THE EFFECTS OF ACUTE ALCOHOL CONSUMPTION ON MUSCULAR PERFORMANCE AND RECOVERY AFTER EXERCISE

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ABSTRACT

The acute consumption of alcohol after sport, particularly team sport, is common place. Indeed, sportspeople regularly consume hazardous levels of alcohol at rates significantly higher than non-sportspeople during this period. Much focus is given to behaviours that may enhance the rate of recovery after sport/exercise however little attention has been given to behaviours that may have the opposite, negative effect such as alcohol consumption. Further, while it is widely recommended that alcohol consumption be avoided if damage/injury to skeletal muscle is present little direct evidence exists to support this recommendation. As acute post-eccentric exercise alcohol consumption has previously been shown to impact the magnitude of force loss associated with such exercise, the overall purpose of this thesis was to further investigate the interaction between acute alcohol consumption and damage to skeletal muscle. Within this thesis the consumption of 1 g, but not 0.5 g, of alcohol per kg bodyweight was found to magnify the force loss typically observed in the days after laboratory based, eccentrically biased exercise. This affect was shown to be due to a combination of decreased neural drive and alterations at the muscle level in the days following the damaging event. Investigation into the effect of alcohol on recovery from an ecologically valid game simulation found that acute alcohol consumption and the stresses induced by a rugby match may interact to reduce lower body power output in the days after the match. Other sports specific performance measures, testosterone and cortisol, and markers of immunoendocrine function were unaffected by this combination. In conclusion, the findings presented in this thesis provide evidence that the consumption of alcohol, even at volumes considerably less than those regularly consumed by sportspeople, has deleterious effects on muscle function when consumed soon after strenuous eccentric exercise. This affect is not seen with a lower dose of alcohol or with modest levels of muscle damage. Important to the wider sporting community, these data support the recommendation of limiting alcohol consumption when damage/injury to skeletal muscle has occurred.

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LIST OF ABBREVIATIONS

A

ACh	Acetylcholine
ACSM	American College of Sports Medicine
ADP	Adenosine diphosphate
Ag/AgCl	Silver chloride
ATP	Adenosine triphosphate
ALAC	Alcohol Advisory Council of New Zealand
ALC	Alcohol
ANOVA	Analysis of variance

B

BAC	Blood alcohol concentration
BrAc	Breath alcohol concentration
BURST	Bath University Rugby Sprint Test
BW	Body weight

C

C°	Degrees centigrade
CAM	Cell adhesion molecule
CBF	Cerebral blood flow
CK	Creatine kinase
CL	Confidence limit
cm	Centimetre
CMJ	Counter movement jump
CNS	Central nervous system
CON	Concentric torque
CV	Coefficient of variation

D

DOMS	Delayed on set of muscle soreness

E

EC	Excitation-contraction
ECC	Eccentric torque
EIMD	Exercise induced muscle damage
EMG	Electromyography
ES	Electrical stimulation
EX	Exercising

G

g	Grams
g	Gravity
GABA	Gamma-Aminobutyric acid

H

h	Hour/s
HID	High intensity drill
HR	Heart rate
HR max	Heart rate maximum
Hz	Hertz

I

ICC	Intra-class coefficient
iEMG	Integrated electromyography
IGF-1	Insulin-like growth factor 1
IL.	Illinois
Inc.	Incorporated
ISO	Isometric tension

K

K_m	Michaelis constant
kg	Kilograms

L

L	Litre

La^-	Lactate
LFF	Low frequency fatigue

M

µm	Micrometer
µs	Micro second/s
mA	Milliamp
MEOS	Microsomal ethanol oxidising system
mmol	Millimole
MU	Motor unit
mV	Millivolt
MVIC	Maximum voluntary isometric contraction
MVC	Maximum voluntary contraction

N

n	Number of subjects
N	Newton
NAD^+	Nicotinamide adenine dinucleotide
NADH	Reduced form of nicotinamide adenine dinucleotide
NADPH	Reduced for of nicotinamide adenine dinucleotide phosphate
Nm	Newton meter
NX	Non-exercising

O

OJ	Orange juice

P

PES	Percutaneous electrical stimulation
P_i	Inorganic phosphate
PPO	Peak power output
PRE	Pre-exercise value

R

rad	Radians

RE	Running economy
RPE	Rating of perceived exertion

S

s	Second/s
SD	Standard deviation
SE	Standard error
SPSS	Statistical Package for the Social Sciences
SR	Sarcomplasmic reticulum
StD	Standard drink

T

TCA	Tricarboxylic acid
TMS	Transcranial magnetic stimulation

U

USA	United States of America

V

V	Volt
VA	Voluntary activation
VO2	Rate of oxygen consumption
VO2 max	Maximal rate of oxygen consumption
v/v	Volume/volume

W

W	Watts

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CHAPTER 1

Overview

The consumption of moderate to large volumes of alcohol by sportspeople after competition is a regular occurrence however little is known about the effect this behaviour has on physical recovery and subsequent performance. While previous research has focused on the effects of alcohol on factors that may influence recovery, there is a lack of understanding regarding how post-exercise alcohol consumption influences measures of physical performance in the days after the exercise bout.

This European style thesis, comprising a literature review and published research, addresses the current gap in the literature by investigating the effects of acute alcohol consumption on recovery after exercise, in particular exercise which is likely to bring about exercise - induced muscle damage (EIMD). For consistency, the published material presented in **Chapters 5, 6, 7 and 8** have been reformatted. This material is otherwise presented in its original published form.

The Literature Review **(Chapter 2)** broadly discusses alcohol, from its production, metabolism and use by the general population and sportspeople, to its general effects on a number of physiological parameters. More importantly, the specific effects acute alcohol consumption has on physical performance and aspects associated with recovery from the stresses of exercise are also discussed. This is followed by a section focussing on exercise - induced muscle damage. This section begins by outlining the mechanics of force production during eccentric muscular contraction and goes on to discuss the potential mechanisms behind EIMD and the implications such damage has on measures of muscular performance. The final section of the literature review focuses on the sport of rugby union. Rugby union has a strong relationship with heavy alcohol consumption and is also associated with high levels of muscle damage and musculoskeletal injury as a result of the physical nature of the sport. The physical demands of rugby union and the possible causes of match related exercise-induced muscle damage are addressed in this section.

Chapter 2 concludes with a summary of the findings of the literature review which form the basis of the specific aims and hypotheses outlined in **Chapter 3**. A brief overview of the common methods used in all studies is provided in **Chapter 4,** after which the series of studies which make up this doctoral thesis, in their published form, are presented in **Chapters 5, 6, 7 and 8.** The details provided in **Chapter 4** have been kept brief to avoid unnecessary repetition as greater detail is provided in the published manuscripts presented in **Chapters 5, 6, 7 and 8**.

Previous work by Barnes et al. (2010), carried out as part of a BSc Hons program, was the first to report that alcohol, at a dose of 1g of alcohol per kg bodyweight, consumed in the hours after strenuous eccentric exercise appears to magnify the force loss typically seen in the days after such exercise**. Chapter 5** follows on from this ground breaking study to investigate whether acute post – exercise alcohol consumption does indeed affect the force loss associated with EIMD and whether this is the result of an interaction between damaged skeletal muscle and alcohol or due to a systemic effect of alcohol on force production. The purpose of **Chapter 6** is to observe whether any such effect is dose dependent so that recommendations for "non-detrimental" levels of alcohol consumption may begin to be developed. The possible mechanisms, central or peripheral in origin, behind the observations of Barnes et al. (2010) are investigated in **Chapter 7**. Then, in the final study of this series, **Chapter 8** uses a rugby game simulation to ascertain whether alcohol impacts functional performance recovery in the days after potentially damaging match specific exercise.

The final chapter, **Chapter 9**, provides a general discussion and conclusions based on the findings of the studies presented in the experimental chapters. Additionally, this chapter includes a small section on topics for future investigation.

CHAPTER 2

Literature Review

This literature review addresses a broad range of topics specific to the aims of this thesis. The initial section (**2.1**) focuses on alcohol, from its production, metabolism and use by the general population and sportspeople, to its general physiological effects and its specific impact on factors that may influence recovery and physical performance. The second section (**2.2**) discusses eccentric EIMD, with particular attention given to possible mechanisms for and physical consequences of this phenomenon. Additionally, this section will discuss exercise - induced muscle damage as it relates specifically to the sport of rugby union.

2.1 ALCOHOL

2.1.1 Production

Ethanol is produced through the anaerobic fermentation of glucose by yeast. Firstly, glucose is converted to pyruvate via glycolysis (Reaction 1). Pyruvate, in turn, is converted to acetaldehyde by pyruvate decarboxylase (Reaction 2). Alcohol dehydrogenase then reduces acetaldehyde to produce ethanol (Reaction 3).

(1) $C_6H_{12}O_6 + 2\ ADP + 2\ P_i + 2\ NAD^+ \rightarrow 2\ CH_3COCOO^- + 2\ ATP + 2\ NADH + 2\ H_2O + 2H^+$

Various enzymes involved in glycolysis

(2) $CH_3COCOO^- + H^+ \rightarrow CH_3CHO + CO_2$

Pyruvate decarboxylase

(3) $CH_3CHO + NADH + H^+ \rightarrow CH_3CH_2OH + NAD^+$

Alcohol dehydrogenase

Three forms of alcoholic beverage are produced for consumption; beer, wine and distilled spirits, generally from different carbohydrate-containing foods. Beer is brewed via the 'malting' of various grains, with barley being the most common. Malt extract, the end product of the 'malting' process and a glucose disaccharide, is boiled and hops are added for flavouring to produce 'wort'. Yeast is then added and alcohol is produced through the fermentation, as outlined above, of the sugars present in the 'wort'. Similarly, wine is produced through the interaction between yeast and 'must', the juice and grape skins produced through crushing.

The yeast used in the brewing process dictates the alcohol content of the end product; typically the yeast used in beer production tolerates environments containing up to 5 or 6 % alcohol (v/v) while those used in the production of wine can exist with higher levels of alcohol, approximately 12 to 15 %, before they begin to die off.

Spirits, such as vodka, whiskey, rum and gin, are created through the distillation of alcoholic beverages that have been produced through the fermentation of a variety of foods, such as barley, rice and fruits. These fermented beverages usually contain 14 – 16 % alcohol, with water making up the remaining volume. As alcohol has a lower boiling point than water the two can be separated by vaporizing the alcohol at 78° C. The vapour is then condensed by cooling and is collected as concentrated liquid alcohol. The distillation process allows for the production of a range of strong spirits with alcohol content ranging from 45 – 75 %.

2.1.2 Absorption and metabolism

When an alcoholic beverage is consumed a small amount of the ethanol in the beverage is immediately metabolised in the gastric mucosa by alcohol dehydrogenase (Reaction 4). The majority of ethanol, however, is absorbed through the upper gastrointestinal tract and is quickly circulated around the body. The water solubility of ethanol allows it to diffuse into the majority of tissues, particularly the liver due to the flow of blood directly from the stomach. The rate of absorption is dependent on a number of factors including gender, body mass, the type of beverage consumed, whether the stomach is empty and the speed at which the alcoholic beverage is consumed (Paton 2005).

Most of the absorbed ethanol is metabolised in the liver by alcohol dehydrogenase resulting in the formation of acetaldehyde and NADH (the reduced form of nicotinamide adenine dinucleotide (NAD^+)) (Reaction 4). Acetaldehyde is toxic and is therefore metabolised rapidly to acetate by the mitochondrial enzyme acetaldehyde dehydrogenase, which has a high affinity for acetaldehyde (K_m = 0.2 - 1 μM) (Reaction 5). The majority of the acetate is released into the blood stream, taken up by other cells and subsequently converted to the tricarboxylic acid (TCA) cycle substrate acetyl-CoA by the enzyme acetyl-CoA synthetase (Reaction 6). Acetyl-CoA is then used in the TCA cycle where it is eventually broken down to carbon dioxide and water.

(4) $C_2H_5OH + NAD^+ \rightarrow CH_3CHO + NADH + H^+$
Alcohol dehydrogenase

(5) $CH_3CHO + NAD^+ + H_2O \rightarrow CH_3COO^- + NADH + 2\ H^+$
Acetaldehyde dehydrogenase

(6) $CH_3COO^- + ATP + CoA \rightarrow AMP + PPi + CH_3CO\text{-}CoA$
Acetyl-CoA synthetase

(7) $C_2H_5OH + NADPH + H^+ + 2O_2 \rightarrow CH_3CHO + 2\ H_2O + NADP^+$
MEOS

An alternative, yet complimentary, pathway for the metabolism of ethanol is the microsomal ethanol-oxidising system (MEOS) occurring in the liver (Reaction 7). This pathway is up-regulated by repeated heavy consumption of alcohol, and is therefore more active in alcoholics than in the general population. The MEOS also results in the production of acetaldehyde, which, as described above, is converted to acetate. However, unlike the alcohol dehydrogenase reaction, the MEOS oxidises its co-factor, the reduced form of nicotinamide adenine dinucleotide phosphate (NADPH), thus altering the energy coupling of ethanol oxidation and oxidative phosphorylation resulting in the generation of heat (Foster and Marriott 2006; Riveros-Rosas et al. 1997).

The metabolism of anything more than a few grams of ethanol can result in disruption in the ratio of NADH and NAD^+ within the cytosol of hepatocytes. Under normal conditions, when ethanol is absent or present in very small amounts, NADH is re-oxidised, in a timely manner, through the shuttling of electrons into mitochondria via the malate-aspartate and glycerophospate shuttles to maintain a low NADH - NAD^+ ratio. However, when moderate to large amounts of ethanol are present the shuttling of electrons is unable to match the production of NADH thus resulting in an increase in NADH within the cytosol. As many of the reactions that occur within the cytosol are close to equilibrium this increase in NADH directly impacts dehydrogenase reactions which rely on NAD^+, in particular the lactate dehydrogenase reaction which usually metabolises circulating lactate to produce pyruvate. Alterations in this reaction may result in elevations in circulating levels of lactate and subsequent lactic acidosis. Additionally, the reduction in concentrations of pyruvate may impact hepatic gluconeogenesis, which under conditions of low hepatic glycogen content, may lead to severe hypoglycaemia (Lieber 2005).

The altered NADH - NAD^+ ratio also impacts a number of other metabolic processes including a shift towards lipogenesis and hepatic fatty acid accumulation and a shift away from fatty acid oxidation, which is usually the main source of energy in hepatocytes. The H^+ produced during the metabolism of ethanol are preferentially used in the TCA cycle so that, together with changes occurring in the cyctosol, the rates of whole body fat, protein and carbohydrate oxidation may be significantly impaired (Paton 2005; Shelmet et al. 1988).

2.1.3 Consumption levels

Globally, alcohol is the most commonly used psychoactive drug. The World Health Organisation (WHO) (2011) estimates that each adult (15 + years of age) consumes, on average, approximately 4.3 L of pure alcohol per year. Use of alcohol is widespread with Europe (10.00 L or greater), Australia, Argentina (both 10.00 – 12.49 L), North America and New Zealand (both 7.5 – 9.99 L) having the highest levels of annual consumption per adult. Of the common forms of alcohol available, beer is the most widely used, however consumption of spirits in the highly populated regions of Eastern Europe and Asia means that this stronger form of alcohol makes up 45.7 % of the total volume of alcohol consumed worldwide annually. Beer (36.3 %),

wine (8.6 %) and other alcoholic beverages (10.5 %), which include traditional and regional varieties of beverage, are all consumed in lower volumes (World Health Organization 2011).

In 2010 it was estimated that each adult in New Zealand, on average, consumed 9.6 L of pure alcohol as: 299 million L of beer; 102.6 million L of wine; 59.5 million L of spirit based drinks, such as ready - to – drink beverages; and 12.6 million L of spirits (Statistics New Zealand 2011).

2.1.4 Guidelines for alcohol use

The WHO classifies safe or low levels of consumption as four standard drinks (StD) per day for males and two StD per day for females, with a StD being classified as any beverage containing 8 g of ethanol. However, the definition of a StD differs between countries. For example, in New Zealand a standard drink contains 10 g of alcohol whereas in Japan a standard drink contains 19.75 g of alcohol. The majority of countries listed in a review of international drinking guidelines defined a standard drink as containing between 8 g and 14 g of alcohol (International Centre for Alcohol Policies 2003). Above the safe levels recommended by the WHO alcohol consumption becomes hazardous (four to six or two to four StD per day for males and females, respectively). Even larger amounts are classified as harmful and may significantly increase the risk of negative mental and physical health issues (Treatment Protocol Project 2000). In New Zealand, the Alcohol Advisory Council (ALAC) recommends that males drink no more than 21 StD and females no more than 14 StD per week, with at least two alcohol free days per week. In the context of this thesis a StD will denote an alcoholic beverage containing 10 g of alcohol, as defined by ALAC.

Heavy episodic or binge drinking is classified as the consumption of 60 g of alcohol in a single drinking episode (World Health Organization 2011) therefore, to avoid the negative health consequences associated with this type of alcohol use, ALAC recommend that no more than 6 StD for males and 4 for females be consumed in any given drinking episode (Alcohol Advisory Council of New Zealand 2007).

2.1.5 Drinking behaviour

2.1.5.1 General population

In their report on alcohol use in New Zealand the Ministry of Health (2009) reported that 85 % of New Zealanders aged between 16 and 64 had consumed alcohol in the past year. Of this group 62 % binged on alcohol at least once during that year, while 13 % of drinkers consumed more than ALAC's recommended amounts at least once a week over this same period. Of the estimated 283,900 adults regularly bingeing on alcohol 33 % of this group were males and 19 % were females aged 18 – 24. Males and females in the 25 - 34 year age group exhibited the next highest rates of weekly binge drinking, 16 and 13 % respectively, suggesting this behaviour is a serious issue with individuals aged between 18 and 34. An earlier report by the Ministry of Health (2008) suggested that 18 % of New Zealand adults had potentially hazardous drinking habits. The rates of heavy episodic drinking in New Zealand are similar to those of other Westernised countries including the United States of America (USA), Australia, Argentina and Canada (World Health Organization 2011).

2.1.5.2 Sportspeople

Large surveys of the drinking habits of college students in the USA have revealed that students involved or partly involved in sports not only drink alcohol more regularly, they also have a higher tendency to drink to get drunk and therefore binge on alcohol more often than those students with no involvement in sports (Ford 2007a; Nelson and Wechsler 2001; Wechsler et al. 1997). Similarly, a survey of French sport science students showed that, although these sporting students drank less frequently than the French general population of a similar age, they reported more frequent episodes of alcohol intoxication. A strong relationship was evident between gender and frequency of intoxication, with males more likely to be intoxicated on ten or more occasions in a year compared to the females surveyed (Lorente et al. 2003).

It is worth noting that, in many countries, the university population as a whole exhibits greater rates of hazardous alcohol consumption than occurs in the general population (Dawson et al. 2004; Karam et al. 2007; Kypri et al. 2005). This suggests that the addition of sporting participation magnifies the hazardous drinking culture that appears to be embedded in university student life.

Regular, hazardous alcohol consumption has been found amongst New Zealand sportspeople, both student and non-student, competing at all levels of competition with at least half of those surveyed reporting regular hazardous drinking behaviour (O'Brien et al., 2005; O'Brien et al., 2007). Given the rate in the general population is 18 % the results of these studies are cause for concern. Of note, hazardous drinking was most evident in males and team sports participants (O'Brien et al. 2007), a fact supported by the high rates of hazardous alcohol use reported by males competing in contact team sports.

Hazardous alcohol consumption has been reported amongst rugby league (Lawson and Evans 1992), rugby union (O'Brien and Lyons 2000; Quarrie et al. 1996) and Australian Rules football (Burke et al. 1991; Dietze et al. 2008; Snow and Munro 2000; Snow and Munro 2006) players with these populations reporting regular, single session alcohol consumption up to nine times the recommended, safe amount. Seen as reward for the hard work put in during training and the match or as an integral part of club culture, the majority of the hazardous alcohol use reported by sportspeople occurs in the hours after the match (Burke et al. 1991; Lawson and Evans 1992; Snow and Munro 2000; Snow and Munro 2006).

An argument often put forward when considering the drinking behaviour of young males participating in contact team sports is that "they are just doing what young men do". However, the rates of hazardous drinking in this population compared to that of their peers in the general population indicates that young sportsmen consume far greater amounts of alcohol, particularly through bingeing behaviour, and therefore put themselves at greater risk of alcohol related harm.

Anecdotally, there may be a belief, albeit a misguided one, amongst sportspeople, managers and coaches that the consumption of alcohol at low to moderate amounts will help the athlete sleep and reduce sensations of pain after their sporting event. These perceived positives of alcohol consumption, along with the use of alcohol to enhance social interaction, may further perpetuate the drinking behaviour of this population.

2.1.6 General physiological effects of alcohol

As will be discussed below, the consumption of alcohol may negatively impact a number of normal physiological processes. However, the evidence behind alcohol's actions on many of these processes comes from *in vitro* experimentation or research with animals using conditions that may be significantly different to those encountered *in vivo*, such as the soaking of muscle tissue in alcohol solution for an extended period of time. Therefore, the results of such research must be considered carefully as they may not necessarily be representative of what happens *in vivo* at orally ingested doses that are realistic.

2.1.6.1 Central nervous system

The popularity of alcohol as a legal drug is due to its depressant effects on the central nervous system (CNS). Once absorbed, alcohol, but not acetaldehyde, readily crosses the blood-brain barrier allowing it to act at a number of locations within the CNS in a dose dependent manner (James 1999; Reilly 2002). Acute alcohol consumption at a dose of 0.75 g of alcohol per kg BW has been shown to significantly alter whole brain glucose metabolism indicating that the functional organisation of the brain is disrupted by what can be considered a moderate dose of alcohol (Volkow et al. 2008). Similar doses have been shown to reduce excitability in the motor (Ziemann et al. 1995) and prefrontal cortices (Kähkönen et al. 2003) while intra-cortical inhibition (Kähkönen et al. 2001; Kähkönen and Wilenius 2007) has also been observed. Alcohol's inhibitory effects are widely believed to be due to the increased action of gamma-aminobutyric acid (GABA) and other inhibitory neurotransmitters (Eckardt et al. 1998; Grobin et al. 1998; Ziemann et al. 1995) along with widespread changes in the function of neurotransmitter gated ion channels (Lovinger 1997). Combined, these changes in cortical excitability are responsible for many of the cognitive impairments associated with alcohol intoxication (Kähkönen 2005; Volkow et al. 2008), as listed in Table 2.1. Additionally, increased activity in the limbic region, through the actions of dopamine and serotonin, reinforce the pleasure associated with alcohol consumption thus making alcohol a highly addictive drug (Eckardt et al. 1998; Volkow et al. 2008).

Elsewhere in the CNS the effects of alcohol are less consistent and, as most studies have been performed on a wide variety of animals, may be species specific (Gruol 1982). At low doses, alcohol has an inhibitory effect on the activity of spinal cord

neurons through GABA mediated changes in synaptic transmission (Davidoff 1973; Gruol 1982; Mehta and Ticku 1988). Similarly, the production of action potentials is negatively affected by changes in Na^+ conductivity (Gage 1965). At the neuromuscular junction (NMJ) alcohol appears to potentiate neuromuscular transmission, possibly through the increased release of acetylcholine (ACh) (Inoue and Frank 1967; Searl and Silinsky 2010), while others have reported an increase in synaptic delay - the time taken for an impulse to pass from the nerve to the muscle fibre (Reed 1980).

Table 1 Alcohol's effects on cognitive function at different blood alcohol concentrations (adapted from Reilly, 2002; James, 1999).

Blood alcohol level (mg/100 ml of blood)	Effects
30	Enhanced sense of well-being; retarded simple reaction time; impaired hand-eye coordination; increased risk of having an accident
60	Mild loss of social inhibition; impaired judgement; increasing cheerfulness
90	Marked loss of social inhibition; co-ordination reduced; noticeably 'under the influence'
120	Apparent clumsiness; loss of physical control; tendency towards extreme responses; definite drunkenness is noted
150	Erratic behaviour; slurred speech; staggering gait; quarrelsomeness; exuberance
180	Loss of control of voluntary activity; impaired vision
400	Oblivion; sleepiness; coma
500	Death possible
600	Certain death

The blood alcohol limit in New Zealand is currently 80mg/100ml of blood (New Zealand Transport Agency 2009)

2.1.6.2 Skeletal muscle

Intracellular Ca^{2+} ($[Ca^{2+}]_i$) plays an essential role in the excitation-contraction (EC) coupling process, the chain of biochemical events occurring after nerve transmission at the NMJ. Studies using skeletal muscle fibres from mice (Nicolás et al. 1998) and human myotubes, similar in form and function to human skeletal muscle fibres (Cofán, 2000), show that alcohol at very high doses acutely affects EC coupling in mammalian skeletal muscle fibres in a dose dependent and reversible manner. In both cases alcohol reduces the concentration of intracellular Ca^{2+} transients. This is due to the direct inhibitory action of alcohol, rather than acetaldehyde (Oba and Maeno 2004) on sarcolemmal voltage-gated Ca^{2+} channels and a decrease in the amount of releasable Ca^{2+} stored within the sarcoplasmic reticulum (SR). Nicolás and colleagues (1998) also observed that high doses of ethanol decreased cellular excitability, possibly due to an inhibition of Na^+ channels, as occurs when cardiac muscle fibres (Habuchi et al. 1995) and frog skeletal muscle are treated with ethanol (Inoue and Frank 1967). The influence alcohol has on EC coupling may be responsible for the decrease in strength occasionally seen immediately after alcohol consumption (American College of Sports Medicine 1982) and the muscular weakness experienced in patients with acute and chronic alcohol myopathies (Urbáno-Marquez & Fernández-Sola, 2004).

As the inhibitory effects of alcohol on the EC coupling process have been reported at blood alcohol levels equivalent to 90 mg / 100 ml or greater, and that these effects no longer occur after alcohol has been washed off the isolated muscle fibres / myotubes (Nicolás et al. 1998; Cofán, 2000), it is unlikely that these direct effects are still evident once blood alcohol drops below this level or when alcohol has been completely metabolised. Whether alcohol's action on EC coupling, and therefore force production, occurs *in vivo* is currently unclear with a number of studies showing acute alcohol use impacts muscular strength while an equal number have failed to see any significant affect during or after alcohol consumption (American College of Sports Medicine 1982; Poulsen et al. 2007).

Further, alcohol may impact skeletal muscle function through its effects on protein synthesis. Both whole body and skeletal muscle protein synthesis, but not degradation (Reilly et al. 1997), have been shown to be altered by acute alcohol treatment in rats

with skeletal muscle protein synthesis decreasing by as much as 75 % for at least 24 h post alcohol treatment (Preedy et al. 1988; Reilly et al. 1997; Tiernan and Ward 1986). In muscle, type II fibres appear to be more susceptible to alcohol than type I fibres (Preedy et al. 1992; Reilly et al. 1997). Similarly, *in vitro* studies have shown a decrease in protein synthesis through the alcohol and acetaldehyde mediated impairment of insulin-like growth factor 1 (IGF-1) and insulin (Hong-Brown et al. 2001). Once again, whether these effects are seen in humans at physiological circulating alcohol concentrations is unknown. To put these findings into perspective, the doses used in the majority of studies investigating alcohol's impact on protein synthesis is 75 mmol of alcohol per kg BW (Preedy et al. 1988; Reilly et al. 1997; Tiernan and Ward 1986). This translates to 3.46 g/kg BW which is equivalent to 241.9 g of alcohol, or 24 StD, for a 70kg individual. While this level of alcohol consumption is not unheard of amongst certain populations, including team sportspeople, ethically it would be extremely difficult to identify whether the same findings hold true *in vivo* in humans. Currently, there is a dearth of information relating to the effects of acute alcohol consumption and protein synthesis in humans. Given the role of protein synthesis in recovery and adaptation after strenuous exercise (Rennie and Tipton 2000) there is considerable scope for future research in this area.

2.1.6.3 Immune function

Chronic alcohol use has long been associated with altered immune function, as evident in chronic alcoholics who often exhibit increased susceptibility to a range of infectious diseases (Cook 1998). However, acute alcohol consumption may also negatively impact immune function thus making the drinker more susceptible to infection (Greiffenstein et al. 2007; Szabo 1999).

Under normal circumstances the innate immune system responds to trauma or infection by initiating a complex inflammatory response. Typically characterised by alterations in hormone and cytokine levels, this process directs phagocytosis, cell proliferation and differentiation to ensure the inflammatory stimulus is dealt with appropriately and subsequent recovery occurs (Giannoudis 2003). Although the specific role each inflammatory molecule plays in this process is not fully understood, the actions of several key molecules are now known. Many of these inflammatory molecules are affected by acute alcohol treatment.

Acute alcohol exposure upsets the balance of normal inflammatory processes resulting in a net shift towards an anti-inflammatory environment through selective alterations in cytokine activity (Greiffenstein et al., 2007). A major contributor to this altered immune state is the alcohol induced down-regulation or impairment of tumour necrosis factor-α (TNF-α) production (Szabo 1999; Szabo et al. 1996; Zhao et al. 2003). Such impairment results in a decrease in endothelial cell activation and therefore a reduction in the expression of cell adhesion molecules (CAMs), thus negatively affecting neutrophil - endothelial cell adhesion. Further, without normal concentrations of TNF-α, the endothelium is unable to produce a number of pro-inflammatory molecules which usually act to magnify the inflammatory response (Saeed et al. 2004). In addition, acute alcohol treatment also inhibits interleukin (IL) - 1β and IL-6 expression (Fitzgerald et al. 2007; Goral et al. 2004; Szabo 1998) further limiting the pro-inflammatory response to infection and trauma.

While negatively affecting pro-inflammatory molecules, the presence of alcohol also increases production of anti-inflammatory molecules. The production of transforming growth factor-β (TGF-β), and therefore its immuno-inhibitory action, is increased at physiologically relevant levels of alcohol (Szabo et al. 1996). Along with inhibiting cellular proliferation, increased TGF-β activity is also likely to inhibit inflammatory cytokine production and lessen the overall effectiveness of the immune response (Szabo, 1999). Acute alcohol related increases in IL-10 levels augment the anti-inflammatory state leading to inhibition of T cell proliferation and down-regulation of pro-inflammatory cytokine activity (Szabo 1998; Szabo et al. 1996). Prostaglandin E_2 (PGE_2) production by monocytes is also increased after acute ethanol stimulation. Similar to the actions of TGF-β and IL-10 increased levels of PGE_2 inhibit inflammatory cytokine production, T cell proliferation and monocyte antigen presentation capacity (Szabo et al. 1992).

Essential to the early stages of the inflammatory process, neutrophils are directly affected by clinically relevant levels of alcohol. As with alcohol's effect on the endothelium's ability to express CAMs, the expression of neutrophil surface adhesion molecules is also inhibited. Such inhibition significantly impacts neutrophil migration. Along with limiting the neutrophils ability to move to the site of

inflammation, alcohol also inhibits superoxide production, an important weapon in the process of phagocytosis (Patel et al., 1996).

2.1.6.4 Hormonal function

As with the innate immune system, the acute consumption of alcohol has been shown to have a profound impact on the endocrine system; a summary of the acute effects of alcohol on a select number of hormones is presented in Table 2.2. Alcohol clearly impacts normal hormonal balance so that a range of factors, including sleep quality, mood, metabolism and cardiovascular function may all be affected during and/or after drinking. Importantly for males, when consumed acutely in large doses (1.5 g alcohol per kg BW), alcohol has a negative effect on testosterone production which, together with an increased conversion rate of testosterone and androstendione to their respective estrogens, leads to feminising effects such as gynecomastia and testicular atrophy (Cicero 1981). Additionally, a decrease in testosterone may also impact skeletal muscle function, bone density and red blood cell numbers (Emanuele and Emanuele 1998). While large doses of alcohol negatively affect testosterone levels, Sarkola and Eriksson (2003) found that a low dose of alcohol (0.5 g per kg BW) has the opposite effect on testosterone while additionally reducing androstenedione levels. Sarkola and Eriksson (2003) proposed that the change in the NADH - NAD^+ ratio in the liver during alcohol metabolism may result in a decreased rate of testosterone oxidation, thus leading to the observed increase in testosterone and decrease in androstenedione. This finding highlights the difficulty associated with understanding the effects alcohol has on normal physiological function as the outcome and mechanisms behind any given observation may be completely different with a different dose.

Table 2 Summary of the effects acute alcohol consumption has on a select number of hormones in males.

Author (Year)	Hormone	Effect on secretion	Physiological implication
Perman (1958) Ireland et al. (1984)	Adrenaline	Increase	Increased heart rate, tachycardia, hypertension
Ylikahri et al. (1978) Rivier & Vale (1988)	Cortisol	Increase	Negatively affect reproductive function, increase blood glucose
Boileau (2003)	Dopamine	Increase	May reinforce alcohol use
Gordon et al. (1978)	Estrogen	Increase	May have feminising effects on males
Leppäluoto et al. (1975) Prinz et al. (1980)	Human Growth Hormone	Decrease	May impact a number of biochemical process
Marks (1978)	Insulin	Increase	Hypoglycemia, if in a fasted state or if consumed with high carbohydrate food
Mendelson et al. (1977) Leppäluoto et al. (1975)	Luteinizing Hormone	Decrease	Decrease testosterone production
Rupp et al. (2007)	Melatonin	Decrease	Sleep fragmentation and disruption
Badawy (1998)	Serotonin	Decrease	Aggression, dysphoria
Mendelson et al. (1977) Gordon et al. (1976) Badr and Bartke (1974)	Testosterone	Decrease	Decreased muscle function, osteoporosis, anaemia, decreased libido, impotence, infertility, feminisation
Kleeman et al. (1955) Eisenhofer & Johnson (1982)	Vasopressin	Decrease	Increase in urine output

2.1.6.5 Circulation

The chronic effect of alcohol on the heart and circulatory system is very much dependent on the drinking behaviour of the individual. Moderate, regular alcohol consumption has been suggested, by association, to protect against myocardial infarction and stroke through a reduction in plaque deposits, platelet aggregation and blood clot formation. Conversely, heavy, chronic alcohol abuse is strongly associated with increased risk of cardiomyopathy, arrhythmia, high blood pressure and hemorrhagic stroke (Zakhari 1997).

Acutely, alcohol consumption may bring about an increase in cardiac output, stroke volume and heart rate through the increased circulation of catecholamines (Child et al. 1979; Kupari 1983). These catecholamine induced changes may serve to compensate for the well documented decrease in left ventricle contractility (Delgado et al. 1975; Horwitz and Atkins 1974; Regan et al. 1966) so that no significant changes in cardiac function are observed with moderate to high levels of alcohol consumption (Child et al. 1979; Kupari 1983)

Alcohol is commonly viewed as a strong vasodilator however its actions have been shown to be tissue specific. Alcohol, but not its metabolites (Altura and Gebrewold 1981), act concurrently as both a vasoconstrictor and vasodilator so that blood flow through skeletal muscle and the internal organs is reduced while at the same time blood flow to the skin increases (Fewings et al. 1966; Gillespie 1967). An increase in peripheral vasodilation could have implications for thermoregulation, however evidence regarding a direct influence of alcohol on thermoregulation in both hot and cold environments is inconclusive (Allison and Reger 1992; Johnston et al. 1996; Scarperi and Bleichert 1983; Yoda et al. 2005; Yoda et al. 2008).

Additionally, cerebral blood flow (CBF) is increased by acute alcohol use (Blaha et al. 2003; Mathew and Wilson 1986; Sano et al. 1993) while regional CBF may increase or decrease depending on the region (Mathew and Wilson 1986; Sano et al. 1993; Tiihonen et al. 1994; Volkow et al. 1988). Alcohol related variations in regional CBF, irrespective of increases in global CBF, are likely to be contribute to the well-known changes in cognitive function that occur during intoxication (Sano et al. 1993; Volkow et al. 1988), as outlined in Table 2.1.

2.1.7 Effects of alcohol on exercise performance

The long standing belief that consumption of alcohol prior to and during exercise or sporting competition improves physical performance dates back to the early Greek and Chinese empires. Surprisingly, only relatively recently has alcohol been viewed as an ergolytic substance; that is, alcohol consumed prior to exercise has a detrimental effect on physical performance (Eichner 1993). The release of the American College of Sports Medicine (ACSM) (1982) Position Stand on the use of alcohol in sports brought attention to this important issue by highlighting the potentially deleterious effects alcohol can have on physical performance. The available literature and trend in research at the time meant that the ACSM Position Stand almost exclusively addressed the effects of pre-exercise alcohol consumption on subsequent physical performance. The ACSM Position Stand (1982) concludes that acute alcohol use may impair strength, power, muscular endurance, speed and cardiovascular endurance. However, there appears to be no detrimental effect of alcohol on maximal oxygen uptake, heart rate, stroke volume, muscle blood flow and respiratory dynamics during exercise.

Since the release of the Position Stand much of the research into the effects of pre-exercise alcohol consumption on performance has proven inconclusive with a number of studies reporting contradictory findings. This is most likely due to the use of different doses of alcohol, different exercise protocols and modalities (i.e. running versus cycling) and variability between subjects for alcohol tolerance. The studies completed since the release of the ACSM Position Stand are summarised in Table 2.3.

