Editorials

CALCIUM SUPPLEMENTATION DURING BREAST-FEEDING

Nursing mothers provide an average of 200 to 250 mg of calcium per day to their infants, and as much as 400 mg per day.\(^1\) Given that women absorb only about a third of the calcium they consume, the amount lost in breast milk represents a substantial proportion of the daily supply for many women and forms the basis for recommending that nursing mothers increase their calcium intake by 400 to 800 mg per day.\(^2\) There is widespread concern that women who do not increase their calcium intake could both jeopardize their own bone health by inducing the mobilization of skeletal calcium and compromise the transfer of calcium to their infants. In this issue of the Journal, however, Kalkwarf et al.\(^3\) report that calcium supplementation had no detectable benefit in lactating women with a usual dietary calcium intake of less than 800 mg per day. This counterintuitive finding challenges the assumption that breast-feeding women need extra calcium and raises intriguing biologic questions about calcium regulation during lactation.

Decreases in bone mineral in breast-feeding mothers are well documented, particularly in skeletal regions with a high proportion of trabecular bone.\(^3,4,6\) Typically, during three to six months of lactation, bone mineral density is reduced by 3 to 5 percent at the lumbar spine and femoral neck and by 1 to 2 percent in the whole body. These dramatic rates of loss of bone mineral density are similar to, or greater than, those in women shortly after menopause. The losses are reversed in subsequent lactation and after the end of breast-feeding, possibly in connection with the return of menses. The discovery is apparent even in women who become pregnant during lactation.\(^7\) These postpartum changes in bone are associated specifically with lactation and do not occur in women who do not breast-feed.\(^5,6\)

The response of bone to lactation is highly variable. In one study, for example, the recorded change in the bone mineral density of the lumbar spine after three months of lactation ranged from a loss of 9 percent to a gain of 2 percent.\(^5\) Similarly, women differ widely in the calcium concentration of breast milk and the daily calcium output in breast milk.\(^1\) There is no convincing evidence that either calcium concentrations in breast milk or maternal changes in bone mineral density are influenced by the consumption of calcium across a broad range of customary intake. Lactation-associated changes in bone mineral occur even in women with a high calcium intake (>1600 mg per day).\(^1\) Clinically, there is little to suggest that breast-feeding has a detrimental effect on long-term bone health or that failure to increase calcium intake during lactation affects the later risk of fracture.\(^1,2\)

Earlier studies of supplementation, which were initially greeted with some skepticism, also detected no effect of increased calcium intake in lactating women.\(^4,8\) A calcium supplement (714 mg per day) consumed for 12 months by lactating Gambian women accustomed to a very low calcium intake (283 mg per day) had no observable effect on the calcium concentration of breast milk, the bone mineral density of the forearm, the efficiency of calcium absorption, or biochemical markers of calcium and bone metabolism, although urinary calcium excretion increased.\(^1,8\) Similarly, a calcium supplement (1000 mg per day) consumed by American women with moderate-to-high calcium intake (1300 mg per day) had no effect on bone mineral density or bone turnover either after three months of lactation or three months after weaning.\(^4\)

The results of the controlled intervention study in lactating and nonlactating mothers by Kalkwarf et al.\(^3\) support these conclusions. Increasing the calcium intake by about 1000 mg per day in women with an average dietary intake of 720 mg per day had no effect on the calcium concentration of breast milk and did not alter the patterns of change in bone mineral density associated with lactation and weaning. There was a small increase in the bone density of the lumbar spine in both groups, possibly due to an effect on the rate of bone remodeling similar to those reported in other groups given calcium supplements. However, this increase was not specific to the breast-feeding women and resulted in only a small, nonsignificant difference in lactation-associated bone mineral loss in the lumbar spine after six months. Furthermore, in a subgroup of these women the calcium supplement did not alter the efficiency of calcium absorption or serum 1,25-dihydroxyvitamin D concentrations.\(^9\)

