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Effect of soybean oligosaccharides on blood lipid, glucose levels and antioxidant enzymes activity in high fat rats

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ABSTRACT

The effect of soybean oligosaccharides on blood lipid levels and oxidative stress in rats fed on high-fat diet was investigated. Rats were divided into five groups of 10 animals each. The high-fat group received a high-fat diet containing 18% (w/w) lipid in the diet (36% of total energy). Animals allocated to the soybean oligosaccharides-treatment groups (I, II and III) received the high-fat diet and orally fed with soybean oligosaccharides at a single dose of 150, 300 and 450 mg/kg body weight, respectively. Control rats received basic diet. Results showed that soybean oligosaccharides significantly reduced abnormal blood glucose, lipid level and oxidative stress in animal models at all doses examined. Soybean oligosaccharides were able to reduce oxidative stress and improve abnormal blood lipid levels induced by high-fat diets. In summary, the present study may be important for reverse cardio-cerebrovascular disease.

1. Introduction

Soybeans are unique foods because of their rich nutrient content. They contain complex carbohydrates, protein, dietary fibre, oligosaccharides, phytochemicals (especially the isoflavones in soy) and minerals (Refstie, Storebakken, & Roem, 1998). Carbohydrates are the second largest component in soybeans. Their complex carbohydrates and dietary fibre contents contribute to their low glycemic index, which benefits diabetic individuals (Jenkins, Wolever, & Taylor, 1981) and reduces the risk of developing diabetes (Salmeron et al., 1997). Soybean seed is a rich source of oligosaccharides, namely raffinose and stachyose: raffinose is a trisaccharide containing galactose linked α-(1–6) to the glucose unit of sucrose; stachyose is a tetrasaccharide containing a galactose linked α-(1–6) to the terminal galactose unit of raffinose (Kim, Kim, & Hwang, 2003). Other reported major sugar of soybeans is sucrose with lower amounts of the monosaccharides, fructose, rhamnose and arabinose; significant levels of glucose occurred only in immature seeds (Van der Riet, Wight, Cilliers, & Datel, 1989).

High-fat diets are reported to increase oxidative stress in a variety of tissues, which may result in many degenerative diseases (Chen, Zhong, Zeng, & Ge, 2008; Lieber et al. 2007; Ma, Liu, Yu, Chen, & Zhang, 2009; Schreibelt et al., 2007). Antioxidant supplementation prevents many diseases attributed to high-fat diet (Chen, Shen, & Chen, in press; Chen, Zhong, Zhu, Zeng, & Dai, 2009; Hong, Wu, Ma, Liu, & He, 2009; Zhu, Wang, Zhang, Pei, & Fen, 2008). There have been several reports describing the biological activities of soybean oligosaccharides such as antioxidant, blood pressure lowering and antidiabetic activities (Huang et al., 2006; Zhao & Yang, 2007). Soybean oligosaccharides and various derivatives can stabilise lipids in formulated foods. Huang et al. (2006) have reported the effect of soybean oligosaccharides on antioxidant enzymes and the immunity activity of broilers. Deng, Mai, Ai, and Zhang (2007) reported that dietary soybean oligosaccharides (SBOS) could decrease the incidences of fatty liver of the fish fed soy protein isolate (SPI)-based diets.

In this work, the effect of soybean oligosaccharides on blood lipid, glucose levels and antioxidant enzyme activity in HF rats was studied.

2. Materials and methods

2.1. Materials

Soybean oligosaccharides (SBOS) were purchased from XiAn ChenXing Plant Science Technology Co. Ltd. (XiAn, China).

Abbreviations: SBOS, soybean oligosaccharides; TC, total cholesterol; LDL-c, low-density lipoprotein cholesterol; TG, triacylglycerols; HDL-c, high density lipoprotein cholesterol; HF, high fat; SOD, superoxide dismutase; CAT, catalase; GSH-Px, glutathione peroxidase; AI, atherosclerosis index; MDA, malondialdehyde.

