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Plant Foods And Cancer Risk — Science And Tradition

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Particular vegetables and fruits have long been believed to be useful in the prevention and cure of disease. Western medicine, until quite recently, largely involved the prescription of specific plants and foods. In ancient Egypt, then later in Greece and Rome, a wide variety of uses were found for many different plants and extracts[1]. Garlic was considered a holy plant in Egypt. Cruciferous vegetables were cultivated primarily for medicinal purposes and were used therapeutically against several acute and chronic disorders[2]. Celery, cucumber, endive, and a variety of herbs and vegetable juices had standard uses. Peas were used in a condition that may have been angina pectoris.

The Romans believed that lentils were a cure for diarrhoea and conducive to an even temper (it is not clear whether these are related). Several fruits also were used therapeutically: the citron (similar to the lemon) was believed to act as an antidote for poisons; raisins and grapes were incorporated into oral preparations, enemas, inhalations and topical applications[3].

The medical traditions of India and China, particularly, have retained and extended much of this therapeutic knowledge. Today, in the developed world once again, there are many interesting properties ascribed to edible plants, herbs, spices and foods. Until relatively recently, however, both ancient and modern therapies and preventive strategies were based on many generations of clinical experience and not on evidence derived from epidemiological and experimental studies.

DIET AND CANCER

In 1981, Doll and Peto produced a wide estimate of 10 to 70 per cent of all cancer as being attributable to diet[4]. This conclusion was based on studies showing increased risk in association with certain foods — particularly foods of animal origin. Over the past decade, however, many studies have examined the effect of plant food consumption on health and disease. High consumption of vegetables and fruit is protective against cancers at many sites; this conclusion appears to be better supported by literature than almost all other dietary hypotheses.

There are many biologically plausible reasons why consumption of vegetables and fruit might deter the occurrence of cancer. These include the presence of potentially anticarcinogenic substances such as carotenoids, vitamin C, vitamin E, selenium, dietary fibre, dithiolthiones, isothiocyanates, indoles, phenols, protease inhibitors, allium compounds, plant sterols, limonene and others. However, the crucial issue is the complexity and variety of the mixtures, not the specific dose of one or two compounds.

This paper presents an update of our earlier review of the role of plant foods — particularly vegetables and fruit[5,6]. It presents, in abbreviated form, the available data on the question of whether the consumption of vegetables and fruit is associated with a lower risk of cancer.

COHORT STUDIES

At least 21 cohort studies[7] have examined the relationship between vegetable and fruit consumption and different types of cancer. Of these, 19 found an inverse association for at least one category of vegetable and/or fruit, and in 12 studies statistically significant reductions in risk were reported. Of these studies, four reported on all cancer sites; four on lung cancer; three each on colorectal and stomach cancers; two each on pancreatic, bladder and breast cancers; and one on prostate cancer.

It is lung cancer for which the cohort study evidence is the most consistent; inverse associations for vegetable and/or fruit consumption have been shown in populations of Norwegian men, postmenopausal US women, Seventh Day Adventists, and members of a retirement community in California. The association between

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colon cancer and vegetable consumption is not as consistent in the cohort studies as in the case-control studies reviewed below.

CASE-CONTROL STUDIES

Most of the evidence for an inverse association with vegetable and fruit consumption comes from case-control studies. As early as 1933, such a study by Stocks and Karn[8] in Britain suggested such an association between intake of certain vegetables against cancer at all sites. Four hundred and sixty-two cancer patients and 435 patients without cancer, each of whom provided a diet history, were compared.

The most common cancers were those of the breast, the colon and rectum, the uterus and the tongue. Cases reported lower consumption of carrots, turnips, cauliflower, cabbage, onions, watercress and beetroot than controls. Intake of these vegetables combined was statistically significantly lower in the cases than in the controls. Although the methods employed in this study were crude by modern standards, it is striking to note that this apparently protective effect of vegetable consumption and of milk, along with the risk-enhancing effects of pipe smoking and beer drinking were the major findings of this study.

Another study, in the same year in India, although less rigorous in design, again found that vegetable consumption was higher in those who did not develop oral cancer compared with those who did[9].

