ORIGINAL ARTICLE

Hypocholesterolemic effects of *Auricularia auricula* ethanol extract in ICR mice fed a cholesterol-enriched diet

Gang Chen · Yang-Chao Luo · Bao-Ping Ji · Bo Li · Wei Su · Zhen-Lei Xiao · Gui-Zhi Zhang

Abstract The cholesterol-lowering properties of *Auricularia auricula* are commonly attributed to the presence of polysaccharides based on previous research. The present study was designed to investigate the effects of ethanol extract of *A. auricula* (AAE) on hypercholesterolemia in ICR mice. AAE contained more than 16% (g/g) polyphenolic compounds, excluding other interfering factors such as polysaccharides, water-soluble fibre and protein. Thirty-six mice were randomly assigned to three groups ($n=12$). The experimental group was fed cholesterol-enriched diet (CED) with oral administration of AAE (150 mg/kg/d b.w.) for 8-week, normal control group and CED control group received either a regular diet (RD) or CED along with oral administration of equal volume distilled water. Serum lipid profiles and antioxidant status were measured in addition to fecal neutral cholesterol and bile acids. AAE showed a remarkable hypocholesterolemic effect, improving antioxidant status, decreasing the level of total cholesterol and atherosclerosis index, increasing the level of high-density lipoprotein cholesterol and fecal excretion of bile acids. No apparent effects on serum triglycerides, low-density lipoprotein cholesterol, fecal excretion of neutral cholesterol and feeding efficiency were observed among all groups. These results indicated that *A. auricula* functional components, which prevented hypercholesterolemia contained polyphenolic compounds, in addition to polysaccharides.

Keywords *Auricularia auricula* · Hypercholesterolemia · Polyphenolic compounds · HMG-CoA reductase · Antioxidant status

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Introduction

Auricularia auricula is a precious macro-fungus and its fruiting bodies have been widely used in Chinese cuisine and are known for their pharmaceutical effects in Chinese traditional medicine. In recent years, *A. auricula* was reported to have many biological activities, including anticoagulant activity (Yoon et al. 2003), antitumor activity (Mizuno et al. 1995), hypoglycemia (Takeuchi et al. 2004) and hypocholesterolemia (Cheung 1996). Up to now, considerable attention of *A. auricula* has been focused on dietary fiber and polysaccharide components, mainly extracted from its fruiting body. Previous studies have also shown that *A. auricula* polysaccharides have similar biological activities as *A. auricula*. Many nutritionists probed into *A. auricula* polysaccharide components (Aletor

1995), conformational change of *A. auricula* polysaccharides (Zhang et al. 1995a), molecular weights of *A. auricula* polysaccharides (Zhang et al. 1995b), improvement of production of *A. auricula* polysaccharides (Wu et al. 2006) and impact on lipid metabolism (Chen et al. 2008). Most recently, some health-promoting diet formulae have been developed using *A. auricula* as the major ingredient with combination of other nutritional herbal foods (Luo et al. 2009a, b). In fact, the fruiting bodies of *A. auricula* contain not only polysaccharides, but also abundant alkaloid, thiamin, riboflavin, ascorbic acid, vitamin D_2 and minerals. Acharya et al. (2004) reported that *A. auricula* ethanol extracts possessed the antioxidant and nitric oxide synthase activation properties. Methanol extract of *A. auricula* could inhibit lipid peroxidation and decrease liver damage in benzo(α)pyrene-treated mice (Chang et al. 1998). However, there is no further investigation on the effect of these substances of *A. auricula* on lipid metabolism.

Therefore, the present study was designed to investigate the hypocholesterolemic effects of *A. auricula* ethanol extract (AAE), we investigated effects of AAE on serum and hepatic lipid profiles, HMG-CoA reductase activity, antioxidant status in addition to fecal excretion of neutral cholesterol and bile acids in ICR mice. The results of this study will advance knowledge of cardiovascular benefits of *A. auricula* to better understand their application in the prevention/treatment of cardiovascular disease using functional foods and nutraceuticals.

