



Diet and Cancer

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ABSTRACT

The large differences in cancer rates among countries, striking changes in these rates among migrating populations, and rapid changes over time within countries indicate that some aspect of lifestyle or environment is largely responsible for the common cancers in Western countries. Dietary fat has been hypothesized to be the key factor because national consumption is correlated with the international differences. However, detailed analyses in large prospective studies have not supported an important role of dietary fat. Instead, positive energy balance, reflected in early age at menarche and weight gain as an adult, is an important determinant of breast and colon cancers, consistent with numerous studies in animals. As a contributor to positive energy balance, and possibly by other mechanisms, physical inactivity has also been shown to be a risk factor for these diseases and in part accounts for the international differences. Although the percentage of calories from fat in the diet does not appear related to risk of colon cancer, greater risks have been

seen with higher consumption of red meat, suggesting that factors other than fat per se are important.

In many case-control studies, a high consumption of fruits and vegetables has been associated with reduced risks of numerous cancers, but recent prospective studies suggest these associations may have been overstated. Among the factors in fruits and vegetables that have been examined in relation to cancer risk, present data most strongly support a benefit of higher folic acid consumption in reducing risks of colon and breast cancers. These findings have been bolstered by an association between incidence of colon cancer and a polymorphism in the gene for methylenetetrahydrofolate reductase, an enzyme involved in folic acid metabolism. The benefits of folic acid appear strongest among persons who regularly consume alcohol, which itself is associated with risk of these cancers. Numerous other aspects of diet are hypothesized to influence the risks of cancers in Western countries, but for the moment the evidence is unclear. *The Oncologist* 2000;5:393-404

The great interest during the last several decades in diet and human cancer derives from the large variations in rates of specific cancers among countries, coupled with the dramatic changes in cancer incidence among populations emigrating to regions with different rates. Such observations indicate the importance of potentially modifiable factors in the cause and prevention of cancer, and a role of diet has been suggested by strong correlations between cancer rates and national per capita intake of specific nutrients. In their comprehensive 1981 review of avoidable causes of cancer mortality in the U.S., Doll and Peto suggested that dietary factors might account for

approximately 35% of cancer deaths, similar to the impact of smoking [1]. However, the estimate for diet was uncertain and they considered that this percentage might be as low as 10% or as high as 70%. Moreover, the specific aspects of diet that were most important were said to be unclear at that time.

In this brief overview, I will attempt to summarize current information on the relation of diet to the most important human cancers in Western countries not due primarily to smoking. Emphasis will be given to prospective epidemiologic studies because these are less subject to serious problems of confounding that can make cross-cultural comparisons

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completely misleading, and they avoid the problem of recall bias that can affect typical case-control studies of diet [2]. Animal and mechanistic studies can also be extremely useful in suggesting hypotheses and providing supportive evidence. Ideally, all diet and cancer-hypothesized relationships would be evaluated by randomized trials in humans, but in most instances such studies may never be feasible because of the need for large sample sizes, long (and uncertain) duration of follow-up, and high levels of compliance.

EXCESSIVE ENERGY INTAKE

For nearly a century, energy (caloric) restriction has been known to reduce the development of mammary tumors in animals [3, 4]. This effect has been observed in a variety of mammary tumor models and also for other tumors [5-9]. The impact of energy restriction can be profound; for example, a 30% lower intake has reduced mammary tumors by as much as 90% [10]. Whether this relationship might also apply to humans is clearly of great interest.

A simple examination of the association between energy intake in human populations and incidence of cancer is likely to be completely misleading because variation in energy intake is determined largely by physical activity [11]. Thus, for example, energy intake is inversely associated with risk of coronary heart disease due to the protective effect of exercise [12]. Fortunately, growth rates and body weight are sensitive indicators of the balance between energy intake and expenditure, and these can be easily measured in epidemiologic studies. Adult height provides an indirect indicator of preadult nutrition, and adult weight gain and obesity reflect positive energy balance later in life.

In comparisons among countries, the average national height of adult women is strongly associated with risk of breast cancer [13], and in case-control and cohort studies greater height has generally been associated with higher risk of breast cancer [14]. Greater height has also been associated with risk of colon and other cancers [15, 16]. In addition, preadolescent height, weight, and rapid growth are strong determinants of age at menstruation [17-19], a well-established predictor of breast cancer, but the composition of diet appears to have little if any effect. Consistent with animal experiments, these studies collectively provide strong evidence that rapid growth prior to puberty plays an important role in determining future risk of breast and probably other cancers [16, 20].

