Effect of moderate Alcohol consumption on recovery from Eccentric Exercise Induced Muscle Damage in Females

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1. Abstract

Introduction: Unaccustomed eccentric contractions produce micro-structural damage to skeletal muscle, initiating a chain of events involving inflammation, pain, and impaired muscle performance. These effects are felt most between 24-72 hours post exercise, but can last anywhere from a few days up to a week. Sports involving a large eccentric component include those that are running-based; with resulting damage often impacting on successive games (common during competition), or on ability to train. Binge-drinking post-games is a common activity seen amongst athletes; particularly those involved in team sports. Both acute and chronic alcohol consumption have known negative impacts on the brain and body organs and being classified as a drug, has associated regulations and restrictions. Athletes require fast recovery from exercise-induced muscle damage for optimal performance in subsequent games or training. The effect of alcohol, exercise, and/or the combination of the two may have a negative effect on muscle recovery post-game following an acute intake of alcohol. While these types of studies have been carried out on males, as of yet no such study has been done on females. Due to physiological differences, there may be variation in muscle response to alcohol compared to that of males. Thus the purpose of this study was to compare the effects of alcohol consumption post exercise-induced muscle damage, with that of an iso-caloric placebo on muscle recovery in females.

Methods: Eight females (mean age 23 years; 65.2 ± 15kg; 164 ± 5.5cm) participated in a controlled, randomized, cross-over design study. Following a prescribed standardised meal, they carried out a damage protocol in which 300 maximal

eccentric contractions of the quadriceps femoris muscle were performed on an isokinetic dynamometer. Post-exercise, an alcoholic beverage or a placebo was consumed. Treatment type and leg of damage were randomly assigned as evenly as possible, with the two trials being separated by a month. Measures of maximal isokinetic (concentric and eccentric) torque and isometric tension produced across the knee were measured in both the exercised and control leg pre-exercise, 36 hours (h) and 60h post-exercise. Creatine kinase activity and muscle soreness ratings (squat and step) were taken prior to damage and post-exercise up to 60h.

Results: A moderate amount of alcohol consumption following eccentric exercise, significantly reduced isometric, concentric and eccentric peak and average peak torque 36h and 60h post-exercise (all $p < 0.05$). Significance differences in force output between time points were seen only in peak and average concentric torque. All three contraction types showed a significant time * treatment interaction effect ($p < 0.05$). Creatine kinase and ratings of perceived muscle soreness did not significantly differ between treatments.

Conclusions: Our results suggest that similar to males, the consumption of alcohol following eccentric exercise-induced muscle damage elicits a greater reduction in muscle performance in the days following damage in females.

2. Acknowledgments

Through the months of late nights, early mornings, times of trial and times of triumph, two men have stood with patience, support, guidance and the hard word when needed. Associate Professor Stephen Stannard and Dr. Toby Mundel – I cannot thank you enough for all you have done to help get this research completed. Another big thank-you goes out to Dr. Andrew Foskett who came on board halfway through and helped keep me on the wagon.

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4. Introduction

A significant number of individual and team sports involve eccentric muscular contractions in which the muscle lengthens while under tension. Eccentric contractions are well documented to bring about varying levels of skeletal muscle damage which is often followed by delayed-onset muscle soreness, circulating markers of muscle damage, and a decrease in muscle performance. In sports that require optimal muscle function over subsequent days, such damage can negatively affect muscle function; thus the rate of recovery from this damage can be crucial to subsequent performance. Within the sporting community, post-game drinking sessions are common. This 'team-bonding' activity is undertaken to celebrate a win, or to share the disappointment of a loss, and frequently involves a large consumption of alcohol referred to as 'binge-drinking'. While the general effects of alcohol on the body are well documented, there is little known of the effect of exercise-induced muscle damage and alcohol consumption on muscle recovery and subsequent performance. For athletes, who require optimal muscle functioning to perform at their best, alcohol consumption post-games may negatively affect muscle recovery time, thus impacting upon their ability to perform in following training and/or games.

While recent studies have investigated possible effects of alcohol consumption on muscle following exercise in men, such studies have yet to be carried out on women. Differing hormonal, metabolic and chemical processes between genders could affect findings; therefore the aim of this study was to investigate the effect of alcohol consumption on skeletal muscle force recovery after strenuous eccentric exercise in females.

Note: skeletal muscle force recovery in this instance is defined as return to 100% of initial (pre-eccentric exercise) torque.

5. Review of Literature

5.1 Introduction

As well being a crucial part of everyday activities, muscle contraction becomes especially important in sporting situations where often rapid movements are required by different parts of the body simultaneously [3]. Optimal muscle functioning is therefore very important for performance during sport, and for this reason research into muscle damage and recovery has become an extensive area of interest. Whilst studying isolated contractions does not fully represent what is happening during activity, it does allow us to experimentally test the effect of an intervention (i.e. nutrition, stimulants, over-training etc) on muscle function. Interventions may have a positive or negative effect on muscle function, or may have no effect whatsoever. In the case of recovery following muscle damage, interventions may either delay or expedite time to regaining full muscle function. The consumption of a large acute amount of alcohol post-sporting events amongst team sport players is becoming an ever increasing concern. Binge-drinking may have the ability to increase time to recovery and therefore performance; with alcohol both directly and indirectly affecting physical (muscular) and mental (nervous system) functioning.

5.2 Eccentric Exercise-Induced Muscle Damage (EEIMD)

Skeletal muscle contractions are categorized into three different types; isometric, concentric and eccentric. Isometric contractions occur where there is tension of the muscle while maintaining length, for example the action of the biceps brachii during arm wrestling. Concentric contractions occur where there is tension of the muscle while shortening and are often used to initiate movement [4], for example the lifting action of the biceps brachii in curling a barbell. Eccentric contractions occur where there is tension of the muscle while lengthening, often being used to slow or stop

movement [4],for example the biceps brachii activity as a barbell is slowly lowered. Out of these three contraction types, activity involving predominantly eccentric action (i.e. prolonged downhill running) have proven to produce greater muscle damage than exercise involving either concentric or isometric contractions[5]. When muscle lengthens under tension, microscopic tears within muscle fibres can occur, resulting in an inflammatory response which is a natural process essential for the repair and recovery of damaged muscle[6]. This inflammatory response can elicit soreness within the muscle, commonly referred to as delayed-onset-muscle-soreness, or 'DOMS'. This soreness is often used as a indication of muscle damage normally peaking after 24 to 72 hours [7, 8] but may last several days or in severe cases up to a week [9]. Soreness can range from being slightly painful to being quite debilitating, and in an athlete this pain can often be an unwanted training hindrance. The associated reduction in muscle function over subsequent days during competition has the ability to affect both individual and team-sports by limiting performance.

5.2.1 Signs of Eccentric Muscle Damage

Eccentric exercise-induced damage to skeletal muscle initiates an inflammatory response causing a flood of inflammatory cells within both the injured muscle and circulation, and appearance of muscle proteins and enzymes in the blood [10]. Invading inflammatory proteins include tumor necrosis factor-alpha (TNF-α), interleukin-1-beta (IL1-β), interleukin-6 (IL-6), and interleukin-1 receptor antagonist (IL-1ra) while the increased appearance of muscle proteins and enzymes within the blood include myoglobin (Mb), creatine kinase (CK), myosin heavy chain fragments (MHC) and skeletal troponin-1 (sTn1) [9-12]; the latter two being contractile proteins which spill into circulation when the sarcoplasm (cell membrane) is disrupted as a result of mechanical damage. Along with these changes, there is often tissue swelling and the

sensation of pain [10, 11]. With CK being found in abundance in the skeletal muscle myoplasm, it is the most commonly used marker of muscle damage within the plasma [13]. The increased blood levels of this, along with other muscle proteins and enzymes is a result of membrane leakage due to damage of muscle plasma membranes during mechanically induced stress (eccentric activity) [14, 15]. Evidence of focal myofibrillar damage (z-line streaming and/or disturbances) can be observed under the electron microscope immediately following eccentric-contraction induced injury (figure 1), with the damage increasing for up to 72 hours post-injury [5, 16].

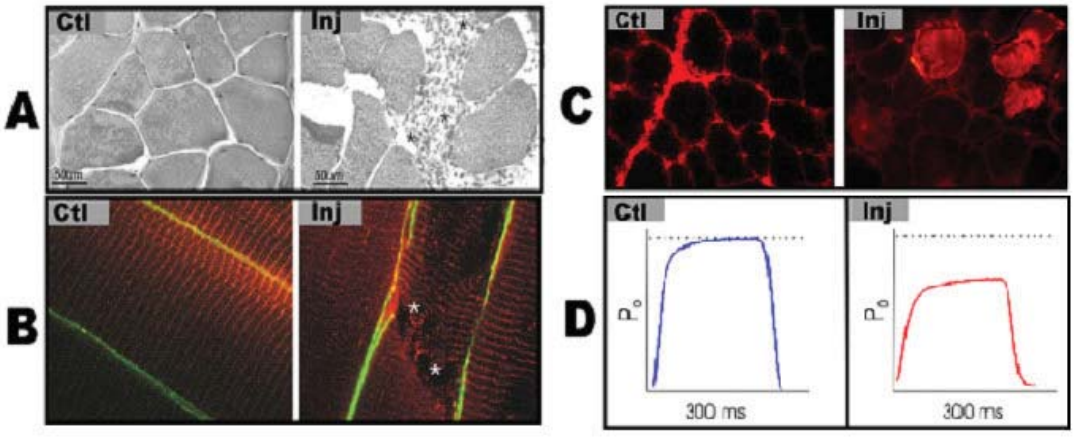

Figure 1 (adapted from Lovering & MacMillan et al. [1]) Difference in skeletal muscle structure and function between control (undamaged) and injured (damaged) 24-hr post eccentric exercise. A: cross-section of tibialis anterior (TA) tissue showing disruption of tissue and infiltration of inflammatory cells. B: longitudinal sections of TA showing change in dystrophin (green) at sarcolemma and disruption of desmin (red). C: indication of membrane damage with injured TA allowing Evans Blue Dye into intracellular space. D: maximal tetanic isometric tension of TA muscle before and after eccentric exercise.

5.2.2 Symptoms of Eccentric Muscle Damage

It has been suggested that DOMS causes decreased voluntary muscle strength following eccentric muscle damage. In humans, it has been concluded that measurements of maximal voluntary contraction torque along with range of motion best quantifies muscle damage [17]. Certain studies have applied electrical stimulation (causing involuntary contraction) to eccentrically damaged muscle fibres and have found that the ability of the muscle to perform work when under these conditions does not change significantly compared to those that are not eccentrically damaged [18, 19]. It therefore appears that the pain experienced in DOMS is the factor most limiting voluntary force output.

5.3 Mechanisms

Over the years, numerous proposed theories have tried to explain reasons behind eccentric induced muscle damage and the subsequent development of DOMS. These theories include the accumulation of lactic acid (a by-product of anaerobic metabolism generated from pyruvate), muscle spasms, muscle damage (z-band disruption and excitation-contraction coupling damage), connective tissue damage and inflammation [20]. While certain theories have very little, if any, evidence for causing muscle damage and DOMS (directly or indirectly), others have shown positive relationships, and continue to be investigated.

5.3.1 'Popping Sarcomere' Theory

A common belief is that because fewer muscle fibres are activated during eccentric contraction compared to that of concentric contraction of the same tension, the high force generated by each activated muscle fibre results in damage to the muscle cell's interior structure and to the surrounding connective tissue [9]. The lengthening of the

fibres under tension can result in 'popping' the weakest sarcomere in a chain-like sequence; consequently negatively affecting the excitation-coupling mechanism in these fibres [21].

5.4 Recovery from Eccentric-Exercise Induced Muscle Damage

The natural course of events that take place in order for full muscle recovery to occur can vary between individuals due to factors such as biological sex [22-24], training status [25], and prior exposure to both damaging [26] and non-damaging [27] eccentric activity. However, studies have shown that various indices of muscle damage including force reduction (maximal voluntary contraction, MVC), changes in range of motion and swelling, are generally resolved between 10 days and 2 weeks post-eccentric damage [18, 28]. Muscle performance (MVC) is the primary indicator of recovery [29] and Clarkson et al [28] have found that muscle recovers approximately 85% of its baseline MVC by day 10, and regains almost full recovery by 2 weeks.

5.5 Alcohol and the Sporting World

5.5.1 DOMS and sport-associated Alcohol consumption

There is little available literature specifically looking into any effects that acute moderate alcohol consumption may have on muscle performance recovery following competitive team games. What *has* been well documented is evidence of DOMS developing within 48-hours following such games, indicating skeletal muscle damage. Binge-drinking within team sports appears to be on the increase, with players often celebrating a win or sharing a loss over excessive alcohol consumption. As a result of excessive alcohol intake, performance over the following days can be significantly affected due to insufficient glycogen replenishment and inadequate rehydration.

