Introduction

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Public policy relating to alterations in established patterns in the nation's food intake is made by government officials guided by their perception of the relevant body of scientific information. Unfortunately, this process of communication between scientists and government officials often takes the form of an advocacy proceeding in which interested scientists, either in oral or written testimony, put forth those elements of the total body of data that support the change in public policy that they favor.

In order to produce a series of documents that would present the basic facts about several issues that relate to the role of nutrition in the nation's health, the American Society for Clinical Nutrition (ASCN) through the action of its Council and its past president, Dr. Jules Hirsch, convened a task force in 1978 that undertook this mission so as to ensure that such documents would avoid the advocacy role and would constitute a consensus that would be of help to public officials in formulating national nutrition policy.

It was evident from the start that there are important limitations to this process. First, it is clear that the scientists with sufficient involvement in these issues to produce such documents have developed their own biases about the data and have strong feelings about the type and degree of intervention in this area that would be of most value to the population. It was hoped that, by having on the committee individuals with a full range of convictions, a consensus document could be produced that would not be dominated by a single point of view. In addition, it was also hoped that by dealing with the evidence component by component, rather than as overall conclusions or recommendations, it would be possible to maintain greater objectivity.

It is also clear that such documents, even if they could establish without doubt a link between a pattern of food intake and disease, would constitute only one aspect of any decision to recommend changes in patterns of food consumption. In some instances, biological uncertainties would exist that would affect the decision. In addition, policy makers would have to take into account potential economic effects—both to the individual consumers and to the national economy as a whole—of major changes in patterns of food consumption and production. Even in the case of the clearest association between nutritional intake and disease, only a subset of the entire population may be at risk. Thus, the wisdom or fairness of policies that apply also to those for whom the interdictum is of no value must be decided.

The panel convened by the ASCN consisted of nine scientists with a wide experience in clinical medicine, human metabolism and nutrition, epidemiology, and animal experimentation. They were asked to examine all available scientific evidence on six dietary issues that are thought to bear heavily on the prevalence in the Western world of arteriosclerotic disease, diabetes, hypertension, liver disease, dental caries, and obesity. They weighed that evidence for its consistency and strength; and they clarified the degree to which that evidence is cohesive and logical in explanation of cause-and-effect relationships.

The panel was chaired by Drs. E. H. Ahrens, Jr., and William E. Connor; each of the seven other panelists was chosen for his experience in respect to a given dietary issue and for his willingness to take part in the project. Over the past year, with the help of 24 additional consultants selected by the panel as a whole, the seven topic leaders have produced a series of Consensus Statements—one on each of the six chosen dietary issues—that are given below. These statements do not represent an official statement of the ASCN or its members, but the views of the panelists.

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The six issues

The dietary issues chosen for review are those that are commonly perceived to be causative factors in certain of the major diseases of the Western world.

1) Dietary cholesterol in relation to arteriosclerotic disease (Dr. Henry McGill).
2) Saturated and unsaturated dietary fat in relation to arteriosclerotic disease (Dr. Charles Glueck).
3) Carbohydrate and sucrose in relation to arteriosclerotic disease, diabetes and dental caries (Dr. Edwin Bierman).
4) Alcohol consumption in relation to liver disease and arteriosclerotic disease (Dr. Norton Spritz).
5) Excess calories in relation to obesity, hypertension, diabetes and arteriosclerotic disease (Drs. Theodore Van Itallie and Jules Hirsch).
6) Dietary sodium in relation to hypertension (Dr. Louis Tobian).

The panel undertook to examine each of these dietary factors separately, and the individual Consensus Statements address each of the factors as if it were an independent risk factor for disease. However, it was evident from the outset that some of these factors are so intimately related to each other in natural foodstuffs that it could be misleading not to consider them as conjoint factors. The association of cholesterol with saturated fats is such a case.

The panel recognized that, even if the reduction in intake of a given factor could be proven to alter the incidence of the disease with which it is related, there are relatively vital social, political, and economic issues related to such alterations. Despite the great importance of such considerations, since they do not clearly come within the purview of clinical nutrition, it was decided to confine the reviews to the relationships of diet to the physical aspects of health and disease.