Positive caloric balance and the resulting accumulation of body fat during adult life also increase the risk of important human cancers. The best-established relationships are with cancers of the endometrium and gall bladder [21-25]. Greater adiposity is also associated with risk of colon cancer

in both women [26] and men, particularly when assessed as abdominal circumference [27]. The relation between body fatness and breast cancer is complex. Prior to menopause, women with greater body fat have reduced risks of breast cancer [28, 29], probably because of more anovulatory menstrual cycles in fatter women [30]. After menopause, a positive weak, overall association with adiposity is seen, which is stronger among women who never used hormone replacement. This interaction with hormone use is probably the result of greater conversion of adrenal androgens to estrogens [31] in overweight women; women taking exogenous hormones have elevated circulating estrogens and a higher risk of breast cancer regardless of adiposity.

DIETARY FAT

A possible relation of dietary fat intake to cancer incidence has also been hypothesized because the large international variations in rates of cancers of the breast, colon, prostate, and endometrium are strongly correlated with apparent per capita consumption [32-34]. However, these associations are due to correlations with intake of animal fat, not vegetable fat [35], raising the possibility that fat per se is not the responsible factor. Dietary fat promotes tumors in many animal models [9, 36-39], but this has been either weak [40] or nonexistent [9] in some studies designed specifically to address the independence of fat and total energy intake.

The relation between dietary fat intake and breast cancer has been examined in many prospective studies. In a pooled analysis [41], no overall association was seen for total fat intake over the range of 15% to >45% of energy from fat; a similar lack of association was seen in analyses restricted to postmenopausal women. Among the small number of women consuming less than 15% of energy from fat, breast cancer risk was elevated twofold. In the 14-year follow-up of the Nurses' Health Study, a weak but statistically significant inverse association was seen for total fat intake [42]. Although total fat intake has not been related to higher breast cancer risk in prospective epidemiologic studies, some evidence suggests that the type of fat may be important. In animal models, the tumor-promoting effect of fat intake has been observed primarily for polyunsaturated fats [43, 44] but such an association was not supported by the pooled data from large cohort studies [41]. In case-control studies conducted in Spain and Greece, women who used more olive oil had reduced risks of breast cancer [45, 46], possibly related to its high content of monounsaturated fat and antioxidants [38].

National rates of colon cancer are also strongly associated with per capita consumption of animal fat and meat, with correlation coefficients ranging between 0.8 and 0.9 [32, 35].

Based on these epidemiologic investigations and animal studies, dietary fat increasing has been hypothesized to act by excretion of bile acids, which can be converted to carcinogens or promoters [47]. However, many studies have found that higher levels of physical activity are associated with reduced risk of colon cancer [48]; thus, the high rates in affluent countries previously attributed to fat intake are probably, at least in part, due to sedentary lifestyle.

In a meta-analysis of 13 case-control studies, a significant association between total energy intake and colon cancer was observed, but saturated, monounsaturated and polyunsaturated fats were not related to colon cancer risk after adjustment for total energy [49]. The relation between diet and colon cancer has been examined in several large prospective studies, which have not confirmed the positive association with total energy intake seen in case-control studies [50-53]. Higher intake of various forms of red meat has been associated with greater risk of colon cancer in most prospective studies [50-53], but not in all [54]. In general, positive associations have been most consistently seen when red meat was specifically examined and when eaten as a main dish. Fairly consistent positive associations have also been observed for processed meats. The apparently stronger association with red meat compared with fat in several recent cohort studies needs further confirmation, but could result if the specific fatty acids or nonfat components of meat (for example the heme iron or carcinogens created by cooking) were the primary etiologic factors. This issue does have major practical implications as some dietary recommendations encourage daily consumption of red meat as long as it is lean [55].

