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Metabolism and Physiological Effects of Pectins

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Non-digestible dietary carbohydrates recently have received much attention. Although most of this attention has focused on the importance of cereal sources of dietary fiber, poorly digested pectic substances derived primarily from fruit and vegetable sources likewise may be of significance in relation to human health.

Pectic substances are complex, colloidal carbohydrate derivatives which occur in or are isolated from plants. They contain a large proportion of anhydrogalacturonic acid units, most likely combined in a chain-like arrangement. The carboxyl groups of the polygalacturonic acids may be partly esterified or may form salts with various cations (1, 2). Pectin is a general term usually employed to designate water-soluble pectinic acids of varying methyl ester content and degree of neutralization capable of forming gels with sugar and acids (1).

The amount of pectic substances in several common fruits and vegetables is shown in Table I. Estimation of the total amount of pectic substances in an average diet is not possible because data are available only for a limited number of foods. Comparison of the content of pectic substances in various foods is further complicated by differences in analytical methods and incomplete descriptions of the foods. In addition to pectic substances found naturally in plant foods, pectin may be added to foods during processing. The most common use of pectin is in making jams and jellies; however pectin is an acceptable additive in a number of other foods. Pectins also have found numerous uses in the preparation of various pharmaceutical products.

Much of what is known about the physiological effect of pectin has evolved as a consequence of interest in the use of pectin for therapeutic purposes. Pectin and combinations of pectin with other colloids have been used extensively to treat diarrheal diseases, especially in infants and children (3 - 6).

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This use of pectin had its origin in the treatment of diarrhea with a diet of scraped apples, a home-remedy practiced for hundreds of years in Europe and introduced into this country in 1933 by Birnberg (7).

The effectiveness of pectin in treating diarrhea subsequently led to investigations to determine the mechanism of its effect and its fate in the alimentary tract. Experiments in dogs showed that when 20g pectin was fed in combination with a mixed diet, only 10 percent of the pectin could be recovered in the feces; however, if the same amount was fed during fasting an average of 50 percent was excreted (Table II). Results obtained with humans fed 50g of pectin daily with a mixed diet were similar to those in dogs with approximately 90 percent apparent decomposition of pectin taking place (8, 9). The degree of decomposition appears to be influenced by the retention time in the intestine, adjustment of the animal to the diet, and the degree of esterification of the pectin (9, 10).

Tests made with human subjects and dogs indicated a lack of enzymes in saliva and gastric juice which could act on pectin. Likewise, trypsin, pepsin and rennet had no effect on pectin *in vitro*; however pectin incubated with feces was rapidly decomposed (11). Results of studies in animals and humans with ileostomies indicated that the breakdown of pectin occurs chiefly in the colon most likely by the action of bacterial enzymes (9). Isolation of the microorganisms which are capable of decomposing pectin revealed that the most active groups were Aerobacillus, Lactobacillus, Micrococcus and Enterococcus (12, 13). The chief products formed during bacterial fermentation are carbon dioxide and formic and acetic acid. If galacturonic acid is produced it apparently is broken down rapidly since only a small amount is present in incubation mixtures. Although a bactericidal action of pectin has been proposed to explain the effectiveness of pectin in treating diarrhea, most experimental results do not support this theory (14, 15). However, recent evidence suggests that under certain *in vitro* conditions, pectins may have a slight antimicrobial action toward E. coli (16).

Most of the recent interest in pectin has been in relation to its effect on lipid metabolism. In 1957 Lin and coworkers (17) first reported that in rats the addition of pectin to a basal diet containing cholesterol increased the excretion of fecal saponifiable and non-saponifiable lipids and decreased the absorption of exogenous cholesterol. About the same time Keys and coworkers (18) proposed that the lower incidence of atherosclerosis associated with "Italian-type diets" might be related to the amount of complex carbohydrates such as pectin, hemicellulose and fiber present in the fruits and vegetables abundant in these diets. Subsequent investigations have thus focused on the effect of pectin on serum and liver cholesterol concentrations and the fecal excretion of lipid and sterols.

