Sports Med (2014) 44:909–919
DOI 10.1007/s40279-014-0192-8

REVIEW ARTICLE

Alcohol: Impact on Sports Performance and Recovery in Male Athletes

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Published online: 19 April 2014

Abstract Alcohol is the most commonly used recreational drug globally and its consumption, often in large volume, is deeply embedded in many aspects of Western society. Indeed, athletes are not exempt from the influence alcohol has on society; they often consume greater volumes of alcohol through bingeing behaviour compared with the general population, yet it is often expected and recommended that athletes abstain from alcohol to avoid the negative impact this drug may have on recovery and sporting performance. While this recommendation may seem sensible, the impact alcohol has on recovery and sports performance is complicated and depends on many factors, including the timing of alcohol consumption post-exercise, recovery time required before recommencing training/competition, injury status and dose of alcohol being consumed. In general, acute alcohol consumption, at the levels often consumed by athletes, may negatively alter normal immunoendocrine function, blood flow and protein synthesis so that recovery from skeletal muscle injury may be impaired. Other factors related to recovery, such as rehydration and glycogen resynthesis, may be affected to a lesser extent. Those responsible for the wellbeing of athletes, including the athlete themselves, should carefully monitor habitual alcohol consumption so that the generic negative health and social outcomes associated with heavy alcohol use are avoided. Additionally, if athletes are to consume alcohol after sport/exercise, a dose of approximately 0.5 g/kg body weight is unlikely to impact most aspects of recovery and may therefore be recommended if alcohol is to be consumed during this period.

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1 Introduction

Globally, alcohol is the most commonly used psychoactive drug; it is estimated that each adult (15+ years of age) consumes, on average, approximately 4.3 L of pure alcohol per year. The use of alcohol is widespread, with Europe, Australia, Argentina, North America and New Zealand having the highest levels of annual consumption per adult [1]. The World Health Organization (WHO) [1] classifies safe or low levels of alcohol consumption as four standard drinks per day for males and two standard drinks per day for females, with a standard drink being classified as any beverage containing 8 g of ethanol. However, the definition of a standard drink differs between countries; for example, in New Zealand and Australia a standard drink contains 10 g of alcohol whereas in Japan a standard drink contains 19.75 g of alcohol. The majority of countries listed in a review of international drinking guidelines defined a standard drink as containing between 8 and 14 g of alcohol [2]. Above the safe levels recommended by the WHO, alcohol consumption becomes hazardous (4–6 or 2–4 standard drinks per day for males and females, respectively). Even larger amounts are classified as harmful and may significantly increase the risk of negative mental and physical health issues, such as a range of cancers, hypertension, stroke and injuries related to violence [3, 4]. In addition to hazardous, chronic alcohol consumption, heavy acute episodic or binge drinking, classified as the consumption of 60 g of alcohol in a single drinking episode, is associated with significant physical, psychological and social harm. Approximately 16.5 % of the world's population are thought to participate in heavy episodic drinking on a weekly basis [1].

While acute and chronic misuse of alcohol are common place in the general population [1], the athletic/sporting population is not exempt from such behaviour; and while

recommendation for abstinence from alcohol for athletes is common, this recommendation may not be overly practical due to the role of alcohol in society [5]. Athletes may not consider alcohol as harmful in the same way they consider other recreational drugs [6]; therefore, in order to educate athletes, and others, a full understanding of the implications alcohol consumption may have on sporting performance, recovery from exercise and, perhaps more importantly, general health, is required. It is therefore the aim of this review to outline the influence alcohol has on sports performance and recovery, while additionally highlighting aspects related to health that may be altered by acute alcohol consumption. Finally, recommendations will be made for safe, appropriate use of alcohol in the sporting environment. Due to a dearth of information relating to the effects of alcohol specifically on female sportspeople, this review will focus primarily on the impact alcohol has on the male athlete; however, many of the effects of alcohol are likely to affect both sexes equally.

