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Alcohol and football

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Abstract
The use of alcohol is often intimately associated with sport, and the association is particularly strong in football. As well as providing a source of energy, alcohol (ethanol) has metabolic, cardiovascular, thermoregulatory, and neuromuscular actions that may affect exercise performance. Its actions on the central nervous system, however, result in decrements in skill and behavioural changes that may have adverse effects on performance. There is also evidence of dose-dependent decrements in aerobic capacity. Although the mechanisms are not well understood, the aftermath of alcohol use (hangover) may also adversely affect performance for many hours after intoxication. Alcohol intoxication may adversely affect the player's dietary choices by displacing carbohydrate from the diet at a time when restoration of glycogen stores should be a priority.

Keywords: *Ethanol, intoxication, exercise, sport, soccer*

Introduction

Alcohol is an energy-supplying nutrient that forms a small but important part of the normal dietary intake of a large part of the world's population. Unlike the other macronutrients, however, alcohol is not an essential part of the human diet, and is absent from the diets of a significant portion of the population. Alcohol intake may be measured in grams or millilitres of ethanol, or in units of alcohol: each unit of alcohol in the UK contains approximately 8 g (10 ml) of ethanol. The UK Department of Health recommends that adult men should not consume more than 3 – 4 units of alcohol per day and women should not consume more than 2 – 3 units daily. In the USA, however, a standard drink delivers about 12 – 14 g of alcohol, and the US Department of Agriculture recommends that men should not drink more than one or two drinks per day and that women should not exceed one drink per day.

Alcohol intake and drinking behaviours vary widely between countries and cultures, and data from the UK will be used here for illustrative purposes. The National Diet and Nutrition Survey (NDNS) of adults aged 19 – 64 years living in private households in Great Britain, carried out between July 2000 and June 2001, found that, among men and women who drank alcoholic beverages, alcohol contributed on average 8.1% and 5.7% respectively of their total energy intake (National Diet and Nutrition Survey, 2003). Mean daily alcohol intake was 21.9 g for men and 9.3 g for women: if those who consumed no alcohol were excluded, the figures were 27.2 g for men and 13.5 g for women. Data from the NDNS suggest that, on average, men were consuming 21.1 units per week and women were consuming 9.0 units per week. The NDNS also highlighted that a number of men and women were drinking more than the UK Department of Health recommended guidelines for safe drinking. Among those people who consumed alcohol, 38% of men and 24% of women had more than the maximum, with younger age groups consuming more units per week than older age groups. It was estimated that 5% of adult men and 10% of adult women in the UK consume no alcohol (National Diet and Nutrition Survey, 2003). About one in 13 adults in the UK, however, is dependent on alcohol (Ibrahim & Gilvarry, 2005).

Alcohol-containing foods – most of which are in liquid form – have a relatively high energy content (29 kJ [7 kcal] per gram of alcohol), but are generally poor in other nutrients. As with other components of the diet, intake of alcohol has implications for health

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and for sports performance, and both acute and chronic alcohol intake in large amounts can be a reason for concern. There is compelling evidence that regular ingestion of moderate amounts of alcohol may confer some health benefits; in particular, there seems to be a 25–40% reduction in the risk of adverse cardiovascular events (Goldberg, Mosca, Piano, & Fisher, 2001). The mechanisms by which alcohol may reduce the incidence of mortality of cardiovascular diseases are the subject of much debate, but there is certainly an increased level of high-density lipoprotein cholesterol in regular moderate consumers (El-Sayed, Ali, & El-Sayed, 2005). Available evidence also suggests that moderate alcohol consumption may have favourable effects on blood coagulation and fibrinolysis. Excessive intake is associated with several negative health outcomes, including an increased risk of various liver disorders, some cancers, and suicide. There are a number of unique features associated with the consumption of alcohol, including its psychological and behavioural effects, its effects on physiological and metabolic functions, and the social connotations of alcohol intake.

