Myth and Magic in Nutrition

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It seems anomalous that the topic “Myth and Magic in Nutrition” should be included in a program on “Planning for Better Nutrition in the 21st Century.” This after all is the age of science. During the past 200 years both society and the environment have been transformed through the acquisition and application of scientific knowledge. Yet, unscientific attitudes are prevalent in many countries. In surveys conducted in the USA and the United Kingdom, less than 10% of the samples of the populations responding met the standard in a test for scientific literacy (1). In a recent Gallup Poll (2), 25% of adults surveyed expressed a belief in astrology and 55% in the existence of the Devil. For magical and supernatural beliefs to be accepted side by side with the critical thinking required for the advancement of science represents inability to distinguish among science, pseudoscience, and superstition.

Resistance to thinking critically about deep-rooted desires, beliefs, and prejudices is not new. Hippocrates and Galen (3), Paracelsus (4), and Claude Bernard (5) all decried the willingness of their medical colleagues to accept speculation as a substitute for accurate observation. It was the stimulus for the original formulation of the scientific method by Bacon (6). Popper (6) has emphasized that objectivity is achieved only through adherence to the method of critical rationalism—the method of science. Current acceptance of unscientific beliefs about foods and diets, nutrition and health, which provides the basis for an immense business in nutritional nostrums for disease prevention and weight reduction (7), must be attributed to failure of much of our society to accept critical thinking and a skeptical attitude as the way of achieving objectivity and accurate knowledge about the natural world.

I propose to tell you where my efforts to understand myth and magic in nutrition have led me (8,9) and of some concerns I have about progress toward better nutrition in the 21st century.

SOME THOUGHTS ON RATIONAL AND MYTHICAL THINKING

The ability of our early ancestors to reason is evident from the tools and other artifacts found in living sites that date 1.5 to 2.0 million years ago (10). They were
able to solve practical problems relating to their immediate needs for survival and undoubtedly acquired much useful information through trial and error coupled with careful observation. The native peoples of the Arctic, for example, discovered that infusions of evergreen needles would cure scurvy (11). Certain Indians in Colombia used salt from a particular deposit as a cure for goiter. It was subsequently found to contain iodine (12). These and other objective and reliable observations, however, were intertwined indiscriminately with a large body of superstitious and magical beliefs.

Although early humans used reason and logic—practical or pragmatic thinking—to solve problems they encountered in meeting their immediate needs for food, they could not deal with most natural calamities by rational thinking. Storms, floods, droughts, illnesses, epidemics, and famine were recurring threats not only to their food supply but to their health and survival. These threats created fear and apprehension—they still do today. This inability to control nature gave rise to beliefs in magical or supernatural beings who were concerned with human affairs and could help or harm human endeavors (13,14).

Animism, a system of beliefs in which animals were endowed with human characteristics, is practiced almost universally among unacculturated societies. It includes magical and supernatural beliefs that are perceived to improve the chances of success in acquiring food (15). Foods were used as offerings to appease vengeful gods and were often endowed with magical properties. The belief that the characteristics of what is eaten can be acquired by the eater has been widespread; for example, nursing Eskimo mothers should eat duck’s wings so the child will become a good paddler (8). Beliefs of this type are based on mythical thinking.

Throughout most of the period of human development, pragmatic thinking and mythical thinking appear to have been accepted as equally useful ways of acquiring knowledge.

**DIETETIC MEDICINE FROM HIPPOCRATES TO PARACELSUS**

During early historical times, major diseases were attributed to supernatural intervention. However, a new attitude toward knowledge began to emerge about 2,500 years ago when Greek philosophers became skeptical of magical and supernatural influences. They concluded that direct observation and logical reasoning were the only reliable ways to learn about the natural world (16,17). This transition was clearly evident in the approach of the Hippocratic physicians whose writings expressed impatience with the idea that disease was a punishment sent by the gods. “Each disease,” Hippocrates stated, “has its own nature and none arise without a natural cause” (3). The Hippocratic physicians recognized the importance of diet in both their diagnosis and treatment. “It is disgraceful,” according to Hippocrates, “not to recognize a patient whose debility is connected with inanition, and to pinch him in his diet” (3).

Despite the many astute observations on health by Greek physicians from the time
of Hippocrates until that of Galen about 600 years later, the Greeks did not perceive the essence of the scientific method. Acceptance of the mystical doctrines that diseases were caused by imbalances of the four humors—blood, phlegm, yellow bile (choler), and black bile (melancholy)—and that health depended on the appropriate balance of the properties (qualities)—warmth, cold, moist, and dry—impeded their understanding of physiological processes and inhibited a transition from myth and pragmatism to science.