2 Methods

A search was performed using the Web of Science, MEDLINE, PubMed, ScienceDirect and GoogleScholar search engines to investigate the drinking habits of athletes and the acute effects alcohol may have on factors associated with sports/exercise performance and recovery. Articles considered for inclusion in this review had to address at least one of the following topics: alcohol use by athletes or sportspeople; the influence of alcohol, at physiologically relevant doses, on post-exercise recovery and/or factors associated with recovery, in vivo or in vitro; the impact of acute alcohol consumption on physical and/or exercise performance. The key search terms 'alcohol' OR 'ethanol' were used in combination with the following: 'athletes', 'sportspeople', 'performance', 'exercise', 'sport', 'recovery', 'immune function', 'inflammation', 'skeletal muscle injury', 'exercise induced muscle damage', 'protein synthesis', 'hormone', 'hydration', 'glycogen repletion', blood flow'. Additional manual searches were carried out from the reference lists of recovered articles. Due to the extensive nature and variety of topics covered by the search, there was no limit to the search period. As the drinking habits of some athletes may not be truly represented within the reviewed literature, anecdotal evidence is presented to provide an indication of occasional, 'extreme' alcohol consumption that may occur in some sports.

3 Alcohol Use in the Sporting Population

Much of the information regarding alcohol use by sportspeople comes from large surveys of US college students. These studies consistently reveal that students involved or partly involved in sports not only drink alcohol more regularly, they also have a higher tendency to drink to get drunk and therefore binge on alcohol more often than those students with no involvement in sports [7–9]. Similarly, while French sporting students reported drinking less frequently than their peers in the general population, they reported more frequent episodes of alcohol intoxication. A strong relationship was evident between sex and frequency of intoxication, with males more likely to be intoxicated on ten or more occasions in a year compared with the females surveyed [10].

It is worth noting that, in many countries, the university population as a whole exhibits greater rates of hazardous alcohol consumption than occurs in the general population [11–13]. This suggests that the addition of sporting participation magnifies the hazardous drinking culture that appears to be embedded in university student life. This fact should be considered when dealing with the well-being of college athletes.

Regular, hazardous alcohol consumption has been found amongst sportspeople, both student and non-student, competing at all levels of competition, with at least half of those surveyed reporting regular hazardous, binge-drinking behaviour [14–16]. Given the rate of such behaviour in the general population is approximately 16.5 % [1] the results of these studies are cause for concern. Of note, hazardous drinking is typically most evident in males and team-sports participants [14, 17], a fact supported by the high rates of hazardous alcohol use reported by males competing in contact team sports.

Hazardous alcohol consumption has been reported amongst rugby league [18], rugby union [19, 20] and Australian Rules football [21–24] players, with these populations reporting regular, single-session alcohol consumption up to nine times the recommended, safe amount. Additionally, such behaviour is regularly reported on by the media when high-profile sportsmen, from a wide range of sporting codes, commit socially unacceptable, alcohol-related offences. Seen as reward for the hard work put in during training and the match, or as an integral part of club culture, the majority of the hazardous alcohol use reported by sportspeople occurs in the hours after the match [18, 21, 23, 24]. Additionally, anecdotal evidence suggests that team bonding and, in some sports, initiation into the club or team, typically involves the consumption of alcohol at high doses. While the majority of alcohol consumption reported by sportspeople occurs in the hours after sport, prolonged periods (2 or more days) of alcohol consumption at the conclusion of a season, or between matches and training, has been reported anecdotally and in the media. While such behaviour has not previously been acknowledged in the scientific literature, the occurrence of such behaviour and

Table 1 Studies investigating the effects of pre-exercise alcohol consumption published since the release of the American College of Sports Medicine's Position Stand on Alcohol and Sport [25]

