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# Epidemiology as a Guide to Clinical Decisions—II. Diet and Coronary Heart Disease

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*Should clinicians prescribe fat-controlled diets to prevent coronary heart disease (CHD), and, if so, which patients should be given this advice? In this report, we use a three-step model to explain the hypothesis that dietary fats are a cause of CHD: dietary saturated fat and cholesterol raise serum cholesterol levels (step 1), which are a cause of subclinical coronary atherosclerosis (step 2), and, in turn, clinically manifest CHD (step 3). An evaluation of the scientific evidence for each step leads us to conclude that dietary fats definitely influence the level of serum cholesterol, and that serum cholesterol is probably a cause of atherosclerosis and CHD.*

*To determine the clinical implications, we examined the potential of various foods to keep cholesterol levels lower, as well as the projected magnitude of reduction in CHD risk. The likelihood of benefit varies among patients, ranging from uncertain or trivial (for those with lower serum cholesterol levels, those who are free of other risk factors and the elderly) to substantial (for patients with higher serum cholesterol levels, those who have other risk factors and those who are young). This analysis supports an individualized approach to clinical management; each decision to prescribe a fat-controlled diet should be a judgment that depends on art—the therapeutic philosophy of each clinician and the particular needs of each patient—as well as on science.*

*The implication for public health policies is that they should promote rather than a uniform eating pattern for all Americans, a uniform environment that enhances individual choices. This should include efforts to educate the medical profession and the public, and more comprehensive and informative food-labeling practices.*

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ABBREVIATIONS USED IN TEXT

CHD = coronary heart disease
HDL = high-density lipoprotein
LDL = low-density lipoprotein

THE SELECT COMMITTEE on Nutrition and Human Needs of the US Senate issued a report in 1977 specifying quantitative dietary goals for all Americans.[1] One of the report's most important recommendations was that Americans reduce their consumption of saturated fatty acids and cholesterol. The historic action of this congressional committee (no federal agency had previously endorsed the view that malnutrition might include *overconsumption* of food) was followed by similar statements from the Surgeon General[2] and from the Departments of Health, Education, and Welfare, and of Agriculture.[3] Together, these reports seemed to establish a public health policy on the role of fats in preventing coronary heart disease (CHD), one whose wisdom was confirmed by the conclusions of many recent reviews,[4-9] and by the recommendations of authorities[10] and professional groups[11] throughout the world.

The policy, however, has not met with uniform acceptance. While some of the opposing viewpoints[12] have been criticized for failing to meet rigorous scientific criteria,[6,13] others are more substantial.[14-18] The most notorious of these, the report of the National Academy of Sciences,[18] is representative; it opposes the universal promotion of fat-controlled diets on the grounds that the evidence for cause and effect is not yet conclusive. This point of view is difficult to dismiss because the evidence linking dietary fats to CHD is largely circumstantial. However, the evidence is also very extensive, and there is reason to believe that more convincing findings are not likely to be forthcoming. It is time to settle the matter.

In this report, we address the controversy as it bears on the practice of individual clinicians who must decide whether dietary prescriptions are likely to help their patients avoid heart disease. We present an evaluation of the scientific evidence for a cause-and-effect relationship between dietary fat and heart disease. We then discuss how clinicians might combine conclusions based on this evidence with their own individual treatment philosophies and the special needs of each patient in order to decide whether—and for whom—dietary intervention is desirable. We close with a note on the implications of this individualized approach for national health policy.

## The Diet-Heart Hypothesis

The idea that dietary fat may be one of the causes of heart disease is referred to here as the diet-heart hypothesis. To examine the evidence with precision, we propose a three-step model (Figure 1): that saturated fats and cholesterol in the diet increase the level of serum cholesterol (step 1), and that higher serum cholesterol levels are a cause of coronary atherosclerosis (step 2) and coronary heart disease (step 3).* The model distinguishes between antecedent subclinical arteriosclerosis and clinically manifest CHD such as myocardial infarction.

