A pilot study investigating the effects of acute sleep restriction and its relationship to markers of muscle recovery from a single session of exercise induced muscle damage in healthy untrained males.

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Abstract

Sleep loss can affect many aspects of human performance including time to exhaustion, muscular strength, focus and mood. These effects can further lead to health risks and medical conditions. With increasingly busy lives humans are often faced with a failure to attain adequate sleep following performance of resistance exercise. This thesis aims to investigate the relationship between disrupted sleep and markers of muscle recovery from a single session of damaging resistance exercise.

16 healthy males (mean age 23 ± 17 years, height 180.6 ± 15.4 cm, mass 87.4 ± 30.7 kg) all performed baseline testing of common recovery markers (pain tolerance, limb volume (LV), serum creatine kinase and maximal voluntary contraction (MVC)), before undergoing a protocol of 5 sets of 10 reps eccentrically induced muscle damage to the Bicep Brachii. Sleep deprived group (SD, n=8) underwent two nights of sleep which was restricted to four hours total time in bed Full sleep group (FS, n=8) adhered to normal sleeping conditions. Throughout the week all baseline tests were retested each day.

Findings from the study confirmed that sleep restriction was achieved ($p=0.001$ $d=0.92$) and muscle damage was shown across all tests except creatine kinase (MVC $p\leq0.001$, $d=0.57$; LV $p=0.006$, $d=0.17$; Pain at 90° $p= 0.003$, $d=0.59$; Pain at 180° $p=0.003$, $d=1.33$; Pain at Full Contraction $p=0.001$, $d=1.59$; General Arm Pain $p=0.005$ $d=0.69$), there were no differences in the recovery pattern for any of the markers observed between the SD group and FS group.

In conclusion, two nights of sleep restriction to four hours does not appear to impair markers of recovery following damaging resistance exercise in the Bicep Brachii, however further investigation is required in order to fully conclude the effectiveness of sleep as a recovery aid.

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Introduction:

Both diet and training can have significant impacts on performance across a range of sports (Spriet et al 2019; Meseguer Zafra et al 2018). Whilst these areas are important in a sporting context, performance can also be impacted by the effectiveness of recovery and depth of fatigue. Performance loss following exercise without adequate recovery can lead to many negative impacts including feeling tired, lethargic or even sore from exercise which in turn can impact subsequent sports participation (Balsom et al 1992). Current recommendations for recovery from muscle damage caused by fatigue include ensuring adequate nutrition, through increasing certain macronutrients (Millard-Stafford et al 2008), adequate hydration through consumption of high carbohydrate electrolyte drinks (Reilly and Ekblom 2005), as well as allowing the body adequate rest in order to recover through the normal healing process. While these recommendations exist, the recovery process from muscle damage has also been claimed to be accelerated by using certain therapies and therapeutic techniques. Practices such as cold water therapy, through reduction in secondary muscle damage (Ingram et al 2009), stretching and massage through increased blood flow resulting in decreased oedema, enhanced blood lactate removal and enhanced tissue healing (Best et al 2008; Pearcey et al 2015), as well as compression garments by impacting the inflammatory response to exercise (Jakeman, Byrne and Eston, 2010; Trenell et al. 2006) all claim to speed up the recovery process. Another area which may also adversely affect recovery, however, is that of sleep disruption.

Sleep disruption is fast becoming a key health issue which faces many individuals around the world. Indeed, sleep disorders alone cost the Australian economy over 5 billion per year, $270 million for health care costs for the conditions themselves, $540 million for care of associated medical conditions attributable to sleep disorders and $4.3 billion attributable to associated productivity losses and non-medical costs resulting from sleep loss-related accidents (Hillman and Lack (2013)). This trend is shown similarly throughout the world. Across the USA, UK, Germany, Canada and Japan, the range of impact on GDP was between 0.85% in Canada and 1.86% in Japan. The UK was found to spend 1.36% of GDP on

sleep productivity losses which equates to around $36.7 billion (Hafner et al. (2017)).

From a sport participation standpoint sleep disruption can create many issues such as reduced neurocognitive factors (Aptowicz et al. 1997; Harrison and Horne 2000) as well as physical factors such as skill execution, strength, power, heart rate and metabolism (Fullagar et al. 2015; Mougin et al. 1991; Reilly and Piercy 1994). The effects of sleep disruption on recovery are still however unknown and as such further research is required.

Currently a large majority of people in Scotland participate in some form of basic physical activity, with 81% of adults performing at least 30 minutes of any physical activity within a 4-week period (Housing and Social Justice Directorate Scottish Government, 2018) while this number is well below the recommended guidelines of 150 mins per week it has increased since 2010 when it was reported as 72% suggesting a trend to more physical activity participation. As well as this increase in physical activity participation, individuals are now also willing to invest in improving sports performance with total sports related spending in Scotland up 41% between 1998 and 2016 (Sportscotland, 2016). However, large numbers of people are achieving less than adequate sleep time as demonstrated in a survey by Steptoe, Peacey and Wardle (2008) where almost 21% of participants reported sleeping less than six hours per night. In contrast The National Sleep Foundation currently recommends that healthy individuals sleep for a total duration of between seven and nine hours for young adults and seven and eight hours for adults (Hirshkowitz et al. 2015). Loss of sleep can place an individual at risk of many issues including both mental and physical health implications, which can lead to affected performance (Oliver et al. 2009; Skein et al. 2011).

The purpose of this thesis is to explore the effects of sleep loss on recovery from eccentrically induced muscle damage. Specifically, the aims of the current project are to assess the effects of partial sleep restriction on:

i) Muscle damage recovery based on pre damage baselines
ii) Muscle damage recovery in participants with and without sleep restriction

1.0 Literature Review

1.1 Skeletal Muscular Structure:

Under normal circumstances a skeletal muscle fibre is composed of many myofibrils, which are formed by sarcomeres arranged in series. This sarcomere comprises several proteins organized in a three-dimensional lattice (Fig. 1). These sarcomeres are comprised of thinner, actin, filaments and thicker, myosin, filaments. Between these filaments exists a cross bridge interaction though which muscle contraction can occur (Rassier, 2017). In this interaction myosin is bound to actin and with the presence of Adenosine triphosphate (ATP) “walks” along the actin filament, sliding closer to the Z line of the sarcomere and thus generating muscle contraction.

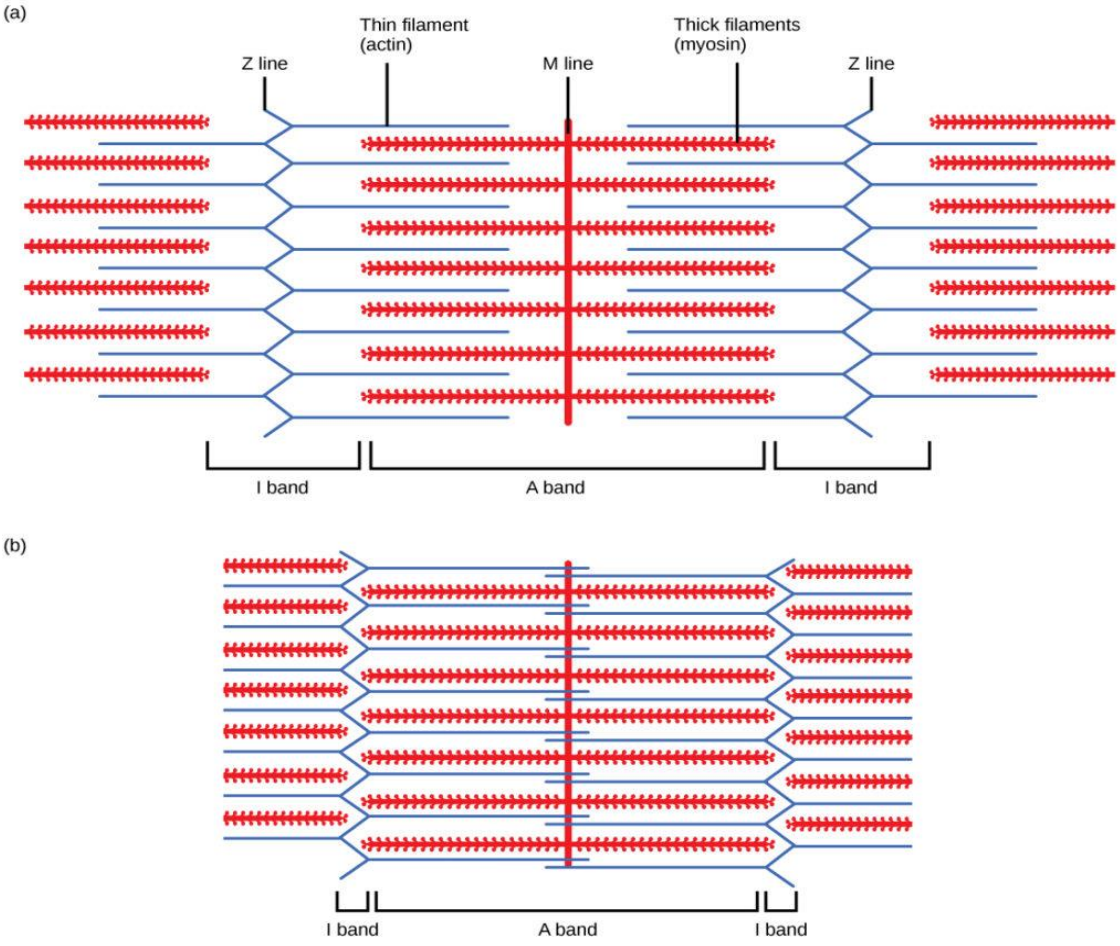

Figure 1: (a) A cross section of a sarcomere, (b) A cross section of a fully contracted sarcomere. adapted from (OpenStax College 2016).

1.2 Muscle Damage:

Various forms of exercise, especially eccentric exercise can lead to temporary muscle damage. This muscle damage is very common and is usually categorised by a number of factors such as: decrease in muscle strength, a decrease in range of motion, presence of Delayed Onset Muscle Soreness (DOMS), increase in swelling, as well as an increase in presence of muscle specific proteins including creatine kinase and myoglobin. This damage is summarised by Howatson and Van Someren (2008) into primary damage, occurring during the exercise bouts, as well as secondary damage which occurs as a result of the inflammatory response.

1.2.1 Primary Muscle Damage:

Damage which occurs as a direct result of eccentric exercise can further be subdivided into two further categories: metabolic damage, resulting from effects of biochemical processes that occur within humans, and mechanical damage, which results from damage to the physical components of muscle itself. Howatson and Van Someren (2008) propose that metabolic damage results from ischaemia (inadequate blood supply) or hypoxia (inadequate oxygen supply) during exercise which is of a prolonged nature. Ischaemia is thought to cause changes in ion concentration, metabolic waste accumulation and adenosine triphosphate (ATP) deficiency. At the onset of exercise heart rate and oxygen uptake will rise until they have reached a higher, steadier state to meet the demands of ATP production (Karvonen & Vuorimaa, 1988). After some time in prolonged exercise, the increased metabolic requirements of a muscle can be balanced by this increased function of the cardiovascular and respiratory systems. More oxygen and fuel will be supplied, and carbon dioxide and other waste products will be removed, this allows aerobic production of ATP (Ament and Verkerke, 2009). Under aerobic conditions, pyruvate which is generated through the breakdown of glucose as one of the end-products can be further processed, generating more ATP, CO_2 and water. However, under anaerobic conditions pyruvate cannot be broken down and as such is converted to lactate alongside hydrogen ions. As exercise continues the process to remove pyruvate becomes increasingly difficult until a certain workload where there is a marked

increase in lactate concentration as well as H^+ ions in the blood and extracellular fluids. While lactate does not directly result in acidosis and in fact slows the process due to the use of H^+ ions to generate pyruvate an increase in lactate is seen at fatigue and therefore can be a good indirect indicator of metabolic acidosis conditions which can cause fatigue (Roberg et al. 2004).

