Dietary Pectin with High Viscosity Lowers Plasma and Liver Cholesterol Concentration and Plasma Cholesteryl Ester Transfer Protein Activity in Hamsters

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ABSTRACT We fed semipurified diets containing pectin with either a high or low in vitro viscosity at a level of 3 g/100 g air-dried diet to hamsters for 8 wk. A control group was fed cellulose and a positive control group was fed psyllium. The pectins used were a calcium-sensitive pectin (CS-pectin) that has a high viscosity and a noncalcium-sensitive pectin (NCS-pectin) that has a low viscosity. In the presence of calcium, CS-pectin has a more than 80-fold higher viscosity than NCS-pectin which offered the opportunity to investigate the possible role of viscosity in the hypolipidemic properties of pectin. The hamsters fed CS-pectin or psyllium had considerably lower plasma cholesterol concentrations (3.69 ± 0.44 and 4.21 ± 0.45 mmol/L, respectively, mean ± SD, n = 14) than those fed NCS-pectin (5.03 ± 1.15 mmol/L) or cellulose (5.72 ± 1.04 mmol/L). Differences in total plasma cholesterol were reflected in both high density lipoprotein and very low density lipoprotein cholesterol. There was no effect of fiber on low density lipoprotein cholesterol levels. Liver cholesterol concentrations paralleled the plasma cholesterol levels and were 9.91 ± 2.48 μmol/g of liver for the CS-pectin group, 15.03 ± 5.75 for the psyllium group, 17.69 ± 10.66 for the NCS-pectin group, and 25.57 ± 9.23 for the cellulose group. Fecal bile acid and neutral steroid excretion tended to be higher in the hamsters fed CS-pectin than in their counterparts fed NCS-pectin. The hamsters fed psyllium had significantly greater fecal excretions of bile acids than the hamsters fed cellulose, CS-pectin or NCS-pectin, whereas the excretion of fecal neutral sterols tended to be lower. Plasma cholesteryl ester transfer protein activity was significantly lower in the hamsters fed CS-pectin than in those fed NCS-pectin. The results of this study suggest that the viscosity of pectins may determine their cholesterolemic effect. J. Nutr. 128: 1944–1949, 1998.

KEY WORDS: ✤ hamsters ✤ dietary fiber ✤ pectin ✤ cholesterol ✤ cholesteryl ester transfer protein activity

Dietary pectins can lower plasma cholesterol concentrations (Anderson et al. 1994, Truswell and Beynen 1992). Different types of pectin can vary in their viscosity, and studies in rats have indicated a positive relationship between viscosity and cholesterol-lowering activity (Ebihara et al. 1979, Judd and Truswell 1985). Studies with other fiber sources have also pointed to a possible role of viscosity. Gallaher et al. (1993) reported that feeding hamsters a diet with hydroxypropyl methylcellulose with high instead of low viscosity lowered plasma cholesterol concentrations. Similar results have been obtained for carboxy methylcellulose of low or high viscosity fed to chickens (Smits et al. 1997).

The viscosity of pectin may affect cholesterol absorption and fecal excretion of sterols and bile acids. Dietary fiber with a high viscosity produces intestinal digesta with a high viscosity (Smits et al. 1997). As discussed by various authors (Carr et al. 1996, Gallaher et al. 1993, Judd and Truswell 1985, Smits et al. 1997), a high viscosity of the digesta may interfere with the formation of micelles and/or lowers the diffusion rate of cholesterol and bile acid containing micelles through the matrix of the digesta. Feeding hamsters diets containing hydroxypropyl methylcellulose with high vs. low viscosity lowered cholesterol absorption (Carr et al. 1996). Several studies have indicated that dietary pectin also lowers cholesterol absorption (Fernandez 1995, Hyun et al. 1963, Kelley and Tsai 1978, Kiriyama et al. 1969, Vahouny et al. 1988) although some studies do not support this (Fernandez et al. 1994, Mathé et al. 1977). Moreover, studies in humans showed that dietary pectin increased the excretion of bile acids and neutral sterols in feces (Kay and Truswell 1977, Miettinen and Tarpila 1977, Stasse-Wolthuis et al. 1980). Similar results were reported in hamsters (Sablé et al. 1990).