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2.2. Animals, diets and experimental design

All animal protocols were approved by the animal care and use committee of Suzhou University (Suzhou City, China). Wistar rats, aged 4 weeks of age, were obtained from the Laboratory Animal Center of our university. The rats were preferred as a model of spontaneous human obesity over other genetically altered animals because when wistar rats are fed high-fat diets (Table 1), they become obese, hyperinsulinemic, and hyperlipidemic and display characteristics of metabolic syndrome. The rats were kept at a constant temperature of 22 °C and exposed to a 12/12 h (light/dark) cycle. After adaptation for 1 week, rats were divided into five groups of 10 animals each: control group; HF model group; oligosaccharides-treated group (I); oligosaccharides-treated group (II); and oligosaccharides-treated group (III).

Rats in control group were allowed free access to basic diet, water and orally administrated with the same volume of physiological saline for 45 consecutive days.

Rats in HF model group were allowed free access to high-fat diet, water and treated with the same volume of physiological saline for that same period.

Rats of oligosaccharides-treated group (I) were allowed free access to high-fat diet, water and were treated by oral infusion with oligosaccharides at a dose of 150 mg/kg BW/day dissolved in physiological saline for that same period.

Rats of oligosaccharides-treated group (II) were allowed free access to high-fat diet, water and were treated by oral infusion with oligosaccharides at a dose of 300 mg/kg BW/day dissolved in physiological saline for that same period.

Rats of oligosaccharides-treated group (III) were allowed free access to high-fat diet, water and were treated by oral infusion with oligosaccharides at a dose of 450 mg/kg BW/day dissolved in physiological saline for that same period.

Body weight was recorded weekly. On completion of the experiment, all rats were weighed and blood was collected via the postcaval vein from anesthetised animals into blood collection tubes between 09:30 and 10:00 h after 12 h of food deprivation. Plasma was prepared by centrifugation of blood at 1000×*g* for 15 min at 4 °C and stored at −80 °C until analysed. Immediately after blood collection, rats were killed by decapitation and livers were then removed, weighed and stored at −80 °C until used for preparation of biochemical analysis.

2.3. Biochemical analysis

2.3.1. Blood lipid profile analysis

Plasma total cholesterol (TC), triacylglycerols (TG), low-density lipoprotein cholesterol (LDL-c), and high-density lipoprotein cholesterol (HDL-c) levels were measured by enzymatic and colorimetric methods, using assay kits from Sigma Diagnostics (St. Louis, MO, USA).

2.3.2. Antioxidant enzymes activities analysis

The SOD activity in the liver and blood was assayed by the inhibition of xanthine/xanthine oxidase mediated reduction of cytochrome c as previously described method (Flohe & Ötting, 1984. One unit of SOD activity in the liver and blood was defined as the amount of enzyme required to give 50% inhibition in the typical calibration curve obtained with standard SOD and was expressed as U/mg protein (ml serum).

The GSH-Px activity was determined in the liver and blood by the method reported by Paglia and Valentine (1975), respectively. In brief, tissue homogenates were centrifuged at 600×*g* for 10 min at 4 °C to remove crude fractions. Then, supernatants were centrifuged at 10 000×*g* for 20 min. One unit of enzyme activity has been defined as *n* moles of NADPH consumed/min/mg protein based on an extinction coefficient of 6.66 mM/cm.

Table 1
Diet composition (g/kg dry matter basis).

Ingredients (g/kg)	Basic diet	High fat
Casein	140	140
Wheat bran	160	160
Sucrose	95	95
Lard	40	160
tert-Butylhydroquinone	0.007	0.007
Minerals mixture	34	34
Vitamin mixture	11	11
L-Cystine	1.8	1.8
Corn starch	435.58	315.58
Choline bitartrate	2.5	2.5
Cellulose	80.12	80.12
Total	1000.07	1000.07

Rats of control group consumed 18.8 ± 0.92 g chows per day, approximately providing 59.7 ± 3.5 kcal daily.
Rats of HF group and oligosaccharides-treated groups (I, II and III) consumed 19.1 ± 0.88 g chows per day, approximately providing 132.5 ± 6.4 kcal daily.
HF: high fat.