A picture is beginning to emerge showing that human lactation is associated with alterations in calcium metabolism, including the temporary mobilization and subsequent restitution of bone mineral, that are independent of dietary calcium intake and unresponsive to increases in calcium intake. The mechanisms involved are uncertain. The serum concentrations of the classic calcitropic hormones, parathyroid hormone and 1,25-dihydroxyvitamin D, are not elevated in early lactation or mid-lactation and increase only during and after weaning.\(^4,9\) Markers of bone resorption are elevated in the early postpartum period, and index of bone formation rise and fall during lactation.\(^10\) Renal conservation of calcium and phosphate is enhanced during lactation, whereas the efficiency of calcium absorption is elevated during weaning.\(^8,9\)

The intake of breast milk by the infant has been identified as a major determinant of the decrease in bone mineral...
mineral, suggesting that stimuli associated with suckling are important in regulating calcium metabolism. Breast-feeding women have elevated serum concentrations of prolactin and parathyroid hormone–related peptide and low serum estradiol concentrations, all of which are likely to influence calcium handling and bone metabolism.

Overall, the evidence suggests that optimal lactation and maternal bone health do not depend on an increase in calcium intake by the breast-feeding mother. This tentative conclusion does not imply that good nutrition, including the maintenance of adequate calcium intake, is unimportant during lactation. However, the accumulating scientific data suggest that breast-feeding women need not consume extra calcium.

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THE PROMISE AND PROBLEMS OF META-ANALYSIS

META-ANALYSIS has acquired a substantial following among both statisticians and clinicians. The technique was developed as a way to summarize the results of different research studies of related problems. Meta-analysis may be applied even when the studies are small and there is substantial variation in the specific issues studied, the research methods applied, the source and nature of the study subjects, and other factors that may have an important bearing on the findings. In this issue of the Journal, LeLorier et al.1 compare the findings of 12 large randomized, controlled trials with the results of meta-analyses of the same problems. They find important discrepancies. When a large randomized, controlled trial — commonly considered the gold standard for determining the effects of medical interventions — disagrees with a meta-analysis, what should the reader conclude? Perhaps more important, when only one of the two tools is used, how much uncertainty should the reader add to the confidence limits and other statistical measures of uncertainty reported by the author?

The core of meta-analysis is its systematic approach to the identification and abstracting of critical information from research reports. Doing a meta-analysis correctly demands expertise in both the method and the substance and hence almost always requires collaboration between clinicians and an experienced statistician. The questions must be defined carefully to maximize the relevance of the reports to be included and to reduce uncertainties about procedures. The investigators must then try to find every relevant report by searching data bases, reviewing bibliographies, and asking widely about unpublished work. The collected reports are then winnowed to the few (often less than 10 percent) that meet the requirements for the meta-analysis. The reports must be searched carefully to identify problems and validate the quantitative findings of interest. These findings must be expressed on a common scale (often as odds ratios), and some reports may have to be dropped for lack of information. Those doing a meta-analysis may also abstract information from each report to produce a quantitative measure of research quality. Each of the individual quantitative estimates must be scrutinized for problems, and this may require the efforts of a range of specialists. When the analysis is completed and submitted for publication, the editor and the reviewers must assure themselves of its quality. A technical review of a meta-analysis requires the reviewer to identify, reabstract, and interpret a fair sample of the original papers. Very few editors and reviewers will do this, which may be one reason why there are so many poor meta-analyses in the literature.

Although some meta-analyses stop with the presentation and discussion of the results of the individual studies, many others proceed further and combine the results into a single, comprehensive “best” estimate, generally with statistical confidence bounds, that is meant to summarize what is known about the clinical problem. This last step — preparing and presenting a single estimate as the distillation of all that
is known — is the one that has drawn the most criticism. This is because there are often biologic reasons, statistical evidence, or both, showing that the studies included in the meta-analysis have in fact measured somewhat different things, so that a combined estimate cannot be meaningful unless additional, doubtful assumptions are made. One such assumption is that the effects reported in the studies actually performed can be seen as a random sample of the effects observed in all possible studies that might have met the author’s criteria. Unfortunately, there is little evidence to support an assumption such as this.