### *Evaluating the Evidence*

Judgments on the validity of each step of the diet-heart hypothesis are based on the three categories of evidence summarized in Table 1 and discussed below. Our focus is on the epidemiologic findings, and on the most informative citations selected from a larger body of published evidence.[7]

*Nonepidemiologic studies.* The general evidence bearing on the diet-heart hypothesis includes pathophysiological, clinical and animal studies.[5,7,19] Within the first set, the demonstration that atheromas contain cholesterol supports the validity of step 2, and the autopsy evidence for coronary arteriosclerosis as a general precursor of CHD supports step 3. Clinical studies of premature CHD in persons with rare lipid disorders such as familial hypercholesterolemia support steps 2 and 3. Ex-

*A more comprehensive formulation of the diet-heart hypothesis, with total cholesterol segregated into its lipoprotein fractions and roles proposed for salt and calories, is provided as an appendix. Because these additional factors do not substantially affect our conclusions, we will use Figure 1 (with total cholesterol as a proxy for low-density lipoprotein) to illustrate our reasoning.

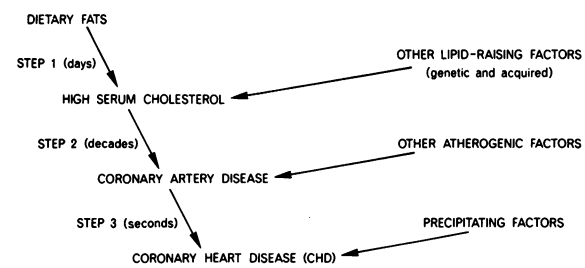


**Figure 1.**—Schematic diagram of the diet-lipid heart hypothesis. The arrows represent the proposed causal links between dietary fats, serum cholesterol level, subclinical atherosclerosis, and clinically manifest coronary heart disease (CHD). Two features that make it difficult to validate the hypothesis are the long duration of step 2 and the multifactorial causes of each step.

periments in animals prove that altering the fat composition of the diet can produce hypercholesterolemia and certain forms of coronary artery disease (steps 1 and 2).

These diverse lines of evidence provide biologic plausibility for the hypothesis, but they do not prove it; there is always some uncertainty in extrapolating the findings of laboratory studies to the clinical arena, or those of rare diseases to the general population. For this reason, the valuation "moderate" in Table 1 is often the maximum that the strength of causal inference from studies of this type can achieve.

*Observational epidemiology.* The science of epidemiology does not lend itself easily to the task of linking eating habits to chronic diseases. It is difficult to demonstrate, from statistical associations, causes that are remote in time and that are neither necessary nor sufficient.[5,7,20,21] Then there are special problems: the food we eat is complex and variable, dietary patterns are often confounded by other aspects of life-style, and it is difficult to measure eating habits accurately. Despite these barriers to drawing conclusions, population-based data are an indispensable component of preventive medicine decisions, and a reasonably convincing picture has now emerged from the many studies on the relationships between diet, lipids and heart disease. Table 1 summarizes this evidence according to the epidemiologic categories of place, time and person.

International studies[22-24] have shown that people living in countries where saturated fats and cholesterol are major components of the diet have relatively high average levels of serum cholesterol (step 1) and rates of CHD (steps 2 and 3 combined). Trends in the United States over the last several decades[25] have pointed to a weak association between national patterns of fat consumption and the average serum cholesterol level of population samples (step 1). Similarly, the average serum cholesterol level appears to have decreased in parallel with the current decline in the CHD rates of the nation (steps 2 and 3).

