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Protective effect of sulfated *Achyranthes bidentata* polysaccharides on streptozotocin-induced oxidative stress in rats

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ABSTRACT

Sulfated *Achyranthes bidentata* polysaccharide (SAbP) is derived from traditional Chinese herbal medicine *A. bidentata* polysaccharide (AbP) by chemical modification. The present study was designed to determine the possible protective effect of SAbP against oxidative damage in streptozotocin (STZ)-treated diabetic rats. Results showed SAbP significantly reduced blood glucose level and malondialdehyde (MDA) concentration in diabetic rats. The activities of GPx and SOD were increased in diabetic rats. But the effects of AbP on blood glucose, MDA concentration and antioxidant enzymes activities are not obvious as SAbP. Therefore, we postulate that SAbP has a better protective effect in diabetes than AbP and this may be attributed to its antioxidative potential.

1. Introduction

Diabetes is the most significant chronic disease and cause of death in modern society (Stratmann, Menart, & Tschoepe, 2007). It is a complicated metabolic disorder characterized by high blood glucose level due to inability of the body cells to utilize glucose properly (Ugochukwu & Babady, 2002). Although several aspects of diabetes can be controlled by insulin treatment and other chemical therapies, numerous complications are common incidents of the disease. Hyperglycemia represents the main cause for these complications of diabetes because elevated glucose concentration directly injures cells and induces lipid peroxidation (Davi, Falco, & Patrono, 2005). Studies have shown that tissue antioxidant status may play an important role in the etiology of diabetes (Al-Azzawie & Saeed, 2006; Valko et al., 2007) and oxidative stress may be a common pathway linking diverse mechanisms for the complications in diabetes (Baynes, 1991). Oxidative stress may constitute a focal point for multiple therapeutic interventions, and for therapeutic synergy.

Renewed attention in recent decades to alternative medicines and natural therapies has stimulated a new wave of research interest in traditional Chinese herbal medicine. Many traditional plant treatments for diabetes mellitus are used throughout the world. But few of them have received scientific evaluation.

Achyranthes bidentata polysaccharide (AbP) is an active component isolated from the root of the Chinese medicinal herb *A. bidentata* Blume. Previous study showed that AbP could strengthen the immune system, restrain tumor metastasis, increase leucocytes number, protect liver cells and activate thoracic cavity macrophages of human (Li & Li, 1997; Lu, Yu, & Jin, 1999).

Chemical modification of polysaccharides provided an opportunity to obtain new pharmacological agents with possible therapeutic uses. Polysaccharides have been chemically modified in various ways to change their physical or biological properties, thus allowing a broader range of applications (Liu & Sun, 2005; Xing et al., 2005).

Sulfated polysaccharide, including the natural or chemically synthesized, is polysaccharide derivative from its hydroxyl by sulfated. In the last decades, the biological properties of polysaccharides and their chemical derivatives, especially sulfated derivatives, have attracted much more attention (Han, Yao, Yang, Liu, & Gao, 2005; Lee, Bae, & Pyo, 2003; Xing et al., 2005). Sulfated polysaccharides have an important bioactivities including antivirus (Urbinati, Bugatti, & Oreste, 2004), antioxidant (Yang, Gao, Han, & Tan, 2005), antitumor (Peng, Zhang, Zeng, & Kennedy, 2005), and anticoagulant activities (Han et al., 2005). Therefore, sulfated modification may be used as a method to improve the biological activities of some polysaccharides and obtain more effective polysaccharides derivatives. Tian, Li, Song, Zheng, and Li (1995) had reported the antivirus activity

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of SAbP. However, there is no further information about property of SAbP.

Present here, the study was to investigate whether management with SAbP has any protective effect on lipid peroxidation and activities of antioxidants in STZ-induced diabetic rats.

2. Materials and methods

2.1. Chemicals

Achyranthes bidentata polysaccharide (AbP) was presented by professor Tian Gen-Yuan, Shanghai Institute of Organic Chemistry. Streptozotocin was obtained from Sigma chemicals (USA). The assay kits for glutathione peroxidase (GPx), superoxide dismutase (SOD) and malondialdehyde (MDA) were purchased from Jiancheng Biologic Project Company, Nanjing, Jiangsu Province, China. Xiaoke pill was procured from the Zhongyi Medicinal Limited Company, Guangzhou, Guangdong Province, China. All the other chemicals were analytical grade.

