equilibrium there is an essentially random mixture of glycerol, mono-, di-, and triglycerides. The reaction cannot be driven to completion by adding an excess of glycerol, because of its limited solubility. It ranges from 22.5% at 200°C. to 40% at 250°C. (28). Most commercial monoglyceride mixtures are prepared at about 200°C. with sodium hydroxide as catalyst and contain 40 to 45% alpha monoglycerides plus about 4% beta monoglycerides. Mutual solvents have been proposed to increase the monoglyceride content (29), but most concentrates are made by molecularly distilling mono- and diglyceride mixtures. The distilled products have a monoglyceride content of 90% or more.

Phosphoric acid has been recommended for destroying the alkaline catalyst (30). The reaction should be halted before any cooling occurs, otherwise glycerol will drop out of solution and reduce the yield of monoglycerides.

### Acidolysis plus Interesterification

Fats like coconut oil, which contain low molecular weight acids, may be modified by displacing the short chain acids with longer chain acids (31). The reaction can be carried out under reduced pressure in a vessel equipped with a fractionating column through which the lower molecular weight acids are continuously removed as they are freed (32). The product has a random distribution of the remaining acids. The usual high-temperature interesterification catalysts are effective. A similar result can be obtained by using monoesters instead of the free acids (32,33).

Interesterification and allied ester reactions are also used quite extensively in the manufacture of paints and plasticizers. These applications were reviewed by Formo in the 1954 A.O.C.S. Short Course (34).


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# Dietary Fat and Heart Disease

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WITHIN THE PAST FEW YEARS all of us in the fats and oils industry have become acutely aware of the research indicating a relationship between heart disease and dietary fats. Besides the potential effects on our markets, these programs are of vital concern in terms of the health of the nation, our families, and ourselves.

This is a controversial topic and an active area of research. There are important differences between experimental animals and human beings in the ways in which blood vessels react to the dietary factors presumed to lead to atherosclerosis and heart disease. Therefore available data are inconclusive to the extent that they provide at least some degree of support for each of several points of view.

My purposes in the present review are to outline the nature of the evidence now available, to indicate the limitations in the data, and to discuss the interpretations and hypotheses on which current clinical research is based.

First of all, there is no doubt that fats are excellent foods. They are highly concentrated sources of energy and contribute much to the enjoyment we get out of our meals. As a matter of fact a certain amount of fat is necessary to provide the essential fatty acids which our bodies are unable to synthesize and, in addition, to carry into our tissues the fat-soluble vitamins which are so important to health. Lipid materials, of which fats are one class, are important constituents of all living cells and life as we know it would be impossible without these fatty materials. Thus, there is no doubt that fats are excellent foods.

However, recent clinical studies have clearly shown that there are important differences in the way in which our bodies metabolize fats from different sources and that these differences may be associated with the development of coronary heart disease.

One of the bits of evidence linking heart disease with dietary fats has come from population and epidemiological studies. Statistics are being amassed in all parts of the world on local incidences of various diseases, the foods people eat, and the conditions under which they live. There are very serious limitations to the accuracy of such data and consequently they must be interpreted with caution. For example, medical records from the United States are not directly comparable to those from India where few people ever see a doctor. In addition, reliable dietary histories are difficult to obtain so the information available generally relates to the quantities of food available in the mar-

kets rather than to the amounts actually eaten by given individuals. Nonetheless, such statistics are valuable as guides in the planning of the clinical research from which we can expect to get the answers to our questions.

Clearly evident from the statistics is an association of coronary heart disease with the prosperous way of life characteristic of industrially advanced countries such as the United States. For example it would be possible to demonstrate a fairly good correlation between the incidence of coronary heart disease and the number of television sets (50). No one has yet claimed that the TV industry is responsible for heart disease. But the pattern becomes clear when we remember that television is a luxury. The mere fact that one can afford to buy a TV set means that he has some extra money which is not absolutely required in paying for housing, food, and clothing. It means he has time enough to sit around and watch a TV program—and incidentally to have frequent snacks while doing so. Thus TV is symbolic of a sedentary way of life with limited physical activity and overeating. One consequence may be obesity, long known to be a factor associated with heart disease.

