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Pharmacological activity of sanchi ginseng (*Panax notoginseng*)

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Abstract

The pharmacological activity and constituents of the sanchi ginseng *Panax notoginseng* have been reviewed. The bulk of pharmacological findings have been based on the saponins or steryl glycosides, although polysaccharides with immunopotentiating activity, proteins with antifungal, ribonuclease and xylanase activity, and a triacylglycerol (trilinolein) with antioxidant activity have been reported. Protective actions against cerebral ischaemia, beneficial effects on the cardiovascular system, and haemostatic, antioxidant, hypolipidaemic, hepatoprotective, renoprotective and estrogen-like activities have been described. Various methods for authentication of *P. notoginseng* are available.

Introduction

Panax notoginseng is a Chinese medicinal herb used for promoting blood circulation. It was first used by minority ethnic groups in Southwest China and became more widely accepted during the Ming Dynasty (Zhang & Fang 2003). Sanchi ginseng (*P. notoginseng*), American ginseng (*P. quinquefolius*), and Chinese and Korean ginseng (*P. ginseng*) are highly prized drugs, with *P. ginseng* being the most expensive. *P. quinquefolius* and *P. ginseng* are better known to Western investigators than the traditional Chinese medicine *P. notoginseng*. For instance, ginseng extract (Seo et al 2005) and ginsenoside Rd from ginseng (Yokozawa et al 2004) have been shown to inhibit expression of inducible nitric oxide synthase and nitric oxide production in lipopolysaccharide-activated murine macrophages and attenuate ageing-associated oxidative damage in senescence-accelerated mice, respectively. Nevertheless, *P. notoginseng* has a number of very important activities including antihypertensive, antithrombotic, anti-atherosclerotic and neuroprotective actions. It is thus timely to summarize research findings on this important Chinese medicinal material. In fact, sanchi ginseng capsules are easily available in the Orient.

Chemical components

Sapogenins have been isolated from leaves (Lei 1984; Hu & Yang 1988), flower buds (Wei 1984b), fruit pedicels (Wei & Cao 1992), and rootlets (Wei 1984a; Wei et al 1985; Chen 1987). The total saponin content in Radix notogeinseng was similar to that in stems and leaves of *P. japonicum* (Chi et al 1992). Flavonoids have been isolated from the leaves (Wei & Wang 1987). Like *P. ginseng*, *P. quinquefolius* and *P. vietnamensis*, *P. notoginseng* contains dammarane saponins as the major constituents (Zhu et al 2004b). Zhou et al (1991) reported the separation of sanchinosides Rb1, Re, R1, Rg1 and Rh1 from *P. notoginseng* cell culture. They were identified by means of a variety of chromatographic and spectroscopic analyses, and found to be the same as those of cultivated sanchi ginseng plants, and ginsenosides isolated from *P. ginseng*.

Wei & Cao (1992) and Wei et al (1992) isolated ginsenosides Rb3, Rc and Re, notoginsenosides-Fe and -R1, and gypenoside 1X from fruit pedicels of *P. notoginseng*. Identification of these compounds was achieved by using chemical and

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spectroscopic methods. Wei et al (2002) subsequently reported the isolation of additional saponins including ginsenoside Rb1, notoginsenosides-Fc and -Fa, and gypenosides XV and XVII.

From the dried roots of *P. notoginseng*, Yoshikawa et al (1997) isolated nine dammarane-type triterpene oligoglycosides, namely notoginsenosides-A, -B, -C, -D, -E, -G, -H, -I and -J, and an acetylenic fatty acid glycoside designated as notoginsenic acid β-sophoroside. Fourteen known dammarane-type triterpene oligoglycosides were isolated at the same time.

HPLC methods were developed for determination of ginsenosides Rb1, Rb2, Rc, Rd, Re, Rf and Rg in Radix notoginseng (Chen & Sorensen 2000). A gradient HPLC method with evaporative light scattering detection (ELSD) for determination of saponins in *P. notoginseng* was described by Li & Fitzloff (2001). Wang et al (2000) described an HPLC-ELSD method for determination of notoginsenoside R1 in Radix notoginseng. It was concluded that the optimal harvest time for *P. notoginseng* was from October to December when the saponin content was high. A reversed-phase HPLC method with diode array detection was developed for the simultaneous determination of notoginsenoside R1, and ginsenosides Rb1, Rc, Rd, Re and Rg1 in raw and steamed *P. notoginseng*. It could be used for the purpose of quality control (Lau et al 2003). Dong et al (2003) reported that the contents of notoginsenoside R1, ginsenosides Rb1, Rg1 and Rd, dencichine, flavonoid and polysaccharide were the highest in unseeded sanchi ginseng plants from southwestern parts of Wenshen in China harvested in September and October.