Associations between fat intake and risk of prostate cancer have been seen in many case-control studies [56-66], but sometimes only in subgroups. In a large case-control study among various ethnic groups within the U.S. [67], consistent associations with prostate cancer risk were seen for saturated fat, but not for other types of fat. The relation of fat intake and prostate cancer risk has been examined in only a few cohort studies with reasonably comprehensive assessments of diet. In a study of 14,000 Seventh-Day Adventist men living in California, a positive association between the percentage of calories from animal fat and prostate cancer risk was seen, but this was not statistically significant [68]. In the Health Professionals Follow-up Study of 51,000 men, a positive association was seen with intake of red meat, total fat and animal fat, which was largely limited to aggressive prostate cancers [69]. No association was seen with vegetable fat. In another cohort from Hawaii, increased risks of prostate cancer were seen with consumption of beef and animal fat [70].

Although further data are desirable, international correlations, case-control, and cohort studies are reasonably

consistent in supporting an association between consumption of fat-containing animal products and prostate cancer incidence. This evidence does not generally support a relation with intake of vegetable fat, which suggests that either the type of fat or other components of these animal products are responsible. In several studies [71, 72] calcium rather than fat appeared to account for an association between consumption of dairy products and risk of prostate cancer. In some studies, animal fat consumption has been most strongly associated with aggressive prostate cancer, which suggests an influence on the transition from the widespread indolent form to the more lethal form of this malignancy.

Rates of endometrium and ovarian cancers and other malignancies that are common in affluent countries are also correlated with fat intake internationally. These have been studied in a small number of case-control investigations; consistent associations with fat intake have not been seen [73-82]. In a prospective study among Iowa women [83], no evidence of relation between fat intake and risk of endometrial cancer was observed.

In summary, support for a major relationship between fat intake and breast cancer risk has weakened considerably as the findings from large prospective studies have become available. For colon cancer, more recent evidence has suggested that this might be explained by factors in red meat other than simply its fat content. Further, the importance of physical activity as a protective factor against colon cancer indicates that international correlations probably overstate the contribution of diet to differences in colon cancer incidence. The available evidence does support an association between animal products and risk of prostate cancer, particularly the aggressive form of this disease, for which fat is a possible explanation. Some have argued that dietary fat must be related to risk of breast and colon cancer because, as an important source of calories, it is a cause of obesity, which is a risk factor for these diseases [84]. However, evidence from longer-term randomized trials indicates that the fat composition of the diet has little if any relation to body fat, and that excessive caloric intake from fat and carbohydrates similarly lead to weight gain [85].

FRUITS AND VEGETABLES

Higher consumption of fruits and vegetables has been associated with a reduced risk of cancers at many sites in numerous epidemiologic studies. Inverse relationships with intake of these foods have been observed in over 200 case-control and prospective cohort studies [86, 87] and additional support comes from studies in which biochemical indicators of fruit and vegetable consumption, such as serum carotenoid levels, are also associated with reduced risks. The studies are particularly numerous and consistent for cancers of the lung [88] and stomach [89], although in a

recent large prospective study from Holland, little relation was seen between consumption of fruits and vegetables and stomach cancer [90]. Inverse associations have also been observed in many case-control studies of colon cancer but prospective data are still limited. Other studies have also suggested possible inverse associations with cancers of the oral cavity, larynx, esophagus, endometrium, cervix, bladder, kidney and breast [91]. Inverse associations have not been seen between overall fruit and vegetable consumption and risk of prostate cancer, but intake of tomato products, the primary source of the carotenoid lycopene, has been related to lower risk in case-control and prospective studies [60, 68, 92].

The evidence that high consumption of fruits and vegetables can reduce the risk of many cancers is strong, but the constituents of these foods that are responsible for these reduced risks are less clear. These foods contain many biologically active chemicals, including recognized nutrients and many more nonnutritive constituents, that could potentially reduce cancer incidence [86]. Potentially protective factors include various carotenoids, folic acid, vitamin C, flavonoids, phytoestrogens, isothiocyanates and fiber (discussed below). The identification of the specific protective constituents, or combination of constituents, is a daunting task and may never be completely possible.

