DIET AND CANCER

A REPORT BY THE AMERICAN COUNCIL ON SCIENCE AND HEALTH

SECOND EDITION

This report is a revised and updated version of a report on diet and cancer published by ACSH in 1985. The original report was written by Michael W. Pariza, Ph.D., of the University of Wisconsin, an ACSH Scientific Advisor.

The new edition was prepared by Kathleen Meister, M.S., a free-lance medical writer and former ACSH Research Associate.

ACSH gratefully acknowledges the comments and contributions of the following individuals who reviewed this report:

Roslyn B. Alfin-Slater, Ph.D.
UCLA

James J. Cerda, M.D.
University of Florida

Ralph E. Dittman, M.D., M.P.H.
Houston, TX

Alfred E. Harper, Ph.D.
University of Wisconsin

Victor Herbert, M.D., J.D.
Mt. Sinai School of Medicine

David Kritchevsky, Ph.D.
The Wistar Institute, Philadelphia, PA

Carolyn Lackey, Ph.D., R.D.
North Carolina State University

Lillian Langseth, Dr. P.H.
Lyda Associates, Palisades, NY

Howard D. Maccabee, Ph.D., M.D.
Radiation Oncology Center, CA

Roger P. Maickel, Ph.D.
Purdue University

Judith A Marlett, Ph.D.
University of Wisconsin, Madison

Donald J. McNamara, Ph.D.
University of Arizona

John A. Milner, Ph.D.
Pennsylvania State University

A. Alan Moghissi, Ph.D.
University of Maryland

Stanley T. Omaye, Ph.D., F. ATS
University of Nevada

Edward G. Remmers, Sc.D.
American Council on Science and Health

Paul D. Saltman, Ph.D.
University of California, San Diego

Fredrick J. Stare, M.D., Ph.D.
Harvard School of Public Health

Robert P. Upchurch, Ph.D.
University of Arizona

Elizabeth M. Whelan, Sc.D., M.P.H.
American Council on Science and Health

Executive Summary

The public has been bombarded with messages urging everyone to make substantial dietary changes to reduce their risk of cancer. Americans have been led to believe that the link between specific dietary factors and cancer is solid and convincing, and that dietary modification should be top priority in cancer prevention. In actuality:

- Smoking cessation — not diet — is the single most important factor in cancer prevention. There is no dietary change which will counteract the harmful effects of cigarette smoking. People who believe that they can safely continue to smoke as long as they eat healthful diets are dangerously misinformed.
- A substantial body of epidemiologic evidence associates low intakes of fruits and vegetables with increased risks of cancer. It would be wise for all Americans to make an effort to include fruits and vegetables in their daily diets. Current recommendations call for a minimum of five daily servings of fruits and vegetables; this is a reasonable goal.
- The current scientific evidence does not warrant recommendations for widespread supplementation with antioxidant nutrients (vitamin C, vitamin E and/or beta-carotene). Clinical trials due to be completed within the next decade should provide definitive evidence on whether or not antioxidant supplementation is beneficial. The one major clinical trial that has already been completed is of little relevance to Americans because it was conducted in a population that has dietary habits and disease patterns dramatically different from those in the U.S.
- Reducing dietary fat intake may reduce the risk of colon cancer but not breast cancer. Evidence for other cancer sites is inconsistent. It is possible that effects attributed to dietary fat may actually be due to related factors such as total caloric intake.
- Dietary fiber has not been convincingly linked with reduced risks of cancer. However, fiber does have other health benefits.
- Reaching and maintaining a desirable body weight is an important health priority. Obesity increases the risk of hypertension, diabetes and coronary heart disease. It may also increase the risk of some types of cancer, especially in women.
- Previous recommendations that Americans should minimize consumption of cured, smoked or pickled foods do not have a sound scientific basis.
- “Chemicals” in food — including naturally occurring substances, intentional additives and contaminants — do not have a significant impact on cancer risk in the United States.
- People who drink alcoholic beverages should do so in moderation. Excess alcohol intake is linked with many health problems, including increased risks of some types of cancer. The current evidence does not warrant a recommendation for abstinence from alcohol for the purpose of preventing breast cancer.

Introduction: A Decade of Recommendations

In 1982, the National Research Council published a report, *Diet, Nutrition, and Cancer,* which included a set of dietary guidelines for cancer prevention. Since then, several organizations, including the National Cancer Institute, the federal government's Departments of Agriculture and Health and Human Services, the Surgeon General's office and the National Research Council, have issued additional recommendations for dietary change. Some of these recommendations were aimed exclusively at cancer prevention, while others focused simultaneously on cancer and other diseases. At various times in the past decade Americans have been advised to:

- Cut back on dietary fat and saturated fat (to no more than 30 percent and 10 percent of total calories, respectively);
- Eat more vegetables and fruits (at least five servings daily, with an emphasis on citrus fruits, dark yellow and leafy dark green vegetables and/or cabbage-family vegetables);
- Increase dietary fiber intake (some authorities recommend 20 to 30 grams daily or 10 to 20 grams of fiber for each 1000 calories consumed);
- Achieve and maintain a healthful body weight;
- Avoid cured, nitrite-preserved, pickled or smoked foods;
- Drink alcohol in moderation, if at all.

In this report, the American Council on Science and Health (ACSH) examines the scientific evidence pertaining to each of these recommendations, to determine whether calls for dietary change are well founded.

Cancer in the United States

Cancer is the second leading cause of death in the United States (cardiovascular diseases are number one) accounting for one out of every five deaths or about 520,000 deaths per year.* The most common types of cancer in the U.S. are listed in Table 1. An estimated 1,130,000 new cancer cases occur each year. About 83 million Americans who are alive today — one-third of the population — will eventually have cancer.

It is commonly believed that the U.S. is experiencing a cancer "epidemic." However, with the exception of lung cancer, this is not the case. Although fluctuations have occurred for some individual types of cancers, there has been little change in the overall cancer death rate in the U.S. over the last 40 years.

