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Whole wheat flour exerts cholesterol-lowering in rats in its native form and after use in bread-making

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Abstract

The ingestion of whole wheat flour (WWF) has been shown to exert lipid-lowering effects in rats but WWF is generally consumed after being processed (fermentation, starch gelatinization, heating). It remains to be assessed whether bread making has an influence on the potential lipid-lowering properties of flours. For this purpose, rats were fed semi-purified diets containing 70% WWF, or the same percentage of desiccated whole wheat bread (WWB) and the control group was fed with fibre-free purified starch diet. All the cereal diets showed a cholesterol-lowering effect in plasma and liver compared to control, but there was a more pronounced plasma triglyceride-lowering effect in rats fed WWB. In parallel, total steroids excretion was significantly enhanced ($P < 0.01$) by the cereal diets, but to a greater extent by WWB. As a result, cholesterol absorption percentage was also markedly reduced in rats fed WWB diet (around 26%, compared to 38% for WWF and 52% for controls). WWF and WWB yielded butyric acid rich fermentations in the cecum, compared to the control diet, and WWB markedly enhanced propionic acid production compared to WWF. In conclusion, the baking process adds significantly to the hypolipemic effects observed previously with WWF, in spite of reduced specific viscosity.

Keywords: Bread-making; Cholesterol; Rat; Whole wheat; Flour

1. Introduction

Whole grains provide a wide range of nutrients and biologically active constituents as dietary fibre, B and E vitamins, selenium, zinc, copper, magnesium and phytochemicals, such as phenolic compounds, which may contribute synergistically to reduce the incidence of various chronic diseases (Slavin, Jacobs, Marquet, & Wiemer, 2001). Epidemiological data show that elevated blood triglyceride and cholesterol, especially low-density-lipoprotein cholesterol (LDL-C), is a major risk factor in development of CHD (Anderson & Tietyen-Clark, 1986). In this view, cereals such as oats, rice, barley or rye and their milling fractions have well-documented lipid-lowering properties in rats (Arjmandi, Craig, Nathani, & Reeves, 1992; Chen, Andersen, & Gould, 1981; Lund, Salt, & Johnson, 1993 ; Topping et al., 1990; Wang & Klopfenstein, 1993). It is generally accepted that, at least, three of the daily grain servings should be whole grains (Welsh, Shaw, & Davis, 1994). Dietary fibre intake, especially from grain sources, has been connected to a reduced risk of coronary heart disease (Wolk et al., 1999) and diabetes (Meyer et al., 2000). Of the different sources of fibre (vegetable, fruit, cereal), cereal fibre has been shown to have the strongest association with a reduced risk of CHD (Rimm et al., 1996).

A series of investigations compiled into a review did not provide clear conclusions about the effects of wheat fibre (Anderson, 1985). Thereafter, studies on wheat fibre have been relatively scarce; rather, they have focused on purified fractions such as bran or germ (Borel, Lairon, Senft, Chauton, & Lafont, 1989a,

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Nomenclature

C	Cholesterol
CHD	Coronary Heart Disease
HDL	High Density Lipoprotein
LDL	Low Density Lipoprotein
SCFA	Short chain fatty acids
TGRLP	Triglycerides-rich-lipoproteins cholesterol
WWB	Whole Wheat Bread
WWF	Whole Wheat Flour

1989b). A recent study has shown that whole wheat flour have lipid-lowering properties in rats (Adam et al., 2001). A crucial question remains about the actual effect of whole grain processing on the lipid-lowering properties. Although processing is often considered to be a negative attribute in nutrition, and some forms of processing reduce nutritional value, whole grains, as harvested, are generally not consumed directly by humans but require some processing prior to consumption.

It appears nutritionally relevant to check whether bread-making does not affect cholesterol-lowering properties of whole wheat flour, because whole wheat bread represents an important food to improve whole grain consumption and daily supply of fibre, minerals and other micronutrients in western countries.

To examine the effects of bread-making on digestive fermentations and lipid metabolism, the present study compared the effects of whole wheat bread (WWB) and the corresponding whole wheat flour (WWF) in rats. To assess the potential of whole wheat to exert hypocholesterolemic effects, the control was a purified starch-based diet.