Numerous experiments in rats (Table III) have shown that

TABLE I

PECTIN CONTENT OF VARIOUS FOODS

Food		Pectic substances
		%
Apples	fresh basis	0.5 - 1.6
Bananas	" "	0.7 - 1.2
Peaches	" "	0.1 - 0.9
Strawberries	" "	0.6 - 0.7
Cherries	" "	0.2 - 0.5
Green peas	" "	0.9 - 1.4
Carrots	dry matter basis	6.9 - 18.6
Orange pulp	" " "	12.4 - 28.0
Potato	" " "	1.8 - 3.3
Tomato	" " "	2.4 - 4.6

Adapted from "The Pectic Substances" (2).

TABLE II

RECOVERY OF PECTIN FROM FECES BY URONIC ACID ESTIMATION

	Per Cent
Pectin fed with basal diet:	
dogs	8.9
humans	8.7
Pectin fed during fasting:	
dogs	51.2
humans	13.8

Adapted from Am. J. Dig. Dis. (9).

TABLE III

Effect of Pectin on Plasma and Fecal Lipids in Rats[1]

Dietary Cholesterol	Dietary Pectin	No. Days on diet	Plasma	Liver		Fecal			Ref
			Cholesterol	Cholesterol	Total Lipids	Total Lipids	Sterols	Bile Acids	
50 mg/day	500 mg/day	6				↑	↑		(17)
0	5%	42	0	0	0				(19)
1%	2.5%	28	↓	↓	0				
1%	5%	28	↓	↓	↓				
1%	5%	28	↓(NS)[2]	↓	↓				(20)
1%	10%	28	↓(NS)	↓	↓				
1%	5%	28	↓	↓	↓(NS)		0	↑	(22)
1%	5%	21	↓	↓	↓				
1%	5%	28	↓(NS)	↓	↓				
1%	5%	28	0	↓	0				(23)
1%	10%	28	↓(NS)	↓	↓(NS)				
1%	5%	28	↓	↓	↓				(25)
0	3%	18	0						(36)
1%	3%	18	↓						
0	10%	12	↓(NS)			↑	↑		(33)

[1]Results expressed as increase or decrease compared to results in rats on same diet without pectin

[2]NS = no statistically significant difference

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addition of 3 to 10% pectin to a diet containing 1% cholesterol counteracts the increase in liver cholesterol and liver total lipid induced by cholesterol feeding (19-25). A decrease in serum cholesterol is usually observed as a result of pectin supplementation although in some experiments the decrease has not been statistically significant. Protopectin and pectins with a low methoxy content do not appear to be effective in lowering serum or liver cholesterol. Supplements of tomato pectin have been reported to produce a smaller decrease in liver cholesterol than citrus pectin (25); however citrus pectin N.F. and apple pectin apparently are equally effective (19).

An anti-hypercholesterolemic action of pectin has also been reported in chickens (26-28) and swine (29). In chickens the addition of 3 to 5% pectin to the diet caused a marked increase in fecal excretion of cholesterol and total lipids irrespective of the cholesterol content of the diet (26, 27). In guinea pigs and hamsters pectin apparently does not have a cholesterol-lowering effect (30). Although long-term feeding of pectin led to a significant decrease in plasma cholesterol in male, but not female, rabbits (31), short-term feeding had no effect (30).

In most experiments pectin has been found to produce an effect on cholesterol and lipid concentrations only in animals receiving added dietary cholesterol (19, 29, 32). However, Mokady (33) has recently reported that in short term feeding experiments in rats, substitution of 10% pectin for starch in a cholesterol-free diet caused a five-to ten-fold increase in fecal total lipids and doubled or tripled sterol excretion. A small decrease in serum cholesterol was observed but results were significant for only one of the pectins tested, a high molecular weight citrus pectin (Table IV).

Few investigations have been conducted in humans to evaluate the effect of pectin supplementation. Keys and coworkers (18) fed middle-aged men controlled diets of natural foods with and without addition of 15g daily of either cellulose or pectin. A three-week period of pectin supplementation resulted in a fall in the mean concentrations of serum cholesterol to levels approximately 5% below the level on the same diet without pectin (Table V). Cellulose supplementation, however, failed to show any significant effect on serum cholesterol concentrations. In a study by Palmer and Dixon (34) an attempt was made to determine the effective dose of pectin which is required to reduce blood cholesterol concentrations. Sixteen men were fed varying amounts of pectin during six 4-week test periods. Other dietary variables and risk factors associated with atherosclerosis were not controlled. Although not all subjects responded equally well to pectin supplementation, daily doses of 8 to 10g pectin caused a significant decrease in serum cholesterol of these men, all of whom had initial values which were either normal or only slightly elevated.