While research into whether there are gender differences in the metabolism of alcohol and its effects on performance recovery is conflicting and very limited there is evidence that suggests that there appears to be a similar drinking attitude within women players to that of men. An article by Howe [30] outlined that post-game binge drinking in elite women's rugby is very much apparent, and may be a result of having 'equal gaming rights' to that of men. Supporting this, Carr and colleagues [31] found no significant gender differences between the frequency and amount of alcohol consumption of athletes. If metabolism varies between genders it could well impact upon muscle force recovery rates in females compared to that of males.

5.5.2 New Zealand guidelines for Alcohol consumption

The Alcohol Advisory Council (ALAC) of New Zealand primarily aims to regulate and minimise alcohol consumption and abuse by monitoring the advertising, sale, and use of alcohol within communities. While the definition of a standard drink differs between countries, within New Zealand, ALAC defines a 'standard drink' as containing 10 grams of pure alcohol (ethanol). This is equal to approximately one 330ml can of beer, one 100ml glass of table wine, or 30ml straight spirits [32].

New Zealand drinking guidelines suggest males should drink no more than 6 standard drinks, and females no more than 4, in any one drinking session. Anything above this number is generally referred to as 'binge drinking', and can have any number of harmful behavioural outcomes. Over a week, no more than 21 standard drinks for males, and 14 for females is recommended. Research suggests athletes [33-36] consume far greater volumes than recommended in any one setting, therefore putting themselves at risk of alcohol related incidents and injuries.

The legal driving limit in New Zealand is set at a level presumably low enough to not affect judgment and response timing, with cognitive impairment beginning at a

relatively 'low' blood alcohol level (figure 2). It is however, interesting to note cases where participants who would be perceived as intoxicated, remain below the legal driving limit; stimulating cause for concern [37]. The rate of alcohol absorption is dependent on factors such as weight, gender, and whether food has been consumed. It is therefore difficult to estimate an average number of standard drinks that would exceed the limit. If it takes a small amount of alcohol to affect cognitive function one would assume that the binge drinking undertaken post sporting events would not only impair the action of proper post-game nutrition and injury prevention/recovery, but could significantly impact upon other areas of the body; namely muscle.

0.02 - 0.03	Few obvious effects; slight intensification of mood.
0.05 - 0.06	Feeling of warmth, relaxation, mild sedation; exaggeration of emotion and behavior; slight decrease in reaction time and in fine-muscle coordination; impaired judgment about continued drinking.
0.07 - 0.09	More noticeable speech impairment and disturbance of balance; impaired motor coordination, hearing and vision; feeling of elation or depression; increased confidence; may not recognize impairment.
0.11 - 0.12	Coordination and balance becoming difficult; distinct impairment of mental faculties and judgment.
0.14 - 0.15	Major impairment of mental and physical control; slurred speech, blurred vision, and lack of motor skills; needs medical evaluation.
0.20	Loss of motor control; must have assistance moving about; mental confusion; needs medical assistance.
0.30	Severe intoxication; minimum conscious control of mind and body; needs hospitalization.
0.40	Unconsciousness; coma; needs hospitalization

Figure 2 Blood alcohol levels, BAC, (in percentage) and the corresponding effects on behaviour [2]

5.5.3 Sport Advertising and Alcohol

Largely contributing to the high alcohol consumption within sporting communities, is the sponsorship and advertising of sport organizations, events, and teams by beer and liquor brewing companies.

A study into alcohol advertising and sport found that during 444 hours of sport programs randomly sampled from network television from 1990 to 1992, a total of 685 alcohol commercials were broadcast, averaging out at 1.5 alcohol commercials per hour of sports programming [38]. Approximately 15% of these commercials used celebrity endorsers (athletes or entertainers). In 2003, the American alcohol industry invested over $540 million in close to 90,000 alcohol advertisements [39] With 60% of all television alcohol advertising in the U.S being screened during sporting events, it is little wonder that the 93% of people between 8 and 17 years who view sports on TV are at a higher risk of being influenced by sporting drinking culture [40]. A 2004 study [41] looked at the impact of televised alcohol commercials on alcohol intake in adolescents. A significant increased risk of excess beer consumption was found as a result of exposure to such advertisements.

In many western cultures, alcohol is used as a measure of manliness [42]; and to drink less, or not be able to 'hold one's liquor' represents a low standing in male society [43]. In fact, New Zealand's beer slogan 'Lion Red – what it means to be a man' summarizes the advertising of masculinity and national pride through beer promotion [44]. Slater and colleagues [45] found the link of masculinity with sports extremely effective; their US study of male teenagers showing consistent preference to televised beer advertisements with sports content compared to those without. There is evidence however that suggests while men appear to be painted as primary binge drinking

offenders in the sporting scene, there appears to be a similar drinking attitude within women players. An article by Howe [30] outlined that post-game binge drinking in elite women's rugby is very much apparent, and may be a result of having 'equal gaming rights' to that of men. Supporting this, Carr and colleagues [31] found no significant gender differences between the frequency and amount of alcohol consumption of athletes

5.5.4 Alcohol and General Athletes

Research suggests that binge drinking is significantly more prevalent in athletes compared to non-athletes, as a result of the social ties of the after-game drinking culture [46-49]. Alcohol consumption by athletes has shown the incidence of sport related injury to be at 54.8% compared with 23.5% in non-drinkers [34]. Dietary surveys regarding alcohol consumption in athletes can often be misleading and skewed; under-reporting is common, and mean alcohol intake does not clearly present between those who abstain from drinking, and those who binge drink on a regular basis [50]. Furthermore, in using averages, such surveys do not distinguish between those who drink throughout competition and those who abstain during training, but drink copious amounts post-competition [51].

On the other hand, several dietary studies comparing different groups of athletes have reported significant differences in alcohol consumption; which is likely due to the nature of the sport and environment in which it is undertaken [52].

5.5.5 Alcohol in Team sports

The results of several dietary studies comparing different groups of athletes have reported that those involved in team sports have a significantly greater mean daily

alcohol intake compared with those involved in individual sports, or those not involved in sport [35, 53, 54].

An early study looking into the alcohol intakes of 45 professional football players from the national Australian Rules Football League leading team found that alcohol intake was confined to weekends, and in particular to after the weekly football match [55]. Alcohol intake immediately after the match averaged at 120g, being the equivalent of 12 standard drinks according to ALAC (10g = 1 standard drink). On a separate occasion, blood tests taken at a 9:00am training session the morning following a weekend match found fourteen of the same 45 players still registered a positive blood alcohol content (BAC)* from the drinking session the night before.

* BAC is determined by the rate of alcohol absorption from the gastrointestinal tract into the bloodstream [56]

5.6 General Effects of Alcohol

5.6.1 Alcohol Absorption

When alcohol is consumed, around 20% is absorbed in the stomach, with the remaining 80% being absorbed in the upper small intestine [57]. Alcohol absorbed in the small intestine passes into the portal vein, and is transported directly to the liver.

Several factors influence the rate of alcohol absorption such as body mass, whether food is present in the stomach at time of consumption (and what type of food) and the amount and type of beverage being consumed [58]. The human body has the ability to clear one standard drink per hour from the blood [59]

5.6.2 Alcohol metabolism (ethanol pharmacokinetics)

Primary oxidation of ethanol occurs in the liver:

Oxidation begins with the rate limiting step of alcohol dehydrogenase (ADH) converting ethanol to acetaldehyde [60, 61].

$CH_3CH_2OH + NAD^+$ --------- $CH_3CHO + NADH + H^+$

Acetaldehyde is further oxidised to acetate by aldehyde dehydrogenase (ALDH)

$CH_3CHO + NAD^+ + H_2O$ --------- $CH_3COOH + NADH + H^+$

This acetate is converted to acetyl-CoA by acetyl-CoA synthase, which is oxidised in the tricarboxylic acid (TCA) cycle. Oxidation of acetyl-CoA produces by-products which are further used in the liver and adipose tissue to synthesise triacylglycerol and fatty acids [62]

$CH_3COO^- + ATP^1 + CoASH$ ---------- $CH_3COSCoA + AMP^{2-} + PP_1^{2-}$

Figure 3 The breakdown of ethanol into acetate within the liver.

A larger acute intake of ethanol results in a higher BAC and therefore enhanced ethanol oxidation within the liver. Because the rate limiting step of ethanol metabolism is the oxidation of AcH, a large acute intake means a greater level of AcH and therefore a significantly larger effect on the nervous system.

5.6.3 Alcohol and the Nervous Systems

1) Central Nervous System

The Central Nervous System (CNS) consists of the brain and the spinal cord; the nerves from which innervate the peripheral nervous system (PNS). As part of the CNS, the brain, which is divided into three basic units (forebrain, midbrain, and hindbrain) is responsible for effective signalling between the mental 'thought' and the subsequent physical 'action' – whether conscious or subconscious.

Even at moderate levels, consumption of alcohol can significantly affect the functioning of certain areas of the CNS. There is evidence of impaired cognition (divided attention, reaction time, memory, and information processing) along with psychomotor performance (eye-brain-hand coordination and body sway) at levels well below 0.05 percent [63]. In fact, it appears that more complex cognitive and psychomotor skills are affected to a greater extent under the influence of moderate alcohol consumption, than are simpler tasks [64]

2) Peripheral Nervous System (PNS)

Acetaldehyde (AcH) is thought to play an important role in the different actions of alcohol with elevated levels causing alcohol sensitivity. Such sensitivity results in vasodilation associated with increased skin temperature, increased heart and respiration rate, decreased blood pressure, headaches, nausea, a sensation of dry throat or mouth and often euphoric feelings [65].

5.7 Alcohol Effects on Muscle

Studies looking at the effect of alcohol on isolated human and rodent muscle cells in-vitro, have found that both acute and chronic alcohol consumption inhibits calcium (Ca^{2+}) transients into the myocyte by inhibiting sarcolemmal Ca^{2+} channel actions. This subsequently impairs excitation-contraction coupling, therefore decreasing strength output [66, 67]. However clinical human trials have failed to support in-vitro evidence. There is also conflicting evidence of alcohol consumption compromising sarcolemmal integrity; a rise in plasma CK post alcohol ingestion and exercise being evident under certain protocols [68] but not in others [69, 70].

5.7.1 Indirect Muscle Damage

1) Alcohol and Reactive Oxygen Species

Alcohol appears to play a secondary role in muscle damage, with ethanol metabolism producing Reactive Oxygen Species (ROS), exacerbating damage to eccentrically worked muscle. ROS are oxygen-containing free radicals which are highly unstable due to an unpaired electron. They are therefore extremely reactive, seeking to achieve a stable state by stealing, sharing, or interacting with hydrogens of lipid membranes, proteins and DNA; damaging them in the process [71]. On top of ethanol breakdown within the liver, consumption of alcohol contributes significantly to the ROS levels within the body in several ways: through interaction and stimulation of other enzymes within the body, by altering the levels of certain metals in the body, and by reducing antioxidants and their ability to combat free radicals [71]. The resulting oxidative state of the cell can expedite cell damage.

2) Alcohol and body metal alterations

Iron and copper, which are naturally occurring metals within the body, can participate in the formation of ROS by 'donating' an electron to hydrogen peroxide, producing a hydroxyl radical. These metals are then regenerated for further availability to other hydrogen peroxide molecules. There is evidence that chronic alcohol consumption can increase the level of free iron in the body both directly; by drinking iron-rich beverages such as red wine, and indirectly, by enhancing iron absorption from food [72, 73]

3) Alcohol and reduced antioxidants

Among the antioxidants that are found within the body's natural antioxidant system, Glutathione (GSH) is thought to be the most important antioxidant present within cells. GSH is a cofactor for glutathione transferase; an enzyme which helps remove certain drugs, chemicals and other reactive molecules in the cell. Its powerful ability to interact directly with and detoxify the hydroxyl radical, helps reduce the level of cellular damage [71].

Alcohol has been shown to deplete levels of GSH, especially within the mitochondria, by interfering with the carrier protein which imports GSH from the cytosol to the mitochondria. Consequently, the ROS generated during respiration within the mitochondria cannot be eliminated [74]. The vast amount of mitochondria found within the liver means generated ROS have a significant damaging effect on liver cells. However, studies have also shown that both acute and chronic alcohol consumption have a significant damaging effect on muscle cells, as they require a reasonable amount of mitochondrion for energy synthesis [75]. Alcohol has also been shown to interfere with other antioxidants such as vitamins C and E [76].

5.7.2 Direct Muscle Damage

The suggestion of a more direct effect of binge drinking on exacerbating soft tissue injury adds to the concern of drinking post-games. Post-game alcohol consumption can have a significant influence upon recovery from soft tissue injury suffered directly from exercise, due to accidents, or as result of tackling and collision involved in certain team contact sports. The standard practice of R.I.C.E (rest, ice, compression, elevation) for soft tissue injury is challenged by the effects of alcohol. Being a potent vasodilator of cutaneous blood vessels, a large acute intake of alcohol has the potential to increase swelling around the injury, possibly impairing repair processes [50].

