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Corn bran dietary fibre modified by xylanase improves the mRNA expression of genes involved in lipids metabolism in rats

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Abstract

The regulation effects of corn bran dietary fibre (CDF) and the xylanase modified form (XMF) in male Sprague Dawley rats on the expression of several genes involved in lipid metabolism were studied. Rats for NF and HF group (8 per group) were fed basic diet (NF) and atherogenic diet (HF), respectively, for six weeks. The rats for CDF or XMF group were fed HF diet, for two weeks first then changed to CDF (10%) or XMF (10%) diet, respectively, for four weeks. XMF ingestion lowered serum total cholesterol (TC), triacylglycerols (TG), low-density lipoprotein cholesterol (LDL-C) and increased high-density lipoprotein cholesterol (HDL-C) significantly ($p < 0.05$) as compared with CDF; The corresponding liver TC, TG and fat in XMF group were also significantly lower ($p < 0.05$). CDF ingestion lowered serum TC, TG, LDL-C, arteriosclerosis index (AI), and liver fat significantly ($p < 0.05$). AI in XMF group was less than 50% of that in CDF group. XMF enhanced the catabolism of lipids by up-regulating the transcription of hepatic cholesterol 7 alpha-hydroxylase (CYP7A1), peroxisome proliferator-activated receptors (PPARα and PPARγ), lipoprotein lipase (Lpl), liver lipase and ileum farnesoid X receptor (FXR), down-regulating the transcription of intestine-bile acid binding protein (I-BABP) and hepatic FXR. While CDF only down-regulated I-BABP transcription, up-regulated ileum FXR, liver PPARα, Lpl and CYP7A1 transcription.

Keywords: Xylanase modification; Corn bran dietary fibre; Hypolipidemic effects; Regulation; mRNA expression; Sprague Dawley rats

1. Introduction

A major cause of cardiovascular turbulence is atherosclerosis, which originates mainly from hyperlipidemia and the turbulent lipids metabolism (Jin, Wen, Tang, & Chen, 1995). The generation and development of atherosclerosis correlate highly with blood total cholesterol (TC) and total triacylglycerol (TG) levels. Among TC, the low-density lipoprotein cholesterol (LDL-C) and very low-density lipoprotein cholesterol (VLDL-C) are the most important actors for coronary heart disease. High-density lipoprotein cholesterol (HDL-C) suppresses the ingestion of LDL-C by body cells, transports excessive cholesterol out of cells in the ester form, and thus prevents the accumulation of cholesterol in the cells (Goldstein, Hazzard, Schrott, Bierman, & Motulsky, 1973).

Hyperlipidemia is tightly connected with dietary factors. It is now well established that certain sources of dietary fibre (such as Psyllium and prune among others), independent of the fat or carbohydrate content of the diet, can lower serum cholesterol concentrations (Kritchevsky & Tepper, 2005; Lucas, Juma, Stoecker, & Arjmandi, 2000). A considerable amount of fibre is processed from wheat bran, fruits, pea hulls and bagasse, among others, all these have been incorporated into food products such as bread and fish products (Sanchez-Alonso, Haji-Maleki, & Borderias, 2007; Sudha, Vetrimani, & Leelavathi, 2007).

Corn bran has higher dietary fibre content than both wheat and rice bran (Wang & Liu, 2000), in which about 40% is heteroxylan, followed by cellulose and some phenolic acids (Saulnier, Marot, Chanliaud, & Thibault, 1995). Shane

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and Walker (1995) reported that corn fibre supplementation of low-fat controlled diet in men with hypercholesterolemia resulted in additional lowering of serum TC, TG and VLDL-C, while serum LDL-C and HDL-C concentrations were not significantly altered. Corn bran fibre hydrolysed by α-amylase was also found to lower the plasma and liver cholesterol concentrations significantly in rats (Ebihara & Nakamoto, 2001). Zhang and Wang (2005) prepared a corn dietary fibre from corn residue using α-amylase and alkali proteinase hydrolysis, and fed it to hyperlipidemic mice. The feeding results showed that with the addition of corn dietary fibre up to 8% of total feed, the levels of serum total cholesterol and total triacylglycerols were lowered, and the HDL-C was increased, as compared with the control.