Materials and methods

Plant material and preparation of AAE The sample of dried *A. auricula* fruiting bodies were cultured in the Daxinganling region, Heilongjiang province, China, and it was authenticated by Dr. B. Zhao, Department of Plant Sciences, China Agricultural University. The procedure for *Auricularia auricula* ethanol extracts was carried out as shown in Fig. 1. The sample of dried *A. auricula* fruiting bodies was ground and sieved through screens (40–60 mesh). Dried-powder of *A. auricula* contained (g/100 g dry weight): carbohydrates 82 (including 54 g total dietary fibres), crude protein 8, fat 1.5 and ash 8.5. This dried powder (200 g) was suspended in 6 l of ethanol (75%, v/v) and refluxed for 2 h at 80 °C. This procedure was repeated thrice and the suspension was filtered through Whatman No. 2 paper to remove the ethanol-insoluble materials such as fiber and polysaccharide compounds. This liquid was concentrated in a rotary evaporator under reduced pressure, and concentrated solution was extracted by pure ethanol for 24 h at 4 °C (proportion of concentrated solution to ethanol was 1:4). The solution of ethanol was filtered through Whatman No. 2 paper to remove deposition such as water-
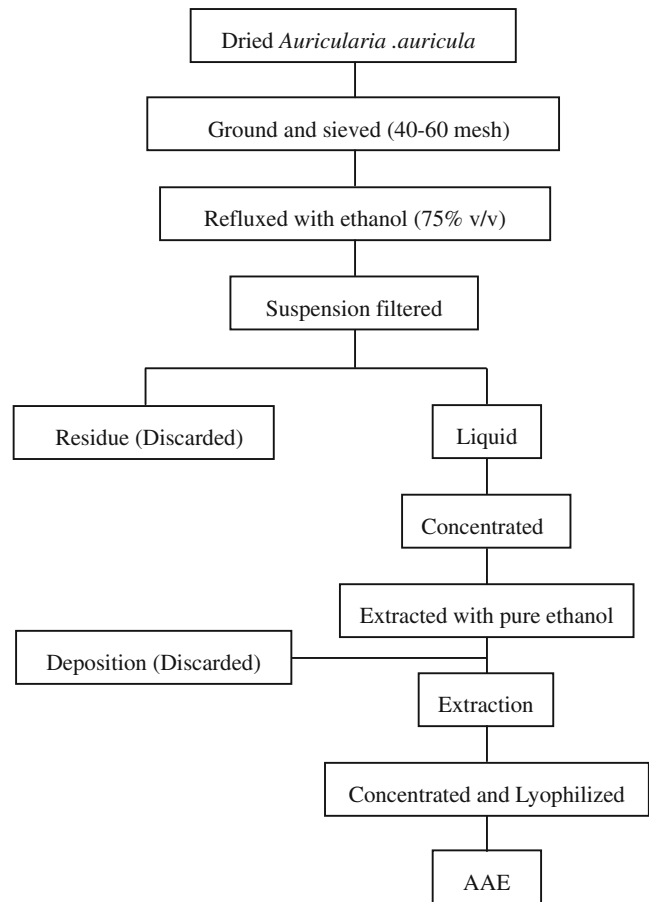

Fig. 1 Flow process chart about preparation of *Auricularia auricular* ethanol extract (AAE)

soluble fibre, polysaccharide, and protein components. This procedure was repeated four times and the ethanol extract was concentrated in a rotary evaporator under reduced pressure and then lyophilized. This preparation is named *A. auricula* ethanol extract (AAE). The total phenol content of AAE was determined using the Folin Ciocalteu method (Zhang et al. 2008). The total phenol content was 183.7 ± 15.6 mg/g, expressed as gallic acid equivalent.