The well-known relationship between serum cholesterol level and subsequent incidence of CHD in cohort studies of individual persons is shown in Figure 2. The strength of this association, its dose-response characteristics and its consistency among studies[21,26,27] all support a causal basis for steps 2 and 3 combined. Evidence for step 2 alone is also found in autopsy[28] and angiographic[29] studies of coronary artery disease. Evidence to support step 1 has been elusive, and some studies have found no association between dietary fats and serum cholesterol level.[30] Although this finding has been cited as evidence against the diet-heart hypothesis,[12] it is more likely a false-negative (type 2 error) caused by the low precision and accuracy of techniques for assessing the dietary habits of populations.[31] Moreover, recent reports have offered positive evidence for step 1[32,33] and for the three steps combined.[33]

The chief barrier to confirming a causal relationship in these epidemiologic studies is the possibility that diet and CHD are both consequences

TABLE 1.—*Quality of Evidence Supporting the Validity of the Diet-Heart Hypothesis in the General Population*

| | *Support for Causal Inference in Humans** | | |
|---|---|---|---|
| *Kind of Evidence* | *Step 1: Diet Fats ↓ Serum Cholesterol* | *Step 2: Serum Cholesterol ↓ Coronary Arteriosclerosis* | *Step 3: Coronary Arteriosclerosis ↓ CHD* |
| Nonepidemiologic Evidence | | | |
| Pathophysiology studies | .... | moderate | strong |
| Clinical studies of rare lipid disorders | .... | moderate | moderate |
| Animal studies | moderate | moderate | .... |
| Observational Epidemiology | | | |
| Place: International comparisons | moderate | moderate | |
| Time: Secular trends | mild | mild | |
| Person: Studies of individuals | mild | moderate | |
| Experimental Epidemiology | | | |
| Metabolic ward studies | strong | .... | |
| Clinical trials of CHD prevention | strong | inconclusive | |

CHD = coronary heart disease.

*The valuations are the judgments of the authors regarding, for each step, the strength of the evidence that a cause-and-effect relationship exists in general human populations.

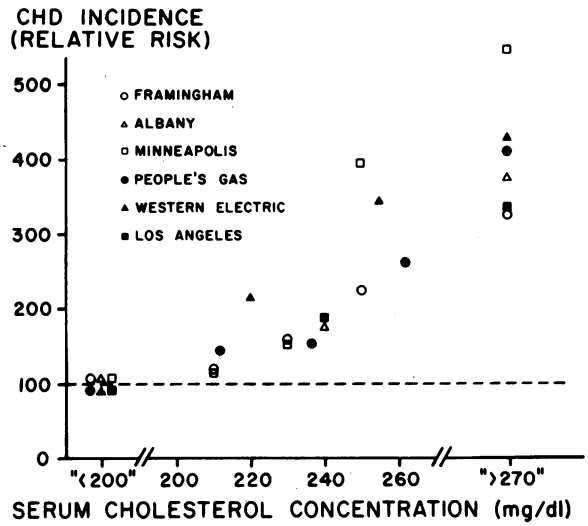


**Figure 2.**—Risk of developing coronary heart disease (CHD) in relation to preexisting serum cholesterol levels in six prospective epidemiologic studies. (Drawn from tabular data from Dayton et al.[26])

of some third (confounding) factor. However, this possibility has been made less likely in many of the studies cited above by showing that the observed associations are independent—that is, that they persist after adjusting statistically for other risk factors. But this calculation cannot indicate the possible influence of factors that were not measured. Observational epidemiology provides evidence that is circumstantial. For this reason, we have chosen the valuation "moderate" as the maximum in Table 1 that studies of this type can achieve.

*Experimental epidemiology.* How can we reduce this uncertainty? The classic resort is controlled clinical trials, a strategy capable of providing direct, rather than circumstantial, evidence. Such research, therefore, can provide a qualitatively different strength of causal inference.

Direct experimentation has been highly successful in resolving the uncertainty of step 1. Metabolic ward studies[34-36] and clinical trials of outpatients[37-39] have demonstrated conclusively that dietary composition governs serum cholesterol level. While other factors (notably heredity) influence this level as well, it is clear that dietary intake of cholesterol and saturated fats raises serum cholesterol levels, and that dietary intake of polyunsaturated fats decreases them.

