CLINICAL ASPECTS

Hydration Status and the Diuretic Action of a Small Dose of Alcohol

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Abstract — Aim: This study was conducted to examine the effect of consuming a dilute alcohol solution (weak beer) on urine production in euhydrated and hypohydrated individuals. Methods: Twelve males completed an intermittent cycle protocol in hot (35.1±0.3°C), humid (68±2%) conditions to dehydrate by 1.9±0.3% body mass in the evening. Twice they were then fed and rehydrated, while on two other occasions they were fed the same meal but remained hypohydrated. The following morning they were given 1 l of beer to drink. On two occasions the beer was alcohol-free, while on the other two occasions the same beer contained 4% ethanol. Participants remained in the laboratory for monitoring over the subsequent 4 h. Blood and urine samples were taken prior to dehydration, prior to drink administration and once every hour of the monitoring period. Results: No difference existed in the volume of urine produced between the alcohol (261±138 ml; mean±SD) and non-alcohol (174±61 ml) beer when hypohydrated (P=0.057), but there was a difference when euhydrated (1279±256 vs 1121±148 ml alcohol and non-alcohol, respectively; P<0.001). Unsurprisingly, more urine was produced on both euhydrated trials than either of the hypohydrated trials (P<0.001). Blood alcohol concentration was elevated (P<0.001) 1 h after drinking to 7.1±1.8 and 6.0±2.7 mmol/l (hypohydrated and euhydrated, respectively) on the alcohol trials. Serum osmolality was higher 1 h after drinking on both the alcohol trials (303±5 and 298±5 mosmol/l) than on their non-alcohol, equivalent hydration trials (290±8 and 284±5 mosmol/l hypohydrated and euhydrated, respectively; P<0.001). Conclusion: These results suggest that the diuretic action of alcohol is blunted when the body is hypohydrated.

INTRODUCTION

It was recognized as far back as the late sixteenth century that the consumption of alcohol leads to an increase in urine output, as evidenced by its effects on the porter in Shakespeare’s Macbeth. Alcohol is one of the two diuretic agents that are commonly present in the modern diet, the other being members of the methylxanthine family, including caffeine. Because of these diuretic actions, advice to the public during hot weather is usually to avoid drinks that contain caffeine because of the belief that these drinks will exacerbate any dehydration due to a stimulation of urine output.

The extent of the diuresis experienced is thought to be dependent upon the amount of alcohol consumed, and its effects on hydration status will be influenced by the concentration of alcohol relative to water in ingested beverages. Shirreffs and Maughan (1997) investigated effects of several beverages with a low alcohol (0, 1, 2 and 4%) content on the restoration of water and electrolyte balance immediately following exercise-induced dehydration. They concluded that there was no difference in the recovery of body water content from dehydration with a beverage of up to 2% alcohol, but that the 4% alcohol drink did increase urine output compared with the alcohol-free drink. This is consistent with the results of Eggleton (1942), who concluded that an additional 100 ml of urine would be produced for each 10 g of alcohol ingested, though this estimate was based on data from only a single subject. Crow (1968) was able to show more convincingly that the diuretic response in rats was proportional to the concentration of alcohol in a fluid bolus delivered by gastric intubation.

Much of the research into alcohol and diuresis has aimed at an evaluation of its mechanism of action rather than investigation of its diuretic effect under a variety of conditions, and most of the current literature refers to individuals who were probably euhydrated, or perhaps even hyperhydrated. Eggleton (1942) suggested that when participants were in a state of slight water deficit (following a light breakfast of tea and toast), the alcohol-induced diuresis was less marked than when in a state of euhydration. Stookey (1999) also concluded, on the basis of limited information, that the diuretic action of alcohol may be less pronounced in individuals who are chronically dehydrated.

Vasopressin (antidiuretic hormone) plays a major role in the regulation of water excretion. It is normally released by the posterior pituitary gland in response to a fall in blood volume or a rise in plasma osmolality and acts to conserve water. This is achieved primarily by increasing the permeability to water of the distal convoluted tubules and collecting tubules in the renal nephrons through insertion of Aquaporin-2 channels into the apical membrane of the tubular epithelial cells (Boone and Deen, 2008). Ingestion of alcohol does increase plasma osmolality, but alcohol also acts directly to inhibit the release of vasopressin, independent of plasma osmolality. Murray (1932) showed that administration of a posterior pituitary extract inhibited the alcohol-induced diuresis, and it is now widely accepted that alcohol exerts its diuretic action by inhibiting vasopressin release. The results of Taivainen et al. (1995), however, do not support the idea that the inhibition of vasopressin secretion is the only mechanism of alcohol-induced diuresis; using a relatively large dose of ethanol (1.2 g kg⁻¹ body mass), they found a marked acute diuresis relative to the control trial with no difference in circulating vasopressin levels. Linkola et al. (1978) also observed some dissociation of plasma AVP concentration and diuresis after alcohol ingestion and suggested that there may be direct effects on renal tubular reabsorption.

