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Pectin and psyllium decrease the susceptibility of LDL to oxidation in guinea pigs

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These studies were undertaken to determine whether pectin (PE) and psyllium (PSY) intake affect the circulating levels of α-tocopherol and the susceptibility of low density lipoprotein (LDL) to oxidation. For that purpose, male Hartley guinea pigs were fed 19 g/100 g of a fat mix with a 2:1:1 ratio of saturated:polyunsaturated: monounsaturated fatty acids and 35 g/100 g total carbohydrate with 80% of the carbohydrate energy contributed by sucrose. Diets were identical in composition except for the fiber source: cellulose (control diet), PE, or PSY. Guinea pigs fed PE or PSY had 36% and 67% lower plasma cholesterol concentrations, respectively, compared with controls ($P < 0.001$). This plasma cholesterol lowering was associated with both very low density lipoproteins and LDL cholesterol fractions. Intake of PE or PSY resulted in 54% lower plasma triacylglycerol (TAG) concentrations compared with the control group ($P < 0.001$). LDL from PE and PSY fed guinea pigs contained fewer molecules of cholesteryl ester, and α-tocopherol concentrations in this particle were 49% and 66% higher, respectively, compared with controls. In addition, LDL from guinea pigs fed soluble fiber exhibited less susceptibility to oxidation than those from the control group, as determined by thiobarbituric acid-reactive substances formation. Hepatic free and esterified cholesterol were 32% lower and hepatic TAG was 25% lower in guinea pigs fed PE and PSY compared with controls. The data from these studies confirm that PE and PSY reverse the hyperlipidemia associated with high fat–sucrose diets and demonstrate a potential antioxidant effect of soluble fiber on circulating LDL. (J. Nutr. Biochem. 10:118–124, 1999)

Keywords: α-tocopherol; guinea pig; LDL oxidation; pectin; psyllium

Introduction

Elevated plasma low density lipoprotein (LDL) cholesterol concentrations represent a major risk factor for the development of atherosclerosis.[1] Increased consumption of dietary fiber has been hypothesized to protect against ischemic heart disease by decreasing plasma LDL cholesterol concentrations.[2] Dietary soluble fiber presumably reduces plasma cholesterol concentrations by different primary effects in the small intestine, such as interruption of cholesterol absorption,[3] interruption of the enterohepatic circulation of bile acids,[4] or both.[5]

Although the beneficial effects of soluble fiber have been known for several years,[6] the mechanisms responsible for its hypocholesterolemic properties have not been completely elucidated. Studies have shown that rats fed fat-cholesterol rich diets supplemented with pectin (PE) had significantly higher fecal excretion of neutral sterols and bile acids than those fed cellulose, which indicates that PE may perform its hypocholesterolemic effects by increasing the excretion of fecal acid and neutral sterols.[7] Increases of fecal bile acid excretion due to PE consumption also have been demonstrated in clinical studies, although with much less of an effect compared with bile acid binding resins.[8] In addition, human studies have shown that psyllium (PSY) consistently reduces total plasma cholesterol by 5 to 20%, and LDL cholesterol by 8 to 20% and these decreases may be related to the ability of PSY to interrupt the enterohepatic circulation of bile acids by micelle disruption or other mechanisms.[9]

Simple carbohydrate (CHO) intake is associated with increases in plasma triacylglycerol (TAG) concentrations[10]

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and decreases in high density lipoprotein (HDL) cholesterol levels.[11] These two parameters under those conditions are associated with increased risk of coronary heart disease. The association between the consumption of soluble fiber and the reduction of elevated plasma TAG levels induced by simple CHO has not been extensively studied; therefore, this represents an important area of investigation.

Oxidatively modified LDL also has been implicated in the development of atherosclerosis.[12] Oxidized LDL plays an important role in the development of atherosclerosis by uptake via the macrophage scavenger receptor and foam cell formation.[13] The oxidative modification involves the linkage of amino groups of lysine residues of apolipoprotein (apo) B with aldehydic lipid peroxidation products and results in an altered apoB conformation.[14] Antioxidants in plasma protect LDL against oxidative modification,[15] making it less likely for LDL to be oxidized in the circulation.