Magical and mystical beliefs remained dominant during the Roman and early Christian eras. Knowledge, then, was to be gained through faith and revelation from authorities, not through reason and observation. Although scholarship flourished in the Arabic (Islamic) culture during the Dark Ages in Europe (16), knowledge of nutrition and dietetic medicine failed to advance beyond the concepts of Galen for 1,000 years.

The intellectual ferment of the Renaissance in the 16th century laid in the groundwork for resurgence of the scientific attitude (16,17). In Padua in 1543, Vesalius was making direct observations on the anatomy of the human body and breaking the pattern of dependence on the opinions of authorities, such as Galen (16). The struggle to escape from the bonds of authority is exemplified by the career of Paracelsus (4). He used his knowledge of alchemy to prepare medicines. He experimented to establish the correct dose and stated "... no thing is without poison. The dosage makes it either a poison or a remedy" (4). He enjoined his colleagues to give up the search for the "elixir of life" and to use their chemical knowledge to discover a specific remedy for each disease. Paracelsus proposed that illnesses resulted from impaired body function or improper food, not from an imbalance of humors, yet he accepted many of the magical beliefs of the day, such as the "doctrine of signatures." The shapes of the leaves in the liverwort and kidneywort were "signatures" indicating the parts of the body for which they were cures.

The belief that foods contained only a single source of nutriment—aliment—persisted through the 18th century (12). This precluded any possibility of developing more than the most rudimentary understanding of nutrition. What passed for nutrition was essentially food lore and dietetic medicine based on a mixture of practical observations and magical beliefs.

SCIENCE AND PSEUDOSCIENCE IN NUTRITION: 1800 TO THE PRESENT

The scientific attitude which developed during the 17th and 18th centuries had an impact on nutrition and food knowledge only during the 19th century. Since then, understanding of nutrition and metabolism has progressed rapidly. Application of the scientific method has enabled us to obtain reliable knowledge about the natural world (6,18,19) and has provided us with a means of distinguishing between myths and realities about food, nutrition, and health. Technological advances achieved by this method of thinking are readily and widely accepted, but the method itself has not become a basic part of our culture nor has it displaced mythical thinking. Failure
to adopt critical rationalism widely as a way of thinking encourages acceptance of pseudoscience—untested speculations and hypotheses without predictive value—essentially magical beliefs clothed in the language of science (18–20). This is evident in current beliefs about foods and diet in relation to health.

Despite medical and health advances, fears of pain, disease, aging, and death—fears our early ancestors dealt with by myth and magic—remain with us. In a commentary on medical care Lewis Thomas (21) stated, “The general belief these days seems to be that the body is fundamentally flawed . . . always on the verge of mortal disease, always in need of continual monitoring and support. . . . There is a public preoccupation with disease that is assuming the dimension of a national obsession.” According to Hilda Bruch (22) anxieties about many aspects of life can increase susceptibility to irrational nutrition and food beliefs. Payne (23) has suggested that the idea of “an optimum state” of nutritional health which can be achieved under some unique set of conditions seems to be of mystical origin and has probably done much to inhibit the growth of understanding of the science of nutrition.

Success in promoting laetrile (the toxic cyanogenic glycoside from apricot pits) and a variety of other worthless dietary treatments for cancer, megadoses of niacin and other essential nutrients for treatment of schizophrenia and many bizarre diets as magical ways for reducing body weight (24), has depended on exploiting fears that may be assuaged by mythical thinking. Diseases for which there are not highly effective treatments are grist for the mills of modern-day sorcerers and magicians (charlatans and quacks) who merchandise nutritional nostrums as cures for them. While scientific knowledge of nutrition has advanced, belief in magical properties of foods has been extended to belief in magical properties of nutrients (7,24). In the USA, the National Council against Health Fraud, in its bimonthly newsletter, has documented claims that range from primrose oil, selenium, orotate, and chelation therapy to remove heavy metals associated with heart disease, to vitamin C, lysine, and cyanogenic glycosides, for cancer.

