Effect of Wheat Bran and Wheat Germ on the Intestinal Uptake of Oleic Acid, Monoolein, and Cholesterol in the Rat

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The effects of fiber-rich wheat bran and wheat germ on the intestinal absorption of dietary cholesterol, free fatty acids, and monoglycerides were studied. Rats were given a test meal containing [^{14}C]oleic acid, [^{14}C]monoolein, and [^{3}H]cholesterol. After a 1-hour digestion period, wheat bran or wheat germ (10% of meal solids) did not significantly modify the gastric emptying of lipids. No effect of wheat bran was evidenced on the amounts of lipids and cholesterol in the intestinal content or the mucosal segments, whereas wheat germ significantly increased the cholesterol in the small intestine content, decreasing its intestinal absorption. Both fractions only slightly influenced the levels of absorbed lipids and cholesterol in the plasma and liver. In vitro *binding measurements showed that the wheat fractions bind only 7% to 15% of both lipids and cholesterol. Results indicate that wheat bran has no direct effect on the mucosal uptake process, whereas wheat germ might decrease the uptake of dietary cholesterol by an as yet unknown mechanism.*

Keywords: Wheat bran; wheat germ; dietary fibers; dietary fats; intestinal absorption

Introduction

Previous studies have shown that adding wheat bran or wheat germ to the diet generally induced a significant decrease in the hepatic storage of cholesterol[1-4] and triglycerides[1,2] in rats. Some other studies have demonstrated that the serum lipoprotein patterns might also be affected in rats[2,5,6] and in humans[7-9] in the presence of the wheat fractions. These data thus pointed out that wheat bran and wheat germ may have long-term effects on lipid metabolism, but the mechanisms involved were not understood until recently.

The presence of wheat bran or wheat germ would result in a decreased lipid uptake by the small intestine mucosa or in a shift in the site of intestinal absorption. Two major mechanisms could be involved in this limiting effect. First, wheat fractions may affect the fat lipolysis process in the stomach[10] and in the small intestine,[10-12] thus reducing the amount of lipolysis products available for mucosal uptake and, in an indirect manner, dietary cholesterol.[10] On the other hand, one may suppose that wheat fractions might directly affect the uptake of fatty acids, monoglycerides, and cholesterol by the intestinal mucosa by interfering either with the micellar phase fats, with the enterocyte surface enzymes, or with the cell re-esterifying enzymes, but no specific work has been dedicated to test this hypothesis. The present study was performed to determine whether wheat bran and wheat germ would affect the uptake of fat lipolysis products and cholesterol by the intestinal mucosa in the rat. Also, the *in vitro* binding of these lipids on both wheat fractions was determined in physiologic conditions.

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Materials and Methods

Enzymatic preparation of radiolabeled lipids

[Carboxyl-^{14}C]triolein (NEN; 98.5% purity, 111.8 mCi/mmol) was used as starting material. Approximately 300 nmol triolein (30 μCi) was added to 10 μl of a 10% arabic gum solution and 5 ml of a 10 mM Tris-HCl buffer at pH 8.5 containing 8 mM taurodeoxycholate. The solution was emulsified by sonication for 30 sec-

onds. The enzymatic reaction was performed at pH 8.5 and 25°C by using a pH Stat titrator with 33 μg porcine pancreatic lipase (300 U/mg) and 13 mg porcine pancreatic colipase (Boehringer Mannheim, Germany). Total lipids present in the reaction mixture were then extracted twice at acidic pH according to the method of Folch et al.[13] The chloroformic lower layer was evaporated to dryness under nitrogen and lipid classes were separated by thin-layer chromatography on silica gel (Ready plastic sheet F 1500, Schleicher et Schuell) by using a development phase heptane/diethyl ether/formic acid (90:60:4, vol/vol/vol). Silicic acid areas of interest were scraped and extracted with chloroform. Re-chromatography of [^{14}C]monoolein (8.4 mCi) and [^{14}C]oleic acid (10.8 mCi) showed a purity of approximately 99%.