Chen et al (2002b) isolated ginsenosides Rg3, Rh1, Rh2, FI and Mc, and daucosterol from *P. notoginseng* leaves. Du et al (2003) separated ginsenosides Rb1, Re and Rg1 and notoginsenoside R1 from the root extract of *P. notoginseng* using high-speed counter-current chromatography. From the leaves of *P. notoginseng*, ginsenosides C-K, Rh1, and notoginsenoside-Fe1V were isolated (Jiang et al 2004). Ginsenosides Rb1, Rg1 and Rh1 in callus were separated by thin layer chromatography (Zheng et al 1989). The saponins in *P. notoginseng* root were similar to those in stems and leaves of *Panax japonicum* var. major (Chi et al 1992). Xiao et al (2004) noted that ginsenosides Re and Rg1 and their isomers could be used as the identifying mark of *P. notoginseng*. The presence of ginsenosides Rb1, Rd and Rg1 and notoginsenoside R1 was demonstrated in urine after oral and intravenous administration of *P. notoginseng* saponin to rats (Li et al 2004b). On the other hand, Yang & Du (2002) reported the synthesis of oligosaccharide derivatives. The three major amino acids in *P. notoginseng* were Arg, Asp and Glu with the abundance being Arg > Asp > Glu (Chen et al 2003).

Authentication

Liquid chromatography/mass spectrometry of malonyl ginsenosides has been used to identify ginseng (Kite et al 2003). DNA from *P. ginseng*, *P. quinquefolius* and *P. notoginseng* was amplified by arbitrarily-primed polymerase-chain reaction (AP-PCR) or random amplified polymorphic DNA analysis (RAPD). Comparison of the fingerprints revealed that *P. ginseng* and *P. quinquefolius* were more closely related. Adulterants gave very different fingerprints (Shaw & But 1995). An authentication procedure based on restriction fragment length polymorphism was developed by Ngan et al (1999). Luo et al (2001) used a method involving CTAB extraction for isolating high-quality genomic DNA from various *Panax* species. The DNA restriction fragments were digested with restriction enzyme, linked up by T4 DNA ligase and amplified by nested PCR. Reproducible amplified fragment length polymorphism genomic DNA fingerprinting was established with the isolated DNA. Cui et al (2003) authenticated *P. notoginseng* by 5S-rRNA spacer domain and RAPD analysis. *P. notoginseng* and its adulterants could be identified by using DNA sequencing (Cao et al 2001).

HPLC was used for fingerprinting (Zhou et al 2001; Wang & Bi 2003; Yang et al 2004) and comparison of raw and steamed *P. notoginseng* (Lau et al 2004). The peak height ratio of Rg1 and Re could be used to differentiate *P. ginseng*, *P. quinquefolius* and *P. notoginseng* (Zhai et al 2001). Species identification by multiplex amplification refractory mutation system was applied by Zhu et al (2004a). Yuan & Hong (2003) noted genetic heterogeneity in Radix notoginseng by fluorescence amplified fragment-length polymorphism analysis. Genetic heterogeneity in rRNA gene and matK gene in *P. notoginseng* was observed also by Fushimi et al (2000).

Protein fingerprints of *P. notoginseng*, *P. ginseng* and *P. quinquefolius* obtained by sodium dodecyl sulfate-polyacrylamide gel electrophoresis, Tris-tricine gel electrophoresis and Western blot were reported by Wu et al (1999). Fingerprints above 58 kDa and below 28 kDa were specific for *P. ginseng* and *P. quinquefolius*, respectively. Fingerprints between 28 kDa and 58 kDa were common to various *Panax* species.

Effect on the liver

P. notoginseng saponins (PNS) exhibited a protective effect on acute hepatic injury induced by carbon tetrachloride, paracetamol, cadmium chloride and allyl alcohol in male mice (Liu et al 1994). PNS from flower buds protected against galactosamine and lipopolysaccharide-induced hepatic injury. From the hepatoprotective saponin fraction five novel dammarane-type triterpene saponins, including notoginsenoside-O, -P, -Q, -S and -T, and nine known protopanaxadiol oligoglycosides, were isolated (Yoshikawa et al 2003). A hot water extract of *P. notoginseng* prevented, after chronic oral administration of ethanol, the rise in serum glutamate–oxaloacetate transaminase and glutamate–pyruvate transaminase and hepatic lipid peroxidation in mice (Lin et al 2003).