The charlatan and the quack exploit widely held beliefs in unsubstantiated attributes of foods. Their success reflects the failure of our educational system to instill the critical attitude needed to counter the ideas prevalent today that “one belief is as good as the next,” and that there is a kind of “subjective truth” that is immune to critical evaluation (20). These ideas represent, as Kurtz (20) has stated, reaction against rigorous standards in the interpretation of evidence. If, in dealing with diet and health issues, all views are given equal consideration, there is no basis for distinguishing between scientific knowledge and pseudoscientific conjecture. Sir Peter Medawar (25) put it this way: It is “as if the discovery . . . of the insufficiency of reason had given a paradoxical validity to nonsense.”

**ARE DIRECTIONS IN NUTRITION TODAY DISCOURAGING MYTHICAL THINKING?**

The discoveries early in the 20th century that many minor constituents of foods were essential for growth, survival, and reproduction provided for the first time the
basis for a rational understanding of relationships between diet and health. In the 1940s, after human requirements for many essential nutrients had been determined, food guides were developed that enabled the public to select, from familiar foods, a nutritionally adequate diet (26). During the 20th century nutritional deficiencies have been virtually eliminated in the industrialized nations; infant and childhood mortality have declined; mature body size has increased, and life expectancy has lengthened (27). These immense improvements in health depended upon advances in nutrition, sanitation, and the control of infectious diseases that were achieved through the scientific method.

These improvements in health have been accompanied by changes in the major causes of death, from infectious diseases to chronic and degenerative diseases (27). Today, chronic and degenerative diseases, which account for two-thirds of all deaths, are our major medical problems. The causes of these diseases are not clearly established, and knowledge of how to treat and prevent them is limited and inadequate. Although they are diseases of aging and their increasing incidence is associated with increased life expectancy, the high probability of dying from them creates apprehension and fear. In addition, governments have recognized that a high proportion of the rising cost of health care is attributable to expenditures for their treatment. Such concerns have led to strong public and political pressures to find effective and less expensive ways of treating and preventing them. One response to these pressures has been to increase funding for basic research needed to establish the underlying causes of chronic and degenerative diseases and disorders; another has been a series of proposals for adoption of population-wide dietary guidelines to prevent them.

It cannot be overemphasized that these guidelines have been proposed, not because there has been an epidemic of such diseases or because health in the industrialized nations has deteriorated, but because crude death rates from chronic and degenerative diseases have risen as health has improved. Mortality from these diseases increases exponentially with increasing age beyond 30 years. Over 80% of newborn infants presently can be expected to survive to age 65 years and beyond, whereas, during the early part of this century, less than 40% survived that long. Most people now live to an age at which they develop chronic and degenerative diseases; 50% of deaths from heart disease, for example, occur after age 75 years, 35% after age 80. The age-adjusted death rate from heart disease, moreover, has been declining for several decades, and those from cancers, except for cancers associated with heavy cigarette smoking and overexposure to ultraviolet light, have remained constant or declined. High total mortality from chronic and degenerative diseases is characteristic of populations with long life expectancies. The assertion that industrialized nations are suffering from an epidemic of chronic and degenerative diseases is a modern-day myth (27,28).

Proposals for population-wide diet modification as a means of preventing chronic and degenerative diseases are based largely on results of epidemiologic studies—observations of associations between disease incidence, especially of heart disease, and various personal and environmental characteristics. Characteristics associated with an increased incidence of a disease are termed "risk factors" for the disease.
In the 1950s, Keys (29) reported a strong association between mortality from heart disease among men aged 55–59 in seven countries and the amounts of fat, saturated fatty acids, animal fat, and cholesterol in their diets. Yerushalmy and Hilleboe (30) pointed out, however, that when 22 countries were included in such an analysis the association was weak. Nonetheless, in clinical and animal studies associations were observed between saturated fatty acid intake and serum cholesterol concentration and, in humans, between high serum cholesterol concentration and mortality from heart disease. These associations provided the basis for identifying high serum cholesterol concentration and a diet high in saturated fatty acids as risk factors for heart disease. Subsequently, heart disease specifically, and chronic and degenerative diseases generally, were purported to be epidemic in Western industrialized nations, and this was attributed to changes that had occurred in the diets of these nations during the first half of this century (28). During this period, however, the saturated fatty acid content of the US food supply at least had not increased appreciably and consumption of meat had declined (28,31).

Something would seem to be a little out of perspective when a purported epidemic of diseases that are responsible for two-thirds of all deaths in most industrialized nations is associated with declining death rates at all ages, an increased proportion of infants surviving beyond the age of 65 years, increased life expectancy at birth, and an increased proportion of elderly people in the population. Despite this, proposals for dietary guidelines for prevention of chronic and degenerative diseases have proliferated as if mortality from such diseases were the only meaningful measure of the state of nutrition and health of the population. Reaven (32) has described this as "looking at the world through LDL-cholesterol-colored glasses."

