayamada, H. & Ueda, H. (1988). In vivo signal transduction of tetrodotoxin-sensitive nociceptive responses by substance P given into the planta of the mouse hind limb. *Cellular and Molecular Neurobiology,* 18, 555–561.

Jacks, D., E., Sowash, J., Anning, J., McGloughlin, T. & Andres, F. (2002). Effect of Exercise at Three Exercise Intensities on Salivary Cortisol. *Journal of Strength and Conditioning Research*, 16, 286-9.

Jones, D., A. (1996). High- and low-frequency fatigue revisited. *Acta Physiologica Scandinavia*, 156, 265–270.

Katz, B. (1939). The relationship between force and speed in muscular contraction. *Journal of Physiology*, (Lond.) 96, 45-64.

Kaufman, E. & Lamster, I. B. (2002). The Diagnostic Applications of Saliva- A Review. *Critical Reviews in Oral Biology & Medicine*, 13, 197-212.

Keeton, R. B., & Binder-Macleod, S, A. (2006). Low Frequency Fatigue. *Physical Therapy*, 86, 1147-1150.

Khalid, W., Amin, M. & Bober, T. (1989). The influence of upper extremities movement on take-off in vertical jump. In: *Biomechanics in Sports V* (edited by L. Tsarouchas, J., Terauds, B., Gowitzke and L. Holt), pp. 375-379. Athens: Hellenic Sports Research Institute.

Kibele, A. (1998). Possibilities and limitations in the biomechanical analysis of countermovement jumps: a methodological study. *Journal of Applied Biomechanics,* 14, 105-117.

Kilduff, L.P., Bevan, H.R., Kingsley, M.I.C., Owen, N.J., Bennett, M., Bunce, P., et al. (2008). Postactivation Potentiation in Professional Rugby Players: Optimal Recovery. *Journal of Strength and Conditioning Research*, 26, 795-802.

Komi, P., V. (2000). Stretch-shortening cycle: a powerful model to study normal and fatigued muscle. *Journal of Biomechanics,* 33, 1197–1206.

Komi, P., V., Horita, T., Kyröläinen, H., & Takala, T., E. & Nicol, C. (1996). Reduced stretch-reflex sensitivity after exhausting stretch-shortening cycle exercise. *European Journal of Applied Physiology & Occupational Physiology*, 72, 401-409.

Kraemer W., J., Fleck, S., J., Dziados, J., E.,(1993). Changes in hormonal concentrations after different heavy-resistance exercise protocols in women. *Journal of Applied Physiology*, 75, 1310–5.

Kraemer, W., J., French, D., N., Paxton, N., J., Hakkinen, K., Volek, J., S., Sebastianelli, W, J., Putukian, M., Newton, R., U., Rubine, M., R., Gomez, A., L., Vescovi, J., D., Ratamess, N. A. Fleck, S.J. Lynch, J. M. & Knuttgen, H. G. (2004). Changes in exercise performance and hormonal concentrations over a big ten soccer season in starters and nonstarters. *Journal of Strength and Conditioning Research*, 18, 121-128.

N., A., Fleck, S., J., Lynch, J., M. & Knuttgen, H., G. (2004). Changes in Exercise Performance and Hormonal Concentrations Over a Big Ten Soccer Season In Starters and Nonstarters. *Journal of Strength and Conditioning Research*, 18, 121-128.

Kraemer, W., J., Fry, A., C., Rubin, M., R., Triplett-McBride, T., Gordon, S., E., Koziris, L., P., J., M., Lynch, Voleck, J., S., Meuffels, D., E., Newton, R., U. & Fleck, S., J., (2001). Physiological and performance responses to tournament wrestling. *Medicine and Science in Sports and Exercise*, 33, 1367–1378.

Kraemer, W., J., Spiering, B., A., Volek, J., S., Martin, G., J., Howard, R., L., Ratamess, N., A., Hatfield, D., L., Vingren, J., L., Ho, J., Y., Fragala, M., S., Thomas, G., A., French, D., N., Anderson, J., M., Häkkinen, K. & Maresh, C., M. (2009). Recovery from a National Collegiate Athletic Association Division I football game: muscle damage and hormonal status. *Journal of Strength and Conditioning Research,* 23, 2-10.

Kraemer, W., J., Gorden, S., E., Fleck, S., J., Marchitelli, L., J., Mello, R., Diziados, J., E., Freidl, K., Harman, E., Maresh, C. & Fry, A., C. (1991). Endogenous anabolic hormonal and growth factor responses to heavy resistance exercise in males and females. *International Journal of Sports Medicine*, 12, 228-235.

Krustrup, P., Ørtenblad, N., Nielsen, J., Nybo, L., Gunnarsson, T., P., Iaia, F., M., Madsen, K., Stephens, F., GreenhaV, P. & Bangsbo, J. (In Press). Maximal voluntary contraction force, SR function and glycogen resynthesis during the first 72 h after a high-level competitive soccer game. *European Journal of Applied Physiology.*

Lac, G., Berthon, P. (2000). Changes in cortisol and testosterone levels and T/C ratio during an endurance competition and recovery. *Journal of Sports Medicine and Physical Fitness*, 40, 139–144.

Lac, G., Pantelidis, D., Robert, A. (1997). Salivary cortisol response to a 30 min sub maximal test adjusted to a constant heart rate. *Journal of Sports Medicine and Physical Fitness*, 37, 56–60.

Lane, A. M., Lane, H. J. & Firth, S. (2002). Relationships between performance satisfaction and post-competition moos among runners. *Perceptual and Motor Skills*, 94, 805-813.

Larsson, B., Karlsson, S., Eriksson, M., Gerdle, B. (2003). Test-retest reliability of EMG and peak torque during repetitive maximum concentric knee extensions. *Journal of Electromyography and Kinesiology*, 13, 281-7.

Lepers, R., Hausswirth, C., Maffiuletti, N., Brisswalter, J. & van Hoecke, J. (2000). Evidence of neuromuscular fatigue after prolonged cycling exercise. *Medicine and Science in Sports and Exercise*, 32, 1880–1886.

Lepers, R., Maffiuletti, N., A., Rochette, L., Brugniaus, J. & Millet, G.Y. (2002). Neuromuscular fatigue during a long-duration cycling exercise. *Journal of Applied Physiology*, 92, 1487-1493.

LeUnes, A. & Berger, J. (1998). Bibliography on the Profile of Mood States in sport an exercise. 1971-1998. *Journal of Sport Behaviour*, 21, 53-70.

Lieber, R., L. (1992). Skeletal muscle structure and function: Implications for rehabilitation and sports medicine. *Baltimore : Williams & Wilkins.*

Lieber, R., L., Thornell, L-E. & Fridén, J. (1996). Cytoskeletal disruption occurs within 15 minutes of cyclic eccentric contractions. *Journal of Applied Physiology*, 80, 278–284.

Linthorne, N., P. (2001). Analysis of standing vertical jumps using a force platform. *American Journal of Physics,* 69, 1198-1204.

Luhtanen, P. & Komi, P., V. (1978). Segmental contribution to forces in vertical jump. *European Journal of Applied Physiology*, 38, 181-188.

MacIntosh, B., R. & Rassier, D., E. (2002). What is fatigue? *Canadian Journal of Applied Physiology,* 27, 42-55.