2.2. Animals

Thirty male Sprague–Dawley (S–D) rats (weight: 200–250 g) were used for this study. They were feeded under controlled environmental conditions of temperature (22 ± 2 °C) and a 12 h light/dark cycle, and maintained on (unless otherwise stated) standard food pellets and tap water ad libitum.

All animal handling procedures were performed in strict accordance with the PR China legislation the use and care of laboratory animals, with the guidelines established by Institute for Experimental Animals of Wenzhou Medical College, and were approved by the College Committee for animal experiments.

2.3. Experimental design

The animals were divided into two groups: a nondiabetic control ($n = 6$) and a diabetic group ($n = 24$). STZ in fresh 0.1 mol/L citrate buffer (pH 4.5) was administered intraperitoneally (i.p.) at a single dose of 55 mg/kg to diabetic group. The control rats were only injected with the citrate buffer. Three days after STZ treatment, development of diabetes was confirmed by measuring blood glucose level. The rats with blood glucose level 16.8 mmol/L or higher were considered to be diabetic and were used in the experiment. The diabetic rats ($n = 24$) were randomly divided into four groups: groups II–V.

Group I ($n = 6$): normal control rats, animals were allowed to free access to a normal diet for 30 days.

Group II ($n = 6$): diabetic control rats, the diabetic animals were allowed to free access to a normal diet for 30 days.

Group III ($n = 6$): Xiaoke pill-treated diabetic rats, the diabetic animals were put on a normal diet and treated with Xiaoke pill (1.5 g/kg/day, p.o.) for 30 days.

Group IV ($n = 6$): SAbP-treated diabetic rats, the diabetic animals were put on a normal diet and treated with SAbP (100 mg/kg/day, p.o.) for 30 days.

Group V ($n = 6$): AbP-treated diabetic rats, the diabetic animals were put on a normal diet and treated with AbP (100 mg/kg/day, p.o.) for 30 days.

On the last day of experiment, blood samples were collected for biochemical estimations. Later the animals were sacrificed. Then the liver and kidney were removed, cleaned and washed in ice-cold normal saline for biochemical study.

The rationale for the selection of the doses was based on the data published by previous workers, wherein, doses of 20–1000 mg/kg have been used (Aguilara et al., 1998).

Body weights of mice were recorded initially (once every week during the experiment).

On the last day of experiment, the animals were deprived of food overnight and sacrificed by cervical dislocation. Blood was collected in sterilized tubes without the anticoagulant. Serum was immediately separated by centrifugation at 3000 rpm at room temperature for 10 min. Samples were stored at −70 °C until assayed.

The kidney and liver were removed quickly and homogenized in nine volumes of ice-cold 0.9% saline solution using a motor-driven Teflon glass homogenizer to yield a 10% (w/v) homogenate. The homogenate was then centrifuged for 10 min at 3500 rpm at 4 °C. The supernatant obtained was used for assays of GPx, SOD and MDA.

2.4. Preparation of sulfated Achyranthes bidentata polysaccharide

AbP was isolated from the roots of *A. bidentata*, sulfated AbP was prepared using the chlorosulfonic acid–pyridine method described by Tian-Gengyuan (Tian et al., 1995). The product was purified by DEAE–Sephadex A-50 and Sephadex G-50. The chemical homogeneity of SAbP was examined by high-performance liquid chromatography (HPLC) and capillary electrophoresis (CE). The structure of SAbP was deduced by methylation analysis and ^{13}C NMR spectroscopy. The sulfur content of the products was calculated by elemental analysis.

2.5. Analytical method

Blood glucose levels were determined by the glucose oxidase method (Trinder, 1969). The serum total cholesterol (TC), HDL-cholesterol (HDLC) and triglyceride concentrations were measured enzymatically using a completely automatic biochemistry analyzer. The serum concentration of LDL was estimated using Friedewald's method (Friedewald, Levy, & Fredrickson, 1972). Lipid peroxidation was determined by quantifying MDA concentrations, which was spectrophotometrically measured by the absorbance of a red-colored product with thiobarbituric acid (Niehaus & Samuelsson, 1968; Yagi, 1994).

SOD and GSH-Px activities were determined using chemical kits. Briefly, the determination of SOD activity was based on the production of O^{2-} anions by the xanthine/xanthine oxidase system (Fairbanks & Klee, 1994). The amount of SOD that inhibits 50% the rate of reduction under the specified conditions was regarded as one enzyme unit.