Since that time, nearly 200 more case-control studies worldwide have examined the relationship between vegetable and fruit consumption and cancer risk. Stomach cancer has been the most studied, followed by cancers of the colon, oesophagus, lung, oral cavity and pharynx, rectum and breast; more than 10 studies have focussed on each of these sites. Statistically significant inverse associations have been reported for one or more vegetable and/or fruit categories in more than 70 per cent of the studies for cancers of the following sites: stomach, oesophagus, lung, oral cavity and pharynx, endometrium, pancreas, colon and skin. Prostate cancer is the only cancer for which the majority of studies have not reported at least one statistically significant inverse association; and indeed, for prostate cancer, no study shows an inverse association. Some of these sites will now be reviewed in more detail.

STOMACH CANCER

Thirty-one case-control studies dealing with the relationship between vegetable and fruit consumption and stomach cancer have been reported. Twenty-eight of these studies found an inverse association between cancer and consumption of one or more vegetable and/or fruit.

The most consistent and abundant findings appear to be for the consumption of fruit, raw vegetables (including lettuce) and allium vegetables. Contrary to the pattern of inverse associations between intakes of the above vegetables and fresh fruits and stomach cancer, several studies suggested that consumption of potatoes and canned fruit were positively associated with risk.

Potential confounders of the relationship between stomach cancer and vegetable and fruit consumption include socioeconomic status. Lower socioeconomic status (as measured by education level, occupation, and/or income) was found to correlate with stomach cancer risk in many of the studies. In addition, the study by Correa *et al*[10] was conducted in Louisiana where stomach cancer rates are much higher among blacks, who are generally poor, as compared to whites in the area. Diet is likely to be correlated with socioeconomic status and the consumption of fresh vegetables and fruits, which tend to be relatively expensive, may be much lower among the lower socioeconomic sections of a population. Nonetheless, several of the odds ratios were adjusted for the confounding effects of occupation, education, ethnicity and/or income as surrogates for socioeconomic status. These studies still noted a statistically significant inverse association between vegetable and fruit consumption and stomach cancer.

Consumption of pickled, fermented, salted and/or smoked foods, consumption of alcohol, consumption of large amounts of carbohydrate (as potatoes or rice), infrequent meals, non-ownership of a refrigerator, poor dentition, residence in a rural vs urban area (which may be tied to socioeconomic status), history of chronic gastritis, blood type O and smoking are other risk factors for stomach cancer elucidated by some of the studies reviewed here. Smoking is not as strong a risk factor for cancer of the stomach as for cancers of the respiratory and upper gastrointestinal tracts.

COLON CANCER

Fifteen of the 21 case-control studies dealing with colon cancer found a statistically significant inverse relationship between cancer and at least one index of vegetable and fruit consumption. Among specific categories of vegetables, the results for cruciferous vegetables were the most striking in that most of the studies presenting findings for cruciferous vegetables found higher intake to be associated with lower risk.

Most of the studies showing no significant association or only positive associations with vegetable and fruit consumption were among the earliest conducted, which may be a reflection of more crude dietary methodology or perhaps less attention paid to detail in gathering fruit and vegetable data (hypotheses regarding these foods were not obvious at the time these earlier studies were conducted).

OESOPHAGEAL CANCER

Fourteen case-control studies of oesophageal cancer have examined the associations with vegetable and fruit consumption. Twelve found a statistically significant inverse relationship between intake of one or more vegetables and/or fruits and oesophageal cancer.

Consumption of vegetables, specifically, was found to be inversely associated in almost all of the studies that reported on this exposure and in most cases, this was statistically significant. The association with potatoes appears to be the most inconsistent. Consumption of fruit in general was inversely associated with oesophageal cancer.

Consumption of red chilli powder was also found to be a risk factor[11]. Although not a vegetable *per se*, such a risk may also apply to whole red chillies themselves. Alternatively, the risk may be derived from

some part of the drying process or use of red chilli powder may be a marker for use of some other spice that is the actual agent.

Smoking and heavy alcohol consumption are well established as the risk factors that account for the majority of oesophageal cancers in the industrialised countries. These habits are associated with less healthful eating patterns and poor nutritional status. Thus, an obvious question is whether the inverse associations found with vegetable and fruit consumption in most of these case-control studies are confounded by alcohol intake and smoking. In fact, several of the studies reviewed here supply evidence that vegetable and fruit consumption has an independent effect with many of the odds ratios being adjusted for smoking or alcohol consumption. Strong consistency is observed across studies: adjustment for smoking and heavy drinking does not reduce risks associated with diet.