Animals and treatment Four-week-old male ICR mice (Experimental Animal Center of Beijing, China) were acclimatized for 1 week before being randomly assigned into the experimental groups. The animals were housed in plastic cages (6 mice/cage) in a room with a 12 h light–dark cycle (8 am–8 pm), temperature of 23 ± 2 °C and a humidity of $55\pm5\%$. During the acclimatization period, each animal was raised on regular diet and water ad libitum. At five-week old, the ICR mice were randomly divided into three groups ($n=12$), the first group (EG) was experimental group fed cholesterol-enriched diet (CED) and orally administered 150 mg/kg/d b w of *A. auricula* ethanol extract; the second group (RD) was fed regular diet; the

third group (CEDC) was CED control group which were only fed CED; meanwhile RD and CEDC were fed along with oral administration of equal volume of distilled water. At the end of the experimental period (8th week), the fecal samples of each group were collected, lyophilized and stored under ambient temperature prior to use. After blooding completely, the animals were sacrificed by cervical dislocation. The livers were excised, weighed and stored at −80 °C until analysis. Composition of animal diets is listed in Table 1 (Experiment Animal Center of Beijing, China). Body weights and daily food intake were measured every 2 days and at every other week blood samples were collected for analysis. The experimental protocols were conducted according to the Guide for the Care and Use of Laboratory Animals established by China Agricultural University, Beijing, China.

Blood sampling On weeks 2, 4, 6 and 8 of the experimental period, blood samples were taken from the orbital venous plexus of ICR mice using a capillary tube without anesthesia, after a 16 h fast. The blood samples were placed in a plastic tube and incubated at 37 °C for 15 min, then centrifuged for 8 min at 4,000 rpm. The serum samples were stored at −20 °C until analysis.

Serum and hepatic lipids Serum lipid profile, including total cholesterol (TC), low density lipoprotein-cholesterol (LDL-C), high density lipoprotein-cholesterol (HDL-C) and triglyceride (TG) was assayed individually using the enzyme kits (Beijing Zhongsheng Hightech Bioengineering Company, Beijing, China) on Alcyon 300 auto-analyzer (Abbott Laboratories Ltd., US). The atherogenic index (AI) was calculated as: (TC−(HDL-C))/HDL-C.

Hepatic lipids were extracted from 300 mg of the liver of ICR mice with chloroform: methanol (2:1, v/v), according to method of Folch et al. (1957). After extraction, 1 ml extraction was dried under a nitrogen stream. The dried lipid residues were dissolved in 500 μl distilled water. Triton X-100 was added to the dissolved lipid solution to produce final concentration of 5 g/L. The hepatic TC and

TG were analyzed with the same enzyme kit as used for the serum analysis.

Hepatic antioxidant status determination The total antioxidant capacity (TAC), superoxide dismutase (SOD) activity and malondialdehyde (MDA) concentration were determined in liver homogenates with commercial kits obtained from Nanjing Jiancheng Bioengineering Institute (Nanjing, China). TAC was measured by the reaction of phenanthroline and Fe^{2+} using a spectrophotometer at 520 nm, a TAC unit is defined as the amount of antioxidants required to make absorbance increase by 0.01 in 1 ml serum. For SOD measurement, superoxide radicals were generated by NADPH and phenazine methosulfate PMS under non-acidic conditions, which reduce (NBT) nitroblue tetrazolium salt and form a blue coloured formazon, which can be measured at 560 nm. Free radical damage was determined by specially measuring MDA. MDA formed as an end product of lipid peroxidation was treated with thiobarbituric acid to generate a colored product that was measured at 532 nm.

Hepatic 3-hydroxy-3-methylglutary CoA (HMG-CoA) reductase activity The activity of HMG-CoA reductase in mice was determined by the method of Edwards et al. (1979). The isolated hepatic microsomes were mixed with an equal volume of 50% glycerol in phosphate buffer (0.1M sucrose, 0.05M KCl, 0.04M potassium phosphate, 0.03M potassium EDTA and 10 mM DTT; pH 7.2). The suspension was homogenized and incubated at 37 °C for 60 min to solubilize HMG-CoA reductase. The activity of the solubilized HMG-CoA reductase was obtained by assaying the enzyme in phosphate buffer (0.2M KCl, 0.16M potassium phosphate, 0.004M EDTA, and 0.01M DTT, pH 6.8) together with 0.2 mM NADPH and 0.1 mM RS-HMG-CoA and determined at 37 °C using a spectrophotometer (GBC scientific equipment Pty Ltd, Australia). The results were expressed as nmol mevalonate synthesized per min per mg protein.

