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The Relationship of Dietary Fat to Atherosclerotic Disease

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No more controversial medical question exists than that of the relationship of diet to atherosclerosis in general, and to coronary atherosclerosis in particular. The following points of view exist:

- 1. In humans, diet bears no relationship to coronary atherosclerosis.
- 2. Dietary factors are the *only* factors of any importance in the etiology of coronary atherosclerosis in humans.
- 3. Dietary factors, in conjunction with hereditary factors and other environmental entities play an important role in the etiology of human atherosclerosis
- 4. Fats are fats and all of them are bad, and therefore, insofar as possible, should be eliminated from the diet if one wishes to keep his coronary arteries in good condition.
- 5. There are major differences in dietary fats, with respect to the pathogenesis of coronary atherosclerosis: Specifically, saturated fats are on the wrong side of the ledger, and the polyunsaturated fats are on the right side of the ledger. (Of the investigators who subscribe to this concept, there are

• Atherosclerosis is the Number One public health problem. Many factors have been implicated in the pathogenesis of this disease. Prominent among these factors is the amount and kind of fat in the diet. The evidence now appears to be conclusive that sufficient quantities of polyunsaturated fat in the diet, with proportional decrease in saturated fat, will result in major decrease in blood lipid. Some evidence indicates that such blood lipid lowering produces a desirable effect upon existing atherosclerosis. Much additional time and work will be required to clarify the prophylactic and therapeutic value of this type of dietary approach.

those who particularly emphasize the inclusion of the polyunsaturated fats, and those who particularly emphasize the elimination of saturated fats.)

- 6. A variety of other dietary factors have been included in a plus-or-minus classification.
- 7. With almost no exception, there is agreement that a diet which results in obesity very significantly increases the tendency to coronary heart disease. This is perhaps the *only* area in which full agreement exists.

One of the major factors that has retarded resolution of the divergent points of view noted above is the existence of pronounced species differences. The same diet administered to a rat and to a mouse can produce very different blood lipid levels and dif-

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ferent patterns of deposition of lipid in various locations. Even greater differences exist between rabbits and rats. Who is to say which of these species will yield information which is applicable to humans?

If, then, the problem fundamentally is one which has to be solved by human investigation, how does one go about obtaining such a solution? Obviously, it is impossible to obtain serial biopsy of specimens of blood vessels in a human subject. Almost the same statement applies to the setting up of a highly acceptable controlled study in which perhaps 1,000 subjects would be given a diet which is regarded as being highly undesirable, and another 1,000 subjects a diet which is regarded as having high "anticoronary" value. Probably the closest approach to such a study is to follow one group which by choice consumes a diet that the investigator regards as being somewhat undesirable, and a second group composed of "motivated persons" who by one means or another can be persuaded to follow a diet which he regards as being in a favorable category. This will receive further consideration (see below).

If one accepts the concept that *the* definitive study is yet to be done, the obvious question arises: How well grounded are any of the preceding conclusions in this field? The answer appears to be: They are grounded upon divergent animal observations which may bear no relationship whatever to the human problem, and/or upon human observations, all of which are more or less fallible.

All human studies which have any possible meaning rest upon one basic assumption, namely, that in humans the level of certain blood lipids bears a definite relationship to blood vessel disease. There is now general acceptance of the statement that of the various blood lipid entities which have been studied, the plasma cholesterol is at least as meaningful as any of the lipo-proteins that are more difficult to measure, possibly more meaningful. On the basis of many studies there appears to be no question that the plasma cholesterol tends to increase with the age of the subject and that the plasma cholesterol level, statistically, is higher in groups with known atherosclerosis than in groups of persons with no detectable atherosclerosis. Unfortunately, there are so many individual exceptions to these statements that the cholesterol level as a dependable index in a given individual leaves much to be desired.

Admitting all the above shortcomings, the point of view of most careful workers in the field at present is that, other things being equal, if by dietary or other means one is able to achieve a low level of plasma cholesterol, this is desirable as compared with a relatively or absolutely high level of plasma cholesterol. Accepting this point of view for the

present, are there dietary measures which predictably will produce and maintain a relatively or absolutely normal level of blood cholesterol. If so, what is the upper limit of normal?

Taking the last question first, if one uses the Sperry-Schoenheimer method, or one of its modifications, for determination of plasma cholesterol, it is the opinion of the authors that any cholesterol level above 220 mg. per cubic centimeter is to be regarded as undesirable, and, if possible, a level of less than 180 mg. is to be achieved. This statement is based upon multiple observations in young normal adults and in older persons with no evidence of vascular disease. Can this be brought about by any reasonable means? The answer appears to be in the affirmative. The principles involved appear to be as follows:

- 1. Avoidance of obesity, and the use of appropriate weight reduction procedures if obesity exists.
- 2. The inclusion in the diet of adequate amounts of polyunsaturated fats.
- 3. The avoidance of excessive amounts of saturated fats in the diet.
- 4. The use of a diet which is nutritionally sound in other respects, with reference to adequate quality and quantity of protein and inclusion of essential vitamins and minerals.