CAT activity was assayed by the method described by Trombino et al. (2009). The enzyme-catalysed decomposition of H_2O_2 was measured. In brief, 0.5 ml aliquot of cold CAT sample and a blank consisting of 0.5 ml distilled water was taken in test tubes and the enzymatic reactions was initiated by adding 5 ml of cold 6 mM H_2O_2 and mixed thoroughly. After exactly 3 min the reaction was stopped by rapidly adding 1 ml 3 M H_2SO_4 and mixed thoroughly. Then 7 ml of 0.01 M $KMnO_4$ reagent was added, mixed thoroughly and the reading was taken at 480 nm within 30–60 s.

Serum glucose were analysed by the enzymatic reaction method using a commercially available kit by DiaSys Diagnostic Systems (Holzheim, Germany).

The concentration of TBARS was measured by a modification of the method of Yagi (1984) and calculated as malondialdehyde (MDA) equivalents using a commercial kit (Oxi-Tek, Zeptometrix Corporation, Buffalo, NY, USA).

Liver index was calculated by the formula: liver index = (liver weight/body weight) × 100. Atherosclerosis index (AI) was calculated by the formula: atherosclerosis index = (serum total cholesterol − HDL-c)/HDL-c.

2.4. Statistical methods

Analysis was performed according to the intention to treat principle. All data are expressed as mean ± SEM. Statistical analyses were performed with SPSS version 11.5 (SPSS Institute, Chicago, IL, USA). Student *t* test was used to assess the statistical significance of the continuous variables. A value of $P < 0.05$ was used as a criterion for statistical significance.

3. Results

3.1. Effect of SBOS on rats' body weight

As shown in Table 2, body weight in the HF group was significantly higher than that in the control group ($P < 0.05$). There were no significant differences between the HF group and SBOS-treated group ($P > 0.05$).

3.2. Effect of SBOS on rats' serum TC, TG, LDL-c, HDL-c levels and AI

Table 3 showed that effect of SBOS on rats' serum TC, TG, LDL-c, HDL-c levels and AI. Serum TC, TG, LDL-c levels, AI in the HF group were significantly higher, whereas HDL-c level was significantly

Table 2
Effect of SBOS on rats' body weight.

Group	Initial weight (g)	Final weight (g)
Control	164.35 ± 13.25	259.78 ± 12.84
HF	169.25 ± 17.03	283.15 ± 20.15[a]
SBOS (I)	158.37 ± 14.26	284.16 ± 17.38
SBOS (II)	158.92 ± 12.58	280.38 ± 19.26
SBOS (III)	166.27 ± 23.51	277.94 ± 22.11

All data are expressed as mean ± SEM of 10 rats. Statistical analyses were performed with SPSS version 11.5 (SPSS Institute, Chicago, IL). Student *t* test was used to assess the statistical significance of the continuous variables.
[a] $P < 0.05$, compared with control group.
HF: high fat; SBOS: soybean oligosaccharides.

lower than those in the control group ($P < 0.01$). The addition of SBOS to the high-fat diet dose-dependently significantly decreased serum TC, TG, LDL-c, levels and AI, and enhanced HDL-c level ($P < 0.05$, $P < 0.01$).

3.3. Effect of SBOS on rats' blood glucose level and liver index

Table 4 shows effect of SBOS on rats' blood glucose level and liver index. Blood glucose level and liver index in the HF group was significantly higher than those in the control group ($P < 0.01$). The addition of SBOS to the high-fat diet dose-dependently significantly reduced blood glucose level and liver index ($P < 0.05$ and $P < 0.01$).

3.4. Effect of SBOS on rats' serum SOD, CAT, GSH-Px activities and TBARS level

As shown in Table 5, serum SOD, CAT, GSH-Px activities in the HF group were significantly lower, whereas TBARS level in the HF group was significantly higher than those in the control group ($P < 0.05$). SBOS dose-dependent significantly enhanced serum SOD, CAT, GSH-Px activities and reduced TBARS level in SBOS-treated groups when compared with the HF groups ($P < 0.01$).

