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'The effects of customary post-game behaviour on rugby specific performance measures following competitive match play'

A report submitted towards the attainment of MPhil

(Sport and Exercise Science)

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Abstract

Background:

Deviant off-field behaviour is now generally accepted as being part of a 'normal' sporting culture. The majority of research into such behaviour has focussed primarily on the immediate impact of alcohol consumption on sporting performance, with such research highlighting the ergolytic nature of alcohol on performance. The fundamental issue with such research however is that the deviant behaviour associated with sports such as rugby union typically occurs the night previous to or following competition, accordingly the effect of such customary behaviour on recovery from competition or on subsequent performance would be more specific to what actually occurs. Initial work has found that moderate alcohol consumption adversely affects the recovery of exercise induced microstructural damage post eccentric exercise, as well a negatively affecting subsequent lower body power output post rugby simulation. Despite conclusions suggesting that alcohol negatively influences both recovery and subsequent performance, such conclusions may not be truly representative of what typically occurs due to the behaviour investigated being far different to what occurs naturally. The question of how customary off-field rugby behaviour affects both recovery and subsequent performance therfore remains unanswered.

Purpose:

The purpose of this study was to investigate whether post-game behaviour, that is customary to rugby union, is detrimental to the subsequent performance of players in the days following competitive match play.

Methods:

Using a naturalistic means of investigation, thirty senior grade club rugby players were allocated to either the standardized post game behaviour (SPGB) or investigated post game behaviour (IPGB) conditions following a competitive rugby match. Players involved in the IPGB condition were left to undergo customary post rugby game behaviour whilst those in the SPGB had their behaviour controlled according to recommended guidelines. Performance measures, behaviour recall and indicators of both muscle damage and hydration status were tested at both twelve and thirty six hours following match play

Results:

Following competitive match play participants in the IPGB condition reported significant (p<0.01) alcohol consumption with a corresponding decrease in sleep when compared with the SPGB. Irrespective of such behaviour, performance measures were not significantly affected. Finally no significant difference was seen between conditions in either hydration status or CK.

Conclusions:

The results of the present naturalistic study indicate that following a competitive match, customary rugby behaviour consisting of significant alcohol consumption and a reduction in sleep failed to significantly affect subsequent rugby specific performance measures in the days following the match.

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1. Introduction

Rugby union is a team sport characterised by its intermittent and physically demanding nature. Due to this there is a considerable physiological effect associated with match play. The intensity and energy expenditure of a typical game of rugby is significantly greater than in other team sports such as soccer ^{25; 4} and field hockey ¹². Moreover, during the course of a rugby game, significant positional variability can be observed ³⁴.

Following a competitive game of rugby there is a significant amount of associated muscle damage, illustrated by a significant increase in creatine kinase activity (CK) ^{135; 47; 128}. It is has been shown that muscle damage can be induced through either eccentric muscular contractions (particularly when unaccustomed) ^{20; 23} or blunt force trauma ^{156; 135; 128}; the erratic and violent nature of rugby suggests that muscle damage results from both.

Following team sport training or competition, optimal nutrition and hydration is recommended to maximise recovery. Such practice would typically rule out alcohol consumption and associated behaviour due to the speculated negative effect on recovery and subsequent performance. Neverthless, it is well documented that both sportsmen and university students demonstrate higher levels of drinking than the general population ^{67; 101; 72; 53; 119} and this often occurs in the post-competition period.

Despite this relationship between rugby and post-match alcohol consumption there is a surprising lack of research into the effects of such potentially detrimental behaviour. The emphasis instead appears to be focused on methods to maximise player's recovery ^{47; 59}. Breaking this trend, recent work has begun investigating the effects of moderate alcohol consumption on recovery^{6; 7; 8}. These primary laboratory based investigations have demonstrated that the loss in both static and dynamic strength following extreme eccentrically induced muscle damage is magnified with alcohol ⁶. Additionally, lower body power output following simulated rugby match play is similarly affected ⁸. Such laboratory based research allows for full control and balancing of conditions; however they may not be indicative of how post-game moderate alcohol consumption effects recovery. Following a game players often consume large quantities of alcohol at a self-administered rate and quantity. Furthermore, such alcohol consumption has been shown to influence diet ^{149; 56} and sleeping patterns ^{120; 143}. Such a combined effect which is characteristic of post rugby match behaviour is therefore extremely difficult to replicate in a laboratory based trial. It is this combined effect that resulted in the suggestion that the use of a naturalistic means of study that exploits the

predictability with which such behaviour occurs¹³⁰. Accordingly the aim of this study was to use a naturalistic experiment design to investigate whether such combined behaviour affects rugby specific performance measures following competitive match play.

2. Literature Review

Rugby Union

Rugby union is an intermittent team sport involving intensive efforts (5-15 seconds) of power and strength separated by relatively short periods of recovery (\approx 40s)⁹¹. Within each team there are a variety of positions each with both unique and common roles and responsibilities ⁵². Positions are typically grouped into 'forwards' and 'backs' according to their general roles and responsibilities. The forwards role is primarily to compete and win the ball which often involves close proximity with the opposition including high impact passages of play such as the scrum and maul. The backs on the other hand are involved with higher levels of intense running e.g. cover defence or accelerating into attacking positions, interspersed with periods of passive recovery ³⁷. To cater for such difference in roles rugby forwards are generally taller, heavier and have a higher body fat percentage than backs

In August 1995 rugby union became professional 84. The switch to professionalism resulted in a game that was played at an increased pace with a significant increase in the amount of physical contact 38; ¹⁰⁹. This change in how the game is played coincides with a corresponding change in the physique of rugby players with players generally increasing in both height and weight and therefore becoming more mesomorphic. Despite a gradually increasing size of the general population, the switch to professionalism has resulted in rugby players increasing in size by a rate of up to 5 times faster than that of the general population ⁹⁸. How this increased size of players has contributed to success is clearly illustrated by the final rankings of the 1999 World Cup. Rugby team success was correlated with the body size of that team ⁹⁸. It is speculated that the financial incentive is being increasingly utilized to lure players of the desired anthropometry into rugby for this very reason. This incentive, as well as an improved knowledge of optimal nutrition and quality of training, is likely to contribute to a larger and more skilled base of players. As a result it can be theorized that countries competing at the world cup who have more resources to allocate to the selection and development of players would potentially have a bigger and more skilful team, which in turn can be related to success. Due to the different positional demands, rugby players are commonly selected according to a wide range of ideal positional specific anthropometric and physiological attributes ¹⁰⁸. This positional selection according to both anthropometric and physiological attributes is far different to other team sports such as soccer where physique is relatively similar to that of the general population 93 and ideal performance attributes are relatively common between members of the squad regardless of position ¹¹².

Effects of a rugby game

When looking at a rugby game there are many factors that could influence the physiological effects displayed. Time-motion analysis was used to compare physical and game specific skill based match demands between 17 elite and 22 semi-elite rugby league players¹²⁷. It was found that for the entire match both physical and game specific demands were similar irrespective of standard of play. It was however found that the elite players performed a greater level of activity during the first half than during the second half. This reduction in level of activity in the second half is possibly through the onset of fatigue following such a strenuous first half ¹²⁷. It can therefore be seen that when looking at the effects of an entire game, as we are in this study, the effects are the same irrespective of level at which it is played.

Previous research has investigated the metabolic stress of competitive rugby union match play. The data show that peak blood lactate concentration during match play was between 6.2-8.8mmol.L⁻¹ ³⁴ and 8.56-9.8mmol.L⁻¹ ⁷⁹, with an estimated energy expenditure of 6.9MJ and 8.2MJ for backs and forwards respectively ²⁸ .From these it is apparent that rugby union is played at a high intensity with energy production being achieved through both aerobic and anaerobic energy systems ^{34; 25}. The total estimated energy expenditure (6.9 -8.2MJ) is slightly higher than that of other intermittent team sports such as soccer ²⁵and field hockey ¹². The high VO₂ (80-85% of VO₂ max) is also higher than the corresponding value for both soccer (\approx 70%)⁴ and field hockey (\approx 78%)¹². The higher exercise intensity and estimated energy expenditure of rugby could be as a result of greater utilization of whole body musculature rather than mainly the primary locomotor muscles as well as energy expended during physical contact (e.g. tackles, scrums etc.) in rugby when compared to other team sports.

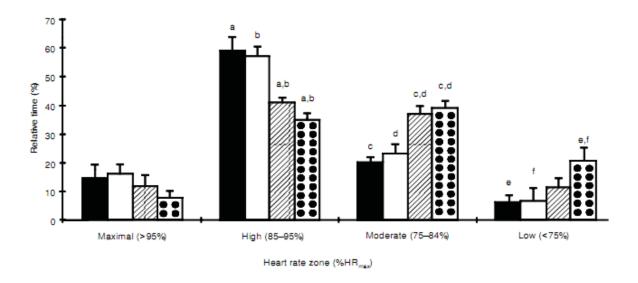


Figure 1 Mean percent times spent in four heart rate zones for four positional groups (props and locks, \blacksquare ; back row forwards, \square ; inside backs, \mathscr{M} ; outside backs! \square . Significant difference (p<0.05) between groups is indicated with lettering. 34 .

Within a game of rugby, it was found that the time spent in particular heart rate zones were significantly related to the players position as illustrated in Figure 1³⁴. It can therefore be proposed that within a rugby game energy expenditure and intensity would vary due to the difference in positional requirements of each particular playing position. Time motion analysis of professional match play highlights this contrast with forwards spending 13% of total match time performing high intensity work in contrast to just 4.5% by outside backs ³³. Accordingly, time spent performing high intensity work was found to be the primary reason why the forwards perform greater total work and have higher heart rate and blood lactate levels than their back counterparts ^{37; 115}. Future studies into rugby of either code should therefore be aware of such positional variability between forwards and backs, as this may significantly influence any findings.

Muscle Damage

Another characteristic that differs between positions is the amount of muscle microtrauma experienced following a game of rugby. This muscle "damage" has been shown to relate to both volume and intensity of exercise, a factor which is different between positions.

As a result of exercise-induced damage to the muscle cell structure, creatine kinase (CK) (the enzyme responsible for the phosphorylation of ADP from creatine phosphate within the muscle cell) is released into the interstitial fluid. This CK is then transported into the general blood circulation via the lymphatic system and so can be measured in a blood test. The general activity of CK in a blood sample indicates the amount of CK in the blood, and in turn, the extent of damage. This increase in blood serum creatine kinase activity therefore provides a reliable method for assessing whether muscle damage has occurred ⁴¹, but, due to individual variabilty to the same exercise, cannot quantify the extent of muscle damage. It is worth noting that due to the method by which the CK is transported there is a lag between the damaging exercise and the associated rise in blood CK levels

Muscle damage is well documented to follow unaccustomed exercise, particularly that comprising a large eccentric component ^{20; 23}. For example, the effect of 24 maximal eccentric contractions of the elbow flexors on ten male subjects was investigated finding CK levels reaching as high as 25244U.L-¹ despite large inter-subject variability⁹⁴. Muscle damage induced by eccentric muscle contractions is thought to be due to the magnitude of strain during the lengthening of an activated muscle rather than simply as a function of peak force ⁷⁰. Such muscle damage results in a reduction in tension and increases in the optimum length for tension generation force within the muscle. Studies comparing the behaviour of intact muscle with that of isolated electrically stimulated muscle have found that the majority of damage takes place within the muscle itself as a result of non-functional sarcomeres, a shift in the length-tension relationship as well as changes in excitation-contraction coupling ⁸⁵. There are two signs of muscular damage immediately following eccentric muscular contraction: the presence of disrupted sarcomeres within the myofibril as well as damage to the excitation-contraction coupling system ¹⁰⁶.

Eccentric muscle contractions occur as part of the general gait cycle ⁷⁰ - running posesses a 50% eccentric demand of the muscles in terms of external work - so it would be expected that markers of damage would be elevated following strenuous running. Indeed, it was found that following a (running) marathon there was a significant increase in mean creatine kinase activity levels (men, woman) at 24hrs (3322U.L-1,946U.L⁻¹) and 48hrs (1787U.L⁻¹, 508U.L⁻¹) respectively ¹¹⁷ (a slight difference between genders is thought to be due to the fact that men have greater muscle mass and

thus correspondingly greater amounts of muscle damage). Given the gait requirements of the game, it seems obvious that rugby players may be exposed to eccentric muscle contractions during even the simplest requirements of the sport. Further, Rugby union is a sport where players are regularly required to change direction ⁵¹ to avoid opposition or exploit opportunities whilst continually varying the pace throughout the duration of the game. This change of direction/pace is achieved through a combination of both concentric and eccentric muscle contractions, and likely the latter will contribute further to any muscle damage experienced. Accordingly, following a competitive rugby match blood CK activity was found to increase significantly ^{47; 128}. The same research noted that there was a significantly higher increase in blood CK in the forwards when compared to the backs, suggesting a positional difference in the amount of muscle damage.

There is increasing scientific evidence suggesting that muscle damage, as indicated by an increase in [CK] may result from physical impact or blunt force trauma 135; 128. An early study 156 compared the hormonal parameters of shadow boxing (simple muscular work) and real boxing (traumatic activity) observing a significant increase in both CK and myoglobin following real boxing indicating the importance of trauma to the liberation of these indices of muscular activity. Later, this effect was studied over the course of a competitive rugby match concluding that serious muscle damage induced during the course of a competitive rugby match is highly dependent on the number of tackles the player is exposed to ¹³⁵. This study neglected physical contact during the game as a result of the scrum, rucking or mauling although highlighting the correlation between blunt force trauma and muscle damage. The scrum is an integral part of the battle for physical and psychological supremacy over the opposition ¹¹⁰, with the average premier scrum exerting an approximate mean force of 10960 N¹¹⁰. It must be considered that the packs (the 8 forwards) form separately approximately 1 meter apart, before fiercely coming together during engagement ¹⁴⁷. Upon engagement approximately 10960 N from each pack is exerted through the shoulders of the front row (the two props and hooker). There is therefore a significant impact that is ignored in the existing literature. Impacts similar to this as well as a higher amount of tackles ³⁷ performed by the forwards would contribute to the significant difference between positions. This difference between positions was further highlighted with the findings that Urease-UV for blood urea nitrogen (BUN) were significantly decreased in the back group but increased in the forward group, indicating that protein catabolism was far greater in the forwards than in the backs⁷⁷.

There is an adaptation in the muscle which occurs following a bout of eccentric exercise such that further such exercise has a reduced impact on muscle function, soreness and damage. It was therefore proposed that the muscle attempts to protect itself against further muscle damage from

subsequent eccentric contractions through increasing the sarcomere number therefore shifting the optimum length for active tension¹⁰⁶. Elite/semi-elite players undergo muscle adaptation as a result of previous trainings and match play. Through such a protective mechanism they may be less susceptible to future eccentrically induced muscle damage. As the season was to progress it would therefore be thought that the eccentric exercise induced component of muscle damage would decrease. Muscle damage resulting from match play ⁴⁷ would therefore vary according to sport specific factors such as training status and position.

Immuno-endocrine markers

It is known that intense exercise can temporarily impair immune function. Following a rugby game there was a significant increase in cortisol (\approx 40%) with a corresponding decrease in testosterone levels (\approx 43%) thus resulting in a significant decrease in testosterone:cortisol ratio (T/C) ratio shown in Figure 2²⁷.

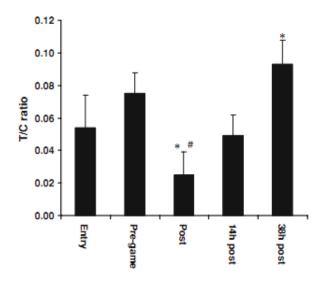


Figure 2 Change in serum testosterone:cortisol (T/C) ratio across time points, # = p < 0.05 from pregame, # = p < 0.05 from entry # = 0.05 fr

There was a significant increase in T/C ratio at 38 hrs. post-game representative of a possible rebound anabolic stimulus. Whether or not this rebound in T/C ratio would be influenced by additional exercise during this period of recovery is unknown. Following the game there was also an

increase in both blood neutrophils and blood monocytes indicating an exercise-induced acute response phase. There was however a drop in the number of NK cells which along with a corresponding drop in lymphocytes may suggest a reduced host protection. This has important follow-on effects indicating a time period of approximately thirty eight hours to minimize susceptibility of players to illness whilst providing sufficient time for repair of muscle damage. It is important to note that this study²⁷ did not differentiate the difference in response between positional groups which could have resulted in different effects on immuno-endocrine response.