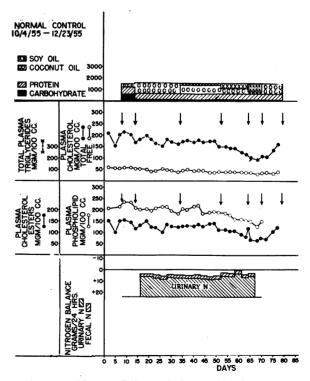


Chart 1.—Plasma cholesterol decreased when 50 per cent of the predominantly saturated fat was replaced by an equal quantity of predominantly polyunsaturated fat.

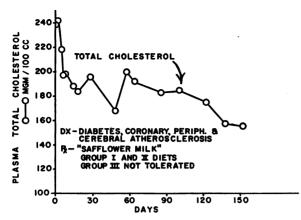


Chart 2.—Effects of a mixed diet containing large amounts of polyunsaturated fat in an elderly male diabetic with extensive vascular disease. Inclusion of fish and fowl is tolerated (Group II diet). Inclusion of meat and eggs in moderate amounts (Group III diet) resulted in prompt elevation of serum lipids. Safflower milk is "Saflac," made by Carnation Co.—a preparation in which butter fat has been replaced by safflower oil (75 per cent linoleic acid). Group I, II and III diets are described elsewhere.²

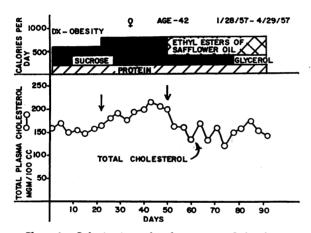


Chart 3.—Substitution of polyunsaturated fat for carbohydrate produces a fall in plasma cholesterol.

5. Adherence to a common-sense program, which includes adequate exercise, avoidance of excessive anxiety, avoidance of excessive use of tobacco and alcohol and avoidance of excessive fatigue. These nondietary factors will not be commented upon further, since this paper is concerned with diet.

What constitutes an adequate amount of polyunsaturated fat and an excessive amount of saturated fat? A single one-sentence answer to this pair of questions is not possible. In a middle-aged person with no evidence of atherosclerotic involvement, a ratio of 50 per cent predominantly unsaturated fat and 50 per cent predominantly saturated fat appears to be compatible with normal levels of plasma cholesterol (see Chart 1). In young normal persons the amount of polyunsaturated fat in relation to saturated fat may be very much less than this. In such persons, ten to twenty grams daily of linoleic acid may be sufficient to "balance" a relatively large quantity of saturated fats.¹

In a person with clinically obvious atherosclerosis, it may be necessary to completely eliminate saturated fat from the diet for several months and to include in the diet very large quantities of polyunsaturated fats. When the desired level of blood cholesterol has been achieved and maintained for a period of perhaps two months, the diet may then be liberalized. Observations in a patient in which this was done are shown in Chart 2. Descriptions of "therapeutic and prophylactic essential fatty acid diets" are published elsewhere.2 In the construction of diets it is to be kept in mind that the use of excessive amounts of concentrated carbohydrate may produce significant elevation of cholesterol as compared with a diet in which polyunsaturated fat is substituted for a portion of this carbohydrate (Chart 3).

What about acceptability of such diets? The answer to this appears to be that any diet which calls for a significant change in one's eating habits is, psychologically speaking, for a time relatively unacceptable. The dietary problem in the case of patients with resistant hypercholesterolemia appears to be essentially of the same type that one faces in dealing with a diabetic patient. Proper education and cooperation are the only answers. The problem is not one that can be solved by instructing a patient to add a tablespoon or two of unsaturated fat daily to his present diet.

What is the evidence that a diet which will achieve normal cholesterolemia will exert either a protective or a therapeutic effect insofar as the atherosclerotic process is concerned in man?

Here the answers are much less absolute than in the case of the blood lipids. In terms of our own experience, it is only possible to say that in a relatively small group of patients with partially occlusive peripheral atherosclerosis, either arrest of progression or actual improvement, as evidenced by functional capacity, has been observed. Several years must elapse before meaningful conclusions can be drawn.

Coincidental with this, work is under way designed to carry out a statistically adequate study of a group of men between the ages of 45 and 55 who do not have clinically obvious atherosclerosis at the time of admission to the study. These persons will be divided into four groups with respect to diet.

Group A will consume a low fat diet.

Group B will consume a low fat diet supplemented with relatively small amounts of unsaturated fat.

Group C will consume a relatively high fat diet with a preponderance of animal fat—essentially an average American diet.

Group D will consume a relatively high fat diet with preponderance of unsaturated fat.

Determination of blood lipids will be done periodically throughout a ten-year year. Repeated clinical evaluation will detect the occurrence of coronary heart disease or other manifestations of atheroscler-

osis. It is believed that this study will supply some of the answers that are so vitally needed.

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