The panel process

The process consisted, first, of a literature review by the topic leader and his advisors that usually resulted in a lengthy and heavily annotated report. Then the panel as a whole discussed and debated each review for completeness and balance. The panel then summarized its views in three ways: the nature of the available evidence was evaluated; the quality and strength of that evidence was examined; and the foreseeable risks and benefits of changing the intake of the given dietary factor were described. The Consensus Statements that follow this introduction have purposely been made brief and concise and, to that end, all references have been omitted (the essential underlying documentation is published here under the authorship of each topic leader). The procedure and the interrelationships of the six dietary issues resulted in some unavoidable repetitiousness from one Statement to another.

The panel was expressly charged not to draw up a set of recommendations. It was agreed at the outset that the panelists would limit their reports to statements of the facts, recognizing that any decision-making on national dietary issues is a highly complicated process that involves many considerations outside medical and nutritional science. For instance, any large change in the nation’s dietary habits could have a world-wide economic impact, not only on agriculture and the food industry, but also on employment, on food prices, and ultimately on the consumer’s pocketbook. The heterogeneity of the human population poses problems of still another kind: is it reasonable to assume that one dietary pattern can best satisfy the health needs of a population that differs, one person from the next, in genetic makeup, in metabolic responsiveness, in ethnic traditions and preferences, in food habits and nutritional needs at different age levels? The retraining of a large population in new dietary practices would involve educational and promotional efforts that will demand the cooperation of the medical profession, nutritionists and educators. These and many other “extra-scientific” considerations convinced the panel that it could perform a useful service by concentrating its attention exclusively on an evaluation of the available scientific evidence.

The Consensus Statements: order of presentation and key definitions

Each position paper considers the relationship of a given dietary factor and disease under three headings: 1) kinds of evidence; 2) quality and strength of the evidence; 3) risks and benefits of changing the intake of the factor.
Kinds of evidence

Epidemiological evidence

This category is limited to the evidence obtained from mortality and morbidity statistics, and by cross-sectional surveys between population groups and within population groups. The methodology is retrospective, passive, and nonmanipulative; while it often reveals important new concepts, the evidence is based on associations and is rarely if ever expected to provide proof of cause-and-effect relationships. In the best of circumstances, seminal ideas are provided that can be put to direct test in animal models and by human experimentation.

Animal experimentation

This category of evidence includes the description of disease in animals, whether natural or induced; morphological and pathological consequences of challenges by the dietary factors in question; metabolic studies; genetic manipulations; and in vitro experimentation with animal tissues.

Human experimentation

The evidence gained prospectively through human experimentation includes descriptive studies of clinical disease; intervention studies in which changes in incidence of new events of disease are measured; autopsy data; metabolic studies; and in vitro manipulations of human tissues.

Quality and strength of the evidence

The various kinds of evidence assembled above were also evaluated in terms of 11 criteria, as defined below.

Consistency among various population groups

Is there an association between the factor in question and occurrence of the disease? Are there important exceptions?

Consistency among individuals within a given population

Is the disease more common in those individuals exposed to the factor in question? How consistent is that association?

Autopsy data in man

Is there evidence from autopsies that supports the putative relationship?

Strength of the association

How strong is the relationship between the factor under consideration and occurrence of disease? Can a correlation coefficient (or other appropriate index of strength) be cited?

Independence of the association

How much of the association relates to the factor under consideration, and how much reflects an associated variable rather than the factor itself?

Temporal association

Does exposure to the factor consistently precede the occurrence of disease?

Effect of new exposure

Do individuals or groups newly exposed to the factor develop a higher incidence of the disease?

Preventive effect of removal

Do apparently healthy individuals who discontinue exposure to the factor show a lower disease incidence?

Improvement effect of removal

Is the course of disease in affected individuals altered favorably when the factor is removed?

Animal models

Can the human disease be simulated in animals by exposing them to the factor under consideration? How closely do the pathological and biochemical alterations mimic the human disease? How consistent is this effect within and among animal species?

Biological explanation

Can the relationship between the factor and disease be explained in terms of accepted pathogenetic mechanisms?

Risks and benefits of reducing the intake of the factor

The accumulated evidence, evaluated for its quality and strength according to the guidelines described above, was also assessed in terms of the possible harm or benefit of removal or reduction in intake of the given dietary factor.
Consensus of the panel

The six statements that follow this Introduction represent the agreements reached by nine scientists. Although it was possible to reach a consensus on each issue, it is not surprising that the separate Statements were viewed with different degrees of enthusiasm by the individual panelists. We concluded that readers of these summaries would find it useful to know how large that variance actually was. To measure it, we devised a rating system that, though far from faultless, gives a sense of the cohesiveness of agreement on the six dietary issues by the nine panel members.