Magalhães, J., Rebelo, A., Oliveira, E., Renato Silva, J., Marques, F. & Ascensão, A. (2010). Impact of Loughborough Intermittent Shuttle Test versus soccer match on physiological, biochemical and neuromuscular parameters. *European Journal of Applied Physiology*, 108, 39–48.

Main, L. & Grove, R. (2009). A multi-component assessment model for monitoring training distress among athletes. *European Journal of Sport Science*, 9, 195-202.

Malm, C., Nyberg, P., Engstrom, M., Sjodin, B., Lenkei, R., Ekblom, B. & Lundberg, I. (2000). Immunological changes in human skeletal muscle and blood after eccentric exercise and multiple biopsies. *Journal of Physiology*, 529, 243–262.

Maso, F., Lac, G., Filaire, E., Michaux, O. & Robert, A. (2004). Salivary testosterone and cortisol in rugby players: correlation with overtraining items. *British Journal of Sports Medicine*, 38, 260-263.

Markovic, G., Dizdar, D., Jukic, I. & Cardinale, M. (2004). Reliability and Factorial Validity of Squat and Countermovement Jump Tests. *Journal of Strength and Conditioning Research*, 18, 551-555.

Martin, V., Millet, G., Y., Martin, A., Deley, G. & Lattier, G. (2004). Assessment of low-frequency fatigue with two methods of electrical stimulation. *Journal of Applied Physiology*, 97, 1923–1929.

Matsumoto, K., Takeyasu, K., Mizutani, S., Hamanaka, Y. & Uozumi, T. (1970). Plasma testosterone levels following surgical stress in male patients. *European Journal of Endocrinology*, 65, 11-17.Mazur, A., Booth, A., & Dabbs, J. (1992). Testosterone and chess competition. *Social Psychology Quarterly*, 55, 70–77.

McBride, T., A., Stockert, B.,W., Gorin, F., A. & Carlsen, R., C. (2000). Stretch-activated ion channels contribute to membrane depolarization after eccentric contractions. *Journal of Applied Physiology*, 88, 91-101.

McGuigan, M., R., Egan, A., D. & Foster, C. (2004). Salivary Cortisol Responses and Perceived Exertion During High Intensity and Low Intensity Bouts of Resistance Exercise. *Journal of Sports Science and Medicine*, 3, 8-15.

McHugh, M., P., Tyler, T., F., Greenberg, S., C. & Gleim, G., W. (2002). Differences in activation patterns between eccentric and concentric quadriceps contractions. *Journal of Sports Sciences*, 20, 83-91.

McIntosh, B., R. & Rassier, D., E. (2002). What is Fatigue? Canadian *Journal of Applied Physiology*, 27, 42-55.

McLean, B., D., Coutts, A., J., Kelly, V. & McGuigan, M., R., Cormack, S., J. (2010). Neuromuscular, endocrine, and perceptual fatigue responses during different length between-match microcycles in professional rugby league players. *International Journal of Physiology and Performance,* 5, 367-83.

McLean, D., A. (1992). Analysis of the physical demands of international rugby union. *Journal of Sports Sciences*, 10, 285–296.

McLellan, C., P., Lovell, D., I., and Gass, G., C. (2010). Creatine kinase and endocrine responses of elite players pre, during, and post rugby league match play. *Journal of Strength and Conditioning Research,* 24, 2908–2919.

McLellan, C., P., Lovell, D., I., and Gass, G., C. (2011). Markers of post-match fatigue in professional rugby league players. *Journal of Strength and Conditioning Research.* 25, 1030-1039.

McLellan, C. P., Lovell, D. I. & Gass, G. C. (2011). Biochemical and endocrine responses to impact and collision during elite rugby league match play. *Journal of Strength and Conditioning Research*, 25, 1553-1562.

McLellan, C. P., Lovell, D. I. & Gass, G. C. (2011). Performance analysis of elite rugby league match play using global positioning systems. *Journal of Strength and Conditioning Research*, 25, 1703-1710.

McLellan, C. P. & Lovell, D. I. (2012). Neuromuscular responses to impact and collision during elite rugby league match play. *Journal of Strength and Conditioning Research*, 26, 1431-1440.

McNair, D. M., Lorr, M. & Droppleman, L. (1971). Manual for the Profile of Mood States. San Deigo, CA. Educational and Industrial Testing Service.

Millet, G., Y., Lepers, R., Maffiuletti, N., A., Babault, N., Martin, V. & Lattier, G. (2002). Alterations of neuromuscular function after an ultramarathon. *Journal of Applied Physiology*, 92, 486–492.

Millet, G., Y., Martin, V., Maffiuletti, N., A. & Martin, A. (2003). Neuromuscular fatigue after a ski skating marathon. *Canadian Journal of Applied Physiology*, 28, 434-45.

Moir, G, Sanders, R., Button, C. & Glaister, M. (2005). The influence of familiarization on the reliability of force variables measured during unloaded and loaded vertical jumps. *Journal of Strength and Conditioning Research*, 19, 40-145.

Moir, G., Button, C., Glaister, M. & Stone, M., H. (2004). Influence of Familiarization on the Reliability of Vertical Jump and Acceleration Sprinting Performance in Physically Active Men. *Journal of Strength and Conditioning Research*, 18, 276-280.

Moore, R., L. & Stull, J., T. (1984). Myosin light chain phosphorylation in fast and slow skeletal muscles in situ. *American Journal of Physiology - Cell Physiology*, 247, C462-C471.

Morgan, W. P., Brown, D. R., Raglin, J. S., O'Connor, P. J., Ellickson, K. A. (1987). Psychological monitoring of overtraining and staleness. *British Journal of Sports Medicine*, 21, 107-114.

Morgan, W.P., Costill, D. L., Flynn, M. G., Raglin, J. S. & O'Connor, P. J. (1988). Mood disturbances following increased training in swimmers. *Medicine and Science in Sports and Exercise*, 20, 408-414.

Morgan, D., L. & Allen, D., G. (1999). Early events in stretch-induced muscle damage. *Journal of Applied Physiology*, 87, 2007–2015.

Morgan, D., L. (1990). New insights into the behaviour of muscle during active lengthening. *Biophysical Journal*, 59, 209-221.

Moritani, T., Muramatsu, S., Muro, M. (1988). Activity of motor units during concentric and eccentric contractions. *American Journal of Physical Medicine & Rehabilitation*, 66, 338-50.

Newham, D., J., Jones, D., A. & Clarkson, P., M. (1987). Repeated high force eccentric exercise effects on muscle pain and damage. *Journal of Applied Physiology*, 63, 1381–1386.

Newham, D., J., Mills, K., R., Quigley, B., M. & Edwards, R., H., T. (1983). Pain and fatigue after concentric and eccentric muscle contractions. *Clinical Science*, 64, 55–62.

Nicolls, A. R., Backhouse, S. H., Polman, R. C. & McKenna, J. (2009). Stressors and affective states among professional rugby union players. *Scandinavian Journal of Medicine and Science in Sports*, 19, 121-128.

Nicol, C., Avela, J. & Komi, P., V. (2006). The Stretch-Shortening Cycle: A Model to Study Naturally Occurring Neuromuscular Fatigue. *Sports Medicine*, 36, 977-999.

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

Nicol, C., Komi, P., V., Horita, T., Kyrolainen, H. & Takala, T., E. (1996). Reduced stretch-reflex sensitivity after exhausting stretch-shortening cycle exercise. *European Journal of Applied Physiology and Occupational Physiology*, 72, 401–409.

