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Perspectives on dietary fat, cancer report

(An outbreak of scientific controversy flared in the popular media last year following a National Academy of Science report in the summer of 1982 on diet and cancer. The following presents a brief summary of the report, along with the responses JAOCS *received when it asked for comments from cancer-session speakers who participated in the 1981 AOCS Conference on Dietary Fats and Health.)*

Explaining that "it is not now possible, and may never be possible, to specify a diet that would protect everyone against all forms of cancer," a National Research Council committee appointed by the National Academy of Sciences last summer issued a report on diet and cancer advising less consumption of fat and cured meat.

The National Cancer Institute had asked the committee to formulate dietary guidelines to minimize risk of cancer. In its report, *Diet, Nutrition, and Cancer*, the committee said it could offer only interim guidelines, not firm solutions. "The evidence suggests that some types of diets and some dietary components (e.g., high fat diets or the frequent consumption of certain fruits and vegetables) tend to decrease it," the committee said, but added that there are not enough data to specify what percentage of cancer risks can be attributed to diet or to what extent improved diets might lower these risks. "It is important . . . that we prepare ourselves for a period of uncertainty, between our present realization that diet affects cancer and our eventual ability to offer the public a precise formula for minimizing the incidence of cancer."

The committee suggested that, in general, Americans should:

– reduce consumption of saturated and unsaturated fats from approximately 40% to 30% of total caloric intake. The major sources of fat in the American diet are fatty cuts of meat, whole-milk dairy products and cooking oils and fats. The committee said both epidemiological and laboratory studies showed higher rates of cancer of the breast, large bowel and prostate in populations that eat foods containing large amounts of both saturated and unsaturated fats.

– eat fruits, vegetables and whole-grain cereal products daily, especially those high in vitamin C and carotene, which converts to vitamin A in the body. The report said frequent consumption of these foods can reduce susceptibility to cancers of the urinary bladder, large bowel, skin, lung, stomach and esophagus.

– consume little salt-cured, salt-pickled and smoked foods.

– drink alcohol in moderation.

The committee concluded that most common cancers are potentially preventable for they appear to be determined more by habit, diet and custom than by genetic differences. It added that since data are incomplete, the National Cancer Institute should review dietary guidelines at least every five years. The committee is expected to complete a second report in June 1983 which will examine priorities for future diet and cancer research.

The report elicited a variety of reactions, from full endorsement to total disagreement. Announcement of the guidelines quickly prompted a challenge by nine food industry trade groups – American Meat Institute, Poultry and Egg Institute of America, United Egg Producers, National Turkey Federation, National Milk Producers Federation, National Cattlemen's Association, National Broiler Council, National Livestock and Meat Board, and National Pork Producers Council – asking that a special task force be formed within NAS "to review and prepare a report" on the dietary guides. Trade groups said the premise that good nutritional practices were likely to reduce the risk of cancer was unproven. (For details, see the *Washington Food Report*, June 19, 1982, p.2.).

Meanwhile, the Council for Agricultural Science and Technology (CAST) asked scientists to comment on the report. The result was publication of *Diet, Nutrition, and Cancer: A Critique*, which included comments from 47 scientists, most from state land grant universities. A chief concern expressed was that the report "makes recommendations of public policy on the basis of inadequate evidence." (See *Food Chemical News*, Oct. 25, 1982, pp. 48-49.)

Another reaction came from the Center for Science in the Public Interest, which asked the Food and Drug Administration and the Department of Agriculture to require fat labeling on all foods. It also asked that labeling specify polyunsaturated, monounsaturated and saturated fatty acid content. (For details, see *Food Chemical News*, Aug. 23, 1982, pp. 11-12.)

In an editorial in the November/December issue of *ACSH News & Views*, Dr. Elizabeth Whelan, executive director of the American Council on Science and Health, called the recommendations for dietary change premature and said, "While some evidence indicates that fat intake and the risk of certain cancers may be linked, there is also considerable evidence that does not support this conclusion."

The following four views on the report were written by specialists on cancer and lipids who were contacted by *JAOCS*.

