

ABSTRACTS

OMEGA 3 FATS AND MORTALITY, CARDIOVASCULAR DISEASE AND CANCER

There is some evidence to suggest that consuming long-chain omega 3 fatty acids, found in fatty fish and fish oils, is linked to a low incidence of coronary heart disease, specifically in the Inuit people of Greenland. Shorter-chain fats may also be protective. These fats are thought to protect against heart disease by lowering blood pressure and heart rate, reducing serum triglycerides, thrombotic tendency, inflammation and arrhythmias. They may also improve endothelial function, insulin sensitivity, paraoxonase concentrations and plaque stability. But, the evidence is not unequivocal.

The authors of this paper in the *British Medical Journal* systematically reviewed the evidence for an effect of long-chain and shorter-chain omega 3 fatty acids on total mortality, cardiovascular events and cancer. They accessed a staggering 15 159 titles and abstracts and found that the trial results were inconsistent. The pooled estimates showed no strong evidence of reduced risk of total mortality or combined cardiovascular events in participants taking additional omega 3 fats. Further, the studies accessed also showed no evidence of a reduction in cardiovascular events. Additionally, none of the trials suggested an increased risk of cancer with a higher intake of omega 3 fats, but the authors say that clinical harm cannot be ruled out.

This meta-analysis found major variation between studies. However, the major findings were clear – there is no evidence that omega 3 fats have an effect on combined cardiovascular events. In fact, there was no clear evidence of a benefit on health. However, as the authors point out, the variation between studies makes full interpretation difficult and they do not call for the current UK guidelines that say that people in general, and those who have suffered from a myocardial infarction in particular, should eat more oily fish. The authors state that to understand the possible effects of omega 3 fats on health more randomised controlled trials of long duration, that take into account other lifestyle factors, are needed.

Hooper L, *et al.* *BMJ* 2006; published online 24 March.

COFFEE, CYP1A2 GENOTYPE, AND RISK OF MYOCARDIAL INFARCTION

The association between coffee intake and risk of myocardial infarction (MI) remains controversial. Coffee is a major source of caffeine, which is metabolised by the polymorphic cytochrome P450 1A2 (CYP1A2) enzyme. Individuals who are homozygous for the *CYP1A2*1A* allele are 'rapid' caffeine metabolisers, whereas carriers of the variant *CYP1A2*1F* are 'slow' caffeine metabolisers.

These authors set out to determine whether the CYP1A2 genotype modifies the association between coffee consumption and risk of acute non-fatal MI.

They looked at 2 014 people who presented with a first acute non-fatal MI and 2 014 healthy people living in Costa Rica between 1994 and 2004, matched for age, sex, and area of residence. Their genotype was examined using restriction fragment-length polymorphism polymerase chain reaction.

They found that 55% of cases and 54% of controls were carriers of the slow **1F* allele and that intake of coffee was associated with an increased risk of non-fatal MI only among individuals with slow caffeine metabolism, suggesting that caffeine is important for these individuals.

Cornelis MC, *et al.* *JAMA* 2006; **295**: 1135-1141.

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SINGLE SUTURE ASPIRIN AND DEAFNESS

Aspirin may reduce hearing loss caused by loud noise and some antibiotics. Apparently the delicate hairs of the inner ear can be damaged by the oxygen free radicals produced throughout life and in response to loud noises and exposure to certain antibiotics. Salicylates, the active ingredients in aspirin, help to neutralise these free radicals. Researchers gave salicylates to patients who were on gentamicin for acute ear infections and used a placebo in controls. Only 3% of those who took salicylates with their antibiotic developed hearing loss, compared with 13% of those taking a placebo.

New Scientist 2006; 13 May: 8.