Table 1: Estimated New Cancer Cases and Deaths in the United States in 1992

Cancer Site	New Cases	Deaths
Lung	168,000	146,000
Colon-Rectum	156,000	58,300
Breast	181,000	46,300
Prostate	132,000	34,000
Pancreas	28,300	25,000
Non-Hodgkin's lymphoma	41,000	19,400
Leukemia	28,200	18,200
Stomach	24,400	13,300
Ovary	21,000	13,000
Liver	15,400	12,300
Brain and central nervous system	16,900	11,800
Kidney	26,500	10,700
Esophagus	11,100	10,000
Bladder	51,600	9,500
Multiple myeloma	12,500	9,200
Oral	30,300	7,950
Skin (Melanoma only)[a]	32,000	6,700
Uterine corpus (including endometrium)	32,000	5,600
Uterine cervix	13,500	4,400
Larynx	12,500	3,650
Thyroid	12,500	1,000

[a] Estimated new cases of non-melanoma skin cancer total over 600,000. Non-melanoma skin cancer is rarely fatal, accounting for only about 2,100 deaths per year.

Source: American Cancer Society, *Cancer Facts and Figures—1992* .

How Important Is Diet?

The proliferation of official dietary recommendations has encouraged the public to conclude that diet is a very important factor in both the causation and prevention of cancer. A widely circulated set of estimates of the proportions of cancer deaths attributable to various factors tends to give the same impression. However, when the scientific basis for these estimates is examined carefully, a different picture emerges.

Table 2 shows those cancer estimates, which were devised by two British epidemiologists, Sir Richard Doll and Richard Peto, in the early 1980s. These estimates, when taken out of context, would seem to indicate that diet is the number one cancer risk factor, more important even than

tobacco. However, this is not what the originators of the table had in mind. In the scientific paper where they presented their cancer estimates, Doll and Peto emphasized that some estimates were much more firmly established than others. In particular, they stressed that the evidence on tobacco was much stronger than that for diet.

With regard to tobacco, their paper stated,

> By far the largest reliably known percentage is the 30 percent of current U.S. cancer deaths that are due to tobacco... No single measure is known that would have as great an impact on the number of deaths attributable to cancer as a reduction in the use of tobacco or a change to the use of tobacco in a less dangerous way.

In contrast, Doll and Peto described the dietary evidence this way,

> For many years there has been strong but indirect evidence that most of the cancers that are currently common could be made less so by suitable modification of national dietary practices... but there is still no precise and reliable evidence as to exactly what dietary changes would be of major importance.

Table 3, which is also derived from the work of Doll and Peto, clearly illustrates the differences in the certainty of the estimates for tobacco and diet. This table lists the reliably established, practicable ways of preventing cancer. Avoidance of tobacco smoke is obviously the most important factor. Dietary change doesn't even appear on the list (except as it may relate to avoidance of obesity, a minor cancer risk factor).

It has been more than a decade since Doll and Peto published their cancer estimates. In that time, a great deal of research has been conducted on diet and cancer. However, it is still not possible to specify with any certainty the proportion of cancer deaths that could be prevented by dietary change. Meanwhile, recent research has suggested that the role of tobacco is even more important than these 1981 estimates would indicate. The death rate from non-respiratory cancers, most of which are unrelated to smoking, has decreased in recent years. At the same time, it has become evident that passive smoking (exposure to tobacco smoke in the environment) plays a role in cancer causation. For these reasons, it is believed that the proportion of U.S. cancer deaths attributable to tobacco in the 1990s is at least 40 percent.

Individuals who set their own health priorities should realize that *the single most important step that people can take right now to reduce their risk of cancer is to abstain from using tobacco in any form.* It is also important to emphasize that *there is no known dietary change which will offset the harmful effects of cigarette smoking.* People who believe that they can safely continue to smoke as long as they adhere to the latest dietary recommendations are deluding themselves.

Table 2: Estimates of the Proportions of Cancer Deaths Theoretically Attributable to Various Factors

Factor	Percent of All Cancer Deaths	
	Best Estimate	**Range of Acceptable Estimates**
Tobacco	30	25-40
Alcohol	3	2-4
Diet	35	10-70
Food additives	<1	(-5) -2[a]
Reproductive and sexual behavior	7	1-13
Occupation	4	2-8
Pollution	2	<1-5
Industrial products	<1	<1-2
Medicines and medical procedures	1	0.5-3
Geophysical factors	3	2-4
Infection	10?	1-?
Unknown	?	?

[a] The -5 figure indicates that some additives may actually be protective against cancer.

Source: Doll, R, and R Peto. The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today. *Journal of the National Cancer Institute* 66:1191-1308, 1981.

Table 3: Reliably Established, Practicable Ways of Avoiding the Onset of Cancer

	Percentage of All U.S. Cancer Deaths
Avoidance of tobacco smoke	30
Avoidance of alcohol	3
Avoidance of obesity	2
Regular cervical screening and genital hygiene	1
Avoidance of non-essential medical use of hormones or radiation	<1
Avoidance of unusual exposure to sunlight	<1[a]
Avoidance of known effects of occupational exposures to carcinogens	<1
Avoidance of known effects of carcinogens in food, water, and air	<1

[a]This table deals only with life-threatening cancers. Avoidance of exposure to sunlight would also lead to a substantial reduction in the incidence of the common types of superficial skin cancer, most of which are not life-threatening.

Source: Peto, R, Cancer Around the World: Evidence for Avoidability, in Hallgren B et al, eds., *Diet and Prevention of Coronary Heart Disease and Cancer*, Raven Press, 1986.

Scientific Studies of Diet and Cancer

To interpret the findings of scientific studies of diet and cancer, it helps to be familiar with the different types of investigations that cancer researchers conduct. One important type of research is animal experimentation, most commonly using rats or mice as test animals.

Experiments conducted since the 1940s have shown that the diet fed to rodents can influence their cancer rates. The effects may be positive (increased incidence of cancer) or negative (reduced incidence of cancer). Sometimes the same dietary factor can either enhance or inhibit cancer in animals depending upon experimental conditions. Researchers use animal experiments to identify factors which might influence cancer risk, to investigate biological mechanisms and to study the effects of specific factors under closely controlled conditions. However, efforts to extrapolate the findings of animal experiments to the human situation are fraught with uncertainty.

To reduce that uncertainty, cancer researchers conduct epidemiological studies in human populations. There are several important types of epidemiological investigations.

Types of Epidemiological Investigations

In *correlational* investigations, scientists compare groups of people with different rates of a specific disease to see whether they differ in a particular risk factor. For instance, researchers might investigate whether levels of fat intake in various countries are correlated with the rates of particular types of cancer in those countries. Studies of this type are useful for generating new ideas, but their findings must be interpreted with caution. It is difficult to determine whether those in whom the disease develops are typical or atypical members of their particular group. It is also important to recognize that a correlation does not necessarily reflect a cause-and-effect relationship. For example, as societies develop, markets for consumer goods such as cigarettes and toilet paper increase. Because of this, there is a correlation between death from lung cancer and *per capita* toilet paper use, although few would seriously propose a cause-effect link.