The game of rugby was shown, using profile of mood states (POMS) testing, to increase mental fatigue ⁷⁷. Mental fatigue can be caused through prolonged periods of cognitive performance ⁷⁴ as well as physiological stress. Mentally the players regardless of position would fatigue as a result of cognitive performance however physically they may differ according to position ⁷⁷. Accordingly, position tailored health management following the game should be considered.

Rugby Culture

Development of rugby specific sub-culture

In the early 1800's British public schools were in a 'pitiful state' ⁹⁰. Outside of the class room the school boys were left to their own devices with tyranny, immorality and uncivilised behaviour common place ⁹⁰. Most public schools of the time such as Rugby, Winchester and Eton began adapting popular village football into their respective institutions ¹³⁷. Thomas Arnold became headmaster of Rugby school in 1828 using the school curriculum as a means by which to instil his ideal of 'Christian manliness' on the students. Arnold addressed the behaviour outside of the classroom by instilling power equivalent to his own on the sixth form in exchange for responsibility ³. It was a general growing concern for social control that resulted in sports such as the Eton wall game and rugby's predecessor 'big side football' being formalized into sports such as soccer and rugby. The genesis of rugby therefore was originally as a means of social control ¹⁵³. Accordingly it is commonly held that Arnold and his concept of muscular Christianity were responsible for the moral reform of public schools as well as influencing the formalization of mob games into less violent and socially accepted 'sports'.

Before 1965, rugby had on field expectations that were concerned with courage and athletic prowess, whilst upholding gentleman, amateur and sportsman like values ³⁵. As the sport evolved away from just a means of social control, the on field ideals remained. Off field however rugby developed a 'deviant sub culture' with a tendency towards the violation of social taboos ¹⁵³. Such

subculture was characterized by drunkenness, public nudity and the vilification of women ³⁵. Such behaviour may be as a result of rugby union being played by primarily the social elite who had far greater access to both alcohol and time in which to endulge. There was an extreme contrast between on field and off field norms and values associated with rugby before 1965.

Post 1965, British society became imbued with meritocratic ideas, with the new generation of players viewing success as the ultimate goal of participation. This change in motivation of players coupled with Britain using sport as a means of attaining national pride in a post-empire period, resulted in the gradual progression from amateurism to professionalism ³⁵. This progression to professionalism was unlike soccer. Rugby embraced amateurism as a form of class barrier to participation i.e. rugby existed as the winter sport for the 'ruling class'. It was this barrier that influenced rugby's unwillingness to turn professional. Due to this lack of democratization, rugby players were able to develop a unique set of rituals and behaviours in the absence of the working class and women. This class continuity and conservatism of the participants, resulted in a sport with a persistent subculture despite social changes in the larger community ³⁵.

Despite such a long period of extreme conservation in the way of change, rugby was eventually rationalized turning professional in 1995. Such rationalization resulted in rugby union moving away from being the winter sport of the ruling class to a sport played by all, at a time when all had access to both alcohol and leisure time. Such a shift on field did not have a corresponding change off field; the 'deviant sub culture' survived along with the consumption of large quantities of alcohol and rowdiness. As such beer drinking is evident throughout rugby (junior level right through to the international level) and remains as an integral component of rugby's off field culture.

As well as a means of providing social cohesion such traditional off field behaviour could persist to survive as a means of resistance to conformity in an ever increasing conforming world. As such off field behaviour as associated with rugby may be representative of a previous era. In an era where there is such a great emphasis on result rather than participation, there is a surprising lack of research on the effects of such behaviour on recovery from game play. Any negative effects that could be associated with such culture should be investigated to determine whether such out dated behaviour is influencing the modern day sport. The history of rugby is unique to the sport involving the growth of a game originally played locally by particular schools to a team sport of international significance. How both on field and off field cultures survived or transformed during such a transition are unknown, as such whether there are any difference between national values/practices associated with rugby is also unknown.

Rugby sub-culture in NZ

Rugby union is globally recognised as "New Zealand's national sport" ¹⁰⁸. Rugby union in New Zealand originated when C.J. Munro (educated in England) returned to New Zealand in 1870 and recommended his local association football team (Nelson Football Club) to try rugby under his tuition ¹³⁷. The sport proved to be attractive to all players involved resulting in the adoption of rugby union as their sport, with the first game between Nelson College and town being played in May 1870 ¹⁸. Rugby's initial success in New Zealand could be attributed to it being a game which provided New Zealanders with a means of recreation allowing respite from the hardships of colonial life.

"The life of the colonial in New Zealand was one of hardship, with recreational outlets being extremely limited. This ruggedness, associated with general living activities such as occupations, was evident in the Rugby games played. Violent physical contact between players was almost an acceptable part of Rugby during the 1880's."

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Such a rugged nature of rugby would have appealed to most New Zealanders of the time due to it conforming to their relatively rugged existence. Unlike in England, rugby in New Zealand was always played with a huge emphasis on result ¹⁸. The game of rugby in New Zealand was played by any one displaying gentlemanly qualities irrespective of class. This attitude by New Zealanders towards gentlemanly values rather than the class to which one belonged is clearly illustrated by Rev. F. Marshall below:

"The word "gentleman" does not connote membership of any social class.... And I have known varsity players who were not gentleman and miners who were. The "Spirit" it would seem contains some of the properties which keep the flame burning in the British way and purpose."

(Rev. F. Marshall cited in Stuart (1977))

In England such embourgeoisement between classes did not occur until after 1965. This involvement of any individual displaying gentlemanly characteristics irrespective of class could have been one reason why the early All Blacks ('the originals') had such great success on the nation's first tour outside of Australasia.

From the evaluation of how rugby emerged in both Noth America and Canada it was concluded that:

"Old country values tend to become 'frozen' at the time of migration such that, within a very few years, the ethnic group no longer reflects the changing values of the country of birth." 35

It could therefore be assumed that off field behaviour associated with English rugby in 1870 such as binge drinking, rowdiness and nudity could have frozen and been adopted by New Zealand, thus providing further escape from colonial hardships while becoming an integral component of post rugby behaviour in New Zealand. Traditional meanings associated with rugby such as this off field behaviour were gradually lost in England in the 1950/60's with the incorporation into the dominant sport ideology. This loss may not be of the same extent in New Zealand with modern day rugby possibly being tied to a more complex set of meaning.

Alcohol

Alcohol use and abuse in sport

Alcohol is the most commonly used recreational drug in New Zealand with approximately 85% of the adult population having consumed at least one drink in the last year ². Moderate alcohol consumption is shown to have a positive effect against coronary artery disease ^{113; 9; 100} as well as many psychological benefits such as mood enhancement, stress reduction and sociability ¹⁰⁰. Hazardous drinking on the other hand can be defined as drinking which confers the risk of either dysfunction or harmful consequences ³⁹ and is associated with a range of negative health and psychological consequences. Hazardous drinking can take place as either involving 60 or more standard drinks in the preceeding month (chronic drinking) or the consumption of five or more standard beverages on one occasion (binge drinking) ³⁶. Binge drinking is high in New Zealand with 60% of the population having drunk until the feeling of drunkenness at least once in the previous year. Alarmingly, 10% of the population reported such 'binging' behaviour at least once per week ². What is interesting is that this 'binge drinking' behaviour is significantly more prevalent in both university students and sports people than it is in the general population ^{146; 67; 72}.

University Students

Previous research has found that hazardous drinking is common place ⁴⁶ and continually increasing ⁸⁹ among university students and the younger population. It was found that in New Zealand universities 60% of males and 58.2% of females typically exceeded the national drinking guidelines⁶⁷. Such a problem was further highlighted by the alcohol use disorders identification test (AUDIT)

results. The AUDIT provides a means to identify persons with hazardous and harmful patterns of alcohol consumption. A total score of 8 or more can be used to classify potentially harmfull drinkers ²². The mean AUDIT scores of 10.9±7.9 and for 7.6±5.9 for male and female students respectively, clearly illustrate harmful alcohol use. Such a relationship does not appear to be unique to New Zealand with studies from both the USA⁶⁶ and the UK ⁵³ yielding similar results. University sport science students in France were found to drink alcohol less frequently than their peers, however reported higher levels of intoxication ⁷². Such a relationship may exist as a result of the students abstaining from alcohol whilst training and studying and then binging following a sport event. The period following the sport event would essentially provide an opportunity for like-minded individuals to indulge in large amounts of alcohol consumption.

Sports People

It has been shown that the involvement in sport is associated with higher alcohol use ^{101; 72}. This hazardous behaviour would be in contrast to the well-known health benefits associated with the increased levels of physical activity ¹⁰⁴ associated with the involvement in sport. The acceptance of such behaviour as part of 'normal' sporting culture should be a cause for concern ¹¹. It would appear that the prevalence within sport is highly influenced by the type and level of sport being played ^{11; 101; 97} Team sports have been found to be significantly more involved with such hazardous drinking than individual sports ⁷². Both Rugby league and Rugby union are both associated with the consumption of large quantities of alcohol as well as hazardous drinking behaviour ¹⁰⁷. It was discovered that a curvi-linear relationship existed between the level of rugby and excessive/hazardous alcohol consumption, illustrated by table 1⁹⁷.

Table 1 Mean(±SD) AUDIT subscale and total scores across all levels of participation⁹⁷

					1	Elite-		
		ıb/social		rovincial		rnational		
	(1)	(N = 912) $(N = 185)$ $(N = 117)$.2) (N = 185)		_		
AUDIT	М	SD	M	SD	M	SD	F-value	P-value
Hazardous alcohol use	5.72	(2.87)	6.22	(2.97)	5.14	(2.82)	2.552	0.078
Dependence symptoms	1.14	(1.64) ^{a,b}	1.43	(1.78) ^a	0.91	(1.22) ^b	3.178	0.041
Harmful alcohol use	2.45	(2.95) ^a	3.50	(3.44) ^b	2.00	(2.34) ^a	8.410	0.0005
AUDIT total	9.32	(6.36) ^a	11.15	(7.00) ^b	8.05	(5.16) ^a	6.479	0.002

^{*}significance within row indicated by subscript letters

Table 1 illustrates that members of the elite-provincial level of participation have significantly higher dependence symptoms, harmful alcohol use and total AUDIT score, whilst only approaching significantly greater hazardous alcohol use. A potentially harmful drinker can be classified as one who scores eight or more on the AUDIT²². Accordingly 53% of club/social, 68% of elite-provincial and 50% of elite-international could be classified as potentially harmful drinkers ⁹⁷. The elite-provincial participants reported the primary reasons behind such behaviour were as a means to deal with sport associated stress. Participants within this group often work/study fulltime on top of the (higher than club/social) level of sport related demands. Through such an increase in time demands the participants would be subject to far less leisure time to relieve such stress, which may result in alcohol consumption being relied upon as a means of relief. Players at the level above (eliteinternational) illustrate a far lower prevalence of harmful drinking. This could be a result of the sport being played at such a level that it takes the place of a job. These players reported that the potential performance decrements and fear of sanctions from team mates, coach or management were the primary reasons behind such a decrease in behaviour 97. This curvi-linear relationship is interesting as players within the 'worst' drinking behaviour category are essentially the elite-international players of the future. Any negative impact of behaviour, on either performance or the ability to improve as a player may result in a decrease in not only the performance of that particular individual but also of the elite level teams. It is extremely interesting that of the 1214 people questioned, only 4% reported abstinence from alcohol ⁹⁷. The 4% reported could relate to the 15% of the population who abstain from alcohol consumption. It would therefore be apparent that any impact of alcohol consumption on rugby play/training may be of huge significance in an age where the result of professional sport is of particular importance . The fact that such a small minority abstain from alcohol suggest greater underlying connections between rugby (both league and union) and alcohol consumption. It would appear that rugby culture as discussed still influences rugby associated behaviour.

Sportsmen often exhibit a weekly drinking pattern where the majority of alcohol consumption and binge drinking behaviour takes place on a Saturday ¹⁵⁴ often following competition. Alcohol consumption could therefore be used as a means for celebration or as a means to unwind after a week dedicated towards a sport. It would be interesting to see whether competition taking place on a night other than Saturday (as becoming ever more common place at the top level)whether such behaviour would remain on the Saturday or follow to the day of competition?

For sports such as rugby union where competitive match play places a significant physiological stress on the individual, the period immediately following competition would be the first and most

important period in which to optimise recovery. Such drinking patterns of the athlete population would therefore potentially take place when recovery is paramount and as such the negative consequences could potentially affect performance particularly during trainings on subsequent days. The effect of alcohol consumption on subsequent performance however has undergone surprisingly little investigation.

Physiological Effects of Alcohol

Harmful alcohol consumption is a common occurrence within both sports people and university students. The physiological effects of such behaviour is therefore of relative importance to determine the effects of such behaviour on performance and recovery.

Hydration

The ingestion of alcohol is commonly associated with a diuretic affect. It was originally found that for a male subject (n=1) diuresis following the consumption of an alcoholic beverage was proportional to the amount of alcohol present in the drink⁴⁰. Such a diuretic action of alcohol is thought to take place via the inhibition of vasopressin secretion ¹²⁵. It is due to this diuretic effect that the avoidance of alcohol is often recommended during rehydration ¹⁵.

The effect of such a diuretic effect on rehydration following exercise-induced dehydration was investigated ¹²⁵. In beverages containing up to 4% v/v alcohol it was found that the diuretic effect of alcohol was substantially blunted when the individuals were in a state of hypohydration. Consumption of beverages up to 2% alcohol would have no impact on rehydration providing that it is consumed in a volume greater than that being lost via sweat. Table 2 illustrates that the urine output time course was different for the 4% trials than for the trials at a lower alcohol percentage. Such a difference was suggested to be as a result of a delay in the rate of gastric emptying and subsequent water absorpion.

Table 2 Cumulative volume of urine produced up to and including each time point from the end of the rehydration period untill the end of the study ¹²⁵

Time After Rehydration, h	Trial 0%	rial 0% Trial 1% Trial 2%		Trial 4%	P-Value
0	33 (19-88)	27 (22-34)	38 (27-69)	34 (12-69)	0.501
1	439 (175-864)	425 (345-588)	520 (357-625)	323 (123-755)	0.643
2	721 (339-1,474)	834 (582-933)	941 (577-1,261)	747 (564-1,435)	0.843
4	875 (473-1,597)	1,062 (673-1,258)	1,131 (672-1,397)	1,348 (698-1,968)	0.32
6	942 (557-1,650)	1,108 (721-1,307)	1,184 (729-1,455)	1,457 (752-2,045)	0.307

^{*}Values are medians with range in parenthesis given in ml.

How alcohol greater than 4% influences the rate of gastric emptying and hence water absorption remains to be seen and could potentially be important in suggesting rehydration practice following dehydration.

It is known that beverage volume and sodium content interact to influence the rehydration process. As such a volume of beverage sufficiently greater than sweat loss is required to rehydrate. However, in the absence of a sufficient sodium content urine output is increased ¹²⁶. It is accepted that euhydration is achieved when the sodium intake is greater than that lost during exercise ¹²⁴. The above study ¹²⁵ used beverages containing only 4.4±0.2 mmol of sodium which was considerably lower than the estimated losses through both sweat and urine. How this relationship would be influenced if the optimal sodium intake was upheld is unknown but of importance.

It was found that the diuretic effect of a small dose of alcohol was blunted when the participants were in a state of hypo hydration⁵⁵ as illustrated by figure 3. It is thought that such a response is brought about in an attempt to restore fluid balance.