The hypolipidemic properties of soluble fiber may be related to the inhibition of cholesterol absorption and to the interruption of bile acid homeostasis in the intestinal lumen, as was described above. Thus, the question related to the effects of PE and PSY on the plasma concentrations of lipid soluble antioxidant micronutrients such as vitamin E remains to be evaluated, because bile acids are required for its physiologic absorption.

In a previous report we evaluated the hypolipidemic effects of PE and PSY and addressed some of the mechanisms involved.[16] The present studies were undertaken to assess the potential beneficial effects of PE and PSY on the susceptibility of LDL to oxidation. We hypothesized that PE and PSY would decrease the susceptibility of LDL to oxidation. This hypothesis was based on our reported data of decreases in plasma LDL cholesterol concentrations observed in guinea pigs after intake of PE and PSY,[16] which could be associated with less lipid available for oxidation.

Guinea pigs were used as the animal model for these studies because they are similar to humans in terms of plasma lipoprotein distribution (i.e., LDL is the major cholesterol carrier lipoprotein) and responses to dietary changes, which are limited to plasma LDL analogous to the human situation.[17]

Material and methods

Materials

TAG assay kits, halothane, and bovine albumin were from Sigma (St. Louis, MO USA). Enzymatic cholesterol assay kits, cholesterol oxidase, and cholesterol esterase were purchased from Boehringer Mannheim (Indianapolis, IN USA). Free cholesterol and phospholipid enzymatic kits were from Wako Pure Chemical (Osaka, Japan). Malonaldehyde bis (diethyl acetal) was from Aldrich (Arlington Heights, IL USA). Quickseal ultracentrifuge tubes were from Beckman Instruments, Inc. (Palo Alto, CA USA). High methoxylated PE obtained from lime peels and containing 6.7% methoxyl groups and 74% galacturonic acid was obtained from Ginsted Products Inc. (Industrial Airport, KY USA). Powdered PSY husk no. 40 (purified 95%, containing less than 3% fat and 1% protein) was obtained from Meer Corporation (North Bergen, NJ USA).

Table 1 Composition of experimental diets

	Diets (g/100 g)			Energy
	Control	Pectin	Psyllium	(%)
Nutrient				
Protein	22.8	22.8	22.8	23.0
Fat mix*	19.0	19.0	19.0	43.2
Starch	7.0	7.0	7.0	7.0
Sucrose	28.0	28.0	28.0	27.9
Vitamins†	1.0	1.0	1.0	1.0
α-Tocopherol	0.001	0.001	0.001	—
Minerals†	8.2	8.2	8.2	—
Cellulose	**14.0**	—	5.6	—
Pectin	—	**14.0**	—	—
Psyllium	—	—	**8.4**	—
Cholesterol	0.17	0.17	0.17	—
Kjoule/g	5.97	5.97	5.97	—
Nutrient Caloric Density (g/kjoules)				
Fiber	8.35	8.35	8.35	
Minerals	4.9	4.9	4.9	
Vitamins	0.6	0.6	0.6	
Proteins	13.7	13.7	13.7	
Cholesterol	0.1	0.1	0.1	

*Fat mix with polyunsaturated/saturated ratio of 0.5 with 2:1:1 saturated:monounsaturated:polyunsaturated fatty acids (23.8 g/100 g lauric, 7.8 g/100 g myristic, 9.2 g/100 g palmitic, 8.6 g/100 g stearic, 19.9 g/100 g oleic, and 26.4 g/100 g linoleic).
†Vitamins and minerals were formulated to meet National Research Council (NRC) specified requirements for guinea pigs. A detailed composition of vitamins and minerals has been reported elsewhere.[18]

