International Journal of Yoga, Physiotherapy and Physical Education
ISSN: 2456-5067
Impact Factor: RJIF 5.24
www.sportsjournal.in
Volume 3; Issue 2; March 2018; Page No. 134-137

Diet, nutrition for prevention of cancer

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Abstract
Objective: To assess the epidemiological evidence on diet and cancer and make public health recommendations.
Design: Review of published studies, concentrating on recent systematic reviews, meta-analyses and large prospective studies. Conclusions and recommendations: Overweight/obesity increases the risk for cancers of the oesophagus (adenocarcinoma), colorectum, breast (postmenopausal), endometrium and kidney; body weight should be maintained in the body mass index range of 18.5-25 kg/m2, and weight gain in adulthood avoided. Alcohol causes cancers of the oral cavity, pharynx, oesophagus and liver, and a small increase in the risk for breast cancer; if consumed, alcohol intake should not exceed 2 units/d. Aflatoxin in foods causes liver cancer, although its importance in the absence of hepatitis virus infections is not clear; exposure to aflatoxin in foods should be minimised. Chinese-style salted fish increases the risk for nasopharyngeal cancer, particularly if eaten during childhood, and should be eaten only in moderation. Fruits and vegetables probably reduce the risk for cancers of the oral cavity, oesophagus, stomach and colorectum, and diets should include at least 400 g/d of total fruits and vegetables. Preserved meat and red meat probably increase the risk for colorectal cancer; if eaten, consumption of these foods should be moderate.

Keywords: Diet, Nutrition, Cancer, Review

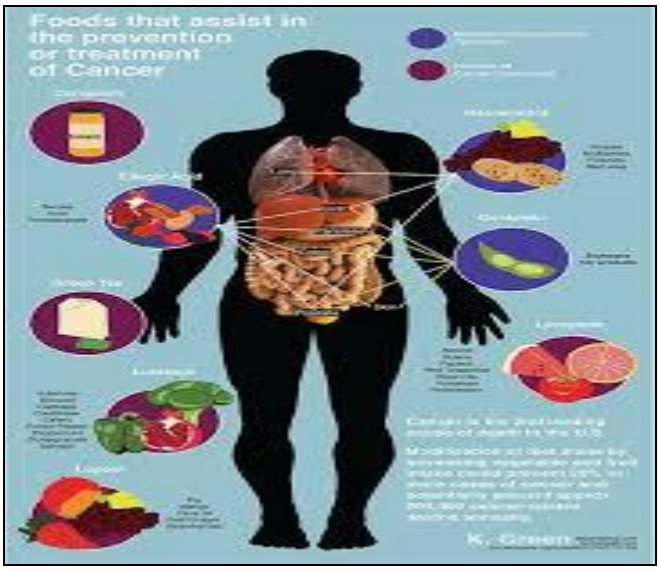
Introduction

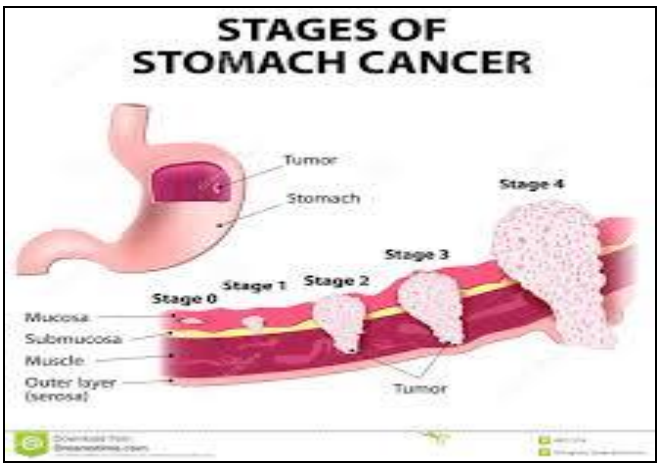
- **Review of the role of diet in the aetiology of the major cancers**

Cancers of the oral cavity, pharynx and oesophagus Cancers of the oral cavity, pharynx and oesophagus were estimated to account for 867,000 cases and 582,000 deaths in 200011. Incidence rates of these cancers vary widely between populations; for example, oesophageal cancer is over a hundred times more common in parts of Central Asia, China and Southern Africa than in most parts of Europe, North America and West Africa. In developed countries, the main risk factors are alcohol and tobacco, and up to 75% of these cancers are attributable to these two lifestyle factors. The mechanism of the effect of alcohol on these cancers is not known, but may involve direct effects on the epithelium. Overweight/obesity is an established risk factor specifically for adenocarcinoma of the esophagus. In developing countries, around 60% of cancers of the oral cavity, pharynx and esophagus are thought to be due to micronutrient deficiencies related to a restricted diet that is low in fruits and vegetables and animal products 3,16; it should be noted, however, that the evidence for a protective effect of fruits and vegetables is largely derived from case-control studies and there are few data yet from prospective studies. The relative roles of various micronutrients are not yet clear, but deficiencies of riboflavin, folate, vitamin C and zinc may all be important. There is also consistent evidence that consuming drinks and foods at a very high temperature increases the risk for these cancers. The results of trials in Linxian, China, aimed at reducing esophageal cancer rates with micronutrient supplements, have been promising but not definitive.