The ultimate answer as to what causes heart disease is certain to be very complex. It is already apparent that almost every aspect of an individual's way of life is involved: how much and what kinds of foods he eats; the nature of the stresses under which he works and plays; whether he gets much exercise or little; various aspects of his hormone balance and metabolism; his personal habits and reactions to environmental pressures. While I do not mean to minimize any of these other factors, the topic will be limited during the present discussion to dietary fats since this is the aspect of greatest interest to the present audience.

At first glance data from epidemiological studies seemed to indicate a correlation between *per capita* fat intake and the incidence of coronary heart disease (27,28,49). This was not too surprising since fats are concentrated foods, providing about 9 calories per gram as compared to about 4 calories per gram of protein or carbohydrate. Hence a high fat diet can very easily be a high calorie diet, favoring gains in body weight. In the United States where fat consumption is of the order of magnitude of 40% of the total calories, the incidence of heart disease is high. By contrast, Japanese who eat very little fat seem to have a low incidence of heart disease.

As a first approximation, this correlation of fat consumption with heart disease was useful. But further study revealed that this is not a simple association of heart disease with total fat intake, as is illustrated in Figure 1. Low fat consumption may coincide with low coronary death rates, as in Japan. It is quite apparent, however, that there is a tremendous variation at the high fat level. For example, the death rate on 38% fat diets may be as low as 200/100,000 in Norway or as high as 550 in Canada.

Data in Figure 1 would seem to indicate that geographical areas fall into two groups with respect to the nature of the dietary fat, *i.e.*, the ratio of saturated to unsaturated fats. a) When this ratio is low, it indicates that the predominant food fats are the more liquid types of plant or marine origin, such as olive, corn, soybean, cottonseed, or fish oils. Coincidentally, coronary death rate is comparatively low even though the total fat consumption may be very high. b) Where

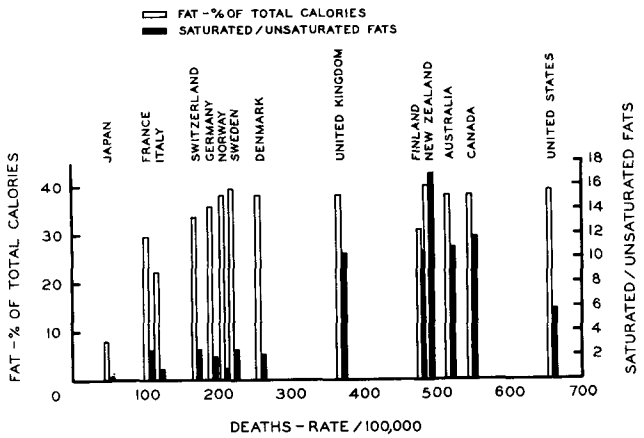


FIG. 1. Death rate from coronary heart disease compared with total fat intake and the nature of dietary fats in various countries. Drawn from data tabulated by Jolliffe and Archer (24) for men aged 55–59 years.

the ratio of saturated to unsaturated fats is high, the predominant food fats are of animal origin, such as butter, cream, and meat fats, or are hydrogenated oils. In these areas, the coronary death rate is high. From Figure 1, one might conclude that the critical ratio is somewhere between 2 and 4. However a great deal more research must be done before the most desirable ratio can be defined. The fact that data for the United States appear to be out of line further emphasizes that many factors, in addition to fats, are involved.

Recently Jolliffe and Archer (24) made a detailed analysis of international statistics in terms of coronary death rates and composition of diets in relation to total daily calories, total fat, saturated fat, unsaturated fat, and animal protein—as well as telephones—per 100 inhabitants. They concluded that the intake of saturated types of fats was the most important variable in accounting for differences in coronary death rates between countries. The next most important variable was the intake of animal protein. Bronte-Stewart (9) has likewise pointed out that the ratio of hard to liquid fats in the diet may be a more important factor than total fat intake.

However, as was noted earlier, there are some serious inherent deficiencies in data of this type. It is important to remember that these data have given valuable leads to the role of dietary fats in the etiology of heart disease, but the hypothesis must still be exhaustively tested by carefully controlled clinical and biochemical studies.

Careful examinations of arteries taken at autopsy have yielded valuable clues about stages in the development of heart disease. In examining tissues from cases of fatal coronary attacks, the pathologist more often than not finds extensive deposits of lipids in the walls of the blood vessels. These deposits are known as atheromatous plaques and the condition is atherosclerosis. The predominant lipid in these plaques is cholesterol. How it gets there and why it accumulates are questions under intensive investigation at the present time.