It is for this reason that scientists frequently talk about "risk factors" rather than "causes." A risk factor is something that is associated with an increased risk of disease. It may or may not be a causative factor. Epidemiological studies can identify risk factors, but epidemiology alone cannot determine whether an association is causal. Findings from epidemiological investigations must be combined with other types of scientific evidence in order to determine whether the association between a risk factor and a disease is one of cause and effect.

Case-control and *cohort* studies focus on individuals rather than population groups. In case-control studies, individuals who have the disease under investigation are compared with otherwise similar individuals who do not have the disease, to see how they differ. These studies are *retrospective* — at the time of the investigation, the cases are already ill, and the focus is on past dietary habits (and other factors) that may have influenced current health status.

In cohort studies, researchers recruit groups of people who do not have the disease under investigation and examine various aspects of their health and lifestyle. They then observe these subjects over a period of time. At the end of the follow-up period, subjects who develop the disease are compared with those who do not. Cohort studies are *prospective,* meaning that information about the subjects is collected before any of them become ill.

Although both of these study designs have potential limitations, cohort studies are generally less susceptible to bias because the data are collected before the disease occurs. However, cohort studies are much more difficult and expensive to perform than case-control studies, they require much larger numbers of subjects, and they take many years to complete. Most studies of diet and cancer are of the case-control type. In the past few years, however, findings from several large cohort studies have become available.

One other important type of study is the *intervention* trial, in which the investigator randomly assigns the subjects to two groups, which are either exposed or not exposed to the factor under investigation. Studies of this type are useful for establishing cause-and-effect relationships, but they are extremely difficult to conduct when the factors of interest are complex, as diet is, or when the disease takes many years to develop, as cancer does. A few long-term intervention studies involving relatively simple diet modifications (such as taking or not taking a vitamin supplement) are

currently in progress, and results should become available within the next decade.

Fruits, Vegetables and Cancer

In the first edition of this report, published in 1985, vegetables and fruits earned only a brief discussion near the back of the booklet. At that time, the evidence that these foods might protect against cancer was not very strong. In the eight years since then, the situation has changed.

Today, a substantial body of scientific evidence links low intakes of fruits and vegetables with increased risks of a wide variety of cancers. Almost 200 epidemiological studies have examined the effects of fruits and vegetables. The vast majority have found that people who eat fewer fruits and vegetables have higher cancer risks. The evidence is particularly strong for cancers of the lung, larynx, oral cavity, pharynx and esophagus. There is also substantial evidence regarding cancers of the pancreas, stomach, colon-rectum, bladder, breast, cervix, ovary and endometrium. In fact, the only major cancer site for which the epidemiological evidence is inconclusive is the prostate.

The association between fruit and vegetable intake and cancer risk is large enough to be of practical importance. For most cancer sites, people with low intakes of these foods have about twice the risk of cancer seen in those with high intakes.

It has been argued that the apparent protective effect of fruits and vegetables might actually be due to the substitution of these foods for other foods associated with increased risk, such as meat or fat. However, enough data have accumulated now to lessen this possibility. A lack of fruits and vegetables has been linked with increased cancer risks in a wide variety of populations from around the world, including many groups that do not have the same pattern of correlated dietary habits found in the U.S. For example, in developing countries, people who do not eat fruits and vegetables compensate by eating more of their staple grain foods, not more meat and fat, yet fruits and vegetables appear to be protective in those populations just as they are in the U.S.

The Role of Antioxidants

There is a strong biological rationale for a protective effect of fruits and vegetables. These foods are the principal dietary sources of vitamin C and carotenoids,* and they are also a major source of vitamin E.# All three of these nutrients are *antioxidants,* meaning that they can prevent or repair damage caused by free radicals and other highly reactive chemical entities produced by oxidation. Free radicals and other reactive oxygen species are formed as by-products of normal metabolism or in response to external stressors such as tobacco smoke. They are believed to play a role in a variety of disease processes, including both the initiation and promotion stages of cancer development.

Other components of fruits and vegetables, including folic acid (a B vitamin) and certain non-nutritive substances such as indoles, may also contribute to cancer prevention; their roles and biochemical effects are not well understood. Some authorities especially have recommended the consumption of cruciferous (cabbage-family) vegetables, such as broccoli and cauliflower, because they are rich in non-nutritive substances which may act as cancer inhibitors. However, the evidence

for benefits attributable to this specific subgroup of vegetables is not very strong.

It may be many years before scientists fully understand the relative importance of various components of fruits and vegetables and the exact actions of each of these substances. However, it is not necessary to wait for a full understanding of the biochemistry before making sensible public health recommendations.

ACSH joins with federal agencies and the National Research Council in recommending that Americans include at least five servings of fruits and vegetables in their daily diets. In most epidemiological studies, this level of consumption has been associated with substantial reductions in cancer risk. It is a good idea to frequently consume fruits and vegetables that are rich in carotenoids and/or vitamin C. The box at the end of this report lists some of the best fruit and vegetable choices and gives information on how to prepare these foods in ways that preserve their vitamin C content.

Current Consumption Levels

The recommendation to eat at least five servings of fruits and vegetables daily may not appear unusual; after all, the traditional Basic Four Food Groups plan, devised in 1955, called for four daily servings of these foods. However, while the new recommendation is not dramatically different from previous advice, it is very different from what Americans actually eat. For example, one major survey of a representative sample of the U.S. population showed that:

- 41% of the people surveyed did not consume any fruit or fruit juice on the survey day.
- 17% ate no vegetables on the survey day.
- Only 28% consumed a fruit or vegetable rich in vitamin C.
- Only 21% consumed a fruit or vegetable rich in carotenoids.
- Only 27% consumed the recommended three servings of vegetables.
- Only 30% consumed the recommended two servings of fruits.
- A mere 10% had three servings of vegetables and two servings of fruits on the survey day.

Clearly, there is room for substantial improvement.

What About Vitamin Supplements?