Nicol, C., Kuitunen, S,. Kyrolainen, H., Avela, J. & Komi, P., V. (2003). Effects of long and short term fatiguing exercises on reflex EMG and force of the tendon-muscle complex. *European Journal of Applied Physiology*, 90, 470–479.

Owen, N. J. (2008). Development of a field test to estimate peak vertical mechanical power of elite male rugby union players. Master of Philosophy Thesis, Swansea University.

Oppliger, R. A. & Bartock, C. (2002). Hydration Testing of Athletes. *Sports Medicine*, 32, 959 – 957.

Papacosta, E. & Nassis, G. P. (2011). Saliva as a tool for monitoring steroid, peptide and immune markers in sport and exercise science. *Journal of Science and Medicine in Sport*, 14, 424-434.

Passelergue, P. & Lac, G. (1999). Saliva cortisol, testosterone and T/C ratio variations during a wrestling competition and during the post-competitive recovery period. *International Journal of Sports Medicine*, 20, 109–113.

Passelergue, P., Robert, A. & Lac, G. (1995). Salivary cortisol and testosterone variations during an official and a simulated weightlifting competition. *International Journal of Sports Medicine*, 16,298–303.

Peterson, K., Hansen, C, B., Aagaard, P. & Madsen, K. (2007). Muscle mechanical characteristics in fatigue and recovery from a marathon race in highly trained runners. *European Journal of Applied Physiology*, 101, 385-396.

Pettorossi, V., E., Della, G., Bortolami, R. & Brunnetti, O. (1999). The role of capsaicin-sensitive muscle afferents in fatigue-induced modulation of the monosynaptic reflex in the rat. *Journal of Physiology,* 515, 599–607.

Pizza, F., X., Davis, B., H., Henrickson, S., D., Mitchell, J., B., Pace, J., F., Bigelow, N., DiLauro, P. & Naglieri, T. (1996). Adaptation to eccentric exercise: effect on CD64 and CD11b/CD18 expression. *Journal of Applied Physiology*, 80, 47-55.

Pizza, F., X., Koh, T., J., McGregor, S., J. & Brooks, S., V. (2002). Muscle inflammatory cells after passive stretches, isometric contractions, and lengthening contractions. *Journal of Applied Physiology*, 92, 1873-8.

Place, N., Lepers, R., Deley, G. & Millet, G.Y. (2004). Time Course of Neuromuscular Alterations during a Prolonged Running Exercise. *Medicine and Science in Sports and Exercise*, 36, 1347-1356.

Place, N., Yamada, T., Bruton, J., D. & Westerblad, H. (2010). Muscle fatigue: from observations in humans to underlying mechanisms studied in intact single muscle fibres. *European Journal of Applied Physiology*, 110, 1-15.

Pyne, D., B. (1994). Regulation of neutrophil function during exercise. *Sports Medicine*, 17, 245–58.

Raglin, J. S., Eksten, F. & Garl, T. (1995). Mood stage responses to a pre-season conditioning program in male collegiate basketball players. *International Journal of Sport Psychology*, 26, 214-224.

Rassier, D., E., McIntosh, B., R. (2000). Coexistence of potentiation and fatigue in skeletal muscle. *Brazilian Journal of Medical and Biological Research*, 33, 499–508.

Ratamess, N, A., Kraemer, W., J., Volek, J.,S., Maresh, C., M., Vanheest, J., L., Sharman, M., J., Rubin, M., R., French, D., N., Vescovi, J., D., Silvestre, R., Hatfield, D., L., Fleck, S., J. & Deschenes, M., R. (2005). Androgen receptor content following heavy resistance exercise in men. *Journal of Steroid Biochemistry and Molecular Biology*, 93, 35–42.

Requena, B., Gonzalez-Badillo, J., J., De Villareal, E., Ereline, J., Garcıa, I., Gapeyeva, H. & Pääsuke, M. (2009). Functional performance, maximal strength, and power characteristics in isometric and dynamic actions of lower extremities in soccer players. *Journal of Strength and Conditioning Research*, 23, 1391–1401.

Roberts, S., P., Stokes, K., A., Weston, L. & Trewartha, G (2010). The Bath University Rugby Shuttle Test (BURST): a pilot study. *International Journal of Sports Physiology and Performance*, 5, 64-74.

Robins, R. W., Hendin, H. M. & Trzesniewski, K. H. (2001). Measuring Global Self Esteem: Construct Validation of a Single-Item Measure and the Rosenberg Self-Esteem Scale. *Personality and Social Psychology Bulletin*, 27, 151-161.

Rodacki, A., L., F., Fowler, N., E. & Bennet, S., J. (2002). Vertical jump coordination: fatigue effects. *Medicine and Science in Sports and Exercise*, 34, 105-116.

Rome, L., C., Morgan, D., L. & Julian, F., J. (1985). Stimulation rate, potentiators, and sarcomere length-tension relationship of muscle. *American Journal of Physiology*, 249, C497–C502.

Ronglan, L.T., Rastaad, T. & Borgesen, A. (2006). Neuromuscular fatigue and recovery in elite female handball players. *Scandinavian Journal of Medicine & Science in Sports*, 16, 267-273.

Rossi, A. & Scarpini, C. (1993). Evidence for presynaptic inhibition on trigeminal primary afferent fibres in humans. *Journal of Neurology Neurosurgery & Psychiatry*, 56, 424–425.

Rotto, D., M. & Kaufman, M., P. (1988). Effect of metabolic products of muscular contraction on discharge of group III and IV afferents. *Journal of Applied Physiology*, 64, 2306-2313.

Rybicki, K., Waldrop, T. & Kaufman, M. (1985). Increasing gracilis muscle interstitial potassium concentrations stimulate group III and IV afferents. *Journal of Applied Physiology*. 58, 936–941.

Sacham, S. (1983). A Shortened Version of the Profile of Mood States. *Journal of Personality Assessment*, 47, 305-315.

Salles, A., S., Baltzopoulos, V. & Rittweger, J. (2011). Differential effects of countermovement magnitude and volitional effort on vertical jumping. *European Journal of Applied Physiology*, 111, 441-8.

Sapolsky, R. M., Romero, M. & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory and preparative actions. *Endocrine Reviews*, 21, 55-89.

Salvador, A., Suay, F., Martinez-Sanchis, S., Simon, V., M. & Brain, P., F. (1999). Correlating testosterone and fighting in male participants in judo contests. *Physiology & Behaviour*, 68, 205–209.

Sánchez-Medina, L., González-Badillo, J., J. (In Press). Velocity Loss as an Indicator of Neuromuscular Fatigue during Resistance Training. *Medicine and Science in Sports and Exercise.*

Sargeant, A., J. & Dolan, P. (1987). Human muscle function following prolonged eccentric exercise. *European journal of applied physiology and occupational physiology*, 56, 704–711.

Skurvydas, A., Dudoniene, V., Kalvenas, A. & Zuoza, A. (2002). Skeletal muscle fatigue in long distance runners, sprinters and untrained men after repeated drop jumps performed at maximal intensity. *Scandinavian Journal of Medicine & Science in Sports*, 12, 34-39.

Skurvydas, A., Jascaninas, J. & Zachovajevas, P. (2000). Changes in height of jump, maximal voluntary contraction force and low-frequency fatigue after 100 intermittent or continuous jumps with maximal intensity. *Acta Physiologica Scandinavia*, 169, 55-62.

