Effect of Polydextrose on Absorption of Triglyceride and Cholesterol in Mesenteric Lymph-Fistula Rats (44112)

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Abstract. **In most experimental designs, the inhibitory effect of water-soluble dietary fibers on lipid absorption is evaluated by the decrease in plasma lipid concentration or the increase in fecal lipid output. The aim of this study was to evaluate the acute effect of a water-soluble polysaccharide, polydextrose, on lipid transport to the mesenteric lymph using lymph-fistula rats. The mesenteric lymph duct of rats was cannulated, and an infusion tube was introduced into the duodenum. After recovery, a lipid emulsion containing radioactive triolein and cholesteryl oleate was infused into the duodenum for 8 hr. The tested group was infused with the lipid emulsion containing 5% or 10% polydextrose as dietary fiber. Samples from the lymph-fistula were collected, and the luminal contents and mucosa were collected at the end of infusion. Lymph flow in the mesenteric lymph decreased in the polydextrose group after the infusion. The amounts of both triglyceride and cholesterol remaining in the lumen were greater in the polydextrose group, due to decreased transport of lipid into the lymph. These effects were dose dependent in the 5% and 10% polydextrose groups. The results of this study indicate that polydextrose retarded the transport of triolein and cholesterol into the mesenteric lymph.** [P.S.E.B.M. 1997, Vol 215]

Water-soluble dietary fibers lower plasma levels of triglycerides as well as those of cholesterol, and several factors are involved in this decrease (1–3). One reason for this decrease is inhibitory action of the dietary fibers on lipid absorption (1–3), the mechanism of which has mainly been demonstrated by *in vitro* studies (1, 4, 5). Compared with *in vitro* studies, *in vivo* studies reflect the properties of the dietary fibers in more physiological terms. Most *in vivo* studies, however, are limited to demonstrating increased fecal lipid output caused by the dietary fibers (1, 6–8).

Several previous studies demonstrated effects of water-soluble dietary fibers on lipid absorption using lymph-fistula rats (9–12). In these studies, chronic feeding of dietary fibers delayed the lymphatic transport of cholesterol and triglyceride (10, 11). In acute feeding studies, Hyun *et al.* (9) demonstrated that pectin, a dietary fiber, suppressed lymphatic transport of cholesterol and triglyceride, and Ikeda *et al.* (12) showed the interrelated effects of dietary fibers and lipid components on lymphatic transport of cholesterol and triglyceride.

Polydextrose is a randomly bonded polymer of glucose, sorbitol, and citric acid that is water soluble and is only slightly digested in the small intestine after ingestion (13). It was demonstrated that polydextrose decreased the serum HDL cholesterol level and serum concentration of apolipoproteins A-I and A-II in healthy subjects (14). Several studies on chronic feeding of polydextrose have shown that it has physiological actions of dietary fibers, such as shortening of the transit time in the gastrointestinal tract (15) and influencing brush-border membrane enzymes (16), glucose absorption (16), and morphology of the large intestine (17). The effects of polydextrose on lipid absorption have not been clearly demonstrated.

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The aim of this study was (i) to examine the acute effect of polydextrose on absorption of cholesterol and triglyceride in the mesenteric lymph-fistula rat, and (ii) to reveal the effect of water-soluble dietary fibers on lipid digestion.

Materials and Methods

Surgery. Male Sprague-Dawley rats (280–330 g), fed normal rat chow, were used. Rats were fasted overnight prior to the surgical procedure. A laparotomy was performed under halothane anesthesia and the intestinal lymph duct was cannulated according to the method of Bollman *et al.* (18). A silicon infusion tube was introduced about 2 cm down the duodenum through the fundus of the stomach. The fundic incision was closed by a purse-string suture. Postoperatively, the animals were infused intraduodenally at 3 ml/hr with a saline solution containing 145 m*M* NaCl, 4 m*M* KCl, and 0.28 *M* glucose (19). The animals were allowed to recover for 1 day prior to lipid infusion in restraining cages.