2. Materials and methods

2.1. Bread making procedures

Flour used for bread making was a reconstituted whole wheat flour (white flour + remilling + bran) from a panel of bread-making cultivars cultivated in France (commercial whole wheat flour). The procedure for the making of yeast bread was: 6 kg whole wheat flour mixed with 3.6 l water and 150 g yeast (*Saccharomyces cerevisiae*). The dough was baked after a 3 h fermentation. The bread was then dried, ground and incorporated into rat diet.

2.2. Animals and diets

Male Wistar rats, weighing approximately 140 g, were used. They were from the colony of laboratory animals of the National Institute of Agronomic Research (INRA of Clermont-Ferrand/Theix, France). They were fed one of the experimental semipurified diets distributed as moistened powder for 21 days (Table 1). The animals were allowed free access to fresh food and tap water. Rats were housed, two per cage, and maintained in temperature-controlled rooms (22 °C), with the dark period from 20.00 to 08.00 h. They were maintained and handled according to the recommendations of the Institutional Ethics Commitee (Clermond-Ferrand University). The body weight of rats was recorded twice per week during the experimental period. During the last 7 days of the experiment period, rats were transferred to metabolic cages and food intake and fecal excretion were recorded over the last 4 days of the experiment.

2.3. Sampling procedures

Rats were first anesthesized with sodium pentobarbital (40 mg/kg) and maintained at 37 °C. An abdominal incision was made and blood was withdrawn from the portal vein (2 ml) and the abdominal aorta (5 ml),

Table 1
Composition of the diets[1]

	Control diet (g/kg)	WWF (g/kg)	WWB (g/kg)
Digestible wheat starch	717.5	195	195
Casein	75	75	75
Wheat Gluten	75	–	–
Mineral mix[2]	26.3	17.9	17.9
Vitamin mix[3]	10	8	8
Groundnut oil	50	32	32
Cholesterol	2.5	2.5	2.5
Whole cereal	–	700	700
Energy (kJ/g)	16.9	16.3	16.3

[1] The first experimental diet consisted of whole wheat flour (WWF), the second one is composed of whole wheat bread (WWB). The bread was dried for 48 h at 80 °C and then ground with a 2 mm grating. For each diet, 700 g/kg of whole flour and whole bread were used. All the diets were equal in carbohydrates (around 75%); only the fibre content varied. For the control, WWF, WWB diets, the starch contents were 75, 71 and 71%, respectively. The protein contents were 15, 16.2 and 16.2%, respectively, and the lipid contents were 5, 4.7 and 4.7%, respectively ; total dietary fibre levels (not reported) were 0, 9.8, 10.1%, respectively.

[2] All diets contained (per kg diet): 4 g Ca, 1.2 g Mg, 5mg Cu, 36 mg Fe, 38 mg Zn. Mineral content of all diets was checked before the beginning of the experiment.

[3] Vitamins supplied (mg/kg control diet) : thiamin, 15 ; riboflavin, 20; pyridoxine, 10; nicotinamide, 100; pantothenate, 70; folic acid, 5; biotin, 0.3; cyanocobalamin; 0.05; retinyl palmitate, 1.5; dl-α-tocopheryl acetate, 125; cholecalciferol, 0.15; menadione, 1.5; ascorbic acid, 50; myo-inositol, 100; choline, 1.36 g. Provided by AIN 76 (Villemoisson, Epinay-sur-Orge, France). For the experimental diets, the vitamin concentration of the whole flour has been taken into account and therefore the vitamin mix supply was reduced to 3 g/kg instead of 10 g/kg.

respectively into heparinized tubes. After centrifugation at 10,000 × ***g***, for 5 min, the plasma was collected and stored at 4 °C for lipid and lipoprotein analysis. After blood sampling, the cecum with its contents was removed and weighed and two samples of cecal contents were transferred to microcentrifuge tubes and immediately frozen at −20 °C.

A portion of liver was freezed-clamped and stored at −80 °C for the measurement of liver lipids.

