Homocysteine and Atherosclerotic Disease: The Epidemiologic Evidence

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Abstract

Homocysteine is a sulphur-containing amino acid formed during metabolism by one of two pathways by remethylation and transsulfuration. Altered homocysteine metabolism may be implicated as a factor in atherosclerosis, cerebrovascular disease or peripheral vascular disease. It is postulated that homocysteine may damage endothelial cells or acts as a direct causal factor in the thromboembolic process. Several studies have reported that there are a number of factors that may influence levels of homocysteine in humans. Serum homocysteine levels may be associated with low levels of folate, vitamin B6 and vitamin B12. These studies showed that serum homocysteine levels were higher in men and older adults, and some showed that there was a direct relationship between homocysteine and cigarette smoking, diabetes, obesity, and hypertension. Subjects who consume larger amounts of coffee were also noted to have higher serum homocysteine levels. Several cross-sectional, case-control, and cohort studies have linked homocysteinaemia with cardiovascular disease morbidity and mortality. In the Framingham Heart Study, the cohort study in Tromso, Norway, and the Atherosclerosis Risk in Communities (ARIC) Study, homocysteine levels were found to be higher in adults with asymptomatic or symptomatic coronary artery disease. In the British Regional Heart Study, homocysteine levels were found to be significantly higher in patients with stroke. Thus, there are suggestions that vitamin therapy and alteration of lifestyle habits such as cigarette smoking may lower homocysteine levels. There may be less coronary heart disease morbidity and mortality with lower homocysteine levels.

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