DIETARY FIBER AND CANCER RISK

Fiber has been hypothesized to reduce risk of colon cancer by diluting potential carcinogens and speeding their transit through the colon, binding carcinogenic substances [93], altering the colonic flora [94-97], reducing the pH [98], or serving as the substrate for the generation of short-chain fatty acids that are the preferred substrate for colonic epithelial cells [99]. However, as epidemiologic evidence has accrued, support for the importance of dietary fiber in reducing risk of colon cancer has dwindled. First, the populations with high fiber consumption and low rates of colon cancer are also typically those of poorer countries where consumption of meat and obesity are low and physical activity is high. Evidence has become clearer that each of these factors reduces risk and thus is likely to explain at least part of the ecological associations between low intake of dietary fiber and colon cancer rates. In case-control studies, intake of cereal products or fiber from grains has not usually been associated with reduced risks of colon cancer, in contrast to the better support for a protective effect of fruits and vegetables [100, 101]. In some studies, higher consumption of grains has been associated with greater risks of colon cancer [102-105]. Also, in several large prospective studies, even overall fiber intake has not been significantly associated with lower risk of colon cancer incidence after adjustment for other risk factors

[50-53, 106]. In these same cohorts, higher grain fiber intake has been associated with lower risks of coronary heart disease [107, 108], indicating that physiologically important effects of dietary fiber can be detected if present. Thus, fiber intake does not appear to account for the apparent protective effect of fruits and vegetables against colon cancer, and available evidence does not support the hypothesis that higher consumption of grain fiber or fiber supplements can reduce risk of colon cancer.

Higher intake of fiber has also been hypothesized to reduce risk of breast cancer by interrupting the enterohepatic circulation of estrogens [109]. However, in prospective studies, little or no relationship has been observed between fiber intake and risk of breast cancer [110-112].

ALCOHOLIC AND CAFFEINATED BEVERAGES

High consumption of alcohol, particularly in combination with cigarette smoking, is a well-established cause of cancer of the oral cavity, larynx, esophagus, and liver [113]. Evidence from many case-control and cohort studies also indicates that even one or two drinks of alcoholic beverages per day increase risk of breast cancer [114]; the increase in risk is approximately 13% for 12 grams/day of ethanol (one average drink). Moderate alcohol consumption increases endogenous estrogen levels, which may account for this effect, at least in part [31, 115, 116]. High alcohol consumption also appears to be associated with risk of cancers of the colon and rectum [101]. Alcohol interferes with the availability of folic acid, which may contribute to its relation with large bowel and breast cancers [117-120].

Coffee contains multiple mutagenic substances [121], which has raised concern that it might be an important cause of cancer in humans. Early evidence suggested a possible positive association with pancreatic cancer, but this has not been supported in most subsequent studies [122-124]. For breast cancer, coffee consumption has been unassociated [125] or even weakly inversely related to risk [126]. Green tea contains polyphenolic compounds that inhibit tumors in experimental animals [127] and inverse associations between green tea consumption and risk of gastric cancer have been seen in case-control studies from Japan [128] and China [129]. Notably, however, rates of gastric cancer in the U.S., where tea consumption is low, are among the lowest in the world; this has been attributed in part to relatively low salt intake [89].

VITAMIN AND MINERAL SUPPLEMENTS

Data on vitamin supplement use and cancer incidence are limited. High-dose supplements of vitamins C and E have not been associated with reductions in breast cancer incidence [111, 130]. In single studies, vitamin E supplements were

associated with a reduced risk of oral cancer [131] and colon cancer [132]. In a large randomized trial conducted in Finland, men receiving vitamin E (50 IU/day) experienced reduced risks of prostate cancer [133]. Although statistically significant, this finding needs to be reproduced because it was not a prior hypothesis and many specific cancers were examined; chance remains a possible explanation. In a large prospective observational study, use of vitamin E supplements (typically 400 IU/day) was not associated with risk of prostate cancer [92]. In a randomized trial conducted in a region of China with very low consumption of fruits and vegetables, a supplement containing beta-carotene, vitamin E, and selenium reduced incidence of stomach cancer [134].

In three large randomized trials, beta-carotene supplements did not reduce risk of lung or other cancers [135-137]; indeed in two of these the incidence of lung cancer was actually increased among those receiving beta-carotene (which in one study was given in combination with vitamin A). These findings have raised interesting mechanistic issues, and some have suggested that they cast doubt on the epidemiologic findings that were used to justify the trials. However, the epidemiologic studies investigated consumption of fruits and vegetables, not beta-carotene supplements; the hypothesis that beta-carotene might be the protective constituent was based on theoretical mechanisms and limited animal experiments. Thus, these trials do not refute beneficial effects of greater fruit and vegetable consumption, but should engender caution in making inferences about supplements based on data relating intake of foods to cancer risk.

