Nutrition: The Need to Define “Optimal” Intake as a Basis for Public Policy Decisions

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Introduction

There has been much controversy as to whether dietary factors affect the risk of various chronic diseases, notably various kinds of cancer.1 We propose that the diverse, sometimes conflicting results from retrospective and prospective studies may be associated with unavoidable measurement errors in nutritional assessment, as well as the homogeneous dietary habits among populations. There is clearly an “optimal” nutritional pattern with a reduced chronic disease risk, especially as regards cardiovascular diseases and those cancers associated with nutritional habits.2-5

As we attempt to define an “optimal” diet, particularly in terms of fat and fiber intake, what contribution can we expect from epidemiology, from experimental studies, and from relevant mechanistic investigations? At the outset, we need to recognize that in the Western world, in general, current food consumption is excessive in terms of total calories consumed as well as fat calories, particularly given the sedentary life-styles of the majority of the people. While myocardial infarction has been linked to obesity, only cancer of the kidney and endometrium have been routinely linked to excessive weight.6,7

Our first solid meal, our first few birthday parties, and our earliest memories of Mother’s cooking, fast food, and gourmet restaurants establish a pattern of eating typical of the Western world. For instance, we tend to eat two or three high-fat/low-fiber meals per day, with the bulk of the calories consumed in the evening. We learned to do so at an early age, just as our parents did. This eating pattern is quite different from that of our prehistoric ancestors; their need to eat as food became available during the day’s activities resulted in a “grazing eating pattern.” Meanwhile, the physiologic processes in the human body most likely have not changed or adapted biologically for thousands, perhaps hundreds of thousands, of years. Our metabolic processes are no different from those of our prehistoric ancestors, determined largely by their existing environment.8

Over the last 10 000 years since the agricultural revolution, there have been massive dietary and physical activity changes. These changes accelerated over the last 150 years with the industrial revolution, primarily due to improvements in food technology, such as extensive food processing and availability of energy-dense foods, and a decrease in physical activity.

In our view, there is good scientific evidence that our current dietary and sedentary habits lead to diseases of metabolic overload: atherosclerosis with its major sequela myocardial infarction, adult-onset diabetes, a variety of specific cancers, and other chronic illnesses. However, most people fail to recognize that current dietary practices are, in reality, “abnormal” and that a low-fat, high-fiber, high-vegetable-and-fruit diet more closely matches our metabolic capacities. In-
stead, we tend to believe that our present diet represents an advancement from our more primitive eating habits. Our concern is with defining the "optimal" nutritional intake commensurate with useful, healthy longevity and compatible with evolutionary processes.

**Issues of Nutritional Assessment**

Prior to a review of human and experimental data on the question of an "optimal" dietary regimen with a lower risk of chronic disease, we will consider the problem of measurement of nutritional intake. Disagreements as to the role of nutrition in health and disease may stem from our inability to determine nutritional intake accurately in homogenous populations, even over short periods. When we consider lifelong dietary habits, the task becomes even more difficult. Even when data are reproducible, reproducibility does not mean validity. In general, insufficient attention is paid to this issue.

Problems associated with measuring dietary intake in individuals and in populations have been extensively reviewed by Bingham. What a person eats from day to day is one of the most complex exposure variables. Not only do people eat different foods containing different nutrients in different combinations, but cooking also is an extremely complicated chemical process. How variations in this process affect the chemical composition of food, from the destruction of nutrients (e.g., vitamin C) to the genesis of carcinogens (e.g., heterocyclic amines), needs to be considered. Thus, it is necessary not only to get good insight into the foods a person eats but also to ascertain the methods of preparation.

Common methods used to assess dietary intake in free-living humans include daily recording, with or without weighing, to estimate amounts consumed, daily recalls, dietary history, and questionnaires on food frequency with or without quantitation. Each procedure has its own strengths and drawbacks, but some problems are common to all methods.