Since fruits and vegetables have been linked to reduced risks of cancer, and since the antioxidant nutrients in these foods are probably the most important protective factors, it is reasonable to ask whether Americans should be advised to take supplements of these nutrients. **ACSH believes that a recommendation for widespread use of antioxidant supplements for cancer prevention is not warranted at the present time.**

The evidence for a cancer-protective effect of individual antioxidant nutrients is not as extensive or convincing as the evidence for a protective effect of fruits and vegetables. Foods are chemically complex mixtures, and it is very difficult to disentangle the effects of specific components. Substances in fruits and vegetables other than vitamin C, carotenoids and vitamin E may prove to be very important in cancer prevention, and a variety of other carotenoids may prove to be at least as

important as beta-carotene, the one carotenoid usually included in supplements. For these reasons, individuals who choose to take supplements instead of eating fruits and vegetables might end up with less effective health protection.

There is some reason for concern about the safety of antioxidant supplementation. Although the scientific evidence indicates that vitamin C, vitamin E and beta-carotene are of low toxicity, even when consumed in doses far beyond those normally obtained from food, a few scientific findings suggest that consumption of large doses of these vitamins might not be safe for all subgroups of the population.

For example, in an experiment in baboons, beta-carotene supplementation worsened the liver damage caused by large doses of alcohol. Whether this finding can be extrapolated to human alcohol abusers is uncertain. A recent human study suggested that vitamin E supplementation might increase the rate at which visual function is lost in patients with the hereditary eye disease retinitis pigmentosa. Vitamin C supplementation may be harmful to individuals with hemochromatosis, a hereditary disorder of iron metabolism. All of these issues require further investigation.

There is also legitimate reason for concern about the likelihood that people might take supplements inappropriately. Many vitamins and minerals can cause serious harm if consumed in excessive doses. An individual who chooses to increase his intake of antioxidants by taking several multivitamin tablets daily instead of the recommended dose of one tablet daily could be in serious trouble. Confusion about beta-carotene and vitamin A may also be a problem. Beta-carotene, a precursor of vitamin A and a strong antioxidant, is relatively nontoxic (although it can cause a reversible yellow discoloration of the skin if consumed in very large amounts). Vitamin A itself is not an antioxidant and is both toxic and teratogenic (capable of causing birth defects) if consumed in large doses.

Several intervention studies of the antioxidant vitamins are in progress, and others are planned. In these studies, large numbers of volunteers have been randomly assigned to take either a supplement or an inactive placebo on a regular basis for a period of years. Results of several intervention trials may be available within the next decade. At that time, it may be possible to make a definitive recommendation for or against the use of antioxidant supplements. By then, it is also likely that the small number of lingering concerns about the safety of the antioxidant vitamins will have been resolved.

While this ACSH report was in preparation, the first results of a major intervention trial were made public. In this trial, conducted in the rural county of Linxian, China, 30,000 people were randomly assigned to take various combinations of nutrient supplements or an inactive placebo, daily for five years. One of the nutrient combinations — beta-carotene, vitamin E and selenium — produced a statistically significant reduction in total mortality. The reduction was mainly due to lower rates of cancer, especially stomach cancer.

These findings are important, but their relevance for the American population is questionable. The people of Linxian, China are extremely different from the people of the U.S. in both dietary habits and disease patterns. The Linxian population has unusually high rates of esophageal and stomach cancers; scientists have long suspected that these cancers may be attributable, at least in

part, to nutritional deficiencies. The nutritional status of the people of Linxian is poor; only a limited variety of foods is available, and the population shows signs of subclinical deficiencies of several nutrients. In contrast, in the United States, esophageal and stomach cancers are relatively uncommon (see Table 1), varied diets are readily available, and nutritional deficiencies are rare.

The findings of the Linxian study should not be extrapolated directly to other populations. It is possible (many scientists would say probable) that the supplement exerted its beneficial effect by correcting a nutritional deficiency, and that similar benefits would not occur in well nourished populations, such as that of the U.S. The Linxian trial is important because it is the first large-scale intervention trial to demonstrate an association between vitamin / mineral supplementation and a reduction in cancer risk in humans. However, the findings of this trial do *not* justify a recommendation for supplement use by the American public.

Dietary Fat, Calories and Cancer

Of all the dietary factors under investigation, dietary fat intake has received the greatest emphasis both in research studies and in advice given to the public for cancer prevention. Yet despite extensive investigation, the scientific evidence relating dietary fat to cancer is far from conclusive.

Colon Cancer

The evidence for an effect of dietary fat is strongest for colon cancer. Animal experiments have suggested plausible mechanisms by which fat might influence the development of cancer at this site. Correlational studies have found strong associations between the amount of fat consumed in various countries and their rates of colon cancer. Many case-control studies have been completed, and most have linked higher total fat intakes with higher risks of colon cancer, although several studies have not found such an association. A very large cohort study — involving more than 80,000 female American nurses — found higher rates of colon cancer in women with higher intakes of animal fat.

Some uncertainties remain, however. One major problem in the interpretation of these studies is that diets high in fat are usually high in calories as well, and it may be that total energy (calorie) intake, rather than fat specifically, is the key factor. In experimental animals, calories are more important than fat; test animals who are raised on a high-calorie, low-fat diet have higher cancer rates than those raised on a low-calorie, high-fat diet. Whether the same is true in humans is uncertain. Because of the high correlation between fat intake and calorie intake in human populations, epidemiological studies have not been able to make a clear distinction between the two factors.

Another possible source of confusion is the varied nature of the fats found in foods. All fats are not identical. They are composed of a variety of fatty acids which may have different effects in the body. It is possible that differences in the effects of specific fatty acids might contribute to the variability of the effects seen in epidemiological studies.

Breast Cancer

The evidence linking dietary fat to breast cancer is less convincing than that for colon cancer. When effects have been found in case-control studies, they have usually been weak. A Canadian cohort study showed some evidence of a positive association between dietary fat intake and breast cancer risk, but three other cohort studies (the U.S. nurses' study, a smaller study of a representative sample of the U.S. population and a large study of postmenopausal women in the Netherlands) found no association.

As is the case for colon cancer, it is possible that total calorie intake may be more important than fat intake in determining breast cancer risk. There is also some reason to suspect that dietary factors might exert effects only in the early stages of life. If this is true, dietary changes later in life would not be worthwhile. It has also been suggested that only a drastic reduction in fat intake would be sufficient to cause a substantial decrease in the incidence of breast cancer. This argument may be valid, but its practical relevance is questionable, since an extreme reduction in dietary fat is unlikely to be achievable or acceptable in Western societies.

Some scientists still hope that it may be possible to decrease the incidence of breast cancer by reducing dietary fat intake. However, many — perhaps most — believe that this approach to breast cancer prevention is unlikely to be successful.

