ORIGINAL ARTICLE

# Effect of beer consumption on plasma magnesium: randomized comparison with mineral water

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## SUMMARY

**Moderate consumption of ethanol lowers mortality from coronary artery disease, and one of the possible mechanisms is an antiarrhythmic action. We therefore investigated the effect of a small daily dose of beer on plasma electrolytes. 52 men who seldom drank alcohol, clinically stable more than one year after coronary bypass surgery, were randomized to drink either 330 mL beer (containing about 20 g ethanol) or mineral water with similar potassium, magnesium, calcium and sodium content daily for 30 days. Plasma electrolytes and liver function indices, and also heart rate, blood pressure and weight, were measured before and after the trial period.**

**The only significant before-and-after difference was in the group consuming beer, whose plasma magnesium rose from 0.89 (SD 0.01) to 0.98 (SD 0.02) mmol/L ($P<0.0025$).**

**This level of beer consumption did no obvious harm to liver function and its possibly beneficial effect on plasma magnesium deserves further investigation.**


## INTRODUCTION

In many populations, moderate consumption of ethanol has been shown to protect against cardiovascular disease[1]. Possible mechanisms include effects on lipids, on clotting and on cardiac conduction. McKee and Britton[2] have lately shown how the direction of these effects is influenced by the pattern of drinking: with binge drinking they tend to be adverse, whereas with steady intake of the same quantity of alcohol they are beneficial. Since much of the mortality in coronary artery disease is due to arrhythmias, and arrhythmias are influenced especially by potassium and magnesium[3–5], we examined the effects of moderate beer consumption on plasma electrolytes. We also did tests of liver function and measured heart rate and blood pressure to detect any harmful effects.

## METHODS

As in previous investigations[6,7], we used Maccabee beer.

The study population was recruited from men aged 48–74 years who had had bypass surgery for coronary artery disease. From 136 such patients we selected 52 on the following criteria:

- Symptoms present for at least 2 years before operation; angina now absent without medication
- Alcohol rarely if ever consumed—not more than once every 2–3 months and not more than 20 g on any occasion
- Bypass surgery done at least twelve months before; no biochemical evidence of liver or kidney dysfunction.

The 52 patients were randomly assigned to an experimental group (EG, $n=26$) and a control group (CG, $n=26$). All patients consumed a diet recommended for patients with coronary artery disease, rich in vegetables and fruit and low in fat (daily energy intake about 1600 kcal) and engaged in similar programmes of physical activity.

For 30 consecutive days EG patients drank 330 mL Maccabee beer (about 20 g of alcohol) once a day, while the CG patients drank instead 330 mL mineral water (Netivot) with the same potassium, magnesium, calcium and sodium content as Maccabee beer. Before and after the experimental phase we measured systolic and diastolic blood pressures, heart rate and weight and took blood for laboratory tests, including plasma sodium, potassium, calcium and magnesium, aminotransferases and alkaline phosphatase, and bilirubin.

Before-and-after comparisons were made with the 'Student'–Fisher $t$ test, $P$ values less than 0.05 being regarded as statistically significant.

## RESULTS

Neither the experimental group nor the control group had significant changes in heart rate, systolic or diastolic blood

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*Table 1* **Laboratory values (means [SD] and 95% confidence intervals)**