1. **Errors in judgment.** Estimating and reporting portion sizes of the food an individual consumes can be highly subjective. Even dieticians need special training to accurately use household measures such as a cup, a teaspoon, and so forth to describe food quantities. It is unrealistic, therefore, to expect different persons to refer to the same quantity of food, for example, when using the terms "small" or "medium" portion. Food models and pictures are used to minimize but not eliminate such judgmental problems. Errors in estimates of weights of food are often in the 50% range.

2. **Problems of recall.** Obviously, this is important only when assessment of diet is retrospective and relies on ability to recall. Prospective daily recording of food is not affected by recall errors. Errors in recall are predominantly underestimates. They can be random (when subjects randomly forget what they have eaten) or systematic (when subjects with certain characteristics consistently underreport). Underestimates, even when random, may diminish true differences between groups, and the errors are not corrected by enlarging the sample size. In case-control studies, cases may recall differently from controls. There are also other incentives for people with different attributes to differentially recall past dietary habits. Thus, obese people and heavy eaters tend to underestimate their dietary intake more than thin people and light eaters. The problem is worse when the period of interest goes farther back in time, as memory will be more distant and real changes in diet may have taken place. Since present diet affects what one recalls of past diets, the more changes that have taken place, the less accurate would be dietary recalls.

3. **Individual variability.** Day-to-day variations in dietary intake within individuals can be large. Sometimes this variation is higher than average interindividual variations. A dietary assessment, whether prospective or retrospective, that does not take this variability into consideration is apt to be quite inaccurate. Methods such as the 24-hour recall capture only a fleeting moment in a person's life. To get a more complete picture of the present diet, 3-day to 14-day records are usually necessary. Added to this daily variability are weekly and seasonal variations. Although some investigators have shown that weekly variations are not as marked as daily variations, seasonal variations can be large for certain nutrients (e.g., vitamin C and fiber, or fruits in general).

Individuals also make dietary changes through the years (e.g., people switching to a vegetarian regime). Disease may be related to diets eaten years ago, which can be substantially different from what a person consumes now. Present diets, therefore, may poorly measure the variables of interest.

4. **Food table errors.** Even if one could get an accurate assessment of the actual foods consumed by individuals, translating this information into nutrients is by no means simple. Depending on the food tables used, nutrient estimates can differ widely because different food tables may use different chemical methods to analyze food, may sample individual food items differently, or may apply distinct conversion factors.

All these factors combine to make dietary assessment one of the most difficult tasks facing nutritional epidemiologists. Measurement errors can sometimes be so large that group differences are simply not detectable.

In addition to the measurement problems, there are individual differences in responsiveness to identical diets. For example, large variations in serum cholesterol exist among subjects consuming the same diet. Thus, even if diet could be measured without error, its correlation with disease risk will be diminished by variations in individual responsiveness.

In conclusion, the diversity of dietary data from both retrospective and prospective studies is, in part, due to the measurement errors as described. Also important is the fact that in a high-risk area many populations free of disease at a given point in time are, nonetheless, at relatively high risk. Thus, useful insights into nutrition and health require studies of populations at different risk for disease and with distinct dietary traditions. Not only are such populations useful tools to explore epidemiologically the nutritional habits that account for the different disease incidence, but they are valuable for studying the underlying mechanisms through biochemical and metabolic investigations.

**Global and Metabolic Epidemiology**

**Fat Levels**

The major epidemiologic understanding of the influence of nutrition on cancer incidence comes from intercultural comparisons. Accordingly, we have continued over many years to investigate comparative cancer epidemiology between the United States and Japan because of large differences in rates of specific cancers and associated food consumption patterns. Good-quality vital statistics are also equally accessible in these two countries.

For example, in 1985, the age-adjusted mortality rates in the United States and Japan per 100,000 people were 27 and 3.5 for breast cancer and 14 and 2.5 for prostate cancer, respectively. The proportion of calories consumed as fat in Japan
was 10% in 1950, about 20% in 1980, and 25% today.\textsuperscript{19,20} In the United States, it has been about 40% for several decades. While these ecological correlations by themselves do not prove causation, such likelihood is underscored by extensive animal studies referred to subsequently.