Radiolabeled [^{14}C]monoolein (monooleoyl-*rac* glycerol) and [^{14}C]oleic acid (*cis*-9 octadecenoic acid) were incorporated into the lipid mixtures used in animal and *in vitro* experiments.

Animals and diets

Adult male Wistar rats were fed for 4 weeks on a usual high-fat diet.[2] To summarize, rats were fed a diet containing 17% protein, 31% starch, 10.7% sucrose, 3% corn oil, 16.5% lard, 0.5% cholesterol, 4.3% cellulose, 1% vitamin mixture, 7% mineral mixture, and 9% water. The rats were then assigned to the experimental groups. The average weights of the rats of the control group (441 ± 16 g), the wheat bran group (478 ± 17 g), and the wheat germ group (428 ± 17 g) did not differ significantly.

The rats were fasted with free access to 50 g/L glucose 40 hours before the experiment and were kept in restraining cages for the last 18 hours with water ad libitum in order to avoid coprophagy. Each test meal was prepared extemporaneously in a disposable syringe as follows: 521 mg of a lipid mixture (53% of meal energy) was added to 1.5 ml of distilled water containing 13.5 mg NaCl, 800 mg sucrose, and 200 mg bovine serum albumin. The lipid mixture was made of 99% pure oleic acid (350 mg) and 0.50 μCi [^{14}C]oleic acid, 98% pure monoolein (150 mg) and 0.17 μCi [^{14}C]monoolein, 99% pure free cholesterol (10 mg) and 2.23 μCi [^{3}H]cholesterol (12.1 Ci/mmol), and 90% pure soybean lecithin (11.2 mg). A total of 170 mg (10% of test meal solids) of finely ground wheat bran (Aria, Paris, France; 42.5% total dietary fiber) or wheat germ (Gerblé, Revel, France; 10.6% total dietary fiber) was or was not added to this mixture. All these materials were emulsified by 15-second sonication and the rats were intragastrically intubated by using a polyethylene catheter.

Sampling and analysis

After 1 hour of digestion, the rats were anesthetized with diethyl ether and then killed by total exsanguination. Blood was drawn by abdominal aorta puncture and collected over 0.1% EDTA as anticoagulant. Plasma was prepared by centrifugation and its volume was estimated at 3.28% of body weight.[14] The stomach content was collected on ice after rinsing with 2 ml ice-cold normal saline and immediately homogenized by 15-second sonication; aliquots were used for scintillation counting. The small intestine was clamped to obtain three segments of approximately equal length (upper, middle, and lower). Each segment was carefully removed and its content was collected on ice by flushing with 10 ml of ice-cold 2 mM taurocholate according to the method of Bennet Clark et al.[15] After sonicating for 15 seconds, 2-ml aliquots were collected for radioactivity determination. The mucosa of each intestinal segment was scraped on ice and acidified with HCl; total lipids were extracted overnight twice with 20 volumes chloroform/methanol 2:1 (vol/vol). Four-milliliter aliquots of mucosa extracts were evaporated to dryness and counted for radioactivity. The liver was removed, weighed, immediately frozen at −60°C, and stored until lyophilization. After grinding, 2 g of dry liver powder were extracted with 20 ml of chloroform/methanol (2:1, vol/vol). Aliquots (2 ml) of the organic extract were used for scintillation counting.

Five rats per experimental group were used. For statistical evaluation of the results, one-way analysis of variance with orthogonal contrasts at a 95% probability level served to determine treatment differences.