PNS were used after intrasplenic hepatocellular autologous transplantation with 70% partial hepatectomy. The hepatocytes were protected against ischaemic perfusion

injury at the early stage after transplantation (Deng et al 2001). Wu et al (2003) reported the effects of PNS on the expression of tumour necrosis factor-α in rats with hepatic fibrosis. Park et al (2005) showed that *P. notoginseng* prevented hepatic fibrosis and microvascular dysfunction in rats.

Effect on the kidneys

PNS inhibited secretion of type 1 collagen, expression of integrin-β1 and proliferation of human kidney fibroblasts. Thus PNS may be useful for prevention and treatment of renal interstitial fibrobosis (Wei et al 2002). PNS inhibited interleukin-1α-induced transdifferentiation of rat renal tubular epithelial cells and secretion of extracellular matrix as seen in reduction of α-smooth muscle actin expression and fibronectin secretion. PNS appeared promising for the prevention and treatment of renal interstitial fibrosis and terminal stage of renal diseases (Wang et al 2004b). Liu & Zhou (2000) observed that PNS protected against cisplatin-induced nephrotoxicity in mice by reducing cytosolic free Ca^{2+} overload and formation of DNA interstrand and DNA–protein crosslinks.

Hypoglycaemic activity and effect on early diabetic nephropathy and diabetic macroangiopathy

Sanchinoside C1 (ginsenoside Rg1) exerted a hypoglycaemic action in alloxan-induced diabetic mice while the circulating level of insulin was not affected. Glucose uptake, glucose oxidation and glycogenesis in the liver were stimulated (Gong et al 1991). After treatment with *P. notoginseng* there was a lowering of thromboxane B2 (T) level and an elevation of 6-ketoprostaglandin F1 α (K) level, resulting in a reduction of the T/K ratio. In addition there were reductions in the urinary levels of albumin and β-2-macroglobulin and blood levels of α-1-microglobulin. The progress of diabetic nephropathy was delayed (Lang et al 1998). Liu et al (2004c) reported the beneficial effects of PNS on patients with type 2 diabetic macroangiopathy.

Immunological adjuvant activity and immunostimulatory action

Notoginsenosides-D, -G, -H and -K increased serum level of IgG in ovalbumin-sensitized mice (Yoshikawa et al 2001). Sun et al (2003) obtained similar findings with PNS. Polysaccharides from *P. notoginseng* exhibited an immunoenhancing action (Li 1991). Sanchinan-A is an arabinogalactan from *P. notoginseng* that activated the reticuloendothelial system (Ohtani et al 1987). Gao et al (1996) reported four heteroglycans with molecular weights ranging from 27 to 760 kDa and made up of glucose, galactose, arabinose, mannose and xylose in different molar ratios. All polysaccharides except one potentiated concanavalin A-induced production of interferon-γ. PNS stimulated splenocyte proliferation in response to concanavalin A, phytohaemagglutinin and pokeweed mitogen. It possessed immunologic adjuvant activity (Sun et al 2003, 2004).

Anti-inflammatory and analgesic activity

Hao & Yang (1986) and Wang et al (1994) reported the anti-inflammatory activity of PNS. Li & Chu (1999) observed that the mechanism of anti-inflammatory action of PNS was related to inhibition of neutrophil Ca^{2+} level and phospholipase A2 activity and diminution of dinoprostone content of exudates in the rat air-pouch acute inflammatory model. However, Wei et al (1999) did not notice any anti-inflammatory effect of PNS in the rat model of complete Freund's adjuvant-induced hind-paw oedema. Lei (1984) and Wang et al (1994) noted an increase in pain threshold in mice with liquid paraffin-induced inflammation after intraperitoneal administration of PNS.

Antioxidant activity and effect on plasma lipid levels

The aqueous extract of *P. notoginseng* demonstrated some haemolysis-inhibiting activity. Its organic extract had some inhibitory activity on lipid peroxidation while its aqueous extract exhibited weaker activity (Ng et al 2004). Lin et al (2003) noted that a hot water extract of *P. notoginseng* inhibited ethanol-induced lipid peroxidation in mouse liver homogenate. Xu et al (1993) reported hypocholesterolaemic and hypotriglyceridaemic activity of PNS in rats and quails. Cicero et al (2003) found that pulverized *P. notoginseng* roots reduced plasma total cholesterol concentration without affecting high-density lipoprotein-cholesterol level in rats.

