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Summary and Implication

Egg lecithin differs from soy lecithin in its phospholipid profile and fatty acid composition. The current study was designed to test whether egg lecithin or soy lecithin decreased the plasma cholesterol concentration in hamsters that are used as a model for humans. Male golden Syrian hamsters were assigned randomly to eight dietary treatments lasting 4 weeks (n=10 per treatment) that contained 0.12% (wt/wt) of cholesterol and varied amount of supplemental lipids. The diet groups were: control with no supplemental lipid, 0.05%, 0.5%, and 5% (wt/wt) of egg lecithin or soy lecithin, respectively, and 5% (wt/wt) of soybean oil. Dietary soy lecithin decreased the plasma cholesterol concentration compared with control in a dosage- and time-dependent manner. The 5% soy lecithin treatment greatly decreased the final plasma total cholesterol and LDL-cholesterol compared with the control ($p<0.0001$). In addition, the 5% soy lecithin supplementation caused greater decrease in plasma total cholesterol than did the 5% soybean oil treatment ($p<0.05$), indicating effects beyond the fatty acid composition. The cholesterol absorption measured on day 24, however, was lower in the control group compared with all other treatments ($p<0.05$). Overall, dietary soy lecithin but not egg lecithin decreased the plasma cholesterol concentration in hamsters via a mechanism other than decreasing cholesterol absorption.

Introduction

It has been long recognized that blood cholesterol concentration is an important risk factor for development of atherosclerosis. Several studies have shown that soy lecithin, a phospholipid mixture extracted from soybean oil, has hypocholesterolemic properties in animals and humans. The underlying mechanism for the cholesterol lowering effect of soy lecithin, however, has not been elucidated.

Cholesterol homeostasis is determined by the balance of intestinal cholesterol absorption, endogenous cholesterol synthesis, and excretion of biliary sterols. Cholesterol absorption is believed to be correlated positively to plasma cholesterol concentration. Recent studies showed that phosphatidylcholine (PC) suppressed cholesterol absorption *in vitro* and *in vivo*, which, in turn, might decrease the blood cholesterol concentration.

Soy lecithin consists of three types of phospholipids (PL): PC, phosphatidylethanolamine (PE), and phosphotidylinositol (PI), with similar amounts of each PL. In contrast, egg lecithin contains PC and PE, with PC as the major component. The concentration of total unsaturated fatty acids is much higher in soy lecithin than in egg lecithin. The objective of this study was to test whether egg lecithin or soy lecithin decreased the plasma cholesterol concentration in hamsters used as a model for human.

Materials and Methods

Animals and diets. Male golden Syrian hamsters (10 weeks old, weighing ~100 g) were housed individually in metabolic rodent cages. All animals were maintained in a temperature-controlled room (23°C) with a 12-hour/12-hour light/dark cycle. Hamsters were provided free access to food and water through the entire experiment period and were allowed 1 week of adaptation to the environment prior to the treatment. Hamsters were assigned randomly to eight dietary treatments lasting 4 weeks (n=10 per treatment) that contained 0.12% (wt/wt) of cholesterol and varied amount of supplemental lipids. The diet groups were: control with no supplemented lipid, 0.05%, 0.5%, and 5% (wt/wt) of egg lecithin or soy lecithin, respectively, and 5% (wt/wt) of soybean oil. The rodent chow was ground to powder by a mechanical blender. Cholesterol and supplemented lipids were added to the ground chow in warm ethanol and hexane, respectively, which were evaporated before feeding the hamsters.

Plasma lipid and lipoprotein analysis. Blood samples were collected on day 0, 14, and 28 of the study period. Plasma total cholesterol, HDL-cholesterol, and TAG concentrations were determined enzymatically. Non-HDL-cholesterol (VLDL + LDL) was calculated by difference.

Cholesterol absorption. Cholesterol absorption efficiency was measured on day 2 and 24 of the study. Hamsters were given intragatrically by gavage 150 µl of medium-chain triacyglecerol (MCT) oil containing a mixture of 1 µCi of [^{14}C] cholesterol and 2 µCi [^{3}H] sitostanol. Feces were collected for the following 4 days, dried for three days, and then finely ground. Lipids were extracted from the ground feces and the original dosing mixtures and dissolved in chloroform. Duplicate aliquots of each sample were transferred to counting vials and dried. After samples were redissolved in methanol, H_2O_2 was added for decolorization. The vials then were incubated at 37 ºC until the color of pigments in the samples disappeared. Methanol and H_2O_2 were evaporated, and scintillation cocktail was added to the vials for counting of

radioactivity. The cholesterol absorption efficiency was calculated as: % cholesterol absorption = {($^{14}C/^{3}H$ dosing mixture - $^{14}C/^{3}H$ feces)/ ($^{14}C/^{3}H$ dosing mixture)}× 100

Statistics analysis. PROC MIXED in SAS was used to analyze the data, which included treatments as fixed effects and blocks as random effects. If the F value of ANOVA was significant, group differences were analyzed further by a multiple comparison test. Differences were considered significant at $p<0.05$.