Together with the conclusions of the ACSM Position Stand (1982), the findings of Lecoultre & Schutz (2009), Kendrick et al. (1994) and McNaughton & Preece (1986) strongly suggest that low to moderate doses of alcohol do not positively influence performance; rather they are likely to decrease endurance performance. It is less clear whether alcohol use impacts measures of strength with the Position Stand (1982) reporting mixed results for measures of strength after alcohol consumption. Although alcohol may act at a number of locations important to force production within the CNS, the recent results of Poulson et al. (2007) further suggest that muscular strength is not affected even at high doses.

Table 3 Summary of research carried out since the ACSM Position Stand (1982) into the effects of alcohol on physical performance.

Author (Year)	Dose	Performance Measure	Effect on Performance
Bond et al. (1984)	0.44ml/kg BW of 95% ethanol	Maximal, progressive cycling test	No effect
Bond et al. (1984)	0.88ml/kg BW of 95% ethanol	Maximal, progressive cycling test	No effect
Houmard et al. 1987	BrAc below 0.5 mg/ml	5 mile treadmill time trial	No effect
Lecoultre & Schutz (2009)	0.5g/kg lean BW	60 minute cycling time trial	Decrease in total work done
Kendrick et al (1994)	25ml 10 minutes before and 30 minutes after the onset of exercise	Treadmill run at 80-85 % VO2max intensity	3 of the 4 subjects failed to complete the allocated time
McNaughton & Preece (1986)	BrAc of 0.01 mg/ml and 0.1 mg/ml	100m run 200m run 400m run 800m run 1500m run	No effect Increased time (dose dependent) Increased time (dose dependent) Increased time (dose dependent) Increase time (dose dependent)
Poulson et al. (2007)	1.59g and 1.48g/kg BW for males and females respectively	Isometric and isokinetic strength	No effect

Low doses of alcohol impact the CNS, and as such have been used to lessen tremor in target sports (O'Brien and Lyons 2000). Further, such doses improve performance during complex cognitive tasks, including the Rapid Visual Information Processing task which tests sustained attention ability (Lloyd and Rogers 1997). These are possibly the result of the calming and sedative effects associated with low doses of alcohol (Lloyd and Rogers 1997). Because of these potentially ergogenic properties, alcohol has been banned by the World Anti-Doping Agency (World Anti-Doping Agency 2007)in target sports such as the modern pentathlon and archery. However, higher doses are associated with decreased performance in a range of psychomotor tasks including hand-eye coordination, tracking tasks, complex decision making and reaction time (Reilly 2002). These detrimental effects mean that alcohol is also banned in sports which rely on fine motor control and fast reaction times such as motorcycle, powerboat and automobile racing (World Anti-Doping Agency 2007). Clearly at higher doses alcohol consumed prior to sport and exercise will have a negative impact on performance by impairing coordination, decision making ability and reaction time. Although alcohol has been shown to improve confidence and feelings of general wellbeing, while also increasing aggression (Reilly 2002), a combination of decreased motor control and inhibition of normal self-preserving cognitive function is likely to contribute to an increased chance of injury during the sporting event (O'Brien and Lyons 2000).

2.1.8 Effects of alcohol on recovery

Unlike research into the effects of pre-exercise alcohol use on performance there is currently limited published scientific literature regarding the effect of post-exercise alcohol use on recovery. Given the drinking habits of sportspeople this scenario is more realistic than that of consuming alcohol prior to exercise. The limited research that has investigated the effects of post-exercise alcohol use on recovery has tended to focus on factors that influence or are associated with recovery, or proxies for recovery, rather than investigating whether a return to optimal performance is impaired by post-exercise alcohol use. For example, strategies such as rehydration, restoration of energy stores and accelerated injury repair are thought to be essential if optimal recovery is to be achieved (Garrett 1990; Reilly and Ekblom 2005). However, whether these indirect measures of recovery reflect a return to pre-exercise performance is debatable.

2.1.8.1 Metabolic recovery

During strenuous exercise liver and muscle glycogen stores may be reduced, while sweating can result in dehydration (fluid loss of 2-5% body mass or greater), which includes electrolyte loss (Maughan and Burke 2002). Dehydration has been shown to impair performance (Armstrong et al. 1985) and so, adequate rehydration and restoration of electrolytes after exercise is important to ensure recovery before the next training session or event (Garrett 1990; Maughan et al. 1994). Equally important to recovery is the repletion of muscle glycogen. Optimal refuelling strategies depend on the type and duration of exercise as well as the time between exercise bouts or events. It has been suggested that the best opportunity for optimising glycogen stores occurs when carbohydrate is consumed in the initial hours after exercise; after that time glycogen storage rates decrease significantly (Maughan and Burke 2002). However, in many sports this period after competition may be spent consuming alcohol instead of following correct nutritional strategies.

Early studies investigating a diuretic effect of alcohol confirmed the long held belief that the consumption of alcohol increases urinary output (Eggleton 1942) through the inhibition of vasopressin (anti-diuretic hormone) (Eisenhofer and Johnson 1982; Kleeman et al. 1955; Rubini et al. 1955). While it appears that the consumption of alcohol has little effect on the excretion of electrolytes during peak diuresis (Rubini et al. 1955; Strauss et al. 1950) alcohol related increases in water loss may result in the osmotic redistribution of water from within cells to the blood stream so that the ionic balance between the two is maintained. Such a change can negatively impact normal cellular function.

A long-standing misconception in the sporting community is that beer, the most commonly consumed alcoholic beverage amongst athletes, is an appropriate form of post-exercise fluid replacement. Beer, however, contains only small amounts of electrolytes, is relatively low in carbohydrates, and has a diuretic effect on the kidneys. It is therefore an inappropriate recovery drink after strenuous exercise (O'Brien 1993).

The negative effect alcohol has on the restoration of fluid balance after exercise has been confirmed by Shirreffs and Maughan (1997). A range of doses, equivalent to

approximately 0, 0.24, 0.49 and 0.92 g of alcohol per kg body weight (BW), were utilised to investigate the effects of acute alcohol consumption on rehydration after dehydrating exercise. Only the highest dose, still less than 1 g of alcohol per kg BW, was found to significantly increase urine output, reduce the recovery rate of blood volume and therefore delay recovery from the dehydrated state, suggesting that alcohol in dilute concentrations has little effect on rehydration. This finding implies that if optimal rehydration is to occur after exercise then a dose of alcohol greater than 0.45 g alcohol per kg BW should be avoided, at least until full rehydration has been attained. However if fluid replacement is not a priority, for example if optimal performance is not required the next day, then the consumption of alcohol post-exercise, from a hydration stand point, may be acceptable. It is worth noting that 0.5 g alcohol per kg BW is equivalent to 3 and a half standard drinks for a 70 kg person.

Although the consumption of alcohol may impact hepatic gluconeogenesis (Lieber, 2005), glucose utilisation and glucose uptake into skeletal muscle (Jorfeldt and Juhlin-Dannfelt 1978; Juhlin-Dannfelt et al. 1977), all of which could contribute to decreased performance if alcohol was consumed prior to exercise, it appears that alcohol has little or no effect on the resynthesis of muscle glycogen after exercise. Burke and colleagues (2003) compared the effects of three recovery diets, two of which contained alcohol, on post-exercise muscle glycogen resynthesis. Consumption of alcohol (1.5 g alcohol per kg BW) in conjunction with a high carbohydrate diet had no impact on post-exercise glycogen storage compared to a diet containing no alcohol. However, an alcohol 'displacement diet' where some carbohydrate-containing food was replaced by an isocaloric amount of alcoholic beverage, was found to impair glycogen repletion. This finding is most likely due to the reduced availability of substrate for glycogen resynthesis rather than any effect alcohol has on the process of glycogenesis. This dietary scenario is similar to that faced by athletes who consume alcoholic beverages post-exercise instead of following appropriate nutritional strategies aimed at replenishing glycogen stores (Burke et al. 2003).

2.1.8.2 Recovery from soft tissue injury

Injury, particularly to skeletal muscle, is a common occurrence in many team sports and may be caused through collision with other competitors, impact with the ground or through eccentric muscular action (Garrett 1990; Gill et al. 2006). From a performance point of view the most important implication of muscle injury is a decrease in muscle function. Any loss in the ability to generate appropriate levels of force during competition or training is likely to be detrimental to the outcome of the event and may impact subsequent adaptation from training. Therefore, optimal recovery from damage to skeletal muscle is essential if a timely return to training and/or competition is required. Additionally, if full recovery is not achieved the likelihood of further injury to the muscle may be increased (Taylor et al. 1993).

Along with other restorative strategies aimed at limiting blood flow to the injured tissue, abstinence from alcohol during the injured state is regularly advised. The evidence for this recommendation, however, appears to be mostly anecdotal, stemming from the known vasodilatory effect of alcohol (Maughan 2006). Although, under normal circumstances, alcohol acts as a peripheral vasodilator at the skin and a vasoconstrictor at the muscle (Fewings et al. 1966; Gillespie 1967) evidence from animal studies suggest that changes in the normal response to trauma, including elevations in vasopressin, adrenaline and noradrenaline, are inhibited when alcohol is consumed prior to trauma/injury (Bilello et al. 2011). This inability to limit blood flow to the site of injury is likely to contribute to the increased oedema that has been observed when alcohol is consumed prior to experimentally induced trauma (Brodner et al. 1981; Flamm et al. 1977). Coupled with alterations in immune function, increased blood flow to the site of injury may increase the severity of the injury and negatively impact the rate and outcome of recovery (Messingham et al. 2002).

Anabolic hormones play an essential role in wound healing, in particular protein synthesis and regeneration of damaged tissue (Demling 2005; Lynch et al. 2008). Therefore it is possible that alcohol's effects on anabolic hormone secretion may negatively impact this phase of the recovery process. As already stated, alcohol attenuates testosterone secretion when consumed at high doses. When consumed after exhaustive aerobic exercise, a dose of 1.5 g alcohol per kg BW has been shown to prolong the inhibitory effect alcohol has on testosterone (Heikkonen et al. 1996).

However, when consumed after resistance exercise, a lower dose (0.83 g alcohol per kg BW) of alcohol prolongs elevations in cortisol, thus extending the post – exercise catabolic state, while at the same time having no impact on testosterone (Koziris et al. 2000). Intriguingly, when consumed after resistance exercise, this dose has been shown to increase free testosterone concentrations above exercise only levels for up to five hours post – exercise (Vingren et al. 2003). Although these studies did not investigate muscle injury after exercise there is a possibility that, when combined with the stresses of sport / exercise, alcohol's negative effect on normal hormone function may be magnified.

Currently there is no direct evidence that alcohol alters recovery processes following injury of skeletal muscle in humans. This is most likely due to the ethical difficulties associated with experimental research, for example, although contusion injury models have been developed in rats (Crisco et al. 1994), it may be difficult to justify bringing about a contusion or impact related injury in human subjects. In observational studies many confounding factors must be taken into account, such as underlying chronic alcohol use and the type and location of the trauma, making it difficult to draw valid conclusions from such research (Li et al. 1997). The use of EIMD has therefore been used to investigate whether alcohol does indeed impact the severity of skeletal muscle damage. Eccentric EIMD provides a dose-controlled, valid, and ethical model for inducing damage to skeletal muscle. It has previously been employed to investigate a number of recovery modalities including cold water immersion therapy (Eston and Peters 1999), non-steroidal anti-inflammatory medication (Gulick et al. 1996), massage (Jönhagen et al. 2004) and compression therapy (Kraemer et al. 2001). Additionally, given the incidence of EIMD in many sports this model may also have direct relevance to the sporting community.

2.1.8.3 Alcohol and EIMD

As with the majority of other studies investigating the interaction between alcohol and exercise, the first study to look at the combination of acute alcohol consumption and EIMD had subjects consume alcohol prior to eccentric exercise. Although this scenario replicates the common occurrence of consuming alcohol and then, as a result of intoxication, sustaining an injury, its application to the sporting environment where alcohol is generally consumed after exercise, may be limited. In this study Clarkson

and Reichsman (1990) had subjects drink either a beverage containing 0.8 g alcohol per kg BW or a non-alcoholic control beverage 35 minutes prior to performing 50 maximal eccentric contractions of the elbow flexor muscles. No difference between treatments was evident in measures of CK, muscle soreness, isometric strength or range of motion.

More recently, Barnes et al. (2010) investigated the opposing, and perhaps more realistic scenario for many sports, of having subjects consume alcohol after damaging exercise. Subjects consumed 1 g alcohol per kg BW or an isovolumetric, isoenergetic non-alcoholic beverage 30 minutes after completing 300 eccentric contractions of the quadriceps. A significant difference in measures of isometric and isokinetic torque was found between the two treatments with greater decrements in muscle function observed with alcohol. An example of the differences in force loss, in this instance isokinetic concentric torque, is presented in Figure 1.

Similar findings were evident for isometric force and isokinetic eccentric torque. This finding suggests that, when consumed after a damaging bout of exercise, alcohol magnifies the typical force loss associated with EIMD. The disparate results of Clarkson and Reichsman (1990) and Barnes et al. (2010) may be due to the timing of alcohol consumption (pre versus post exercise), the dose used or the greater volume of exercise and larger muscle mass used by Barnes et al. (2010). Further research is required to confirm the findings of Barnes et al. (2010) and additionally to better understand the interaction between post – exercise alcohol consumption and EIMD.

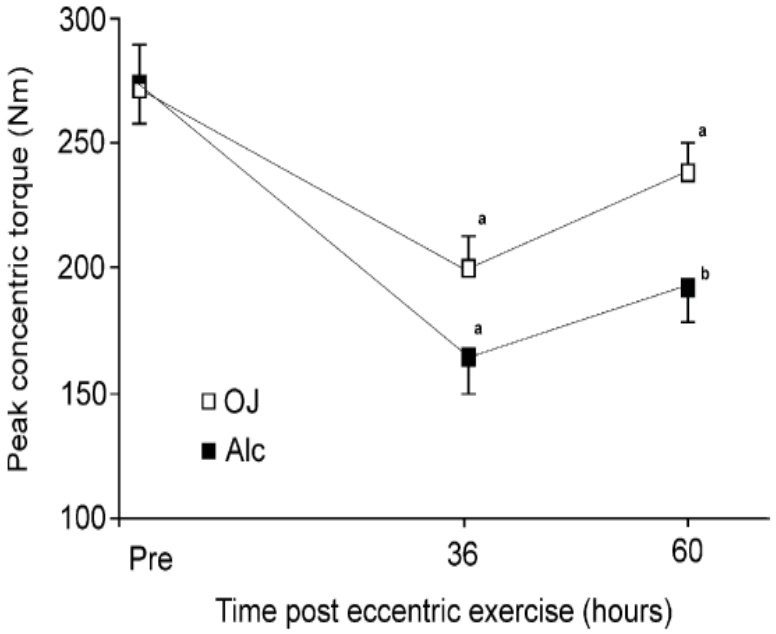

Figure 1 Peak concentric torque (mean ± SE) measurements made before and 36 and 60 h after 300 eccentric contractions of the quadriceps under control (OJ) and alcohol (ALC) conditions. Significant differences in values occur over time ($p < 0.001$) and between trials ($p < 0.05$) exist. A significant interaction effect exists ($p < 0.05$). Significantly different from preceding values – a $p < 0.001$, b $p < 0.05$ (Barnes et al. 2010).

2.2 ECCENTRIC EIMD

2.2.1 Voluntary force production

Under normal conditions, the intent to voluntarily generate force involves the sequence of tightly regulated events originating in the brain and ending with the activation of skeletal muscle and, usually, movement around a joint. This sequence of events will, depending on the external load or task, result in the muscle remaining the same length (isometric), shortening (concentric) or lengthening (eccentric) under tension.

2.2.1.1 Neural control of force

Briefly, voluntary force production originates in the cerebral cortex, basal ganglia and cerebellum. Activity in these regions may be altered by input from a number of sensory fibres including group I and II afferents, which sense changes in muscle length and tension, and group III and IV afferents which sense pain. Convergent input from the pre - motor cortex, basal ganglia and cerebellum leads to excitation, or inhibition, of neurons in the primary motor cortex. Output from the primary motor cortex brings about depolarisation of motor neurons in the pyramidal tract, located in the spinal cord, resulting in excitation of anterior (α) - motor neurons. Each muscle may be innervated by a motor neuron pool made up of a large number of α - motor neurons; for example the gastrocnemius is controlled by approximately 580 α - motor neurons. Depolarisation of the axonal membrane, through opening of voltage gated Na^+ channels, allows for action potential propagation along the axon to the terminal branches. Branching of the axon may result in the innervation of up to 1000, or more, muscle fibres depending on the particular muscle. Together the α - motor neuron and the fibres it innervates make up a motor unit (MU) (Grabowski and Tortora 2003; Komi 2003; McArdle et al. 2009).

The magnitude of force generated during voluntary muscular contraction is dependent on the number of MUs recruited and the firing frequency (rate coding) of these active MUs. Recruitment occurs in an orderly manner with small, low-threshold MUs recruited first and as greater force, or faster muscular action, is required more MUs are recruited until the large, high-threshold MUs are active (Henneman et al. 1965; Milner-Brown et al. 1973b). Additional force is produced by an increase in the firing frequency of active MUs (Milner-Brown et al. 1973a) however the contribution of MU recruitment and firing frequency as strategies to increase force development differs between muscles (De Luca et al. 1982; Kukulka and Clamann 1981).

2.2.1.2 EC coupling

The arrival of an action potential at the presynaptic membrane opens voltage – gated Ca^{2+} channels resulting in an increase in $[Ca^{2+}]_i$. This in turn leads to exocytosis of synaptic vesicles which deposit ACh into the synaptic cleft. ACh diffuses across the synaptic cleft and binds with ACh receptors which open allowing for Na^+ influx and

K^+ efflux, thus depolarising the end plate. Any remaining ACh is either taken up by the presynaptic membrane or broken down to acetate and choline by acetylcholinesterase. The end plate potential propagates across the sarcolemma into the T-tubule system. Here, the activation of dihydropyridine receptors leads to activation and opening of ryanodine receptors which release Ca^{2+} from the SR into the cytoplasm (Latash 2008; McArdle et al. 2009).

2.2.1.3 Cross-bridge cycle

Beginning at the end of a "power stroke", binding between actin and myosin (actomyosin) is broken when adenosine triphosphate (ATP) binds to the nucleotide "pocket" of the myosin subdomain (S1). The increase in $[Ca^{2+}]_i$, brought about by depolarisation of the end-plate, activates the troponin complex of the actin filament. The binding of Ca^{2+} to troponin C brings about a conformational change allowing S1 access, albeit weak, to its binding site on the actin molecule. Further conformational changes on the actin filament result in movement of the tropomyosin coil so that S1 has direct access to the binding site. The hydrolysis of ATP to adenosine diphospahte (ADP) and inorganic phosphate (P_i) by the myosin ATPase brings about a swing in the lever arm (cocking) of approximately 60^o which allows S1 to come into close proximity with the binding sites on the actin filament. Binding at this stage remains weak until the release of P_i results in tight binding and the formation of a force - generating state. The release of ADP returns S1 back to its original position, however because it is strongly bound to the actin filament, this action results in movement of the actin filament towards the centre of the sarcomere. This step is typically referred to as the "power stroke". Binding of ATP to the now empty S1 "pocket" releases the binding between myosin and actin so that the cycle may continue. Cross-bridge formation and cycling will continue unabated as long as $[Ca^{2+}]_i$ is high enough to inhibit the troponin-tropomyosin system that would otherwise block the actin - myosin binding sites. Withdrawal of CNS stimulation leads to a decrease in $[Ca^{2+}]_i$ as Ca^{2+} release from the SR is halted and the action of SR Ca^{2+} ATPase returns Ca^{2+} back into the SR from the cytosol (Billeter and Hoppeler 2003; Cooke 1997; McArdle et al. 2009).

The rate at which ATP is utilised in type IIB fibres is approximately four times that of type I fibres (Stienen et al. 1996). This is due to the fibre type specific characteristics

of myosin and SR Ca^{2+} ATPases which have greater rates of ATP hydrolysis in type IIX and IIB fibres compared to those of type I and IIA fibres (Stienen et al. 1996; Szentesi et al. 2001). These specific characteristics, along with increased rates of Ca^{2+} release from the SR in type II fibres (Li et al. 2002), are responsible for the greater rates of shortening, and therefore power production (Figure 2), and specific tension (kN m^{-2}) found in the fast fibre types (Bárány 1967; Bottinelli et al. 1996).

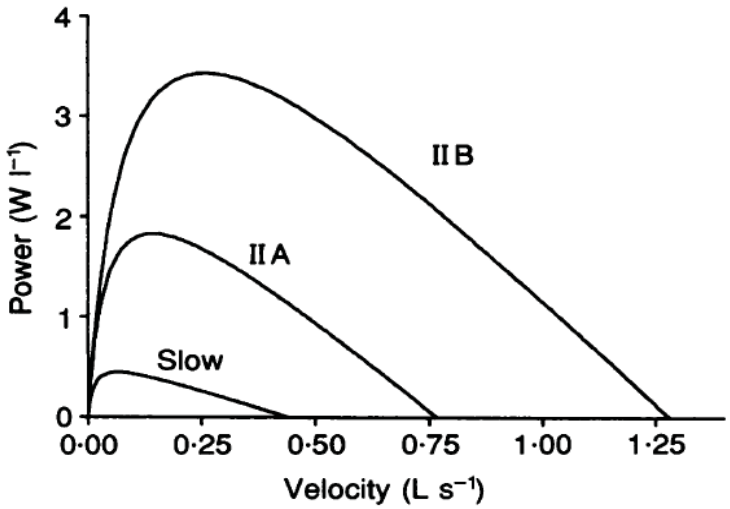

Figure 2 Velocity – power curve for isolated human type I (slow), IIA and IIB fibres at 12˚ C (Bottinelli et al. 1996).

2.2.2 Eccentric muscular contraction

Eccentric muscular contraction occurs when the external force is greater than the force produced by the muscle so that the muscle lengthens under tension (Lindstedt et al. 2001). Eccentric contractions are an essential component of movements occurring in everyday life, as well as in sport and exercise. During the majority of movements, including locomotion, jumping, and resistance training (Byrne et al. 2004), eccentric contractions store potential energy as part of the stretch shortening cycle. This energy is subsequently used during the concentric contraction provided it occurs immediately after the eccentric contraction (Komi 2000). If there is no 'stretch-shortening cycle', all the energy absorbed by the muscle ends up as heat. Eccentric contractions also act to decelerate a limb during coordinated movement, for example during sprinting (Yu et al. 2008) or throwing a ball (Mikesky et al. 1995), or act as a shock absorber, such

as occurs when walking down a steep hill (Lindstedt et al. 2001). The latter, again, results in significant heat production in the muscle.

2.2.2.1 Force – velocity relationship

In isolated muscle fibres, once the load exceeds the maximal isometric force the muscle begins to yield at a very slow rate producing a relatively flat region on the force – velocity curve, at this point the force developed is very similar to isometric force values (Figure 3). However, as the rate of lengthening increases the force produced may be twice that of isometric force production (Edman 1988).

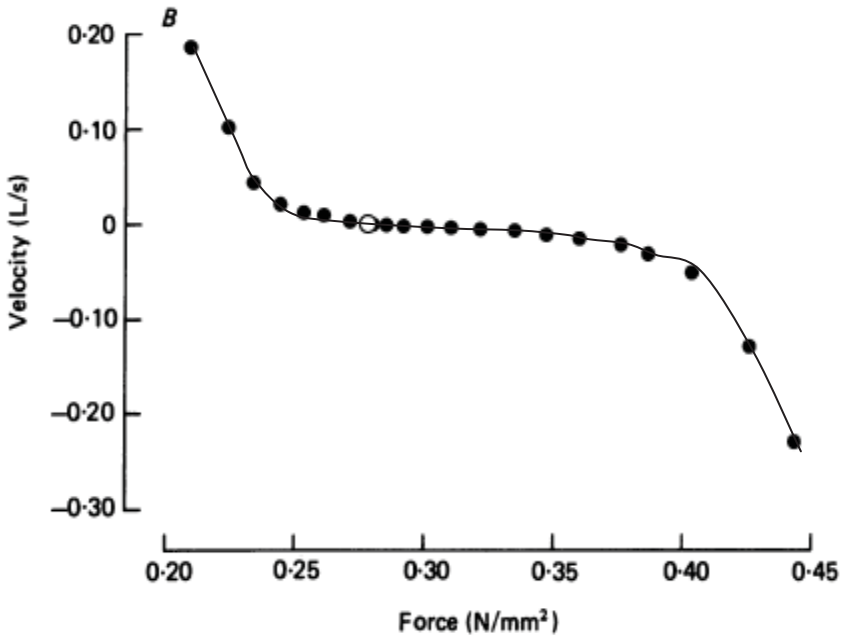

Figure 3 Force velocity curve in a single muscle fibre from a frog (adapted from Edman 1988).

Similarly, eccentric force generated during voluntary contraction in a range of human muscle groups is generally greater than or equal to isometric force (Doss and Karpovich 1965; Griffin 1987; Linnamo et al. 2006; Walmsley et al. 1986). However, unlike the force - velocity curve in isolated fibres, this relationship is less clear during maximal voluntary contraction (MVC). A number of studies have shown that eccentric force is independent of lengthening velocity (Cress et al. 1992; Pinniger et al. 2003; Westing et al. 1990; Westing et al. 1988) while others have found that force

increases as the lengthening velocity increases (Colliander and Tesch 1989; de Ruiter et al. 2000; Walmsley et al. 1986). To confound matters further, several studies have shown that as velocity increases there is an initial increase in eccentric force production, however force then decreases towards (Griffin 1987) or below (Singh and Karpovich 1966) isometric values as the rate of lengthening continues to rise. These contradictory results illustrate the complexity and muscular specificity that appears to control eccentric force production at different movement velocities. Further, the strength (Hortobágyi and Katch 1990) and sex (Colliander and Tesch 1989) of the individuals being tested also appears to play a role in how eccentric force is produced.

2.2.2.2 Neural strategies during eccentric contraction

When electrical stimulation is used to produce a supra-maximal eccentric contraction the force velocity curve more closely represents that seen in isolated muscle fibres (Seger and Thorstensson 2000; Westing et al. 1990). That truly maximal forces are unable to be generated during eccentric contraction suggests that neural inhibition may limit force production as the velocity of lengthening increases (Seger and Thorstensson 2000; Westing et al. 1991; Westing et al. 1990; Westing et al. 1988). Additionally, electromyographic (EMG) activity (Grabiner and Owings 2002; Kellis and Baltzopoulos 1998; Komi et al. 2000; Westing et al. 1991) and voluntary activation (VA) (Babault et al. 2001; Beltman et al. 2004) during eccentric contraction are lower than that produced during concentric contraction at the same velocity, even though the force being generated eccentrically is substantially higher. The level of muscle activation appears to be set prior to the onset of muscular contraction and related changes in muscle length (Grabiner and Owings 2002) suggesting the CNS utilises unique methods of recruitment during eccentric contraction compared to the other modes of contraction (Enoka 1996). Additional evidence comes from the findings of Fang et al. (2001, 2004) whose works shows that the brain requires more time and greater levels of activity to prepare and carry out eccentric contractions at both maximal and submaximal intensities compared to concentric contraction, even though EMG is lower during eccentric movements. This extra activity and preparation time may play a role in limiting muscle activity, as mentioned above, so that the risk of injury associated with eccentric contraction is minimised (Fang et al. 2004).

A possible explanation for the force/EMG relationship seen during eccentric contraction is the preferential recruitment of high-threshold (type II) motor units (Nardone and Schieppati 1988). Recruitment usually follows a clearly defined order from low to high threshold motor units (Henneman et al. 1965). However Nardone and Schieppati (1988), Nardone et al. (1989) and Howell et al. (1995) have demonstrated that this order may be reversed during eccentric contractions. The majority of studies investigating the preferential recruitment of type II fibres have, however, failed to observe any change in the order of recruitment during eccentric contraction (Bawa and Jones 1999; Beltman et al. 2004; Komi et al. 2000; Kossev and Christova 1998; Pasquet et al. 2006; Søgaard et al. 1996; Stotz and Bawa 2001). It therefore appears that a reversal of Henneman's "size principle" is not a typical strategy in force development during eccentric contraction (Byrne et al. 2004). As with the other contraction types, the control strategy used during eccentric contraction appears to be task specific with changes in motor unit recruitment / de-recruitment and discharge rates dictating force production as required (Duchateau and Enoka 2008). The difference between eccentric compared to concentric and isometric force production is that, because of the greater force producing characteristics of the muscle while lengthening (as discussed below), motor unit recruitment and discharge rates are lower for any given force output (Kossev and Christova 1998; Moritani et al. 1987; Søgaard et al. 1996; Stotz and Bawa 2001).

2.2.2.3 Mechanisms of force production

The greater force observed during eccentric contraction, typically referred to as force enhancement during stretch (Edman 2010), is the result of specific cross-bridge dynamics and the contribution of the muscles elastic components in absorbing energy.

2.2.2.3.1 Cross-bridge contributions

As previously stated, in isolated fibres the lengthening arm of the force – velocity curve is characterised by two slopes, an initial steep slope that is velocity dependent and a flatter slope that is independent of lengthening velocity (Pinniger et al. 2006; Ranatunga et al. 2010; Roots et al. 2007). The initial steep rise in tension is believed to be due to increased strain on all cross-bridges as the load increases above isometric levels. Because lengthening at this point occurs rapidly cross-bridge cycling is unable to be completed and so all cross-bridges are extended (Pinniger et al. 2006).

The second slope is characterised by cross-bridge dynamics that are different to those seen during isometric and concentric contractions. According to Jones and Round (1990) stretch in the S2 region of the myosin filament contributes to eccentric force production up to a certain length, after which cross-bridge detachment occurs through mechanical processes rather than through the hydrolysis of ATP. That eccentric contraction relies less on ATP than the other forms of contraction has been known since the 1920s and provides evidence for the mechanical nature of cross-bridge detachment (Fenn 1924). Flitney and Hirst (1978) observed a rapid decrease in sarcomere stiffness, which is representative of the number of cross-bridges, as lengthening increases. Flitney and Hirst (1978) considered this "give" in the sarcomere to be the result of forcible detachment of cross-bridges that occurs when the sarcomere is stretched to a critical length. Similar results were seen by Edman (1999) and reinforced the view that a critical point is reached when cross-bridges may be dragged along the actin filament prior to detachment (Flitney and Hirst 1978; Lombardi and Piazzesi 1990). During lengthening, sarcomere stiffness increases as a result of rapid cross-bridge detachment and re-attachment (Lombardi and Piazzesi 1990; Linari et al. 2000). Cross-bridges that are forcibly detached may re-attach 200 times faster than cross-bridges that complete the normal cross-bridge cycle that involves splitting ATP (Lombardi and Piazzesi 1990). Additionally, the number of cross-bridges during lengthening has been shown to be greater than that of isometric contractions and it has been theorised that both heads of the myosin molecule may be active at the same time thus individually contributing to force development (Linari et al. 2000). Further, pre-power stroke cross-bridges significantly contribute to the tension that occurs during lengthening due to their lower force state which may allow them to be stretched to longer lengths than cross-bridges in the post-power stroke state (Pinniger et al. 2006; Ranatunga et al. 2010; Rassier 2008). It therefore appears that cross-bridges contribute to eccentric force production through a combination of stretch in the S2 region, greater cross-bridge numbers and increased rates of detachment and re-attachment.

An alternative mechanism to that provided by the evidence above is the model Morgan (1990) proposed that relies on the stretching, and subsequent "popping", of sarcomeres. In this model, as the muscle is stretched the weaker sarcomeres lengthen rapidly and "pop". Tension is then taken up by the next weakest sarcomeres which

have a greater yield tension, thus increasing total tension in the myofibril. Initially, a steep rise in force will be observed during lengthening as weaker sarcomeres no longer contribute to force development, however as the muscle lengthens further the yield tension difference between sarcomeres is less and so force begins to plateau. Although this model fits the lengthening region of the force - velocity curve nicely recent studies have failed to observe rapid lengthening and "popping" of sarcomeres during stretch. Several of these recent studies will be discussed below.

2.2.2.3.2 Passive tension

The classic length – tension relationship developed by Gordon et al. (1966) (Figure 4a) shows that the force produced by a muscle fibre is dependent on the overlap of the thick (myosin) and thin (actin) filaments of a sarcomere. Starting from a maximally shortened length, force rises to a plateau as the overlap increases until the greatest number of cross-bridges are able to be formed. As the fibre continues to be lengthened the overlap begins to decrease, the number of sites for myosin – actin interaction is reduced and, correspondingly, force production decreases. This model has been shown to be accurate for whole muscle (Maganaris 2001; Marginson and Eston 2001) when isometric contractions at a range of angles are used to develop the length – tension (angle – torque) curve. However the classic length – tension curve is not representative of force production in isolated fibres (Figure 4b), single sarcomeres and whole muscle (Figure 4c).

The difference in the length tension relationship observed during eccentric contractions, compared to the isometric length tension relationship, is a result of passive components within the sarcomere, cytoskeleton, connective tissue and tendon (Brughelli and Cronin 2007).

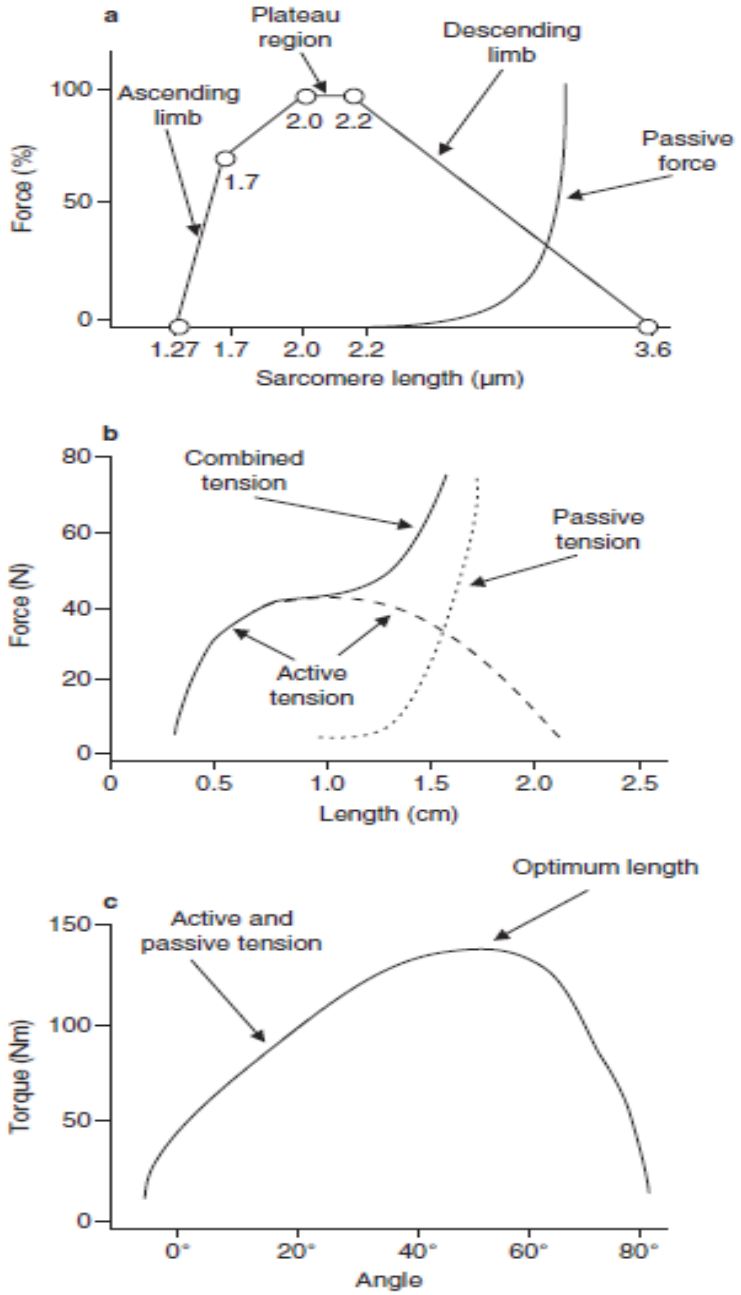

Figure 4 Length – tension relationships of isolated frog skeletal muscle fibres (a), whole muscle (b) length tension curves and single joint torque – angle curves (Brughelli and Cronin 2007).

At the sarcomere level the extra tension observed during active stretch is provided, almost exclusively, by the large sarcomeric protein titin (Linke et al. 1996). Titin spans the entire length of the half sarcomere, from Z – disk to M – line, thus limiting length change in half sarcomeres and maintaining sarcomere integrity. The contribution titin makes to tension appears to be dependent on activation by Ca^{2+} and cross – bridge cycling. At very long sarcomere lengths, when actin – myosin overlap is non – existent, activation of titin results in force development 3 to 4 times greater than forces generated passively (Leonard and Herzog 2010). Additionally, deletion of titin leads to a complete loss of force, both passive and active, in stretched myofibrils (Leonard et al. 2010).

Recently, Nishikawa et al. (2012) introduced the "winding filament hypothesis" which proposes that rotation of the actin filament during the cross – bridge cycle results in titin winding around the thin filament so that its length shortens and stiffness increases. Then, when the sarcomere is lengthened, the PEVK region of titin, which acts like a spring, would become stretched leading to storage of elastic potential energy and force enhancement. Currently no direct experimental evidence exists to support this theory; however the results of computer modelling have produced a length – tension curve very similar to those obtained experimentally in isolated fibres.