There is a long history of association between alcohol and sport (Collins & Vamplew, 2002). Sponsorship by alcohol companies remains an important source for many football clubs, further emphasizing the association. Alcohol consumption is associated in particular with celebration, but consumption can also be used as a shield to escape from the realization of failure. In this regard, sport is not different from most other social contexts, but the actions of intoxicated high-profile sportsmen and women are likely to attract attention from the media. Excessive alcohol intake is also likely to have a negative effect on a footballer's ability to perform the tasks for which he or she is employed.

Alcohol use in football and in sport

It is not entirely clear whether the prevalence of alcohol use is different in football players or other athletes from that of the general population, and the contradictory information in the literature reflects the variability in alcohol use in different population groups. It may also reflect the social connotations associated with alcohol use, and questionnaire-derived data on alcohol use may be even more prone to misrepresentation than those relating to use of other food groups. Reports from American school populations suggest a greater prevalence of alcohol use in team sports participants than in non-participants (Garry & Morrissey, 2000), and data from a New Zealand student population show higher rates of hazardous drinking behaviours in elite sportspeople (O'Brien, Blackie, & Hunter, 2005). In contrast, it seems that sports participation delays drinking and intoxication debut in Norwegian youngsters (Hellandsjo Bu *et al.*, 2002). Data from Spain (Pastor, Balaguer, Pons, & Garcia-Merita, 2003) suggest that alcohol use in athletic students is less than in their non-athletic peers. There are data from France that show a lower prevalence of alcohol use in athletic students (Lorente, Peretti-Watel, Griffet, & Grelot, 2003) but also data that show a greater prevalence of use (Lorente, Souville, Griffet, & Grelot, 2004). Moore and Werch (2004) have highlighted some of the complexities that influence analyses of the relationship between sports participation and substance use. In their survey, school-sponsored, male-dominated sports were associated with increased alcohol use, but out-of-school, mixed-gender sports participation was associated with greater use among females.

There are few surveys of the prevalence of alcohol use in senior football players and in those playing in professional leagues, and the reliability of these surveys is unknown. Maughan (1997) reported an average intake of about $10-12\,g \cdot day^{-1}$, representing about 2–3% of total energy intake in the first team squads of two Scottish Premier League teams. There was, however, an individual value in excess of 10% of energy intake, as well as several players who consumed no alcohol. Burke and Read (1988) reported that alcohol accounted for about 4% of total energy intake in elite Australian Rules football players, about the same as in the general population. In a somewhat alarming report, Ama, Betnga, Ama Moor and Kamga (2003) found that 25% of amateur football players surveyed in Yaounde, Cameroon, admitted drinking methylated spirits, and that 16% drank alcohol before matches (including 12% of "elite" players).

Care is needed in the interpretation of alcohol intake figures. "Moderate drinking", usually defined as the ingestion of about 10–40 g of ethanol per day for men and $10-20\,g \cdot day^{-1}$ for women, is not generally considered to be harmful to health. Indeed, those who drink alcohol in moderation have lower overall mortality rates than those who drink more heavily or those who do not drink at all (Macdonald, 1999). Problems arise, however, when intake at these levels is episodic – that is, when the average intake for a week is compressed into a short space of time. Unfortunately, binge drinking is commonly found in football, and the culture of the game is such that many players will abstain from alcohol in training but will drink copious amounts after a game (Burke & Maughan, 2000). This pattern was demonstrated in a dietary survey of Australian Rules football players whose mean daily intake of alcohol was reported to be a modest 20 g or two standard drinks (Burke & Read, 1988). However, almost all this intake occurred on

match day, when the mean intake was 120 g (range = 27 – 368 g); alcohol provided a mean contribution of 19% to total energy intake on match day (range = 3 – 43%). These self-reports of excessive alcohol intake after the game were confirmed when blood alcohol levels were estimated at a training session on the morning after a game: 34% of players registered a positive blood alcohol level with 10% of players showing a level above the legal limit for driving a motor vehicle.