Diets

Diets were prepared and pelleted by Research Diets (New Brunswick, NJ USA). Three different high-fat diets were used in these studies. Protein, fiber, vitamins, minerals, and cholesterol amounts in these diets were adjusted to the same energy density. Dietary cholesterol was adjusted to 0.1 g/1,000 kjoules, which is equivalent to an amount of absorbed cholesterol equal to the daily synthesis rates in guinea pigs[18] (*Table 1*). Three different sources of fiber were tested within these studies: the insoluble fiber cellulose (control) and the two soluble fibers PE and PSY. The fat mixture was formulated with 24 g/100 g olive oil, 49 g/100 g palm kernel oil, and 27 g/100 g safflower oil. Because PE and PSY similarly reduce plasma cholesterol when PE is given as the sole fiber source, as does PSY at a concentration of 60 g/100 g total soluble fiber,[16] the amounts of fiber were adjusted accordingly in these diets.

Animals

Male Hartley guinea pigs (Harlan Sprague Dawley Inc., Indianapolis, IN USA) weighing 300 to 400 g (10 per group) were randomly assigned to one of three dietary groups for 4 weeks. Animals were housed in a light cycle room (7:00 AM to 7:00 PM) and had free access to water and diets. Guinea pigs were anesthetized with halothane vapors and euthanized by heart puncture. Blood was utilized for the measurement of plasma lipids, lipoproteins, plasma thiobarbituric acid-reactive substances (TBARS) formation, and LDL-α-tocopherol. Hepatic tissue was harvested for the measurement of cholesterol and TAG concentrations. Animal experiments were conducted in accordance with U.S. Public Health Service and U.S. Department of Agriculture guidelines. Experimental protocols were approved by the University of Connecticut Institutional Care and Use Committee.

Plasma and hepatic lipid determinations

Total plasma cholesterol[19] and TAG concentrations were determined by enzymatic analysis. Plasma HDL was determined using the precipitation method of Warnick et al.[20] with a slight modification, which is the use of 2 mol/L dextran sulphate. LDL was separated by ultracentrifugation in a LE-80K ultracentrifuge (Beckman Instruments, Inc.) at 125,000 × g at 15°C for 19 hours in a Ti-50 rotor. Separation was based on d = 1.019 − 1.090 kg/L.

Hepatic total and free cholesterol and TAG were determined according to Carr et al.[21] after extraction of hepatic lipids with chloroform:methanol (2:1). Hepatic cholesteryl ester concentrations were calculated by subtracting hepatic free cholesterol from total cholesterol.

LDL characterization

LDL composition was calculated by determining free and esterified cholesterol, TAG, and phospholipids by use of enzymatic kits[18]; protein was calculated by a modified Lowry procedure.[22] The number of constituent molecules of LDL was calculated on the basis of one apoB per LDL with a molecular mass of 412,000 kD.[23] The molecular weights used were: 885.4, 386.6, 645, and 734 g/mol for TAG, free and esterified cholesterol, and phospholipids, respectively.

In vitro determination of LDL oxidation susceptibility

LDL from individual samples was isolated using agarose-heparin affinity columns (LDL-direct method) and dialyzed in ethylenediamine tetraacetic acid (EDTA)-free phosphate buffered saline (PBS; 10 nmole/L $NaPO_4$ buffer, pH 7.4, containing 0.15 M/L NaCl). Copper-mediated oxidation of LDL was performed by adding 0.5 mM $CuCl_2.2H_2O$ solution to 0.2 mg protein/mL LDL. The effect of dietary treatments on the extent of oxidation was measured by incubating samples for 3 hours at 37°C. The lipid peroxide content of oxidized LDL was determined by analyzing TBARS expressed as malonaldehyde (MDA) equivalents.[24] The TBARS assay was conducted by adding 2 mL of TBARS reagent (26 mmol/L TBA, 0.92 trichloracetic acid in 0.25 N HCL) to 550 μL incubation mixture and heating in a boiling water bath for 15 minutes. After removing the tubes from the water bath, 0.025 L of n-butanol were added. After shaking briefly, the phases were separated by centrifugation at 1,500 × g for 15 minutes. The pink color developed in the organic layer was read in a spectrophotometer at 532 nm.