2.2.3 EIMD related force loss

A number of measures have been used in an attempt to quantify the magnitude of muscle damage and the timeline of recovery after damaging bouts of exercise. Many of these measures have become standard practice in research relating to EIMD and include, but are not limited to, changes in muscle function, elevations in circulating levels of a number of myofibril proteins including creatine kinase (CK) and myoglobin, delayed onset of muscle soreness (DOMS), swelling, and altered range of motion. The use of these measures and their relevance for investigating EIMD was reviewed by Warren et al. (1999b). This document has been cited over 300 times since it was published and is still commonly referred to when justifying the choice of measures used when studying EIMD.

Given the implications of impaired muscle function on everyday tasks as well as athletic training and competition, measures of muscle function are widely regarded as

the most important factor associated with EIMD (Twist and Eston 2005; Warren et al. 1999b). While elevations in circulating levels of myofibril proteins and soreness are observed for days after eccentric exercise these changes are often poorly related to alterations in muscle function and therefore may be unreliable indicators of how a muscle has been affected by EIMD (Nosaka et al. 2002; Warren et al. 1999b). Further, the importance and relevance of other, non-functional measures of EIMD must be questioned if observed in the absence of altered muscle function. The remainder of this section will therefore focus on the structural and functional changes associated with EIMD and the effect these alterations have on voluntary force production and other functional performance measures.

2.2.3.1 Mechanisms of force loss

Decrements in voluntary force production occurring as a result of EIMD may be caused by alterations either proximal (CNS) or distal (peripheral/muscle) to the NMJ. Historically, changes at the muscle level have been implicated as the cause of the significant loss in force seen in the hours and days after eccentric exercise with two theories, sarcomere over stretching (Proske and Morgan 2001) and EC uncoupling (Warren et al. 2001) predominating. Additionally, alterations in CNS function may also contribute to force loss (Prasartwuth et al. 2005).

2.2.3.1.1 Structural changes

It is generally accepted that mechanical damage is responsible for initiating the sequence of events that lead to significant losses in force after damaging exercise (Byrne et al. 2004; Friden and Lieber 2001; Grabiner 2008; Lieber and Fridén 1999; Proske and Allen 2005; Warren et al. 2001), however a consistent model of events is yet to be determined.

Sub-cellular damage to skeletal muscle has been observed within the first 5 – 15 minutes of eccentric exercise and is typically characterised by disrupted sarcomeres, Z – disk streaming and extension into adjacent A-bands, A-band widening and fibre enlargement (Fridén and Lieber 1998; Lieber et al. 1996; Lieber et al. 1991; McCulley and Faulkner 1985; Takekura et al. 2001; Wood et al. 1993). Figure 5 demonstrates the commonly observed morphological abnormalities found after eccentric exercise.

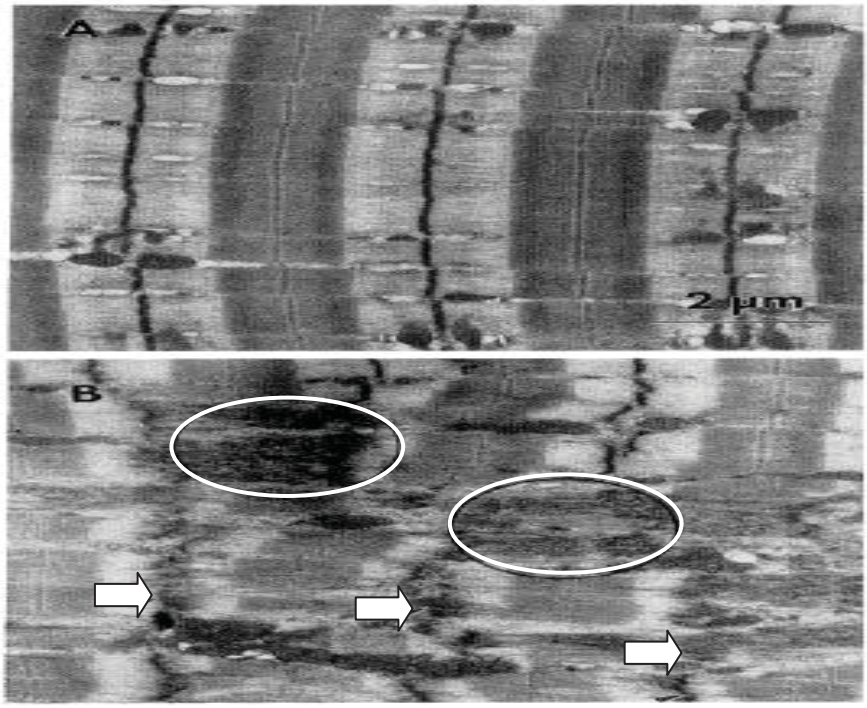

Figure 5 Electron micrograph of rabbit tibialis anterior muscle subjected to isometric (A) or eccentric (B) contractions. No change to normal morphology is evident after isometric contraction however Z – disk streaming and smearing (arrows) and extension of Z – disks into adjacent A – bands (circled areas) is clearly evident in muscle exercised eccentrically (adapted from Lieber et al. 1991).

Greater morphological changes have been consistently observed in type II fibres indicating that these fibres are selectively damaged during eccentric exercise (Fridén et al. 1983; Jones et al. 1986; Lieber and Fridén 1988; Lieber et al. 1991; Takekura et al. 2001). The reason for this phenomenon is unclear ,however it appears that the fibre - type specific composition of the cytoskeleton and myofilament lattice, such as a thinner Z – disk, may predispose type II fibres to greater damage during eccentric contractions (Choi and Widrick 2010). The Z – disks of type I fibres have approximately twice the desmin content of type II Z – disks (Barash et al. 2002) so this theory is plausible. Additionally, it has been suggested that the greater rate of fatigue in type II fibres during eccentric exercise may also make them susceptible to damage (Fridén and Leiber 1992).

2.2.3.1.2 The "popping sarcomere theory"

The most popular mechanism used to explain the structural changes and related losses in force associated with EIMD is the "popping sarcomere theory", as discussed above. Morgan (1990) suggests that the additional stress caused by the popping of sarcomeres along the length of the myofibril will result in direct damage to the sarcomere and may eventually lead to damage of the sarcoplasmic reticulum and / or sarcolemma (Morgan 1990; Morgan and Allen 1999; Proske and Morgan 2001).

There are several contentious issues surrounding the hypothesis that the "popping" of sarcomeres is responsible for structural damage and subsequent force loss. Firstly, the force loss observed after eccentric contraction, even when very high numbers of contractions occur, has been shown to be poorly related to the magnitude of histological damage (Hesselink et al. 1996; Lieber et al. 1991; Peters et al. 2003; Warren et al. 1993b). Secondly, recent advances in technology have provided a better understanding of how individual sarcomeres and half sarcomeres behave at different lengths during isometric and dynamic force production (Telley and Denoth 2007). The findings of a number of these studies challenge the "popping sarcomere theory" and have fuelled interesting debate (Herzog and Leonard 2007; Morgan and Proske 2006, 2007; Telley et al. 2006b).

In the first study to directly investigate whether over–stretched sarcomeres contribute to the force loss associated with lengthening contractions, Panchangam and Herzog (2011) found that although tension decreased significantly in isolated myofibrils, after repeated stretch - shortening cycles, these deficits were not related to sarcomere over extension. Also absent, and in agreement with the findings of Telley et al. (2006a) and Ranatunga et al. (2010), was the rapid, uncontrolled lengthening and "popping" of sarcomeres that is fundamental to the "popping sarcomere theory". Finally, those myofibrils that had over-extended sarcomeres had less tension loss than those lacking over-extension. The greater tension observed with sarcomere over- extension supports earlier findings (Leonard et al. 2010; Leonard and Herzog 2010) regarding titin's contribution to active force production during lengthening contractions. If sarcomeres do indeed "pop", although no direct evidence exists, the findings of these recent studies suggest that the force developed by a damaged muscle will be increased, not reduced as is typically seen with EIMD.

Although these studies do not directly show how changes in sarcomere dynamics contribute to force loss, Panchangam and Herzog (2011) propose that tension loss is the result of changes within the sarcomere such as a decrease in average cross – bridge tension or a decrease in the number of cross – bridges. Studies investigating EC coupling have shown that when damaged fibres are potentiated with caffeine, which increases $[Ca^{2+}]_i$ concentrations (Allen and Westerblad 1995), contraction takes place as usual (Balnave and Allen 1995; Ingalls et al. 1998b; Warren et al. 1993b). Therefore, although the mechanisms proposed for eccentric force development suggest cross – bridges are mechanically broken it appears that the ability of actin and myosin to interact is unaffected by eccentric exercise. With the development of new technology and direct evidence regarding the behaviour of sarcomeres during active lengthening it is likely that the "popping sarcomere theory" will, at some stage, be re - evaluated and perhaps upgraded to better fit findings such as those discussed above.

2.2.3.1.3 Direct damage to the sarcolemma

An alternative to the "popping sarcomere theory" is that the high levels of strain placed upon fibres, as a result of lower MU recruitment, results in direct damage to the sarcolemma during lengthening contractions. The release of CK and myoglobin into circulation (Warren et al. 1999), along with the influx of fibronectin into damaged muscle fibres (Fridén and Lieber 1998; Komulainen et al. 1998; Lovering and De Deyne 2004; Peters et al. 2003) suggests that sarcolemmal permeability is compromised by EIMD. If this is indeed the case an increase in $[Ca^{2+}]_i$ and intracellular sodium ($[Na^+]_i$) concentrations immediately after eccentric contractions would be expected, due to the electrochemical gradient that exists across the sarcolemma (Allen et al. 2010). However, although $[Ca^{2+}]_i$ and $[Na^+]_i$ are increased 10 – 20 minutes after eccentric exercise, this increase is not immediate (Balnave and Allen 1995; Yeung et al. 2003) as would be expected given the rapid damaging effects of eccentric contractions (Lieber et al. 1996). Similarly, movement of CK and fibronectin across the sarcolemma is greatest hours and days after eccentric exercise ceases.

Also, if sarcolemmal integrity is significantly affected by eccentric contraction then the propagation of the end plate potential to the T-tubule system would likely be

impaired and the resting membrane potential of the fibre would be lowered. This is generally not the case with a number of studies failing to see a reduction in EMG activity in the hours and days after damaging exercise (Deschenes et al. 2000; Hubal et al. 2007; Piitulainen et al. 2008; Warren et al. 1999a). Conversely, others have reported reductions in action potential conductance velocities after eccentric exercise, particularly in type II fibres, providing evidence that sarcolemmal integrity may in fact be affected by the damaging process (Piitulainen et al. 2010). These changes, however, where only observed 2 h post - exercise and therefore may reflect fibre type specific fatigue brought about by the exercise protocol rather than EIMD. The weight of evidence therefore suggests that if eccentric exercise affects the sarcolemma its electrochemical properties are unaffected. Any damage to the sarcolemma may be resolved fairly rapidly, within a minute (Bansal et al. 2003), so that its functional characteristics may essentially remain unchanged.

2.2.3.1.4 Ca^{2+} mediated muscle damage

Elevations in $[Ca^{2+}]_i$ have been observed after eccentric exercise in human and animal skeletal muscle (Allen et al. 2005; Allen et al. 2010). Such elevations have been shown to activate calpain proteases, in particular μ - calpain which is capable of cleaving dystrophin, desmin and titin *in vitro* (Murphy 2010; Verburg et al. 2009). Recently, Raastad et al. (2010) demonstrated that calpain activity is elevated in human muscle 30 minutes after eccentric exercise and remains elevated for up to 96 h. Elevations in calpain activity soon after eccentric exercise may be responsible for the loss of desmin, dystrophin and titin observed after eccentric contractions *in vitro* (Zhang et al. 2008) and *in vivo* (Barash et al. 2002; Beaton et al. 2002; Crenshaw et al. 1994; Lovering and De Deyne 2004; Peters et al. 2003; Trappe et al. 2002). However, unlike desmin, the early loss in dystrophin *in vivo* is not necessarily caused by calpian activity, as treatment with a calcium channel blocker does not appear to limit the loss of dystrophin at any time point post - eccentric exercise (Beaton et al 2008). A significant loss in dystrophin has been shown to occur within minutes of eccentric exercise and may last up to 21 days post exercise (Beaton et al. 2002; Komulainen et al. 1998; Lovering and De Deyne 2004). Lovering and De Deyne (2004) suggest that the mechanical strain that occurs during lengthening contractions could directly damage proteins involved in force transduction, in particular dystrophin. If dystrophin is damaged soon after the onset of eccentric exercise its loss

may affect membrane permeability through alterations in stretch activated and Ca^{2+} leak channels (Turner et al. 1991; Yeung et al. 2005) resulting in the gradual elevation in $[Ca^{2+}]_i$ that has been observed over the first 30 minutes after exercise (Balnave and Allen 1995; Yeung et al. 2005). The resulting elevated $[Ca^{2+}]_i$ will then bring about an increase in calpain activity which in turn sets about degrading desmin and titin.

The loss of desmin after eccentric exercise has been extensively reported (Barash et al. 2002; Beaton et al. 2002; Fridén and Lieber 1998; Komulainen et al. 1998; Lieber et al. 1996; Lovering and De Deyne 2004; Peters et al. 2003; Zhang et al. 2008) however the time line of changes varies between studies making it difficult to predict whether losses in desmin are the direct result of lengthening contraction or due to the secondary actions of calpain proteolysis. Similar to losses in dystrophin, decreases in desmin have been observed as early as 5 minutes after eccentric exercise (Lieber et al. 1996; Zhang et al. 2008) however, generally, the number of fibres displaying substantial desmin loss peaks between 4 and 12 h post –exercise (Barash et al. 2002; Beaton et al. 2002; Komulainen et al. 1998; Peters et al. 2003). That desmin loss is greatest several hours after the end of exercise supports the theory that changes in desmin content are calpain mediated.

Titin degradation has also been observed soon after eccentric exercise (Fridén and Lieber 1998; Zhang et al. 2008) as well as 24 h post – exercise (Crenshaw et al. 1994; Trappe et al. 2002). As with the losses in dystrophin and desmin, it is unclear whether the proteolytic action of calpain degrades titin or if this process is necessary for the removal of structural proteins that have been directly damaged as a result of mechanical strain. Such a process would be essential for the regeneration of new proteins.

Komulainen et al. (1998) found that, along with losses in dystrophin and desmin, damaged fibres displayed disorganised actin staining, however others have failed to see any change in actin content (Barash et al. 2002). Changes in actin may reflect the degradation of nebulin, which has also been shown to be degraded 24 h post-eccentric exercise (Trappe et al. 2002). Similarly, but on a different time scale, the actin content of damaged muscles is reduced significantly up to 14 days post-eccentric exercise (Ingalls et al. 1998a). As changes in actin occur later than the loss of dystrophin and

desmin, these findings led Komulainen et al. (1998) to hypothesise that the commonly observed deformation of contractile proteins is the result of stretch induced changes in sarcolemmal and cytoskeletal structures, rather than direct damage to the sarcomere. The findings of others, who have shown that alterations in cytoskeletal structure precede disruption to contractile proteins, go some way to confirming this theory (Fridén et al. 1983a; Lieber et al. 1991; Newham et al. 1983a).

Along with activating calpains, increased $[Ca^{2+}]_i$ is thought to activate phospholipase A_2 (PLA_2) which, along with loses in cytoskeletal proteins, may play a significant role in reducing membrane permeability, leading to further elevations in $[Ca^{2+}]_i$ and release of myofibrillar proteins into the interstitium thence to the circulation (Allen et al. 2010; Gissel and Clausen 2001).

Given the role of titin in passive force development and desmin and dystrophin in lateral and longitudinal force transmission (Bloch and Gonzalez-Serratos 2003; Monti et al. 1999), the loss of the structural proteins discussed here are likely to contribute to the morphological deformities seen after eccentric exercise (Figure 5). It could be expected that such radical changes in a number of proteins essential to the transmission of force, both radially and longitudinally, would significantly contribute to a loss in tension however this is not typically the case (Hesselink et al. 1996; Ingalls et al. 1998a; McCulley and Faulkner 1985; Peters et al. 2003).

2.2.3.1.5 EC uncoupling

In the absence of a definitive link between histological evidence of damage and changes in muscle function, Warren et al. (2001) propose that alterations in EC coupling are responsible for the losses in force that occurs when a muscle is damaged eccentrically. Failure of this sequence of events, at any point in the process, would result in sub-optimal Ca^{2+} release from the SR and a decrease in the potential for actin–myosin interaction.

The use of isolated fibres from muscles damaged *in vivo* has shown that, when stimulated, damaged fibres have a reduced rate of Ca^{2+} release from the SR and decreased tetanic force. This loss in force can be reversed by the application of caffeine indicating that changes in EC coupling, rather than changes in contractile

protein function, are at least partly responsible for force loss with EIMD (Balnave and Allen 1995; Ingalls et al. 1998b; Warren et al. 1993b). It appears that EC uncoupling occurs at or before the point where dihydropyridine receptors trigger Ca^{2+} release by ryanodine receptors, most likely at the triad junction, an area where the T-tubule is flanked by the SR on either side (Lamb 2009). Elevations in resting $[Ca^{2+}]_i$, as observed with EIMD (Balnave and Allen 1995; Ingalls et al. 1998b; Lynch et al. 1997), have been shown to reduce T-tubule depolarisation thus limiting Ca^{2+} release from the SR (Lamb et al. 1995; Verburg et al. 2006). Additionally, this rise in $[Ca^{2+}]_i$ may lead to an increase in calpain 3, which is able to disrupt Ca^{2+} release from the SR, as well as cleaving titin (Verburg et al. 2005). The mechanism responsible for increased $[Ca^{2+}]_i$ is not precisely known however it is believed that leakage of Ca^{2+} from within the SR and influx from the T- tubule system (Lamb 2009), possibly due to morphological changes brought about by mechanical damage to the T-tubule system and SR membrane (Friden and Lieber 2001; Takekura et al. 2001), are involved in this process. Recently, a significant relationship between damage to junctophilins, which are thought to be the primary proteins involved in localisation of triadic membranes, and EC uncoupling, and subsequent force loss, have been reported by Corona et al. (2010). An increase in membrane permeability, which itself may be due to increased $[Ca^{2+}]_i$ and related proteolysis, as discussed above, is also likely to contribute to increased resting $[Ca^{2+}]_i$ (Allen et al. 2005). The mechanism for EC uncoupling presented here may account for the long lasting low frequency fatigue (LFF) commonly seen after strenuous eccentric exercise.

2.2.3.1.6 Low frequency fatigue

Jones (1996) defines LFF as a long lasting loss in force at low, compared to high, frequencies of stimulation in the absence of metabolic or electrical changes in the muscle. The presence of LFF is most commonly associated with eccentric exercise, rather than exercise comprising concentric or isometric contractions (Newham et al. 1983b; Rijkelijkhuizen et al. 2003), although it has also been observed after isometric exercise at long muscle lengths (Jones et al. 1989). Its association with eccentric exercise and, in particular, EIMD has resulted in measures of LFF being widely used to investigate changes in EIMD related EC uncoupling (Avela et al. 2001; Brown et al. 1997b; Brown et al. 1996; Dundon et al. 2008; Jones et al. 1989; Nielsen et al. 2005; Sargeant and Dolan 1987; Skurvydas et al. 2006). The comparison of force

produced by percutaneous electrical stimulation (PES) at low frequencies, usually between 10 and 50 Hz, with force generated at high frequencies, 80 – 100 Hz, provides a valid and reliable measure of LFF; a comparison of the force produced at 20 Hz with that at 100 Hz is particularly common as it provides a good indication of the force frequency relationship (Brown et al. 1997a; Brown et al. 1997b; Brown et al. 1996; Dundon et al. 2008; Jones et al. 1989). The decrease in the ratio between low and high frequency forces, indicating the presence of LFF, may last up to 3 or 4 days post-eccentric exercise (Brown et al. 1997b; Brown et al. 1996; Jones et al. 1989; Verster and Roehrs 2007).

Westerblad et al. (1993) provided a possible mechanism for LFF, and confirmed the theory of Jones (1981) that LFF was related to EC uncoupling, by illustrating that reductions in $[Ca^{2+}]_i$ are directly related to reductions in tetanic force generation at low frequencies of stimulation. Further, Balnave and Allen (1995) showed that the LFF seen in isolated fibres after repeated stretch was due to a decrease in Ca^{2+} release implicating changes in the SR as the cause of LFF. Similarly, the release rate of Ca^{2+} from the SR has been found to be closely related to the magnitude of LFF and decrease in twitch forces observed after lengthening contractions (Nielsen et al. 2005). These studies, along with those discussed above, have narrowed the location of EC coupling failure to an area in the proximity of the T-tubule voltage sensors and the SR Ca^{2+} release channels (Westerblad et al. 2000).

The sigmoidal force - $[Ca^{2+}]_i$ relationship means that a reduction in $[Ca^{2+}]_i$ during low frequency stimulation will have a profound effect on force production due to the steepness of the curve at low concentrations. On the other hand, changes in $[Ca^{2+}]_i$ will have less effect on force production during high frequency stimulation because of the relative flatness of the curve at high $[Ca^{2+}]_i$ (Jones 1996; Westerblad et al. 2000). The functional implications of these changes may include a reduced ability to generate forces in response to low frequency voluntary activation. Given motor unit discharge rates are typically low (< 30 Hz) it could be predicted that LFF would have a considerable impact on muscle function during all levels of force production (Keeton and Binder-Macleod 2006), as occurs with EIMD. To compensate for this loss in force generating ability greater motor unit recruitment and increased firing

rates may be necessary to make up for the short fall in force (de Ruiter et al. 2005; Dundon et al. 2008).

2.2.3.1.7 Altered CNS function

A number of alterations in CNS function have been observed after eccentric exercise. Firstly, VA, the ability to activate a muscle to produce maximal force (Taylor et al. 2006), may be depressed in the days after eccentric exercise (Endoh et al. 2005; Prasartwuth et al. 2006; Prasartwuth et al. 2005; Racinais et al. 2008). Equally, others (Hubal et al. 2007; Newham et al. 1987; Saxton and Donnelly 1996) have failed to see a change in VA leading them to conclude that EIMD related force loss is due solely to peripherally based changes such as those discussed above.

The use of transcranial magnetic stimulation (TMS), PES or direct motor nerve stimulation allows for a comparison of the force/torque generated voluntarily to that produced with an interpolated stimulus. If an additional force is observed as a result of the interpolated stimulus then it is assumed that VA is incomplete and that central fatigue may be present (Taylor et al. 2006). The equation below provides a reliable measure of voluntary activation:

$$\text{Voluntary activation (\%)} = (1 - a / b) \times 100$$

where a is the maximum force from the interpolated stimulus and b is the force generated by the same stimulus train in the resting muscle (Herbert and Gandevia 1999).

Long lasting, significant reductions, ranging from 5.3 % (Racinais et al. 2008) to 20 % (Prasartwuth et al 2005; Prasartwuth et al. 2006), in VA have been detected during maximal voluntary isometric contractions (MVIC) of short (Prasartwuth et al. 2005; Prasartwuth et al. 2006; Racinais et al. 2008) and long duration (Endoh et al. 2005). The differing responses obtained by stimulating the motor cortex and motor nerve indicate that VA is impaired through inhibition at the motor cortex and/or motoneuron level (Prasartwuth et al. 2005). Similarly, persistent changes in VA have been shown to be supraspinal in origin, rather than due to alterations at the spinal level (Racinais et al. 2008), with specific increases in excitability of intracortical inhibitory pathways

reported by Endoh et al. (2005). Whether these changes are related to DOMS is unclear as the timeline of changes in VA and subjective ratings of pain have differed between studies (Prasartwuth et al. 2005; Racinais et al. 2008). Experimentally induced pain has been shown to reduce MVIC via a centrally mediated mechanism (Graven Nielsen et al. 2002) which may include inhibition of cortical and spinal motoneurons (Le Pera et al. 2001). Therefore, it is possible that sensations of pain play some part in the reduced VA discussed here.

Similarly, sensations of pain are also likely to contribute to the altered perceptions of effort observed in the damaged state. During force matching tasks subjects regularly fail to generate the target force/torque for a given level of effort or, alternatively, greater levels of effort are required to produce a given amount of force. The failure to sense effort, rather than a reduced ability to sense muscle tension, is responsible for this inaccuracy and is most likely the result of alterations in excitability of the motor cortex (Carson et al. 2002; Proske et al. 2003; Proske et al. 2004; Weerakkody et al. 2003). Group III and IV muscle afferents inhibit neural activity upstream of the motor cortex therefore it is likely that an increase in activity of these sensory fibres is behind the alterations in perception reported in these studies (Carson et al. 2002). Further supporting this theory is the finding that experimentally induced pain produces a similar reduction in force matching ability (Weerakkody et al. 2003; Proske et al. 2003). Changes in the perception of effort could contribute to the reductions in VA discussed above; subjects believe they are giving a maximal effort during MVIC however the additional force produced by an interpolated stimulus suggests otherwise.

During sub-maximal force matching a larger EMG signal is produced for a given force output indicating that a greater neural output is required to generate the target force (Carson et al. 2002; Dartnall et al. 2008; Proske et al. 2004). Likewise, neuromuscular efficiency, the relationship between force and EMG (N/mV), is altered during MVIC with EIMD so that less force is produced for a given EMG signal (Deschenes et al. 2000). This is most likely due to LFF so that, although EMG is either greater or the same as pre-exercise values, EC uncoupling causes a reduction in force (Dundon et al. 2008). Increases in EMG may be representative of an increase in MU recruitment earlier in the contraction and/or an increased rate of MU

synchronisation and firing frequency (Proske et al. 2004). Indeed, significant changes in MU behaviour have been observed with EIMD.

Reductions in MU recruitment thresholds, the force at which a MU discharges, along with increases in MU discharge rates (Dartnall et al. 2009) and MU synchronisation (Dartnall et al. 2008) have been observed 24 h after damaging exercise. As mentioned earlier these changes may be a necessary strategy to overcome LFF so that appropriate force production is achieved. The mechanisms behind these changes are not clearly understood however it is believed that, as with the other EIMD related changes in CNS behaviour, feedback from group III and IV afferents may be involved (Dartnall et al. 2009).

2.2.3.1.7 Inflammation, repair and regeneration

Before the regeneration and repair process begins further degradation and tissue necrosis occurs via an inflammatory response initiated by changes to the sarcolemma (Charge and Rudnicki 2004). This response is accompanied by oedema which produces a measureable increase in muscle cross sectional area (Chleboun et al. 1998; Takahashi et al. 1994; Takekura et al. 2001). Within an hour of damage, neutrophils, the first of the inflammatory cells to infiltrate the damaged tissue, start to degrade cellular debris, including disrupted saromeric proteins, cytosolic organelles and plasmalemma, through a combination of phagocytosis, the production of proteases and the release of cytotoxic and cytolytic molecules, such as superoxide (Newham et al. 1983a; Peake et al. 2005; Tidball 2005). Phagocytic degradation of muscle tissue, in particular contractile proteins, around the site of damage is believed to contribute further to the already lowered force generating ability of the effected muscle fibres (Faulkner et al. 1993; MacIntyre et al. 1996). Approximately 48 h after the initial neutrophil response macrophages enter the damaged tissue and continue the removal of cellular debris. More importantly, macrophages are thought to play an essential role in the repair process as they are associated with a wide range of growth factors and cytokines (Tidball, 2005).

Once muscular degeneration is complete the process of regeneration is initiated by changes in the activity levels of a number of trophic factors including IL-6, TGF- β, hepatocyte growth factor, TNF-α and IGF-1 (Charge & Rudnicki, 2004). The

combined actions of these and a number of other proteins bring about activation of muscle satellite cells. Myogenic proliferation and differentiation leads to the development of new muscle fibres which fuse with damaged fibres so that, once the new fibres increase in size, no morphological or functional difference is evident between old and new fibres (Charge & Rudnicki, 2004).

2.2.3.1.8 The sequence of events

The precise sequence of events that occur after eccentric exercise is contentious and extremely difficult to fully understand given the complexity and multi-factorial nature of the issue. Several models have been put forth however, from the evidence provided above, the model proposed by Warren et al. (2001) may be the most accurate at this time. Incorporating the timeline of events and the relative contribution each factor has on force loss, Warren et al. (2001) suggest that strength loss over the first three days is predominantly (≥ 75 %) caused by EC uncoupling while structural changes to force-bearing elements account for the remaining loss in strength. By day three degradation of contractile proteins (40–50 %) as a result of ongoing calpain activity and the inflammatory process, along with continued EC uncoupling, are the main contributors to continued force decrements. Between day 5 and day 14 EC uncoupling is resolved and contractile protein loss is solely responsible for any remaining loss of strength. Force generation returns to pre-exercise levels approximately 14 days after exercise.

Given the contribution structural damage is likely to make to EC uncoupling this model is very similar to that set forth by Proske and Allen (2005) in which structural damage initiates the uncontrolled release of Ca^{2+} leading to decreases in force development. While Proske and Allen (2005) support the theory that changes in Ca^{2+} homeostasis play a major role in events they also attribute early force loss to the "popping sarcomere theory" which, as has been discussed, has little direct experimental foundation.

Not considered in either model is the contribution altered CNS function makes to force loss in response to EIMD. It is likely that EC uncoupling, damage to force bearing proteins and changes in CNS function combine to reduce force production with each factor contributing to a greater or lesser extent over the post – exercise period.

2.2.3.2 Implications of EIMD on force production

Reductions in the ability to voluntarily generate maximal force in the hours and days after strenuous, repetitive eccentric exercise have been widely reported in studies using a number of different eccentric exercise protocols (Byrne and Eston 2002a; Byrne et al. 2001; Davies and White 1981; Deschenes et al. 2000; Hamlin and Quigley 2001a; MacIntyre et al. 2001; Sargeant and Dolan 1987; Sayers and Clarkson 2001). The majority of studies have investigated changes in the force produced by MVIC at a single joint angle. Additionally, isokinetic concentric and eccentric torque production is also often reported (Warren et al. 1999b).

Few studies have directly compared the EIMD related response of the three contraction types however it appears that the magnitude of force loss and time to recovery are similar between maximal isometric, concentric and eccentric contractions (Byrne and Eston 2002a; Hortobágyi et al. 1998). Michaut et al. (2002) investigated the difference in force loss for all contraction types after eccentric exercise however, as they compared pre and immediately post–exercise values, their results only provide an indication of the effect eccentric exercise induced fatigue has on the different contraction types.

The general time line of force loss after high-force eccentric exercise is remarkably similar between studies, irrespective of the damaging protocol used. A reduction in force of between 45 and 76 % has been reported immediately post – exercise (Cleak and Eston 1992; MacIntyre et al. 2001; Newham et al. 1987; Nosaka et al. 2002) however the majority of this force loss is likely to be related to metabolic fatigue rather than EIMD (Nosaka et al. 2006; Warren et al. 1999b). Force decrements tend to peak around 24 h post – exercise with reductions in force ranging from 25 to 65 % observed at this time (Cleak and Eston 1992a; Nosaka et al. 2002; Sargeant and Dolan 1987). Significant, but incomplete, recovery is usually observed 5 days post – exercise however forces at this time may still be considerably less than pre –exercise values (MacIntyre et al. 2001). Force improves slowly towards baseline levels at a fairly linear rate until values are not different to those measured prior to exercise. In most cases, full recovery has been achieved within two weeks of exercise however very long lasting force loss has been found three to four weeks after exercise (Newham et al. 1987; Sayers and Clarkson 2001).

2.2.3.2.1 Shift in the length – tension relationship

EIMD results in a shift in the length – tension relationship of the damaged muscle so that the optimal length for force production occurs at longer muscle lengths (Brockett et al. 2001; Chen et al. 2007a; Child et al. 1998; Jones et al. 1997; Philippou et al. 2004; Prasartwuth et al. 2006; Saxton and Donnelly 1996; Yeung and Yeung 2008). Acute changes in the length – tension relationship tend to be relatively short lived with a return to pre-exercise values normally occurring within two weeks of exercise (Chen et al. 2007a; Philippou et al. 2004; Prasartwuth et al. 2006; Yeung and Yeung 2008). The reported shifts in optimal length range from 4^{o} (Jones et al. 1997) up to 18^{o} (Chen et al. 2007a; Philippou et al. 2004) and appear to depend on the intensity of the exercise used to bring about EIMD (Chen et al. 2007a; Philippou et al 2004). As a result of the shift in optimal length, the loss in force is not equal across the full range of motion with greater losses seen when the muscle is contracted at short lengths (Byrne and Eston 2002b; Chen et al. 2007a; Child et al. 1998; Saxton and Donnelly 1996). A comparison of voluntary and twitch torques at the pre - exercise angle of measurement and the new optimal angle suggests that the contribution of the shift to force loss is very small. Therefore, neglecting to take the shift in optimal length into account during subsequent post–exercise measures may result in only small underestimations of actual force loss, if only one joint angle is measured (Prasartwuth et al. 2006).

The mechanisms behind the acute changes in optimal length have not clearly been proven however several mechanisms have been suggested including sarcomereogenesis, a greater contribution from passive tension at long muscle lengths, disrupted sarcomeres and altered Ca^{2+} sensitivity (Brughelli and Cronin 2007).

2.2.3.2.2 Force – velocity relationship

As previously mentioned, a number of studies have illustrated that type II fibres are preferentially damaged by eccentric exercise (Fridén et al. 1983b; Jones et al. 1986; Lieber and Fridén 1988; Takekura et al. 2001). Given the greater force and velocity characteristics of these fibres it could be expected that torque production at high angular velocities would be affected to a greater extent than during contractions at low angular velocities. The findings of several early studies supported this theory by

demonstrating that the rate of torque recovery for concentric (Fridén et al. 1983b; Golden and Dudley 1992) and eccentric (Eston et al. 1996) contractions performed at high angular velocities is slower than that occurring for low angular velocities. This, however, is not necessarily always the case with Gibala et al. (1995) and Byrne et al. (2001) reporting that the magnitude and recovery of force loss is similar between MVIC and concentric contractions at high and low angular velocities. Alternatively, greater losses in torque and slower recovery have been found during concentric (Deschenes et al. 2000; Paddon-Jones et al. 2005) and eccentric (Paddon-Jones et al. 2005) contractions at low angular velocity, compared to faster rates of contraction.

2.2.3.2.3 Factors influencing the magnitude of force loss

The differences in the magnitude of force loss reported in the literature may be due to the different protocols used to bring about damage. It is not unreasonable to suggest that all damaging protocols are not equal in their ability to bring about damage to a group of target muscles. For example, it is possible that the completion of 300 eccentric contractions of the quadriceps on an isokinetic dynamometer (Barnes et al. 2010; McIntyre et al. 1996) or 100 barbell squats with a load of 80 % of subjects concentric one repetition maximum, as employed by Byrne and Eston (2002b), is more damaging than 150 unweighted walking lunge steps (Malm et al. 1999), 40 minutes of downhill walking (Nottle and Nosaka 2007) or 100 vertical jumps (Twist and Eston 2005; 2007). Indeed, research has revealed that a number of variables contribute to the severity of EIMD related force loss.

The high force and low motor unit recruitment that occurs during eccentric contraction is likely to place large mechanical strain on the sarcomeres and connective tissue responsible for generating force (Enoka 1996). Whether strain or force is principally responsible for EIMD, and associated losses in force, is unclear with findings confirming that each factor plays a role in the damaging process. The magnitude of strain a muscle is exposed to as it lengthens, rather than the high force being produced, has been suggested as the main contributing factor (Lieber and Fridén 1993), conversely others have shown that force loss, and therefore EIMD, is closely associated with the force generated during eccentric exercise (Deschenes et al. 2000; McCulley and Faulkner 1985; Warren et al. 1993a).

The length of the muscle during contraction also influences the magnitude of damage with muscles exercised at long lengths displaying greater changes in strength, and other characteristics associated with EIMD, than those exercised at shorter lengths (Child et al. 1998; McHugh and Pasiakos 2004; Newham et al. 1988; Nosaka and Sakamoto 2001; Panchangam et al. 2008; Paschalis et al. 2005a). Additionally, when time under tension is controlled for, the rate at which the muscle lengthens during eccentric work has also been shown to influence the magnitude of EIMD (Chapman et al. 2006). Finally, the number of eccentric contractions (Baker et al. 2007; Chapman et al. 2008; Hesselink et al. 1996; Nosaka et al. 2002) and intensity (Paschalis et al. 2005c) during the exercise bout also impacts the severity of damage seen in the days after exercise; more contractions and higher intensities result in greater levels of damage. Together these factors suggest that significant EIMD is likely to occur when a muscle repeatedly and rapidly lengthens under high force at long lengths, as occurs when the hamstring contracts eccentrically during sprinting (Yu et al. 2008), for example.

2.2.3.2.4 Implications of EIMD on functional performance

As discussed above, the effect of EIMD on voluntary force production is well established however the performance of an individual muscle group under isokinetic or isometric conditions may not be representative of muscle function during dynamic, multi-joint movement, as occurs during exercise and sport. This lack of application is behind the relatively recent increase in research investigating changes in a variety of functional performance measures after strenuous eccentric exercise.