It is known that dietary fibre increases bile acid and cholesterol excretion by decreasing bile acid and cholesterol absorption in the intestinal tract, thus enhancing bile acid synthesis from cholesterol, hence acts as a hypocholesterolemic agent (Marlett, 2001). In a previous study, a new corn bran fibre XMF, which was moderately hydrolysed by xylanase, presented significantly increased bile salts binding capacity than its origin corn bran dietary fibre (CDF) in vitro (Hu, Wang, & Xu, 2008). However, the general mechanisms of hypolipidemic activity of dietary fibre are still uncertain. Lipids (cholesterol and fatty acids) are essential nutriments and have a major impact on gene expression. Cholesterol intracellular concentration is precisely controlled by some complex mechanisms involving transcriptional regulations (Lobaccaro et al., 2001). Soluble phytochemicals have been reported to act as hypocholesterolemic agents through regulating some genes involved in lipids metabolism (Cha et al., 2006; Mezei et al., 2003; Takahashi et al., 2002). The hypocholesterolemic effect of dietary beet fibre is associated with diminished expression of the hepatic apoliprotein A-I gene (Sonoyama, Shuhachi, & Niki, 1995). Corn bran is mainly composed of insoluble fibre (Hu & Wang, 2006). There is no report about whether corn bran dietary fibre improves lipid homeostasis through influencing the transcription of genes involved in lipids metabolism to date. Thus, the purpose of this study was to investigate the influences of corn bran dietary fibre CDF and its xylanase modified form XMF on the regulation of mRNA expression of several genes involved in cholesterol and triacylglycerols catabolism.

2. Methods and materials

2.1. Corn bran dietary fibre (CDF)

Corn bran was provided by Dancheng Caixin Group Co., Ltd. (Henan, PR China) and was milled through a 250 μm screen, then processed as reported previously (Hu et al.).

2.2. Xylanase modified corn bran fibre (XMF)

A 10 L container with a working volume of 8 L of 50 mmol/L phosphate buffer at pH 5.3, containing 0.7 g/100 g CDF of xylanase (NCB 77, 8000 IU/g, from *Bacillus subtilis,* main enzyme activity EC 3.2.1.8), supplied by Hunan New Century Biochemical Co., Ltd., Yueyang, PR China. Eight hundred grams CDF were added to the freshly prepared xylanase enzyme solution. The reaction mixture was incubated in a super water bath thermostatic vibrator (Model 501, Shanghai Experimental instrument Co., Shanghai, PR China) at 50 °C with 145 rpm agitation for 1.75 h, and the reactants handled as in Section 2.1 to obtain the xylanase modified fibre (called XMF).

2.3. Animals and diets

The Jiangnan University Animal Care and Use Committee approved all rat studies. All rats weighing 100 ± 10 g (male Sprague Dawley, from Shanghai Slac Laboratory Animal Co., Ltd., Shanghai, PR China) were placed individually in stainless steel wire mesh cages in a room maintained at 23 °C ± 2.0 °C, with relative humidity of 55 ± 10%, and a daily photo period from 7:00 am to 7:00 pm. Rats consumed food and water ad libitum and were acclimated to the animal facility for one week. All were then randomly assigned to NF (negative control), HF (positive control), CDF or XMF group with eight rats per group. Rats in NF and HF group were fed with basic feed (NF diet) and an atherogenic diet (HF diet), respectively, for six weeks. The rats for CDF or XMF group were fed HF diet, for two weeks first then changed to CDF or XMF diet, respectively, for four weeks. The components of diets were as shown in Table 1. HF diet was an atherogenic diet. Cholesterol, sodium cholate, yolk powder and basic feed were purchased from Shanghai Slac Laboratory Animal Co., Ltd., Shanghai, China). Lard was purchased from a local market in Wuxi, PR China.