Antitumour activity

Yu (1993) reported that the drug Hua-sheng-ping, composed of herbs including *P. notoginseng*, was effective on precancerous patients. Konoshima et al (1999) described the inhibitory effect of *P. notoginseng* root extract on mouse skin tumours induced by 7, 12-dimethylbenz[a]anthracene as initiator and fumonisin B1 as promoter, and also on mouse skin tumours induced by a nitric oxide donor ((+/−)-(E)-methyl-2[(E)-hydroxyimino]-5-nitro-6-methoxy-3-hexen amide) as initiator and 12-O-tetradecanoyl phorbol-13-acetate as promoter. *P. notoginseng* extract and ginsenoside Rb1 sensitized KHT sarcoma in mice to radiation. Chen et al (2001a) observed in mice that *P. notoginseng* extract and ginsenoside Rb1 increased the sensitivity of an experimental tumour, KHT sarcoma, to ionizing radiation. Chung et al (2004) noted that *P. notoginseng* was the most cytotoxic component of Pc SPES, an eight-component herbal combination for the treatment of prostate cancer, toward PC3 human prostate cancer

cells. Wang et al (2004c) reported that the serum of a dog fed with *P. notoginseng* extract inhibited proliferation of human gastric mucosa epithelium GES-1 cells and GES-1 cells transformed by N-methyl-N′-nitroso-N-nitrosoguanidine. The dammarane glycoside 20-O-β-D-xylopyranosyl (1→6)-β-D-glucopyranosyl-20(S)-protopanaxadiol showed toxicity against breast cancer MCF-7 cells (He et al 2005). *P. notoginseng* powder increased gastric secretion and gastric mucosal blood flow and reduced malondialdehyde formation in rats with a precancerous stomach lesion (Shi et al 2003).

Effect on sperm motility and estrogen-like activity

The aqueous extract, n-butanol extract and polysaccharide fraction of *P. notoginseng* improved sperm motility (Chen et al 1999). Ginsenosides Rc and Rb2 enhanced sperm motility, with ginsenoside exerting a somewhat delayed action (Chen et al 2001b). The estrogen-like activity of ginsenoside Rg1 was disclosed by Chan et al (2002). Rg1, but not its aglycone, stimulated proliferation of the estrogen receptor-positive MCF-7 cells. The stimulation was counteracted by the estrogen antagonist ICI182780. Rg1 also stimulated estrogen response element-luciferase reporter gene activity in HeLa cells. However, the binding of tritiated estradiol-17β to MCF-7 cell lysates could not be inhibited by Rg1, indicating that Rg1 did not interact directly with the estradiol receptor.

Hemostatic activity and effect on haemorrhagic shock

External application of PNS to the transected rat tail reduced the bleeding time (White et al 2000). The alcohol extract of *P. notoginseng* gave the shortest bleeding time (White et al 2000, 2001). PNS had only low haemolytic activity (Sun et al 2003). *P. notoginseng*, alone or in combination with *Salvia miltiorrhiza* and ligustrazine, could lessen the lipoperoxidative damage to tissues. Combination therapy was more efficacious than a single treatment (Wang et al 1997).

Effect on platelet aggregation and anti-atherosclerotic activity

Wang et al (2004a) reported that PNS reduced platelet activation, adhesion and aggregation. It prevented thrombosis, and improved microcirculation in patients with blood hyperviscosity syndrome. Zhao et al (1994) reported similar effects of the drug Xijian Tongshuan pill on patients with cerebral thrombosis. The drug contained *P. notoginseng* as one of its components. PNS inhibited the proliferation of cultured aortic smooth muscle cells stimulated by hypercholesterolaemic serum. Thus it could delay the progression of atherosclerotic lesions (Lin et al 1993).

Effect on the fibrinolytic system

Notoginsenoside R1 promoted the production of tissue-type plasminogen activator without affecting urokinase-type plasminogen activator or plasminogen activator inhibitor-1 in cultured human umbilical vein endothelial cells (Zhang et al 1994). R1 also increased the fibrinolytic potential of cultured human pulmonary artery smooth muscle cells by enhancing the synthesis of plasminogen activators. This action of R1 may explain the beneficial effect of *P. notoginseng* for the treatment of cardiovascular disease (Zhang et al 1997).