Determination of fecal neutral cholesterol and bile acids The fecal neutral cholesterol was extracted and analyzed as per the method of Martensson (AOAC 2000) with slight modification. Fecal samples (0.2 g) were homogenized with 2 ml of a mixture of 10 mol/l NaOH and 96% ethanol (1:2, v/v) and kept at 70 °C for 45 min. The samples were allowed to cool to room temperature followed by centrifugation at $1,000 \times g$ for 5 min. The supernatant was removed and extracted with n-hexane thrice. The combined hexane phases were washed with 70% ethanol until neutral for neutral cholesterol analysis; and the lower phases were acidified with HCl and extracted with chloroform:methanol (2:1, v/v) for bile acids analysis. The cholesterol and

Table 1 Chemical composition of fed diets (g/100 g d.m.)

Ingredients	RD	CED
Corn starch	59	38
Casein	21.1	21.1
Cellulose	4.9	4.9
Fat	4.2	4.2
Minerals	10.8	10.8
Yolk powder	–	10
Cholesterol	–	1
Lard	–	10

RD Regular diet, *CED* Cholesterol enriched diet

coprostanol were analyzed using a Shimadzu GC-14 gas chromatography, Japan with a flame ionization detector and a 30 m×0.25 mm DM-5 capillary column maintained at 290 °C using nitrogen gas as carrier. The initial column temperature was 220 °C and was increased to 300 °C at a rate of 2 °C/min. The final temperature was held for 10 min. Column flow rate was 1.5 ml/min. Pure cholesterol and coprostanol were used as standards (Sigma-Aldrich, St. Louis, MO). Total neutral cholesterol was expressed as percent of total fecal cholesterol plus coprostanol. Fecal acidic steroids were determined by an enzyme kit (Total Bile Acid Kit, Wako, Japan).

Statistical analysis Data were presented as mean ± SD for 12 ICR mice per group. Statistical analysis was performed using one-way analysis of variance followed by Duncan's multiple range tests. P-values of less than 0.05 were considered to be statisticaly significant. Analysis was performed with SPSS 10.0.

Results and discussion

Body weight, food intake, and feeding efficiency As shown in Table 2, after the 8-week experimental period, the final body weights of the EG and CEDC (39±1.6 and 41±2.4 g, respectively) were not significantly different from that of RD (39±2.2 g), and the levels of food intake of the EG and CEDC (2.1±0.7 and 2.1±0.3 g, respectively) were not significantly different from that of RD (2.2±0.1). The levels of feeding efficiency of the EG and CEDC were (14.2±0.5 and 15.2±0.4%, respectively) also not significantly different from that of RD (13.6±0.6%).

Table 2 Changes in body weight and food intake of mice during 8-week experimental period

	RD	CEDC	EG
Initial body wt, g	20±1.2[a]	20±1.2[a]	20±1.2[a]
Final body wt, g	39±2.2[a]	41±2.4[a]	39±1.6[a]
Weight gain, g/day	0.3±0.1[a]	0.3±0.2[a]	0.3±0.1[a]
Food intake, g/day	2.2±0.1[a]	2.1±0.3[a]	2.1±0.7[a]
Feeding efficiency,%	13.6±0.6[a]	15.2±0.4[a]	14.2±0.5[a]

$n=12$ mice per group; Values in a row with different superscript letters differ significantly ($p<0.05$). Feeding efficiency: (daily weight gain/daily food intake)×100; RD: Regular diet control group; CEDC: Cholesterol-enriched diet control group; EG: Cholesterol-enriched diet + oral administration of *Auricularia auricula* ethanol extract experimental group

Table 3 Effect of *Auricularia auricula ethanol extract* on serum lipids in mice during 8-week experimental period