(Copies of *Diet, Nutrition, and Cancer*, 488 pages, are available from National Academy Press, 2101 Constitution Ave., NW, Washington, DC 20418, for $13.50. The 80-page

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CAST Publication No. 13, *Diet, Nutrition, and Cancer: A Critique*, is available for $4.50 from CAST, 250 Memorial Union, Ames, IA 50011.)

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Although the concept that diet and nutrition might influence cancer is not a new one, this relationship has received surprisingly little detailed attention. During the 1930s, a number of laboratories were interested in the possible influence exerted by nutritional factors on susceptibility to cancer, but the question soon lost the interest of both scientific and lay communities. Now, there is a growing belief that dietary factors play a predominant role in the causation of cancer in humans.

I have followed closely the previous suggestions and/or reports by the NRC's Food and Nutrition Board entitled *Toward Healthful Diets*, the American Heart Association's *Prudent Diet*, and the Senate Select Committee's *Dietary Goals for the United States*. In the last few years, remarkable advances have been made in our understanding of nutrition as it relates to the risk of certain types of cancer. Much has been learned with respect to the natural history and basic understanding of nutrition-related cancer. Whereas these prior dietary suggestions were directed mainly toward reducing the risk for cardiovascular disease, the present suggestions are directed toward the prevention of certain types of cancer.

This scientific review pertains to the NRC's report, *Diet, Nutrition, and Cancer*. First, let me comment on the members of the NRC's Committee on Diet, Nutrition, and Cancer. These committee members are highly qualified experts in their field and competent to assess the scientific evidence. The purpose of this committee was to evaluate the current state of research on nutrition and its relationship to cancer, to determine the possible causes that have been established, to question when inconsistencies remained and to propose preventive measures on the basis of present evidence. This report, in contrast to the previous report, *Toward Healthful Diets*, by the NRC's Food and Nutrition Board, is, indeed, a comprehensive assessment and detailed appraisal of the current knowledge concerning the dietary components as they relate to certain types of cancer.

It is my belief that the committee has evaluated the evidence from all types of studies, namely, human epidemiological, and experimental animal model studies. It made a careful evaluation of all parameters whereby our diet does or could influence cancer development. It has discussed in detail the limitations of each procedure and arrived at the conclusion that the results of the epidemiological studies in humans and the experimental animal model studies provided convincing evidence as to the role of dietary and metabolic factors in the development of certain types of cancer. Particularly within the last decade, epidemiological evidence supported by extensive experimental studies has steadily advanced the concept that nutrition, in many ways, affects human carcinogenesis. When the animal model studies complement the epidemiological observations of human risks, there is no reason, in my view, to doubt the validity of the evidence. It is quite obvious to those in the area of nutrition and cancer that it represents a significant document and a valuable resource to everyone concerned with this subject. It is likely to serve as a significant impetus to future progress in an important area of cancer prevention. Thus, as discussed in the report, many of the gaps in our knowledge could be eliminated.

The following review considers certain aspects relating to the report and to the conclusion reached. It focuses on those aspects that pertain to my area of interest and research.

1) With regard to nutritional studies that assess the relation of certain dietary components to cancer, the committee realized that the nutritional intake within a given population cannot readily be studied because of inherent difficulties with nutritional surveys. The problem is enhanced by various dietary components being interrelated. When evaluating one nutrient parameter, it is difficult to isolate it from other dietary factors because of the complex interactions among them. Thus people who get most of their calories from fat are likely to have a low intake of starches, and vice versa. It also has been recognized that it is difficult to relate diet to cancer, since cancer has a long latency period and current diet histories might not represent what people ate in the distant past. We are interested in learning not only what people consumed recently, i.e., during the previous week, but also what they ate in the more distant past. The belief that individuals can report accurately not only what they usually eat but also what they actually consumed is untested. However, recent information suggests that recall of a diet consumed in the more distant past may closely reflect present food choices. Conclusions were reached by comparing nutritional intakes not only from one population group to another, but from migrant populations, special groups within the population, and also from case-control and cohort studies. The relationship between dietary factors and cancer has been investigated by correlational, case-control and cohort studies. The committee placed more emphasis in the data from case-control and cohort studies which, in my opinion, were more definitive and reliable. The results obtained from these reliable human studies were complemented by the convincing evidence in animal model studies that have been reproduced in different laboratories.