5.7.3 Muscle Recovery

While many studies have found significant declines in muscle force output post eccentric exercise [77, 78] very little research has looked into the effect of post-game alcohol consumption on muscle force recovery following eccentric exercise. A very recent study looked into muscle performance recovery in eleven males who, after completing 300 eccentric contractions of the quadriceps muscle, consumed either 1g/kg bodyweight ethanol (alcohol group, ALC) or orange-juice (control group, CON) in a post-match style scenario [79].On a separate occasion, participants carried out the same protocol on the other leg, swapping treatment groups. Results of the cross-over study found that CK concentrations and muscle soreness ratings did not significantly differ between treatment groups; however peak strength loss was significantly greater in the ALC group post-36h. Such results indicate that even moderate consumption of alcohol following eccentric based exercise enhances loss of dynamic and static strength, negatively impacting upon performance. Further study found that under the same protocol, a lower dose of alcohol per kg of body weight (0.5g/kg) appeared to have no significant effect on muscle force loss post eccentric

exercise; indicating a dose-dependent effect on post-eccentric exercise muscle force recovery [80].

5.7.4 Alcohol on Recovery – A nutritional perspective

Alcohol consumption during post-game drinking sessions tends to substitute adequate CHO and fluid replacement, compromising recovery and subsequent performance.

Sport nutrition guidelines recommend the consumption of 100g carbohydrate (CHO) within 2 hours of finishing a game and a total consumption of 10g CHO per kg body weight over 24 hours in order to replace depleted muscle glycogen stores [81]. It is also crucial to keep hydrated during, and rehydrate immediately after a game to avoid the effects that dehydration has on the body both physically and mentally.

1) Dehydration

Dehydration of team sport players before, during, and post-game is a common problem. Research has suggested that a mere 2% drop in body mass due to water loss significantly impacts upon sporting performance at both a physical and mental level [82, 83]. During exercise, working muscles cause core body temperature to rise, and the resulting heat is dissipated through conduction, convection, radiation to the environment, and/or through the evaporation of sweat [84]. Added to this is an increase in respiration; all resulting in body water loss. Without adequate hydration, the body is unable to dissipate heat via the sweating mechanism. The subsequent continual rise in core temperature as well as an imbalance of water to electrolytes within the blood, can have severe consequences [85]. Sweating is the primary source of water loss, along with essential electrolytes such as potassium and sodium. Though sweat losses in team sport differ according to the amount and intensity of exercise actually performed throughout a game, length of game, and environment; average fluid deficits post-game suggest a significant concern for performance [82]. Team players appear to often begin

a game inadequately hydrated as a result of having a practice session prior to the game, or due to improper rehydration from the previous day's training [86]. Voluntary fluid intake during a game does not sufficiently replace water loss and many studies have found negative fluid imbalance in players from pre-match to post-match [87-90]. Added to this pre- and during-game hydration imbalance, is the concern that players do not fully rehydrate post-game. The recommended replacement of 1.5 times sweat lost via post-match fluid ingestion (often with added electrolytes) is rarely voluntarily met, impacting upon sporting performance over subsequent days [91].

The suggestion of alcohol being a strong diuretic has been extensively studied, dating back as far as the early 1930's. It has been proposed that alcohol may inhibit the release of vasopressin, a water-reabsorbing hormone, from the posterior pituitary gland, therefore enhancing water loss within the kidneys [92, 93]. Eggleton [94] found that despite administering the same volume of liquid to participants over several trials, increasing alcohol content in the given volumes resulted in significantly higher urinary output. It has however, been argued that because alcohol is ingested in such large quantities by team players post-game, that rather than further dehydrating players, it may in fact bring about faster, and a much larger level of rehydration than would voluntary intake of water or sports-drink [95]. Sheirreffs and Maughan [95] found that participants who drank a 1-2% alcoholic beverage post-exercise in a volume equal to 1.5 times the amount of sweat lost, showed no significant difference in urine output compared to those consuming an alcohol-free beverage in the same volume. Only when the alcohol content went above 2%, was there an obvious increase in urine output. While these results can be useful for team players who enjoy 'light' alcoholic

beverages, majority of those who drink post-match consume beverages of and above 5%.

2) Glycogen Stores

Glycogen levels are depleted post-match due to the high requirement by muscle to convert into ATP and therefore energy. Alcohol intake can replace proper food intake, compromising replacement of muscle glycogen thus affecting energy supplied to the muscle and consequently (indirectly) influencing performance. However, the combined effect of a large acute intake of alcohol and damaged muscle may have a far more severe effect on muscle recovery and subsequent performance.

Possible effects of acute alcohol consumption on skeletal muscle glycogen stores have been studied using various protocols. The few human studies carried out have investigated the possible effect of alcohol on muscle glycogen stores under various exercise types and duration, timing and dosage of alcohol, training status and number of participants, and the time period of muscle analysis. Similarly, rat studies have also used a range of variables and environments to test this theory. Analysis of muscle samples post-exercise in human studies have found that while there is no apparent direct effect of alcohol on glycogen stores, an indirect effect of alcohol seems to exist as a result of displacing CHO intake from optimal recovery nutrition practices[96]. Rodent studies tend to use intravenous infusion rather than oral administration therefore it can be hard to relate findings to a sporting context in humans. Many rodent studies have reported an apparent impairment of alcohol on insulin signalling and/or the glucose transport system, therefore negatively impacting upon glucose uptake and muscle glycogen stores [97-102]. It could be suggested therefore that in humans, the combined effect of inadequate CHO ingestion and

alcohol consumption risks the sufficient replacement of depleted muscle glycogen levels; affecting energy levels and thus sporting performance.

5.8 Gender differences

5.8.1 Alcohol metabolism and effects

With hormonal, metabolic and physical make-up differences between females and males, there is a possibility of a gender-based effect on the interaction between post EIMD alcohol consumption and muscle force recovery. Clarkson and Reichsman [103] found no significant difference between strength output in the bicep of 10 females in the days following EIMD alcohol consumption compared to that of a non-alcoholic beverage. While dose may be a determining factor (0.8g/kg body mass), this study also did not account for possible menstrual cycle effects (trials being separated by 10 days), and the lower number of eccentric contractions performed by a smaller muscle group may have produced unreliable results.

1) Alcohol Metabolizing Enzyme differences

It appears that the same absolute oral dose of alcohol affects women to a greater extent than men for a number of reasons. While studies have shown no difference in *peak* blood alcohol levels, blood alcohol levels in women tend to persist longer in women than in men [104-106]. Lower activity of alcohol dehydrogenase (ADH) within the stomach increases the bioavailability of ethanol [104] while a higher percentage of body fat (therefore decreased body water) compared to that of men results in decreased volume of ethanol distribution, contributing to higher blood alcohol levels [107]. Not contributing to a higher blood alcohol level within women, is the suggestion by some studies of a enhanced rate of ethanol oxidation in the liver, resulting in the generation

of AcH, possibly aggravating alcohol toxicity [108] along with a slower alcohol gastric emptying rate.

2) Body composition and Liver Volume

Alcohol elimination rate (AER) is expressed as the concentration of alcohol cleared from the blood per unit of time. Kwo and collegues [109] proposed that aspects of ethanol pharmacokinetics that may result in higher alcohol sensitivity in women, is due less to a difference in the AER of the liver and more to do with a lower lean body mass to fat ratio and therefore ethanol absorption seen in women. This lower ethanol absorption is a result of alcohols' dispersal in body water; hence a lower body water content (higher body fat content), a higher BAC [110]. They found that both genders between the tested ages of 22-30 years had livers of nearly equal volume and similar AERs (g/h), however lean body mass was 42% greater in men. Similar findings have been reported by Li and Beard et al [111]. It has been suggested that this lower lean body mass in women give a higher liver volume per lean body mass compared with men, results in a greater clearance of alcohol per unit of lean body mass, which supports the idea of a greater generation of acetaldehyde. Relating to this a study by Kwo and collegues.[109] found that as a result of men having the 42 percent greater lean body mass, women had a 33% higher mean AER and a 38% higher liver volume per kg lean body mass.

3) Effects on the CNS

Under moderate alcohol consumption, intoxicated women appear to be significantly more impaired than men on delayed recall [112] while also responding significantly more slowly on cognitive decision tasks [113] Women have also shown slower short term memory function recovery than men [105]. Higher alcohol doses show this gender

difference to be more apparent, while lower doses (BAC of 0.03) show no gender differences in cognitive impairment [114].

5.8.2 The Menstrual Cycle - Is it to blame?

1) The Natural Cycle

For the average woman, the menstrual cycle is between 21-28 days in length, with ovulation (release of an ovum) occurring on average at day 14. Ovulation separates the two primary menstrual phases; the follicular and luteal phase. On average, the follicular phase runs from day 1 to ovulation (day 14) while the luteal phase runs from ovulation (day 14) to day 1 of the next menstrual cycle. These two phases involve varying levels of the sex steroid hormones oestrogen, progesterone follicular stimulating hormone (FSH) and luteinising hormone (LH); release of which is controlled primarily by the hypothalamus and pituitary gland.

During the follicular phase, a rise in FSH causes maturation of several ovarian follicles, maturation of the ovum, and signals the ovaries to start producing oestrogen. The rise in oestrogen stimulates a surge in LH levels around day 14 of the cycle, causing an ovarian follicle to burst and the release of the most mature ovum into one of the fallopian tubes (ovulation). The luteal phase that follows ovulation, involves the formation of the corpus luteum from the ruptured follicle as a result of LH levels. The corpus leuteum in turn produces progesterone, and the combined effect of oestrogen and progesterone stimulates the generation of a thick layer of blood vessels by the endometrium to support a fertilized egg should pregnancy come about. Without fertilization, the corpus luteum deteriorates, causing a drop in progesterone and oestrogen levels and the onset of menstruation.

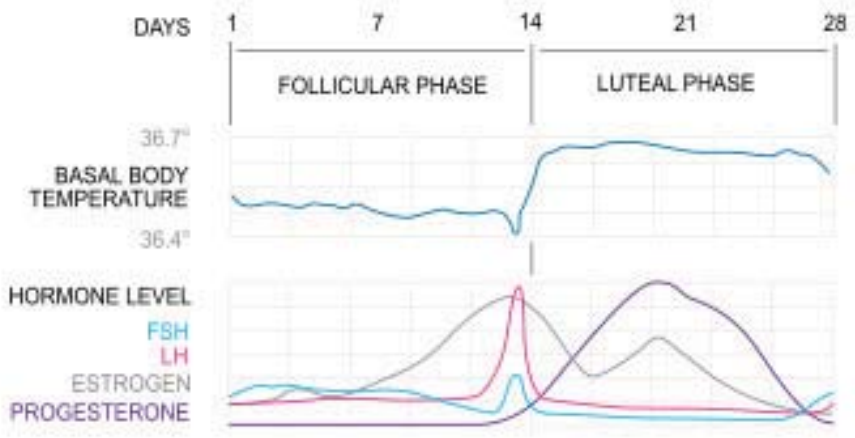

Figure 4 Phases of the menstrual cycle and changes in hormone levels.

There is conflicting evidence of a possible hormonal effect on alcohol metabolism in women as a result of changes in levels of sex-steroid hormones. Some studies have observed an oestrogen-associated hepatic AcH elevation in woman when alcohol is consumed in the ovulatory and luteal phase of the menstrual cycle [108]. A review by Lammers and Mainzer et al. [115], found that out of the 11 reviewed studies, only 2 were methodologically valid. Both of these studies found the only affected parameter of alcohol metabolism to be the elimination rate which averaged 14% higher in the luteal phase [116, 117]. This may explain the observed rise in hepatic AcH, and the suggested accompanying heightened alcohol sensitivity [108]. Similar effects have been observed when synthetic oestrogens are administered prior to alcohol consumption in animals [118, 119] and in humans [120]. Such elevations in AcH influence alcohol sensitivity in women compared to that of men, thus affecting the level of CNS impairment and subsequent behaviour.