In many animal studies, high intakes of selenium have reduced risks of various tumors [138], and ecological studies have suggested inverse associations with breast and colon cancers [139]. However, in case-control and cohort studies, levels of selenium in blood or nails have not been associated with incidence of these cancers [140, 141]. In a recent study designed to assess the use of selenium supplements in the prevention of skin cancers, those receiving supplements developed somewhat greater numbers of these cancers [142]. Even though the number of participants was small, a significant, large, and rapid reduction in incidence of other cancers was seen among those receiving selenium; reductions were particularly great for cancers of the lung, colon, and prostate. Although extremely interesting, these findings also require confirmation. Arguing strongly against a large and rapid effect of selenium supplementation is the experience of Finland during the last decade. Because of very low selenium content of soils in that region, and thus in the food supply, selenium was systematically applied with fertilizers in the mid-1980s. Despite

large and abrupt increases in blood selenium levels, there has been no apparent decline in incidence or mortality rates of prostate or colon cancer in the subsequent years (*P. Pietinen*, personal communication).

Because of promising animal studies, calcium has been considered as a promising agent to reduce risk of colon cancer. Calcium supplements modestly reduced recurrence of colon adenomas in a recently reported randomized trial [143]. However, in large prospective studies, only weak and nonsignificant inverse associations have been seen between calcium intake and risk of colon cancer [144]. Further evaluation of this relationship is needed, but current evidence suggests that, if a benefit exists, it is modest. Calcium supplement use was positively associated with risk of prostate cancer in one study [71]; although this needs to be confirmed, it raises concern about widespread use of calcium supplements by men.

Many lines of evidence indicate that folic acid intakes in the U.S. have been substantially less than optimal. The average intake from foods has been only about half the current RDA of 400 micrograms per day. Multivitamin supplements containing folic acid dramatically reduce risk of neural tube pregnancies and substantially lower blood levels of homocysteine, a risk factor for coronary heart disease. Also, *Blount et al.* [145] have found that persons with low red cell folate levels have a 10-fold incorporation of uracil into DNA, which is reversible by a folic acid supplement (normally folic acid provides a methyl group for synthesis of thymine from uracil). Lower intakes of folic acid have been associated with increased risks of colon adenoma and colon cancer [117-119]. In reports [146, 147], use of multiple vitamins containing folic acid was associated with lower risk of colon cancer; little apparent effect was seen for the first 15 years of use, but

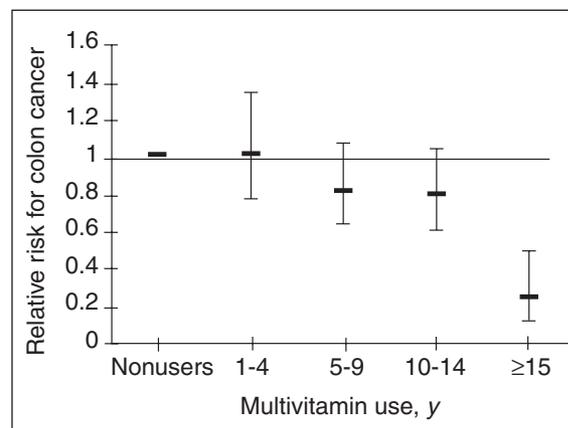


Figure 1. Multivariate relative risk for colon cancer according to years since the start of use of multivitamins containing folic acid in the Nurses' Health Study (1980 to 1994) [146]. Reproduced with permission [146].