2.4. Analytical procedure

Short-chain-fatty-acid (SCFA) concentrations were measured by gas–liquid chromatography after ethanolic extraction of the supernatants (8.000 × ***g***, 5 min at 4 °C) of cecal contents as described by Rémésy and Demigné (Remesy & Demigne, 1974). Bile acids and sterols were extracted from cecal contents and feces by 40 volumes ethanolic KOH (4 mol/l) and quantified using the reaction catalyzed by the 3 α-hydroxysteroid dehydrogenase (E.C. 1.1.1.50; Sigma, L'Isle D'abeau Chesnes, France) (Turley & Dietschy, 1978). Neutral steroids were extracted three times with 1 ml hexane from a 100 μl aliquot of the alkaline ethanolic extract, after addition of 5 α-cholestane as an internal standard. The solvent was evaporated under N_2 and the residue dissolved in hexane. Extract (200 μl) was injected into the gas chromatograph (Danieducational, Paris, France) fitted with a 12 m × 0.25 mm (inner diameter) fused silica capillary column (BP10; SGE, Villeneuve-St-Georges, France) and a flame-ionization detector. Helium was used as the carrier gas, and the sterols were separated using a temperature gradient from 240 to 280 °C. Sterol concentrations were calculated from the peak areas relative to the area of the internal standard. Triglyceride and total cholesterol were determined in plasma by enzymatic procedures using commercial kits (Biotrol, Paris, France and BioMerieux, Charbonnières-les-bains, France, respectively). Liver triglyceride and cholesterol were extracted and analysed as described by Mazur, Rémesy, Gueux, Levrat, and Demigné (1990) control serum (Biotrol-33 Plus, Biotrol, Paris, France) was treated in parallel to check the accuracy of the analyses.

Plasma lipoproteins were separated on a density gradient by preparative ultracentrifugation as described (Serougne, Ferezou, & Rukaj, 1987) in a TST 41.14 swinging-bucket rotor (Kontron, Zürich, Switzerland) at 100,000 *g* for 24 h (15 °C). The gradient was then fractionated into 500 μl fractions and the cholesterol and triglyceride contents of each fraction were determined by the method described for plasma samples. Because of low level of plasma LDL and the partial overlapping of HDL1 and HDL2 fractions in rats, only two fractions were considered: the $d < 1.040$ kg/l fraction (chiefly triglyceride-rich lipoprotein, TGRLP, together with some LDL) and the $d > 1.040$ kg/l fraction (HDL).

The total dietary fibre was analysed by the method approved by the Association of Official Analytical Chemists (Prosky et al., 1984). The total fibre content was 11.5 g/100 g whole wheat flour and 12.0 g/100 g whole wheat bread, leading to fibre percentages between 8.0% and 8.4% in the experimental diets (Table 1). The specific viscosity of bread was measured by the Biochemistry Laboratory of the ITCF (Boigneville, France) according to (Saulnier, Peneau, & Thibault, 1995). Values were 3.95 and 1.55 ml/g for WWF and WWB, respectively.

2.5. Calculation and data analysis

The cecal pool was calculated as: cecal concentration (μmol/l) × cecal contents volume (l). The food conversion efficiency (FCE) was calculated as: weight gain (g/day)/food intake (g/day). The cholesterol apparent absorption (μmol/day) = daily cholesterol intake (μmol/day) −daily total steroid excretion (μmol/day). Values are given as the means ± SEM and, where appropriate, significance of differences ($P < 0.05$) between mean values was determined by analysis of variance (ANOVA), coupled with the Student–Newman–Keuls' Test. The data were tested for normality by using the Kolmogorov and Smirnov method before the parametric statistical tests were performed.

3. Results

3.1. Effects of cereals on food intake and weight gain and digestive fermentations

The presence of 70% whole flour or whole bread in the diet did not affect daily food intake, weight gain or the apparent food conversion efficiency (FCE) (Table 2). Rats fed the cereal diets had a greater fecal excretion of dry matter than controls ($P < 0.001$), and rats fed bread had greater fecal excretion than rats fed whole flour ($P < 0.001$). Ceca were heavier in rats fed the cereal diet, this was chiefly due to an enlargement of the contents but also to a slight hypertrophy of the cecal wall. The cecal pH was significantly acidified in rats fed the cereals diets, down to around 6.5 (Table 2).

