AN ABSTRACT OF THE THESIS OF

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Title: LECITHIN SUPPLEMENTATION AND PLASMA PHOSPHOLIPIDS

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Dr. Elisabeth Yearick

The effect of supplementary lecithin on plasma lipids was studied. Thirty-one men between the ages of 38 and 56 were given 7.2 gm of supplemental lecithin per day in the form of Soya Lecithin. Supplementation lasted for a period of seven and a half to eight weeks. Blood was analyzed both before and after supplementation in order to determine total plasma phospholipid, triglyceride, cholesterol and total lipid levels. Supplementary information was obtained about smoking habits, activity levels and percent of desirable weight for each subject. The phospholipid profile of randomly selected samples was also determined in order to find any possible changes in the individual phospholipids.

There was no significant change in either phospholipid, triglyceride, total lipids, or the individual phospholipids. However, there was a significant increase in cholesterol. A positive correlation between plasma triglycerides and both

cigarette smoking and the percent of desirable weight was discovered. There was also a negative correlation between activity and plasma triglycerides.

Lecithin Supplementation and Plasma Phospholipids

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INTRODUCTION

The close association between atherosclerosis and hypercholesterolemia has caused a great deal of interest in finding a means to lower the plasma cholesterol and to maintain it within an acceptable range. One product which is currently reported by popular writers to have such an effect is lecithin. Dr. Lester Morrison (1971), a strong proponent of lecithin, has declared that "lecithin is one of the most important nutritional supplements developed in the last fifty years." Dr. Roger Williams (1971), another popular writer, has stated that lecithin has the power of "removing atherosclerosis."

In spite of the vast amount of publicity that lecithin has received, there is a dearth of scientific data on the effects of lecithin taken as a dietary supplement. What little research is reported has been done using doses which either exceed or fall below those that most people are taking.

Although there is some evidence that lecithin may inhibit the absorption of cholesterol in the intestine, most of the theories for the action of lecithin deal with its effect after it has been absorbed. It has been postulated that lecithin may act by increasing the total phospholipid

concentration of plasma or by changing the concentrations of the individual phospholipids.

Because of the two possible modes of action of lecithin, this study was undertaken in order to determine the effect of lecithin supplementation on: 1) the total plasma phospholipids, and 2) the phospholipid "profile". It was part of a larger group effort in which total plasma lipid, cholesterol, and triglyceride concentration, as well as dietary intake, activity level and cigarette smoking of the subjects were determined.

REVIEW OF LITERATURE

Blood Lipid Values in Healthy Men

The major lipid constituents of plasma (phospholipids, cholesterol and triglycerides) have been measured in many different populations in a wide variety of conditions. As may be seen in Table 1, the reported values for groups of healthy men vary greatly. This variation is due, in part, to differences in analytical methods. Nevertheless, certain factors which influence plasma lipid concentrations of healthy individuals have been well demonstrated.

Age has a notable effect on plasma lipids. In adult males, concentrations of cholesterol, phospholipid, and triglyceride increase with age until about 55 years (Adlersberg et al., 1956; Carlson, 1960; Berlin, Oldfelt and Vikrot, 1969b; Bottiger, 1973). After this age the levels remain constant or may even decrease slightly.

Plasma lipids are also affected by exercise. Hoffman, Nelson and Goss (1967) studied men between the ages of 45 and 54 with sedentary occupations. Those having a regular program of exercise, even if the exercise period was short, had lower levels of total lipids, cholesterol, and triglycerides. Phospholipids were not significantly affected. Lopez et al. (1974) found values very similar to those of Hoffman et al. (1967). They did not measure phospholipids but did note a significant increase in the alpha

Table 1. Lipids in plasma or serum of apparently healthy males.

Investigator	Age	Phospho-lipids (mg/100 ml)	Cholesterol free	Cholesterol total	Triglycer-ides (mg/100 ml)	Total Lipids (mg/100 ml)
Ahrens and Kunkel (1949)		219	56	185	86.6	636
Sperry and Brand (1955)		230	46	166	--	732
Adlersberg (1956)	48-52	288		239	--	
Carlson (1960)	46-55	262		258	95.2	
Phillips (1962)	Adults	258	51	195	59.9	
Hoffman, Nelson and Goss (1967)	40-55, low exercise	200		224	113.9	660
	40-55, high exercise	197		213	100.7	628
Phillips and Dodge (1967)		222	61	181	70.3	
Berlin, Oldfelt and Vikrot (1969a)	53-87, men and women	286	85	283	86.6	
Berlin, Oldfelt and Vikrot (1969b)	42-60	257	84	266	94.7	
Kunz and Strumvoll (1971)	Adults	218		215	91	
MacDonald (1972)	18-21	187		181	103.7	
Bottiger (1973)	48-52	290		259	114	
Billimoria et al. (1975)	30-60, non-smokers	215	44	215	82	
	30-60, smokers	241	31	236	134	

lipoproteins with exercise. Gertler (1967) obtained slightly different results in his study of young Marine corps recruits. At the end of basic training, there was a significant decrease in plasma triglycerides and an increase in phospholipids. Plasma cholesterol remained essentially unchanged, although the men consumed an average of 4500 Kcal, about 45 percent from fat, much of which was of animal origin.

Because of the high correlation between cigarette smoking and deaths due to coronary heart disease, much research has been done to determine the effect of smoking on blood lipids. Although Page, Lewis and Moinuddin (1959) reported no significant immediate change in serum lipids due to cigarette smoking, the effects of chronic use are well documented. Elevated cholesterols have been reported in dogs due to sustained nicotine administration (Kershbaum and Bellet, 1964) and in smokers as compared to nonsmokers (Karvonen et al., 1959). A recent study (Billimoria et al., 1975) has shown that, compared with non-smokers, men who smoked twenty cigarettes or more per day had higher phospholipids, triglycerides, and esterified cholesterol. It also appeared that moderate smoking exerted an intermediate effect, depending on the number of cigarettes smoked.

It is well known that sex is another important factor influencing lipid levels. Plasma cholesterol and triglycerides are significantly lower in women from the ages of

20 to 35 than in men of the same age (Berlin et al., 1969b). Phospholipids have been reported to be the same (Berlin et al., 1969b; Bottiger, 1973) or lower (MacDonald, 1972) in young women than in young men. Cholesterol and phospholipid levels of women, aged 45-63, tend to increase and may even surpass those of men in the same age range (Adlersberg et al., 1956; Berlin et al., 1969b; Bottiger, 1973).

Blood lipids are greatly affected by diet. Dietary fats have an especially large influence. Dietary cholesterol is a lipid component which is positively correlated with plasma cholesterol levels (Erickson et al., 1964). The findings in humans have been corroborated and extended by animal studies. For instance, a study of rats has shown that, as dietary cholesterol is increased, it is the percentage of cholesterol esters that becomes elevated (Fraser, 1974). Stange, Agostini and Papenberg (1975), studying rabbits, found that a diet high in cholesterol, or high in cholesterol and saturated fat, resulted in a marked increase in the cholesterol of all lipoprotein fractions.