Recommending diet modification as a means of preventing diseases that are not of nutritional origin represents an extension of the use of diet therapy to ameliorate effects of impaired metabolic function in patients who are ill, to its use as a prophylactic measure in people who are healthy. In the USA and some other countries, major food companies have realized this and have used the proposals for dietary guidelines as justification for touting their individual products as agents for preventing major diseases. This encourages mythical thinking about panaceas for disease prevention on the part of the public in much the same way as do the pseudo-scientific claims of purveyors of untested nutritional products as magical cures for diseases. It is reminiscent of the approach to diet in the prescientific era when foods were assumed to consist of nutriment, medicines, and poisons. At a time when the state of "scientific literacy" of the population is being decried, it does not seem inappropriate to ask if the current public health approach to diet and disease is fostering scientific illiteracy by encouraging acceptance of myth and magic in nutrition.

Atherogenesis is accepted as the major disorder responsible for ischemic (coronary) heart disease but, as it is not possible to assess the severity of atherosclerosis routinely in epidemiological studies, mortality from heart disease or clinical evaluation of ischemic heart disease is taken as an indicator of the incidence of atherosclerosis. Stehbens (33) has pointed out that this is inappropriate because: the se-
verity of atherosclerosis overlaps greatly in men with and without ischemic heart disease; evidence from autopsy studies indicates that most men over 50 years of age have pronounced atherosclerosis; and many individuals with extensive coronary artery disease do not exhibit clinical evidence of ischemia. The incidence of atherosclerosis is thus grossly underestimated from measures of the incidence of ischemic heart disease. Therefore, in risk factor analysis, when risk factors for ischemic heart disease are taken as risk factors for atherosclerosis, the degree of overlap in atherosclerosis between individuals with and without ischemic heart disease cannot be taken into account.

Risk factors may be conditions that influence the course of a disease, result from the disease, or are only coincidentally associated with occurrence of the disease. Over 200 risk factors have been reported for ischemic heart disease. Stehbens (33) emphasizes that only if a risk factor occurs in every individual with the disease is it appropriate to infer a causal role for it; and that failure to distinguish between risk and cause exaggerates the importance of noncausative agents. This has led to the conclusion that chronic and degenerative diseases, and atherosclerosis in particular, have multiple causes, i.e., are multifactorial in etiology.

Epidemiologists, by using the term “risk factor,” acknowledge that associations cannot be assumed to represent causes. They have, however, postulated criteria based on epidemiologic evidence for establishing causal relationships between risk factors and diseases. These include measures of the strength, consistency, specificity, and chronological relationship of the association, the plausibility of the risk factor as a cause, and the coherence of the total body of evidence. How well does the evidence reviewed by Stehbens (33) support the view that risk factors for ischemic heart disease can be equated with causes of atherosclerosis?

Hypercholesterolemia is accepted as a major risk factor for both atherosclerosis and ischemic heart disease, but it is not present in every individual with these diseases. Mortality from ischemic heart disease among individuals with low serum cholesterol concentrations is about one-third that among individuals with “moderately high” (6.2–6.5 mM, 240–250 mg/dl) levels (34). Also, in the Framingham study, ischemic heart disease is the major cause of death among men in the lowest quintile of serum cholesterol values (35). Thus the strength of the association cannot be considered great enough to support a causative role for this major risk factor.

Inconsistencies in the degree of association between risk factors and ischemic heart disease abound in epidemiological investigations (31,34). The relative importance of risk factors is found to differ among populations. When ischemic heart disease mortality and serum cholesterol concentration, for example, are plotted as a function of age, the lines do not parallel each other.

The fact that most deaths from ischemic heart disease occur among individuals with moderately low concentrations (200–250 mg/dl) of serum cholesterol (35,36) has been the basis for lowering the standard for the concentration above which risk of ischemic heart disease is considered to increase significantly. The highest number of individuals without evidence of ischemic heart disease, however, is also included within this range of concentrations. Specificity could hardly be much lower.
Identification of risk factors for atherosclerosis is established from associations with ischemic heart disease—the end-stage of the disease. A chronological relationship has not been established between risk factors and development of the initial lesion of atherosclerosis. Also, even though risk factors may be modifying factors that can influence the course and severity of atherosclerosis and susceptibility to ischemic heart disease, the plausibility of risk factors as causes and the coherence of the evidence supporting a causal relationship are low. Risk factors for ischemic heart disease cannot, therefore, be considered as causes of atherosclerosis.