2) Oestrogen on muscle damage and recovery

Oestrogen has been implicated as a possible factor in preventing exercise-induced muscle damage. This hormone may protect tissues from oxidative stress damage by increasing membrane stability via direct interaction with membrane phospholipids [121, 122]

a) Heat Shock Proteins

In rodent studies it has been observed that oestrogen attenuates specific heat shock proteins (HSP) expression as well as HSP70 messenger RNA synthesis in skeletal muscle following exercise [123, 124]. While present in cells under normal conditions, HSP's are rapidly synthesized in various tissues including skeletal muscle in response to stress, including exercise induced muscle damage. HSP's play a role in stabilizing proteins and are involved in the folding of denatured proteins. Under stress, such as that caused by muscle damage, the process of forming structures becomes difficult for proteins; therefore increased expression on HSP's can help deal to this problem. A lower HSP expression in females suggests that oestrogen may elicit a protective effect on muscle damage following exercise.

b) Muscle Structure Dysfunction

While a limited number of animal studies have looked at oestrogen as an attenuating influence in muscle structure disruption, one animal study showed that for up to 96h post downhill running, male rodents generally experienced earlier histopathological indices of damage to muscle structural proteins (actin, myosin and desmin) to a greater extent as well as a greater degree of muscle fibre swelling than in females [125]. Similarly, Amelink et al. [126] found a greater disruption of histochemically determined muscle banding patterns in males versus female rats 48h post exercise. These muscle banding patterns refer to the structural layout and interaction of mysosin and actin

(muscle contractile proteins) within muscle filaments. These two proteins form structural lines called z-bands, which are noticeably disrupted post muscle damage.

c) Muscle Force Reduction

Moran and colleagues [127] found that in mice, oestradiol (form of oestrogen) status had no effect on the degree of soleus muscle isometric and eccentric tension/torque force loss immediately after eccentric exercise in mice whose ovaries had been removed. However they did find a positive influence of oestrogen on maximum isometric muscle forces. Long-term force loss and recovery was not measured. A human study carried out by Sayers and Clarkson [78] involving 50 maximal eccentric contractions of the elbow flexors found when testing a large number females and males (94 and 48 respectively), a disproportionally higher number of females than males demonstrated initially large force losses, yet force recovery in these same female participants was more rapid than in males who showed similar initial force losses. The researchers suggested that the enhanced force recovery observed in these females could be due to a gender mediated attenuation of the inflammatory response via oestrogen and its antioxidant function [125]. In contrast, Rinard and Clarkson [128] found no significant difference in force reduction or recovery between women (n = 83) and men (n = 82) following 70 maximal eccentric contractions of the elbow flexors ($p > 0.05$). However, this protocol only looked at isometric tension. It must be noted that while these two similar protocols [78, 128] reported contrasting results, neither controlled for menstrual cycle in females.

d) Markers of Muscle Damage

A series of rodent based studies have demonstrated a direct effect of oestrogen on decreasing leakage of CK directly from skeletal muscle tissue post eccentric exercise induced muscle damage [129]. An earlier study [130] reported female rats had significantly

lower levels of circulating CK activity than males rats following the same running exercise. Furthermore when male rats were supplemented with oestrogen prior to exercise, there was an attenuation of post-exercise circulating CK activity similar to that shown in normal female rats. More recent studies have also shown significantly diminished post-exercise or post-injury elevations in serum CK activities [131-133].

A separate study carried out by Salimena et al. [134] suggested that oestrogen was at least partially responsible for lower levels of muscle damage indices found in female mice compared to that of male mice. With CK being a main indication of muscle damage, this further suggests the protective effect that oestrogen may play on preventing the same extent of muscle damage seen in males under the same eccentric protocol.

While these study results are yet to be confirmed in the human population, the majority of rodent studies have shown that following exercise, females present lower levels and fewer indicators of muscle damage than males.

Further research needs to be carried out to examine the potential of oestrogen to diminish exercise induced structural muscle damage and resulting force reduction and recovery in humans.

3) Oral Contraceptives

Oral contraceptives (OCs) work to suppress the natural monthly hormonal cycle. Studies investigating the effect of OC use on BAC in women have brought forward varying results. Jones and Jones [135, 136] Zeiner and Kegg [137] all found a significantly lower BAC and slower alcohol elimination rates in women taking OCs. In contrast, Cole-Harding and Wilson [138], Hay and Colleagues [139], Jeavons and Zeiner [120], and

Niaura et al. [105] reported no effect on BAC levels or alcohol elimination rate in those taking OCs.

4) Effects on the CNS

The few studies that have looked into the effect of moderate consumption of alcohol on the CNS have revealed that in general, neither menstrual cycle stage nor use of synthetic oestrogens appear to influence psychomotor or cognitive performance in intoxicated women compared with men [105, 112, 140-142].

Gender differences in the effect of alcohol on muscle damage recovery has of yet not been done. However, recent studies on this topic in males, has opened the door to this exciting new area of research.

6. Hypothesis

That moderate alcohol consumption following exercise-induced eccentric muscle damage in females would negatively influence (increase length of time to) skeletal muscle force recovery.

7. Methods

7.1 Subjects

Eight healthy females (mean age 23 years; 65.2 ± 15kg; 164 ± 5.5cm) were recruited via word-of-mouth and poster advertising to participate in this study. Strict criteria were established and made known during recruitment: 1) participants had to have been on the combined oral contraceptive pill for a minimum of 3 months (and had to remain on it for the duration of the study), 2) participants had to be habitual consumers of alcohol, and 3) participants must have been between the ages of 18-30 and be engaged in physical activity regularly. Information sheets were given to those interested. All participants were screened using a Health Screening Questionnaire to rule out those who were at risk physically, culturally, or religiously in following the protocol. Those who passed the Questionnaire were asked to give written consent. Approval for this study was granted by the Massy University Human Ethics Committee (09/73).

7.2 Experimental Protocol

7.2.1 Pre-testing procedures

The week preceding the first trial, subjects attended a familiarization session in which they carried out the required movements that were to be used for performance testing on the Biodex isokinetic dynamometer (Biodex Medical Systems Inc., NY). Appropriate seat positions were determined using recommendations made by the manufacturer (Biodex Medical Systems Inc., 2004) and were recorded for subsequent use throughout the study. Menstrual cycle was also recorded in order to test the subjects during the luteal phase (day 14 until day 1 of next period) of each trial. It has been suggested that hormone fluctuations in females may be a causal factor in

performance and exercise capacity, therefore testing participants in the same phase controlled for this [143].

Participants were asked to abstain from any form of exercise apart from necessary walking and from alcohol consumption 48h prior to, and until 60h post trial. Two trials were separated by at least a month, dependent on the individual's menstrual cycle. A month was deemed long enough as this would account for fluctuating hormone levels. Also, previous studies have used a washout period of one month (or less) and have considered it valid since the testing of different legs (right or left) in each trial avoids the issue of muscle adaptations to eccentric work [79, 80, 144]. Participants were randomised into trials; however it was difficult to blind trials due to the nature of the intervention.

On the day of the trial, participants were required to consume a 'Cookie Time' One-Square Meal and 'Sanitarium' Up & Go (flavour, vanilla) as a standardized meal (3700kJ; CHO 121.2 g, fat 27.3 g, protein 25.7 g, fat), 4h prior to testing. Participants were asked to attend the laboratory in the evening, where two lots of 4 ml bloods were withdrawn by venepuncture. If undergoing the alcohol trial, they were then required to take two pregnancy tests to rule out any chance of pregnancy and the subsequent risk of alcohol consumption on the fetus [145, 146]. Upon observation of two negative results, participants were asked to complete a muscle soreness scale of the leg to be tested by a simple step and squat test. They were then asked to complete a 5-minute warm up on the Monark cycle ergometer before pre-exercise performance testing was carried out. This involved 5 MVC each of isometric, concentric and eccentric contractions of the quadriceps femoris muscle while seated on the isokinetic dynamometer. Performance measures for both legs were taken in order to use one leg

as a control. Once completed, participants performed an eccentric bout of exercise that involved 300 maximal eccentric repetitions using the quadriceps femoris muscle to elicit muscle damage. Once the eccentric bout was completed, participants completed another soreness scale and were allowed to consume an Up & Go and muesli bar (Mother Earth Baked Oaty Slices). This standardized meal (1452kJ; CHO 50.4 g, fat 11.4 g, protein 11.5 g) helped avoid a sharp peak in BAC once drinking had commenced, while also preventing hunger as participants had to fast overnight. Thirty minutes post-exercise participants consumed 6 equal amounts of either an alcohol (ALC) beverage containing 0.88g of alcohol per kg of body weight as vodka in orange juice or a non-alcoholic (OJ) beverage of orange juice mixed with glucose powder and water. Equal amounts of orange juice were consumed in both trials; however glucose powder and water were added to the OJ beverage to match energy content and volume of ALC beverage respectively. Drinks were consumed over a 90 minute period; one drink every 15 minutes. Twenty minutes after the final beverage was consumed, participants were asked to rate another soreness scale, and those on the alcohol trial were required to give 3 breath tests using a digital alcohol tester (Digitech Professional Fuel Cell Alcohol Tester, QM-7300) to determine BAC. They were then free to leave the laboratory and were either driven home by a 'buddy' or the primary researcher.

Participants returned to the laboratory the following three mornings (12h, 36h, and 60h post-exercise) for follow up blood samples, performance tests (36h & 60h), ratings of muscle soreness, and a standardized breakfast (Up & Go and muesli bar).

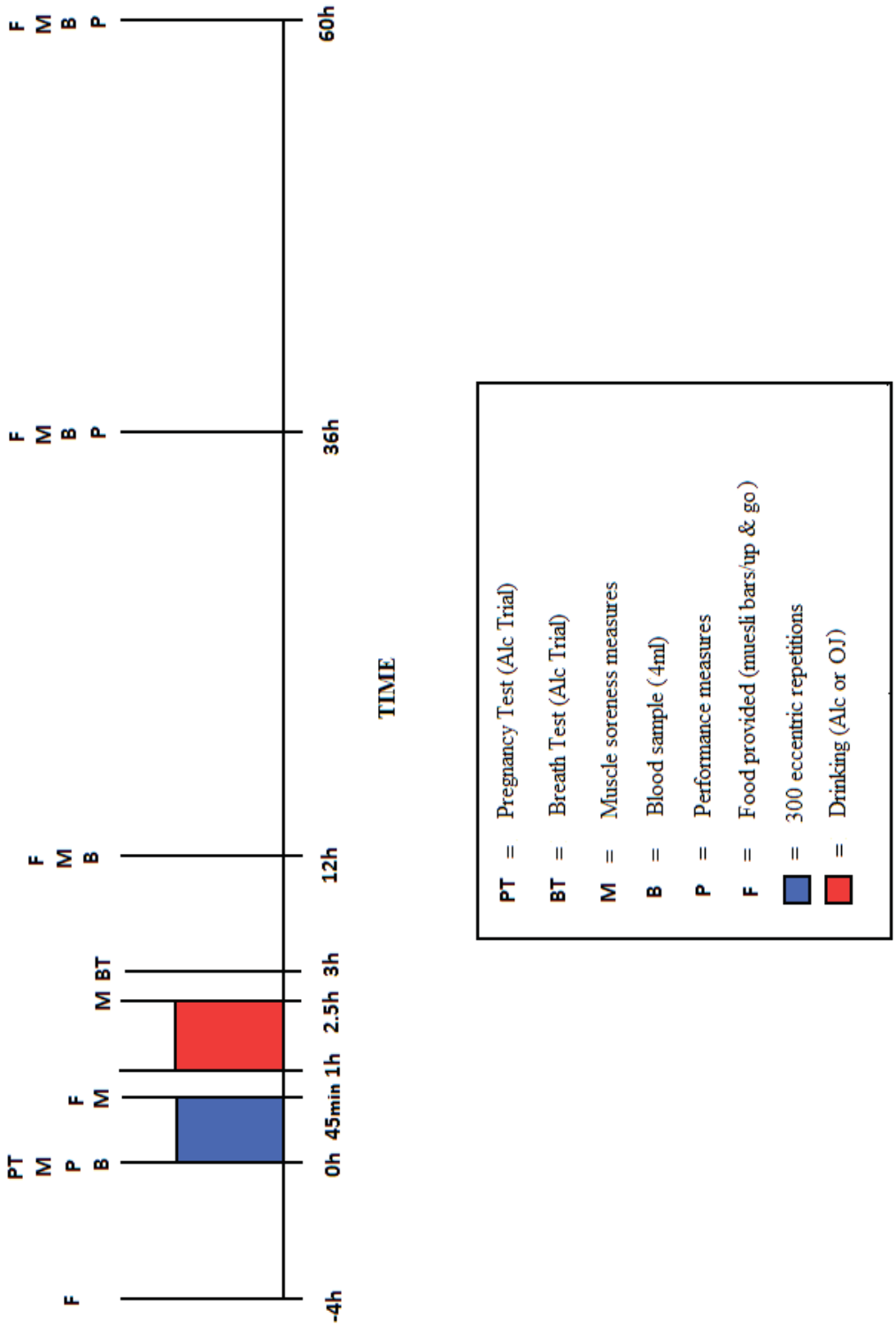

Figure 5 Timeline of testing procedure

7.2.2 Blood sampling

A venous blood sample was obtained from the antecubital vein into a 4 mL red vacutainer tube containing clotting factors (micronized silica particles). The tube was left at room temperature for 5-10 minutes before being centrifuged (Eppendorf, Hamburg) at 4ºC for 10 minutes at 2000 rpm. Plasma was removed, aspirated into three 300µL aliquots and frozen at -80ºC for later analysis. These aliquots were analysed for creatine kinase (marker of muscle damage).

7.2.3 Performance tests

Participants were required to complete a 5-minute warm-up on a bicycle ergometer (Monark, Varberg, Sweden) at 100W prior to all performance tests. Upon completion, the participant was seated on the isokinetic dynamometer at the previously recorded seat adjustments so that the femoral epicondyle was aligned with the dynamometers axis of rotation and the ankle strap positioned 5cm proximal to the medial malleolus. Along with the ankle, straps were placed around the chest, hips and the leg to be tested in order to isolate the quadriceps femoris muscle. Range of motion of the leg was set at 60º for concentric and eccentric contractions, and at 75º for isometric contractions, which allowed the weight of the leg to be determined. The subject then performed 5 maximal contractions of each type with each set separated by two minutes of passive recovery. Concentric and eccentric torque was measured at an angular velocity of 30º/s [147]. Absolute peak torque/tension (PT); the peak torque out of the 5 contractions and average peak torque/tension (APT); the average peak torques taken from the 5 contractions were recorded. This was then repeated on the other leg as a control.