The amount of polyunsaturation in the dietary fats also has an important influence on plasma lipids. Kinsell et al. (1953) showed that polyunsaturates are capable of producing a reduction in serum cholesterol. This finding was confirmed by Wilson et al. (1971) in a study of 59 nonobese men who adhered for six months to the American Heart Association's fat controlled diet, which has a high ratio of

polyunsaturated to saturated fats (P/S ratio). At the end of the study, the following decreases in mean plasma lipids were observed: cholesterol 9.6 percent, triglyceride 11.7 percent, pre-β lipoprotein 5.7 percent, β lipoprotein 15.6 percent, α lipoportein 6.2 percent. Chiat et al. (1974) found a 35 percent decrease in mean serum triglyceride and a 16 percent reduction in cholesterol when unsaturated fat was isocalorically substituted for saturated fat in both hyperlipidemic and normal men. They found some evidence to indicate that polyunsaturated fats may act, at least in part, by decreasing the rate of secretion of very low density lipoprotein (VLDL) triglycerides into plasma. Stange et al. (1975) found that when cholesterol was administered with a diet high in saturated fat, the electrophoretic mobility of the VLDL and low density lipoproteins (LDL) was reduced compared to that of animals on diets high in cholesterol and polyunsaturated fats, or on control diets. Also, the diet high in saturated fats was associated with an abnormal stacking of the VLDL, LDL and high density lipoproteins (HDL). The animals on the control and the high-polyunsaturate diets showed no such abnormal pattern. The authors suggested that these findings may be related to the lower incidence of atherosclerosis associated with the polyunsaturated diet.

The effect of dietary carbohydrate on plasma lipids is more controversial. Anderson (1967) found that increasing

the carbohydrate in the diet led to significantly higher serum triglycerides. It appears that not only the quantity, but also the type of carbohydrate in the diet is important. Groen et al. (1966) and Grande (1967) both found that when sucrose or sucrose plus lactose was substituted for the more complex carbohydrates in the diet the levels of serum cholesterol increased significantly. Yudkin and Roddy (1964) have, moreover, found a correlation between dietary sucrose and the incidence of occlusive atherosclerotic disease. However, other investigators have not been able to duplicate their results (Burns et al., 1969; Howell and Wilson, 1969).

The interaction of dietary fats and carbohydrates may also be an important factor. MacDonald (1972) studied six different types of dietary patterns. The plasma lipid response to certain types of carbohydrate varied, depending on whether the diet was high in saturated or polyunsaturated fat.

Blood Lipids and the Risk of Atherosclerotic Heart Disease

Many efforts have been made to determine a correlation between certain blood lipid parameters and the risk of atherosclerotic heart disease. Although the results of these studies remain somewhat controversial, there seem to be some trends worth noting.

High plasma or serum cholesterol is the lipid abnormality most commonly cited as predictive of coronary heart disease. In the Framingham study, Dawber, Moore and Mann (1957) found that a cholesterol above 260 mg/100 ml plasma was associated with a greater incidence of atherosclerotic heart disease. A later analysis in the Framingham study showed a significant correlation between atherosclerotic heart disease and high levels of each of the following components of plasma: cholesterol, phospholipids and the S_f0-12 fraction of the lipoproteins. Brown, Kinch and Doyle (1965) found a close correlation between cholesterol concentrations and the incidence of ischemic heart disease (IHD). Although there also seemed to be a high correlation between high triglycerides and IHD, the strong relation between triglyceride and cholesterol levels made a simple correlation difficult to determine.

Bottcher (1967) is representative of a number of investigators who feel that plasma cholesterol and atheroschlerotic heart disease may not necessarily be correlated. Indeed, a recent study by Kunz and Strumvoll (1971) showed the mean cholesterol levels of a healthy control group and persons suffering from peripheral occlusive arterial disease (POAD) to be essentially the same. Albrink, Meigs and Man (1961) found a much higher correlation of the incidence of coronary heart disease with high triglycerides than with high cholesterol. They also found a poor correlation

between cholesterol and triglyceride values in the plasma. Hatch et al. (1966) and Brown and Doyle (1967) confirmed the correlation of high triglycerides with cardiovascular disease.

Plasma Phospholipids

The plasma phospholipids are composed almost entirely of six types of phosphatides. The major plasma phosphatide is phosphatidyl choline (PC), comprising about 65-75 percent of the total phospholipids. Next in concentration are sphingomyelin (SM) at 15-30 percent and lysolecithin (LPC) at 3-14 percent. Phosphatidyl ethanolamine (PE), phosphatidyl serine (PS), and phosphatidyl inositol (PI) are minor phospholipid constituents of plasma (Table 2).

Age and sex seem to affect the distribution of the fractions. There is a greater concentration of LPC in young men (less than 37 years of age), than in older men or in females of any age (Berlin et al., 1969b). Bottiger (1973) found a significantly higher level of LPC in men of all ages than in women. The absolute amount of LPC in the plasma increased with age, but the proportion did not.

Spritz and Mishkel (1969) have shown that the feeding of a diet containing 40 percent of the calories as polyunsaturated fats caused a change in the fatty acid composition of the plasma lipids. The relative amounts of PC, PE, SM, and LPC were not affected by the unsaturated diet, but

Table 2. Plasma or serum phospholipid[a] distribution in health and disease (percent of lipid phosphorus).

Investigator	Comment	PC	LPC	SM	PS	PI	PE
Phillips (1962)		68.2	7.7	19.0	-	-	5.0
Doizake and Zieve (1963)		64.8	10.8	21.9	-	-	2.4
Robinson and Phillips (1963)		65	14.5	14.7	-	-	5.8
Phillips and Dodge (1967)		70.4	6.2	17.4	1.9	-	3.6
Berlin et al. (1969a)	normals	71.4	5.9	21.3	-	-	1.4
	acute myocardial infarction	72.1	3.7	22.9	-	-	1.2
Berlin et al. (1969b)		71.0	6.5	21.5	-	-	1.6
Spritz and Mishkel (1969)	saturated fat diet	66.4	2.6	28.7	-	-	2.4
	unsaturated fat diet	64.8	2.6	30.2	-	-	2.4
Kunz, Matt and Hacki (1970)	normals	67.8	5.8	21.3	0.4	0.8	2.1
	hyperlipemic (HL)	74.7	3.3	15.0	0.6	1.4	3.0
	increasing lipids (HL)	75.4	3.5	13.2	0.8	1.9	3.3
	decreasing lipids (HL)	73.9	3.0	17.1	0.5	1.2	2.6
Kunz and Strumvoll (1971)	normals	68.1	5.5	18.9	0.5	1.1	2.4
	peripheral occlusive arterial disease	70.4	4.2	16.7	0.6	1.2	3.3
	hypercholesterolemic, healthy	68.1	5.3	17.2	0.5	1.0	2.9
Vikrot, Berlin and Oldfelt (1971)	normals	70.7	6.3	21.5	-	-	1.5
	hypercholesterolemic	68.1	5.2	25.2	-	-	1.4

[a]PC = Phosphatidyl choline, LPC=lysolecithin, SM = sphingomyelin, PS = phosphatidyl serine, PI = phosphatidyl inositol, PE = phosphatidyl ethanolamine.

the molecular species of PC was significantly altered, becoming more highly unsaturated. The ratio of phospholipid: protein and cholesterol:protein in the low density lipoproteins was decreased during the unsaturated feedings. Because the polyunsaturated fatty acids occupy a greater area than the saturated acids the authors hypothesized that the lipid lowering by the polyunsaturates was due, at least in part to their spatial configuration. On the other hand, Nye (1969) found significant changes in phospholipid concentrations, especially in the SM and PC fractions, when the quantities of fat, protein, ethanol, and calories in the diet were changed.

Changes in the phospholipid profile have been observed in patients with cardiovascular disease (Table 3). LPC was lowered in patients with hyperlipoproteinemia (Kunz, Matt and Hacki, 1970), in subjects with hypercholesterolemia and peripheral occlusive arterial disease (Kunz and Strumvoll, 1971), and in patients suffering from atherosclerotic diseases (Gillett and Besterman, 1975). Studies by Kunz and Strumvoll (1971) and Nothman and Proger (1962) have shown significantly increased levels of PE in atherosclerotic patients. Kunz and Strumvoll (1971) were able to show that elevated PE was more closely related to the incidence of peripheral occlusive arterial disease (POAD) than any other lipid parameter.