The aim of this investigation was to examine the effect of the consumption of a small dose of alcohol on urine production in individuals in both euhydrated and hypohydrated states in order to further the understanding of the effects of alcohol ingestion in different physiological states.
MATERIALS AND METHODS

Participants and ethical approval

Twelve healthy male volunteers participated in this study after giving written informed consent. Approval for the study was obtained from the Loughborough University Ethical Research Committee (ref: R08-P112). The mean±SD physical characteristics of the participants were as follows: age 23 (4) years, height 1.82 (0.04) m and body mass 76.6 (6.8) kg. All subjects were physically active on a recreational basis, and all were accustomed to drinking alcoholic beverages on an occasional basis, though none reported regular large intakes of alcohol.

Protocols

Subjects undertook one familiarization trial and four experimental trials. The experimental trials were performed at least 7 days apart in a randomized, cross-over design. Briefly, each trial consisted of an initial period of exercise in the evening to induce hypohydration, after which fluid was either provided (euvhylrated trials (E)) or restricted (hypohydrated trials (H)): this was then followed by overnight recovery. On the following morning, subjects ingested the appropriate test drink, which consisted of 1 l of alcohol-free beer (Bavaria Alcohol Free Beer, Holland) (non-alcohol trials (NA)) or 960 ml of the same alcohol-free beer, with 40 ml of 100% ethanol (BDH Laboratory Supplies, Poole, UK) added (alcohol trials (A)). Therefore, the four experimental conditions were as follows: euhydrated without alcohol (ENA), euhydrated with alcohol (EA), hypohydrated without alcohol (HNA) and hypohydrated with alcohol (HA).

Subjects completed an activity and diet record for the 24-h period prior to arriving at the laboratory for the familiarization trial. They were asked to replicate the same activity patterns and dietary intakes for the 24 h prior to each subsequent trial. They were also asked to refrain from alcohol consumption and unaccustomed strenuous activity during these 24 h. Subjects arrived in the early evening (5–6 pm) following a 4 h fast. They had also been instructed to drink 500 ml of plain water 2 h before arriving at the laboratory. Upon arrival, subjects were seated in a comfortable temperature and a blood sample (5 ml) was drawn from an antecubital vein after 15 min. Subjects were asked to consume the entire volume over a period of 30 min, after which the clock was reset for the 4-h monitoring period. They remained seated in the laboratory during this time and read, worked on a computer or watched television. At the end of the observation period, final nude body mass was measured and the subjects were then provided with food and drinks to consume before leaving the laboratory.

Blood and urine sampling

Blood and urine samples were collected at hourly intervals during the 4-h monitoring period. Participants remained seated for 15 min before the sample was drawn. Each 5-ml sample was drawn into a dry syringe. Of this, 2.5 ml was dispensed into a plain tube and left to clot for over an hour at room temperature before being centrifuged at 3000×g for 15 min. The resulting serum was separated and stored at 4°C for the analysis of osmolality. The other 2.5 ml was dispensed into K2EDTA-treated tubes and mixed well. From this, 4×100 μl aliquots of whole blood were deproteinized into 4×1 ml 0.3 N perchloric acid for the analysis of blood glucose and alcohol concentrations. The remaining whole blood was used to determine haemoglobin concentration and haematocrit. For each urine sample, subjects were asked to empty their bladder as fully as possible. The volume was measured and 2×5 ml samples were kept for the analysis of pH, osmolality, and sodium and potassium concentrations.