Experimental Plan. Rats were infused with a lipid emulsion in phosphate-buffered saline (PBS) containing 5% or 10% polydextrose (Pfizer Inc., Tokyo, Japan). Control rats were infused with the lipid emusion in PBS without polydextrose. The lipid emulsion contained 0.354 g of triolein (400 μmoles of triolein), 1 μCi of glycerol tri[9,10(n)-^{3}H]oleate (New England Nuclear, Boston, MA), 0.051 g of cholesteryl oleate (78 μmoles of cholesteryl oleate), 10 μCi of [1-^{14}C] cholesteryl oleate (New England Nuclear), 68 mg of egg phosphatidylcholine (78 μmoles), and 570 μmoles of sodium taurocholate in 30 ml. The PBS contained 6.75 m*M* Na_2HPO_4, 16.5 m*M* NaH_2PO_4, 115 m*M* NaCl, and 5 m*M* KCl (pH 6.4). The emulsion was infused at 3 ml/hr for 8 hr. Seven rats were studied in each experimental group: PBS with 5% and 10% polydextrose, and PBS without polydextrose.

On the day of the experiment, stock lipid solutions were mixed. After evaporation, sodium taurocholate dissolved in PBS with or without polydextrose was then added to reach the required lipid concentration, and the mixture was sonicated. Samples of the infusate were analyzed for triglyceride and cholesterol, and radioactivity at the beginning and end of infusion (reproducibility: ±5%).

Lymph was collected into pre-cooled tubes for 1 hr before the lipid infusion was begun. This lymph sample was analyzed as the fasting lymphatic output of lipid. Additional lymph samples were collected 1, 2, 3, 4, 5, 6, 7, and 8 hr after the beginning of lipid infusion. At the end of the lipid infusion, the animal was anesthetized and then sacrificed by exsanguination. The stomach and colon were excised separately after tying off both ends. The small intestine was divided into four segments of equal length by tying. The luminal contents of each segment were collected in conical test tubes for measuring the volume. The lumen of each segment was washed thoroughly with three separate washings, each of 3.0 ml of 10 m*M* sodium taurocholate, and the washings were combined in the conical test tubes. The radioactive lipid recovered in the luminal contents represented the lipid that was infused but not yet absorbed by the small intestine. The lipid from the intestinal segment was then extracted according to the procedure of Folch *et al.* (20). The lipid recovered from the small intestinal wall (mucosa) represented the lipid absorbed, but not yet transported, by the small intestine.

Thin-Layer Chromatography. Thin-layer chromatography (TLC) was used to separate the lipid of the luminal contents into different lipid classes (21). The TLC was performed using silica gel G plates, and the solvent system used was composed of hexane:anhydrous diethyl ether:methanol:glacial acetic acid (40:10:3:1, v/v/v). The lipids were visualized after staining with iodine vapor. The appropriate lipid spots were then scraped into scintillation vials, and 1 ml of absolute ethanol was added before the addition of 10 ml of scintillation cocktail.

Radioactivity Determination. Radioactivity was measured in an aqueous miscible scintillant (Opti-Flour; Packard Instrument Co., Downers Grove, IL). The samples were counted for 5 min in a liquid scintillation spectrometer (460 CD; Packard).

Statistics. The results of lymph flow and lipid output were evaluated by two-way analysis of variance with replication in which orthogonal decomposition for linear comparison was carried out (22). Other data were evaluated by one-way analysis of variance in which multiple comparisons were carried out using the method of least significant difference (22). Differences were considered significant if the probability of the difference occurring by chance was less than 5 in 100 ($P < 0.05$).

Results

Lymph Flow and Luminal Volume. As shown in Figure 1, lymph flow decreased slightly during the first

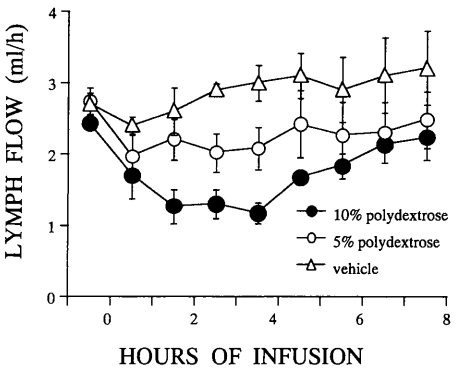

Figure 1. Lymph flow before and after lipid infusion. Values are expressed as the mean ± SEM. Polydextrose decreased the lymph flow in a dose-dependent manner.