Metabolism of ethanol

Alcohol (ethanol) is readily absorbed from the gastrointestinal tract and quickly becomes distributed throughout all body tissues. It is absorbed faster than it can be metabolized, so blood (and tissue) concentration increases, but there is no storage depot for alcohol, so it must be removed by oxidation. After a single drink, the blood alcohol concentration usually reaches a peak after about 40 min, but this time will vary with prior exercise, the co-ingestion of other nutrients, and habitual alcohol intake. A small amount (less than 10% of an ingested dose) may be lost in the urine and via the lungs.

Metabolism of alcohol occurs primarily in the liver, and it can be oxidized at a rate of about 100 mg per kilogram of body mass per hour. The first step in the metabolism of alcohol is the oxidation of ethanol to acetaldehyde ($CH_3CH{=}O$), a reaction catalysed by alcohol dehydrogenase (Reaction 1 below), with conversion of the coenzyme NAD^+ to NADH in the process. The acetaldehyde is further oxidized to acetate in a reaction catalysed by aldehyde dehydrogenase (Reaction 2 below), and the acetate so formed can then enter the citric acid cycle, where it is converted to carbon dioxide and water. A number of the metabolic effects of alcohol are directly linked to the production of an excess of both NADH and acetaldehyde. Acetaldehyde is thought to be responsible for many of the adverse effects of alcohol.

$$CH_3CH_2OH + NAD^+ \rightarrow CH_3CH{=}O + NADH + H^+ \quad (1)$$

$$CH_3CH{=}O + NAD^+ + H_2O \rightarrow CH_3COO^- + NADH + 2H^+ \quad (2)$$

Alcohol also can be metabolized by the microsomal ethanol-oxidizing system (MEOS) within the liver. The MEOS plays a minor role in alcohol metabolism when alcohol intake is small or absent, but its activity is upregulated by chronic intake of alcohol in high doses. Once the pathway is activated, MEOS greatly enhances the rate of conversion of alcohol to acetaldehyde: in doing so, it can disrupt the oxidation-reduction status of the cell by reducing the available pool of the reduced cofactor NADPH. The rate at which ethanol is cleared by the liver varies between individuals, and the response of the individual will depend on a number of factors, including the amount of ethanol consumed in relation to their habitual intake. There is conflicting information about whether the rate of metabolism of alcohol is increased by exercise.

Alcohol and performance

It is generally assumed that performance is impaired in individuals who are intoxicated, but there is a substantial body of anecdotal evidence to suggest that alcohol intake before exercise will not have an adverse effect on performance. Because of the understandable reluctance of institutional ethics committees to approve studies involving the administration of alcohol, there is limited experimental evidence relating to the effects of alcohol on exercise performance. There are reasons to believe that acute alcohol intake may impair performance of endurance exercise because of effects on metabolic, cardiovascular, or thermoregulatory function, and that it may affect performance of skilled tasks because of effects on reaction time, fine motor control, levels of arousal, and judgement.

There generally seem to be no effects of alcohol ingestion, at least in moderate amounts, on muscle strength and power (Reilly, 2003). An exception, however, is an early study by Hebbelinck (1963) that showed no effect of alcohol (0.6 ml of 94% ethanol $\cdot$ kg body mass^{-1}) on isometric strength, but a 6% reduction in vertical jump height and a 10% decrease in performance in an 80 m sprint. In a study of the effects of alcohol on sprinting and middle-distance running performance, McNaughton and Preece (1986) showed a progressive impairment in performance at distances of 200, 400, 800, and 1500 m with increasing doses of alcohol in amounts that resulted in blood alcohol concentrations between 0.01 and 0.1% (i.e. $10 - 100\,mg \cdot 100\,ml^{-1}$); performance in the 100 m sprint was not affected. In a study by Houmard, Lagenfield and Wiley (1987), performance time in a 5 mile (8 km) treadmill run was 28 s slower after low-dose alcohol ingestion (blood alcohol count below $0.05\,g \cdot 100\,ml^{-1}$): this effect was not statistically significant, but is nonetheless meaningful to the athlete. Kendrick, Affrime and Lowenthal (1993) reported that runners who drank alcohol before a treadmill run had higher heart rates, lower blood sugar levels, and more trouble finishing their runs compared with runners who had non-alcoholic drinks. Performance in events that

require skill, accuracy, judgement, and information processing are more obviously affected than are tasks that do not require these attributes.