In parallel, there were alterations in the concentrations and molar ratios of cecal SCFA (Table 3). Compared with the controls, the cereals group had around 30% higher total cecal SCFA concentration, which was due to a higher concentration of butyric acid ($P < 0.01$) and propionic acid. In rats fed either cereal diet, the molar proportion of butyric acid in the cecum was relatively high (15–19%) and WWB-fed rats exhibited the highest propionic acid concentration in the cecum (51 mmol/l).

Table 2
The growth and cecal variables in rats consuming WWF, WWB and control diet[1]

Diet	Food intake[2] (g/day)	Weight gain[2] (g/day)	Apparent FCE	Cecum			Faeces
				Total weight[3] (g)	Wall weight[3] (g)	pH[3]	Daily excretion[2] (g dry matter/day)
Control	19.1±0.6[a]	5.4±0.4[a]	0.29±0.03[a]	2.70±0.14[b]	0.80±0.05[c]	7.19±0.05[a]	0.42±0.03[c]
WWF	18.7±1.2[a]	5.8±0.5[a]	0.31±0.02[a]	3.40±0.25[a]	0.94±0.03[b]	6.47±0.08[b]	1.38±0.10[b]
WWB	20.9±0.6[a]	5.6±0.4[a]	0.27±0.02[a]	3.88±0.17[a]	1.00±0.03[b]	6.54±0.05[b]	1.97±0.08[a]

[1] Values are means±SEM; $n=8$. Values in a column not sharing a letter are different, $P<0.05$.
[2] Variables measured during study.
[3] Variables measured postmortem.

Table 3
The cecal SCFA variables in rats consuming WWF, WWB and control diet[1]

Diet	Acetic acid (mmol/l)	Propionic acid (mmol/l)	Butyric acid (mmol/l)	Total SCFA (mmol/l)	SCFA molar ratio	Cecal SCFA pool (μmol)
Control	75±5[a]	29±2[b]	9±1[b]	113±7[a]	66/26/8	209±21[b]
WWF	81±11[a]	38±2[b]	29±6[a]	149±17[a]	54/25/19	393±29[a]
WWB	74±6[a]	51±4[a]	22±3[a]	147±11[a]	50/35/15	431±39[a]

[1] Values are means±SEM; $n=8$. Values in a column not sharing a letter are different, $P<0.05$.

Table 4
The cholesterol fecal excretion and cholesterol absorption variables in rats consuming WWF, WWB and control diet[1]

Diet	Daily cholesterol intake (μmol/day)	Fecal excretion					
		Bile acids (μmol/day)	Cholesterol (μmol/day)	Coprostanol (μmol/day)	Total steroïds (μmol/day)	Cholesterol apparent absorption (μmol/day)	(% of intake)
Control	124±4[a]	17.8±0.8[b]	22.5±1.8[a]	25.7±2.7[b]	60.9±3.5[c]	66.5±4.2[a]	52.4±3.0[a]
WWF	127±8[a]	24.4±2.0[b]	28.2±2.2[a]	26.0±1.9[b]	78.5±4.0[b]	48.4±3.5[b]	37.9±2.4[b]
WWB	135±4[a]	44.5±3.6[a]	23.4±1.9[a]	39.3±3.8[a]	107.2±5.4[a]	27.1±4.8[c]	20.7±3.1[c]

[1] Values are means±SEM; $n=8$. Values in a column not sharing a superscript are different, $P<0.05$