Some areas of the world, such as parts of Iran, South Africa and China, experience very high rates of oesophageal cancer that are not necessarily attributable to smoking and alcohol consumption. In further support of an independent effect of vegetable and fruit consumption, inverse associations were found[12] between intake of many vegetables and fruits and oesophageal cancers in Iran although the study in Lin Xian, China, was essentially null. In these areas, identified risk factors include low socioeconomic status, consumption of opium and of known carcinogens in food, perhaps consumption of extremely hot beverages and diets high in wheat and corn.

LUNG CANCER

Thirteen case-control studies of lung cancer have examined the effects of vegetable and fruit consumption. Eleven found lower risk associated with one or more vegetables and/or fruits. Many of the studies found consumption of carrots (or dark yellow-orange vegetables) to be associated with lower risk and somewhat fewer reported a similar result for leafy green vegetables (also described as kale, dark green vegetables or spinach). Carrots and greens are two of the most concentrated dietary sources of a variety of carotenoids.

In these studies, the benefit conferred by high intake of vegetables was often more evident in smokers than nonsmokers but not all studies examined associations within the smoking strata. One explanation for the trend for stronger associations in smokers might be that the majority of cases in most studies of lung cancer are smokers and there may not have been enough power to detect true associations in nonsmokers. Another possibility is that the benefit conferred by vegetable consumption is a late-stage event that occurs after initiation by substances in cigarette smoke. Interestingly, a study of only nonsmoking female patients in Hong Kong (64 per cent of female lung cancer patients in Hong Kong have never smoked), also found inverse associations for intakes of vegetables[13].

ORAL AND PHARYNGEAL CANCER

Thirteen case-control studies of vegetable and fruit consumption and oral and pharyngeal cancer have been reported. Ten of these studies reported an inverse association between oral and pharyngeal cancer and intake of one or more vegetables and/or fruits.

Consumption of fruit appeared to be especially consistent, with an approximate doubling of risk for consumers of the least vs the most amounts of fruit. No clear pattern emerged for specific vegetables with the possible exception of green vegetables which were associated with reduced risk in five of six studies.

As with oesophageal and laryngeal cancer, the predominant risk factors for oral and pharyngeal cancer are tobacco and alcohol. In most areas of the world, smoking, chewing (including betel chewing) and drinking are estimated to account for 75 per cent or more of the cancers of these sites. Each of the studies reviewed here found these to be the major risk factors. Poor dentition was also found to be a risk factor in a few studies. Because, as noted before, tobacco and alcohol habits may correlate inversely with food intake and the nutritional quality of the diet, the potential for a confounding effect of these habits on the relationship between vegetable and fruit consumption and oral and pharyngeal cancer is great. Nonetheless, there is ample evidence of an independent inverse association with vegetable and fruit consumption.

BREAST CANCER

Thirteen case-control studies of breast cancer in 11 different countries have examined the potential protective effect of vegetable and fruit consumption. Of these, nine found a significant reduction of risk in association with at least one vegetable or fruit category.

Most of the breast cancer studies focussed on fat intake as well as vegetable intake in order to shed light on the controversial hypothesis concerning a role for a high-fat diet in the etiology of breast cancer. An interesting difference between the studies that found associations between vegetable and fruit consumption and breast cancer and those that did not, is that odds ratios were adjusted for potential confounders in the studies which found a relationship. Theoretically, high vegetable and fruit consumption may be associated positively with risk factors for breast cancer such as upper social class, body weight, and later age at first birth. Such correlations might obscure any inverse association between vegetable and fruit consumption and breast cancer in the absence of adjustment.