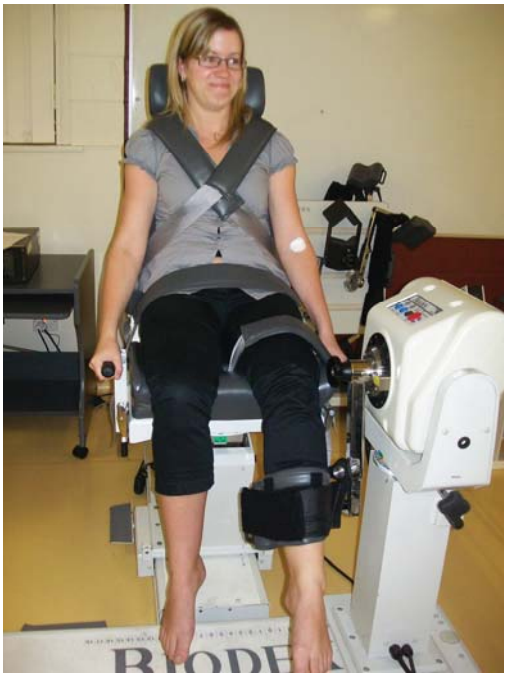

Figure 6 Participant completing 5 maximal eccentric repetitions of the quadriceps muscle on the Biodex isokinetic dynamometer. The same eccentric motion was carried out for the damage protocol

7.2.4 Muscle soreness

Perceived muscle soreness ratings were recorded on two subjective scales (stepping and squatting) from 0 to 10 (0 = no soreness, 10 = excruciatingly painful) prior to exercise-induced eccentric damage, straight after damage, and after the 90 minute drinking period. Perceived soreness was also recorded over the three mornings following damage (12h, 36h and 60h post). For the step test, participants were asked to step up (concentric muscle action) onto a 40cm box then step down (eccentric muscular contraction) and the soreness in doing so was rated. For the squatting test, participants were asked to do one full squat at a controlled pace and rate their perceived soreness in the tested leg. The six individual ratings (for the six separate occasions) were presented on six separate pieces of paper, each containing two scales;

one for stepping and one for squatting. The separate pieces of paper ensured that there was no comparison between time points and all values were subjective.

7.2.5 Eccentric-exercise muscle damage

The range of motion was set at 60º from maximal knee flexion (0º) to 60º extension (using the dynamometers inbuilt goniometer), with repetitions being performed at an angular velocity of 30º/s; a range and speed proven to effectively bring about a high level of muscle damage and subsequent soreness [148].

Participants performed 3 sets of 100 eccentric repetitions of the quadriceps femoris muscle. Each set was separated by 5 minutes of passive recovery during which time participants remained seated on the dynamometer and were allowed water ad lib. During the sets participants were encouraged to exert maximal effort through the full range of motion, resisting the downward pull of the dynamometer arm. Their produced torque was displayed on the computer screen to which they had full visual access during the duration of the exercise. It has been suggested that visual feedback enhances effort during protocols such as this [149].

7.2.6 Intervention

Thirty minutes post-exercise participants consumed 6 equal amounts of either an alcohol (ALC) beverage containing 0.88g of alcohol per kg of body weight as vodka (37.5% alcohol/volume; Smirnoff, Australia) in orange juice (Citrus Tree; Frucor, New Zealand) or a non-alcohol (OJ) beverage of orange juice mixed with glucose powder (King) and water. The amount of 0.88g was based on previous studies done in males at 1g of alcohol per kg body weight [79] and was adjusted for lean body mass differences in females. The total amount of vodka consumed in the ALC trial averaged at 151 ± 13.9ml, equivalent to 5 ± 0.4 standard drinks. Equal amounts of

orange juice were consumed in both trials; however glucose powder (60 ± 14.7 g) and water (151.5 ± 36.8 ml) were added to the OJ beverage to match energy content and volume of ALC beverage respectively.

7.3 Biochemical analysis

7.3.1 Creatine kinase

Analysis of the muscle damage marker creatine kinase (CK) was carried out at MedLab Central, Palmerston North, by enzymatic method using reverse reaction. This 'reverse reaction' method photometrically measures the rate of NADPH formation as a final product of the last of three reactions, to quantify CK activity [150].

7.4 Statistical Analyses

Data were analysed using Statistical Analysis Software (SAS) 9.2 for Windows (version 6.0.6001). Using a repeated measures analysis of variance (ANOVA), comparison between conditions (alcohol and control) over time for each measure (independent variable) were determined, providing levels of significance for Trial effect, Treatment effect, and interaction effect between Treatment and Trial. Where significance permitted, post-hoc tests were performed to identify significant differences at each time point. Represented values are means ± or standard error (used to estimate the true population mean) for $n = 10$ at a 95% significance level ($p = 0.05$). The Holm-Bonferroni method (multiple comparison test) was carried out on all significant p-value in a step-wise manner to determine true significance.

Peasons Product Moment Correlation Coefficient's were determined using Microsoft Excel (Version 2003) for Windows. This allowed us to investigate any relationships

between certain variables (i.e. ratings of muscle soreness with muscle performance measures) by giving an r-value between 0.0 and 1.00 (or -0.0 and -1.00).

8. Results

8.1 Performance measures

8.1.1 Isometric tension

The completion of 300 eccentric muscular contractions of the quadriceps muscle appears to have no effect on peak ($p = 0.2829$) or average ($p = 0.1938$) isometric tension over time in either trial (figures 7, 8). Furthermore, no trial effects (difference between trials) exist for either peak isometric tension (PT) or average peak isometric tension (APT) with $p = 0.1938$ and $p = 0.5283$ respectively. There was however, a significant interaction (time x trial) effect in PT ($p = 0.0041$) but not APT ($p = 0.0935$) suggesting that the consumption of alcohol after eccentric exercise results in a greater PT reduction compared to the consumption of a control beverage.

A significant decrease in the ALC trial at 36h in PT from pre-values of 30 ± 9% ($p = 0.0041$) was followed by significant recovery to 13 ± 4% ($p < 0.0001$) at 60h. Comparatively a decrement of 5 ± 9% ($p = 0.6$) from pre-values was seen in the OJ trial with less recovery than ALC residing -2 ± 6% below initial values at 60h.

As with PT, APT decrement of 33 ± 9% between pre-values and 36h was significantly different in the ALC ($p = 0.0022$) but not in the OJ ($p = 0.5443$) trial (-2 ± 10%). Significance was also seen in the increase in APT to 16 ± 4% from 36h to 60h in the ALC trial ($p < 0.0001$). Values did not significantly improve in the OJ trial.

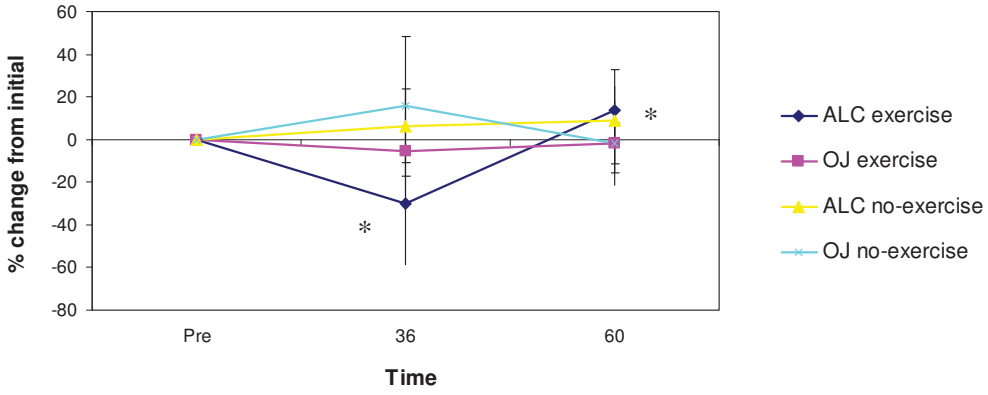

Figure 7 Mean peak isometric tension changes from pre-exercise measures (%) at 36h and 60h post-eccentric exercise (±SD). No significant differences occur in values over time (p = 0.2829), nor between trials (p = 0.1938). An interaction between time and treatment exists with p = 0.0041 at 36h.

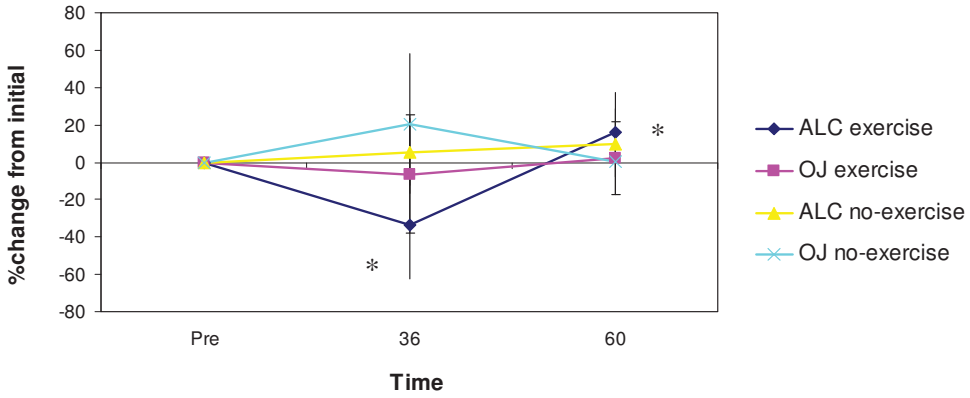

Figure 8 Mean average peak isometric tension changes from pre-exercise measures (%) at 36h and 60h post-exercise (±SD). No significant differences occur in values over time (p = 0.2094), between trials (p = 0.5283), nor is there any interaction between time and treatment (p = 0.0935).

NB: * on figures marks significance between time points
For all results, p-values stated come from comparison of both non-exercised and exercised legs in the ALC and OJ trial.

8.1.2 Concentric torque

Unlike isometric PT, there appeared to be a significant time and interaction effect on concentric PT ($p = 0.0107$, $p = 0.0306$). There was no evident treatment effect ($p = 0.2761$). PT in the ALC trial significantly decreased by 33 ± 7% from pre- to 36h ($p = 0.0012$) and remained significant from 36h to 60h with recovery to 9 ± 3% ($p < 0.0001$). No significant changes were seen in the OJ trial with a 13 ± 11% drop in torque at 36h followed by an increase to 6 ± 11% at 60h ($p > 0.05$ for both).

Similarly, concentric APT showed both a time ($p = 0.0007$) and interaction ($p = 0.0181$) effect, yet a p-value of 0.3029 showed no treatment effect. A significant decrease in torque for ALC by 30 ± 10% at 36h ($p = 0.001$) recovered to 15 ± 5% by 60h ($p > 0.0001$). As with PT, no significance was found in from pre- to 36h (14 ± 9%, $p > 0.05$) or from 36h to 60h (2 ± 7%, $p > 0.05$).

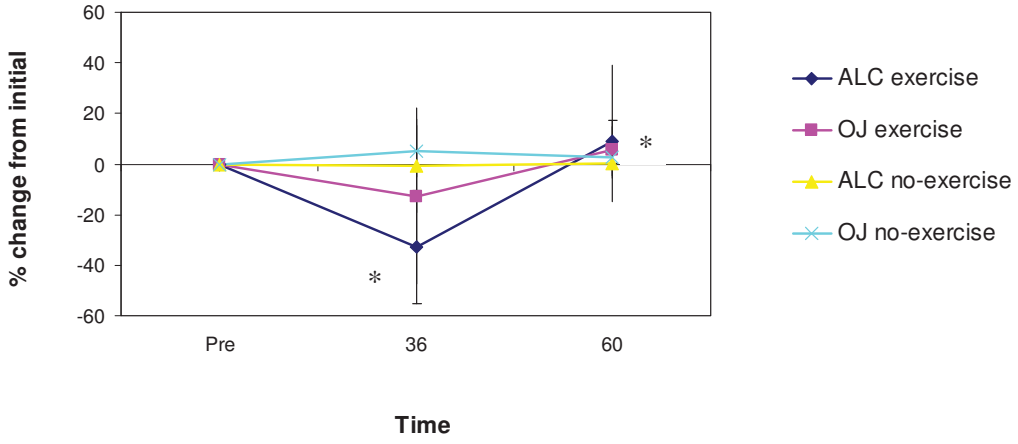

Figure 9 Mean peak concentric torque changes from pre-measures (%) at 36h and 60h post-exercise (±SD). Significant differences in torque values occur over time (p = 0.0107) and the interaction of time*treatment (p = 0.0306), however no treatment effect is evident (p = 0.2761).