Sample analysis

Urine pH was measured as soon as possible after the sample was produced (Corning pH meter 140, Corning, Essex, UK). Urine sodium and potassium concentrations were measured by flame photometry (Corning clinical flame photometer 410C, Corning, Essex, UK). Urine and serum osmolality was analysed by freezing point depression (Osmomat 030 cryoscopic osmometer, Gonotec, Berlin, Germany). Blood glucose concentration was determined using the GOD–PAP method ( RANDOX laboratories Ltd, Co Antrim, UK). Blood alcohol concentration was measured on the deproteinized blood extract using an NAD-linked enzymatic assay with fluorometric analysis (Model 8-9 Fluorimeter, Locarte, London, UK) based on the principles of the Bonnichsen (1963) method. Blood haemoglobin concentration was analysed using the cyanmethaemoglobin method and haematocrit was
measured by microcentrifugation. These values were then used to calculate changes in blood, red cell and plasma volumes according to the equations developed by Dill and Costill (1974), using the pre-dehydration values on each trial as the baseline. All assays were performed in duplicate, except haematocrit which was measured in triplicate.

Statistics
All data sets were tested for normality of distribution before being subjected to statistical analysis. Where data were found not to be normally distributed, a natural log transformation was performed on the data and distribution was checked again. If found to be normally distributed, appropriate parametric tests were performed, while if the natural log transformation failed to normalize the data then they were analysed using the Wilcoxon signed rank post hoc test. Data were analysed by a two-factor repeated measures analysis of variance to identify whether there were significant differences between trials or significant changes over time. Where significance was found, t-tests were performed and a Bonferroni correction for multiple comparisons was applied as appropriate. A significance level of $P=0.05$ was accepted as indicating a statistically significant difference. All data are reported as mean±standard deviation, except where not normally distributed when they are reported as median (range).

RESULTS

Body mass
All subjects underwent the dehydration protocol successfully and achieved a similar level of hypohydration (Table 1). The level of hypohydration at the point of consuming the drink was significantly different between the euhydrated and hypohydrated trials ($P<0.001$). At the end of the observation period, there was no statistically significant difference in the levels of hypohydration between any of the trials.

Urine volume
Urine output was measured after each hour of the observation period. The total urine volumes produced in the four hours can be seen in Fig. 1 and the volume produced each hour can be seen in Fig. 2.

Figure 1 clearly shows that, as expected, there is a greater urine output on the euhydrated trials compared with the hypohydrated trials ($P<0.001$). It can also be seen that more urine was excreted on the EA trial (1279±256 ml) than the ENA trial (1121±148 ml; $P<0.001$). The volume of urine excreted on the HA trial was not significantly different from that excreted on the HNA trial ($P=0.057$). Figure 2 shows that peak urine output occurred 1 h after the end of the drink consumption on all trials, with outputs being significantly ($P<0.001$) greater at that point on both of the euhydrated trials.
(EA 643±303; ENA 599±205) than on either of the hypohydrated trials (HA 113±81; HNA 70±30) but not different within hydration status trials (euhydrated \( P=0.82 \), hypohydrated \( P=0.069 \)). When total urine output for EA and HA combined is compared with ENA and HNA combined, significantly more urine was produced on the alcohol trial than on the non-alcohol trials (1540±345 vs 1295±147, respectively; \( P=0.022 \)).

**Urine analysis**

Table 2 shows various physiological parameters measured in the urine samples taken before and each hour after drink consumption. Urine pH increased over time for all trials \(( P<0.05 \) but had started to decline again in ENA and HNA by the end of the observation period. The urine pH values in ENA and HNA were higher 2 h after consuming the drinks than their respective hydration status alcohol trials \(( P<0.003 \). As expected, urine osmolality was greater on both the hypohydrated trials than on the euhydrated trials \(( P<0.001 \). This was the case before drinking and for all 4 h of the observation period. Urine osmolality decreased after the ingestion of the test drink on both euhydrated trials \(( P<0.003 \) and started to rise again at hour 3 of the observation period.

During the rehydration period, urine sodium loss decreased over time \(( P=0.02 \) but there was no effect of trial on sodium excretion. Urine potassium loss was significantly greater in all trials 1 h after consuming any of the test beverages than at 3 or 4 h after drinking \(( P=0.006 \). This is not surprising due to the greater urine output at the start of the monitoring period compared with the later time points. There was also a significant effect of the alcohol on urinary potassium loss. At 2 h after drinking, urine output on trial ENA was greater than on trial EA \(( P=0.001 \) and was also greater on trial HNA than HA \(( P=0.003 \), suggesting that alcohol consumption reduced urinary potassium loss. At 2 h after drinking, urinary potassium output was greater on ENA than on any of the other trials \(( P<0.002 \).