Effect on patients with coronary angina pectoris

Treatment of patients with a mixture of *P. ginseng* roots and *P. notoginseng* roots produced improvements in general symptoms and physical strength together with changes in lipid metabolism (Yuan et al 1997).

Effect on cardiovascular system

PNS exerted an anti-arrhythmic action (Liu & Chen 1984; Li & Zhang 1988). Kubo et al (1984) reported the effect of *P. notoginseng* on experimental disseminated intravascular coagulation. Li et al (2004e) noted that PNS reduced procoagulant activity in NB4 cells. Wang & Chen (1984) described the cardiac and haemodynamic effect of PNS. The depressant actions of PNS on vascular smooth muscles were observed by Wu & Chen (1988a), and their negative chronotropic and inotropic actions by Wu & Chen (1988b). Rao et al (1987) reported the calcium antagonist action of PNS. However, Zhang et al (1999) presented evidence that ginsenoside Rg1 was not a Ca^{2+} channel antagonist.

PNS shortened the duration of the fast action potential. It reduced the contractile force, the maximum upstroke velocity and magnitude of the slow action potential, and ^{45}Ca uptake by cultured myocardial cells (Li & Shi 1990). Gao et al (1992) observed that panaxatriol saponins from *P. notoginseng* demonstrated marked anti-arrhythmic activity in the rat model of coronary artery ligation-induced ischaemic and reperfused arrhythmias. The saponins decreased the size of myocardial infarcts, and protected against $CaCl_2$-acetylcholine-induced atrial fibrillation and flutter in mice. Hu et al (1992) showed that the alcoholic extract of cultured *P. notoginseng* reduced the heart rate and inhibited noradrenaline-stimulated constriction of aortic strips. PNS increased monophasic action potentials recorded from the myocardial surface in open-chest rabbits by contact electrodes, and inhibited the inotropic action of ouabain in isolated

guinea-pig left atria (Chen et al 1992). Li et al (1993) examined the electrophysiological actions of panaxatriol saponins from *P. notoginseng* on Purkinje fibres in ovine heart. The saponins increased the duration of action potential without affecting the amplitude, and blocked the delayed (outward) rectifier potassium channel. PNS inhibited total myocardial ATPase, but did not affect myocardial Na^+–K^+ exchanging ATPase. Ginsenoside Rb1 inhibited total myocardial ATPase and automaticity and contractility of isolated guinea-pig atria. Ginsenoside Rg1 inhibited total myocardial ATPase to a lesser extent (Chen et al 1994).

PNS increased prostacyclin level in carotid artery and reduced thromboxane A2 in blood platelets in rabbits. The anti-atherosclerotic action of PNS may be attributed to correction of unbalance between the two prostaglandins (Shi et al 1990). PNS protected the rat myocardium from injury induced by cardiac ischaemia and reperfusion. This was seen in a decrease in myocardial infarct size, reductions in malondialdehyde generation, cardiac release of creatine phosphokinase and myocardial Ca^{2+} accumulation (Li et al 1990).

PNS and Rb1, but not Rg1, blocked calcium channels (Xiong & Sun 1989; Xiong et al 1989). In patients with essential hypertension, PNS treatment improved left ventricular diastolic function. The activity of calcium pump on sarcoplasmic reticulum was increased. Myocardial intracellular calcium level was reduced. Left ventricular muscle mass was increased (Feng et al 1997).

Panaxadiol saponins of *P. notoginseng* inhibited isoproterenol-induced beating in isolated rat right atria and Ca^{2+}-induced increase of contractile force in isolated guinea-pig colon. The saponins inhibited the release of intracellular Ca^{2+} and the influx of extracellular Ca^{2+} by blocking the potential-dependent and receptor-operated calcium channel in smooth muscle (Dan et al 1993). *P. notoginseng* extract lowered blood pressure in rabbits and rats (Lei & Chiou 1986). Guan et al (1994) and Kwan (1995) reported that PNS might act as a novel and selective Ca^{2+} antagonist that did not interact with the L-type Ca^{2+} channel as in the case of KCl-induced contraction, but might instead interact with the putative receptor-operated Ca^{2+} channel as in the case of phenylephrine-induced contraction.

PNS increased the action of the calcium pump on sarcoplasmic reticulum reduced myocardial intracellular Ca^{2+} concentration, and decreased left ventricular muscle mass (Feng & Jiang 1998). PNS inhibited endothelium-dependent relaxation by preventing the rise in Ca^{2+} concentration in endothelial cells via the receptor-operated Ca^{2+} channels in the presence of acetylcholine or cyclopiazonic acid-opened non-selective cation channels (Kwan & Kwan 2000).