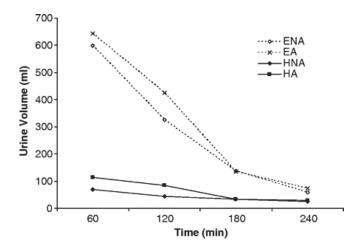


Figure 3 Hourly urine output for 4hrs post drink ingestion, ENA=Euhydrated without alcohol, EA= Euhydrated with alcohol, HNA = Hypohydrated without alcohol & HA = Hypo hydrated with alcohol. 55

When looking at the effects of alcohol on hydration it is important to consider the effects on hydration and performance over a longer period of time than just the immediate effects (e.g. 180 mins following ingestion) as this would be more applicable to how alcohol consumption may influence recovery from an event. As such a unique study¹³⁴ looking at diuresis over the twelve hours following alcohol ingestion (1.2g/kg BW) found that during the first three hours alcohol induced diuresis occured. The diuretic action of alcohol is thought to take place through the inhibition of vasopressin secretion ^{87; 114; 55}. Between 3 and 9 hours after alcohol ingestion a phase of antidiuresis was obtained. After 9 hours the subjects were loaded with water (20ml/kg BW). Following such loading antidiuresis was still maintained resulting in a significantly greater water retention in the alcohol condition than in the control condition, with water retentions of 44±6% and 12±4% respectively ¹³⁴. It was therefore hypothesised that alcohol induced diuresis is not brought about through the inhibition of vasopressin secretion alone. The antidiuresis and water retention on the contrary is thought to be as a result of stimulated vasopressin secretion ¹³⁴.

It can therefore be concluded that immediately following alcohol consumption there is an associated diuretic action. There are many factors that influence such an action with the main reason thought to be through the inhibition of vasopressin secretion. Following such diuretic action it is interesting that there is a period of antidiuresis resulting in significantly greater water retention following water loading.

It is clear therefore that the body increases urinary output to help the body deal with alcohol consumption. Further studies need to be undertaken to determine whether subsequent antidiuresis

occurs following the removal of alcohol from the system and accordingly whether the amount of alcohol consumed would influence the time taken for such antidiuretic action to take place. If such a relationship existed the antidiuretic action would occur following removal of alcohol from the system whether it be through metabolism or excretion and the occurrence of such action would be dependent on the amount of alcohol consumed. When considering the effects of alcohol consumption following a competitive rugby match, the hydration status upon completion of the game, the amount of alcohol consumed and how long after the consumption the testing is taking place must be considered. Whether or not this increased water retention would influence hydration status between an alcohol consuming group and a control group the day following a game needs to be established. This could potentially influence subsequent performance as it is well documented that dehydration results in an associated performance decrement.

Thermoregulation

Alcohol has been long regarded as a good peripheral vasodilator ⁴⁸. As a result of both increased vasodilation and increased diuresis following the ingestion of alcohol, blood pressure decreases resulting in a corresponding increase in heart rate as a result of the baroreflex ^{50; 60; 125; 151}. As well as a decrease in heart rate there is a corresponding decrease in regulated body temperature ^{26; 152} following the ingestion of alcohol.

During exposure to extreme temperature environments alcohol has been shown to disrupt thermoregulation thus facilitating subsequent hyper/hypothermia ⁸⁸. The effect of alcohol on thermoregulatory responses and thermal sensations following mild heat exposure was investigated ¹⁵¹. The effect of alcohol consumption (0.36g/kg body weight) is shown in figure 4.

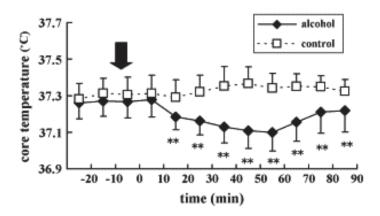


Figure 4 Mean core body temperature (±SEM) during both alcohol and control trials. Arrow illustrates time of ingestion, ** indicates significant (P<0.01) difference ¹⁵¹

It can be seen that even at mild heat exposure core body temperature is decreased following the ingestion of alcohol (even relatively low quantities). This heat loss associated with alcohol ingestion is thought to be through augmented sweating and vasodilation. Accordingly, even a low dose of alcohol modulates both autonomic heat loss and behavioural processes for thermoregulation resulting in body temperature being regulated at a significantly lower level ¹⁵¹.

Using the same protocol the thermoregulatory responses following exposure to the cold (18°C) were investigated¹⁵². The study found that core body temperature decreased regardless of treatment condition (figure 5).

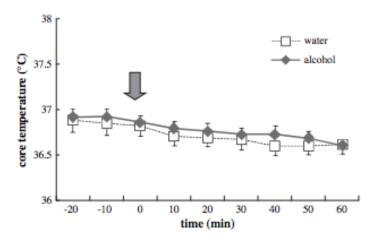


Figure 5 Mean core body temperature (±SEM) during both alcohol and control trials. Arrow illustrates time of ingestion ¹⁵²

It can be seen that there is no significant autonomic response to cold exposure. Body sensations of cold and thermal discomfort were successively stronger in the control treatment however this diminished after the alcohol treatment. Such perceptions may be a result of the effect of alcohol on the central nervous system rather than the blood vessels themselves. It would appear that alcohol influences thermoregulation differently dependant on the temperature. Alcohol has been shown to have a thermoregulatory effect during exercise in the cold ⁴⁹. The effect of alcohol on man's ability to adapt to exercise in a cold environmentwas investigated, finding the alcohol treatment resulted in a significantly greater heat loss than the control, thus indicating that alcohol impairs thermoregulation under such conditions⁴⁹. This impairment dissipates when the blood alcohol is metabolized with further heat loss similar between conditions.

Following alcohol ingestion and exercise in a warm environment (35°C and 45% relative humidity) There was no significant change in skin temperature, core temperature or sweat rate suggesting no fundamental alteration to thermoregulation as a result of alcohol. This observation of no effect was unique to exercise in a warm environment³².

It is clear that alcohol influences thermoregulation in man. The degree to which the individual is influenced depends on the temperature and whether exercise is being undertaken. Accordingly studies looking at the effect of alcohol on performance should be aware of the effects on thermoregulation so as not to confuse effects occurring as a result of this thermoregulatory effect rather than the ability to perform work.

Metabolism

Alcohol is a readily accessible source of energy (29kJ per gram) ¹³⁹. The majority (over 90%) of ingested ethanol is metabolised to acetaldehyde via both alcohol dehydrogenase (ADH) and the microsomal ethanol-oxidising system (MEOS) in the liver ⁴⁵as shown by figure 6.

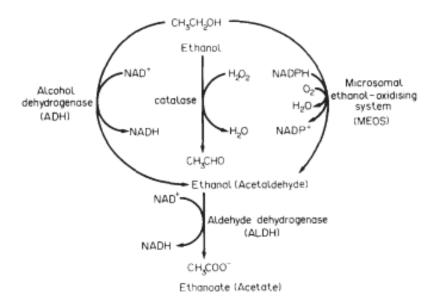


Figure 6 The basic pathways of ethanol metabolism ¹⁴

The remainder (<10%) of ingested ethanol is removed un-changed via breath, sweat or urine. The metabolism of ethanol via alcohol dehydrogenase (ADH) and consequently aldehyde dehydrogenase (ALDH) result with a disturbance in the NADH/NAD+ ratio through increased production of reduced NAD+¹⁴. This altering of NADH/NAD+ ratio promotes the conversion of pyruvate to lactate such a conversion can subsequently inhibit both gluconeogenesis and ketogenesis⁹². The existing literature therefore suggests that alcohol does not provide a substrate for glycogen reformation rather its effect is one of an indirect nature. At rest the Ingestion of alcohol has been shown to replace fat as the predominant fuel for oxidation whilst preventing the oxidation of carbohydrate following an intravenous glucose load, providing approximately two thirds of basal caloric requirements ¹²³.

It is accepted that the rate of muscle glycogen resynthesis is influenced by the magnitude of glycogen depletion especially during the early hours of recovery ¹⁵⁵. It is however not clear exactly how the consumption of alcohol following exercise influences muscle glycogen resynthesis and any corresponding practical implications. The following research illustrates what is known.

Post exercise studies on skeletal muscle in rats identified an ethanol-mediated impairment in post exercise glycogen repletion in both the liver and oxidative skeletal muscle. It was later found that this defect in both oxidative skeletal muscle and liver metabolism was mediated by the ethanol molecule itself ¹⁴⁸. Little effect was however seen in type II muscle fibres following exercise ¹⁰².

Acute alcohol has also been shown to dampen the stimulatory effect of exercise on glucose uptake by the exercising muscle possibly through the enhancement of the inhibitory effects of exercise on insulin secretion ⁶³. After prolonged exercise and acute alcohol treatment a significant proportion of released acetate is taken up by the muscles, there was found to be a corresponding reduction of equal magnitude in the uptake of glucose⁶².

The effect of alcohol on glycogen repletion in humans was investigated ¹⁶. Six well trained cyclists undertook an eight hour (2 meals) protocol whilst a further nine undertook a twenty four hour (4 meals) protocol. All subjects completed the following three trials in cross over order: Control diet (1.75g/kg CHO), alcohol diet (1.5g/kg alcohol) and an alcohol and carbohydrate diet (1.75g/kg CHO & 1.5 g/kg alcohol). The effects on glycogen repletion during recovery are shown in table 3.

Table 3 Mean (±SE) muscle glycogen concentrations after exercise depletion, recovery and different dietary interventions. ¹⁶

	Post exercise	Post recovery	Net storage (Post recovery minus post exercise)
8h Recovery study control	11.5 ± 2.8	56.0 ± 3.6	44.6 ± 6
Alcohol displacement	15.8 ± 6.9	40.2 ± 2.8	24.4 ± 7*
Alcohol + CHO	16.2 ± 5.3	52.5 ± 6.6	36.2 ± 8
24h Recovery study			
control	20.8 ± 5.5	102.5 ± 8.8	81.7 ± 5
Alcohol displacement	19.9 ± 4.2	88.2 ± 5.8	68.4 ± 5
Alcohol + CHO	21.8 ± 5.1	106.9 ± 10.0	85.1 ± 9

^{*} represents alcohol displacement diet resulting in significantly less muscle glycogen repletion than following either the control or alcohol-carbohydrate diet(p<0.05)

The significantly lower level of muscle glycogen repletion in the diet where dietary carbohydrate was replaced with alcohol (alcohol vs. control) illustrates the indirect effect on post exercise glycogen resynthesis if alcohol was used for isoenergetic displacement of carbohydrate in the post exercise recovery period. The non-significant increase in glycogen repletion following the alcohol & carbohydrate diet compared to the carbohydrate control illustrates that despite an increased supply of energy through alcohol there was no associated increase in glycogen repletion. From this it can be concluded that the consumption of alcohol in unison with the recommended carbohydrate intake does not provide a substrate for glycogen synthesis nor does it result in the sparing of carbohydrate for a non-oxidative fate.

Neurological effects

Alcohol has a variety of neurological effects. Alcohol is a known depressant through its ability to reduce CNS excitability and cerebral activity ^{121; 139}. Altered CNS activity as a result of alcohol has been shown to effect balance, reaction time, memory and accuracy of fine motor skills ¹³¹. Alcohol has also been shown to have a negative influence on quality of sleep ^{116; 139}. This influence can be a result of a reduction in the amount of sleep, as well as through the disruption of a normal sleeping pattern (decreasing REM sleep and increasing deep sleep) ^{116; 133; 105}. Considering the relationship between alcohol consumption and lack of sleep a negative effect on the quality of sleep could compound any associated negative effects. These neurological effects of alcohol could potentially have a negative effect on the recovery of athletes following the ingestion of alcohol ¹⁶.

The hangover

The 'hangover' is the term used to describe the collection of both unpleasant physical and mental symptoms that occur following acute alcohol administration. A summary of hangover symptoms currently exists within the literature¹⁰⁵. The symptoms and severity show great intra and inter individual variability ¹⁰⁵. Despite this variability in symptoms and severity the occurrence of the hangover is common place.

A study of 350 college undergraduates found that, 25.5% had experienced a hangover in the week previous to being surveyed⁸³. Despite numerous studies into what a hangover is and how it comes about there is still no conclusive answer. Accordingly, neither a unitary or multi-factorial hypothesis can be confirmed, as such it is often referred to as a phenomenon that is specific to the individual⁵⁷. Three classifications influencing the hangover has been proposed: characteristics of the beverage(s), characteristics of the drinking occasion and characteristics of the individual ⁵⁸.

Characteristics of the beverage

Alcoholic beverages often contain congeners which are substances often used to flavour and colour drinks. Despite a lack of research in the area, the relationship between hangover severity and congener content can be illustrated by figure 7¹⁴¹.

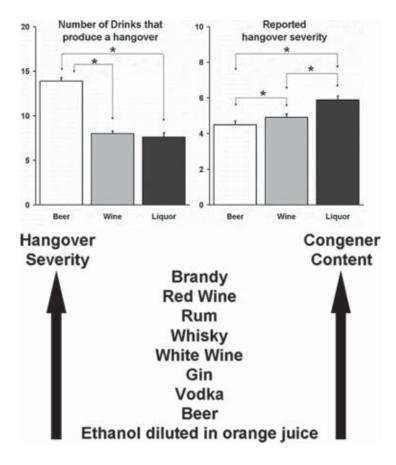


Figure 7 Number of drinks required to produce a hangover and associated hangover severity. (* representing a significant difference (p<0.05) ¹⁴¹.

It can be seen therefore that alcoholic beverages with higher congener content produce more severe hangovers. It was also shown that it takes fewer drinks of high-congener content to get a hangover¹⁴⁰. This effect on the intensity of hangover was shown however to have no effect on the next day performance, sleep or perceived impairment. It is commonly accepted that larger doses result in more severe symptoms, although the hangover is not solely dose related ¹⁵⁰.

Characteristics of the drinking occasion

Howland et al. ⁵⁷reported no literature investigating the effects of occasional characteristics on the hangover, however they proposed factors such as drinking alone or with others; food consumption and pre-drinking mood or fatigue status as being factors that should be considered and further investigated. It is known that the drinking of alcohol often takes place at the expense of sleep. As such some of the symptoms associated with the hangover may be attributed to a significant reduction in sleep duration and quality ⁶⁴. This interaction between sleep and alcohol consumption therefore needs to be considered when investigating following a 'normal' nights drinking⁷⁸.

Characteristics of the individual

One factor that can be agreed upon is that the peak blood alcohol concentration reached during the drinking session $^{69;\,133}$ has a significant influence on the subsequent hangover. It is also known that susceptibility to hangover can vary across individuals who consumed equivalent quantities of alcohol therefore there must be more contributing factors unique to the individual that contribute to both incidence and intensity 5 .

It is known that there is a gender difference in alcohol metabolism ¹³⁶. Potentially resulting in a difference in both hangover presence and severity between genders. It was found that woman experienced significantly higher hangover intensity than their male counterparts¹⁴². A study investigated gender differences in the sheer quantity of consumption found that following a normal nights drinking males reported an average consumption of 14.7 units compared with 10.5 of females⁷⁸. This clearly shows a gender difference in the sheer amount of alcohol consumed, whether this results in a higher peak breath alcohol content (BrAC) and subsequent difference in the presence or severity of a hangover is yet to be investigated. The fact that males generally have a greater body mass may result in the peak BrAC being similar between genders.

A survey found an association between the incidence of hangover and family history of drinking problems¹³³ reflecting biological differences in the response to alcohol. Studies have also found that some drinkers may be resistant to hangover (20-30% of the drinking population) ⁵⁷It is therefore obvious that there is large variation in how the individual responds to alcohol and further research needs to be done in this field to allow a full identification of compounding co-factors.

Cognitive effects of the hangover

There is currently little evidence determining whether any effects on cognitive performance are attributable to the hangover⁴². The hangover effect on next-morning memory function following a single episode of drinking however was investigated by Verster et al. ¹⁴². 48 participants (24 woman & 24 men) underwent a single blind study comprising of baseline testing followed by either a hangover condition or placebo condition the following morning. Immediate recall and recognition were unimpaired, delayed recall was significantly reduced as a result of alcohol consumption indicating memory retrieval processes are significantly impaired during the alcohol hangover. This would indicate that cognitive function is affected by hangover and further research should be done in establishing the effects of such detriments on behaviour.