While intercultural comparisons provide the best opportunity to examine large-scale differences in disease rates, there are examples within a country that can make the same point. For instance, a study among strict vegetarians (vegans) in New Jersey indicated that although 30% of their calories came from fat, they had a low intake of saturated fats (5% of calories), and serum cholesterol in adults averaged only about 130 mg/dl.\textsuperscript{21} Their fiber intake averaged about 45 g/day. There are no reliable data on heart disease and cancer mortality in vegans, but the metabolic data suggest that their risk is likely to be low. Ornish,\textsuperscript{22} following the guidelines of Pritikin, recently showed some regression of atherosclerosis after placing patients on a diet with 10% of total calories from fat for 1 year.

**Fiber Levels**

Studies of international colon cancer rates revealed that Japan and Finland, particularly rural Finland, had low rates of colon cancer compared with most of the Western world. In Japan, the explanation may hinge on a low-fat intake, with a good part of the fat derived from fish high in omega-3 fatty acids, which are protective against cancers in several animal models.\textsuperscript{23,24} In Finland, a high saturated fat intake is common, particularly from dairy products, and the rate of coronary artery disease is among the highest in the world. However, the Finns traditionally have a high cereal (wheat bran) and total fiber intake (32 g/day). The stool bulk in subjects in rural Finland was measured to be over 200 g/day, compared with 80 g/day for subjects in New York.\textsuperscript{25-27} Because of similar total fat intakes in the United States and Finland, the daily total bile acids in stools were similar. However, because the larger stool bulk in Finland has a diluting effect, the concentration of fecal bile acids was about a third of that in New York, and actually very similar to that found in Japan. The low concentration (4 mg/g of feces) in Japan stems from a low fat intake with a fiber intake similar to that in the United States. Thus, bile-acid concentration in stools fairly parallels risk for colon cancer development.\textsuperscript{26,27}

For breast cancer, serum levels of estrogen, particularly unbound estrogen, may be useful biomarkers for risk. Rose\textsuperscript{28} has shown that when premenopausal women were switched to a 20% fat diet from a conventional higher fat diet, a significant reduction in estradiol was obtained. Key et al.\textsuperscript{29} reported that among rural Chinese women consuming only 15% of their calories as fat, estradiol levels were much lower than for British women. Estradiol levels were 36% higher in premenopausal women (35 through 44 years of age), 90% higher in perimenopausal women (45 through 54 years of age), and, 171% higher in postmenopausal women (55 through 64 years of age) in the British compared with the Chinese women. This suggests that dietary fat is particularly important as an estrogen regulator in the postmenopausal period. Such data have an important bearing on intervention trials for breast cancer. Rose\textsuperscript{30} also found that wheat bran fiber supplements, given to increase the total daily fiber intake to approximately 30 g/day, significantly lowered the estrogen levels in premenopausal women after 2 months.

**Experimental Evidence Bearing on an “Optimal” Diet**

Strong collateral and supporting evidence for “optimal” levels of nutrients comes from experimental approaches in animal models that, in turn, provide the basis for mechanistic considerations. When animal experiments generate hypotheses, these can be tested in human observations. When the National Academy of Sciences Committee on Diet, Nutrition, and Cancer recommended, in 1982, a diet of 30% fat calories, there were few data to support this recommendation.\textsuperscript{31} In fact, studies in a colon cancer model, using between 30% and 60% fat calories, suggested that cancer risk is similar at 30% and 40% fat calories.\textsuperscript{32}

