Lectin May Contribute to the Atherogenicity of Peanut Oil

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ABSTRACT: Peanut oil is unexpectedly atherogenic for rats, rabbits, and primates. The lesions it produces are more fibrous than fatty. The mechanism underlying the atherogenicity of peanut oil has been elusive. Randomization of peanut oil reduces significantly its atherogenic properties, but native and randomized peanut oils have similar rates of lipolysis, and rats fed the two oils absorb and transport lipids in a similar fashion. Peanut oil differs from other oils in having a relatively high lectin content, and the randomization process markedly reduces the lectin content as well. The biologically active lectin of peanut oil has an affinity for glycoproteins found specifically on arterial smooth muscle cells. Peanut lectin has been shown to stimulate growth of smooth muscle and pulmonary arterial cells. Vigorous washing of peanut oil reduces its lectin content by 46%. Compared to rabbits fed cholesterol and peanut oil, rabbits fed cholesterol and washed peanut oil exhibited less severe atherosclerosis in the aortic arch (by 9%) and in the thoracic aorta (by 31%). The data suggest that peanut oils' endogenous lectin may contribute significantly to its atherogenic properties.

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Peanut oil has been shown to be unexpectedly atherogenic when fed to rats (1,2), rabbits (3), or monkeys (4,5) on an atherogenic diet. Peanut oil is more atherogenic than corn oil even when fed as part of a cholesterol-free atherogenic diet (6). Steiner and Dayton (7) found that a diet containing 50–75% ground peanuts was hypercholesterolemic and hyperlipoproteinemic for rabbits, resulting in some aortic sudanophilia. We found that when peanut oil was randomized (autointeresterified) it became significantly less atherogenic for rabbits (8) and vervet monkeys (5). The aortic lesions of the rabbits fed peanut oil contain a relatively small amount of lipid and are characterized by thick, fibromuscular plaques.

The unexpected atherogenicity of peanut oil and its reversal by randomization could not be ascribed to its triglyceride structure since the five principal triglycerides of native (mol%) and randomized (mol%) peanut oil (listed respectively, as follows) contained oleic and linoleic acids almost exclusively (9): OOO (16.3) and OOL (15.9); OLO (11.8) and OOO (13.1); OOL (9.1) and OLL (9.6); POO (8.0) and OLO (7.9); PLO (7.6) and POO (5.3); OLL (6.6) and LOL (4.8) where O = oleic acid; L = linoleic acid; P = palmitic acid. Our recent studies (10) suggest that the presence of palmitic acid at the *sn*-2 position of a triglyceride increases its atherogenic potential, but the principal fatty acids present at the *sn*-2 position of peanut oil are linoleic and oleic. Lipolysis rates of peanut oil, randomized peanut oil, and corn oil are similar (11). Lymphatic absorption of cholesterol is the same in rats fed native or randomized peanut oil (12,13). We began to investigate the possibility that peanut lectin might predispose this oil to become atherogenic. We found that peanut oil had 22% greater lectin-like activity than soybean oil, 129% more than corn oil, and 57% more than randomized peanut oil (14).

MATERIALS AND METHODS

Male, New Zealand White rabbits (8/group), initial weight 3–3.5 kg, were fed a diet containing 92% commercial ration, 6% oil, and 2% cholesterol. This diet was chosen so that results would be comparable to earlier studies (3,8). After 60 d the rabbits were bled under light anesthesia, then killed, and livers and aortas removed. Serum total cholesterol and triglycerides were analyzed using commercial kits (Sigma, St. Louis, MO). Aliquots of liver were homogenized in chloroform/methanol 2:1 (15), and the lipid extract was analyzed for free and total cholesterol (16) and triglycerides (17). Aortas were graded visually on a 0–4 scale (18).

Peanut oil (10 L) was mixed with 5 L of phosphate-buffered saline (pH 7.3) for 2 h then separated by centrifugation. The washed peanut oil (8 L) was mixed with 1.6 L high- purity water for 2 h then separated by centrifugation. Lectins present in the oils were determined as described previously (14) using the method of Lotan *et al.* (19) in which various concentrations of phosphate-buffered saline extracts of the oils are used to agglutinate human erythrocytes. Presence of lectins in the oils was verified by polyacrylamide gel electrophoresis (20). Lectin-like activity (μg/kg) of the oils used was: corn oil, 24.0; peanut oil, 55.0; and washed peanut oil, 29.5.

RESULTS AND DISCUSSION

Necropsy results are given in Table 1. There were no significant differences in any parameter. Effects of the fats on atherosclerosis are presented in Table 2. Severity of aortic arch atherosclerosis in rabbits fed peanut oil was 17% greater than that seen in corn oil and 10% greater than in rabbits fed

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TABLE 1
Necropsy Results. Rabbits Fed 2% Cholesterol and 6% Corn Oil (CO), Peanut Oil (PNO), or Washed Peanut Oil (WPNO) (for 60 d)[a]

	Group		
	CO	PNO	WPNO
Number	8/8	7/8	8/8
Weight gain (g)	694 ± 126	585 ± 101	691 ± 71
Liver weight (g)	128 ± 6	120 ± 7	125 ± 6
Liver as body weight %	4.36 ± 0.16	4.21 ± 0.19	4.28 ± 0.19
Serum lipids (mmol/L)			
Cholesterol	85.2 ± 8.6	99.9 ± 14.8	92.5 ± 9.0
Triglycerides	3.06 ± 0.68	2.03 ± 0.37	2.55 ± 0.61
Liver lipids (mmol/100 g)			
Cholesterol	1.88 ± 0.12	1.67 ± 0.11	1.89 ± 0.20
Ester %	66.0 ± 2.8	64 ± 3.3	65 ± 4.7
Triglycerides	0.07 ± 0.006	0.05 ± 0.003	0.06 ± 0.003

[a]Data ± SEM.

TABLE 2
Severity of Atherosclerosis in Rabbits Fed 2% Cholesterol and 6% CO, PNO, or WPNO (for 60 d) (graded on a 0–4 scale)

	Group		
	CO (24.0)[a]	PNO (55.0)[a]	WPNO (29.5)[a]
Aortic arch	3.25 ± 0.27	3.79 ± 0.15	3.44 ± 0.18
Thoracic aorta	2.00 ± 0.33	2.71 ± 0.26[b]	1.88 ± 0.26[b]

[a]Lectin-like activity, μg/kg.
[b]$P < 0.05$ using the Mann-Whitney test. All values ± SEM. See Table 1 for abbreviations.

washed peanut oil. Severity of atherosclerosis in the thoracic aorta of rabbits fed peanut oil was 36% greater than in those fed corn oil and 44% greater than in those fed washed peanut oil ($P < 0.05$, Mann-Whitney test). The oils used in this experiment were analyzed for lectin-like activity, which in peanut oil was 129% higher than in corn oil and 86% higher than in washed peanut oil. The presence of lectin-like activity provides a rational reason for the atherogenicity of peanut oil. That activity is reduced significantly during the randomization process as is the oil's atherogenicity.

Sanford and Harris-Hooker (21) showed that peanut lectin stimulated growth of smooth muscle and pulmonary arterial cells but not aortic endothelial cells. The structure of peanut lectin has recently been described by Banerjee *et al.* (22). Peanut lectin exhibits a specificity for D-galactose residues and preferentially for the β-D-Gal(1-3)D-Gal-NAC sequence which occurs on arterial smooth muscle cells. Peanut lectin, in the presence of hypercholesterolemia, could enhance proliferation of arterial smooth muscle cells. Testing the effects of peanut lectin added to atherogenic diets containing other oils would be of interest.

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