Table 1
Components of animal diets

	NF	HF	CDF	XMF
Corn powder	35.5	30	25	25
Soy bean pomace	21.6	20	20	20
Lard	2	15	15	15
Fish powder	2	4	4	4
Cellulose	10	3	–	–
Flour	26	13.8	11.8	11.8
$CaHPO_4$	1	1	1	1
$CaCO_3$	1.3	1.1	1.1	1.1
Lysine	0.12	0.12	0.12	0.12
Methioline	0.13	0.13	0.13	0.13
Choline	0.1	0.1	0.1	0.1
Mineral mixture	0.03	0.03	0.03	0.03
Vitamin mixture	0.02	0.02	0.02	0.02
NaCl	0.2	0.2	0.2	0.2
Cholesterol	–	2	2	2
Sodium cholate	–	0.5	0.5	0.5
Yolk powder	–	9	9	9
CDF	–	–	10	–
XMF	–	–	–	10

2.4. Sampling and analytical procedures

Serum was analysed for TC, TG and HDL-C using appropriate commercially available kits (Zhejiang Dongou Biotech Co., Ltd., Wenzhou, PR China). LDL-C was calculated according to Friedwald equation (Anna, Riitta, Marjukka, Kirsi, & Oksman, 2003). On day 42, rats were weighed and sedated by barbiturate injection. They were exsanguinated, the livers removed and weighed. Serum was analysed for TC, TG, HDL-C and LDL-C. Arteriosclerosis index (AI) was calculated as described by Queiroz-Monici, Costa, da Silva, Reis, and de Oliveira (2005). Aliquots of liver were extracted with chloroform-methanol 2:1 (W/V) and the lipid extract was analysed for TC, TG and fat (Kritchevsky & Tepper, 2005). Other aliquots were kept at −70 °C for mRNA analysis of farnesoid X receptor (FXR), cholesterol 7 alpha-hydroxylase (CYP7A1), lipoprotein lipase (Lpl), hepatic lipase (Lipc), peroxisome proliferator-activated receptors (PPARα and PPARγ). Aliquots of ileum wall were kept at −70 °C for mRNA analysis of FXR and intestine-bile acid binding protein (I-BABP).

2.5. RNA preparations and reverse transcription (RT-PCR)

RNA was extracted using TRIzol kit (GK 3003, GeneRay Biotech (Shanghai) Co., Ltd., Shanghai, PR China). M-MLV reverse transcriptase (Premega, Madison, WI, USA) was used to reverse-transcribe the total RNA according to the manufacturer's instructions. The oligonucleotide primers of rattus target genes were designed using a PCR primer selection program at the website of the Virtual Genomic Center from the GenBank database (Table 2). Amplification was performed by PCR with a gradient thermal cycler (Techne TC-512, Durviz, Valencia, Spain). The reaction solution (30 μL final volume) contained 10 × PCR buffer 2.5 μL, dNTP 2.4 μL, 25 mmol/L $MgCl_2$ 0.6 μL, 20 pmol forward primer 0.6 μL and reverse primer 0.6 μL, 5 U/μL AmpliTaq DNA polymerase (Takara Shuzo Co., Shiga, Japan) 0.25 μL, cDNA 1.5 μL, dH_2O 24.05 μL. After predenaturation at 94 °C for 4 min, the PCR conditions were set as follows: denaturation at 94 °C for 30 s, annealing at 55 °C for 30 s, extension at 72 °C for 45 s, 35 cycles, at last extension at 72 °C for 5 min, then hold at 4 °C. A sample (1.5 μL) of each amplified product was subjected to electrophoresis in a 2% agarose gel, stained with ethidium bromide, and visualized under UV illumination system (Model FR-200A, Shanghai FURI Science & Technology Co., Ltd., Shanghai, PR China).

2.6. Statistical analysis

Comparison of the means was performed by one-way ANOVA using the honestly significant difference (HSD) of Tukey's ad-hoc test. The statistical analyses were done using SPSS 13.0 for Windows software (SPSS Institute Inc., Cary, NC).

3. Results and discussion

3.1. Serum and liver lipids

Results of serum and liver lipids are shown in Table 3. As compared with the HF and NF groups, the addition of CDF and XMF to a HF diet could both depress the increase of serum TG significantly ($p < 0.05$). TC values in treatment groups were significantly higher than that in NF group ($p < 0.05$), but significantly lower than that in HF group ($p < 0.05$). Furthermore, the TC value in XMF group was only 80% of that in CDF group with a