Wu et al (1995) reported that ginsenoside Rg1 prolonged ventricular refractoriness and repolarization, and increased ventricular fibrillation threshold. Huang et al (1999) reported that PNS improved early post-burn cardiac function in rats as reflected in reduction of cardiac troponin T and a less severe deterioration of cardiac contractile functions. Zhang et al (2003) found that PNS increased myocardial Gsalpha mRNA expression, adenyl cyclase and ATPase activity and cAMP level in myocardium of burned rats.

Trilinolein, a triacylglycerol from *P. notoginseng*, had antithrombotic, anti-arrhythmic and antioxidant activity. It reduced atherogenesis-associated free radical damage, and myocardial injury due to ischaemia and reperfusion (Chan et al 2002). Angiotensin II-induced protein synthesis, β-myosin heavy chain promoter activity and production of intracellular reactive oxygen species were inhibited. Angiotensin II- or H_2O_2-activated mitogen-activated protein kinase phosphorylation and activator protein-1- (or nuclear factor-kappa B)-reporter activities were reduced (Liu et al 2004a). The aforementioned processes, induced by noradrenaline instead of angiotensin II, were also inhibited by trilinolein (Liu et al 2004b).

PNS protected rabbit iliac artery against balloon endothelial denudation injury. It promoted regeneration of the endothelium, decreased intimal thickness, and down-regulated the expression of vascular endothelial growth factor and matrix metalloproteinase-2 (Chen et al 2004). PNS upregulated the activity of human endothelial nitric oxide synthase gene promoter activity in NIH 3T3 cells (Zhang et al 2004).

Effects on the brain and eyes

PNS protected against scopolamine-induced amnesia (Hsieh et al 2000). An increase in spontaneous behaviour was observed in rats after oral administration of *P. notoginseng* (Cicero et al 2000). Lipid peroxidation and cell damage were reduced by a herbal preparation containing *P. notoginseng* in a rat model of incomplete cerebral ischaemia involving bilateral ligation of common carotid arteries (Leung et al 1991). Han & Hu (1996) reported the protective action of PNS on ischaemic brain damage. Administration of PNS within 5 h after the onset of cerebral ischaemia decreased brain oedema, infarct size and neurological deficit score (He et al 2004). The panaxatriol saponins from *P. notoginseng* protected against focal cerebral ischaemia in the rat brain by alleviating cerebral oedema, up-regulating heat shock protein HSP70 expression, down-regulating transferring expression and maintaining the blood–brain barrier (Yao & Li 2002). Auxiliary treatment of patients with severe craniocerebral injury with Xuesaitong, a preparation of *P. notoginseng*, produced a marked therapeutic effect. It resulted in a lower intracranial pressure, a higher Glasgow coma score, and a better Glasgow outcome scale (Ai et al 2004). PNS prevented Ca^{2+} overload and Ca^{2+}–calmodulin complex formation in nerve cells following craniocerebral injury (Han et al 1999).

Ma et al (1997) demonstrated that PNS inhibited uptake by Ca^{2+} by rat cerebral cortical synaptosomes

and that the inhibition could be reversed by calcium, suggesting that PNS was a calcium-channel blocker in neurons. PNS, Rb1 and Rg1 activated rat brain synaptosomal Na^+–K^+ ATPase. PNS and Rb1, but not Rg1, inhibited Ca^{2+}–Mg^{2+} ATPase (Jin & Shi 1991).

The neuroexcitotoxic nonprotein amino acid, β-N-oxalo-L-α, β-diaminopropionic acid, was found in *P. notoginseng* as well as other *Panax* species (Long et al 1996). PNS protected cultured rat cortical neurons from glutamate neurotoxicity (Ma et al 1999). PNS protected cultured chick embryo neurons from NaCN-induced hypoxic cell damage. The breakdown of ATP was inhibited and ATP restoration was accelerated. The release of creatine kinase was diminished (Jiang & Qian 1995). PNS reduced apoptosis and necrosis in neurons and neuronal $[Ca^{2+}]i$ and leakage of lactate dehydrogenase (Zhu et al 2003).

P. notoginseng root increased neurite outgrowth in SKN-SH cells. Notoginsenoside R increased the percentage of cells with multipolar neuritis. Rb1, Rb3 and Rb4 increased the number of varicosities (Tohda et al 2002). Ginsenoside Rd promoted astrocyte differentiation from neural stem cells (Shi et al 2005). PNS is a vasodilator of blood vessels in the brain (Wu & Sun 1992).

