Diet and Cancer*

The causes of cancer are complex and incompletely understood. Much research has been carried out in an attempt to gain an insight into the nature and aetiology of this disease. Particular attention has been focused on the role of certain dietary components and how they might influence an individual's risk of developing cancer. These components include fat, alcohol, fibre, vitamins A, C and E and selenium. The British Nutrition Foundation has recently produced a briefing paper which reviews the work on this topic.

The evidence for an association between dietary fat and cancer comes mainly from epidemiological studies which show a positive correlation between total fat intake per capita and incidence of cancer, particularly of the breast and colon. The paper cites several studies which give conflicting results. The inconsistency of these findings may be a reflection of the crudity of the dietary data and the fact that an accurate estimation of fat intake is very difficult. A number of studies have examined dietary fat intake in individuals in relation to specific types of cancer. Case-control and prospective studies have also shown both positive and negative associations between fat intake and breast and bowel cancer. It is possible that some of the inconsistencies in the data relating to bowel cancer are due to the effects of other dietary constituents such as fibre. It has been suggested that high fat and low fibre intakes may act synergistically.

Both international and case-control studies have shown a weak positive association between calorie intake and cancer incidence, particularly of the colon, rectum and breast. However, people who have a higher calorie intake tend to consume more of all the major nutrients making it difficult to separate the effects of specific dietary components from the effects of total calorie intake. There is recent evidence that obesity may be a risk factor for certain cancers, particularly endometrial, gall bladder, breast, ovarian, colo-rectal and prostate cancers. The opposite relationship seems to occur for lung cancer, with the thinnest people having the highest risk. One of the mechanisms proposed for the observed relationship between breast cancer and overweight is that the adrenal hormones are converted into oestrogens in adipose tissue. Oestrogens are believed to play a key role in the aetiology of breast cancer.

The findings from a number of studies relating protein or meat consumption and cancer risk indicate that such an association is very weak. Some correlations have been observed between high cholesterol intakes and certain cancers and also between high serum cholesterol and cancer mortality. Conversely, there is evidence that individuals with very low levels of serum cholesterol have a greater risk. It has been suggested that a low serum cholesterol level is an early indicator of incipient cancer and is not, in itself, a cause of cancer. However, this explanation has not been entirely substantiated and the evidence of a link between dietary or serum cholesterol and cancer risk remains uncertain.

**Source: National Dairy Council Nutrition Service, London.*

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The relationship between vitamin A and beta-carotene intakes and cancer risk has also been investigated. Studies which have measured blood levels of retinol have been criticised on the grounds of poor dietary data, inadequate sample numbers and poor reliability of the method for retinol determination. Furthermore, blood retinol does not reflect dietary intake under normal conditions due to the substantial store of vitamin A in the liver. Circulating beta-carotene, on the other hand, is influenced directly by dietary intake and is currently receiving more interest. However, one notable review suggests that the balance of evidence indicates a protective effect of higher blood concentrations of retinol and higher dietary intakes of beta-carotene. Conflicting evidence for a link between vitamin C intake, vitamin E and risk of cancer also exists and these studies have been criticised because of their unreliable dietary data. However, there appears to be considerable support for the protective effect of selenium against cancer.

Studies investigating the role of dietary fibre and cancer risk have generally indicated a protective effect. A few studies have suggested that different fibre fractions may have different physiological effects. Recently, attention has focused on the relationship between faecal bile acids and fibre intake, since faecal bile acid concentration is positively associated with the incidence of bowel cancer. The briefing paper states that more work is required to ascertain which factors other than fibre binding capacity influence bile acid concentration in the colon. Several studies have confirmed a relationship between alcohol consumption and cancer of the oesophagus and possibly cancers of the buccal cavity, pharynx, breast and rectum and an interaction with the risk of smoking. In addition, different alcoholic beverages appear to carry distinctive risks.

It is possible that interactions between dietary components might effect the carcinogenic process. Examples include the involvement of vitamins C and E with selenium and, possibly, beta-carotene in protecting against oxidative processes; the effects of fat and dietary fibre on the bile acid levels; and the interaction of nitrite from, for example, vegetables, with other food components to form potentially carcinogenic nitrosamines. Many studies are therefore complicated by the fact that the dietary data is based on groups of foods which, obviously, contain more than one dietary component. Correlations between cancer risk and spceific dietary components are very difficult to ascertain. The BNF briefing paper concludes that current evidence on diet and cancer risk remains weak and advice can only be given to the public in most cautious terms. A comprehensive list of references on the topic is given in the paper.

Reference

The British Nutrition Foundation (1988). Are there dietary influences in the causation of cancer? Briefing Paper No. 14.