Unfortunately, however, it has been more difficult to validate steps 2 and 3. Because of difficulties in measuring the extent of coronary artery disease under the circumstances of a clinical trial, we are forced to rely on the rate of CHD as the index of benefit. To be able to observe a change in this index, there must be a reasonable frequency of its occurrence; this means that the populations studied must be made up of persons who already have coronary artery disease. Even studies of this kind, which focus on a late stage of the disease and are, therefore, invariably a secondary form of prevention insofar as coronary arteriosclerosis is concerned, require that enormous numbers of participants be studied for a long time.

It should come as no surprise, then, that no definitive randomized trial of preventing CHD by dietary change has yet been carried out.[7,15,40,41] Of the studies that have been conducted, some have shown no benefit at all,[42,43] while others[39,44,45] have provided at best suggestive evidence for the efficacy of a fat-controlled diet. Some investigators have used cholesterol-lowering drugs as surrogates for dietary manipulation; such studies can take advantage of a double-blind design to guard against various biases. The major drug trials have not settled the issue, however. They have shown either no substantial benefit[46,47] or a reduction in CHD rates that was unexpectedly accompanied by an increase in overall mortality.[48]

In short, while we can conclude that reducing saturated fats and cholesterol in the diet does lower serum cholesterol levels, we have not yet established beyond doubt, probably because of technical barriers to conducting definitive research on the issue, that such diets will prevent heart disease.

## Making a Judgment on Causality

Is this body of knowledge sufficient to establish policy on whether fat-controlled diets should be recommended to the American public? In our judgment, it is. The existing evidence, while circumstantial, is extensive and coherent in its support for the hypothesis. Furthermore, the prospects for stronger evidence emerging in the future are limited.

This is not to say that there will be no new information. Promising clinical trials in progress include a study of cholestyramine for preventing coronary arteriosclerosis[41] (which uses angiography to isolate step 2 data), and trials of cholestyramine[49] and intestinal bypass[50] for preventing CHD (steps 2 and 3 combined). These studies, however, are all surrogate tests in which a drug or an operation serves as a proxy for dietary change, and all involve a special group of par-

ticipants selected for their high levels of serum cholesterol. The only current tests of diet change per se address several risk factors simultaneously,[51,52] and these will only test the efficacy of the multifactor program as a whole. It is unlikely that a trial of diet alone will ever be undertaken, given barriers such as the estimated sample size (50,000) of such a study, unless novel approaches to design are developed.[53]

If new evidence in the foreseeable future is not likely to settle the matter conclusively, where does that leave us? Some experts propose that the residual uncertainty means that an intervention policy is not warranted. In our judgment, however, and in that of many others,[1-11,13] the evidence supporting the diet-heart hypothesis is sufficiently persuasive to justify active nutritional intervention now.

## Developing a Nutritional Policy

Having reached the judgment that dietary fats are at least one of the causes of heart disease, we now turn to the practical aspects of developing and implementing a nutritional policy: What foods should be recommended to which patients?

### *The Projected Impact of Specified Foods on CHD*

The general approach to dietary management of serum cholesterol levels is well known: Replace animal fats with vegetable products and, for those who are overweight, lower the intake of calories.[54] To decide which particular foods are most effective in lowering serum cholesterol levels, it is helpful to take a more quantitative approach.[55,56] By how much is serum cholesterol lowered (or raised) by various actual foods (step 1), and by how much is the risk of CHD lowered when serum cholesterol is reduced by a given amount (steps 2 and 3)?

*Effects of foods on serum cholesterol level.* The chief dietary precepts of a fat-controlled diet are illustrated in Table 2, where we provide examples of commonly eaten high-fat foods and low-fat alternatives. To estimate the relative importance of eating a low- rather than a high-fat food, we have calculated (based on metabolic ward data) the theoretical contribution to the serum cholesterol level if each food were eaten daily. The foods that raise serum cholesterol levels the most are those that have the highest amounts of saturated fat and cholesterol: meats and animal fat, organ meats, butter and egg yolks.

