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Effects of *Panax notoginseng* saponins on receptor-operated Ca^{2+} channels in vascular smooth muscle[1]

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ABSTRACT The effects of saponins of *Panax notoginseng* (PNGS) on the α-adrenoceptor agonists-induced contractile responses and Ca^{2+} movement were studied in dog mesenteric artery (MA) and saphenous vein (SV). PNGS reduced the contractions and the ^{45}Ca influx (from 0.36±0.03 to 0.14±0.05 μmol·g^{-1} wet strip) induced by phenylephrine (Phe) without effect on KCl-induced contraction and ^{45}Ca influx which were nearly completely inhibited by nifedipine 0.1 μmol·L^{-1}. PNGS did not change the ^{45}Ca efflux induced by Phe and the K_d value (from 0.76±0.04 to 0.72±0.15 nmol·L^{-1}) for [^{3}H]prazosin binding on the microsomal membrane isolated from MA. Our results indicate that PNGS selectively inhibites Ca^{2+} entry through receptor-operated Ca^{2+} channel.

KEY WORDS ginseng; saponins; vascular smooth muscle; calcium channels; alpha-adrenergic receptors; phenylephrine; nifedipine; prazosin

The saponins extracted from *Panax notoginseng* (PNGS) dilated the blood vessels and reduced the blood pressure in rat, cat and dog. In 1985, we first reported that PNGS inhibited norepinephrine (NE)-induced contractile response without effect on the contractile response to KCl in rabbit aorta, and suggested that PNGS might be a Ca^{2+} entry blocker which could cause vasodilation by inhibiting adrenoceptor operated Ca^{2+} influx[1]. Later, we found that PNGS did not block Ca^{2+} entry through the Ca^{2+} channel on rat motor nerve terminal which belongs to *N*-subtype of voltage-dependent Ca^{2+} channel (VDC)[2]. We redetermined the selectively inhibitory effect of PNGS on Ca^{2+} entry in dog vessels and further suggested that PNGS maybe act by blocking the Ca^{2+} entry through receptor-operated Ca^{2+} channel (ROC) without affecting Ca^{2+} entry through VDC and Ca^{2+} release from the intracellular store[3].

In the present studies, we further determined the effects of PNGS on phenylephrine (Phe)-, talipexole (Tal, BHT-920)-, and KCl-induced contractile responses, and on ^{45}Ca influx and efflux in dog mesenteric artery (MA) and saphenous vein (SV). We also tested whether PNGS altered the affinity of postsynaptic α-adrenoceptor in subcellular membrane of MA.

MATERIALS AND METHODS

Vascular tissues Mongrel dogs (10—20 kg) were killed with injected iv sodium pentobarbital 100 mg·kg^{-1}. The superior mesenteric arteries and saphenous veins were cut into spiral strips (about 2 mm in width and 3.5 mm in length) in Krebs' solution: NaCl 115.0, KCl 4.6, $MgSO_4$ 1.16, NaH_2PO_4 1.10, $NaHCO_3$ 21.9, $CaCl_2$ 2.5, glucose 11.0 mmol·L^{-1} and propranolol 1 μmol·L^{-1}, pH 7.4. Vascular endothelium was removed by rubbing the internal sur-

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face of vessels., which was confirmed by the absence of endothelium-dependent relaxation in the presence of acetylcholine 10 $\mu mol \cdot L^{-1}$, which in fact caused a slight contraction. The preparations were suspended in 3-ml organ baths containing Krebs' solution at 37 C and aerated with 95 % O_2 + 5 % CO_2. The preparations were allowed to equilibrate for 2 h under resting tension of 3 g for SV and 5 g for MA.

Contractility experiments During the equilibration period, the bath solution was changed every 20 min. Stable contractile response induced by KCl 100 $mmol \cdot L^{-1}$ was obtained. The contraction was considered reproducible if the maximal tension of 2 consecutive contractions differed by <10 %. The preparations which failed to produce reproducible contractions were discarded.