Treatment of patients suffering from retinal vein occlusion with a combination of PNS and isovolumic haemodilution reduced retinal circulation time, and promoted disappearance of retinal haemorrhage, oedema, capillary leakage and cystoid macular oedema (Xi et al 2000).

Antifungal activity

A small chitinase-like antifungal protein with a molecular weight of 15 kDa (Lam & Ng 2001a) and a 35-kDa antifungal protein (Lam & Ng 2002a) were isolated from *P. notoginseng* roots. The former inhibited mycelial growth in *Fusarium oxysporum*, *Coprinus comatus* and *Mycosphaerella arachidicola* but not in *Rhizoctonia solani*. The latter inhibited fungal growth in *C. comatus*, *F. oxysporum*, *Botrytis cinerea* and *Physalospora piricola*, and exerted an inhibitory activity on HIV-1 reverse transcriptase.

Ribonuclease and xylanase activity

A heterodimeric ribonuclease composed of a 27-kDa subunit and a 29-kDa subunit was isolated from *P. notoginseng* roots (Lam & Ng 2001b). It exhibited potent ribonuclease and cell-free translation-inhibitory activity. Antiproliferative activity against leukemia L1210 cells and antifungal activity against *C. comatus* and *P. piricola* were demonstrated.

A low-molecular-weight xylanase with HIV-1 reverse transcriptase inhibitory activity was isolated from *P. notoginseng* roots (Lam & Ng 2002b). It exhibited an optimal temperature at 50°C and an optimum pH between 5 and 6.

Pharmacokinetics and bioavailability

The pharmacokinetics and bioavailability of ginsenosides from *P. notoginseng* in rats were studied by Xu et al (2003a) and Li et al (2004b). Its excretion profile was reported by Li et al (2004c). PNS was absorbed in rabbits to a greater extent when administered by the intranasal route as compared with other routes. The formulation in microcrystalline cellulose had the advantages of high bioavailability and low irritation (Xu et al 2003b).

Ginsenoside biosynthesis

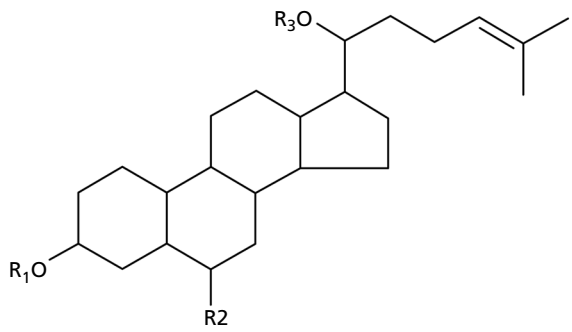
Calli induced from *P. notoginseng* leaves, petioles, flowers, stems, roots and tubers synthesized saponins (Zheng et al 1989). Hu et al (2001) described an improved method for production of saponin and polysaccharide of various ginseng species by high-density cultivation in pneumatically-agitated bioreactors. Addition of methyl jasmonate to *P. notoginseng* cell culture increased the production of ginsenosides (Wang et al 2005). Ginsenoside biosynthesis was also affected by the calcium concentration of the medium (Yue & Zhong 2005). Zhang & Zhong (2004) described scaling up of a centrifugal impellar bioreactor for hyperproduction of saponin and polysaccharide by cultured *P. notoginseng* cells. Gao et al (2003) pointed out the importance of industrialization of medicinal plant tissue culture for the modernization of traditional Chinese medicine.

Culture conditions and ways to prevent root rot and infection

Information in these areas was provided by Cui et al (1992, 1998), Wang et al (1998, 2000), Li et al (1998, 1999, 2000), and Chen et al (2002a, 2002c). The heavy metal content of *P. notoginseng* was below the national standard of Peoples' Republic of China. Organochlorine pesticide multi-residue content was low (Wang et al 1999; Zhang et al 2000).