3.5. Effect of SBOS on rats' liver SOD, CAT, GSH-Px activities and TBARS level

As shown in Table 6, liver SOD, CAT, GSH-Px activities in the HF group were significantly lower, whereas TBARS level in the HF group was significantly higher than those in the control group ($P < 0.05$). SBOS dose-dependently significantly enhanced liver SOD, CAT, GSH-Px activities and reduced TBARS level in SBOS-treated groups when compared with the HF groups ($P < 0.05$ and $P < 0.01$).

4. Discussion

High-fat diets have been shown to produce more rapid weight gain in rodents (Akbay et al., 2004). In the present study, we

Table 4
Effect of SBOS on rats' blood glucose level and liver index.

Group	Blood glucose (mmol/l)	Liver index
Control	5.26 ± 0.38	3.83 ± 0.21
HF	18.32 ± 1.[05a]	4.37 ± 0.12[a]
SBOS (I)	15.37 ± 1.11[c]	4.31 ± 0.09
SBOS (II)	13.24 ± 0.93[c]	4.11 ± 0.10[b]
SBOS (III)	10.28 ± 1.04[c]	3.92 ± 0.09[c]

All data are expressed as mean ± SEM of 10 rats. Statistical analyses were performed with SPSS version 11.5 (SPSS Institute, Chicago, IL). Student *t* test was used to assess the statistical significance of the continuous variables.
[a] $P < 0.01$, compared with control group.
[b] $P < 0.05$ and [c] $P < 0.01$, compared with HF group.
HF, high fat; SBOS, soybean oligosaccharides.

Table 5
Effect of SBOS on rats' serum SOD, CAT, GSH-Px activities and TBARS level.

Group	SOD (U/ml)	CAT (U/ml)	GSH-Px (U/ml)	TBARS (nmol/ml)
Control	153.58 ± 11.42	17.49 ± 1.07	9.48 ± 0.53	10.05 ± 0.88
HF	87.71 ± 4.82[a]	10.22 ± 0.82[a]	4.13 ± 0.22[a]	18.42 ± 1.43[a]
SBOS (I)	107.51 ± 11.52[b]	14.63 ± 1.21[b]	5.94 ± 0.33[b]	15.11 ± 0.72[b]
SBOS (II)	137.31 ± 8.37[b]	15.83 ± 0.94[b]	7.27 ± 0.53[d]	13.44 ± 0.93[b]
SBOS (III)	161.13 ± 9.35[b]	17.05 ± 1.31[b]	8.27 ± 0.71[b]	10.73 ± 1.53[d]

All data are expressed as mean ± SEM of 10 rats. Statistical analyses were performed with SPSS version 11.5 (SPSS Institute, Chicago, IL). Student *t* test was used to assess the statistical significance of the continuous variables.
[a] $P < 0.01$, compared with control group
[b] $P < 0.01$, compared with HF group.
HF, high fat; SBOS, soybean oligosaccharides; SOD, superoxide dismutase; CAT, catalase; GSH-Px, glutathione peroxidase; TBARS, thiobarbituric acid reactive substances.

Table 6
Effect of SBOS on rats' liver SOD, CAT, GSH-Px activities and TBARS level.

Group	SOD (U/ml)	CAT (U/ml)	GSH-Px (U/ml)	TBARS (nmol/ml)
Control	211.47 ± 7.69	22.69 ± 1.63	11.73 ± 0.79	8.89 ± 0.48
HF	125.32 ± 11.72[a]	13.56 ± 1.27[a]	6.96 ± 0.47[a]	15.47 ± 1.03[a]
SBOS (I)	139.41 ± 16.92[b]	16.25 ± 0.94[c]	8.71 ± 0.77[c]	13.39 ± 1.71[b]
SBOS (II)	172.08 ± 14.27[c]	19.63 ± 1.04[c]	10.36 ± 1.53[c]	10.38 ± 0.91[c]
SBOS (III)	207.44 ± 11.58[c]	21.32 ± 1.41[c]	14.03 ± 1.05[c]	9.03 ± 0.58[c]

All data are expressed as mean ± SEM of 10 rats. Statistical analyses were performed with SPSS version 11.5 (SPSS Institute, Chicago, IL). Student *t* test was used to assess the statistical significance of the continuous variables.
[a] $P < 0.01$, compared with control group.
[b] $P < 0.05$ and [c] $P < 0.01$, compared with HF group.
HF, high fat; SBOS, soybean oligosaccharides; SOD, superoxide dismutase; CAT, catalase; GSH-Px, glutathione peroxidase; TBARS, thiobarbituric acid reactive substances.