Overall, the associations appear to be weaker than those seen for respiratory and digestive cancers. It may be that the potential anticarcinogenic actions of vegetables and fruits are less applicable to breast cancer than non-hormone related epithelial cancers. For instance, most lung and upper aerodigestive cancers are strongly associated with smoking, which provides a direct exposure to initiating agents, whereas for breast cancer, the known risk exposures are mainly related to exposure to unopposed estrogen, which may have more of a promotional effect. If the effect of potential anticarcinogenic agents in vegetables and fruit is to block initiating events then these agents may be more effective in preventing, say, lung than breast cancer. Nonetheless, an effect of indoles, which are found in cruciferous vegetables, on estrogen metabolism has been demonstrated and may be relevant to the etiology of breast cancer.

Thus, the majority of the studies show no more than a halving of risk

with high consumption of vegetables and fruit. It is commonly the case that dietary risk or protective factors are not shown to be as strong as other risk factors in epidemiological studies. This may be in part due to attenuation of odds ratios due to the inability of dietary assessment methods to classify diets of individuals with great precision. It may also be due in part to the narrow range of levels of exposure to dietary factors within most populations.

Therefore, odds ratios consistently suggesting a doubling or halving of risk pertinent to dietary exposures may in fact be signaling a stronger relationship. Even if the risk of cancer were only doubled by low vegetable and fruit consumption, the importance would remain because the attributable risk would be high due to widespread exposure to low vegetable and fruit consumption and because diet is a relatively modifiable entity and thus a potential target for change.

MECHANISMS

There are many biologically plausible reasons why consumption of vegetables and fruit might reduce the likelihood of cancer. These include the presence of potentially anticarcinogenic substances such as carotenoids, ascorbate, tocopherols, selenium, dietary fibre, dithiolthiones, isothiocyanates, indoles, phenols, protease inhibitors, allium compounds, plant sterols, limonene and others which are increasingly being called, collectively, phytochemicals or bioactive compounds — chemicals of plant origin that play a crucial role in mammalian metabolism[6].

When the body is first exposed to specific carcinogens, many are not in their active form; the steps between exposure to the pro-carcinogen and the conversion of a normal cell into a cancer-prone cell (transformation) can be considered as follows: the pro-carcinogen is activated to the ultimate carcinogen form by P450 enzymes. (It is worth keeping in mind that the body is not trying to make carcinogens — it is trying to solubilise insoluble foreign compounds and therefore enable excretion in the urine.) However, this is complicated by the fact that the same enzyme can often make one compound less carcinogenic and another more carcinogenic; either of the forms of the carcinogen — pro-carcinogen or ultimate carcinogen — may be converted by Phase II enzymes into a form that is relatively inert and even more easily excreted.

These are typified by glutathione S-transferase; if not excreted, the carcinogen can pass through the cell membrane and the nuclear membrane; it can then interact with the DNA-forming adducts and/or produce mutations; DNA synthesis and replication (or DNA repair) subsequently occurs. Repair has varying degrees of fidelity; if the DNA is not repaired accurately, cell replication — producing daughter cells with copies of the mutated DNA — occurs. These cells then synthesise an abnormal protein or fail, altogether, to synthesise a protein crucial to the normal function of the cell or even crucial to controlling cell replication itself (this is almost certainly what happens when a tumour suppressor gene [for example, P53] mutates or is deleted).

This sequence of stages brings a cell a step closer to becoming a cancer cell (alternatively, even with abnormal DNA, the cell may cease to replicate and then undergo differentiation or apoptosis). DNA damage probably has to occur several times before a cell becomes completely free of growth restraint and a fully cancerous one. Finally, the abnormal cells obtain a growth advantage over the normal cells and steadily increase in numbers (promotion) — often becoming more malignant and able to spread (progression). These are steps that themselves involve further changes in the cellular DNA.

At almost every one of the above stages, known phytochemicals/bioactive compounds can alter the likelihood of carcinogenesis, occasionally in a way that enhances risk, but usually in a favourable direction. For example, such substances as glucosinolates and indoles, isothiocyanates and thiocyanates, phenols and coumarins can induce a multiplicity of solubilising and (usually) inactivating enzymes; ascorbate and phenols block the formation of carcinogens such as nitrosamines; flavonoids and carotenoids can act as antioxidants, essentially disabling carcinogenic potential; lipid-soluble compounds such as carotenoids and sterols may alter membrane structure or integrity; some sulphur-containing compounds can suppress DNA and protein synthesis, carotenoids suppress DNA-synthesis and enhance differentiation.