The objective of the present study was to compare two types of pectin, a so-called calcium-sensitive pectin (CS-pec-
NCS-pectin, noncalcium sensitive pectin. Calcium sensitive pectin; HMG-CoA, 3-hydroxy-3-methylglutaryl coenzyme A; Glore et al. (1995) and hamsters (Daggy et al. 1997). We used psyllium as a positive control fiber because it lowers concentrations in hamsters (Daggy et al. 1997). In addition, because it does not affect plasma and lipoprotein cholesterol activity. In this study we used cellulose as a control fiber. Dietary fibers such as guar gum, psyllium, and pectin that lower cholesterol concentrations in hamsters (Daggy et al. 1997). Fernandez et al. (1997) reported in studies with guinea pigs that dietary fibers such as guar gum, psyllium, and pectin that lower plasma cholesterol concentrations also lowered plasma CETP activity. In this study we used cellulose as a control fiber because it does not affect plasma and lipoprotein cholesterol concentrations in hamsters (Daggy et al. 1997). In addition, we used psyllium as a positive control fiber because it lowers effectively plasma cholesterol levels in humans (Glore et al. 1994) and hamsters (Daggy et al. 1997).

MATERIALS AND METHODS

The experimental protocol was approved by the animal experiments committee of the Faculty of Veterinary Medicine, Utrecht University, The Netherlands.

Animals and diets. Golden Syrian hamsters (56 5-wk-old males) (HsdCpb:ShGa) were obtained from Harlan CPB (3700 AD Zeist, The Netherlands) and were fed a commercial rodent diet (Hope Farms, Woerden, The Netherlands) for 2 wk. The hamsters were subsequently divided into four groups of 14 on the basis of their energy distribution of the diets was: fat, 38%; protein, 17%; carbohydrates, 16.74 kJ per gram; fat, 37.66 kJ per gram. The diets contained 100 mg of cholesterol per 1,974 kJ. The energy distribution of the diets was: fat, 38%; protein, 17%; carbohydrate, 45%. The calculated polysaturated/saturated ratio of the diets was 0.26.

The fibers were added in hydrated form. Fiber (30 g) was mixed into 1 L of water and added to 970 g of the diet ingredients, not including the fibers.

Composition in mg/kg of food: FeSO4; 7H2O, 174; (Fe, 35), MnO2, 79, (Mn, 50) ZnSO4; H2O, 33 (Zn, 12); NiSO4; 6H2O, 13 (Ni, 3), NaF; 2 (F, 1); KI, 0.2 (I, 0.15); CuSO4; 5H2O, 15.7 (Cu, 4); Na2SeO3; 5H2O, 0.3 (Se, 0.10); CrCl3; 6H2O, 1.5 (Cr, 0.30); SnCl2; 2H2O, 1.9 (Sn, 1); NH4VO3, 0.2 (V, 0.1); corn meal (carrier material), 9872.9.

Composition in mg/kg of food: thiamine hydrochloride, 4; riboflavin, 3; nicotinamide, 20; calcium pantothenate (purity 45%), 17.8; pyridoxine hydrochloride, 6; cyanocobalamine (purity 0.1%), 50; choline chloride (purity 50%), 2000; folic acid, 1; biotin, 2; menadione, 0.05; all-rac-α-tocopheryl acetate (purity 50%), 60; retinyl acetate and palmitate (500 IU per mg), 8; cholecalciferol (500 IU per mg), 2; corn meal (carrier material), 9,826.15.

Studies in humans have shown that pectin administered in the form of a gel together with a meal was more effective in lowering plasma cholesterol concentrations than pectin given in a dry form (Keys et al. 1961). The semipurified diets were first prepared without the fiber source. Cellulose (30 g), psyllium, CS-pectin or NCS-pectin was added to 1 L of water. The pectin and psyllium solutions were left overnight at 4°C to allow the fibers to dissolve completely, whereas

TABLE 1

<table>
<thead>
<tr>
<th>Type of pectin</th>
<th>DE2</th>
<th>% AGA3</th>
<th>Molecular weight4</th>
<th>Viscosity5 without added Ca2+</th>
<th>Viscosity5 with added Ca2+</th>
</tr>
</thead>
<tbody>
<tr>
<td>CS-pectin</td>
<td>71</td>
<td>85</td>
<td>13 · 104</td>
<td>13</td>
<td>590</td>
</tr>
<tr>
<td>NCS-pectin</td>
<td>58</td>
<td>86</td>
<td>5 · 104</td>
<td>7</td>
<td>7</td>
</tr>
</tbody>
</table>