Table 2
Sequences of PCR primers and GenBank accession numbers

Gene[a]	GenBank accession number	Forward (FP), reverse (RP)[b]	Product size
CYP7A1	X17595	FP: GGCATTTGGACACAGAAGCATT RP: TGGTCCCTCACACACAGGTTCT	435 bp
I-BABP	L22788	FP: TCCGTCCTCAGTTGCCTCTC RP: TCCATCTTCACGGTTGCCTT	304 bp
FXR	NM_021745	FP: TTCACAAAAAGACTTCCAGGGT RP: CTTCTCCACTGCCTCTCTATCC	318 bp
PPARα	NM_013196	FP: GACCTGGAAAGTCCCTTATCTG RP: GCATTGTGTGACATCCCGAC	458 bp
PPARγ	NM_013124	FP: CTCCAAGAATACCAAAGTGCGA RP: AAACCTGATGGCATTGTGAGAC	336 bp
Lpl	NM_012598	FP: TGAGAACATTCCCTTCACCCT RP: CAGCGGAAGTAGGAGTCGTT	132 bp
Lipc	NM_012597	FP: GAGCCCAGTCCCCCTTCA RP: ATGTCATTCTTTGCTGCGTCTC	366 bp
β-Actin	NM_031144	FP: GTCAGGTCATCACTATCGGCAAT RP: AGAGGTCTTTACGGATGTCAACGT	147 bp

[a] FXR, farnesoid X receptor; CYP7A1, cholesterol 7 alpha-hydroxylase; Lpl, lipoprotein lipase; Lipc, liver lipase; PPARα, peroxisome proliferator-activated receptor α; PPARγ, peroxisome proliferator-activated receptor γ; I-BABP, intestine-bile acid binding protein; β-actin, positive control.

[b] The oligonucleotide primers of rattus target genes were designed using a PCR primer selection program at the website of the Virtual Genomic Center from the GenBank database.

Table 3
Serum and liver lipids in rats (8 per group) fed NF or HF or test fibre diets for 42 days (mean ± SD)

	NF	HF	CDF	XMF
Serum lipids				
TG (mmol/L)[A]	1.48 ± 0.19^{a}	5.58 ± 0.58^{c}	3.32 ± 0.21^{b}	2.99 ± 0.27^{b}
TC (mmol/L)	1.94 ± 0.22^{a}	7.54 ± 0.96^{d}	6.12 ± 0.84^{c}	4.89 ± 0.65^{b}
LDL-C (mmol/L)	0.61 ± 0.06^{a}	5.86 ± 1.16^{d}	4.10 ± 0.77^{c}	2.89 ± 0.54^{b}
HDL-C (mmol/L)	1.01 ± 0.23^{a}	0.82 ± 0.11^{a}	0.91 ± 0.11^{a}	1.40 ± 0.24^{b}
AI	0.89 ± 0.41^{a}	8.27 ± 1.36^{d}	5.82 ± 1.28^{c}	2.54 ± 0.48^{b}
Liver lipids				
TC (μmol/g)	8.37 ± 0.58^{a}	87.39 ± 7.02^{c}	81.18 ± 12.19^{c}	47.21 ± 2.30^{b}
TG (μmol/g)	5.75 ± 0.98^{a}	50.23 ± 5.17^{c}	44.0 ± 4.04^{c}	33.06 ± 2.06^{b}
Fat (%)	5.27 ± 0.03^{a}	25.72 ± 0.29^{d}	19.05 ± 0.41^{c}	17.63 ± 0.29^{b}

[a,b,c,d] Same superscript letters in a row show values that do not differ significantly ($p > 0.05$, $n = 8$).
[A] TC, total cholesterol; TG, total triacylglycerol; AI, arteriosclerosis index.

significance of $p < 0.05$. When compared with the HF diet, CDF and XMF efficiently decreased serum LDL-C level, and XMF was significantly more efficient than CDF ($p < 0.05$). Among TC, LDL-C and VLDL-C play as the most important factors for coronary heart disease (Goldstein et al., 1973). From Table 3, it was easy to deduce that the main cause of hypercholesterolemia was the high level of LDL-C in this study. During the four weeks feeding period, the studied dietary fibre CDF and XMF lowered TC mainly through decreasing the LDL-C level.

The high lipid diet did not lower HDL-C in rats significantly as reported elsewhere (Dong et al., 2000). There was a slight decrease of HDL-C in rats fed the HF and CDF diets at the end of week six but not significantly when compared with that in rats fed the NF diet ($p > 0.05$). However, HDL-C in rats fed XMF diet was increased significantly ($p < 0.05$). AI in the HF group was 9.3 folds of the NF group. The addition of CDF and XMF decreased AI to 70.4% and 30.7% of that in the HF group with a significance of $p < 0.05$ (Table 3), respectively.