2) With respect to overnutrition and cancer risk, the NRC committee concluded that the evidence from human and animal model studies linking total caloric intake to the risk of certain types of cancer is largely indirect and does not permit a clear-cut interpretation of the direct involvement of caloric intake. It has long been suspected that being overweight is associated to some degree with the risk of death from certain types of human cancer and this recently was confirmed by the American Cancer Society study. However, there is little data relating total caloric intake to cancer risk. In several of these studies, it was not possible to evaluate the relative importance of overweight or obesity in comparison to total caloric intake. Human

studies that have compared both caloric and fat intake suggest that fat intake is more important than the caloric intake. In the middle 1940s, Tannenbaum and Silverstone were engaged in a series of elegant studies demonstrating the effect of diet in terms of total calories on breast tumor growth in animals. Studies in animal models indicate that restricting the intake of food (calorie restriction) without modifying the proportion of the individual nutrients reduces the cancer incidence. Because the intake of all nutrients was simultaneously reduced in these animal model studies, the observed reduction in tumor incidence might have been due to the reduction of other nutrients, such as fat. Thus the committee's conclusion that neither the human nor the animal model studies permit a clear interpretation of the effect of total caloric intake on the risk of cancer is, in my opinion, reasonably accurate.

3) Evidence for the importance of total dietary fat as a risk factor for cancer of the large bowel, breast and pancreas comes from both human and animal model studies, whereas the evidence for the prostate cancer emerges from human studies. Available evidence also suggests that high dietary fiber (mainly from whole grain cereals) acts as a protective factor in populations consuming a high amount of total fat. Case-control studies indicated an elevated risk for those with an increased intake of total fat and saturated fat. In animal models, high-fat diets enhance the development of colon, mammary and pancreatic tumors. Polyunsaturated fats enhance mammary tumors and stimulate tumor growth more effectively than do saturated fats. However, diets containing small amounts of polyunsaturated fat and a high level of saturated fat increase mammary tumors as effectively as do diets containing a high level of polyunsaturated fat. Animal model studies provide some evidence that at low dietary fat levels, diets high in polyunsaturated fats are more effective colon tumor promoters than diets rich in saturated fats, irrespective of the source of the saturated fat. In general, these results support a role for total dietary fat in the incidence of certain types of cancer. Both migrant studies in humans and animal model studies clearly suggest that the stage of carcinogenesis at which the effect of dietary fat is exerted definitely appears to be during the promotional phase of carcinogenesis, rather than during the initiation phase. However, no clear-cut experiments have been conducted in animal models to indicate that the dietary fat has no effect during initiation. The fact that ubiquitous environmental carcinogens are present at very low concentrations suggests that promoting factors may have a preponderant influence on the eventual outcome of the cancer process in humans. Due to the wide variety of initiating agents and the possible difficulties in removing them from the environment, the promotional phase of carcinogenesis may be a more promising area for the development of preventive measures. In practical terms, this suggests that reducing fat intake should decrease cancer of the colon, breast, prostate and pancreas, regardless of whether it is achieved by eliminating fats of animal or vegetable origin.

4) With respect to the relationship between the dietary cholesterol and cancer, data from several studies equivocally suggest that decreased serum cholesterol may be associated with increased mortality of colon cancer in man. Whether low serum cholesterol levels in these patients precede or follow colon cancer is not completely determined. One also wonders if decreased serum cholesterol may be linked with increased cholesterol excretion. This might be a more accurate indication of total cholesterol burden imposed by diet and actual body production. Thus, excess cholesterol excretion could be the link between low serum cholesterol levels and an increased risk of colon cancer. Additional studies are warranted to settle this issue.