*Effects of lowering serum cholesterol levels on*

TABLE 2.—*General Precepts of a Fat-Controlled Diet, With Examples to Indicate the Relative Impact Each Food Change Might Have on Serum Cholesterol Levels*

| *General Precepts* | *Example of a High-Fat Food** | *Approximate Contribution to Serum Cholesterol if Eaten Daily† (mg/dl)* | *Example of a Fat-Controlled Food* | *Approximate Contribution to Serum Cholesterol if Eaten Daily† (mg/dl)* | *Approximate Change in Serum Cholesterol Level (mg/dl)* |
|---|---|---|---|---|---|
| 1. Trim all visible fat | 6 oz untrimmed choice porterhouse steak | 38 | 6 oz trimmed choice porterhouse steak | 16 | −22 |
| 2. Substitute fish or chicken for beef, pork & lamb | 4 oz lamb chop | 20 | 4 oz skinned chicken | 7 | −13 |
| 3. Eliminate organ meats (liver, kidney, heart, brain, and sweetbreads) | 3 oz liver | 28 | 3 oz tuna | 5 | −23 |
| 4. Substitute polyunsaturated oils for solid cooking fats and monounsaturated oils | 1 oz bacon fat or lard | 9 | 1 oz safflower oil | − 9 | −18 |
| 5. Substitute polyunsaturated margarine for butter | 1 oz butter | 15 | 1 oz polyunsaturated margarine | − 2 | −17 |
| 6. Substitute egg whites for whole eggs | 1 whole egg | 19 | 1 egg white | 0 | −19 |
| 7. Substitute low-fat desserts for ice cream | 4 oz ice cream | 8 | 4 oz skim yogurt | 0 | − 8 |
| 8. Choose lean meats | 4 oz hamburger (35% fat) | 25 | 4 oz hamburger (10% fat) | 12 | −13 |
| 9. Substitute low-fat cheese for regular cheese | 2 oz cheddar cheese | 12 | 2 oz low-fat cheese | 2 | −10 |
| 10. Substitute skim milk for regular milk | ½ pint whole milk | 5 | ½ pint skim milk | 0 | − 5 |

*The foods listed here were selected because they are common parts of the American diet and because they have a relatively strong influence in raising cholesterol levels. We have not included processed combinations of food (such as hot dogs), whose effects will depend on their ingredients, nor foods that are rare or have little influence on serum cholesterol. These have been presented in detail elsewhere.[54,55] Grains, fruits and vegetables are omitted because they do not raise serum cholesterol levels unless eaten in quantities sufficient to induce obesity (exception: coconut).

†The average contribution of each food, if it were eaten daily, is calculated as $2.16S - 1.65P + 0.0677C - 0.5$, where S = % of calories from saturated fat, P = % of calories from polyunsaturated fat, and C = mg of dietary cholesterol per day. This simplified formula is based on metabolic ward studies of the effects of varying dietary composition while calories are kept constant,[34,35] and it projects averages that do not reflect the possibility of biologic variability among individual persons in their responses to a given food.

TABLE 3.—*Mean Levels of Plasma Cholesterol in United States Populations Reported by the Lipid Research Clinics,* by Age and Sex*

| Age (Years) | Plasma Cholesterol Level (mg/dl) | |
|---|---|---|
| | Women | Men |
| 20-24 | 162 | 162 |
| 25-29 | 174 | 179 |
| 30-34 | 174 | 193 |
| 35-39 | 188 | 201 |
| 40-44 | 196 | 205 |
| 45-49 | 205 | 213 |
| 50-54 | 222 | 213 |
| 55-59 | 231 | 215 |

*From Heiss et al.[62]

*CHD risk.* The beneficial effect of avoiding foods that increase levels of cholesterol can be projected from the cohort studies shown in Figure 2. The amount by which a person's risk changes will vary with the individual factors described below. On the average, however, a person with a moderately high initial serum cholesterol level (such as 250 mg per dl) has about a 1 percent lower risk of CHD for each 1 mg per dl decrease in the lipid level. In other words, dietary intervention that achieves a reduction in serum cholesterol of 25 mg per dl, may prevent (or postpone) a quarter of the CHD deaths in such persons.

