REDUCTION OF HYPERCHOLESTEROLEMIA BY *HIGH*-FAT DIET PLUS SOYBEAN PHOSPHOLIPIDS

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SOME INVESTIGATORS advise a low-fat diet to relieve hypercholesterolemia (48, 58, 38). We gave our hypercholesterolemic patients the opposite diet, one *high* in fat and cholesterol, plus soybean phospholipids. This paper presents 91 such cases and 31 controls: the blood cholesterol came down notably in the treated cases, but not in the controls.

COMPOSITION OF DIET

The average daily diet contained over 4,000 calories. About 30% to 40% of this was fat, mostly animal fat. The patients were advised to eat internal organs including at least a tablespoon of raw liver and one of raw brains daily; these foods are rich in cholesterol. The daily diet also included a teaspoon of a B-vitamin concentrate extracted from rice-bran. The patients took one teaspoon of soy bean phospholipids with each meal. Soy bean phospholipids include a mixture of 29% lecithin which contains choline, 40% inositol phospholipids, and 31% cephalin (56). This mixture, plus a low concentration of phytosterols, is used widely in the food industry under the name of "lecithin," and we shall refer to it as such here.

MODE OF ACTION

Lecithin affects at least two organs that manufacture cholesterol and one that excretes it. Lecithin relieves lipid dyscrasias of the skin* (29) and of the liver (44, 39, 33, 34) which two organs manufacture more cholesterol than any of the other organs in the body (57). We can get an idea of the amount of cholesterol the body makes from the finding that the liver of the rat synthesizes enough cholesterol to meet all the requirements of that animal (7), and the skin, testes, and kidneys also make it (57). The arteries synthesize cholesterol (11), but we do not know what lecithin does directly to arterial metabolism. The liver excretes cholesterol in the bile, and biliary obstruction causes hypercholesterolemia in man, dog, and rat (32, 30, 19, 20, 10, 41, 9). Byers, Friedman, and Michaels presented evidence that the liver controls the level of free plasma cholesterol (9). So, dietary lecithin may act through the liver to reduce blood cholesterol.

DIETARY FAT

Other investigators have shown that a *high*-fat diet plus proteins and B-vitamins will relieve fatty degeneration of the liver (12, 37, 5, 6, 22). This paper suggests that a similar diet plus lecithin will relieve hypercholesterolemia. A high-fat diet will not raise the blood cholesterol (62, 61, 40, 60, 59, 26); it will not cause fatty degeneration of the liver if the diet contains certain nutritional factors, including proteins (37), especially methionine (6, 22), and lecithin (1, 44, 39, 33, 34); and it will not interfere with liver regeneration (54). On the other hand, a fat-*free* diet will cause fatty degeneration of the liver by taking the unsaturated fats out of the diet; and one may cure it by returning the unsaturated fats to the diet (17). Fats, then, help to correct lipid dyscrasias.

FAT-SOLUBLE VITAMINS

Fat in the diet also helps the intestines absorb fat-soluble vitamins A, D, E, and K (4, 63, 2, 35). These vitamins help prevent lipid dyscrasias. Vitamin E, which is present in animal fat (51), prevents a type of fatty infiltration of the aorta (42). It also affects blood cholesterol beneficially: One measure of the tendency to atherosclerosis is the serum phospholipid : cholesterol ratio. For safety the concentration of phospholipid should be greater than the concentration of cholesterol (48). Vitamin E raises the concentration of phospholipid over that of cholesterol in the chick; adding cholesterol to the diet causes the opposite effect without raising the absolute amount of cholesterol in the serum (13).

Vitamin E has other functions in preventing lipid dyscrasias. It helps relieve unsaturated fatty acid deficiencies in rats (53). It acts as an anti-oxidant to protect unsaturated carbon bonds in fats during their mobilization, metabolism, and storage within tissue cells (36, 43). In rats and rabbits a deficiency of vitamin E increases the cholesterol and total lipid content of the muscle and brain (31, 47). A deficiency of vitamin A acts similarly. In lipid degeneration of peripheral acini of the liver, there is a deficiency of vitamin A in the peripheral areas of degeneration, but a normal amount in the inner normal regions (55).

Fat-soluble vitamins were present equally in the diets of the lecithin-treated cases and of the controls presented here. So, while these vitamins may have aided the treatment, they were not responsible for the differences noted.

RESULTS

72 of the 91 hypercholesterolemic patients who took lecithin and fat benefitted: their blood cholesterols dropped 15 mg.% or more. In the remaining 19 patients, the cholesterol stayed the same ± 15 mg.% in 13 patients and increased 15 mg.% or more in 6. The control patients were also on a high-fat, high cholesterol diet, but they received no lecithin. As the graph shows, approximately the same number of these patients had an increase in cholesterol as had a decrease. The high-fat diet did not affect the blood cholesterol level. Adding lecithin to the diet reduced the cholesterol in 79% of the patients so treated.