As is evident from Figure 2, fatty deposits (fatty streaks) in the blood vessel walls occur frequently even in very young children in this country. The number and extent of these fatty streaks increases sharply in the male during adolescence. Later, plaques

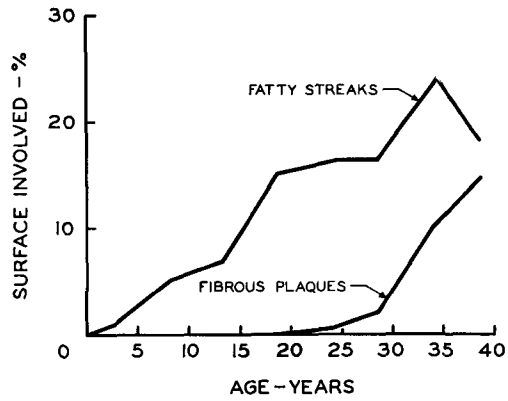


FIG. 2. Aortic atherosclerosis in white males in New Orleans. Average percentage of surface covered by fatty streaks and fibrous plaques in 162 cases (23).

overlaid with fibrous material begin to appear, presumably at sites of earlier fatty streaks. Eventually the plaque may ulcerate and perhaps become the locus for a blood clot. While this is the natural history of the lesion, the severity of atherosclerosis in a given individual is not necessarily a good index of his chances of having a heart attack. All we can say is that clinical symptoms are usually associated with extensive atherosclerosis.

The important point for the present discussion is that autopsies done in countries like Guatemala (17), where incidence of atherosclerotic heart disease is low, indicate far fewer fatty streaks than are shown in Figure 2 and a much later appearance of fibrous plaques. As was noted above, these are the regions where diets are predominantly vegetarian, low in fat, or relatively poor in saturated fats.

While no one yet knows how or why these cholesterol-rich atheromatous plaques form, it is noteworthy as is shown in Figure 3, that, on an average, above normal concentrations of cholesterol in the blood are associated with atherosclerosis and coronary heart disease (15,18,26,31,33,38,43,44). Furthermore the average serum cholesterol level of Americans is far above the average of peoples in nations where the incidence of coronary heart disease is low (8,10,26,27,28,40,45). Significantly, in the few instances when it has been possible to induce myocardial infarction (*i.e.*, a condition analogous to coronary heart disease) in experimental animals, this has been achieved by the use of diets which contained all of the factors acting to keep serum cholesterol concentrations at a high level over an extended period of time (19,47).

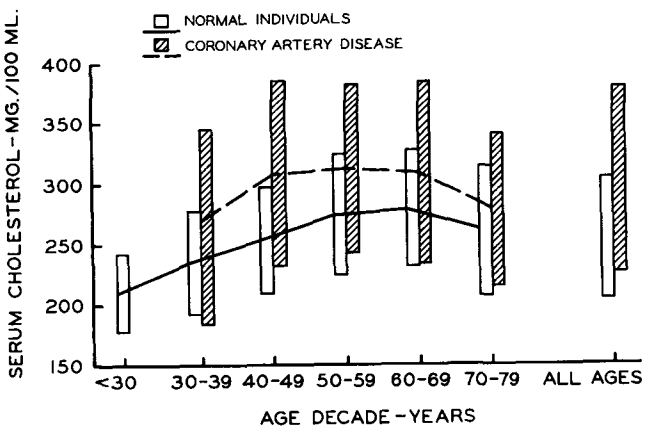


FIG. 3. Average serum cholesterol values at various ages in normal subjects and patients with coronary artery disease (41).

In spite of these implications it must be emphasized that the association of elevated serum cholesterol levels with coronary heart disease is as yet merely a working hypothesis. It is not an established fact. There is absolutely no way of predicting from a single determination of an individual's serum cholesterol level whether or not he has or is a candidate for coronary heart disease. Nonetheless the association is impressive. As a result, for many years, doctors have recognized elevated cholesterol levels as a danger signal and have adopted various means of bringing the cholesterol levels back to normal.