Experimental period	Serum lipids mmol/L		
	RD	CEDC	EG
TG			
2	0.21±0.03[a]	0.16±0.02[b]	0.16±0.02[b]
4	0.22±0.05[a]	0.15±0.04[b]	0.16±0.04[b]
6	0.21±0.06[a]	0.16±0.05[b]	0.18±0.07[c]
8	0.23±0.04[a]	0.17±0.03[b]	0.21±0.04[c]
TC			
2	0.70±0.03[a]	1.58±0.08[b]	1.34±0.02[c]
4	0.71±0.04[a]	1.62±0.07[b]	1.45±0.04[c]
6	0.69±0.08[a]	1.64±0.06[b]	1.54±0.03[c]
8	0.72±0.06[a]	1.68±0.09[b]	1.63±0.06[b]
HDL-C			
2	0.25±0.03[a]	0.37±0.04[b]	0.39±0.03[b]
4	0.26±0.04[a]	0.36±0.08[b]	0.41±0.08[c]
6	0.25±0.06[a]	0.36±0.04[b]	0.45±0.07[c]
8	0.26±0.04[a]	0.38±0.06[b]	0.48±0.09[c]
LDL-C			
2	0.03±0.01[a]	0.06±0.01[b]	0.05±0.01[b]
4	0.03±0.02[a]	0.06±0.02[b]	0.06±0.01[b]
6	0.02±0.01[a]	0.05±0.02[b]	0.05±0.02[b]
8	0.02±0.01[a]	0.05±0.01[b]	0.04±0.02[b]
AI			
2	1.80±0.04[a]	3.27±0.08[b]	2.44±0.05[c]
4	1.73±0.05[a]	3.50±0.10[b]	2.52±0.04[c]
6	1.76±0.04[a]	3.56±0.09[b]	2.42±0.06[c]
8	1.76±0.03[a]	3.42±0.11[b]	2.40±0.08[c]

$n=12$ mice/group; Values in a row with different superscript letters differ significantly ($p<0.05$). AI: atherosclerotic index; RD: CEDC, EG: See Table 2

Serum lipids and atherogenic index profiles As shown in Table 3, the serum concentrations of total cholesterol increased significantly ($p<0.05$) after feeding CED. The administration of AAE (150 mg/kg/d bw) significantly attenuated the increase of serum TC ($p<0.05$) during initial 6-week, however, its level was elevated gradually and close to the CEDC level at 8th week. Oral administration of AAE gradually increased the level of HDL-C since 4th week significantly ($p<0.05$), and the difference became greater over time. Nevertheless, the consumption of AAE had no significant effect on serum TG and had a tendency to regulate the levels of TG. In addition, the administration of AAE had no effect on serum levels of LDL-C in ICR mice fed CED. Additionally, the oral AAE administration decreased AI in EG group significantly ($p<0.05$), compared with that in CEDC group.
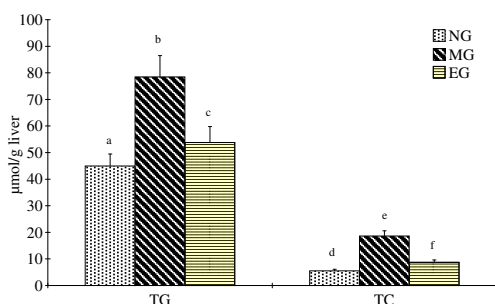
Fig. 2 Effect of *Auricularia auricular* ethanol extract on hepatic lipids profile in hyperlipidemia mice $n=12$ mice/group. Values not sharing the same letter within TG or TC are significantly different ($p<0.05$); RD; CEDC; EG: As in Table 2
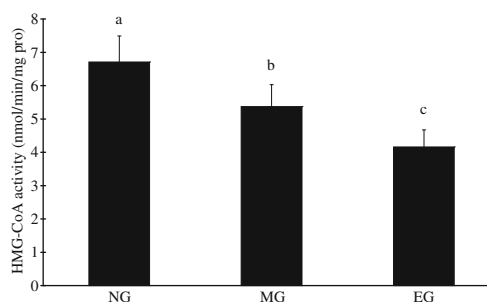

Fig. 3 Effect of *Auricularia auricular* ethanol extract on HMG-CoA activity on hyperlipidemia mice $n=12$ mice/group. Values not having the same letter are significantly different ($p<0.05$); RD; CEDC; As in Table 2

Hepatic lipid profiles Hepatic TC and TG levels were significantly ($p<0.05$) elevated by CED (Fig. 2). Expectedly, the consumption of AAE in EG group significantly ($p<0.05$) decreased TC and TG content in the whole liver by 113.80% and 45.86%, respectively.

Hepatic antioxidant status Cholesterol-enriched diet significantly reduced the levels of TAC, SOD and elevated MDA concentration ($p<0.05$) in CEDC (Table 4). Oral administration of AAE significantly improved the hepatic antioxidant status through elevating TAC and SOD levels with lowering MDA concentration ($p<0.05$).