Determination of LDL α-tocopherol concentrations

α-Tocopherol concentrations were determined in LDL because this is the major α-tocopherol carrier lipoprotein in circulation.[25]

LDL was separated by ultracentrifugation at a density of 1.019 to 1.090 kg/L and stored at −80°C until analysis. Plasma LDL (0.25 mL) was mixed thoroughly with the same volume of methanol and hexane to release the α-tocopherol contained within the lipoprotein and to precipitate the protein contained in the sample. The phases were separated by centrifugation. The upper layer was transferred to another tube. The hexane extraction was repeated twice more. The combined hexane extracts of each sample were evaporated under nitrogen, and the residue was re-dissolved in 0.25 mL methanol:dichloroethane (4:1) for the high performance liquid chromatography (HPLC) injection.

An aliquot of 0.05-mL sample was analyzed by HPLC using a Rainin Microsorb 3 μm C18, 15-cm column, with 100% methanol as the mobile phase and absorbance detection (292 nm) to quantitate standards and samples. Linear regression between the peak area and the amount of sample injected was used for quantitation. The slope of the standard curve was used to calculate the final concentration of α-tocopherol in samples.

Statistical analysis

Analysis of variance was used to test significant differences between cellulose, PE, and PSY effects on plasma cholesterol, lipoproteins, LDL-TBARS formation, plasma α-tocopherol, hepatic cholesterol, and TAG. The Newman-Keuls test was used as post hoc test to evaluate differences among means. Because there was not a parametric distribution in the calculated values for α-tocopherol in LDL, the Kruskal-Wallis statistic test was used as a post hoc test to evaluate the differences among means. Data are presented as the mean ± SD for the number of animals tested. Differences were considered significant at *P*-values less than 0.05.

Results

Effects of PE and PSY on plasma lipids and lipoproteins

Guinea pigs fed PSY diets gained less weight than those fed PE or cellulose (217 ± 71, 228 ± 56, and 192 ± 103 g for control, PE, and PSY groups, respectively), although these numbers were not significantly different ($P > 0.05$) in this particular study. However, because the PSY group gained significantly less weight in our previous study,[16] we decided to use body weight as a covariant to ensure that the significant differences in the measured parameters persisted. When body weight was used as a covariant, we found that significant differences between control and PSY were present in plasma and hepatic parameters.

Guinea pigs fed PE or PSY had lower plasma cholesterol concentrations than those in the control group. In guinea pigs fed PE, plasma cholesterol concentrations were 36% lower than guinea pigs fed cellulose. PSY enriched diets had stronger hypocholesterolemic properties than either cellulose or PE enriched diets ($P < 0.001$) (*Table 2*). Plasma TAG concentrations were lower in guinea pigs fed PE or PSY compared with the control group ($P < 0.001$); however, PE and PSY had no differences in the hypotriglyceridemic responses (*Table 2*).

By analyzing the effects of dietary treatments on plasma lipoprotein fractions, it was demonstrated that very low density lipoprotein (VLDL) cholesterol concentrations were lower in guinea pigs fed PE and PSY compared with controls ($P < 0.001$). However, PSY was more effective in reducing VLDL cholesterol than PE (*Table 2*). In addition, PE and PSY intake resulted in a reduction in plasma LDL cholesterol that paralleled that which was observed for total cholesterol, with a more pronounced effect associated to PSY consumption (*Table 2*). HDL cholesterol concentrations were not affected by any of the fiber treatments (*Table 2*).

LDL number of molecules was modified in guinea pigs by intake of soluble fiber. Guinea pigs fed PE and PSY had LDL particles with a lower number of cholesteryl ester molecules than guinea pigs fed the control diet ($P < 0.05$; *Table 3*). In contrast, the rest of the LDL components were not affected by any of the treatments.