Means of studying the hangover

From the literature it is clear that there are two forms of investigating the effects of the hangover: laboratory based and naturalistic trials. Laboratory based trials allow for the blinding of consumption, typically have found no effect on performance. Naturalistic trials however allow the drinker to dictate how much, what and the pace of the drinking. Such a method best represents what occurs naturally and does show an effect on performance, there are however no means by which alcohol consumption could be blinded ¹³⁰. The utilization of the naturalistic method of investigation was therefore recomended to take advantage of the predictability with which social drinking occur¹³⁰.

Effects of alcohol on sport performance

The majority of the research regarding the effects of alcohol on performance has focussed primarily on the immediate impact of alcohol consumption/intoxication. In 1982 the American College of Sports Medicine released the following conclusions regarding the effect of alcohol on sports performance ¹:

- The acute ingestion of alcohol has a deleterious effect on many psychomotor skills
- Alcohol consumption does not substantially influence physiological function crucial to physical performance [maximal oxygen uptake (VO₂ max), respiratory dynamics and cardiac function].
- Alcohol ingestion will not improve muscular work capacity and may decrease performance levels.
- Alcohol may impair temperature regulation during prolonged exercise in a cold environment.

This positional stand highlighted the potential negative affect of alcohol consumption on sport performance. Subsequent research substantiates ACSM's position. The effects of alcohol on metabolic responses to treadmill running in men was investigated⁶⁵. Beverages containing 25ml of ethanol in 125ml of grapefruit juice (test condition) or 150ml of grapefruit juice (placebo condition) were randomly assigned to 4 well trained endurance runners. The conditions were administered 10 minutes prior as well as after 30 minutes of running at between 80 and 85% of VO₂max. Three of the four participants failed to complete the run. Results indicated that alcohol resulted in a significantly higher heart rate, as well as illustrating that after alcohol intake at 30 minutes blood glucose dropped significantly by 24%. It was therefore theorized that alcohol negatively influences

endurance based exercise by eliciting a hypoglycaemic effect after 30 minutes of exercise. The effects of alcohol on shorter duration events has also been investigated ⁸¹, finding a detrimental but inconsistent relationship between alcohol dose and running time of the sprinting group (200m & 400m). Such an effect was not visible however for a 100m event ⁸¹, it was thaught that the negative effect of alcohol was as a result of a negative effect on complex motor skills involved during running. For middle distance running (800m & 1500m) a decrement was also observed. This was speculated to result from decreased myocardial contractivity as well as an inhibitory effect on gluconeogenesis, and was greater in magnitude the greater the distance ran.

Such endurance based activities are influenced by a variety of variables including cardiac function. The direct negative effect of alcohol consumption on endurance activity would suggest that one or more of these variables are influenced through the consumption of alcohol. The absence of effect on more anaerobic activity such as the 100m, would possibly suggest that the observed effect was associated with a variable more specific to aerobic exercise. It is difficult using the above exercise modes to test whether muscular performance is affected ¹⁰³. Accordingly the effects of acute alcohol intoxication on skeletal motor performancewas investigated ¹⁰³. The comparison of maximal isometric strength of the a) wrist and b) knee is illustrated by figure 8.

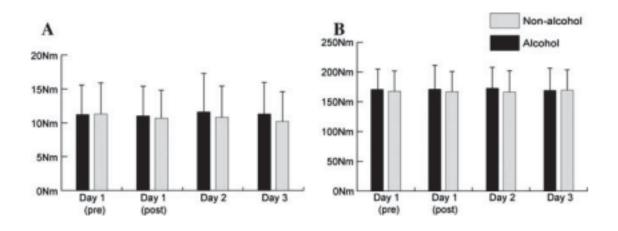


Figure 8 Mean (±SD) Maximal isokinetic muscle strength of a) wrist and b) knee ¹⁰³

Figure 8 shows that no significant difference between either the alcoholic condition or control condition was observed. This would therefore indicate that alcohol consumption has little to no effect on muscular strength. Results of the same condition on isometric endurance also indicated no

significant difference as a result of alcohol intoxication. Thus, it was therefore proposed that moderate alcohol intoxication has no significant effect on motor performance^{73; 103}. This would suggest the negative effect observed previously ^{81; 65} occurred through the effect of alcohol on variables other than muscular performance. This could potentially provide a reason why there was no observed negative decrements in the 100m sprint⁸¹ as it is an event that relies primarily on muscular performance. Although there are no effects of alcohol intoxication on muscular performance, there is a negative effect of alcohol on endurance based exercise. Such an effect would result in alcohol being ergolytic as opposed to ergogenic (previously theorized).

The majority of athletes at any level of competition do not consume alcohol immediately prior to competition rather consumption usually takes place either the night before ⁹⁶, or following competition ⁶. As such only extreme alcohol consumption will still be present in the circulation thus negatively affecting performance. Due to the predominant occurrence of drinking in sports people following exercise/competition the effects of alcohol on recovery would be of great importance. Despite such practice being prevalent in sporting society there is a surprising lack of research into the effect of alcohol on recovery from exercise.

Alcohol's effect on subsequent performance

As previously discussed, eccentric muscle action results in a significant amount of muscle damage. Barnes et al. (2010) looked at how acute moderate alcohol consumption affects muscular performance during recovery from eccentric induced muscle damage. Eleven healthy males performed 300 maximal eccentric contractions of the quadriceps muscles of one leg before being allocated to either an alcohol condition (1g/kg bodyweight) or control condition (isocaloric quantity of orange juice). On another occasion the participants underwent the same exercise protocol followed by the remaining condition. As expected the 300 eccentric contractions resulted in significant decreases in isometric, concentric and eccentric peak torque over time. All post exercise strength measures were significantly different between conditions with the greatest decrement observed with alcohol. It is therefore apparent that even moderate acute alcohol consumption magnifies losses in both dynamic and static strength thus having a detrimental effect on recovery from eccentric exercise induced muscle damage ⁶. It must be noted that there were no observed increases in creatine kinase activity as a result of the alcohol condition. Accordingly, the changes in muscular performance appeared to be unrelated to creatine kinase activity. A further study ⁶used the same protocol measuring maximal dynamic and static torques of both exercising and non-

exercising quadriceps. It was concluded that it is the recovery of exercise induced microstructural damage that is adversely affected rather than a weakening of all muscles. A low dose of alcohol (0.5g/kg body weight) has however been shown to have no effect on the loss of force associated with eccentric exercise ⁷. It is interesting to note that the detrimental effect on recovery from eccentric exercise induced muscle damage is only apparent after a moderate dose of alcohol suggesting a dose dependent relationship. These studies support the theory that alcohol does not adversely affect motor performance ¹⁰³. How alcohol consumption greater than 1g/kg body weight affects such performance is unknown, despite being common place (Snow and Munro (2006) cited in

Sports such as rugby union have a high amount of associated muscle damage following competitive match play. It could be theorized therefore that alcohol would negatively affect recovery. What needs to be considered however is that the eccentrically induced muscle damage^{6; 7} is particularly extreme and isolated to one particular muscle group (the quadriceps). It must be noted that it is extremely difficult to quantify muscle damage. As discussed, during rugby muscle damage occurs from both eccentric muscle action and blunt force trauma, the associated damage is therefore not isolated to one particular muscle or group of muscles. Taking this into consideration supposing muscle damage was the same across both this isolated eccentric protocol (isolated muscle group) and a competitive game of rugby (whole body eccentricly induced muscle damage and blunt force trauma induced muscle damage) would there be a similar effect on performance or is a muscle group's ability to generate peak torque determined by the muscle damage to that particular muscle? Accordingly whether such a magnified loss in both dynamic and static strength following both acute alcohol consumption and a competitive rugby game still needs to be established.

The majority of the research has focused solely on the effects of alcohol on recovery following relative simple sporting exercise whether it be eccentric muscle action, or running at a variety of % of VO₂ max's or distances. Results would therefore only be indicative to those sports. Research into the prevalence of drinking illustrate that athletes of a team sport are more likely to indulge in postgame alcohol consumption than individualistic sports. As such it would be important to determine whether such behaviour influences the recovery from game play. To date only the effects of acute moderate alcohol consumption on physical performance, creatine kinase and immuno-endocrine function during recovery from a simulated rugby match⁸ has been investigated

The study looked at ten male senior rugby players who completed a modified version of the BURST protocol (modified to include game specific contacts), before consuming either an alcohol beverage (vodka and orange juice, 1g alcohol per kg BW) or an isoenergetic orange juice control. Performance

measures (CMJ, agility, 15m sprint time and scrummaging performance) were measured pre, 4hrs and 48hrs post. Creatine kinase and immuno-endocrine function were measured pre-simulation, 30 minutes, 12hrs, 24hrs, and 36hrs and 48hrs post. Creatine kinase and immuno-endocrine function were different as a result of the game simulation however there was no difference as a result of alcohol consumption on the magnitude of these changes. Following the simulation and moderate alcohol consumption CMJ height was significantly decreased indicating that alcohol negatively affects lower body power output following simulated rugby match play. No other measures of physical performance were seen, thus suggesting movements requiring repeat maximal muscular action are not affected as a result of alcohol consumption following simulated match play 8. This study provided an insight into the effects of a game however it must be noted that although the simulation (modified BURST protocol) was designed to emulate a game as such it would be extremely difficult to replicate the severity of contact a player would experience during a game. Participants had just completed their competitive season therefore by having a relatively high level of match fitness, it could be proposed that these participants were relatively accustomed to motions and hence the eccentric component of match play. Through a lack of severity experienced in the simulated contact the participants could have finished the simulation with considerably less muscle damage than following a competitive game carried out at the same time of the season. It must also be noted that the study was limited by the amount of alcohol subjects were ethically allowed to consume thus consuming a quantity far short of what would be consumed 'normally'. Despite such draw backs the study provides an insight into the influence alcohol consumption has on recovery from a rugby match.

A study by ⁹⁵ found that aerobic performance of rugby players was significantly affected, performing on average 11.4% worse when the players were exposed to 'normal' Friday night drinking habits (large range between 1 and 38 units of alcohol). The players anaerobic performance however was unaffected. Such results would indicate that the only effects of alcohol on performance would be on the ability to work aerobically^{81; 103}.

Counter movement jump (CMJ) height has been shown to be significantly reduced following Australian rules football ³⁰ and rugby league ⁸⁰. Such reduction has also however not been seen in Australian Rules football²⁴ with the only significant reduction existing in the contraction time of the countermovement jump, and not in height.

Simulated rugby union match play failed to elicit a reduction in CMJ Height⁸, rather the only significant reduction was observed following simulation and moderate alcohol consumption. The effect of match play on CMJ therefore has been seen to vary from study to study. However with only

one study looking at the effects of alcohol on this ability to jump, a detrimental finding would be proposed however subject to further investigation.

Alcohol associated behaviour

Adequate sleep is often recommended as being essential for optimal performance. Studies investigating the effect of both sleep loss and sleep deprivation have found a negative impact on neurobehavioral performance with both significant psychomotor changes ⁴³ and negative effects on selective attention ⁷¹. When trying to quantify such impairment it was found that moderate levels of fatigue produced performance decrements greater to or equal to those observed at levels of alcohol intoxication deemed unacceptable whilst driving or in the work place ⁶⁸. Physical performance differences however are not seen until participants have been at a state of wakefulness for 36 hours. The effect of 36hrs sleep deprivation on prolonged exercise was found to be an 11% decrement in the time in which 80% of VO₂ max could be maintained. A failure to find any differences in both heart rate and metabolic rate along with a corresponding significant increase in perceived exertion suggest that a decrement in prolonged exercise was a psychological result rather than a physiological one ⁷⁶. It has previously been shown that 36 hours of sleep results in a negative effect on anaerobic performance ¹²⁹, reducing both peak and mean power. Blood lactate concentrations were not affected by sleep loss, time of the day or an interaction between the two. The authors therefore attributed this decrement in anaerobic performance to either cumulative fatigue working against circadian rhythm, sleep deprivation on arousal or the results being influenced by the time of day at which the testing took place. The study failed however to measure how the subjects perceived the particular testing. The decrement seen at 36 hours could be a result of an increased perception of difficulty as seen in the effect on prolonged exercise. This effect on anaerobic performance could therefore be a result of an increase in psychological difficulty. Such a decrement in anaerobic performance was not observed when a Wingate test was performed following partial sleep deprivation ⁸⁶. It would therefore appear that only extreme sleep deprivation adversely affects anaerobic performance whereas partial deprivation as commonly experienced in conjunction with alcohol consumption would induce no effect on anaerobic performance.

Post-game behaviour whether it is alcohol consumption or lack of sleep regardless of effect on physical performance or recovery may have a follow on effect by affecting optimal nutritional practice. The recommended guidelines on optimal nutritional practice whether it be everyday or sport specific are well documented¹⁷. One study investigating how alcohol use affects both diet and

health status found that alcohol consumption displaces other energy sources of the diet subsequently reducing the nutrient density of the diet ⁵⁴. Such a reduction in nutrient density of the diet would have a subsequent negative effect on health status as is evident through blood chemistry. The effect of such an alteration following demanding exercise on recovery has yet to be investigated.

Summary of literature

Sport participation is associated with a higher alcohol consumption than that of the general population ^{101; 72}. Such a relationship is influenced by many factors such as the sport played ¹⁰¹ and the level at which it is being played ⁹⁷. Team sports such as rugby have been found to be associated with the consumption of large amounts of alcohol ⁷². Within such sports a curvi-linear relationship appears to exist between the level of participation and excessive/hazardous alcohol consumption ⁹⁷. Rugby evolved as a means of social control ¹⁵³ adopting on-field expectations based around courage, athletic prowess and the upholding of gentlemanly and sportsman like values ³⁵. Off-field a contrary subculture was adopted with the tendency towards the violation of social taboo's characterized by such behaviour as excessive alcohol consumption and public nudity ¹⁵³.

Rationalization of rugby union in August 1995 resulted in rugby shifting from amateurism to professionalism. This shift resulted in a corresponding change in on-field values with success being viewed as the ultimate goal of participation ³⁵. A prevalent off-field deviant subculture remained however and is now generally accepted as being part of a 'normal' sporting culture ¹¹. The majority of research into such behaviour has focussed primarily on the immediate impact of alcohol consumption on sporting performance, with such research highlighting the ergolytic nature of alcohol on performance. The fundamental issue with such research however is that the deviant behaviour associated with sports such a rugby typically occurs the night previous to 96 or following competition ⁶, accordingly the effect of such customary behaviour on recovery from competition or on subsequent performance would be more specific to what actually occurs. Initial work^{6; 7; 8} has begun investigating how alcohol consumption would affect recovery from both eccentric exercise and simulated rugby match play. It was found that moderate alcohol consumption adversely affected the recovery of exercise induced microstructural damage ⁶ as well a negatively affecting subsequent lower body power output post rugby simulation 8. Despite such conclusions suggesting that alcohol negatively influences both recovery and subsequent performance it must be considered that such investigations were laboratory based thereby controlling all aspects of behaviour whilst only varying the amount of alcohol consumed. As such conclusions are applicable to behaviour similar to that tested and they are not truly representative of what typically occurs following rugby. The question of how customary off-field rugby behaviour affects both recovery and subsequent performance remains unanswered. The purpose of this study was therefore to use a naturalistic means of investigation to determine whether customary rugby behaviour would affect either recovery or subsequent performance.

3. Hypotheses

It was hypothesized that:

- i. Rugby's subculture of excessive alcohol consumption, sleep deprivation and interference of dietary practice remains within New Zealand.
- ii. The investigated behaviour customary to rugby (combined effect of alcohol consumption, sleep deprivation and interference with regular dietary practice) would adversely affect the player's subsequent ability to perform rugby specific measures (counter movement jump height, maximal strength & repeated sprint ability) in the days following a competitive game.
- iii. Such investigated behaviour would have a negative effect on hydration status following competitive match play.
- iv. The investigated behaviour in the absence of competitive match play would have a neglible effect on rugby specific performance measures.

4. Methods

4.1. Participants

Thirty senior grade club rugby players (age = 21.7±1.4 years, mass = 87.2±9.3kg, height = 182.0±5.9cm) from two senior grade rugby teams volunteered to participate in the post-game behaviour study. The players were allocated to either the standardized post game behaviour group (SPGB) or investigated post game behaviour group (IPGB) by team. The study took place immediately following the final game of the 2011 season, ensuring a sufficient level of rugby specific fitness.