When muscles are contracting there will be an increase, regardless of method of ATP production, in Adenosine Diphosphate (ADP) and inorganic phosphate (Pi) which are by-products of ATP usage (Chance et al, 1981). This, as well as the previously discussed production of lactate, can lead to an increase in H^+ ions and therefore a decrease in intracellular pH (Lindinger, 1995). The presence of ADP, Pi and H^+ can all lead directly to reductions in efficiency of cross bridge interaction of the actin and myosin filaments therefore resulting in less efficient muscle usage. This decreased efficiency requires the muscle to work harder in order to achieve the same results, further compounding the issue in a cascade effect leading to faster fatigue (Ament and Verkerke, 2009).

When looking at the individual muscle cells, the ability to generate a high frequency of action potentials, systems by which nerve impulses are passed along, is extremely important in exercise. The ability to sustain an action potential at high frequency is primarily dependent on the ability to both pass potassium ions back into the cell as well as to expel excess sodium ions, which enter during the action potential. (Fitts, 1994). These electrochemical gradients are powered by an electrogenic pump which, in turn, is powered by ATP. As more ATP is used the body is required to reproduce more, however the efficiency with which this is done decreases resulting in less efficient fibre recruitment (Thomas, 1972).

As well as the metabolic issues which would be associated with primary muscle damage it has also been shown that damage can be caused by surrounding damage to the muscle cell itself. This is thought to be as a result of a non-uniform lengthening of sarcomeres, which can result in some of the actin and myosin filaments being stretched and no longer able to overlap within the sarcomere itself (Morgan and Proske, 2004) (figure 2). As a result of this passive structures may assume more tension and undergo “popping” resulting in Z-band streaming (Friden et al. 1984). This excessive tension from repeated eccentric contractions

can cause failure of the structure and therefore a reduction in the muscles ability to generate force.

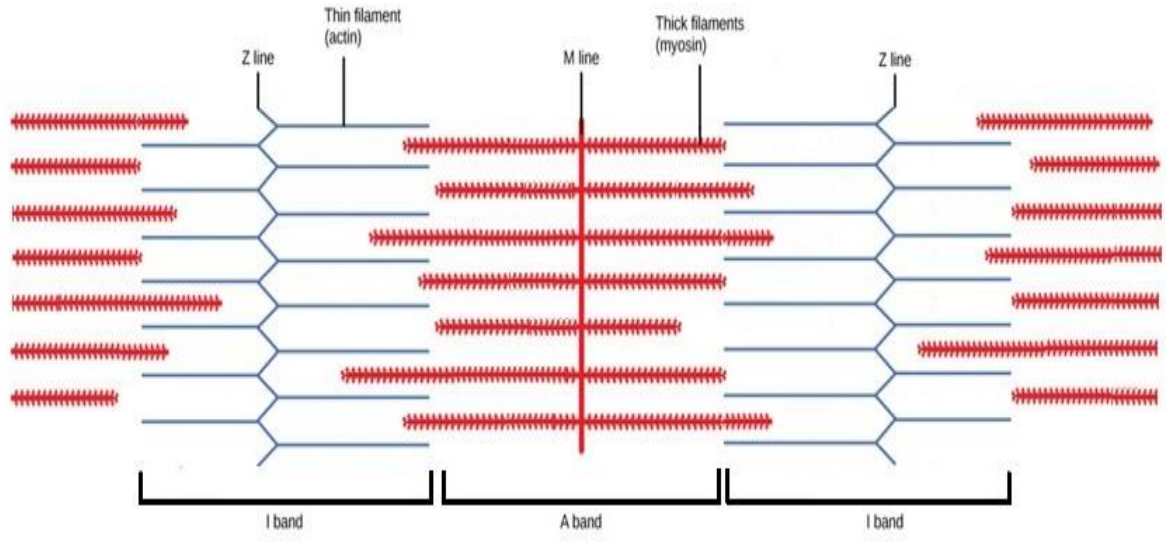

Figure 2: A Cross section of a sarcomere damaged through EIMD showing stretched muscle filaments.

The reduction in force generated by primary muscle damage can therefore lead to assessment of muscle damage through assessment of pre and post exercise Maximum Voluntary Contraction.

1.2.2 Secondary Muscle Damage:

Processes which follow the primary phase of damage appear to be as a result of the disruption of intracellular calcium ions. It has been suggested by Baird et al. (2012) that with the depletion of ATP and the leaking of extracellular calcium ions into the intracellular space that intracellular proteolytic enzyme activity will begin to promote muscle protein degradation and cell permeability which can allow some cell contents to leak into circulation. Eccentric exercise has been shown to lead to a loss of sarcoplasmic reticulum membrane integrity (Howatson and Van Someren, 2008). This loss of integrity can lead to an influx of Ca^{2+} into the cytosol causing a cascade of events that further damage the cell causing alterations to the cytoskeleton, sarcoplasmic reticulum, mitochondria and myofilaments (Howatson and Van Someren, 2008). This cell permeability can allow leakage of various intramuscular proteins such as creatine kinase and myoglobin and

therefore could be used to show levels of muscle damage directly as a result from exercise.

Following the rise in intracellular Ca^{2+} it is suggested that proteolysis is triggered thus facilitation breakdown of damaged fibres (Proske and Allen, 2005). The inflammatory response can result in invasion of the area by macrophages and monocytes. This inflammation is accompanied by oedema and as a result can be presumed to be the cause of muscle swelling.

Secondary muscle damage can also lead to measurable assessment of induced muscle damage both through the inflammatory response generating measurable pain as well as swelling and increased cell permeability allowing the measurement of intramuscular proteins found in the circulatory system.

1.3 Fatigue:

Fatigue when referencing exercise can be broken generally into two categories: central fatigue and peripheral fatigue (Davis, 1995). Central fatigue is caused by issues relating to the central nervous system while peripheral fatigue is caused by limiting factors which are not related to the central nervous system.

1.3.1 Central Fatigue:

When performing intense exercise, the body is reliant on the complex recruitment of motor units at high firing frequencies. As fatigue begins to take effect from a neurological standpoint a failure to coordinate the motor drive would be reflected in lack of skill and efficiency.

While performing prolonged exercise the body will synthesise and metabolise central monoamines such as serotonin, dopamine and noradrenaline (Taylor et al. 2016). Bahr et al (1987) suggested that during prolonged exercise increased brain serotonergic activity may cause an individual to become more lethargic and result in a loss of drive and therefore a reduction in motor unit recruitment. This loss of recruitment would then affect the physical and mental efficiency of the exercising individual. Davis and Bailey (1997) further hypothesised that fatigue, rather than as a result of purely serotonin, resulted from an increase in the brain content ratio of serotonin to dopamine which would result in feelings of tiredness and lethargy and thus accelerating the onset of fatigue. As a result, this process

could be described as central fatigue. This has since been expanded upon in some reports including that of Taylor et al. (2016) to include noradrenaline as a further mediator of central fatigue to create a more complex mix of central monoamines resulting in fatigue.

1.3.2 Peripheral fatigue:

A further limiting factor of exercise which may be a mediator of fatigue can be the availability of muscle glucose. Blood glucose is maintained at constant levels in the body through insulin pathways, however glucose uptake in exercising muscles is mediated by glucose transporters (Hayashi et al. 1997). Upon contraction of the muscle, glucose transporter activation increases which results in an increase in glucose uptake. While undergoing endurance exercise intracellular glycogen stores will be decreased until eventually glucose availability is smaller than the consumption. At this point exercise will become extremely difficult to continue (Ament and Verkerke, 2009). It is suggested this comes as a direct reaction from the brain to a decreased blood glucose level due to the need of brain tissue for a minimum glucose supply (Ament and Verkerke, 2009). While this is less relevant for individual bouts of resistance training, repeated bouts would lead to some level of fatigue in resistance training also (Lambert and Flynn, 2002).

A final impact of exercise is the increased temperature of the muscle cell. This is as a result of the muscle contraction and can lead to an increase in core body temperature. Gonzalez-Alonso et al. (1999) has suggested that above 40°C the central drive of subjects is decreased, and they are unable to maintain workload.

1.4 Recovery:

Recovery following exercise can be seen as a return to the ability to meet or exceed performance in a particular activity (Bishop et al. 2008). Several factors in an individual can lead to a reduction in performance such as dehydration (Barr, 1999), waste product production (Ament and Verkerke, 2009), as well as damage to muscle cells (Morgan and Proske, 2004). These factors while all products of exercise can lead to fatigue in exercise and restrict the return of an individual to previous performance markers thus creating fatigue.

1.4.1 Recovery following exercise:

While the processes involved in recovery are generally unknown, some patterns do exist allowing theories to be drawn.

Following the end of static or dynamic exercise, muscle blood flow will immediately increase for the initial seconds before decreasing to resting levels. This is dependent on the type and duration of exercise. During short periods of contraction maximal blood flow has been seen to occur within 4-6 cardiac cycles. However, following longer periods of contraction (30 seconds) maximal blood flow occurred during the first heartbeat (Bangsbo and Hellsten, 1998). Walloe and Wesche (1988) found that blood flow was greater and lasted longer following an exercise of increased duration and higher tension following two-minute intermittent contraction at intensities of 10% and 30% of maximal voluntary contraction force (MVC). Following this initial increase however muscular blood flow has then been seen to decrease at a moderate rate and reach resting levels within 20-30 minutes following exercise (Bangsbo and Hellsten, 1998).

Following exercise, pulmonary and muscular oxygen uptake will increase rapidly, however similar to muscular blood flow, this can remain elevated compared to resting levels for some time in the immediate cessation of exercise (Laforgia et al. 2006). This has been termed excess post-exercise oxygen uptake (EPOC). Depending on the type of exercise there have been findings appearing to show differences on level of EPOC with interval exercise resulting in higher levels than continuous exercise (Jung et al. 2019). Bangsbo and Hellsten (1998) have also suggested that further evidence is required as some effect may be caused by an increase in temperature, catecholamine effects or extra oxygen use in other tissue types.

Following exercise, the central nervous system must undergo recovery. This is as a result of the observed role of central fatigue. Minett & Duffield (2014) have suggested that, following exercise, declines in motor unit activity can be sustained for between 1 and 24 hours. Rampinini et al. (2011) further confirmed this by showing that, following a professional soccer match, decreases in muscle activation as measured through EMG were of a similar level to the decreases

seen in MVC thus suggesting a relation between MVC recovery and central driving factors.