Skurvydas, A., Kamandulis, S., Masiulis, N. (2010). Two series of fifty jumps performed within sixty minutes do not exacerbate muscle fatigue and muscle damage. *Journal of Strength and Conditioning Research*, 24, 929-35.

Skurvydas, A., Sipaviciene, S., Krutulyte, G., Gailiuniene, A., Stasiulis, A., Mamkus, G. & Stanislovaitis, A. (2006). Dynamics of indirect symptoms of skeletal muscle damage after stretch-shortening exercise. *Journal of Electromyography and Kinesiology*, 16, 629-36

Smart, D., J., Gill, N., D., Beaven, C., M., Cook, C., J. & Blasevich, A., J. (2008). The relationship between changes in interstitial creatine kinase and game-related impacts in rugby union, *British Journal of Sports Medicine*, 42, 198-201.

Smith, L., L. (1991). Acute Inflammation: The Underlying Mechanism in Delayed Onset Muscle Soreness? *Medicine and science in sports and exercise,* 23, 542-51.

Snegovskaya, V. & Viru, A. (1993). Steroid and pituitary hormone responses to rowing: relative significance of exercise intensity and duration and performance level. *European Journal of Applied Physiology*, 67, 59–65.

Søgaard, K., Blangsted, A., K., Jørgensen, L., V., Madeleine, P. & Sjøgaard, G. (2003). Evidence of long term muscle fatigue following prolonged intermittent contractions based on mechano- and electromyograms. *Journal of Electromyography and Kinesiology*, 13, 441-50.

Steenberg, A., Fischer, C., P., Keller, C., Møller, K. & Pedersen, B., K. (2003). IL-6 enhances plasma IL-1ra, IL-10, and cortisol in humans. *American Journal of Physiology - Endocrinology and Metabolism*, 285, E433–E437.

Stokes, P. (1995). The potential role of excessive cortisol induced by HPA hyperfunction in the pathogenesis of depression. *European Neuropsychopharmacology Supplement*, 77-82.

Stone, M. H., K. Sanborn, O'Bryant, H., S., Hartman, M., Stone, M., E., Proulx, C., Ward, B. & Hruby, J. (2003). Maximum strength-power-performance relationships in collegiate throwers. *Journal of Strength and Conditioning Research,* 17, 739-745.

Street, G. (2001). Sources of error in determining countermovement jump height with the impulse method. *Journal of Applied Biomechanics*, 17, 43-54.

Strojnik, V. & Komi, P., V. (2000). Fatigue after submaximal intensive stretch-shortening cycle exercise. *Medicine and Science in Sports and Exercise*, 32, 1314-1319.

Strojnik, V. & Komi, P.V. (1998). Neuromuscular fatigue after maximal stretch-shortening cycle exercise. *Journal of Applied Physiology*, 84, 344-350.

Suay, F., Salvador, A., Gonzalez-Bono, E., Sanchis, C., Martinez, M., Martinez-Sanchis, S., V., M. & Montoro, J., B. (1999). Effects of competition and its outcome on serum testosterone, cortisol and *prolactin. Psychoneuroendocrinology,* 24, 551–566.

Suzuki, M., Umeda, T., Nakaji, S., Shimoyama, T., Mashiko, T. & Sugawara, K. (2004). Effect of incorporating low intensity exercise into the recovery period after a rugby match. *British Journal of Sports Medicine*, 38, 436-440.

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

Terry, P. C., Lane, A. M., & Fogarty, G. (2003). Construct validity of the Profile of Mood States-A for use with adults. *Psychology of Sport and Exercise*, 4, 125-139.

Thorlund, J., B., Aagaard, P. & Madsen, K. (2009). Rapid Muscle Force Capacity Changes after Soccer Match Play. *International Journal of Sports Medicine*, 30(4): 273-278.

Thorlund, J., B., Michalsik, L., B., Madsen, K. & Aagaard, P. (2008). Acute fatigue-induced changes in muscle mechanical properties and neuromuscular activity in elite handball players following a handball match. *Scandinavian Journal of Medicine and Science in Sports*, 18, 462–472.

Toumi, H., Poumarat, G., Best, A., Martin, G., Fairclough J. & Benjamin, M. (2006). Fatigue and muscle-tendon stiffness after stretch-shortening cycle and isometric exercise. *Applied Physiology Nutrition and Metabolism*, 31, 565-572.

Tremblay, M.S., Copeland, J.L. & Van Helder, W. (2005). Influence of exercise duration on post-exercise steroid hormone responses in trained males. *European Journal of Applied Physiology*, 94, 505-551.

Turner, D., C., Wallimann, T. & Eppenberger, H. (1973). A Protein That Binds Specifically to the M-Line of Skeletal Muscle Is Identified as the Muscle Form of Creatine Kinase. *Proceedings of the National Academy of Sciences of the United States of America*, 70, 702-705.

Twist, C. & Eston, R. (2005). The effects of exercise-induced muscle damage on maximal intensity intermittent exercise performance. *European Journal of Applied Physiology,* 94, 652–658.

Vanhelder, W., .P., Radomski, M., W., Goode, R., C. & Casey. K. (1985). Hormonal and metabolic responses to three types of exercise of equal duration and external work output. *European Journal of Physiology,* 54, 337–342.

Vanrenterghem, J., DeClercq, D. & Van Cleven, P. (2001). Necessary precautions in measuring correct vertical jumping height by means of force plate measurements. *Ergonimics*, 44, 814-818.

Verin, E., Ross, E., Demoule, A., Hopkinson, N., Nickol, A., Fauroux, B., Moxham, J., Similowski, T. & Polkey, M.I. (2004). Effects of exhaustive incremental treadmill exercise on diaphragm and quadriceps motor potentials evoked by transcranical magnetic stimulation. *Journal of Applied Physiology*, 96, 253-259.

Viitasalo, J., P., Komi, P., V., Jacobs, I. & KArlsson, J. (1982). Effects of prolonged cross-country skiing on neuromuscular performance. In: Exercise and Sport Biology, International Series on Sport Sciences, Vol. 12, and P. V. Komi (Ed.). Champaign, IL. Human Kinetics Publishers, 1982, pp. 191–198. In: Lepers, R., Hausswirth, C., Maffiuletti, N., Brisswalter, J. & van Hoecke, J. (2000). Evidence of neuromuscular fatigue after prolonged cycling exercise. *Medicine and Science in Sports and Exercise*, 32, 1880–1886.

Viitasalo, J., T. (1982). Effects of pretension on isometric force production. *International Journal of Sports Medicine,* 3, 149-52.

Vøllestad, N., K. (1997). Measurement of human muscle fatigue. *Journal of Neuroscience Methods*, 74, 219-27.

Vuorimaa, T., Vasankari, T., Mattila, K., Heinonen, O., Hakkinen, K. & Rusko, H. (1999) Serum hormone and myocellular protein recovery after intermittent runs at the velocity associated with VO2max. *European Journal of Applied Physiology*, 80, 575–581.

Warren, G., L., Lowe, D. A. Hayes, D., A., Karwoski, C., J., Prior, B., M. & Armstrong, R., B. (1993). Excitation failure in eccentric contraction induced injury of mouse soleus muscle. *Journal of Physiology*, 468, 487–499.

Westerblad, H. & Allen, D., G. (1994). The role of sarcoplasmic reticulum in relaxation of mouse muscle; effects of 2,5-di(tert-butyl)-1,4- benzohydroquinone. *Journal of Physiology*, 474, 291–301.