Studies of large population groups indicate that three factors show up repeatedly in association with heart disease, namely obesity, elevated blood pressure, and elevated serum cholesterol level. Data from one such study are summarized in Figure 4. During the

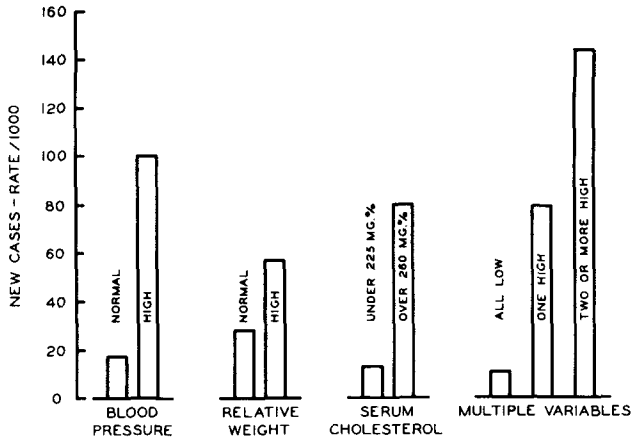


FIG. 4. Incidence of deaths from atherosclerotic heart disease among men aged 45-62 years in relation to blood pressure, weight, and serum cholesterol level (13).

period of this study there were only 10 new heart cases for every 1,000 individuals in whom blood pressure, weight, and serum cholesterol level were all normal or low. When just one of these values was high, the number of new heart attacks was 79 but when two were high the rate was 143—more than 14 times greater than among the normal group.

Of these three factors, weight and cholesterol levels are easily affected by what and how much one eats. If more food is eaten, that is, more calories are ingested than are needed for physical activities and the maintenance of body functions, the excess is stored as fat. Although this fat is derived from all classes of foods—carbohydrates and proteins as well as fats—fats are frequently the culprits in obesity since they have the highest caloric value. For this reason, most reducing diets are planned to contain very little fat-rich foods such as cream, pastries, and fried foods. As will be seen later, this restriction has the extra benefit for a coronary-prone person of decreasing the intake of the more saturated types of fats.

Once again it should be emphasized that diet is only part of the story. Physical activity, emotional stress, hormone balance, and many other factors also may

TABLE I

Average Daily Fat Use in United States in 1955 [a]

| Food group | Fat (grams) | Fatty acid (grams) | | | L/S[b] |
|---|---|---|---|---|---|
| | | Saturated | Oleic | Linoleic | |
| Beef, veal, lamb | 22.1 | 11.0 | 8.8 | 0.4 | 0.04 |
| Pork (except bacon, salt pork) | 15.1 | 6.0 | 7.6 | 1.5 | 0.25 |
| Poultry, fish | 4.4 | 1.2 | 1.6 | 0.8 | 0.67 |
| Bacon, salt pork | 13.4 | 5.4 | 6.7 | 1.3 | 0.25 |
| Lard | 9.2 | 3.7 | 4.6 | 0.9 | 0.24 |
| Other shortenings | 9.0 | 2.2 | 5.8 | 0.7 | 0.32 |
| Oils, salad dressings | 9.3 | 1.9 | 2.8 | 4.6 | 2.40 |
| Margarine | 10.4 | 2.6 | 6.4 | 0.8 | 0.31 |
| Butter | 10.6 | 7.0 | 2.9 | 0.4 | 0.06 |
| Milk, cream, ice cream, cheese | 28.0 | 18.5 | 7.6 | 1.0 | 0.05 |
| Eggs | 5.6 | 2.0 | 2.5 | 0.4 | 0.20 |
| Other foods (purchased baked goods, nuts, fruits, vegetables, etc.) | 18.0 | 3.6 | 9.0 | 3.6 | 1.0 |
| Total | 155.1 | 65.1 | 66.3 | 16.4 | 0.25 |
| Per cent of total calories | 43.6 | 18.3 | 18.6 | 4.5 | ..... |

[a] Based on data from individual dietary histories (48).
[b] Weight of linoleic acid divided by weight of saturated acids.

affect serum cholesterol levels. However research of the past 10 years leaves no doubt that dietary fat is one of the most important factors and that its effect is related to its composition. Fats which are rich in polyunsaturated fatty acids, specifically linoleic acid, characteristically act to decrease serum cholesterol levels whereas fats which are rich in the saturated fatty acids raise the cholesterol levels. In fact, several equations have been derived which are reasonably successful in predicting the effect of a given fat mixture from the distribution of the fatty acids (2,30).