Reference	Dose consumed	Performance measure	Effect on performance
Bond et al. [101]	0.44 ml/kg BW of 95 % ethanol	Maximal, progressive cycling test	No effect
Bond et al. [101]	0.88 ml/kg BW of 95 % ethanol	Maximal, progressive cycling test	No effect
Houmard et al. [102]	Breath alcohol below 0.5 mg/ml	5-mile treadmill time trial	No effect
Lecoultre and Schutz [27]	0.5 g/kg lean BW	60-min cycling time trial	Decrease in total work done
Kendrick et al. [28]	25 ml 10 min before and 30 min after the onset of exercise	Treadmill run at 80–85 % VO_{2max} intensity	Three of the four subjects failed to complete the allocated time
McNaughton and Preece [29]	Breath alcohol of 0.01 mg/ml and 0.1 mg/ml	100-m run	No effect
		200-m run	Increased time (dose-dependent)
		400-m run	Increased time (dose-dependent)
		800-m run	Increased time (dose-dependent)
		1,500-m run	Increased time (dose-dependent)
Poulsen et al. [30]	1.59 g and 1.48 g/kg BW for males and females respectively	Isometric and isokinetic strength	No effect

BW body weight, VO_{2max} maximal oxygen uptake

its impact on player health, safety and performance warrants attention in the future.

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

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

4 Alcohol and Sports Performance

The release of the American College of Sports Medicine (ACSM) Position Stand [25] on the use of alcohol in sports highlighted the potentially deleterious effects alcohol can have on physical performance; up until then, alcohol was often viewed as having ergogenic properties [26]. The available literature and trend in research at the time meant that the ACSM Position Stand almost exclusively addressed the effects of pre-exercise alcohol consumption on subsequent physical performance. The ACSM Position Stand [25] concludes that acute alcohol use may impair strength, power, muscular endurance, speed and cardiovascular endurance. However, no detrimental effect of alcohol on maximal oxygen uptake, heart rate, stroke volume, muscle blood flow and respiratory dynamics during exercise was reported.

Since the release of the Position Stand, much of the research into the effects of pre-exercise alcohol consumption on performance has proven inconclusive, with a number of studies reporting contradictory findings. This is most likely due to the use of different doses of alcohol, different exercise protocols and modalities (i.e. running vs. cycling) and variability between subjects for alcohol tolerance. The studies completed since the release of the ACSM Position Stand are summarised in Table 1.

Together with the conclusions of the ACSM Position Stand [25], the findings of Lecoultre and Schutz [27], Kendrick et al. [28] and McNaughton and Preece [29] strongly suggest that low to moderate doses of alcohol do not positively influence performance; rather, they are likely to decrease endurance performance. It is less clear whether alcohol use impacts measures of strength, with the Position Stand [25] reporting mixed results for measures of strength after alcohol consumption. Although alcohol may act at a number of locations important to force production within the central nervous system (CNS), the results of Poulsen et al. [30] further suggest that muscular strength is not affected even at high doses.

Given what is now known about the alcohol use of athletes, that they are more likely to consume alcohol after exercise, the findings of previous research, including the ACSM [25] Position Stand and the subsequent research outlined above, seem to have limited application to much of the sporting population. Perhaps more important and

relevant is how the consumption of alcohol after exercise alters recovery and adaptation.

5 Alcohol, Recovery and Subsequent Performance

Investigations into the effects of post-exercise alcohol use on recovery have tended to focus on factors that influence or are associated with recovery, or proxies for recovery, rather than investigating whether a return to optimal performance is impaired by post-exercise alcohol use. For example, strategies such as rehydration, restoration of energy stores and accelerated injury repair are thought to be essential if optimal recovery is to be achieved [31, 32]. Whether these indirect measures of recovery reflect a return to pre-exercise performance is debatable.

5.1 Metabolic Recovery

During strenuous exercise, liver and muscle glycogen stores may be reduced, while sweating can result in dehydration (fluid loss of 2–5 % body mass or greater) [33]. Dehydration has been shown to impair performance [34] and so adequate rehydration and restoration of electrolytes after exercise is important to ensure recovery before the next training session or event [31, 35]. Equally important to recovery is the repletion of muscle glycogen. Optimal refuelling strategies depend on the type and duration of exercise as well as the time between exercise bouts or events. It has been suggested that the best opportunity for optimising glycogen stores occurs when carbohydrate is consumed in the initial hours after exercise; after that time, glycogen storage rates decrease significantly [33]. However, in many sports this period after competition may be spent consuming alcohol instead of following correct nutritional strategies.