To infer cause and effect relationships from evidence of associations, no matter how strong and consistent they may be, does not conform with scientific principles. The epidemiological criteria for establishing cause do not meet the standard of the Henle and Koch postulates that require the cause be present in each individual suffering from the disease (33). They also do not conform with the strictures of Bernard (5) and Popper (6) that a single exception to a hypothesis is evidence that the hypothesis is inadequate. In addition, the concept of multifactorial causation of chronic and degenerative diseases implies that such diseases could occur from a variable mix of host and environmental characteristics without any one being essential. This is contrary to the essence of the scientific method in that it virtually precludes disproof of a causal role for any one factor (33).

To illustrate this problem, Klevay (37) prepared a list of characteristics associated with pellagra prior to the discovery of niacin. The list is as long as many for ischemic heart disease today. Strong beliefs in the validity of some of these “risk factors” as causes created great resistance to accepting the evidence that pellagra was a nutritional deficiency disease. One cannot help but wonder what effect advocacy of multifactorial causation of pellagra at that time would have had on public health policy for its control.

Quite apart from the inadequacy of the evidence of a causal role for risk factors in atherosclerosis, serious questions have been raised about how well these risk factors predict the probability of coronary occlusion and how effective institution of population-wide dietary guidelines is likely to be in preventing ischemic heart disease. Shortcomings of the evidence marshalled in support of proposals for adoption of such guidelines have been identified by Mann, Yerushalmy, Ahrens, Corday, Oliver, Goldboult, Reiser, Olson, Stehbens, Kritchevsky, McNamara, McCormick, Harper, and others, all of whom are cited in the extensive and thorough critique of reports on the subject by Smith and Pinckney (31). A popular version of much of this information by Moore (38) was published under the title The Cholesterol Myth.

The evidence cited most frequently as supporting claims that institution of dietary guidelines will be effective in preventing chronic and degenerative diseases is from the results of intervention trials for control of ischemic heart disease (35). These are studies in which dietary modifications and drug treatments known to lower serum cholesterol concentration have been tested over a period of several years for their ability to reduce the incidence of heart attacks and mortality from ischemic heart disease in large groups of people, usually in men considered to be at high risk for heart disease. Advocates of dietary guidelines claim that the treatments have reduced
risk of heart attacks or death by 20% to 40%. When the results are examined closely, however, it is evident that even with cholesterol-lowering drugs, serum cholesterol concentration was usually reduced, even in the most effective trials, by less than 10%, and that reductions in the incidence of heart attacks and deaths from ischemic heart disease were rarely significant (31). This is not surprising when it is realized that a 25% decrease frequently represents a decrease in death rate from 2/1,000 to 1.5/1,000 persons/year.

In a combined analysis (meta-analysis) of six of the best intervention trials, mortality from ischemic heart disease was not significantly reduced by the treatments, although a significant reduction was observed when the drug studies were analyzed separately. Even in these, total mortality was the same in the treatment and control groups (39). In another meta-analysis of 19 such trials, in which about 30,000 men were treated for from 5 to 10 years with either diet modification or drugs to lower serum cholesterol concentration, the treatments decreased the number of heart attacks just significantly, but mainly owing to the effects of the drugs. Again, no decrease in total mortality was detected (40). Forette et al. (41) reported that in elderly women, mortality from ischemic heart disease was much higher among those with the lowest serum cholesterol values than among those with concentrations of about 7.0 mM or 270 mg/dl. In a study (42) of 4,400 men with serum concentrations of cholesterol in the normal range (mean 5.94 mM, 230 mg/dl), the average decrease from consuming a cholesterol-lowering diet was 13.6% but, over a five-year period, the treatment did not reduce either mortality from ischemic heart disease or total mortality. St. Leger et al. (43), in an analysis of information from 18 industrialized nations, noted, as others had, a positive correlation between fat intake and mortality from ischemic heart disease, but the main significant association they observed overall was a strong negative correlation between mortality from several chronic and degenerative diseases and wine consumption.