In some experiments, the preparation was washed with Ca^{2+}-free Krebs' solution plus egtazic acid 50 $\mu mol \cdot L^{-1}$ [4]. In Ca^{2+}-free solution, the agonists were repeatedly used to deplete intracellular Ca^{2+} store, and then, in the presence of agonist, $CaCl_2$ 2.5 $mmol \cdot L^{-1}$ was added to obtain the contractile response presumably due to extracellular Ca^{2+} entry. When the contractile response reached the plateau, the preparation was thoroughly washed with Krebs' solution and left standing in Krebs' solution for 20 min to refill intracellular Ca^{2+} storage sites.

^{45}Ca influx and efflux The experiments were carried out as described in our previous reports[5,6].

Subcellular membrane binding experiment Radioligand-receptor binding was done in the plasma membrane fractions (microsome-2) obtained from MA. Plasma membrane vesicles was prepared[7]. Mg-MOPS buffer (MOPS 50 $mmol \cdot L^{-1}$ and $MgCl_2$ 10 $mmol \cdot L^{-1}$, pH 7.2) was used in the incubation media. [^{3}H]Prazosin was used as the radioligand. It was freshly diluted from stock solution with cold Mg-MOPS buffer. The incubation media contained 100 μl of Mg-MOPS buffer with or without PNGS and 50 μl of diluted radioligand. The reaction was started by adding 100 μl of membrane suspension to make a final volume of 250 μl. Incubation was carried out in a gyratory shaker water bath at 25 C for 25 min. The reaction was terminated by adding 2.0 ml of cold (4 C) Mg-MOPS buffer to the entire incubation mixture. Aliquots of 2 ml from this tube were filtered rapidly over Whatman glass fiber GF/C filters. Each filter was washed with cold Mg-MOPS buffer thrice (5 ml each time). Total time of filtration and washing averaged 20 s. Filters were dried overnight at 20 C. Then filters were left to equilibrate in the scinitillation cocktail at 20 C for about 10 h and then counted in the liquid scinitillation counter. In the blank tubes, the membrane was replaced by sucrose-MOPS buffer (sucrose 0.25 $mol \cdot L^{-1}$, and MOPS 10 $mmol \cdot L^{-1}$, pH 7.4). Specific binding for [^{3}H]prazosin was calculated[8].

Nifedipine (Sigma) was dissolved and stored in dark in 100 % ethanol at 10 $mmol \cdot L^{-1}$ stock solution and freshly diluted to desired concentrations with demineralized water. Prazosin-HCl (Pfizer) was dissolved in HCl 0.01 $mol \cdot L^{-1}$ and further diluted to desired concentration with demineralized water. Egitazic acid (Sigma), *dl*-propranolol (Sigma), cocaine-HCl (Sigma), phenylephrine (Sigma), yohimbine (Sigma) and talipexole (Boehringer Ingelheim) were prepared in demineralized water. ^{45}Ca and [^{3}H]prazosin (647.5 -1036 $TBq \cdot mol^{-1}$) were purchased from New England Nuclear (Boston MA, USA). PNGS with 7 spots by thin layer chromatography which contains panaxadiol and panaxatriol was obtained from Wu-Zhou Pharmaceutical Co, Wu-Zhou City, Guangxi Chuang Autonomous Region, China[1].

RESULTS

Contraction in Krebs' solution In MA, Phe 1 and 10 $\mu mol \cdot L^{-1}$ induced the contractile responses of 69 ± 8 and 86 ± 11 $g \cdot g^{-1}$ wet tissue respectively. PNGS 0.6 $g \cdot L^{-1}$ reduced these responses to 37.0 ± 2.5 (reduced by 46 ± 6 %; $n = 10$ experiments on 15 strips of 10 dogs; $P < 0.01$) and 32 ± 6 (reduced by 62 ± 5 %; $n = 10$ experiments on 12 strips of 10 dogs; $P < 0.001$) $g \cdot g^{-1}$ wet tissue respectively. But PNGS 0.6 $g \cdot L^{-1}$ decreased Phe (10 $\mu mol \cdot L^{-1}$)-induced contractile responses only by 19 ± 3 % (from 84 ± 13 to 68 ± 11 $g \cdot g^{-1}$ wet tissue; $n = 10$ experiments on 19 strips of 10 dogs; $P < 0.05$) in SV. After washout of PNGS, the original resting tension was restored and the Phe-induced contractions returned to control level. PNGS also caused re-

laxation of the MA strips precontracted with Phe 10 $\mu mol \cdot L^{-1}$ (relaxed by 37.1±1.8 %; n = 8 experiments on 8 strips of 8 dogs; $P <$ 0.001). However, KCl (100 $mmol \cdot L^{-1}$)-induced contractile responses were not altered by PNGS (Fig 1).