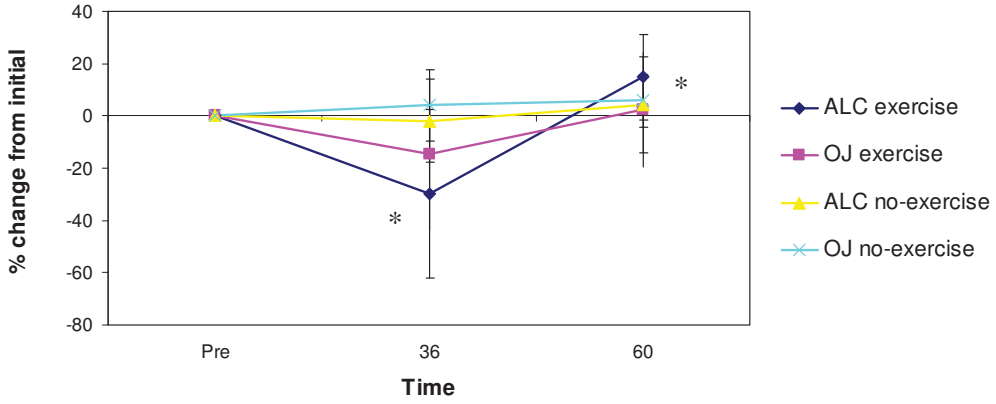

Figure 10 Mean average peak concentric torque changes from pre-measures (%) at 36h and 60h post-exercise (±SD). Significant differences in torque values occur over time (p = 0.0007) and the interaction of time*treatment (p = 0.0181), however no treatment effect is evident (p = 0.3029).

NB: * on figures marks significance between time points
For all results, p-values stated come from comparison of both non-exercised and exercised legs in the ALC and OJ trial.

8.1.3 Eccentric torque

Unlike concentric, but similar to isometric, no significant time ($p = 0.2318$) or treatment ($p = 0.3425$) effects were observed for eccentric PT (figure 11). There was however, a significant interaction effect of time*treatment on PT ($p = 0.0007$). A significant decline in ALC PT was seen at 36h (26 ± 8%) followed by recovery to 12 ± 5% at 60h ($p = 0.0025$ and <0.0001 respectively). Once again, no significant differences were noted between any time points within the OJ trial; 12 ± 5% decrement at 36h and a return to 1 ± 7% at 60h ($p > 0.05$ for both).

In the same way, eccentric APT showed no time ($p = 0.2757$) or treatment ($p = 0.2240$) effects but had a significant interaction effect ($p = 0.0011$). Significant torque decrease at 36h in the ALC trial (26 ± 8%) gave a p-value of 0.0029, while the increase at 60h to 12 ± 5% gave a significance of $p < 0.0001$. As with PT, no significance was found in from pre- to 36h (9 ± 7%, $p > 0.05$) or from 36h to 60h (1 ± 5%, $p > 0.05$) in the OJ trial.

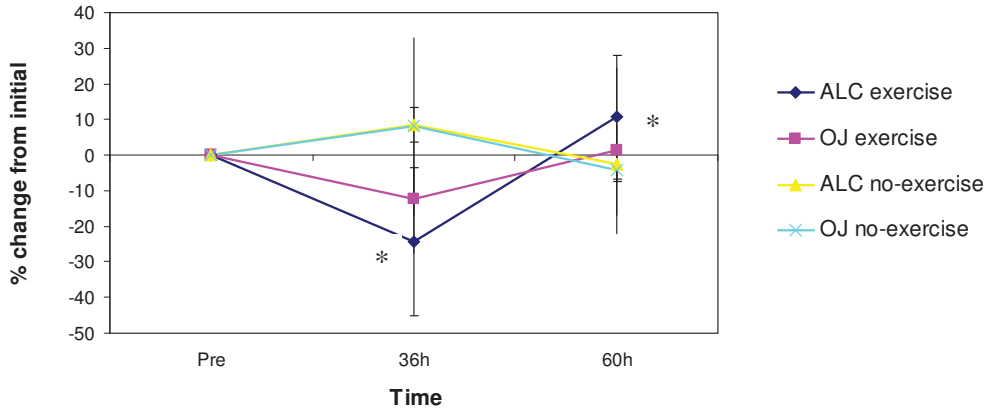

Figure 11 Mean peak eccentric torque changes from pre-measures (%) at 36h and 60h post-exercise (±SD). Significant differences do not exist in torque values over time (p = 0.2318) nor is any treatment effect evident (p = 0.3425). There is however a significant interaction of time*treatment (p = 0.0007).

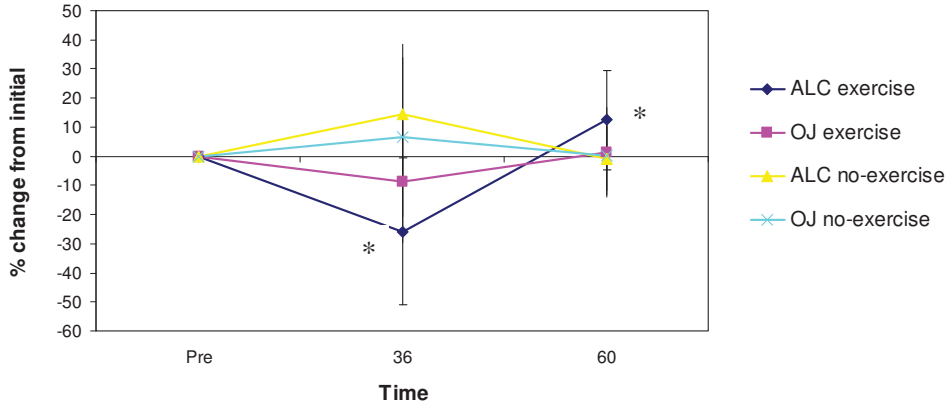

Figure 12 Mean average peak eccentric torque changes from pre-measures (%) at 36h and 60h post-exercise (±SD). Significant differences do not exist in torque values over time (p = 0.2757) nor is any treatment effect evident (p = 0.2240). There is however a significant interaction of time*treatment (p = 0.0011).

NB: * on figures marks significance between time points
For all results, p-values stated come from comparison of both non-exercised and exercised legs in the ALC and OJ trial.

8.2 Blood markers

Creatine Kinase

Creatine kinase activity significantly increased from pre-exercise values over time ($p = 0.0217$). Differences between 36h and 60h post in the OJ group were significant ($p = 0.0414$), however no other within treatment significances existed in either OJ or ALC groups. No significant differences were observed in CK concentration between trials ($p = 0.8444$) nor was there a significant interaction between time and treatment ($p = 0.9951$). No correlation was found to exist between serum CK activity and ratings of muscle soreness of muscular performance.

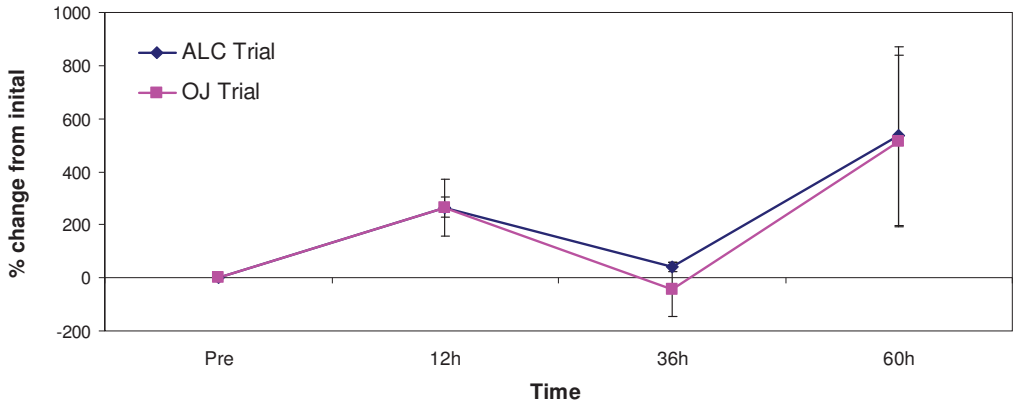

Figure 13 Mean blood CK changes (%) from pre-measures (U/L) at 12h, 36h and 60h post-exercise (±SD). Significant difference was seen in blood levels over time ($p = 0.0217$) however no treatment effect was evident ($p = 0.8444$). Furthermore, no significant interaction (time*treatment) effect exists ($p = 0.9951$).

8.3 Muscle soreness

While ratings of perceived muscle soreness (RPS) significantly differed between participants (stepping $p = 0.0007$; squatting $p < 0.0001$) in the ALC and OJ groups, there was no significant interaction effect between treatment groups and time (stepping $p = 0.2298$; squatting $p = 0.4797$) No correlation was found to exist between ratings of soreness and performance measures.

Stepping

Post 12h, 36h and 60h RPS values differed significantly from pre-values in the ALC group ($p = 0.0077$, < 0.0001, 0.0003 respectively) with similar findings in the OJ group ($p = 0.0008$, <0.0001 and 0.0057 respectively). Comparatively, post eccentric-exercise and post-drinking values were significantly different from pre-values in the OJ group ($p = 0.0003$, 0.0014) but not in the ALC group. Also within the ALC group, significant differences were seen between post-exercise and post alcohol consumption values at both 36h ($p = 0.0014$, 0.0023) and 60h ($p = 0.0216$, 0.0319) and between 12h and 36h ($p = 0.0428$).

Between trials, soreness values were showed significant difference post-exercise ($p = 0.0098$) and following drinking ($p = 0.0483$).

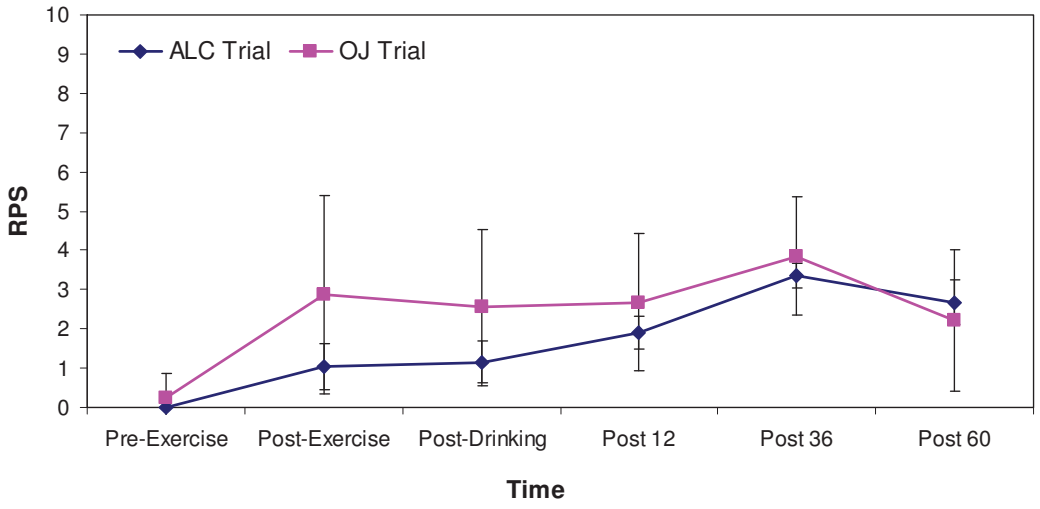

Figure 14 Mean ratings of perceived soreness while stepping (± SD), pre- and post-exercise, post-drinking and 12h, 36h and 60h post-exercise.

Squatting

As with stepping, post 12h, 36h and 60h RPS values differed significantly from pre values in the ALC group ($p = 0.0027$, <0.0001, 0.0014 respectively) with similar findings in the OJ group ($p = 0.0001$, <0.0001 and 0.0138 respectively). Values in the ALC group significantly differed from post-exercise to 36h ($p=0.0005$), post drinking to 36h ($p = 0.002$) and 12h to 36h ($p = 0.0218$); however unlike stepping, significant findings were not evident between these time points and 60h. Significance was found between 36h and 60h ($p = 0.0375$) values. Within the OJ group, differences between post-exercise to 36h and from 36h to 60h were found to be significant ($p = 0.0072$, 0.0008).

Between trials, only at the post-drinking time point was a significant difference observed ($p = 0.0477$).

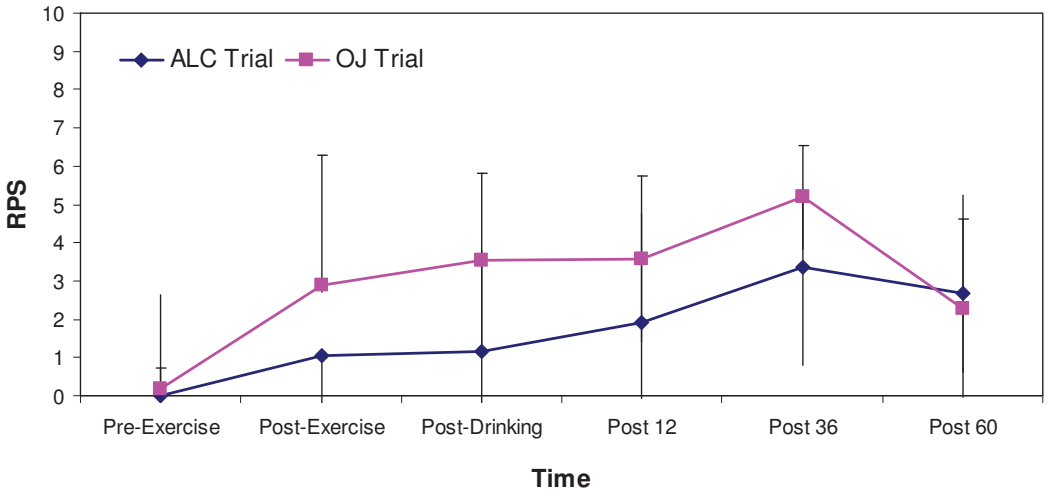

Figure 15 Mean ratings of perceived soreness while squatting (± SD), pre- and post-exercise, post-drinking and 12h, 36h and 60h post-exercise.