5.1.1 Post-Exercise Rehydration

Early studies investigating a diuretic effect of alcohol confirmed the long-held belief that the consumption of alcohol increases urinary output [36] through the inhibition of vasopressin (antidiuretic hormone) [37–39]. More recently, Hobson and Maughan [40] reported that the diuretic effect of alcohol, at least at a dose of 4 % alcohol, is dependent on hydration status, with a reduced alcohol-related diuresis seen when participants were in a hypohydrated state.

The negative effect alcohol has on the restoration of fluid balance after exercise has been confirmed by Shirreffs and Maughan [41]. A range of doses, equivalent to approximately 0, 0.24, 0.49 and 0.92 g of alcohol/kg body weight (BW), were utilised to investigate the effects of acute alcohol consumption on rehydration after dehydrating exercise. Only the highest dose was found to significantly increase urine output, reduce the recovery rate of blood volume and therefore delay recovery from the dehydrated state, suggesting that alcohol in dilute concentrations has little effect on rehydration.

The reduced diuresis [40] and lack of effect on rehydration [41] at low doses of alcohol may allow athletes to consume small volumes of alcohol, at a dose less than 0.49 g/kg BW, after exercise without negatively impacting rehydration. However, if fluid replacement is not a priority, for example if optimal performance is not required the next day, then the consumption of alcohol post-exercise in larger volumes may be acceptable, at least from a hydration stand point.

5.1.2 Post-Exercise Glycogen Resynthesis

Although the consumption of alcohol may impact hepatic gluconeogenesis [42], glucose utilisation and glucose uptake into skeletal muscle [43, 44], all of which could contribute to decreased performance if alcohol was consumed prior to exercise, it appears that alcohol has little or no effect on the resynthesis of muscle glycogen after exercise. Burke and colleagues [45] compared the effects of three recovery diets, two of which contained alcohol, on post-exercise muscle glycogen resynthesis. Consumption of alcohol (1.5 g alcohol/kg BW) in conjunction with a high carbohydrate diet had no impact on post-exercise glycogen storage compared with a diet containing no alcohol. However, an alcohol 'displacement diet' where some carbohydrate-containing food was replaced by an isocaloric amount of alcoholic beverage, was found to impair glycogen repletion. This finding is most likely due to the reduced availability of substrate for glycogen resynthesis rather than any effect alcohol has on the process of glycogenesis. This dietary scenario is similar to that faced by athletes who consume alcoholic beverages post-exercise instead of following appropriate nutritional strategies aimed at replenishing glycogen stores [45].

5.2 Recovery from Soft Tissue Injury

Injury, particularly to skeletal muscle, is a common occurrence in many sports, especially contact team sports, and may be caused through collision with other competitors, impact with the ground or through eccentric muscular action [31, 46]. From a performance point of view, the most important implication of muscle injury is a decrease in muscle function. Any loss in the ability to generate appropriate levels of force during competition or training is likely to be detrimental to the outcome of the event and may impact subsequent adaptation from training.

Therefore, optimal recovery from damage to skeletal muscle is essential if a timely return to training and/or competition is required. Additionally, if full recovery is not achieved, the likelihood of further injury to the muscle may be increased [47]. As with metabolic recovery, much of the evidence for the influence of alcohol on recovery from muscle injury is via indirect measures and association.

5.2.1 Immune Function

Under normal circumstances, the innate immune system responds to trauma by initiating a complex inflammatory response. Typically characterised by alterations in hormone and cytokine levels, this process directs phagocytosis, cell proliferation and differentiation to ensure the inflammatory stimulus is dealt with appropriately and subsequent recovery occurs [48]. Although the specific role each inflammatory molecule plays in this process is not fully understood, the actions of several key molecules are now known. Many of these inflammatory molecules are affected by acute alcohol treatment.