Acute effects of alcohol that may influence performance

Metabolic effects

It is widely recognized that ethanol has a variety of effects on carbohydrate metabolism in skeletal muscle and in liver. Much of the available evidence comes from animal models, showing that synthesis of glycogen in both liver (Cook *et al.*, 1988) and oxidative skeletal muscle (Xu, Heng, & Palmer, 1993) is impaired in the presence of even relatively low levels of ethanol, though there seems to be no effects on Type II muscle fibres. Alcohol also delays gastric emptying, delaying the delivery of co-ingested glucose to the small intestine, and also appears to impair intestinal glucose absorption (Cook *et al.*, 1988). Ingestion of alcohol will increase the risk of hypoglycaemia due to the suppression of glucose production by the liver (Jorfeldt & Juhlin-Dannfelt, 1978). This may be of particular concern during prolonged, moderate-intensity exercise when glucose output from the liver is an important source of energy.

The animal data on impairment of glycogen storage in liver and muscle are often used as a reason for athletes to avoid alcohol during the recovery period after exercise, but it is not entirely clear that these data can be extrapolated to post-exercise recovery in humans. One of the key priorities during recovery between training sessions or games is the replacement of the limited carbohydrate stores, and this is achieved by ensuring an adequate intake of carbohydrate from the diet during the recovery period. Burke *et al.* (2003) reported the effects of alcohol intake on muscle glycogen storage in humans over 8 h and 24 h of recovery from a prolonged cycling bout that resulted in a substantial reduction of carbohydrate stores. The athletes who participated in this study undertook three different diets following their glycogen-depleting exercise: a high-carbohydrate diet intended to optimize recovery, an alcohol displacement diet (reduced carbohydrate, in which about 210 g of dietary carbohydrate was replaced by about 120 g alcohol), and an alcohol + carbohydrate diet (about 120 g alcohol added to the high-carbohydrate diet). Muscle glycogen storage was significantly reduced (by almost 50% at 8 h and about 16% at 24 h) on the alcohol displacement diet when the amount of carbohydrate provided by the diet was less than adequate (Figure 1). On the other hand, when the high-carbohydrate diet was eaten, there was no clear evidence that alcohol intake caused a reduction in muscle glycogen storage: there was a small, not statistically significant reduction at 8 h and no effect at all at 24 h. There was, however, a large variability of the responses of different participants, and it may well be that some individuals are unable to effectively replenish their glycogen reserves between daily training sessions if substantial amounts of alcohol are consumed.

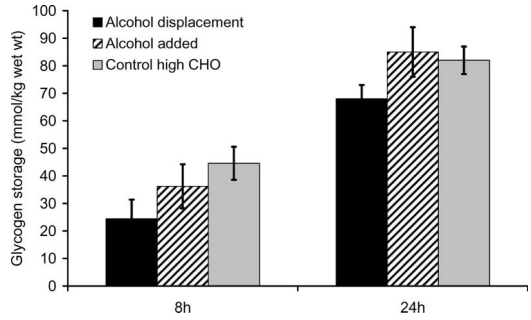

Figure 1. Effects of alcohol on post-exercise muscle glycogen storage. After glycogen-depleting exercise, participants were fed a control high-carbohydrate diet, an isoenergetic diet with 1.5 g alcohol $\cdot$ kg^{-1} displacing carbohydrate, or the high-carbohydrate diet with 1.5 g alcohol $\cdot$ kg^{-1} added. Displacing carbohydrate reduced glycogen storage at 8 h and 24 h; adding alcohol tended to reduce glycogen storage at 8 h, but there was no effect at 24 h. Drawn from the data of Burke *et al.* (2003).