In vitro binding measurements

Bile was collected from anesthetized cannulated rats, and the pooled bile used contained 58.75 mM bile salts, 7.67 mM phospholipids, and 0.43 mM cholesterol. The pool of rat bile was diluted with 10 mM Tris-HCl buffer, 150 mM NaCl, pH 7.0, in order to obtain an 8-mM final bile salt concentration, a figure close to that reported in the small intestine.[16] The lipid mixture (see above), containing labeled oleic acid, monoolein, and cholesterol, was added to the pool of rat bile and incubated overnight at room temperature. After centrifugation, the final solution mixture contained 8.0 mM bile salts, 1.05 mM bile phospholipids, 0.052 mM bile cholesterol, 0.032 mM soybean lecithin, 0.052 mM cholesterol containing 11.9 nCi [^{3}H]cholesterol, 2.47 mM oleic acid containing 2.66 nCi [^{14}C]oleic acid, and 0.84 mM monoolein containing 0.91 nCi [^{14}C]-monoolein. The bile salts to phospholipids to fatty acids ratio and the fatty acids to monoglycerides ratio were close to those found for the small intestinal content of rat during fat digestion.[17]

Binding experiments were performed by mixing 25 mg of wheat bran or wheat germ after hydration with distilled water with 2.5 ml of the above described solution. The mixture was incubated under gentle shaking for 1 hour at 37°C. This period allowed for maximal binding. After centrifugation for 15 minutes at 15,000 × g and 4°C, the supernatant was collected. [^{3}H]cholesterol was directly counted from aliquots of the supernatant. Lipids were extracted according to the method of Folch et al.[13] at acidic pH. [^{14}C]oleic acid and [^{14}C]monoolein were separated by thin layer chromatography and counted as described above.

Each experiment was repeated three times, and controls consisted of incubations without fiber sources. ^{3}H and ^{14}C radioactivity (dpm) was measured in the presence of 15 ml Aquasure liquid scintillation cocktail (NEN, Dupont de Nemours).

Results and Discussion

Rat experiments

The present study aimed to determine whether wheat bran and wheat germ would lower the intestinal absorption of fatty acids, monoglycerides, and cholesterol. Since free cholesterol is directly absorbed by the small intestine and since dietary fats were provided in the form of absorbable long-chain free fatty acids and monoglycerides, no effect of fiber sources on the gastric and intestinal enzymatic lipolysis of fat would interfere, allowing direct determination of the influence of wheat fractions on lipid uptake by the intestinal mucosa. In that respect, a 1-hour digestion time was selected, since the rate of lipid mucosal uptake is maximal in the early hours of digestion and the effect of wheat fractions is greater in the first hour of triglyceride digestion in the rat.[10] As described below, 22% to 30% of the ingested labeled lipids had emptied from the stomach in these conditions.

Gastric emptying of lipids. The amount of radioactivity emptied from the stomach was estimated as the difference between the amount of label actually ingested and the amount of label remaining in the stomach after 1 hour. In the control group, the stomach emptying of [^{14}C]lipids was 22.18% (± 3.41), corresponding to 2.89 (± 0.44) 10^5 dpm. The figures obtained from the rats in the wheat bran group (22.5% ± 2.9%) and in the wheat germ group (29.9% ± 3.8%) were not statistically different. The stomach-emptying rate of [^{3}H]cholesterol (26.2% ± 3.6%) in the control rats was slightly higher than those of oleic acid and monoolein and did not differ from those of the rats in the wheat bran (27.6% ± 2.2%) and wheat germ (30.5% ± 3.4%) groups. Thus, the effects of wheat fractions on lipid assimilation cannot be considered as being the result of a delayed or accelerated output from the stomach of cholesterol or products of gastric lipolysis.

Lipids in the intestinal content. As shown in *Figure 1A*, 18.5%, 17.3%, and 22.8% of [^{14}]lipids evacuated from the stomach were present in the whole small intestine of rats of the control, wheat bran, and wheat germ groups, respectively. No significant differences were seen between the groups regarding the amounts of lipolysis products present. Comparable amounts of dietary [^{3}H]cholesterol were found in the small intestine (*Figure 2A*) in the presence or the absence of wheat bran, accounting for 25.9% and 29.7% of the quantity emptied from the stomach, respectively. In contrast, the presence of wheat germ significantly increased the amount of [^{3}H]cholesterol in the upper segment and in the whole small intestinal content, representing 38.6% of the dose having left the stomach.