- **Nasopharyngeal cancer**

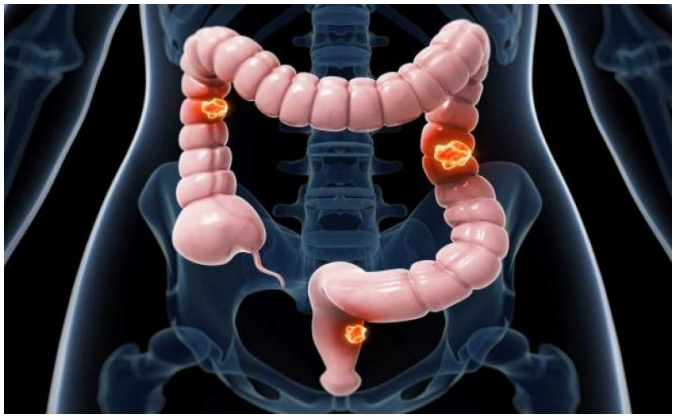
This is particularly common in Southeast Asia, and has been consistently associated with a high intake of Chinesestyle salted fish, especially during early childhood, as well as with infection with the Epstein–Barr virus. Chinese-style salted fish is a special product which is usually softened by partial decomposition before or during salting; other types of salted fish have been studied and not found to be convincingly associated with the risk for developing nasopharyngeal cancer.





- **Stomach cancer**

Stomach cancer was estimated to account for 876,000 cases and 647,000 deaths in 200011. Until about 20 years ago stomach cancer was the most common cancer in the world, but mortality rates have been falling in all Western countries and stomach cancer is now much more common in Asia than in Europe or North America. Infection with the bacterium H. pylori is an established risk factor, but not a sufficient cause, for the development of stomach cancer. Diet is also thought to be important in the aetiology of this disease, and dietary changes are implicated in the recent decline in stomach cancer incidence and mortality rates in many countries. Substantial evidence, mainly from case-control studies, suggests that risk is increased by high intakes of some traditionally preserved salted foods, especially meats and pickles, and with salt per se, and that risk is decreased by high intakes of fruits and vegetables, perhaps due to their vitamin C content. However, evidence from prospective studies does not clearly support a protective effect for fruits and vegetables. The introduction of refrigeration has also been associated with decreased risk, probably through reducing intakes of salted foods and facilitating year-round fruit and vegetable availability.



- **Colorectal cancer**

Colorectal cancer is the third most common cancer in the world and was estimated to account for 945,000 cases and 492,000 deaths in 200011. Incidence rates are approximately fold higher in developed than in developing countries. It has been suggested that diet-related factors may account for up to 80% of the between-country differences in rates. The best established dietary-related risk factor is overweight/ obesity. Alcohol probably causes a small increase in risk. Adult height, which is partly determined by the adequacy of nutrition in childhood and adolescence, is weakly associated with increased risk, and physical activity
has been consistently associated with a reduced risk. These factors together, however, do not explain the large variation between populations, and there is almost universal agreement that some aspects of a Western diet are a major determinant of risk.

- **Meat**

International correlation studies show a strong association between per capita consumption of meat and colorectal cancer mortality, and several mechanisms have been proposed through which meat may increase cancer risk. Mutagenic heterocyclic amines and polycyclic aromatic hydrocarbons can be formed during the cooking of meat at high temperatures and nitrites and their related compounds found in smoked, salted and some processed meat products may be converted to carcinogenic N-nitroso compounds in the colon. In addition, high iron levels in the colon may increase the formation of mutagenic free radicals. The results of observational studies of meat and colorectal cancer have varied3; a recent systematic review concluded that preserved meat is associated with an increased risk for colorectal cancer but that fresh meat is not and most studies have not observed positive associations with poultry or fish. However, mortality
rates for colorectal cancer are similar in Western vegetarians and comparable non-vegetarians. Overall, the evidence is not conclusive but suggests that high consumption of preserved and red meat probably increases the risk for colorectal cancer.

