DIET AND CANCER

Diet has been linked to the development of cancer in clinical and experimental studies. The foods comprising the diet of humans are complex mixtures of chemicals modified by many events that occur between the field and the table. The overall incidence of cancer and the incidence of cancers of specific organ sites differ strikingly from one country to another and often from one region to another within a country. It is absolutely clear that cigarette smoking is the cause of approximately one quarter of all the fatal cancers in the United States and approximately 20% of all deaths caused by cancer. The public is now asking about the causes of cancer that are not associated with smoking. What are the causes, and how can these cancers be avoided? Unfortunately, it is not yet possible to make firm scientific document regarding the association between diet and cancer. We are in an interim stage of knowledge. The search for the causes of cancer has become an important branch of cancer research. In the course of research, it has become clear that most cancers have external causes and, in principle, should therefore be preventable. For example, Blacks and Japanese residing in the United States develop the spectrum of cancers that is typical for the United States but different from that in Africa and Japan. But what might these external causes be? Many factors in our environment are potential causes of cancer. They include substances in the air we breathe, the water we drink, the region in which we work and live, and the foods we eat. The results of the studies of the association between diet and cancer show that the differences in the rate at which various cancers occur in different human populations are often correlated with differences in diet. However, the influence of diet on spontaneous and experimentally-induced cancers is not easily investigated because the underlying mechanisms and molecular biology of cancers are still not fully understood.

DIETARY PATTERNS AND COMPONENTS OF FOOD

Since the turn of the century, new methods of processing and storage have resulted in a proliferation of the kinds and numbers of food items. In attempting to determine which constituents of food might be associated with cancer, some evidences suggest that some types of diets and some dietary components (e.g., high fat diets, or the frequent consumption of salt-cured, salt-pickled, and smoked foods) tend to increase the risk of cancer.
The mechanisms responsible for these effects are not fully understood.

**TOTAL CALORIC INTAKE**

The epidemiological evidence supporting total caloric intake as a risk factor for cancer is slight and largely indirect. Much of it is based on associations between body weight and obesity and cancer. Studies that have been evaluated both caloric and fat intake suggest that fat intake is the more relevant variable. (1, 2)

There are neither epidemiological nor experimental studies which show a clear relationship between caloric intake and the risk of cancer. The studies conducted in animals show that a reduction in total fat intake decreases the age-specific incidence of cancer. The evidence for human is less clear (3, 4).

**LIPIDS (FAT AND CHOLESTEROL)**

**FATS:** Epidemiological studies have repeatedly shown an association between dietary fat and the occurrence of cancer at several sites, especially the breast, prostate, and large bowel. The high incidence mortality rate of breast cancer have been shown to correlate strongly with higher per capita fat consumption (5, 6).

Like breast cancer, increased risk of large bowel cancer has been associated with high fat intake in both correlation and case control studies (7). The data on prostate cancer are more limited, but they too suggest that an increased risk is related to high levels of dietary fat. Animal consuming low fat diets have a lower tumor incidence and polyunsaturated fats appear to be more effective than saturated fat in enhancing tumorigenesis (8).

**CHOLESTEROL:** The relationship between dietary cholesterol and cancer is not clear both in animal and human.

**PROTEIN**

Dietary protein has often been associated with cancers of the breast, endometrium, prostate, colorectum, pancreas, and kidney. However, since the major dietary sources of protein contain a variety of other nutrients and non-nutritive components, the association of protein with cancer at these sites may not be direct but, rather, could reflect the action of another constituent concurrently present in protein rich foods. On the other hand, there are evidences that high intake of dietary protein increases risk of cancers at a number of different sites because of the very high correlation between fat and protein in the diets. It seems likely that dietary fat is the more active component, but evidence does not completely preclude the existence of an independent effect of protein (1, 5, 8).