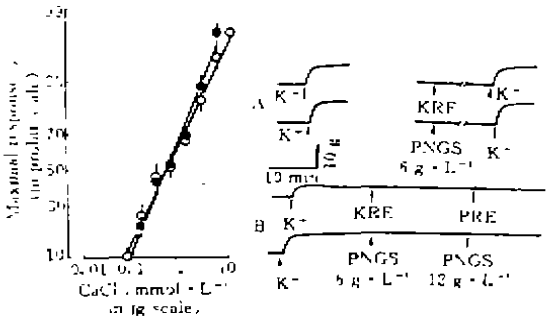

Fig 1. $CaCl_2$-induced contractile responses by accumulatively adding $CaCl_2$ in Ca^{2+} free solution containing KCl 100 $mol \cdot L^{-1}$ and egtazic acid 50 $\mu mol \cdot L^{-1}$ before (○) and after (●) addition of PNGS 0.6 $g \cdot L^{-1}$. n=6 strips of dogs. A) KCl 100 $mmol \cdot L^{-1}$ 20 min after adding PNGS (lower tracing) and Krebs' solution (50 μl, upper tracing). B) additon of Krebs' solution and PNGS during the plateau of KCl-induced contraction.

Contraction induced by Ca^{2+} In Ca^{2+}-free containing egtazic acid solution, addition of α-adrenoceptor agonists induced contractile responses which were due to intracellular Ca^{2+} release. After depleting the intracellular calcium store by repeatedly using α-adrenoceptor agonists, subsequent addition of $CaCl_2$ evoked contractile response which was considered to be due to extracellular Ca^{2+} entry and was markedly inhibited by PNGS in the presence of Phe 10 $\mu mol \cdot L^{-1}$ in MA. In the presence of Tal 10 $\mu mol \cdot L^{-1}$, subsequent addition of $CaCl_2$ did not evoke a contractile response in MA, but did in SV and this response was inhibited by PNGS to 42 % (Tab 1). This effect of PNGS was concentration-dependent.

PNGS had no significant effect on the contractile response induced by subsequent addition of $CaCl_2$ in the presence of KCl 100 $mmol \cdot L^{-1}$ which was due to Ca^{2+} entry through VDC (Tab 1). However, this contractile response was nearly completely inhibited by nifedipine 0.1 $\mu mol \cdot L^{-1}$ which can selectively block Ca^{2+} entry through VDC. In the presence of Phe and Tal, PNGS significantly decrease $CaCl_2$-induced contractile responses and the maximum contractile responses (Fig 2). On the other hand, PNGS did not significantly change the $CaCl_2$-induced contractile responses in the presence of KCl 100 $mmol \cdot L^{-1}$ (Fig 1).

^{45}Ca influx and efflux KCl 100 $mmol \cdot L^{-1}$ and Phe 10 $\mu mol \cdot L^{-1}$ evoked ^{45}Ca

Tab 1. Effects of PNGS and nifedipine on the Ca^{2+}-dependent contraction (g tension/g wet tissue) induced by Phe, Tal, and KCl. $\bar{x} \pm s$. $^cP < 0.01$ vs pretreatment.

