THE INFLUENCE OF DIETARY CALCIUM AND VITAMIN E ON COLON CANCER RISK

G.H. McINTOSH, N.A. MAZZEO and J. TURNER

Colon and rectal cancers taken together are the most common form of internal malignancies in populations on a 'western' diet. They outnumber lung cancer in men and breast cancer in women. Australians are amongst the high risk nations, with 8,000 people affected annually. Epidemiological studies have shown that like breast cancer, mortality figures for colon cancer are correlated with fat intake (Cohen, 1987).

Two nutrients, Calcium and Vitamin E, are being studied for their potential to provide protection (Newmark and Mergens, 1980; Newmark, 1987). Both nutrients interact with fat in the diet. Vitamin E is a powerful lipid phase antioxidant and prevents the oxidation of fatty acids. Calcium combines with fatty acids and bile acids to produce nonreactive salts, which are then excreted. Both of the latter are implicated in colon cancer, due to their toxic effects in the hind gut.

We examined the effect of Ca and vit E at human RDI and 5-10 times RDI, using Sprague Dawley rats on a fat modified (20%) AIN76 rodent diet. Calcium (as Ca HPO4) was supplied at 0.1 and 0.5% and vit E (as d-α-tocopherol acetate) at 10 and 100 ppm in a 2x2 factorial experiment. Fat and calcium metabolism and colonocyte proliferation rate (using a colchicine metaphase arrest technique) were measured as end points, the last being a useful marker of colon cancer risk.

Increasing Ca significantly increased fat excretion and increasing vit E had an opposite (and smaller) effect. Vitamin E increase reduced colonocyte proliferation rate by 30%, and increasing Ca had a similar (but not additive) effect.

In a second rat experiment DMH (Dimethylhydrazine) was used to induce colon tumours. Tumour incidence was affected by vit E levels, the low vit E showing a reduced incidence. Calcium, however, produced a reduction in tumour burden at high levels. In this respect, Ca and vit E had opposite effects.

We conclude that calcium content of diet has a significant influence on fat utilisation and shows evidence at high levels of lowering the tumour burden. Vitamin E and calcium were effective in lowering colonocyte proliferation rate, if supplemented to high fat diets at levels above current RDI recommendations. Proliferation rate in the first experiment did not predict tumour incidence in the second experiment.

The optimal levels of these nutrients for prevention and the nature of interaction needs further elucidation.

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CSIRO Division of Human Nutrition, PO Box 10041 Gouger Street, Adelaide SA 5000