**CARBOHYDRATES**

The principal carbohydrates in foods are sugars, starches, and cellulose. Some studies suggest that a high intake of refined sugar or starch increases the risk of cancer at certain sites: pancreas, breast, and stomach. The results are insufficient to permit any firm conclusion to be drawn. However, excessive carbohydrate consump-
tion contributes to caloric excess, and this in turn has been implicated as a modifier of carcinogenesis. In one study, the intake of sugar was correlated with increased mortality from pancreatic cancer in women only, and the intake of potatoes was correlated with increased mortality from liver cancer in both sexes\(^5\). In other studies, a high intake of refined sugar and a low intake of starch have been associated with an increased incidence of breast cancer\(^3\). Frequent consumption of starch has been associated with a high incidence of gastric cancer in one case-control study\(^10\) and with esophageal cancer in another\(^11\). A few recent studies suggest that dietary lactose combined with vitamin A deprivation and long-term feeding of high levels of sucrose and xylitol may contribute to carcinogenesis.

**DIETARY FIBER**

Recently, attention has been directed toward the physiological significance of dietary fiber, which generally included indigestible carbohydrates and carbohydrate-like components of food such as cellulose, lignin, hemicellulose, pentosans, gums, and pectins. The principal foods that provide dietary fiber are vegetables, fruits, and whole grain cereals. Most epidemiological studies on fiber have examined the hypothesis that high fiber diets protect against colorectal cancer. Results of correlation and case-control studies of dietary fiber have sometimes supported and sometimes contradicted this hypothesis. However, the correlation study indicated that the incidence of colon cancer was inversely related to the intake of one fiber component—pentosan fraction, which is found in whole wheat products and other food items. Laboratory experiments also have indicated that the consumption of some high fiber ingredients (e.g., cellulose and bran) inhibits the induction of colon cancer by certain chemical carcinogens. Unfortunately, the results are inconsistent\(^10,12\).

**VITAMINS**

In recent years, there has been considerable interest in the role of vitamins A, C, and E in the genesis and prevention of cancer. In contrast, less attention has been paid to the B vitamins and others such as vitamin K.

**VITAMIN A**: A growing accumulation of epidemiological evidence indicates that there is an inverse relationship between the risk of cancer and the consumption of foods that contain vitamin A (e.g., liver) or its precursors (e.g., the carotenoids in green and yellow vegetables). Most of the data do not show whether the effects are due to carotenoids, to vitamin A itself, or to some other constituent of these foods. Studies in laboratory animals indicate that vitamin A deficiency generally increases susceptibility to chemically induced neoplasia and that an increased intake of the vitamin appears to protect against carcinogenesis in most, but not all cases. Because high doses of
vitamin A are toxic, many of these studies have been conducted with its synthetic analogues (retinoids), which lack some of the toxic effects of the vitamin. Retinoids have been shown to inhibit chemically induced neoplasia of the breast, urinary bladder, skin, and lung in animals.\textsuperscript{13,14} The epidemiological evidence is sufficient to suggest that foods rich in carotenoids or vitamin A are associated with a reduced risk of cancer.

\textit{Vitamin C}: The epidemiological data pertaining to the effect of vitamin C on the occurrence of cancer is not extensive. Furthermore, it provides mostly indirect evidence since it is based on the consumption of foods, especially fresh fruits and vegetables, known to contain high concentrations of the vitamin, rather than on actual measurements of vitamin C intake. The results of several case-control studies and a few correlation studies suggest that the consumption of vitamin C containing foods is associated with a lower risk of certain cancers, particularly gastric and esophageal cancer.\textsuperscript{15,16,17}

\textit{Vitamin E}: Vitamin E, like ascorbic acid, inhibits the formation of nitrosamines in vivo and in vitro. Vitamin E is present in a variety of commonly consumed foods (particularly vegetable oils, whole grain cereal products, and eggs). However, there are no reports about the effect of this vitamin on nitrosamine-induced neoplasia. Limited evidence from studies in animals suggests that vitamin E may also inhibit the induction of tumorigenesis by other chemicals. The data are not sufficient to permit any firm conclusion to be drawn about the effect of vitamin E on cancer in humans.

\textbf{MINERALS}

Of the many minerals present in the diet of humans, there are 9 minerals that have been suspected of playing a role in carcinogenesis i.e. Selenium, Iron, Copper, Zinc, Molybdenum, Iodine, Asenic, Cadmium and Lead. The data is still insufficient to make definite conclusion about the causation and the prevention of cancers of those minerals.

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