5) In spite of evidence from human and animal model studies on the inverse relationship between dietary fiber and colon cancer, the NRC Committee concludes that there is no conclusive evidence for the protective effect of certain dietary fibers against colon cancer in humans. Dietary fibers comprise a heterogenous group of carbohydrates, including cellulose, hemicellulose and pectin, and a noncarbohydrate substance, lignin. The composition of fibers differs from one source to another. Vegetable fibers, which are highly fermentable, have little indigestible residue; whole grain cereal brans are less fermentable and have more indigestible residue. Thus, cereal grains, vegetables and fruit fibers have different percentages of cellulose, hemicellulose, pectin and lignin. The major problem with several human studies on dietary fiber and colon cancer is that there is no published information on the total dietary fiber content of various food items. Most of the information was obtained using crude fiber values, which are useless. In other studies, most fiber analyses have been based on total fiber consumption calculated by grouping foods such as fruits, vegetables and cereals according to their fiber content. Thus, the results often appear confusing because general misuse of fiber terminology and lack of analysis of various fiber sources.

In conclusion, the NRC's report, *Diet, Nutrition, and Cancer*, is a significant document. It may also be concluded that dietary deficiencies or excesses of certain nutrients could play an important role in cancer causation. The human data are particularly plausible because extensive animal model studies have demonstrated the effect of nutrition and nutrients on experimental carcinogenesis. Thus, we frequently have been surprised at the relative lack of attention given to this field when, in fact, the relationship had considerable biological plausibility, rationale, and internal consistency from the very beginning.

(Dr. Reddy's remarks prepared for *JAOCS* include materials previously provided for the Council for Agricultural Science and Technology Publication No. 13, *Diet, Nutrition, and Cancer: A Critique*, published in 1982.)

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Introduction

In the past few years, concern has been raised that a cholesterol-lowering diet may increase the risk of cancer. Although this hypothesis was examined and dismissed by most nutritionists and cancer specialists, nevertheless, the alarm was

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sounded and led to the following review of the facts.

Results of 8 International Dietary Experiments

The first well controlled trial was conducted at the Veterans Administration facility in Los Angeles (Pearce and Dayton, 1971). Among 420 men placed on a diet with increased polyunsaturated fats for 8 years, the reduction of heart attacks, both fatal and nonfatal, was significant in comparison to 420 control men. But in contrast to this expected outcome, the review of a total of 10 years' observation of this population revealed a higher cancer death rate in the diet group (31 men) than in the control group (with only 17 men). What had happened? As in every long-term experiment, when people are randomized into a study group, they retain their freedom to drop out, or to choose only to participate a fraction of time in the diet. The investigators kept meticulous adherence-to-diet records on each man. A summary of these records, with the adherence expressed in percent of the entire time they could have attended the meals served in the Special Study Dining Hall, is shown below.

Adherence to diet (%)	No. of cancer deaths	
	Diet group	Control group
0-10	10	2
11-20	2	1
(All cancer deaths)	(31)	(17)

Thus, 12 cancer cases occurred among men in the Diet Group who adhered less than 20% of the time to the diet. We now recall that of all men assigned to the diet group, 31 later died from cancer. However, the 12 men who died from cancer and who only rarely or never ate the meals in the Study Dining Hall can hardly be called dieters. They were not exposed to any significant degree to the diet enriched with polyunsaturated fats, and must have eaten the "control food." If we now deduct these 12 men from the 31 cancer cases, only 19 cancer patients remain in the experimental group. When these 19 are compared to 17 cancer patients in the control group, there is no longer any significance in the difference in cancer frequency among the two groups. In fact, we can now add to the original 17 cancer patients on "control food," the 12 men who were supposed to, but hardly ever consumed the diet!

The combined experience of the dietary studies among men in Oslo, London, Helsinki and Faribault, all of them with increased consumption of polyunsaturated fats, was reported immediately following publication of the first trial results, by Ederer et al., also in 1971. The frequency of cancer during the years of diet and the so-called post-diet phase was lower in the dietary experimental groups with 7.7% in comparison to the control groups with 10.9%.