Even if there is no direct metabolic effect of ethanol on glycogen storage when dietary carbohydrate intake is high, it is likely that players who consume large amounts of alcohol during the recovery period after training or a match will have a reduced carbohydrate intake, either as a result of a decreased total (non-alcohol) energy intake or because of a failure to follow the recommended eating strategies at this time.

Aerobic function

Acute ingestion of moderate doses of alcohol appears to have little effect on cardiorespiratory function and exercise performance (ACSM, 1982). In a study by Bond, Franks and Howley (1984), 12 males (six moderate drinkers and six abstainers) undertook three separate maximal exercise tests consisting of progressive workloads on a bicycle ergometer. Before each work bout, the participants consumed a placebo (0.0 ml $\cdot$ kg^{-1}), a small (0.44 ml $\cdot$ kg^{-1}), or a moderate (0.88 ml $\cdot$ kg^{-1}) dose of a 95% ethanol solution. Neither amount of alcohol had any significant effect on heart rate, blood pressure, ventilation, oxygen uptake, or work performance. Other studies, however, have suggested a decreased performance in events demanding a high aerobic capacity, such as middle-distance running (see Reilly, 2003).

Hydration effects

The diuretic action of ethanol is well recognized, and Eggleton (1942) estimated an excess urine production of about 10 ml for each gram of ethanol ingested. Alcohol acts via suppression of the release of antidiuretic hormone from the pituitary (Roberts, 1963). Alcohol has a negligible diuretic effect when consumed in dilute solution following a moderate level of hypohydration induced by exercise in the heat (Shirreffs & Maughan, 1997). There appears to be no difference in recovery from dehydration whether the rehydration beverage is alcohol-free or contains up to 2% alcohol, but drinks containing 4% alcohol tend to delay the recovery process by promoting urine loss. Based on the data of Eggleton (1942), however, it is apparent that concentrated alcohol solutions will result in net negative fluid balance: a 25 ml measure of spirits (40% ethanol) contains 10 ml of alcohol and 15 ml of water, resulting in a urine output of about 100 ml and net negative water balance of 85 ml. Ingestion of large volumes of dilute alcohol will result in a water diuresis, but should promote restoration of fluid balance provided that there is also an intake of sodium. The alcohol content of some standard drinks is shown in Table I.

Effects on thermoregulatory function

The 1982 ACSM Position Stand on the use of alcohol in sports identified perturbations of thermoregulatory mechanisms, especially in the cold, as one of the reasons to abstain from alcohol prior to exercise. Small doses of ethanol, given to human volunteers at rest in the absence of a thermal stress, have very little effect on body temperature, but large doses administered before exercise at low ambient temperatures result in increased peripheral vasodilatation and a marked fall in core temperature. In combination with the concomitant fall in blood glucose concentration that is normally observed in this situation, there is clearly potential for an adverse effect on performance. Graham (1981) showed that ingestion of alcohol ($2.5\,ml \cdot kg^{-1}$) before prolonged (3 h) exercise in the cold resulted in increased heat loss, though this effect was somewhat attenuated by co-ingestion of glucose. In animal studies, the administration of alcohol to animals exposed to ambient temperatures both above and below the thermoneutral zone has shown that alcohol acts to impair adaptation to both heat and cold (Kalant & Lê, 1983).

In another animal study, rats were given 0, 4, 8, 12, or 16% ethanol as the sole source of drinking water for 14 days. Time to fatigue in treadmill running in the heat (35°C) of rats drinking 4% ethanol was similar to that of rats consuming water (32 and 32.9 min respectively), but the running time of rats drinking 16% ethanol was reduced (Francesconi & Mager, 1981). There appear to be no further recent studies in this important area.

Table I. A standard drink contains approximately 10 g of alcohol.