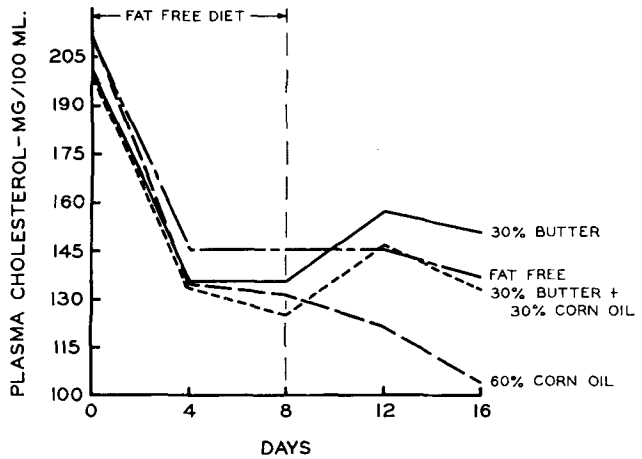


FIG. 5. Average plasma cholesterol levels in groups of subjects receiving liquid formula diets containing either no fat, butter, corn oil, or a mixture of corn oil with butter as the indicated proportion of total calories (6).

This conclusion is based on data from clinical studies under a wide variety of conditions. For example, Figure 5 summarizes some of the results when groups of subjects were given a fat-free liquid formula diet, which was essentially a mixture of skim milk and simple carbohydrates, as the only food and then were given similar diets in which part of the carbohydrate was replaced by butter, corn oil, or a mixture of these fats. There was the expected fall in cholesterol values during the fat-free diet period. When 30% of the calories were derived from butter, serum cholesterol levels rose. However with corn oil the cholesterol levels continued to fall below values during the fat-free period even though corn oil supplied 60% of the calories, *i.e.*, the level fell even though this was a high fat diet. At the high fat level, corn oil was only moderately effective in counteracting the action of butter.

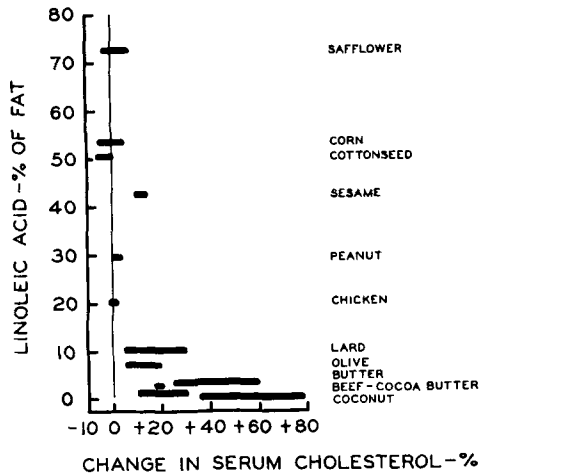


FIG. 6. Effects of dietary fats of various linoleic acid contents on human serum cholesterol values in comparison with the effect of corn oil. Based on studies with liquid formula diets reported by Ahrens *et al.* (1,2).

In Figure 6 are summarized the serum cholesterol responses found in a series of clinical tests in which single fats were fed in liquid formula diets. In this series, corn oil was the standard against which other fats were compared. The length of the bar for corn oil indicates that the cholesterol level in a given subject on the formula diet containing corn oil was highly reproducible within $\pm 5\%$. It is apparent that all oils containing a high proportion of linoleic acid behaved like corn oil. Fats yielding 10% or less of linoleic acid tended to raise cholesterol values. Thus butter, beef tallow, and coconut oil, for example, had effects opposite to that of corn oil.

On the basis of the available data, it is not possible to state how or why dietary fats have such different effects. At present we know only that a diet containing a high ratio of polyunsaturated to saturated fatty acids will decrease serum cholesterol levels in the majority of individuals. This is true whether the diets are liquid formulas exclusively (1,2,4,20), low fat diets to which are added selected oils (3,35,36), or reasonably normal diets containing a variety of cooked foods selected and prepared so as to supply more