Recovery from the damage caused by exercise can, unlike other mediators of fatigue, take several days to be fully repaired which suggests a repair of contractile elements, the sarcoplasmic reticulum and the connective tissues must occur (Clarkson and Trembley 1988). As with other mechanical damage repair this is thought to be associated with the inflammatory response of the human body (MacIntyre et al. 2001). Studies have also shown however that following this repair, after repeated bouts of the same exercise the negative effects, primarily pain, of damaging exercise are diminished. This therefore suggests an adaptation effect following muscle damage (Byrnes et al. 1985). Through this, exercise can result in a rapid remodelling of skeletal muscle (McGlory et al. 2017). This can be maintained by changes to both gene expression and levels of protein synthesis. Following resistance exercise rates of Muscle Protein Synthesis (MPS) have been shown to rise above levels of Muscle Protein Breakdown (MPB). The adaptation is partly thought to be as a result of increased amino acid transportation (Biolo et al. 1995). Further to this resistance exercise has also been shown to alter gene expression resulting in further muscular adaption to resistance training. Nader et al. (2014) reported that regulatory mechanisms that may dictate when genes are expressed were influenced by a training programme. It is suggested then that this may establish a hierarchy of genomic responses involved in initiating or maintaining training-induced adaptations.

With all of these changes it is thought that recovery is intended to undo the damage incurred during training and as such create adaptations in the body to further withstand fatigue causes in the future (Bishop et al. 2008). However, some elements of recovery are still to be researched, with different factors thought to impact recovery and either speed up or slow down recovery in both athletes and untrained individuals. This can happen through manipulation of certain processes in the body such as diet, tissue manipulation or anti-inflammatory properties or functions such as sleep.

1.5 Optimal Recovery Strategies:

In order to return to the levels of performance which existed prior to exercise fatigue which took place in a fast and effective manner, many techniques have been explored. The primary purpose of these techniques are to increase blood flow, reduce tension in the muscle and to reduce neurological excitability (Weerapong, Hume and Colt 2012). Many can also aid recovery by combatting issues such as inflammation, high heart rate, disposal of waste products, rehydration and nutrient recovery.

1.5.1 Active and passive recovery:

Active recovery is the promotion of continued low-level exercise in order to promote the recovery of the body (Spencer et al. 2006). Martin et al. (1998) found that active recovery produced significant decreases in blood lactate when compared with both massage and rested (passive recovery) groups. However, while this was backed up by Bond et al. (1991) they were unable to find any differences between groups in measures of isokinetic strength, work output and muscle fatigue. In a meta-analysis conducted by Dupuy et al. (2018) it was suggested that active recovery had no effect on perceived fatigue however had a significant effect on Delayed Onset Muscle Soreness (DOMS). This therefore indicates that while active recovery can aid in the removal of lactate and reduction in the pain associated with muscle damage this does not reduced the overall damage to the muscle and further recovery strategies should be carried out no fully recovery from damaging exercise.

1.5.2 Diet and rehydration:

Dehydration can occur both before and during exercise though the latter can cause reduction in aerobic endurance, increased body temperature, heart rate and perceived exertion (Barr, 1999). These negative impacts of dehydration have been seen to be proportional to the degree of dehydration and as such ensuring adequate rehydration is of critical importance following exercise.

When restoring lost liquid it is necessary to replace more than 100% of the liquid lost through exercise (Bishop et al. 2008). This is due to the bodies inability to completely retain all liquid taken in. Most rehydration researchers will recommend

that as well as replacement of total liquid lost the replacement of electrolytes should be undertaken following exercise (Bishop et al. 2008). This can be done through consumption of rehydration beverages with increased sodium and potassium. Further to this, the inclusion of carbohydrates should also be undertaken in rehydration beverages in order to aid restoration of muscle glycogen stores (Bishop et al 2008).

Diet can also aid recovery however this can vary extremely based on the type of exercise performed and as such various recommendations exist for individual sports and activities. Beck et al. (2015) provide some general guidelines. In order to enhance production of muscle glycogen which will be used during exercise it is necessary to consume adequate carbohydrates. Millard-Stafford et al. (2008) suggest a minimum of 1.2 grams per kilogram of body weight per hour for a four-hour period.

Beck et al. (2015) also suggest that following either resistance or endurance exercise there exists an increase in protein turnover. However, until feeding the protein balance can remain negative. Therefore, it is suggested that protein consumption following exercise can enhance protein synthesis. Phillips and Van Loon (2011) suggest that protein intake in the range of 1.3-1.8 grams per kilogram of bodyweight per day will maximise muscle protein synthesis. Finally Beck et al. (2015) recommend that since exercise induced protein synthesis is elevated for 24-48 hours after resistance and 24-28 hours following high intensity aerobic exercise, as well as the “Muscle full” effect of protein synthesis returning to baseline despite circulating amino acids around 90 minutes after consumption, that multiple feedings over the day post exercise may maximise muscle growth.

It is important to note the consumption of other nutrients may be necessary on an individual sport basis with Nachtigall et al. (1996) also suggesting an increase in iron intake in distance runners to counter deficiencies which may be associated with this discipline.

Further, there are some fields of research undertaking investigations into the effects of antioxidants, especially vitamins C and E, on muscle recovery. This field can be seen to stem from the hypothesis that muscle damage is the result of reactive oxygen species instead of the conventional thoughts of mechanical

damage. Positive results have been seen by Jakemanl and Maxwell (1993) as well as Meydani et al (1993). Further promising evidence on the effect of antioxidant supplementation was seen in a review article by Merry and Ristow (2016) where it was concluded that when optimal muscle performance is required following short recovery intervals antioxidant consumption was able to aid in immediate recovery. In particular in the study by Cobley et al. (2011) participants were able to maintain a higher level of performance in repeated bouts of intermittent damaging exercise. However, Merry and Ristow (2016) did further conclude that antioxidant use may suppress the positive adaptations of resistance training and as such should only be taken to aid recovery during short sub-optimal recovery times.

1.5.3 Massage:

Massage in general can be used to promote health and well-being. Cafarelli and Flint (1992), describe massage as “a mechanical manipulation of body tissues with rhythmical pressure and stroking for the purpose of promoting health and well-being.” This has anecdotally been seen in a sporting context to promote recovery, however massage is still controversial among researchers with several theories existing as to why it may be an effective recovery strategy. Bio-mechanically the theory behind massage is that it elongates shortened or adhered connective tissue in the muscle (Weerapong, Hume and Colt, 2012). Based on research carried out by Magnusson (1998) in increased muscle compliance elongated connective tissue can result in a less stiff muscle tendon unit and as a result can aid basic recovery.

Massage has also been shown to improve blood flow in the massaged area (Mori et al. 2004). This may aid recovery in numerous ways including increased oxygen delivery, thus aiding healing as well as the removal of certain metabolites (Hinds et al 2004).

However, many studies dispute the real physiological effects of massage and instead suggest the use of massage to be based on anecdotal evidence and instead that massage promotes psychological feelings of relaxation from athletes (Hemmings et al 2000), indicating more of a placebo effect from massage. This may be due to evidence of parasympathetic activity following massage which can

then impact other physiological effects such as the release of endorphins (Kaada and Torstein 1989), reduced heart rate (Corley et al 1995) and reduced systolic and diastolic blood pressure (Labyak and Metzger 1997).

1.5.4 Temperature Changes:

There has been some evidence of increased muscular recovery following temperature changes. This evidence has included work on temperature increase, decrease or contrasting therapy, in which the temperature is cycled between hot and cold.

Predominantly this research has taken place within water immersion systems. While research into hot water immersion has thus far provided results which suggest a lack of benefit in recovery (Versey et al. 2013) there has been some suggestion that hot water immersion can benefit a recovery of isometric force (Vaile et al. 2008) (Viitasalo et al. 1995).

Promising early indicators for cold water immersion was seen through meta-analysis by Machado et al. (2016), in which it was observed to be better than passive recovery at managing muscle soreness. This is backed up in numerous studies with benefits across a range of sports (Heyman et al. 2009) (Montgomery et al. 2008), immersion temperatures (Yeargin et al. 2006) or repeated or single immersions (Eston and Peters, 2009). It has been suggested that cold water immersion is able to benefit through the recognised reduction in inflammatory response of cryotherapy (Meeusen and Lievens, 1986) which can alleviate muscle spasm and pain. Further it has been suggested that following exercise cold therapy promotes recovery by accelerating decreases in post exercise heat storage to homeostasis (Peiffer et al. 2009).

Dupuy et al. (2018) concluded following meta-analysis that contrast therapy, the application of one temperature change followed by the opposite for example hot followed by cold, was successful at reducing DOMS, as well as perceived pain and production of creatine kinase. This may be due to contrast therapy inducing successive peripheral vasoconstriction and vasodilation which has been proposed to increase lactate clearance (Cochrane, 2004)(Bieuzen et al. 2013). This therefore suggests that contrast therapy may be the most effective temperature change technique at reducing muscle damage following exercise.

1.5.5 Anti-inflammatories:

Current theories around anti-inflammatory based recovery suggest that due to their ability to minimize oedema in the muscle and act as a pain killer they allow a return to training to happen quicker. Thus, resulting in faster recovery back to full potential as is required by Bishop et al. (2008)'s definition. Studies into this field have however had mixed results. Semark et al (1999) showed that there was not a significant difference following the administering of flurbiprofen, a non-steroidal anti-inflammatory drug (NSAID) on muscle soreness and damage. This is contrary to Lanier (2003) who concluded through review that NSAID use for brief periods of time could be beneficial for short term recovery of muscle function, it should be noted that Lanier suggests a prophylactic use of NSAIDs may be more effective compared with a therapeutic use.

1.6 Trained versus Untrained:

The ability of a person to recover can depend on their previous level of training. However, this can depend on the type of exercise carried out as well as the individuals level of training in this type of exercise.

1.6.1 Endurance trained:

Endurance training has been shown to have an impact on the length of time needed to adequately recover. Tomlin and Wenger (2001) found in a review of existing literature that when individuals with high levels of aerobic fitness, based on VO_{2max}, were able to better recover from high intensity intermittent exercise. This may be due to an increased lactate response, an ability to regenerate phosphocreatine faster as well as an increased aerobic response to exercise. This increased aerobic response suggests that an individual can adequately supply oxygen for a longer duration of exercise. With this increased supply of oxygen there is less accumulation of waste products and as such fatigue will cause less damage requiring less recovery (Stangier et al. 2016)

An increased aerobic response has been shown by Hamilton et al. (1991) in which individuals who were endurance trained consumed a higher level of oxygen during repeated bouts of six second sprints. This increased oxygen consumption also coincided with a significantly smaller decrement in power over

the ten sprints. As such it was concluded that increased oxygen consumption allowed for less reliance on glycolysis and therefore less lactate production. This therefore would allow for a faster ability to recover from high intensity but low duration exercise.

With a more efficient aerobic system it was also noted that a more efficient lactate removal system exists (MacRae et al 1992) (Hoshtrup and Bangsbo 2017). With an increased lactate removal system this can create favourable conditions to allow for increased levels of muscular recovery.

McCully et al. (1989) and Tomlin and Wenger (2001) have supported the theory of increased phosphocreatine regeneration in endurance trained individuals. This would allow for individuals to be more prepared to return to baseline levels and as such could be seen as a recovery measure.

Although endurance training can be seen to improve recovery from exercise in the long term these adaptions will not be able to aid in the short term.

1.6.2 Resistance trained:

With regards to the effects of resistance training on recovery there is a lack of literature on how it may affect recovery from endurance events. However, Glowacki et al. (2004) suggested that the addition of resistance training may hinder the development of maximal aerobic capacity compared with endurance training alone.