The relation of cancer risk to consumption of plant foods is probably most usefully considered in an evolutionary/adaptational context. The argument in this regard has been developed in more detail elsewhere[14,15].

SUPPLEMENTATION

As evidence emerged in the 1960s and 1970s of a role for vegetables and fruits as protective agents, particularly against stomach and lung cancers, a number of investigators began to ask about potential mechanisms and agents. The animal experimental data were already suggesting the presence of a whole variety of active agents but humans have always liked simple solutions and magic bullets[16]. Researchers wondered whether β-carotene might be the relevant agent[17]. They began with retinol data on humans and animals, but drifted to β-carotene as oxygen damage to DNA and antioxidant status became possible players in carcinogenesis. This nicely framed hypothesis was enough to spark a whole industry.

The hypothesis was boldly stated and testable, and a number of well-conducted studies have now rendered the verdict. β-carotene supplementation does not lower rates of lung cancer[18,19,20] and among high-risk individuals appears to increase incidence and mortality[18,19]. β-carotene also does not reduce recurrence of adenomatous polyps[21] and may increase risk of larger polyps[22]. β-carotene does not reduce the risk of recurrence of skin cancer[23].

In striking contrast, as the constituents of tobacco smoke were enumerated, it became increasingly clear that making a noncarcinogenic cigarette was not an achievable goal and that behavioural, economic and legislative programmes to eliminate smoking were more appropriate. It may suggest that behavioural approaches (paralleling the successful tobacco programmes) to increasing plant food intake may be more fruitful. Economic incentives to increase human plant-food production are also worthy of consideration.

There are other arguments enumerated above, based on the biology

of cancer, to support an increased intake of plant foods as a primary strategy. The use of single agents has proved to be ineffectual and ultimately counterproductive in the treatment of many cancers; this is largely because resistant clones arise readily in the presence of potent cytotoxic/chemotherapeutic agents. In rapidly proliferating tissues, with elevated levels of cell death, selection for survival in the presence of the agent will occur rapidly. In tissues where there are large numbers of initiated cells, it is plausible that similar selection will happen in the presence of a single chemo-preventive agent.

If the action of the agent is to induce differentiation, those cells that are incapable of differentiation may gain a proliferative advantage. Similar arguments apply to a single agent that increases the rate of apoptotic cell death; again, those cells that are resistant to apoptosis may continue to proliferate[24].

It follows that, while we should continue experimental studies to understand the role of specific agents in cancer prevention, we should not be surprised if the effect of the agent differs at different stages of carcinogenesis or if it differs when used alone vs in combination with other compounds.

The safest public health strategy seems to be to advocate increased intake of intact plant foods with the multiplicity of agents that they contain. It is less likely that any clone of malignant cells can survive the polypharmacy of plant foods[15,24].

IMPLICATIONS FOR INDIVIDUALS AND COMMUNITIES

The implication for individuals and societies around the world are clear — produce and eat more vegetables and fruit — not more pills.

At present, it is not clear what quantity we should eat each day — it is clear that many people around the world do not eat enough. Therefore, setting a population target of perhaps 400 g may be a useful interim goal. Certainly, at this level, there are few dangers for any part of the population. In India, maintenance of the traditional plant-based diet, at a level of energy intake sufficient to ensure the absence of childhood malnutrition, is, along with the avoidance of tobacco use, likely to make a profound contribution to continuing the low rates of cancer that are a feature of Indian health.

Excerpts from the Gopalan Oration delivered at the XXIXth Annual Meeting of the Nutrition Society of India at the National Institute of Nutrition, Hyderabad, on November 21, 1996.

The full text of the oration will appear in the proceedings of the Nutrition Society of India.

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NUTRITION NEWS

● **International Symposium by Rank Prize Funds:** This Symposium on: 'Feeding a World Population of more than Eight Billion People: A Challenge to Science' was held in the UK from December 6-9, 1996. It was attended by 104 delegates. The proceedings are expected to be published shortly.

● **The 23rd Kamla Puri Sabharwal Memorial Lecture** was held on December 18, 1996, at the Lady Irwin College. Dr Prema Ramachandran spoke on: 'Current Concerns in Maternal Nutrition'. Dr C. Gopalan presided.