Acute alcohol exposure upsets the balance of normal inflammatory processes, resulting in a net shift towards an anti-inflammatory environment through selective alterations in cytokine activity [49]. A major contributor to this altered immune state is the alcohol-induced downregulation or impairment of tumour necrosis factor (TNF)-α production [50–52]. Such impairment results in a decrease in endothelial cell activation and therefore a reduction in the expression of cell adhesion molecules (CAMs), thus negatively affecting neutrophil–endothelial cell adhesion. Furthermore, without normal concentrations of TNF-α, the endothelium is unable to produce a number of pro-inflammatory molecules which usually act to magnify the inflammatory response [53]. In addition, acute alcohol treatment also inhibits interleukin (IL)-1β and IL-6 expression [54–56], further limiting the pro-inflammatory response to trauma.

While negatively affecting pro-inflammatory molecules, the presence of alcohol also increases production of anti-inflammatory molecules. The production of transforming growth factor (TGF)-β, and therefore its immuno-inhibitory action, is increased at physiologically relevant levels of alcohol [52]. Along with inhibiting cellular proliferation, increased TGF-β activity is also likely to inhibit inflammatory cytokine production and lessen the overall effectiveness of the immune response [50]. Acute alcohol-related increases in IL-10 levels augment the anti-inflammatory state, leading to inhibition of T-cell proliferation and downregulation of pro-inflammatory cytokine activity [52, 56]. Prostaglandin E_2 (PGE_2) production by monocytes is also increased after acute ethanol stimulation. Similar to the actions of TGF-β and IL-10, increased levels of PGE_2 inhibit inflammatory cytokine production, T-cell proliferation and monocyte antigen presentation capacity [57].

Essential to the early stages of the inflammatory process, neutrophils are directly affected by clinically relevant levels of alcohol. As with the effects of alcohol on the endothelium's ability to express CAMs, the expression of neutrophil surface adhesion molecules is also inhibited. Such inhibition significantly impacts neutrophil migration. As well as limiting the neutrophil's ability to move to the site of inflammation, alcohol also inhibits superoxide production, an important weapon in the process of phagocytosis [58].

In addition to these acute alterations in immune function, which may negatively affect the outcome of skeletal muscle injury, the chronic use of alcohol at harmful or hazardous levels may further compromise the immune system so that an individual's susceptibility to illness and infection is greatly increased [59]. It is possible that, when combined with acute and chronic alcohol-related changes to the immune system, the temporary immunosuppression [60] associated with exercise may further reduce an athlete's ability to deal with illness. Further research is required to understand this relationship.

5.2.2 Skeletal Muscle Blood Flow

Along with other restorative strategies aimed at limiting blood flow to the injured tissue, such as compression and contract water therapy [46, 61], abstinence from alcohol during the injured state is routinely advised. The evidence for this recommendation, however, appears to be mostly anecdotal, stemming from the known vasodilatory effect of alcohol [62]. Although, under normal circumstances, alcohol acts as a peripheral vasodilator at the skin and a vasoconstrictor at the muscle [63, 64], evidence from animal studies suggests that changes in the normal response to trauma, including elevations in vasopressin, adrenaline and noradrenaline, are inhibited when alcohol is consumed prior to trauma/injury [65]. This inability to limit blood flow to the site of injury is likely to contribute to the increased oedema that has been observed when alcohol is consumed prior to experimentally-induced trauma [66, 67]. Coupled with alterations in immune function, increased blood flow to the site of injury may increase the severity of the injury and negatively impact the rate and outcome of recovery [68].

5.2.3 Endocrine Effects

As with the innate immune system, the acute consumption of alcohol has been shown to have a profound impact on the endocrine system; a summary of the acute effects of

Table 2 The effects of alcohol on a select number of hormones, and the physiological implications of these changes