2.2.3.2.4.1 Power output

While the generation of maximal forces during exercise is important, optimal power development, the rate at which force is generated, is a critical component of many sports (Duchateau and Hainaut 2003). The rate of force development and peak power output (PPO) have been suggested as key variables to sporting success, particularly in sports that rely heavily on strength and power (Stone et al. 2002) such as throwing and jumping sports and the contact football codes which include American football, rugby union, rugby league, Gaelic football and Australian Rules football. Stone et al. (2002) suggest that PPO is likely to be the most important attribute in determining the outcome of sporting performance. Given the relationship between power output and

force production (Power = force x distance / time) it is likely that any condition that limits the generation of force, such as EIMD, would directly impact PPO.

Research investigating the effect of EIMD on PPO during maximal anaerobic cycle tests, such as the 30 s Wingate anaerobic test, has been contradictory with a lack of change reported by Malm et al. (1999) and Nottle and Nosaka (2007). Conversely, a decrease in PPO in the days following damaging exercise has been reported by Sergeant and Dolan (1987), Byrne and Eston (2002b) and Twist and Eston (2005; 2007). Additionally, the latter study by Twist and Eston (2007) found that time to PPO was impaired 48 h after the completion of a repeated vertical jump protocol. The inability to produce peak power, particularly at the start of exercise, may be due to an inability to produce maximal forces as a result of a combination of peripheral and central mechanisms including the preferential damaging of type II fibres, decreased voluntary activation and EC coupling failure (Byrne and Eston 2002b; Twist and Eston 2007), as discussed above.

These incongruous results are difficult to explain given the corresponding changes in other measures commonly associated with EIMD, in particular decreases in voluntary force production and elevations in CK, reported by Nottle and Nosaka (2007), Byrne and Eston (2002b) and Twist and Eston (2005; 2007). It is worth noting that, although the subjects in Malm et al. (1999) reported significant levels of DOMS two days post exercise, no significant change in performance or CK was evident at any time post - exercise. Therefore it is unclear whether the protocol used was sufficient to bring about significant levels of EIMD.

More conclusive are the decrements in vertical jump height observed up to three days post-exercise. Vertical jump performance is closely related to sprinting speed (Cronin and Hansen 2005) and provides an accurate measure of lower body power output when combined with body mass and the appropriate regression equation (Harman et al. 1988; Sayers et al. 1999). While decreases in jump performance have been seen with squat, counter movement (CMJ) and drop jumps the magnitude and mechanism of change may differ between the three jump types (Byrne and Eston 2002a). Decrements in squat jump height, which relies more on force production than the other two jump types, appears to be most affected. This is possibly due to the lack of

contribution from the muscles elastic elements during this concentric only jump (Byrne and Eston 2002a; Marginson et al. 2005). On the other hand, decreases in drop jump performance (Byrne and Eston 2002a; Horita et al. 1999; Horita et al. 2003; Twist and Eston 2007), which relies on the stretch – shortening cycle, and accompanying increases in ground contact time (Twist and Eston 2007) have been observed up to 72 h post – exercise. Along with decrements in force production, changes in drop jump height may be impaired by the muscles decreased tolerance to impact and stretch. During the drop jump, for example, ground contact time may increase as a result of subjects attempting to dampen impact with the ground thus reducing the stretch reflex (Byrne and Eston 2002a; Twist and Eston 2007).

Reductions in power output provide some insight into the functional effects of EIMD; however few sports involve single bouts of explosive movement. Perhaps more relevant to most sports is how repeated power output is altered in the days following a bout of damaging exercise. Surprisingly, repeated (10 x 10 s) sprint cycle performance was found to improve in the days following a bout of damaging exercise (Malm et al. 1999). However, as previously stated, whether the exercise model used by Malm et al. (1999) was appropriate to bring about EIMD is somewhat questionable. Others have shown repeated sprint ability, both on a cycle ergometer (10 x 6 s) and on foot (10 x 10 m), to be negatively impacted up to 48 h after damaging exercise (Twist and Eston 2005). Similarly, speed over 10 m and agility, which is reliant on PPO (Stone et al. 2002), was also reduced in the presence of EIMD (Highton et al. 2009). Not surprisingly, the same mechanisms as those responsible for changes in PPO have been implicated in these reductions in repeated sprint and agility performance (Twist and Eston 2005; Highton et al. 2009). Irrespective of the mechanism it is clear that EIMD results in significant alterations in both one off and repeated power output; outcomes which are likely to have serious consequences for sports performance.

2.2.3.2.4.2 Running economy

As with force and power production alterations in running economy (RE), as measured by steady state oxygen consumption during fixed, sub-maximal running speeds, may negatively impact sports performance (Saunders et al. 2004). Once again the results of studies investigating this topic have proven contradictory with a number

of studies (Hamill et al. 1991; Marcora and Bosio 2007; Paschalis et al. 2005b; Vassilis et al. 2008) failing to see any change in RE after damaging exercise. Although Marcora and Bosio (2007) reported a decrease in the distance covered during a treadmill time trial this was unrelated to changes in RE, but instead appeared to be the result on an altered perception of effort. That is, subjects reported higher ratings of perceived exertion (RPE) at lower speeds when running after damaging exercise compared to pre-exercise values. This result is similar to the changes in force matching and sense of effort (Carson et al. 2002; Proske et al. 2004; Proske et al. 2003; Weerakkody et al. 2003) with EIMD reported earlier. Paschalis et al. (Paschalis et al. 2005b) and Vassilis et al. (2008) suggest that the lack of change in RE in their study may have been intensity related with sub-maximal running economy likely to be influenced by type I motor units whereas type II motor units may be preferentially recruited and therefore damaged during eccentric exercise. This implies that only at maximal intensities, such as during time trial performance, as observed by Marcora and Bosio (2005), would the impact of EIMD truly be seen. This may be a valid explanation however this does not fit with the findings of other studies, discussed below, that tested RE at a similar range of intensities. A more likely reason, as Paschalis et al. (2005) and Vassilis et al. (2008) also propose, is that the exercise used to bring about damage only targeted the quadriceps and therefore, because running involves many more muscles than just the quadriceps, this exercise protocol may not have been appropriate for use in investigating RE. This highlights the specific nature in which EIMD affects certain physical parameters and suggests that both the exercise model and performance measures used should be considered carefully when investigating the effects of EIMD on physical performance.

Contrary to the findings discussed above, Braun and Dutto (2003) and Chen et al. (2007b) found that RE may be reduced by between 3 and 7 % for up to three days post-exercise when downhill running is used to bring about EIMD. This affect was correlated with alterations in running form, in particular a decrease in stride length, stride frequency and joint range of motion, and occurred on a different time line to changes in muscle function. Chen et al. (2007b) speculate that reductions in RE may be due in part to impairment in the stretch–shortening cycle and increased recruitment of muscle fibres to compensate for those damaged during exercise. It is also possible that the reduced stride length and increased stride frequency reduces impact during

running, in a similar way that increasing the contact time during jumping does, so that the sensations of pain that are typically associated with EIMD are reduced.

2.2.3.2.4.3 Cycling performance

Endurance cycling performance is affected by EIMD. As with short duration sprint cycling (Bryne and Eston 2005; Twist and Eston 2005; 2007), PPO and average power output, and therefore distance covered, during five (Twist and Eston 2009) and 15 (Burt and Twist 2011) minute cycling time trials is reduced 48 h after damaging exercise. Similarly, cycling time to exhaustion is also reduced at the 48 h point (Davies et al. 2009). At sub-maximal intensities the VO_2 response to cycling is not altered by EIMD (Davies et al. 2009; Gleeson et al. 1995; Schneider et al. 2007; Twist and Eston 2009) suggesting that maximal, but not sub - maximal cycling is affected in the days after eccentric exercise. As with changes in running time trial performance (Marcora and Bosio 2007), reduced cycling performance appears to be closely related to altered perceptions of effort, at both sub-maximal (Twist and Eston 2009) and maximal intensities (Burt and Twist 2011; Davies et al. 2009; Twist and Eston 2009) such that subjects believe that they are performing at a higher intensity than they actually are. Together these findings indicate that EIMD may influence pacing ability during endurance cycling and running, an outcome that has direct implications for competition and training (Twist and Eston 2009).

2.2.4 Rugby Union

The sport of Rugby Union, or simply rugby as it will be referred to from here on, has a close and historic relationship with alcohol (Collins and Vamplew 2002). As previously discussed, rugby players are amongst the group of sports people who regularly consume hazardous amounts of alcohol through binge drinking behaviour (O'Brien and Lyons 2000; Quarrie et al. 1996). While the physical nature of rugby means that players are susceptible to muscle injury, through what is often violent contact with other players and/or the ground and as a result of eccentric muscular contraction, it is currently not known how the combination of alcohol and the physical stress induced by a rugby match impacts recovery in the days following the match.

2.2.4.1 Rugby – the sport

Globally over 5 million people in more than 117 countries play rugby. The sport has seen substantial growth over the last few years with large increases in playing numbers seen across Africa, South America, Asia and North America. Much of this growth is believed to be due to the inclusion of Rugby 7's in the 2016 Olympic Games and increased funding from rugby's governing body, the International Rugby Board, which has seen many countries starting to provide greater resources into development of the sport (Chadwick et al. 2010). Rugby is played at a number of levels starting with age grade competition through to the senior club level and on to elite provincial/regional and international levels. Rugby is played professionally in many countries with major professional competitions found in the United Kingdom, Australia, New Zealand, South Africa, Japan, France and Italy. Many of these countries have more than one level of professional competition such as in New Zealand where players are paid to play in the ITM Cup, Super 15 and, if selected, at the international level. The large variety of grades available to participants makes rugby a sport that caters to a large proportion of the population including the young, the old and males and females alike.

Unless the rules are modified to suit a particular population, for example in different age grades, a rugby match is contested over two 40 minute halves by teams of 15 players and seven reserve players, who can be used for tactical and injury related substitutions throughout the match. On the field, each team has five tight forwards, three loose forwards, four inside backs and three outside backs. Averaged height and weight data from Jarvis et al. (2009), Scott et al. (2003), Austin et al. (2011) and Holway and Garavaglia (2009) illustrates the typical trend in positional differences found at most levels of competition. Tight forwards (props, hooker and locks) are typically the tallest (average 183.4 cm) and heaviest (104 kg) players in the team, back row forwards (flankers and number 8) are the next largest (183.6 cm, 98.8 kg) while inside backs (178.8 cm, 84.8 kg) and outside backs (180.6 cm, 88.1 kg) tend to be shorter and lighter. These characteristics are dictated by the specific role each position has within the team.

2.2.4.2 Physical demands of rugby

The large volume of contact that occurs in rugby makes it difficult to directly measure the physiological demands of a match, particularly because of the potential danger associated with instrumenting players. That said, Deutsch et al. (1998), Doutreloux et al. (2002) and most recently Cunniffe et al. (2009) measured the heart rate (HR) response during colts (Under 19 age group), university and elite professional level competition, respectively. In their pilot study (n = 2) Cunniffe et al. (2009) found that the mean HR for the players (one back and one forward) being observed was 172 beats per minute across the 80 minutes of the match. During the match the forward spent more time in the 95 - 100 % HR max zone compared to the back, while the back spent more time in the 80 – 90 % HR max zone. Although the number of subjects observed by Cunniffe et al. (2009) was very small their findings are in line with the observations of Deutsch et al. (1998) and Doutreloux et al. (2002), who both reported that forwards spend longer periods of the game at or above 85 % heart rate maximum (HR max) compared to backs. In fact forwards may spend anywhere from 15 to 20 % of the match in the 95–100 % HR max zone, possibly as a result of their frequent involvement in rucks, mauls, scrums and tackles (Cunniffe et al. 2009; Deutsch et al. 1998).

While research using direct measures of intensity, such as HR and lactate, is scarce the use of time - motion analysis from video and more recently Global Positioning System has allowed researchers to quantify game demands indirectly. A summary of the results of such research is displayed in Table 4. The data presented in Table 4 represents mean data for forwards and backs with data from front row and back row forwards pooled and, similarly, data from inside and outside backs pooled together. There are differences, due to positional specificity, in the distances covered and time spent in each component of the game with forwards spending more time walking and jogging compared to backs, while backs spend relatively more time sprinting compared to forwards. Consistent with the HR data presented above, on the whole forwards spend significantly more of the match involved in high intensity activity, by way of their involvement in high numbers of contacts, compared to backs (Austin et al. 2011; Duthie et al. 2006). Analysis of work : rest ratios illustrates the intermittent nature of the sport with forwards typically spending less time resting than backs. Given the role of forwards is to contest for the ball at rucks, mauls and scrums this is

not surprising. Work : rest ratios have been shown to range from 1 : 2 – 1 : 7.4 for forwards and from 1 : 2.2 – 1 : 21.8 for backs (Austin et al. 2011; Deutsch et al. 2007; Deutsch et al. 1998). Austin et al. (2011) suggest that the large variation in reported work : rest ratios, particularly at the professional level, is most likely due to the changing nature of the sport as a result of recent rule changes. This has resulted in a faster game with shorter rest periods and an increase in the number of high intensity activities (Austin et al. 2011).

Table 4 Summary of the total distance covered, percentage of time spent in different phases of locomotion and the total number of contacts, including rucks, mauls, tackles and scrums, for forwards and (backs) during matches at various levels of competition. Classification of walking, jogging, striding and sprinting differs between studies however these components usually relate to speeds of approximately 0-6, 6-12, 12-18 and > 18 km/h, respectively. * Venter et al. (2011) included contacts with the ground in addition to those reported by others.

Author (Year)	Level of competition	Total distance (m)	Stand (%)	Walk (%)	Jog (%)	Cruise (%)	Sprint (%)	Number of contacts
Deutsch et al. (1998)	Under 19	4240 (5640)	45.0 (40.0)	45.9 (27.3)	20.0 (18.7)	1.9 (2.6)	0.4 (1.0)	108.0 (10.5)
Duthie et al. (2005)	Super 12	_	41.0 (41.0)	27.0 (38.0)	20.0 (16.0)	1.7 (2.1)	0.5 (1.5)	91.0 (30.0)
Deutsch et al. (2007)	Super 12	_	47.7 (41.1)	17.9 (32.4)	20.1 (17.3)	1.2 (2.5)	0.2 (0.6)	128.0 (32.5)
Roberts et al. (2008)	English Prem	5581 (6127)	31.0 (30.0)	35.0 (45.0)	16.7 (16.7)	3.9 (3.9)	0.4 (0.5)	89.0 (24.0)
Venter et al. (2011)	Under 19	4487 (4452)	22.3 (20.8)	44.5 (57.0)	24.8 (17.8)	7.8 (4.5)	0.4 (0.9)	683.0 (474.0) *
Austin et al. (2011)	Super 14	4960 (5422)	37.0 (32.5)	28.5 (41.0)	15.0 (14.5)	6.0 (7.5)	2.0 (4.0)	128.0 (32.5)

2.2.4.3 Physical cost of a rugby match

The physical demands of rugby would suggest that in the days after the match physical performance may be impaired due to a combination of factors including muscle soreness, injury and fatigue. Surprisingly very little is known about the effect a match has on measures of performance in the days after the event. In fact, unlike the similar contact football codes of rugby league (McLellan et al. 2011; Twist et al. 2011), American football (Hoffman et al. 2002) and Australian Rules football (Cormack et al. 2008; Dawson et al. 2005), to date it appears that no attempt has been made to quantify the physical cost of a rugby match or the relationship between game demands and decrements in performance.

The physiological and psychological stress brought about by a rugby match results in an acute phase inflammatory response characterised by elevations in IL-6 and cortisol in the hours immediately after the match (Cunniffe et al. 2010; Elloumi et al. 2003). Additionally, total leukocyte, neutrophil and monocyte numbers are elevated up to 38 h post-match, possibly in response to skeletal muscle damage (Cunniffe et al. 2010; Suzuki et al. 2004). Although immune cell concentrations are higher, neutrophil function is suppressed suggesting players may be more susceptible to infection during the post-match period (Cunniffe et al. 2010; Suzuki et al. 2004). On the other hand, a lack of change in salivary immunoglobulin A, a marker of mucosal immunity, after a rugby match suggests this may not necessarily be true (Koch et al. 2007). In fact the high training loads players experience throughout the competitive season, rather than the stresses of a match, may be responsible for increased rates of upper respiratory illness at particular times throughout the year (Cunniffe et al. 2011). More research is needed to provide a better understanding of the stress competitive rugby has on acute and chronic immune states at all levels of competition.

The increase in cortisol observed immediately post-match coincides with a decrease in testosterone resulting in an acute catabolic state. This change in the testosterone/cortisol ratio is likely to increase the availability of energy substrates during the important post-match recovery period. Up to five days after the match testosterone levels are elevated while at the same time cortisol levels are decreased (Cunniffe et al. 2010; Elloumi et al. 2003). Elloumi et al. (2003) suggest that this shift to an anabolic state occurs to restore homeostasis during an extended recovery period.

2.2.4.3.1 Match induced muscle damage

Several studies have investigated markers of stress after a rugby match with particular focus on CK in an attempt to quantify match related muscle damage (Banfi et al. 2007; Cunniffe et al. 2010; Gill et al. 2006; Smart et al. 2008; Suzuki et al. 2004; Takarada 2003). In the sport and exercise context, elevations in CK are typically associated with EIMD, however Zuliani et al. (1985) demonstrated that contact, by way of boxing impacts, results in significant elevations in CK. Similar findings have been reported in rugby with post-match elevations in CK found to be closely related to the number of contacts, in particular tackles, a player is involved in during the match (Smart et al. 2008; Takarada 2003). These findings are unsurprising given the wealth of information implicating contacts, especially tackles, as the main cause of musculoskeletal injuries in the sport (Bathgate et al. 2002; Brooks et al. 2005b; Fuller et al. 2008; Fuller et al. 2007; Quarrie and Hopkins 2008). Contusion injuries to the leg, including thigh haematomas, are the most common injury reported by both forwards and backs (Bathgate et al. 2002; Brooks et al. 2005b; Fuller et al. 2008). The thigh and lower leg areas are particularly susceptible to injury given the general aim of the tackler is to bring the ball carrier to ground as quickly as possible so that possession for the ball can be contested. The most effective way to do this is to tackle the player by wrapping both arms around the thighs or lower legs.

The contribution contact with the ground makes to match related muscle damage may also be substantial with van Rooyen (2008) recently reporting that this type of contact was the most prevalent in professional provincial rugby in South Africa, compared to scrum, lineout or tackle contacts. Similarly, a comparison between the contacts observed by Venter et al. (2011) and those seen in other studies (Table 2.4) highlights the high number of ground contacts made by players throughout a match. Unlike other types of contact, ground contact is likely to affect players in all positions as it may occur when tackling or being tackled, during rucks and mauls or simply through the player losing their footing. Contact with the ground may contribute more to muscle damage, and other injuries, early in the season when grounds are harder (Alsop et al. 2005; Alsop et al. 2000; Takemura et al. 2007) and players are less conditioned to match specific contacts.

Recently, Elmer and colleagues (2011) confirmed the findings of earlier work (Beiner et al. 1999; Crisco et al. 1996; Crisco et al. 1994) by demonstrating that experimentally induced contusion injuries result in significant reductions in isometric force, muscular power and work up to 48 h after the injurious event. Elmer et al. (2011) propose that type II fibres, which are more prevalent in superficial regions of the muscle, are affected the most by this type of injury. Therefore, it appears that the consequences of contusion injuries are very similar to those occurring with eccentric EIMD, namely reduced muscle function, elevations in CK and an acute inflammatory response (Smith et al. 2008). Further, Elmer et al. (2011) suggest that the mechanisms behind this loss in muscle function may be the same as those thought to be responsible for force deficits associated with EIMD, such as EC uncoupling and disruption to sarcomeric structure and function.

A third contributor to muscle damage during matches is repeated eccentric muscular contractions. To date there has been no attempt to directly quantify the frequency of eccentric loading during a match and therefore the contribution this type of contraction makes to match related muscle damage is unknown. However, given the frequent short sprints occurring during the match, which are likely to either end in the player rapidly decelerating, being tackled or evading the opposition by side-stepping/cutting, it is highly plausible that eccentric muscular contraction contributes to indirect measures of muscle damage, such as elevations in CK. Indeed, elevations in CK and DOMS have been reported after simulated team sport protocols that involve intermittent sprinting, as occurs during rugby, suggesting this type of activity, in the absence of match related contacts, is enough to bring about significant levels of muscle damage (Bailey et al. 2007; Magalhães et al. 2010; Thompson et al. 1999; Thompson et al. 2003).

Colby et al. (2000) found that large forces, in excess of 160% MVC, are produced eccentrically by the quadriceps when stopping, sidestep - cutting, cross - cutting and landing while sprinting. The rapid decelerations that occur during these manoeuvres may contribute to EIMD in the quadriceps, as well as playing a role in injury to the anterior cruciate ligament, a common injury in rugby players (Brooks et al. 2005a). Additionally, players who are involved in the highest number of short duration sprints, namely outside backs, have the highest rate of hamstring injuries of any of the

playing positions in rugby (Brooks et al. 2006). Hamstring injuries may be brought about through eccentric loading of the hamstring muscles during the late swing phase and/or late stance phase of sprinting (Yu et al. 2008) as the hamstring group act to break the forward swing of the leg (Garrett 1990; Stanton and Purdham 1989). It may be speculated that micro-structural damage, brought about by eccentric loading of the quadriceps and hamstrings during movements that involve rapid decelerations and sprinting, respectively, may occur regularly throughout a match so that collectively each action contributes to what may be substantial EIMD.

Whether these three factors contribute to EIMD equally throughout the season is unknown. The repeated bout effect, which provides protection or adaptation to the damaged muscle so that further bouts of eccentric exercise are less damaging (McHugh 2003; McHugh et al. 1999), would suggest that players may become less susceptible to all factors contributing to muscle damage as the season progresses. Therefore a combination of changes in ground conditions and an increased ability to cope with contacts and eccentric loading later in the season may contribute to the decrease in injuries occurring at this time. Whether the unpredictable nature of the sport provides the appropriate, specific stimulus to bring about the repeated bout effect, so that subsequent matches are dealt with more efficiently from a muscle damage point of view, is unclear. Anecdotally muscle soreness is much greater in preseason and during early season matches than later in the season however this belief was not supported by the findings of Kavaliauskas (2010) who failed to see any difference in subjective ratings of lower body muscle soreness in professional rugby players over the course of a rugby season.

2.2.4.3.2 Post-match recovery from muscle damage

Although elevations in CK have been consistently reported after rugby matches (Banfi et al. 2007; Cunniffe et al. 2010; Gill et al. 2006; Smart et al. 2008; Suzuki et al. 2004; Takarada 2003) there is no evidence that performance is impaired in the hours or days after the match. Several studies have investigated the use of various post – rugby match recovery modalities on markers of muscle damage but once again these studies neglected to investigate actual physical performance (Banfi et al. 2007; Gill et al. 2006). These studies make the assumption that elevations in CK are representative of a decrease in muscle function and suggest that training loads should

be modified while CK levels remain high so that full recovery can be achieved (Smart et al. 2008; Suzuki et al. 2004). However, the use of CK alone as a measure of muscle damage and over training has been widely questioned (Brancaccio et al. 2007; Fridén and Lieber 2001; Hartmann and Mester 2000; Urhausen and Kindermann 2002). Until more information regarding the physical cost of a rugby match is available it is difficult to draw conclusions as to the need of recovery modalities post-match.

2.2.4.4 Rugby match simulations

The random nature of rugby means that the total work done, number of contacts and time spent in different modes of locomotion may differ significantly between matches. Coupled with the unpredictability of weather and ground conditions, particularly as rugby is played outdoors during the winter, these factors make it difficult to use actual matches for intervention studies when replicating conditions across trials is critical. To get around this issue a number of studies have utilised match simulations based on time–motion analysis to investigate the effects of a range of interventions such as carbohydrate-protein supplementation (Roberts et al. 2011), caffeine supplementation (Stuart et al. 2005), carbohydrate-caffeine supplementation (Roberts et al. 2010a) and intermittent hypoxic training (Hamlin et al. 2008; Hinckson et al. 2007). The use of such protocols maximises ecological validity and accurate replication of match related components across a number of trials. Although changes in markers of muscle damage have been observed after contact team sport simulations (Roberts et al. 2011; Twist and Sykes 2011), the majority of simulations have focused on the intermittent running involved in the sport and have tended to pay less attention to the contact aspect which, as discussed above, plays a significant role in the match. Where attempts have been made to replicate match related contacts studies have tended to use tackle bags and/or hit shields (Hinckson et al. 2007; Singh et al. 2010; Stuart et al. 2005) which are cushioned and therefore do not provide the same violent contact that player on player contact produces. As simulations are generally performed indoors, so that climate can be controlled, there may be significant risk of injury associated with attempting to simulate actual match specific contact between players. However, by not employing realistic contacts these studies are only addressing one of the contributors, eccentric muscle contraction, to muscle damage and are missing components that will cause considerable fatigue to players throughout the course of the match.

That said, with the exception of Roberts et al. (2011), all of the rugby match simulations discussed above have investigated interventions that may improve performance rather than looking at the effects of different modalities on recovery from a simulated match. Therefore, while a key component of the match is missing in these simulations it may be more important to include realistic contacts if the aim of the research is to identify the effectiveness of post-match behaviour on subsequent performance recovery.

2.3 SUMMARY

Due to the limited research investigating the influence alcohol consumption has on post –exercise recovery, in this case recovery from EIMD, the review of literature presented here has been broad. While this lack of previous, similar research provides wide scope for developing the earlier work of Barnes et al. (2010), direct comparison to the findings of others is difficult. In fact, the work of Clarkson and Reichsman (1990) is the only previous study to investigate the combination of eccentric exercise and alcohol consumption. However, as the consumption of alcohol by sportspeople, particularly those involved in contact team sports, tends to occur post–exercise or competition the relevance of this study to the sporting community may be limited.

The results of Barnes et al. (2010) were the first to demonstrate that the consumption of 1 g alcohol per kg BW after eccentric exercise magnified the force loss typically associated with damaging exercise. However, a single finding, in isolation, provides little to expand our knowledge of how alcohol impacts recovery. According to the "scientific method" the development and testing of hypotheses should be followed by modification to the original hypothesis and further experimental investigation so that, if results are consistent between experiments, the hypothesis may become theory, or at least greater weight can be given to the original findings. Therefore, in the current context, further studies are required to either confirm or refute the findings of Barnes et al. (2010).

The consumption of alcohol, often at hazardous levels, is engrained in sporting culture, particularly in team sports that have traditionally been male dominated. Therefore, any finding that implies that the use of alcohol in the sporting setting may

be less than favourable is likely to be met by at least some opposition. The recommendation for alcohol abstinence by sports people, especially after competition when alcohol consumption is at its highest, is unrealistic. What is required, therefore, is a far greater understanding of how different doses of alcohol influence recovery so that research based recommendations can be made for the appropriate use of alcohol during the post exercise/competition period.

The force loss associated with EIMD is the result of a complex combination of alterations in peripheral and central function which appears to be initiated by eccentric exercise induced structural deformities within skeletal muscle. The Literature Review identified three main sites at which alterations in normal function may result in a loss in voluntary force production. Firstly, voluntary activation may be diminished as a result of a decrease in neural output. Whether this is due to the sensations of pain associated with EIMD is currently unclear however similar decrements to those seen with EIMD have been observed with experimentally - induced pain. Secondly, alterations in EC coupling, as a result of modifications at the T-tubule/SR interface, may play a significant role in EIMD force loss and is responsible for EIMD related LFF. Finally, changes in the contractile characteristics of sarcomeres would additionally influence force production through an altered ability to form cross-bridges.

The depressant effects of alcohol on the CNS are responsible for its global popularity as a recreational drug. The effects of alcohol are dose dependent and range from social inhibition, loss of coordination, memory loss and eventually coma and death. *In vitro* studies have shown that EC coupling, through altered Ca^{2+} handling by the SR, and membrane integrity may be modified by physiologically relevant doses of alcohol. As with EIMD, these changes have the potential to reduce voluntary force production however *in vivo* studies have failed to confirm this hypothesis. Whether EIMD related changes to neural output, EC coupling and membrane integrity are worsened by alcohol, when the CNS and muscle may be more susceptible to alcohol's actions, is conceivable.

While laboratory based experimentation provides a means of tightly controlling variables such as intensity and work between trials, the application of alcohol's

impact on EIMD resulting from maximal isokinetic eccentric exercise to a sporting context may be limited. The strong relationship between the sport of rugby and heavy alcohol consumption is fairly well known. Significant levels of damage to skeletal muscle may occur as a result of a rugby match through contact with opponents and / or the ground and through repeated eccentric contractions during rapid deceleration and direction changes. The physical stress brought about by a rugby match initiates an inflammatory response and alterations in testosterone/cortisol ratios, both factors which alcohol may negatively impact thus impairing recovery. Unfortunately, the random nature of the sport means that investigation of the effects of alcohol on recovery from actual rugby matches is fraught with difficulties therefore the use of rugby simulations may provide a valid and controlled means of investigating this interaction.

The complete lack of research quantifying the effect of a rugby match on muscle function makes it difficult to predict whether the muscle damage induced by contact sport is similar in magnitude to that brought about experimentally in a laboratory setting. It may be possible that alcohol's interaction with EIMD is dependent on the severity of damage and therefore, assuming the EIMD induced by Barnes et al. (2010) is similar to that occurring in a rugby match, alcohol may detrimentally impact functional recovery after a match. Alternatively, if rugby induced muscle damage is less severe alcohol's effects may be negligible**.**

CHAPTER 3

Aims and Hypotheses

3.1 HYPOTHESES

Based on the information gathered from the literature review, as summarised above, it is hypothesised that that the consumption of alcohol after exercise will negatively impact performance in the days following the eccentrically biased exercise.

Specific hypotheses:

One When consumed in the hours after intense eccentric exercise the consumption of 1 g alcohol per kg BW will magnify the loss in force typically observed in the days after such exercise **(Chapter 5 and 7**).

Two Alcohol's actions on force loss will be due to an interaction between alcohol and EIMD rather than due to the systemic effect of alcohol on force production **(Chapters 5 and 7).**

Three A low dose (0.5 g alcohol per kg BW) of alcohol will not affect force loss in the days following strenuous eccentric exercise **(Chapter 6).**

Four The consumption of 1 g alcohol per kg BW, when consumed after strenuous eccentric exercise, will negatively affect measures of neuromuscular function through alterations at the muscle level, rather than through changes in the CNS **(Chapter 7).**

Five In the days following the drinking episode neuromuscular function will not be altered by the consumption of 1 g alcohol per kg BW, if EIMD is absent **(Chapter 7).**

Six Sport specific measures of speed, power and agility will be negatively affected by the consumption of 1 g alcohol per kg BW after a simulated rugby game **(Chapter 8).**

Seven Immunoendocrine function will be negatively altered by the consumption of 1 g alcohol per kg BW after a simulated game of rugby **(Chapter 8).**

3.2 AIMS

The general aim of this thesis is to investigate the effects of acute, post-exercise alcohol consumption on measures of physical performance in the days after the exercise bout. To investigate the previously stated hypotheses the specific aims of this series of studies are to:

Aim One Investigate the potential interaction between post-exercise alcohol consumption and EIMD **(Chapter 5 and 7)**.

Aim Two Ascertain whether a low dose of alcohol impairs force development when combined with EIMD **(Chapter 6).**

Aim Three Examine the mechanisms behind alcohol's interaction with EIMD, in particular whether the interaction is due to alterations at the muscle or CNS level **(Chapter 7).**

Aim Four Examine the effects of alcohol, in the absence of EIMD, on neuromuscular performance **(Chapter 7)**.

Aim Five Investigate whether post-exercise alcohol consumption impacts sport specific performance in the days after a simulated game of rugby **(Chapter 8).**

Aim Six Identify whether post-exercise alcohol consumption alters the immunoendocrine response to sports specific exercise **(Chapter 8).**

CHAPTER 4

General Methodology

4.1 GENERAL METHODOLOGY

This chapter outlines the common methodology used in this series of studies. Details specific to each particular study are discussed in the appropriate experimental chapters.

4.2 PRE-EXPERIMENTAL CONTROL

For **Chapters 5, 6 and 7**, leg dominance and treatment were allocated using a randomised, cross-over design so that equal numbers of subjects completed their first trial using either their dominant and non-dominant leg and additionally equal numbers completed their first trial with either the ALC or OJ treatment. For **Chapter 8**, a randomised cross-over design was also used so that equal numbers of subjects underwent the ALC or OJ trial in their first trial.

In all studies, subjects were provided with a standardised pre-exercise meal which they consumed four hours prior to the start of each trial. Subjects were also given a standardised small meal immediately after exercise, prior to the consumption of the treatment beverage. The food provided was the same for all subjects, rather than being relative to BW. Although this method has limitations due to larger individuals requiring greater levels of energy after exercise nutritional conditions were the same between trials. Subjects were instructed to abstain from exercise, except for any necessary walking, and alcohol use in the 48 h period prior to each trial and throughout the entire post-exercise period. To ensure the same diet was followed in each trial, subjects were required to complete a diet diary (Appendix E) for the duration of the first trial; although subjects diets were not analysed constant reminders were given throughout each trial so that the diet consumed over the course of the first trial was replicated during the second trial. Additionally, subjects were instructed to abstain from behaviours that may have altered their recovery in the days

following the exercise bout. This included the use of anti-inflammatory medication, compression garments, stretching, ice/contrast therapy and nutritional supplements. Subjects underwent familiarisation of all procedures at least one week before their first trial.

4.3 EXPERIMENTAL MEASURES

All trials were carried out in the late afternoon/early evening period so that the consumption of alcohol would not impact subject's daily activity. In all studies, performance measures were made immediately before the start of the exercise bout and again at 36 and 60 h **(Chapter 5, 6 and 7)** or 24 and 48 h **(Chapter 8)** post-exercise. Based on the earlier work of Barnes et al (2010), these timelines were chosen as they represent the pattern of training that may occur after a game of contact sport. That is, soon after a game/event athletes may consume a large volume of alcohol which may induce a hangover the next day. Often the day after a game/event is spent recovering and training begins again in earnest the next day, approximately 36 h after the event. By 60 h the athlete is likely to be back training at an optimal level and in the case of team sportspeople team trainings are likely to have also begun by this time.

By reassessing performance at least 24 h post-exercise the hang-over period, which may have influenced subsequent performance, was avoided. Additionally, by beginning the retesting process at least 24 h post-exercise any effect of fatigue related to the exercise bout is likely to have been avoided. A schematic representation of the time line used in all studies is presented in Figure 6.

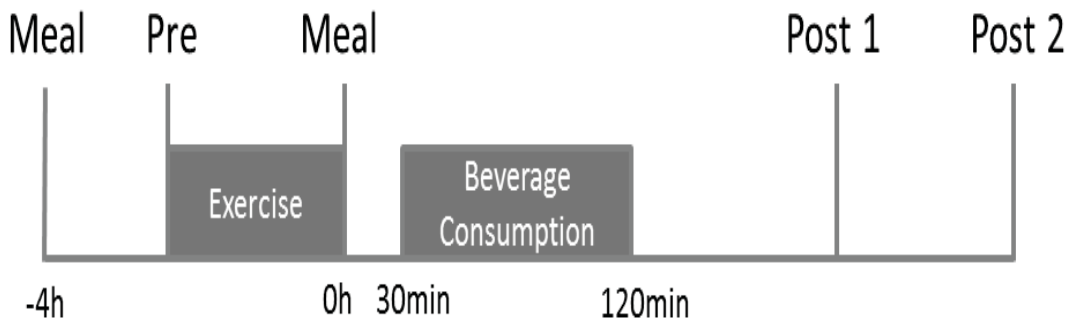

Figure 6 Schematic representation of the time line used in all studies (not to scale). Performance measures were made immediately before exercise (Pre). Follow up measures (Post 1 and Post 2) were made at either 24 h and 48 h (Chapter 8) or 36 h and 60 h (Chapters 5, 6 and 7), respectively.

4.4 EXERCISE PROTOCOLS

4.4.1 Eccentric exercise

The eccentric exercise model used in **Chapter 5, 6** and **7** has previously been shown to bring about significant decreases in voluntary force generation, DOMS, inflammation and elevations in circulating CK concentrations (Barnes et al. 2010; MacIntyre et al. 1996). Thus this model provides a reliable means of bringing about EIMD in a controlled manner so that differences between legs, and therefore treatments, can be accurately measured.

4.4.2 Rugby simulation

The rugby game simulation used in **Chapter 8** utilised a modified version of the Bath University Rugby Sprint Test. This protocol has previously been shown to have a good level of repeatability (Roberts et al. 2011; Roberts et al. 2010b). The addition of rugby specific contact, by way of player on player tackling and mauling, added a specific contact component that until now has been missing from rugby simulations. A simulation was chosen, rather than a real game, as it provides an appropriate level

of control, in terms of matching work and intensity, between trials so that a direct comparison could be made between treatments.