Drug	Strip	Strips (Dogs)	Before treatment	PNGS 0.6 $g \cdot L^{-1}$	Nifedipine 0.1 $\mu mol \cdot L^{-1}$	Krebs' sol 30 μl
Tal 10 $\mu mol \cdot L^{-1}$	SV	13 (13)	30±4	18.8±1.9^c	—	—
		15 (13)	32±3	—	—	31±3
Phe 10 $\mu mol \cdot L^{-1}$	MA	15 (13)	77±9	48±7^c	—	—
		11 (11)	79±13	—	20±4^c	—
		9 (9)	66±15	—	—	64±15
KCl 100 $mmol \cdot L^{-1}$	MA	8 (8)	51±5	51±5	—	—
		6 (6)	49±4	—	4.4±0.6^c	—
		9 (9)	44.5±1.9	—	—	41.6±2.0

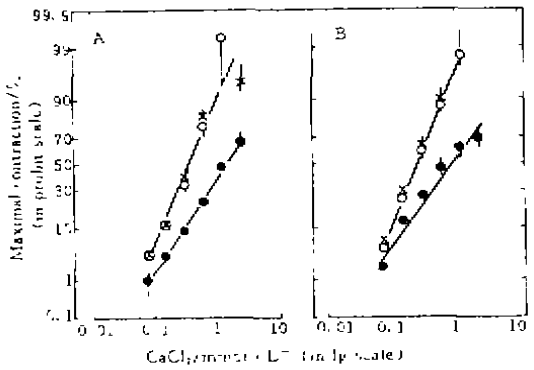

Fig 2. Effects of PNGS (●) on $CaCl_2$-induced contractile responses in the presence of Tal 10 μmol·L^{-1} (in SV; $n=7$ experiments on 14 strips of 7 dogs; A) and Phe (in MA; $n=7$ experiments on 21 strips of 7 dogs; B) (×) pretreatment and (○) control.

influxes of 0.44±0.03 and 0.36±0.03 μmol·L^{-1}·g^{-1} wet tissue respectively. The effect of Phe was significantly inhibited by PNGS. PNGS did not alter the KCl-evoked ^{45}Ca influx which was completely inhibited by nifedipine 0.2 μmol·L^{-1} (Tab 2).

Tab 2. Effects of PNGS 0.6 g·L^{-1} and nifedipine 0.2 μmol·L^{-1} on ^{45}Ca influx (μmol·g^{-1} wet wt of strip) induced by KCl 100 mmol·L^{-1} and Phe 10 μmol·L^{-1}. $n=8$ dogs. $\bar{x}\pm s$. $^aP>0.05$, $^cP<0.01$; *vs* Vehicle.

	KCl	Phenylephrine
Vehicle	0.44±0.03	0.36±0.03
PNGS	0.44±0.04^a	0.14±0.05^c
Nifedipine	0.03±0.01^c	—

Phe increased the loss rate of ^{45}Ca which reflected intracellular Ca^{2+} release, and PNGS did not significantly change Phe effect (Tab 3).

Affinity of α-adrenoceptor In isolated vessel strip experiments, PNGS markedly inhibited the contractile responses induced by different concentrations of Phe (in MA) or Tal (in SV) in the Krebs' solution, and decreased

Tab 3. Loss of ^{45}Ca (pmol·g^{-1}·min^{-1}) after Phe 1 μmol·L^{-1} and PNFS 0.6 g·L^{-1}. $n=3$ dogs. $\bar{x}\pm s$. $^cP<0.01$; *vs* control.

Time	Control	Phe	Phe+PNGS
10 min	90±6	92±6	95±4
20 min	56±6	58±5	60±5
30 min	30±4	30±3	35±5
40 min	14±2	15±3	16±3
50 min	10±2	25±3^c	25±3^c
60 min	8±1	10±2	10±2
70 min	5±1	6±1	6±1
80 min	5±1	6±1	6±1

the maximum contractile responses. Prazosin 0.1 μmol·L^{-1} and yohimbine 0.1 μmol·L^{-1} changed these responses of Phe and Tal, respectively, without inhibition of maximum contractile responses in a competitive manner (Fig 3).

