Hyperhomocysteinaemia, Helicobacter pylori, and coronary heart disease

Sirs,—Sung and Sanderson have suggested that the development of hyperhomocysteinaemia in subjects with Helicobacter pylori infection (due to malabsorption and consequent vitamin deficiency) might provide a link between these two possible causes of coronary heart disease. We have examined this issue within the framework of our earlier prospective case control study of H pylori infection, based on a comparison of incidence cases of myocardial infarction (fatal and non-fatal) and controls within the British regional heart study.

Residual serum samples were available for 110 of 135 cases of myocardial infarction and 118 of 136 controls for measurement of total homocysteine by reverse phase high performance liquid chromatography with fluorescence detection.

Within the control group, geometric mean total homocysteine concentrations were very similar in subjects seronegative (n = 63) or seropositive (n = 55) for H pylori (11.9 ± 1.9 µmol/l; p = 0.98), i.e. an analysis adjusted for age and town, total homocysteine was positively related to risk of myocardial infarction. For each 5 µmol/l increase in total homocysteine, the odds ratio (OR) increased by 1.38 (95% confidence interval CI 1.02 to 1.86; p = 0.03). However, mutual adjustment had little effect on the respective odds ratios: for total homocysteine, adjusted OR = 1.35; 95% CI 1.00 to 1.83; p = 0.04; for H pylori, adjusted OR = 1.47; 95% CI 0.81 to 2.68; p = 0.20.

The findings of this study, while consistent with earlier reports of an association between homocysteinaemia and coronary risk, do not provide strong support for the hypothesis that hyperhomocysteinaemia and H pylori infection have interrelated effects on coronary risk.

Analyses of total homocysteine were carried out in the department of pharmacology, University of Bergen, Norway (Professors H Refsum and P M Ueland).

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