Results and Discussion

The total plasma cholesterol concentrations in hamsters were affected by dietary treatments in a time- and dosage-dependent manner (Table 1). Feeding 5% egg lecithin for two weeks resulted in a higher plasma cholesterol concentration compared with that of the other treatments ($p<0.05$), but the final cholesterol concentration in the 5% egg lecithin group did not significantly differ from that of the control. Supplementing 5% soy lecithin or 5% soybean oil for four weeks decreased the plasma cholesterol concentration in hamsters compared with control ($p<0.0001$ and $p<0.05$, respectively). In addition, the final cholesterol concentration in the 5% soy lecithin treatment group was lower than that in the 5% soybean oil group ($p<0.05$). The 0.5% and 0.05% dosages of both soy lecithin and egg lecithin supplementations did not result in any significant differences compared with control.

Table 1. Treatment effects on hamster plasma cholesterol concentration at different time points[1].

Treatment	Plasma Cholesterol Concentration (mg/dl)		
	Day 0	Day 14	Day 28
Control	86.07	148.07^{b,c}	226.71^{a}
0.05% egg lecithin	81.31	151.91^{b,c}	235.44^{a}
0.5%egg lecithin	87.07	156.4^{b}	223.37^{a,b}
5% egg lecithin	82.34	166.29^{a}	234.09^{a}
0.05% soy lecithin	83.09	151.25^{b,c}	219.76^{a,b}
0.5% soy lecithin	83.88	140.80^{c}	218.11^{a,b}
5% soy lecithin	84.74	139.81^{c}	189.77^{c}
5% soybean oil	80.06	148.9^{b,c}	208.53^{b}
S.E.M.	4.81	4.73	11.29

[1]Values in the same column not sharing a common superscript differed at $p<0.05$; n=10 for each treatment; S.E.M., standard error of the mean.

There were no significant differences in the final concentrations of plasma HDL-cholesterol and triacylglycerol (TAG) between the different treatment groups (Table 2). In contrast, dietary 5% soy lecithin decreased ($p<0.05$) the non-HDL (VLDL and LDL)-cholesterol concentration compared with those of the other treatment groups except the 5% soybean oil treatment ($p>0.05$). These data suggest that 5% soy lecithin decreased the total cholesterol by decreasing the non-HDL-cholesterol.

Lipid supplements did not result in significant differences in cholesterol absorption in the short term (Table 3). In contrast, cholesterol absorption measured by the end of the study (day 24) was lower in the control group compared with that of the other groups ($p<0.05$), which indicated that dietary soy lecithin decreased the plasma total and non-HDL-cholesterol concentration via a mechanism other than decreasing cholesterol absorption.

Table 2. Treatment effects on hamster plasma HDL-cholesterol (chol), non-HDL-cholesterol, and TAG concentration on day 28[1].

Treatment	HDL-Chol (mg/dl)	Non-HDL-Chol (mg/dl)	TAG (mg/dl)
Control	56.10	170.59^{a,b}	179.59
0.05% egg lecithin	51.92	183.47^{a}	192.73
0.5% egg lecithin	57.15	166.19^{a,b}	160.89
5% egg lecithin	52.05	182.01^{a,b}	196.50
0.05% soy lecithin	54.50	164.72^{b}	204.37
0.5% soy lecithin	53.96	164.97^{b}	160.72
5% soy lecithin	50.08	139.66^{c}	189.00
5% soy bean oil	55.76	152.75^{b,c}	178.68
S.E.M.	5.28	10.79	13.67

[1]Values in the same column not sharing a common superscript differed at $p<0.05$; n=10 for each treatment; S.E.M., standard error of the mean.

Table 3. Treatment effects on cholesterol absorption at different time points[1].

Treatment	Cholesterol absorption efficiency (%)	
	Day 2	Day 24
Control	55.22	44.81^{c}
0.05% egg lecithin	59.22	62.46^{a,b}
0.5% egg lecithin	57.96	66.93^{a,b}
5% egg lecithin	67.84	72.65^{a}
0.05% soy lecithin	56.29	60.67^{b}
0.5% soy lecithin	54.77	58.65^{b}
5% soy lecithin	52.06	62.99^{a,b}
5% soy bean oil	61.01	61.45^{a,b}
S.E.M.	4.69	4.13

[1]Values in the same column not sharing a common superscript differed at $p<0.05$; n=10 for each treatment; S.E.M., standard error of the mean.

Numerous studies have shown that dietary unsaturated fatty acid (UFA) can decrease blood cholesterol concentration, whereas high intake of saturated fatty acids, especially C12:0, C14:0, and C16:0, will result in elevated blood cholesterol concentration. Soybean oil and soy lecithin contain high percentages of UFA, which may contribute to their cholesterol lowering properties. The

decrease of plasma cholesterol concentration, however, was more obvious in the hamsters of the 5% soy lecithin group than in those of the 5% soybean oil group, indicating effects beyond the content of UFA.

Phosphatidylcholine (PC) plays an important role in the emulsification and solubilization of cholesterol in small intestine. Several studies, however, showed that adequate supply of PC inhibits, instead of increases, cholesterol absorption. This inhibition is relieved by the hydrolysis of PC by pancreatic phospholipase A_2 ($pPLA_2$). Our data suggest that the supplemented lipids increased the cholesterol absorption by acting as an emulsifier and solubilizer for cholesterol. The possible inhibition of PC on cholesterol absorption was abolished by sufficient hydrolysis of PC in the small intestine of hamsters.

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