- **Fat**

As with meat, international correlation studies show a strong association between per capita consumption of fat and colorectal cancer mortality. Possible mechanisms proposed to explain such an association are that a high fat
intake may increase the levels of cytotoxic free fatty acids or secondary bile acids in the lumen of the large intestine. However, the results of observational studies of fat and colorectal cancer have, overall, not been supportive of an association with fat intake, especially after adjusting for total energy intake.

- **Fruits, vegetables and fibre**

Burkitt suggested that the low rates of colorectal cancer in Africa were due to the high consumption of dietary fibre, and there are several plausible mechanisms for a protective effect. Fibre increases stool bulk and speeds the transit of food through the colon, thus diluting the gut contents and perhaps reducing the absorption of carcinogens by the colonic mucosa. Fermentation of fibre (and resistant starch) in the large intestine produces short chain fatty acids such as butyrate, which may protect against colorectal cancer through the ability to promote differentiation, induce apoptosis and/or inhibit the production of secondary bile acids by reducing luminal pH. Many case-control studies of colorectal cancer

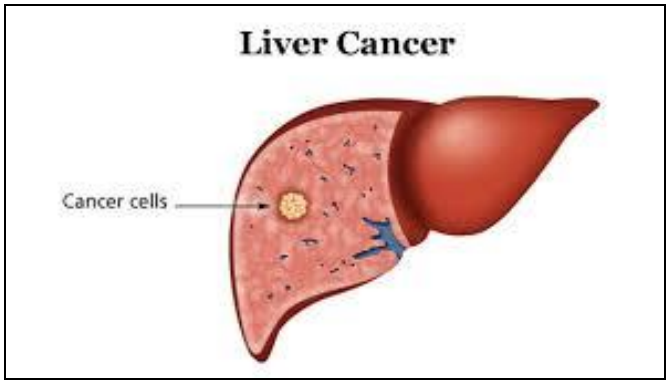
have observed moderately lower risk in association with high consumption of dietary fibre, and/or fruits and vegetables but the results of recent large prospective studies have been inconsistent.

- **Folate**

Some recent prospective studies have suggested that a methyl-deplete diet (i.e. a diet low in folate and methionine and high in alcohol) is associated with an increased risk of colon cancer. Also, use of folic acid containing
multiple vitamin supplements has been associated with lower risk of colon cancer. A diminished folate status may contribute to carcinogenesis by alteration
of gene expression and increased DNA damage and chromosome breakage. The finding that a common polymorphism in the methylenetetrahydrofolate reductase gene involved in folic acid metabolism may also be associated with colorectal cancer strengthens the hypothesis that dietary folate may be an important factor in colorectal carcinogenesis.

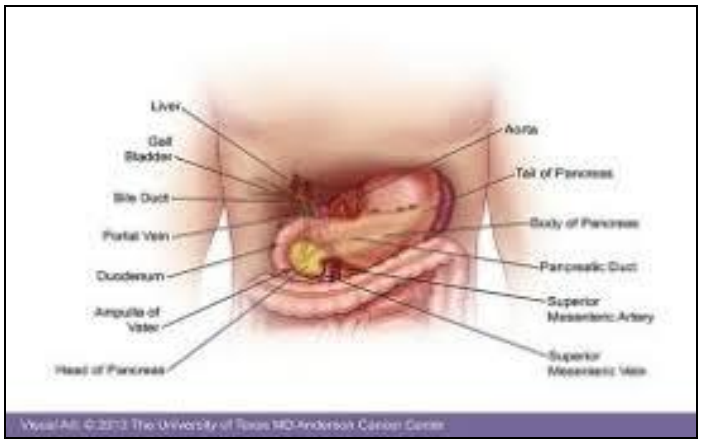
- **Calcium**

Another promising hypothesis is that relatively high intakes of calcium may reduce the risk for colorectal cancer, perhaps by forming complexes with secondary bile acids in the intestinal lumen or by inhibiting the hyper proliferative effects of dietary haem. Several observational studies have supported this hypothesis and two trials have suggested that supplemental calcium may have a modest protective effect on the recurrence of colorectal adenomas. More data are needed to evaluate this hypothesis.