Hepatic HMG-CoA reductase activity Hepatic HMG-CoA reductase activity was significantly decreased in CEDC compared with RD ($p<0.05$), oral administration of AAE significantly decreased HMG-CoA reductase activity compared with CEDC ($p<0.05$) (Fig. 3).

Fecal excretion of neutral cholesterol and bile acids The daily fecal dry mass showed no significant differences among groups (Table 5). The fecal excretion of neutral steroids and bile acids were increased in CEDC compared with RD. However, oral administration of AAE could

promote fecal bile acids output significantly by 4.5-fold, compared with CEDC ($p<0.05$) but no significant effect was observed on fecal excretion of neutral steroids.

Recent studies in animals and human subjects have shown a significant hypocholesterolemic effect of dietary *A. auricula*. In this study, we further determined the effects of *A. auricula* ethanol extract on the serum lipid profiles, hepatic lipid profiles, antioxidant status and fecal excretion of neutral cholesterol and bile acids in ICR mice fed with CED. The AAE made with this purification process contained more than 16% (g/g) *A. auricula* polyphenolic compounds, excluding other interfering components such as polysaccharides, water-soluble fibre and protein components.

Polyphenolic compounds have been reported to exert an inhibitory effect on food intake and growth by decreasing protein digestibility (Tebib et al. 1994). In this experiment, administration of AAE (150 mg/kg/d b. w.) to the CED-fed ICR mice showed no significant difference on levels of weight gain, food intake and feeding efficiency. This observation is in agreement with other investigators, who found no effect in food intake and weight gain of dietary fiber and polyphenols, such as catechin, tannic acid or condensed tannins (Bravo et al. 1994a, b).

High serum TC and LDL-C are the main risk factors in the pathogenesis of coronary heart disease. The results of the present investigation have shown that a CED intake led to an increase of cholesterol content both in serum and liver. Somewhat different from most studies (Wei et al. 2003; Zhao et al. 2006), serum HDL-C levels were increased significantly in CEDC compared with RD. This HDL-C elevating effect in hyperlipidemia ICR mice was also reported by Lee et al. (2006). It might be due to cholesterol-enriched diet causing stress reaction in ICR mice, which is associated with high levels of serum cholesterol, including LDL-C and HDL-C. Oral administration of AAE (150 mg/kg/d bw) had a distinct cholesterol-

Table 4 Effect of *Auricularia auricula* ethanol extract on hepatic antioxidant status of CED-fed mice after 8 weeks

	TAC, U/mg pro	SOD, U/mg pro	MDA, nmol/mg pro
RD	1.91 ± 0.26^a	175.09 ± 10.53^a	0.75 ± 0.08^a
CEDC	1.03 ± 0.17^b	91.22 ± 8.27^b	1.54 ± 0.09^b
EG	1.75 ± 1.62^c	138.32 ± 12.27^c	0.88 ± 0.06^a

$n=12$ mice/group; Values in a column with different superscript letters differ significantly ($p<0.05$). RD: CEDC: EG: As in Table 2

Table 5 Effect of *Auricularia auricula* ethanol extract on fecal neutral sterol and total bile acids concentrations of CED-fed mice

$n=12$ mice/group; Values in a row with different superscript letters differ significantly ($p<0.05$) RD: CEDC: EG: As in Table 2

Neutral steroids	RD	CEDC	EG
Cholesterol, mg/d	0.20 ± 0.03^a	0.97 ± 0.05^b	0.95 ± 0.04^b
Coprostanol, mg/d	0.52 ± 0.07^a	1.30 ± 0.25^b	1.35 ± 0.11^b
Total, mg/d	0.72 ± 0.06^a	2.27 ± 0.26^b	2.30 ± 0.21^b
Total bile acids, mg/d	0.48 ± 0.03^a	1.51 ± 0.10^b	6.97 ± 0.12^c
Dry feces weight (g/mouse/d)	0.45 ± 0.03^b	0.43 ± 0.02^b	0.46 ± 0.01^b

lowering effect by decreasing the levels of serum TC, hepatic TC and TG, accordingly reduced the risk of CHD. Similar cholesterol-lowering results had been previously reported in many polyphenolic compounds from different plant foods (Auger et al. 2005; Suzuki et al. 2005). AI has direct correlation with the cardiovascular disease. AAE was able to decrease AI significantly, contributing to the lower risk in pathology of hyperlipidemia and atherosclerosis.