Table 3
Effect of SBOS on rats' serum TC, TG, LDL-c, HDL-c levels and AI.

Group	TC (mmol/l)	TG (mmol/l)	LDL-c (mmol/l)	HDL-c (mmol/l)	AI
Control	2.17 ± 0.17	0.97 ± 0.04	0.44 ± 0.03	1.96 ± 0.11	0.12 ± 0.04
HF	4.10 ± 0.27[b]	1.91 ± 0.08[b]	0.93 ± 0.05[b]	1.05 ± 0.08[b]	2.91 ± 0.39[b]
SBOS (I)	3.58 ± 0.14[d]	1.65 ± 0.19[c]	0.79 ± 0.08[c]	1.27 ± 0.07[d]	1.83 ± 0.41[a]
SBOS (II)	2.99 ± 0.21[d]	1.33 ± 0.12[d]	0.66 ± 0.05[d]	1.56 ± 0.08[d]	0.93 ± 0.18[d]
SBOS (III)	2.52 ± 0.11[d]	1.13 ± 0.13[d]	0.53 ± 0.07[d]	1.91 ± 0.12[d]	0.32 ± 0.05[d]

All data are expressed as mean ± SEM of 10 rats. Statistical analyses were performed with SPSS version 11.5 (SPSS Institute, Chicago, IL). Student *t* test was used to assess the statistical significance of the continuous variables.
[b] $P < 0.01$, compared with control group.
[c] $P < 0.05$ and [d] $P < 0.01$, compared with HF group.
HF, high fat; SBOS, soybean oligosaccharides; TC, total cholesterol; LDL-c, low-density lipoprotein cholesterol; TG, triacylglycerols; HDL-c, high-density lipoprotein cholesterol; AI, atherosclerosis index.

evaluated whether a high-fat diet could induce pathogenesis in the obese rat compared with normal controls. The results showed that over a period of 1 month, the high-fat fed animals weighed significantly more than the control group, which demonstrated that exposure to a high-fat diet led to increased weight gain and impairments in blood glucose regulation. The results of the present study still showed that SBOS can reduce blood glucose level and liver index in experimental animals, but did not alter rats' body weight.

Antioxidant enzymes are capable of scavenging reactive oxygen species and products of lipid peroxidation, thereby protecting cells and tissues from oxidative damage. To prevent oxidative stress, there is an ongoing balance between antioxidants and ROS. When there is an imbalance, ROS may accumulate and trigger oxidative injury by lipid peroxidation, and protein oxidation, accompanied by increased toxic product synthesis and cell death (Abadie, Malcom, Porter, & Svec, 2001; Choi, Park, & Park, 2002). Superoxide dismutases convert superoxide radicals to molecular oxygen and H_2O_2, and catalase decomposes H_2O_2 to molecular oxygen and water (Tian, Cai, & Wei, 1998). There is a variety of evidence indicating that antioxidant enzyme activities are lower in animals fed with HF diet (Malheiros et al., 2003). A lipid profile measures total cholesterol, HDL cholesterol, LDL cholesterol, and triacylglycerols are specific concern about cardiovascular disease, especially coronary artery disease. In this work, oxidative stress occurred in rats fed HF diet as evident by elevated levels of malondialdehyde and decreased antioxidant activities in serum and livers, suggesting increased lipid peroxidation and oxidative stress. Abnormal blood lipid levels were also observed in all rats fed HF diet. A study showed that a long-term of HF diet administration could increase blood glucose level in animals, which was confirmed by our work. Activities of antioxidant enzymes were lowest in rats fed HF diet, which is likely to exacerbate existing oxidative stress. Huang et al. (2006) reported that the activities of antioxidant enzymes in liver such as catalase, SOD, and GPx were significantly elevated when SBOS was administered to rats orally once per day for 6 weeks. Zhao and Yang (2007) reported that soybean oligosaccharides can decrease blood lipid levels in patients with hyperlipidemia. Our *in vivo* study showed that SBOS can dose-dependently reduce oxidative stress and improve abnormal blood lipid levels in the SBOS-treated rats. The positive correlation between polyphenolic content of polysaccharide and its antioxidant activity is well documented (Huang & Mau, 2006). Therefore, the content of total phenolic compounds in the extracts might explain their high antioxidant activities. In this study SBOS also showed a remarkable antioxidant activity, one of the possible mechanisms is polyphenolic-associated polysaccharide (formation of non-extractable complex between high molecular weight phenolics and polysaccharides). Those kinds of phenolic compounds show antioxidant activity due to their redox properties, which play an important role in absorbing and neutralising free radicals, quenching singlet and triple oxygen or decomposing peroxide. It may be suggested that SBOS reduced abnormal blood glucose levels in HF rats by enhancing antioxidant enzymes activities and decreasing oxidative injury.