It is interesting to note that Mohan and Chakravarti (1975) found a significant difference in the phospholipid profile of Rhesus monkeys exhibiting spontaneous atherosclerosis and that of animals suffering cholesterol-induced atherosclerosis. Those with spontaneous atherosclerosis had increased PC with decreased LPC and SM+LPE. On the other hand, those with cholesterol-induced atherosclerosis were found to have elevated PE and decreased SM+LPE. This difference prompted the authors to suggest that different mechanisms were responsible for the two types of atherosclerosis.

Lecithin

The term "phospholipids" encompasses a large number of compounds. The phosphoglycerides (Figure 1) are the largest class of these phospholipids, and are derivatives of phosphatidic acid. In the phosphoglycerides, the phosphoric acid is esterified to a nitrogenous compound or inositol (Figure 2) to form the polar head group of the molecule. The sphingolipids make up another important class of phospholipids. They are of the general formula shown in Figure 3. The most common sphingolipid is sphingomyelin, in which choline forms the polar head group.

Historically, the term lecithin had has two meanings. It is most properly used to refer to a mixture of phospholipids which are soluble in most organic solvents, but not

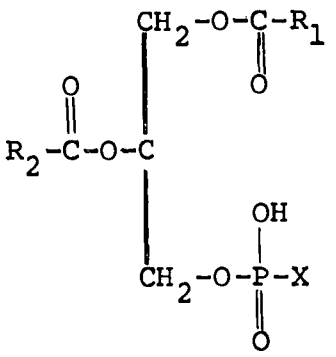

Figure 1. General formula for a phosphoglyceride.

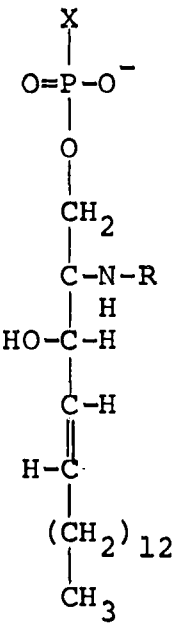

Figure 3. General formula for a sphingolipid.

Ethanolamine: $HOCH_2CH_2NH_2$

Choline: $HOCH_2CH_2\overset{+}{N}(CH_3)_3$

Serine $HOCH_2CHNH_2COOH$

Inositol:

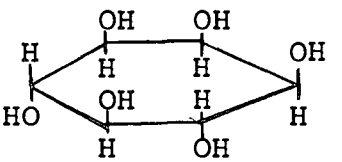

Figure 2. Substituents contributing their polar X groups in the major phosphoglycerides and sphingomyelin.

in acetone. The term has been used interchangeably with phosphatidyl choline (PC). However, throughout this paper PC will be used to denote phosphatidyl choline, while the word "lecithin" will be used to describe the mixture of phosphorus-containing lipids.

Lecithin derived from soybeans is widely produced and used commercially. Such lecithin, when marketed, must contain at least fifty percent acetone-insoluble material. It may also contain various amounts of other substances such as triglycerides, fatty acids, and carbohydrates. According to the Food Chemicals Codex, the amounts and types of these substances present will vary with the method of fractionation used (Food Protection Committee, 1966). The molecular species of phosphatides present in soy lecithin is also quite variable (Table 3).

Table 3. The phospholipid content of commercially produced soy lecithin (as percent of lipid).

Investigator	PC	PI	PE	Unknown	PA
Stanley (1951)	21	20	8	11	
Morrison (1958)[a]	29.5	31.6	29.5		
Erdahl et al. (1973)	29.0	15.1	23.5	7.9	7.0

[a]Method of reporting not specified, may be as percent of recovered phosphorus.

Lecithin as a Dietary Supplement

Soya lecithin has always been valuable commercially as an emulsifying agent. However, at various times it has also been a popular dietary supplement. Currently it is receiving a great deal of publicity as a health food. Davis (1965) reported that lecithin will either prevent or cure obesity, multiple sclerosis, nephritis, psoriasis, and atherosclerosis (by lowering plasma lipids).

For use as a dietary supplement, lecithin can be obtained in either liquid, granular, powder, or capsule form. Recommended dosages of lecithin vary widely. To reduce serum cholesterol, Morrison (1958) advised the use of two to four tablespoons (12-24 gm) of powdered lecithin daily. Davis (1965) recommended half this amount. A daily intake of 7.2 gm is suggested on the label of one commercially available powdered lecithin. On the other hand, a local pharmacist advised the use of four capsules, or 4.8 gm per day.

Several scientific studies have been done to determine the effectiveness of phospholipid supplementation in lowering plasma lipids. These investigations fall into two categories, depending on the manner of lecithin administration. The lecithin may be given either orally or through intravenous infusion.

The most successful use of oral lecithin supplementation was made by Dr. Lester Morrison in a study reported

in 1958. Doses of 36 gm lecithin per day were administered to hypercholesterolemic patients. After four months, twelve of the fifteen patients showed the following changes: greatly lowered plasma cholesterol, a relative increase in plasma phospholipids and thus a significant increase in the phospholipid to cholesterol ratio. In contrast, Davies and Murdoch (1959) showed that phospholipid supplementation at a level of 0.7 to 2.0 grams per day had no effect on plasma cholesterol of patients suffering from ischemic heart disease. Butler et al. (1960) confirmed the results of Davies and Murdoch. Enticknap (1962) and ter Welle et al. (1974) found no changes in plasma phospholipids or cholesterol in hypercholesterolemic subjects when doses of 0.6 to 2.4 grams were given.

Studies in which lecithin was administered intravenously have all been done on animals. Friedman and Byers found that the sustained infusion of four percent colloidal phosphatide caused a significant increase in plasma cholesterol of rats (1956) and of rabbits (1957). Further investigation showed that this hypercholesterolemia was only temporary. The rabbits that received the infusion were found to have far less atherosclerotic plaque material in their arteries than paired rabbits that received only a dextrose infusion. The cholesterol content of the arteries in the phosphatide-infused rabbits was one-fourth that of the dextrose-infused rabbits, suggesting that the phosphatide

had caused a mobilization of cholesterol in the aorta (Friedman, Byers and Rosenman, 1957).

Patelski et al. (1970) reported similar findings. The following changes were induced in rabbits fed an atherogenic diet without lecithin: in the aortic wall there was decreased cholesterol esterase, enhanced incorporation of free fatty acid into cholesterol esters, increased accumulation of esterified and free cholesterol, and increased lipase and phospholipase A activities; in plasma there was an increase in cholesterol and in phospholipase activity. No change was observed in liver phospholipase A or in lipase activities of serum or liver. However, when animals were fed the atherogenic diet and given injections of 0.5-1.0 ml of a phospholipid supplement every other day, there were no changes in enzyme levels except for increased serum and liver lipase activity. Autopsies of the rabbits revealed that phospholipid-injected rabbits suffered less severe atherosclerosis, and that the incorporation of free fatty acids into the aorta was reduced. Howard and Patelski (1974) later showed that although the activity of aortic acyl Co-A cholesterol acyl transferase was increased in animals with atherosclerosis, injections of polyunsaturated phosphatides inhibited that enzyme.

Theories of Action of Lecithin

The main problem in atherosclerosis is the accumulation of fatty material, especially cholesterol and

triglycerides, in the arteries. When this buildup is great enough, it is not possible for an adequate supply of blood to pass through the artery. Anoxia can occur, resulting in damage to the surrounding tissues.

Popular writers suggest that ingesting extra lecithin will increase the concentration of lecithin in the blood. They feel that, because lecithin acts as an emulsifier in food systems, it will carry away excess cholesterol and triglycerides from the arteries. Although such a theory is very simplified, and does not take into account the complex organization of the system within the blood vessels, it is given some credence by the reports that intravenous infusion of phosphatides causes removal of cholesterol from aortic tissue (Friedman, Byers and Rosenman, 1957). Adding still more credence to their arguments is the fact that in atherosclerosis, although there is an increase in the absolute amounts of both phospholipid and cholesterol in the plasma, the ratio of phospholipid to cholesterol decreases (Gertler and Oppenheimer, 1954). However, some other investigators have disputed this generalization (Thomas et al., 1965).