There was a slight trend for the CDF diet to suppress the increase of liver TG and TC. On the other hand, the level of liver fat in rats fed the CDF diet was significantly lower than in rats fed the HF diet ($p < 0.05$, Table 3). Whereas the level of liver TG, TC and fat in rats fed the XMF diet were significantly lower than in rats fed the CDF diet ($p < 0.05$).

Supplementing a low-fat diet with corn bran was effective in reducing the serum lipid concentration in men with hypercholesterolemia (Shane & Walker, 1995). In type II diabetes patients, consumption of corn bran lowered the plasma very low-density lipoprotein cholesterol level. On the other hand, consumption of corn bran did not reduce the plasma cholesterol concentration in 10 healthy men (Ebihara & Nakamoto, 2001). A fibre supplement containing corn bran provided significant and sustained reductions in LDL-C without reducing HDL-C or increasing TG in subjects with mild to moderate hypercholesterolemia (Knopp et al., 1999). In the present study, XMF was significantly more efficient in suppressing the increase of serum TC and LDL-C, liver TC, TG and fat than CDF ($p < 0.05$). Furthermore, XMF increased HDL-C, and as a result decreased AI efficiently. Therefore, it is concluded that the hypolipidemic effects of XMF was significantly better than CDF.

3.2. Transcription of genes involved in cholesterol catabolism

Farnesoid X receptor (FXR) protein is an orphan nuclear receptor expressed predominantly in the liver, kidney, intestine, and adrenals. Activated FXR represses transcription of the gene encoding cholesterol 7 alpha-hydroxylase (CYP7A1), which is the rate-limiting enzyme in bile acid synthesis, and activates the gene encoding of intestinal bile acid binding protein (I-BABP), which is a candidate bile acid transporter. The activation of FXR was specific and limited to the primary bile acid chenodeoxycholic acid and to a much lesser extent to the secondary bile acids deoxycholic acid and lithocholic acid (Makishima et al., 1999). Thus, the transcriptions of the genes encoding I-BABP and CYP7A1 are related to the level of bile acids in body.

To observe the expression of genes from the liver or ileum as shown in Fig. 1, β-actin was used as a positive control. The mRNA expression level of every gene was a relative concept compared with the positive control.

The transcription level of I-BABP in the HF diet group was up-regulated when compared with the NF diet group, and was down-regulated a lot by the CDF diet. While the XMF diet down-regulated the transcription of I-BABP to the lowest level. (Fig. 1). The order of mRNA abundance of hepatic CYP7A1, from the highest to the lowest, was that in rats fed the XMF, CDF, HF, and NF diet, respectively. XMF could bind bile acids studied here more efficiently than CDF, especially the binding against deoxycholate and chenodeoxycholate (Hu et al., 2008). The presence of XMF or CDF in the intestine can decrease the intestinal concentration of deoxycholate and chenodeoxycholate, decrease the activation of ileum FXR and then down-regulate the transcription of I-BABP gene as shown in Fig. 1, so as to consequently lower the intestinal re-absorption of bile acids. The decreased enterohepatic