LeLorier et al. searched four leading general medical journals to identify all the large randomized, controlled trials (those with 1000 subjects or more) whose results were published from 1991 through 1994, then searched for meta-analyses of similar topics that were published before each trial. Twelve large randomized, controlled trials and 19 meta-analyses met their criteria. Because some of the trials and meta-analyses reported on more than 1 outcome, they studied a total of 40 outcomes. Overall, there was somewhat better than chance agreement between these meta-analyses and the subsequent large randomized, controlled trials, with kappa values in a range commonly considered to represent “fair-to-slight agreement.” In terms of an ordinary diagnostic test, the results could be described as average. The results obtained with the two approaches usually pointed in the same direction, and there were no cases in which they gave statistically significant results in opposite directions. However, the discrepancies with regard to the estimated size of an effect were sometimes quite substantial, and occasionally they differed significantly despite their agreement in direction. Stewart and Parmar have shown how some such differences can arise.

It is impossible to say, on the basis of present evidence alone, whether the results of a large randomized, controlled trial or those of a meta-analysis of many smaller studies are more likely to be close to the truth. Much depends on the details of both the research studies and the analyses. When both the trial and the meta-analysis seem to be of good quality, however, I tend to believe the results of the trial. A history of 40 years of generally successful use of randomized, controlled trials that have made important contributions to progress in many branches of medicine must not be overlooked. In addition, major problems with the implementation of meta-analyses have been common. There have been a wide variety of these, including failure of the investigator performing the meta-analysis to understand the basic issues, carelessness in abstracting and summarizing appropriate papers, failure to consider important covariates, bias on the part of the meta-analyst, and, perhaps most often, overestimations of the strength and precision of the results. It is not uncommon to find that two or more meta-analyses done at about the same time by investigators with the same access to the literature reach incompatible or even contradictory conclusions. Such disagreement argues powerfully against any notion that meta-analysis offers an assured way to distill the “truth” from a collection of research reports.

Other observers, including policy makers, also have reservations about meta-analyses, and there is some general concern about the credibility of the findings of meta-analysis. I know of no instance in medicine in which a meta-analysis led to a major change in policy before the time when a careful, conventional review of the literature led to the same change. Showing that a sequence of meta-analyses performed over time converged to have some value as published findings accumulated does not mean that it was the meta-analyses that gave a convincing answer to the particular clinical question.

In my own review of selected meta-analyses, problems were so frequent and so serious, including bias on the part of the meta-analyst, that it was difficult to trust the overall “best estimates” that the method often produces. On present evidence, we can generally accept the results of a well-done meta-analysis as a way to present the results of disparate studies on a common scale (as is shown by the two figures in the article by LeLorier et al.), but any attempt to reduce the results to a single value, with confidence bounds, is likely to lead to conclusions that are wrong, perhaps seriously so. I still prefer conventional narrative reviews of the literature, a type of summary familiar to readers of the countless review articles on important medical issues.

Meta-analysis may still be improved, by a combination of experience and theory, to the point at which its findings can be taken as sufficiently reliable when there is no other analysis or confirmation available, but that day seems to be well ahead of us. LeLorier et al. also imply, however, that large randomized, controlled trials should be regarded more circumspectly than published reports commonly suggest. We never know as much as we think we know.

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BARRY H. SLAVEN, M.D., PH.D.
Clinical Debate

Should a Low-Fat, High-Carbohydrate Diet Be Recommended for Everyone?

Low-fat diets are often advocated for weight reduction and to lower the risk of coronary heart disease and certain forms of cancer. However, there is no universal agreement about the value of low-fat diets. In this Clinical Debate, Connor and Connor argue in favor of the public health benefits of low-fat diets, whereas Katan, Grundy, and Willett present a different point of view.

THE CASE FOR A LOW-FAT, HIGH-CARBOHYDRATE DIET

The association between the dietary intake of fat and cholesterol and the extent of atherosclerosis and coronary heart disease has been recognized since 1907, when de Langen found little atherosclerosis in native Javanese but extensive atherosclerosis in the Dutch settlers in Java. As might be expected, the diets of the two groups of people differed greatly in fat content. At almost the same time in Russia, Anitschkow produced extensive atherosclerosis in rabbits by feeding them cholesterol and fat. In both humans and animals fed high-fat, high-cholesterol diets, serum cholesterol levels greatly increased. Subsequently, after World War II, Keys emphasized that mortality from coronary disease in various countries was directly related both to the amount of dietary fat consumed and to serum cholesterol levels. An extensive data base on this subject was reported in the Seven Countries Study. Mortality from coronary disease also correlated well with dietary cholesterol intake in another analysis of data from 35 countries.