The rise of cholesterol in liver and serum may be due to increased uptake of exogenous cholesterol and subsequent deposition and decreased cholesterol catabolism of cholesterol to bile acids (Jaganathan et al. 1974). To elucidate the underlying mechanism of the cholesterol-lowering action of AAE, we investigated the hepatic HMG-CoA reductase activity and fecal cholesterol and bile acids excretion. HMG-CoA reductase is the rate-limiting enzyme in the cholesterol biosynthetic pathway. The inhibition of HMG-CoA reductase decreases cholesterol synthesis and is very effective in lowering plasma cholesterol (Goldstein and Brown 1990). High cholesterol diet itself inhibited HMG-CoA reductase activity in our study for the feedback regulation of cholesterol, which was also illustrated by Hayashi et al. (2004). Previous studies demonstrated that polyphenols extract from virgin olive oils (Benkhalti et al. 2002) and naringenin (Lee et al. 2003), a kind of phenolic compound, decreased liver microsome HMG-CoA reductase activity. Similarly, AAE significantly reduced the activity of HMG-CoA reductase compared with CEDC, suggesting the hypocholesterolemic effects of AAE phenolic compounds through suppressing the rate of cholesterol synthesis. Cholesterol can be excreted into the bile directly or after conversion to bile acids. Bile acid synthesis occurs exclusively in the liver, and cholesterol 7α-hydroxylase is the first rate-limiting enzyme in this pathway. EG significantly increased the fecal excretion of bile acids compared with CEDC; we conjectured the hypocholesterolemic effect of AAE phenolic compounds might be due to the accelerated rate of catabolism of cholesterol to bile acids and also be related to enhance the activity of cholesterol 7α-hydroxylase, which would be another mechanism of the hypocholesterolemic effect of AAE. Del Bas et al. (2005) also reported that polyphenols in red wine induce cholesterol 7α-hydroxylase expression and increase of cholesterol elimination via bile acids. The present results are consistent with above research. Further, *A. auricula* was previously reported with the ability to increase the fecal excretion of neutral steroids and bile acids in rats (Cheung 1996). The author considered that β-glucans and glucuronoxylomannan of *A. auricula* interfered with the absorption of cholesterol from the digestive tract of the animal, so increased the fecal neutral steroids output, but he doubted that some other components in *A. auricula* having the responsibility for increasing the fecal bile acids output. In the present study, we found AAE had no obvious effect on fecal excretion of neutral cholesterol but significantly increased fecal excretion of bile acids (Table 5). Therefore, it was assumed that polyphenolic compounds were able to increase fecal excretion of bile acids, but the active compounds and the underlying mechanism require further analysis.

Furthermore, it is well reported that the cholesterol-enriched diet would appear to induce free radical production, followed by oxidative stress and hypercholesterolemia (Tarladgis et al. 1964), such as decreasing the activities of catalase and SOD and thereby elevating the lipid peroxide contents, resulting in the production of toxic intermediates. In the present study, administration of AAE (150 mg/kg/d bw) could significantly lower the levels of lipid peroxides, MDA, and enhanced the activities of the hepatic SOD and TAC (Table 4). Further research is in progress, aimed at characterizing the polyphenolic compounds and isolation of the active compounds.

Conclusion

Ethanol extract of *A. auricula* is rich in polyphenolic compounds and thus possesses potent hypocholesterolemic effects. Through the in vivo study, it was found that consumption of AAE could significantly improve the antioxidant status and lipids profile, inhibit cholesterol synthesis in liver and elevate fecal bile acids excretion. However, the study on the polyphenolic compounds in *A. auricula* is very limited. Further study on AAE is being carried out to elucidate the specific compounds contributing to hypolipidemic activity and to explore other nutritional properties of *A. auricula*, such as anti-diabetes and anti-atherosclerosis.

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