In summary, an HF diet caused hyperglycemia and oxidative injury occurred in rats fed HF diet, as did elevated blood glucose production and oxidative stress. A 30-day treatment with SBOS can reduce oxidative stress and improve abnormal blood lipid levels in the SBOS-treated rats.

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References

Abadie, J. M., Malcom, G. T., Porter, J. R., & Svec, F. (2001). Can associations between free fatty acid levels and metabolic parameters determine insulin resistance development in obese Zucker rats? *Life Science, 69*, 2675–2683.

Akbay, E., Ulusu, N. N., Töröner, F., Ayvaz, G., Taneri, F., Aktürk, M., et al. (2004). Effects of rosiglitazone treatment on the pentose phosphate pathway and glutathione-dependent enzymes in liver and kidney of rats fed a high-fat diet. *Current Therapeutic Research, 65*, 79–89.

Chen, Y. G., Shen, Z. J., & Chen, X. P. (2009). Evaluation of free radicals scavenging and immunity-modulatory activities of Purslane polysaccharides. *International Journal of Biological Macromolecules*. doi:10.1016/j.ijbiomac.2009.07.009.

Chen, X., Zhong, H. Y., Zeng, J. H., & Ge, J. (2008). The pharmacological effect of polysaccharides from *Lentinus edodes* on the oxidative status and expression of VCAM-1mRNA of thoracic aorta endothelial cell in high-fat-diet rats. *Carbohydrate Polymers, 74*, 445–450.

Chen, X., Zhong, H. Y., Zhu, H., Zeng, J. H., & Dai, P. (2009). Effect of *Curcuma kwangsiensis* polysaccharides on blood lipid profiles and oxidative stress in high-fat rats. *International Journal of Biological Macromolecules, 44*, 138–142.

Choi, S. B., Park, C. H., & Park, S. (2002). Effect of cola intake on insulin resistance in moderate fat-fed weaning male rats. *Journal of Nutritional Biochemistry, 13*, 727–733.

Deng, J. M., Mai, K. S., Ai, Q. H., & Zhang, W. B. (2007). Effects of soybean oligosaccharides on lipid metabolism of Japanese flounder (*Paralichthys olivaceus* Temminck et Schlegel) fed animal or plant protein source-based diets. *Frontiers of Agriculture in China, 1*, 315–323.

Flohe, L., & Ötting, F. (1984). Superoxide dismutase assays. *Methods in Enzymology, 105*, 93–104.

Hong, Y.-K., Wu, H.-T., Ma, T., Liu, W.-J., & He, X.-J. (2009). Effects of *Glycyrrhiza glabra* polysaccharides on immune and antioxidant activities in high-fat mice. *International Journal of Biological Macromolecules, 45*, 61–64.

Huang, S. J., & Mau, J. L. (2006). Antioxidant properties of methanolic extracts from *Agaricus blazei* with various doses of g-irradiation. *LWT, 39*, 707–716.

