The argument about the "diet-heart disease question" has left the scientific community exhausted and the public confused. It is time to take a look at the process whereby we have reached this impasse. In doing so, I do not intend to review in detail all the facts and fancies that have previously been brought to our attention. Instead, I wish to propose a resolution of the stalemate between the adherents and detractors of the diet-heart disease relationship.

The evidence supporting the conclusion that diet and heart disease are causally linked is based roughly on the following types of observations: 1) Examination of various population groups has established an association between the consumption of cholesterol, saturated fat, and total fat with the prevalence of coronary heart disease in these countries. 2) These same dietary variables are also positively associated with serum cholesterol concentrations in these populations. 3) The feeding of various fat-modified diets to a variety of animals leads to variable degrees of hyperlipidemia. 4) In humans, as well as in experimental animals, there is a strong correlation between the concentration of serum cholesterol, or that of certain lipoproteins, and the development of arterial lesions and/or arteriosclerotic diseases. 5) Arterial lesions of humans and of experimental animals with certain types of hyperlipidemias contain excessive amounts of cholesterol. 6) Patients with certain types of hyperlipidemias established early in life show premature development of coronary heart disease.

The arguments usually presented to cast doubt on the relation of dietary lipids to heart disease are along the following lines: 1) Population studies establish correlations but do not necessarily prove causal relations. 2) Associations between dietary lipids and serum cholesterol concentrations and/or heart disease within population groups are frequently found to be statistically nonsignificant. 3) Intervention trials with lipid-lowering drugs and/or diet modifications have generally produced statistically nonsignificant changes in mortality and marginally significant improvements in morbidity. 4) Animal experiments cannot be extrapolated to humans.

I do not want to argue here, as I usually would, that the objections raised by the skeptics can in part be "explained" by the insufficient statistical power of the various tests, and that the majority of clinical trials, even though statistically inconclusive, have pointed in the same direction, and that none of them have given even tentative evidence to disprove a diet-heart disease relationship. I also do not want to argue that if animal experiments cannot, with the proper caution, be extrapolated to humans, then much of medical science would have to be disregarded. Instead, I will examine the criteria by which we normally judge the validity of conclusions derived from large-scale diet trials and other relevant evidence. In this examination I would raise two crucial questions: 1) What level of evidence should one require in support of the presumed relation between diet and heart disease before the public is advised by the government or by various medical groups that changes in diet may have beneficial results? 2) Why do only the "prudent diet" protagonists have to carry the full burden of proof? Let me briefly explain what I mean by these questions.
The level of evidence required for experimental proof in various areas of scientific investigation is commonly set at $p$ levels of 5% or even 1%. Few people within the biomedical community have serious reservations about these limits, although they are arbitrary. The reason is that, for the pursuit of science, they seem to work reasonably well. It is not clear, however, that they work equally well in decision areas such as public health, economic planning, or politics, to name only a few. In my opinion, more attention should be given to the probability criteria by which we decide whether the evidence is sufficiently strong to proceed, for instance, with either advice to the public, a legislative program, or taxation of high-risk foods. Clearly, the choice of the probability level should depend greatly on the risks and payoffs of the contemplated actions.

Consequently, is there any reason to maintain very strict criteria of evidence if the maintenance of the status quo involves suffering for millions of people and premature death for many more? Would the conclusions of the various clinical trials be altered if the probability levels for significance were relaxed to 10% or even 20% or 30%? After all, if physicians would tell us that they are reasonably (~70%) confident that, on the average, consumers of a lipid-rich diet and cigarette smokers could significantly improve their health by adopting a different lifestyle, involving little risk, would we counter that such advice should not be given unless the evidence had reached the 95% level?

Now let me briefly address the second question. Why is it always the "prudent diet" protagonists that are called upon to prove their hypothesis with costly experiments? Would it not appear reasonable, in view of the large body of (perhaps incomplete) evidence, and the acceptance of a diet-heart disease relation by many of the workers in the field, to ask the other parties to come forth with their evidence that the current diet of the U.S. population is superior to the "prudent diet." This would, of course, involve much effort, in particular, if the same rigid criteria of evidence were demanded as those presently required to prove the merits of the "prudent diet." It is likely that in the next 10 years no one would be able to come up with "proof" that the current American diet is superior to the so-called "prudent diet."

In view of this preposterous situation, would it not seem reasonable to take a different approach to the problem? If neither side of the diet-heart disease question has the resources to provide incontrovertible proof that one diet or the other is superior, should we not try to settle for a goal that is within our reach? Suppose that we wipe the slate clean and stack up all the evidence in favor of the "prudent diet" and then stack up all the evidence demonstrating the beneficial effects of the present U.S. diet. As reasonable people, living in a reasonable society, we would furthermore agree to give our support to whichever diet is supported by the best evidence. If in the meantime some individual or group decides that their economic, social, or psychological interests are not well served by the resulting situation, they are free to contribute more evidence to support their point of view. This would assign the burden of proof to the party with the weakest evidence rather than demand it by habit, lassitude, or self-interest solely from the protagonists of the "prudent diet."

Donald B. Zilversmit
Division of Nutritional Sciences
Savage Hall
Cornell University
Ithaca, New York 14853

(Arteriosclerosis 2: 83–84, March/April 1982)
Diet and heart disease. Prudence, probability, and proof.
D B Zilversmit

Arterioscler Thromb Vasc Biol, 1982;2:83-84
doi: 10.1161/01.ATV.2.2.83

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://atvb.ahajournals.org/content/2/2/83.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Arteriosclerosis, Thrombosis, and Vascular Biology can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at: http://www.lww.com/reprints

Subscriptions: Information about subscribing to Arteriosclerosis, Thrombosis, and Vascular Biology is online at: http://atvb.ahajournals.org//subscriptions/