The 13-year results from the "Anti-Coronary-Club" in New York deserves special attention (Singman et al., 1973). The authors wrote: "Our observations lend no confirmation to the alleged association between cancer mortality and high polyunsaturated fatty acid diet." In brief, 1,764 men 40-49 years of age were followed. They included an active experimental group of 378 men keeping a relatively high polyunsaturated fat intake throughout the study; an inactive group of 853 men who were once active dieters but lately had limited their participation annually for examinations; a control group of 533 men who were never advised on diet. Six cases of cancer among 378 active dieters amount to a cancer frequency of 1.59%; 15 men with cancer in 853 inactive persons translates into a cancer incidence of 1.75%, and 10 cancer patients in the controls equal an incidence of 1.88%. The slightly lower risk of the active dieters to develop cancer, therefore, agrees well with the previously quoted results from three European and one American dietary intervention studies.

A 5-year study, again from Oslo (Hjermann et al., 1981), not only confirmed the 47% lower frequency of both heart attacks and sudden death in the diet group, but also took all-cause deaths and cancer mortality in consideration. The total death rate per 1,000 men was 26 in the diet intervention and 38 in the control group; cancer death rates were 8 and 13 per 1,000, respectively. Again, unequivocally, cancer was *not* increased in men placed on a polyunsaturated to saturated fat diet in the ratio of 1 to 1 — if anything, cancers occurred less frequently among dieters!

A dietary trial in Minnesota (Frantz et al., 1975) in 7 mental hospitals, lasting 4 years, proved unsuccessful in preventing coronary heart disease to a significant degree in women and in men over age 50. However, highly significant differences were obtained in men below age 50 on the diet, with 2.5 per 1,000 men experiencing heart attacks, strokes or sudden death, whereas 9 per 1,000 men among the controls either suffered from or succumbed to heart and blood vessel diseases. The all-cause death rate which in-

cludes cancer was significantly higher in control men (10.8/1,000) compared to the active diet group (1.7/1,000).

Finally – although only an observation, not an experiment – the 20-year follow-up of 2,000 men in the Chicago Electric Company (Shekelle et al., 1981) is important in this context. It received wide attention with its remarkable finding of a 30% reduction of coronary heart deaths in men who had reported low consumption of dietary cholesterol and a relatively high intake of polyunsaturated fats at the beginning of the 20-year follow-up. At the Conference of the National Institute of Health in Bethesda, May 1981, the cancer incidence in 5 different cholesterol groups in this population was presented and showed no difference in the frequency of cancer over the 20-year period in men with either low, medium or high cholesterol levels.

Conclusion

The lowering of blood cholesterol levels through changes in the fat quality which has proven beneficial in the prevention of heart attacks has had no undesirable side effects. Even more important, the reports on the frequency of cancer in the international dietary studies, between 1970 and 1982, have demonstrated a slight (and insignificant) reduction in cancer deaths, an unexpected bonus for adherence to a diet with less animal fat and more vegetable fats and oils.

Additional Comments

The "estimate that diet contributes from 30 to 60% of all cancers" is unfounded and should never have been entered into final publication of the report. This type of unwarranted speculation only confuses the public and serves no useful purpose.

The evidence for a causal role of dietary fat in the development of breast cancer is lacking and is clearly based on opinions and statistical associations rather than on facts. The recent publication from the Kaiser-Permanente Medical Care Program (Hiatt et al., 1982) concluded that serum cholesterol and breast cancer in 1,035 patients were not associated. "The postulated causal relation between dietary fat and breast cancer does not act via an effect on circulating lipid levels." (JNCI 68:885-889, 1982.) How prostate cancer was drawn into this discussion remains particularly annoying when considering the vast racial differences in the prostatic cancer mortality between Orientals or Jews on the one hand, and Blacks in both countries, South Africa and in the United States, and Scandinavians on the other hand – the former with very low rates, the latter with higher than expected rates.

The discussion of Vitamin A and its potential role is well balanced and the position taken on dietary fibers represents the present debate of unresolved issues.

