

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 B₆ 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 B₆, 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.

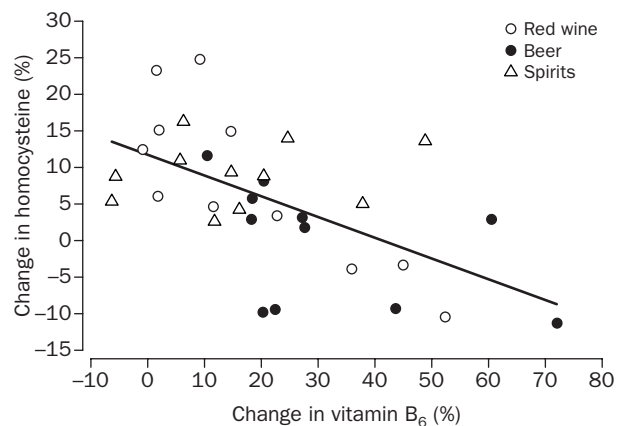
Homocysteine concentrations were raised after 3 weeks' consumption of red wine and spirits by 8% and 9%, respectively, as compared with water consumption, whereas no increase was recorded after beer consumption (table). Such an increase in homocysteine coincides with a 10–20% increase in cardiovascular disease (CVD) risk.² However, moderate alcohol consumption is associated with a lowered CVD risk. The cardioprotective effects of moderate drinking could exceed the increase in risk by higher homocysteine concentrations. Alternatively, slightly and transiently raised homocysteine concentrations might be beneficial. Homocysteine could act as a mediator of tissue repair and as a regulator of blood cells and cells of the vascular wall.³

Homocysteine concentrations could rise by inhibition of its two major breakdown pathways, both dependent on B-vitamins. The remethylation pathway depends on folate

	Red wine	Beer	Spirits	Water
Homocysteine (μ mol/L)	14.2¶**	12.9†	14.2‡§	13.0†
Folate (nmol/L)	18.4	17.8	16.5¶§	18.3‡§
Vitamin B ₁₂ (pmol/L)	223	218	209	232
Vitamin B ₆ (nmol/L)	56.5‡§	62.9†§	55.6‡§	48.3§¶

*Median homocysteine concentrations in serum samples. Vitamins have been measured in plasma; different from †water, ‡beer, §red wine, ||spirits ($p < 0.01$), and from ¶water, **beer, ††red wine, ‡‡spirits ($p < 0.02$).

Mean blood concentrations of homocysteine,* folate, vitamins B₆ and B₁₂ after 3 weeks consumption of red wine, beer, spirits and water



Relation between changes in homocysteine and changes in vitamin B₆ concentrations

Individual changes were computed by subtracting outcome after 3 weeks' consumption of each alcoholic beverage from outcome after 3 weeks' water consumption and expressed as percentage of outcome after water consumption, per alcoholic beverage.

and vitamin B₁₂, whereas vitamin B₆ is essential in the breakdown via trans-sulphuration.⁴ We assessed beverage specific effects on plasma values of these vitamins. No significant differences in vitamin B₁₂ 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 B₆, analysed as pyridoxal-5'-phosphate, was increased after beer consumption by about 30%. Surprisingly, vitamin B₆ concentrations were also higher after intake of wine and spirits—17% and 15%, respectively (table). Changes in vitamin B₆ showed a significant inverse correlation with changes in homocysteine ($r = -0.58$, $p = 0.0004$; figure), suggesting that vitamin B₆ 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 B₆ itself is inversely associated with CVD risk, independently of homocysteine.⁵ So, the increase in plasma vitamin B₆, as seen after beer and to a lesser extent after red wine and spirits consumption might even contribute to a lower CVD risk.

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