When resistance training the methods of recovery differ compared to endurance training. Fatigue in resistance training is caused both by muscular fatigue as well as, more predominantly than in endurance training, peripheral and central nervous system fatigue. As a result, this requires different methods of recovery. Morán-Navarro et al. (2017) showed that resistance training to failure resulted in longer recovery even when compared to training the same total volume of work done. This would suggest impairment to the recovery system only present with muscular failure. Similarly, this was seen by Mayo et al. (2016) in which results suggested exercise type and inter-repetition rest design could blunt the decrease of cardiac vagal activity thus increasing the total time of recovering and leading to hypotension.

1.6.3 Untrained:

The ability of a person to recover from muscle damage differs greatly between trained and untrained. Newton et al. (2008) were able to show in a comparison of elbow flexor muscles following maximal contraction performed in isometric dynamometer that resistance trained individuals were able to recover to baseline strength after three days while untrained individuals remained 40% lower at the same timepoint.

This is backed up in endurance trained individuals as suggested by Darr et al. (1988) in which it was suggested that heart rate in trained individuals recovered significantly faster than in untrained individuals.

For untrained individuals the results of exercise will provide similar negative impacts, however these results may be more severe. This is a result of the various physiological adaptions that arise from exercise such as increased cardiovascular efficiency (Lavie et al. 2019) and muscular efficiency (Balnave and Thompson 1993). In a review article by Wilson et al. (2016) it was concluded that regular exercise can lead to benefits in many aspects of the cardiorespiratory system including increased blood flow, increased efficiency of the circulatory system and increased aerobic capacity. These adaptions can lead to increased ability to heal through reduction in inflammation and therefore less severe and faster recovery.

It is important to note that untrained individuals will be more responsive to the negative effects of exercise and as such may be more responsive to effects which can compound poor recovery such as sleep loss. Further, these individuals may be more likely to receive inadequate sleep quality and time due to perceived busy lifestyles and barriers which could potentially lead to lack of physical activity as well as sleep. Measurement of recovery in otherwise healthy however untrained individuals could then provide clearer data with regards to assessment of new areas which can impact recovery either positively or negatively.

1.7 Recovery measurement:

In order to adequately assess recovery, it is necessary to establish methods of measurement which can provide informative results. Many different tests exist with varying levels of effectiveness depending on situation and individual.

1.7.1 Maximal Voluntary Contraction (MVC):

Maximal voluntary contraction (MVC) measures the maximum contractile force of a specific muscle group in an individual. MVC is an extremely useful tool in assessing recovery as it is directly related to the Bishop (2008) definition of a return to baseline levels of pre-fatigue ability. MVC can also allow specific testing of individual muscle groups, thus allowing accurate measurements of fatigue. In assessment of MVC it would be assumed that following muscle damage the tested levels would decrease below baseline immediately after testing before slowly returning to the pre fatigue levels over the course of the following days. When testing MVC it is necessary to test with accurate dynamometers. Testing of MVC with accurate dynamometers and a repeatable experience can lead to good repeatability of results. Todd, Gorman & Gandevia (2004) estimated a coefficient of variation between 2 and 11% for measure of MVC. However, while MVC can provide sensitivity to small changes in muscular strength (Meldrum et al 2007) the main drawbacks include a reliance on expensive equipment and researcher knowhow as well as being time intensive (Conable and Rosner, 2011). Due to the individuality of participants involved in recovery, tests can often rely on setting baselines before damage/exercise is carried out and as such these are not practical for use in the general population. However, within the laboratory setting these provide very accurate and repeatable results and therefore allow a good assessment of muscular damage.

1.7.2 Limb assessment:

Other research methods can include assessment of the damaged limb, this can be achieved through measurement of certain factors such as limb volume. The basis for this measurement comes from the inflammation of the muscle in order to repair following damage. This inflammation can lead to a swelling of the muscle and therefore an increase in volume. All four limbs of the body (both arms and legs) increase in circumference following eccentric muscle damage (Chen et al

(2011)). While this is a well-established method of measuring muscle damage there does exist certain flaws, namely accurate repeatability, is difficult to achieve through simple tests of circumference measurement or use of a basic volumeter. This could be due to the high variation of volumes in some regions of the body. Pasley & O'Connor (2008) reported as much as a 14% change in certain regions of upper body limbs with muscle volume measurements. It is however possible to mitigate some of this high variability through simple measures such as accurate marking of the limb for rerecording as well as more complex and more sensitive methods including using expensive or high-tech equipment such as DEXA scanners or complex volumeters. While the repeatability of some measurements can be questioned, following correct protocol with these measures can mitigate some of this as well as provide very simple assessment of muscle damage which can be carried out simply without the need for technical knowledge or complex machinery.

1.7.3 Blood Analysis:

With individual muscle cell damage various substances that are normally found in the muscle such as myoglobin, creatine kinase and lactate dehydrogenase can leak into the circulation and as such could be used as markers of damage. Blood analysis can therefore be a method of assessing muscle damage.

Myoglobin is predominantly used to bind to oxygen and is located in muscle tissue. However following muscle damage myoglobin is released into the blood stream (Hyldahl and Hubal 2014). Myoglobin is not naturally occurring in the blood stream and as such baseline levels should be extremely low (between 12-100ng/ml) however this is dependent on gender, race and age. Following damaging exercise however, the muscle can release myoglobin into the blood stream through increased muscle membrane permeability (Hyldahl and Hubal 2014). This can be measured and can provide an accurate measurement of the level of muscle damage occurred, with more damage there should be higher levels of myoglobin in the blood. Myoglobin has a reasonably high coefficient of variation thus providing high variation in samples provided Moller and Sylven (1981) found this to be around 18%.

Creatine Kinase is an enzyme which is used to generate Phosphocreatine and ADP following the breakdown of ATP. This breakdown produces energy and therefore is used during exercise. Creatine Kinase has been observed to be present in blood serum in higher concentrations following muscular damage as a result of the increased muscle permeability that occurs following exercise. Indeed, it has been assessed as a relatively simple and easy method of assessing muscle damage (Koch, Pereira and Machado, 2014). Similar to myoglobin this is caused through a release of contents of the muscle cell through damage (Baird et al 2012). Koch Pereira and Machado (2014) however, concluded that previous literature such as (Margaritis et al. 1999) have found Creatine Kinase activity to be poorly related to functional measures of muscle soreness, strength and range of motion. Instead Koch, Pereira and Machado (2014) suggest that Creatine Kinase instead should be used as a qualitative marker to show muscle trauma has occurred rather than as an assessment of how much trauma. Further, discussion on the validity of Creatine Kinase measurements of muscle damage suggests that results can vary based on many factors such as genotype, body composition (Heled et al. 2007), sex (Tiidus 2011), muscle group used (Chen et al. 2011) and age (Webber et al. 1989). Warren et al. (1999) are further able to back up the conclusion of Koch Pereira and Machado by suggesting creatine kinase does not correlate with decrease in muscle function. Similar to myoglobin, creatine kinase has also been found to have a large coefficient of variation (35%) this is due in part to the large variation between groups thus repeatability of tests may become difficult (Jackson et al. 1987). However, due to the increased sensitivity of creatine kinase tests, as well as the concentration of creatine kinase remaining elevated in the blood over a longer period this test is generally used in assessment of muscle damage.

Less research has been carried out on Lactate dehydrogenase as a method of assessment, this could be due to increased difficulty and costs compared to a comparatively similar enzyme in Creatine Kinase, which could be seen to be the easier option. In the occasions it has been used however it has produced results similar to the more commonly used Creatine Kinase. With Chen and Hseih (2001) showing a similar trend to that of Creatine Kinase.

1.7.4 Pain Assessment:

A common aspect of muscle damage can be the presence of pain in the associated muscle. Therefore, it is possible to assess the level of pain in order to provide a broad scale of the extent of the muscle damage. Commonly this can be achieved through use of Visual Analogue Scales (VAS) (Bijur et al. 2001). VAS has long been an effective measure of chronic pain (McCormack et al. 1988). However recently it has since been shown to also be valid in instances of acute pain. VAS has also been shown to be extremely reliable in terms of reproduction of results with less than 5% variation in ratings (Bijur et al. 2001). VAS however can sometimes rely too much on correct understanding of the test by the participant and as such can lead to inaccurate results. (Bijur et al. 2001). VAS is used commonly despite this due to the simplicity of the test. Requiring very little equipment or knowledge by the participant in order to be repeatable. As well as providing a finer distinction of the pain due to the non-restricted nature of the scale (Kersten et al. 2012).

1.8 Sleep and the impact on performance:

A newly developed area for performance improvements by athletes is that of sleep. Sleep can both hinder and help an athlete and as such requires further research in order to provide optimal situations for recovery and performance (Fullagar et al. 2015).

1.8.1 Guidelines versus reality:

The National Sleep Foundation currently recommends following a review of over 300 scientific articles that healthy individuals sleep for a total duration of between seven and nine hours for young adults and seven and nine hours for adults (Hirshkowitz et al. 2015).

Whilst these recommendations may exist from the National Sleep Foundation, these guidelines are not always met. Steptoe, Peacey and Wardle (2008) found that in a self-reported study of 17465 students between ages of 17 and 30 that 20% of respondents slept less than the recommended seven hours. This is backed up by Bansil et al (2011) who report that of the 10,000 surveyed participants 33% reported that they slept less than seven hours per night.

Further to these findings Ford, Cunningham and Croft (2015) in a review of data from the National Health Interview Survey between 1985 and 2014 showed that the percentage of adults sleeping less than six hours had increased by 31%, thus showing a trend of shorter sleep duration in modern society. This is believed to be associated with busier work schedules, the advent of the electronic age creating competition for time with electronic toys/technology and an increased prevalence of stress.

1.8.2 Sleep Quality:

The quality of sleep can be equally important in performance. Sleep quality refers to how effective the sleep was for the individual (Krystal and Edinger 2008). Shorter sleep latencies, fewer awakenings and reduced wake after sleep onset were viewed as indicators of good sleep quality, regardless of age in a review of current literature by Ohayon et al. (2017).

Sleep quality is often assessed by clinicians through the use of the Pittsburgh Sleep Quality Index (PSQI) (Mollayeva et al. 2016). This consists of a questionnaire regarding a subject's sleep habits over the previous month. While the PSQI can provide good reliability of tests with a Cronbach's alpha of 0.83, it is limited, in its over reliance on the subject filling in the questionnaire. Both exaggeration or minimising issues can take place by the individual also there is a reliance on good cognitive function and memory of the previous month (Mollayeva et al. 2016)

1.8.3 Sleep states:

Throughout the night humans will undergo several different stages of sleep (Figure 2). A Typical night of sleep will be composed of 90-minute cycles divided into periods of rapid eye movement sleep (REM) and non-rapid eye movement sleep (NREM) (Fullagar et al. 2015). NREM sleep is then further subclassified into 4 different stages depending on further characteristics such as blood pressure and brain activity.