Evaluation of the effect of dietary pectin on plasma and fecal lipids in humans was made in an investigation conducted at

TABLE IV

EFFECT OF PECTIN ON BLOOD CHOLESTEROL AND FECAL LIPIDS IN RATS FED A CHOLESTEROL-FREE DIET

	Relative[1] values for		
	Blood Cholesterol	Fecal Lipids	Total Fecal Sterols
	%	%	%
Control	100	100	100
Low MW Pectin[2]	91	458	278
High MW Pectin	76	735	372
Low M Pectin	86	390	222
Pectin S	83	553	270
Pectin MR	83	478	293

[1]Average values for 8 rats/group expressed as a percentage of control values (for animals fed a pectin-free diet)

[2]MW = molecular weight; M = methoxy; S = slow setting; MR = medium-rapid setting

Adapted from Nutr. Metabol. (33).

TABLE V

MEAN SERUM CHOLESTEROL CONCENTRATIONS IN 24 MEN FED 15gPECTIN/DAY

Major source of dietary carbohydrate	Serum cholesterol, mg %		
	No Pectin	+ Pectin	Δ
Legumes	202.4	192.7	-9.7
Sucrose	221.5	211.3	-10.2

Adapted from Proc. Soc. Exper. Biol. and Med. (18).

the University of Iowa (35). During an initial period of 4 weeks three healthy male subjects were fed controlled diets having a composition similar to that of the average American diet. During a second 5-week period the men received the same diet plus 20 to 23g pectin N.F. which was incorporated into the foods in the diet. The final 5 weeks represented a second control and recovery period. Results showed that after pectin feeding there was a significant decrease of 13% in the mean plasma cholesterol compared to the value for the control period (Table VI). No appreciable effect on the plasma triglyceride level was observed. During the period of pectin ingestion the men showed an increase in total fecal fat, stool volume, fecal sterol and fecal digitonide precipitable sterols.

Several mechanisms have been proposed by which dietary pectin may lower plasma and liver concentrations of cholesterol in various species. Possible mechanisms would include: 1) reduction in cholesterol absorption; 2) alteration in intestinal microflora and 3) depression of bile acid absorption or recirculation. Experimental results reported by Leveille and Sauberlich (22) indicate that the most important effect of pectin in the rat appears to be its influence on bile acid absorption. In cholesterol-fed rats the addition of pectin increased fecal bile acid excretion by 32%, whereas fecal neutral sterol excretion was not altered (Table VII). Additional experiments with inverted intestinal sacs demonstrated that pectin decreased *in vitro* taurocholic acid transport by 50%.

Further support for the importance of the inhibition of bile acid absorption was provided by the similarity in response of cholesterol-fed rats to pectin and cholestyramine, a known inhibitor of bile acid absorption (Table VIII). More recently, however, Phillips and Brien (36) suggested that there may be different mechanisms for the action of pectin and cholestyramine since in their experiments pectin did not affect vitamin A absorption whereas cholestyramine is known to limit absorption of the vitamin.

Dietary pectin somewhat decreases absorption of cholesterol-4-^{14}C in cholesterol-fed rats, as evidenced by decreased deposition of radioactive cholesterol in the liver and increased fecal excretion of cholesterol-4-^{14}C (22). However, impaired cholesterol absorption induced by dietary pectin apparently is only partially responsible for the hypocholesterolemic effect of pectin. Since pectin effectively lowers liver cholesterol even when cholesterol and pectin are fed separately on alternate days (19, 22), impaired absorption of the added cholesterol would not appear to be the most critical means by which pectin exerts its effect. In rats fed a cholesterol-free diet Mokady (37) recently reported that hepatic biosynthesis of cholesterol, as measured by conversion of acetate-1-^{14}C to cholesterol, was substantially higher in rats fed 10% pectin. *In vitro* incorporation of label into triglycerides, phospholipids and total lipids in the liver was also significantly higher in the pectin-fed animals. The

