Diet, Disease, and Definitions of 'Proof'

By ACSH Staff — October 1, 1997

The public is often faced with conflicting "expert" opinions on how dietary components allegedly contribute to the cause, prevention, and cure of a host of health and even social problems. These supposedly nutrition-related problems range from Alzheimer's disease and immunologic disorders to juvenile delinquency and homicidal behavior. Every year the popular press provides appealingly simple "explanations" of purported dietary causes of such problems, blaming an excess or lack of specific food constituents or ingredients and portraying dietary supplements or specific exclusionary diets elimination diets as beneficial to sufferers. The nutrition scientists the media interview about such claims typically express skepticism. Small wonder that the public is confused.

Such disagreements often stem from differences in what people regard as proof. The primary definition in Merriam *Webster's Collegiate Dictionary* for "proof" is "the cogency [convincingness or plausibility] of evidence that compels acceptance by the mind of a truth or a fact." Accordingly, the testimonial of a trusted friend might provide "proof" for some people; for others, proof might come from nothing less than a long-term, double-blind, placebo-controlled, peer-reviewed study published in a prestigious medical or scientific journal. For scientists, evidence that is not objective is, at best, a poor source of proof.

Rabbit, Eggplant, and Bacon

Historically, human societies have tried to explain illnesses according to folk logic. In the absence of knowledge of biological processes, such logic provides the best "explanations." For example, according to folk logic, if a pregnant woman ate too much rabbit, she might bear a child with a harelip; if she overconsumed eggplant, she might have a blue baby (an infant with a bluish tint, usually from a cyanotic heart defect).

During the 16th century, scientist-philosopher-poet Sir Francis Bacon declared that, without confirmation from objective testing, such subjective beliefs were mere nonvalidated hypotheses. Modern scientific researchers must consider all information bearing on a hypothesis both information that increases the believability of the hypothesis and information that casts doubt on it. Because an absolute determination of whether a hypothesis is true is impossible, scientists use statistics to rate the probability that the hypothesis is correct. But reliance on statistical probabilities invites carping from believers who insist that certain nonvalidated hypotheses merit belief if they have not been totally disproved.

Clearing the Air

Before the 19th century, there were few avenues for exploring the causal mechanisms of disease. Anton van Leeuwenhoek, the Dutch fabric merchant who developed the first microscope and became the first human to see microorganisms, described the structure of bacteria in 1676, but

their potential for causing disease was not considered. According to the folk logic of the time, infectious diseases were caused by inhalation of malodorous emanations, or *miasmas*, from decayed matter; the worse a neighborhood smelled, the higher the risk of inhaling a pathogenic miasma.

The authors of virtually all popular dietÂ®Â¢disease books . . . advance hypotheses that are untested, ill-tested, unfounded, unlikely, or disproved.

The miasmatic theory of disease lost favor in 1854, when British physician John Snow, considering an epidemic of cholera that had caused 500 diarrheal deaths in London's Broad Street area, concluded that water delivered from the Thames River to the communal pump on Broad Street was the source of the disease agent. Snow did not have proof of the cause-and-effect relationship he proposed. But the strength of the association between Thames water and the Broad Street epidemic was sufficient for him to persuade the local authorities to remove the handle of the communal pump. The subsequent abatement of the epidemic suggested that the culprit was not a miasma but the transported water or something in it.

In 1876 German physician Robert Koch showed that the bacterium associated with anthrax in cattle could cause disease in mice. According to Koch, to prove that a particular bacterium causes a particular disease, one had to determine that the indicted bacterium was present in every case of the disease in question, was absent from individuals with any other disease, and caused the same disease if transmitted to other individuals. And scientists began attempting to prove that other diseases had a single, distinctive causal agent.

But while many diseases have a clear-cut primary cause, many others, particularly the major diseases in developed countries today, are multifactorial; that is, they result from interactions of risk factors: hereditary, biologic, sociologic, environmental, and behavioral conditions. For example, scientists recognize that coronary heart disease (CHD) may result from genetic factors, from a high saturated-fat intake, from cigarette smoking, or from a combination of many risk factors. In contrast, most pop nutrition authors, using modern folk logic, portray such diseases simplistically.

"Risky" Business

Scientists conduct studies of various kinds to determine how important a factor is in increasing the likelihood of a particular disease. In *prospective* studies, researchers monitor, over long periods, individuals whose exposure to a factor is high to see whether those individuals develop a particular disease. In *retrospective* studies researchers try to see (a) whether the exposure of individuals with a particular disease to a particular factor had been greater than the exposure of comparable individuals without that disease and (b) whether the incidence of the disease among individuals who had had the greatest exposure to the factor was higher than the incidence among otherwise comparable individuals whose exposure had been lower. In other studies researchers monitor changes in disease incidence in populations whose migration involves a change in exposure to the factor.