4.5 TREATMENT

A dose of 1 g alcohol per kg BW, as vodka (37.5% alcohol/volume; Smirnoff, Australia) and orange (Frucor Beverages, New Zealand), was used in **Chapter 5, 7** and **8**; in **Chapter 5** a dose of 0.5 g alcohol per kg BW was used. Although the higher dose is considerably lower than the levels of alcohol regularly consumed by athletes after sports, this dose has previously been shown to negatively impact force generation in the days following the exercise bout (Barnes et al. 2010). Therefore, ethically it is difficult to increase the dose knowing that it already has a detrimental effect. Ethical approval for alcohol related research is often difficult to obtain and this series of studies was no exception. This may be one reason recent research into alcohol's effect on performance is scarce. The control beverage used in all studies was an isovolumetric, isoenergetic amount of orange juice. To ensure the volume of beverage consumed was the same between treatments an additional volume of water was consumed with the alcohol beverage. In each trial, after the beverage was consumed subjects were driven home and required to go to bed. Additionally, when returning home from the laboratory after the consumption of alcohol a "buddy system" was employed whereby someone at the subject's home took responsibility for their behaviour and safety overnight.

CHAPTER 5

Post Exercise Alcohol Ingestion Exacerbates Eccentric-Exercise Induced Losses in Muscular Performance

Publication:
Barnes MJ, Mündel T, Stannard SR (2010). European Journal of Applied Physiology (2010): 108; 1009-1014.

5.1 ABSTRACT

The effect of acute alcohol intake on muscular performance in both the exercising and non-exercising leg in the days following strenuous eccentric exercise was investigated to ascertain whether an interaction between post-exercise alcohol use and muscle damage causes an increase in damage related weakness. Ten healthy males performed 300 maximal eccentric contractions of the quadriceps muscles of one leg on an isokinetic dynamometer. They then consumed either a beverage containing 1g/kg bodyweight ethanol (as vodka and orange juice) (ALC) or a non-alcoholic beverage (OJ). At least two weeks later they performed an equivalent bout of eccentric exercise on the contralateral leg after which they consumed the other beverage. Measurement of peak and average peak isokinetic (concentric and eccentric) and isometric torque produced by the quadriceps of both exercising and non-exercising legs was made before and 36 and 60 h post-exercise. Greatest decreases in exercising leg performance were observed at 36 h with losses of 28.7, 31.9 and 25.9% occurring for OJ average peak isometric, concentric, and eccentric torque respectively. However, average peak torque loss was significantly greater in ALC with the same performance measures decreasing by 40.9, 42.8 and 44.8% (all $P < 0.05$). Performance of the non-exercising leg did not change significantly under either treatment. Therefore, consumption of moderate amounts of alcohol after damaging exercise magnifies the loss of force associated with strenuous eccentric exercise. This weakness appears to be due to an interaction between muscle damage and alcohol rather than the systemic effects of acute alcohol consumption.

5.2 INTRODUCTION

The mechanisms and consequences of exercise-induced muscle damage (EIMD) have received considerable scientific attention over the past 20 years. Strenuous eccentric muscle action is now known to cause micro-structural damage resulting in delayed onset muscle soreness (DOMS), inflammation and, more importantly, impaired muscle function that typically lasts for a number of days, depending on the severity of the damage (Cleak and Eston, 1992; Proske and Morgan, 2001). Over this same period EIMD has been employed as a model of soft tissue injury where a number of modalities aimed at improving the rate of recovery (e.g. cold water immersion therapy (Eston and Peters 1999), non-steroidal anti-inflammatory medication (Gulick et al. 1996), massage (Jönhagen et al. 2004) and compression therapy (Kraemer et al. 2001) have been tested. This research has provided mixed and often inconclusive results. Surprisingly, compared to these recovery modalities, and others, less attention has been afforded to post-exercise behaviours that may negatively impact the recovery process after eccentric exercise.

One such behaviour is post-exercise alcohol use. While the consumption of large amounts of alcohol by sportspeople, often after competition or training, is common place (Nelson and Wechsler 2001; Snow and Munro 2006; O'Brien et al. 2007) little is known about how this behaviour effects recovery and subsequent performance in the days after exercise. We have recently shown that moderate amounts of alcohol (1 g alcohol per kg BW) consumed immediately after strenuous eccentric exercise exacerbates the weakness normally seen in the days following such exercise (Barnes et al. 2010). This finding suggests that athletes competing in sports that involve strenuous eccentric muscular work, or those suffering from soft tissue injury, should avoid alcohol consumption, at least immediately after exercise or injury, if optimal recovery is to occur. However, the design of this previous study did not allow us to ascertain whether this alcohol-induced weakness was due to delayed recovery from strenuous eccentric exercise or whether alcohol alters the ability to recruit and create tension in any muscle group in the 60 h after drinking.

Previous research (Poulsen et al. 2007) suggests that the acute consumption of clinically relevant levels of alcohol does not affect muscular performance in the days

following the drinking session. Further, indices of EIMD are unaffected when alcohol is acutely consumed prior to a bout of damaging exercise (Clarkson and Reichsman 1990). The results of these studies suggest that our previous findings may be due to an interaction between the already damaged muscle and the acute post-exercise consumption of alcohol. The purpose of this study therefore, was to investigate whether alcohol interacts with damaged skeletal muscle to magnify the typical weakness associated with EIMD or whether our previous findings are due to the systemic effects of acute alcohol consumption, independent of EIMD.

5.3 METHODS

5.3.1 Subjects

Twelve males volunteered to participate in this study. However, due to an obvious learning effect the results of two subjects were excluded from statistical analysis, as the subjects' strength was higher than pre-exercise values in the days after the damaging protocol. This suggests that either these subjects had failed to produce sufficient force during the exercise protocol to bring about significant levels of EIMD or that at some stage during the exercise protocol they had "learned" how to generate greater levels of force, possibly through neural adaptation to the exercise (Gabriel et al. 2006) . This suggests that these subjects "learned" how to generate Analysis was therefore carried out on the results of 10 subjects (age 23.5 ± 5.1 years, body mass 76.9 ± 12.9 kg). All subjects were healthy and had at least two years of resistance training experience at a recreational level (minimum twice per week). The protocol was approved by the Massey University Human Ethics Committee and written consent was obtained from each subject.

Familiarisation of the protocol was carried out at least one week before the first trial. Subjects were instructed to abstain from alcohol consumption and any form of exercise from 48 h before and until 60 h after the exercise bout. Subjects were also instructed to abstain from practices that could potentially improve or worsen their recovery during the 60 h post exercise period. Subject's diets were replicated between trials by way of a food diary that was filled out from the morning of the first trial until the last measurements were taken at 60 h post exercise. Utilising a single cross-over design, treatment and leg were randomly allocated in a counter-balanced fashion.

5.3.2 Overview

The current study utilised a modified version of the protocol described by Barnes et al. (2010). Adapted from the work of MacIntyre and colleagues (1996), this exercise protocol has previously been shown to bring about significant levels of muscle damage as characterised by decreased muscular performance, DOMS and elevations in circulating CK concentrations (MacIntyre et al. 1996; Barnes et al. 2010). Briefly, subjects performed 300 maximal eccentric contractions of the quadriceps muscles of one leg on an isokinetic dynamometer. They then consumed either an alcoholic beverage or a non-alcoholic control beverage. At least two weeks later they performed an equivalent bout of eccentric exercise on the contralateral leg after which they consumed the other beverage. Muscular performance of the damaged (exercising) and non-damaged (non-exercising) leg was measured prior to the damaging exercise bout and at 36 and 60 h post-exercise.

5.3.3 Muscular performance

Four hours after consuming a standardised, solid meal (3765 kJ; CHO 133 g, Fat 28 g, Protein 25 g) subjects returned to the laboratory in the evening and warmed up on a cycle ergometer (Monark, Varberg, Sweden) for five minutes at 100W. They were then seated on a Biodex® isokinetic dynamometer (Biodex Medical Systems, New York, USA) and straps were fixed across the chest, hips and active leg to isolate movement to the quadriceps. Knee joint range of motion was set and recorded for use in subsequent follow-up tests. Subjects then performed separate sets of five maximal isometric, concentric and eccentric contractions of the quadriceps muscles of both the non-exercising and the exercising leg. Each set was separated by two minutes of passive recovery. Isometric tension was measured at a knee angle of 75º (1.31 rad). Concentric and eccentric torque was measured at an angular velocity of $30°\cdot s^{-1}$ (0.52 $rad\cdot s^{-1}$). Absolute peak torque and average peak torque over five contractions was recorded. Muscular performance was measured again at 36 and 60 h post-exercise.

5.3.4 Exercise protocol

Subjects remained on the Biodex and completed 3 sets of 100 maximal eccentric contractions, over a 60° (1.05 rad) range of motion at an angular velocity of $30°\cdot s^{-1}$, using the quadriceps muscles of one leg. Each set was separated by 5 min of passive recovery. Subjects were verbally encouraged to resist the downward action of the

dynamometer arm as hard as possible and had access to visual feedback of their torque throughout the protocol to ensure continuous maximal effort. Total work completed during the eccentric exercise bouts was not significantly different between trial one (34.9 ± 11.5 kJ) and trial two (36.7 ± 11.1 kJ) (P = 0.14) or between control (OJ) (35.2 ± 11.5 kJ) and alcohol (ALC) trials (36.5 ± 11.5 kJ) (P = 0.29).

5.3.5 Treatment

Thirty minutes after completion of the exercise bout, and having consumed a standardised meal immediately after exercise (1532 kJ; CHO 50.4 g, Fat 9.1 g, Protein 7 g), subjects began drinking a beverage containing either 1 g of alcohol per kg of BW as vodka (37.5% alcohol/volume; Smirnoff, Australia) in orange juice (Frucor Beverages, New Zealand) (ALC) or a control beverage of orange juice alone (OJ). Equivalent to 8 ± 2.8 standard drinks, the mean volume of vodka consumed per subject was 211.9 ± 51.4 ml. In order to balance total energy value (2794.5 ± 476.1 kJ) and fluid volume (1637.9 ± 268.3 ml) between trials subjects consumed a greater volume of orange juice in the OJ trial while in the ALC trial they consumed an additional volume of water (751.5 ± 128.0 ml) along with the alcoholic beverage. An equal volume of beverage was consumed every 15 min over a total time of 90 min. Once the required amount of beverage was consumed participants were driven home and instructed to go directly to bed.

For the second trial the contralateral leg was exercised and the other beverage was consumed using the same protocol, as outlined above.

5.3.6 Statistical analysis

Data was analysed using the Statistical Program for Social Sciences (SPSS) for Windows (version 15.0, SPSS Inc., Chicago, IL.). A general linear-model three-way repeated-measures ANOVA (Treatment x Time x Leg) was used to compare conditions over time for each performance measure. This analysis provided main effects of Time, Treatment and Leg; thus Treatment x Time, Treatment x Leg, Time x Leg and Treatment x Time x Leg interactions were also investigated. If conditions differed significantly, post-hoc pairwise comparisons using Bonferroni adjustment were performed to identify differences between time points within each treatment and leg. As no significant change was seen in muscular performance of the non-exercising

leg (see results section for details) and to allow comparison between the results of the current study and our previous findings (Barnes et al 2010), exercising leg data was analysed separately with two-way repeated-measures ANOVA providing additional Treatment x Time interactions. As different legs were used for each trial, resulting in significantly different pre-exercise values between treatments, data was analysed as absolute change in torque relative to pre-exercise values. Paired t-tests were carried to compare total work done between trial one and two and between the ALC and OJ trials. Reported values are means ± SD. Statistical significance was set at $P < 0.05$.

5.4 RESULTS

Completion of 300 eccentric muscular contractions of the quadriceps resulted in significant decreases in isometric, concentric and eccentric peak and average peak torque over time in the exercising leg only (all $P < 0.001$, Table 5). No significant change in non-exercising leg performance was observed at any time point under either treatment (all $P > 0.2$). Significant Treatment x Time x Leg interactions were found for isometric ($P = 0.036$) and eccentric ($P = 0.02$) peak torques as well as for isometric ($P = 0.032$), concentric ($P = 0.032$) and eccentric ($P = 0.023$) average peak torques.

Analysis of the exercising leg data, independent of the Leg variable, found significant changes over time for all performance measures (all $P < 0.001$). Significant Treatment effects (all $P < 0.02$) and Treatment x Time interactions (all $P < 0.05$) were seen for all performance measures except peak concentric torque ($P = 0.16$ and 0.42 respectively). Greatest decreases in performance were seen with ALC at 36h while no significant change in performance was observed between 36 and 60 h under either treatment.

Repeated-measures ANOVA of trial one versus trial two found no significant order effect for any of the muscular performance measures (all $P > 0.15$).

Table 5 Absolute changes in torque (Nm) following strenuous eccentric exercise (mean ± SD).

	EX			NX		
	Pre	36h	60h	Pre	36h	60h
Peak ISO						
OJ	275.8 ± 48.9	-78.8 ± 38.7*	-72.9 ± 48.7*	286.0 ± 51.2	-3.4 ± 16.0	-0.1 ± 19.6
ALC	295.6 ± 49.8†	-113.7 ± 45.3*†	-108.9 ± 84.4*	289.6 ± 62.6	-10.0 ± 21.8	-3.0 ± 42.7
Peak CON						
OJ	227.8 ± 50.3	-71.5 ± 32.4*	-57.9 ± 22.8*	255.9 ± 69.4	-11.5 ± 26.0	-12.1 ± 23.2
ALC	240.9 ± 50.4	-88.3 ± 44.4*	-78.2 ± 64.6*	238.3 ± 45.3	-11.4 ± 24.2	-3.5 ± 37.7
Peak ECC						
OJ	284.9 ± 77.3	-77.1 ± 59.3*	-63.5 ± 51.9*	311.0 ± 81.7	-0.3 ± 32.2	4.9 ± 37.8
ALC	345.6 ± 81.7‡	-150.2 ± 58.8*‡	-133.2 ± 88.2*†	335.6 ± 79.0	-22.6 ± 47.7	-33.0 ± 73.1
Average ISO						
OJ	256.9 ± 39.0	-73.8 ± 41.5*	-72.0 ± 55.7*	271.3 ± 40.1	-7.8 ± 18.7	-1.5 ± 22.1
ALC	281.1 ± 37.7†	-115.1 ± 50.3*†	-111.7 ± 86.8* †	271.3 ± 54.1	-8.9 ± 15.3	-3.7 ± 43.7
Average CON						
OJ	205.1 ± 31.2	-65.6 ± 32.7*	-55.3 ± 32.7*	230.7 ± 50.2	-10.0 ± 19.7	-9.9 ± 32.8
ALC	226.8 ± 37.9†	97.0 ± 47.7*†	91.0 ± 64.0*†	222.7 ± 36.2	-11.6 ± 16.8	-9.4 ± 40.5
Average ECC						
OJ	266.9 ± 76.9	-69.1 ± 93.6#	-63.6 ± 47.4*	291.3 ± 84.3	2.6 ± 33.7	6.4 ± 41.5
ALC	320.4 ± 70.1†	-143.5 ± 68.6*†	-127.8 ± 91.6*†	306.3 ± 75.9	-8.6 ± 37.1	-23.8 ± 60.1

ISO isometric, CON concentric, ECC eccentric torque (Nm), OJ control, ALC alcohol treatment

* Significantly different to pre-exercise value ($P < 0.01$). # Significantly different to pre-exercise value ($P < 0.05$). ‡ Significantly different between trials ($P < 0.01$). † Significantly different between trials ($P < 0.05$)

No significant difference between 36 and 60 h values under either treatment

5.5 DISCUSSION

The aim of the present study was to investigate whether the systemic effects of alcohol bring about muscular weakness in the days following alcohol consumption after strenuous eccentric exercise or whether alcohol interacts with EIMD to exacerbate the loss of muscular performance, as previously observed (Barnes et al. 2010).

Completion of 300 maximal eccentric contractions of the quadriceps resulted in significant decreases in all performance measures in the exercising leg only (Table 1). In accordance with the results of Poulsen et al. (2007) our data confirms that a moderate dose of alcohol has no effect on muscular performance in the days following a drinking episode provided the muscle has not been damaged as a result of strenuous eccentric work. The results of our previous study (Barnes et al. 2009) are thus due to an interaction between post-exercise alcohol consumption, the damaged muscle and/or the recovery processes initiated by EIMD.

Confirming our previous observations, in the current study significant differences in post-exercise muscle performance between treatments were seen after 36 h. At this time point isometric and eccentric peak torques were 38.5 and 43.5% lower than pre-exercise measures, respectively, with ALC compared to losses of 28.6 and 27% for the same measures with OJ. Perhaps more important than a single all-out effort, the ability to generate force repeatedly was greatly reduced, with losses in average peak torque of 40.9 (isometric), 42.8 (concentric) and 44.8% (eccentric) with ALC compared to 28.7, 31.9 and 25.9% with OJ, respectively. Together with the results of our previous work (Barnes et al. 2009) the current study reinforces the observation that the consumption of moderate amounts of alcohol after damaging exercise magnifies the loss in force production capability typically associated with EIMD.

To date considerable effort has been made to identify modalities that improve the rate of performance recovery after strenuous eccentric exercise. The majority of this research, however, has failed to conclusively show that losses in performance can be minimised if a particular modality is used during the post-exercise period (Barnett 2006; Cleak and Eston 1992b; Wilcock et al. 2006). An alternative, as suggested by

Reilly and Ekblom (2005), is to adhere to proper nutritional strategies including moderation when drinking alcohol. Indeed, given our current and previous findings moderation of alcohol after strenuous, damaging exercise is sound advice if a timely return to optimal performance is desired. However, whether moderation alone is sufficient to avoid alcohol's negative effects on recovery after damaging exercise is currently unclear as only one dose (1 g alcohol per kg BW) has been investigated. In fact the dose used in the current study is considerably lower than levels of alcohol consumption frequently reported by sportspeople (Snow and Munro 2006; O'Brien et al. 2005; O'Brien et al. 2007) suggesting that alcohol use would have to be restricted to an even greater extent if results such as those observed in the present study are to be avoided. Further research is warranted to investigate the dose effects of alcohol use in the post-exercise period.

Although the mechanisms behind our findings are not yet understood previous research into acute alcohol use suggests that a number of similarities may exist between the separate effects of EIMD and acute alcohol consumption on skeletal muscle. Alterations in EC coupling and CNS function have been proposed as contributors to the force loss associated with EIMD (Deschenes et al. 2000; Carson et al. 2002; Prasartwuth et al. 2005; Racinais et al. 2008; Dartnall et al. 2009). Similarly, acute alcohol exposure has been shown to negatively affect sarcoplasmic Ca^{2+} transport, thus altering EC coupling (Cofán et al 2000); while alcohol acts on the CNS to impact axonal conductance and neurotransmission leading to dose dependent impairment of psychomotor and cognitive skills. These actions contribute to alcohol's popularity as a recreational drug (Reilly 2002; Valenzuela 1997). Finally, a well-coordinated immune response is initiated by EIMD to facilitate repair and recovery of damaged tissue (Tidball 2005). As acute alcohol use has been shown to adversely affect recovery from trauma/injury by altering the normal inflammatory response (Szabo and Mandrekar 2009) our results may be due to alcohol related impairment of the normal recovery processes. Whether any or all of these factors combine to bring about the results observed in the current study is worth further investigation.

5.5.1 Conclusion

When consumed after strenuous damaging exercise, a moderate dose of alcohol magnifies the typical loss of muscular performance associated with EIMD.

Conversely, in the absence of muscle damage alcohol does not affect muscular performance in the days following exercise indicating that post-exercise alcohol consumption interacts with muscle damage. The mechanisms behind our findings are yet to be elucidated.

CHAPTER 6

A Low Dose of Alcohol Does Not Impact Skeletal Muscle Performance After Exercise Induced Muscle Damage

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Barnes MJ, Mündel T, Stannard SR (2011). European Journal of Applied Physiology: 111 (4):725-729.

6.1 ABSTRACT

Moderate, acute alcohol consumption after eccentric exercise has been shown to magnify the muscular weakness that is typically associated with EIMD. As it is not known whether this effect is dose dependent the aim of this study was to investigate the effect of a low dose of alcohol on EIMD related losses in muscular performance. Ten healthy males performed 300 maximal eccentric contractions of the quadriceps muscles of one leg on an isokinetic dynamometer. They then consumed either a beverage containing 0.5 g alcohol per kg BW (as vodka and orange juice) or an isocaloric, isovolumetric non-alcoholic beverage. At least two weeks later they performed an equivalent bout of eccentric exercise on the contralateral leg after which they consumed the other beverage. Measurement of peak and average peak isokinetic (concentric and eccentric) and isometric torque produced by the quadriceps was made before and 36 and 60 h post-exercise. Significant decreases in all measures of muscular performance were observed over time under both conditions (all $P < 0.05$); however no difference between treatments was evident at any of the measured time points (all $P > 0.05$). Therefore, consumption of a low dose of alcohol after damaging exercise appears to have no effect on the loss of force associated with strenuous eccentric exercise.

6.2 INTRODUCTION

The consumption of large amounts of alcohol by sportspeople, particularly those involved in team sports, after competition and/or training is common place. This population is further reported as consuming such hazardous levels of alcohol more frequently than the general population (Maughan 2006; O'Brien et al. 2007; Quarrie et al. 1996; Snow and Munro 2006). As with the non-sporting population, these drinking patterns are associated with increased risk-taking behaviours which often result in alcohol-related injury (Cherpitel 1993; Nelson and Wechsler 2001). For sportspeople however, acute alcohol use, particularly after strenuous exercise, provides another risk through the influence alcohol can have on processes involved in recovery and adaptation.

The physical nature of many team sports engenders a high incidence of EIMD and soft tissue injury due to repeated rapid deceleration, changes in direction, and collisions with other players and/or the ground (LaStayo et al. 2003). Although both EIMD and alcohol consumption are common in many sports, very little is known about how the two interact to affect recovery (of performance) in the days after the injurious event.

When alcohol's ingestion is quickly followed by trauma/injury, alcohol is known to increase the chance of infection and medical complication resulting in delayed recovery through alterations in the trauma induced inflammatory response (Szabo and Mandrekar 2009). However, this combination of pre-exercise alcohol use and soft tissue injury (as EIMD), investigated by Clarkson & Reichsman (1990), found that a moderate dose of alcohol (0.8 g alcohol per kg BW) had no effect on recovery of performance in the days after a damaging bout of exercise, when compared to a non-alcoholic beverage.

Perhaps more relevant to the practices of sportspeople is how the consumption of alcohol *after* strenuous damaging exercise impacts muscular performance and recovery. We recently investigated this scenario (Barnes et al. 2010) and **Chapter 5** and found that a moderate dose of alcohol (1 g alcohol per kg BW) consumed after

strenuous eccentric exercise interacts with EIMD to magnify the force loss typically associated with such exercise.

As well as having an exacerbating effect on EIMD related losses in force, a dose of 0.92 g alcohol per kg BW has been shown to negatively influence the ability to rehydrate after dehydrating exercise (Shirreffs and Maughan 1997) while a dose of 1.5 g alcohol per kg BW impairs glycogen repletion after prolonged exercise if energy from alcohol replaces energy from carbohydrate in the post exercise diet (Burke et al. 2003). Alterations in the post-resistance exercise hormone response have been observed after a dose of 0.83 g alcohol per kg BW including a prolonged elevation in cortisol (Koziris et al. 2000) and elevated free testosterone (Vingren et al. 2003), the latter potentially due to an effect of alcohol on the androgen receptor. Both are thought to negatively affect adaptation to resistance exercise (Vingren et al. 2003) and could partly explain our previous findings (Barnes et al. 2010) and **Chapter 5**.

These deleterious effects of post-exercise alcohol consumption provide evidence that the use of alcohol after strenuous exercise should be managed carefully, however, more information is required if recommendations on appropriate alcohol use during the post-event period are to be made. The aim of this study, therefore, is to add to this limited knowledge base by investigating whether a low dose of alcohol interacts with damaged skeletal muscle to magnify the typical weakness associated with EIMD, as previously observed with a moderate dose of alcohol.

6.3 METHODS

6.3.1 Subjects

Ten males (age 20.8 ± 1.6 years, body mass 83.9 ± 12.9 kg) volunteered to participate in this study. All subjects were healthy and regularly (minimum twice per week) participated in recreational level resistance training. The protocol was approved by the Massey University Human Ethics Committee and written consent was obtained from each subject.

Familiarisation was carried out at least one week before the first trial. Subjects were instructed to abstain from alcohol consumption and any form of exercise from 48 h

before and until 60 h after the experimental bouts. Subjects were also instructed to abstain from practices that could potentially improve or worsen their recovery, for example the use of anti-inflammatory medication, massage, stretching and cryotherapy, during the 60 h post exercise period. Subjects recorded their diet from the morning of the first trial until 60 h post exercise and were instructed to replicate this for the second trial. Utilising a single cross-over design, treatment and leg (dominant vs. non-dominant) order were randomly allocated in a counter-balanced fashion.

6.3.2 Overview

The current study utilised a protocol previously described in **Chapter 5**. Briefly, this required each subject to perform 300 maximal eccentric contractions of the quadriceps muscles of one leg on an isokinetic dynamometer. They then consumed either an alcoholic beverage, equivalent to 35 g alcohol for a 70 kg person, or an isocaloric, isovolumetric amount of a non-alcoholic control beverage. At least two weeks later they performed an equivalent bout of eccentric exercise on the contralateral leg after which they consumed the other beverage. Muscular performance was measured prior to the damaging exercise bout and at 36 and 60 h post-exercise.

This 'one-legged' exercise model allows the subject to act as their own control therefore ensuring that the only difference between treatments is the leg used, i.e. dominant vs. non-dominant. The design also negates any residual effects in the muscle from the previous trial, such as the well-known repeated bout effect (McHugh 2003). A limitation of this model, however, is that only a single dose of alcohol is able to be compared to the control beverage.

6.3.3 Muscular performance

Four hours after consuming a standardised, solid meal (3700 kJ; CHO 50.1%, Fat 27.1%, Protein 11.7%) subjects presented at the laboratory in the evening and warmed up on a cycle ergometer (Monark, Varberg, Sweden) for five minutes at 100W. Subjects then performed separate sets of five maximal isometric (ISO), concentric (CON) and eccentric (ECC) contractions of the quadriceps muscles of one leg on a Biodex® isokinetic dynamometer (Biodex Medical Systems, New York, USA). Knee

joint range of motion was recorded for use in subsequent follow-up tests. Each set was separated by two minutes of passive recovery. Isometric tension was measured at a knee angle of 75º (1.31 rad). Concentric and eccentric torque was measured at an angular velocity of $30°\cdot s^{-1}$ ($0.52\ rad\cdot s^{-1}$). Absolute peak and average peak torque over five contractions were recorded. Muscular performance was measured again at 36 and 60 h post-exercise.

6.3.4 Exercise protocol

Immediately after muscular performance was measured, subjects completed 3 sets of 100 maximal eccentric contractions, over a 60° (1.05 rad) range of motion at an angular velocity of $30°\cdot s^{-1}$, using the quadriceps muscles of the tested leg. Each set was separated by 5 min of passive recovery.

6.3.5 Treatment

Immediately after the completion of the exercise protocol subjects consumed a standardised meal immediately after exercise (1468 kJ; CHO 51.1%, Fat 12.9%, Protein 28.2%). Thirty minutes later subjects began drinking a beverage containing either 0.5 g alcohol per kg of BW as vodka (37.5% alcohol/volume; Smirnoff, Australia) in orange juice (Frucor Beverages, New Zealand) (ALC) or a control beverage of orange juice alone (OJ). The mean volume of vodka consumed per subject was 112.8 ± 17.4 ml, this was diluted in 714 ± 109.9ml of orange juice and consumed in 6 equal volumes every 15 min over a total time of 90 min. Total energy value (2043.5 ± 314.3 kJ) and fluid volume (1285.2 ± 197.7 ml) were balanced between trials. To achieve this, an additional volume of water (458.4 ± 70.4ml) was consumed along with the alcoholic beverage. Thirty minutes after the consumption of the alcoholic beverage blood alcohol concentration (BAC) was estimated from breath alcohol content using a Digitech fuel cell alcohol tester (Electus Distributions PTY, Ltd, NSW, Australia). Subjects were then driven home and required to go directly to bed. For the second trial the contralateral leg was exercised and the other beverage was consumed using the same protocol, as outlined above.

6.3.6 Statistical analyses

Data was analysed using the Statistical Program for Social Sciences (SPSS) for Windows (version 15.0, SPSS Inc., Chicago, IL.). A general linear-model repeated-

measures ANOVA (Treatment x Time) was used to compare conditions over time for each performance measure. Similarly, repeated-measures ANOVA (trial 1 vs. trial 2) was carried out to identify possible order effects for each performance measure. If significant differences were found, post-hoc pairwise comparisons were made using Bonferroni adjustment to further investigate changes in performance over time within each condition. As different legs were used for each trial, resulting in significantly different pre-exercise values between treatments, data was analysed as absolute change in torque (Nm) (Table 1) as well as percentage change from pre-exercise values. Reported values are means ± SD. Statistical significance was set at $P < 0.05$.

6.4 RESULTS

Completion of 300 eccentric muscular contractions of the quadriceps resulted in significant decreases in isometric ($p = 0.004$) and concentric ($p = 0.005$) peak torque and isometric ($p = 0.02$), concentric ($p = 0.007$) and eccentric ($p = 0.004$) average peak torque over time under both treatments (Table 6). At 36 h ISO OJ was 12.3 % ($p = 0.05$) and ALC 11.1 % lower than pre values while at 60 h ISO was not significantly lower than pre values for OJ (4.1%) or ALC (5.2 %). Similarly, CON OJ and ALC decreased by 21 % ($p = 0.009$) and 16.4 % ($p = 0.017$) at 36 h, respectively, before improving to be down by 12.2 % and 11.7 % at 60 h. Smaller decrements were seen for ECC OJ (4.2 %) at 36 h compared to 14.2 % ($p = 0.032$) for ECC ALC at the same time point. These values had improved at 60 h so that ECC OJ was 6.1 % higher than pre-values while ALC was still 6.1% lower than pre-values. Similar decrements were seen for average peak torque measures.

No significant Treatment or Treatment x Time effect was evident for any of these performance measures (all $p > 0.2$). Repeated-measures ANOVA of trial one versus trial two found no significant order effect for any of the muscular performance measures (all $p > 0.1$). Total work completed during the eccentric exercise bouts was not significantly different between trial one (37.9 ± 5.5 kJ) and trial two (38.8 ± 6 kJ) ($p = 0.7$) or between OJ (39.3 ± 6.6 kJ) and ALC trials (37.6 ± 4.6 kJ) ($p = 0.3$).
Average BAC concentration after the consumption of 0.5g alcohol per kg BW was 0.011 ± 0.0013 g/dL (2.42 ± 0.29 mmol/L).

Table 6 Changes in torque (Nm) over time following strenuous eccentric exercise (mean ± SD).

	Pre	36h	60h
Peak ISO			
OJ	301.4 ± 48.9	-40.1 ± 44.0*	-17.1 ± 45.2
ALC	284.2 ± 49.8	-37.5 ± 45.3	- 19.1± 59.1
Peak CON			
OJ	265.1 ± 50.3	-54.9 ± 47.1*	-35.3 ± 41.4
ALC	249.5 ± 50.4	-42.8 ± 38.8*	-31.5 ± 41.4
Peak ECC			
OJ	336.5 ± 77.3	-20.6 ± 89.6	15.9 ± 66.0
ALC	330.9 ± 81.7	-49.4 ± 51.9*	-20.4 ± 55.4
Average ISO			
OJ	269.7 ± 39.0	-33.2 ± 35.9*	-15.4 ± 45.2
ALC	264.4 ± 37.7	-35.3 ± 45.1	-18.8 ± 54.7
Average CON			
OJ	239.5 ± 31.2	-46.1 ± 42.8*	-30.2 ± 36.3
ALC	236.1 ± 37.9	-51.7 ± 45.2*	-34.4 ± 42.1†
Average ECC			
OJ	308.4 ± 76.9	-51.4 ± 76.0	17.4 ± 77.3†
ALC	310.3 ± 70.1	-54.7 ± 46.1*	-24.8 ± 51.6

ISO isometric, CON concentric, ECC eccentric torque (Nm), OJ control, ALC alcohol treatment

* Significantly different to pre-exercise value ($p < 0.05$). † Significantly different to pre-exercise value ($p < 0.05$).

6.5 DISCUSSON

The aim of the present study was to investigate whether a low dose of alcohol (0.5 g alcohol per kg BW) consumed after strenuous eccentric exercise exacerbates EIMD related decrements in muscular performance in young resistance trained males, as previously observed with a dose of 1 g alcohol per kg BW (Barnes et al. 2010; **Chapter 5)**. As observed in our previous work, the completion of 300 maximal eccentric contractions of the quadriceps resulted in significant decreases in all performance measures, except peak eccentric torque, in the days following the exercise bout (Table 6). Unlike our previous results (Barnes et al. 2010; **Chapter 5**), however, the dose of alcohol used in the current study did not increase the magnitude of this decrement.

Although the mechanisms behind our previous findings are still unclear, similar volumes of alcohol to those consumed in the current study have been shown to inhibit neutrophil function (Patel et al. 1996) and whole muscle action potential responses to nerve stimulation (Pagala et al. 1995), while volumes slightly higher than this are known to alter the immune response to a bacterial challenge (Szabo 1998). Comparable doses to those used in our previous work have been found to decrease calcium transients (Nicolas et al. 1998) as well as negatively impacting the regulation of a number of chemokines (Szabo et al. 1999). Separately, a humoral effect, importantly on the androgens, has also been previously described (Vingren et al. 2003). The inflammatory response to EIMD is well documented (Proske and Morgan 2001) as is the potential effect this damage has on EC coupling (Warren et al. 2001), processes that rely heavily on the factors discussed above. We may therefore speculate that acute alcohol use could alter the EIMD related inflammatory response while further suppressing the EC coupling process to magnify force loss and delay recovery, as previously observed with a dose of 1 g alcohol per kg BW. However, further investigation is required to define the mechanisms behind the interaction between acute alcohol use and EIMD.

The aim of this series of studies was to experimentally investigate the effects of alcohol on EIMD related losses in force in a well-controlled manner so that any potential confounding factors were minimised and any effects clearly isolated.

However, the findings of these studies are limited in their application to a relatively homogenous group of subjects and how other populations might be affected is not yet understood. The population investigated to date, young recreationally trained males, is representative of the sporting demographic most often reported as binging on alcohol after competition (Nelson and Wechsler 2001; O'Brien et al. 2007). However, this fact should not limit future research to this group at the expense of other populations, for example females participating in team sports also report regularly consuming alcohol at hazardous levels (Ford 2007b; O'Brien et al. 2005; Quarrie et al. 1996).

Further, the protocol utilised has been used extensively to investigate the physiological effects and mechanisms of EIMD (Beaton et al. 2002; Jönhagen et al. 2004; Paulsen et al. 2005); however, future research should investigate more ecologically valid measures of performance such as sports specific measures of power, endurance and speed thus providing the wider sporting community with results directly applicable to a sports setting.

Together, the findings of the present study and the results of our previous work (Barnes et al. 2010; **Chapter 5**) indicate that an acute dose of alcohol of 1 g, but not 0.5 g, alcohol per kg BW, when consumed after strenuous eccentric exercise, negatively impacts the magnitude of force loss associated with EIMD. Until more information is available on how different doses of alcohol impact EIMD related decrements in force we tentatively make the recommendation that if alcohol is to be consumed in the hours after damaging/injurious exercise then the dose of alcohol consumed should be kept at or below 0.5 g alcohol per kg BW to ensure force loss is minimised. Collectively, the research carried out on the effects of acute alcohol use after strenuous exercise has shown only negative outcomes at a range of doses higher than that used in the current study but significantly lower than the volumes regularly reported as being consumed by sportspeople (Nelson and Wechsler 2001; O'Brien et al. 2005). The management of alcohol after sporting events should therefore be given greater attention than may currently be the case if recovery is to occur at an optimal rate.

6.5.1 Conclusion

When consumed after strenuous damaging exercise, a low dose (0.5 g alcohol per kg BW) of alcohol had no impact on the EIMD related losses in muscular performance.

CHAPTER 7

The Effects of Acute Alcohol Consumption and Eccentric Muscle Damage on Neuromuscular Function

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7.1 ABSTRACT

Voluntary and electrically stimulated muscular performance was examined to identify the effects of acute alcohol consumption on neuromuscular function in the presence and absence of EIMD. After initial neuromuscular performance measures were made, 12 subjects (EX) completed a bout of eccentric exercise using the quadriceps muscles of one leg while the remaining 11 subjects (NX) did not exercise. Subjects then consumed either a beverage containing 1 g alcohol per kg BW (ALC) or a non-alcoholic beverage (OJ). On another occasion the contralateral leg of both groups was tested and those in the EX group performed an equivalent bout of eccentric exercise after which the other beverage was consumed. Measurements of neuromuscular function were made pre-exercise and 36 and 60 h post-beverage consumption. CK was measured pre-exercise and at 12, 36 and 60h. Significantly greater ($p < 0.01$) decrements in maximal voluntary isometric contraction were observed with EX ALC at 36 and 60h compared to EX OJ, no change was seen in the NX group. Significant decreases in voluntary activation were observed at 36 ($p = 0.003$) and 60h ($p = 0.01$) with EX ALC only. Elevations in CK were observed at all post-eccentric exercise time points (all $p < 0.05$) under both EX OJ and ALC. No change in electromyography or low frequency fatigue was observed under either treatment in either group. These results suggest that decreased neural drive appears to contribute to alcohol's effect on the magnitude of EIMD related decrements in voluntary force generation.