In two of these trials, 840,000 men, 31 to 59 years of age, were screened in order to obtain 16,700 judged to be at high risk for ischemic heart disease. Thus only 2% of the population screened met the criteria. From an analysis of one of these studies of men treated with a cholesterol-lowering drug, Brett (44) concluded that the reduction in cardiovascular morbidity and mortality in the high risk group, in absolute terms, was 1% to 2%. In his assessment he did not take into account that total mortality was the same for both treated and untreated subjects owing to higher death rates in the treatment group from noncardiovascular causes, in part related to violence. This raises a question as to whether cholesterol-lowering treatments may have adverse effects, possibly on behavior, in a segment of the population (39).

In connection with the benefits predicted from dietary control, a few other questions should be considered (27). Why can the downward trends in ischemic heart disease mortality in Canada, Australia, and the United States during the past two decades not be clearly related to changes in risk factors? Why are there upward trends in ischemic heart disease mortality in Denmark and in Sweden where a strong program of risk-factor control was instituted almost 20 years ago? Why in selected populations that differ by as much as threefold in ischemic heart disease mortality
is there no difference in life expectancy? Why are life expectancies of the populations of several countries in Europe with close to the highest amounts of fat and sugar in their diets among the top ten in the world?

The limited effectiveness of diet modification may have to do with the fact that age, male sex, and genetic background are three of the major “risk factors” for ischemic heart disease. Hopkins and Williams (45) have concluded that 50% to 80% of early deaths from ischemic heart disease are from only 5% to 15% of families and are from diseases that are resistant to treatment. Most females are resistant to ischemic heart disease until after the menopause; and in both sexes after 60 years of age the association between risk factors and ischemic heart disease mortality is weak (46). In addition, Naito (47) has reported that 35% of patients selected for coronary bypass surgery have no obvious risk factors. Thus only a small portion of the population would be expected to benefit significantly even from effective interventions. From an analysis of information on diet modification as a preventive measure for ischemic heart disease, Taylor et al. (48) concluded that the anticipated lengthening of life-span from life-long adherence to a serum cholesterol-lowering diet would be a few days to a few months.

Why is it that ardent advocates of dietary guidelines for prevention of chronic and degenerative diseases and disorders ignore these discrepancies and disregard publications at variance with their viewpoint (27,31,33)? Some comments on the Consensus Conference sponsored by the US National Heart, Lung, and Blood Institute, at which a recommendation for instituting dietary guidelines for lowering serum cholesterol levels of all Americans over the age of two was approved unanimously, might be enlightening. Ahrens (49) and Oliver (50), who participated in the conference, identified inadequacies in the evidence and shortcomings of the procedures used in reaching “consensus.” Oliver emphasized that the final report of the panel provided “no place for the expression of genuine doubts, caveats, or frank disagreements.” Le Fanu (51) expressed distress that Steinberg, chairman of the conference, in his rebuttal to Oliver and Ahrens in support of the conclusions of the panel, failed to deal with trials that had not produced supportive results but, instead, drew on two sets of “unpublished observations.” Experiences of this type led Krichevsky (52) to select “unobserved publications” as the title for a recent presentation in which he reviewed several seldom-cited papers discussing the validity of assumptions about the dietary guideline approach to control of cardiovascular diseases.

Selective use of scientific information, an adversarial attitude in defense of rather than a skeptical attitude toward extrapolations, ready access to—and acceptance by—media that thrive on sensationalism rather than on probing the meaning and assessing the accuracy and reliability of statements by guideline proponents, all ensure that the public will not benefit from the process of critical rationalism which scientists rely on to evaluate and correct questionable assumptions and unjustifiable extrapolations about diet and health relationships. Skrabaneck (53) and McCormick (54) conclude that the long list of risk factors for ischemic heart disease is a tribute to our ignorance, and that this and the multifactorial concept of causality that evolved from inability to deal with risk factors objectively gives rise to false promises and
unrealistic expectations. It is for these very reasons that criticism is leveled at promoters of nutritional nostrums.

Despite the knowledge that has been gained about factors associated with the occurrence of chronic and degenerative diseases, and the remarkable advances that have been achieved in understanding the genetic basis of defects in lipid metabolism associated with arterial diseases, it is difficult to conclude otherwise than that the title of Moore’s article (38), “The Cholesterol Myth,” is an apt description of the dietary guideline approach to control of chronic and degenerative diseases.

LOOKING TOWARD THE 21ST CENTURY

What can we envision as directions in the science of nutrition, public health nutrition, and public understanding of nutrition in the 21st century? What is the outlook for displacement of myth and magic from nutrition?