Table I. Total Lymph Flow during the 8-hr Lipid Infusion and Luminal Volume of the Small Intestine after the End of the Infusion

	10% polydextrose (ml)	Vehicle (ml)	10% polydextrose − vehicle (ml)
Lymph flow	17.7 ± 0.9	27.1 ± 0.9	−9.4 ± 0.7
Luminal volume	14.6 ± 0.7	9.0 ± 0.9	5.6 ± 0.8

Note. Values are the mean ± SEM.

hour of lipid infusion in vehicle-control rats, and increased from 1 hr onward. Both experimental groups, infused with 5% and 10% polydextrose, had a lower lymph flow than the control animals ($P < 0.05$ in each), and the decrease was more in the rats infused with 10% polydextrose than in those with 5% polydextrose ($P < 0.05$). Table I shows the total lymph flow during the 8-hr experimental period and the total luminal volume in the gastrointestinal tract after the infusion in the control and 10% polydextrose groups. The decrease in lymph flow during the 8-hr infusion in the experimental rats compared with the vehicle-controls was 9.4 ± 0.7 ml, which was more than the increase in the luminal volume in the experimental rats (5.6 ± 0.8 ml).

Lymph Radioactive Lipid Output (Percentage of Hourly Infused). Lymph output of triolein (expressed as a percentage of hourly radioactive triolein infused) is shown in Figure 2. The lymph radioactive triolein output increased steadily in the vehicle-control animals and reached a constant output (about 75%) during the third and eighth hour of lipid infusion. The experimental animals in both the 5% and the 10% polydextrose groups had a significantly depressed lymphatic triolein output compared with the control animals ($P < 0.01$ in each). The decrease in triolein output was more in the 10% group than in the 5% group ($P < 0.05$), indicating that the effect of polydextrose on the triolein output in the

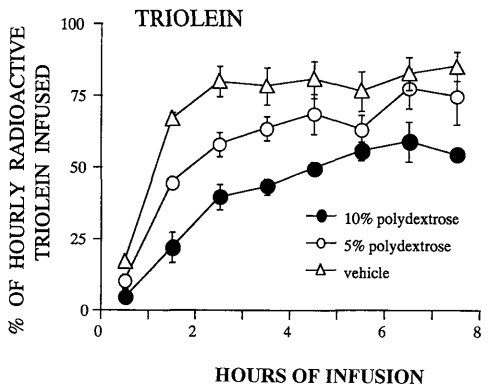

Figure 2. Lymphatic radioactive triolein output. Values are expressed as the mean ± SEM. Polydextrose suppressed triolein output in a dose-dependent manner.

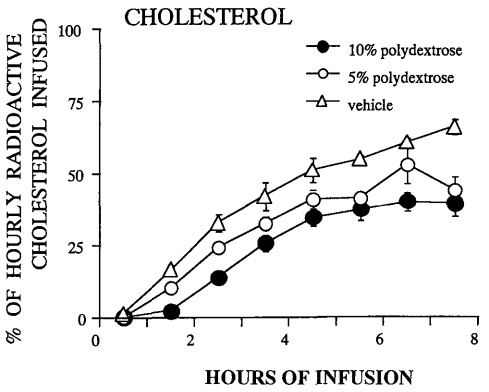

Figure 3. Lymphatic radioactive cholesterol output. Values are expressed as the mean ± SEM. Polydextrose suppressed cholesterol output in a dose-dependent manner.

lymph was dose dependent. The decrease in the total triolein output in the lymph during the 8-hr experimental period was also dose dependent ($P < 0.01$ in each; 42.2% ± 2.3% in the 10% polydextrose group, 55.3% ± 2.4% in the 5% polydextrose group, and 74.3% ± 3.3% in the controls).