9. Discussion

9.1 Muscle Performance

Previous investigation into the effects of alcohol consumption post eccentric exercise induced damage have found a dose-dependent reduction of muscle force recovery in males. Up until now, no studies have looked at this same effect in females. The primary aim of this study therefore, was to determine whether a similar effect of alcohol ingestion (the same relative amount as males) on muscle force reduction and time to recovery post eccentric exercise would be seen in females.

Both legs (control and exercised) were tested for performance measures in the ALC and OJ trials to account for any learning effect over the trials. No significant changes in muscle force output were evident within the control legs of both trials.

Significant interaction effects for peak and average peak isometric, concentric and eccentric tension/torque suggests that alcohol consumption adds to the normally observed decline and recovery rate of muscle force output post eccentric exercise.

The greatest loss in peak and average torque/tension following 300 eccentric contractions of the quadriceps were observed at 36h from pre values in the ALC trial. In contrast, no significance was observed in peak and average strength loss in the OJ trial at these same time points. In fact strength loss values appeared to be unusually low for a protocol that was designed to produce maximal muscle damage. These findings contrast those that have looked at force output after eccentric exercise without addition of an intervention such as alcohol. Clarkson and Hubal [77] and Sayers and Clarkson [78] both found significant force decline following eccentric protocols that

used a less severe protocol. We would therefore assume a protocol involving a larger number of eccentric contractions would elicit at least the same (if not more) level of damage. Similar eccentric protocols such as that carried out by Barnes and Mundel et al. [79] elicited sufficient muscle damage to see significant force reduction in concentric torque (28%) eccentric torque (19%) and isometric tension (12%) following exercise completion in the placebo trial. Our study tends to contrast these findings. It is therefore likely that participants failed to exert enough voluntary effort for maximal damage to occur.

It may be suggested that a lack of significance in force reduction and recovery relating to muscle damage has an underlying explanation. Based on interaction significance, we could conclude that alcohol does negatively affect force output post eccentric exercise regardless of the level of damage. Maximal or not, any muscle damage resulting from eccentric activity results in decreased voluntary muscle force output and alcohol consumption to the level of 0.88 g per kg body weight in females appears to increase force reduction 36h post compared to no alcohol consumption. As investigated by Barnes and Mundel et al. [80], the effect of alcohol on exacerbating damage post eccentric exercise appears to be dose-dependent in males. Whether this also exists for females requires further investigation.

Gender differences in alcohol pharmacokinetics and resulting effects on the body, along with possible hormonal influences on muscle damage and recovery may account for the outcome of this study. While few human studies have looked at how oestrogen may or may not have a protective effect on muscle damage post eccentric exercise, research into this area using rodents has shown a positive influence of

oestrogen on lower indices of muscle damage, reduced muscle structural damage and force output immediately following exercise. The suggested effect of oestrogen on increasing membrane stability and therefore protecting tissues from oxidative stress damage (an indirect effect of alcohol) [121, 122] could be used to explain why our results showed non-significant changes in force output in the OJ trial. However oxidative stress only contributes a certain amount to muscle damage, with majority of eccentric damage occurring as a direct effect of load on muscle fibres. Studies carried out by Barnes et al. [79, 80] looking at similar protocol in men have showed significant time effects across all contraction types indicating the occurrence of muscle damage resulting from the eccentric protocol. Other studies have also given significant time effects, therefore if equal and maximal effort was put into our study as with previous studies, the apparent low level of 'damage' suggests some kind of protective mechanism in women against eccentric damage; whether it be oestrogen, or something yet unknown.

Significant interaction effects for isometric, concentric and eccentric tension/torque indicate that despite a low level of muscular damage achieved, alcohol still negatively affects force reduction post eccentric exercise. These results are in agreement with Barnes [144] and Barnes and Mundel [79] who found the combined effect of alcohol and eccentric exercise to have a greater muscle force reduction in the days following, compared to that of eccentric exercise alone.

9.2 Blood markers

Creatine kinase, an enzyme found in abundance within skeletal muscle tissue, is commonly used as the prime indicator that muscle damage has occurred due to its rapid leakage out of ruptured skeletal muscle cells. Studies that have brought about a significant level of muscle damage have all reported elevated serum CK activity up until around 72h post eccentric exercise. CK activity significantly increased from pre-exercise values over time with the only within treatment significance being seen in the OJ group between 36h and 60h. No significant difference existed between treatments nor was there any time x treatment effects. This suggests CK is not the best variable to determine the level of muscle damage achieved. Also worth noting is the high level of serum CK recorded prior to eccentric exercise in one participant This participant had engaged in eccentric activity 40h prior to testing which may have contributed to higher pre-exercise levels. The higher initial levels may have affected averages and therefore significant differences between time points.

It could be speculated that lack of within treatment time significances are a result of the debate of oestrogen on attenuating markers of muscle damage.

The problem remains however, that no credible human studies have yet directly investigated the influence of oestrogen or oestrogen supplementation on serum CK levels or muscle membrane disruption post exercise. However it has been seen that in human females, *resting* CK levels inversely correlate with oestrogen levels[151]. In relation to our study, the majority of research that uses running and eccentric quadriceps muscle exercises has shown decreased serum CK elevations post-exercise in females compared to that of males [152, 153] while other studies appear not to show such gender differences [154].

9.3 Muscle soreness

Ratings of muscle soreness increased significantly from pre-exercise values when stepping up and squatting in both trials. Figures 14 and 15 show an apparent greater overall perceived soreness within the OJ group compared with that of the ALC group up until 60h. It has been hypothesised that the products of alcohol metabolism have similar effects as opiates on certain brain receptors; with the resulting blocking of pain and induction of euphoria [155]. This may explain the levels of perceived pain in the ALC group, with the ongoing lower levels possibly being a result of mental comparison to previously measured pain. Average perceived ratings did not exceed a value of 6 therefore suggesting moderate levels of pain. It was evident that participants found it difficult to distinguish from soreness and weakness particularly at the post-exercise and post-alcohol consumption time points. This may have limited accuracy of ratings. Appropriate measures were taken to ensure ratings at each time point were subjective rather than comparative between time points.

While within treatment soreness significance was found when stepping and squatting between time points of both treatment groups, less significance was found between treatment groups. Only at post-exercise and post-drinking (squatting) was perceived soreness significantly different between those who consumed alcohol and those who did not. Post-drinking significant differences between groups supports the research behind the idea of a temporary effect of acute alcohol consumption on pain; however this same theory cannot explain post-exercise/pre-alcohol consumption significance. A possible 'placebo' effect may have occurred with a few participants verbally stating they would find it easier on the alcohol trial. Such a mindset at pre and post-exercise time points may have influenced perceived ratings.

No correlation was evident between ratings of perceived muscle soreness and performance measures suggesting this method of soreness rating is poorly correlated to muscle function.

9.4 Limitations of Study

As with all research, limitations during data collection can greatly affect the outcome of results.

The greatest limiting factor in this study was the low number of participants being tested (n = 8). The greater the sample size, the more accurate results become in terms of significance.

Testing on women required a month between trials to eliminate possible hormonal effects. While exercise and alcohol intake was limited and/or standardized 48h prior to and during the trials, what occurred between trials was uncontrollable. This included exercise levels; which could influence muscle strength gain or loss, muscle injury or trauma, and alcohol intake. Increased or decreased levels of physical activity from one month to another may affect muscle tissue response to the level of eccentric activity undertaken in the study protocol.

Bloods were unable to be drawn from one participant due to small deeply situated veins. While effort was made initially, the participant's level of comfort and stress became the deciding factor in not proceeding with blood sampling. For this reason, the blood sampling pool dropped to n = 7. This is turn affected significance due to an even smaller sample size for statistical analysis.

Another factor affecting CK results was the undertaking of eccentric exercise by one participant 48h prior to testing. While the study requirements included the request of

no exercise 48h prior to testing, limiting exercise 72h prior may have avoided pre-levels of force output, soreness and CK activity being affected. In turn, averages would not have been skewed, and significant differences may have been observed.

For some participants, there were large discrepancies between their pre-exercise data over the two trials. Also, some showed a marked *increase* in certain performance measures post-eccentric exercise compared to pre-values. These inconsistencies may be result of poor familiarisation. While participants were told of the absolute need to give maximal effort, it is highly possible that some did not give 100% for one, both or neither of the trials.

10. Practical Application

Findings from this study have practical implications for female athletes who compete over successive days as well as the general sporting community. Results give insight into the benefits of moderating alcohol intake in female athletes during game season and particularly during heavy training times. This is predominantly so for sports involving a large component of eccentric muscular contraction (such as running downhill). Reduced performance seen in this study should encourage athletes to avoid post-game drinking during times where subsequent games or training will be affected. In situations where alcohol consumption cannot be avoided, limiting intake may prevent reduced performance.

Furthermore, results from this study suggest a possible protective effect from muscle damage in females, therefore a lower relative impact of alcohol on eccentrically damaged muscle.

11. Conclusions

Results from this study provide evidence to suggest that moderate alcohol consumption post eccentric-exercise induced muscle damage has a significant effect on the reduction and recovery of peak and average concentric torque over time, while having no significant effect on isometric or eccentric tension/torque. Our findings also show that consuming alcohol following eccentric exercise a gives rise to a greater reduction in quadriceps muscle force output 36h post-exercise compared to no alcohol consumption. Females who consume alcohol post eccentric exercise appear to have insignificant reductions in force output in the days following a contrast to that seen in similar studies carried out in males.

While a significant rise in serum CK activity from pre values reflects muscle damage, alcohol consumption does not affect CK activity compared to that of no alcohol consumption. Similarly, ratings of muscle soreness clearly mirror muscle damage; however alcohol does not have any affect on perceived pain.

Results from this study suggest that while females may not experience the same level of muscle damage as males, whether it be due to gender effects or not; alcohol still reduces muscle force output up to 36h following eccentric exercise. Therefore post-game drinking among females should be monitored to avoid training and/or performance hindrances in the days following eccentric based activity.

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Figure References

Figure 3
http://img.medscape.com/pi/emed/ckb/pediatrics_cardiac/1331339-1331349-1010220-1766148.jpg Retrieved 13th April 2011 from http://www.medscape.com/

Figure 4
http://womenshealth.about.com/cs/menstruation/a/understandmenst.htm Retrieved 23rd April 2011 from http://womenshealth.about.com/

13. Appendix I

Raw data for performance measures (n = 8)

Peak Isometric Tension (Exercised Leg)

	ALC			OJ		
Subject	**Pre**	**36h**	**60h**	**Pre**	**36h**	**60h**
1	256.8	180.2	245	177.8	210.8	207.5
2	97.1	119	123.3	243.1	191.1	195.5
3	106.6	87.2	123.3	158.3	122.2	152.4
4	158.5	136.9	145.9	168.2	114	123.9
5	177.1	76.9	95.1	120.4	128.6	116.9
6	157	92	90	120	117	111
7	266	178	217	171	252	178
8	134.8	39.1	59.3	125.3	79.8	104.3
Average	**169.2**	**113.6**	**137.3**	**160.5**	**151.9**	**148.6**
SD	**62.8**	**49.5**	**63.8**	**41.0**	**58.9**	**40.6**

Peak Isometric Tension (Control Leg)

	ALC			OJ		
Subject	**Pre**	**36h**	**60h**	**Pre**	**36h**	**60h**
1	264.4	235.2	266.7	166.4	213.4	236.5
2	127.6	146	228.4	242.3	203	218.6
3	101.4	133.4	139.4	150.6	155.9	155
4	168.4	169.8	173.9	168.4	160.7	163.3
5	186	147.2	137.7	112.4	197.7	177.2
6	167	193	175	100	150	144
7	193	196	218	233	221	201
8	137.2	164	149.1	132.4	123.1	120.6
Average	**168.1**	**173.0**	**186.0**	**163.1**	**178.1**	**177.0**
SD	**49.6**	**33.4**	**47.0**	**51.8**	**35.2**	**39.2**

Average Isometric Tension (Exercised Leg)

	ALC			OJ		
Subject	**Pre**	**36h**	**60h**	**Pre**	**36h**	**60h**
1	274.1	146.5	234.3	166.4	213.4	236.5
2	79.3	93.1	108.5	229.5	163.8	185.2
3	93.4	80.6	108.3	148.8	118.2	135.2
4	146.6	111	131.3	151.8	84	109
5	157.9	61.8	81.2	105.6	110	105.5
6	144	84	69	94	94	85
7	222	166	194	157	229	167
8	126.2	31.7	52.5	116.6	75.1	97.7
Average	**155.2**	**96.8**	**122.3**	**146.2**	**135.9**	**140.1**
SD	**64.6**	**43.6**	**62.7**	**42.6**	**59.2**	**52.1**