Exceptions to these associations were observed, however. The intake of saturated fat and animal fat correlated better with the incidence of coronary disease than did total fat intake, as is now widely recognized. It was subsequently shown that saturated fats, as well as dietary cholesterol, elevate plasma total and low-density lipoprotein (LDL) cholesterol by down-regulating the LDL receptors in the liver. Another exception was observed in Crete, where there was a high intake of olive oil (a monounsaturated fat) but a low rate of death due to coronary disease in a population adapted to a frugal lifestyle. The Greenland Eskimos also had a low incidence of coronary disease despite a diet rich in fat and cholesterol. The Eskimo diet was protective against coronary disease because it contained n-3 fatty acids from fish and seal. These fatty acids are antithrombotic and lower serum lipid levels.

Experiments in monkeys and other animals confirmed these epidemiologic observations. Feeding fat and cholesterol (e.g., eggs and butterfat) to rhesus monkeys produced high serum cholesterol levels and severe coronary atherosclerosis, even myocardial infarction. That these conditions were largely reversible was documented when a low-fat, cholesterol-free diet was subsequently fed to the monkeys, proving that a drastic reduction in serum cholesterol levels would ultimately be followed by considerable reversal of coronary atherosclerotic lesions (from 60 percent to 20 percent occlusion). Years later, angiographic evidence of improvement in lesions was found in humans treated with low-fat diets alone or with diet plus lipid-lowering drugs.

Various scientific bodies, including the American Heart Association and the National Heart, Lung, and Blood Institute, have recommended reductions in dietary fat intake to treat or prevent coronary disease. The suggested level of dietary fat is 30 percent or less of total energy intake, reduced from the previous high population intake of 40 percent in the 1960s. Even the American Cancer Society has suggested a 50 percent reduction in fat intake (from 40 to 20 percent of energy intake) to prevent cancer of the colon and breast and other cancers. There is universal agreement that the dietary level of saturated fat should be greatly reduced, from the current intake of 11 percent to 6 to 8 percent of energy intake, along with a reduction in dietary cholesterol. Most recommendations have specified that the saturated fat eliminated from the diet be replaced by carbohydrates from grains, vegetables, legumes, and fruits. This change would diversify the diet and add protective constituents from plant sources.

If the saturated fat were replaced by monounsaturated or polyunsaturated fat, as some have suggested, the fasting serum total and LDL cholesterol levels would be decreased without significantly decreasing total fat intake. Is this an appropriate idea? The strategy of replacing fat with fat does not take into account the disadvantages of a high-fat diet. First, postprandial lipemia is atherogenic and directly reflects the amount of dietary fat ingested. A lower-fat diet would reduce postprandial lipemia composed of chylomicrons and atherogenic remnants that circulate in the plasma after the ingestion of dietary fat. Second, although obesity remains an unsolved problem, there is good evidence that Americans who follow a low-fat diet high in plant foods lose weight more easily than those whose fat intake is higher. Third, the energy cost of metabolizing a dense nutrient like fat is lower than the energy expenditure needed to metabolize carbohydrate from plants. Finally, the advantages of a lower-fat diet in the prevention of cancer and the control of
hypertension are lost when saturated fat is replaced by other fats, rather than by vegetable foods.

Since 1980, the recommendations of scientific bodies have been translated for the public by the Department of Agriculture (USDA) and the Department of Health and Human Services and published every five years as “Dietary Guidelines for Americans,” and both the public and the food industry have attempted to follow them. According to the USDA’s Continuing Survey of Food Intakes by Individuals, dietary fat intake has decreased to about 33 percent of energy intake from a previous 40 percent, and dietary carbohydrate has increased from 45 to 52 percent of energy intake from the 1960s to 1995. These changes bring the average diet close to the goal of obtaining no more than 30 percent of energy intake from fat and 55 percent from carbohydrate. However, dietary fiber intake is still low at 15 g per day (the goal is 20 to 35 g per day).