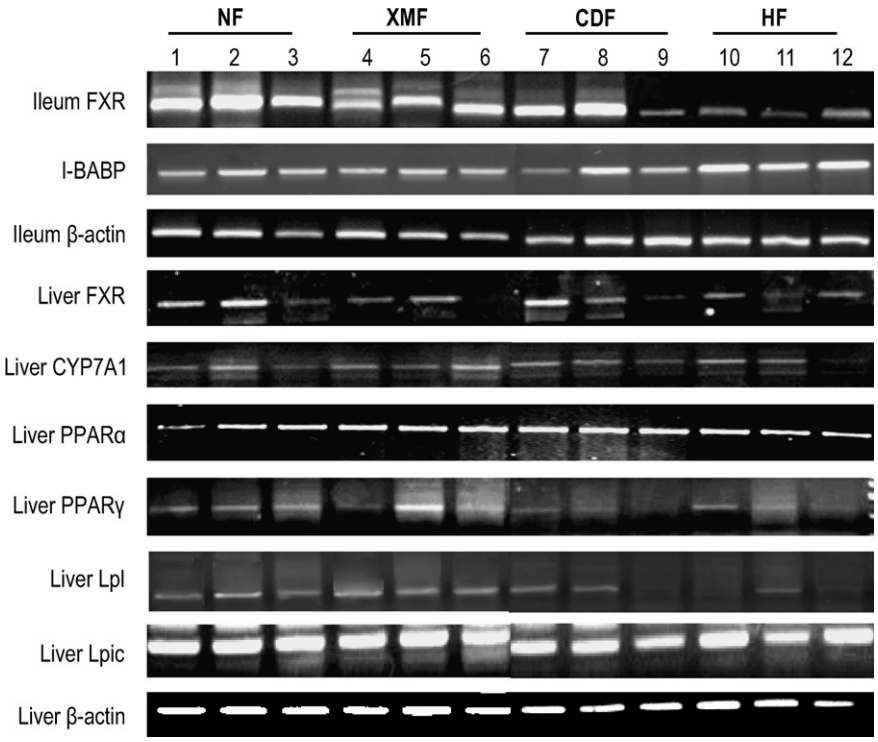

Fig. 1. mRNA transcription of genes involved in cholesterol metabolism. FXR, farnesoid X receptor; CYP7A1, cholesterol 7 alpha-hydroxylase; Lpl, lipoprotein lipase; Lipc, liver lipase; PPARα, peroxisome proliferator-activated receptor α; PPARγ, peroxisome proliferator-activated receptor γ; I-BABP, intestine-bile acid binding protein; β-actin, positive control. Three rats in each diet group were chosen randomly to excise aliquots of liver and ileum for mRNA analysis. NF, basic diet; HF, atherogenic diet; CDF, HF diet with an addition of 10% of CDF (corn bran fibre); XMF, HF diet with an addition of 10% of XMF (xylanase modified corn bran fibre). Rats in NF group were fed NF diet all the time; rats in HF group were fed HF diet all the time. Rats in CDF and XMF groups were fed HF diet in the first two weeks, then changed to CDF and XMF diet, respectively, and fed for four weeks.

circulation of bile acids might be in favour of the export of bile acids synthesized in liver to form a low concentration status of bile acids, which keeps FXR at a low activated level. So the transcription of CYP7A1 is up-regulated. Increased expression of CYP7A1 mRNA enhances bile acids biosynthesis from cholesterol, reduces circulation levels of cholesterol. High cholesterol diet was found to up-regulate the CYP7A1 mRNA expression in mice (Li, Hou, Tang, & Ling, 2004). The HF diet also made rats up-regulate the expression of hepatic CYP7A1 gene in the present study. CDF enhanced the expression slightly when compared with HF diet, whereas XMF up-regulated the CYP7A1 transcription more significantly than CDF, which explains why XMF prevented the progression of hyperlipidemia in rats more efficiently than CDF.

The ileum FXR transcription level was relatively highest in rats fed the NF diet, and then in rats fed the CDF and XMF diet, it was weakest in rats fed the HF diet (Fig. 1). Such an expression trend of ileum FXR mRNA indicated that high fat and cholesterol diet with cholic acid addition used here could down-regulate the transcription of the FXR gene in the ileum, the addition of CDF and XMF decreased the down-regulation efficiently. In the liver, the transcription of FXR was stronger on the NF diet than on the HF diet. However, the addition of XMF in HF diet enhanced the down-regulation further, and the addition of CDF up-regulated the FXR mRNA expression when compared with HF diet. The transcription trend of the hepatic FXR in NF, HF and XMF groups was just opposite to that of CYP7A1, which indicates that the ingestion of XMF could regulate the expression of FXR and CYP7A1 genes positively at the same time. This might be one of the deep reasons that XMF in HF diet lowered TC, TG and AI more efficiently than CDF.