TABLE VI

CHANGES IN PLASMA AND FECAL LIPIDS OF THREE MEN IN RESPONSE TO PECTIN SUPPLEMENTATION

	Periods		
	I	II	III
	Basal Diet	Basal Diet + 20-23 g Pectin	Basal Diet
Plasma cholesterol, mg %	226 ± 6.8[1]	196 ± 9.9[2]	211 ± 8.4
Plasma, triglycerides, mg %	84 ± 12.8	90 ± 10.9	98 ± 9.5
Total fecal fat, g/24 hr	3.4 ± 0.31	5.1 ± 0.41[2]	3.2 ± 0.41
Fecal sterols, g/24 hr	1.06 ± 0.12	1.23 ± 0.12[3]	0.82 ± 0.15
Fecal digitonide precipitate, mg/24 hr	69 ± 8.3	126 ± 12.4[2]	91 ± 18.5

[1]Mean ± standard deviation for 3 subjects.

[2]Differs significantly from mean values for Periods I and III ($p < 0.05$).

[3]Differs significantly from mean value for Period III ($p < 0.05$).

Unpublished data adapted from (35).

TABLE VII

EFFECT OF PECTIN SUPPLEMENTATION IN CHOLESTEROL-FED RATS

	Cholesterol (1%)	Cholesterol (1%) + pectin (5%)
Plasma cholesterol, mg/100 ml	128 ± 3[1]	116 ± 5
Liver fat, %	7.4 ± 0.3	6.6 ± 0.3
Liver cholesterol, mg/g	10.3 ± 0.5	7.5 ± 0.3
Fecal sterols, mg/day	142 ± 5[2]	141 ± 8
Fecal bile acids, mg/day	17.9 ± 2.4[2]	23.6 ± 3.3

[1]Mean ± SE of mean for 10 rats.

[2]Values are means for 5 animals.

Adapted from J. Nutr. (22).

TABLE VIII

COMPARISON OF THE EFFECT OF PECTIN AND CHOLESTYRAMINE IN CHOLESTEROL-FED RATS

Dietary treatment	Liver		Plasma Cholesterol
	Total Lipid	Cholesterol	
	%	mg/g	mg/100 ml
1% cholesterol	7.9 ± 0.3	10.3 ± 0.5	128 ± 14
1% cholesterol + 5% pectin	7.1 ± 0.4	7.2 ± 1.1	91 ± 4
1% cholesterol + 1% cholestyramine	5.8 ± 0.2	4.0 ± 0.2	86 ± 4

[1]Mean for 5 rats ± SE of mean

Adapted from J. Nutr. (22).

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author suggested that the higher rate of hepatic lipogenesis observed in the pectin-fed rats might be due to a reduction in the absorption of dietary fat, since absorbed lipid is known to inhibit hepatic fatty acid synthesis.

The effect of dietary pectin on the number and types of intestinal microorganisms has not been thoroughly investigated; however, the failure of antibiotics to prevent the lowering of blood cholesterol by pectin (19, 22) indicates that intestinal microflora do not contribute significantly to its effect.

The effect of pectin on the absorption of nutrients other than lipids has received little attention. In rats the utilization of β-carotene and absorption of vitamin A was not impaired by pectin supplementation (36). Addition of 12 or 24% pectin to diets of rats has been found to decrease the digestibility of protein (38). Viola and coworkers (39) reported that slow setting pectin (55% esterified) decreased the apparent digestibility of protein but did not impair its utilization. However, addition of 10% medium-rapid-acting pectin (65% esterified) led to an impairment of protein utilization as shown by a decrease in weight gain per gram of digested protein. Both pectin preparations decreased the apparent retention of calcium by approximately 30%.

In summary, experimental results in various species indicate that ingested pectin is nearly completely broken down in the colon most likely by bacterial enzymes. The products formed apparently are not extensively utilized since pectin makes a neglible contribution to the energy value of the diet (39). The digestibility and utilization of pectin, however, needs to be re-evaluated using more sensitive and specific methods. The lowering of plasma and liver cholesterol concentrations by pectin supplementation appears to be related primatily to its effect on bile acid absorption. Results showing an increased excretion of bile acids would suggest the possibility of an increased hepatic conversion of cholesterol to bile acids thus reducing serum and liver cholesterol concentrations. By removing the feedback inhibitor of cholesterol on HMG-CoA reductase these changes would explain the increase in hepatic biosynthesis of cholesterol which has been observed. Further investigations are needed to evaluate cholesterol synthesis and turnover as well as the activity of enzymes important in the regulation of cholesterol synthesis.

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