Nineteen senior grade club rugby players (age = 20.8 ± 1.6 years, body mass = 88.5 ± 15.9 kg, height = 182.4 ± 6.3 cm) from the previous study volunteered to undergo the subsequent trial investigating the effect of investigated behaviour on performance measures and hydration in the absence of competitive match play.

Voluntary consent and the completion of a health screening questionnaire were obtained for each participant prior to the commencement of the study. The study was approved by Massey University Human Ethics Committee.

4.2. Study Design

The study utilized a naturalistic method of investigation to take advantage of when customary rugby behaviour occurs (i.e. following competitive match play). Following the completion of game play the participants from two senior grade club teams were split by team into either the SPGB or IPGB. Such an allocation allowed all the participants to compete in the one game, removing any potential match related differences.

One week prior to baseline testing all participants underwent familiarisation of the entire testing protocol.

The participants were instructed to abstain from exercise and alcohol for at least forty eight hours prior to testing. Forty eight hours prior to commencing the study all participants reported to the laboratory to determine bodyweight, baseline performance measures, alcohol use disorders identification test (AUDIT), urine specific gravity, and a twenty four hour food and behaviour recall.

Following the rugby match, participants returned to the laboratory for urine specific gravity and blood samples before being split into condition by team. The first group (IPGB) involved participants undergoing 'customary' post-game behaviour with no further contact with the investigators apart from follow up testing at 12hrs and 36hrs. The second group (SPGB) involved participants undergoing a standardized behaviour intervention involving a controlled meal (1g CHO per kg BW & 20gPRO¹) with unlimited non-alcoholic beverages and evening entertainment before being returned home and instructed to be in bed by 11pm. All participants returned to the laboratory at both twelve and thirty six hours post-game for repeat performance measures, urine specific gravity, blood sample and food and behaviour recall for the previous twenty four hours. The protocol can be illustrated with the below timeline (Figure 9):

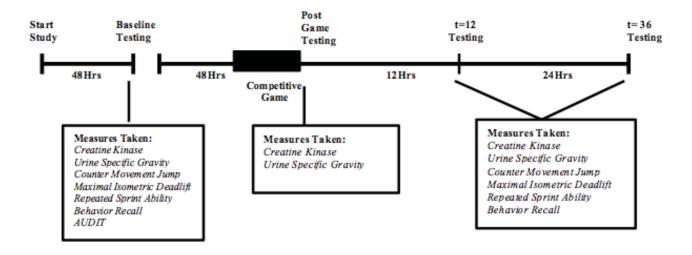


Figure 9 A Schematic representation of the protocol used. Testing took place at baseline, immediately post, twelve hours post and thirty six hours post.

The opportunity arose to investigate the effects of solely 'customary rugby behaviour' in the absence of competitive match play (IB). The participants underwent the same protocol investigating the effect of such behaviour on performance measures following the end of season function.

4.3. Experimental Measures

4.3.1. Creatine kinase

¹ Based on practice tips for recovery after training and competition ¹⁷

Venous blood was collected from the antecubital vein into 4ml K₃EDTA vacutainer tubes (Beckton Dickinson, UK) which was then centrifuged at 4°C for 10 minutes at 1650g. Plasma was separated and frozen at -80°C for later analysis. A Vitalab Flexor clinical chemistry analyser (Vitel Scientific NV, Netherlands) and Roche CK-NAC liquid assay kit (Roche Diagnostics GmbH, Mannheim, Germany) were then used to determine Creatine Kinase activity.

4.3.2. Performance Measures

Each participant then gave a mid-stream urine sample before commencing a warm up. Prior to each testing session each subject underwent a five minute warm up at 100 Watts using a Monark cycle ergometer (Monark, Stockholm, Sweden) followed by movement specific stretching. Each participant then underwent performance measures in the following order:

Counter Movement Jump

Three countermovement jumps ¹⁰ were performed using an electronic jump mat (Smart Jump, Fusion Sport, Australia). Each jump was separated by 30 seconds. The maximum jump height was recorded.

Maximal isometric deadlift

The maximal isometric deadlift exercise was performed on a platform consisting of modified barbell and chain connected to a tension s-beam load cell (Muller, Germany) connected with a custom made amplifier and Power lab (ADInstruments, Australia). Each participant was instructed on the proper technique of lifting, with the feet shoulder width apart, knees at approximately 30° of flexion and elbows in full extension. Each participant performed three maximal efforts separated by 1 minute of rest. The maximum value was recorded.

Repeated sprint Ability

Participants underwent six forty metre sprints departing every thirty seconds as described by ⁴⁴. The sprints took place inside the lab using a photoelectric timing system (Smart speed, Fusion Sport, Australia). The timing gates were programed to allow participants to start each repetition from the same end as they finished their last repetition therefore removing the need to return to a common starting point for the commencement of subsequent repetitions. During each testing session two participants would participate inducing an element of competition and it was ensured that each participant always ran against the same participant throughout testing. Each repetition took place from a standing start 40cm from the timing gate. The timing of each repetition started and finished

when the participant broke the light beam. Each repetition time as well as total time spent running was recorded electronically.

4.3.3. Urine Specific Gravity

Midstream urine samples were taken at baseline, immediately post-game and at twelve hours and thirty six hours post-game/ end of season function. Urine specific gravity (U_{sg})was determined from each sample using an Atago optical urine specific gravity refractometer (Atago, New York, USA) calibrated using deionized water. U_{sg} were compared to the indexes of hydration status ¹⁹ to indicate hydration status.

4.3.4. Behaviour Recall

At each testing period, the previous twenty four hours of behaviour (sleep, alcohol consumption and level of physical activity) was established using a behaviour recall questionnaire (see appendices). The behaviour was categorised according to table 4, to allow for statistical analysis.

Table 4 Categorisation of behaviour recall

Category	Alcohol	Sleep
1	0	0
2	1 to 3	1 to 4
3	3 to 5	4 to 6
4	6 to 10	6 to 8
5	10 to 20	8 to 10
6	20 or more	10 or more

4.3.5. AUDIT

During baseline testing each participant answered the AUDIT, allowing an analysis of habitual alcohol use to be undertaken.

4.4. Standardized Behaviour Intervention

Participants allocated to the SPGB condition reported to the laboratory thirty minutes following the completion of the game. Participants were then provided with a controlled meal based upon practice tips for recovery after training and competition ¹⁷. Each participant was provided with a meal and drink consisting of 1g of carbohydrate per kg body weight and 20g of protein (R-Line, New Zealand & Subway Sandwich's, New Zealand) with the volume of sports drink provided being varied according to participant's bodyweight. The participants then watched a movie before being returned home and instructed to be in bed by 11 pm.

4.5. Data Analysis

The changes both within and between conditions (SPGB vs. IPGB) were analysed using a two-way repeated measures analysis of variance (ANOVA) (Condition * Time). The differences between time points within each condition were further analysed using a post-hoc pairwise comparison (Bonferroni adjustment). AUDIT questionnaire results were analysed using a one way ANOVA to establish whether any significant differences existed between each of the three subscales (Hazardous alcohol use subscale, Dependence symptoms subscale & Harmful alcohol use subscale) as well as between the experimental conditions total scores. T-tests were used to establish whether there was a statistical difference between the two conditions. All analysis was carried out using the Statistical Program for Social Sciences (SPSS) for Windows (Version 18.0.0, SPSS Inc., Chicago, IL) with the reported values given as mean ±SD, with statistical significance set at p<0.05.

5. Results

Two SPGB participants and four IPGB participants did not complete the testing and were subsequently excluded from the results.

5.1. Creatine Kinase Activity

Competitive match play resulted in significant changes in CK activity illustrated by table 5 below:

Table 5 Changes in CK (IU.L-1) following a senior grade competitive rugby match (mean±s).

	Baseline	IMPG	12 Hrs. Post	36 Hrs. Post
IPGB	230.8±54.2	248±80.9	534±245.8	410.9±149.5
SPGB	264.9±51.6	448.9±77.1*	897.3±234.3*	686.1±142.5*#
Total	247.9±37.4	344.5±55.9	715.7±169.8*#	548.5±103.3*#

Significant difference from baseline measures: *P<0.05

Significant difference from IMPG: #P<0.05

A main effect of time (p<0.001) was observed for CK following competitive match play. No difference was observed between conditions (p=0.22) or as a result of the time*condition interaction (p=0.376). Total mean CK activity was significantly different between baseline and both 12 hours post (p=0.034) and 36-hour post (p=0.011) as well as between immediately post game and both 12 hours post (p=0.042) and 36 hours post (p=0.009). A significant difference was observed for the SPGB condition between baseline and immediately post game (p=0.031), 12 hours post (p=0.039) and 36 hours post (p=0.010) as well as between immediately post game and 36 hours post (p=0.034).

5.2. Performance Measures

5.2.1. Counter Movement Jump

At twelve hours following the IB trial there was a significant decrease in jump height (p=0.041) with a subsequent significant increase at 36hrs post behaviour (p=0.003) Clearly illustrated by figure 10 below:

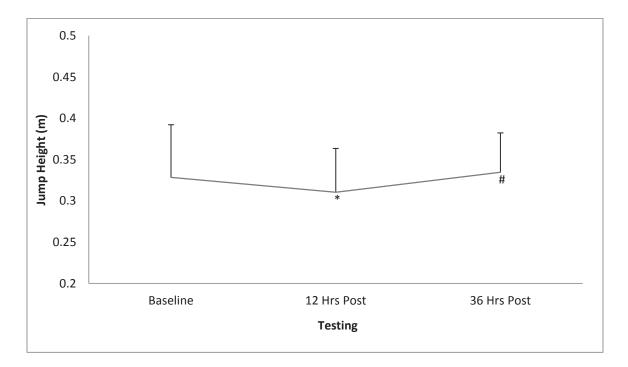


Figure 10 Maximal countermovement jump height (m) for participants in the IB condition at baseline, 12hrs and 36hrs post behaviour. (* Indicates a significant difference from baseline, # indicates a significant difference from 12Hrs Post)

Countermovement jump height for both post game conditions is illustrated by figure 11.

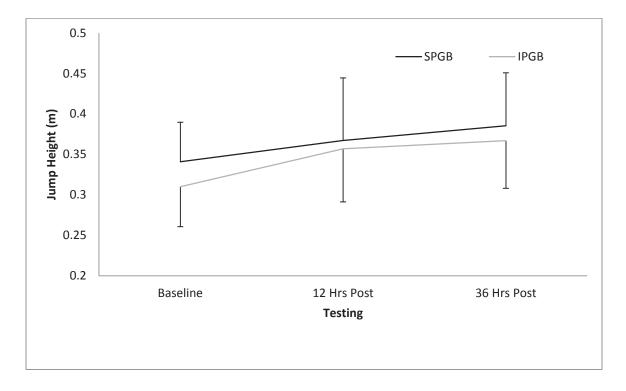


Figure 11 Maximal countermovement jump height (m) for participants in both the IPGB and SPGB conditions at baseline, 12hrs and 36hrs post behaviour.

There was no significant difference as a result of time (p=0.217)or of the time*condition interaction (p=0.764). Neither the game or post game behaviour therefore had any significant effect on countermovement jump height.

5.2.2. Max Strength

Results from both the post-game conditions and the post behaviour trial indicate no significant effect of time in the maximal force generated (p=0.239, p=0.387 respectively). There was also no significant difference between conditions (p=0.289).

5.2.3. Repeated Sprint

Mean sprint time and percentage decrement were calculated for both trials (table 6) following both game and behaviour only trials. Following the behaviour only trial t-tests indicate no significant difference between either mean sprint times or percentage decrements (p=0.221 & 0.675 respectively). Post-game there was no significant difference in percentage decrement over time or between conditions over time (p=0.706 & 0.15 respectively). Total sprint time was not significantly different as a result of time (p=0.767) or the time *condition interaction (p=0.070).

Table 6 Repeat sprint results: Individual sprint times, total sprint time, mean sprint time and percent decrement between initial and final sprint for each behaviour condition. Values are means (±SD)

		Time(s)						Mean	Total	%
coco		rep. 1	2	3	4	5	6	Time (s)	Time (s)	Decrement
SPGB	Baseline	5.91	6	6.21	6.39	6.59	6.47	6.26	37.57	6.04
	визенне									
		0.39	0.43	0.57	0.47	0.91	0.41	0.46	2.74	3.48
	12hrs post	5.67	5.86	5.97	6.18	6.21	6.32	5.67	36.21	6.42
		0.3	0.39	0.33	0.45	0.41	0.39	0.31	2.19	1.35
	36hrs post	5.98	6.01	6.09	6.15	6.31	6.48	5.92	37.02	4.19
		0.4	0.49	0.51	0.35	0.48	0.75	0.4	2.59	1.69
IPGB										
	Baseline	5.82	5.89	6.15	6.26	6.27	6.37	6.13	36.77	5.3
		0.34	0.35	0.46	0.42	0.44	0.43	0.38	2.29	2.1
	12hrs post	6.1	6.16	6.4	6.37	6.63	6.68	6.39	38.34	5.04
		0.33	0.3	0.47	0.33	0.5	0.29	0.34	2.05	2.08
	36hrs post	6.3	6.48	6.61	6.47	6.79	6.81	6.58	39.45	5.91
	•	0.46	0.64	0.7	0.31	0.42	0.92	0.52	3.14	4.33
IB										
	Baseline	5.92	6.00	6.27	6.42	6.58	6.67	6.31	37.86	6.69
		0.42	0.45	0.49	0.49	0.57	0.55	0.46	2.78	2.44
	12hrs post	6.38	6.49	6.55	6.74	6.86	6.87	6.65	39.90	5.82
		0.70	0.68	0.65	0.68	0.88	0.70	0.63	3.79	3.33
	26hrs nost									
	36hrs post	6.15	6.34	6.40	6.63	6.42	6.67	6.44	38.61	7.33
<u> </u>		0.48	0.77	0.53	0.62	0.64	0.65	0.53	3.16	6.71

Mean sprint time following the game showed no significant change between all three time points (as seen in table 6 above) however a visible trend is attributed to the time, condition interaction (p = 0.058) illustrated by figure 12.

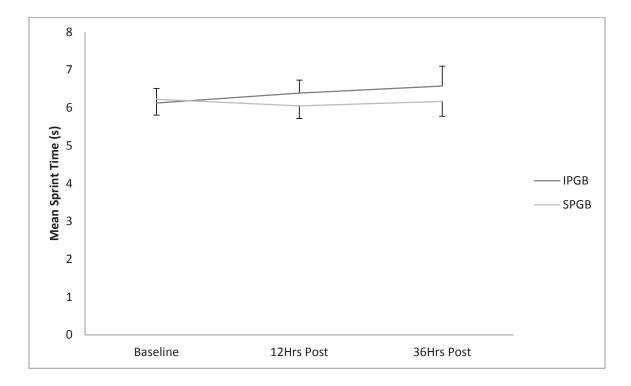


Figure 12 The effects of behaviour on mean sprint time. Mean Sprint time at baseline as well as at 12 hours and thirty six hours post competitive match play for both the IPGB and SPGB conditions.

5.3. Urine Specific Gravity

The T-test revealed no significant difference as a result of time (p=0.431 post game & 0.515 behaviour only) however post game there was a significant difference due to the time* condition interaction (p=0.038). Post-hoc further showed that the only significant difference between conditions following the game occurred immediately post game (p=0.045).

Table 7 Mean urine specific gravity post-game at base line, immediately post game, 12 hours and 36 hours post competitive match play. Values are means (±SD)

Condition		n	Baseline	Immediately Post Game	12hrs Post	36hrs Post
Post-Game						_
	SPGB	11	1.019	1.021*	1.018	1.016
			0.007	0.004	0.007	0.006
	IPGB	13	1.021	1.016*	1.022	1.020
			0.007	0.006	0.004	0.009
IB		19				
			1.023		1.019	1.021
			0.005		0.005	0.006

• Indicates a significant difference

Immediately following the game the partcipants in the SPGB condition were significantly more dehydrated than the IPGB condition with U_{sg} of 1.021 ± 0.004 and 1.016 ± 0.006 respectively.