Prostate Cancer

The evidence for an association between dietary fat and prostate cancer is much less extensive than that for cancers of the breast and colon. Links between fat intake and prostate cancer risk have been seen in some case-control studies, but in some of these, effects were seen only in certain age groups or for specific fat sources. Cohort studies have had inconsistent results.

The ways in which fat might influence prostate cancer risk are poorly understood because there is no good animal model for this disease. Another complicating factor affecting all studies of prostate cancer is the high rate of latent disease. The ways in which men with latent prostate cancer may differ from those with clinically apparent, life-threatening disease are not well understood. In summary, the evidence on dietary fat intake and prostate cancer is inconclusive.

Other Cancer Sites

Animal experiments indicate that high dietary fat intake may increase the risk of pancreatic cancer. Whether this finding can be extrapolated to humans is unknown. Human pancreatic cancer is rapidly fatal; few patients live long enough to be interviewed by epidemiological researchers. Because investigation of this disease is so difficult, little is known about its risk factors.

Since cancers of the ovary and endometrium (lining of the uterus) are influenced by hormonal factors, which in turn can be influenced by diet, it is reasonable to propose that dietary fat intake might be associated with these cancers. However, only a very small number of studies of dietary

factors and endometrial or ovarian cancer have been completed, and their findings are inconclusive.

Recommendations on Dietary Fat Intake

Virtually all health authorities have recommended decreases in total fat intake, usually to 30 percent of total calories. This recommendation is not completely unreasonable, but its value in cancer prevention has been oversold to the public. **ACSH believes that moderation is desirable in all aspects of diet, including dietary fat intake. However, the case for reducing fat consumption to prevent cancer is not as strong as the public has been led to believe. High-fat diets have been linked to only one type of cancer, colon cancer, and even for that site, the evidence is not entirely conclusive.**

Some scientists argue that the evidence linking dietary fat and cancer does not need to be conclusive in order to warrant making recommendations to the public. They contend that cutting back on fat is justified for controlling obesity and reducing blood cholesterol levels. If it proves to reduce cancer risk as well, so much the better. If not, little has been lost.

There is some merit to this argument. However, it is also important to recognize that the dietary priorities for prevention of cancer are not exactly the same as those for the prevention of obesity or heart disease. For weight control, the key factor is the balance between calorie intake and calorie expenditure; cutting back on fat intake may be helpful (because fat is high in calories), but it is not sufficient. For cholesterol reduction, the key factor appears to be *saturated* fat intake, not *total* fat intake; switching from saturated to unsaturated fats may be at least as effective as cutting back on total fat. Moreover, it is still uncertain whether reduction of blood cholesterol levels is of value for all segments of the population and whether it is effective by itself in reducing total mortality.

Obesity

Since high calorie intakes increase cancer rates in experimental animals, it might be expected that obesity would increase the risk of cancer in humans. There is some epidemiologic evidence in support of an association between overweight and cancer. A large study by the American Cancer Society showed that cancer death rates increased progressively with increasing degrees of overweight in both sexes. Obesity has been linked to increased risks of endometrial cancer and postmenopausal (but not premenopausal) breast cancer in women. Data on other types of cancer are either limited or inconsistent.

The current evidence linking obesity to cancer, if considered alone, would be insufficient to justify any recommendations to the public. However, obesity is also associated with several other diseases, including diabetes, high blood pressure and coronary heart disease. **ACSH recommends that all Americans maintain a healthful body weight through appropriate eating and exercise habits.**

It is suggested occasionally that people should make a deliberate effort to maintain their body weight substantially below the normal range in an effort to reduce cancer risk and extend life span.

Although dietary restriction of this type does inhibit cancer and increase longevity in experimental animals, its risks and benefits in humans are not known. Extreme dietary restriction in an effort to prevent cancer is not recommended.

Dietary Fiber

The idea that increased consumption of dietary fiber may reduce the risk of cancer has become part of the conventional nutrition wisdom in the U.S. Yet there is a lack of good scientific evidence to support this claim.

Researchers find dietary fiber difficult to study because it is not one substance but many, and different types of fiber can have different effects in the body. Also, people do not consume fiber in isolation; it occurs as a component of grain products, vegetables and fruits that also contain many other substances, some of which may influence cancer risk. For these reasons, the results of studies of dietary fiber have been complex and confusing.

Official recommendations often state that fiber may help to prevent "some" types of cancer. In fact, the "some" refers only to two closely related cancer sites — the colon and rectum. There is no substantial scientific evidence linking fiber intakes to decreased risks of cancer at any other body sites in humans.

The epidemiological evidence on dietary fiber and cancers of the colon and rectum is inconsistent. Correlational studies have generally shown that groups of people who eat more fiber in general or more grain foods have lower colon cancer rates. In these studies, vegetables and fruits and the fibers that they contain, seemed to have little or no effect. In case-control studies, on the other hand, protective effects have generally been associated with vegetable fiber intake, while cereal fibers have often shown no effect or have been associated with actual *increases* in colon cancer risk. Only a few cohort studies have been completed, and none has shown a strong effect of fiber.

Like the epidemiological findings, the results of animal experiments have been inconsistent. In fact, under certain experimental conditions, some types of fiber actually increased colon cancer rates in experimental animals.

The 1985 edition of this report described the scientific evidence on fiber and colon cancer as "exceptionally equivocal and inconsistent." That conclusion remains valid today. Certainly, dietary fiber is a valuable component of the diet. A moderate intake of fiber contributes to the maintenance of normal digestive function; fiber has been recommended for this purpose for decades. Many Americans could benefit from including more fiber-rich foods in their diets; however, the evidence that increases in fiber would reduce cancer risk is weak.

ACSH believes that a recommendation to increase intake of dietary fiber in an effort to prevent colon cancer is not warranted on the basis of current evidence. Specific dietary recommendations should be given only when there is sufficient basis to expect that they will in fact accomplish what they promise. To do otherwise is to risk confusing the public and diverting attention from more important dietary and health priorities.

Cured, Smoked and Pickled Foods

The original 1982 dietary guidelines for cancer prevention proposed by the National Research Council included a recommendation to minimize intake of foods preserved by salt-curing, salt-pickling or smoking. Dietary recommendations issued by the National Cancer Institute repeated this advice, but it was not included in the *Dietary Guidelines for Americans* or the 1988 *Surgeon General's Report on Nutrition and Health,* and it was deemphasized in the 1989 National Research Council report *Diet and Health: Implications for Reducing Chronic Disease Risk.* There is good reason for this change in emphasis.