Reduced glutathione (GSH) was catalyzed by the glutathione peroxidase (GPx) in the presence of hydrogen peroxide. GSH-Px activity was estimated by the analysis of GSH in the enzymatic reaction (Sedlak & Lindsay, 1968). One unit of enzyme activity of the enzymes unit represents a decrease in GSH concentration of 1 μmol/L per minute after subtraction of non-enzymic mode (Rotruck, Pope, Ganther, & Swanson, 1973).

2.6. Statistical analysis of the data

Results were expressed as means ± standard deviations (*SD*). Data were analyzed using Analysis of Variance (ANOVA) and *T*-test to the statistical significance ($P < .05$).

3. Results

3.1. Characterization of AbP and SAbP

AbP is a fructan that is comprised of a β-D-fructofuranosyl backbone having residues linked (2 → 1) and (2 → 6) with branches and

Table 1
Effect of SAbP-treated on diabetic rats' body weight[a]

Group	Days				
	−7	0	10	20	30
Control	238.5 ± 7.2	267.5 ± 10.1	302.0 ± 26.2	341.5 ± 19.1	381.5 ± 20.5
DM	231.3 ± 9.2	250.8 ± 7.5*	267.2 ± 12.4**	259.3 ± 14.0**	254.3 ± 10.5**
XK	229.6 ± 10.5	248.2 ± 12.8*	272.0 ± 13.4**	288.6 ± 14.0$^{**,\Delta\Delta}$	310.6 ± 20.5$^{**,\Delta\Delta}$
S-AbP	227.5 ± 8.3	249.2 ± 7.3*	273.8 ± 10.5**	295.5 ± 15.4$^{**,\Delta\Delta,\square\square}$	327.5 ± 18.0$^{**,\Delta\Delta,\square\square}$
AbP	232.7 ± 10.7	252.5 ± 11.5*	268.8 ± 15.2**	265.8 ± 18.0**	273.0 ± 16.7**

$^{*}P < .05$, $^{**}P < .01$, compared with normal control (group I).
$^{\Delta}P < .05$, $^{\Delta\Delta}P < .01$, compared with diabetic control (group II).
$^{\square\square}P < .01$, compared with the corresponding value for AbP-treated control (group V).
[a] Data represent mean ± *SD* ($n = 6$ for each group).

α-D-glucopyranose residue on the nonreducing end of the fructan chain. Each branch is terminated by a β-D-fructofuranose residue. Sulfated AbP was purified by DEAE–Sephadex A-50 and Sephadex G-50 to give SAbP. It was concluded by methylation and ^{13}C NMR spectroscopy that the hydroxyl groups of AbP were mainly substituted on 6-position of the glucopyranose and fructofuranose, 1-position of the fructofuranose and parts of 4-position of fructofuranose. The sulfur content of SAbP was found to be 18.9% and the degree of substitution was 2.4.

3.2. Body weight gain of rats

As shown in Table 1, there was no obvious difference in initial body weights between different groups. After 30 days of experiment, the diabetic control rats gained less body weight than normal control rats ($P < .01$). The body weight gains were significantly increased ($P < .01$) both in SAbP-treated animals and ($P < .01$) Xiaoke pill-treated ones compared with diabetic control rats. No significant difference was observed in body weight gains between AbP-treated rats and diabetic control ones.

3.3. Change of blood glucose level in diabetic rats after SAbP-treated

As shown in Table 2, the diabetic groups (groups II–V) rats significantly ($P < .01$) increases the blood glucose levels compared with the normal control group. AbP-treated group reduced the blood glucose level compared with diabetic control rats. In both SAbP-treated and Xiaoke pill-treated groups of animals, the blood glucose levels were significantly decreased ($P < .01$) after 30 days of experiment, compared with diabetic control rats. Moreover, the blood glucose level of post-treatment was significantly decreased compared with that of pre-treatment in SAbP-treated rats. Furthermore, the blood glucose level in SAbP-treated rats was lower than that of Xiaoke pill-treated and AbP-treated ones.

Table 2
Effect of SAbP-treated on blood glucose level in diabetic rats[a]

Group	Pre-treatment	Post-treatment
I	6.85 ± 0.51	6.98 ± 0.46
II	22.80 ± 2.69**	28.61 ± 0.74**
III	23.93 ± 1.59**	20.01 ± 2.54$^{**,\Delta\Delta}$
IV	23.60 ± 2.40**	16.36 ± 1.92$^{**,\Delta\Delta,\#,\square\square}$
V	21.67 ± 2.77**	26.36 ± 2.22$^{**,\Delta}$

$^{**}P < .01$, compared with normal control (group I).
$^{\Delta}P < .05$, $^{\Delta\Delta}P < .01$, compared with diabetic control (group II).
$^{\#}P < .05$, compared with the corresponding value for Xiaoke pill-treated control (group III).
$^{\square\square}P < .01$, compared with the corresponding value for AbP-treated control (group V).
[a] Data represent mean ± *SD* ($n = 6$ for each group).