1 Data provided by the manufacturer (Copenhagen Pectin, Lille, Denmark).
2 Degree of esterification, i.e., the percentage of galacturonic acid subunits with methyl ester groups.
3 Percentage (wt%) of anhydrous galacturonic acid.
4 Determined by relative viscosity.
5 Viscosity (mPa s) of a pectin solution (5 g/L) in the absence and presence of Ca2+ (7.14 mmol/L, added as CaCl2) at a pH of 4.2.
the cellulose solution was used directly. Then 970 g of powdered semipurified diet without the fiber source was added and mixed into the fiber solution with a kitchen mixer. The dough-like diets were divided into small portions and kept frozen at −20°C. The frozen portions of diet were fed to the hamsters every other day. The food intake of three or four hamsters (we had two cages with three hamsters and two cages with four hamsters per dietary group) per 2 d was measured, and the average food intake per hamster per day for each cage was calculated. The food intakes were averaged for the entire 8-wk experimental period.

**Analytical methods.** Blood was collected into heparinized tubes from the retro-orbital sinus of the hamsters while they were under light ether anesthesia. Food was removed at 1700 h and any food in the cheek pouches was also removed. Blood samples were taken the next day between 900 and 1100 h. The hamsters were anesthetized at the end of the experiment with a mixture of ketamine (140 mg per kg body weight), xylazine (24 mg per kg) and atropin (0.4 mg per kg) that was administered intraperitonally. The hamsters were then exsanguinated from the abdominal aorta and the livers were removed.

Plasma cholesterol (Allain et al. 1974) and triglyceride (Buccolo and David 1973) concentrations were measured enzymatically. Plasma very low density lipoprotein (VLDL) + low density lipoprotein (LDL) were precipitated with phosphotungstic acid/MgCl₂ (Sigma Diagnostics, St. Louis, MO, catalog number 352-4) according to Weingand and Daggy (1990), and the supernatant (HDL) was assayed for cholesterol. The concentration of cholesterol in the VLDL + LDL fraction was calculated as the difference between whole plasma and HDL. At the end of the experiment, plasma samples for each group were pooled and lipoproteins were isolated by density gradient ultracentrifugation (Terpstra et al. 1981). The various lipoprotein fractions were collected from the centrifuge tube by aspiration and the cholesterol concentration was measured.

Livers were homogenized with 15 mL of distilled water in a Turrax blender (Janke & Kunkel, Ika Werke, Staufen, Germany) and 50 μL of the homogenate was added to 500 μL of ethanol containing 6 mL/L of a KOH solution (8.91 mol/L). The mixture was incubated at 37°C for 55 min, after which 1 mL of petroleum ether/diethylther (1:1, v/v) was added. The mixture was thoroughly vortexed and centrifuged. Supernatant (700 μL) was removed and evaporated to dryness under nitrogen. The residue was solubilized in ethanol in a sonification bath and the cholesterol was measured enzymatically as described above.

**CETP.** CETP activity was measured with endogenous plasma lipoproteins as acceptor for cholesteryl esters. Hamster plasma (400 μL) and a tracer amount of cholesteryl (1-14C) oleate-labeled human LDL (1.7 kBq, 81 nmol of cholesteryl ester) were added to a small conical tube and incubated in a shaking waterbath of 37°C. An aliquot of 50 μL was removed from the tube after 4 h of incubation and added to 200 μL of whole human plasma as a carrier. A phosphotungstic acid/MgCl₂ solution (50 μL) (Sigma Diagnostics) was added, and the VLDL + LDL were precipitated. The supernatant (HDL) and 50 μL of the incubation mixture were assayed for radioactivity and the fraction of cholesteryl (1-14C) oleate transferred from LDL to HDL was calculated.