7.2 INTRODUCTION

Injury or trauma to skeletal muscle that occurs during alcohol intoxication often results in an increased risk of infection and medical complication which may prolong recovery (Szabo and Mandrekar 2009). Although this alcohol related injury/trauma is common, little is known about the opposite situation, where alcohol is consumed after a damaging event, as may occur after intense eccentrically based exercise.

Recent research has shown that the acute consumption of 1 g alcohol per kg BW in the hours following a bout of strenuous eccentric exercise magnifies the typical force loss associated with damaging exercise (Barnes et al. 2010; **Chapter 5**). The design of these studies did not permit insight into the mechanisms behind these findings, however a number of possibilities exist due to similarities in the reported, separate effects that EIMD and acute alcohol bingeing may have on processes involved in the expression of muscular force.

Separately, acute (heavy) alcohol consumption and EIMD have been shown to bring about acute muscular weakness, soreness and elevations in circulating concentrations of CK and myoglobin that may last for several days after the initial event (Armstrong 1984; Urbano-Marquez and Fernandez-Sola 2004). Further, alterations in neuromuscular function appear to originate at both the peripheral (muscle) and CNS level under both alcohol and EIMD conditions separately.

Peripherally, EC coupling may be affected by acute alcohol consumption in a dose dependent and reversible manner through alterations in sarcolemmal membrane integrity (Preedy et al. 2001) and reductions in cellular excitability (Inoue and Frank 1967). Intracellular Ca^{2+} transients may also be affected, with ethanol known to inhibit sarcolemmal voltage-gated Ca^{2+} channels and decrease the amount of releasable Ca^{2+} stored within the SR (Cofan et al. 2000; Nicolas et al. 1998). However, unlike these *in vitro* studies, Poulsen et al. (2007) recently failed to observe a change in EC coupling, plasma free calcium concentrations and muscular performance, *in vivo*, after the consumption of a moderate dose of alcohol. Similarly, comparisons between the force generated by electrical stimulation at low (10-20 Hz) and high (80–100 Hz) frequencies after eccentric exercise suggest that changes in EC

coupling, via alterations in Ca^{2+} transport between the SR and sarcoplasm, contribute to the force loss experienced with EIMD (Hill et al. 2001; Sargeant and Dolan 1987). Sarcolemmal integrity may also be compromised by EIMD (Lovering and De Deyne 2004), however EMG has failed to consistently show a change in sarcolemmal depolarization as a result of muscle damage (Deschenes et al. 2000; Hamlin and Quigley 2001b; Piitulainen et al. 2008).

Centrally, alcohol acts at a number of locations in a dose dependent manner (Reilly 2002). Acute alcohol exposure may contribute to muscular weakness through alterations in acetylcholine receptor function at the post synaptic membrane as well as through inhibition of synaptic potentials (Bradley et al. 1980; Gruol 1982). Further, acute alcohol neuropathy may also contribute to this weakness through hyperalgesia and impairments in axonal transport (Koike and Sobue 2006).

Central fatigue, defined as a decrease in neural drive during muscular contraction (Taylor et al., 2006), and represented by a reduction in VA, has recently been proposed as a contributor to the early force loss seen after EIMD. These observed changes in VA are believed to result from supraspinal modulation due to inhibition at the motor cortex and/or motoneurones (Prasartwuth et al. 2006; Prasartwuth et al. 2005; Racinais et al. 2008). However, whether this decrease is associated with muscle pain during effort is currently unclear due to evidence for (Racinais et al. 2008) and against (Prasartwuth et al. 2005) this theory.

It is currently not known whether factors that contribute to alcohol related weakness interact with an already damaged muscle group, which may be more susceptible to alcohol's actions, to magnify EIMD related force loss. Therefore, the purpose of this study was firstly to investigate whether the combination of EIMD and post-exercise alcohol consumption magnify damage related losses in force, as observed previously (Barnes et al. 2010; **Chapter 5**). If this was indeed the case then the second aim of this study was to investigate the potential mechanisms behind this interaction. Finally, to allow us to draw more accurate conclusions from the study we also investigated whether acute alcohol consumption, in the absence of EIMD, may alter neuromuscular function and therefore be the cause of our previous observations (Barnes et al. 2010; **Chapter 5**). We hypothesized that alcohol would interact with

EIMD related alterations at the muscle level to exacerbate the typical force loss observed after strenuous eccentric exercise.

7.3 METHODS

7.3.1 Subjects

Twenty three healthy males (24.1 ± 5.6 years of age; 83 ± 12.6 kg, mean ± SD) volunteered to participate in this study. Subjects were randomly allocated into an exercising (EX) group (n = 12) or a non-exercising (NX) group (n = 11) so that the effect of alcohol on neuromuscular function, in the presence and absence of EIMD respectively, could be investigated. All subjects had at least 3 years of resistance training experience at a recreational level (at least twice per week) and were regular (1 or 2 drinking episodes per week) consumers of alcohol. Subjects were instructed to abstain from any form of exercise and alcohol consumption from 48 h before each trial until the last measures were made 60 h post beverage consumption. At least 2 weeks prior to the first trial subjects underwent familiarization of the exercise and neuromuscular performance measures used in the study. Identical familiarization was performed on both legs so that any eccentric exercise related repeated bout effect (McHugh 2003) or contralateral effect (Howatson and van Someren 2007) would be similar between legs. Leg dominance was determined during the familiarization process and subsequently used during trial allocation. The exercise protocol and all possible risks associated with participation were explained to each subject prior to them providing written consent. The experimental protocol was approved by the Massey University Human Ethics Committee and is in accordance with the guidelines set forth by the Declaration of Helsinki.

7.3.2 Overview

In order to allow us to directly compare the findings of this study with those observed in our earlier work a similar protocol was utilised in the present study (Barnes et al. 2010; **Chapter 5**). Subjects came to the laboratory in the evening, having consumed a standardized meal (3765 kJ) 4 hours earlier, to begin the first trial. After a pre-exercise venous blood sample was taken and baseline measures (PRE) of neuromuscular function made, subjects in the EX group completed a strenuous bout

of eccentric exercise. They then consumed either an alcoholic beverage or an isoenergetic, isovolumetric control beverage. Subjects in the non-exercising group (NX) completed the same process however they did not undertake the eccentric exercise. Follow-up blood samples were taken at 12, 36 and 60 h post-beverage consumption and neuromuscular performance measures were made 36 and 60 h post beverage consumption. These time points were originally chosen by Barnes et al. (2010) in an attempt to replicate the times at which optimal muscular performance may be realistically required after a team sport. That is, EIMD may occur during a game (Takarada 2003) after which large volumes of alcohol may be consumed (Maughan 2006). Training often begins again in earnest approximately 36 h post-game while by 60 h team training is likely to have recommenced.

At least two weeks after the first trial subjects returned to the laboratory and completed the second trial using the contralateral leg and the other beverage. Beverage consumed and leg dominance were randomly allocated in a counter balanced fashion so that equal numbers of subjects in the EX group completed their first trial with a combination of alcohol (ALC) or control beverage (OJ) and the dominant or non-dominant leg. Equal numbers of subjects in the NX group completed their first trial using their dominant leg and ALC, dominant leg and OJ or non-dominant leg and OJ while 2 subjects completed their first trial using their non-dominant leg and ALC.

7.3.3 Neuromuscular performance

7.3.3.1 Voluntary and electrically stimulated muscle tension

Subjects were seated on an adjustable, custom made straight back chair so that the hip and knee joints were positioned at an angle of 90°. An inextensible strap, connected to a load cell (Sensortronics, CA, USA), was attached slightly above the level of the lateral malleolus and a seat belt was fixed across the hips to isolate movement to the knee joint. Maximum voluntary isometric contraction (MVIC) and electrically stimulated (ES) isometric muscle tension was measured by the load cell connected to a custom made DC amplifier. The amplified signal was recorded and displayed using a Powerlab 8/30 and Chart for Windows (version 6.0) software (ADInstruments, Australia), respectively. Load cell calibration was performed in Chart for Windows prior to each use. In preparation for EMG and ES the thigh was shaved and the

location of the electrodes abraded and cleansed with an alcohol wipe. The time course of neuromuscular performance measures is illustrated in Figure 7.

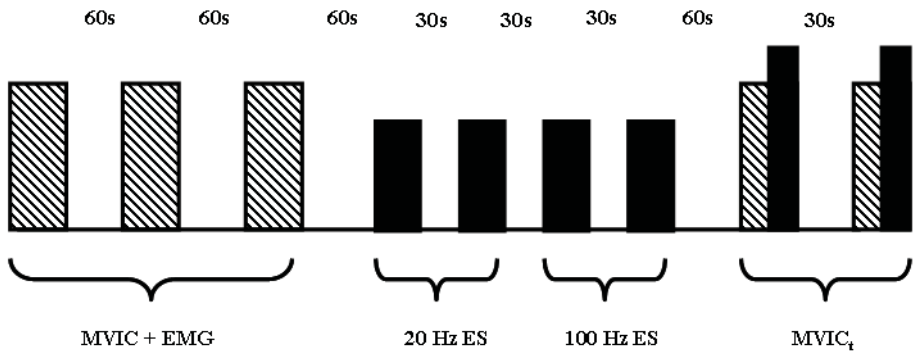
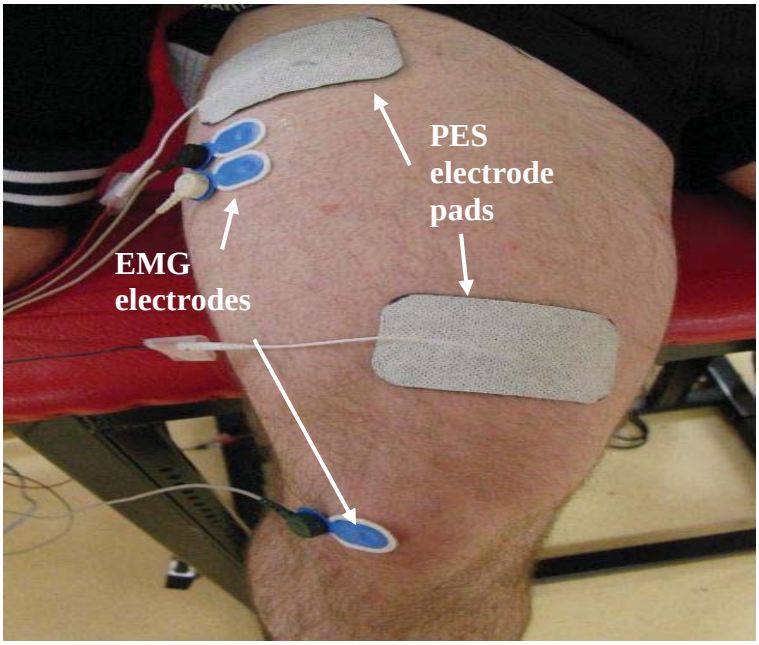


Figure 7 Schematic view of the neuromuscular performance protocol. Rest periods are included. See Methods for details. MVIC (hatched bars), maximal voluntary isometric contraction; iEMG, integrated electromyography; ES, electrical stimulation (solid bars); $MVIC_t$, MVIC with 100 Hz interpolated tetanus.

7.3.3.2 Percutaneuous electrical stimulation

Stimulation of the quadriceps was achieved via two 45 x 90 mm electrodes (Empi, MN, USA) placed longitudinally over the distal *vastus medialis* and the proximal *vastus lateralis* muscles (Lewek et al. 2001). Chart for Windows was programmed to send stimulus trains through a constant current stimulator (Digitimer DS7, Digitimer Ltd, England) to the electrodes. The square-wave pulse duration (100 μs) and source voltage (400 V) remained constant throughout testing. Output current ranged from 200-300 mA depending on each subject's maximal tolerable amplitude at 100 Hz, as obtained during earlier familiarization sessions. To ensure identical pad placement between tests, a permanent marker pen was used to trace around each electrode pad as well as the electrodes used for EMG recording. The experimental set up utilised is demonstrated in Figure 8.

A



B

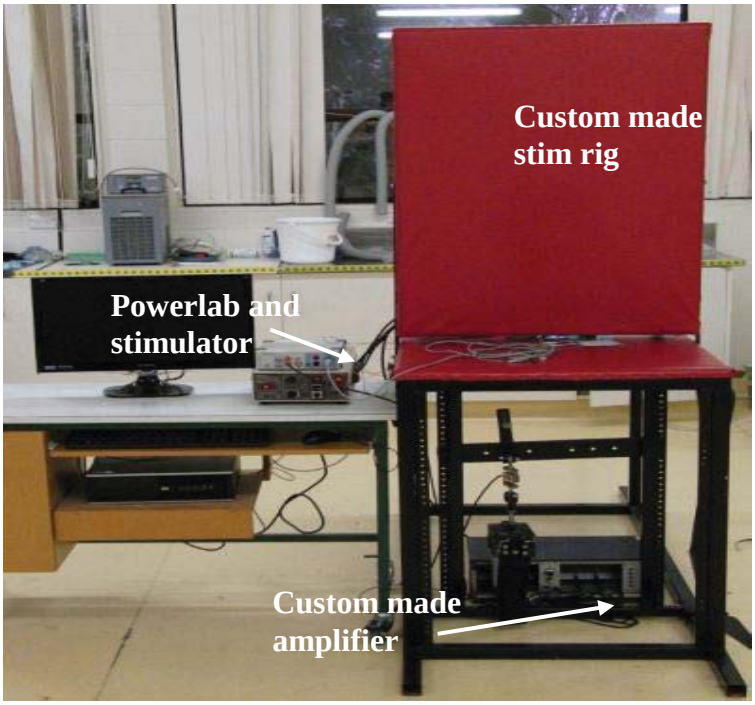


Figure 8 Positioning of percutaneous electrical stimulation and EMG electrodes (A) and customised electrical stimulation rig with accompanying Powerlab (ADInstruments, Australia) data acquisition unit, electrical stimualtor (Digitimer DS7, Digitimer Ltd, England) and customised amplified (B).

7.3.3.3 MVIC

Subjects performed three 3 s maximal isometric contractions of the quadriceps muscles separated by 30 s rest. The maximum value of the three efforts was used for further analysis.

7.3.3.4 EMG

Surface electromyography was recorded using 30 x 20 mm electrolyte gel filled Ag/AgCl electrodes (Ambu Blue Sensor, Denmark) placed 20 mm apart on the *vastus lateralis*, 1/3 the distance from the knee to the hip. A ground electrode was placed on the patella. The EMG signal was sampled at 1000 Hz during each 3 s MVIC. The signal was amplified (BioAmp, ADInstruments, Australia), band-pass filtered (10-500 Hz) and integrated in Chart for Windows (v6). A 50 Hz notch was applied for DC noise removal. Mean integrated EMG (iEMG) was analysed over a 500 ms period centred on the maximum MVIC value. Neuromuscular efficiency (N/mV), the mean MVIC divided by mean iEMG during this 500 ms period, was also calculated.

7.3.3.5 Voluntary activation

Two MVICs with an interpolated supramaximal 100 Hz tetanus of 500 ms duration were performed ($MVIC_t$), each separated by 1 min rest (Hill et al. 2001). The stimulus train was triggered by the investigator when a plateau in maximal force output was observed. VA was quantified as a percentage using the following formula:

$$\text{Voluntary activation (\%)} = (1 - a / b) \times 100$$

Where a is the maximum force from $MVIC_t$ and b is the force generated by the 100 Hz stimulus train in the resting muscle (Herbert and Gandevia 1999).

7.3.3.6 Low frequency fatigue

ES muscle tension of the quadriceps was carried out using 500 ms stimulus trains of 10 and 50 pulses discharged at frequencies of 20 and 100 Hz. Each frequency was repeated twice with a 30 s rest between stimuli. The ratio of the maximum force generated at 20 Hz and 100 Hz was analysed to investigate changes in the force frequency relationship and therefore the presence of LFF (Brown et al. 1997a). The

voltage used at 100 Hz induced a force of at least 40% MVIC when initially measured prior to the exercise bout.

7.3.4 Creatine kinase

Venous blood samples were obtained from the antecubital vein of subjects in the EX group. Blood was collected into a 4 ml EDTA-containing vacutainer, placed on ice for 10 min, and centrifuged at 4 °C for 10 min at 805×*g*. Plasma was aspirated into 300 µl aliquots and frozen at −80 °C for later analysis. CK activity was determined using a Vitalab Flexor clinical chemistry analyser (Vital Scientific NV, Netherlands) and a Roche CK-NAC liquid assay kit (Roche Diagnostics GmbH, Mannheim, Germany).

7.3.5 Eccentric exercise

Subjects in both groups warmed-up on a cycle ergometer (Monark, Varberg, Sweden) for five minutes at 100W before neuromuscular performance was assessed. Subjects in the EX group then completed 3 sets of 100 maximal eccentric contractions of the quadriceps of one leg on an isokinetic dynamometer (Biodex Medical Systems, New York, USA). Each repetition was performed over a 60° range of motion at an angular velocity of $30°\cdot s^{-1}$. Each set was separated by 5 min passive recovery. This protocol has previously been shown to bring about significant alterations in isokinetic and isometric force, elevations in CK and white blood cell concentrations (Barnes et al. 2010; MacIntyre et al. 1996).

7.3.6 Treatment

Having consumed a small standardized meal (1468 kJ) 30 minutes after exercise, subjects consumed either a beverage containing 1 g alcohol per kg BW (ALC), as vodka and orange (223 ± 35 mL vodka, 706 ± 111 mL orange juice, 2917.3 ± 459.2 kJ) or an isovolumetric, isoenergetic non-alcoholic control beverage of orange juice (OJ). This dose is equivalent to 8.3 ± 1.3 standard drinks (10 ml of alcohol). In order to balance treatments for both volume and energy content an additional volume of water (1007 ± 158 mL) was consumed along with the alcoholic beverage. Six equal volumes of the beverage were consumed at 15 minute intervals over a total time of 90 minutes. Thirty minutes after the consumption of the alcoholic beverage, blood alcohol concentration was estimated from breath alcohol content using a Digitech fuel

cell alcohol tester (Electus Distributions PTY, Ltd, NSW, Australia). Subjects were then driven home and required to go directly to bed.

Prior to beverage consumption in the first trial subjects were not told what beverage they would be consuming. However, due to the dose used, we have previously found it difficult to blind subjects as to which beverage they are consuming (Barnes et al. 2010; **Chapter 5**). Therefore no attempt was made to blind subjects in the present study. We therefore cannot rule out the possibility of expectancy effects, that is, subjects may have expected to perform differently because of the consumption of alcohol.

7.3.7 Statistical analyses

Analysis was carried out using the Statistical Program for Social Sciences (SPSS) for Windows (version 17.0, SPSS Inc., Chicago, IL.). A general linear-model three-way mixed-model analysis of variance (ANOVA) (Group x Treatment x Time) was used to compare treatments over time between groups for each measure. Additionally, a four-way mixed-model ANOVA (Group x Treatment x Time x Type (MVIC vs. $MVIC_t$)) was carried out to compare the force generated by MVIC and $MVIC_t$ between and within groups over time. Post-hoc pairwise comparisons using Bonferroni adjustment were performed to identify differences between groups, time points and treatments. Similarly, a three-way mixed-model ANOVA (Group x Trial (trial 1 vs. trial 2) x Leg (dominant vs. non-dominant leg)) was carried out to investigate possible order effects. As different legs were used in each trial percentage change (2 Times x 2 Treatments) was used for analysis of MVIC, LFF and iEMG. Data was checked for assumptions of normality and, as CK values were not normally distributed values were base-10 log transformed prior to statistical analysis. Additionally, the Pearson's product-moment correlation (r) was calculated to investigate the relationship between CK and MVIC. Reported values are means ± standard deviation (SD), except for Fig 7.2 which reports means ± standard error (SE) due to large inter-subject variation. Raw values for measures of neuromuscular performance are reported in Table 7.1. Statistical significance was set at the 95% level of confidence ($p < 0.05$).

7.4 RESULTS

7.4.1 MVIC

A significant Treatment effect (p = 0.005) and Treatment x Time (p = 0.017) and Time x Group (p = 0.016) interactions were found for percentage change in MVIC. At 36 h a greater decrease in force was evident under EX ALC (- 32.0 ± 11.8 %) compared to EX OJ (- 27.4 ± 13.0 %; p = 0.059). The greatest difference between the two treatments was seen at 60 h (p = 0.004) with MVIC 22.1 ± 12.5 % and 31.2 ± 10.9 % lower than PRE values for EX OJ and EX ALC, respectively. At 60 h EX OJ had improved from 36 h values (p = 0.002) however EX ALC had not (p = 0.383). In the NX group, a small but significant difference (6.24 %, p = 0.034) between treatments was evident at 60 h however MVIC at this time point was not significantly different from 36 h values under either OJ (p = 0.737) or ALC (p = 0.072). No other changes in MVIC were seen in the NX group under either treatment.

No main or interaction effects were found when comparing the force generated during MVIC to that generated during the voluntary contraction component of $MVIC_t$ (all $p > 0.05$).

Table 7 Raw values (mean ± SD) for maximum voluntary isometric contraction (MVIC), integrated EMG (iEMG), voluntary activation (VA) and low frequency fatigue (LFF) for the exercise (EX) and non-exercise (NX) groups under the control (OJ) and alcoholic treatments (ALC).

	EX (n = 12)			**NX (n = 11)**		
	Pre	36h	60h	Pre	36h	60h
MVIC (N)						
OJ	644.8 ± 131.7	471.7 ± 138.1*	504.5 ± 137.1*	659.5 ± 134.0	653.6 ± 120.6	656.1 ± 123.4
ALC	718.5 ± 156.2†	488.4 ± 156.4*	494.0 ± 130.1*	686.7 ± 141.3	666.3 ± 141.2	640.4 ± 139.8
iEMG (mV.s)						
OJ	0.252 ± 0.139	0.260 ± 0.139	0.254 ± 0.154	0.200 ± 0.088	0.207 ± 0.101	0.182 ± 0.081
ALC	0.230 ± 0.154	0.224 ± 0.081	0.179 ± 0.068	0.346 ± 0.084†	0.376 ± 0.127†	0.353 ± 0.109†
LFF (20:100 Hz)						
OJ	0.760 ± 0.098	0.635 ± 0.108*	0.666 ± 0.097*	0.750 ± 0.063	0.778 ± 0.053	0.763 ± 0.053
ALC	0.739 ± 0.050	0.627 ± 0.102*	0.627 ± 0.102*	0.762 ± 0.052	0.750 ± 0.060	0.758 ± 0.063
VA (%)						
OJ	83.1 ± 12.4	78.4 ± 9.9	77.5 ± 11.5	83.4 ± 8.7	89.5 ± 88.1	88.1 ± 4.7
ALC	86.5 ± 12.9	67.1 ± 17.5*†	69.4 ± 15.9*	86.9 ± 8.8	86.3 ± 7.8	88.4 ± 6.2

Note: Significantly different to Pre * $p < 0.05$

Significantly different to OJ † $p < 0.05$

7.4.2 iEMG

No difference in percentage change in iEMG was seen between groups, treatments or times (all $p > 0.05$). However, neuromuscular efficiency was significantly altered over time ($p < 0.001$) under both treatments in the EX group. Neuromuscular efficiency had decreased by 27.6 ± 11.7 % and 28.8 ± 18.8 % for OJ and ALC respectively, at 36 h. Neuromuscular efficiency had improved at 60 h so that it was not significantly different to pre-exercise values under either treatment (OJ $p = 0.19$, ALC $p = 0.39$). No change was seen in the NX group.

7.4.4 VA

A significant Time effect ($p = 0.008$) and Time x Group ($p < 0.001$) and Treatment x Time interactions ($p = 0.002$) were observed for VA. VA did not change over time under EX OJ, NX OJ or NX ALC (all $p > 0.05$). However, at 36 h EX ALC was 19.5 ± 18.4 % ($p = 0.003$) lower than PRE values. At 60 h VA was not different to 36 h values ($p = 1.00$) and was still 17.2 ± 16.7 % ($p < 0.001$) lower than PRE values.

7.4.5 LFF

A significant main effect of Time ($p = 0.035$) and a Time x Group ($p = 0.012$) interaction was seen for percentage change in the 20/100 Hz ratio. Values at both 36 and 60h were both lower than PRE values for both EX ALC and EX OJ (all $p < 0.05$). Due to a small non-significant ($p > 0.05$) increase in LFF under NX OJ, at 36 h a 5.8 % difference was observed between treatments in the NX group. However, no other changes were found in this group.

A significant reduction in the peak force generated by ES at both 20 and 100 Hz was observed over time under both EX OJ and ALC (all $p < 0.05$).

7.4.6 CK

Elevations in CK were observed over time under both treatments ($p = 0.004$, Fig. 9) with values at 36 and 60 h significantly greater than PRE values (all $p = 0.05$). No difference in CK was observed between treatments ($p = 0.723$). CK was not related to changes in MVIC (Pearson's correlation coefficient $r = - 0.145$, $p = 0.13$).

Analysis found no significant order effect of trial (all $p > 0.1$) or leg (all $p > 0.5$) for any of the criterion measures. Total work completed during the eccentric exercise bouts was not significantly different between trial one (35.7 ± 11.5 kJ) and trial two (37.6 ± 11.1 kJ) ($p = 0.15$) or between OJ (35.9 ± 11.5 kJ) and ALC trials (36.5 ± 11.5 kJ) ($p = 0.25$).

Breath alcohol levels were 0.0569 ± 0.0045 g/dL (12.35 ± 0.97 mmol/L) 30 minutes after the completion of alcohol consumption.

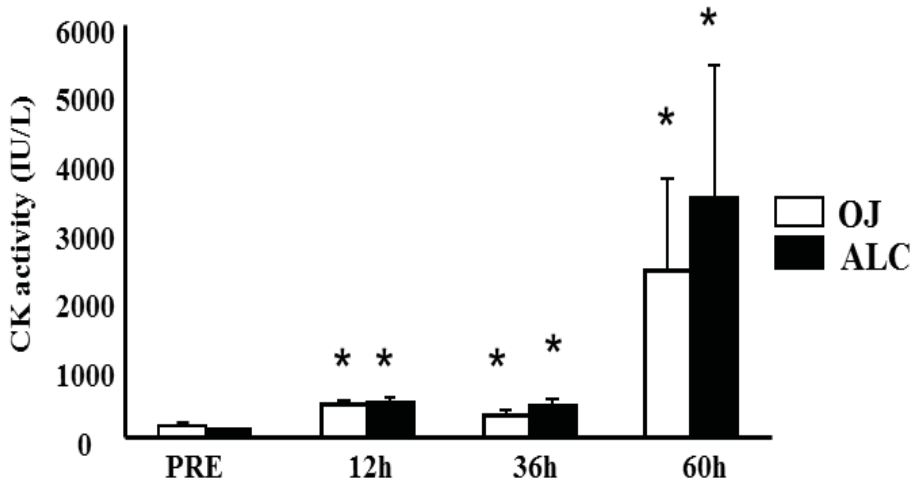


Figure 9 CK activity prior to (PRE) and 12, 36 and 60h post eccentric exercise under OJ (control) and ALC (alcohol) treatments. * Significantly different to pre-exercise values ($p < 0.05$). Values are mean ± SE.

7.5 DISCUSSION

Completion of 300 maximal eccentric contractions of the quadriceps produced a significant decrease in MVIC at 36 and 60 h post exercise. As previously observed (Barnes et al. 2010; **Chapter 5**) the consumption of 1 g of alcohol per kg BW after strenuous eccentric exercise resulted in a greater reduction in force at both 36 and 60 h compared to OJ. The observed decreases in MVIC occurred in the absence of changes to iEMG while decreases in LFF were seen under both treatments. As there

was no difference in MVIC and the voluntary contraction component of $MVIC_t$, which may have indicated subjects were anticipating the interpolated stimulus, the observed changes in VA at 36 and 60 h suggest that alterations in neural output may contribute to the greater force loss that occurs when alcohol is consumed acutely after eccentric exercise. Conversely, the consumption of the same dose of alcohol, in the absence of EIMD, had no effect on measures of neuromuscular performance. This finding is consistent with our previous results (Barnes et al. 2010) as well as the earlier work of Poulsen et al. (2007).

As typically occurs after strenuous eccentric exercise, CK increased significantly from PRE at all post-beverage time points under both OJ and ALC. Although elevations in CK have been observed in rat skeletal muscle tissue after alcohol administration (Amaladevi et al. 1995), as with the findings of Clarkson and Reichsman (1990), we found no difference in CK between ALC and OJ. Had a difference between treatments been observed it would be difficult for us to conclude that this was due to the additional effect of alcohol on the already damaged sarcolemma as it could equally indicate that the level of damage and/or balance between CK leakage and clearance differed between trials. As reported elsewhere (Brancaccio et al. 2007; Clarkson and Ebbeling 1988) large inter-subject variability was found in the CK response to the bout of eccentric exercise performed in this study, in particular at 60 h.

As previously reported (Deschenes et al. 2000; Hubal et al. 2007; Piitulainen et al. 2008), the force loss associated with strenuous eccentric exercise does not appear to be related to changes in sarcolemmal depolarisation, as illustrated by the absence of change in iEMG in the EX group in the present study. Additionally, as reported by Poulsen et al. , the dose of alcohol consumed by subjects in this study had no effect on iEMG, even in the EX group where we may have expected the sacrolemma to have been more susceptible to alcohol's actions because of the presence of structural damage. Although acute alcohol administration *in vitro* has been shown to elicit changes in sarcolemmal integrity and cellular excitability (Inoue and Frank 1967; Preedy et al. 2001; Urbano-Marquez and Fernandez-Sola 2004) identifying whether this effect also occurs *in vivo* is difficult. Whether a larger dose, representative of the quantities consumed by heavy alcohol consumers during binge drinking episodes

(Black et al. 1999; Nelson and Wechsler 2001; O'Brien et al. 2005) has an effect on force loss and iEMG, both with and without EIMD, is currently unknown.

During MVIC significant decrements in neuromuscular efficiency were evident at 36 and 60 h in the EX group under both OJ and ALC due to a large decrease in force production without concomitant changes in iEMG. The significant decrease in force produced by both low (20 Hz) and high (100 Hz) frequency ES of the relaxed quadriceps suggests that although muscular activity is unchanged damage to the contractile properties within the muscle as well as a change in the length–tension relationship may contribute to the observed force loss (Prasartwuth et al. 2005).

Low frequency fatigue, defined as a decreased ability to generate force at low (10 – 20 Hz) but not high (50 – 100 Hz) ES frequencies, is believed to contribute to the prolonged force loss associated with EIMD and is representative of a decrease in the ability of the SR to release and take up Ca^{2+} (Keeton and Binder-Macleod 2006). Similar to studies investigating the link between LFF and EIMD (reviewed by Jones (1996) the present study observed changes in the relationship between force generated at low and high frequencies in a damaged muscle group. Acute alcohol use has been shown, *in vitro*, to negatively affect sarcoplasmic Ca^{2+} transport (Cofan et al. 2000; Nicolas et al. 1998) however, as with iEMG, the results from the NX group in this study are similar to those of Poulsen et al. (2007) and suggest that acute alcohol related changes in Ca^{2+} transport do not contribute to muscular weakness or skeletal muscle contractility, even in the presence of EIMD.

Central fatigue, characterised as a decrease in VA, has been shown to contribute to the force loss that typically occurs during and immediately after eccentric exercise (Prasartwuth et al. 2005; Racinais et al. 2008). Given VA was not measured immediately after exercise in the present study it is difficult to predict whether the completion of 300 eccentric contractions of the quadriceps resulted in central fatigue as occurs after other damaging protocols (Prasartwuth et al., 2005; Endoh et al., 2005; Racinais et al., 2008) . The timeline of changes in VA in response to eccentric exercise are not well defined, possibly due to the different exercise protocols and methods used to quantify changes during and after exercise. Similar to the timeline of changes in VA presented here, Endoh et al. (2005) reported a decrease in VA during

fatiguing tests 2 and 4 days after a bout of eccentric exercise. However, the novel nature of our findings, that the combination of alcohol and EIMD, but not EIMD alone, appears to directly or indirectly impact the CNS to reduce VA, makes it difficult to compare our results to those of Endoh et al. (2005) and others (Prasartwuth et al., 2005; Racinais et al., 2008).

Although the mechanisms behind central fatigue have not been completely elucidated the use of TMS has shown that alterations in VA originate upstream of the motor cortex (Taylor et al. 2006). Similarly, studies using TMS have illustrated that acute alcohol consumption, at similar doses to that used in the present study, results in reduced motor cortex excitability 30 minutes after alcohol consumption (Kähkönen et al. 2003). How long alcohol continues to act on the motor cortex is currently unclear, however we cannot discount the fact that acute alcohol related changes within the motor cortex may combine with exercise induced central fatigue to prolong decreases in VA.

Along with the potential interaction between central fatigue and acute alcohol related changes in the motor cortex, inhibition of synaptic potentials (Gruol 1982), reduced axonal transport (Koike and Sobue 2006) and neurotransmission (Bradley et al. 1980) have been reported, *in vitro*, at physiologically tolerable doses. Whether the same is true *in vivo* is currently unclear. Changes in nicotinic acetylcholine receptor (AChR) numbers have been shown to correlate with alterations in skeletal muscle function (Zhang et al. 2010). As acute alcohol exposure alters AChR function (Bradley et al., 1980) we may speculate that alcohol induced changes at the neuromuscular junction may result in, what would presents as, reduced VA and related force loss. Although alcohol may act directly on the CNS it is unlikely that its action would continue to affect these factors once alcohol is fully metabolised. Given the lack of change in VA in the NX group and the fact that the dose used in the current study would have been fully metabolised within ~12 h of being consumed, it is unlikely that alcohol continues to directly influence these factors at 36 and 60 h.

Alternatively, changes in the perception of pain may have contributed to the reduction in VA seen here. Experimentally induced pain reduces maximal voluntary contraction force via a centrally mediated process (Graven Nielsen et al. 2002). This reduction is

likely the result of inhibition of motor cortex excitability, along with spinal modulation as reported by Le Pera et al. (2001). Similarly, the ability to sense effort, and therefore match force, in the presence of muscle pain is altered with subjects reportedly underestimating the level of force being generated by the sore muscle group. Again, it has been suggested that this is the result of a decrease in motor cortex excitability due to nociceptive afferent output from the sore muscle (Weerakkody et al. 2003).

Acute alcohol related weakness may be associated with muscular pain and tenderness similar to that caused by EIMD related DOMS. Alcohol related hyperalgesia is believed to be due to an increase in the production of a number of proteins (Dina et al. 2007; Eriksson 2001; Koike and Sobue 2006), many of which are also believed to be involved in inflammatory related pain, as occurs with DOMS, via their influence on nociceptor afferent output (Aley and Levine 1999; Lieber and Fridén 2002). Although the consumption of alcohol prior to eccentric exercise has been shown to have no effect on subjective, general ratings of soreness (Clarkson and Reichsman 1990) we cannot discount the possibility that alcohol consumed after damaging exercise may result in an increase in sensations of pain during maximal muscular contraction, thus resulting in a reduction in VA. Indeed, anecdotally a number of subjects in the EX group remarked that all measurements, both voluntary and electrically induced, at 36 and 60 h under ALC were more painful than the same measures made under OJ. The fact that there was no difference between forces generated during MVIC and $MVIC_t$ under any of the treatments, within either group suggests that anticipation of electrical stimulation did not contribute to the decrease in VA. The relationship between muscle damage, acute alcohol consumption and pain warrants further investigation in the future.

7.5.1 Conclusion

In conclusion, the ability to generate maximum voluntary isometric force with the quadriceps was greatly reduced when post-exercise alcohol consumption was combined with EIMD. Conversely, the consumption of alcohol in the absence of EIMD had no effect on any of the measures made in this study. Together with the findings of our earlier work (Barnes et al. 2010; **Chapter 5**) , the results of this study further suggest that, when combined with damaged skeletal muscle, the consumption

of 1 g alcohol per kg BW has a detrimental effect on muscular performance in the days following exercise. Alcohol's effect appears to be mainly due to alterations in neural output, in conjunction with damage related changes in muscle contractile properties. It is proposed that this prolonged effect may be due to alcohol related hyperalgesia.

CHAPTER 8

The Effects of Acute Alcohol Consumption on Recovery from a Simulated Rugby Match

Publication:
Barnes MJ, Mündel T, Stannard SR (2012). Journal of Sports Sciences: 30 (3):295-304. www.tandfonline.com

8.1 ABSTRACT

We investigated the effects acute post-exercise alcohol consumption has on measures of physical performance, CK and immunoendocrine function in the 48 hours following a rugby game simulation. Ten male senior rugby union players completed a rugby game simulation after which they consumed either 1 g alcohol per kg BW or a non-alcoholic control beverage. Agility, 15 m sprint, CMJ and srummaging performance was measured pre-simulation and 24 and 48 h post-simulation. White blood cell count, testosterone, cortisol and CK were measured pre-simulation and 30 minutes, 12, 24, 36 and 48 h post-simulation. One week after the first trial subjects completed the second simulation after which the other beverage was consumed. Treatment was allocated using a cross-over design so that equal numbers of subjects completed either the alcohol or control treatment during their first trial. The acute consumption of alcohol after a rugby game simulation negatively affected CMJ performance in the days following the simulation ($p = 0.028$). No differences between treatments were observed for the other criterion measures made in this study. In conclusion, when consumed after 80 minutes of a simulated rugby game the consumption of 1 g alcohol per kg BW negatively impacts lower body vertical power output, however performance of tasks requiring repeated maximal muscular effort is not affected.