Drink	Volume
Standard beer (4% alcohol)	250 ml
Low-alcohol beer (2% alcohol)	500 ml
Cider, wine coolers, alcoholic soft drinks	250 ml
Wine	100 ml
Champagne	100 ml
Fortified wines, sherry, port	60 ml
Spirit	30 ml

Psychomotor and behavioural effects

Alcohol has a number of direct effects – rather than via any of its metabolites – on central neurotransmitter synthesis and release. These actions have been reviewed by Reilly (2003). It is not surprising, therefore, that ingestion of large doses of alcohol will affect some or all of the actions of the central nervous system. The American College of Sports Medicine published a position stand on the use of alcohol in sports (ACSM, 1982) in which it was concluded that small to moderate amounts of alcohol – that is, less than the amount necessary to cause intoxication – result in impaired reaction time, hand–eye coordination, accuracy, balance, and gross motor skills. The evidence that has accumulated since then has confirmed these findings. The same mechanisms may operate to increase the risks associated with driving while under the influence of alcohol: about a half of all fatal road traffic accidents involve drivers who are intoxicated (National Institute on Alcohol Abuse and Alcoholism, 2000). An initial response to ingestion of small doses of alcohol is an enhanced sense of well-being, but even low doses (sufficient to elevate the blood alcohol concentration to about $30\,ml \cdot dl^{-1}$) will impair hand–eye coordination. Progressively increasing the dose leads to loss of social inhibition, loss of fine motor control, erratic behaviour, increased aggression, and finally loss of control of voluntary activity.

The aftermath of alcohol use

According to O'Brien and Lyons (2000), 74% of a population of football players reported that they drank alcohol, and 65% reported drinking the day before training or a match. There is limited and conflicting evidence on the effects of post-alcohol/hangover effects on functional capacity, but there is sufficient

evidence of adverse effects the day after a heavy drinking session for such activities to be discouraged (Barker, 2004). O'Brien (1993) showed reductions in aerobic exercise performance of rugby players the day after an evening bout of drinking involving an intake of 1 – 38 units of alcohol, though anaerobic performance was unaffected. The negative effect on aerobic performance was apparent at even the smallest dose of alcohol. For obvious reasons, there are few studies of the effects of high alcohol intakes on soccer-specific performance, and there appear to be no studies on well-trained footballers. Symptoms of hangover are thought to be due to dehydration, acid – base disturbances, disruption of cytokine and prostaglandin pathways, and alterations in glucose metabolism via effects on circulating insulin and glucagon levels (Wiese, Shlipak, & Browner, 2000). There are also cardiovascular effects during the hangover phase, including increased heart rate, decreased left ventricular performance, and increased blood pressure (Kupari, 1983).

Chronic effects of alcohol

Alcoholism is a complex disorder, with a strong genetic predisposition that interacts with precipitating factors in the environment (Macdonald, 1999). Some alcoholics consume alcohol in relatively moderate amounts on a regular basis while others engage in episodes of binge drinking that may vary in their frequency. Chronic intake of large amounts of ethanol is usually associated with multiple nutritional deficiencies as well as various muscle, liver, and cardiac pathologies. Animal studies show muscle atrophy, primarily in Type II fibres, in response to chronic alcohol exposure (Preedy, Duane, & Peters, 1988). It is unclear whether the habitual intake of small amounts of alcohol may adversely affect the metabolic adaptations in muscle that result from training. Although the management of the nutritional deficiencies is clinically relevant in the treatment of alcoholism, it has little practical relevance to the athlete. Impairment of performance because of the other effects of alcohol abuse is likely to be apparent long before nutrient deficiencies begin to be a concern.

Effects of alcohol on injury and incapacity

The ingestion of alcohol is likely to have a number of behavioural and other effects that may influence the risk of injury and the recovery process after injury. The history of the game of football contains many instances of players taking part in games while under the influence of alcohol. Prior alcohol consumption appears to increase the risk of sports-related injury, with an injury prevalence of 55% in drinkers compared with 24% in non-drinkers ($P < 0.005$) (O'Brien & Lyons, 2000). The mechanisms by which this association may be mediated are not entirely clear, but the increased risk of injury, and the increased severity of injuries that do occur, may be a consequence of increased risk-taking behaviours, as alcohol removes some of the restraints that normally operate (O'Brien, 1993). This may also account for the increased aggression often displayed by young men while under the influence of alcohol.