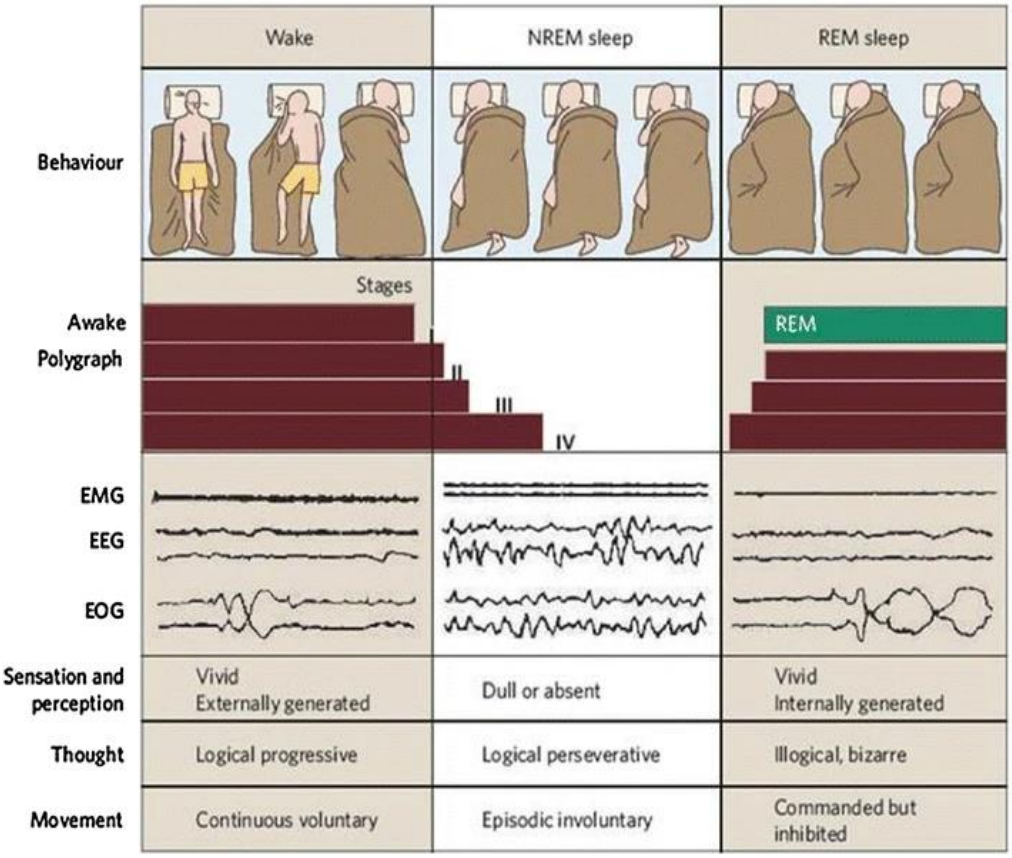

Figure 3: The stages of sleep throughout a sleep wake cycle. Adapted from Fullagar et al. (2015, p 161–186) The first row depicts movements throughout sleep stages. The second row illustrates REM sleep and the four stages of NREM sleep. The third row includes sample polysomnography tracings (each ~20 s) of an electromyogram, an electroencephalogram, and an electrooculogram to help determine the presence or absence of each stage. Rows four, five, and six portray a range of subjective and objective state variables.

1.8.4 Athletes versus Untrained individuals:

Current trends in athletes sleeping patterns are significantly different to those of the average person. Leeder et al. (2012) found that when comparing 47 Olympic level athletes with a non-athletic control group that sleep among athletes was inferior quality to that of a non-trained individual, while getting a comparative duration of sleep. Further to this, Sargent et al. (2014) reports that in a survey of 70 nationally ranked athletes across a range of seven different sports the average amount of sleep was only 6.5 hours, less than the recommended guideline of the National Sleep Foundation.

1.9 Sleep disruption on athletic performance:

Sleep loss has been shown in several instances to have a negative impact on an athletic performance, this includes both endurance and resistance athletes. Similarly, it is also seen with both Sleep Restriction, whereby an individual loses a small amount of sleep but is still able to sleep a small period, and Sleep Deprivation, where the individual will not sleep at all for a prolonged period (Fullagar et al. 2015).

1.9.1 Physical Performance & Sleep:

While current theories around sleep loss on performance are still under discussion, a number of prevailing arguments have arisen discussing both the type of exercise undertaken as well as the method of sleep loss, sleep restriction or sleep deprivation.

Overall while sleep restriction has been shown to not affect singular bouts of aerobic performance (Fullagar et al. 2015) some discrepancy in this still exists. Oliver et al. (2009) and Skein et al. (2011) both suggested that a single night of sleep deprivation was able to negatively impact performance in a time trial scenario, whereby participants were able to cover less distance in the same amount of time following either 30h sleep deprivation (Oliver et al. 2009). Or a timed sprint scenario with no sleep over the course of a day between trials (Skein et al. 2011). Similarly, sleep restriction has been shown to impact sports-specific skill execution, submaximal strength and muscular and anaerobic power (Fullagar et al. 2015). This can be evidenced by Reilly and Piercy (1994) who were able to show that following sleep restriction of three hours for three nights there was a significant reduction in maximal bench press, leg press and dead lift as well as performance degradation in submaximal lifts across the same lifts as well as bicep curl. This is however in contrast to Blumert et al. (2007) and Taheri and Arabameri (2012) who suggested that one night of sleep deprivation had no negative effects on weightlifting performance and anaerobic power respectively.

Sleep restriction has also been shown to have a negative effect on the accuracy of an individual, a key component of many sports. Reyner and Horne (2013) reported a loss in accuracy of tennis serves directly following a night of only five

hours sleep. Edwards and Waterhouse (2009) saw similar results when investigating a four hour decrease in sleep on accuracy of dart throwing.

As well as the above, sleep deprivation has also been shown to be particularly detrimental with regards to time to exhaustion trials (Fullagar et al. 2015), in a review of current existing studies Fullagar was able to conclude that time to exhaustion trials saw a reduction of around 11% in terms of time following 36 hours of total sleep deprivation. These results are supported by other studies highlighting reduced time to exhaustion (mean ~20%) during incremental exercise protocols following sleep deprivation.

Similarly, both sleep restriction and sleep deprivation have been shown as well as exercise to act as an additional stress on the body which can cause increase in heart rate, minute ventilation and plasma lactate concentration (Mougin et al. 1991). This increased metabolic demand can further impact the physical performance of an individual participating in exercise.

Further sleep deprivation has been shown to blunt the full restoration of muscle glycogen stores of team sport athletes by around 25% (Skein et al. 2011). Without adequate intake, this could hinder the ability of athletes to compete for sustained periods and could also suggest a reduced recovery ability.

1.9.2 Pain tolerance:

Pain and athletic performance can be linked through an athlete's ability to overcome a pain barrier. Whether this allows the athlete to train harder, longer or to fight through painful injury rehabilitation, A reduction in pain tolerance could therefore cause a decrease in performance. Sleep loss has been shown by Onen et al. (2001) to decrease pain threshold for subjects. Interestingly in this study it was also seen that pain threshold underwent a rebound during the nights in which recovery sleep was allowed.

While sleep loss has been shown to impact areas of athletic performance there is still doubt as to how it may impact other areas of exercise including that of recovery. Many of the damaging impacts of sleep loss which contribute to athletic loss have been hypothesised to also increase recovery time and inhibit a full recovery system.

1.10 Sleep and the impact on recovery:

Recent evidence has surfaced with regards to the importance of sleep on athletic principles and much of this focuses on the impact of sleep on systems which are crucial to recovery from exercise such as impacts on the inflammatory system and skeletal muscle health.

Sleep deprivation has been shown to have an impact on both the immune system and inflammation as well as increased risk factors of diabetes, cardiovascular disease and obesity (Van Leeuwen et al. 2009). In the study by Van Leeuwen a decrease was seen in NK cells. NK Cells are phagocytes of innate immunity which recognize and destroy intracellular pathogens. An increase was also seen in B cells which are responsible for orchestrating adaptive immunity through cellular and humoral responses. A decrease in NK cells has been shown to increase the chance of infections while an increase of B cells has been shown in mice to enhance immunity and help the individual to survive through extraordinary conditions in the short term, however prolonging this situation leads to increased inflammation, and tissue injury (Van Leeuwen et al. 2009) increased inflammation and tissue injury are crucial factors relating to recovery from exercise induced muscle damage.

Further to these changes in the immune system Van Leeuwen also was able to show after 5 nights of sleep restriction an elevation in inflammatory response immediately after sleep restriction through comparison of C-reactive protein. C-reactive protein has also been shown in humans to be a risk factor in cardiovascular disease as well as playing a role in the development of atherosclerosis and thrombosis suggesting prolonged sleep restriction may change both immune functions as well as inflammatory proteins. Similar to Van Leeuwen, Faraut et al. (2013) in a review of effects of shift work, found that melatonin was a key mediator of both oxidative stress and circadian rhythm disruption. Melatonin production is shown to adjust to light and dark cycles which therefore can act as a circadian rhythm synchroniser throughout the day, however melatonin is also a power anti-oxidative (Faraut et al. 2013). Faraut summarised by suggesting that because night shift workers experience upwards of 20% melatonin suppression due to the exposure of light at night that shift work,

therefore sleep disruption may act as an oxidative stressor and thus making an individual more susceptible to cardiovascular disease and inflammation. This has also been backed up by Zouaoui Boudjeltia et al (2011) in which the recovery process after chronic sleep restriction are associated with changes in blood biomarkers of both oxidative stress and increased cardiovascular risk.

A further implication of sleep deprivation is a reduced level of skeletal muscle health. In a review by Aisbett et al. (2017) it was hypostheised that through altered light exposure, sleep patterns, food and beverage consumption, and impaired drivers of skeletal muscle health such as protein intake, resistance training, and hormone release that shiftwork and therefore sleep deprivation may significantly impair skeletal muscle health through multiple physiological pathways resulting in a reduction of protein synthesis and an increase in protein degradation in the muscle. With a reduced level of protein synthesis and increased protein degradation it would be hypothesised that slower recovery from exercise induced muscle damage may take place.

While little research has been carried out on the impact of sleep loss on an individual's ability to recover from muscle damage as a result of exercise specifically there is some evidence to back up this theory. Specifically with regards to NREM sleep there is reason to believe that sleep loss results in a reduced ability to recovery from exercise. NREM sleep has previously been associated with growth hormone, a product crucial to muscle regeneration, secretion. Similarly, NREM sleep has been shown to be a stimulus for other anabolic hormones which increase synthesis of protein as well as prevention of catabolism of amino acids.

Several studies have been carried out showing an impact of sleep loss on the processes which are important in the immediate recovery from fatiguing processes. These processes include slow breathing and heart rate (Akerstedt and Nilsson 2003) as well as metabolism impacts such as those suggested by Dattilo et al. (2011). Dattilo suggested, following a review of current literature on endocrinology of sleep, that due to the increased stimulation of protein degradation and its impact on protein synthesis, individual muscle cell recovery and adaption would be impacted. Due to the impact of peripheral fatigue during

exercise this hypothesis is still to be investigated with emphasis on resistance exercise damage due to the increased need for protein synthesis.

Similarly, McMurray and Brown (1984), assessed that following 24 hours of wakefulness, ventilation and oxygen uptake remain higher after exercise. As well as this blood glucose was seen to be higher during the trial for those on the sleep protocol. Both blood glucose and oxygen are key contributors to initial recovery from exercise as shown by Bangsbo and Hellsten (2002). This therefore could further suggest evidence for impacted recovery following sleep loss.

The increasing evidence, as provided initially by Walker and Stickgold (2005) that sleep may play an important role in learning and other cognitive functions could suggest a role of sleep in recovery of these cognitive functions which can play crucial roles in exercise. Finally, with the increased metabolic demands of sleep loss as shown by Mougin et al. (1991) due to the stress imposed by sleep loss an inability for metabolic recovery could also hinder full return to performance of an individual who has undergone lack of sleep.

