

The effect of red wine and beer on plasma homocysteine levels: a randomised controlled trial

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Regular moderate alcohol consumption may protect against cardiovascular disease (CVD), while heavy consumption contributes to an increased risk. This apparently biphasic response has made it difficult to elucidate the mechanisms involved. There is much controversy surrounding the effects of beverage specific constituents found in beer and red wine. Recent research has explored the possible relationship between alcoholic beverage type and total plasma homocysteine levels (tHcy), an independent risk factor for CVD. Beer, a source of folate (6–9 µg/100 ml), has been negatively associated with tHcy (1). The notion that components in beer may modulate tHcy levels has been supported by a 3-week cross-over study (n = 11) where both red wine and spirits (40 g alc./day) raised tHcy whilst beer (40 g alc./day) had no effect (2). In contrast a 4-treatment parallel intervention (n = 60) found that beer, red wine and spirits (30 g alc./day) for 6 weeks all raised tHcy (3). Red wines are rich in polyphenols, and high doses of polyphenols found in tea and coffee have been shown to raise tHcy (4). We therefore aimed to compare the effects of the regular consumption of red wine, de-alcoholised red wine and beer on tHcy.

Community volunteers entered a crossover study with four treatment periods: control-abstinence, 375 ml red wine/day (39 g alcohol; 760 mg polyphenols), 375 ml de-alcoholised red wine/day (0 g alc.; 785 mg polyphenols) and 1125 ml beer/day (41 g alc.). Each subject received each of the four treatments for 4 weeks in random order. tHcy, biomarkers of alcohol consumption (γ-GT and HDL-cholesterol) and 24hr urinary excretion of 4-O-methylgallic acid (4OMGA), a marker of red wine polyphenol absorption were measured.

Twenty four healthy men aged 53 ± 2y with BMI 25.3 ± 0.5 kg/m² completed the study. Compliance with drinking protocol was confirmed with increased γ-GT and HDL during red wine and beer periods, and increased 4OMGA during red wine and de-alcoholised red wine periods. Repeated measures ANOVA found no statistically significant effects of red wine, de-alcoholised red wine or beer on tHcy. Increases in urinary excretion of 4OMGA after red wine and de-alcoholised red wine were not correlated with any change in tHcy.

	Control abstinence	Red wine	De-alcoholised red wine	Beer
γ-GT (U/L)	17.7 ± 0.7	21.3 ± 0.8 ^a	18.0 ± 0.7 ^{b,c}	22.2 ± 0.9 ^a
HDL-C (mmol/L)	1.21 ± 0.05	1.34 ± 0.06 ^a	1.22 ± 0.06 ^{b,c}	1.37 ± 0.07 ^a
4OMGA (µg/day)	71 (141)	405 (480) ^a	438 (638) ^a	142 (287) ^{a,b,c}
tHcy (µmol/L)	8.94 (8.02, 9.88)	9.81 (8.90, 10.80)	9.35 (8.54, 10.34)	9.61 (8.84, 10.45)

^a different from control abstinence, ^b different from red wine, ^c different from beer (P < 0.05). Data, mean ± SEM; 4OMGA data is median (interquartile range); tHcy data is geometric mean (95% CI).

A non-significant trend for both red wine and beer to elevate tHcy prompted *post hoc* comparison of the averaged results from the 2 alcohol periods (ie beer and red wine) and non-alcohol periods (ie abstinence and dealcoholised red wine). Alcohol increased tHcy from 9.17 (8.4, 10.0) to 9.7 (8.9, 10.6) µmol/L [geometric mean (95% CI); P = 0.006]. We conclude that alcohol, but not beverage specific constituents, increases tHcy in healthy men.

References

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