3.2. Effects of cereals on fecal steroids

Daily cholesterol intake was not significantly different among the groups namely around 130 μmol/day (Table 4). The fecal bile acids excretion was not significantly enhanced by the WWF diet, compared to controls, whereas rats fed the WWB had significantly higher fecal excretion of bile acids than controls (+150%, $P<0.001$). Cholesterol excretion did not differ among the groups. The coprostanol excretion was higher in rats fed WWB (around +51%) than in rats fed the control or the WWF diet. Consequently, total steroid excretion was significantly higher in rats fed the cereal diets, and notably the bread diet, than in controls. Furthermore, rats fed WWB had significantly higher fecal steroids excretion than rats fed WWF (+29%, $P<0.01$). Accordingly, estimated cholesterol absorption was significantly depressed in rats fed WWF and WWB (−27%, −59%, respectively) compared to controls, especially in rats fed WWB ($P<0.001$). The percentage of cholesterol absorbed, relative to that consumed, was significantly lowered by the cereal diets, but this percentage was still lower in rats fed WWB than in rats fed WWF ($P<0.01$).

3.3. Effects of cereals on lipid metabolism

Plasma cholesterol was lower in rats fed WWF, WWB (−22 and−29%, respectively, $P<0.001$) than in controls, as well as hepatic cholesterol which was also markedly decreased (−47.5 and−54%, $P<0.001$) in rats fed WWF and WWB, respectively (Table 5). Triglyceride concentrations were also altered significantly in plasma (−33%, $P<0.001$) and liver (−32%, $P<0.001$) in rats fed WWB, compared to controls. Hepatic trigly-

Table 5
The plasma and hepatic lipid variables in rats consuming WWF, WWB and control diet[1]

Diet	Plasma		Liver	
	Cholesterol (mmol/l)	Triacylglycerols (mmol/l)	Cholesterol (mg/g)	Triacylglycerols (mg/g)
Control	2.59 ± 0.15^{a}	2.00 ± 0.17^{a}	8.0 ± 0.7^{a}	21.6 ± 1.2^{a}
WWF	2.02 ± 0.09^{b}	$1.64\pm0.14^{a,b}$	4.2 ± 0.4^{b}	13.1 ± 0.6^{b}
WWB	1.84 ± 0.08^{b}	$1.33\pm0.08^{b,c}$	3.7 ± 0.3^{b}	14.7 ± 0.6^{b}

[1] Values are means±SEM; $n=8$. Values in a column not sharing a superscript are different, $P<0.05$

ceride were significantly lower in WWF fed rats (−39%, $P<0.001$) compared to the control group, but not significantly lower in plasma.

Plasma lipoproteins were fractionated by ultracentrifugation and a pool was further analyzed for lipid content. The TGRLP fraction ($d<1.040$ kg/l) in the WWF or WWB fed groups had 33% less cholesterol than controls, whereas no difference was observed in the HDL fraction (Fig. 1a). TGRLP triglyceride were also markedly lower than in controls in rats fed the WWF or WWB diets (−14 or−24%, respectively) (Fig. 1b).