**Haematological analysis**

No measurable level of blood alcohol was detected throughout the entire trial on ENA or HNA. Blood alcohol concentration was higher at 1, 2 and 3 h after drinking than either of the non-alcohol trials \(( P<0.001 \) and this was highest in the

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**Table 2. Various physiological parameters measured in the urine at each hour post-drink consumption for the four trials**

<table>
<thead>
<tr>
<th>Hours after drink</th>
<th>ENA</th>
<th>EA</th>
<th>HNA</th>
<th>HA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine pH</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>6.32±0.46</td>
<td>6.20±0.76</td>
<td>6.43±0.77</td>
<td>5.96±0.54</td>
</tr>
<tr>
<td>1</td>
<td>6.37±0.39</td>
<td>6.24±0.47</td>
<td>6.47±0.88</td>
<td>5.98±0.60</td>
</tr>
<tr>
<td>2</td>
<td>7.06±0.35</td>
<td>6.58±0.39</td>
<td>7.23±0.71</td>
<td>6.25±0.99</td>
</tr>
<tr>
<td>3</td>
<td>7.36±0.51</td>
<td>6.89±0.74</td>
<td>7.16±0.91</td>
<td>6.57±1.10</td>
</tr>
<tr>
<td>4</td>
<td>7.11±0.79</td>
<td>6.99±0.87</td>
<td>6.67±0.88</td>
<td>6.63±1.00</td>
</tr>
<tr>
<td>Urine osmolality (mosmol kg(^{-1}))</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>311±144</td>
<td>338±193</td>
<td>837±166</td>
<td>880±198</td>
</tr>
<tr>
<td>1</td>
<td>104±30</td>
<td>113±32</td>
<td>802±269</td>
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<td>790±149</td>
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<tr>
<td>4</td>
<td>543±154</td>
<td>575±156</td>
<td>924±129</td>
<td>890±138</td>
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<tr>
<td>Urine Na(^+) (mmol) median (range)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>9 (3–21)</td>
<td>11 (1–108)</td>
<td>6 (1–8)</td>
<td>7 (1–37)</td>
</tr>
<tr>
<td>2</td>
<td>8 (5–14)</td>
<td>7 (2–68)</td>
<td>4 (0–6)</td>
<td>5 (0–33)</td>
</tr>
<tr>
<td>3</td>
<td>7 (4–11)</td>
<td>6 (1–20)</td>
<td>3 (2–5)</td>
<td>3 (1–6)</td>
</tr>
<tr>
<td>4</td>
<td>5 (1–9)</td>
<td>7 (2–17)</td>
<td>3 (1–5)</td>
<td>3 (1–6)</td>
</tr>
<tr>
<td>Urine K(^+) (mmol)</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>11±7</td>
<td>8±4</td>
<td>7±3</td>
<td>8±5</td>
</tr>
<tr>
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<td>8±3</td>
<td>4±2</td>
<td>5±2</td>
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<tr>
<td>4</td>
<td>6±2</td>
<td>6±3</td>
<td>4±1</td>
<td>4±1</td>
</tr>
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</table>
first hour after ingestion on both trials (7.1±1.8 mmol/l on HA and 6.0±2.6 mmol/l on EA; Fig. 3). There was no difference in blood alcohol concentration between EA and HA at any time point. On both of the alcohol trials, there was no difference in blood alcohol concentration from the pre-ingestion baseline to 4 h after drink ingestion.

Blood glucose concentration generally followed the same pattern during each trial, with no difference between trials (P=0.593). There was a significant fall in blood glucose on both alcohol trials between 1 and 2 h after drinking (P<0.001). There was no significant change in blood glucose concentration with time (P=0.378), and there was no difference between trials (P=0.749). Plasma volume also did not differ over time (P=0.446) or between the trials (P=0.762; Fig. 4), although plasma volume was lower in the morning after hypohydration compared with before the cycling exercise, and both of the alcohol-containing trials show sharp decreases in plasma volume after drinking.

Based on serum osmolality measures made prior to exercise, all participants started the trials in a similar hydration state (Fig. 5). At 1 and 2 h after drinking, serum osmolality was greater on both alcohol trials than on their equivalent hydration status non-alcohol trials. Serum osmolality tended to always be lowest on the euhydrated non-alcohol trial: at 1, 2 and 3 h after drink consumption, it was significantly lower than on either EA or HA.