For this evaluation the evidence on each issue was weighed in still another manner than by kinds and quality, namely:

1) Associations among various population groups
2) Associations among individuals within a given population

In each of these two categories, the net weight of the evidence was considered in terms of consistency, autopsy data, strength and independence of the association, temporal association, and effect of new exposure (using the definitions described above).

3) Intervention studies

In this category, all available evidence was assessed in terms of preventive and improvement effects of removal, i.e., an evaluation of the results obtained in primary and in secondary prevention studies.

4) Animal models
5) Biological explanation

To each of these five categories we assigned a maximum score of 20, i.e., from 0 (weakest) to 20 (strongest)—so that a total score of 100 on a given dietary issue could be awarded by the panelist who perceived the evidence to be rock-solid in each of the five categories listed above.

For purposes of rating the Consensus Statements it became necessary to set up 10 ballots, rather than six. For carbohydrates we had to arrange three separate ballots—one for carbohydrate and atherosclerotic disease, another for carbohydrate and diabetes, and a third for carbohydrate and dental caries. For alcohol we balloted twice: one rating on alcohol and liver disease, and another on alcohol and atherosclerotic disease. And finally, because the issues of cholesterol and saturated fat are justifiably considered as con-joint putative factors in atherogenesis, we rated these issues jointly as well as separately. That gave us a total of 10 ballots; five categories of evidence on each ballot; nine panelists voting; hence, 540 bits of information.

The unsigned ballots were coded, then computer-analyzed. The numerical results are seen in the table.

These data in the table and their spread are better presented graphically. In the figure the stars designate the medians of the ratings. The length of each box indicates the middle 50% of the ratings, i.e., the 25th to the 75th percentile limits, and the crosses show the median distance from the median.

In ballots six and seven, which represent the ratings on carbohydrates and caries, and on alcohol and liver disease, the panelists were in close agreement that a causal association was strongly supported by the evidence. In ballots nine and ten, the mean scores on salt and on excess calories as health hazards were somewhat lower, and on these ballots the panel voted less cohesively than it had on ballots six and seven. However, there was even less agreement among us on the three ballots at the left: cholesterol, saturated fat, and the conjoint issue of cholesterol and fat, but the mean scores were rather high. In marked contrast, ballots four, five, and eight, which rated carbohydrate and atherosclerotic disease, carbohydrate and diabetes, and alcohol in relation to atherosclerotic disease, showed that the panelists were of nearly one
INTRODUCTION

mind in judging that the evidence was extremely weak.

However, note also the five outlying votes marked as solid circles: for instance, the score of 54 on the carbohydrate-diabetes relationship, even though the mean of nine ballots on this issue was only 13, and the two unusually high scores on alcohol and atherosclerosis, and the one discordantly low rating on excess calories. These outliers make the point that in any committee report any widely disparate viewpoints are hidden in a display of consensus. The existence of honest disagreements—or perhaps misunderstandings—is part of any decision-making process.

We hasten to emphasize the obvious fact that the rating system described above is completely arbitrary in its weightings of the five categories: the proportions adding to 100 could have been quite differently ordered, if, for instance, it were believed that human intervention studies deserve twice as much weight in a decisional process as all the epidemiological observations. The procedure was devised and undertaken merely as a way to display the cohesiveness of opinion of the panelists themselves, who had met on numerous occasions, read the literature, and debated the evidence from various angles. It seemed important, by some such means, to indicate to the reader where the panelists agreed most strongly, and where the widest variations of opinion persisted despite this intense exposure to the evidence. We emphasize that the scores have no intrinsic merit and should not be taken out of proper context.

Conclusion

The six Consensus Statements and ratings represent the views of the nine scientists chosen by the Council of the ASCN to prepare this report. They are not an expression of an official position of the ASCN or its members. Detailed reports with appropriate references will be published separately under the authorship of the individual panelist responsible for each topic review.

It is the panel’s hope that this approach to the pressing problem of the nation’s nutrition goals will prove useful and instructive. Perhaps the methodology used (with modifications and improvements) will serve as a model for other interactions between scientists, the government, and the public.