Westerblad, H., Allen, D., G. & Lannergren, J. (2002). Muscle Fatigue: Lactic Acid or Inorganic Phosphate the Major Cause? *News in physiological sciences*, 17, 17-21.

Westerblad, H., Duty, S. & Allen, D., G. (1993). Intracellular calcium concentration during low-frequency fatigue in isolated single fibres of mouse skeletal muscle. *Journal of Applied Physiology,* 75, 382-388.

Westgaard, R,. H., de Luca, C.,J., (1999). Motor unit substitution in long duration contractions of the human trapezius muscle. *Journal of Neurophysiology*, 82, 501-4.

Wilkerson, J., E., Horvath, S., M. & Gutin, B. (1980). Plasma testosterone during treadmill exercise. *Journal of Applied Physiology*, 49, 249-53.

Wilson, G., J. & Murphy, A., J. (1996). The use of isometric tests of muscular function in athletic assessment. *Sports Medicine*, 22, 19-37.

Zemkova, E. & Hamar, D. (2009). The effect of soccer match induced fatigue on neuromuscular performance. *Kinesiology*, 41, 195-202.

CHAPTER EIGHT

8.0 Appendices

Appendix A: Swansea University ethical approval documents

SPORT AND EXERCISE SCIENCE
SCHOOL OF ENGINEERING, SWANSEA UNIVERSITY

ETHICAL ADVISORY COMMITTEE

APPLICATION FOR ETHICAL COMMITTEE APPROVAL OF A RESEARCH PROJECT

In accordance with Departmental Safety Policy, all research undertaken in the department must be approved by the Departmental Ethics Advisory Committee **prior to** data collection. **Applications for approval should be typewritten on this form using the template available in the Public Folders.** The researcher(s) should complete the form in consultation with the project supervisor. Where appropriate, the application must include the following appendices:

(A) subject information sheet;
(B) subject consent form;
(C) subject health questionnaire.

After completing sections 1-12 of the form, 1 copy of the form should be handed-in to the Department Administrator who will then submit copies of the application for consideration by the Departmental Ethics Advisory Committee. The applicant(s) will be informed of the decision of the Committee in due course.

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1. DRAFT TITLE OF PROJECT

Neuromuscular, endocrine and mood responses to an elite rugby union match

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2. NAMES AND STATUS OF RESEARCH TEAM

Dr Liam Kilduff: Supervisor

Mr Nick Owen: Biomechanist

Dr Christian Cook: UKSport Consultant

Mr Brad Harrington: Scarlets Rugby

Mr Phil Scott: Postgraduate student

Miss Charlotte Finn: Postgraduate student

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3. RATIONALE

Published literature abounds with reports concerning the effects of competitive high intensity intermittent performance on both neuromuscular and endocrine markers of fatigue. Several studies have investigated both the neuromuscular and endocrine responses to high intensity intermittent sports such as American Football (Hoffman et al ., 2002, 2005)

Australian Rules Football (Cormack et al ., 2008) and Rugby Union (Elloumi et al ., 2003; Takarada 2003). These sports have a high intensity, intermittent nature, showing specific work intensities and recovery periods that vary depending on player position (Glaister, 2005). During a rugby match, players perform repeated high intensity stretch shortening cycle movements (Duthie, Pyne & Hooper, 2003; Komi, 2000), and are exposed to a high frequency of contact, both of which are associated with muscle damage (Peake et al ., 2005; Feasson et al ., 2002; Fielding et al ., 1993; Takarada, 2003), typically characterised by morphological alterations within muscle tissue (Peake et al ., 2005), followed by an inflammatory response (Doussett et al ., 2007). A common trend in the literature regarding elite high intensity intermittent team sport is that of a bimodal pattern of force recovery after intensive stretch-shortening cycle movements (Doussett et al ., 2007; Nicol et al ., 1991). The immediate decline in performance is generally associated with acute, metabolically induced reactions followed by a short term recovery. This is followed by a secondary reduction associated with inflammatory and muscle remodelling processes, the effects of which last over a period of 2-6 days (Dousset et al ., 2007).

Competitive team sports including Rugby Union, American Football and Australian Rules Football are also associated with fluctuations in anabolic and catabolic hormones including testosterone and cortisol (Hoffman et al ., 2002, 2005; Elloumi et al ., 2003; Cormack et al ., 2008), indicative of an athlete's anabolic/catabolic state (Crewther et al ., 2009) as well as an endocrinological marker of the physiological stress associated with exercise (Hoffman et al., 2002). Studies have reported consistent elevations in cortisol in response to competition; however the response of testosterone is unclear. Some studies have reported increases in testosterone in response to competition and exercise (Elias, 1985; Kraemer et al ., 2001; Gray et al ., 1993; Hakkinen & Pakarinen, 1993; Hoffman et al ., 1997), others have reported decreases (Webb et al ., 1984; Kraemer, 1992; Kraemer et al ., 2001; Elloumi et al ., 2003), whereas other studies have reported no changes in response to competition (Hoffman et al ., 2002). This equivocality may be a result of different sports used to examine the responses of varying sports (wrestling vs. rugby union), different testing protocols (lab based vs. field based), and varying levels of subject motivation (competition vs. exercise based protocols), as well as the use of non-elite subjects with poor training histories.

Additionally, some studies have shown fluctuations in psychological variables in response to physical exertion. However, the vast majority of research in this area has focussed on overtraining and over-reaching, rather than psychological responses to matches over the post competition recovery period. Some literature suggests that this is an important factor in athlete's recovery (Suzuki et al., 2004; Maso et al., 2006).

It is well established that elite athletes must train aggressively to perform at a high level, and are in a state of training-fatigue adaptation (Fowles, 2006). Therefore the aim of this study is to characterise the effects of a professional rugby union match on neuromuscular, endocrine and mood markers of fatigue. Results from this study will provide information on fatigue arising from a rugby match, which is important because it allows coaching staff to determine an athlete's ability to recover, help identify appropriate training

loads to maximise performance, as well as assess the effectiveness of recovery strategies to prepare athletes for subsequent bouts of training and/or competition.

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4. REFERENCES

Doussett, E., Avela, J., Ishikawa, M., Kallio, J., Kuitunen, S., Kyrolainen, H., Linnamo, V. & Komi, P. (2007). Biomodal Recovery Pattern in Human Skeletal Muscle Induced by Exhaustive Stretch-Shortening Cycle Exercise. *Medicine & Science in Sports and Exercise*, **39**, 453-460.

Duthie, G.M., Pyne, D. & Hooper, S. (2005). Time motion analysis of 2001 and 2002 Super 12 Rugby. *Journal of Sports Sciences*, **23**, 523-530.

Elias, M. (1981). Serum cortisol, testosterone and testosterone-binding globulin responses to competitive fighting in human males. *Aggressive Behaviour*, **7**, 215-224.

Feasson, L., D. Stokhom, D., Freysssenet, Richard, I., Duguez, S., Beckmann, J.S. & Denis, C. (2002). Molecular adaptations of neuromuscular disease-associated proteins in response to eccentric exercise in human skeletal muscle. *Journal of Physiology*, **543**, 297-306.

Fielding, R. A., Manfredi, T. J., Ding, W., Fiatarone, M. A., Evans, W. J. & Cannon, J. G. (1993). Acute phase response in exercise III. Neutrophil and IL-beta accumulation in skeletal muscle. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, **265**, 166-172.

