Effect of consumption of red wine, spirits, and beer on serum homocysteine

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Serum homocysteine increases after moderate consumption of red wine and spirits, but not after moderate consumption of beer. Vitamin B₁₂ in beer seems to prevent the alcohol-induced rise in serum homocysteine.

Homocysteine concentrations are affected by lifestyle factors such as diet—eg, inadequate intake of B vitamins involved in the homocysteine breakdown. Alcoholics have raised homocysteine concentrations that are either caused by low vitamin B intake or chronic excessive alcohol consumption. Beer is a rich source of folate and vitamin B₁₂, whereas red wine and spirits contain negligible amounts of these vitamins. We postulated that moderate alcohol consumption could affect homocysteine metabolism, and that these effects are beverage specific. In a randomised, diet-controlled, crossover trial,¹ 11 healthy, non-smoking men (aged 44–59 years) who were moderate alcohol drinkers, consumed four glasses of red wine, beer, or spirits (Dutch gin), or sparkling mineral water (control) with dinner. Beverages were switched every 3 weeks in a randomised order, according to a Latin square design. All food and drink was supplied for 12 weeks. The diet, which was essentially the same during all four periods, contained adequate amounts of macronutrients and micronutrients. Alcohol intake equalled 40 g daily (with exception of the water period), which did not affect activities of the liver enzymes γ-glutamyltransferase, alanine aminotransferase, and aspartate aminotransferase. Treatment effects were assessed by analysis of variance, by use of general linear modelling, in which homocysteine measurements were log transformed. No carry-over effects were seen.

Homocysteine concentrations were raised after 3 weeks' consumption of red wine and spirits by 8% and 9%, respectively, as compared with water consumption, whereas consumption of red wine and spirits, but not after moderate consumption of beer. Vitamin B₁₂ in beer seems to prevent the alcohol-induced rise in serum homocysteine.

Homocysteine concentrations could rise by inhibition of its two major breakdown pathways, both dependent on B vitamins. The remethylation pathway depends on folate and vitamin B₁₂, whereas vitamin B₁ is essential in the breakdown via trans-sulphuration.¹ We assessed beverage specific effects on plasma values of these vitamins. No significant differences in vitamin B₁ were reported. A 10% fall in folate occurred after spirits consumption only (table), and no correlation was found between changes in homocysteine values and changes in folate (Pearson correlation coefficient, p=0.99). Plasma vitamin B₁₂, analysed as pyridoxal-5'-phosphate, was increased after beer consumption by about 30%. Surprisingly, vitamin B₁₂ concentrations were also higher after intake of wine and spirits—17% and 15%, respectively (table). Changes in vitamin B₁ were shown to have a significant inverse correlation with changes in homocysteine (r=−0.58, p=0.0004; figure), suggesting that vitamin B₁ might be a rate-limiting factor for homocysteine breakdown after moderate alcohol consumption. Interestingly, prospective data from the Atherosclerosis Risk in Communities study suggest that vitamin B₁₂ itself is inversely associated with CVD risk, independently of homocysteine.² So, the increase in plasma vitamin B₁₂ as seen after beer and to a lesser extent after red wine and spirits consumption might even contribute to a lower CVD risk.

This study was funded by the Dutch Foundation for Alcohol Research. Martijn S van der Gaag died in January, 2000.


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