This projection, however, may exaggerate the effectiveness of an intervention begun in the middle years of life when coronary arteriosclerosis is already established. It is also a relatively modest benefit when measured against the gains of 50 percent and more that may result from eliminating other major risk factors such as hypertension and smoking.[57] Nevertheless, larger reductions in cholesterol level (and CHD risk) would be likely to result from changing to extremely low-fat diets such as those recommended by Pritikin and eaten in many parts of the world.[58] Clearly, even a small reduction in risk would be considered worthwhile by many persons and would have an important impact on the health of the population as a whole.

### *Tailoring Dietary Advice to Particular Patients*

It seems likely that some patients will benefit more than others from a fat-controlled diet; the following considerations are relevant to deciding who should receive such dietary advice.

*Serum cholesterol level.* The most useful index for deciding how extensive a fat-controlled diet to recommend is the serum cholesterol level. (Low-density lipoprotein cholesterol might be a more precise criterion [see appendix], but its theoretical advantages have been difficult to demonstrate in practice, and total cholesterol remains the more available, economic and familiar laboratory test.) Middle-aged patients with serum total-cholesterol levels higher than 210 mg per dl are more likely to benefit from dietary advice than those with lower levels because the risk-benefit ratio is marginal or conceivably adverse below this point; through cause and effect seems unlikely, low levels of serum cholesterol have been associated with increased risk of cancer and stroke.[59,60] Patients with very high cholesterol levels have the potential for a greater reduction in risk per unit of serum cholesterol lowered than do those with moderate levels, because the relationship between serum cholesterol level and CHD incidence seems to fit an exponential-type curve.[61]

The choice of 210 mg per dl for the cutoff point is a somewhat arbitrary interpretation of the data in Figure 2. The value is close to the population mean for middle-aged men and women. Because younger people tend to have lower levels, it seems reasonable to recommend that the persons who are likely to benefit substantially from dietary advice are those with plasma cholesterol levels (confirmed by testing on several occasions) that are above the mean for their age and sex. Population-based data for such decisions are provided in Table 3.

*Other risk factors.* Hypertension, cigarette smoking, low levels of high-density lipoprotein, physical inactivity, and male sex are relevant because of the multiplicative interaction of risk factors. This means that the amount of risk associated with a given increment in serum cholesterol concentration is increased in the presence of other risk factors.[56,57,61]

*Age and prior heart attack.* Young people seem to be an attractive target for dietary intervention because primary prevention can be started before atherogenesis begins, and also because dietary habits learned in childhood may persist for a lifetime. On the other hand, there are psychological and pragmatic drawbacks to focusing the attention of selected children on adult diseases.

The elderly are at particularly high risk, but altering serum cholesterol levels may not be effective if advanced coronary arteriosclerosis has already developed. Those who have already had a heart attack have a relative risk of recurrence for a given increment of serum cholesterol that is much smaller than that for first infarction,[63] due perhaps to the presence of advanced coronary

arteriosclerosis. However, this observation is biased by special selection factors that operate in studies of men who have already had heart attacks,[64] and the high overall level of risk for such patients means that even a small proportionate reduction in mortality may save many lives.

## Conclusions

*Scientific and Public Health Considerations*

Our discussion thus far has dealt with the *science* of the diet-heart issue. We have shown that excessive dietary fat, eaten over a long time, is very likely to be a cause of heart disease. Further, that certain persons—particularly those above the 50th percentile of the population distribution with regards to serum cholesterol concentration and those with other risk factors—will probably have meaningful reductions in risk if they reduce their habitual intake of animal fat. At the same time, we suggest that there are also groups of people for whom such dietary intervention will be only marginally—if at all—beneficial.