3.4. Change of blood lipids level in diabetic rats after SAbP-treated

In the present model, as shown in Table 3, diabetic control group of rats significantly ($P < .01$) increased the total cholesterol, triglyceride, and LDL-cholesterol level in blood and decreased ($P < .01$) the HDL-cholesterol level compared with the normal control group after 30 days of experiment. Both SAbP and Xiaoke pill significantly reversed the values of TG, TC, LDL-c, HDL-c closely to normal level. Moreover, based on the numerical values of the reversed levels of TG and HDL-c, SAbP appeared to be more efficacious than Xiaoke pill ($P < .05$, $P < .01$). However, AbP was only reversed the HDL-c levels to normal.

3.5. Change of MDA level in diabetic rats after SAbP-treated

Table 4 shows the level of malondialdehyde (MDA), a secondary product of lipid peroxidation in the serum, liver and kidney tissue homogenate. The MDA levels of the experiment groups were significantly ($P < .01$) increased. But AbP-treated did not induce any significant change in the MDA level in blood, liver and kidney, whereas SAbP-treated resulted a significant ($P < .01$) decreasing the MDA level in blood and tissue of diabetic rats, compared with diabetic control rats. The effects of SAbP on reducing MDA level in liver and kidney were better than XK pill ($P < .05$).

3.6. Change of GPx content in diabetic rats after SAbP-treated

As shown in Table 4, the activity of GPx in diabetic control rats was significantly ($P < .01$) decreased in blood, liver and kidney compared with the normal control rats (group I). Both SAbP-treated and Xiaoke pill-treated diabetic rats showed significant increase ($P < .01$) the activities of GPx in blood, liver and kidney tissue compared with diabetic control rats, and SAbP-treated showed better effect in restoring the activity of GPx in kidney than Xiaoke pill. AbP-treated in diabetic rats also produced a significant ($P < .01$, $P < .05$) decrease the activity of GPx in blood and liver compared to diabetic control rats, however, the effect was not so significantly ($P < .01$) as SAbP-treated.

3.7. Change of SOD level in diabetic rats after SAbP-treated

As shown in Table 4, there was a significant ($P < .01$) reduction of the activity of SOD in diabetic control rats compared with the normal control rats (group I). Both SAbP-treated and Xiaoke pill-treated significantly ($P < .01$ or $P < .05$) increased the activities of SOD in blood, liver and kidney of diabetic rats compared to diabetic control rats, however no significant difference was observed between these two groups (group III and group IV). The liver SOD level in diabetic rats significantly increased ($P < .01$) with treatment of AbP, but no significant effect was observed the activities of SOD in blood and kidney, compared with diabetic control rats.

Table 3
Effect of SAbP-treated on blood fat level in diabetic rats[a]

Group	TG	TC	HDL-c	LDL-c
I	1.17 ± 0.21	2.2 ± 0.14	0.73 ± 0.02	0.95 ± 0.10
II	2.41 ± 0.35**	3.26 ± 0.18**	0.56 ± 0.12*	1.61 ± 0.34**
III	1.45 ± 0.15$^{\Delta}$	2.43 ± 0.13$^{\Delta\Delta}$	0.71 ± 0.05$^{\Delta\Delta}$	1.03 ± 0.12$^{\Delta\Delta}$
IV	1.08 ± 0.11$^{\Delta\Delta,\#,\square\square}$	2.32 ± 0.13$^{\Delta\Delta,\square\square}$	0.83 ± 0.07$^{*,\Delta\Delta,\#\#,\square}$	1.20 ± 0.16$^{\Delta\Delta}$
V	2.24 ± 0.34**	3.16 ± 0.35**	0.70 ± 0.03$^{\Delta}$	1.44 ± 0.18**

$^{**}P < .01$, compared with normal control (group I).
$^{\Delta}P < .05$, $^{\Delta\Delta}P < .01$, compared with diabetic control (group II).
$^{\#}P < .05$, compared with the corresponding value for Xiaoke pill-treated control (group III).
$^{\square\square}P < .01$, compared with the corresponding value for AbP-treated control (group V).
[a] Data represent mean ± *SD* ($n = 6$ for each group).

