

“Cancer Is Not Inevitable”

National Research Council panel recommends changes in diet to reduce the risk of cancer

“It is highly likely that the United States will eventually have the option of adopting a diet that reduces its incidence of cancer by approximately one-third,” according to a new report issued this month by the National Research Council.* “The evidence is increasingly impressive,” says Clifford Grobstein of the University of California, San Diego, chairman of the panel that prepared the report, “that what we eat does affect our chances of getting cancer, especially particular kinds of cancer. This is . . . good news because it means that by controlling what we eat we may prevent such diet-sensitive cancers.”

The bad news, he continues, is that the committee “does not yet think it possible to say . . . how much the incidence of particular cancers might be reduced by dietary alteration. Certainly we have no ideal cancer-preventing diet to announce.” What the panel did announce was some modest suggested changes in dietary habits that should at the very least, in Grobstein’s words, “reduce anxiety” about cancer. The suggestions are similar to the “prudent diet” recommended by the American Heart Association to minimize heart disease, to the “Dietary Goals for the United States” issued in 1977 by the Senate Select Committee on Nutrition and Human Needs, and to the unofficial guidelines recommended in 1979 by Arthur Upton, then director of the National Cancer Institute, in testimony before the Senate committee. Nonetheless, the report marks the first time that any official body has suggested that the risk of cancer can be alleviated by dietary changes.

The “interim dietary guidelines” fall into four major categories:

► The proportion of calories in the diet provided by fats should be reduced from 40 percent to 30 percent. “Of all the dietary compounds [the panel] studied, the combined epidemiological and experimental evidence is most suggestive for a causal relationship between fat intake and the occurrence of cancer”—especially cancers of the colon, breast, and prostate. This is in sharp contrast to a 1980 report *Toward Healthful Diets* by

the Food and Nutrition Board of the National Academy of Sciences, which concluded that “there is no basis for recommendations to modify the proportions [of fat] in the American diet at this time.” Questioned about this at a press conference, Grobstein said “We know more now.”

► The daily diet should include whole-grain cereals, fruits, and vegetables, especially those high in vitamin C and β -carotene, which is converted by the body into vitamin A. These foods include citrus fruits, dark-green and deep-yellow vegetables, and members of the family Cruciferae (such as cabbage, broccoli, cauliflower, and Brussels sprouts). “In laboratory experiments,” says Grobstein, “these vitamins, the mineral selenium, and some nonnutritive chemicals present in cruciferous vegetables inhibit the formation of cancer-causing chemicals or reduce cancer incidence in other ways.” The report strongly recommends against injudiciously supplementing diets with these substances because of potential side effects.

► The consumption of salt-cured, salt-pickled, and smoked foods should be minimized because they are associated with an increased incidence of cancers at certain sites, particularly the stomach and the esophagus. In the United States, such foods include sausages, smoked fish and ham, bacon, and hot dogs.

► Excessive consumption of alcohol should be avoided, particularly in combination with cigarette smoking. Such consumption has been associated with an increased risk of cancer of the upper gastrointestinal and respiratory tracts, as well as with other adverse health effects.

The report was commissioned by the National Cancer Institute during the Carter Administration, in 1980. Its fate under the Reagan Administration seems problematic. That Administration’s view is thought to have been summarized by Agriculture Secretary John R. Block, who testified during his confirmation hearings that “I’m not so sure government should get into telling people what they should or shouldn’t eat.” The report’s principal value may thus lie not in its guidelines but rather in its summarization of the current state of knowledge about dietary links to cancer.

The subject is a particularly difficult one to study—a “minefield” in the words of the panel’s vice chairman, John Cairns of the Harvard School of Public Health. The complex nature of cancer initiation, the formidable task of determining what an individual consumed 20 or even 30 years before the onset of a tumor, the difficulties of regulating the components of diet for experimental animals, the heterogeneity of the human population, and the wide variety of changing life-styles are just some of the problems that confront researchers.

The foundation for the link between diet and cancer rests on two key facts. The first is that, with the exception of lung cancer resulting from smoking, the incidence of most types of cancer has not changed appreciably during the 20th century. This, and the observation that the incidence of many types of cancers is higher in nonindustrialized countries such as New Zealand than in the United States, indicates that the most common cancers “are related, for the most part, not to industrialization but to various other long-standing features of our life-style, especially diet.”

The second key fact is that the incidence of various types of cancers varies from country to country. When people migrate from one country to another, “they tend to acquire the pattern of cancer that is characteristic of their new homes. This is surely the most comforting fact to come out of all cancer research, for it means that cancer is, in large part, a preventable disease.”

The evidence associating fats with cancer, in fact, first came from studies of Japanese who migrated to the United States, abandoning their traditional low-fat diet in favor of a meatier one in the United States. Evidence for the link was subsequently strengthened by a large number of epidemiological studies as well as by studies of laboratory animals. Most investigators now believe that dietary fats act as promoters, agents that do not themselves cause cancer but that enhance the activity of carcinogens.

The recommendation to reduce dietary fat has so far been the most controversial aspect of the report. The American Meat Institute, a trade group of cattle growers, charged the panel with

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promoting "misleading advice which does no service to the public." Commented Grobstein: "I don't think we're disseminating unproved theories." The panel may also come into conflict with the American Heart Association, which has been promoting not only a low-fat diet but also a shift to polyunsaturated fats. Animal evidence cited in the report indicates that in a low-fat diet, polyunsaturated fats are more effective than saturated fats in promoting tumor formation. Panel member Anthony B. Miller of the National Cancer Institute of Canada predicted that it will be some time before the effects of polyunsaturated fats is known in humans.