Therefore, we conducted systematic experiments on this question and found that the incidence of mammary cancer and tumor multiplicity at 10% and 20% levels of dietary fat was similar, and much lower than that obtained with 30% and 40% fat calories.\textsuperscript{33} There was a sharp discontinuity between 20% and 30% fat calories. Observations in a large-scale study of breast cancer in women that ranged from 32% to 46% of calories revealed no difference attributable to fat.\textsuperscript{34} These results agree with the data in rats, which show similar breast cancer incidence at 30% and 40% of fat calories. Studies by Carroll, Ip, and Cohen further showed important differences in mammary gland cancer incidence for various types of fats and oils.\textsuperscript{35-37} In animal colon cancer models, Reddy et al. observed that a level of 25% fat calories yielded a cancer incidence and multiplicity similar to that at 10% fat calories and much lower than that seen at 40%.\textsuperscript{38} In this model, the colonic bile-acid concentration reflected the dietary fat intake, and bile acids have been shown to act as promoters of colon cancer. Addition of wheat bran, or of cellulose, to the diet lowered the incidence and multiplicity of colon cancer.\textsuperscript{39,40} Experimental studies have shown that different fibers have varying effects on the action of different types of carcinogens. For example, several studies have demonstrated that dietary wheat bran and cellulose, insoluble fiber fraction, protected against colon carcinogenesis in laboratory animal models, whereas dietary soybean, rice bran, or oat bran had no such inhibitory effect.\textsuperscript{39} Dietary fiber has also been shown by Cohen et al. to exert a protective effect in an animal mammary cancer model.\textsuperscript{41} This effect was seen primarily in animals fed a high-fat diet, but not in those fed a low-fat diet, suggesting that increased fiber intake may block the cancer promoting effects of high-fat diets. These model studies on the role of fat and certain fibers fairly reflect and support the results obtained in human populations, as discussed above.

**Conclusions**

In view of the present evidence, what should be our recommendation as to an “ideal” or “optimal” intake of dietary fat and fiber? The “ideal” diet may be so low in fat (<20% of calories) and high in fiber (>35 g) that from a practical and economic standpoint, we could not expect a Western population to accept such levels at this time. “Optimal,” on the other hand, would be levels of fat and fiber still associated with a low risk of disease, and yet involve a food consumption that can be implemented in a modern Western society.

The epidemiologic evidence for such low-risk levels comes largely from global comparisons supported by metabolic epidemiologic studies. The contributions of case–control studies, for reasons described, are relatively limited. The epidemiologic evidence is strengthened by extensive studies in laboratory animals with comprehensive mechanistic backing. On the basis of available data, then, we suggest that an “optimal” diet for low rates of coronary artery disease would provide...
25% of calories from fat. This level of total fat would also reduce risk of certain cancers, notably breast, ovary, endometrial, pancreas, prostate, and colon. For prevention of cancers of the colon and breast, we recommend a fiber intake of 25 g/day. Insoluble fiber, notably wheat bran fiber, has been shown experimentally to be of particular benefit for lowering the risk of breast and colon cancers.\(^{39-41}\) Cholesterol levels, however, have been reported to be more responsive to soluble fiber such as is found in oat bran.\(^{42}\)

In summary, we recommend an "optimal" diet with an intake of fat at 25% of total calories with the percentage of different types of fats requiring further delineation. At low intakes of fat, the type of fat appears to be less significant. It appears that a low-fat, high-fiber diet might be mutually supportive. To simplify, we suggest a 25/25 diet—25% fat and 25 g fiber. Some people from academia and industry may challenge that recommendation. What makes science so rewarding is that constructive debates will further knowledge so that, in the end, truth will generally prevail. The opportunities to prevent disease through dietary manipulation are so great that all parties that can influence our diet should collaborate toward testing and then implementing an "optimal" diet for healthful longevity.

For future studies on neoplastic diseases, we should focus more on reliable, yet easily determined, biomarkers, much as has been done in the case of coronary artery disease and cholesterol, which may establish standards and criteria that will provide a sound basis for risk prediction. Such markers would also permit determination of adherence to health-promoting regimens.