The aquaretic effect of the ingested alcohol whilst in differing hydration states was also assessed. Unsurprisingly, there was a significant difference in the rate of free water clearance between the euhydrated trials and the hypohydrated trials (P<0.001). There was also a significant influence of time on the rate of free water clearance (P=0.001) and a significant interaction between trial and time (P<0.001). Two hours after drinking the beverages, there was a significant difference in the rate of free water clearance between EA and ENA (5.12±1.95 and 3.19±1.77 ml/min, respectively; P=0.021) as well as between HA and HNA (−0.34±0.94 and −1.28±0.44 ml/min, respectively; P=0.014; Fig. 6).

**DISCUSSION**

The results of the present study suggest that hypohydration appears to blunt the diuretic action of a small dose of alcohol. Total urine production over the four hours after drink consumption was greater (on average by 158 ml; 10 of the 12
participants produced more urine) on the euhydrated trial with alcohol than without, in line with previous research. The urine volume produced on the hypohydrated trials was, not unexpectedly, less than on the euhydrated trials, but there was no difference in the total volume of urine produced with (261±138 ml) or without (174±61 ml) alcohol in the beer ingested.

It is also clear that there is a greater individual variation in the urine volumes produced on both of the alcohol trials than on their equivalent hydration status non-alcohol trial. We have investigated this pattern further to determine if this is an indication of individual sensitivity to the action of alcohol. It has been suggested that habitual intake may affect the diuretic response to ingested alcohol, in much the same way as regular consumers can become habituated to the psychological effects of alcohol (Stookey, 1999). However, this does not seem to be the case in the present study. When the volume of urine produced on the alcohol trials is expressed as a percentage of that produced on the equivalent hydration status, non-alcohol trial, there seemed to be no relationship between the values, suggesting that there is not a consistent effect within individuals of consuming a litre of 4% alcohol beer on urine production. The sample population in this study was selected to be relatively homogeneous in terms of their habitual consumption of alcohol, and this, together with the well-recognized limitations to the validity of self-reports of alcohol intake (Khadjesari et al., 2009) limits the value of any attempt to relate the possible diuretic action to habitual intake. Notwithstanding these limitations, the use of alcohol consumption diaries by participants in the weeks or month before the start of an experiment might allow better interpretation of inter-individual differences in responses and could also facilitate the comparison of results between studies.

Based on data from a single individual, Eggleton (1942) calculated that 10 g of alcohol causes an additional 100 ml of urine to be produced. Based on this estimate, addition of 40 ml of 100% ethanol (31.6 g) to 960 ml of fluid would have been expected to result in an additional urine output of about 320 ml. In the euhydrated state, participants in the present study passed an average additional volume of only 158 ml, but the individual response ranged from −165 to 468 ml. When hypohydrated, the addition of the same volume of alcohol caused an average additional volume of 87 ml to be produced, with a range of individual responses from −165 to 411 ml. We do not know the characteristics of the single participant in the study by Eggleton, including potentially highly influential information such as gender, body size or habitual alcohol intake, all of which may influence urine production and alter this generalized relationship. Eggleton also showed a day-to-day variation in urine output brought about by the diuretic effect of alcohol consumption within individuals. There are likely to be too many factors affecting the diuretic response to alcohol consumption for a single value to be applied to all individuals.

The mechanisms that underpin the diuretic action of alcohol are not clear. Murray (1932) suggested that alcohol may exert its diuretic action by a direct inhibitory effect of ethanol on vasopressin release from the posterior pituitary gland. However, the increase in plasma osmolality caused by alcohol ingestion and absorption would be expected to stimulate antidiuretic hormone release, thereby reducing the urine production. Kleeman et al. (1955) attempted to elucidate the exact action of the diuresis by infusing participants with hypertonic saline prior to alcohol administration, which resulted in the diuretic response being blocked. Roberts (1963) further confirmed this by showing that the mechanism of dehydration in chronic and acute alcoholic states...
is caused by inhibition of antidiuretic hormone. She showed, using sodium chloride ingested concurrently with alcohol, that the diuretic effect of alcohol is not due to the loss of solutes in the urine.

It took 4 h after drink consumption for the difference in urine production between EA and ENA trials to reach statistical significance. Serum osmolality was significantly greater in EA compared with ENA for 3 h after drinking but by the end of the 4-h monitoring period, serum osmolality was not different between treatments. Blood alcohol concentration peaked at 1 h after ingestion and remained significantly elevated in EA compared with ENA for 3 h after drinking. Therefore, based on the present results, whichever of these two factors influences the alcohol-induced diuresis, there is a time delay in its action when administering a low dose of alcohol.