In some parts of the world where heavily smoked, salted and/or pickled foods are consumed on a daily basis as major dietary items, the death rates from cancers of the esophagus and stomach are very high. Carcinogens are present in foods preserved in these traditional ways, and they are believed to contribute to the high risks of stomach and esophageal cancers.

However, in the United States, esophageal cancer is rare, and the death rate from stomach cancer has been declining steadily for more than half a century. During the same time period, consumption of processed meat in the U.S. has increased substantially. There is no epidemiological evidence linking increased consumption of processed meats in the U.S. with increased risks of any types of cancer.

Why is the relationship between cured/smoked foods and cancer different in the U.S. than in some other countries? The main reason seems to be differences in the food products themselves. The cured or smoked foods most commonly consumed in the U.S., such as frankfurters, ham and bacon, are very different from the foods that have been linked with increased cancer risks in countries such as China, Japan and Iceland. Most of the cured, smoked or salted foods sold in the U.S. have received only mild treatments that give them their characteristic flavor and appearance; few products are treated to the extent needed to render them stable without refrigeration.

Because there is no evidence linking the cured, smoked or pickled foods commonly consumed in the U.S. with increased risks of any type of cancer, ACSH believes that a recommendation to avoid these foods is unwarranted.

Food "Chemicals" and Cancer

None of the dietary recommendations issued by the groups of scientific experts previously mentioned in this report advised Americans to avoid foods containing artificial additives, preservatives, pesticide residues or other "chemicals" in an effort to prevent cancer. This consensus is based on the fact that there is no good evidence linking added chemicals in the U.S. food supply with increased risks of any type of cancer. Unfortunately, many Americans continue to believe just the opposite. The misconception that "synthetic chemicals" cause most cases of cancer is remarkably widespread and persistent.

In some non-Western societies, certain "chemicals" in food have been linked with increased cancer risks. For example, certain parts of China have unusually high rates of nasopharyngeal,

esophageal and stomach cancers, which have been linked to the heavy consumption of certain traditionally preserved (salted or fermented) foods. These foods may contain carcinogenic chemicals produced by the preservation process or by the actions of contaminating microorganisms. In some parts of Asia and Africa, high rates of liver cancer may be related to the consumption of foods contaminated with aflatoxin or other chemical substances produced by molds. The molds can form toxins in grains, peanuts or other foods stored under less-than-optimal conditions, particularly in warm, moist climates.

In the U.S., mold contamination is limited by good storage practices. Foods are preserved primarily by refrigeration, freezing and canning, rather than by traditional methods of fermentation. Exposure to harmful contaminants is minimal.

Coffee and Tea

Two important sources of "chemicals" in the U.S. diet are coffee and tea, both of which contain a wide variety of chemical substances. However, there is no convincing evidence linking consumption of either coffee or tea with increased cancer risks in humans. In some epidemiological studies, increased intake of coffee has been associated with higher risks of bladder cancer, but experts suspect that this may reflect a bias rather than a cause-and-effect relationship. A recent combined analysis of eight well-designed epidemiological studies of coffee and bladder cancer found no evidence of increased risk. Coffee consumption is associated with an increased risk of fibrocystic breast disease but not with breast cancer.

Natural Carcinogens in Food

Much attention has been devoted recently to the discovery that the human diet contains a great variety of *natural* carcinogens. The quantity of natural mutagens and carcinogens in food is believed to be greater, by several orders of magnitude, than the quantity of man-made mutagens and carcinogens. Yet there is no evidence linking these natural carcinogens with increased cancer risks in humans. The human body appears to have defense and repair mechanisms capable of coping with these substances. This is fortunate since natural mutagens and carcinogens are widespread and no human diet could ever be entirely free from them.

In summary, the available evidence indicates that "chemicals" in the diet — including both man-made substances and naturally occurring food components — do not have a significant impact on cancer risk in the United States.

Alcohol and Cancer

Heavy drinking of alcohol is associated with increased cancer risks. Excessive alcohol intake, especially in combination with cigarette smoking, dramatically increases the risks of cancers of the mouth, larynx and esophagus. Alcohol abuse is associated with an increased risk of liver cancer, but

it is unclear whether this is a cause-and-effect relationship.

Some epidemiological studies have associated the drinking of large amounts of beer with an increased risk of rectal cancer; others have not shown this relationship. If the association is real, it may be attributable to carcinogenic substances called nitrosamines that may be produced during brewing. The nitrosamine content of beer sold in the U.S. has decreased in recent years as a result of improvements in brewing methods. Therefore, it is uncertain whether prior epidemiological findings are applicable to the types of beer currently on the market.

Breast Cancer

Reports that the consumption of moderate amounts of alcohol might increase a woman's risk of breast cancer have aroused understandable concern. However, the scientific evidence on this issue is far from conclusive.

The majority of epidemiological studies have found an association, usually a weak one, between alcohol intake and breast cancer, but others have found no association. In some studies, the association was seen only in women with premenopausal breast cancer, not in the larger number who develop the disease after menopause. A few reports, including one from the large ongoing cohort study of U.S. nurses, indicate that breast cancer risk is increased even in women who have fewer than seven alcoholic drinks per week, but others suggest that only heavy drinkers of alcohol (those consuming roughly three drinks or more per day) have a significant increase in risk. No biological mechanism by which alcohol might influence breast cancer risk has been established, and it is unclear whether the increased risk seen in some epidemiological studies is attributable to alcohol *per se* or to some other characteristic of women who drink alcohol as opposed to those who abstain.

The evidence associating alcohol with breast cancer is not sufficiently conclusive to warrant any public health recommendations. Even if the evidence were conclusive, it still might not prompt authorities to recommend that all women abstain from the use of alcohol. The reason for this is that moderate alcohol intake is also associated with a *decreased* risk of heart disease, and heart disease is far more common than breast cancer. Thus, the benefits of moderate alcohol intake might exceed the risks, even if alcohol were proven to contribute to breast cancer.*

Recommendations

All authorities that issue dietary guidelines for the American public concur in recommending that if people choose to drink alcohol, they should do so in moderation. This is sound advice. Heavy drinking of alcohol is clearly associated with many risks to health and safety, including an increased risk of some types of cancer.

ACSH joins with other health authorities in recommending moderation in the use of alcohol. The current evidence does not justify a recommendation that women abstain from drinking alcohol in an effort to prevent breast cancer.

