Clinical Study

Influence of Soy Lecithin Administration on Hypercholesterolemia

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Recent studies suggest that lecithin-rich diet can modify cholesterol homeostasis and hepatic lipoprotein metabolism. Considering the phytotherapeutic impact of lecithin, this work hypothesizes that lecithin administration in hypercholesterolemic patients may reduce cholesterol concentrations by increasing biliary secretion. Total cholesterol and LDL were evaluated after soy lecithin administration in hypercholesterolemic patients. One soy lecithin capsule (500 mg/RP-Sherer) was administrated daily. One-two months before the treatment beginning, blood samples were collected for total lipids and cholesterol fractions analysis. The results showed a reduction of 40.66% and 42.00% in total cholesterol and of 42.05% and 56.15% in LDL cholesterol after treatment for one and two months, respectively. A significant reduction in total cholesterol and LDL-cholesterol concentrations was observed during the first month of treatment, suggesting that the administration of soy lecithin daily may be used as a supplemental treatment in hypercholesterolemia.

1. Introduction

Lipid metabolism studies include a vision for the lipoprotein structure, function, and a description of the lipid metabolism forms, indicating that the dyslipidemias are important risk factors in the context of cardiovascular disease and that appropriate intervention can have a significant impact on clinical treatment [1].

Atherosclerosis, the most serious cardiovascular disease, may affect individuals at an early age (20–29 years). One of the disease symptoms is greasy striations, and its evolution depends on several factors, such as heredity addition to the diet, stress, and aging resulting from vascular serious imbalances. The relationship between hypercholesterolemia and coronary atherosclerosis disease has been demonstrated in many clinical trials [2, 3]. Moreover, the reduction in the number of events and mortality or coronary disease, interruption, or even the decline in atherosclerotic disease by plasma cholesterol-lowering drugs has also been reported by numerous studies [4, 5].

One of dietary risk factors for dyslipidemias and atherogenesis is the deficiency in the antioxidant intake, such as selenium [6], vitamin E [7], in addition to the low consumption of unsaturated fats [8, 9] and fiber [10]. More recently, it has been suggested that antioxidant substances are capable of reversing endothelial dysfunction caused by hypercholesterolemia [11–13] and also reduce the number of coronary events [14], although their use in medical practice still needs more conclusive information.

Considering the high cost of drugs to reduce the plasma cholesterol and the prospect of their prolonged use, patients have relied on alternative treatments for the control of
hypercholesterolemia [15, 16]. These treatments have been used in an empirical way by the population in general due to the lack of methodologies that allows more reliable conclusions.

Recent studies suggest that a lecithin enriched diet can modify the cholesterol homeostasis and lipoprotein metabolism. Lecithin diet modifies the cholesterol homeostasis in the liver, increasing the activity of HMG-CoA reductase and cholesterol 7 alpha-hydroxylase, and decreasing the microsomal ACAT activity [17].

One of the most spectacular properties of lecithin is its ability to reduce the excess of LDL cholesterol. It also promotes the synthesis in the liver of great amount of HDL, the beneficial cholesterol [18].

Bile acid secretion with high levels of cholesterol and phospholipids is encouraged by lecithin-rich diets when compared with diets without lecithin [17]. Therefore, this study evaluates hypercholesterolemic effect of soy lecithin on patients with pure or combined hypercholesterolemia.

2. Materials and Methods

Volunteers (n = 30) were chosen from a group previously diagnosed with hypercholesterolemia. Placebo capsules, containing 500 mg of soy oil and 500 mg soy lecithin capsules (22% phospholipid (phosphatidylethanolamine), 10% triacylglycerol, and 68% phosphatidylcholine), were kindly donated by RP-Sherer (Brazil). Blood was collected using Vacutainer System (Bd, Brazil). Analytical determinations were performed using commercial enzymatic kits donated by Labtest (Brazil). Statistical analysis were done using SigmaStat and the graphs were built in Microsoft Excel software.

2.1. Patients. Thirty volunteers (58–70 years old) were selected for administration of lecithin. The groups were chosen from patients diagnosed with hypercholesterolemia, participants of a project at the University Camilo Castelo Branco (Unicastelo, SP, Brazil). These voluntary patients were previously informed about the use of biological material to carry out the work. The project was approved by the Ethics Committee of the Faculty of Medical Sciences, State University of Campinas under the protocol number 792/08. Every volunteer was given one capsule (n = 20) of 500 mg soy lecithin daily and placebo group received one capsule placebo. One and two months after treatment with beginning, the blood samples were withdrawn and the lipid profile was performed.

This was a double-blind study, in which only one person in the group knew the patients who received placebo or soy lecithin capsule; after the data analysis, it was allowed access to the primary research groups.

Blood was collected in tubes for serum obtention; serum was separated by centrifugation at 2500 rpm for 10 minutes. Serum samples were stored in a freezer (20°C) until the determination completion. The volume of blood required to perform the analysis was 10 mL per withdrawn. For placebo influence determination blood was withdrawn before and two months after administration. Patients taking soy lecithin capsules had blood withdrawn before and after the administration (one and two months).

2.2. Lipid Determination. Total cholesterol and triglyceride concentrations in the plasma were determined by enzymatic methods, using commercial reagents (Labtest diagnostics, Brazil).