The tools of molecular biology are being applied in studies of physiological processes that underlie nutrition. As they are applied more intensively, we can expect steady advances in understanding of genetic control of regulatory processes, the basis for differences among individuals in susceptibility to hypertension, osteoporosis, atherosclerosis, diabetes, and obesity, the ways in which inadequate intakes of nutrients influence immunocompetence, the capacity and limitations of metabolic mechanisms that protect the body from dietary and environmental agents, the genetic-environmental interactions that limit the tolerance of some segments of the population for certain food components, and the changes during aging that occur in the responsiveness of homeostatic mechanisms. It will become possible to distinguish among effects of genetic limitations, disease, and senescence; to devise genetic probes that will permit early identification of individuals at high risk for chronic and degenerative diseases; and to improve greatly the therapeutic measures available to the physician for their treatment. The end result will be a realistic assessment of what can and what cannot be accomplished through nutrition. This will reduce the basis for exaggerated claims for foods as medicines and should limit the growth of nutritional myth and magic.

In the realm of public health nutrition, selective intervention for individuals who are at high risk of developing chronic and degenerative diseases will become increasingly effective as basic knowledge of the underlying causes of these diseases expands. Emphasis on population-wide dietary guidelines for disease prevention will then decline. The latest edition of Dietary Guidelines for Americans represents a trend in this direction. It is primarily a set of recommendations for maintenance of health with only modest emphasis on disease prevention. This is not a reason to be sanguine about the likelihood that conflict over mass dietary intervention versus screening and selective intervention will fade rapidly; health organizations continue to vigorously promote “cholesterol education programs” for entire populations. Passmore (55), noting the moral righteousness of advocates of disease prevention through diet restriction, labeled them “the new Puritans”; Skrabanek (53) observed
that "as religious faith declines, the dogmas of medicine grow" and "shepherds of the soul have been replaced by the priests of prevention." Evangelical and moralistic health movements that have taken us by storm intermittently for at least a century have generally grown rapidly, then declined slowly as the public has realized how exaggerated their claims for benefits are (56). The dietary guidelines movement will probably decline more slowly than most because it is also fueled by vested interests—political interests that see guidelines as a simple way of reducing costs of medical care and services; industry interests that see them as a vehicle for introducing highly profitable products; professional interests that see them as a means of acquiring a disproportionate amount of funding from, and power in, the health care system. Balance, however, will be restored during the 21st century, and appropriate dietary guidelines for assuring nutritional health will be retained without exaggerated claims for disease control.

As we approach the end of the 20th century, mythical and magical views of nutrition are more prevalent than they were at the beginning of the century (57). Paradoxically, forces that foster such views have multiplied as the science of nutrition has progressed and their influence shows little sign of waning. I am, therefore, skeptical that progress in the science of nutrition and improved understanding of health problems in the 21st century will have much impact on public understanding of nutrition. Promotion of nutritional nostrums through pseudoscientific claims for health benefits, a constant source of misinformation, has become an immense business. The food industry, which has capitalized on dietary guidelines for disease prevention to promote products, advertises purported health benefits of individual foods in misleading ways. Health agencies and organizations that promote dietary guidelines for the public as disease prevention measures encourage their acceptance by creating fears and providing simplistic solutions. In addition, the media, in reporting on science, present unrelated fragments of research in sensational ways without providing the basic information needed to place the facts in a meaningful context. It is little wonder that much of the public views nutrition as a mythical source of the elixir of life and expects scientists to devise magical solutions for health problems on demand.

Advocating simple or simplistic solutions for complex health problems, by creating apprehension, fear of food, and fear for health, and then offering salvation through adherence to a set of commandments, conjures up images of panaceas and elixirs. It reinforces the idea that mythical thinking and rational thinking are equally valid ways of acquiring knowledge. It promotes a view of life without risk. It encourages acceptance of authority (38 professional organizations, the Cholesterol Education Panel asserts, have endorsed their report) in place of critical thinking. It is distressing when impatience with the rate of progress in solving health problems through science is undermining understanding and acceptance of the scientific attitude. To improve public understanding of relationships among diet, nutrition, health, and disease in the 21st century, and encourage rejection of dogma and mythical thinking about them (58), we must make clear in discussing this subject, as Thomas James (59) has chided
us, when our advice is based on factual evidence, when it is derived from logical
deductions, and when it is conjectural.

REFERENCES

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DISCUSSION

Dr. Leathwood: How do you think the cholesterol story arose in mythical terms?