Another potentially controversial aspect of the report involves dietary fiber. Several investigators, led by Denis P. Burkitt of St. Thomas's Hospital in London, argue that a high proportion of nonnutritive fibers, such as cellulose, lignin, gums, and pectins, in the diet protect against cancer of the colon and rectum. The fiber is believed to act primarily as a bulking agent, diluting the concentration of potential carcinogens in the feces and hastening their passage through the bowels.

The panel, however, concluded that there is "no conclusive evidence to indicate that dietary fiber . . . exerts a protective effect." If such an effect does exist, furthermore, it is most likely to be "related to the intake of one fiber component—the pentosan fraction," found primarily in whole wheat products. Burkitt contends that the evidence is sufficiently strong to advocate a significant daily consumption of fiber.

The status of other dietary components reviewed in the report is less controversial but no less complex. For most of them, the evidence is suggestive but inconclusive. Protein is a good example. Epidemiological studies have suggested an association between high consumption of protein and increased risk of cancer at several sites, including breast, large bowel, pancreas, prostate, and kidney. These studies are clouded, however, by the close association of protein and fats in the diet. Studies in laboratory animals indicate that carcinogenesis is suppressed when protein consumption is at or below the minimum level required for optimal growth. The panel thus concludes that "high protein intake *may* be associated with an increased risk of cancer at certain sites."

A similar situation exists for carbohydrates. High consumption of sugar is associated with an increased risk of pancreatic cancer in women, and a high intake of potatoes is associated with liver



A prudent meal

Salmon mousse and vegetables are good for the heart and minimize cancer risk.

cancer in both sexes. Frequent consumption of starches has also been associated with gastric and esophageal cancer, while a high intake of sugar combined with a low intake of starch has been associated with an increased incidence of breast cancer. The total evidence, however, "is too sparse to suggest a direct role for carbohydrates in carcinogenesis. However, excessive carbohydrate consumption contributes to caloric excess, which in turn has been implicated as a modifier of carcinogenesis."

There is little information about vitamins, apart from A and C. Vitamin E, like C, inhibits the formation of carcinogenic nitrosamines, but it is present in so many different commonly consumed foods that it is difficult to identify population groups that do not receive enough. There is virtually no information about the B vitamins.

Results are also scanty for minerals. Both epidemiological and laboratory studies suggest that selenium may offer some protection against cancer, but many of those studies used near toxic levels of selenium. Iron deficiency has been indirectly associated with cancer of the upper alimentary tract, and perhaps also with gastric cancer. Zinc may be associated with tumor formation, but experiments in animals suggest that it both enhances and retards carcinogenesis, depending on concentration and experimental design. Molybdenum deficiency may be associated with an increased risk of esophageal cancer. Both an excess and a deficiency of iodine may be associated with an increased risk of thyroid cancer.

Of perhaps greater concern in the long run is the large number of hazardous extraneous chemicals in the diet. These include hazardous constituents of foods themselves, environmental contaminants, mutagens produced during cooking, and both intended and inadvertent food additives. There are some 3000 food additives that are used intentionally, an

estimated 12,000 that are added inadvertently during processing and packaging, and an unknown but presumably large number of natural constituents and environmental contaminants. Most of these, fortunately, are present at quite low levels and their effects may be negligible. It is "reassuring," the panel argues, that cancer incidence rates have not increased during this century, for this suggests that these nonnutritive substances have either a small constant effect or little effect at all. The panel cautions, however, that any effects attributable to the greatly increased use of processed foods and changes in cooking habits in the last 20 to 30 years may not yet have become apparent.

One of the most important questions that remains to be answered is the relation between mutagenesis and carcinogenesis. Mutagens are widely distributed in the diet. Many vegetables contain mutagenic flavonoids, and mutagenic activity has been observed in extracts from many foods, including coffee, tea, and alcoholic beverages. Mutagens are produced in meat and fish by the high-temperature pyrolysis of proteins, and recent studies have shown that even low-temperature cooking also leads to production of mutagens.

Most of the mutagens that have been found in foods have not been tested for carcinogenic activity, but there is a growing body of evidence suggesting that mutagens are likely to be carcinogens. Some scientists, however, argue that the link is inconclusive and that the association should not be used as a basis for regulation. Cairns espouses what seems to be a majority viewpoint when he notes that it would be foolish to believe that any compound which is a potent mutagen in bacterial or animal systems is not a hazard to humans. Even if mutagens should prove carcinogenic, though, difficult decisions will be necessary. Cooking of meat and fish, for example, may produce mutagens, but it also destroys pathogenic microorganisms and parasites. Some vegetables that contain mutagens also have high nutritive value.

The report emphasizes that "the weight of evidence suggests that what we eat during our lifetimes strongly influences the probability of developing certain kinds of cancer, but that it is not now possible, and may never be possible, to specify a diet that protects all people against all forms of cancer." Nonetheless, concludes Grobstein, "it is time to further spread the message that cancer is not as inevitable as death and taxes."—THOMAS H. MAUGH II