Table 2 Plasma lipids and lipoproteins of guinea pigs fed diets containing cellulose (control), pectin, or psyllium as the fiber source*,†

Diet	Cholesterol (mmol/L)				TAG (mmol/L)
	TC	VLDL	LDL	HDL	
Control	7.41 ± 1.23[a]	0.15 ± 0.02[a]	7.19 ± 0.92[a]	0.13 ± 0.02[a]	1.64 ± 0.32[a]
Pectin	4.71 ± 1.32[b]	0.09 ± 0.03[b]	4.53 ± 0.81[b]	0.11 ± 0.04[a]	0.76 ± 0.22[b]
Psyllium	2.48 ± 0.73[c]	0.07 ± 0.02[c]	2.58 ± 0.32[c]	0.13 ± 0.03[a]	0.61 ± 0.18[b]

*A detailed description of the diets is presented in *Table 1*.
†Data are presented as mean ± SD for 10 guinea pigs. Values in a column with different superscipts are significantly different as determined by analysis of variance and Newman-Keuls as posthoc test ($P < 0.05$).
TC–total cholesterol. VLDL–very low density lipoprotein. HDL–high density lipoprotein. TAG–triacylglycerol.

Effects of PE and PSY on the susceptibility of LDL to oxidation

LDL susceptibility to oxidation measured by TBARS formation after 3 hours of incubation in the presence of Cu^{2+} was 63% and 50% lower in guinea pigs fed PE or PSY, respectively, than guinea pigs fed cellulose ($P < 0.05$; *Table 4*). No differences between PE and PSY treatments were observed for this parameter.

Both PE and PSY had LDL particles with higher α-tocopherol concentrations than LDL particles isolated from guinea pigs fed cellulose. LDL isolated from guinea pigs fed PE contained 49% higher LDL-α-tocopherol concentrations than animals fed cellulose ($P < 0.05$; *Table 5*). Similarly, guinea pigs fed PSY diets had LDL particles with 66% higher α-tocopherol concentrations than animals fed cellulose ($P < 0.05$; *Table 4*). No significant differences in LDL-α-tocopherol concentrations between PE and PSY were observed.

Effects of PE and PSY on hepatic lipids

Hepatic cholesterol was significantly altered by soluble fiber intake. Lower hepatic total cholesterol was observed in guinea pigs fed PE and PSY (29% and 36%, respectively) compared with cellulose fed guinea pigs (*Table 5*; $P < 0.01$). No differences in hepatic total cholesterol between PE and PSY were observed. Hepatic free and cholesteryl ester concentrations were also lowered by PE and PSY intake compared with controls ($P < 0.05$; *Table 5*).

Guinea pigs fed either PE or PSY had lower hepatic TAG concentrations, on the order of 43% and 23%, respectively, compared with controls ($P < 0.05$; *Table 5*).

Discussion

Effects of PE and PSY on plasma lipids and lipoproteins

This study confirms our previous findings in guinea pigs[15,26,27] and in other animal models[28,29] in which PE and PSY had a beneficial effect on plasma lipids by lowering cholesterol and TAG concentrations. The hypocholesterolemia may be the result of distinct mechanisms between PE and PSY, because PSY had a more pronounced effect in lowering plasma LDL cholesterol concentrations. The specific plasma cholesterol lowering differences between PE and PSY may be due to distinct effects of these fibers on cholesterol absorption,[29] micelle disintegration,[30] or disruption of the enterohepatic circulation of bile acids.[4,31]

The main secondary effect of PE and PSY fibers appears to be related to the reductions in hepatic cholesterol concentrations observed in the present investigation and in other reported studies from our laboratory,[16,17,26,27] as will be discussed below.