There are several theories as to the possible mechanisms by which increased phospholipid in the plasma could prevent the accumulation of fats in the arteries or other cardiovascular problems. Ahrens and Kunkel (1949) have shown that various serum samples may have a different appearance, even though the total lipid concentration is

similar. One sample might have been quite clear, while the other appeared cloudy. The amount of cloudiness seemed to be related to the proportion of phospholipid in the total lipid of the serum. The authors felt that the phospholipid helped to maintain the serum lipids in smaller, more stable particles which were less likely to be deposited in atherosclerotic plaques.

Weiner et al. (1973) has suggested that lecithin may exert a protective influence that prevents the autoxidation of cholesterol in cell membranes. The oxidation products of cholesterol appear to be even more atherogenic than cholesterol itself (MacDougal et al., 1965).

Another theory proposes that lecithin may influence the action of lecithin:cholesterol acyl transferase (LCAT). Although the significance is still unclear, it is felt that LCAT may be important in esterifying the cholesterol and by doing so, it may prevent cholesterol from accumulating in the arteries. A study by Soloff, Rutenberg and Lacko (1973) indicates that in patients with coronary heart disease, the rate of cholesterol esterification by LCAT is lower than in normal subjects. However, the authors were unable to say whether the lower activity was causally related to atherosclerosis. It should be noted that with lipid-protein aggregates in which the lecithin to cholesterol ratio exceeds 1:1, LCAT activity is greater than when the ratio is less than 1:1 (Nichols and Gong, 1971). On the other hand,

although injections of lecithin and phosphatidyl choline have been shown to cause significant changes in some enzyme activities (Patelski et al., 1970; Howard and Patelski, 1974), none of these effects was related to LCAT. In addition, Portman et al. (1970) found an increased LCAT activity associated with atherosclerotic conditions.

It is possible that lecithin supplementation might change the phospholipid profile. This also could be significant because certain abnormalities of the phospholipid profile have been associated with atherosclerotic heart disease. Doviasova et al. (1975) showed that, by increasing the lysolecithin content of the plasma through administration of phospholipase A, the free cholesterol content of the plasma was increased, while the proportion of the esterified cholesterol had been mobilized from plasma and erythrocyte membranes and transferred to the liver, as a significantly higher level of cholesterol was found in the liver of animals treated with phospholipase A. These results are particularly interesting in light of the findings of Kunz et al. (1970), Kunz and Strumvoll (1971) and Gillett and Besterman (1975). All found lower levels of LPC in patients suffering from cardiovascular disease. Also, Besterman and Gillett (1972) showed that LPC seemed to inhibit platelet aggregation, and to slow the erythrocyte sedimentation rate, thus possibly decreasing the likelihood of thrombus formation. A thrombus, or clot, is

particularly dangerous as it can block the already narrowed blood vessels and cut off blood circulation to certain parts of the body. The other phospholipid fraction which is changed in cases of cardiovascular disease is PE. Kunz and Strumvoll (1971) felt that PE may enhance thrombus formation, thus increasing the danger of peripheral occlusive arterial disease (POAD).

MATERIALS AND METHODS

Experimental Subjects

The experimental group consisted of thirty-one male volunteers who ranged in age from thirty-eight to fifty-six years. Criteria for acceptance were that the subject 1) have no history of heart disease, 2) have no known abnormalities of fat metabolism, and 3) not be taking any hyperlipidemic drugs. The subjects further agreed to maintain a constant body weight and to adhere to a stable pattern of exercise and diet for the duration of the study.

Experimental Design

The experimental period lasted eight weeks from the first to the last blood sample. Four blood samples were taken for lipid analysis in the course of the study. Two samples were taken within a two- or three-day time span at the beginning of the study, and again at the end of the study. It was hoped that the duplicate sampling would minimize the effect of day-to-day fluctuations in blood lipids which naturally occur. Each sample was analyzed, then the pre-treatment values were averaged, and the post-treatment values were averaged.

Supplemental lecithin was given in the form of "Natural Needs" Soya Lecithin[1], a gelatin capsule containing 1.2 gm

[1]Western Wholesalers Co., Portland, Oregon.

of soya lecithin in soy bean oil. The subjects were asked to take two capsules three times a day for a total of six capsules daily (7.2 gm lecithin/day). Supplementation began on the day after the subject had given his second blood sample, and continued until the fourth sample had been taken, a period of approximately seven and a half weeks.

Dietary Analysis

Subjects were asked to maintain two three-day diet records: one during the first week of the study, and another within one week of the last blood contribution. The dietary analysis had two purposes: first, to evaluate the participant's usual nutrient intake, and second, to insure that the diet of the individual was indeed relatively constant. Because the eating patterns of an individual may differ significantly between weekdays and weekends, the subjects were asked to record their diet on two weekdays and on one day of the weekend. The diets were analyzed by computer for food energy, protein, fat, calcium, vitamin A, thiamin, riboflavin, niacin, ascorbic acid, iron, cholesterol, saturated fat, linoleic and oleic acids, using a nutrient data bank compiled by Ohio State University.

Supplementary Information

Activity levels were estimated from responses to questions concerning type of work, planned exercise, mode of

travel to and from work, participation in sports and work around the home. These activity levels were classified according to the outline given by Bogert, Briggs and Calloway (1973) shown in Appendix I. The subject was initially placed in an activity category which corresponded to the type of work he did. If, in addition, he participated in at least 45 minutes of strenuous activity three or more days a week, he was classified in the next higher activity category.

Percent of desirable weight was calculated from the figures compiled by the Metropolitan Life Insurance Company, (Keys and Grande, 1973). All men were assumed to be of medium frame. If a person was above the desirable weight, the percent overweight was calculated from the highest figure given as desirable. If the subject was below the desirable weight, his percent underweight was calculated from the lowest figure given as desirable.

Collection of Blood Samples

Twenty milliliters of fasting venous blood were collected in two 10 ml Vacutainers treated with ethylenediaminetetraacetic acid (EDTA) powder. Filled Vacutainers were surrounded with ice until they could be returned to the laboratory for processing. Hemoglobin was determined by the cyanomethemoglobin method as described in Oser (1965). Hematocrit was assessed by the method outlined in

Richterich (1969). These parameters were measured in one sample at the beginning and in one sample at the end of the study.

The blood was centrifuged at approximately 35,000 RPM for 35-40 minutes. The plasma was then transferred to small vials and stored in -10°C until further processing could be carried out.

Lipid Extraction and Determination of Total Lipids

Lipids were extracted according to the procedure described by Chiu (1969). Two milliliters of plasma were extracted into chloroform:methanol (2:1). The extract was concentrated to a volume of about one milliliter in an Ehrlenmeyer flask under a stream of pure nitrogen, quantitatively transferred to a tared one milliliter volumetric flask, and completely dried under a stream of nitrogen. Total lipids were determined gravimetrically. The dried extracts were stored at -10°C.

Eighteen extracts that had been randomly selected for chromatographic analysis of phospholipids were sampled prior to drying. This procedure was necessary in order to prevent deterioration of PS and PE fractions (Nelson, 1972). For these samples, the extract from two milliliters of plasma was reduced to a volume of one milliliter. Five hundred microliters were transferred to a tared one

milliliter volumetric flask, dried and weighed as described above. Total lipids are reported as mg/100 ml plasma.

Determination of Lipid Phosphorus

The dried extracts were reconstituted to one milliliter with chloroform. For the lipid phosphorus determination, forty microliters of the extract were diluted to one milliliter with chloroform.