Roberts (1963) saw that alcohol continued to have a dehydrating effect in chronically hypohydrated individuals. The current study, however, found that the diuretic effect of alcohol was blunted in the acutely dehydrated state. Therefore, it seems that the extent of the diuresis is dependent either upon the duration of the presence of the alcohol in the body and/or the extent of the hypohydrated state. The hypohydrated subjects used by Roberts were chronic alcoholics admitted to an alcoholic clinic and therefore the true level of the dehydration cannot be quantified. It would be expected that the diuretic response would be blunted as the level of dehydration increases due to renal conservation mechanisms, but it is also possible that the relative concentration of alcohol in the blood increases in the hypohydrated state due to the reduced body water content, thereby causing a more pronounced diuresis. Although there was no significant difference in the volume of urine produced on the two hypohydrated trials, there was a tendency for more to be produced on the alcohol trial than on the non-alcohol trial. This indicates that there may still be an effect of the alcohol consumption but that this effect is blunted when the body is in water deficit.

Saini et al. (1995) found that the arginine vasopressin response to low-intensity exercise was reduced after ingestion of alcohol (1.2 g kg⁻¹ body mass) but that there was no effect on aldosterone or atrial natriuretic peptide. They also reported that since the significant increase in plasma osmolality caused by the alcohol ingestion did not influence the response of aldosterone or atrial natriuretic peptide, that total osmolality does not have a major influencing role over the release of these hormones.

However, Taivainen et al. (1995) suggest that the inhibition of vasopressin secretion is not the only mechanism of alcohol-induced diuresis. They saw that after administering alcohol (1.2 g kg⁻¹ body mass) there was an alcohol-induced diuresis followed by a period of antidiuresis, and when participants were then water loaded the antidiuresis continued but to a lesser extent on the alcohol trial than on the control trial. During the alcohol-induced diuresis, plasma arginine vasopressin levels did not differ from the control experiment but were higher during the initial phase of antidiuresis. With the small dose of alcohol administered in the present study, no such effect may be apparent.

Weak beer has been reported to be the preferred rehydration drink in many industrial workplaces where sweat losses are high (Maughan and Griffin, 2003). Indeed, industry sales figures back this up with sales increasing during the summer months (www.allbusiness.com, www.bbc.co.uk/news). Most beers have a sodium content of 3–5 mmol/l and a potassium content of 8–10 mmol/l (Holland et al., 1991). Although higher levels of sodium and potassium will reduce urine output when ingested with a large bolus of water, these levels of electrolytes are too low to have a significant effect on water retention when the body is in a state of hypohydration (Shirreffs et al., 1996). Alcohol also acts as a peripheral vasodilator that could, theoretically at least, aid heat loss, therefore making it more appealing to those working where sweat rates and the need to lose body heat is high. However, a study by Desruelle et al. (1996) suggests that alcohol does not alter the capacity of the body to deal with exogenous or endogenous heat loads.

The present study found no effect of alcohol consumption on the urinary excretion of sodium, but alcohol consumption did result in lower potassium excretion than on the non-alcohol trials after 2 h. A reduction in urinary electrolyte output in response to alcohol ingestion was shown by Rubini et al. (1955) who found a rapid reduction in the excretion rate of sodium, potassium and chloride when alcohol (∼48 g) was consumed. The onset of responses seen in the present study was not as rapid as in the Rubini study, though this could be due to the different volumes of fluid consumed (1 l compared with 120 ml), different rates of consumption (up to 30 min compared with up to 10 min) or different amounts of alcohol (32 g compared with ∼48 g). Our finding of an alcohol-induced reduction in potassium output is also supported by a trend in the study of Shirreffs and Maughan (1997), which showed lower potassium output when using 4% beer as a rehydration drink than with a 0% placebo at 1, 2, 4 and 6 h after rehydration.

This reduction in potassium excretion due to the alcohol consumed also corresponds well to the finding that, after 120 min, alcohol increases the rate of free water clearance compared with trials conducted when in the same hydration status but without the consumption of alcohol. Assessment of the aquaretic effect of alcohol in future research may also allow improved comparisons to be drawn between studies that have used differing methodologies.

**CONCLUSION**

In conclusion, it appears that when the body is in a state of water deficit the diuretic action of alcohol is blunted in order to attempt to restore fluid balance. The physiological mechanisms and influences of this response remain unclear but are likely to be closely interlinked.

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