Lymph output of cholesterol (expressed as a percentage of hourly radioactive cholesterol infused) is shown in Figure 3. The lymph radioactive cholesterol output increased steadily in the vehicle-control animals during the 8-hr infusion period and reached 70% in output. Lymphatic cholesterol output in the 5% and 10% polydextrose groups decreased significantly during the experiment compared with the controls ($P < 0.05$ in each). The effect of polydextrose was dose dependent, as shown by the finding that the cholesterol output in the 10% dextrose group was less than that in the 5% dextrose group ($P < 0.05$). In regard to cholesterol absorption, the decrease in the total lymph output during the experiment was dose dependent ($P < 0.05$ in each; 24.4% ± 1.2% in the 10% polydextrose group, 30.2% ± 2.5% in the 5% polydextrose group, and 40.3% ± 3.0% in the controls).

Distribution of Radioactive Lipid in the Wall (Percentage of Total Radioactivity Infused). Recovery in the small intestinal wall (mucosa) is shown in Table II. Mucosal recovery at the end of lipid infusion did not differ significantly between the experimental and control groups.

Table II. Distribution of Infused Radioactive Lipid in the Small Intestinal Mucosa (% of Total Dose Infused)

	10% polydextrose	5% polydextrose	Vehicle
Triolein	3.9 ± 0.7	5.5 ± 1.4	4.0 ± 0.5
Cholesterol	22.8 ± 1.8	19.4 ± 1.3	17.0 ± 1.4

Note. Values are the mean ± SEM.

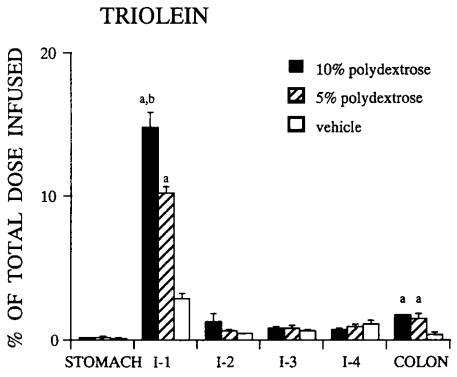

Figure 4. Percentage distribution of infused radioactive triolein in the lumen of the stomach, small intestine, and colon. Values are expressed as the mean ± SEM. Small intestine was divided into four equal segments, with I-1 being the most proximal segment. $^{a}P <$ 0.01, compared with the corresponding vehicle-control value. $^{b}P <$ 0.01, compared with the corresponding 5% polydextrose value.

Distribution of Radioactive Lipid in the Lumen of Different Intestinal Segments (Percentage of Total Radioactivity Infused). We measured the amount of radioactive triolein and cholesterol in the feces excreted during the lipid infusion and found negligible amounts of radioactive isotope in all three groups. Figure 4 shows the distribution of radioactive triolein in the lumen of the stomach and colon, and the four segments of the small intestine. Most of the luminal radioactive triolein was recovered in the proximal first quarter of the small intestine in all three groups. The recovery in the proximal first quarter of the small intestine in both experimental groups was more than the vehicle-control group ($P < 0.01$ in each), and this effect was dose dependent ($P < 0.01$). The recovery of triolein in the colon was more in the two polydextrose groups than in the control group ($P < 0.01$ in each), but recovery in the colon did not differ between the 5% and the 10% polydextrose groups.

Figure 5 shows the distribution of radioactive cholesterol in the lumen of the stomach and colon, and the four segments of the small intestine. In the control rats, the luminal radioactive cholesterol was recovered in all four segments of the small intestine. In the 10% polydextrose group, the recovery of radioactive cholesterol in the lumen was greater in the proximal first and second quarters of the small intestine and in the colon, compared with the control group ($P < 0.05$ in each). In the 5% polydextrose group, the recovery of luminal cholesterol was greater in the proximal first segment of the small intestine compared with the controls ($P <$ 0.05), but the recovery of the 5% group was less than that of the 10% group ($P < 0.05$). The recovery of cholesterol in the colon in the 10% polydextrose group tended to be greater than that in the control group, but this was not a notably significant increase.