5.4. Behaviour Recall

Post-game behaviour

Table 8 Table illustrating the categorized participant behaviour at baseline, 12 hours and 36 hours post testing.

		Baseline	12Hrs Post	36Hrs Post
	IPGB	4.6	2.3*	4.1
	IPGB	0.5	0.6	0.7
		4.2	4.3	4.1
Sleep	SPGB	0.6	0.7	0.4
			t	
	IB	4.4	2.9*	4.4
		0.5	0.7	0.8
	IPGB	1	5.6*	1
	II Gb	0	0.5	0
		1	1	1
Alcohol	SPGB			
		0	0	0
	IB	1	5.4*	1.1
	ID	0	0.6	0.3

Significant difference from baseline measures *P<0.01

Following a game it is evident that those participants assigned to the SPGB condition demonstrated no significant changes (p>0.05) in either sleep experienced or alcohol consumed when compared to baseline measures. The IPGB condition exhibited significant decreases in sleep experienced (p<0.01) as well as a significant increase in the amount of alcohol consumed (p<0.01). In the absence of match play (IB Condition) there was a significant increase in the amount of alcohol consumed (p<0.01) and decrease in the amount of sleep experienced (p<0.01).

5.5. AUDIT

Of the thirty participants in the study six failed to complete the AUDIT questionnaire and were therefore removed from the analysis. Mean total and subscale scores were calculated for the twenty four participants and are presented in table 9.

Table 9: AUDIT (total, hazardous subscale, dependance subscale, and harmful subscale) scores for both IMPG and SPGB conditions

		Audit Sc	ores		
	,	Total	Hazardous	Dependence	Harmful
Condition	n	Score	Subscale	subscale	Subscale
		15.8			
SPGB	13	(2.5)	8.7 (1.3)	2.1* (1.2)	5.1 (1.9)
		19.7			
IPGB	11	(6.1)	9.2 (1.2)	4.2 *(1.2)	6.4 (4.2)

Scores are presented as means (standard deviation in parenthesis)

Results of the one way ANOVA show no significant difference in the total, hazardous subscale and harmful subscale (p=0.067, 0.359 and 0.372 respectively) between the two different groups of participants. There was a significant difference in the score on the dependence scale (p= 0.012) with the IPGB group eliciting a higher score. The combined total average for the entire test population (24 participants) was 17.7 ± 5 .

5.6. Testing Conditions

The average temperature and humidity was 19.4±0.4°C and 81.8±2.0% respectively with no significant difference between trials (p>0.05).

^{*} indicates a statistical difference

6. Discussion

The main finding of this study was that rugby specific performance measures (counter movement jump, maximal isometric deadlift and RSA test) were not significantly affected by behaviour customary to post rugby union game play. These findings are contrary to recent laboratory based findings showing that moderate alcohol consumption negatively impacts lower body power output following a simulated rugby match ⁸ as well as magnifying losses in performance after eccentric induced muscle damage ^{6; 7}. We speculate that, given the extreme levels of exercise induced muscle damage found by Barnes et al. (2010, 2011), the results and practial recomendations of previous research may not be representative of what actually occurs in sport.

Competitive match play was found to illicit no significant changes in counter movement jump, maximal isometric deadlift exercise or RSA percent decrement. Such lack of change in CMJ has been previously observed post game ²⁴ however the effect of match play on maximal strength or repeat sprint ability has not previously been studied. Accordingly the present study would suggest that 12 and 36 hours after match play a player's ability to perform exercise involving maximal strength or repeated maximal sprints is not significantly different than prior to the match. There was no significant difference between conditions for CMJ, maximal strength or repeated sprint ability thus indicating that performance measures were unaffected by post game behaviour, contrary to the hypothesis.

Previous research has found that the 'hangover' does not significantly affect sprint performance ^{99; 8}. These studies measured a single maximal effort sprint following moderate alcohol consumption of laboratory based and naturalistic based trials respectively. An observed trend in mean sprint time in this study suggests that there was a tendency for customary post game behaviour (IPGB condition) to result in an increased mean sprint time when combined with competitive match play. The increase in mean sprint time following customary behaviour therefore is contrary to the existing literature and was not accompanied by a corresponding increase in percent decrement in the repeat sprint ability test. It can be theorized that such an increase in sprint time could either be a result of a far greater prior alcohol consumption in this study than previous work or that the participants in the 'hung over' state felt that they had to hold back initially in order to complete the testing, thus resulting in a greater mean sprint time at 12 and 36 hours post.

Following the IB condition, maximal strength, mean sprint time and repeat sprint decrement were not significantly different from baseline. Strength and sprint performance therefore appear to be

unaffected by customary behaviour. Contrary to the results following competitive match play there was a significant decrease in counter movement performance at 12 hours post followed by a significant increase at 36 hours post behaviour. The IB condition had a negative effect on countermovement jump that was not observed when this behaviour was combined with competitive match play. The naturalistic design and resulting lack of control over behaviour, may have resulted in differences in behaviour, possibly explaining the difference in result between the two customary behaviour trials. Despite both customary behaviour trials (IPGB, IB) reporting substantial alcohol consumption (prevalence of 20+ recall) we were unable to compare whether there was a difference between quantities greater than 20 standard drinks between trials. This presents a limitation of the present study as well as potentially explaining why there was observed differences in counter movement jump between the two customary behaviour trials.

From the existing literature it was hypothesised that alcohol consumption associated with post game customary behaviour would result in the dehydration of the players following match play. The average baseline U_{sg} illustrates that players in the SPGB condition were minimally hypo hydrated (U_{sg} =0.019) whereas players in the IPGB condition were significantly hypo hydrated (U_{sg} =1.021) at baseline ¹⁹. There was no significant difference between the two conditions and these results illustrated that the players did not have a good hydration level even at baseline, agreeing with previous literature reporting dehydration within the adult population ^{61; 144}. Immediately following the game there was a significant difference in U_{sg} between the IPGB condition and the SPGB condition. It was found that players in the IPGB condition's hydration status improved from baseline whilst those in the SPGB condition's declined. This could have resulted from better pre and during game hydration, this however was not investigated. Following the IPGB condition the hydration status of the players returned to baseline levels (more hypo hydrated than post game). The absence of significant hypo hydration resulting from the IGB condition would suggest that the body was able to maintain its hydration status despite relatively large levels of alcohol consumption, which are thought to promote urine loss ¹²⁵ Immediately post game the players were 'minimally dehydrated' (U_{sg}=1.016±0.06) which was of similar magnitude to the hypo hydrated condition used by Hobson & Maughan (2010), as such alcohol associated diuresis is blunted to prevent further hypo hydration. In the IB condition the players were significantly hypo hydrated at baseline (U_{sg}=1.023±0.005), following customary behaviour only there was no significant change in hydration status. Irrespective of whether match play was undertaken, players at this level of competition appear to be habitually hypo hydrated. The effects of customary behaviour on players in such a state are minimal. Whether such behaviour would elicit a different response in players in a euhydrated state such as players at a higher level of play would require further investigation. The players in the SPGB condition became more hypo hydrated as a result of match play, possibly through adherence to recommended guidelines for hydration during exercise ¹⁷.

The competitive match play in this study resulted in a significant increase in CK activity, peaking at 12 hours post game. Such an increase was similar to that following collegiate match play ¹³² and rugby simulation ⁸, however lower than observed following provincial ^{47; 128} and international match play ²⁷. Such results indicate that muscle damage did occur as a result of match play during this study. Despite illustrating that muscle damage did occur as a result of match play great variability amongst individuals ¹³ as well as the fact that changes in CK do not provide a means to accurately quantify muscle damage ^{138; 145} would result in it not being possible to compare the level of muscle damage between participants. Accordingly muscle damage resultant from match play irrespective of magnitude did not illicit significant decrements in performance measures ⁸ regardless of post-game behaviour.

The behaviour recall illustrated significant alcohol consumption and reduction in sleep following the IPGB condition as well as the IB condition. It is therefore clear that rugby union is still associated with high levels of alcohol consumption and reduction in sleep ^{107; 97} following both competitive match play and other rugby associated functions. Alcohol use as investigated by the AUDIT resulted in a mean score of 17.7±5, a score far greater than previously found for New Zealand rugby payers ⁹⁷or university students ⁶⁷ and is indicative of alcohol use disorders ¹⁰⁷. A score of 8.9±1.3 in the hazardous alcohol use subscale is of particular concern indicating a considerable binge drinking behaviour that may be unique to this population. It is therefore apparent that particularly in the population investigated a unique culture characterized by excessive alcohol consumption and reduction in sleep has prevailed. How this culture would compare with other groups of rugby players at a range of levels would require further investigation.

Application of findings

Existing practical guidelines suggest that alcohol both directly and indirectly affects optimal recovery in the athlete ¹⁷. The present study found that customary rugby behaviour following competitive match play or functions was characterised by both excessive and hazardous alcohol consumption and a reduction in the amount of sleep experienced.

Accordingly it was hypothesized that customary behaviour following match play would adversely affect subsequent performance. Results however showed that despite such behaviour, neither subsequent performance nor hydration status were adversely affected. Such results would suggest that customary rugby behaviour does not affect optimal recovery following a game of rugby.

Alcohol has been shown to increase high risk behaviour ¹⁷ as well as having long term adverse effects on health ^{111; 118}. As such recommended guidelines regarding responsible alcohol use for the general population could suffice following sport participation.

Limitations

The design of the study resulted in the investigation of typical post-game behaviour, such as the amount of alcohol consumed, timing of consumption and amount of sleep which was self-selected by the players and therefore not controlled by the investigators. As such, behavioural recall was used to record such individually dictated behaviour. Research has shown that the recall of alcohol consumption tends to be reported reliably by non-alcoholics ^{82; 29; 31}. The results of the AUDIT would suggest that the players sampled could potentially be classified as alcoholics. As such the ability of the players to report alcohol consumption accurately must be questioned. The means of measuring alcohol consumption in this study was designed to reduce this inaccuracy. The recall questionnaire was developed with the assumption that rugby players may typically drink ten or more drinks ¹⁰⁷, accordingly the highest category was set at 20 or more standard drinks. The results illustrate that this category was prevalent and therefore the results of this study are limited further by the inability to quantify drinking behaviour greater than this.

Existing literature highlighted inter positional variability in both energy expenditure and intensity ³⁴; and it could therefore be assumed that customary behaviour post match play could affect players differently according to position. This study due to a limited sample size did not categorize players according to their position, therefore any relationship between post-match behaviour and position remains to be investigated.

The participants of this study were aware that their behaviour was being investigated. Accordingly as a result of the Hawthorne effect, 'customary behaviour' as observed during this study may be influenced just through participation in the study.

The authors acknowledge that the sample used in the present study was one of university senior grade rugby players and as such may not be representative of the entire New Zealand rugby playing population.

Through the use of two teams one being allocated to a behavioural intervention and one into a control condition the effects of such behaviour on baseline measures could be compared. There is therefore the limitations that both conditions were not done using the same players in a randomized blinded manner as well as a small sample size therefore the sample tested could have potentially been outliers.

Finally this study investigated the effect of a single evening of customary behaviour on rugby specific performance measures, the conclusions drawn are therefore specific to an acute exposure to such behaviour, the effects of chronic behaviour for example over the duration of the competitive season on performance measures and other rugby season related adaptations would require further investigation.

7. Conclusions

The purpose of this study was to investigate whether post-game behaviour, that is customary to rugby union, is detrimental to the subsequent performance of players in the days following competitive match play. The naturalistic design of this experiment allowed the effects of realistic uninfluenced behaviour on subsequent rugby specific performance to be investigated. To the authors knowledge this is the first time in which such an approach has been utilized, the findings would therefore provide a key insight into whether prevalent post-game rugby behaviour affects subsequent performance.

The results of the present naturalistic study indicate that following a competitive match, customary rugby behaviour consisting of significant alcohol consumption and a reduction in sleep failed to significantly affect subsequent rugby specific performance measures in the days following the match. The naturalistic means of investigation allowed for customary behaviour to be investigated without influence, allowing for a true representation of what typically occurs following competitive match play.

The present study was limited by the use of a relatively small size removing the ability for inter positional variation to be investigated as well as potentially the teams being looked at being outliers compared to the entire population. Finally the study was limited to the effects of a singular customary behaviour experience, the long term effect of regular experiences may therefore have an effect on physical recovery this however would require further investigation.

Results of this study would indicate that previous recommendations regarding the negative affect of customary post game behaviour on physical recovery are unfounded. The effect of such consumption on risk taking behaviour as well as the long term consequences of such high alcohol consumptions would however represent the negatives of such consumption. Accordingly it would be recommended that the guidelines for responsible alcohol consumption following physical activity should be no different to that of the general population.

8. References

- 1. (1982) American College of Sports Medicine Position Statement on: The Use of Alcohol in Sports. *Medicine & Science in Sports & Exercise* **14**, ix-xi.
- 2. (2009) Alcohol use in New Zealand: key results of the 2007/08 New Zealand Alcohol and Drug Use Survey, [Mo Health, editor]. Wellington.
- 3. Bamford TW (1957) Discipline at rugby under Arnold. Educational Review 10, 18-28.
- 4. Bangsbo J, Mohr M & Krustrup P (2006) Physical and metabolic demands of training and matchplay in the elite football player. *Journal of Sports Sciences* **24**, 665-674.
- 5. Barker C (2004) The alcohol hangover and its potential impact on the UK armed forces: a review of the literature on post-alcohol impairment. *Journal of the Royal Army Medical Corps* **150**, 168-174.
- 6. Barnes MJ, Mundel T & Stannard SR (2010) Acute alcohol consumption aggravates the decline in muscle performance following strenuous eccentric exercise. *Journal of Science and Medicine in Sport* **13**, 189-193.
- 7. Barnes MJ, Mundel T & Stannard SR (2011) A low dose of alcohol does not impact skeletal muscle performance after exercise-induced muscle damage. *European Journal of Applied Physiology* **111**, 725-729.
- 8. Barnes MJ, Mundel T & Stannard SR (2012) The effects of acute alcohol consumption on recovery from a simulated rugby match. *Journal of Sports Sciences* **30**, 295-304.
- 9. Berger K, Ajani UA, Kase CS *et al.* (1999) Light-to-moderate alcohol consumption and the risk of stroke among US male physicians. *New England Journal of Medicine* **341**, 1557-1564.
- 10. Bevan HR, Owen NJ, Cunningham DJ *et al.* (2009) Complex training in professional rugby players: Influence of recovery time on upper-body power output. *The Journal of Strength & Conditioning Research* **23**, 1780.
- 11. Black D, Lawson J & Fleishman S (1999) Excessive alcohol use by non-elite sportsmen. *Drug and Alcohol Review* **18**, 201-205.
- 12. Boyle P, Mahoney C & Wallace W (1994) The competitive demands of elite male field hockey. *The journal of sports medicine and physical fitness* **34**, 235.
- 13. Brancaccio P, Maffulli N & Limongelli FM (2007) Creatine kinase monitoring in sport medicine. *British Medical Bulletin* **81-82**, 209-230.
- 14. Bullock C (1990) The biochemistry of alcohol metabolism, ÄîA brief review. *Biochemical Education* **18**, 62-66.
- 15. Burke LM (1997) Nutrition for post-exercise recovery. *Australian Journal of Science and Medicine in Sport* **29**, 3.
- 16. Burke LM, Collier GR, Broad EM *et al.* (2003) Effect of alcohol intake on muscle glycogen storage after prolonged exercise. *Journal of Applied Physiology* **95**, 983-990.
- 17. Burke LM & Deakin V (2006) Clinical sports nutrition: McGraw-Hill.
- 18. Carter S (1977) The history of Rugby Union football in New Zealand, California State University, Long Beach.
- 19. Casa DJ, Armstrong LE, Hillman SK *et al.* (2000) National Athletic Trainers' Association position statement: fluid replacement for athletes. *Journal of athletic training* **35**, 212.
- 20. Clarkson PM & Hubal MJ (2002) Exercise-induced muscle damage in humans. *American Journal of Physical Medicine & Rehabilitation* **81**, S52.
- 21. Clarkson PM, Nosaka K & Braun B (1992) Muscle function after exercise-induced muscle damage and rapid adaptation. *Medicine and science in sports and exercise* **24**, 512.
- 22. Conigrave KM, Saunders JB & Reznik RB (1995) Predictive capacity of the AUDIT questionnaire for alcohol, Äêrelated harm. *Addiction* **90**, 1479-1485.
- 23. Connolly DAJ, Sayers SP & Mchugh MP (2003) Treatment and prevention of delayed onset muscle soreness. *Journal of Strength and Conditioning Research* **17**, 197-208.