The HDL cholesterol level was also determined by an enzymatic method (Labtest diagnostics, Brazil), after the precipitation of LDL and VLDL fractions. The Friedewald equation (1), below, was used to obtain the concentration of LDL cholesterol [19]:

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Ch_{LDL} = Ch_{Total} - (Ch_{VLDL} + Ch_{HDL}),
\]

where Ch stands for cholesterol and Ch_{VLDL} represents triglycerides divided by five.

2.3. Statistical Analysis. Results were calculated using average ± standard deviation. The statistical analysis, which was considered significant, used the paired t-test of the Sigma Stat software (P < .001). The graphs were built in Microsoft Excel software.

3. Results and Discussion

Patients who took placebo capsules showed no differences in lipid profile after two months of administration (Figure 1), total cholesterol was reduced in 11.4%, but this decrease is not statically significant.

For patients taking soy lecithin capsules significant decrease in the total cholesterol concentration and in HDL cholesterol during the first and the second months of administration suggests that the administration time did not influence the results (Figure 2). Also, triglyceride concentration did not change as seen in relation to cholesterol (data not shown).

Figure 2 also shows the concentration of lipoprotein cholesterol, showing a significant reduction in the LDL cholesterol concentration after administration of soy lecithin capsules, which was not dependent on the administration time.
Scientific community believes that the cholesterol concentration is a risk factor of cardiovascular disease. It was observed that a decrease of 10% in cholesterol is associated with a 27% reduction in cardiovascular disease risk [20]. The main determinants of plasma cholesterol are saturated fatty acids polyunsaturated and dietary cholesterol. Plasma cholesterol can be further reduced by specific dietary supplements, such as fiber, garlic, and fish oils [21].

Thus, many scientists have explored the possibility of increasing hypocholesterolemic components of foods; recent studies have shown that soy protein and soy sterols have hypocholesterolemic effect [22, 23].

In this work, soy lecithin effect on the serum cholesterol concentration was evaluated. The results showed a decrease of 40.65% and 42.60% in total cholesterol and 42.65% and 56.11% in LDL cholesterol, one and two months after administration, respectively. The results of this study are in line with studies of soy protein as a whole; there is no work in literature on the isolated effect of lecithin.

Soybean presents a number of advantages compared with other sources of vegetable protein [24]. It has high-protein content (38%–42%) of low cost and high quality as well as isoflavones that help in reducing blood cholesterol. The daily intake of 25 grams of soy protein dramatically reduced the total cholesterol over a period of approximately three weeks, that is, one month after treatment beginning. It was also shown that this effect was not dependent on time, since there was no increase two months after the treatment end. The daily intake of soy protein may reduce the LDL concentration by 30%, while occurs a stimulus for the HDL production [25, 26].

Soybean protein increases the cholesterol-lowering effects of plant sterols on rats fed cholesterol; the combination of plant sterols and soy protein increases fecal neutral sterols and bile acid excretion compared with the sterol and soy protein alone; therefore, the combination of sterol and soy protein shows a more ostensible decrease in plasma lipids than the isolated ingredients [27].

A decrease in the cholesterol intestinal absorption and an increase in the bile acid excretion have been suggested as possible mechanisms for the effects in the reduction of lipids by soy protein [28].

Jiang et al. [29] demonstrated the inhibition of cholesterol absorption in diets rich in phosphatidylcholine. This study suggests that the high degree of saturation of acyl groups of the soybean phosphatidylcholine decreases the cholesterol intestinal absorption.

Lecithin is one of the nature elements that have dispersing properties. That is why it can emulsify fat, avoiding its absorption. Lecithin is capable of reducing LDL-cholesterol. It also promotes the HDL-cholesterol synthesis [27]. In addition to be used to help reduce cholesterol and triglycerides and protect the liver in the prevention of kidney stone formation, it is used as a tonic for the nervous system and brain activities. The Food and Drug Administration-(FDA) USA, and the World Federation of Cardiology recommended the use of 25 grams per day of soy protein, which corresponds to approximately 60 g of soybeans for cardiovascular disease prevention. However, it is not yet clear which components of soy are responsible for their antiatherogenic purposes.

Recent studies suggest that a lecithin-rich diet can modify the cholesterol homeostasis and lipoprotein metabolism in liver. Lecithin diet modifies the cholesterol homeostasis in the liver, increasing the HMG-CoA reductase and alpha 7 hydroxylase cholesterol activities and decreasing the microsomal ACAT activity. The LDL concentration and size are also significantly reduced and the bile acid pool and bile lipid secretion are increased [17].

4. Conclusion

This work suggests that soy lecithin-rich diets can be used as an adjunct in the treatment of hypercholesterolemia; however, further works with a large number of patients should be carried out towards finding the ideal dose-response.

Lecithin-rich diets can stimulate the fatty acid secretion with high levels of cholesterol and phospholipids when compared with diets without lecithin, considering the lecithin performance as phytotherapeutic, with a large spectrum of activity. The results showed significant reduction in the concentration of total cholesterol and LDL-cholesterol during the first month, suggesting that the daily administration of lecithin capsule could be used as an adjuvant treatment in hypercholesterolemia, possibly by reducing the intestinal absorption or by the increased secretion of bile acids with high levels of cholesterol and phospholipids.

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References


