ABSTRACT

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Diet and Cancer – New findings

The association between diet and cancer continues to be investigated and debated. International differences in cancer incidence and changes seen with migration suggest that environmental factors may be involved. Diet has been proposed to influence cancer risk in various ways – food may contain cancer-causing constituents; excessive intake of food may increase the risk of cancer; certain dietary constituents may protect against cancer. Recently new information has accumulated regarding the association between diet and cancer. While total fat intake has been implicated in some types of cancer the evidence is inconsistent. Moreover, some new findings suggest that total energy intake may be a more important factor. Furthermore, the consumption of very low fat diets to prevent diseases such as cancer or coronary heart disease, has been associated with higher total mortality.

Findings from both human and experimental animal studies suggest that a high intake of fat increases the incidence of cancers of the breast, colon, prostate, and possibly the respiratory tract. In countries, such as Japan, where per capita fat consumption is traditionally low (10% of energy), the risk of cancers of the breast and colon is much lower but stomach cancer is much higher than in western nations where fat contributes about 40% of energy. Similarly, the cancer mortality of Japanese who have migrated to the United States increases to a level comparable to that of the host nation. A high fat diet has been associated with poorer survival time, following the diagnosis of breast cancer. Findings of an association between dietary fat and cancer are by no means consistent and this can be explained in part by the long latency period between exposure to cancer-causing agents and the detection of the disease. In addition, fat in the diet does not exist in isolation and other food constituents may modify cancer risk.

Studies of individuals or populations within a country, as opposed to comparisons between countries, often fail to support an association between fat and cancer. For example, colon and breast cancer incidence is similar in American Mormons and Seventh Day Adventists, despite their different fat intakes. Similarly a difference in the incidence of cancer between non-Maoris and Maoris in New Zealand has been observed despite comparable fat intake of these two populations. Similar inconsistencies have arisen in studies with experimental animals.

Unlike most human epidemiological investigations, animal studies reveal that the type of fat may be significant. When fat intake is low, polyunsaturated fatty acids stimulate mammary and colon tumours to a greater extent than saturated fatty acids. In contrast, when fat intake is higher and the need for essential fatty acids is met, there is little distinction between the different types of fat. The relative importance in man of the different types of fat needs to be resolved.

Recent human studies have observed an association between low blood cholesterol (less than 185mg/100ml) and cancer mortality, particularly colon cancer in males. While this association has been observed in most populations investigated, it is not yet clear whether low blood cholesterol is simply a metabolic response to preclinical cancer.

The mechanism by which dietary fat influences tumorigenesis is unclear. There is support from animal experiments for a 'promoter' effect following exposure to a carcinogen. Among the theoretical mechanisms for the involvement of fat in breast cancer are alterations in the hormonal

environment, changes in the composition of cellular membranes, and inhibition of the immune response (especially by polyunsaturated fatty acids). In colon cancer, effects of fat on bile acid stimulation and on the intestinal flora have been suggested as possible mechanisms.

The influence of energy intake on tumour growth has been recognised for many years. However, recently a long term prospective study of 75,000 overweight (by 40%) men and women was carried out by The American Cancer Society, it revealed that overweight women had higher mortality rates for cancers of the ovary, breast, endometrium, gall bladder and cervix than controls of normal weight. Obesity seems to be associated with an increased risk of breast cancer in post-menopausal women, whereas in pre-menopausal women it has an opposite effect. Obese men in the American Cancer Society study had higher mortality rates for colorectal and prostate cancers compared with controls. Findings from several experimental animal studies suggest that energy intake may be a more critical determinant of some types of cancer than dietary fat.

How far cancer can realistically be reduced by implementing dietary modifications in general and changes in fat intake in particular is unknown. In various coronary heart disease intervention studies employing a fat-modified diet, the decrease in coronary heart disease was offset by a corresponding increase in death from other causes, such as cancer. As a result the total mortality in the study population was unchanged. The long term consequences of consuming a diet low in total fat, saturated fat and high in polyunsaturated fat are unknown. Diets high in polyunsaturated fat, for example, have been implicated in gallstone formation, cancer incidence and changes in the composition and fluidity of cell membranes. When establishing dietary recommendations for the general population, it is important to consider not only the potential benefits but how changes may influence other diseases in the long-term.

Reference

Diet, Nutrition and Cancer: New Findings (1986) Dairy Council Digest (USA) 57, (2), 7-12.
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