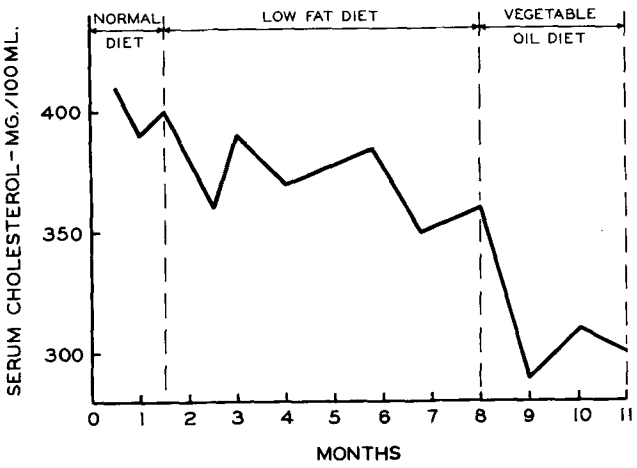


FIG. 7. Comparison of effects on serum cholesterol level of a low fat diet and a vegetable oil diet (11).

polyunsaturated than saturated fatty acids (11,12, 25,32,37,39).

The activities of the fats have been ascribed to total unsaturation (1,2), content of linoleic acid (31,32,42), relative proportions of saturated and polyunsaturated fatty acids (30), and content of sterols and other unsaponifiables (5,6). However none of these is a completely adequate parameter for defining the effect of a fat on serum cholesterol since other components of the diet are also involved. Perhaps it is significant that in some studies linoleate-rich oils have been found to promote the excretion of cholesterol metabolites parallel to the decrease in the concentration of cholesterol circulating in the blood, whereas more saturated fats had the opposite effect (16,21,22,34).

Granted that fats differ in effects on serum cholesterol levels, what does this mean in terms of American food habits? As is evident from Table I, the average American diet provides about 40% of the calories as fat, predominantly of animal origin and oils hardened by hydrogenation. Thus the ratio of linoleic to saturated fatty acids is only about 0.25. In other words, this is just the sort of a diet which has been found in clinical studies to produce high levels of cholesterol in the blood. As Figure 1 showed, this is also the type of diet associated with a high incidence of heart disease.

Since there is some basis for believing that high blood cholesterol levels are related to the development of coronary disease, it obviously follows that we might be wise to consider changing the American diet to decrease the intake of saturated fatty acids or, conversely, to increase the proportion of the polyunsaturates.

The desired effect cannot be achieved merely by adding polyunsaturated vegetable oils to an already abundant fat-rich diet. In such a regimen, any beneficial effects from unsaturates could be completely submerged by the increased caloric intake.

For many years clinicians have been recommending that their hypercholesterolemic patients adopt a low fat diet. In effect, this means cutting out most of the dairy and meat products since these are the main sources of fat in the normal American diet. Low fat diets can be effective in bringing serum cholesterol levels down to normal as was illustrated in Figure 5. Unfortunately such diets are highly restricted and most of us would rebel against eating them day in and day out for the rest of our lives. It is encouraging, therefore, to note that in recent clinical studies serum cholesterol levels have been effectively lowered with diets containing appreciable amounts of fat, provided this is rich in polyunsaturates, particularly linoleates.

A typical example is shown in Figure 7. In this hypercholesterolemic individual, the low fat diet had only a moderate effect on the cholesterol level. However the level was much lower when the diet contained an appreciable amount of a polyunsaturated fat, in this case, cottonseed oil. As would be expected, not every individual shows exactly this response. In some cases, even a modest change in diet can cause a prompt change in serum cholesterol level. In other cases, changes occur more slowly or only after quite extensive diet modification. Apparently the vegetable oil diet is most effective in lowering cholesterol levels in patients having high serum levels of both cholesterol and triglycerides (12).

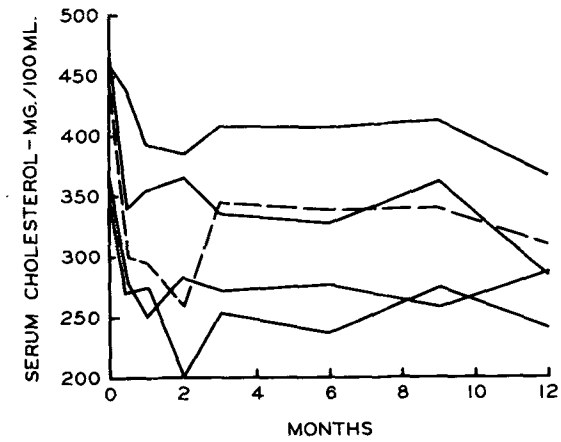


FIG. 8. Effect on serum cholesterol levels of ingesting a vegetable oil diet for one year (35).