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*Several investigators have shown that psoriasis is a lipid dyscrasia (40A, 29A, 29B) and lecithin may relieve this disease (29).

AGES OF LECITHIN TREATED PATIENTS

All age groups were represented in these cases. The exact distribution was as follows:

Age of Patients	*Number of patients in this group*
0-19 years	28
20-39 years	13
40-59 years	38
60 years	9
age not known	3
	91 patients

In six of the treated cases the cholesterol rose 15 mg. or more. The age of these patients was: 7, 33, 35, 55, 65, 78 years, respectively. It might appear that lecithin succeeds regardless of age, and fails regardless of age. We believe some failures occurred because the patients did not take their lecithin regularly as instructed.

LENGTH OF TREATMENT

In a few cases the blood cholesterol dropped 15 mg. or more after only two or three months of treatment. Usually it takes longer. The cases reported here were treated as follows:

Length of Treatment	*Number of Patients so treated*
0- 6 months	26
7-12 months	31
13-24 months	28
25 months	6
	91 patients

In many cases the cholesterol had fallen 15 mg. or more in less than six months. We continued to treat these patients because the cholesterol had not yet come down to normal.

The six patients whose blood cholesterol rose 15 mg. or more were treated for the following lengths of time: 4, 7, 9, 12, 12, 45 months, respectively. This might suggest that when lecithin fails, it fails regardless of how long it is prescribed.

We also checked the phospholipid : cholesterol ratio in 4 patients who had suffered from marked hypercholesterolemia complicated by other evidences of lipid dyscrasia: severe eczema in 3 cases and cirrhosis of the liver in the fourth. After treatment the phospholipid : cholesterol ratio was greater than unity in all cases, indicating that these patients were protected against atherosclerosis according to the criterion (48).

We sought to help these patients use lipids to their advantage and avoid at least one disease of bad lipid metabolism: hypercholesterolemia. Further investigation is needed to determine whether the program presented here protects against other lipid dyscrasias, such as atherosclerosis.

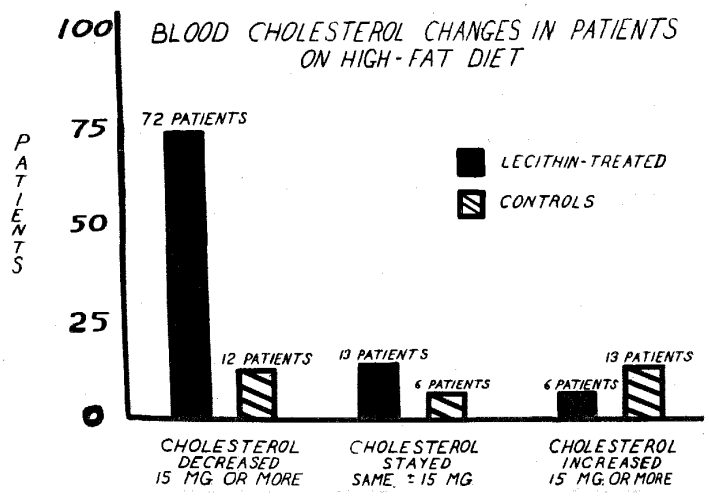
One group of investigators has found a special form of cholesterol that may be important in this respect: it is the lipoprotein molecule of large size and low density which can be detected with the ultra-centrifuge (28). Gofman, Jones, Lindgren et al report that lipoprotein molecules do not vary with the total serum cholesterol level; and they are at higher concentrations in cases of coronary thrombosis, diabetes mellitus, nephrosis, and hypothyroidism (28): diseases commonly accompanied by atherosclerosis (8, 27, 64, 15, 52, 16). But other investigators report that hypercholesterolemia also accompanies some of these diseases (8, 3, 50, 45, 14, 27, 64, 15, 52, 46, 49, 16).

In any case, the question is how to reduce the concentration of cholesterol and lipoprotein molecules in the blood. Several workers have reported that they reduced the blood cholesterol by giving their patients a low-fat diet (40, 58, 38, 18, 21). Gofman, Jones, Lindgren, et al, reported that they reduced the concentration of lipoprotein molecules in the blood by treating their patients similarly (28). This paper presents a series of cases in which we reduced the blood cholesterol by giving the patients a *high*-fat diet plus lecithin. We hope to find whether or not this treatment will also reduce the concentration of lipoprotein molecules in the blood.

SUMMARY

1. 122 hypercholesterolemic patients followed a *high*-fat diet. 91 of them took a teaspoon of soy bean phospholipids with each meal. The remaining 31 served as controls.

2. The blood cholesterol came down in 79% of the patients who took phospholipids. In the controls there was no tendency for the cholesterol to either rise or fall.



3. This indicates that it may be possible to correct hypercholesterolemia while the patient eats a high-fat, high-cholesterol diet.

4. Evidence is reviewed which suggests that the fat may aid in correcting this lipid dyscrasia.

5. This therapy tends to reduce hypercholesterolemia by promoting healthy lipid metabolism.

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