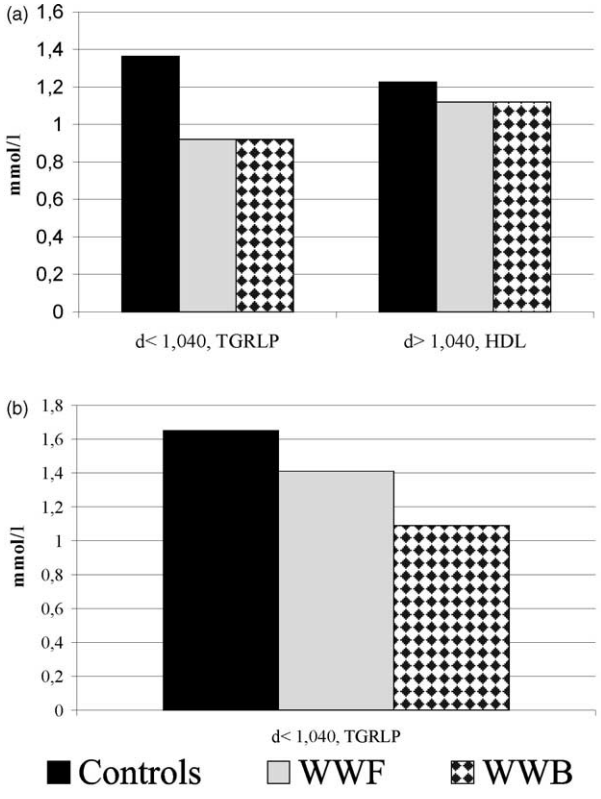

Fig. 1. Differences in the repartition of cholesterol (panel A) and triglycerides (panel B) in plasma lipoprotein fractions of rats fed the control, WWF (whole wheat flour) and WWB (whole wheat bread) diets. Each value is a mean of a triplicate analyses of a pool of eight plasma. The fractions with a density less than 1.040 kg/l corresponded chiefly to triglyceride-rich lipoproteins with a lower contribution of LDL. The fractions with a density higher than 1.040 kg/l corresponded essentially to HDL.

4. Discussion

It is noteworthy that, in the present experiment, whole wheat bread induced a greater decrease of the cholesterol absorption (% of intake) than whole wheat flour. Numerous investigators have documented the cholesterol-lowering effects of dietary fibre (Anderson, Deakins, & Bridges, 1990), and the mechanisms responsible for these effects are relatively complex. Several hypotheses have emerged regarding the cholesterol-lowering mechanisms of soluble fibres, including: alteration of bile acid absorption, modification of lipid absorption and metabolism, effects of short chain fatty acids (SCFA) resulting from fibre fermentations or cholesterol or lipoprotein metabolism (Anderson et al., 1990) and changes in insulin or other hormone concentrations, or tissue sensitivity to hormones (Jenkins, Rainy-MacDonlad, Jenkins, & Benn, 1986; Wolever & Jenkins, 1986).

Rats fed the bread diet had significantly higher fecal excretion of bile acids than controls ($P<0.001$) or rats fed the WWF ($P<0.01$), but cholesterol excretion did not differ among the groups. The coprostanol excretion was enhanced only in rats fed WWB (+51%) but not in those fed WWF. Consequently, total steroid excretion was significantly higher in rats fed the cereal diets, especially the bread diet, than in controls. This could be explained by the 0.5% greater content of total dietary fibre (TDF) in bread (12% instead of 11.5% for WWF) and also by the characteristics of dietary fiber amenable to changes during processing, including molecular, structural and functional properties. The role and properties of cell wall-degrading enzymes, such as xylanolytic enzymes or β-glucanases, as well as their inhibitors in grains, are beginning to be better understood (Poutanen, 2001). Since dietary fibre components act as substrates for these enzymes, depolymerisation and solubilisation of fibre occurs when suitable conditions are afforded. In particular wet processes at moderate temperatures, such as wet milling or prolonged baking procedures (e.g. sourdough baking), may activate the enzymes in grain. Increased solubilisation of the major dietary fiber component (arabinoxylan) has been observed during baking of wheat (Westerlund, Theander, Andersson, & Aman, 1989). Various reaction products of polysaccharides arise during baking of bread and they have significant effects on product structure and quality. Formation of retrograded starch (resistant starch), as a result of gelatinisation and cooling (Englyst, Wiggins, & Cummings, 1982; Ranhotra & Gelroth, 1988), represents about 30% of the total apparent dietary fibre of white bread. Other starch alterations, such as formation of glucosaccharides by breaking of intramole-

cular linkages in starch (Siljeström, Björck, & Westeslund, 1989; Theander & Wesserlund, 1987) can be envisaged as a result of baking in the crust, but their possible effects on enterohepatic-bile acid cycling have not yet been shown. Glucosaccharides have β-1,6-anhydro-pyranose end units, and they may react further by transglycosidation with hydroxyl groups of starch or other carbohydrates. Resulting branched glucan structures represent an irreversible chemical modification of starch, increasing the non-starch glucan content of the fibre fraction (Theander & Westerlund, 1987), which is in accordance with our results (0.5% greater content of TDF in bread).

