

Re: the business of carbs or is it the conspiracy or carbs?

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- *From:* GMCarter <fiar@xxxxxxxxxxxx>
 - *Date:* Fri, 16 Jun 2006 10:24:31 GMT
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On Fri, 16 Jun 2006 01:12:48 GMT, Jim Chinnis <jchinnis@xxxxxxxxxxxx> wrote:

snip

But studies on the whole seem to show that reducing homocysteine worsens rather than improves risk of heart disease and stroke.

What studies? I understood the situation was to the contrary. Elevated homocysteine is associated with CHD, albeit weakly.

What data are you referring to?

George M. Carter

Am Heart J. 2006 Feb;151(2):282-7. Related Articles, Links
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Homocysteine-lowering trials for prevention of cardiovascular events: a review of the design and power of the large randomized trials.

B-Vitamin Treatment Trialists' Collaboration.

BACKGROUND: Dietary supplementation with folic acid and vitamin B12 lowers blood homocysteine concentrations by about 25% to 30% in populations without routine folic acid fortification of food and by about 10% to 15% in populations with such fortification. In observational studies, 25% lower homocysteine has been associated with about 10% less coronary heart disease (CHD) and about 20% less stroke. **METHODS:** We reviewed the design and statistical power of 12 randomized trials assessing the effects of lowering homocysteine with B-vitamin supplements on risk of cardiovascular disease. **RESULTS:** Seven of these trials are being conducted in populations without fortification (5 involving participants with prior CHD and 2 with prior stroke) and 5

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in populations with fortification (2 with prior CHD, 2 with renal disease, and 1 with prior stroke). These trials may not involve sufficient number of vascular events or last long enough to have a good chance on their own to detect reliably plausible effects of homocysteine lowering on cardiovascular risk. But, taken together, these 12 trials involve about 52,000 participants: 32,000 with prior vascular disease in unfortified populations and 14,000 with vascular disease and 6000 with renal disease in fortified populations. Hence, a combined analysis of these trials should have adequate power to determine whether lowering homocysteine reduces the risk of cardiovascular events within just a few years. CONCLUSION: The strength of association of homocysteine with risk of cardiovascular disease may be weaker than had previously been believed. Extending the duration of treatment in these trials would allow any effects associated with prolonged differences in homocysteine concentrations to emerge. Establishing a prospective meta-analysis of the ongoing trials of homocysteine lowering should ensure that reliable information emerges about the effects of such interventions on cardiovascular disease outcomes.

Mayo Clin Proc. 2006 Feb;81(2):177–82. Related Articles, Links

Association of plasma homocysteine with coronary artery calcification in different categories of coronary heart disease risk.

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OBJECTIVE: To Investigate the association of plasma homocysteine with coronary artery calcification (CAC) in strata based on 10-year risk of coronary heart disease (CHD) in a cohort enriched in persons with hypertension. PARTICIPANTS AND METHODS: Fasting plasma homocysteine was measured by liquid chromatography electrospray tandem mass spectrometry. Coronary artery calcification was measured noninvasively by electron beam computed tomography and CAC score calculated using the method of Agatston et al. The 10-year CHD risk was calculated based on the Framingham risk score. The association of homocysteine with log-transformed CAC score was assessed in the pooled sample and within each risk stratum by linear regression after adjustment for conventional risk factors. RESULTS: In the 1071 participants studied, homocysteine was associated with CAC quantity ($P = .01$) after adjustment for CHD risk factors (age, male sex, total and high-density lipoprotein cholesterol, diabetes, history of smoking, body mass Index, and systolic blood pressure), serum creatinine, and statin and hypertension medication use. When the association was

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assessed in strata based on 10-year CHD risk, homocysteine was significantly ($P = .003$) associated with CAC quantity in participants at Intermediate 10-year risk of CHD (6%–20%) independent of other risk factors but not in those at lower risk or higher risk. **CONCLUSION:** Plasma homocysteine is associated with quantity of CAC Independent of CHD risk factors. When studied in categories of 10-year CHD risk, the association was significant in participants at intermediate risk but not in those at low or high risk. Plasma homocysteine levels may have clinical utility as a marker of CHD risk in such individuals.

Atherosclerosis. 2006 Jun 13; [Epub ahead of print] Links

Homocysteine and coronary heart disease risk in the PRIME study.

Troughton JA, Woodside JV, Young IS, Arveiler D, Amouyel P, Ferrieres J, Ducimetiere P, Patterson CC, Kee F, Yarnell JW, Evans A; on behalf of the PRIME Study Group.

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INTRODUCTION: Despite recent meta-analyses suggesting that homocysteine is an independent predictor of coronary heart disease (CHD), there is debate regarding whether elevated homocysteine may be deleterious only in the presence of other risk factors, with which it acts synergistically to exert a multiplicative effect on CHD risk, emerging only as a CHD predictor in patients with pre-existing risk factors. The Prospective Epidemiological Study of Myocardial Infarction (PRIME) Study is a multicentre prospective study of 10593 men from France and Northern Ireland, investigating cardiovascular risk factors. We investigated: (1) whether higher homocysteine is associated with increased CHD risk in the PRIME case-control cohort; (2) whether homocysteine interacts synergistically with pre-existing CHD risk factors. **METHODS:** Homocysteine was measured in 323 participants who had developed CHD at 5-year follow-up and in 638 matched controls. **RESULTS:** There was no significant difference in homocysteine between cases and controls ($p=0.18$). Homocysteine was significantly higher in current smokers (geometric mean $\mu\text{mol/l}$ (interquartile range $\mu\text{mol/l}$) 9.45 (7.43, 11.75)) compared with non-smokers (8.90 (7.32, 10.70); $p=0.007$). There was a significant interaction between homocysteine, smoking and CHD risk ($\chi^2=10.29$, $d.f.=2$, $p=0.006$). **CONCLUSIONS:** These findings suggest that elevated homocysteine is significantly associated with CHD risk in current smokers.

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