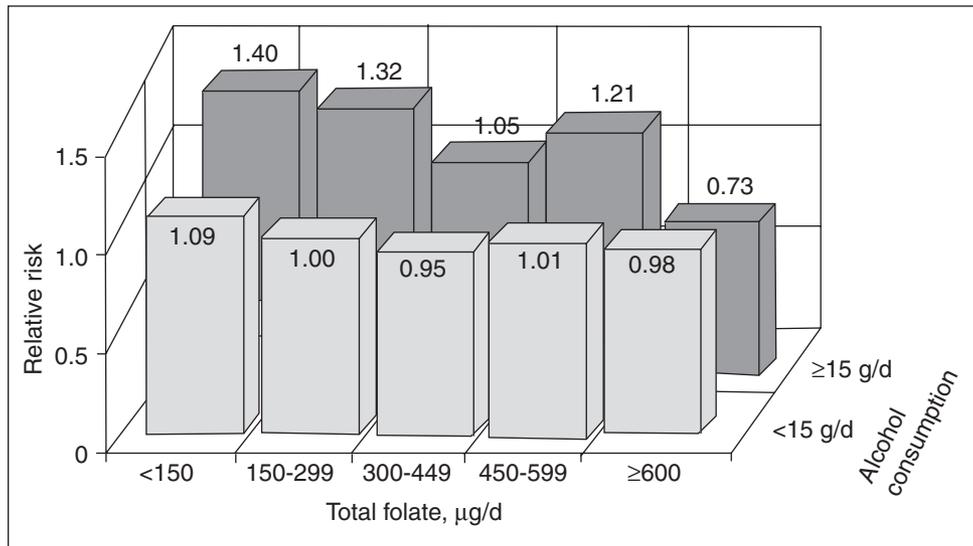


Figure 2. Multivariate relative risk of breast cancer by total folate intake and alcohol consumption. The reference group for all comparisons was women who consumed 150 to 299 g/d of total folate and less than 15 g/d of alcohol. Reproduced with permission [120].

longer use was associated with a 75% lower risk (Fig. 1) [146]. It is highly unlikely that randomized trials of sufficient duration will ever be done to evaluate such a relation directly. However, observations that a functionally important polymorphism in a gene involved in folic acid metabolism (methyltetrahydrofolate reductase) is associated with incidence of colon cancer [148, 149] provide important support for a causal effect of folic acid; if a genetically determined variant in an enzyme is associated with risk of disease, the substrate for the enzyme should also be related to risk of disease. In contrast to evidence relating beta-carotene intake to reduced risk of cancer, the use of a supplement containing folic acid, not only intake of folic acid calculated from foods, was associated with reduced risk. Because this was a multiple vitamin, the possibility remains that other components, such as vitamin D, might have contributed to lower risk of colon cancer. As noted above, higher intake of folic acid may also mitigate the elevated risks of breast cancer seen with daily alcohol consumption (Fig. 2) [120].

As of 1998, the U.S. food supply has been fortified with folic acid by adding an amount to flour that is estimated to increase the mean intake by about 100 micrograms per day, but fortification has not happened in Northern Ireland and most parts of Europe. Although this will be helpful, most persons will still not achieve the RDA level through diet alone. Recommendations to increase intakes of fruits and vegetables, while justified for many reasons, are unlikely to have a major impact on folic acid status, both because of practical barriers to higher intakes and the lower bioavailability of folate from foods [150].

SUMMARY AND SUGGESTIONS

During the past two decades, data on diet and cancer have greatly increased, and this continues to support the

[84, 151] as suggested earlier by *Doll and Peto* [146]. However, much more is known about the specific aspects of nutrition that may be important. Initially, greatest weight was given to the avoidance of harmful constituents of foods, such as carcinogens in cooked food and dietary fat. Subsequently, the protective factors in the diet, mainly in fruits and vegetables, were appreciated and this received primary attention. As prospective studies have become available, some of the benefits from fruits and vegetables seen in case-control studies appear to have been overstated, although for some cancers the evidence of benefit has been substantiated further. However, the increased risks due to excessive energy intake, represented by rapid growth in childhood and overweight in adults, and lack of physical activity have become more firmly established, particularly for cancers of the colon and breast. These clearly deserve greater emphasis.

In making dietary suggestions for cancer prevention by nutritional means, the effects of dietary change on risk of cardiovascular disease, the major cause of death in Western countries, must be carefully considered. Strategies that will reduce both types of disease deserve greatest emphasis. Also, the level of certainty about the evidence, including the likely quantitative benefit, should be considered in creating recommendations and communicating these objectives. With these general considerations in mind, the following suggestions are offered.