Scientific researchers often try to determine whether exposure of experimental animals to a dietary factor can induce in those animals something similar to the human disease with which the factor is associated. But the media typically report the findings of such animal studies with inadequate reserve. If researchers find, say, that 12 of 50 rats exposed to a dietary factor develop breast cancer within a year but that only 6 of 50 unexposed rats develop breast cancer, the press might "report" that the dietary factor in question "causes breast cancer" or "doubles risk of breast cancer." A more cautious description of the researchers' finding would be to say that the dietary factor may contribute to breast cancer in rats and that it therefore deserves scientific attention as a possible contributor to human breast cancer.

To determine whether particular findings from such animal experiments are relevant to human health, scientists conduct small-scale experiments in which they limit volunteers' exposure to the dietary factor and monitor the subjects for incidence of the disease. The researchers' findings are published in quality scientific journals only when expert reviewers have judged that scientific ground rules have been followed. These reviewers ask such questions as: Was the number of subjects sufficient? Were they monitored long enough and closely enough? Did the researchers take into account the placebo effect an improvement in condition that is caused by the mere act of undergoing treatment rather than by the specific treatment? (Many people experience relief of symptoms simply because they believe they are doing something that is palliative or curative.)

If the hypothesis is coherent, biologically plausible, and supported by epidemiologic and animal studies, large-scale human studies may follow. But, for example, while studies in which exposure to the major risk factors for CHD has been lessened have resulted in a decrease in the incidence of CHD, the decrease has often been discouragingly modest, suggesting that the lessening of exposure began too late and/or that other risk factors are important.

Of course, lessening exposure to any established risk factor is at least a step in the right direction. For instance, although some cigarette smokers will not develop lung cancer and some nonsmokers will, the risk of developing lung cancer is about 10 times higher for smokers. *Individual dietary* risk factors are usually much less potent contributors to disease. Routine exposure to a single dietary risk factor seldom results in more than a doubling or tripling of risk.

How to Spot a Dubious Diet Book

The authors of virtually all popular diet-disease books ignore such details and advance hypotheses

that are untested, ill-tested, unfounded, unlikely, or disproved. These books tend to have the same format: The author gives an informal, subjective account of at least one well-known health problem that he or she has had and/or has treated and that conventional medical experts allegedly failed to cure or alleviate. Then the author claims that a dietary supplementation regimen and/or a diet that excludes specific foods or food groups effected a complete cure. Next with scant, overly selective, or no citing of scientific evidence the author offers anecdotes about, and/or testimonials purportedly from, laypersons who similarly experienced relief. The author may bemoan the medical establishment's reputed inattention to, dismissal of, or opposition to the alleged low-cost remedy. He or she may even declare, or at least imply, that a conspiracy to maintain dependence on physicians, synthetic drugs, and processed foods exists between the American Medical Association and the pharmaceutical and food industries. Menus, recipes, dietary-supplement lists, supplier lists, and/or a recommended-reading list of trade books that are compatible with the author's hypothesis follow.

Such authors usually respond to adverse criticism (which virtually never comes from proponents of other fringe methods) with the equivalent of: "If you don't believe it, disprove it." But few scientists have the inclination, time, and resources to attempt disproving an implausible hypothesis.

Below are some questions one might want to ask the next time one leafs through a diet book.

** Does the text focus on studies published in reputable scientific journals as it should or does it focus on anecdotes, testimonials, case histories, and/or the author's personal beliefs?*
** Do the author's credentials lend credibility to his/her claims? Are the credentials even related to the claims? Does the author hold a master's degree? A doctorate? From an "accredited" institution? A correspondence school? Is the source of the institution's accreditation certified by a reputable entity specifically, the U.S. Secretary of Education or the Council for Higher Education Accreditation (CHEA)? Are the tenets that underlie the author's credentials worthy of confidence? Chiropractic and naturopathic "credentials," for example, may not be credentials at all.*
** Does the author reference case histories? Case histories published in reputable medical journals are reliable. Other case histories may be erroneous, as with respect to diagnosis. Many people un- or misdiagnosed with cancer, for example, claim to have been cured of it.*

** Might the relief claimed in testimonials have been unrelated to the alleged remedy proposed by the author?*
** Does the author stand to benefit financially from sales of products brand-name dietary supplements, meal-replacements bars, a newsletter related to his/her recommendations?*

The Bottom Line

Numerous details concerning diet and disease remain to be elucidated. That paperback writers will elucidate a significant number of these details is extremely unlikely. Ten years from now, preventive dietary and nutritional strategies will be fine-tuned. Until then, as a population we are better-off following the recommendations of scientific organizations respected by scientists than the recommendations of mavericks, entrepreneurial dissidents, and quacks.

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