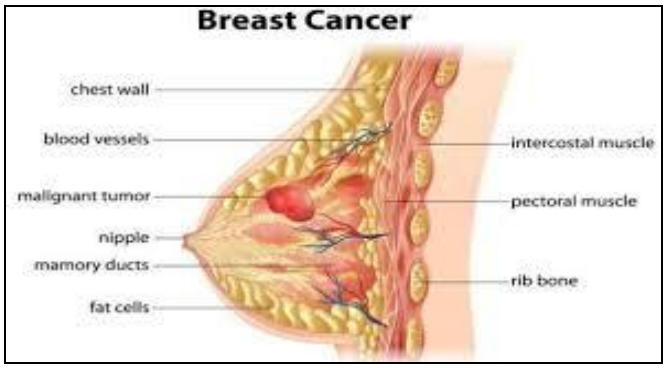
- **Cancer of the liver**

Liver cancer was estimated to account for 564,000 cases and 549,000 deaths in 200011. Approximately 75% of cases of liver cancer occur in developing countries, and liver cancer rates vary over fold between countries, being much higher in sub-Saharan Africa and Southeast Asia than in Europe and North America. The major risk factor for hepatocellular carcinoma, the main type of liver cancer, is chronic infection with hepatitis B, and to a lesser extent, hepatitis C virus. Ingestion of foods contaminated with the mycotoxin aflatoxin is an important risk factor among people in developing countries with active hepatitis virus infection. Excessive alcohol consumption is the main diet-related risk factor for liver cancer in Western countries, probably via the development of cirrhosis and alcoholic hepatitis. Little is known about possible nutritional cofactors for viral carcinogenesis, but this may be an important area for research.



- **Cancer of the pancreas**

Cancer of the pancreas was estimated to account for 216,000 cases and 214,000 deaths in 2000 and is more common in Western countries than in developing countries. Time trends suggest that both incidence and mortality for cancer of the pancreas are increasing in most parts of the world, although some of this apparent increase may be due to improvements in diagnostic methods. Overweight/obesity possibly increases the risk. Some studies have suggested that risk is increased by high intakes of meat, and reduced by high intakes of vegetables, but these data are not consistent and come mostly from case-control studies. Over the next few years there will be substantially more prospective data on diet and cancer of the pancreas, and it is possible that more clear-cut associations with dietary factors will emerge.



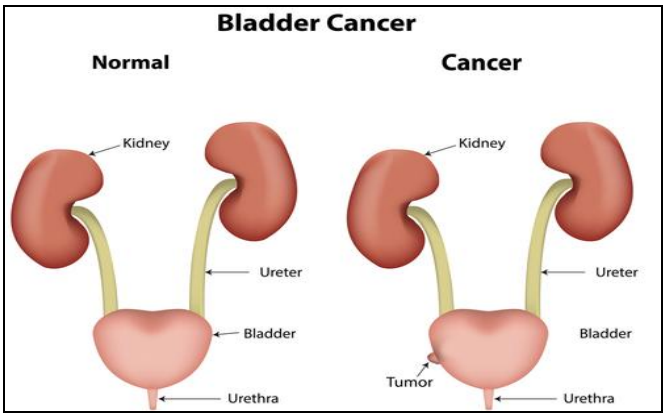
- **Breast cancer**

Breast cancer is the second most common cancer in the world and the most common cancer among women. Breast cancer was estimated to account for 1,105,000 cases and 373,000 deaths in women in 200011. Incidence rates are about five times higher in Western countries than in less developed countries and Japan7. Much of this international variation is due to differences in established reproductive risk factors such as age at menarche, parity and age at births, and breastfeeding, but differences in dietary habits and physical activity may also contribute. In fact, age at menarche is partly determined by dietary factors, in that restricted dietary intake during childhood and adolescence leads to delayed menarche. Adult

height, also, is weakly positively associated with risk, and is partly determined by dietary factors during childhood and adolescence. Oestradiol and perhaps other hormones play a key role in the a etiology of breast cancer, and it is possible that any further dietary effects on risk are mediated by hormonal mechanisms.

- **Overweight/obesity**

Obesity increases breast cancer risk in postmenopausal women by around 50%, probably by increasing serum concentrations of free oestradio. Obesity does not increase risk among premenopausal women, but obesity in premenopausal women is likely to lead to obesity throughout life and therefore to an eventual increase in breast cancer risk.



- **Bladder cancer**

Cancer of the urinary bladder was estimated to account for 336,000 cases and 132,000 deaths in 200011. The geographic variation in incidence is about fold, with relatively high rates in Western countries. Smoking increases the risk for bladder cancer. Studies suggest that high intakes of fruits and vegetables may reduce risk, but this is not established and more prospective data are needed.

Conclusions

Since the 1981 Doll and Peto review on diet and cancer mortality, about one third of cancers have generally been thought to be related to dietary factors. More recent evidence suggests that this number may be too high, but a revised quantitative estimate is beyond the scope of this review. Among the diet-related factors, overweight/obesity convincingly increases the risks of several common cancers. After tobacco, overweight/obesity appears to be the most important avoidable cause of cancer in populations with Western patterns of cancer incidence. Among non-smoking individuals in these populations, avoidance of overweight is the most important strategy for cancer prevention.

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