Diet and Cancer: Conclusions

This report has examined the information relating to diet, nutrition and cancer, and the debate among scientists as to whether recommendations on specific issues are warranted based on current knowledge. It is important to emphasize that scientific knowledge of diet and cancer is still developing, and much remains to be learned.

The 1985 edition of this report concluded that there was “insufficient evidence to warrant establishment of a public policy of guidelines for diet modification for all Americans for the purpose of reducing the risk of cancer.” For most aspects of diet, this conclusion remains valid. However, the evidence linking fruit and vegetable intake with reduced risk of cancer has become substantially stronger in the past eight years. On the other hand, the evidence linking fiber to colon cancer and fat to breast cancer is actually weaker now than it was eight years ago.

ACSH continues to believe that it is important to make recommendations to the public only when there is a sound, scientific basis for the proposed course of action. ACSH opposes recommendations that go beyond a prudent interpretation of current knowledge. The temptation to substitute wishful thinking for valid scientific conclusions must be resisted. Authorities who issue recommendations to the public should clearly distinguish between lifestyle changes with firmly established benefits and those of merely hypothetical value.

For Good Nutrition, Experts Recommend that Everyone Should Have at Least 3 Servings of Vegetables and at Least 2 Servings of Fruit Every Day

What Counts as One Serving?

1 cup of raw leafy green vegetables
1/2 cup of other raw or cooked vegetables
1 piece of fruit
1 wedge of melon
3/4 cup of juice
1/2 cup of canned fruit
1/4 cup of dried fruit

The Best Sources of ß-Carotene and other Carotenoids Include:

orange-yellow vegetables (carrots, sweet potatoes, winter squash)
dark-green leafy vegetables (spinach, kale, collards)
broccoli
orange-yellow non-citrus fruits (canteloupe, apricots)

The Best Sources of Vitamin C Include:

citrus fruits (oranges, grapefruit, tangerines) and their juices
strawberries
certain vegetables (peppers, tomatoes, cabbage, broccoli, cauliflower; potatoes also provide some vitamin C)

To Preserve the Vitamin C Content of Foods:

Eat some fruits and vegetables raw, since cooking decreases vitamin C levels
Cook vegetables only until they reach the crisp-tender stage. Use minimal amounts of water, or steam or microwave vegetables.

SELECTED BIBLIOGRAPHY

Recent Reviews of the Scientific Literature

Adami, HO et al, Report from the Working Group on Diet and Cancer, *Pharmacology and Toxicology* 72:S176-S179, 1993.

Ames, BN, Dietary Carcinogens and Anticarcinogens. Oxygen Radicals and Degenerative Diseases, *Science* 221:1256-1264, 1983.

Ausman, L, Fiber and Colon Cancer: Does the Current Evidence Justify a Preventive Policy? *Nutrition Reviews* 51:57-63, 1993.

Block, G, B Patterson, and A Subar, Fruits, Vegetables, and Cancer Prevention: A Review of the Epidemiological Evidence, *Nutrition and Cancer* 18:1-29, 1992.

Block, G, The Data Support a Role for Antioxidants in Reducing Cancer Risk, *Nutrition Reviews* 50:207-213, 1992.

Block, G, Vitamin C Status and Cancer. Epidemiologic Evidence of Reduced Risk, *Annals of the New York Academy of Sciences* 669:280-290, 1992.

Byers, T, and G Perry, Dietary Carotenes, Vitamin C, and Vitamin E as Protective Antioxidants in Human Cancers, *Annual Review of Nutrition* 12:139-159, 1992.

Etherton, GM and MS Kochar, Coffee. Facts and Controversies, *Archives of Family Medicine* 2:317-322, 1993.

Freudenheim, JL and S Graham, Toward a Dietary Prevention of Cancer, *Epidemiologic Reviews* 11:229-235, 1992.

Howe, G et al, The Association Between Alcohol and Breast Cancer Risk: Evidence from the Combined Analysis of Six Dietary Case-Control Studies, *International Journal of Cancer* 47:707-710, 1991.

Howe, GR, Dietary Factors and Risk of Breast Cancer: Combined Analysis of 12 Case-Control Studies, *Journal of the National Cancer Institute* 82:561-569, 1990.

Howe, GR, High-Fat Diets and Breast Cancer Risk. The Epidemiologic Evidence, *Journal of the American Medical Association* 268:2080-2081, 1992.

Kelsey, JL and MD Gammon, Epidemiology of Breast Cancer, *Epidemiologic Reviews* 12:228-240, 1990.

Knekt, P et al, Vitamin E and Cancer Prevention, *American Journal of Clinical Nutrition* 53:283S-286S, 1991.

Kritchevsky, D, Caloric Restriction and Experimental Tumorigenesis, *Nutrition Today*, Jan/Feb 1993, pp. 25-27.

Kritchevsky, D, Undernutrition and Chronic Disease: Cancer, *Proceedings of the Nutrition Society* 52:39-47, 1993.

Nomura, AMY and LN Kolonel, Prostate Cancer: A Current Perspective, *American Journal of Epidemiology* 13:200-227, 1991.

Roebuck, BD, Dietary Fat and the Development of Pancreatic Cancer, *Lipids* 27:804-806, 1992.

Rose, DB and JM Connolly, Dietary Fat, Fatty Acids and Prostate Cancer, *Lipids* 27:798-803, 1992.

Scheuplein, RJ, Perspectives on Toxicological Risk — An Example: Food-borne Carcinogenic Risk, in Clayson DB et al, eds., *Progress in Predictive Toxicology*, Elsevier Science Publishers B.V., 1990.

Trock, B, E Lanza, and P Greenwald, Dietary Fiber, Vegetables, and Colon Cancer: Critical Review and Meta-analyses of the Epidemiologic Evidence, *Journal of the National Cancer Institute* 82:650-661, 1990.

Viscoli, CM, MS Lachs, and RI Horwitz, Bladder Cancer and Coffee Drinking: A Summary of Case-Control Research, *Lancet* 341:1432-1437, 1993.

Ziegler, RG, Vegetables, Fruits, and Carotenoids and the Risk of Cancer, *American Journal of Clinical Nutrition* 53:251S-259S, 1991.

Sources of Dietary Recommendations

National Research Council, *Diet and Health: Implications for Reducing Chronic Disease Risk*, Washington, DC: National Academy Press, 1989.

National Research Council, *Diet, Nutrition and Cancer*, Washington, DC: National Academy Press, 1982.