Reference	Hormone	Effect on secretion	Physiological implication
Perman [103] Ireland et al. [104]	Adrenaline	Increase	Increased heart rate, tachycardia, hypertension
Ylikahri et al. [105] Rivier and Vale [106]	Cortisol	Increase	Negatively affect reproductive function, increase blood glucose
Boileau [107]	Dopamine	Increase	May reinforce alcohol use
Gordon et al. [108]	Estrogen	Increase	May have feminising effects on males
Leppäluoto et al. [109] Prinz et al. [110]	Human growth hormone	Decrease	May impact a number of biochemical process
Marks [111]	Insulin	Increase	Hypoglycemia, if in a fasted state or if consumed with high carbohydrate food
Mendelson et al. [112] Leppäluoto et al. [109]	Luteinizing hormone	Decrease	Decrease testosterone production
Rupp et al. [113]	Melatonin	Decrease	Sleep fragmentation and disruption
Badawy [114]	Serotonin	Decrease	Aggression, dysphoria
Mendelson et al. [112] Gordon et al. [115] Badr and Bartke [116]	Testosterone	Decrease	Decreased muscle function, osteoporosis, anaemia, decreased libido, impotence, infertility, feminisation
Kleeman et al. [39] Eisenhofer and Johnson [37]	Vasopressin	Decrease	Increase in urine output

alcohol on a select number of hormones is presented in Table 2. Alcohol detrimentally impacts normal hormonal balance so that a range of factors, including sleep quality, mood, metabolism and cardiovascular function, may all be affected during and/or after alcohol consumption. Importantly for males, when consumed acutely in large doses (1.5 g alcohol/kg BW), alcohol has a negative effect on testosterone production which, together with an increased conversion rate of testosterone and androstenedione to their respective estrogens, leads to feminising effects such as gynecomastia and testicular atrophy [69]. Additionally, a decrease in testosterone may also impact skeletal muscle function, bone density and red blood cell numbers [70]. While large doses of alcohol negatively affect testosterone levels, Sarkola and Eriksson [71] found that a low dose of alcohol (0.5 g/kg BW) has the opposite effect on testosterone while additionally reducing androstenedione levels. Sarkola and Eriksson [71] proposed that the change in the ratio of nicotinamide adenine dinucleotide (NAD^+) and its reduced form, nicotinamide adenine dinucleotide plus hydrogen (NADH), in the liver during alcohol metabolism may result in a decreased rate of testosterone oxidation, thus leading to the observed increase in testosterone and decrease in androstenedione.

Anabolic hormones play an essential role in wound healing, in particular protein synthesis and skeletal muscle regeneration [72, 73]. Therefore, it is possible that the effect of alcohol on anabolic hormone secretion may negatively impact this phase of the recovery and adaptive process. When consumed after exhaustive aerobic exercise, a dose of 1.5 g alcohol/kg BW prolongs the inhibitory effect alcohol has on testosterone [74]. However, when consumed after resistance exercise, a lower dose (0.83 g alcohol/kg BW) of alcohol alters elevations in cortisol, thus extending the post-exercise catabolic state, while at the same time having no impact on testosterone [75]. Intriguingly, when consumed after resistance exercise, a dose of 1.09 g alcohol/kg BW results in an increase in free and total testosterone [76], a response opposite to that seen in the absence of resistance exercise. While the findings of Vingren et al. [76] suggest alcohol consumption has the potential to aid in the recovery and adaptation after resistance exercise, until further research is carried out it is premature to consider alcohol as a post-exercise tonic for enhancing muscular recovery.

The often contradictory responses to alcohol consumption highlight the difficulty and complexity associated with understanding the effects this drug has on normal physiological function as the outcome and mechanisms behind any given observation may be dependent on dose, sex, age and previous level of habitual alcohol use.

5.2.4 Protein Synthesis

Acute alcohol consumption may impact skeletal muscle recovery via its effects on protein synthesis. Both whole body and skeletal muscle protein synthesis, but not degradation [77], have been shown to be altered by acute