Fowles, J.R. (2006). Technical Issues in Quantifying Low-Frequency Fatigue in Athletes. *International Journal of Sports Physiology and Performance*, **1**, 169-171.

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

Hoffman, J.R., Maresh, C. M., Newton, R. U., Rubin, M. R., French, D. N. & Volek J. S. et al . (2002). Performance, biochemical and endocrine changes during a competitive football game. *Medicine and Science in Sports and Exercise*, **34**, 1845-1853.

Komi, P. (2000). Stretch shortening cycle: a powerful model to study normal and fatigued muscle. *Journal of Biomechanics*, **33**, 1197-1206.

Kraemer, W.J, Fry, A.C. & Rubin, M.R. et al . (2001). Physiological and performance responses to tournament wrestling. *Medicine and Science in Sports and Exercise*, **33**, 1367-1378.

Nicol, C., Komi, P.V. & Marconnet, P. (1991). Fatigue effects of marathon running on neuromuscular performance I: Changes in force integrated electromyographic activity and endurance capacity. *Scandinavian Journal of Medicine and Science in Sports*, **1**, 18-24.

Peake, J.M., Suzuki, K., Wilson, G., Horden, M., Nosaka, K., Mackinnon, L. & Coombes. J.S. (2005). Exercise-Induced Muscle Damage, Plasma Cytokines, and Markers of Neutrophil Activation. *Medicine and Science in Sports and Exercise*, **37**, 737-745.

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

5. AIMS and OBJECTIVES

The aim of the proposed study is to assess the neuromuscular, endocrine and mood responses to an elite rugby union match.

1) Assess the effects of a competitive rugby match on a countermovement jump as an indicator of neuromuscular performance

2) Assess the effects of a competitive rugby match on endogenous free testosterone and cortisol concentrations

3) 2) Assess the effects of a competitive rugby match on mood state with the use of a Brief Assessment of Mood questionnaire.

6. METHODOLOGY

6.1 Study Design

The study will take place over a 72 h period around one competitive rugby match. Data will be collected at the same time of day on each trial to avoid the effects of circadian rhythms. The subjects used in this study will be elite rugby union players (n = 20). Should we see clear differences in neuromuscular, hormonal and mood states, this may help in quantifying the time course of recovery of these variables. Athletes who are not suffering from illness or injury are able to participate having completed a pre-participation screening questionnaire (AHA/ACSM Health/Fitness Facility Pre-participation Screening Questionnaire) as well as

subject information and subject consent form. Subjects will also be familiarized with testing procedures prior to collecting any data.

6.2 Experimental Procedures

On arrival participants will be required to fill in a questionnaire supplying information on mood state (Brief Assessment of Mood), as well as provide saliva samples having not ingested any food/fluids for 30 min prior to supplying the sample. Saliva samples will be obtained for subsequent enzyme linked immunoassay for testosterone and cortisol, before completing a standardized warm up. Following the warm up, subjects will perform 1 maximal effort CMJ on a portable force platform (Kistler Instruments, Hampshire, UK) for subsequent analysis of power (eccentric and concentric phase), rate of force development (eccentric and concentric phase), and jump height. Measurements will be repeated at the same time of day for 60 h following the match.

6.3 Data Analysis Techniques

Saliva samples will be analysed in duplicate via enzyme linked immunoassay (ELISA) having been stored at -80°C, and force traces will be analysed via numerical integration to determine variables including peak power (W) (concentric and eccentric), rate of force development ($N.s^{-1}$) (concentric and eccentric), and jump height (m). Values will be presented as mean ± standard deviation (SD). Data will be collated and processed in a Microsoft Excel spreadsheet (Microsoft Corporation, USA). Data will be used to derive descriptive statistics and graphical interpretations. Statistical analysis of the data will be carried out using a Statistical Package for the Social Sciences (SPSS 16.0 for Windows, Release 16.0 1 December 2007) to determine any differences and/or relationships between the data sets. Normally distributed data will allow a one way, repeated measures analysis of variance (ANOVA) to determine differences in neuromuscular, endocrine or mood data. If significant differences are found, a Bonferroni post hoc will be used to identify the location of these differences.

6.4 Storage and Disposal of Data and Samples

All data collected concerning members of Scarlets Rugby will be kept strictly confidential with professionalism and anonymity assured. The data will be used solely for this study and will be kept under the Data Protection Act. Access to this data will be strictly exclusive to members of the research team, Scarlets Rugby Coaching Staff and the participants at their request. Data will be deleted at the end of the study. Saliva samples will be stored in line with the Human Tissues and disposed of as medical waste once assayed.

6.5 Dietary supplementation

No dietary supplements are involved in this research.

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7. LOCATION OF THE PREMISES WHERE THE RESEARCH WILL BE CONDUCTED.

The testing for this investigation will take place at Parc Y Scarlets, Llannelli. Force platform and ELISA techniques for saliva sample analysis will be carried out at the Sports Science laboratory at Swansea University. Testing and data analysis will be supervised by Dr. Kilduff.

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8. SUBJECT RISKS AND DISCOMFORTS

Participants will be fully informed of all procedures and risks, and will be required to complete a medical questionnaire. Written consent before must also be given before any testing occurs. Participants will be required to perform maximal effort on countermovement jumps which carries with it little physical risk; bearing in mind these subjects are well trained. However, a standardised warm up will be completed before any testing commences to minimise risk of injury. Subjects will also be required to supply saliva as well as information on mood state, but only if content with doing so. Any participants who do not feel comfortable with this may withdraw from the study at any time.

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9. INFORMATION SHEET AND INFORMED CONSENT

Have you included a Subject Information Sheet for the participants of the study? YES

Have you included a Subject Consent Form for the participants of the study? YES

If written consent will not be obtained, explain why.

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10. COMPUTERS

Are computers to be used to store data? YES

If so, is the data registered under the Data Protection Act? YES

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11. STUDENT DECLARATION

Please read the following declarations carefully and provide details below of any ways in which your project deviates from them. Having done this, each student listed in section 2 is required to sign where indicated.

1. I have ensured that there will be no active deception of participants.

2. I have ensured that no data will be personally identifiable.

3. I have ensured that no participant should suffer any undue physical or psychological discomfort

4. I certify that there will be no administration of potentially harmful drugs, medicines or foodstuffs.

5. I will obtain written permission from an appropriate authority before recruiting members of any outside institution as participants.

6. I certify that the participants will not experience any potentially unpleasant stimulation or deprivation.

7. I certify that any ethical considerations raised by this proposal have been discussed in detail with my supervisor.

8. I certify that the above statements are true with the following exception(s):

Student signature: (include a signature for each student in research team)

Date:

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12. SUPERVISOR'S DECLARATION

In the supervisor's opinion, this project (delete those that do not apply):

- Does not raise any significant issues.
- Raises some ethical issues, but I consider that appropriate steps and precautions have been taken and I have approved the proposal.
- Raises ethical issues that need to be considered by the Departmental Ethics Committee.
- Raises ethical issues such that it should not be allowed to proceed in its current form.

Supervisor's signature: Date:

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13. ETHICS COMMITTEE DECISION (COMMITTEE USE ONLY)

ETHICAL APPROVAL: GRANTED REJECTED (delete as appropriate)

The ethical issues raised by this project have been considered by members of the Departmental Ethical Approval Committee who made the following comments:

.....