It is therefore recommended that further research be carried out into this topic in order to provide adequate recommendations as to sleep as a tool for recovery from exercise. Due to the higher level of responsiveness of untrained individuals to damage following exercise these individuals will provide the clearest level of effect of sleep loss on recovery. Similarly, in evaluation of any muscle damage both performance and non-performance based markers when assessed together would provide the strongest basis of assuring muscle damage is recorded.

1.11 Research aims:

The aim of this research is to investigate the relationship between sleep restriction and markers of muscle recovery from a single session of eccentric induced muscle damage in the bicep brachii.

Specifically, the aims of the current project are to assess the effects of partial sleep restriction on:

i) Muscle damage recovery based on pre damage baselines
ii) Muscle damage recovery in participants with and without sleep restriction

2.0 Methods

2.1 Participants

16 males with no history of resistance training within the past six months (mean age 23 ± 17 years, height 180.6 cm ± 15.4, mass 87.4 ± 30.7 kg) volunteered after providing informed consent. Participants were excluded from this study if they had any history of sleep disorders or known health issues which restricted participation in exhaustive exercise. The study was performed according to the declaration of Helsinki and was approved by the University of Stirling NHS, Invasive or Clinical Research (NICR) committee, paper: NICR 17/18 – Paper No.09

2.2 Experimental Procedure

Participants visited the lab over a period of six days. Prior to the first laboratory visit all participants were asked to refrain from: (1) performing any resistance training, and (2) caffeine consumption for a 24h period. All testing was performed on participants' non-dominant arm to ensure there was minor obstruction to participant and to achieve maximal damage. During the first laboratory visit (Day 0) baseline measures were obtained for muscle soreness, limb volume, blood plasma creatine kinase and maximal voluntary contraction of the Biceps Brachii. Following these baseline tests participants carried out an Exercise Induced Muscle Damage (EIMD) protocol. Upon completion participants were randomly assigned one of two groups: Sleep Deprived (SD) and Full Sleep (FS). Those who were assigned to the SD group were asked to adhere to no more than four hours total Time In Bed (TIB) for the following two nights. Those in the FS group were asked to continue with individual normal sleeping pattern. All participants were asked to return to the lab on days 1, 2, 4 and 5 to repeat all baseline tests.

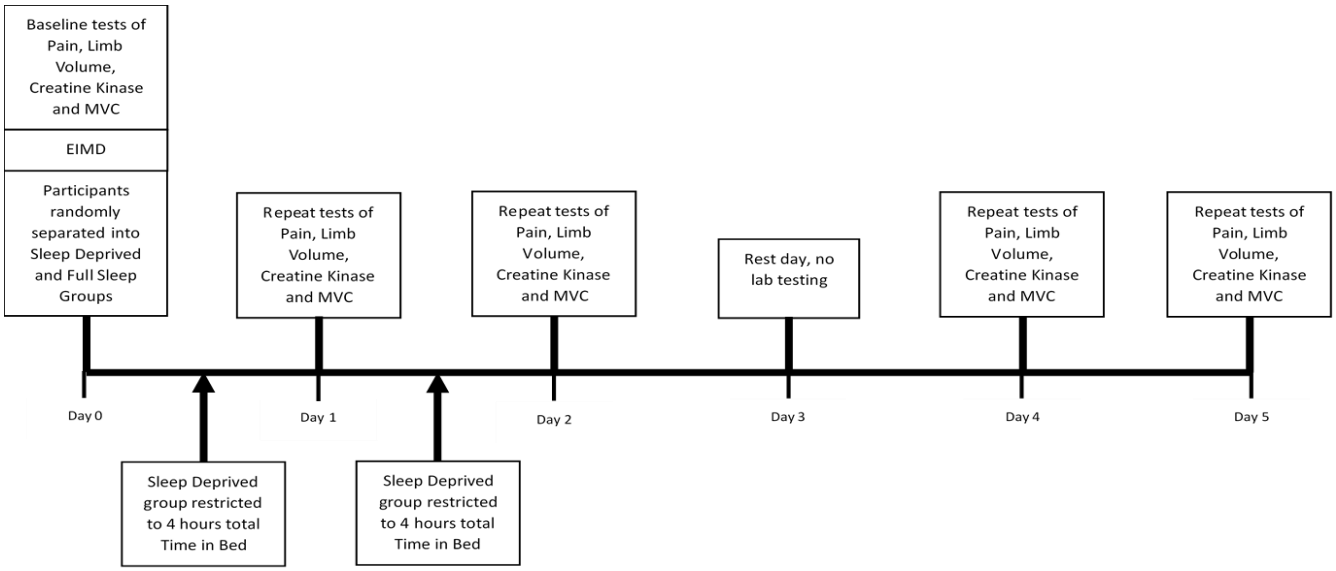

Figure 4: Study week outline. Day 0 respresentive of first day reporting in lab. Participants performed all baseline tests on days 0, 1, 2, 4 and 5. Exercise Induced Muscle Damage was performaed on day 0. Any sleep restriction was taken place in between day 0 and 1 & 1 and 2. Day 3 was reserved for recovery from sleep restriction and as such no testing took place.

2.3 EIMD

On day 0 following MVC, EIMD was induced in participants by completion of five sets of ten maximal eccentric isokinetic contractions with 60s between sets at a range of motion from fully contracted to fully extended. At a velocity of 30° per second. This was performed on an isokinetic dynamometer (Biodex System 3, Medical Systems, USA) This protocol was previously carried out in our lab by Hunter et al. (2012) and has been shown to induce muscle damage. Contractions were started at fully contracted position and were performed continuously within the sets. All participants received verbal encouragement throughout the contractions.

2.4 Sleep Deprivation

Individuals who were assigned to the sleep deprivation group were asked to adhere to four hours total TIB. This was recorded by individuals in a sleep diary which was also used in the week before testing in order to create baseline sleep patterns. Participants were asked to record time to bed, wake up time, time to fall asleep and total times awoken during the night. As a further regulatory method

all participants were asked regardless of group allocation to inform a member of the research group through text message when they were both going to bed and waking up thus ensuring the individual was indeed awake at times included in the sleep diary.

2.5 Muscle Soreness

Participants were asked to rate their soreness on a visual analogue scale (VAS) in four different positions; Overall pain, at 90°, at 180° and at fully contracted. Participants rated their muscle soreness using a 200mm VAS with the far left tether point representing “No Pain Whatsoever” and the far right tether point representing “Worst Pain Imaginable”. This was in accordance to a scale validated by (Bijur et al. 2001). Distance was then measured using standard ruler from “No Pain Whatsoever” tether in order to create value.

2.6 Limb Volume

Arm volume was measured through an arm volumeter (custom built) along with a measuring cylinder (Merck Measuring Cylinder, Merck & Co., US). The volumeter was filled with water (30°C ± 2°C) until it reached a preinstalled outlet. This was allowed to rest still while all excess water flowed out of water outlet. Water temperature was controlled using a thermometer (Fisher Scientific™ Traceable™ Lollipop™ Shock/Waterproof Thermometer, Thermo Fisher Scientific, USA).

In order to ensure consistency a mark was drawn on the anterior part of the participants’ arm level with the tip of the axilla. This mark was redrawn on each subsequent visit to ensure it did not fade. Participants lowered their arm into the into water until it was level with drawn mark and at this point were asked to remain still so as to allow water to flow out of the outlet into the measuring cylinder. This water was allowed to flow until there was less than one drop per two seconds. Limb volume was then calculated as the volume of water in the measuring cylinder.

2.7 Blood Sampling

Five ml blood samples were collected from the antecubital vein of the participant following limb volume measurement and before MVC. Blood was collected into a

5ml vacutainer tube (Becton Dickinson, USA), containing EDTA for plasma separation. The tube was centrifuged at 2500 rpm for 10 min at 4°C and Plasma Stored at -70°C until analysis.

When analysis took place all samples were assessed for creatine kinase levels using an iLab Aries automatic biochemical analyzer (Instrumentation Laboratory, USA) and using a commercially available reagent (CK-liquid, Werfen,USA).

2.8 Biodex Positioning and MVC

Participants were secured in an isokinetic dynamometer (Biodex System 3, Medical Systems, USA) for measurement of MVC and for performing eccentric contractions to induce EIMD. The seat position was adjusted to suit the participant's anthropometric characteristics. The upper arm was placed on a padded support anterior to the body and the elbow flexed at an angle of 90° where 0° was at full extension. Velcro straps were used to secure the participants body and limbs into the required positions. The position of the participants was recorded on the initial visit and kept constant throughout the experimental period. The dynamometer was calibrated daily according to the manufacturer.

Following blood sampling, soreness and limb volume assessment participants were instructed to perform a standard submaximal warmup protocol on an isokinetic dynamometer. This consisted of two sets of three 5 second contractions with 60 seconds rest. This was performed at a perceived intensity of 50%, 75% twice.

Following the warm up participants performed isometric MVCs, this consisted of three maximal five second contractions with 60 second rest in between. Participants were all instructed to contract quickly and maximally following command and all participants received verbal encouragement to ensure maximal contraction of the arm. The Peak torque as recorded by the dynamometer was recorded as MVC.

2.9 Statistical Analysis

Data was analysed using Statistical Package for Social Sciences 25 (IBM SPSS, Chicago, IL). Area under the curve (AUC) was calculated using the trapezoid method for MVC, CK, Limb Volume and all four individual pain assessments.

Data was then tested for normality using Shapiro-Wilk normality test. Adequate EIMD was checked across all variables by assessing Paired Sample T-tests between pre-test data and day1 data across all variables. Differences between the two groups for all measures were assessed using Independent T-tests. Alpha was set at 0.05 Following assessment required sample size and actual power was calculated using G*Power3 (Faul, Erdfelder, Lang, & Buchner,2007) effect size was calculated in the standard fashion.

3.0 Results

3.1 Baseline:

Baseline values for all tests when separated by sleep group are presented in Table 1. There was no significant difference between Reduced Sleep (RS) and Full Sleep (FS) groups prior to any sleep deprivation or muscle damage taking place.

	FS	RS	P	Cohen's D	Required sample size	Actual power
Maximum Voluntary Contraction (N·m Torque)	70 ± 22	77 ± 25	0.595	0.27	434	0.08
Plasma Creatine Kinase (U/L)	317 ± 273	245 ± 185	0.547	0.31	330	0.09
Limb Volume (mL)	3173 ± 650	3195 ± 827	0.953	0.03	34886	0.05
General Arm Pain (mm)	0 ± 1	6 ± 17	0.370	0.48	140	0.15
Pain at 90 Degrees (mm)	1 ± 1	4 ± 12	0.394	0.44	166	0.13
Pain at 180 Degrees (mm)	1 ± 1	0 ± 0	0.250	0.63	82	0.22
Pain at Full Contraction (mm)	1 ± 2	0 ± 0	0.265	0.61	88	0.21

Table 1: *Baseline testing results on Day 0 for Restricted sleep (RS) (n=8) and Full Sleep (FS) (n=8). Data Presented as mean ± SD. No significant differences between groups.*

3.2 Sleep characteristics:

Sleep Duration is presented in Figure 2. Habitual sleep (before testing) presented with mean time in bed 8 hrs 3 minutes ± 1hr 48 min. This was comparable to during the Full Sleep (FS) condition where mean time in bed was 7 hrs 55 min ± 1 hr 46 min (p=0.500). Mean time in bed was lower in the Restricted Sleep (RS) group: 6hrs 33 across the whole test (4 hrs 09 on sleep deprived nights and 8 hrs 16 on recovery nights) and was significantly lower than pre-test sleep (p=0.001).