The plethora of “fat-free” foods has offered people the opportunity to consume more sugar in, for example, sweet rolls, cookies, and frozen yogurt. According to recent USDA food-consumption data, the intake of sugar and other refined sweeteners increased from about 55 kg (120 lb) per person per year in 1970 to 68 kg (150 lb) per person per year in 1995. There has been a small increase in the consumption of plant foods containing protective factors such as antioxidants, soluble fibers, and saponins from grains, legumes, vegetables, and fruits and in the intake of the n-3 fatty acids found in seafood, dark green vegetables, nuts, and oils, but all are short of the recommended goals.

Table 1 shows which of the recommended dietary changes for optimal health throughout life the U.S. population has adopted, as well as recommended changes not yet made. Although there has been progress, further steps need to be taken to achieve a more healthful diet.

There is little doubt that Americans have improved their health by lowering their dietary intake of fat and cholesterol. Serum cholesterol levels are lower, and mortality due to coronary disease is 20 to 30 percent lower than 25 years ago. The directions for change are clear, and further implementation of the currently recommended dietary goals is indicated.

**TABLE 1. RECOMMENDED DIETARY CHANGES.**

<table>
<thead>
<tr>
<th>ADOPTED BY THE PUBLIC</th>
<th>NOT YET ADOPTED BY THE PUBLIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consume leaner meats</td>
<td>Consume less sugar in foods and beverages</td>
</tr>
<tr>
<td>Eat more chicken</td>
<td>Eat more seafood</td>
</tr>
<tr>
<td>Use oils for cooking</td>
<td>Reduce intake of added fats</td>
</tr>
<tr>
<td>Eat fewer egg yolks</td>
<td>Use low-fat cheeses</td>
</tr>
<tr>
<td>Drink less whole milk</td>
<td>Drink skim milk</td>
</tr>
<tr>
<td>Eat frozen yogurt</td>
<td>Double intake of grains, legumes, vegetables, and fruits</td>
</tr>
</tbody>
</table>

*Recommendations are adapted from Department of Agriculture data.15

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**BEYOND LOW-FAT DIETS**

Excess intake of fat is considered an important cause of chronic diseases, but will a reduction in total dietary fat intake provide the health benefits implied by supporters of this strategy? We are concerned that popular belief and scientific evidence on this question have diverged to an alarming extent. Studies in the 1950s and 1960s showed that replacing saturated fat with polyunsaturated fats low-
erased serum total cholesterol levels, and clinical trials subsequently suggested that this dietary change may reduce the incidence of coronary heart disease. Consequently, diets high in polyunsaturated fats were widely recommended for the prevention of coronary heart disease. However, concern developed about the safety of polyunsaturated fats, whereas the perception grew that carbohydrates were innocuous. The focus of recommendations therefore shifted to diets low in fat and high in carbohydrates. Interest in monounsaturated fat also increased, but at present the dominant dietary advice is to replace foods high in total or saturated fat and cholesterol with high-carbohydrate foods such as pasta, potatoes, rice, and bread.

**High-Carbohydrate Diets and Cholesterol Levels**

Diets that lower serum total cholesterol levels are believed to lower the risk of coronary heart disease. This belief is justified if the cholesterol being lowered is LDL cholesterol; high LDL cholesterol levels cause coronary heart disease, and treatments that lower LDL cholesterol reduce the incidence of coronary heart disease. Replacement of saturated fats by unsaturated oils reduces mainly LDL cholesterol. However, as illustrated in Figure 1, low-fat, high-carbohydrate diets lower not only LDL cholesterol but also high-density lipoprotein (HDL) cholesterol levels. HDL cholesterol levels are lowered by both sugars and complex carbohydrates (starch), and the depression of HDL cholesterol lasts for as long as the low-fat diet is eaten.