| | EG | | CG | |
|---|---|---|---|---|
| | B | A | B | A |
| Potassium (mmol/L) | 4.3 (0.2) [3.9–4.7] | 4.32 (0.2) [3.91–4.73] | 4.33 (0.2) [3.92–4.74] | 4.31 (0.2) [3.9–4.72] |
| Calcium (mmol/L) | 2.45 (0.05) [2.35–2.55] | 2.46 (0.05) [2.36–2.56] | 2.46 (0.05) [2.36–2.56] | 2.45 (0.05) [2.35–2.55] |
| Sodium (mmol/L) | 141.1 (3.6) [133.8–148.8] | 141.0 (3.6) [133.6–148.4] | 141.0 (3.6) [133.6–148.4] | 140.9 (3.6) [133.5–148.3] |
| Magnesium (mmol/L) | 0.89 (0.01) [0.87–0.91] | 0.98 (0.02) [0.94–1.02] | 0.90 (0.01) [0.88–0.92] | 0.89 (0.02) [0.85–0.93] |
| AST (iu/L) | 19.96 (1.5) [16.88–23.04] | 20.01 (1.5) [16.93–23.09] | 19.9 (1.5) [16.82–22.98] | 20.0 (1.5) [16.92–23.08] |
| ALT (iu/L) | 20.0 (1.4) [17.12–22.88] | 19.9 (1.4) [17.02–22.78] | 19.9 (1.4) [17.02–22.78] | 20.0 (1.4) [17.12–22.78] |
| APH (IU/l) | 52.1 (2.8) [46.34–57.86] | 52.3 (2.85) [46.44–58.16] | 51.9 (2.8) [46.14–57.66] | 52.1 (2.8) [46.34–57.86] |
| T bilirubin (μmol/L) | 7.92 (0.7) [6.58–9.36] | 7.88 (0.7) [6.44–9.32] | 7.9 (0.7) [6.46–9.34] | 7.92 (0.7) [6.58–9.36] |
| D bilirubin (μmol/L) | 3.1 (0.2) [2.69–3.51] | 3.08 (0.19) [2.69–3.48] | 3.09 (0.19) [2.7–3.48] | 3.11 (0.2) [2.7–3.52] |
| Total protein | 68.7 (2.92) [62.7–74.7] | 68.8 (3.0) [62.6–75.0] | 68.6 (3.0) [62.4–74.8] | 68.7 (3.0) [62.5–74.9] |
| Albumin | 51.2 (2.5) [46.1–56.3] | 51.3 (2.4) [46.4–56.2] | 51.3 (2.5) [46.2–56.4] | 51.2 (2.5) [46.1–56.3] |
| Globulin | 15.2 (1.6) [11.9–18.5] | 15.1 (1.6) [11.8–18.4] | 15.1 (1.6) [11.8–18.4] | 15.2(1.6) [11.9–18.5] |

EG=experimental group; CG=control group; B=before; A=after
AST, ALT=aspartate and alanine aminotransferase; APH=alkaline phosphatase; T, D bilirubin=total, direct bilirubin
*$P < 0.0025$. All other before-and-after differences non-significant

pressure or weight. The only recorded value that did change significantly from the beginning to the end of the study was plasma magnesium in the experimental group (Table 1). In particular, beer consumption had no obvious effect on indices of liver function.

## DISCUSSION

Since a large number of variables were examined, we must first ask whether the change in plasma magnesium in the beer-drinking group arose by chance. We think not, because the observation has been replicated in a separate investigation of patients with coronary artery disease, not yet completed.

Might the beneficial influence of alcohol on coronary disease mortality be explained partly by an action on magnesium? A low plasma magnesium is proarrhythmic[8], and magnesium has been successfully used in prevention and treatment of arrhythmias. For example, a 3-day intravenous infusion with subsequent oral therapy achieved a significant decrease in ventricular ectopics[9]; and oral magnesium alone had a moderate antiarrhythmic effect[10]. According to some reports, magnesium infusions started early after onset of myocardial ischaemia can limit infarct size, prevent serious arrhythmias and reduce mortality[11–15], but the very large ISIS-4 study showed no benefits[16].

Unlike moderate drinking, heavy drinking is associated with a low plasma magnesium. Perhaps this accounts for the paradoxical effects described by McKee and Britton[2]. In our experiment the explanation for the raised magnesium after beer-drinking is not clear. Increased intake can be ruled out (since the magnesium content of beer and mineral water was the same); increased absorption is a possibility, but we suspect that beer increases plasma magnesium by decreasing the intracellular concentration. This mechanism deserves further investigation.

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