PE and PSY had similar hypotriglyceridemic properties in these studies. This TAG lowering is consistent with the hypothesis that soluble fiber may delay the absorption of TAG from the small intestine[32] or may delay glucose and fructose absorption.[33]

Lipoprotein composition also was altered by soluble fiber intake. Cholesteryl ester depleted LDL particles were found in these studies in response to PE and PSY intake. LDL with these features is associated metabolically with faster LDL turnover [34] and negatively associated with increases in the incidence of atherosclerosis in African green monkeys.[35]

Table 3 LDL number of molecules per particle of guinea pigs fed diets containing cellulose (control), pectin, or psyllium as the fiber source*

Diet	LDL number of molecules			
	FC	CE	TAG	PL
Control	172 ± 122	624 ± 266[a]	198 ± 179	228 ± 180
Pectin	106 ± 91	281 ± 102[b]	63 ± 23	133 ± 52
Psyllium	251 ± 239	324 ± 141[b]	162 ± 84	375 ± 255

*Data are presented as mean ± SD for 10 guinea pigs. Values in a column with different superscripts are significantly different ($P < 0.05$) as determined by one-way analysis of variance and Newman-Keuls as post hoc test.
LDL–low density lipoprotein. FC–free cholesterol. CE–cholesterol ester. TAG–triacylglucerol. PL–phospholipids.

Table 4 LDL α-tocopherol concentrations and plasma TBARS of guinea pigs fed diets containing cellulose (control), pectin, or psyllium as the fiber source*

Diet	LDL–α-tocopherol (nmol/L)	TBARS (nmol MDA/non-HDL protein)
Control	1.99 ± 1.18[a]	5.98 ± 3.68[a]
Pectin	3.91 ± 1.20[b]	2.18 ± 1.37[b]
Psyllium	5.78 ± 4.42[b]	3.02 ± 2.05[b]

*Data are presented as mean ± SD for 10, 9, and 7 guinea pigs fed cellulose, pectin, and psyllium, respectively, for the low density lipoprotein (LDL)–α-tocopherol concentrations, and 10 guinea pigs per group in the thiobarbituric acid-reactive substances (TBARS) measurement. Values in a column with different superscripts are significantly different ($P < 0.05$) as determined by one-way analysis of variance and the Newman-Keuls as post hoc test. Values for α-tocopherol were analyzed by the Kruskal-Wallis statistic test for nonparametric distribution. MDA–malonaldehyde. HDL–high density lipoproteins.

Effects of PE and PSY on LDL susceptibility to oxidation

The hypolipidemic mechanisms of soluble fiber are associated with decreases in cholesterol absorption or interruption of the enterohepatic circulation of bile acids due to specific physicochemical properties of dietary fibers associated with micelle disruption and increased viscosity in the intestinal lumen.[3,31] Based on this information, we decided to explore the possible effects of dietary fiber on important lipid soluble micronutrients such as α-tocopherol, an important antioxidant that may be associated with the prevention of atherosclerosis.[36]

Studies conducted in hypertensive patients by Maggi et al.[37] have shown that LDL α-tocopherol depleted particles exhibit enhanced susceptibility to oxidation compared with those from normal subjects, which may explain the incidence of atherosclerosis in hypertensive patients. In addition, studies conducted by Nenseter et al.[15] have demonstrate the antioxidative properties of α-tocopherol in studies performed in rabbits.

In these studies, we observed that guinea pigs fed PE or PSY exhibited LDL particles with higher α-tocopherol concentrations and more resistance to oxidation, as evidenced by the decrease of TBARS formation after incubation in the presence of Cu^{+2}. Taken together, these two observations, along with the hypothesis that LDL cholesteryl ester depleted particles are negatively associated with atherosclerosis,[35] it is reasonable to speculate that the cholesteryl ester depleted LDL obtained from guinea pigs fed PE or PSY may not be a major target for free radical formation. Thus, we can explain the presence of higher α-tocopherol concentrations in these LDL particles compared with the LDL particles obtained from guinea pigs fed cellulose.