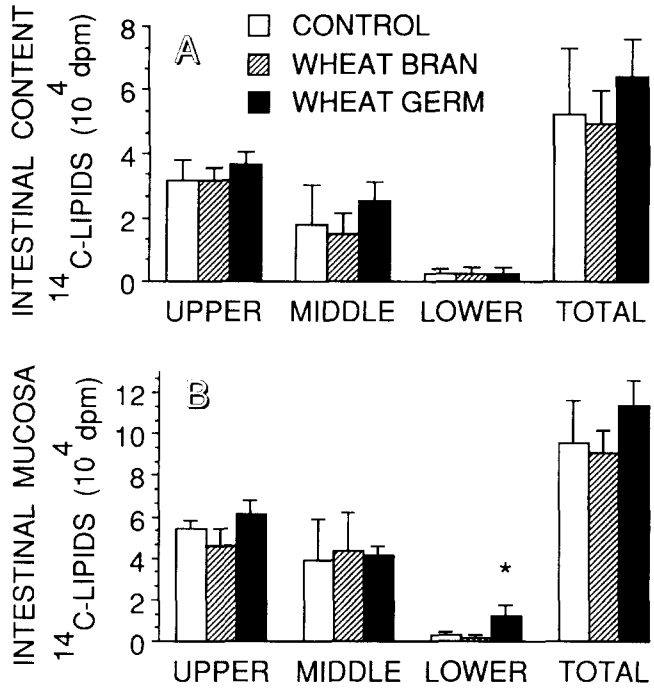

Figure 1 Total [^{14}C]lipid radioactivity (dpm) in the rat intestine. Values are mean ± SEM of five individual values. Range lines indicate 1 SEM. Significance ($P < .05$) is indicated by an asterisk. (A) Luminal content, (B) mucosal content.

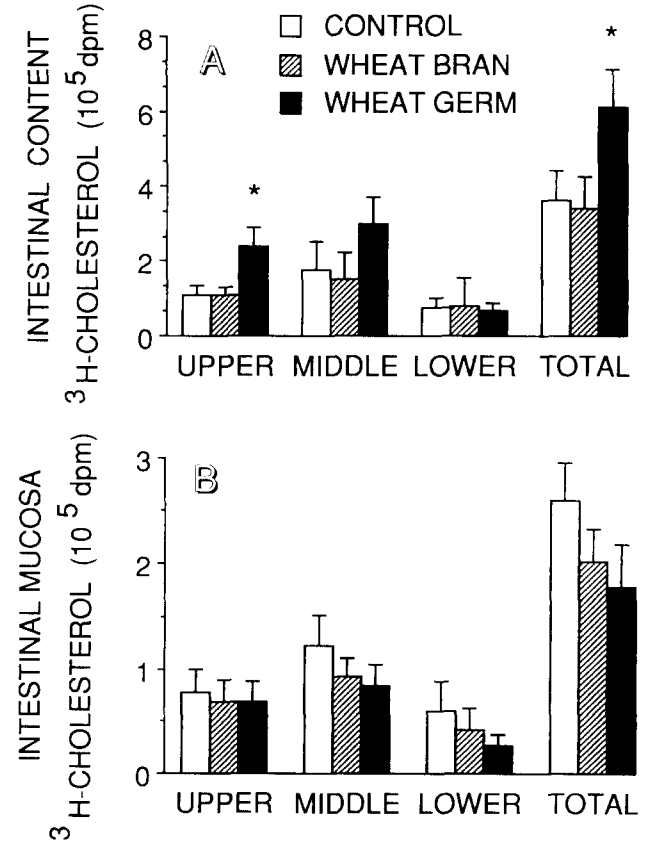

Figure 2 [^{3}H]cholesterol radioactivity (dpm) in the rat intestine. Values are mean ± SEM of five individual values. Range lines indicate 1 SEM. Significance ($P < .05$) is indicated by an asterisk. (A) Luminal content, (B) mucosal content.