alcohol treatment in rats, with skeletal muscle protein synthesis decreasing by as much as 75 % for at least 24 h post alcohol treatment [77–79]. In muscle, type II fibres appear to be more susceptible to alcohol than type I fibres [77, 80]. Similarly, in vitro studies have shown a decrease in protein synthesis through the alcohol- and acetaldehyde-mediated impairment of insulin-like growth factor (IGF)-1 and insulin [81]. Whether these effects are seen in humans at physiological circulating alcohol concentrations is unknown. To put these findings into perspective, the doses used in the majority of studies investigating alcohol's impact on protein synthesis is 75 mmol alcohol/kg BW [77–79]. This translates to 3.46 g/kg BW, which is equivalent to 241.9 g of alcohol, or 24 standard drinks, for a 70 kg individual. While this level of alcohol consumption is not unheard of amongst certain populations, including team sportspeople, ethically it may be difficult to identify whether the same findings hold true in vivo in humans. Recently, Parr et al. [82] investigated the effects of 1.5 g alcohol/kg BW, equivalent to 12 ± 2 standard drinks, on muscle protein synthesis after a bout of concurrent training. Even when nutritional status was optimised in the post-exercise period, alcohol consumption was found to impair normal post-exercise muscle protein synthesis. This finding suggests alcohol consumption, post-exercise, may detrimentally impact recovery and adaptation. Given the importance of protein synthesis in recovery and adaptation after strenuous exercise [83], there is considerable scope for further research in this area.

5.2.5 *Impact on Exercise-Induced Muscle Damage*

The success of interventions aimed at improving recovery rates in a sport/medical setting is difficult to measure due to the random nature of injury, timing of presentation to a medical practitioner, and compliance by the patient in the days after assessment [84]; therefore, the use of exercise-induced muscle damage (EIMD) has been used as a model of skeletal muscle injury to investigate a number of recovery modalities [85–87]. The use of EIMD provides a level of control and repeatability that allows for direct assessment of the efficacy of a treatment on functional measures of recovery, rather than indirect measures that may or may not influence recovery such as those discussed in the previous section.

As with the majority of other studies investigating the interaction between alcohol and exercise, the first study to examine the combination of acute alcohol consumption and damage to skeletal muscle, via EIMD, had subjects consume alcohol prior to eccentric exercise. Although this scenario replicates the common occurrence of consuming alcohol and then, as a result of intoxication, sustaining an injury, its application to the sporting environment where alcohol is generally consumed after exercise may be limited. Clarkson and Reichsman [88] had subjects drink either a beverage containing 0.8 g alcohol/kg BW or a non-alcoholic control beverage 35 min prior to performing 50 maximal eccentric contractions of the elbow flexor muscles. No difference between treatments was evident in measures of creatine kinase, muscle soreness, isometric strength or range of motion.

Investigating the opposite and perhaps more realistic scenario for many sports, Barnes et al. [89] had subjects consume either 1 g alcohol/kg BW or an isovolumetric, isoenergetic non-alcoholic beverage 30 min after completing 300 eccentric contractions of the quadriceps. A significant difference in measures of isometric and isokinetic torque was found between the two treatments, with greater decrements in muscle function observed with alcohol. Further studies by Barnes et al. [90, 91] confirmed this initial finding that 1 g of alcohol/kg BW, but not 0.5 g/kg BW [92], magnifies the loss in force typically associated with strenuous eccentric exercise. The mechanisms behind the effects of alcohol on muscle function, when combined with EIMD, are unknown; however, given the apparent negative influence of alcohol on immunoendocrine function, blood flow and protein synthesis, a combination of these factors, along with alterations in voluntary activation of skeletal muscle [91], may contribute to the greater force loss described by Barnes et al. [89–91].

The divergent findings of Clarkson and Reichsman [88] and Barnes et al. [89–91] may be due to several factors, including the magnitude of the damage induced by exercise (50 vs. 300 contractions), the muscle mass exercised (elbow flexors vs. knee extensors), the dose used (0.8 g vs. 1 g/kg BW) and the timing of alcohol consumption (pre- vs. post-exercise). It is not known whether the dose used by Clarkson and Reichsman [88] would impact recovery if consumed after exercise; indeed, more research is required to fully understand the dose response of alcohol impact on EIMD.