The distribution of radioactive lipid among the different lipid classes in the lumen of the first intestinal segment was compared between the 10% polydextrose group and the control group using TLC analysis. The results of triolein are indicated in Figure 6. Most of the infused triolein in the vehicle-control group was hydrolyzed by pancreatic lipase to free fatty acids and partial glycerides at the end of the lipid infusion. Most of the triolein was digested in the 10% polydextrose group, but the recovery in the triglycerides fraction and the diglycerides fraction of the 10% polydextrose group was greater than that of the control group ($P < 0.01$). Recovery in the free fatty acids fraction and the monoglyceride fraction of the 10% polydextrose group was less than that of the control group ($P < 0.01$ in each). These data indicated that the digestion of the 10% polydextrose rats was less efficient than that of the control rats, whereas most of the triolein was digested even in the polydextrose group. In regard to the mucosa, there were no significant differences among the lipid classes (data not shown), indicating that polydextrose did not affect the re-esterification of monoglyceride and free fatty acids in the intestinal mucosa.

Figure 7 shows TLC analysis of distribution in the luminal cholesterol in the first segments at the end of the lipid infusion. The distribution of cholesterol esters and free cholesterol did not differ among the 10% polydextrose group and control group. Further, distribution of the mucosal cholesterol did not differ between the two groups (data not shown).

Total Recovery for Triolein and Cholesterol (Lymph + Lumen + Wall). Total recovery of lipids is shown in Table III. Total recovery of triolein and cholesterol did not differ significantly among the 5% polydextrose, 10% polydextrose, and control groups.

Discussion

The average molecular weight of polydextrose is around 1500, which is less than other natural dietary

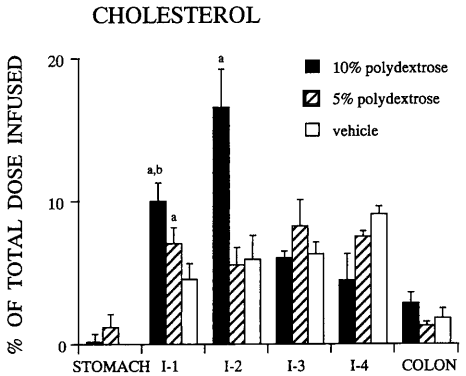

Figure 5. Percentage distribution of infused radioactive cholesterol in the lumen of the stomach, small intestine, and colon. Values are expressed as the mean ± SEM. Small intestine was divided into four equal segments, with I-1 being the most proximal segment. $^{a}P <$ 0.05, compared with the corresponding vehicle-control value. $^{b}P <$ 0.05, compared with the corresponding 5% polydextrose value.

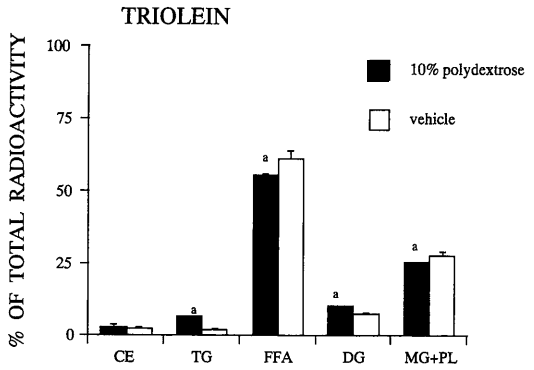

Figure 6. Distribution of radioactivity of infused triolein in different lipid classes in the intestinal lumen of I-1. Values are expressed as the mean ± SEM. Small intestine was divided into four equal segments, with I-1 being the most proximal segment. CE, cholesterol esters; TG, triglycerides; FFA, free fatty acids; DG, diglycerides; MG+PL, monoglyceride + phospholipids. $^{a}P < 0.01$, compared with the corresponding vehicle-control value.

fibers (23). This partially explains why the effect of polydextrose on the increase of fecal weight is less than that for other natural dietary fibers (15, 17). However, polydextrose increases the water-holding capacity of the diet and feces (15, 17), inducing diarrhea with diets containing high levels of polydextrose (15). In the present experiments, we used 5% or 10% polydextrose, which did not cause diarrhea in rats.