Avoidance of Overweight and Weight Gain During Adulthood

The evidence that excess body fat substantially increases risk of several important cancers is convincing, and this is also a major cause of cardiovascular disease and diabetes. Simply staying within standard weight guidelines for adults (BMI = 19-25 kg/m²) is not sufficient because

concept that a substantial proportion of cancer is potentially avoidable by nutritional means. Quantitative estimates of the preventable proportion in Western countries remain approximately 30% to 40%

many persons who are not overweight upon entering adulthood could still gain 30 to 40 pounds and remain within the guidelines [152]. For such persons, who comprise the majority in the U.S., staying within five to 10 pounds of their weight at age 20 is a simple guide. Although this may sound extreme to many Americans, who have come to regard major midlife weight gains as normal aging, it is the norm in Japan and most parts of the world.

Be Moderately to Vigorously Active For at Least 30 Minutes on Most Days

Physical activity is a primary method of weight control, and this goal is thus closely linked with weight control. However, evidence is strong that physical activity independently reduces risk of colon cancer, and higher levels of activity may reduce breast cancer risk, although the data are less consistent. Benefits for cardiovascular disease are well-established. The widely recommended goal of half an hour per day of moderate to vigorous activity is a minimum, and is very low compared to lifestyles in traditional societies.

Consume Five Servings of Fruits and Vegetables Daily

Evidence is strong that increasing consumption of fruits and vegetables to five servings a day will reduce risks of some cancers, including cancers of the lung and esophagus, and possibly breast and other cancers. However, for nonsmokers who are at low risk of some of these cancers, the benefits may be modest. More data from prospective studies are needed on specific fruits and vegetables and their quantitative relation with risk because different vegetables and fruits almost certainly have distinct biological effects; some may be particularly beneficial for certain cancers and some could even be harmful. Current U.S. guidelines include potatoes as a vegetable and these (as french fries) account for most of the recent increases in consumption; however, there is no good evidence that increasing potato consumption, or starch in general, will reduce cancer or cardiovascular disease risk.

Replace Red Meat with Chicken, Fish, Nuts, and Legumes, and Consume Dairy Products at Most in Moderation

Limiting consumption of red meat to several times a month at most by replacement with chicken, fish, legumes, and nuts will probably reduce risk of colon cancer and possibly prostate cancer, although the evidence is not conclusive. This dietary change will improve blood lipids and is highly likely to reduce risk of coronary heart disease. The role of dairy products in both cancer and heart disease prevention is not clear. High consumption of dairy products has been associated with prostate cancer

risk in many studies, and several recent studies suggest that fat may not be the responsible factor. The strategy of encouraging use of low-fat rather than high-fat dairy products will have little impact on population rates of any disease because the fat from milk remains in the food supply, and is even often eaten by the same people but in a different form. A general recommendation to increase calcium intake by greater consumption of dairy products is likely to increase rates of coronary disease because production and consumption of dairy fat would rise. At present, a prudent strategy would be to consume dairy products in modest amounts at most, and use supplements if higher intake of calcium is required.

Limit Alcohol Consumption to One Drink a Day for Women and Two for Men

The evidence is strong that high alcohol consumption increases risk of many cancers, and even intakes as low as one drink a day can increase risk of breast cancer. Personal decisions about alcohol consumption are complex and, in addition to the pleasures of consuming these beverages, should include consideration of benefits for coronary heart disease and risks of accidents and addiction.

Consider Taking a Multiple Vitamin Containing Folic Acid, Particularly if Alcohol is Consumed Daily

Substantial evidence indicates that consuming folic acid in the form of an RDA-level multiple vitamin (400 micrograms/day) will reduce risks of colon cancer and coronary heart disease. Alcohol antagonizes the bioavailability of folic acid, and epidemiologic studies suggest that the benefits of additional folate are greatest for those consuming alcohol on a daily basis. In principle, substantially increasing fruits and vegetables could also increase folic acid consumption, but folic acid from these foods has lower bioavailability than that from supplements. Also, behavioral and economic barriers to greatly increasing consumption of fruits and vegetables precludes large increases by the population as a whole in the foreseeable future. At present, the evidence is insufficient to recommend use of other vitamin or mineral consumption for cancer prevention.

Consume Cereal Products in a Minimally Refined, Whole Grain Form

The evidence that higher intake of cereal fiber will reduce risk of colon or other cancer has weakened greatly. However, this recommendation can be justified by the strong and consistent inverse relationships between cereal fiber consumption and risk of coronary heart disease and diabetes.

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