The 4th paragraph on page 4 (of the National Research Council's press release on the report), however, is not quite correct. It should read: In the U.S., overall age-adjusted cancer rates have remained fairly stable over the last 30 to 40 years . . . However, it is estimated that these rates could have declined by one fourth to one third if smoking-related cancers were eliminated (notably lung, oral cavity, bladder-kidney, pancreas, esophagus cancer).

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In preparing *Diet, Nutrition, and Cancer*, the Committee established by the National Research Council in conjunction with The National Cancer Institute has responded to the request that it (1) "review the state of knowledge and information pertinent to diet/nutrition and the incidence of cancer and (2) develop a series of recommendations related to dietary components (nutrients and toxic contaminants) and nutritional factors which can be communicated to the public."

This review of the state of knowledge is comprehensive and covers epidemiological and experimental evidence on the relationship between cancer and various aspects of nutrition, including total caloric intake, lipids, protein, carbohydrate, fiber, vitamins, minerals, alcohol, food additives and contaminants. The committee also has summarized the information on the basis of its relevance to different types of cancer.

In addition, it has included discussions on the nature and causes of cancer, on methodology for studying relationships between nutrition and cancer, on naturally occurring carcinogens and mutagens that may be present in food, and on various other related topics. Each chapter is followed by a list of references to pertinent literature.

The committee has provided a valuable service in collecting a large amount of the information scattered through the literature. This report should serve to focus additional attention on possible relationships between nutrition and cancer, and will be a useful source of information for investigators in this field of research.

Interpretation of the data and the conclusions drawn from it will no doubt stimulate controversy. The committee has proposed dietary recommendations as requested, and has wisely labeled them as interim guidelines. Even so, the provision of dietary guidelines for the general public is fraught with danger. Such recommendations are subject to change as more information becomes available and each change leads to loss of credibility and a greater tendency by the public to ignore such pronouncements.

Like the committee, I feel that diet has an important influence on cancer and I think that evidence linking specific constituents such as dietary fat to carcinogenesis deserves serious consideration. The evidence to date is perhaps insufficient to offer assurance that any particular dietary modification will lessen the chance of developing cancer, but dissemination of this information makes it possible for people to consider dietary options. It should also stimulate further research which could in turn lead to better methods of combating cancer.

I think that members of the committee are to be commended for the effort they have made to summarize the literature on diet, nutrition and cancer, and I look forward with anticipation to their second report, in which they will consider potentially profitable areas for further research. They expect to complete this report in approximately a year.

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I am in broad agreement with the observations of the NRC Committee, and I am in broad agreement with its conclusions but for one important matter.

It is my personal opinion that people eat their current diet because they enjoy it and prefer it to alternatives. Consequently, any change in the diet of the general population is, of necessity, going to be toward one which is less palatable or less convenient. In particular, throughout the world people are tending to eat a diet as rich in fat and meat as they can reasonably afford and I am sure that this is on the basis of palatability rather than any other reason. Eating is one of the major joys of life and therefore before suggesting any change in diet which reduces palatability (and therefore the pleasure associated with eating), we must be very sure of our facts. I am not sure that we are justified in trying to change the diet of the whole population. We can easily justify a low fat diet to persons who have raised serum lipid levels, etc. I do not believe that we can justify recommending the proposed dietary changes to fit, healthy, slim persons on the grounds that it will prevent them from developing cardiovascular disease, breast or bowel cancer, etc., because we simply do not have the data.

I believe that the balance of the evidence indicates a causal relationship between a high fat/low fiber/low vitamin/high salt diet and a range of diseases and I would be happy to see the diet recommended to high risk groups. However, most of us will *not* develop any of the diseases in question.

We do not know why, for example, 5% of us will develop bowel cancer and the other 95% will not, but we do know of some risk factors. I would prefer not to impose a relatively unpalatable diet on 95% who do not need it. It would be better to identify a high risk group (e.g., persons with colorectal adenomas and relatives who have already developed colorectal cancer) and urge *them* to change their diet, leaving the rest of the population to enjoy their food.

I am sure that this is a minority view among persons working in the field of fat and disease, but it might be put forward nevertheless.