Huang, R. L., Zhang, Z. H., Chen, P. G., Ma, K. W., Yang, L., Chen, H., et al. (2006). Effect of soybean-oligosaccharides on antioxidation and the immunity of broliers. *Journal of Agricultural University of HeBei, 29*, 88–94 (in Chinese).

Jenkins, D. J. A., Wolever, T. M. S., & Taylor, R. H. (1981). Glycemic index of foods: A physiological basis for carbohydrate exchange. *American Journal of Clinical Nutrition, 34*, 362–366.

Kim, S., Kim, W., & Hwang, I. K. (2003). Optimization of the extraction and purification of oligosaccharides from defatted soybean meal. *International Journal of Food Science & Technology, 38*, 337–342.

Lieber, C. S., Leo, M. A., Cao, Q., Mak, K. M., Ren, C. L., Ponomarenko, A., et al. (2007). The combination of S-adenosylmethionine and dilinoleoylphosphatidylcholine attenuates nonalcoholic steatohepatitis produced by a high-fat diet in rats. *Nutrition Research, 27*, 565–573.

Ma, M., Liu, G. H., Yu, Z. H., Chen, G., & Zhang, X. (2009). Effect of the *Lycium barbarum* polysaccharides administration on blood lipid metabolism and oxidative stress of mice fed high-fat diet in vivo. *Food Chemistry, 113*, 872–877.

Malheiros, R. D., Moraes, V. M. B., Collin, A., Janssens, G. P. J., Decuypere, E., & Buyse, J. (2003). Dietary macronutrients, endocrine functioning and intermediary metabolism in broiler chickens: Pair wise substitutions between protein, fat and carbohydrate. *Nutrition Research, 23*, 567–578.

Paglia, D. E., & Valentine, W. N. (1975). Characteristics of a pyrimidine-specific 5′-nucleotidase in human erythrocytes. *Journal of Biological Chemistry, 250*, 7973–7979.

Refstie, S., Storebakken, T., & Roem, A. J. (1998). Feed consumption and conversion in Atlantic salmon (*Salmo salar*) fed diets with fish meal, extracted soybean meal or soybean meal with reduced content of oligosaccharides, trypsin inhibitors, lectins and soya antigens. *Aquaculture, 162*, 301–312.

Salmeron, J., Manson, J. E., Stampfer, M. J., Colditz, G., Wing, A. L., & Willett, W. C. (1997). Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *The Journal of the American Medical Association, 277*, 472–477.

Schreibelt, G., van Horssen, J., van Rossum, S., Dijkstra, C. D., Drukarch, B., & de Vries, H. E. (2007). Therapeutic potential and biological role of endogenous antioxidant enzymes in multiple sclerosis pathology. *Brain Research Reviews, 56*, 322–330.

Tian, L. Q., Cai, Q. Y., & Wei, H. C. (1998). Alterations of antioxidant enzymes and oxidative damage to macromolecules in different organs of rats during aging. *Free Radical Biology & Medicine, 24*, 1477–1484.

Trombino, S., Cassano, R., Bloise, E., Muzzalupo, R., Tavano, L., & Picci, N. (2009). Synthesis and antioxidant activity evaluation of a novel cellulose hydrogel containing trans-ferulic acid. *Carbohydrate Polymers, 75*, 184–188.

Van der Riet, W. B., Wight, A. W., Cilliers, J. J. L., & Datel, J. M. (1989). Food chemical investigation of tofu and its byproduct okara. *Food Chemistry, 34*, 193–202.

Yagi, K. (1984). Assay for blood plasma or serum. *Methods in Enzymology, 105*, 328–331.

Zhao, Y. J., & Yang, Y. H. (2007). Random double blind test on effect of soybean oligosaccharides in regulation of blood lipid in patients with hyperlipidemia. *Progress in Modern Biomedicine, 7*, 1526–1535 (in Chinese).

Zhu, M. Y., Wang, C. J., Zhang, H. S., Pei, X. W., & Fen, J. M. (2008). Protective effect of polysaccharides from *Morinda officinalis* on bone loss in ovariectomized rats. *International Journal of Biological Macromolecules, 43*, 276–278.