5.3 Recovery from Contact Team Sport

When significant muscle damage is not present it appears that alcohol consumed after strenuous exercise does not impact physical recovery and performance in the days after the exercise bout/drinking episode. Using contact team-sports as a model, Barnes et al. [93] found no effect of 1 g of alcohol/kg BW on recovery of strength, speed, power and agility in the days after a simulated rugby match, compared with a non-alcoholic control beverage. Additionally, this dose did not alter the endocrine and inflammatory responses to the game simulation. This finding is supported by the work of Murphy et al. [94] who also

reported no effect of the same dose of alcohol on recovery when consumed after a competitive game of rugby league.

Careful control over diet and alcohol consumption in these studies provides scientific rigour necessary to further our understanding of the impact of alcohol in the post-exercise/competition environment; however, the doses used and control of the social environment does not provide a realistic setting to fully understand how alcohol is consumed and how it may subsequently influence recovery and performance after sport/exercise. To avoid such limitations, Prentice et al. [95] used a naturalistic approach to observe 'normal' alcohol use by a group of rugby players after a competitive game. The effects of this behaviour on hydration, strength, power and repeated sprint ability were compared with a group of players undertaking a controlled, optimal recovery strategy. Subjects in this study reported consuming ~20 standard drinks in a single drinking episode after a game; however, this behaviour had no impact on hydration status and measures of anaerobic performance in the 2 days after the game. A lack of change in anaerobic performance the day after alcohol consumption has previously been described by O'Brien [96]; however, whether the hazardous alcohol consumption reported by Prentice et al. [95] impacts aerobic exercise, as previously reported by O'Brien [96], is currently unknown.

Alcohol consumption may be associated with decreased quality and duration of sleep, either directly through the impact of alcohol on the sleep process [97] or indirectly as a result of staying awake longer while consuming alcohol at night. While sleep deprivation may not alter anaerobic performance under normal circumstances [98, 99], partial sleep deprivation after a rugby league match has recently been shown to negatively affect lower body power output the next day [100]. However, the hazardous alcohol use reported by Prentice et al. [95] was associated with a significant decrease in sleep hours, suggesting that, for anaerobic activities, the combination of heavy alcohol consumption and reduced sleep does not impact performance. More research is required to better understand the interaction between alcohol, sleep, and physical and cognitive performance.

6 Conclusions and Future Directions

Alcohol use by athletes often occurs during the post-competition period at hazardous levels in excess of those seen in the general population. Such behaviour may increase the athlete's risk of alcohol-related illness and other harm while also detrimentally altering normal endocrine and immune function. Particularly important for males, in both athletic and general populations, is the reduced production of testosterone and subsequent effects on body composition, protein synthesis and muscular adaptation/regeneration; these effects are likely to inhibit recovery and adaptation to exercise. Low doses of alcohol, approximately 0.5 g/kg BW, post-exercise are unlikely to be detrimental to repletion of glycogen, rehydration and muscle injury; however, the effects of alcohol are dependent on the timing of consumption, nutritional status and the priority given to optimal rates of recovery. Higher doses, around 1 g/kg BW, should be avoided if injury to skeletal muscle has occurred. While very high, hazardous doses of alcohol consumed after strenuous exercise may not directly impact performance in the days after exercise, such bingeing behaviour is associated with long-term physical, psychological and social harm [5] and should therefore be avoided; it should be remembered that alcohol is a poison and as such should be treated as one. While less likely to occur than drinking large volumes of alcohol after sports, the consumption of even low doses of alcohol prior to athletic endeavour should be discouraged due to the ergolytic effects of alcohol on endurance performance.

Future research should investigate the effects alcohol has on recovery and performance in female athletes as very little information currently exists on this topic. Further research into the effects of both chronic and acute alcohol consumption, at levels relevant to the athletic population, on adaptation to exercise, particularly protein synthesis, and immune function are required so a more complete recommendation can be made on the safe use of alcohol by athletes.

Acknowledgments The author would like to acknowledge the support, guidance and contributions of Dr. Toby Mündel and Professor Stephen R. Stannard towards the development of this topic of research. The author has no conflicts of interest that are directly relevant to the content of this manuscript.

No sources of funding were used to assist in the preparation of this review.

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