Intoxication during match-play is fortunately rather rare, at least at the higher levels of the game, though it is not completely absent. It is perhaps not so unusual, though, for players training the morning after a high alcohol intake the previous night to be still under the influence of alcohol, and several high-profile players have publicly admitted to alcohol addiction.

Some degree of muscle damage, either of intrinsic or extrinsic origin, is commonplace in both training and competition. This may often be in the form of minor damage that results in efflux of muscle-specific proteins into the vascular space, accompanied by some degree of pain and disability that may persist for hours or days (Clarkson & Hubal, 2002). This damage results in turn in an inflammatory response that involves an increase in local blood flow and macrophage infiltration of the damaged area. Recommended treatments include application of ice, compression, and elevation of the limb to reduce blood flow. Because alcohol can act as a peripheral vasodilator, it is often stated that alcohol intake should be avoided after any exercise that may have resulted in muscle damage. There appears, however, to be no experimental evidence to support these anecdotal observations. Clarkson and Reichsman (1990) investigated the effects of alcohol ingestion on muscle damage induced by eccentric exercise of the upper arm muscles. Female participants exercised one arm on two separate occasions, with alcohol ($0.8\,g \cdot kg^{-1}$) ingested 35 min before exercise on one occasion and a non-alcoholic drink on the other. Muscle damage was assessed by leakage of muscle-specific enzymes into the circulation, function was assessed by force generation, and subjective soreness and stiffness were also assessed. The exercise resulted in muscle damage, pain, loss of strength, and decreased range of motion, but there was no effect of alcohol on any of these responses. Nonetheless, intoxication is likely to result in inappropriate behaviours that may exacerbate existing muscle damage and delay the recovery process, and is unwise.

As more serious injury or surgical intervention results in longer-term inability to train or play, footballers may face some special problems. There may be a temptation to drink more, perhaps because

of the absence of a requirement to prepare for upcoming games or because of depression at being absent from the game and from the routine of training. This may result in unwanted weight gain, apart from the potential for negative effects on the repair process in muscle and other tissues.

Alcohol and the spectator

Alcohol consumption affects not only those on the field but also those spectators on the terraces and those watching at home on television. One consequence of a ban on alcohol sales at an (American) university football stadium was a decrease in security problems: decreases in arrests, assaults, ejections from the stadium, and student disciplinary hearings were reported after implementation of the ban (Bormann & Stone, 2001). These positive outcomes were not associated with any reduction in season ticket renewals in spite of a generally negative perception from spectators. An earlier study (Spaite *et al*., 1990) had shown no effect on the overall incidence of medical incidents among spectators after a ban on bringing alcohol into a college football stadium; there were, however, some changes in the nature of the medical emergencies treated.

Alcohol was banned from football grounds in the UK in the mid-1980s, but this has not prevented intoxication among supporters attending games. A review of medical consultations with stadium medical staff at a major Scottish football ground in a single season revealed a total of 127 casualties seen at 26 games (Crawford *et al*., 2001). ''Alcohol excess'' was identified as a major contributing factor in 26 (20%) cases.

The 1998 World Cup in Paris had effects on supporters and television spectators around the world. A total of 151 patients attended the Accident and Emergency Department of Edinburgh Royal Infirmary, Scotland, over a 5 week period with conditions that were identified as being related to the World Cup (Mattick, 1999). The majority of patients were suffering from alcohol-related trauma. During the 2002 World Cup held in Korea and Japan, 47 patients attended the Emergency Department of the University College Hospital, Galway, Ireland (Mattick, Mehta, Hanrahan, & O'Donnell, 2003); more than half of all cases were alcohol-related.

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