- 24. Cormack SJ, Newton RU & McGuigan MR (2008) Neuromuscular and endocrine responses of elite players to an Australian rules football match. *International journal of sports physiology and performance* **3**, 359.
- 25. Coutts A, Reaburn P & Grant A (2003) Hearth rate, blood lactate concetration and estimated energy expediture in a semi-profesional rugby league team during a match: case study. *Journal of Sports Sciences* **21**, 97-103.
- 26. Crawshaw LI, Wallace H & Crabbe J (1998) Experimental Biology 1997 Symposium on Neurobiology of Thennoregulation: Role of Stress: ETHANOL, BODY TEMPERATURE AND THERMOREGULATION. *Clinical and experimental pharmacology and physiology* **25**, 150-154.
- 27. Cunniffe B, Hore AJ, Whitcombe DM *et al.* (2010) Time course of changes in immuneoendocrine markers following an international rugby game. *European Journal of Applied Physiology* **108**, 113-122.
- 28. Cunniffe B, Proctor W, Baker JS *et al.* (2009) An evaluation of the physiological demands of elite rugby union using global positioning system tracking software. *The Journal of Strength & Conditioning Research* **23**, 1195-1203.
- 29. Czarnecki DM, Russell M, Cooper ML *et al.* (1990) Five-year reliability of self-reported alcohol consumption. *Journal of studies on alcohol and drugs* **51**, 68.
- 30. Dawson B, Gow S, Modra S *et al.* (2005) Effects of immediate post-game recovery procedures on muscle soreness, power and flexiblity levels over the next 48 hours. *Journal of Science and Medicine in Sport* **8**, 210-221.
- 31. Del Boca FK & Darkes J (2003) The validity of self,Äêreports of alcohol consumption: state of the science and challenges for research. *Addiction* **98**, 1-12.
- 32. Desruelle AV, Boisvert P & Candas V (1996) Alcohol and its variable effect on human thermoregulatory response to exercise in a warm environment. *European journal of applied physiology and occupational physiology* **74**, 572-574.
- 33. Deutsch M, Kearney G & Rehrer N (2007) Time, Älmotion analysis of professional rugby union players during match-play. *Journal of Sports Sciences* **25**, 461-472.
- 34. Deutsch M, Maw G, Jenkins D *et al.* (1998) Heart rate, blood lactate and kinematic data of elite colts (under-19) rugby union players during competition. *Journal of Sports Sciences* **16**, 561-570.
- 35. Donnelly P & Young KM (1985) Reproduction and transformation of cultural forms in sport a contextual analysis of rugby. *International Review for the Sociology of Sport* **20**, 19.
- 36. Duncan DF (1997) Chronic drinking, binge drinking and drunk driving. *Psychological Reports* **80**, 681-682.
- 37. Duthie G, Pyne D & Hooper S (2003) Applied Physiology and Game Analysis of Rugby Union. *Sports Medicine* **33**, 973-991.
- 38. Eaves S & Hughes M (2003) Patterns of play of international rugby union teams before and after the introduction of professional status. *International journal of performance analysis in sport* **3**, 103-111
- 39. Edwards G, Arif A & Hodgson R (1981) Nomenclature and classification of drug related and alcohol related problems a WHO memorandum. *Bulletin of the World Health Organization* **59**, 225-242.
- 40. Eggleton MG (1942) The diuretic action of alcohol in man. *The Journal of Physiology* **101**, 172-191.
- 41. Ehlers GG, Ball TE & Liston L (2002) Creatine kinase levels are elevated during 2-a-day practices in collegiate football players. *Journal of athletic training* **37**, 151.
- 42. Finnigan F, Schulze D, Smallwood J *et al.* (2005) The effects of self administered alcohol induced hangover in a naturalistic setting on psychomotor and cognitive performance and subjective state. *Addiction* **100**, 1680-1689.
- 43. Fiorica V, Higgins EA, Iampietro P *et al.* (1968) Physiological responses of men during sleep deprivation. *J Appl Physiol* **24**, 167-176.

- 44. Fitzsimons M, Dawson B, Ward D *et al.* (1993) Cycling and running tests of repeated sprint ability. *Australian Journal of Science and Medicine in Sport* **25**, 82-82.
- 45. Gellert J & Teschke R (1988) The biochemistry of alcohol metabolism]. *Zeitschrift Gastroenterologie* **26**, 22.
- 46. Gill JS (2002) Reported levels of alcohol consumption and binge drinking within the UK undergraduate student population over the last 25 years. *Alcohol and Alcoholism* **37**, 109-120.
- 47. Gill N, Beaven C & Cook C (2006) Effectiveness of post-match recovery strategies in rugby players. *British journal of sports medicine* **40**, 260-263.
- 48. Gillespie J (1967) Vasodilator properties of alcohol. British medical journal 2, 274-277.
- 49. Graham T & Baulk K (1980) Effect of alcohol ingestion on man's thermoregulatory responses during cold water immersion. *Aviation, space, and environmental medicine* **51**, 155.
- 50. Grassi G, Somers V, Renk W *et al.* (1989) Effects of alcohol intake on blood pressure and sympathetic nerve activity in normotensive humans: a preliminary report. *Journal of hypertension. Supplement: official journal of the International Society of Hypertension* **7**, S20.
- 51. Green BS, Blake C & Caulfield BM (2011) A Comparison of Cutting Technique Performance in Rugby Union Players. *The Journal of Strength & Conditioning Research* **25**, 2668.
- 52. Greenwood J (1997) Total rugby: A & C Black.
- 53. Heather N, Partington S, Partington E *et al.* (2011) Alcohol use disorders and hazardous drinking among undergraduates at English universities. *Alcohol and Alcoholism* **46**, 270.
- 54. Hillers VN & Massey L (1985) Interrelationships of moderate and high alcohol consumption with diet and health status. *The American journal of clinical nutrition* **41**, 356-362.
- 55. Hobson RM & Maughan RJ (2010) Hydration status and the diuretic action of a small dose of alcohol. *Alcohol and Alcoholism* **45**, 366.
- 56. Hofmann W & Friese M (2008) Impulses got the better of me: alcohol moderates the influence of implicit attitudes toward food cues on eating behavior. *Journal of Abnormal Psychology* **117**, 420.
- 57. Howland J, Rohsenow DJ, Allensworth Davies D *et al.* (2008) The incidence and severity of hangover the morning after moderate alcohol intoxication. *Addiction* **103**, 758-765.
- 58. Howland J, Rohsenow DJ & Edwards EM (2008) Are some drinkers resistant to hangover? A literature review. *Current Drug Abuse Reviews* **1**, 42-46.
- 59. Ingram J, Dawson B, Goodman C *et al.* (2009) Effect of water immersion methods on post-exercise recovery from simulated team sport exercise. *Journal of Science and Medicine in Sport* **12**, 417-421.
- 60. Iwase S, Matsukawa T, Ishihara S *et al.* (1995) Effect of oral ethanol intake on muscle sympathetic nerve activity and cardiovascular functions in humans. *Journal of the autonomic nervous system* **54**, 206-214.
- 61. Jafari Nadoushan S & Shakiba Mehrdad LMH (2008) Hydration status of adult population of yazd. JOURNAL OF SHAHID SADOUGHI UNIVERSITY OF MEDICAL SCIENCES AND HEALTH SERVICES.
- 62. Jorfeldt L & Juhlin-Dannfelt A (1978) The influence of ethanol on splanchnic and skeletal muscle metabolism in man. *Metabolism* **27**, 97-106.
- 63. Juhlin-Dannfelt A, Ahlborg G, Hagenfeldt L *et al.* (1977) Influence of ethanol on splanchnic and skeletal muscle substrate turnover during prolonged exercise in man. *American Journal of Physiology Gastrointestinal and Liver Physiology* **233**, G195-G202.
- 64. Kavouras SA (2002) Assessing hydration status. *Current Opinion in Clinical Nutrition & Metabolic Care* **5**, 519-524.
- 65. Kendrick Z, Affrime M & Lowenthal D (1993) Effect of ethanol on metabolic responses to treadmill running in well-trained men. *The Journal of Clinical Pharmacology* **33**, 136.
- 66. Knight JR, Wechsler H, Kuo M *et al.* (2002) Alcohol abuse and dependence among US college students. *Journal of studies on alcohol and drugs* **63**, 263.
- 67. Kypri K, Langley JD, McGee R *et al.* (2002) High prevalence, persistent hazardous drinking among New Zealand tertiary students. *Alcohol and Alcoholism* **37**, 457.

- 68. Lamond N & Dawson D (1999) Quantifying the performance impairment associated with fatigue. *Journal of Sleep Research* **8**, 255-262.
- 69. Lemon JIM (1993) Alcoholic hangover and performance: a review. *Drug and Alcohol Review* **12**, 299-314.
- 70. Lieber RL & Friden J (1993) Muscle damage is not a function of muscle force but active muscle strain. *Journal of Applied Physiology* **74**, 520-526.
- 71. Linde L & Bergstrome M (1992) The effect of one night without sleep on problem-solving and immediate recall. *Psychological research* **54**, 127-136.
- 72. Lorente FO, Souville M, Griffet J et al. (2004) Participation in sports and alcohol consumption among French adolescents. *Addictive behaviors* **29**, 941-946.
- 73. Lundin L, Hällgren R, Lidell C *et al.* (1986) Ethanol reduces myoglobin release during isokinetic muscle exercise. *Acta medica Scandinavica* **219**, 415-419.
- 74. Marcora SM, Staiano W & Manning V (2009) Mental fatigue impairs physical performance in humans. *Journal of Applied Physiology* **106**, 857-864.
- 75. Markovic G, Dizdar D, Jukic I *et al.* (2004) Reliability and factorial validity of squat and countermovement jump tests. *Journal of Strength and Conditioning Research* **18**, 551-555.
- 76. Martin B (1981) Effect of sleep deprivation on tolerance of prolonged exercise. *European journal of applied physiology and occupational physiology* **47**, 345-354.
- 77. Mashiko T, Umeda T, Nakaji S *et al.* (2004) Position related analysis of the appearance of and relationship between post-match physical and mental fatigue in university rugby football players. *British journal of sports medicine* **38**, 617-621.
- 78. Mc Kinney A & Coyle K (2006) Alcohol hangover effects on measures of affect the morning after a normal night's drinking. *Alcohol and Alcoholism* **41**, 54.
- 79. McLean D (1992) Analysis of the physical demands of international rugby union. *Journal of Sports Sciences* **10**, 285-296.
- 80. McLellan CP, Lovell DI & Gass GC (2011) Biochemical and Endocrine Responses to Impact and Collision During Elite Rugby League Match Play. *The Journal of Strength & Conditioning Research* **25**, 1553.
- 81. McNaughton L & Preece D (1986) Alcohol and its effects on sprint and middle distance running. *British journal of sports medicine* **20**, 56.
- 82. Meier SE, Brigham TA & Handel G (1987) Accuracy of drinkers' recall of alcohol consumption in a field setting. *Journal of Studies on Alcohol* **48**, 325-328.
- 83. Meilman PW, Stone JE, Gaylor MS *et al.* (1990) Alcohol consumption by college undergraduates: current use and 10-year trends. *Journal of Studies on Alcohol*.
- 84. Mellalieu., Trewartha G & Stokes K (2008) Science and rugby union.
- 85. Morgan D & Allen D (1999) Early events in stretch-induced muscle damage. *Journal of Applied Physiology* **87**, 2007.
- 86. Mougin F, Bourdin H, Simon-Rigaud M *et al.* (1996) Effects of a selective sleep deprivation on subsequent anaerobic performance. *International journal of sports medicine* **17**, 115-119.
- 87. Murray MM (1932) The diuretic action of alcohol and its relation to pituitrin. *The Journal of Physiology* **76**, 379-386.
- 88. Myers R (1981) Alcohol's effect on body temperature: hypothermia, hyperthermia or poikilothermia? *Brain research bulletin* **7**, 209-220.
- 89. Naimi TS, Brewer RD, Mokdad A *et al.* (2003) Binge drinking among US adults. *JAMA: The Journal of the American Medical Association* **289**, 70-75.
- 90. Neddam F (2004) Constructing masculinities under Thomas Arnold of Rugby (1828,Äì1842): gender, educational policy and school life in an early,ÄêVictorian public school. *Gender and Education* **16**, 303-326.
- 91. Nicholas C (1997) Anthropometric and physiological characteristics of rugby union football players. *Sports medicine (Auckland, NZ)* **23**, 375.

- 92. Norbert F & Arky R (1966) Effects of Alcohol on Carbohydrate Metabolism in Man. *Psychosomatic Medicine* **28**, 551-563.
- 93. Norton K, Olds T, Olive S *et al.* (1996) Anthropometry and sports performance. *Anthropometrica*, 287-364.
- 94. Nosaka K & Clarkson P (1996) Variability in serum creatine kinase response after eccentric exercise of the elbow flexors. *International journal of sports medicine* **17**, 120-127.
- 95. O'Brien CP (1993) Alcohol and sport. Sports Medicine 15, 71-77.
- 96. O'Brien CP & Lyons F (2000) Alcohol and the athlete. Sports Medicine 29, 295-300.
- 97. O'Brien KS, Ali A, Cotter JD *et al.* (2007) Hazardous drinking in New Zealand sportspeople: level of sporting participation and drinking motives. *Alcohol and Alcoholism* **42**, 376.
- 98. Olds T (2001) The evolution of physique in male rugby union players in the twentieth century. *Journal of Sports Sciences* **19**, 253-262.
- 99. Pecha T, Sweere K, Conlon K *et al.* (2007) Effects of Alcohol Consumption on the Motor Performance College,Äiaged Athletic Females. *Journal of Undergraduate Kinesiology* **3**, 1.
- 100. Peele S & Brodsky A (2000) Exploring psychological benefits associated with moderate alcohol use: a necessary corrective to assessments of drinking outcomes? *Drug and alcohol dependence* **60**, 221-247.
- 101. Peretti-Watel P, Beck F & Legleye S (2002) Beyond the U-curve: the relationship between sport and alcohol, cigarette and cannabis use in adolescents. *Addiction* **97**, 707-716.
- 102. Peters T, Nikolovski S, Raja G *et al.* (1996) Ethanol acutely impairs glycogen repletion in skeletal muscle following high intensity short duration exercise in the rat. *Addiction Biology* **1**, 289-295.
- 103. Poulsen MB, Jakobsen J, Aagaard N *et al.* (2007) Motor performance during and following acute alcohol intoxication in healthy non-alcoholic subjects. *European Journal of Applied Physiology* **101**, 513-523.
- 104. Powell KE & Pratt M (1996) Physical activity and health. Bmj 313, 126-127.
- 105. Prat G, Adan A & Sanchez-Turet M (2009) Alcohol hangover: a critical review of explanatory factors. *Human Psychopharmacology: Clinical and Experimental* **24**, 259-267.
- 106. Proske U & Morgan D (2001) Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. *The Journal of Physiology* **537**, 333-345.
- 107. Quarrie K, Feehan M, Waller AE *et al.* (1996) The New Zealand Rugby Injury and Performance Project: alcohol use patterns within a cohort of rugby players. *Addiction* **91**, 1865-1868.
- 108. Quarrie K, Handcock P, Toomey M *et al.* (1996) The New Zealand rugby injury and performance project. IV. Anthropometric and physical performance comparisons between positional categories of senior A rugby players. *British journal of sports medicine* **30**, 53-56.
- 109. Quarrie K & Hopkins W (2007) Changes in player characteristics and match activities in Bledisloe Cup rugby union from 1972 to 2004. *Journal of Sports Sciences* **25**, 895-903.
- 110. Quarrie K & Wilson B (2000) Force production in the rugby union scrum. *Journal of Sports Sciences* **18**, 237-246.
- 111. Rehm J, Room R, Graham K *et al.* (2003) The relationship of average volume of alcohol consumption and patterns of drinking to burden of disease: an overview. *Addiction* **98**, 1209-1228.
- 112. Reilly T, Bangsbo J & Franks A (2000) Anthropometric and physiological predispositions for elite soccer. *Journal of Sports Sciences* **18**, 669-683.
- 113. Rimm EB, Klatsky A, Grobbee D *et al.* (1996) Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine, or spirits? *Bmj* **312**, 731-736.
- 114. Roberts KE (1963) Mechanism of dehydration following alcohol ingestion. *Archives of Internal Medicine* **112**, 154.
- 115. Roberts SP, Trewartha G, Higgitt RJ *et al.* (2008) The physical demands of elite English rugby union. *Journal of Sports Sciences* **26**, 825-833.
- 116. Roehrs T, Yoon J & Roth T (1991) Nocturnal and next,Äêday effects of ethanol and basal level of sleepiness. *Human Psychopharmacology: Clinical and Experimental* **6**, 307-311.