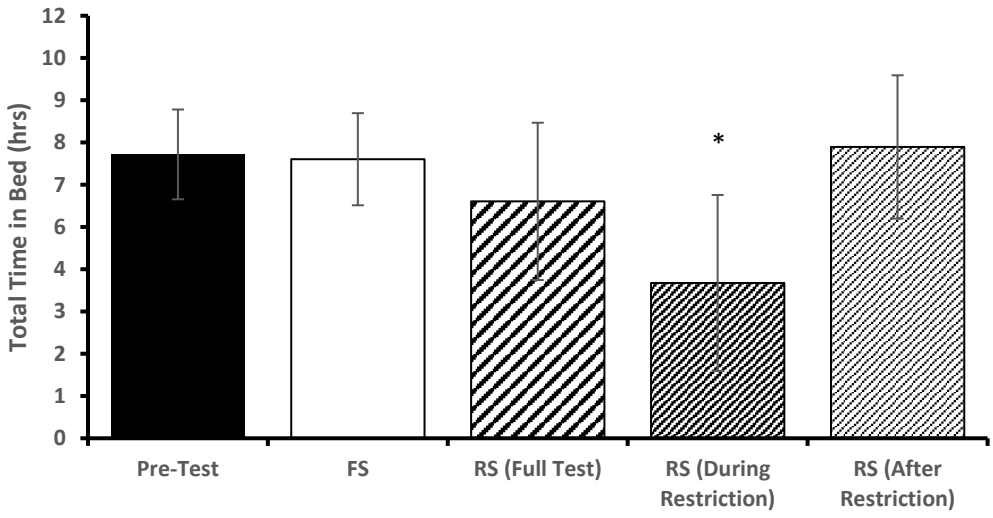

Figure 5: *Total Time in Bed for each group; Pre-Test (all participants)(n=16), Full Sleep group (FS)(n=8), Restricted Sleep reported across the whole testing period (RS Full Test)(n=8), Restricted Sleep during nights when sleep was restricted (RS During Restriction)(n=8) and restricted sleep group following Restriction taking place (RS After Restriction)(n=8). Pre-Test and Full sleep groups were not significantly different (p=0.500). Restricted sleep was significantly different from Pre-Test Sleep (p=0.001). *denotes significance*.

3.3 Exercise Induced Muscle Damage:

Results of testing following Exercise Induced Muscle Damage (EIMD) are displayed in Table 2. Following EIMD there was a significant reduction in MVC and increase in Limb Volume and Arm Pain across all four testing points. Plasma Creatine Kinase however, saw no significant difference on the following day of testing.

	Day 0 (Pre-testing)	**Day 1**	**P**	**Cohen's D**	**Required Sample size**	**Actual power**
Maximum Voluntary Contraction (N·m Torque)	73 ± 23	61 ± 19	<0.001 *	0.57	27	0.57
Plasma Creatine Kinase (U/L)	281 ± 228	337± 320	0.298	0.20	199	0.12
Limb Volume (ml)	3184 ± 718	3310±745	0.006 *	0.17	274	0.1
General Arm Pain (mm)	3 ± 12	12 ± 14	0.005 *	0.69	19	0.73
Pain at 90 Degrees (mm)	2 ± 8	8 ± 12	0.003 *	0.59	25	0.59
Pain at 180 Degrees (mm)	0 ± 1	16 ± 17	0.003 *	1.33	7	0.99
Pain at Full Contraction (mm)	0 ± 1	18 ± 16	0.001 *	1.59	6	0.99

Table 2: *Testing results across both sleep status at baseline and following EIMD. Data Presented as mean ± SD. N=16.*
** denotes Significance.*

3.4 Effects of Sleep Restriction:

Area Under the Curve (AUC) results for Full Sleep Group and Restricted Sleep Group when testing for all parameters are presented in Table 3. Visual analogue scale results for pain tolerance, MVC results, CK activity and LV results across both RS and FS groups throughout the full testing week are presented in Figure 3. Across all parameters there was no reported impact of sleep loss on the RS Group when compared to the FS Group. As seen in Table 3, increases were seen across all AUC analysis for each parameter for both groups. However, the changes were not significantly different between the two groups.

	AUC FS	AUC RS	P	Cohen's D	Required sample size	Actual power
Maximum Voluntary Contraction (N·m Torque)	270.77 ± 70.62	276.84 ± 88	0.881	0.08	4908	0.05
Creatine Kinase (U/L)	1433.06 ± 832	3292.78 ± 5846	0.388	0.45	158	0.13
Limb Volume (ml)	12997.5 ± 2676	13168.75 ± 3472	0.06	0.47	146	0.14
General Arm Pain (mm)	20.63 ± 19	39.63 ± 45	0.298	0.55	106	0.17
Arm Pain at 90 Degrees (mm)	13.81 ± 10	27.06 ± 33	0.294	0.54	110	0.17
Arm Pain at 180 Degrees (mm)	32.25 ± 27	41.69 ± 44	0.612	0.26	468	0.08
Arm Pain at Full Contraction (mm)	34.44 ± 28	49.75 ± 43	0.416	0.42	180	0.12

Table 3: *AUC results for all parameters across five days of testing following EIMD. Results are separated between undergoing 2 nights of restricted Sleep (RS) and undergoing full sleep (FS). Data Presented as total AUC ± SD. N=8 for both groups. No significant differences between groups.*

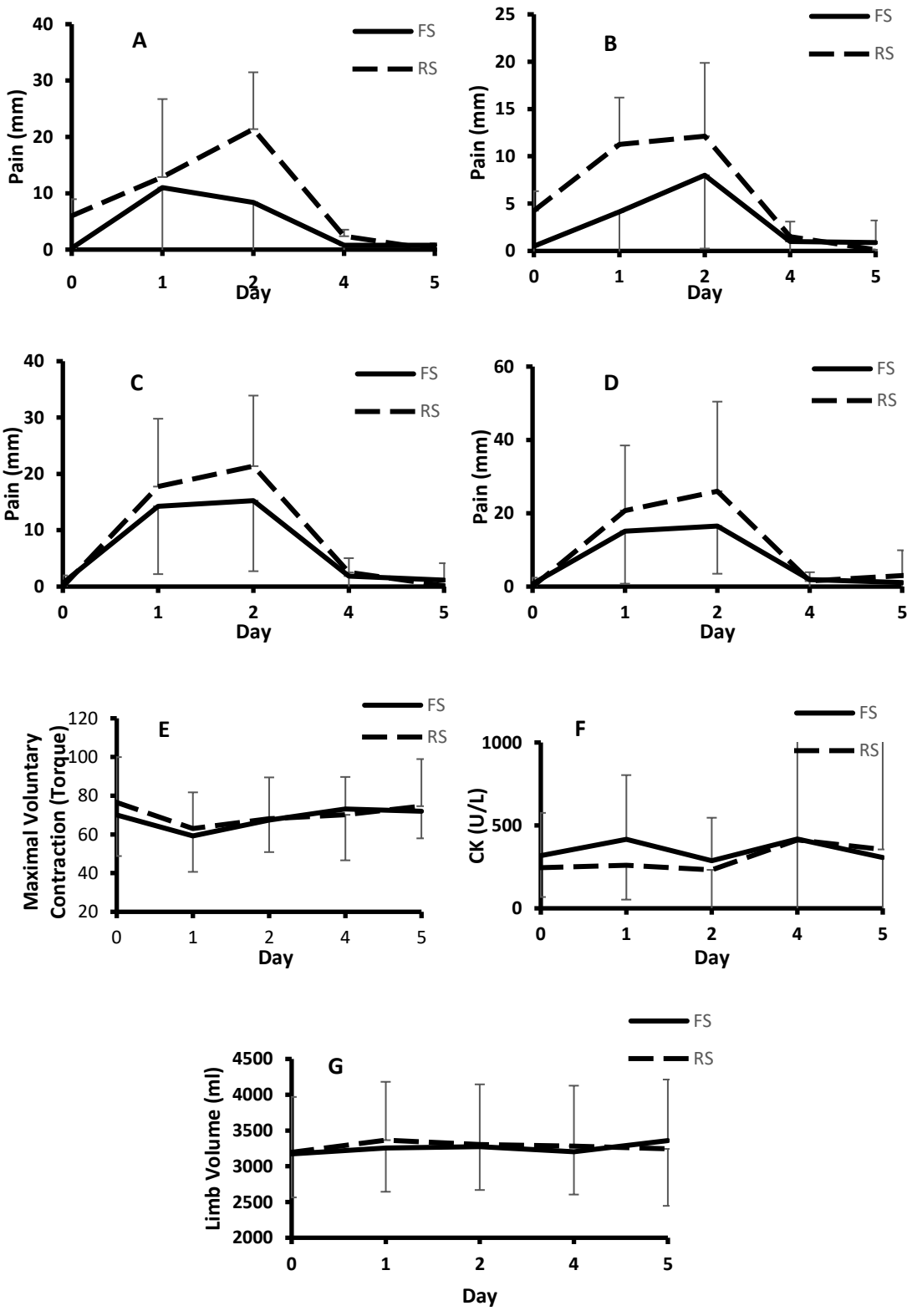

Figure 6: *Characteristics of muscle recovery in Bicep Brachii across five days of testing following Exercise Induced Muscle Damage on Day 0 while undergoing either Full Sleep (FS) (n=8) or Restricted Sleep (RS) (n=8). Data is presented as Mean ± SD. (A) General Pain in Arm. (B) Pain when arm held at 90 Degrees. (C) Pain when arm held at 180 degrees. (D) Pain when arm Fully Contracted. (E) Maximum Voluntary Contraction. (F) Plasma Creatine Kinase. (G) Limb Volume.*

4.0 Discussion

The aim of this study was to assess whether two nights of sleep restriction to four hours total time in bed had any effect on the recovery from eccentric induced muscle damage in untrained healthy young males when compared to pre damage baselines and participants who carried out a normal night of sleep. The findings demonstrate that two nights of sleep restriction did not impair the recovery process in the body across any of the measured factors of muscle damage. This contrasts the suggestion by previous studies such as that of Dattilo et al. (2011), Nédélec et al. (2015) and Fullagar et al. (2015) that sleep may aid the recovery of muscle damage through a potential modification of metabolic systems, damage repair, hormonal responses or inflammatory response. The results of our study therefore suggest that following EIMD in biceps brachii, sleep does not seem to impair markers of muscle recovery. However, evidence still exists of sleep as a recovery aid and as such further investigation in this topic is still required.

This study reports no significant effects of the impact of sleep restriction on each of the chosen characteristics of muscle damage. This contrasted with the hypothesis that sleep restriction would result in impairment of the recovery process as was suggested by Dattilo et al. (2011) as well as the suggestion by Fullagar et al. (2015). Dattilo et al. had made this recommendation based on the increased stimulation of protein degradation and its impact on protein synthesis which can be caused by a hindrance to sleep. Further Fullagar et al. expanded on this through suggestion of sleep playing a role in repair of the nervous and metabolic systems. These systems are very prevalent in muscle damage with impairment of repair therefore impacting on overall recovery from this damage.