The public can be educated as to risky dietary traditions and become more accustomed to beneficial nutritional habits. The recent, widely advertised change in fat content of "fast food" restaurants is a step in the right direction, as is the broad availability of fat-free desserts. New policies of the Food and Drug Administration can help in consumer education. The emphasis should be on grams of fat per portion. For example, an 1800-calorie diet in which 25% of the calories are fat translates into 50 grams of fat per day. The consumer must have the opportunity to become knowledgeable about fat content by meaningful food labeling.

Nutritionists and food industries have the challenge to help in lowering the incidence of diseases attributable to metabolic overload. Through changes in production and marketing of foods, food preparation, and food processes that are more compatible with our metabolic capacities, they can contribute enormously to our health. Consumers should be educated accordingly, through proper nutrition education, beginning very early in life.

On the basis of worldwide observations in humans and considering the results of laboratory experiments in animals, it seems clear that the proposed 25/25 diet certainly can do no harm. Also, the material discussed provides evidence that adoption of such a lower total fat, high-fiber, vegetables and fruit scheme may further lower the incidence of cardiovascular disease and decrease the risk of major human cancers, such as colon, prostate, breast, ovary, endometrium, and pancreas. Further likely benefits may be better weight control and avoidance of constipation and its accompanying adverse intestinal effects.

An equally important consideration is that if no action is taken, this itself is an action. Such inaction would allow the higher incidence and mortality from major preventable chronic diseases in the Western world to continue. This is clearly unacceptable in light of current, sound knowledge on the mechanisms of nutritional carcinogenesis. □

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Report Questions Tax Equity in Health Insurance

Federal tax laws that discriminate against people who do not have employer-provided health insurance are a major reason millions of Americans are uninsured, according to a report issued by the nonpartisan group, Health Care Solutions for America.

"The real issue here is fairness," said Aaron Trippler, Executive Director of Health Care Solutions. "Current tax policy allows corporations to deduct the entire cost of health insurance for their employees, while individuals purchasing their own insurance are not allowed any deduction at all."

The study found that approximately 9.8 million more people would purchase health insurance if federal tax policy treated everyone the same by allowing all taxpayers to deduct the amount they paid for health insurance.

"Today, millions of part-time workers, employees of small businesses, self-employed and unemployed individuals have to purchase health insurance with after-tax dollars," said Trippler. "This can double the effective cost of obtaining insurance. For example, a middle-income family may need to earn an additional $8200 to have enough net income after paying federal, state, and social security taxes to purchase the same policy which costs a corporation $4000."

Currently, two thirds of workers who must use after-tax dollars to purchase health insurance are uninsured. Their families are 24 times as likely to be uninsured as those eligible for employer-provided coverage, according to the report.

"Existing federal tax policy provides the most help in purchasing health insurance to those who need it least: full-time and higher paid employees of large corporations," added Trippler. "But for middle-class and low-income families who work for employers who do not offer health insurance, federal tax policy provides no help at all."

Trippler noted that under existing law, the self-employed are allowed to deduct just 25% of the cost of their health insurance, with the result that more than a quarter of them are uninsured as well. "And even this small deduction will expire on July 1 of this year."

The study finds the burden falls most heavily on low-income families and minorities, who are much less likely to work for employers who provide health insurance. "Unfair tax laws make it all but impossible for the working poor and lower middle class to buy health insurance," said Health Care Solutions President John Weiss.

"Providing tax equity would insure more people, at less cost, than any other health insurance reform proposals being discussed," said Weiss. "The net cost of providing individuals the same tax deduction that corporations get would be about $8 billion a year, or $30 a year for the average American. That's a very modest cost to reduce the number of uninsured by nearly 10 million and enable many more to continue their coverage, despite ever-increasing premium costs."

Health Care Solutions for America is a nonprofit, nonpartisan organization based in Washington, DC. It was established to perform research and develop alternative solutions for problems with the US health care delivery system. Copies of the report, "Federal Tax Policy and The Uninsured," are available for $9.95 postpaid from Health Care Solutions for America, 1155 Connecticut Ave, NW, Washington, DC 20036. (Tel: 202/429-6529.)