Cholesteryl (1-14C) oleate-labeled LDL was prepared as described previously (Terpstra et al. 1989). Briefly, cholesteryl (1-14C) oleate (Amersham, Bucks, United Kingdom, specific activity of 2.04 TBq/mol) was dissolved in 50 μL of ethanol and added to a mixture of LDL and lipoprotein-free serum from cholesterol-fed hypercholesterolemic rabbits. Lipoprotein-free serum from hypercholesterolemic rabbits has high CETP activity (Son and Zilversmit 1986) which facilitates the incorporation of cholesteryl (1-14C) oleate into LDL. The mixture was incubated in a shaking waterbath of 37°C for 24 h, and the radiolabeled LDL were resiolated by density gradient ultracentrifugation.

**Fecal bile acids and neutral sterols.** Fecal bile acid and neutral sterol excretion were determined after the hamsters had been fed the diets for 3 and 7 wk. The feces from cages with three or four hamsters were collected for 48 h and bile acids were measured per cage. The results were expressed as means ± SD for four cages per dietary group. The fecal neutral sterols were determined in one pool of feces for each dietary group.

Bile acids were determined using an enzymatic method (Mashige et al. 1976). This procedure only measures the 3α-hydroxy bile acids which compromise about 60% of the fecal bile acids in the hamster (Daggy et al. 1997). Feces were lyophilized and 50 mg of the lyophilized feces was added to a tube containing 1 mL of a mixture of t-butanol of water (1:1, v/v) (van der Meer et al. 1985). The tubes were vigorously vortexed, incubated for 20 min in a shaking waterbath at 37°C, and centrifuged for 2 min. The supernatant was decanted into another tube and the 3α-hydroxy bile acids were measured (Mashige et al. 1976).

Fecal cholesterol, coprostanol and epicoprostanol, which comprise about 75% of the cholesterol-derived neutral sterols in the feces of the hamster (Daggy et al. 1997), were determined by gas chromatography. Lyophilized feces (50 mg) was added to a tube containing 700 μL methanol and 220 μL of a NaOH solution (5 mol/L); 5α-cholestane was used as internal standard. The mixture was vigorously vortexed and incubated for 2 h in a shaking waterbath at 80°C. NaCl was added after cooling to prevent gel formation, and the neutral sterols were extracted with three times 3 mL of petroleum ether (60–80). The extracts were evaporated to dryness and the sterols were silylated as described by Setchell et al. (1983). The sterols were analyzed on a Hewlett-Packard (HP 5890) (Palo Alto, CA) gas chromatograph equipped with a flame-ionization detector and a capillary column (DB 1701, 30 m, 0.25 i.d., 0.15 μm film thickness). The column was operated at the following temperatures: 50°C, increase of 6°C/min to 250°C, increase of 2°C/min to 280°C, then 40 min at 280°C. Injection temperature was set at 225°C and detector temperature at 325°C. Helium gas was used as carrier at a pressure of 85 kPa. A 1 μL sample dissolved in hexane was injected using a split ratio of 1:100.

**Statistical analysis.** Plasma lipid concentrations and fecal bile acid excretion were measured at different time points. Therefore, we used a two-way (diet and week as factors) Repeated Measures Analysis of Variance (ANOVA) on one variable (lipid concentrations or fecal bile acid excretion) to analyze statistically the diet-induced differences in lipid concentrations at 2, 4, 6 and 8 wk after the beginning of the experiment (wk 0). The other results were analyzed with a one-way (diet as factor) ANOVA on one variable (measured parameter). A multiple-comparison procedure (t test with the Bonferroni adaptation) was used to determine the groups or time points that were significantly different when the ANOVA test indicated a significant effect. Correlations between parameters were statistically analyzed with the Pearson product moment correlation test. The level of significance was preset at P < 0.05. Statistical analyses were done with the SigmaStat® statistical software package (Jandel Corporation, San Rafael, CA).

**RESULTS**

The four diets were fed to the hamsters in the form of a dough which contained the various fiber sources in a hydrated form and were well accepted. The food intake tended to be somewhat lower in the groups of hamsters that were fed the two types of pectin (Table 3). Final body weights were not significantly different.

On wk 0, the hamsters were transferred from the commercial diet to one of the four semipurified diets. The hamsters fed CS-pectin had plasma cholesterol levels that were significantly lower than those in the hamsters fed NCS-pectin and that were not different from those in the hamsters fed psyllium (Fig. 1).