There was a statistical reduction in Maximal Voluntary Contraction (MVC) on Day 1 after damage and following one night of restriction thus confirming that muscle damage had occurred, but this was similar in both groups. This damage was also seen to have recovered to pre-baseline values in both groups by day 4. This data is in line with what has been seen in previous studies. Following damaging exercise MVC has been seen to reduce by 37% on the first day (Hunter et al. 2012). Similarly, Howatson (2010) reported a reduction in MVC of 17% after 48

hours following eccentric induced EIMD on the Bicep Brachii this is comparable to the present study in which a 17% drop off was seen. It is also worth noting that the sample size in this group was similar to these studies with 12 participants in Howatson's study and 19 in Hunter et al's. this further confirms a similar power and effect size as in these studies which showed reduction in MVC. Due to the use of an accurate dynamometer and repeated experience as well as showing similar decrements in MVC with the previous studies this data is detailed in showing significant damage to the bicep brachii.

Limb volume assessments saw relatively small increases in both groups suggesting an increase in swelling following muscle damage. Swelling would normally be expected following muscle damage as shown by Chen et al. (2011) and discussed by Proske and Allen, (2005) as an inflammatory response which is accompanied by oedema and as a result can be presumed to cause of muscle swelling. This damage was almost fully recovered in both groups by Day 4. Our results contrast with those reported by Hunter et al. (2012) using a similar protocol. They report increases of over 1cm in medial limb girth on day 3 as measured by upper arm circumference following the same muscle damaging exercise protocol. In the present study the use of a custom built volumeter resulted in difficulty attaining highly accurate results. This was due to an issue in design in which any movement by the participant would cause water to flow out of the system, which may have increased readings on each use. Participants were instructed to remain as still as possible while in use of the volumeter however this design flaw cannot be ruled out as a reason for the high variability in results. Chromy et al. (2015) in a review of limb volume measurements had previously suggested that limb immersion was the best method for assessment due to its increased accuracy and repeatability. However, Chromy et al. (2015) also recommended that due to shivering as well as an inability to measures specific muscles that when facing these issues to use other methods such as MRI or CT scans, in the present study these were not available as an option despite facing issue with shivering and the false water droplets caused from this. The issues faced in the current study could result in a lack of accuracy in limb volume assessments.

Interestingly when investigating pain, area under the curve data was 92% higher for general arm pain, 96% higher in 90 degree, 29% higher in 180 degree and 45% higher upon full contraction of the arm when sleep restriction was occurring. While these results were not significantly different, the higher area under the curve results particularly on the days of restricted sleep suggest further testing be required which may result in a further in-depth analysis into pain. This could include increasing the testing points of when pain occurs throughout the day to investigate tolerance of participants to the established pain of muscle damage. The hypothesis of increased pain rankings following sleep restriction would be in line with what is suggested in numerous areas of research suggesting that a loss of four hours sleep, similar to in the present study, could be hyperalgesic on the following day (Roehrs et al. 2006). While this may not affect the practical implications of recovery such as MVC or swelling it may result in a feeling of recovering slower.

Exercise-induced muscular damage was seen to be induced in all markers of muscle damage regardless of sleep status, except for creatine kinase which did not see a significant increase in values. A lack of increase in creatine kinase levels is contrary to what would be expected of a person undergoing muscle damage (Koch, Pereira and Machado, 2014). Creatine Kinase has also been seen to be particularly effective in studies involving muscle damage in the arms as opposed to legs (Jamurtas et al. 2005). Jamurtas et al. suggest that this may be as a result of reliance of leg muscles in daily activities. In this instance the repeated bout effect of muscle damage will play an impact in reduction of any negative association of muscle damage including a reduction in cell permeability and therefore a reduction in Creatine Kinase leakage into the blood stream. This is further contrary to what was seen in the present study where not only arms were used but also the use of non-dominant arm to further limit the previous use of the muscle group. Further backup as to the use of arms in the present study is shown by Nosaka and Clarkson (1992) whereby muscle mass is shown to have no correlation in post exercise Creatine Kinase secretion. Koch, Pereira and Machado (2014), also suggest that several studies have reported variations between individuals in creatine kinase levels based on genotype, body composition, (Heled et al. 2007) or age (Webber et al. 1989) While we were able

to limit these factors in our study (age 23 ± 17, mass 87.4 ± 30.7 kg) we cannot rule out the effects these may have caused on the variability of creatine kinase. Similarly, creatine kinase can have a high coefficient of variation (Jackson et al. 1987). Further studies have also shown a lower correlation with upper body work done compared to serum creatine kinase levels (Machado et al. 2012). As well as this Baird et al. (2012) has suggested that serum creatine kinase levels alone do not provide an adequate reflection of structural damage to muscle cells as it can be too varied between individuals.

Participants adhered to the protocol of four hours total time in bed (TIB) when in the sleep restricted group through self-reported sleep diaries and the “Check-In” method used by researchers. This was in comparison to reported mean time in bed of slightly over eight hours on a habitual basis. Eight hours was also adhered to in the full sleep group with participants reporting normal sleeping habits during the test. However during testing throughout the study there were a small amount of days where individuals did not achieve a full 8 hours TIB, this occurred across both groups and both in the recovery or pre testing recording of sleep. This therefore may have influenced results. However, as the only significant difference found was during testing of the restricted sleep group we can conclude that sleep restriction was achieved in the sleep loss group and that this group was able to achieve significantly less sleep over the two nights of investigation.

The results of this study may have been affected by certain limitations within the study design. Importantly due to the nature of the study it was impossible to blind participants to the sleep protocol which was undergoing. This may have therefore created a bias in participant reported tests such as pain. Research in the area of circadian rhythms has also suggested that sleep restriction can alter circadian rhythms (Mizuno, 2014) similarly Mizuno suggests in this review that exercise performed at certain times of the day can differ in terms of performance. While we tried to test/retest at the same time of day across the study there were times where participants were tested at different times during the day. There was always at least 1 full overnight period between measurements. This difference in performance result could have impacted MVC in particular. Previous research in the form of a meta-analysis of strength throughout the day by Grgic et al. (2019) found that measures taken in the evening hours tended to be higher than those

taken in the morning and as such may have created higher measurements for our own study when investigating participants later in the day. Further CK has been shown to be elevated up to 48 hours following testing (Nosaka et al. 1992) while testing was always achieved within this range we cannot rule out that peaks of creatine kinase may have been missed due to the difference in time course of testing. Pain levels have also been shown to be affected by time of day in healthy young males (Aviram et al. 2013). Aviram was able to show that pain sensitivity was highest in the evening and lowest in the morning. Therefore, it may be useful to further investigate levels of pain over the course of the day at regular intervals following sleep restriction to provide a profile of the true extent of a potential impact of sleep disruption on post exercise pain.

Further limitations may have also existed with regards to control of further recovery interventions. While all participants were requested not to apply any topical anti-inflammatories or use any other recovery strategies this cannot be ruled out. Further we were unable to control individual nutrition or any physical activity recovery strategies on an individual participant basis. Instead individuals were encouraged to remain as close to normal dietary strategies during the study and to avoid introduction of any new supplements or recovery aids as well as to avoid exercise which may hinder recovery. These limitations mean that results of this study were presented with a low statistical power and therefore restrict the conclusions we can make about the effects of sleep loss exercise recovery.

The findings of this study in conjunction with the further existing literature identifies other areas which may benefit from further investigation. While we were unable to confirm an effect of sleep loss on recovery in the present study, we believe a more severe sleep loss or differing system of sleep loss could warrant a statistically relevant impairment in recovery. Previous research has hypothesised that Non-Rapid Eye Movement (NREM) sleep may be key in recovery in various of exercise (Fullagar et al. 2015), currently it is believed that humans will cycle between NREM sleep and REM sleep throughout a night. These sleep cycles range from between 90 minutes and 120 minutes. Due to this it would therefore be of interest to investigate to what extent if any a disruption to specific periods of sleep, REM periods versus NREM periods may impact recovery. This could be achieved through investigation into either less total time

in bed or an interval-based sleep disruption such as 1 hour awake followed by 1 hour asleep.

It may also be of interest to investigate various other choices with regards to population of the study. For this study the chosen group was untrained males this was in order to provide as much recognisable muscle damage while negating any possible effects of repeated bout effect, whereby exposure to a repeated bout of eccentric exercise can result in attenuated symptoms of muscle damage (Gleeson et al. 2003). However, Gleeson also suggests that prior concentric training, which is more commonplace among trained individuals, can actually lead to increased susceptibility of EIMD. Therefore, further investigation could also lead to differing results between untrained and trained individuals in this instance. Similarly while a decision was made in the present study to perform the EIMD on the non-dominant arm of participants in order to provide both highest recordable levels of damage (Jamurtas et al. 2005), while also providing minimum disruption to participants, this may also lead to differing results due to the nature of higher use of dominant arm. Howatson and van Someren, (2007) has previously shown evidence of a contralateral repeated bout effect in the arm. This therefore may have weakened our anticipated EIMD and could provide basis for further investigation into EIMD in dominant arms. While other studies have shown muscle damage to be higher in arms following EIMD it may be useful to investigate the effects of sleep disruption on larger muscles particularly in those in the legs of untrained individuals. This could generate differing results due to the lack of repeated bout effect as described by Howatson and van Someren, (2007). Finally, in the present study we chose to investigate males due to the suggested advantages of oestrogen in muscle recovery (Tiidus, 2005,2011) as well as the implications of oral contraception blunted responses to MVC (Joyce et al. 2014). However further studies have found that gender in fact does not impact muscle recovery and that this area requires further investigation (Kendal and Eston. 2002).

In conclusion, this study investigated recovery from EIMD through measurement of limb pain, limb volume, post-exercise serum creatine kinase and MVC in untrained individuals. This population did not show a difference in recovery markers between sleep restriction and full sleep despite a proven increase on all

markers of EIMD except creatine kinase. This is contrary to what was suggested in previous literature, namely Datillo et al. (2011) as well as Fullagar et al. (2015) whereby a hypothetical situation is suggested in which sleep restriction would impact on recovery from damaging exercise. Unfortunately, because of the certain limitations in this study we were presented with a low statistical power and as such we are unable to confirm that sleep loss does not have an impact on recovery from exercise induced exercise. The findings of this study are therefore presented as pilot data and should be used to further clarify the systems of post exercise recovery and suggests less reliance on damage repair and the inflammatory response as well as less reliance on the protein degradation and synthesis which is suggested by Datillo et al. (2011). Further investigation however should be paid to studies involving both more severe sleep loss as well as different population groups. Further investigation into the effects of sleep loss on pain sensitivity is also recommended due to the potential impact of sleep and time of day on pain reporting. This research in conjunction with the present study will aid in providing a framework for recovery from damaging exercise which can involve other systems less reliant on achieving adequate sleep.

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Appendices

Appendix 1- Participant sleep diary

Participant Name: _____ Participant Age: ____

Researcher: Greg Atkinson

Date	Time to bed	Time awake (next day)	How long did it take you to fall asleep?	How many times did you wake up through the night?	Total time asleep
Eg: 13/3/17	1030pm	6:00am	15 mins	2	7.5 hours

Activity Diary **Date**	**Activity Type**	**Duration**
Eg: 13/3/3	5 a-side football	30 mins