The present results indicate that the percentage of cholesterol absorption may be significantly depressed by a bread diet (25% vs 38% in rats fed WWF, and 52% in controls). The mechanisms of inhibition of cholesterol absorption, in which viscosity is an important factor, have been well documented: disturbance of micelle formation, slowing of cholesterol transfer to the brush border across the unstirred layer and inhibition of ileal bile acid reabsorption (Stedronsky, 1994). However, in the present case, the specific viscosity of the bread was reduced compared to that of the corresponding whole wheat flour. The water-soluble arabinoxylans in wheat bread have a higher degree of arabinose substitution than those in the flour and dough, indicating increased solubilisation of highly substituted arabinoxylans during baking (Westerlund, Andresson, Aman, & Theander, 1990). A mathematical model proposed by (Bengtsson, Andersson, Westerlund, and Aman, 1992) indicates that, the higher proportion of di-substituted xylose residues the greater the viscosity of the polymer. Increased solubilisation of highly substituted arabinoxylans during baking may explain the decrease of the specific viscosity of the bread. Viscosity is not the only mechanism whereby dietary fibres increase fecal bile acid excretion through binding bile acids or interfering with micelle formation. Soluble fibres undergo various types of interactions with steroids, even with ionized bile acids at the physiological pH in the small intestine (Stedronsky, 1994). In rats the fecal excretion of secondary bile acids has been reported to increase after feeding resistant starch (Verbeek, De Deckere, Tijburg, Van Amelsvoort, & Beynan, 1995). It has been proposed that helical structures in starch act as binding sites for bile salts (Abadie, Hug, Kubli, & Gains, 1994). Soluble fibres and resistant starch are extensively broken down by the microflora. Resistant starches are viewed as butyric-yielding precursors (Mathers & Dawson, 1991; Silvester, Englyst, & Cummings, 1995), but in a rat model, also they have been found effective in promoting high propionic acid fermentations (Morand, Levrat, Besson, Demigne, & Rémésy, 1994). It has been speculated that SCFA could constitute a mediator of the lipid-lowering effect of fibres. In vitro experiments have demonstrated that propionate is an effective inhibitor of both fatty acid and cholesterol synthesis (Demigne et al., 1995; Levrat et al., 1994), but in vivo investigations have been less conclusive (Berggren, Nyman, Lundquist, & Bjovck, 1996).

The WWF and WWB diets contained ≈2% wheat germ, and it is conceivable that this fraction could play a significant role in the observed lipid-lowering effects. In short-term studies in rats, ingestion of raw wheat germ lowered plasma triglyceride and cholesterol (Cara, Bovel, Armand, Senft, & Riottot, 1991). The actual mechanism may be an inhibition of pancreatic lipase by wheat germ proteins, which could interact with the emulsified substrate and hinder the adsorption of the enzyme on the interface (Borel, Lairon, Termine, Grataroli, & Lafont, 1989).

WWF, WWB diets induced a reduction in both liver and plasma triglyceride levels, compared with the control group fed a fibre-free diet. Several mechanisms could be involved, such as impaired dietary triglyceride absorption from the small intestine as described above, increased chylomicron remnant uptake, reduced liver very-low-density lipoprotein (VLDL) secretion, or increased VLDL catabolic rate (Mahley, 1982). It has been shown, in rats, that adding wheat bran to a test meal increased the ileal output of dietary fats (Borel et al., 1989a), and decreased the amounts of labelled lipids of dietary origin in intestinal mucosa and postprandial serum (Borel et al., 1989a). Thus, from in vivo (Borel et al., 1989a) and in vitro (Isaksson, Lundquist, & Ihse, 1982) studies, these effects could be interpreted as the result of a partial inhibition of gastrointestinal lipases.

In conclusion, the baking process adds significantly to the hypolipidaemic effects observed previously with whole wheat flour. Indeed, the apparent cholesterol absorption related to dietary cholesterol intake was significantly reduced in WWB-fed rats compared to the WWF and control groups. This increase of the effects may be explained by the characteristics of resistant starch and dietary fibre amenable to changes during processing, including molecular, structural and functional properties. Despite a reduced specific viscosity, this does not imply that the soluble fibre content is decreased. Bread-making has long been identified as a favourable process in various respects (starch gelatinization, phytic acid breakdown and better mineral bioavailability) and it also appears to be interesting because the potential lipid-lowering effects of cereal flours are maintained or even increased during this process.

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