3.3. Transcription of genes involved in triacylglycerols catabolism

Peroxisome proliferator-activated receptors (PPARs) are ligand-activated transcription factors of a nuclear receptor superfamily. PPARs have three subtypes of actors: α, σ and γ (Kersten, Desvergne, & Wahli, 2000). PPARα regulates the expression of target genes involved in lipid catabolism and plays a role in the clearance of circulating and cellular lipids. PPARγ activation in adipose tissues induces a decrease in circulating lipid levels. The transcription of genes involved in lipid metabolism such as Lpl was correlated with the activation of PPARγ (Takahashi et al., 2002). Recently, it was thought that a dual or cross activation of both PPARα and PPARγ is important for the improvement of hyperlipidemia and diabetes associated with obesity.

The mRNA expression of hepatic PPARα in rats fed the XMF diet was close to that in those fed the CDF diet, while that in rats fed the NF diet was close to that in those fed the HF diet. The abundance in the former two groups was higher (Fig. 1), which indicated that XMF and CDF ingestion could both improve the PPARα pathway, enhanced the clearance of circulating and cellular lipids.

For the relative expression level of hepatic PPARγ, it was down-regulated by the HF diet as compared with the NF diet. However, the highest expression was in rats fed the XMF diet, and the lowest was in rats fed the CDF diet (Fig. 1). In rats fed the HF diet, there was only a trace mRNA expression of Lpl gene. The relative transcription of Lpl was highest in rats fed the XMF diet, followed by in those fed the NF diet, and was significantly lower in rats fed the CDF diet. These indicated that the ingestion of XMF and CDF could increase the activation of PPARγ, and XMF was more efficient than CDF. Furthermore, it might be deduced that the HF diet can interfere with the PPARγ pathway, and XMF enhances lipid catabolism through protecting the PPARγ pathway efficiently. Soy isoflavones reportedly exerted antidiabetic and hypolipidemic effects through the PPAR pathways in obese Zucker rats and murine RAW 264.7 Cells (Mezei et al., 2003), but there is no previous report about the mechanism of hypolipidemic effects of dietary fibre on this aspect. However, we are unable to explain why the expression of hepatic PPARγ was the weakest in rats fed the CDF diet.

As compared with the NF diet group, the HF diet down-regulated the mRNA expression of the hepatic Lipc gene apparently. Its strongest expression occurred in rats fed the NF or/and XMF diet, while the lowest was observed in those fed the CDF diet. Liver lipase encoded by the Lipc gene is released in the endothelium cells of hepatic antrum, which plays an important role in liver triacylglycerols catabolism (Anonymous, 2007-1-03 20:00). The down-regulation of Lipc by HF diet accounts for the injured liver function after six weeks' ingestion of HF diet, which is in line with the high content of liver fat. While the ingestion of XMF could protect the hepatic tissue from being damaged by the HF diet, maintain the hepatic lipase activity and as a result improve the lipid metabolism. This also explained why XMF presented a better hypolipidemic profile than CDF. Just like for the PPARγ, we could not explain the lowest mRNA expression of Lipc in rats fed the CDF diet.

It was suggested that the diminished absorption of bile acid caused by beet fibre in the intestine might alter ileal apolipoprotein A-I gene expression (Sonoyama et al., 1995). The expression of these genes studied here might also be influenced by the decreased re-absorption of bile acids in the intestine. On the other hand, some special hydrolysate might be concluded to be efficient for the regulation of genes investigated in the present study. Some further investigations are being undertaken to understand the two above deductions.

4. Conclusions

The hypolipidemic effects of corn bran fibre modified by xylanase (XMF) and the original corn bran dietary fibre (CDF), and the regulating effects of XMF and CDF on the mRNA expression of several genes involved in lipids metabolism in rats were examined. CDF decreased the increase of serum TG, TC and LDL-C in rats by acting as bile acid sequestrants, down-regulating the transcription of I-BABP, up-regulating the transcription of ileum FXR, hepatic CYP7A1, Lpl and PPARα. While through efficiently up-regulating the transcription of CYP7A1, PPARα, PPARγ, Lpl and Lipc in liver and FXR in ileum, down-regulating the expression of hepatic FXR and ileum I-BABP, XMF appeared to be more capable than CDF, and then repressed the enterohepatic transport of bile acids, reduced the rates of lipogenesis, enhanced the catabolism of cholesterol and triacylglycerols. Our findings reported here not only provide a significant molecular basis of how corn bran dietary fibre improve hyperlipidemia, but also suggest the possibility that modified corn bran fibre-XMF has therapeutic applications in lipid abnormalities, such as hyperlipidemia.

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