4. Discussion

The present study investigated the effects of SAbP on oxidative of STZ-induced diabetic rats. Diabetics and experimental animal models exhibit high oxidative stress due to persistent and chronic hyperglycemia, which depletes the activity of antioxidative defense system resulting in elevated levels of oxygen free radicals (Hong et al., 2004; Ihara et al., 1999). There was reported that reactive oxygen species (ROS) and free radicals were increased in both type of diabetes (Johansen, Harris, Rychly, & Ergul, 2005). Moreover, the onset of diabetes has been confirmed closely associated with oxidative stress in both clinical and experimental diabetes mellitus (Rosen et al., 2001). Attacking of high levels of free radicals and simultaneously decreasing expression of antioxidant enzymes, which may enhance membranes susceptibility to lipid peroxidation and lead to pancreatic β-cell dysfunction as well as other cellular organelles damage (Baynes, 1991; Lenzen, Drinkgern, & Tiedge, 1996; Maritim, Sanders, & Watkins, 2003).

STZ, an antibiotic produced by *Streptomyces achromogenes,* has been widely used for inducing diabetes in the experimental animals through its toxic effects on pancreatic β-cells (Kim et al., 2003; Rakieten, Rakieten, & Nadkarni, 1963; Yamagishi, Nakayama, Wakatsuki, & Hatayama, 2001). Although the mechanism of β-cell cytotoxic action of STZ and the induced hyperglycemia is not fully understood, several lines of evidences indicate that STZ can stimulate free radicals generation, which may be one of the most essential causes of β-cell damage and diabetogenic effect of STZ (Ohkuwa, Sato, & Naoi, 1995; Szkudelski, 2001). Moreover, islet cells, because of relatively low activity of antioxidant enzymes such as superoxide dismutase, catalase and glutathione peroxidase are much more vulnerable to free radicals attack (Tiedge, Lortz, Drinkgern, & Lenzen, 1997). As a result, most islet cells are impacted to death or dysfunction after streptozotocin injection. The lack of compensation of insulin resistance by augmented insulin secretion results in rise in blood glucose.

In our study, we observed a significant increase in the concentration of blood glucose and decrease in body weight, as well as elevation in urinary production, hydroposia and food intake in streptozotocin-induced diabetic rats. Diabetes mellitus is also strictly related to other metabolic abnormalities, among which the most important ones are lipid abnormalities, characterized mainly by high triglyceride, high cholesterol levels and low HDL-cholesterol, which were also observed in our streptozotocin-induced diabetic animals. Our study showed that SAbP not only lowered the level of blood glucose, but also improved the body weight loss due to diabetes and lipid metabolism by causing a significant decrease in serum triglycerides, total cholesterol, LDL-cholesterol and rise in HDL-cholesterol levels. And the effects to some extent are better than AbP and XiaoKe pill, a traditional therapeutic drug for diabetes mellitus. The results may be associated with its potential protection effect to pancreatic β-cells, which will be demonstrated by later explanation. Therefore, glycometabolism and lipid metabolism may be improved due to increase in insulin secretion along with renovation of β-cells.

Since there is a strong correlation between oxidative stress and diabetes occurrence as mentioned above, supplementation of an antioxidant could be helpful to treatments for diabetes by gearing up the detoxification machinery. In recent years natural plants with antioxidative property have been the center of focus. It is believed that these plants can prevent or protect tissues against damaging effect of free radicals (Osawa & Kato, 2005). Many chemicals from these plants such as polysaccharides have shown anti-diabetic effect in experimental research (He, Li, Guo, Lin, & Lin, 2006; Hong, Xun, & Wutong, 2007; Li, 2007; Ou et al., 2007). However, some chemical groups may play an important role in the activity of polysaccharides. Sulfate is one of these, which has been proved to be closely related to antioxidant activity (Hu, Gen, Zhang, & Jiang, 2001; Qi et al., 2005; Rupérez, Ahrazem, & Leal, 2002; Zhang et al., 2004). Similar results were observed in our study, our data indicated a significant increase in MDA concentration and reduction in antioxidant enzyme activities of SOD and GPx in the serum, liver and kidney tissue of STZ-induced diabetic rats. SAbP tended to lower the MDA concentration and restored the activities of SOD and GPx to near normal levels. Moreover, it showed to some extent better effect than XK pill. However, in spite of partly improving antioxidant enzyme activities in blood and liver of diabetic rats, AbP did not change lipid peroxidation products such as MDA concentration. The results above corroborate that AbP obtains better antioxidative effect by sulfated modification. Intro-