The primary inhibitory effects of water-soluble dietary fibers on the absorption of lipids occur through several mechanisms, including interference of emulsification and lipolysis (1–3). In the present experiments, we demonstrated that the lipid emulsion that contained polydextrose, the water-soluble dietary fibers, retarded mesenteric lymph transport of both triolein and cholesterol (Figs. 2 and 3). Several mechanisms can be considered in this acute inhibitory effect of polydextrose on absorption of lipids.

Tso *et al.* demonstrated that a dehydrated matrix retarded the transport of chylomicrons by the small

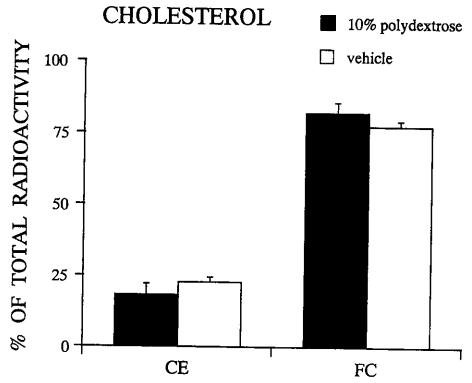

Figure 7. Distribution of radioactivity of infused cholesterol in different lipid classes in the intestinal lumen of I-1. Values are expressed as the mean ± SEM. Small intestine was divided into four equal segments, with I-1 being the most proximal segment. CE, cholesterol esters; FC, free cholesterol.

Table III. Total Recovery (Lymph + Lumen + Wall) for Triolein and Cholesterol (% of Total Dose Infused)

	10% polydextrose	5% polydextrose	Vehicle
Triolein	75.2 ± 2.5	76.4 ± 1.8	79.6 ± 3.1
Cholesterol	77.2 ± 1.9	78.2 ± 3.3	80.2 ± 2.4

Note. Values are the mean ± SEM.

intestine into the lymph (24, 25). In the present studies, the mesenteric lymph flow decreased significantly after infusion of the polydextrose-containing diet, which might contribute to the reduction of lipid transport to the mesenteric lymph (24). Regarding the decrease in lymph flow, the water-holding capacity of polydextrose was important. As shown in Table I, luminal volume of the gastrointestinal tract increased after an 8-hr infusion of the lipid emulsion containing polydextrose. However, the decrease in the lymph flow caused by polydextrose was more than the increase in luminal volume, indicating that factors other than the water-holding capacity of polydextrose contributed to the decreased lymph flow induced by polydextrose.

In regard to nutrient absorption, polydextrose did not inhibit glucose absorption in the rat as previously demonstrated (16). However, in the present experiments, luminal content of triolein and cholesterol, which was not absorbed by the small intestine, was greater in the polydextrose group than in the control group. This increase was mainly observed in the proximal small intestine, suggesting that the absorption of lipids was retarded. In the present experimental design, we demonstrated the delay of lipid absorption, and the same inhibitory effect of the dietary fiber on lipid absorption was observed in other water-soluble dietary fibers, such as guar gum (9) and pectin (10). However, we did not demonstrate an effect of chronic feeding of polydextrose on lipid absorption. This warrants further exploration to obtain sufficient evidence of a decrease in lipid absorption from polydextrose.

The evidence from *in vitro* studies suggested that most dietary fibers can alter the activities of pancreatic enzymes (26). Most of the triolein was digested in both the control and polydextrose groups in the present studies, although there was less digestion in the polydextrose group than in the control group. This result indicated that the inhibitory effect of polydextrose on digestion was not important in retardation of lipid absorption.

In the present study, we demonstrated that a diet containing polydextrose retarded the lymphatic transport of triolein and cholesterol in the mesenteric lymph-fistula rat. This acute effect of polydextrose was mainly due to an inhibitory effect on lipid absorption.

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