8.2 INTRODUCTION

Hazardous, acute alcohol consumption occurs regularly in many sports, in particular male dominated team sports, as part of post-game celebration, team culture, or associated with sponsorship commitments (Maughan 2006). Participants in these sports have reported regular bingeing on alcohol at higher rates than the general, non-sporting population (Nelson and Wechsler 2001; O'Brien et al. 2005). Although this behaviour is prevalent and often seen as an acceptable part of a team culture, surprisingly little research has been carried out to investigate the effects of such behaviour on performance recovery after exercise or competition. Recently, Barnes et al. (2010; **Chapters 5 and 7**) investigated the effects of an acute alcohol binge episode on recovery of skeletal muscle performance after a bout of strenuous eccentric exercise. These studies revealed that alcohol magnifies the level of force loss typically associated with exercise-induced muscle damage through an interaction between alcohol and the damaged skeletal muscle. Others investigating alcohol's impact on recovery have focused on rehydration, (Shirreffs and Maughan 1997) glycogen repletion (Burke et al. 2003) and hormonal changes (Heikkonen et al. 1996) rather than looking at its effect on subsequent performance directly. Yet the true index of recovery is repeat performance.

Historically, a strong relationship has existed between heavy alcohol consumption and the sport of rugby union (Collins and Vamplew 2002) with many players reporting higher levels of alcohol use than participants in other sports or the general population (Black et al. 1999; O'Brien and Lyons 2000; Quarrie et al. 1996). Like a number of other football codes, rugby union is characterized by repeated short duration, high intensity activity, including short sprints and physical contact, interspersed with periods of jogging, walking and standing (Deutsch et al. 2007). Although such activities, particularly the contact components, are likely to have a detrimental effect on performance in the days following the game very little research has confirmed/investigated this. In fact the majority of research investigating recovery after a rugby match has quantified changes in markers of muscle damage (Cunniffe et al. 2010; Gill et al. 2006; Smart et al. 2008) and endocrine (Cunniffe et al. 2010; Elloumi et al. 2003) and immune function (Cunniffe et al. 2010; Suzuki et al. 2004) rather than characterizing the effects such strenuous activity may have on physical

performance. Due to the unpredictable nature and variability of the physical demands of each game, simulated rugby games have been used to quantify changes in performance brought about by various interventions (Hamlin et al. 2008; Hinckson et al. 2007; Roberts et al. 2010a; Stuart et al. 2005). Based on time-motion analysis of game play, this strategy ensures ecological validity while allowing the number and duration of each component of the game to be controlled in a repeatable manner.

Although acute alcohol consumption has previously been shown to impair recovery when consumed after a strenuous bout of laboratory-based eccentric exercise (Barnes et al. 2010; **Chapters 5 and 7**), it is currently not known if the same is true when whole body, sport specific exercise is used to bring about fatigue and muscle damage, as occurs in the field. Therefore, the primary aim of this study was to investigate the effects of post-game alcohol consumption on whole body, sport specific performance in the days following a simulated rugby game. Additionally, the secondary aim of this study was to investigate the effectiveness of a modified version of the Bath University Rugby Shuttle Test (BURST) (Roberts et al. 2010b) as a means to bring about changes in markers of muscle damage, performance and immunoendocrine function, as may occur after a game of rugby.

8.3 METHODS

8.3.1 Subjects

Ten male senior grade club rugby players (age = 21.3 ± 2.4 years, mass = 90.2 ± 16.4 kg, height = 178.2 ± 9.6 cm) volunteered to participate in this study. Subjects had completed their rugby season three weeks before the beginning of their first trial and therefore had a good level of "game specific fitness". Familiarization of the protocol was carried out two weeks before the first trial and involved subjects completing half of the simulation and all performance measures. Subjects were instructed to abstain from exercise and alcohol consumption from 48 h before the simulation until the last performance measures were taken 48 h post-simulation. Subjects recorded their diet from the morning of the first trial until 48 h post-simulation and were instructed to replicate this for the second trial. Additionally, subjects were also instructed to abstain from practices that could potentially improve or worsen their recovery, for example the use of anti-inflammatory medication, massage, stretching, protein

supplementation and cryotherapy during the 48 h post-simulation period. One week separated trials, and treatment was allocated randomly in a balanced fashion. All subjects signed an informed consent document prior to their involvement, and the study was approved by the Massey University Human Ethics committee.

8.3.2 Overview

Subjects were randomly allocated into pairs of similar playing position and presented to the laboratory three hours after consuming a standardized meal (3700 kJ; CHO 110 g, Fat 11 g, Protein 26 g). Body mass was recorded (Jadever Scale Co. Ltd., Taiwan), a venous blood sample collected and a heart rate monitor (Polar Electro Oy, Finland) fitted. Subjects then warmed up and performance measures were made after which subjects immediately began the simulation. Subjects completed the simulation in their allocated pairs and alternated contact activities so that an equal number of each activity was completed by subjects throughout the simulation. At the completion of the simulation subjects were reweighed, a venous blood sample was collected and they then consumed a standardized meal (1532 kJ; CHO 50.4 g, Fat 9.1 g, Protein 7 g). Thirty minutes later subjects began consuming a set amount of either an alcoholic or non-alcoholic beverage which they consumed over 90 minutes. Each participant was then driven home and required to go directly to bed. They then returned to the laboratory 12, 24, 36, and 48 h post-simulation for blood sampling while follow up performance measures were made 24 and 48 h post-simulation. One week after the first trial (Trial One) subjects completed the second trial (Trial Two) in which the alternative beverage was consumed.

8.3.3 Game simulation

In an attempt to replicate the physical demands of a rugby union game we modified the BURST protocol (Roberts et al. 2010b) to include direct game specific contact between subjects. This important component has been absent from previous rugby game simulations (Hamlin et al. 2008; Hinckson et al. 2007; Roberts et al. 2010b; Singh et al. 2010). Based on time motion analysis of English senior elite level rugby, this protocol has been shown to have a good level of reproducibility across repeated trials (Roberts et al. 2010b). To our knowledge this is the first time the BURST protocol has been used in this manner.

Summarized in Figure 10, the simulation was divided into quarters, each comprising four blocks of five exercise cycles. Quarters 1 and 2 and quarters 3 and 4 were separated by a four minute break consisting of two minutes of passive recovery and two minutes of walking. A ten minute half time break, with seven minutes passive recovery and three minutes walking, separated quarters two and three. The mean time taken to complete each simulation was 122 ± 1 minutes. As two subjects completed the simulation at the same time, 24 minutes of the total time taken to complete the simulation was spent resting by one participant or the other (16 x 45 s rest time each) during which time the other participant completed the HID. Subtracting this rest time, and the time spent resting between quarters, our subjects were actively engaged in the simulation for 80 minutes.

Measurements of HR, lactate (La^-) (LactatePro, Arkray Inc, Japan), and RPE (Borg 1970) were taken at the beginning of each break and at the completion of the simulation. This data was later analysed to compare relative intensities between trials. During each break subjects consumed 2 mL of water per kg body mass (180 ± 33 mL).

In each exercise cycle subjects walked 20 m then turned 180° and ran 20 m. They then either pushed in a dynamic or static fashion (Scrum or Ruck, respectively) against a cart dynamometer (Grunt 3000, E-Type Engineering, New Zealand), tackled or were tackled around the thighs (Tackle) onto a 5 cm gymnastics mat or wrestled against the other participant for possession of a rugby ball for 10 s (Maul). For safety reasons, subjects were instructed to tackle at ~70% of their normal game effort. Subjects had 20 s to complete the given contact activity before jogging 20 m and resting for 5 s before beginning the next cycle. Each cycle was 51 s in duration. Pacing was maintained by a set of infrared, programmable timing gates (Smartspeed, Fusion Sport, Australia). At the completion of every fifth exercise cycle subjects completed a High Intensity Drill (HID) comprised of an agility task and a 15 m sprint (see Performance Measures section for details). Within trial HID performance was recorded and analysed for reliability between the two trials.

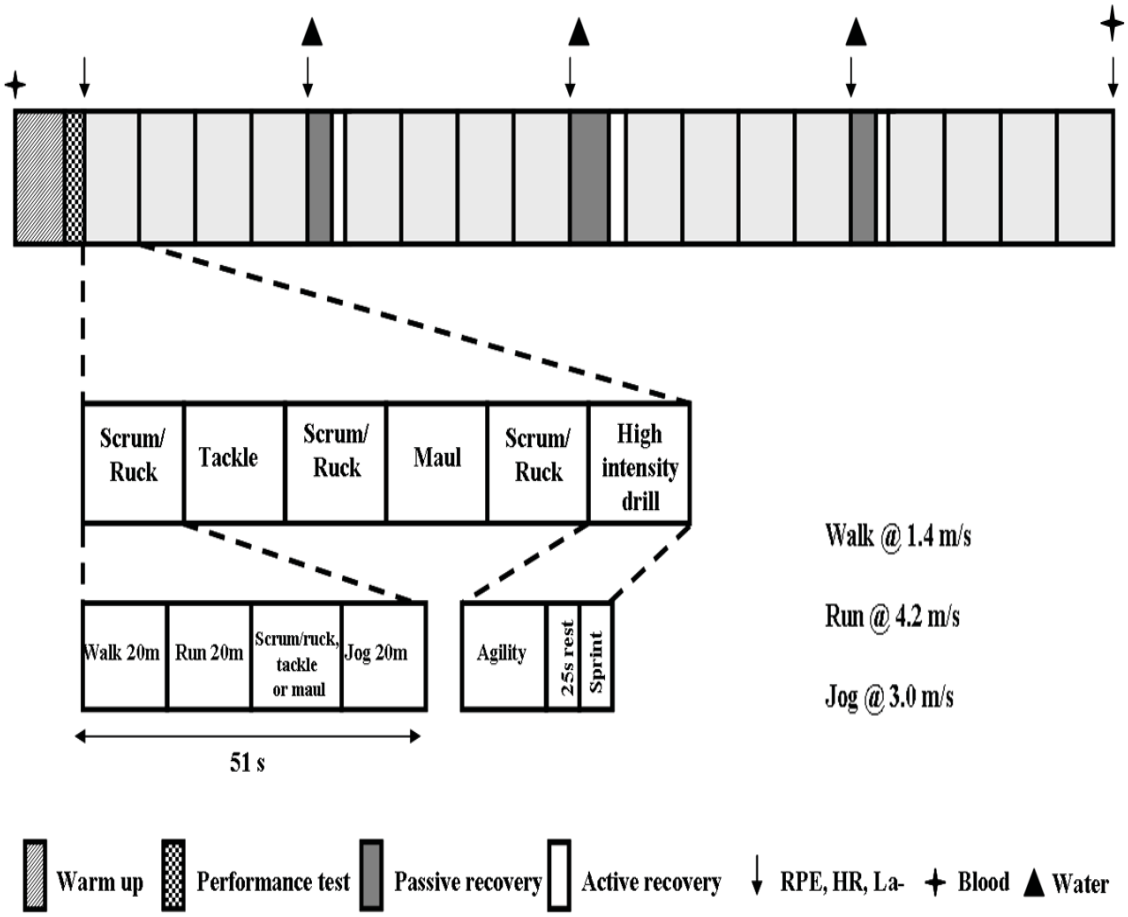


Figure 10 Schematic view of the simulation. Subjects completed 16 blocks of exercise (four blocks per quarter). Each block involved subjects performing five circuits in which they were required to walk 20 m, run 20 m, perform a contact drill and then jog 20 m. Each circuit took 51 s. At the end of the fifth circuit subjects performed a high intensity drill (Figure 10) which included an agility task and 15 m sprint. After blocks 4 and 12 subjects rested for 4 minutes (quarter time) while between blocks 10 and 9 subjects rested for 10 minutes (half time). Each simulation took 122 ± 1 minutes to complete.

8.3.4 Performance measures

Prior to the measurement of performance at each time point subjects performed a standardised five minute warm-up, which included jogging, sprinting and stretching, after which they completed one block of five exercise cycles (4 minutes 15 s, as outlined in the previous section). CMJ height, scrummaging power and high intensity drill performance were then measured, in that order, for both subjects.

8.3.4.1 Counter movement jump

Immediately after their warm-up subjects performed three CMJs (Harman et al. 1990), each attempt was separated by 30 s. Jump height was measured with an electronic jump mat (Swift Performance, NSW, Australia) and the maximum value was recorded.

8.3.4.2 Scrummaging power

Horizontal power output was measured using the Grunt 3000 cart dynamometer. Subjects placed their shoulders against the pads of the cart at a set height and were instructed to push against the cart as rapidly as possible using their legs, arms and shoulders (Stuart et al. 2005). Force and acceleration outputs from the cart dynamometer were collected and converted to power (W) using a Powerlab 8/30 data acquisition system and Chart for Windows (version 6.0) software (ADInstruments Pty, NSW, Australia). Three attempts, separated by 30 s, were made with the greatest peak power output recorded.

8.3.4.3 High intensity drill

Adapted from Roberts et al. (2010b) and summarised in Figure 11, subjects completed an agility task which involved carrying a15 kg tackle bag (Powa Products, New Zealand) 9 m twice before weaving through three cones as fast as possible. After a 25 s rest subjects performed a 15 m Sprint. Agility task and 15 m sprint times were recorded with infrared timing gates (Swift Performance, NSW, Australia) and analysed separately. Other than the 25 s recovery period the HID was performed at maximal speed.

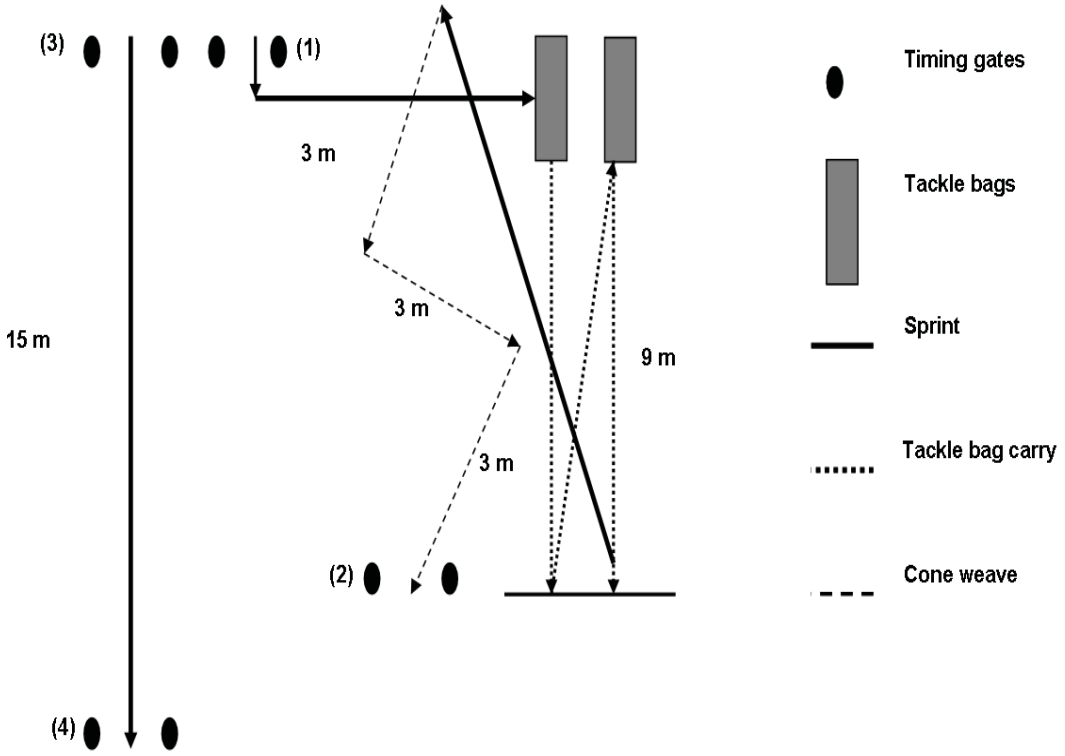


Figure 11 Schematic view of the High Intensity Drill. Subjects picked up the first tackle bag and carried it 9 m. They then collected the second tackle bag, carried it 9 m and then weaved through 3 cones set 3 m apart at an angle 45 °. Agility task time was recorded with timing gates (1) and (2). Subjects then rested for 25 s before sprinting 15 m through timing gates (3) and (4). Not to scale.

8.3.5 Treatment

Thirty minutes after completion of the simulation, and having consumed a standardised meal subjects began drinking a beverage containing either 1 g alcohol per kg BW as vodka (37.5% alcohol/volume; Smirnoff, Australia) in orange juice (Frucor Beverages, New Zealand) (ALC) or an isovolumetric, isoenergetic control beverage of orange juice alone (OJ). The mean volume of vodka consumed per participant was 239.5 ± 51.4 ml. In order to balance total energy value (3131.3 ± 559.8 kJ) and fluid volume (1840.1 ± 329.0 ml) between trials subjects consumed a greater volume of orange juice in the OJ trial while in the ALC trial they consumed an additional volume of water (842.1 ± 150.6 ml) along with the alcoholic beverage. An

equal volume of beverage was consumed every 15 minutes over a total time of 90 minutes.

Thirty minutes after the consumption of the alcoholic beverage, blood alcohol concentration (BAC) was estimated from breath alcohol content using a Digitech fuel cell alcohol tester (Electus Distributions PTY, Ltd, NSW, Australia).

8.3.6 Blood measures

8.3.6.1 Sample collection

Venous blood samples were obtained from the antecubital vein and collected into 4 mL K_3EDTA vacutainer tubes (Beckton Dickinson, UK). Samples were kept at room temperature for 15 minutes before standard haematological analysis was carried out. For other measures (CK, testosterone and cortisol) a second 4 mL K_3EDTA vacutainer tube was collected at the same time and subsequently centrifuged at 4° C for 10 minutes at 1650 *g*. Plasma was aspirated into appropriate aliquots and frozen at −80° C for later analysis.

8.3.6.2 Creatine kinase

CK activity was determined using a Vitalab Flexor clinical chemistry analyser (Vital Scientific NV, Netherlands) and a Roche CK-NAC liquid assay kit (Roche Diagnostics GmbH, Mannheim, Germany).

8.3.6.3 Immune cell concentrations

Analysis of total and differential leukocytes was performed using an automated cell counter (A^{c}T 5diff Hematology Analyzer, Beckman Coulter, USA).

8.3.6.4 Hormonal variables

Radioimmunoassay (1470 Automatic Gamma Counter, Perkin Elmer, MA, USA) using commercially available kits (DiaSorin, MN, USA) was carried out to analyze for plasma cortisol and testosterone.

8.3.7 Statistical analysis

Data was analysed using the Statistical Program for Social Sciences (SPSS) for Windows (version 17.0, SPSS Inc., Chicago, IL.). A general linear-model two-way repeated-measures ANOVA (treatment x time) was used to compare changes within and between trials (OJ vs. ALC, trial 1 vs. trial 2). Post-hoc pairwise comparisons using Bonferroni adjustment were performed to identify differences between time points within each treatment. Reported values are mean ± SD. Statistical significance was set at $p < 0.05$. Reliability measures between trials 1 and 2 for 15 m sprint and agility drill performance were calculated using methods described by Hopkins (2000). This analysis provided change in mean, coefficient of variation (CV %), intraclass coefficient and related 95 % confidence limits (CL). Data were also analysed using inferential statistics with 90 % CL to produce magnitude-based inferences about the observed outcomes. Threshold values were derived from the smallest standardised (Cohen) difference in mean (0.2) multiplied by between-subject pre-simulation SD. The likelihood of the outcome being beneficial, trivial or harmful was determined using the spread sheet of Hopkins (2007). The following schema was used to determine the likelihood of the outcome being beneficial, harmful or trivial: < 0.5%, most unlikely; 0.5 - 5 %, very unlikely; 5-25 %, unlikely; 25 - 75 %, possibly; 75 - 95 %, likely; 95 – 99.5 %, very likely; > 99.5 %, most likely.

8.4 RESULTS

8.4.1 Simulation

As outlined in Table 8, subjects covered a total of 6988 m and completed 80 contact activities throughout the simulation.

Lactate (La^-), but not HR, changed significantly over the course of each simulation ($p < 0.01$) however no difference in either variable was seen between OJ and ALC trials (La^-, $p = 0.74$; HR, $p = 0.62$). Similarly, RPE increased over time ($p < 0.001$) during each simulation. Unlike La^- and HR, a significant difference between OJ and ALC trials ($p = 0.001$) was found for RPE. Post-hoc analysis found that this difference was the result of participants rating the third quarter of the ALC trial easier than the same period during the OJ trial (OJ = 15.2 ± 1.6; ALC = 16.5 ± 1.2; $p = 0.015$).

No significant order effect in La^- or RPE was seen between Trials One and Two, however HR was significantly different between trials (p = 0.011). Mean HR at the end of the second and third quarters of Trial Two was 5 (p = 0.001) and 4 (p = 0.012) beats per minute lower, respectively, than equivalent measures in Trial One.

Table 8 Summary of distances covered and number of contacts made during each simulation.

ACTIVITY	
Tackles made	8
Tackled	8
Scrum	24
Ruck	24
Maul	16
Walk	2140 m
Jog	2284 m
Run	1660 m
Sprint	904 m
Total Distance	6988 m

Changes in body mass pre- to post-simulation were 1.82 ± 0.82 kg for OJ and 1.45 ± 0.51 kg for ALC. There was no significant difference between OJ and ALC trials (p = 0.25) or between Trial One and Trial Two 2 (p = 0.86).

Analysis for reliability found a mean change of -0.07 s (95 % CL, -0.04 to -0.09; ICC r = 0.51 and CV of 4.4 % (3.01, 3.77) for 15 m sprint performance and a mean change of – 0.42 s (95 % CL, -0.55 to -0.29), ICC r = 0.52 and CV of 3.29 % (95 % CL, 4.04 to 5.08) for agility drill performance. This indicates that both sprint and agility drill

performance throughout Trial Two was faster than the same effort in Trial One. The low ICC values suggest a poor relationship and, therefore, low reliability for these measures between the two trials.

Average ambient conditions in the gymnasium were not different between trials for temperature (OJ, 16.6 ± 0.5° C; ALC, 6.36 ± 0.4° C; p= 0.47) and relative humidity (OJ, 72.3 ± 3.4 %; ALC, 71.4 ± 3.4 %; p = 0.49) or between Trial One (16.4 ± 0.4° C, 71.5 ± 3.9 %) and Trial Two (16.5 ± 0.6° C (p = 0.81), 72.2 ± 2.8 % (p = 0.60)).

8.4.2 Performance measures

Scrummaging and 15 m sprint performance did not change significantly over time (p = 0.25 and 0.36, respectively). Agility drill performance improved over time under both OJ and ALC (p = 0.001), however no difference between treatments was observed for any of these three performance variables (all p > 0.05). CMJ did not change over time (p = 0.075) however a significant treatment x time effect (p = 0.028) was found. Decreases of 5.1 and 3.8 % (pre = 39.6 ± 4.5 cm, 24 hours = 37.6 ± 3.98 cm, 48 hours = 38.1 ± 4.7 cm) were observed at 24 and 48 hours, respectively, under ALC. No difference in CMJ was seen under OJ (pre = 37.7 ± 5.2 cm, 24 hours = 37.8 ± 5.3 cm, 48 hours = 37.8 ± 6.0 cm). The results of performance measures are summarized in Table 2. No order effect of trial was seen for any of the performance measures (all p > 0.8).

Analysis of performance measures using inferential statistics showed that the effects of alcohol on performance were 100 % most likely trivial (CMJ 0.12 ± 0.2 cm, 90 % CL; Agility Drill 0.004 ± 0.039 s, 90 % CL; Scrummaging 0.1 ± 0.18 W, 90 % CL) or unclear (15 m Sprint 0.003 ± 0.037 S, 90 % CL).

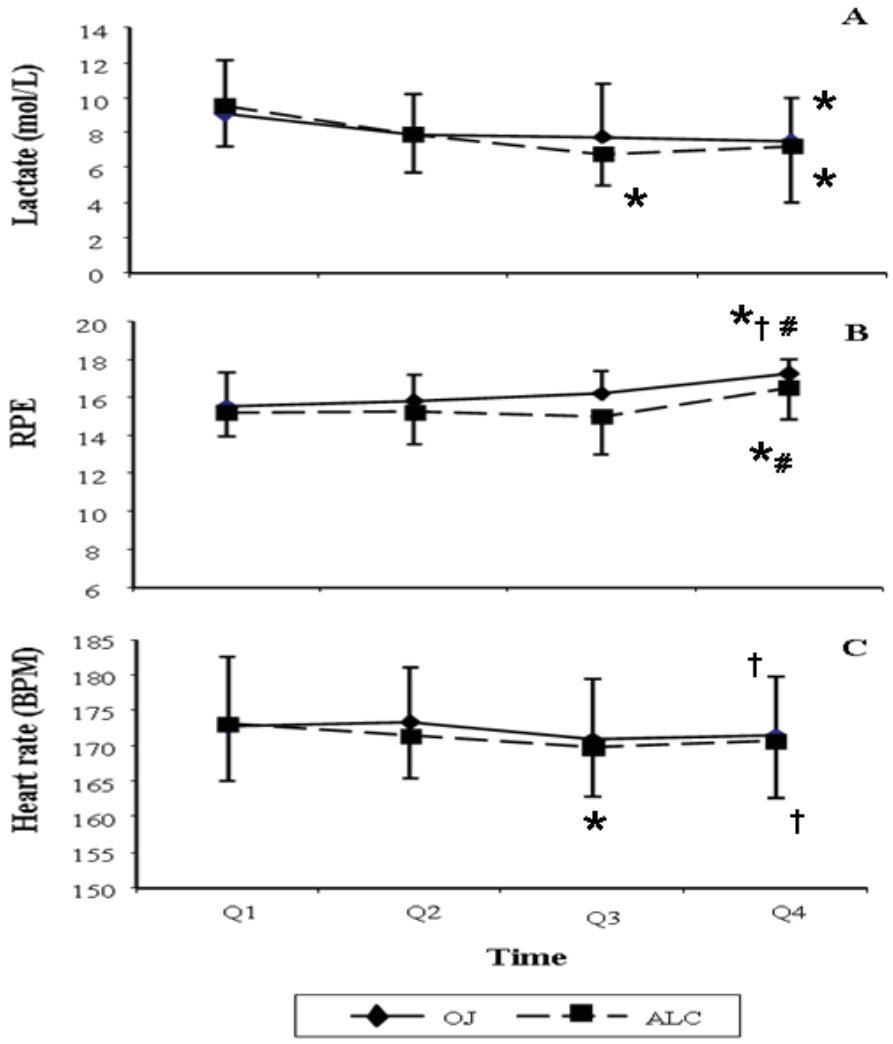


Figure 12 Between treatment (control (OJ) vs. alcohol (ALC)) comparisons of lactate (A), RPE (B) and heart rate (C) responses over four quarters (Q1-Q4) of a rugby game simulation (mean ± SD). * Significantly different to Q1 value ($p < 0.05$). † Significantly different to Q2 value ($P < 0.05$). # Significantly different to Q3 ($p < 0.05$).

Table 9 Scrummaging power, counter movement jump height (CMJ), 15m sprint time and time to complete an agility drill before and 24 and 48 h after a rugby game simulation (mean ± SD).

	Treatment	Pre	24 h	48 h
Scrummaging (W)	OJ	1189 ± 247	1218 ± 223	1205 ± 195
	ALC	1202 ± 264	1230 ± 281	1292 ± 259
CMJ (cm)	OJ	37.7 ± 5.2	37.8 ± 5.2	37.6 ± 6.0
	ALC	39.6 ± 4.5	37.6 ± 3.8	38.1 ± 4.7
15m Sprint(s)	OJ	2.64 ± 0.08	2.66 ± 0.13	2.72 ± 0.13
	ALC	2.74 ± 0.16	2.46 ± 0.11	2.65 ± 0.11
Agility Drill (s)	OJ	17.72 ± 0.60	16.88 ± 0.35*	17.00 ± 0.41*
	ALC	17.57 ± 0.54	17.06 ± 0.74	16.89 ± 0.55*

* Significantly different to pre-simulation values ($p < 0.05$).

8.4.3 Blood measures

A main effect of time was observed for all blood parameters (Table 10 and 11, all $p < 0.001$) however no difference between treatments was evident for any of these measures. Total leukocyte ($p = 0.02$), neutrophil ($p < 0.001$) and monocyte ($p < 0.039$) concentrations increased significantly post-simulation before returning towards pre values at 12 h. Plasma CK activity was elevated post-simulation under both treatments however, due to large variation with OJ, significant elevations in CK were only seen with ALC over the subsequent 48 h period. Closer inspection of CK values revealed that the large variation observed with OJ was due to two subjects who had significantly higher values in response to the OJ trial compared to the ALC trial. It is unclear why this difference occurred. If the CK data for these two subjects is removed the CK response to both trials is similar (Time, $p < 0.001$; Treatment, $p = 0.98$; Treatment x Time, $p = 0.65$). Compared to pre-values cortisol increased significantly under both treatments at 12 h (both $p = 0.001$) after which time values returned to pre levels ($p = 1.00$). A second rise in cortisol levels ($p = 0.01$) was seen at 36 h under ALC only. Unlike cortisol, testosterone levels were not significantly different to pre-values at any given time under either treatment.

Table 10 Immune cell concentrations pre-game simulation, immediately post and 12, 24, 36 and 48 h post-game simulation (mean ± SD).

	Treatment	Pre	Post	12 h	24 h	36 h	48 h
Total Leukocytes	OJ	7.85 ± 1.11	13.67 ± 4.44*	8.11 ± 2.65	7.90 ± 1.25	6.91 ± 2.41	7.85 ± 2.4
(10^9/L)	ALC	7.86 ± 1.13	13.49 ± 2.29*	7.78 ± 1.55	7.94 ± 1.74	6.69 ± 0.81	7.71 ± 1.37
Neutrophils	OJ	4.26 ± 1.05	10.61 ± 3.23*	4.25 ± 1.52	4.04 ± 0.68	3.49 ± 2.04	4.61 ± 2.10
(10^9/L)	ALC	4.38 ± 0.85	9.79 ± 2.00*	4.12 ± 0.85	4.22 ± 1.76	3.80 ± 1.66	4.02 ± 1.03
Monocytes	OJ	0.64 ± 0.14	1.03 ± 0.32*	2.77 ± 1.00	0.67 ± 0.14	0.64 ± 0.22	0.69 ± 0.22
(10^9/L)	ALC	0.66 ± 0.14	1.00 ± 0.25*	2.60 ± 0.65	0.68 ± 0.14	0.79 ± 0.51	0.64 ± 0.19
Total Lymphocytes	OJ	2.69 ± 0.74	2.23 ± 0.58*	0.74 ± 0.22	2.85 ± 0.80	2.48 ± 0.73	2.40 ± 0.56
(10^9/L)	ALC	2.58 ± 0.70	2.25 ± 0.49*	0.73 ± 0.15	2.72 ± 0.66	2.65 ± 1.07	2.57 ± 0.62

OJ control, ALC alcohol treatment

* Significantly different to pre-simulation values ($p < 0.05$).

Table 11 CK, testosterone and cortisol pre-game simulation, immediately post and 12, 24, 36 and 48 h post-game simulation (mean ± SD).

	Treatment	Pre	Post	12 h	24 h	36 h	48 h
CK	OJ	120 ± 61	359 ± 154*	648 ± 470	658 ± 474	473 ± 380	392 ± 308
(IU/L)	ALC	138 ± 76	277 ± 105*	481 ± 188*	452 ± 196*	395 ± 157*	312 ± 128*
Testosterone	OJ	11.0 ± 4.6	14.7 ± 6.9	12.9 ± 4.1	11.0 ± 5.3	12.6 ± 3.2	10.7 ± 5.4
(nmol/L)	ALC	10.4 ± 4.1	13.1 ± 5.1	13.5 ± 3.9	11.6 ± 5.3	13.8 ± 5.0	9.8 ± 4.1
Cortisol	OJ	333 ± 154	621 ± 247*	663 ± 147	345 ± 207	608 ± 180	336 ± 157
(nmol/L)	ALC	299 ± 142	517 ± 260*	702 ± 193	308 ± 119	543 ± 181	376 ± 147

OJ control, ALC alcohol treatment

* Significantly different to pre-simulation values ($p < 0.05$).

8.4.4 Blood alcohol concentration

The average BAC recorded 30 minutes after the consumption of 1g of alcohol per kg bodyweight was 0.067 ± 0.003 g/dL (14.6 ± 0.72 mmol/L).

8.5 DISCUSSION

The primary purpose of this study was to investigate whether an acute dose of alcohol impacts physical performance, markers of skeletal muscle damage and immunoendocrine function in the days following a rugby game simulation, when compared to a non-alcoholic beverage. The main finding of this study was that, when consumed after a simulated game of rugby, a dose of 1 g alcohol per kg BW negatively impacts lower body power output, as measured by CMJ. Having previously shown that this dose of alcohol interacts with damaged skeletal muscle to magnify losses in performance after eccentric exercise (Barnes et al. 2010; **Chapters 5 and 7**) we may speculate that the decrease seen with alcohol in the current study is similarly due to an interaction between alcohol and muscle damage brought about by the simulation. Decreases in CMJ of similar magnitude have previously been observed after damaging eccentric exercise (Byrne and Eston 2002a). Although the completion of 80 minutes of rugby game simulation brought about post-exercise changes in CK, testosterone, cortisol and immune cell concentrations the consumption of alcohol had no effect on the magnitude of these changes.

Currently little is known about the effect post-exercise alcohol consumption has on performance as the majority of research in this field has investigated the effects of pre-exercise alcohol consumption on physical performance (American College of Sports Medicine 1982) or factors related to recovery without directly measuring performance (Burke et al. 2003; Heikkonen et al. 1996, 1998; Maughan and Shirreffs 2006). Similarly, to date a number of studies have investigated game related changes in blood borne markers of stress, inflammation and muscle damage, however to our knowledge the detrimental effect a game of rugby may have on actual physical performance has not been quantified. The lack of research in these areas makes it difficult to compare the changes in the performance measures made in our study with changes that may occur after an actual game or other forms of exercise. Several

studies have quantified changes in physical performance after other forms of football with conflicting results. Decrements in CMJ have been observed up to 24 h post-game (Dawson et al. 2005; McLellan et al. 2011) while others have failed to see any effect of a game on subsequent performance (Cormack et al. 2008). Comparisons of pre to post-game performance have typically focused on measures of lower body vertical power (Cormack et al. 2008; Dawson et al. 2005; McLellan et al. 2011) so that the effects of a game on other important physical components of the sport, such as endurance, horizontal power, agility, strength and sprinting ability, are currently unknown. The changes in CMJ observed in the present study suggest lower body power output is negatively affected by post-simulation alcohol consumption, however given this change is most likely trivial it is unlikely to greatly influence performance. Similarly, no change in any other performance measures were seen suggesting that performance in tasks that require repetitive maximal muscular action, such as sprinting and dynamic scrummaging, are not affected by post-exercise alcohol consumption.

Similar to an international game of rugby (Cunniffe et al. 2010), we observed an acute phase inflammatory response, characterized by elevations in leukocytes, neutrophils and monocytes, 30 minutes post-simulation. This response, in particular infiltration of neutrophils to the site of injury, is believed to be important in the repair of damaged skeletal muscle (Tidball 1995). Acute alcohol consumption has been shown to negatively affect the innate immune system, and thus impact the rate of recovery after injury or trauma, via alterations in neutrophil, leukocyte and monocyte function (Szabo 1999). However, the dose used in our study did not affect the innate immune response to rugby specific exercise. This finding suggests that the consumption of 1 g alcohol per kg BW post-game is unlikely to detrimentally affect the inflammatory processes typically associated with strenuous exercise and the repair of damaged skeletal muscle.

Game related changes in cortisol and testosterone have been noted after rugby games at various levels of competition. Typically, elevations in cortisol and decreases in testosterone are seen immediately post-game before returning to baseline levels the following day.(Cunniffe et al. 2010; Elloumi et al. 2003). As occurs after other forms of exercise, this shift towards a catabolic state ensures increased energy demands are

met during the recovery period (Koziris et al. 2000). In the present study a significant elevation in cortisol was seen 12 h post simulation however no change in testosterone was observed at any time. Although acute post-exercise alcohol use has been shown to negatively affect testosterone, but not cortisol (Heikkonen et al. 1996), no effect of alcohol was seen in our study. This may be due to the dose used in the present study as alcohol's influence on testosterone has been observed at a dose of 1.5 g alcohol per kg BW and is known to occur in a dose dependent manner (Heikkonen et al. 1996).