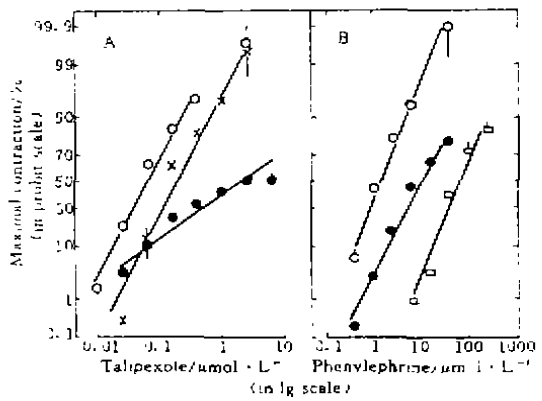

Fig 3. Effects of PNGS (●), yohimbine (×; A), and prazosin (□; B) on contractile responses to different concentrations of Tal (in SV; $n=7$ experiments on 13 strips of 7 dogs; A) and Phe (in MA; $n=7$ experiments on 7 strips of 7 dogs; B) (○) pretreatment.

In subcellular membrane radioligand binding experiments, analysis of the Scatchard plots of the specific binding data of [^{3}H]prazosin in microsome-2 fraction of MA yielded the B_{max} estimate of 56±18 fmol/mg protein, and the K_d value of 0.76±0.04 nmol·L^{-1} (data from 3 different experiments). PNGS did

not significantly change the K_d value (0.72 ± 0.15 nmol·L^{-1}), but decreased the B_{max} to 31 ± 11 fmol/mg protein (decreased by 44.5 ± 2.6 %). Inhibition of [^{3}H]prazosin binding by PNGS was dependent on the concentration of PNGS, and was nearly maximal at 1 g·L^{-1} (Fig 4).

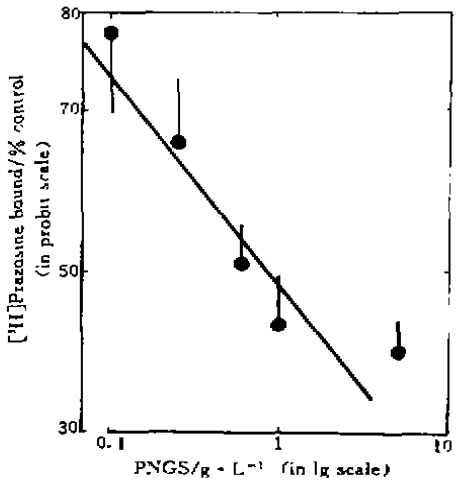

Fig 4. Inhibition of [^{3}H]prazosin (0.7 − 1.0 nmol ·L^{-1}) binding by PNGS. n=3−4 experiments.

DISCUSSION

In previous work, we have found that PNGS had an inhibitory effect of NE-induced contractile responses in Krebs' solution and no effect on KCl-induced contractile responses. These effects of PNGS were different from the blockade effects of general Ca^{2+} entry blockers[1]. In the present study, we further found that PNGS specifically inhibited contractile responses which were induced by subsequent addition of Ca^{2+} in the presence of Phe and Tal after depleted intracellular Ca^{2+} store, and Ca^{2+} influx induced by Phe. However, PNGS did not significantly alter the $CaCl_2$-induced contractile response in the presence of KCl 100 mmo · L^{-1}, and KCl-induced Ca^{2+} influx. These effects of KCl were blocked by nifedipine. Moreover, PNGS did not decrease the ^{45}Ca efflux which reflected intracellular Ca^{2+} release induced by Phe. These results are consistent with our previous results which suggested PNGS had a selective effect on ROC[1,3].

In contractile response and subcellular membrane binding experiments, PNGS did not change the affinities of postsynaptic α-adrenoceptors. In fact, the results exclude the possibility that the effects of PNGS were induced through the change of α-adrenoceptor affinity. In membrane radioligand binding experiments, although PNGS decreased specific binding sites of [^{3}H]prazosin, this effect did not play an important role in the inhibition of α-adrenoceptor agonists-induced contractions and ^{45}Ca influx by PNGS. First, although PNGS 0.6 g·L^{-1} decreased the amount of postsynaptic α-adrenoceptor by 44 %, the postsynaptic α-adrenoceptor reserve was so large that activation of only 40 % of the receptors produced 90 % maximum contractile response induced by Phe in MA[9]. If PNGS inhibited agonist-induced contractile responses by decreasing the amount of receptors, PNGS 0.6 g·L^{-1} should only inhibit these contractile responses by less than 10 %. In fact, PNGS 0.6 g·L^{-1} inhibited Phe-induced contractile response by 46.3 %. Second, compared with MA, there was little receptor reserve in SV[9], but Phe (10 μmol·L^{-1})-induced responses in MA were more sensitive to PNGS than that in SV. Finally, in subcellular membrane binding experiments, the effect of PNGS 1 g·L^{-1} on decreasing the amount of receptors was near maximum (Fig 5). But in isolated tissue experiments, inhibitory effect of PNGS was increased with raising concentration of PNGS, even the concentration level was over 1 g·L^{-1} (data not shown). According to our results, it is excluded that PNGS inhibited α-adreno-