Typical variations in individual responses to a corn oil diet are shown in Figure 8. In these cases, consumption of meat and dairy products was restricted so the diet was essentially a low fat one to which was added corn oil as a shortening and salad dressing and in the forms of a tablespread, filled milk, "ice cream" and "cheese." This diet caused a prompt fall in the serum cholesterol level followed by a rebound and establishment of a new level appreciably below that observed when the patient was on a "normal" diet. It is noteworthy that this new level was maintained for at least a year.

In brief, then, clinical studies have proved that the composition of the dietary fat influences the amount of cholesterol circulating in the blood. Polyunsaturated vegetable oils, such as corn, cottonseed, safflower, and soybean, give lower cholesterol levels than do the more saturated fats of animal origin. Fish fats appear to have the same effect as the vegetable oils. The picture with respect to hydrogenated fats is not clear but the indications are that hydrogenation can markedly decrease the effectiveness of an oil (9,29,35). The ratio of polyunsaturated to saturated fatty acids appears to be the critical factor, rather than the absolute amounts of these components in the diet.

In planning diets for clinical studies, the guiding principles have been: replacement of a portion of the more saturated fats with polyunsaturated vegetable oils with a simultaneous reduction in total fat intake, and, if necessary, reduction in total caloric intake (7,11,12,18,25,26,32,37,39,40,45,46). In other words there has been a substitution of one type of fat for another, rather than addition of a new fat and, hence, no drastic change in normal dietary habits. Whether or not this modification will have a desirable effect on the development of coronary heart disease is a matter that can be determined only after many years of study of such diets. Several large studies of this type are currently in progress and, although no definite conclusions are yet possible, the trend seems to be toward a lower incidence of new heart cases in the vegetable oil diet group.

Regardless of what these studies eventually prove, it seems obvious that the tendency in the United States is going to be toward a less rich diet, if only to control obesity. Fats are the most concentrated sources of calories and are added in large quantities during food preparation. It is reasonable to believe that the use of tablespreads, shortenings, and the like could be cut appreciably without nutritional loss and, in fact, with a possible benefit. The present American diet containing 40% of calories from fat is unnecessarily rich and the tendency at present would seem to be toward recommending a fat intake of 25 to 30% of the total calories.

Members of the fats and oils industry know, of course, that there are important differences in shortening value, stability, and so forth, between the ordinary animal fats, the hydrogenated oils, and the natural edible oils. Recently it has become obvious there are also important nutritional differences which can be related to fatty acid composition.

The evidence now indicates that the time is approaching when diet planning will include a balancing of the fatty acids just as we already balance amino acids, vitamins, and minerals to ensure that intakes of all essential ones are adequate. It seems probable that the present American diet has too low a ratio of linoleic to saturated fatty acids and would be improved by a cut in total fat calories with an increased use of the unhydrogenated vegetable oils in place of a portion of the more saturated solid fats.

In particular we are becoming increasingly aware of the unique value of linoleic and other polyunsaturated fatty acids. There now seems little doubt that these are very important constituents of the fats, far too valuable to be destroyed by such processes as hydrogenation. Recognizing the requests of nutritionists for higher linoleate-content shortenings and margarines, many companies in the industry have been working on the development of such products and several are already in the markets.

Because the immediately obvious merits of such new products are related to the cholesterol problem, a recent statement of the Food and Drug Administration (14) deserves comment. This states in part: "The role of cholesterol in heart and artery diseases has not been established. A causal relationship between blood cholesterol levels and these diseases has not been proved. The advisability of making extensive changes in the nature of the dietary fat intake of the people of this country has not been demonstrated."

This is a laudably conservative version of the same conclusions reached in the present discussion. Unfortunately this ruling has been interpreted in many quarters as a) discrediting all the evidence that vegetable oils do differ from animal fats in effects on serum cholesterol levels and b) discounting indications from clinical and epidemiological studies that coronary-prone individuals may be benefited by diets lower in saturated fats. Such an interpretation is not in accord with the facts. Many clinicians are convinced that the evidence is impressive enough to warrant large-scale testing and that, meanwhile, the prudent coronary-prone individual is well advised to make some changes in his diet with respect to fats. The interpretation put on the FDA statement has to some extent tended to hamper development of new high-linoleate food products just at the time when these are most needed for clinical study.