Conclusion and future perspectives

Many of the constituents of *P. notoginseng* including the ginsenosides are in common with those of *P. ginseng* and *P. quinquefolius* (Figure 1). Recent investigations have disclosed several bioactive proteins in *P. notoginseng* roots including two antifungal proteins and two enzymic proteins, a ribonuclease and a xylanase. Immunostimulating polysaccharides are produced by *P. notoginseng*, as in some other plants. Trilinolein is a



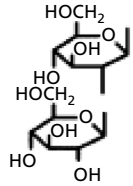
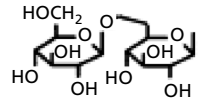
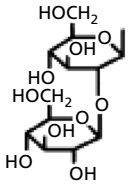
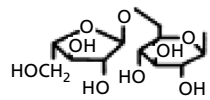
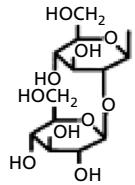
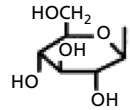
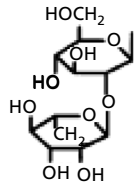
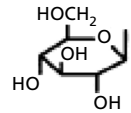
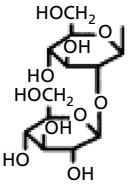
Ginsenosides	R^1	R^2	R^3
Rb1		H	
Rc		H	
Rd		H	
Re	H		
Rg3		H	H

Figure 1 Structures of some components of sanchi ginseng.

triacylglycerol produced by *P. notogeinseng* with anti-arrhythmic, antithrombotic and antioxidant activity. *P. notoginseng* saponins exert protective effects on the central nervous and cardiovascular systems, partly by reducing free radical damage. They also display hypoglycaemic, hypolipidaemic, immunostimulatory, anti-tumour, anti-inflammatory, analgesic, antioxidant, haemostatic, antithrombotic, anti-atherosclerotic, fibrinolytic, anti-arrhythmic, hypotensive, estrogen-like and sperm motility enhancing activity (Table 1). Chromatographic methods for identifying and determining constituents are useful for quality control.

Although *P. notoginseng* is mainly used for its cardiovascular action, it has a host of other activities which could be exploited. The mechanisms of some of these activities await elucidation.

Table 1 Biological activity of sanchi ginseng

Biological activity	Chemical constituent/fraction	Representative reference (s)
Hypoglycaemic	Ginsenoside Rg1	Gong et al (1991)
Antidiabetic nephropathy	Crude extract	Lang et al (1998)
Antidiabetic macroangiopathy	Crude extract	Liu et al (2004c)
Hepatoprotective	Saponin	Deng et al (2001), Yoshikawa et al (2003), Park et al (2005)
Renoprotective	Saponin	Liu & Zhou (2000), Wei et al (2002), Wang et al (2004b)
Immunomodulatory	Polysaccharides	Ohtani et al (1987), Li (1991), Gao et al (1996)
	Saponin	Sun et al (2003, 2004)
Immunological adjuvant	Saponin	Yoshikawa et al (2001), Sun et al (2003)
Anti-inflammatory	Saponin	Hao & Yang (1986), Wang et al (1994), Li & Chu (1999)
Analgesic		Lei (1984), Wang et al (1994)
Antioxidant	Trilinolein, aqueous extract	Chan et al (2002), Lin et al (2003), Ng et al (2004)
Hypolipidaemic	Saponin	Xu et al (1993)
Antitumour	Extract	Konoshima et al (1999), Chen et al (2001a)
	Ginsenoside Rb1	Chen et al (2001a)
Estrogen-like	Ginsenoside Rg1	Chan et al (2002)
Enhancing sperm motility	Polysaccharide fraction	Chen et al (1999)
	Ginsenosides Rc & Rb2	Chen et al (2001b)
Haemostatic	Saponin	Chen et al (2001)
Antithrombotic	Saponin, trilinolein	Chen et al (2002b), Wang et al (2004a)
Anti-atherosclerotic	Saponin	Lin et al (1993)
Fibrinolytic	Notoginsenoside R1	Zhang et al (1997)
Anti-arrhythmic	Saponin, trilindein	Shi et al (1990), Gao et al (1992), Chan et al (2002)
Cardioprotective	Saponin	Li et al (1990)
Negative chronotropic and inotropic	Saponin	Wu & Chen (1988b)
Improving early post-burn cardiac function	Saponin	Huang et al (1999)
Blocking Ca channels	Saponin	Xiong et al (1989)
Hypotensive		Lei & Chiou (1986)
Selective Ca^{2+} antagonist	Saponin	Kwan (1995)
Neuroprotective	Crude extract	Leung et al (1991)
	Saponin	Han & Hu (1996), Han et al (1999), Ma et al (1999), Yao & Li (2002)
Antifungal	Protein	Lam & Ng (2001a, 2002a)
Ribonuclease	Protein	Lam & Ng (2001b)
Xylanase	Protein	Lam & Ng (2002b)

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