Table 1 Ratio (%) of luminal content lipids versus mucosa lipids in the rat intestine

Lipid groups	Upper Segment	Middle Segment	Lower Segment	Whole Intestine
[^{14}C]lipids				
Control	58.4 ± 11.9[a]	31.7 ± 11.5[a]	100.9 ± 15.6[a]	51.0 ± 11.0[a]
Wheat bran	68.5 ± 5.8[a]	31.3 ± 8.90[a]	150.0 ± 30.2[a]	50.0 ± 15.3[a]
Wheat germ	61.5 ± 2.0[a]	69.3 ± 15.6[b]	32.4 ± 15.9[b]	58.4 ± 8.1[a]
[^{3}H]cholesterol				
Control	96.9 ± 21.3[a]	94.4 ± 11.4[a]	166.6 ± 32.5[a]	143.1 ± 43.2[a]
Wheat bran	156.5 ± 18.9[ab]	157.6 ± 20.4[a]	226.5 ± 50.8[a]	178.7 ± 40.2[a]
Wheat germ	272.2 ± 42.5[b]	377.1 ± 60.1[b]	301.5 ± 82.3[a]	310.6 ± 44.8[b]

Values (mean percentage ± SEM of five individual values) bearing different superscript letters in the same column are significantly different ($P < .05$).

Table 2 Amounts (dpm) of ^{14}C and ^{3}H radioactivity in the serum and the liver of rats

Lipids	Control Group	Wheat Bran	Wheat Germ
Serum			
[^{14}C]lipids	17,708 ± 2,397[a]	11,751 ± 1,972[a]	12,933 ± 1,655[a]
[^{3}H]cholesterol	5,061 ± 430[a]	5,788 ± 344[a]	4,980 ± 475[a]
Liver			
[^{14}C]lipids	3,435 ± 478[a]	2,820 ± 718[a]	3,197 ± 590[a]
[^{3}H]cholesterol	10,308 ± 1,214[a]	9,027 ± 935[a]	10,261 ± 1,754[a]

Values (mean ± SEM of five individual values) bearing different superscript letters in the same horizontal row are significantly different ($P < .05$).

Lipids in the intestinal mucosa. The incorporation of [^{14}C]oleic acid and [^{14}C]monoolein in the intestinal mucosa lipids (*Figure 1B*) was not affected in the upper and middle segments by the presence of wheat bran or wheat germ. Wheat germ significantly increased the [^{14}C]labeling of the lipids in the lower part of the small intestinal mucosa, but the physiologic importance of this observation was difficult to evaluate given the negligible amount of labeled lipids recovered in the distal part of the intestinal mucosa. However, the total amount of [^{14}C]lipids in the whole intestinal mucosa and the distribution of these lipids along the intestine were not significantly modified in the presence of both wheat fractions. In all cases, the upper and middle parts of the small intestine were the main sites of fat and cholesterol absorption. The calculation of the 1-hour ratio of luminal content lipids versus mucosa lipids in the intestine (*Table 1*) indicates that the absorption of fatty acids and monoglycerides was not significantly affected by the presence of either wheat fraction, although wheat germ increased the ratio in the middle segment and decreased it in the lower segment of the small intestine.

As shown in *Figure 2B*, wheat bran and wheat germ only slightly decreased the amount of [^{3}H]cholesterol incorporated in the middle and lower parts of the intestinal mucosa and thus had no significant effect on total [^{3}H]cholesterol in the whole mucosa. As shown in *Table 1*, wheat bran did not modify the ratio of luminal content versus mucosa cholesterol. The figure obtained with wheat germ was different, since this ratio was significantly lowered in the presence of wheat germ in the upper and middle segments of the small intestine (*Table 1*). However, this effect remains to be further documented since no significant decrease in the mucosal or plasma cholesterol content was observed.