This effect of carbohydrates on HDL cholesterol is a cause for concern. Low HDL cholesterol levels are strongly associated with coronary heart disease, and many factors that produce lower HDL cholesterol levels increase the risk of coronary heart disease; examples are smoking, obesity, lack of physical activity, abstinence from alcohol, and male sex. Premature coronary heart disease is seen in most genetic HDL-deficiency syndromes, especially in those in which LDL cholesterol levels are normal or high. In trials of lipid-lowering agents, drug-induced changes in HDL cholesterol levels independently predicted changes in the risk of coronary heart disease. Also, the induction of high HDL cholesterol levels in animals retards atherogenesis, and the infusion of HDL protein retards the development of fatty streaks. Thus, lowering HDL cholesterol levels usually increases the risk of coronary heart disease, and diets that lower HDL cholesterol levels must be viewed with concern.

People in China and rural Japan have both low total dietary fat intake and low rates of coronary heart disease. However, such populations are also highly active and extremely lean — both factors that raise HDL cholesterol levels and reduce plasma triglyceride levels, thus offsetting the adverse changes caused by low-fat diets. The low rates of coronary heart disease in the Chinese and other rural populations may therefore be due largely to their high levels of physical activity and low body fat, plus their low intake of saturated and trans fats, rather than to low total fat intake. (Trans fats are created during the partial hydrogenation of oils; they raise LDL cholesterol and lower HDL cholesterol levels.)

**Obesity**

A major argument for low-fat diets is that they should promote weight reduction. Fat is calorically dense and often is hidden in food products. Theoretically, high-fat diets could facilitate the overconsumption of calories and promote weight gain; however, controlled trials have not supported this idea.
In a free-living population, when calories from fat are intentionally restricted, they appear to be largely replaced by carbohydrate. A limited weight reduction is seen after people start a fat-restricted diet, but weight loss stops after a few months, and the long-term net weight loss is only 0.8 to 2.6 kg (Fig. 2). The prevalence of obesity has increased by one third in the United States since 1976, whereas the percentage of total energy intake from fat has declined. Thus, fat restriction does not invariably produce weight reduction; obesity is a complex problem that will not be solved solely by reducing the percentage of fat in the diet.\textsuperscript{15}

**Cancer**

Evidence that dietary fat may cause cancer in humans derives primarily from comparisons of rich and poor nations; these comparisons, like those of the incidence of coronary heart disease, are potentially confounded by many differences in lifestyle. In large prospective studies, fat intake has had no overall relation to the risk of breast cancer.\textsuperscript{16} For colon cancer, more detailed evidence suggests a role of red meat but not of total fat, and for prostate cancer associations have been seen with animal but not vegetable fat.\textsuperscript{16}

**The Alternatives to Low-Fat Diets**

Replacement of fat by carbohydrates has not been shown to reduce the risk of coronary heart disease,\textsuperscript{2} and benefits are unlikely, because this change similarly lowers HDL and LDL cholesterol\textsuperscript{4} and reduces the intake of vitamin E and essential fatty acids. Beneficial effects of high-carbohydrate diets on the risk of cancer or on body weight have also not been substantiated. Alternatively, the replacement of fats high in saturated and trans fatty acids by unhydrogenated unsaturated oils improves the ratio of HDL to LDL cholesterol. Overweight persons can decrease their intake of saturated and trans fat by reducing their consumption of fat from dairy products, meats, and partially hydrogenated oils; they should also eat less sugar and highly refined starch. Carbohydrates should be consumed mainly in the form of fruits, vegetables, legumes, and whole grains. In people who are close to their ideal weight, saturated and trans fats could be replaced largely by unsaturated vegetable oils and highly refined carbohydrates by fruits, vegetables, and whole grains.

Everything we know about lipoproteins and heart disease tells us that such diets will reduce the risk of coronary heart disease. For oils high in polyunsaturated fats, this effect is supported by controlled clinical trials.\textsuperscript{2} However, studies in animals have aroused concern about a relation between polyunsaturated fats and cancer, even though observations in humans are reassuring. At present it appears prudent to replace the majority of saturated and trans fats by oils high in monounsaturated fats such as rapeseed (canola) oil and olive oil. The experience in Mediterranean countries shows that diets high in monounsaturated fats, such as those in Mediterranean countries, show a lower risk of coronary heart disease than those in the United States, where the intake of dietary fat is lower. The following diagram illustrates the effects of dietary fat reduction on body weight in long-term randomized, controlled trials.