Ten microliters of the diluted extract were ashed with 3.5 percent perchloric acid in 5N sulfuric acid. When the sample was not completely oxidized by the perchloric acid mixture, 10 ul of nitric acid were added to all tubes in the assay. After this step, the samples were returned to the 165°C oven for one hour before being treated with the color reagent.

Phosphorus was analyzed by the procedure of Lowry et al. (1954), as modified by Hawthorne, Smith and Pescadore (1963). The assay is based on the formation of phosphomolybdic acid, which is reduced to a molybdenum blue complex by ascorbic acid. The color so formed was measured spectrophotometrically. Lipid phosphorus was calculated from the regression equation determined from standards. Phospholipids were estimated by multiplying the lipid phosphorus by a factor of 25, and are reported in mg/100 ml plasma.

Determination of the Phospholipid Profile

Plasma phospholipids were separated by thin layer chromatography of the total lipid extract on Brinkman Silica Gel G plates, coated to a thickness of 0.25 mm. A developing solution of chloroform: methanol: glacial acetic acid: water in a ratio of 25:15:4:2 by volume was used. Chromatography chambers lined with filter paper were equilibrated with 92 ml of the developing solution for one hour prior to insertion of the plates. The plates were rinsed with chloroform, and then activated in an oven at 115°C for one-half hour before spotting. Ten microliters of the total lipid extract were applied to the plate, in duplicate, with a Hamilton syringe. Plates were allowed to develop until the solvent front had moved 16 cm from the point of origin. Spots were removed with a spot collector[2]. Phospholipids were then eluted from the silicic acid into a 25 ml Ehrlenmeyer flask using 10 ml of chloroform: methanol (2:1). Except in the case of PC, the entire eluate from each spot was dried under nitrogen for the phosphorus analysis. For the PC spots, one-twentieth of the eluate was analyzed.

Phosphorus was determined by the method described earlier. When ashing from the chromatography spots was not complete, 25 ul of fuming nitric acid were added to all

[2]Brinkman Instruments, Inc., Westbury, New York.

tubes in the assay, and samples were returned to the 165°C oven for one hour. Then, two microliters of perchloric acid were added to all tubes and the samples were returned to a 185°C oven for two hours before adding color reagent.

Phospholipids were reported as percent of the recovered phosphorus. Preliminary studies were carried out to determine the proportion of applied phospholipid which was recovered.

Cholesterol and Triglycerides

Cholesterol was determined by the ferric chloride color reaction (Block, Jarrett and Levine, 1966) using the Technicon Autoanalyzer[3]. Plasma triglycerides were quantified by the Van Handel and Zilversmit (1957) micromethod[4].

Analysis of "Natural Needs" Soya Lecithin

One capsule of "Natural Needs" Soya Lecithin was cut open and the contents were dissolved in 100 ml of chloroform. Twenty microliters of this solution were used for chromatography of the individual phospholipids in the lecithin. One half milliliter of this solution was diluted to 10 ml with chloroform in order to determine the total phosphorus content of the capsule.

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[3]Analyses carried out by Ms. Lisa Holden.

[4]Analyses carried out by Dr. Elisabeth Yearick.

RESULTS AND DISCUSSION

Supplementary and Dietary Information

A description of the subjects appears in Table 4. The vast majority of the participants in this study worked for the University, and this was reflected in generally low levels of physical activity. Twenty-one of the thirty-one men had light activity patterns, while four were judged to be sedentary. Moderate and strenuous lifestyles were each attributed to three men. Fourteen of the subjects were of desirable weight for height; twelve others were within 10 percent of desirable weight for height. Of these twelve, three were slightly below the desirable weight. Only one subject was more than 25 percent above the desirable limits. Four subjects smoked cigarettes at the time of the study and all of the four smoked at least twenty cigarettes per day. All participants had normal hemoglobin concentrations and hematocrits.

Twenty-two subjects completed the dietary records at the beginning and at the end of the study. Six participants turned in only one of the two diets, and three did not return any of the dietary information.

Although the subjects were asked to maintain a fairly constant diet there were great fluctuations in the intake of almost all nutrients between the first and second diets. Such fluctuation is probably not unusual. Yudkin (1951)

Table 4. Description of Subjects.

Subject Number	Age	Relative Weight[a]	Activity Level[b]	Smoking Habits	Hemoglobin (gm/100 ml)	Hematocrit (%)
1	50	106	2	non-smoker	16.4	47
2	55	99	2	non-smoker	16.7	46
3	46	109	2	non-smoker	17.0	48
4	44	109	4	non-smoker	15.7	44
5	38	100	2	non-smoker	15.6	45
6	52	100	2	non-smoker	16.2	47
7	52	94	2	non-smoker	15.1	46
8	51	109	1	non-smoker	16.6	46
9	40	114	2	non-smoker	15.0	44
10	45	100	2	non-smoker	15.4	46
12	51	100	1	non-smoker	17.2	47
13	41	100	2	non-smoker	15.4	44
14	46	129	1	smoker	17.3	48
15	45	103	2	non-smoker	15.6	44
16	45	100	2	non-smoker	15.9	45
17	41	96	4	non-smoker	15.6	45
19	38	100	2	non-smoker	15.2	44
20	49	100	3	non-smoker	15.8	44
21	47	113	2	non-smoker	15.6	46
23	43	108	2	smoker	14.1	40
24	56	109	2	non-smoker	16.2	46
25	44	100	2	non-smoker	15.8	46
26	53	100	3	non-smoker	15.7	46
28	46	104	2	non-smoker	17.0	46
29	54	115	2	non-smoker	15.2	42
30	55	100	2	smoker	15.9	46
31	54	103	1	smoker	17.2	48
33	54	113	2	non-smoker	15.4	42
35	56	100	4	non-smoker	15.6	45
36	48	100	2	non-smoker	16.5	48
37	45	100	3	non-smoker	15.3	44
mean	46	104	2		15.9	45

[a]Percent of desirable weight for height (from tables prepared by Metropolitan Life Insurance Company, 1959).

[b]Activity ratings: 1=sedentary, 2=light activity, 3=moderately active, 4=strenuous activity (Bogert, Briggs and Calloway, 1973).

found that dietary intakes of fat, carbohydrates and protein could vary greatly from week to week for one individual.

When two dietaries were returned, the values from the two were averaged. The average nutrient intake of each subject appears in Appendix II. All of the diets were adequate in protein, iron, niacin and ascorbic acid. However, reported intakes of calories and riboflavin were each low (less than 67 percent of the recommended dietary allowance) in one instance. Calcium, thiamin and vitamin A were each estimated to be low in four cases. For the group, fat provided an average of 37 percent of the calories, although fat intake ranged from 23-48 percent of caloric intake. The range of the ratio of polyunsaturated fatty acids to saturated fatty acids (P/S) was 0.12 to 1.15 with the mean being 0.37. These figures are quite close to the average national intakes. Pike and Brown (1975) state that in the United States 40 percent or more of the dietary calories come from fat. The typical P/S ratio in the United States ranges from 0.3 to 0.4 (Karvonen, 1972). The P/S ratio commonly recommended for lowering cholesterol is 1.0 (Cristakis et al., 1966; Karvonen, 1972).

Mean Plasma Lipids

The mean phospholipid concentration at the end of the study was 187 mg/100 ml, compared with 181 mg/100 ml at the

beginning (Table 5). Both values were within the normal range reported by other investigators (Thomas et al., 1965; Hoffman et al., 1967; MacDonald, 1972) although MacDonald's results are for younger individuals. Average total lipids and triglycerides prior to supplementation were 655 mg/100 ml, and 91 mg/100 ml, respectively. The final values were 645 mg/100 ml, and 105 mg/100 ml, respectively. All values were within the normal range found by other investigators (Ahrens and Kunkel, 1949; Hoffman et al., 1967). Plasma cholesterol concentration before treatment was 204 mg/100 ml and after was 213 mg/100 ml. Again, both values were within the normal range reported by other investigators (Hoffman et al., 1967; Kunz and Strumvoll, 1971; Macdonald, 1972).