Plasma CK activity in the present study was elevated immediately post-simulation however the magnitude of change was considerably smaller than that observed after international (Cunniffe et al. 2010) and provincial (Gill et al. 2006; Smart et al. 2008) level rugby games. Our values are similar to those observed in collegiate level rugby players in the days following a game (Suzuki et al. 2004). However, due to the large variability in CK between and within subjects, in particular two potential outliers, and the fact that CK may not truly represent the magnitude of damage, (Brancaccio et al. 2007; Warren et al. 1999b) we cannot reliably infer that the simulation used in this study brought about a similar level of muscle damage to that experienced during a collegiate level game of rugby. While our intention was to closely mimic the contact that is encountered in an actual game, the intensity of the contact during the simulation was significantly less than that experienced under game conditions. This lack of realistic game specific contact is perhaps the major limitation of using simulations to investigate the effects of various interventions on performance or recovery during and/or after contact team sport. Irrespective of the magnitude of change in CK, in this study we have shown that an elevation in CK does not necessarily mean that sprint performance, agility, and horizontal power output is negatively impacted. This brings into question the value of quantifying CK after games and/or training in order to better manage workloads and training intensities as suggested by Suzuki et al. (2004) and Smart et al. (2008).

To our knowledge, this is the first time a modified version of the BURST protocol (Roberts et al. 2010b) has been used to elicit fatigue and muscle damage. Although reliability in both 15 m sprint and high intensity drill times between trials was poor, unlike previous studies using the BURST protocol (Roberts et al. 2010b), in this study these were not outcome measures of performance, but rather were used to simulate

movements and velocities encountered during rugby game play. Performance of these tasks was faster in the Trial Two, probably due to a learning effect, however this improved performance did not influence La^- and RPE, while HR was, in fact, lower at the ends of the second and third quarters in Trial Two compared to the same time points in Trial One. We therefore believe that the small differences in performance of these tasks between trials is unlikely to have had any bearing on the outcome of either trial given the majority of each trial was matched for distances, velocities and time. However, these between trial differences highlight the difficulty of precisely replicating intensity between trials/treatments when aspects of the simulation are self-paced.

As illustrated by elevations in CK, cortisol and immune cell concentrations the protocol used in this study appears to provide similar stresses to those encountered in a game of rugby. However, the limitations encountered here, namely realistic levels of game specific contact and good reliability in sprint performance between trials, must be addressed when considering this protocol as a means of bringing about rugby specific fatigue and muscle damage to investigate various interventions on within game performance and/or post-game recovery.

An attempt to control diet was made with the use of self-reported food diaries during the first trial and encouragement to repeat the diet during the second trial. However, this method of dietary control is inferior to providing food, and indeed has been suggested to be unreliable when investigating repeated performance measures (Jeacocke and Burke 2010). Whether this significantly influenced the results is unknown, however we cannot discount that small differences in the macronutrient make up of each subjects diets between trials may have influenced the outcome of the performance measures made 24 and 48 h post simulation.

Finally, it is important to mention that the dose used in the present study is considerably lower than the volume of alcohol regularly consumed by rugby players and other team sports subjects after competition and/or training. The measurement of BAC 30 minutes after the consumption of the alcoholic beverage resulted in a BAC of 0.067 g/dL which, depending on the country, is either above or close to the legal limit for driving.

8.5.1 Conclusion

The results of the present study indicate that the consumption of 1 g of alcohol per kg body weight after a simulated rugby game has a detrimental, yet trivial, effect on CMJ; however, other performance measures that require repeated maximal efforts are not affected. Similarly, although changes in cortisol, CK and immune cell concentrations occurred in response to the simulation no effect of alcohol was observed suggesting the dose of alcohol used here may not be detrimental to recovery after a game of rugby. Whether a more realistic, higher dose of alcohol combined with an actual rugby game affects these measures is worth investigating in the future. The simulation related changes reported here suggest that the use of the BURST protocol is a valid means of bringing about rugby specific stress; however, consideration should be given to the limitations discussed when using this protocol in the future.

CHAPTER 9

General Discussion and Conclusion

9.1 GENERAL DISCUSSION

Previous research into the effect of alcohol on physical performance has typically concentrated on the impact pre-exercise alcohol use has on measures of endurance, speed and strength (*Section 2.1.7*). However, consumption of large volumes of alcohol after competition and/or training is common place in many sports, in particular team sports such as the various football codes (*Section 2.1.5.2*). Given this pattern of alcohol use the previous research into pre-exercise alcohol use on performance has little direct application to sport.

Sports that have the strongest relationship with heavy alcohol use also typically involve a high level of eccentric muscular contraction through a combination of deceleration, jumping, changing direction and body. As with other exercise related alcohol research the earliest study to investigate alcohol's interaction with eccentric exercise induced muscle damage focused on pre-exercise alcohol consumption and found no effect of alcohol on any of the criterion measures made, compared to a non-alcoholic beverage (Clarkson & Reichsman 1990). More recently Barnes et al. (2010) found that when consumed soon after damaging exercise the consumption of 1 g alcohol per kg BW magnifies the typical loss of force associated with such exercise. This study however did not provide an insight into the mechanisms behind this observation. Therefore, the aim of **Chapter 5** was to confirm the earlier finding of Barnes et al. (2010) and further investigate whether the observed effects of alcohol on force production are localised and the result of an interaction between damaged skeletal muscle and alcohol or whether alcohol's effects on force production are systemic in nature. Akin to the initial findings of Barnes et al. (2010), alcohol ingestion post-exercise exacerbated the force loss observed in the days following the damaging exercise. Given alcohol had no effect on the non-damaged leg while a greater loss in force was seen with alcohol in the damaged leg, compared to a control

beverage, these results appear to be due to an interaction between alcohol and processes involved in force generation and/or repair of damaged skeletal muscle.

Therefore, the evidence that the consumption of 1 g alcohol per kg BW after eccentric exercise impacts force loss in the days after exercise is strong. However from a practical stand point, to better educate those in the wider sporting community as to a 'safe' level of alcohol use, and given complete abstinence may not be a realistic recommendation, an understanding of how different doses may impact recovery and force loss is required. To begin this process a dose of 0.5 g alcohol per kg BW was used in **Chapter 6**. Unlike the higher dose, no difference in force loss was observed between this dose and a non-alcoholic beverage indicating that this lower dose does not affect force loss and may therefore be a safe dose if alcohol is to be consumed after damaging exercise. Whether a threshold, at which point alcohol begins to impact force loss, exists or whether alcohol's effect on force loss occurs in a linear fashion above 0.5 g alcohol per kg BW is currently unknown. It is worth noting that the magnitude of force loss after eccentric exercise in **Chapter 6** was considerably less than that observed in **Chapters 5 and 7**. Therefore it is possible that the level of damage was insufficient to observe the interaction reported in **Chapters 5 and 7.** This theory supports the conclusions made in **Chapter 8**.

Although the results of **Chapter 5** indicate that alcohol at a moderate dose interacts with exercise induced muscle damage the precise nature and location of this interaction was unclear. **Chapter 7** attempted to determine whether these effects are the result of alcohol's actions at the muscle level, CNS level, or a combination of both. Additionally the effects of alcohol, in the absence of muscle damage, on measures of neuromuscular performance were made to provide a better understanding of the interaction between alcohol and damaged skeletal muscle. Changes in measures of MVIC, neuromuscular efficiency and LFF were observed with both alcohol and the control beverage in the days following eccentric exercise. However, the greatest change in MVIC was observed when alcohol had been consumed after exercise. Similarly, a significant change in VA was observed when alcohol and muscle damage were combined. No change in VA was evident with the control condition. These results suggest that a decrease in neural output, combined with changes occurring at the muscle level, contribute to the greater force loss observed when alcohol is

consumed after damaging exercise. Conversely, as previously reported (Poulsen et al. 2007), the consumption of alcohol alone had no effect on measures of neuromuscular performance in the days following the drinking session.

While alcohol consumed after strenuous eccentric exercise in an isolated muscle group is detrimental to muscular force generation, for these findings to have a wider application it is important to understand whether subsequent performance and recovery is affected when whole body, sport specific exercise is used to stress and damage the body, as occurs during contact team sport. Therefore **Chapter 8** examined the interaction between the effects of a simulated game of rugby and post-game (moderate) alcohol consumption on measures of sports specific performance and immunoendocrine function in the days following the simulation. Of all the measures made only CMJ was affected by the consumption of alcohol in the hours following the game simulation. These data thus indicate that lower body power generation is affected by this combination however the lack of change in other measures suggests the dose of alcohol used does not impact immune and endocrine function or measures of performance that involve repeated muscular efforts. It may also be postulated that alcohol's ergolytic effects occur only when high levels of muscle damage are present prior to alcohol consumption and not when the level of exercise induced muscle damaged is relatively low, as occurred in **Chapter 8**.

Given the similar, yet separate, physiological responses and outcomes of eccentric EIMD (*Section 2.2.3*) and contusion injury (*Section 2.2.4.3.1*) it is possible that the findings of **Chapters 5 and 7**, along with those of Barnes et al. (2010), may be applied to impact related soft tissue injuries, as well as severe EIMD. Although the precise mechanism responsible for the loss of force development under each condition is not precisely known similarities have been suggested (Elmer et al. 2011). Additionally, while it is likely that the inflammatory response to each form of injury/damage is not exactly the same (Nosaka and Clarkson 1996) broad similarities do exist, such as elevations in neutrophils, monocytes and various interleukins (Charge and Rudnicki 2004; Tidball 2005; Smith 2008). Although the influence of alcohol on the innate immune response to severe EIMD, as occurred in **Chapters 5, 6 and 7**, has not been established it can be speculated that alcohol has a negative effect on this process. It is possible that, by changing the inflammatory response (*Section*

2.1.6.3), alcohol may impact the severity of an injury irrespective of whether it is consumed prior to or after an injurious event. Due to the lack of direct research in this area, there is significant potential for extending the line of work presented in this thesis, particularly when the clinical application and importance of such work is considered.

While abstinence from alcohol is widely recommended for individuals suffering from soft tissue injury very little direct evidence exists to support this directive (*Section 2.1.8.2*). In fact, the findings of Barnes et al. (2010) and **Chapters 5 and 7** provide the first direct evidence that this recommendation is warranted. It is therefore suggested that greater emphasis be placed on managing alcohol consumption during the hours after potentially damaging exercise and/or events resulting in musculoskeletal injury, rather than focusing on recovery modalities whose effectiveness is questionable (Gulick et al. 1996; Barnett 2006).

9.2 LIMITATIONS

While every attempt was made to ensure data collection in this series of studies was carried out as accurately as possible several limitations exist. Firstly, the major limitation was that subjects may have expected alcohol to negatively impact their recovery and subsequent performance in the days after exercise. As reference to the findings of Barnes et al. (2010) was included in all information sheets provided to potential subjects across the four studies it is possible that the knowledge that the dose of alcohol being used in **Chapters 5, 7 and 8** impacted subjects perceptions of recovery thus resulting in greater decrements in performance measures, as found in **Chapters 5 and 7**. To avoid such an issue pilot testing was performed in an attempt to mask the taste of alcohol between the two treatment beverages. However these attempts were unsuccessful as even the lowest dose used, 0.5 g per kg BW, was detectible. Therefore no additional attempt to conceal the alcohol was made. The only attempt to limit the subjects knowledge of which beverage was to be consumed was made during the first trial of each study. This ensured that the subject was unaware of the beverage they would be consuming prior to carrying out the first exercise bout. This, of course, was only able to be done for each subject's first trial as it soon became apparent to the subject which beverage they were consuming. Although the

inability to completely blind subjects to the beverage being consumed is a limitation, it is no different to the challenges faced in all intervention based research.

Secondly, a strict, but limited amount of dietary and behavioural control was placed on subjects during the pre and post–exercise periods. While subjects were instructed to abstain from exercise, the use of practices that may impact the severity of EIMD and consumption of alcohol it is unclear how successful this instruction was in controlling subject's behaviour. Similarly, in the absence of funding to supply subjects with a controlled diet throughout the experimental period, subjects were required to replicate their diet between trials using a food diary. This practice has recently been questioned by researchers who suggest that is method is in adequate when replicating diets between trials (Jeacocke and Burke 2010). To limit the variation in diet between trials subjects were provided with the same pre–exercise meal and additionally a controlled small, post–exercise meal during each trial.

In **Chapter 7** iEMG was recorded using a PowerLab data acquisition system (ADInstruments, Australia). The application of a 50 Hz notch filter prior to signal processing, in an attempt to limit background noise, may have resulted in the loss of EMG signal power therefore limiting the scope to investigate change in this measurement (Konrad 2005). Although the settings were the same between trails for all subjects it is unclear whether this had a significant effect on results. Unfortunately once data was collected it could not be altered to remove the 50 Hz notch filter.

Finally, a potential limitation for all studies is the lack of performance measurement immediately post-exercise. Given the treatment was applied after the exercise bout, an understanding of the effect the exercise had on performance measures would provide an indication of whether subjects were in the same physiological state prior to the application of the treatment. If, for example, a subjects performance was worse immediately post-exercise in one trial, compared to the other, this difference may influence changes at later time points. However, as the legs exercised in **Chapters 5, 6 and 7** were different between trials/treatments, it would be expected that the fatigue occurring in the dominant and non-dominant leg would differ to some extent even though the same protocol was used for each leg. This issue may be eliminated through the counter balanced and randomised allocation of dominant and non-dominant leg

between both treatment and trial, as used in this series of studies. Whether measuring performance immediately post-exercise in these studies would have provided an accurate measure of damage is questionable given the large contribution metabolic fatigue has on decreases in performance after such strenuous exercise (Proske and Morgan 2001). That said, measurement of performance decrements after the rugby game simulation (**Chapter 8**) would have provided a valuable measure of reliability between trials.

9.3 CONCLUSIONS

The aim of this thesis was to conduct a series of studies to further investigate the effects of post-exercise alcohol consumption on physical performance in the days following a bout of strenuous, damaging exercise. The findings of this thesis showed that

(1) When consumed after a strenuous bout of eccentric exercise, the consumption of 1 g alcohol per kg BW appears to interact with damaged skeletal muscle and/or processes involved in recovery to magnify the loss of force typically associated with strenuous eccentric exercise.

(2) A dose of 0.5 g alcohol per BW does not impact muscular performance in the days following a bout of eccentric exercise.

(3) A decrease in neural output appears to contribute to alcohol's interaction with exercise induced muscle damage to magnify damage related force loss.

(4) In the absence of exercise induced muscle damage, the consumption of 1 g alcohol per kg BW does not impact measures of neuromuscular function in the days following the drinking episode.

(5) The consumption of 1 g alcohol per kg BW after a simulated rugby game has a detrimental effect on CMJ; however, other sports specific performance measures that require repeated maximal efforts are not affected. This dose does not impact the immunoendocrine response to a simulated rugby match.

(6) Post-exercise alcohol consumption appears to effect performance in the days following exercise when high levels of muscle damage are already present; no effect is seen after a strenuous bout of exercise which brings about low levels of muscle damage.

(7) Greater attention to the management of alcohol after potentially damaging exercise and/or during the injured state is recommended.

9.4 FUTURE DIRECTIONS

Following on from the findings presented in this thesis a number of areas for future research have been identified.

Although **Chapter Five** investigated a low dose of alcohol other doses are worth investigating so that eventually guidelines may be developed for the use of alcohol after exercise. Very little is known about the effects post exercise alcohol consumption on recovery from other forms of exercise, most information comes from indirect measures as discussed in the literature review. This lack of information provides large scope to investigate any number of doses and scenarios that may be particularly relevant to the sport and exercise field.

While an attempt to investigate the effects of alcohol on immune function after potentially damaging exercise was made in **Chapter 8** it is unclear whether the EIMD experienced as a result of the match simulation was as severe as may occur in a real game or even if it was similar to that induced by the specific eccentric only exercise utilised in **Chapters 5, 6 and 7**. Therefore, two areas of research are required to address this limitation. Firstly, it is unclear how the dose used in **Chapters 5, 7 and 8** impacts immune function when consumed after very strenuous and damaging eccentric exercise, as performed in **Chapters 5, 6 and 7**. Given alcohol's negative effect on many of the proteins involved in the innate immune response to EIMD it is possible that this dose has an effect on what is likely to be a substantial inflammatory response.

Secondly, the use of a rugby match simulation has a number of limitations and therefore may not be truly representative of the stresses that occur during a real rugby match. Investigation of how the dose used in **Chapter 8** or higher doses affect recovery from actual rugby matches would add valuable information to the limited evidence provided by the findings of **Chapter 8**.

Finally, it is currently unknown how regular, acute and/or chronic alcohol consumption impacts adaptation to exercise. Although a training study of this magnitude would require considerable resourcing to ensure compliance and control of a number of factors, including diet, activity and alcohol use, throughout the training period the results may provide valuable information relevant to the wider sport and exercise community.

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APPENDIX A

Ethics Application Documentation

CHAPTERS 5 AND 7

Massey University

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18 February 2009

Mr Matthew Barnes
IFNHH
PN452

Dear Matthew

Re: **HEC: Southern A Application – 08/64**
Contribution of the central nervous system to alcohols effect on recovery of force after eccentric exercise-induced muscle damage

Thank you for your letter dated 16 February 2009.

On behalf of the Massey University Human Ethics Committee: Southern A, I am pleased to advise you that the ethics of your application are now approved. Approval is for three years. If this project has not been completed within three years from the date of this letter, reapproval must be requested.

If the nature, content, location, procedures or personnel of your approved application change, please advise the Secretary of the Committee.

Yours sincerely

J. O'Neill

Professor John O'Neill, Chair
Massey University Human Ethics Committee: Southern A

cc Dr Stephen Stannard
IFNHH
PN452

Prof Richard Archer, HoI
IFNHH
PN452

CHAPTER 6

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FILE

22 July 2009

Mr Matthew Barnes
IFNHH
PN452

Dear Matthew

Re: HEC: Southern A Application – 09/43
The effects of low dose alcohol consumption on muscle function after strenuous eccentric exercise

Thank you for your letter dated 20 July 2009.

On behalf of the Massey University Human Ethics Committee: Southern A, I am pleased to advise you that the ethics of your application are now approved. Approval is for three years. If this project has not been completed within three years from the date of this letter, reapproval must be requested.

If the nature, content, location, procedures or personnel of your approved application change, please advise the Secretary of the Committee.

Yours sincerely

Professor Julie Boddy, Chair
Massey University Human Ethics Committee: Southern A

cc Dr Stephen Stannard
IFNHH
PN452

Dr Toby Mündel
IFNHH
PN452

Prof Richard Archer, HoI
IFNHH
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26 November 2009

FILE

Dr Stephen Stannard
IFNHH
PN452

Dear Stephen

Re: HEC: Southern A Application – 08/65
Effect of acute alcohol intake on recovery after rugby specific exercise

Thank you for your letter dated 25 November 2009 outlining the change you wish to make to the above application.

The change, to replace approved exercise with 80 minutes of intermittent short sprints, jogs and walks, has been approved and noted.

If the nature, content, location, procedures or personnel of your approved application change, please advise the Secretary of the Committee. If over time, more than one request to change the application is received, the Chair may request a new application.

Yours sincerely

JmBoddy

Professor Julie Boddy, Chair
Massey University Human Ethics Committee: Southern A

cc Prof Richard Archer, HoI
IFNHH
PN452

APPENDIX B

Statements of Contribution

CHAPTER 5

DRC 16

MASSEY UNIVERSITY
GRADUATE RESEARCH SCHOOL

STATEMENT OF CONTRIBUTION
TO DOCTORAL THESIS CONTAINING PUBLICATIONS

(To appear at the end of each thesis chapter/section/appendix submitted as an article/paper or collected as an appendix at the end of the thesis)

We, the candidate and the candidate's Principal Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of Candidate: Matthew J Barnes

Name/Title of Principal Supervisor: A/Prof Stephen Stannard

Name of Published Paper: Post-exercise alcohol ingestion exacerbates eccentric-exercise induced losses in performance

In which Chapter is the Published Work: Chapter Five

What percentage of the Published Work was contributed by the candidate: 85%

M Barnes
Candidate's Signature

06/07/11
Date

S Stannard
Principal Supervisor's signature

06/07/11
Date

CHAPTER 6

DRC 16

MASSEY UNIVERSITY
GRADUATE RESEARCH SCHOOL

STATEMENT OF CONTRIBUTION
TO DOCTORAL THESIS CONTAINING PUBLICATIONS

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We, the candidate and the candidate's Principal Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of Candidate: Matthew J Barnes

Name/Title of Principal Supervisor: A/Prof Stephen Stannard

Name of Published Paper: A low dose of alcohol does not impact skeletal muscle performance after exercise-induced muscle damage

In which Chapter is the Published Work: Chapter Six

What percentage of the Published Work was contributed by the candidate: 85%

Candidate's Signature — 06/07/11 Date

Principal Supervisor's signature — 06/07/11 Date

GRS Version 2– 1 December 2010

CHAPTER 7

DRC 16

MASSEY UNIVERSITY
GRADUATE RESEARCH SCHOOL

STATEMENT OF CONTRIBUTION TO DOCTORAL THESIS CONTAINING PUBLICATIONS

(To appear at the end of each thesis chapter/section/appendix submitted as an article/paper or collected as an appendix at the end of the thesis)

We, the candidate and the candidate's Principal Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of Candidate: Matthew John Barnes

Name/Title of Principal Supervisor: A/Prof Stephen Stannard

Name of Published Research Output and full reference:

The effect of acute alcohol consumption and eccentric muscle damage on neuromuscular function

In which Chapter is the Published Work: Chapter Seven

Please indicate either:

- **The percentage of the Published Work that was contributed by the candidate:** 90%
  and / or
- **Describe the contribution that the candidate has made to the Published Work:**

Candidate's Signature — 16/12/11 Date

Principal Supervisor's signature — 16/12/11 Date

GRS Version 3– 16 September 2011

CHAPTER 8

MASSEY UNIVERSITY
GRADUATE RESEARCH SCHOOL

STATEMENT OF CONTRIBUTION TO DOCTORAL THESIS CONTAINING PUBLICATIONS

(To appear at the end of each thesis chapter/section/appendix submitted as an article/paper or collected as an appendix at the end of the thesis)

We, the candidate and the candidate's Principal Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of Candidate: Matthew John Barnes

Name/Title of Principal Supervisor: A/Prof Stephen Stannard

Name of Published Research Output and full reference:

The effects of acute alcohol consumption on recovery from a simulated rugby match

In which Chapter is the Published Work: Chapter Eight

Please indicate either:

- **The percentage of the Published Work that was contributed by the candidate:** 90%
  and / or
- **Describe the contribution that the candidate has made to the Published Work:**

MBarnes
Candidate's Signature

16/12/11
Date

S Stannard
Principal Supervisor's signature

16/12/11
Date

GRS Version 3– 16 September 2011

APPENDIX C

Publications

CHAPTER 5

Eur J Appl Physiol (2010) 108:1009–1014
DOI 10.1007/s00421-009-1311-3

ORIGINAL ARTICLE

Post-exercise alcohol ingestion exacerbates eccentric-exercise induced losses in performance

Matthew J. Barnes · Toby Mündel · Stephen R. Stannard

Accepted: 23 November 2009 / Published online: 11 December 2009

Abstract The effect of acute alcohol intake on muscular performance in both the exercising and non-exercising legs in the days following strenuous eccentric exercise was investigated to ascertain whether an interaction between post-exercise alcohol use and muscle damage causes an increase in damage-related weakness. Ten healthy males performed 300 maximal eccentric contractions of the quadriceps muscles of one leg on an isokinetic dynamometer. They then consumed either a beverage containing 1 g of ethanol per kg bodyweight ethanol (as vodka and orange juice; ALC) or a non-alcoholic beverage (OJ). At least 2 weeks later they performed an equivalent bout of eccentric exercise on the contralateral leg after which they consumed the other beverage. Measurement of peak and average peak isokinetic (concentric and eccentric) and isometric torque produced by the quadriceps of both exercising and non-exercising legs was made before and 36 and 60 h post-exercise. Greatest decreases in exercising leg performance were observed at 36 h with losses of 28.7, 31.9 and 25.9% occurring for OJ average peak isometric, concentric, and eccentric torques, respectively. However, average peak torque loss was significantly greater in ALC with the same performance measures decreasing by 40.9, 42.8 and 44.8% (all $p < 0.05$). Performance of the non-exercising leg did not change significantly under either treatment. Therefore, consumption of moderate amounts of alcohol after damaging exercise magnifies the loss of force associated with strenuous eccentric exercise. This weakness appears to be due to an interaction between muscle damage and alcohol rather than the systemic effects of acute alcohol consumption.

Keywords Ethanol · Muscle strength · Soft tissue injuries

Communicated by William Kraemer.

M. J. Barnes (✉) · T. Mündel · S. R. Stannard
Institute of Food, Nutrition, and Human Health,
Massey University, Private Bag 11-222,
Palmerston North, New Zealand
e-mail: M.Barnes@massey.ac.nz

Introduction

The mechanisms and consequences of exercise-induced muscle damage (EIMD) have received considerable scientific attention over the past 20 years. Strenuous eccentric muscle action is now known to cause micro-structural damage resulting in delayed onset muscle soreness (DOMS), inflammation and more importantly, impaired muscle function which typically lasts for a number of days, depending on the severity of the damage (Cleak and Eston, 1992; Proske and Morgan, 2001). Over this same period EIMD has been employed as a model of soft tissue injury where a number of modalities aimed at improving the rate of recovery (e.g. cold water immersion therapy (Eston and Peters 1999), non-steroidal anti-inflammatory medication (Gulick et al. 1996), massage (Jönhagen et al. 2004) and compression therapy (Kraemer et al. 2001)) have been tested. This research has provided mixed and often inconclusive results. Surprisingly, compared to these and other recovery modalities, less attention has been afforded to post-exercise behaviours that may simultaneously impair the recovery process after EIMD.

One such behaviour is post-exercise alcohol use. While the consumption of large amounts of alcohol by sportspeople, often after competition or training, is common place (Nelson and Wechsler 2001; Snow and Munro 2006; O'Brien et al. 2007) little is known about how this behaviour effects recovery and subsequent performance in

CHAPTER 6

Eur J Appl Physiol (2011) 111:725–729
DOI 10.1007/s00421-010-1655-8

SHORT COMMUNICATION

A low dose of alcohol does not impact skeletal muscle performance after exercise-induced muscle damage

Matthew J. Barnes · Toby Mündel · Stephen R. Stannard

Accepted: 13 September 2010 / Published online: 28 September 2010

Abstract Moderate, acute alcohol consumption after eccentric exercise has been shown to magnify the muscular weakness that is typically associated with exercise-induced muscle damage (EIMD). As it is not known whether this effect is dose-dependent, the aim of this study was to investigate the effect of a low dose of alcohol on EIMD-related losses in muscular performance. Ten healthy males performed 300 maximal eccentric contractions of the quadriceps muscles of one leg on an isokinetic dynamometer. They then consumed either a beverage containing 0.5 g of alcohol per kg bodyweight (as vodka and orange juice) or an isocaloric, isovolumetric non-alcoholic beverage. At least 2 weeks later, they performed an equivalent bout of eccentric exercise on the contralateral leg after which they consumed the other beverage. Measurement of peak and average peak isokinetic (concentric and eccentric) and isometric torque produced by the quadriceps was made before and 36 and 60 h post-exercise. Significant decreases in all measures of muscular performance were observed over time under both conditions (all $P < 0.05$); however, no difference between treatments was evident at any of the measured time points (all $P > 0.05$). Therefore, consumption of a low dose of alcohol after damaging exercise appears to have no effect on the loss of force associated with strenuous eccentric exercise.

Keywords Ethanol · Muscle strength · Soft tissue injuries

Communicated by William Kraemer.

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Introduction

The consumption of large amounts of alcohol by sportspeople, particularly those involved in team sports, after competition and/or training is common place. This population is further reported as consuming such hazardous levels of alcohol more frequently than the general population (Maughan 2006; O'Brien et al. 2007; Quarrie et al. 1996; Snow and Munro 2006). As with the non-sporting population, these drinking patterns are associated with increased risk-taking behaviours which often result in alcohol-related injury (Cherpitel 1993; Nelson and Wechsler 2001). For sportspeople, however, acute alcohol use, particularly after strenuous exercise, provides another risk through the influence alcohol can have on processes involved in recovery and adaptation.

The physical nature of many team sports engenders a high incidence of exercise-induced muscle damage (EIMD) and soft tissue injury due to repeated rapid deceleration, changes in direction, and collisions with other players and/or the ground (LaStayo et al. 2003). Although both EIMD and alcohol consumption are common in many sports, very little is known about how the two interact to affect recovery (of performance) in the days after the injurious event.

When alcohol's ingestion is quickly followed by trauma/injury, alcohol is known to increase the chance of infection and medical complication resulting in delayed recovery through alterations in the trauma-induced inflammatory response (Szabo and Mandrekar 2009). However, this combination of pre-exercise alcohol use and soft tissue injury (as EIMD), investigated by Clarkson and Reichsman (1990), found that a moderate dose of alcohol (0.8 g per kg bodyweight) had no effect on recovery of performance in the days after a damaging bout of exercise when compared to a non-alcoholic beverage.

CHAPTER 7

The effects of acute alcohol consumption and eccentric muscle damage on neuromuscular function

Matthew. J. Barnes, Toby Mündel, and Stephen R. Stannard

Abstract: Voluntary and electrically stimulated muscular performance was examined to identify the effects of acute alcohol consumption on neuromuscular function in the presence and absence of exercise-induced muscle damage (EIMD). After initial neuromuscular performance measures were made, 12 subjects completed a bout of eccentric exercise (EX) using the quadriceps muscles of 1 leg while the remaining 11 subjects did not exercise (NX). Subjects then consumed either an alcoholic beverage containing 1 g·kg^{-1} body weight (ALC) or a nonalcoholic beverage (OJ). On another occasion the contralateral leg of both groups was tested and those in the EX group performed an equivalent bout of eccentric exercise after which the other beverage was consumed. Measurements of neuromuscular function were made pre-exercise and 36 and 60 h post-beverage consumption. Creatine kinase (CK) was measured pre-exercise and at 12, 36, and 60 h. Significantly greater ($p < 0.01$) decrements in maximal voluntary isometric contraction were observed with EX ALC at 36 and 60 h compared with EX OJ, and no change was seen in the NX group. Significant decreases in voluntary activation were observed at 36 h ($p = 0.003$) and 60 h ($p = 0.01$) with EX ALC only. Elevations in CK were observed at all posteccentric exercise time points (all $p < 0.05$) under both EX OJ and ALC. No change in electromyography or low-frequency fatigue was observed under either treatment in either group. These results suggest that decreased neural drive appears to contribute to alcohol's effect on the magnitude of EIMD-related decrements in voluntary force generation.

Key words: ethanol, neuromuscular function, exercise induced muscle damage, percutaneous electrical stimulation.

Résumé : Chez des sujets présentant ou non une lésion musculaire suscitée par l'exercice physique (EIMD), on analyse l'effet de la consommation d'alcool sur la fonction neuromusculaire telle que révélée par la réponse des muscles à la stimulation électrique ou à la contraction volontaire. À la suite des mesures initiales de performance neuromusculaire, on demande à 12 sujets (EX) de participer à une séance d'exercice pliométrique des muscles du quadriceps fémoris d'une seule cuisse et on invite les 11 autres sujets (NX) à ne pas faire d'exercice. Puis les sujets prennent une boisson contenant de l'alcool (ALC) à raison de 1 g·kg^{-1} de masse corporelle ou une boisson non alcoolisée (OJ). À un autre moment, on évalue chez les deux groupes le membre controlatéral, puis les sujets du groupe EX participent à une autre séance d'exercice pliométrique, équivalente à la première, avant de prendre l'autre boisson. On évalue la fonction neuromusculaire avant la séance d'exercice, puis 36 h et 60 h après avoir pris la boisson. On évalue aussi l'activité de la créatine kinase (CK) avant la séance d'exercice, puis 12 h, 36 h et 60 h plus tard. Chez le groupe EX avec ALC, on observe à la 36^e h et à la 60^e h une plus grande baisse significative ($p < 0,01$) de la tension isométrique maximale volontaire, comparativement au groupe EX avec OJ; on n'observe aucune modification chez le groupe NX. Chez le groupe EX avec ALC seulement, on observe une diminution significative de l'activation volontaire à la 36^e h ($p = 0,003$) et à la 60^e h ($p = 0,01$). Dans les deux groupes EX (OJ et ALC), on observe une augmentation de la CK à toutes les périodes de mesure consécutives à la séance d'exercice. Dans les deux groupes et dans les deux conditions, on n'observe aucune modification de l'activité myoélectrique (EMG) ni de fatigue à faible fréquence. D'après ces observations, il semble que la diminution de la commande nerveuse s'inscrit dans l'effet de l'alcool sur l'ampleur de la baisse de la force volontaire associée à l'EIMD.

Mots-clés : éthanol, fonction neuromusculaire, lésion musculaire suscitée par l'exercice physique, stimulation électrique percutanée.

[Traduit par la Rédaction]

Introduction

Injury or trauma to skeletal muscle that occurs during alcohol intoxication often results in an increased risk of infection and medical complication, which may prolong recovery (Szabo and Mandrekar 2009). Although this alcohol-related injury or trauma is common, little is known about the opposite situation, where alcohol is consumed after a damaging event, as may occur after intense eccentrically based exercise.

Recent research has shown that the acute consumption of 1 g of alcohol per kg body weight in the hours following a

Received 29 June 2011. Accepted 16 October 2011. Published at www.nrcresearchpress.com/apnm on xx December 2011.

M.J. Barnes, T. Mündel, and S.R. Stannard. School of Sport and Exercise, Massey University, Palmerston North, New Zealand.

Corresponding author: Matthew. J. Barnes (e-mail: M.Barnes@massey.ac.nz).

Appl. Physiol. Nutr. Metab. 37: 63–71 (2012) doi:10.1139/H11-137 Published by NRC Research Press

CHAPTER 8

Journal of Sports Sciences, 2011; 1–10, i*First* article

Routledge
Taylor & Francis Group

The effects of acute alcohol consumption on recovery from a simulated rugby match

MATTHEW J. BARNES, TOBY MUNDEL, & STEPHEN R. STANNARD

School of Sport and Exercise, Massey University, Palmerston North, New Zealand

(Accepted 31 October 2011)

Abstract
In this study, we investigated the effects of acute post-exercise alcohol consumption on measures of physical performance, creatine kinase, and immunoendocrine function in the 48 h following a rugby game simulation. Ten male senior rugby union players completed a rugby game simulation after which they consumed either 1 g of alcohol per kilogram of body mass or a non-alcoholic control beverage. Agility, 15 m sprint, countermovement jump, and srummaging performance were assessed pre-simulation and 24 and 48 h post-simulation. White blood cell count, testosterone, cortisol, and creatine kinase were measured before the simulation and 30 min, 12, 24, 36, and 48 h after the simulation. One week after the first trial, participants completed the second simulation after which the other beverage was consumed. The acute consumption of alcohol after a rugby game simulation negatively affected countermovement jump performance in the days following the simulation ($P=0.028$). No differences between treatments were observed for the other criterion measures made in this study. In conclusion, after 80 min of a simulated rugby game, the consumption of 1 g of alcohol per kg body mass negatively impacts lower body vertical power output. However, performance of tasks requiring repeated maximal muscular effort is not affected.

Keywords: *Contact sport, alcohol, recovery, rugby*

Introduction

Hazardous, acute alcohol consumption occurs regularly in many sports, in particular male-dominated team sports, as part of post-game celebration, team culture or in association with sponsorship commitments (Maughan, 2006). Participants in these sports have reported regular bingeing on alcohol at higher rates than the general, non-sporting population (Nelson & Wechsler, 2001; O'Brien, Blackie, & Hunter, 2005). Although this behaviour is prevalent and often seen as an acceptable part of a team culture, surprisingly little research has been carried out into the effects of such behaviour on performance recovery after exercise or competition. Recently, Barnes and colleagues (Barnes, Mündel, & Stannard, 2010a, 2010b) investigated the effects of an acute alcohol binge episode on recovery of skeletal muscle performance after a bout of strenuous eccentric exercise. These studies revealed that alcohol magnifies the level of force loss typically associated with exercise-induced muscle damage through an interaction between alcohol and the damaged skeletal muscle. Others investigating alcohol's impact on recovery have focused on rehydration (Shirreffs & Maughan, 1997), glycogen repletion (Burke et al., 2003), and hormonal changes (Heikkonen et al., 1996) rather than looking at its effect on subsequent performance directly. Yet the true index of recovery is repeat performance.

Historically, a strong relationship has existed between heavy alcohol consumption and the sport of rugby union (Collins & Vamplew, 2002), with many players reporting higher levels of alcohol use than participants in other sports or the general population (Black, Lawson, & Fleishman, 1999; O'Brien & Lyons, 2000; Quarrie et al., 1996). Like a number of other football codes, rugby union is characterized by repeated short-duration, high-intensity activity, including short sprints and physical contact, interspersed with periods of jogging, walking, and standing (Deutsch, Kearney, & Rehrer, 2007). Although such activities, particularly the contact components, are likely to have a detrimental effect on performance in the days following the game, very little research has confirmed/investigated

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http://dx.doi.org/10.1080/02640414.2011.637949

APPENDIX D

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APPENDIX E

24 Hour Diet Recall

Name _____ **Date** _____

Meal/Snack	Food item	Amount
Breakfast		
During Morning		
Lunch		
During Afternoon		
Evening Meal		
Evening Snack		