.....

.....

.....

.....

.....

Please ensure that you take account of these comments and prepare a revised submission that should be shown to your supervisor/ resubmitted to the Department Ethical Approval Committee (delete as appropriate).

Signed: Date:

(Chair, Departmental Ethics Advisory Committee)

Appendix B: Swansea University Subject Information Sheet & Consent Form

DEPARTMENT OF SPORTS SCIENCE

SUBJECT INFORMATION SHEET

Date: 01/02/2010

Contact Details:

Supervisor: Dr. Liam Kilduff

Swansea University

Singleton Park

Swansea

SA2 8PP

Wales, UK

Tel. 01792 205678

Postgraduate: Charlotte Finn (07746503127)

1. Study title

Neuromuscular, endocrine and mood responses to an elite rugby union match

2. Invitation paragraph

This study aims to assess neuromuscular, endocrine and mood responses to an elite rugby union match. Should we see changes in any of these markers, it may help assess the use of active recovery in team sports and identify an athlete's ability to recovery and prescribe appropriate training loads following intense bouts of competition.

3. What is the purpose of this study?

The purpose of this study is to neuromuscular, endocrine and mood responses to an elite rugby union match in a group of elite rugby players following a competitive match. The results obtained may help coaches to identify appropriate recovery strategies and training loads following competition.

4. Why have I been chosen?

You have been chosen to participate in the study due to your elite performance in the Rugby Union. The study is completely voluntary and you may withdraw at any time during testing without giving reason.

5. What will happen to me if I take part?

Subjects will be required to participate in a total of 4 testing sessions before, and following 1 rugby match. Sessions will involve providing saliva samples for hormonal analyses and completing a questionnaire regarding mood state. Participants must complete a standardized warm up before completing 1 maximal effort CMJs.

6. What are the possible disadvantages of taking part?

Participants will be required to complete a medical questionnaire and provide written consent before any testing begins. Participants must ensure that they are content with supplying information before they agree to partake in this study. Participants may withdraw from the study at any time without giving reason. Any participants who develop illness or injury during testing may be asked to withdraw to minimise risk of further injury/illness to the participants as well as inaccuracy of data collected. Due to the elite status of these athletes, it is unlikely that this study carries with it any risk of injury; however precautions will be taken by ensuring the participants have completed a standardised warm up before any testing occurs.

7. What are the possible benefits of taking part?

Upon completion of the study, participants may gain an improved understanding of their ability to recover from a competitive rugby match. Findings from this study will potentially help inform future studies in monitoring fatigue following a rugby match, and also help direct training loads following elite rugby matches with the aim to recover in preparation for the next bout of competition. Participants may arrange to meet after the testing to provide an explanation and interpretation of their own results.

8. Will my taking part in the study be kept confidential?

All personal information acquired during the course of the study will be kept strictly confidential within the research team. Upon completion of the study, written documentation that leaves research team will have all personal information removed so that it will not be possible to identify the subjects.

DEPARTMENT OF SPORTS SCIENCE
SUBJECT CONSENT FORM

Contact Details:

Complete details including name and contacts

Project Title:

Title of the project

Please initial box

1. I confirm that I have read and understood the information sheet dated ...../...../..... (Version number .....) for the above study and have had the opportunity to ask questions. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐
3. I understand that sections of any of data obtained may be looked at by responsible individuals from the University of Wales Swansea or from regulatory authorities where it is relevant to my taking part in research. I give permission for these individuals to have access to these records. ☐
4. I agree to take part in the above study.

Name of Subject	Date	Signature
Name of Person taking consent	Date	Signature
Researcher	Date	Signature

Appendix C: Force Platform Raw Data

Table 6.0: Body weight (kg) at baseline, 12, 36 and 60 h post-match.

Subject	Game Time (min)	Body Weight (kg)			
		Baseline	12 h	36 h	60 h
1	73	117.4	115.0	116.8	117.4
2	51	111.8	114.2	114.7	113.6
3	88	119.5	117.3	121.0	119.5
4	69	123.5	122.3	123.9	122.9
5	88	119.4	115.5	116.9	116.1
6	88	110.2	108.4	109.3	109.9
7	75	98.8	96.5	96.6	97.1
8	82	92.9	91.4	93.0	93.8
9	88	96.6	95.1	96.3	95.5
10	88	96.8	98.7	98.6	97.0
11	88	106.2	107.0	108.0	106.4
12	88	92.7	92.1	92.9	92.3
13	88	106.6	106.9	108.3	108.1
14	88	80.1	81.6	81.4	81.7
Mean	81.57	105.18	104.43	105.55	105.09
S.D	11.03	12.67	11.99	12.58	12.31

Table 6.1: Peak concentric power (W) at baseline, 12, 36 and 60 h post-match

	Peak concentric power (W)			
Subject	Baseline	12 h	36 h	60 h
1	6010	5540	5601	5682
2	5757	5799	5451	5875
3	5211	4834	4562	4864
4	6775	6536	6765	6471
5	6421	5867	6168	6217
6	6112	5765	5678	6021
7	6511	6149	6503	6519
8	5488	4866	5175	5497
9	6582	5890	5868	5967
10	6089	4808	5483	5935
11	6847	6105	6413	6572
12	5490	5595	5435	5751
13	6751	6598	6496	6574
14	5360	5169	5051	5352
Mean	6100.4	5679.9	5760.5	5949.7
S.D	565.1	588.6	638.5	504.5

Table 6.2: % change in peak concentric power at baseline, 12, 36 and 60 h post-match

	Peak Concentric Power % change			
Subject	Baseline	12 h	36 h	60 h
1	100	92.2	93.2	94.5
2	100	100.7	94.7	102.0
3	100	92.8	87.5	93.3
4	100	96.5	99.8	95.5
5	100	91.4	96.1	96.8
6	100	94.3	92.9	98.5
7	100	94.4	99.9	100.1
8	100	88.7	94.3	100.2
9	100	89.5	89.2	90.7
10	100	79.0	90.0	97.5
11	100	89.2	93.7	96.0
12	100	101.9	99.0	104.8
13	100	97.7	96.2	97.4
14	100	96.4	94.2	99.8
Mean	100	93.2	94.3	97.7
S.D	0	5.8	3.8	3.6

Table 6.3: Peak jump height (cm) at baseline, 12, 36 and 60 h post-match

	Jump Height (cm)			
Subject	Baseline	12 h	36 h	60 h
1	0.35	0.30	0.29	0.31
2	0.33	0.32	0.32	0.34
3	0.28	0.28	0.26	0.26
4	0.34	0.35	0.34	0.33
5	0.32	0.27	0.31	0.35
6	0.39	0.36	0.36	0.39
7	0.46	0.44	0.48	0.46
8	0.37	0.31	0.36	0.34
9	0.45	0.43	0.40	0.45
10	0.42	0.24	0.35	0.41
11	0.48	0.44	0.44	0.47
12	0.45	0.45	0.46	0.47
13	0.41	0.40	0.43	0.42
14	0.41	0.39	0.39	0.41
Mean	0.39	0.35	0.37	0.39
S.D	0.06	0.07	0.07	0.06