The change in concentration of phospholipids, total lipids and triglycerides was not significant, thus agreeing with the results of Enticknap (1962) and ter Welle et al. (1974). The lack of significant change in these three parameters can be interpreted as an inability of supplemental lecithin to change phospholipids, total lipids or triglycerides. This is the conclusion reached by Enticknap (1962) and ter Welle et al. (1974). Indeed, Bragdon (1958) and Scow, Stein and Stein (1967) showed that the amount of phospholipid in the lymph after ingestion of a test meal was not related to the dietary intake of phospholipid. According to this observation, it would seem that

Table 5. Lipid concentrations in plasma before and after lecithin supplementation (mean of 31 subjects and standard deviation).

Lipid Component	Before	After	Percent Change[b]
Phospholipid mg/100 ml	181± 32	187± 27	+ 6
Triglyceride mg/100 ml	91± 43	105± 59	+21
Cholesterol mg/100 ml	204± 53	213[a]± 39	+ 5
Total lipid mg/100 ml	655±164	645±170	0
Phospholipid/ Cholesterol	0.90±0.20	0.89±0.13	

[a]Significantly higher ($p \leq 0.05$)

[b]Mean of individual percent changes (Appendix IV).

supplementary lecithin may not reach the blood stream, and thus could not affect plasma lipids. However, it must be noted that Bragdon (1958) proposed that it was possible that the absorbed phospholipid which was not used in chylomicron formation entered the portal vein directly. This theory has neither been proved or disproved.

It is possible that the lack of effect was due to the relatively short experimental period (eight weeks). However, Morrison (1958) found a significant increase in plasma phospholipids with a significant decrease in cholesterol after only three months of supplementation. On the other hand, neither ter Welle et al. (1974) nor Enticknap (1962) was able to show a significant change in plasma lipids after nine months or two years of supplementation, respectively.

The other factor that may have accounted for the lack of significant change in plasma lipids is the amount of lecithin administered. Morrison (1958), who gave large doses (36 gm lecithin/day) found changes in a relatively short time. The investigators who used smaller doses (maximum of 2.5 gm lecithin/day) found no change, even over long periods of time. If the effect of lecithin supplementation is dosage-dependent, the 7.2 gm/day given in this study was not large enough to change phospholipid, total lipid or triglyceride concentrations during the experimental period.

A rather surprising result was the significant increase ($p \leq 0.05$) in plasma cholesterol from 204 mg/100 ml to 213 mg/100 ml. There are two possible reasons for this increase. First of all, the study was initiated at the beginning of the school term, a period which was relatively free from stress for the majority of the participants. The end of the study coincided with a time of much more stress, the end of the term. Such stress is commonly associated with increased levels of cholesterol (Pike and Brown, 1975).

Much more interesting than the stress theory is the hypothesis that lecithin supplementation actually mobilized cholesterol from the artery wall. This hypothesis was proven when phospholipid was given to animals by intravenous infusions (Friedman et al., 1957) but has not yet been established when lecithin is administered orally. Much more experimental work would be necessary in order to prove this hypothesis, especially in light of the fact that there was no significant increase in plasma phospholipids.

In order to obtain more positive results, it would be advantageous to repeat a similar study for a much longer period of time using several different levels of supplementation as well as a placebo.

Correlations Between Plasma Lipids and Other Variables

Simple correlations were run on all independent variables. The plasma lipid concentrations of each subject

before (A) and after (B) supplementation are shown in Appendix III. Each value is the average of two blood samples. The percent change in each subject's lipid fraction is given in Appendix IV. Correlations were computed between plasma lipids, dietary lipids, weight, activity, and smoking practices. Significant correlations are shown in Table 6. All correlation coefficients are shown in Appendix V.

At both sampling periods there was a strong correlation between phospholipids, cholesterol, total lipids, and triglycerides, thus verifying the generalization that elevation of one lipid parameter is usually associated with an increase in other plasma lipids (Albrink et al., 1961; Thomas et al., 1965). Higher initial values of phospholipid, cholesterol, triglycerides, and total lipids were correlated with a greater decline in total lipid values.

Cigarette smoking was associated with elevated triglycerides during both experimental periods. This is in agreement with the results of Karvonen et al. (1959) and Billimoria (1975). However, no other lipid parameter was significantly raised in smokers.

Percent of desirable weight was also associated with elevated triglycerides, but not with elevation of any other lipid parameter. Moderate and strenuous activity levels were negatively correlated with triglyceride concentrations. This is in accordance with the results of Gertler (1967), Hoffman et al. (1967) and Lopez et al. (1974). None of the

other lipid parameters was significantly affected by activity level.

Unfortunately, the small sample made it difficult to determine other significant correlations. The populations of smokers and men with moderate and strenouos activity patterns were very small. Although the correlations between these lifestyle patterns and lipid parameters were high, only the triglyceride correlations were high enough to fit within the 95 percent confidence interval established.

It should be noted that the correlation between physical activity and P/S ratio in the diet was due to two men who had very high ratios and thus affected the average for the whole group. When these two men were eliminated from the sample there was no significant correlation.

As could be expected, high plasma cholesterol was found to be negatively related to the P/C ratio. Examination of the results for phospholipid determination showed a tendency for the people with the highest initial phospholipids to exhibit a decrease in phospholipids, while those with lower initial phospholipids were more likely to show an increase in phospholipids at the end of the study. This resulted in a negative correlation between the initial phospholipid concentration and the final P/C ratio.

Composition of "Natural Needs" Soya Lecithin

The determination of phosphorus in one capsule of "Natural Needs" Soya Lecithin yielded 51 mg P/capsule.

Table 6. Positive and negative correlations of independent variables. A refers to data prior to supplementation. B refers to data after supplementation. Correlations are significant ($p \leq 0.01$) unless otherwise indicated.

		Total lipids		Cholesterol		Triglycerides		P-lipid / Cholest		Percent Change						
	B	A	B	A	B	A	B	A	B	P-lipid	Total lipid	Cholest	Triglyc	Smoking	Activity	% Desirable Weight
A P-lipids	+	+	+	+	+	+			-	-	-		-			
B P-lipids		+	+	+	+	+	+									
A Total lipids			+	+	+	+	+				-					
B Total lipids				+	+	+	+									
A Cholesterol					+	+	+[a]	-[a]	-		-[a]					
B Cholesterol						+			-				-[a]			
A Triglycerides							+				-			+	-	+
B Triglycerides														+		+
A P-lipid / Cholest										-		+				
B P-lipid / Cholest										+		-[a]	+			
% Change P-lipid											+					
% Change Total lipids													+			
% Change Cholesterol													+			
% Change Triglyceride																
Smoking																
Activity																
% Desirable weight																
% Fat in diet																
P/S ratio in diet															+[a]	

[a] $p \leq 0.05$.

Multiplication of this figure by a factor of 25 gave a concentration of 1.278 gm phospholipid. This is slightly higher than the amount of phospholipid listed on the label of the capsule jar. However, the factor of 25, which is proper when applied to human plasma phospholipids, is not quite correct for this type of phospholipid mixture. The phospholipids from soya lecithin are more highly polyunsaturated than those from humans. Also, the distribution of the different classes of phospholipids is different than in plasma lipids. As each class of phospholipid has a somewhat different molecular weight, the conversion factor will be different.