Average Isometric Tension (Control Leg)

	ALC			OJ		
Subject	**Pre**	**36h**	**60h**	**Pre**	**36h**	**60h**
1	255.6	220.2	249.2	147	182.7	199.1
2	109.5	120.7	199.2	210.7	187.1	195.5
3	88.1	120.4	133.8	141.6	143.4	148.5
4	159.2	166	168.9	150.3	148.5	150
5	170.8	137.4	127.7	82.6	156.2	147.9
6	155	180	156	75	125	129
7	177	172	188	213	212	188
8	129.7	145.8	135	124.2	118.6	114.2
Average	**155.6**	**157.8**	**169.7**	**143.0**	**159.1**	**159.0**
SD	**50.7**	**33.7**	**41.3**	**50.9**	**32.3**	**31.6**

Peak Concentric Torque (Exercised Leg)

	ALC			OJ		
Subject	**Pre**	**36h**	**60h**	**Pre**	**36h**	**60h**
1	189	142.5	180.9	150	131.6	150.6
2	125.6	109.3	116.9	206	176.1	175.4
3	80	79.7	92.4	132.8	110.1	122.2
4	154.5	127.6	126.8	156	101.1	124.9
5	137.7	64.6	87	92.2	86	130.6
6	168	76	69	129	71	71
7	229	146	168	159	264	158
8	111.1	41.5	49	104.7	66.6	95.7
Average	**149.3**	**98.4**	**111.2**	**141.2**	**125.8**	**128.5**
SD	**46.6**	**38.5**	**46.2**	**35.3**	**66.1**	**33.8**

Peak Concentric Torque (Control Leg)

	ALC			OJ		
Subject	**Pre**	**36h**	**60h**	**Pre**	**36h**	**60h**
1	214	169.9	190.4	161.9	181.1	178.5
2	128.9	148.9	177.6	177.9	179.9	164.5
3	90	112	124.6	128.6	146.8	145.1
4	161.9	144.1	136.9	148.8	136.6	155.5
5	159.9	163.8	145.6	117.8	131	148.9
6	166	138	145	114	135	133
7	215	176	146	213	211	229
8	122.9	146.8	122.9	112.9	103.9	103.8
Average	**157.3**	**149.9**	**148.6**	**146.8**	**153.1**	**157.2**
SD	**43.3**	**20.3**	**23.8**	**35.8**	**34.6**	**36.4**

Average Concentric Torque (Exercised Leg)

	ALC			OJ		
Subject	**Pre**	**36h**	**60h**	**Pre**	**36h**	**60h**
1	144.8	101.8	127.8	161.9	181.1	178.5
2	87.8	93.6	105.1	182.4	137.9	134.6
3	63.7	77.1	109.8	105	88.3	105
4	125.3	96	103.7	136.4	90.3	103
5	122.6	49.6	72.6	70.5	62.6	78.2
6	139	56	52	111	61	58
7	181	135	151	127	181	124
8	103.2	32.6	38.6	100.9	60.6	83.9
Average	**120.9**	**80.2**	**95.0**	**124.3**	**107.8**	**108.1**
SD	**36.2**	**33.1**	**38.0**	**35.7**	**51.8**	**37.6**

Average Concentric Torque (Control Leg)

	ALC			OJ		
Subject	**Pre**	**36h**	**60h**	**Pre**	**36h**	**60h**
1	201.1	145.1	150.3	141.2	152.8	162.5
2	109.6	114.2	148.7	178.1	160.3	145.4
3	79.2	91.3	115.6	116	135.5	133.8
4	129.3	124.1	129.6	120.1	109.9	130.3
5	120.5	121.2	104.9	88.4	109.2	122.3
6	131	128	130	98	113	115
7	159	128	123	169	161	191
8	114.6	135.5	113.4	106.2	97.1	94.3
Average	**130.5**	**123.4**	**126.9**	**127.1**	**129.8**	**136.8**
SD	**36.2**	**15.9**	**16.2**	**32.7**	**25.7**	**29.7**

Peak Eccentric Torque (Exercised Leg)

	ALC			OJ		
Subject	**Pre**	**36h**	**60h**	**Pre**	**36h**	**60h**
1	309.5	241	276.6	194.8	172.3	232.6
2	237.7	231.1	303.6	307.3	244.6	228.3
3	149.7	154.9	192.6	185.3	163.5	157.7
4	185.2	172.1	188.1	182.5	106.4	136.5
5	229.6	114.8	148.5	200.7	193.6	182.6
6	214	119	65	146.6	162	102
7	317	227	291	242	237	212
8	141.1	79.5	80	135.6	112.1	149
Average	**222.9**	**167.4**	**193.1**	**199.3**	**173.9**	**175.0**
SD	**65.6**	**60.9**	**92.5**	**54.5**	**50.7**	**46.8**

Peak Eccentric Torque (Control Leg)

	ALC			OJ		
Subject	**Pre**	**36h**	**60h**	**Pre**	**36h**	**60h**
1	302.5	274.9	217.7	224.7	301.3	306
2	244.3	251	309.2	324.9	233.5	230.1
3	125.4	179	193.1	181.8	200.7	187.9
4	188.6	204.5	205.8	194.6	179.8	191.1
5	236.2	243.4	227.2	176.3	233.8	232.5
6	218	215	215	169	233	204
7	254	291	229	242	207	212
8	163.2	174.2	165	146.6	146.1	137.3
Average	**216.5**	**229.1**	**220.2**	**207.4**	**216.9**	**212.6**
SD	**55.9**	**42.9**	**41.5**	**56.3**	**45.8**	**48.1**

Average Eccentric Torque (Exercised Leg)

	ALC			OJ		
Subject	**Pre**	**36h**	**60h**	**Pre**	**36h**	**60h**
1	287.9	194.6	219.2	218.3	272	276.5
2	200.4	224.8	269.2	269.1	215.1	208.6
3	131.9	136.6	181.5	180	140.9	148.5
4	173.5	156.4	172.6	182.5	106.4	136.5
5	222.1	102.5	134.4	168	183.7	171.8
6	201	101	57	134.3	119	92
7	278	202	279	204	215	194
8	132.7	67.9	75.1	122.7	105.7	140.1
Average	**203.4**	**148.2**	**173.5**	**184.8**	**169.7**	**171**
SD	**58.6**	**55.9**	**82.1**	**46.7**	**61.2**	**56.0**

Average Eccentric Torque (Control Leg)

	ALC			OJ		
Subject	**Pre**	**36h**	**60h**	**Pre**	**36h**	**60h**
1	278.1	256.7	282.8	218.3	272	276.5
2	229.8	231.6	254.6	302.4	216.5	199.8
3	115.4	173.7	178.5	168.7	181.3	177.9
4	166.6	178	192.5	165.3	141.2	174.8
5	234.9	222.7	213.8	155.9	222.5	222.3
6	117	179	173	147	205	172
7	202	224	182	204	160	194
8	147.2	156.2	143.2	134.2	137.9	130.2
Average	**186.4**	**202.7**	**202.6**	**186.9**	**192.0**	**193.4**
SD	**59.4**	**35.4**	**46.0**	**54.4**	**45.9**	**42.8**

Raw Data for Blood Markers

	ALC				OJ			
Subject	**Pre**	**12h**	**36h**	**60h**	**Pre**	**12h**	**36h**	**60h**
1	28	89	77	133	316	104	101	83
2	70	252	204	199	74	130	118	100
3	58	169	121	105	155**	149	86	103
4*								
5	76	197	163	861	73	305	192	331
6	105	649	1457	4315	69	340	330	2327
7	88	305	171	109	88	110	80	79
8	77	276	206	208	115	335	283	898
Average	**71.7**	**276.7**	**342.7**	**847.1**	**127.1**	**210.4**	**170.0**	**560.1**
SD	**24.2**	**179.4**	**493.5**	**1552.4**	**88.8**	**110.2**	**101.2**	**833.5**

*Bloods unable to be obtained from participant due to deep veins.
** High initial reading resulting from eccentric based exercise 48 hours prior to protocol.

Ratings of Perceived Muscle Soreness

Stepping

	ALC						OJ					
Subject	**Pre-Ex**	**Post-Ex**	**Post-D**	**Post 12**	**Post 36**	**Post 60**	**Pre-Ex**	**Post-Ex**	**Post-D**	**Post 12**	**Post 36**	**Post 60**
1	0	5.5	4.9	4.8	2.4	2.6	0	7.8	2.3	4.2	2.1	1.2
2	0	0	0	1.4	2.7	0.3	0	2.9	1.5	2.2	5.4	1
3	0	0	0	2	2.6	1.3	0	0.6	1.9	2.7	6.8	3.2
4	0	0	0	0.3	2.4	2.6	0	0.8	0.9	1.2	3.4	2
5	0	1.2	1.1	1.2	4	0.9	0	0.8	0.9	0.2	2.9	1.1
6	0	1	2.3	2.1	3.9	4.8	0	5.3	6.9	5.9	3.6	6.3
7	0	0.3	0.1	1.3	3.6	3.9	1.8	2	2.1	2.3	3.5	1.1
8	0	0.3	0.8	2.2	5.2	5	0	2.9	3.9	2.7	3.1	1.8
Average	**0**	**1.0**	**1.1**	**1.9**	**3.3**	**2.7**	**0.2**	**2.9**	**2.6**	**2.7**	**3.9**	**2.2**
SD	**0**	**1.8**	**1.7**	**1.3**	**1**	**1.8**	**0.6**	**2.5**	**2**	**1.8**	**1.5**	**1.8**

Squatting

	ALC						OJ					
Subject	**Pre-Ex**	**Post-Ex**	**Post-D**	**Post 12**	**Post 36**	**Post 60**	**Pre-Ex**	**Post-Ex**	**Post-D**	**Post 12**	**Post 36**	**Post 60**
1	0	7.7	8.6	6	8.4	2.6	0	10	4.6	6.2	4.9	1.6
2	0	0.2	0	1.4	3	0.4	0	0.2	3.2	2.2	7.2	0.9
3	0	0	0.5	0.5	1.2	0.3	0	1.9	3.2	1.9	4.9	1.3
4	0	0	0	0.3	1.6	2.4	0	0.5	1.6	4.4	4.8	1.8
5	0	2.6	1	1.5	7	2.1	0	0.8	0.9	0.6	3.9	1.6
6	0	1	2.4	4	5.1	4.6	0	6	8.1	6.2	6.9	7.7
7	0	0.3	0.8	3.6	3.9	3.4	1.5	1.8	2.3	2	3.3	0.3
8	0	0.4	1.7	3.4	6.1	6.4	0	1.9	4.5	5.1	5.6	3.1
Average	**0**	**1.5**	**1.9**	**2.6**	**4.5**	**2.8**	**0.2**	**2.9**	**3.6**	**3.6**	**5.2**	**2.3**
SD	**0**	**2.6**	**2.8**	**1.9**	**2.6**	**2.0**	**0.5**	**3.4**	**2.3**	**2.2**	**1.4**	**2.3**

***** Ex = exercise, D=drinking

14. Appendix II

Results of Performance Measures Statistical Analysis (n = 8)

Significance Levels for Performance Measures Effects

	Time Effect	Treatment Effect	Interaction Effect
Isometric Peak Torque	p = 0.2829	p = 0.1938	p = 0.0041
Isometric Average Torque	p = 0.1355	p = 0.1346	p = 0.0013
Concentric Peak Torque	p = 0.0107	p = 0.2761	p = 0.0306
Concentric Average Torque	p = 0.0007	p = 0.3029	p = 0.0181
Eccentric Peak Torque	p = 0.2318	p = 0.3425	p = 0.0007
Eccentric Average Torque	p = 0.2757	p = 0.2240	p = 0.0011

Levels of significance for performance measures at time points

	% change at 36h	Significance Level	% change at 60h	Significance Level
ALC Iso PT	-30	p = 0.0041	13	p < 0.0001
ALC Iso AT	-34	p = 0.0022	16	p < 0.0001
OJ Iso PT	-5	p = 0.6000	-2	p = 0.0918
OJ Iso AT	-6	p = 0.5425	2	p = 0.4187
ALC Con PT	-33	p = 0.0012	9	p < 0.0001
ALC Con AT	-30	p = 0.0010	15	p < 0.0001
OJ Con PT	-13	p = 0.1923	6	p = 0.0596
OJ Con AT	-14	p = 0.0979	2	p = 0.0543
ALC Ecc PT	-26	p = 0.0029	12	p < 0.0001
ALC Ecc AT	-22	p = 0.0029	13	p < 0.0001
OJ Ecc PT	-12	p = 0.1171	1	p = 0.0863
OJ Ecc AT	-8	p = 0.3001	-3	p = 0.2312

Average performance changes (percentage) from pre-exercise values and level of significance of these changes from preceding time point (n = 8)

15. Appendix III

Rating Perceived Muscle Soreness

Name …….

Trial ……...

Treatment ……..

Pre-exercise post-exercise post-drinking 12h post 36h post 60h post

Stepping

0 ——————————————————————— **10**

Squatting

0 ——————————————————————— **10**

0 = No soreness

10 = Excruciatingly painful