Lipids in the serum and liver. As reported in *Table 2*, the presence of wheat bran and wheat germ in the test meal slightly but insignificantly decreased the amount of [^{14}C]lipids in the serum of rats after 1 hour of digestion; the amount of [^{14}C]lipids in the liver was unchanged. The same pattern was observed for [^{3}H]cholesterol, which appeared in equal amounts in the serum or in the liver of the rats in the three groups (*Table 2*).

In vitro binding experiments

During the *in vitro* experiments, the water-holding capacity of the wheat bran and wheat germ used were estimated respectively as 3.2 and 2.3 g water/g. The extent of binding of labeled oleic acid, monoolein, and cholesterol was simultaneously determined in the presence of rat bile. As shown in *Table 3*, with the 1% fiber source, approximately 12% of monoolein present was bound on both wheat fractions, whereas the binding of oleic acid was higher on wheat germ (14.59%) than on wheat bran (6.96%). Labeled cholesterol was bound at a comparable extent (approximately 15%) on both wheat fractions. This is in good agreement with

Table 3 *In vitro* binding of labeled oleic acid, monoolein, and cholesterol by wheat fractions in the presence of rat bile

Binding Substance	Lipid Bound (%)		
	[^{14}C]Monoolein	[^{14}C]Oleic Acid	[^{3}H]Cholesterol
Wheat bran (1%)	11.27 ± 1.21	6.96 ± 0.87	14.26 ± 1.17
Wheat germ (1%)	12.65 ± 1.12	14.59 ± 1.15	15.70 ± 1.34

Values are mean ± SEM of three measurements.

a previous study performed with synthetic mixed micelles.[18]

Given the data obtained in the present physiologic conditions, it can be calculated that 17.2 nM oleic acid, 9.5 nM monoolein, and 1.57 nM cholesterol are bound per milligram of wheat bran. Biliary phospholipids and bile salts are known to play an important role in the intestinal processing of fats, but both *in vitro* studies[12,19] and a study in the rat[20] have already shown that the extent of the binding of phospholipids and bile salts by wheat bran is rather low.

From the *in vitro* lipid binding capacities we have determined that the wheat bran (170 mg) added to the rat test meal was able to bind only approximately 0.23% of oleic acid, 0.37% of monoolein, and 1% of cholesterol ingested. Similar figures were obtained with wheat germ, suggesting that intraluminal binding of fat lipolysis products and cholesterol by wheat fractions would be too slight to impair their uptake by the intestinal mucosa.

In conclusion, the 1-hour uptake of oleic acid and monoolein by the rat intestinal mucosa was not significantly affected by the presence of wheat bran or wheat germ in the test meal, and no important shift was observed in the site of cholesterol and fat lipolysis product absorption. No significant change was observed in the serum or the liver-labeled lipids, indicating that no impairment of long chain-free fatty acid and monoglyceride processing occurred, whether it was mucosal uptake, re-synthesis of lipids and excretion from the enterocyte, transport by chylomicron, or liver uptake. Thus, these new findings point out that fiber-rich wheat fractions would affect fat assimilation,[21] mainly by a direct effect on the enzymatic lipolysis in the stomach and in the small intestine, as previously shown *in vitro*[11,12] and *in vivo*.[10]

In order to explain the sole effect of wheat germ (i.e., apparent decrease in dietary cholesterol absorption rate), it may be hypothesized that phytosterols present in the wheat kernel, which are concentrated as other neutral lipids in the germ,[22] would exert a well-known lowering effect on cholesterol absorption by the intestine.[23] This hypothesis needs to be tested by a specifically designed study.

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