Thin layer chromatography of lecithin showed the following proportions of phosphorus from the individual phospholipids: PC 37.9 percent, PE 40.6 percent, PI 13.9 percent, unknown (a small spot between the origin and PC) 3.2 percent, and solvent front 4.5 percent. These proportions are similar to those reported by other investigators (Table 3). In comparing the results of Erdahl et al. (1973) with those obtained in this laboratory, it should be noted that Erdahl et al. (1973) used gravimetric determinations of the chromatographed lecithin, while our results are reported as percent of recovered phosphorus. Another fact is that the composition of lecithin is quite variable, depending on the method of extraction (Food Protection Committee, 1966).

Plasma Phospholipid Profile

Thin layer chromatography of the total lipid extract led to the following sequence of phospholipid separation in ascending order: origin (O), LPC, SM, PC, PS and PI, PE and the solvent front (SF). A small quantity of unidentified phosphorus was found in the solvent front and the origin. Preliminary studies revealed that 83 percent of the phospholipid was recovered when chromatographed samples were compared to unchromatographed spots.

Results of the thin layer chromatography are shown in Table 7. All values were within the normal range except SM, which was lower than other authors have reported (Table 2). It is interesting to note that Kunz et al. (1970) found similar levels of SM in patients suffering from hyperlipidemia during the phase of increasing lipids. However, investigation of the lipid values of the subjects whose lipids were chromatographed revealed no significant trends towards increasing lipids. Nor was a low SM necessarily associated with men whose plasma lipids increased during the experimental period.

Another possible explanation for the low SM value is that extraction of the lipid may not have been complete. SM is known to be one of the most difficult phospholipids to solubilize.

An anomalous finding was the low PC and high LPC in the first sample from subject number 12. Because of

Table 6. Phospholipid[a] profile of experimental subjects (as percent of the recovered phosphorus).

Sample number	PC		LPC		SM		PS & PI		PE		O		SF	
	before	after	before	after	before	after	before	after	before	after	before	after	before	after
3	67.7	73.8	7.7	4.4	17.4	14.9	1.7	1.5	3.6	4.3	0.4	0.2	1.5	0.8
12	56.4	73.6	20.0	6.2	9.2	8.8	0.8	2.7	2.3	6.1	-	-	3.2	2.6
20	69.4	76.1	9.8	8.3	13.8	9.7	1.4	1.4	2.9	2.5	0.9	1.1	1.9	0.8
21	69.6	69.8	6.7	7.8	17.6	15.2	1.5	1.7	4.0	4.2	-	0.4	0.6	1.0
26	69.1	70.6	4.3	8.2	20.4	17.7	1.2	0.2	3.8	1.6	-	-	1.2	1.6
29	78.2	75.3	8.6	6.7	9.2	13.8	0.2	0.9	2.0	2.5	-	-	1.7	1.4
31	77.3	74.0	6.2	7.8	10.4	12.6	0.7	1.3	2.5	3.2	-	0.3	2.9	0.7
35	60.4	67.0	13.7	12.3	18.8	14.7	-	0.7	4.6	3.4	-	-	2.4	1.9
37	72.5	73.5	10.7	5.5	8.2	14.9	1.8	0.7	4.0	3.0	1.3	0.9	1.4	1.4
Mean	69.1	72.6	10.6	7.5	13.9	13.6	1.0	1.2	3.3	3.4	0.3	0.3	1.5	1.4
S.D.	6.7	2.9	7.3	2.2	4.7	2.8	0.6	0.7	0.9	1.3	0.5	0.4	0.9	0.6

[a]PC=phosphatidyl choline, LPC=lysolecithin, SM=sphingomyelin, PS=phosphatidyl serine, PI=phosphatidyl inositol, PE=phosphatidyl ethanolamine, O=origin, SF=solvent front.

technical difficulties this plasma sample was thawed and refrozen several times before extraction. Vogel, Zieve and Carleton (1962) found that repeated freezing and thawing of serum resulted in conversion of some of the PC to LPC. Berlin et al. (1969b) also found that when the plasma was held at 37°C for a period of four hours there was a significant increase in LPC concentration. Thus, when the 12A plasma sample was held at room temperature, some of the PC may have been converted to LPC.

There were some general trends in the profiles from the beginning to the end of the sampling period. These trends were: an increase in PC (seven out of nine people), a decrease in SM (six out of nine people), and a decrease in LPC (six out of nine people). However, these changes were not great enough to be significant. There are two possible reasons for this lack of effect of the supplemental lecithin. First, either the time period of the level of supplementation may not have been adequate for effecting a change. Second, more rigid controls should have been placed on the diet. Nye (1969) found that variations in the fat proportions and types of fat, carbohydrate and protein in the diet cause significant variation in plasma phospholipid profiles.

SUMMARY

An exploratory study of the effect of supplementary lecithin on plasma lipids was carried out. Subjects for the study were thirty-one healthy men from the ages of 38 to 56. Each participant received 7.2 gm of supplemental lecithin per day in the form of "Natural Needs" Soya lecithin (six capsules). In order to take into account the day-to-day fluctuations two samples of plasma were taken prior to supplementation and two were similarly taken afterwards. Each sample was analyzed for plasma phospholipid, triglyceride, cholesterol and total lipids. Supplementary information was collected and used to determine smoking habits, activity levels and percent of desirable weight for each subject. In order to observe any possible changes in the phospholipid profile thin layer chromatography was performed on blood samples of nine randomly chosen individuals.

Analysis of the plasma lipids showed no change in phospholipid, triglyceride, or total lipids. No significant change in the phospholipid profile was found. However, there was a significant increase in cholesterol after supplementation. This increase was attributed to either an increase in stress for the subjects or to possible action of the ingested lecithin upon the cholesterol in the arteries.

Calculation of correlation coefficients for all of the independent variables showed positive correlations between all lipid concentrations. Also, higher initial concentrations of phospholipid, cholesterol and triglycerides were negatively correlated with a change in total lipids. There was a negative correlation between activity and plasma triglycerides. Plasma triglycerides were positively associated with both cigarette smoking and percent of desirable weight.

The findings of this study could be greatly clarified and enhanced by further investigations which would extend the time during which supplementary lecithin was administered. Also, because it appears that the effect of lecithin may be dosage-dependent, it would be advantageous to administer several different doses of lecithin as well as a placebo. Stricter controls on diet and activity patterns would eliminate an important extraneous source of variability.

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APPENDICES

APPENDIX I. CRITERIA FOR ASSIGNMENT TO ACTIVITY CATEGORIES (from Bogert, Briggs and Calloway, 1973).

Very light (sedentary): Sitting most of day, studying, talking; about two hours of walking or standing.

Light activity: Typing, teaching, shop work, laboratory work, some walking.

Moderate activity: Walking, housework, gardening, carpentry, light industry; little sitting.

Strenuous activity: Unskilled labor, forestry work, skating, outdoor games, dancing; little sitting.

Very strenuous activity: Tennis, swimming, basketball, football, running, lumbering; little sitting.

APPENDIX II. DIETARY INTAKE OF SUBJECTS COMPARED TO NATIONAL RESEARCH COUNCIL RECOMMENDED DIETARY ALLOWANCES, 1974 (VALUES ARE THE AVERAGE OF TWO THREE-DAY DIETARIES UNLESS OTHERWISE INDICATED).