Of course if foods are to have a higher linoleic to saturated fatty acid (L/S) ratio there will be shelf-life problems. The linoleate-rich oils naturally oxidize or become rancid more rapidly than do the more saturated, linoleate-poor fats. It is well known that from a nutritional point of view rancid fats have several undesirable properties. Thus, if a higher L/S ratio is desirable, new ways may have to be found to stabilize foods containing these fats, perhaps with new antioxidants or new packaging materials. As you well know, this is going to be a complex problem because the edible oils vary widely in stability. For example refined soybean oil reverts easily whereas refined corn and cottonseed oils are quite stable, presumably because of their high tocopherol contents.

## Summary

So far as the industry is concerned, the evidence indicating a relationship of dietary fat to heart disease presents some interesting challenges. Undoubtedly it portends a change in the fat consumption pattern toward a lower *per capita* use coupled with a shift from solid fats toward a higher proportion of edible oils. Most important of all, however, is the growing recognition that fats and oils are nutritionally valuable foods, intimately related to health and well-being, and should by no means be regarded merely as a source of calories.

Although there are innumerable factors involved in the etiology of heart disease, dietary fat is an important one and fortunately is one that can be modified in whatever way proves desirable. Because the more saturated types of fats lead to higher serum cholesterol levels than do the polyunsaturated oils, and because cholesterol is somehow involved in the course of atherosclerotic heart disease, clinical tests are now in progress to determine whether prolonged use of a diet rich in these oils will lead to fewer heart attacks than does the usual American diet rich in saturated fats. So far, data are encouraging enough to merit recommendation of the modification in dietary fat to the coronary-prone individual and to justify development of new high-linoleate fat products by the industry.

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# A Nutritive Evaluation of Over-Heated Fats

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*According to the tests used, harmful substances do not* occur in fried foods or in fats used in preparing foods. It is possible to obtain biologically undesirable materials by excessively heating and/or oxidizing fats in the laboratory, but the conditions required for the production of such materials differ greatly from those used in practical cooking or processing of foods. There appears to be no reason to believe that fats are nutritionally damaged when handled by normally-accepted good practice in present-day food preparation.

FATS ARE IMPORTANT and essential in the diet. They are not something we can take or leave alone; they provide energy, supply essential fatty acids, carry fat-soluble vitamins, improve flavors, modify textures, and add satiety values to meals.

Much of their utility depends upon their stability to heat. In frying operations they prevent sticking and transfer heat from hot surfaces to food. The stability of fats at high temperatures (up to 200°C. in some frying operations) invites repeated or continuous use, and questions have been raised concerning the nutritive value and wholesomeness of fats after long usage. Some data in the scientific literature show that undesirable changes occur in fats if they are heated in the laboratory to high temperatures for long periods or if they are subjected to severe oxidizing conditions. Other reports indicate that fats which have been used for prolonged commercial or home cooking retain their nutritive value and remain wholesome.

There are two major reasons why food technologists handling fats should be familiar with this subject. The nutritive values of fats at all stages of processing and use should be known, and there are significant and frequently adverse public relation aspects which must be handled. Publicity problems usually arise from misinterpretations or from unjustified extrapolations of laboratory findings. Even though the implied effects may not be true, headlines such as "The Carcinogenic Action of Heated Fats and Lipoids" (1) cannot be considered in the best interests of the fat industry. The facts must be known in order to understand the problems and to combat misleading reports or inferences.

It is not our intention to review this subject exhaustively. Instead we plan to consider published and unpublished research findings which indicate typical changes that can occur in food fats during laboratory treatments or cooking procedures and to contrast the findings with those obtained when food fats are tested. Noncritical review of the scientific literature relating to heated fats can lead to very erroneous conclusions since reports show that it is possible to mistreat fats experimentally with sufficient heat and/or oxygen to cause, when the abused fat is fed to test animals, retarded growth, poor feed efficiencies, rough, greasy matted coats, diarrhea, starvation, enlarged livers and kidneys, abnormal fat depots, impaired enzymatic functions, abnormal water metabolism, papillomas and other growth formations, and shortened life spans. In extreme cases animals may die in a few days after severely abused fats have been fed. There is thus no question whatsoever that fat can be damaged by purposeful abuse. The critical question is: "are fats damaged during processing or cooking operations?" To answer that question we must examine some of the conditions which produce the effects listed above. They may have been