In our earlier report,[16] it was demonstrated that PE and PSY lower not only plasma LDL cholesterol concentrations but also plasma apoB levels, which suggests a decrease in the number of LDL particles. It could be that a smaller number of LDL particles containing less cholesterol, as a result of PE and PSY intake, would not be so susceptible to oxidation and plasma α-tocopherol would be spared.

In addition, we suggest that the length of time that LDL remains within the circulation may be another factor associated with the susceptibility of LDL to oxidation. The longer the LDL remains in plasma, the higher the possibility that it will be oxidized. Because PE and PSY increase the number of apoB/E receptors,[16] LDL clearance is possibly increased and therefore there is less opportunity for the LDL particle to become oxidized.

Most studies addressing the mechanisms of LDL oxidation focus on lipid oxidation because it is believed that it occurs prior to oxidative modifications of apoB.[38] The effect of PE and PSY on reducing LDL susceptibility to oxidation could be due to a combined effect on the reduction of LDL cholesterol, the compositional modification observed in LDL, and the higher number of LDL receptors.

Effects of PE and PSY on hepatic lipids

Hepatic cholesterol concentrations were altered by the type of soluble fiber. PE and PSY significantly decreased hepatic cholesteryl ester and free cholesterol. Studies conducted by Huff et al.[39] suggested that decreases in hepatic cholesterol concentrations are associated with reductions in the synthesis of VLDL particles, which may be associated with the lower VLDL and LDL cholesterol fractions observed in guinea pigs fed PE or PSY. We have observed that alterations in hepatic cholesterol homeostasis generated by the intake of soluble fiber will alter the activity of regulatory enzymes involved in cholesterol synthesis and degradation, as well as esterification, and on the number of hepatic apoB/E receptors.[17,26,27]

Guinea pigs fed PE and PSY diets had lower hepatic TAG concentrations than animals fed cellulose. The mechanisms most likely associated with this hepatic TAG depletion may be related to the capacity of soluble fiber to delay sucrose absorption in the intestinal lumen.[32] In addition, Mamo et al.[10] postulated that fructose skips the phospho-

Table 5 Hepatic lipids of guinea pigs fed diets containing cellulose (control), pectin, or psyllium as the fiber source*

Diet	Hepatic lipids (μmol/g)			
	Total cholesterol	Free cholesterol	Esterified cholesterol	Triacylglycerol
Control	12.53 ± 1.33[a]	11.10 ± 1.47[a]	1.06 ± 0.23[a]	16.11 ± 4.90[a]
Pectin	8.80 ± 2.75[b]	7.69 ± 2.73[b]	0.69 ± 0.41[b]	14.72 ± 2.62[b]
Psyllium	7.97 ± 2.57[b]	6.97 ± 2.68[b]	0.72 ± 0.15[b]	10.86 ± 2.43[b]

*Data are presented as mean ±SD for 10 guinea pigs. Values in a column with different superscripts are significantly different ($P < 0.05$) as determined by one-way analysis of variance and the Newman-Keuls as post hoc test.

fructokinase regulatory step in glycolysis, increasing acetyl-CoA concentrations and therefore lipogenesis. Because fructose is one of the components of sucrose, we suggest that PE and PSY, by inhibiting simple CHO absorption, also may decrease hepatic lipogenesis and therefore reduce hepatic TAG concentrations.

Conclusion

From these studies we can conclude that in addition to the plasma and hepatic cholesterol lowering properties of PE and PSY observed in guinea pigs fed high fat–sucrose diets, these sources of soluble fiber introduce another important alternative to prevent atherosclerosis by sparing the α-tocopherol concentrations in LDL and by decreasing the susceptibility of LDL to oxidation. Therefore, PE and PSY have two beneficial effects that could be related to decreased risk for coronary heart disease: (1) the lowering of plasma lipids and (2) the observed increases in LDL resistance to oxidation associated with the α-tocopherol sparing effects.

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