Plasma HDL cholesterol concentrations paralleled total plasma cholesterol levels as did VLDL + LDL cholesterol concentrations (Fig. 1). The proportion of HDL cholesterol was negatively correlated with the plasma total cholesterol concentrations (r = −0.69, P < 0.001, n = 56) when all hamsters were considered. Isolation of VLDL, LDL and HDL from pooled plasma samples (one pool per dietary group) by ultracentrifugation indicated that group differences in plasma total cholesterol concentrations were reflected in HDL and
VLDL cholesterol (Fig. 2). LDL cholesterol concentrations did not differ among the dietary groups.

Plasma triglyceride concentrations increased when the hamsters were transferred from the commercial diet to the semipurified diets (Fig. 1). The effects of dietary fiber source on plasma triglyceride concentrations were similar to those on plasma cholesterol concentrations. The cellulose-fed hamsters had the highest triglyceride concentrations and the CS-pectin group the lowest (P < 0.05). The concentrations of cholesterol in the liver paralleled the concentrations of cholesterol in the plasma (Table 3). The lowest liver cholesterol concentrations were found in the hamsters fed the CS-pectin that also had the lowest cholesterol concentration in the plasma, whereas the cellulose-fed hamsters that had the highest cholesterol concentration in the plasma also had the highest amount of liver cholesterol (P < 0.05). A positive correlation existed between the concentrations of cholesterol in the plasma and that in liver (r = 0.81, P < 0.01, n = 56).

The hamsters fed the diet containing CS-pectin tended to have a higher excretion of fecal bile acids than the hamsters fed the NCS-pectin (P = 0.054) (Fig. 3). The excretion of neutral sterols in the CS-pectin-fed hamsters was also higher than in the NCS-pectin-fed hamsters, but this difference could not be tested statistically because neutral sterols were measured in one pool of feces per dietary group. The hamsters fed psyllium had significantly higher excretion of bile acids than the other three dietary groups (P < 0.05), but fecal excretion of neutral sterols was lowest in this group.

Plasma CETP activity was significantly lower in the hamsters fed CS-pectin compared with those fed NCS pectin (Table 3). CETP activities in the groups fed cellulose, psyllium and CS-pectin were not significantly different.

**DISCUSSION**

CS-pectin, which has a high in vitro viscosity, was more effective in lowering plasma cholesterol concentrations than was NCS-pectin, with a lower in vitro viscosity. Further, the feeding of CS-pectin vs. NCS-pectin was associated with a tendency toward a higher excretion of bile acids and neutral sterols. Thus, the viscosity of pectin may determine its cholesterolemic properties, which may be mediated by raising the excretion of fecal bile acids and neutral sterols. A higher excretion of bile acids and neutral sterols may be the result of a higher viscosity of the intestinal contents; this interferes with the formation of micelles and/or lowers the diffusion rate of bile acid and cholesterol-containing micelles through the viscous matrix of the digesta. As a consequence, the mucosal uptake of cholesterol and bile acids is diminished (Carr et al. 1996, Gallaher et al. 1993, Judd and Truswell 1985, Smits et al. 1997).

We used psyllium as a positive control and found that it lowered plasma cholesterol almost as effectively as did CS-pectin. Both psyllium and pectin are soluble fibers with a high in vitro viscosity, but the mechanism of the cholesterol-lowering effect might be different. Psyllium instead of CS-pectin in the diet raised fecal bile acid excretion and tended to lower the excretion of neutral sterols by the hamsters. Studies in humans (Miettinen and Tarpila 1989) and hamsters (Daggy et al. 1997) have shown that psyllium enhanced the excretion of fecal bile acids but not that of neutral sterols. Moreover, psyllium did not affect cholesterol absorption in studies with humans (Everson et al. 1992, Miettinen and Tarpila 1989, Stanley et al. 1973), monkeys (McCull et al. 1992), guinea pigs (Fernandez 1995) and hamsters (Turley et al. 1994).