Dr. Harper: One source was the study of Keys (1), which showed the relationship between the incidence of heart disease and saturated fat intake for seven countries. A beautiful straight-line relationship was found. However, when another 21 countries were added the situation changed completely. It was possible to plot a line showing the same fatty acid intake but widely differing heart disease incidence, and another showing the same incidence of heart disease but widely differing fat intakes (2). The American Heart Association has made a lot of money out of the promotion of the diet-heart hypothesis and continues to reiterate Keys' results. To question the hypothesis would threaten its income, and probably also that of the heart associations of Canada, Australia, and the European countries. When you pose such a threat, the reaction is strictly emotional. At a conference two years ago epidemiologists
were vigorous in their attack on "heretics" but they did not respond to the scientific issues. Moore (3) was attacked similarly for his article "The Cholesterol Myth."

Dr. Goldberg: This is a disease with a molecular and a genetic involvement. There are many genetic defects that predispose people to atherosclerosis. In certain populations an increase in serum cholesterol is not the cause of the disease but a clear indication in genetically susceptible people that they will get it. For example, people with type II hypercholesterolemia, who are unable to remove cholesterol from the blood, invariably die by the second decade of life. We are only just beginning to understand the very complex etiology of this disease. Lovastatin was promoted as a panacea for heart disease and this may have been a driving force behind the cholesterol-lowering hysteria.

Dr. Harper: There are many modifying factors and the genetic one is very important. On the other hand there has not been an impressive response in intervention trials with treatments to lower serum cholesterol. In most instances there has been no decrease in the total death rate, even though there may have been a barely significant decrease in reported coronary artery disease. I think one has to look very skeptically at this effect.

Dr. Goldberg: In the large cholestyramine study, the Lipid Research Center's Primary Prevention Program, there was a decrease in coronary mortality but an increase in mortality due to violent death. Recent evidence is beginning to show that people and monkeys on low cholesterol diets have increased aggression. We start to wonder what is really going on.

Dr. Olson: In my laboratory we have looked at the evidence that risk factors are contributory to coronary heart disease in a matrix of multiple etiology. As a result of this we assigned 35% of the risk of coronary heart disease to genetics, and I think the explosion in genetic analysis reflects the importance of factors other than lipid disorders. As far as modifiable risk factors are concerned, we assigned about 25% of the risk to smoking and 20% to hypertension. Hypercholesterolemia and related changes in lipoproteins accounts for another 20% of the risk. These modifiable risk factors have been linked to the pathogenesis of atherosclerosis and may be causes. These data are summarized in a recent paper (4), showing that one can have coronary heart disease without any known risk factors.

Dr. Harper: You can have coronary heart disease without any of these risk factors. None of them are causes—they are only modifying or associated factors, including cigarette smoking.

Dr. Olson: Causes can be described in general terms. Snow identified the cause of cholera in London in 1849 as water from the Broad Street pump. The agent was described much later as the cholera vibrio. Of course association does not mean causation but association may lead to evidence of causation. Epidemiologists have not yet defined the agent for coronary heart disease. Some years ago I suggested that the agent might be β-lipoproteins, but now I am not so sure (5).

Dr. Fischler: I am delighted by what Dr. Harper has to say. However, I should like to modify his assertion that magic and myth, ignorance and superstition, are obstacles to rational thinking. Magical thinking is part of human nature and part of the progress of civilization. I would suggest, paradoxically, that magical thinking is not an obstacle but a necessary condition to the development of scientific thinking. If it is part of human nature why should scientists be immune? They are not, so there is myth and magic in scientific thinking and the cholesterol affair is a perfect illustration. Scientists often dismiss previous theories as popular beliefs. The cholesterol story may soon be another example.

Dr. Harper: I did not intend to imply that mythical thinking is an obstacle to rational thinking. My hope is that it will not dominate rational thought. I am concerned about irrational
and anti-intellectual attitudes dominating in society. Only through rational thinking can we solve the problems of the natural world.

Dr. Walker: I am grateful for this attention to lack of scientific logic. The same concerns apply to the association now being drawn between aluminum and Alzheimer’s disease. This is just as illogical as the cholesterol story. The fact that aluminum is found in the brain in Alzheimer patients does not mean it must be causal. The same logic would make melanin carcinogenic. You expose a white person to the sun and get melanin accumulation. There is also an increase in tumors. Those tumors contain high levels of melanin, so why isn’t melanin causal!

REFERENCES