Table 6.4: % change in peak jump height at baseline, 12, 36 and 60 h post-match

	% change jump height			
Subject	Baseline	12 h	36 h	60 h
1	100	85.8	83.8	87.5
2	100	97.3	97.6	104.2
3	100	96.8	90.8	92.6
4	100	101.8	100.0	98.5
5	100	86.0	97.1	109.8
6	100	92.6	92.3	99.0
7	100	96.3	105.9	99.6
8	100	84.4	97.3	92.3
9	100	94.2	88.0	98.9
10	100	55.9	81.8	98.1
11	100	92.4	92.0	97.9
12	100	100.7	102.7	106.3
13	100	96.8	103.6	101.0
14	100	95.1	94.2	99.5
Mean	100	91.2	94.8	98.9
S.D	0	11.4	7.2	5.7

Appendix D: Salivary Testosterone and Cortisol Raw Data

Table 7.0: Salivary testosterone concentrations ($pg \cdot mL^{-1}$) at baseline, 12, 36 and 60 h post-match

	Testosterone ($pg \cdot mL^{-1}$)			
Subject	Baseline	12 h	36 h	60 h
1	61.3	41.5	90.9	80.1
2	120.2	71.4	51.9	90.5
3	256.6	153.2	187.7	232.3
4	190.1	145.0	170.7	162.1
5	181.6	156.4	167.5	150.7
6	270.3	175.9	185.5	123.6
7	349.5	183.0	285.7	195.2
8	177.2	158.5	36.9	181.7
9	149.3	252.4	210.8	133.0
10	250.3	184.0	219.2	273.7
11	357.5	196.2	254.5	202.6
12	142.7	67.9	171.2	175.3
13	263.9	172.8	219.3	362.5
14	219.4	152.1	164.5	215.1
Mean	213.6	150.7	172.6	184.2
S.D	84.2	56.1	71.0	74.0

Table 7.1: % change in salivary testosterone concentrations at baseline, 12, 36 and 60 h post-match

	Testosterone % change			
Subject	Baseline	12 h	36 h	60 h
1	100	67.7	148.2	130.6
2	100	59.4	43.2	75.3
3	100	59.7	73.1	90.5
4	100	76.3	89.8	85.3
5	100	86.1	92.2	83.0
6	100	65.1	68.6	45.7
7	100	52.4	81.7	55.8
8	100	89.5	20.8	102.5
9	100	169.0	141.1	89.0
10	100	73.5	87.6	109.3
11	100	54.9	71.2	56.7
12	100	47.6	120.0	122.8
13	100	65.5	83.1	137.4
14	100	69.3	75.0	98.0
Mean	100	74.0	85.4	91.6
S.D	100	29.8	34.0	27.8

Table 7.2: Salivary cortisol concentrations ($\mu g \cdot dL^{-1}$) at baseline, 12, 36 and 60 h post-match

Cortisol ($\mu g \cdot dL^{-1}$)				
Subject	Baseline	12 h	36 h	60 h
1	0.29	0.39	0.42	0.30
2	0.41	0.63	0.88	1.17
3	0.31	0.49	0.80	0.26
4	0.25	0.60	0.63	0.44
5	0.35	0.75	0.46	0.54
6	0.58	0.68	0.78	0.60
7	0.41	1.01	0.49	0.59
8	0.50	0.62	0.28	0.51
9	0.29	0.53	0.67	0.43
10	0.39	0.39	0.52	0.46
11	0.39	0.54	0.81	0.55
12	0.41	0.51	0.37	0.43
13	0.48	0.44	0.42	0.50
14	0.47	0.75	0.79	0.54
Mean	0.39	0.59	0.59	0.52
S.D	0.09	0.17	0.20	0.21

Table 7.3: % change in salivary cortisol concentrations at baseline, 12, 36 and 60 h post-match

	Cortisol % change			
Subject	Baseline	12 h	36 h	60 h
1	100	134.2	145.0	103.6
2	100	155.2	216.3	288.1
3	100	159.8	260.5	85.2
4	100	237.2	247.2	173.4
5	100	215.6	132.6	155.2
6	100	118.2	134.8	103.8
7	100	246.8	121.4	145.0
8	100	122.6	55.8	100.5
9	100	182.8	231.6	148.9
10	100	99.2	132.8	116.9
11	100	137.0	205.4	139.6
12	100	125.0	90.8	105.2
13	100	92.1	87.1	104.5
14	100	158.9	167.2	114.6
Mean	100	156.0	159.2	134.6
S.D	100	48.7	63.8	51.0

Table 7.4: Salivary T/C ratio at baseline, 12, 36 and 60 h post-match

	T/C			
Subject	Baseline	12 h	36 h	60 h
1	211.2	106.5	215.8	266.3
2	295.8	113.2	59.1	77.3
3	833.0	311.2	233.9	885.5
4	750.8	241.3	272.6	369.3
5	521.9	208.5	362.9	279.1
6	469.6	258.5	239.1	206.9
7	858.0	182.1	577.9	330.5
8	351.5	256.6	131.3	358.5
9	513.3	474.7	312.8	307.0
10	641.0	474.9	422.6	599.5
11	905.3	362.7	313.8	367.5
12	349.8	133.2	462.2	408.4
13	553.8	393.8	528.5	728.1
14	462.3	201.7	207.2	395.5
Mean	551.2	265.6	310.0	398.5
S.D	219.3	123.0	148.0	210.2

Table 7.5: % change in T/C ratio at baseline, 12, 36 and 60 h post-match

	T/C % change			
Subject	Baseline	12 h	36 h	60 h
1	100	50.4	102.2	126.1
2	100	38.3	20.0	26.1
3	100	37.4	28.1	106.3
4	100	32.1	36.3	49.2
5	100	39.9	69.5	53.5
6	100	55.1	50.9	44.1
7	100	21.2	67.4	38.5
8	100	73.0	37.4	102.0
9	100	92.5	60.9	59.8
10	100	74.1	65.9	93.5
11	100	40.1	34.7	40.6
12	100	38.1	132.1	116.7
13	100	71.1	95.4	131.5
14	100	43.6	44.8	85.5
Mean	100	50.5	56.2	72.3
S.D	100	20.0	31.7	36.0

Appendix E: Mood Disturbance Raw Data

Table 8.0: Mood disturbance at baseline, 12, 36 and 60 h post-match

	Mood Disturbance			
Subject	Baseline	12 h	36 h	60 h
1	7	11	8	13
2	4	6	6	3
3	10	19	10	11
4	4	2	11	6
5	4	6	4	2
6	2	3	1	1
7	5	6	5	2
8	4	8	5	4
9	7	8	10	5
10	2	4	4	5
11	4	9	5	5
12	6	10	7	5
Mean	5	8	6	5
SD	2	4	3	4

Table 8.1: % change in mood disturbance at baseline, 12, 36 and 60 h post-match

	% change mood disturbance			
Subject	Baseline	12 h	36 h	60 h
1	100	157	114	186
2	100	150	150	75
3	100	190	100	110
4	100	50	275	150
5	100	150	100	50
6	100	150	50	50
7	100	120	100	40
8	100	200	125	100
9	100	114	143	71
10	100	200	200	250
11	100	225	125	125
12	100	167	117	83
Mean	100	156	133	108
SD	0	49	60	65

Appendix F: Brief Assessment of Mood Questionnaire

Brief Assessment of Mood (BAM)

Circle the number that best describes how you are feeling right now

	Not at all	A little	Moderately	Quite a bit	Extremely
How **anxious** do you feel?					
How **depressed** do you feel?					
How **confused** do you feel?					
How **angry** do you feel?					
How **energetic** do you feel?					
How **fatigued** do you feel?					