- 117. Rogers MA, Stull GA & Apple FS (1985) Creatine kinase isoenzyme activities in men and women following a marathon race. *Medicine and science in sports and exercise* **17**, 679.
- 118. Room R, Babor T & Rehm J (2005) Alcohol and public health. The lancet 365, 519-530.
- 119. Rowland B, Allen F & Toumbourou JW (2011) Impact of alcohol harm reduction strategies in community sports clubs: Pilot evaluation of the good sports program. *Health Psychology*, No Pagination Specified.
- 120. Rundell OH, Lester BK, Griffiths WJ *et al.* (1972) Alcohol and sleep in young adults. *Psychopharmacology* **26**, 201-218.
- 121. Saino K, Leino T, Huttunen MO *et al.* (1976) Electroencephalographic changes during experimental hangover. *Electroencephalography and Clinical Neurophysiology* **40**, 535-538.
- 122. Scott AC, Roe N, Coats AJS *et al.* (2003) Aerobic exercise physiology in a professional rugby union team. *International journal of cardiology* **87**, 173-177.
- 123. Shelmet JJ, Reichard GA, Skutches CL *et al.* (1988) Ethanol causes acute inhibition of carbohydrate, fat, and protein oxidation and insulin resistance. *Journal of Clinical Investigation* **81**, 1137.
- 124. Shirreffs SM (2003) The optimal sports drink. *Schweizerische zeitschrift fur sportmedizin und sporttraumatologie* **51**, 25-30.
- 125. Shirreffs SM & Maughan RJ (1997) Restoration of fluid balance after exercise-induced dehydration: effects of alcohol consumption. *Journal of Applied Physiology* **83**, 1152.
- 126. Shirreffs SM, Taylor A, Leiper J et al. (1996) Post-exercise rehydration in man: effects of volume consumed and drink sodium content. *Medicine & Science in Sports & Exercise* 28, 1260-1271.
- 127. Sirotic AC, Coutts AJ, Knowles H et al. (2009) A comparison of match demands between elite and semi-elite rugby league competition. *Journal of Sports Sciences* 27, 203-211.
- 128. Smart D, Gill ND, Beaven CM *et al.* (2008) The relationship between changes in interstitial creatine kinase and game-related impacts in rugby union. *British journal of sports medicine* **42**, 198-201.
- 129. Souissi N, Sesboue B, Gauthier A et al. (2003) Effects of one night's sleep deprivation on anaerobic performance the following day. European Journal of Applied Physiology 89, 359-366.
- 130. Stephens R, Ling J, Heffernan TM *et al.* (2008) Review: A review of the literature on the cognitive effects of alcohol hangover. *Alcohol and Alcoholism* **43**, 163.
- 131. Suter P & Schutz Y (2008) The effect of exercise, alcohol or both combined on health and physical performance. *International Journal of Obesity* **32**, S48-S52.
- 132. Suzuki M, Umeda T, Nakaji S *et al.* (2004) Effect of incorporating low intensity exercise into the recovery period after a rugby match. *British journal of sports medicine* **38**, 436-440.
- 133. Swift R & Davidson D (1998) Alcohol hangover. Alcohol Health Res World 22, 54-60.
- 134. Taivainen H, Laitinen K, Tahtela R *et al.* (1995) Role of plasma vasopressin in changes of water balance accompanying acute alcohol-intoxification. *Alcoholism-Clinical and Experimental Research* **19**, 759-762.
- 135. Takarada Y (2003) Evaluation of muscle damage after a rugby match with special reference to tackle plays. *British journal of sports medicine* **37**, 416-419.
- 136. Thomasson HR (2002) Gender differences in alcohol metabolism. *Recent developments in alcoholism*, 163-179.
- 137. Turley A (2008) Rugby,ÄiThe Pioneer Years: Auckland, NZ: HarperCollins.
- 138. Van der Meulen J, Kuipers H & Drukker J (1991) Relationship between exercise-induced muscle damage and enzyme release in rats. *Journal of Applied Physiology* **71**, 999-1004.
- 139. Vella LD & Cameron-Smith D (2010) Alcohol, Athletic Performance and Recovery. *Nutrients* **2**, 781-789.
- 140. Verster JC (2006) Congeners and alcohol hangover: differences in severity among Dutch college students after consuming beer, wine or liquor. *Alcoholism: Clinical and Experimental Research* **30**.
- 141. Verster JC (2008) The alcohol hangover—a puzzling phenomenon. *Alcohol and Alcoholism* **43**, 124.

- 142. Verster JC, van Duin D, Volkerts ER *et al.* (2003) Alcohol hangover effects on memory functioning and vigilance performance after an evening of binge drinking. *Neuropsychopharmacology* **28**, 740-746.
- 143. Vitiello MV (1997) Sleep, alcohol and alcohol abuse. Addiction Biology 2, 151-158.
- 144. Volpe SL, Poule KA & Bland EG (2009) Estimation of prepractice hydration status of National Collegiate Athletic Association Division I athletes. *Journal of athletic training* **44**, 624.
- 145. Warren GL, Lowe DA & Armstrong RB (1999) Measurement Tools Used in the Study of Eccentric Contraction-Induced Injury. *Sports Medicine* **27**, 43-59.
- 146. Wechsler H, Davenport AE, Dowdall GW *et al.* (1997) Binge drinking, tobacco, and illicit drug use and involvement in college athletics: A survey of students at 140 American colleges. *Journal of American College Health*.
- 147. Wetzler MJ, Akpata T, Laughlin W *et al.* (1998) Occurrence of cervical spine injuries during the rugby scrum. *The American journal of sports medicine* **26**, 177.
- 148. Xu D, Heng J & Palmer T (1993) The mechanism (s) of the alcohol-induced impairment in glycogen synthesis in oxidative skeletal muscles. *Biochemistry and molecular biology international* **30**, 169.
- 149. Yeomans MR (2004) Effects of alcohol on food and energy intake in human subjects: evidence for passive and active over-consumption of energy. *British Journal of Nutrition* **92**, 31-34.
- 150. Ylikahri R, Leino T, Huttunen M *et al.* (1976) Effects of Fructose and Glucose on Ethanol,Induced Metabolic Changes and on the Intensity of Alcohol Intoxication and Hangover. *European Journal of Clinical Investigation* **6**, 93-102.
- 151. Yoda T, Crawshaw LI, Nakamura M et al. (2005) Effects of alcohol on thermoregulation during mild heat exposure in humans. Alcohol 36, 195-200.
- 152. Yoda T, Crawshaw LI, Saito K *et al.* (2008) Effects of alcohol on autonomic responses and thermal sensation during cold exposure in humans. *Alcohol* **42**, 207-212.
- 153. Young K (1988) Performance, control, and public image of behavior in a deviant subculture: The case of rugby. *Deviant behavior* **9**, 275-293.
- 154. Yusko DA, Buckman JF, White HR *et al.* (2008) Alcohol, tobacco, illicit drugs, and performance enhancers: A comparison of use by college student athletes and nonathletes. *Journal of American College Health* **57**, 281-290.
- 155. Zachwieja J, Costill D, Pascoe D et al. (1991) Influence of muscle glycogen depletion on the rate of resynthesis. *Medicine & Science in Sports & Exercise* **23**, 44.
- 156. Zuliani U, Bonetti A, Franchini D *et al.* (1985) Effect of boxing on some metabolic indices of muscular contraction. *Int J Sports Med* **6**, 234-236.

Appendices



Participant Information Sheet

Project title: The effects of a carbohydrate sports drink on recovery following a competitive rugby match

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My name is Chris Prentice and as part of my MPhil (Sport and Exercise) I am investigating the effects of a carbohydrate sports drink in comparison with standard behaviour following a competitive game of rugby. I would like to invite you to take part in my study, which is on a voluntary basis and you have the right to pull out or ask questions at any time.

Why are we doing this study?

In sport such as rugby union, optimal postgame recovery strategies are often practised with inappropriate behaviour such as the consumption of large quantities of alcohol, disruption of appropriate nutritional practice and/or disregard for sleep.

Earlier work in this field has looked primarily at the effects of moderate alcohol consumption on strenuous eccentric exercise and simulated rugby performance with relatively mixed results. The unique nature of competitive rugby union results in both fatigue as well as a relatively high level of muscle damage. The overall effect of post game behaviour on recovery from a rugby game would therefore only truly become clear following a game played under competitive conditions.

Published work looking at the effect of post-match rugby behaviour has used primarily laboratory based methods such as monitoring recovery following match simulation rather than following behaviour after an actual competitive game.

What is the aim of this study?

The aim of this project is to look at the difference between a controlled post rugby game behaviour intervention and standard post match behaviour within New Zealand.

If I agree to take part, what will I be asked to do?

Time Commitments

You will be asked to come into the laboratory on 5 occasions.

The first session will involve a familiarisation to the experimental protocol. This familiarisation will take place midweek and will take approximately 1hr 30 mins.

Session 2 will take place on Saturday immediately following your game of rugby and will involve collection of blood and urine. This process will take approximately 30 mins.

Players from the control condition will then be left to undergo standard post game behaviour whereas those in the test group will be requested to report to the lab at 9pm for a standardized behaviour intervention involving a CHO sports drink, controlled meal and movie, they will then be driven home and required to go straight to bed. This intervention will take 3 hours.

On the following three mornings (12,36 and 60 hours after exercise) you will be picked up from home and driven to the laboratory to undergo repeat measures of venous blood, urine, repeated sprint ability, lower limb explosive power and whole body isometric strength. You will also be asked to fill out both a 24 hr diet recall questionnaire as well as a 24 hr behaviour recall questionnaire. No exercise will be allowed to be undertaken in the 60 hours following the competition match with no alcohol consumption allowed over this time. Each of these three testing periods will last approximately 1 hour.

Exercise Testing

During testing of rugby specific measures, 3 tests will be used:

Phosphate Decrement Test (Morton, 1986)

The phosphate decrement test will be testing the participants repeated sprint ability. The test will consist of 6*40m Sprints starting every 30s and performance will be monitored using electronic timing lights.

Countermovement Jump ⁷⁵

The countermovement jump will be performed three times on a force platform with the average height for each testing being used to test lower limb explosive power

Maximal Isometric Dead Lift

The maximal isometric dead lift is a strength measurement of the back and legs. The test will be carried out using a platform dynamometer, with the participant standing on the platform performing two maximal efforts keeping their back straight and their knees bent at approximately 30 degrees.

What are my rights?

- You can ask questions on any aspect of the project at any time, and we will do our best to answer them to your satisfaction.
- As a participant in the study you will provide information on the understanding that your name will not be used unless you give permission to the researcher.

- You have the right to view your own data at any stage and have it explained to you.
- You will also be given access to a summary of the project findings when it is concluded
- You can withdraw from the project at any time, without giving any reason and without penalty.

What about compensation for injury?

If physical injury results from your participation in this study, you should visit a treatment provider to make a claim to ACC as soon as possible. ACC cover and entitlements are not automatic and your claim will be assessed by ACC in accordance with the Injury Prevention, Rehabilitation and Compensation Act 2001. If your claim is accepted, ACC must inform you of your entitlements, and must help you access those entitlements. Entitlements may include, but not be limited to, treatment costs, travel costs for rehabilitation, loss of earnings, and/or lump sum for permanent impairment. Compensation for mental trauma may also be included, but only if this is incurred as a result of physical injury.

Am I eligible?

Although voluntary, your participation will also be confirmed on criteria relating to health and safety; namely, in this study we are seeking healthy, moderately active (exercise a minimum of 3 days per week) males. For health/safety reasons, you should **not** participate if any of the following apply to you:

- You have any known heart or cardiovascular condition or if a member of your family died below the age of fifty (50) as a result of a heart condition.
- You have (a) condition(s) that could be made worse by exercise.
- You have ever had an injury or any medical condition that you think may affect your ability to sense pain or discomfort.
- You are taking prescribed medication.
- You have any other reason to consider that you are not in good health and of average, or better than average, fitness.
- You have previously had to seek medical advice after exercising in cold conditions.
- You have hemorrhoids, diarrhea or have undergone rectal surgery.

All data obtained from this study will be kept strictly confidential. Data will be identified as a code only. Results will be made available to you at the completion of the study.

If you are interested in taking part:

Contact:

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Pre-Exercise Health Screening Questionnaire

Name:	 	 	
Address:			
Phone:	 		
Age:			

This questionnaire has been designed to identify the small number of persons (15-69 years of age) for whom physical activity might be inappropriate. The questions are based upon the Physical Activity Readiness Questionnaire, originally devised by the British Columbia Dept of Health (Canada), as revised by Thomas *et al.* (1992) and Cardinal *et al.* (1996), with the added requirements of the Massey University Human Ethics Committee. The information provided by you on this form will be treated with the strictest confidentiality.

You should be aware that even amongst healthy persons who undertake regular physical activity there is a risk of sudden death during exercise. Though extremely rare, such cases can occur in people with an undiagnosed heart condition. If you have any reason to suspect that you may have a heart condition that will put you at risk during exercise, you should seek advice from a medical practitioner before undertaking an exercise test.

Please read the following questions carefully. If you have any difficulty, please advise the exercise specialist who is conducting the exercise test. Please answer all of the following questions by ticking only <u>one</u> box for each question:

1.	Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?
	Yes No
2.	Do you feel a pain in your chest when you do physical activity? Yes No
3.	In the past month have you had chest pain when you were not doing physical activity? Yes No No
4.	Do you lose your balance because of dizziness or do you ever lose consciousness? Yes No
5.	Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition? Yes No
6.	Do you have a bone or joint problem that could be made worse by vigorous exercise? Yes No
7.	Do you know of any other reason why you should not do physical activity?

	Yes	No	
8.	Have any immediate a	family had heart problems prior to the ag	ge of 60?
9.	Have you been hospit	alized recently?	
	Yes	No	
10	. Do you exercise 3 or	more days per week?	
	Yes	No	
I have	read, understood and co	impleted this questionnaire.	
Signatu	ıre:		Date:

References

Thomas S, Reading J and Shephard RJ. (1992) Revision of the Physical Activity Readiness Questionnaire (PAR-Q). *Can J Sport Sci* 17(4): 338-345.

Cardinal BJ, Esters J and Cardinal MK. (1996) Evaluation of the revised physical activity readiness questionnaire in older adults. *Med Sci Sports Exerc* 28(4): 468-472



The effects of a carbohydrate sports drink on recovery following a competitive rugby match

PARTICIPANT CONSENT FORM - INDIVIDUAL

I have read the Information Sheet and have had the details of the study	explained to me. My	
questions have been answered to my satisfaction, and I understand that I ma	y ask further questions	
at any time.		
I agree to participate in this study under the conditions set out in the Information	n Sheet.	
Signature:	Date:	
Full Name - printed		



24 Hr Behaviour Recall

Name:	Date:	

	Amount
Alcohol	
Sleep	
Physical Activity	

24 Hour Diet Recall

Name	Date

Meal/Snack	Food item	Amount
David Cont		7
Breakfast		
During		
Morning		
Lunch		
During		
Afternoon		
Evening		
Meal		
Evening		
Snack		