Psyllium specifically increases the excretion of bile acids. Thus, a mechanism involving a decreased mixing of the intestinal digesta through an increased viscosity, as suggested for pectin, seems unlikely since it would inhibit cholesterol absorption and raise excretion of neutral sterols. Daggy et al. (1997) reported that ~90% of the bile acids in hamsters is excreted as unconjugated bile acids and that psyllium mainly increases the excretion of unconjugated bile acids. This indicates that psyllium does not bind bile acids in the small intestine as is the case with cholestyramine which predominantly increases the fecal excretion of conjugated bile acids (Daggy et al. 1997). The lower excretion of neutral sterols by hamsters after consuming psyllium rather than pectin could be due to a better absorption of dietary cholesterol when psyllium is fed, because in the case of pectin feeding, cholesterol might be entrapped in the small intestine. Hepatic bile acid synthesis is increased after feeding psyllium and cholesterol (Matheson et al. 1995), possibly accounting for the increased fecal bile acid output observed in our study.
Fermentation of soluble fibers may influence bile acid metabolism by stimulating deconjugation of bile acids. Studies in chickens have indicated that feeding rye, which is rich in soluble fiber, results in an increased microbial cholyltauryl hydrolase activity in the intestine (Feighner and Dashkevicz, 1988), possibly caused by an increased intestinal fermentation activity because of the presence of a fermentable fiber source (Smits et al. 1997). Further, intestinal fermentation products of soluble fibers, short-chain fatty acids, may be responsible for the cholesterol-lowering effect by lowering the cholesterol synthesis in the liver (Chen et al. 1984). In vitro studies have indicated that incubation of liver slices with propionic acid lowers 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase activity (Bush and Milligan 1971). On the other hand, feeding trials have shown that dietary pectin increases the activity of HMG-CoA reductase activity in the liver (Fernandez et al. 1994). Moreover, pectin administered intracecally in pigs does not lower serum cholesterol levels (Ahrens et al. 1988), and cecal infusion of propionate in pigs does not lower plasma cholesterol concentrations (Beaulieu and McBurney 1992). In addition, pectin still lowers serum cholesterol concentrations in germ-free mice (Komai and Komura 1987). These findings suggest that intestinal fermentation products probably do not play a major role in the cholesterol-lowering properties of dietary pectin.

A study in guinea pigs fed different fibers such as cellulose, pectin, psyllium and guar gum has indicated a direct association between plasma cholesterol levels and plasma CETP activity (Fernandez et al. 1997). In this study we found that

**FIGURE 1** Plasma concentrations of total cholesterol, high density lipoprotein (HDL) cholesterol, very low density lipoprotein (VLDL + LDL) cholesterol, and triglycerides in hamsters fed semipurified diets containing either cellulose, psyllium, calcium-sensitive pectin (CS-pectin) or noncalcium sensitive pectin (NCS-pectin). The results are means ± SD, n = 14. Dietary groups and time points that do not share a superscript are significantly different (P < 0.05).

**FIGURE 2** Cholesterol concentrations in high density lipoproteins (HDL), low density lipoproteins (LDL) and very low density lipoproteins (VLDL) of hamsters fed semipurified diets containing cellulose, psyllium, calcium-sensitive pectin (CS-pectin) or noncalcium sensitive pectin (NCS-pectin). The results are from pooled plasma samples (14 hamsters per group) taken after the hamsters had been fed the diets for 8 wk. The recovery of cholesterol in the lipoprotein fractions was 104 ± 4% (mean ± SD, n = 4).

**FIGURE 3** Excretion of fecal 3α-hydroxy bile acids (which comprise about 60% of the total fecal bile acids), fecal neutral sterols (cholesterol, coprostanol and epicoprostanol, which comprise about 75% of the cholesterol-derived neutral sterols) and fecal total steroid excretion (the sum of the bile acid and neutral steroid excretion) in hamsters fed semipurified diets containing cellulose, psyllium, calcium-sensitive pectin (CS-pectin) or noncalcium sensitive pectin (NCS-pectin). The bile acids data are means ± SD of four feces pools per dietary group and those of the neutral sterols represent one pool per dietary group. Each dietary group had 14 hamsters. Fecal bile acid excretion of dietary groups and time points that do not share a superscript are significantly different (P < 0.05).
hamsters fed CS-pectin instead of NCS-pectin had lower plasma cholesterol levels and also significantly lower CETP activities. There were, however, no significant differences in CETP activity between the hamsters fed cellulose, psyllium, and CS-pectin, whereas these groups had different plasma cholesterol concentrations. The conclusion is that in hamsters the cholesterolemic effects of different fibers studied are not directly related to their effects on CETP activity.

LITERATURE CITED