Subject number	Calories % RDA	Protein % RDA	Fat as % Calories	Calcium % RDA	Iron % RDA	Vitamin A % RDA	Thiamin % RDA	Riboflavin % RDA	Niacin % RDA	Ascorbic acid % RDA	P/S
1	72	145	36	48	127	74	54	60	103	111	0.24
2	94	149	40	99	140	80	100	97	139	205	0.32
3[a]	63	127	44	82	114	131	96	96	92	247	0.30
4	102	170	38	172	118	175	66	152	109	168	0.26
5	83	160	29	126	160	64	96	119	141	354	0.60
6	122	190	41	146	142	116	136	159	130	398	0.20
8	80	128	37	100	113	114	85	105	112	302	0.52
9	77	175	27	42	133	289	77	138	142	154	0.34
10	88	164	37	104	136	144	84	112	122	260	0.26
12	94	169	41	96	135	95	1325[b]	1116[b]	754[b]	1536[b]	0.24
13	92	192	38	191	147	130	64	141	123	136	0.20
14	76	152	42	105	157	100	94	91	103	278	0.30
15	106	168	38	80	171	146	86	110	114	230	0.32
16[a]	84	169	32	139	207	180	122	131	168	406	0.29
17	86	162	37	159	134	182	105	153	92	815[b]	1.15
20[a]	74	111	23	121	122	92	177	164	103	522	0.85
23	80	154	42	90	144	216	68	90	122	251	0.28
24	128	168	46	104	157	117	144	102	128	434	0.37
25[a]	121	187	41	137	181	174	114	128	106	279	0.27
26	128	244	35	234	248	335	1383[b]	876[b]	489[b]	2144[b]	0.24
28[a]	89	184	43	167	140	82	79	161	104	172	0.12
29[a]	130	226	40	149	189	53	270	232	176	446	0.40
30	116	184	45	104	373[b]	230[b]	316[b]	317[b]	304[b]	802[b]	0.66
31	101	156	34	66	138	82	106	106	154	112	0.24
33	115	172	48	69	152	60	108	105	190	294	0.45
35	139	236	32	142	250	116	216	176	194	242	0.32
36	93	138	32	140	185	197[b]	344[b]	330[b]	264[b]	998[b]	0.33
37	74	150	42	158	99	112	50	128	120	170	0.26

[a] Values from these individuals are taken from only one dietary.
[b] Figures include dietary intake plus vitamin supplementation.

APPENDIX III. PLASMA LIPID CONCENTRATIONS OF INDIVIDUAL SUBJECTS, BEFORE (A) AND AFTER (B) SUPPLEMENTATION.

Subject Number	P-lipids mg/100 ml		Total lipids mg/100 ml		Cholesterol mg/100 ml		Triglycerides mg/100 ml		P-lipids/ cholesterol	
	A	B	A	B	A	B	A	B	A	B
1	168	200	770	607	211	195	84	81	0.80	1.03
2	89	132	287	372	114	95	22	68	0.78	1.40
3	226	250	1282	1146	235	245	168	263	0.96	1.02
4	153	172	624	676	178	232	58	97	0.86	0.74
5	174	162	602	574	209	209	62	61	0.83	0.78
6	163	180	532	536	178	198	64	58	0.92	0.91
7	155	198	618	626	155	202	95	126	1.00	0.98
8	152	183	594	580	209	196	85	112	0.73	0.93
9	186	202	677	620	213	204	70	106	0.94	0.99
10	142	174	430	512	159	190	40	40	0.89	0.92
12	163	162	630	570	200	204	78	76	0.82	0.79
13	219	194	780	708	268	269	135	90	0.82	0.72
14	174	200	819	846	211	207	209	224	0.82	0.97
15	200	190	584	604	220	225	66	72	0.91	0.84
16	194	263	688	702	250	187	106	157	0.78	0.92
17	140	180	542	549	178	188	36	55	0.79	0.96
19	164	190	620	620	226	217	100	80	0.73	0.88
20	182	182	676	602	176	209	85	96	1.03	0.87
21	176	173	589	682	184	210	100	142	0.96	0.82
23	199	228	715	702	264	226	127	285	0.75	1.00
24	210	179	598	550	173	207	56	51	1.21	0.86
25	209	152	637	560	183	189	101	93	1.14	0.80
26	200	182	778	657	241	232	98	88	0.89	0.78
28	164	174	573	598	198	175	88	150	0.83	1.00
29	220	194	771	714	258	237	158	110	0.85	0.82
30	196	172	609	653	221	236	62	80	0.89	0.73
31	268	214	875	834	258	291	174	151	1.04	0.74
33	174	160	588	540	158	193	112	65	1.10	0.83
35	182	172	550	645	203	213	56	56	0.90	0.81
36	192	218	702	796	257	280	62	65	0.75	0.78
37	163	154	564	606	150	153	60	84	1.09	1.01
mean	181	187	655	645	204	213	91	105	0.90	0.89
S.D.	32	27	164	170	53	39	43	59	0.20	0.13

APPENDIX IV. PLASMA LIPIDS: POST-TREATMENT CONCENTRATION AS PERCENT OF PRE-TREATMENT CONCENTRATION.

Subject number	Phospholipids	Triglycerides	Total Lipids	Cholesterol
1	119	96	78	92
2	148	309	130	83
3	111	157	89	104
4	112	167	108	130
5	93	98	95	100
6	110	91	101	111
7	128	133	101	130
8	120	76	98	94
9	109	151	92	96
10	123	100	119	119
12	99	97	90	102
13	89	67	91	100
14	115	107	103	98
15	95	109	103	102
16	136	148	102	115
17	129	153	101	106
19	116	80	100	96
20	100	113	89	119
21	98	142	116	114
23	115	224	98	86
24	85	91	92	120
25	73	92	88	103
26	91	90	84	96
28	106	170	93	88
29	88	70	93	92
30	94	129	107	107
31	80	87	95	113
33	91	58	92	122
35	95	100	117	105
36	114	105	113	109
37	94	140	107	102
mean	106	121	100	105

APPENDIX V. CORRELATIONS OF INDEPENDENT VARIABLES. A REFERS TO DATA PRIOR TO SUPPLEMENTATION; B REFERS TO DATA AFTER SUPPLEMENTATION.

	Phospho-lipids	Total Lipids		Cholesterol		Triglycerides		P-lipid/ Cholest		Percent Change						% Desir-able
	B	A	B	A	B	A	B	A	B	P-lipid	Total lipid	Chol-ester	Tri-glyc	Smok-ing	Acti-vity	Weight
A P-lipids	.522	.705	.483	.725	.762	.621	.340	.340	-.587	-.685	-.500	.074	-.452	.346	-.217	.177
B P-lipids		.694	.510	.696	.726	.526	.628	-.241	-.053	.222	-.254	.045	-.031	.240	-.200	.168
A Total lipids			.705	.637	.611	.756	.637	.057	-.221	-.245	-.564	-.039	-.234	.238	-.194	.333
B Total lipids				.466	.544	.621	.544	-.064	-.255	-.129	-.099	.114	-.153	.305	-.091	.133
A Cholesterol					.829	.559	.414	-.384	-.460	-.249	-.376	-.296	-.302	.347	-.240	.149
B Cholesterol						.479	.267	-.102	-.681	-.281	-.276	.276	-.428	.270	-.116	.044
A Triglycerides							.719	.033	-.173	-.266	-.411	-.113	-.266	.477	-.457	.570
B Triglycerides								-.136	.210	.131	-.172	-.221	.329	.521	-.294	.464
A P-lipid/ Cholest									-.180	-.592	-.205	.508	-.234	-.069	.073	.038
B P-lipid/ Cholest										.699	.275	-.413	.721	-.091	-.066	0.33
% Change P-lipid											.426	-.091	.582	-.107	.036	-.112
% Change Total lipids												.115	.472	.04	.184	-.124
% Change Cholesterol													-.271	-.128	.237	-.132
% Change Triglyceride														.122	.179	-.082
Smoking															-.332	.311
Activity																-.313
% Fat in Diet	-.176	-.090	.090	-.347	-.234	-.128	.104	.196	.127	.071	.102	.176	.206	.190	-.071	-.044
P/S Ratio in Diet	-.135	-.150	-.115	-.174	-.082	-.268	-.233	-.043	-.064	.115	.015	.185	-.003	-.006	.358	-.161