U.S. Department of Health and Human Services, *The Surgeon General's Report on Nutrition and Health*, DHHS (PHS) Publication No. 88-50210, 1988.

U.S. Departments of Agriculture and Health and Human Services, *Nutrition and Your Health: Dietary Guidelines for Americans*, Third Edition, 1990.

Selected Individual Studies of Diet and Cancer

Blot, WJ et al, Nutrition Intervention Trials in Linxian, China: Supplementation with Specific Vitamin/Mineral Combinations, Cancer Incidence, and Disease-Specific Mortality in the General Population, *Journal of the National Cancer Institute* 85:1483-1492, 1993.

Carstensen, JM, LO Bygren, and T Hatschek, Cancer Incidence Among Swedish Brewery Workers, *International Journal of Cancer* 45:393-396, 1990.

Chu, SY et al, Alcohol Consumption and the Risk of Breast Cancer, *American Journal of Epidemiology* 130:867-877, 1989.

Freudenheim, JL et al, Lifetime Alcohol Intake and Risk of Rectal Cancer in Western New York, *Nutrition and Cancer* 13:101-109, 1990.

Friedenreich, CM et al, A Cohort Study of Alcohol Consumption and Risk of Breast Cancer, *American Journal of Epidemiology* 137:512-520, 1993.
Hiatt, RA and RD Bawol, Alcoholic Beverage Consumption and Breast Cancer Incidence, *American Journal of Epidemiology* 120:676-683, 1984.
Howe, GR et al, A Cohort Study of Fat Intake and Risk of Breast Cancer, *Journal of the National Cancer Institute* 83:336-340, 1991.
Jones, DY et al, Dietary Fat and Breast Cancer in the National Health and Nutrition Examination Survey. I. Epidemiologic Follow-up Study, *Journal of the National Cancer Institute* 79:465-471, 1987.
Lew, EA and L Garfinkel, Variations in Mortality by Weight among 750,000 Men and Women, *Journal of Chronic Diseases* 32:563-576, 1979.
Simon, MS et al, Alcohol Consumption and the Risk of Breast Cancer: A Report from the Tecumseh Community Health Study, *Journal of Clinical Epidemiology* 44:755-761, 1991.
Thun, MJ et al, Risk Factors for Fatal Colon Cancer in a Large Prospective Study, *Journal of the National Cancer Institute* 84:1491-1500, 1992.
van den Brandt, P et al, A Prospective Cohort Study on Dietary Fat and the Risk of Postmenopausal Breast Cancer, *Cancer Research* 53:75-82, 1993.
Willett WC, Moderate Alcohol Consumption and the Risk of Breast Cancer, *New England Journal of Medicine* 316:1174-1180, 1987.
Willett, WC et al, Dietary Fat and Fiber in Relation to Risk of Breast Cancer. An 8-Year Follow-up, *Journal of the American Medical Association* 268:2037-2044, 1992.
Willett, WC et al, Relation of Meat, Fat, and Fiber Intake to the Risk of Colon Cancer in a Prospective Study among Women, *New England Journal of Medicine* 323:1664-1672, 1990.

Other References

American Cancer Society, *Cancer Facts & Figures—1992.*
Bendich A, Safety Issues Regarding the Use of Vitamin Supplements, *Annals of the New York Academy of Sciences* 669:300-310, 1992.
Bendich A and M Cohen, Ascorbic Acid Safety: Analysis of Factors Affecting Iron Absorption, *Toxicology Letters* 51:189-201, 1990.
Berson, EL et al, A Randomized Trial of Vitamin A and Vitamin E Supplementation for Retinitis Pigmentosa, *Archives of Ophthalmology* 111:761-772, 1993; see also the accompanying editorial Massof RW and D Finkelstein, Supplemental Vitamin A Retards Loss of ERG Amplitude in Retinitis Pigmentosa, *Archives of Ophthalmology* 111:751-754, 1993.
Block, G, Dietary Guidelines and the Results of Food Consumption Surveys, *American Journal of Clinical Nutrition* 53:356S-357S, 1991.
Doll, R, and R Peto, The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today, *Journal of the National Cancer Institute* 66:1191-1308, 1981.
Leo, MA et al, Interaction of Ethanol with ß-Carotene: Delayed Blood Clearance and Enhanced Hepatotoxicity, *Hepatology* 15:883-891, 1992.
Murphy, SF, AF Subar, and G Block, Vitamin E Intakes and Sources in the United States, *American Journal of Clinical Nutrition* 52:361-367, 1990.
Peto, R, Cancer Around the World: Evidence for Avoidability, in Hallgren B et al, eds., *Diet and Prevention of Coronary Heart Disease and Cancer,* Raven Press, 1986.
Ravenholt, RT, Tobacco's Impact on Twentieth-Century U.S. Mortality Patterns, *American Journal of Preventive Medicine* 1:4-17, 1985.
U.S. Departments of Agriculture and Health and Human Services, *Healthy People 2000. National Health Promotion and Disease Prevention Objectives,* 1991.

* All estimates in this paragraph are based on American Cancer Society projections for the year 1992.

* Carotenoids are a group of red, orange and yellow pigments found in plants. They are responsible for the colors of many vegetables and a few fruits, including carrots, sweet potatoes, tomatoes, corn and cantaloupe. Dark green vegetables, such as spinach and broccoli, also contain carotenoids, but in these foods the characteristic colors of the carotenoid pigments are masked by the green color of chlorophyll. Some carotenoids can be converted to vitamin A in the body; others cannot. Current evidence indicates that it is the antioxidant activity of carotenoids, rather than conversion to vitamin A, that may be relevant to cancer prevention.

[#] It is difficult for people to increase their intake of vitamin E from dietary sources because the foods that are richest in this vitamin (vegetable and seed oils; products made with these oils such as mayonnaise and margarine; nuts; and wheat germ) are high in fat and calories. Increased intake of these foods is generally not desirable. Fruits and vegetables provide smaller amounts of vitamin E, but they are low in fat and calories and therefore can be eaten in generous amounts. Some experts recommend that people who need to increase their vitamin E intake should do so by eating more fruits and vegetables.

[*] Of course, this balancing of benefits and risks applies only to individuals who can drink alcohol safely. Many women, including those who are pregnant, lactating or attempting to conceive; those who have a history of alcoholism or who cannot keep their alcohol intake moderate; and those who take certain types of medication, must avoid alcohol completely.