The implications of these scientific judgments for public policy follow from the idea that, to be effective, public health measures should be applied uniformly to the entire population. We support existing nutrition guidelines[1-3] designed to educate the general public and to create a climate that encourages those people who choose to modify their diets. We propose the enactment of more informative food-labeling regulations that include a wider selection of foods and their ingredients and that specify quantities of saturated fat and cholesterol as well as standard nutrients. To that end, we believe that all foods—including meat, butter, eggs and other products listed on Table 2—should be sold with labels that enable consumers to estimate the potential increase of cholesterol level after eating an average serving. These policies will create an environment that supports an informed choice in eating habits throughout the population.

*The Art of Medicine*

We now turn to the *art* of applying these scientific judgments to the actual practice of medicine. At least two persons are involved in any clinical decision—the clinician and the patient. The decision to prescribe a low-fat diet depends on the individual characteristics of each.

The therapeutic philosophy of each *clinician* is based on years of educating and advising patients,

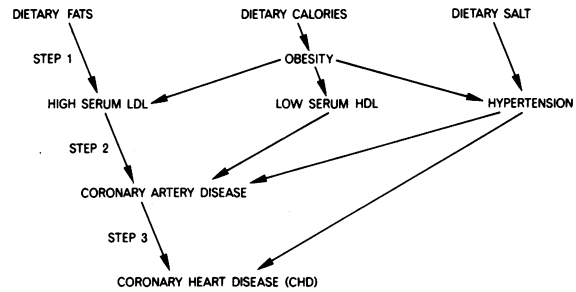


**Figure 3.**—Schematic representation of a more comprehensive diet-lipid-heart hypothesis, which presents lipoproteins in the place of total cholesterol and which includes proposed roles for dietary factors other than fats. (CHD = coronary heart disease; HDL = high-density lipoprotein; LDL = low-density lipoprotein)

and of observing the behavioral outcome. This experience creates a value system that will determine both the vigor of an intervention stance and the choice and method of presenting health information.

The ultimate determinant of the clinical decision, however, is the individual preference of the *patient*. Many will welcome the opportunity to make dietary changes directed at their future health, even if the likelihood of benefit is small. Others may be unwilling or unable to alter their habits. The patient's family situation bears on the decision, for there are practical, cultural and, sometimes, medical advantages to a common eating pattern for an entire household. Virtually all patients like to think of food as a good and satisfying part of life, and dietary advice should be tempered to enhance this natural and rewarding goal.

We have attempted in this report to reduce the confusion created by conflicting dietary recommendations[1-18] by suggesting that dietary intervention is more appropriate in some circumstances than in others, and that clinicians should use their professional skills to tailor advice based on scientific considerations to the particular needs and beliefs of each patient.

## Appendix

*Additional Aspects of the Diet-Heart Hypothesis*

A more comprehensive form of the diet-heart hypothesis is provided in Figure 3. Low-density lipoprotein (LDL), which carries most of the blood's cholesterol, occupies the same position as total cholesterol does in Figure 1; indeed, these two variables are usually comparable for clinical purposes.[65]

A growing body of evidence suggests that the

inverse relationship between high-density lipoprotein (HDL) levels and CHD incidence has a causal basis.[66] HDL does not bear on the discussion in this paper, however, because its relationship with CHD is independent of the LDL-CHD connection[65] and because those changes in dietary fat that lower the level of LDL- (or total-) cholesterol do not appear to influence HDL-cholesterol concentration.[67] The third major class of lipoproteins, those of very-low-density lipoprotein (VLDL), are not included in Figure 3 because the evidence that they are atherogenic is relatively weak.[21]

It appears that HDL and LDL are both determined in part by obesity.[54,67] However, the influence of calories on these two lipoproteins and on disease is beyond the scope of this paper, as is the concern with salt as a determinant of hypertension. Clinical decisions regarding calories and salt require the same combination of science and art noted in this report for advice on dietary fat. We refer readers to a recent review for the scientific facts necessary for this task.[7]

We should also note that dietary fats may have a role in the development of diseases other than CHD. The evidence for a causal connection between animal fats and cancer[68] is much less extensive than that discussed in this report, however, and we believe it is premature to use this rationale for recommending dietary changes.

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