![Figure 2. Effects of Dietary Fat Reduction on Body Weight in Long-Term Randomized, Controlled Trials. The studies are those reviewed by Willett. Values for the fat-reduction group have been corrected for weight changes in the concurrent control group. Values in parentheses are the percentages of energy from fat in the control group and the fat-reduction group, respectively. NDH denotes National Diet–Heart Study.](image-url)
fats can be attractive and that they are associated with longevity and a low incidence of coronary heart disease and cancer.

Finally, the intense focus on total fat intake not only is unlikely to be beneficial but also distracts people from lifestyle changes that can have real benefits. These include specific dietary reductions in saturated and trans fat, increases in the consumption of fruits, vegetables, and whole grains, and the prevention of excessive weight by greater physical activity and reductions in overall caloric intake.

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REBUTTALS

Dr. Connor and Ms. Connor respond:

Although there is refreshing agreement about increasing the consumption of plant-derived foods, we suggest that a greater amount be consumed, to provide optimal intake of fiber, antioxidants, and other protective factors. In the study cited by Katan et al., subjects were fed a high-fat diet (41 percent of total energy), most of this from olive oil, which would dilute the effect of grains, beans, vegetables, and fruits.

We emphasize that Americans have already cut fat intake to 33 percent of total energy consumption, but the fat has been replaced largely with sugar, not complex carbohydrates and fiber. Fat intake should be reduced further. A low-fat diet (27 percent of energy intake from fat) is even advantageous for blood-pressure reduction.

There are no data showing that the physiologic reduction of HDL cholesterol levels with a low-fat diet is detrimental. Diet-induced lowering of HDL cholesterol does not confer the same risk of atherosclerosis as do low HDL cholesterol levels in Americans consuming a high-fat diet. Lowering HDL cholesterol levels by the consumption of a low-fat diet results in more rapid clearance of HDL and decreased transport of HDL apoproteins. Populations eating a low-fat diet that have low HDL cholesterol levels do not have an increased incidence of coronary disease. Neither do patients with some forms of inherited low plasma HDL cholesterol levels, including Tangier disease, in which HDL is virtually absent.

Finally, the recommendations of Katan et al. invite the American public to increase fat intake when all health agencies are advocating the consumption of less fat and more complex carbohydrates and fiber. Such conflicting recommendations only compound public health education about nutrition. Ninety years of research should permit the scientific community to speak with a consistent voice about diet and the prevention of coronary disease. The public deserves the opportunity to implement fully the current, scientifically sound recommendations.

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Drs. Katan, Grundy, and Willett respond:

We agree that the intake of fruits, vegetables, and high-fiber foods should be increased and that the high amounts of saturated and trans fatty acids consumed by Americans in the 1950s were unhealthful. However, one cannot generalize from those fats to all fats, because unsaturated fats, which make up the majority of fats in the current U.S. diet, lower LDL cholesterol levels. Reductions in saturated fat and cholesterol in the diet cannot by themselves account for the decline in mortality due to coronary heart disease since the 1950s; increases in the consumption of unsaturated vegetable oils may have been important as well.

Connor and Connor object to substituting unsaturated fats for unhealthful fats because fats produce postprandial lipemia. However, long-term substitution of carbohydrate, instead, does not reduce postprandial lipemia, probably because high carbohydrate intake increases the levels of endogenous triglycerides that delay postprandial clearance of lipoproteins. HDL cholesterol levels are also reduced. In the study cited by Connor and Connor, a low-fat, high-fiber diet reduced weight after 12 weeks, but this effect was transient in longer studies (see our Fig. 2). There is no good evidence that reducing total dietary fat will prevent cancer or hypertension.

Like Connor and Connor, we lament the plethora of fat-free products high in sugar and the avoidance of foods such as nuts and oil-based salad dressing that provide n-3 fatty acids. However, the failure to distinguish among types of fat and the emphasis on total fat reduction are the very causes of these problems.

Lower consumption of saturated and trans fats is desirable, but nonspecific recommendations to reduce total fat consumption have no strong scientific basis and could be harmful if unsaturated fats are avoided.

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