ceptor agonists-induced contractile responses and ^{45}Ca influx by a change in affinity and amount of α-adrenoceptors. Effect of PNGS on amount of α-adrenoceptors appears to be a nonspecific effect. It has been found that saponins of quillaja, which have been commonly used to permeabilize the cell membranes of vascular smooth muscle[10,11], also decreased the B_{max} of [^{3}H]prazosin binding in subcellular membrane of MA by 10—25%[12].

We have noticed the report that PNGS inhibited not only NE-induced but also KCl-induced contractile responses in the rabbit vessels[13]. However, in that study, there are some problems. First, they did not determine whether the preparations could produce reproducible contractile responses before collection of data. Secondly, to determine effect of PNGS on KCl-induced contractile response, they did not use some drugs to block NE transmitter release from sympathetic nerve terminal or postsynaptic α-adrenoceptors. It has been suggested that in vascular smooth muscle, 30—60 % contractile response to KCl is due to depolarization of presynaptic membrane following by the transmitter release from nerve terminal[14,15]. Thus, the inhibition of KCl-induced contractile response by PNGS may only reflect a fact that PNGS inhibited the part of contractile response induced by activation of postsynaptic α-adrenoceptor following transmitter release from presynaptic terminal. We re-determined the effect of PNGS on KCl-induced contractile response in rabbit mesenteric artery under the same conditions reported. We found that PNGS (1 g $\cdot L^{-1}$) inhibited KCl (100 mmol $\cdot L^{-1}$)-induced contractile response to 87.9±3.9 % ($n=6$ experiments on 6 strips of 6 rabbits, $P<0.05$) when the transmitter release and the postsynaptic α-adrenoceptors were not blocked. However, when using prazosin 1 μmol $\cdot L^{-1}$ plus rauwolzine 1 μmol $\cdot L^{-1}$ to block postsynaptic α-adrenoceptors, PNGS 1 g $\cdot L^{-1}$ no longer altered KCl (100 mmol $\cdot L^{-1}$)-induced contractile response (98.4±1.7 %, $n=5$ experiments on 5 strips of 5 rabbites, $P>0.05$). Furthermore, PNGS did not significantly inhibit KCl-induced ^{45}Ca influx which was completely blocked by nifedipine (Tab 2). Thus, the inhibition of KCl-induced contractile response by PNGS[13] is in question.

Present data further confirm that PNGS seletively inhibits the Ca^{2+} entry through ROC without affecting intracellular Ca^{2+} release, affinity of α-adrenoceptors and Ca^{2+} entry through VDC.

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三七皂甙对血管平滑肌上受体操纵 Ca^{2+} 通道的特异性作用[1]

R965.2

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摘要 三七皂甙能明显抑制狗肠系膜动脉及大隐静脉 α 肾上腺素能受体触发的收缩反应及 ^{45}Ca 内流（从0.36±0.03 降至 0.14±0.05 $\mu mol \cdot g^{-1}$组织湿重），但对高 K^+引起的 Ca^{2+} 内流无影响. 三七皂甙不影响 Ca^{2+} 释放及受体的亲和力. 提示三七皂甙具有特异性阻断受体操纵 Ca^{2+} 通道的特性，对电位依赖性 Ca^{2+} 通道无作用.

关键词 人参；皂甙类；血管平滑肌；α 肾上腺素受体；钙通道；苯福林；硝苯地平；哌唑嗪