Table 4
Effects in the concentration of malondialdehyde (MDA) and the activities of glutathione peroxidase (GPx), superoxide dismutase (SOD) in pancreas of control and experimental animals[a]

Tissue and enzyme	I	II	III	IV	V
Blood SOD (U/ml)	267.85 ± 5.41	206.16 ± 5.89**	236.59 ± 19.93$^{**,\Delta\Delta}$	232.55 ± 12.88$^{**,\Delta,\square}$	211.23 ± 9.65**
GPx (U/ml)	868.38 ± 33.36	528.21 ± 50.14**	825.64 ± 59.80$^{\Delta\Delta}$	871.80 ± 26.49$^{\Delta\Delta,\square\square}$	671.80 ± 73.76$^{**,\square\square}$
MDA (nmol/ml)	7.72 ± 1.24	12.45 ± 1.39**	9.15 ± 1.17$^{\Delta\Delta}$	8.63 ± 1.62$^{\Delta\Delta,\square\square}$	11.79 ± 1.42**
Liver SOD (U/mg protein)	299.24 ± 23.58	230.77 ± 30.29**	305.27 ± 16.92$^{\Delta\Delta}$	285.79 ± 14.74$^{\Delta\Delta}$	277.36 ± 11.53$^{\square\square}$
GPx(U/mg protein)	545.24 ± 33.76	391.10 ± 23.59**	501.94 ± 36.69$^{\Delta\Delta}$	516.16 ± 27.75$^{\Delta\Delta,\square\square}$	433.71 ± 18.96$^{**,\square}$
MDA(nmol/mg protein)	1.33 ± 0.23	2.39 ± 0.30**	1.97 ± 0.25*	1.33 ± 0.27$^{\Delta\Delta,\square\square,\#}$	2.36 ± 0.44**
Kidney SOD (U/mg protein)	317.25 ± 23. 32	251.79 ± 15.03**	290.73 ± 26.53$^{\Delta}$	302.89 ± 20.97$^{\Delta\Delta}$	267.71 ± 25.17*
GPx (U/mg protein)	317.69 ± 17. 17	201.03 ± 11.15**	288.79 ± 9.96$^{**,\Delta\Delta}$	305.29 ± 7.21$^{\Delta\Delta,\#,\square\square}$	211.93 ± 12.50**
MDA (nmol/mg protein)	2.00 ± 0.20	3.24 ± 0.32**	2.40 ± 0.20$^{*,\Delta\Delta}$	2.04 ± 0.1 7$^{\#,\square\square}$	2.88 ± 0.23**

$^{**}P < .01$, compared with normal control (group I).
$^{\Delta}P < .05$, $^{\Delta\Delta}P < .01$, compared with diabetic control (group II).
$^{\#}P < .05$, compared with the corresponding value for Xiaoke pill-treated control (group III).
$^{\square\square}P < .01$, compared with the corresponding value for AbP-treated control (group IV).
[a] Data represent mean ± *SD* ($n = 6$ for each group).

duction of sulfate groups to polysaccharide may change its physicochemical characteristics and chain conformation, and the forces among sulfate groups induce a relatively chain expanded and more hydroxy groups exposed. As a result, the ability of AbP to scavenge free radicles is elevated. This may be one of the possible mechanisms of the antioxidative effect from SAbP.

Lipid peroxidation products such as MDA are generated under high levels of un-scavenged free radicals and may bring about protein damage and inactivation membrane bound enzymes, so they play an important role in pancreatic damage associated with diabetes.

SOD and GSH-Px are two important enzymatic antioxidant defense mechanisms by decomposing superoxide peroxide, blocking lipid peroxidation and being involved in the cellular defense mechanisms, thus protecting the tissue against oxidative damage. The enhanced activity of SOD and GSH-Px in the present study may contribute to the potent effect of SAbP on scavenging oxygen free radicals. Pancreatic β-cells may be protected from oxidative damage induced by STZ according to this reciprocal mechanism. Therefore, SAbP has a direct and indirect preventive and protective effect in diabetes by decreasing oxidative stress and preservation of pancreatic β-cell integrity, and this may be one of the reasons that SAbP improves glycometabolism.

In conclusion, SAbP exert protective effects in experimental diabetes, possibly by reducing oxidative stress and hence protects organism from oxidative damage and dyslipidemia. Furthermore, the effects are stronger to some extent than AbP due to introduction of sulfate groups. However, further studies are necessary to confirm these effects.

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