Research Progress of *Panax notoginseng* Saponins (PNS) in the Treatment of Cardiovascular Diseases

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Abstract *Panax notoginseng* saponins (PNS) is the primary active component of the traditional Chinese medicine *P. notoginseng*. This compound exhibits a range of pharmacological effects, including anti-inflammatory, antioxidant, and antiplatelet aggregation properties, as well as the enhancement of microcirculation. The extensive research on the modernization of traditional Chinese medicine has garnered significant attention regarding the application of PNS in the treatment of cardiovascular diseases. Research has demonstrated that PNS interventions significantly improve the pathological progression of cardiovascular disease through synergistic effects involving multiple targets and pathways. This paper summarizes the pharmacological mechanisms, clinical research advancements, safety, and potential adverse reactions associated with PNS in the treatment of cardiovascular diseases, in order to provide theoretical references for future research and practical applications in this field.

Key words *Panax notoginseng* saponins (PNS), Cardiovascular diseases, Platelet aggregation, Oxidative stress, Lipid metabolism, Myocardial fibrosis, Inflammatory response, Cardiomyocyte

1 Introduction

Cardiovascular disease (CVD) is one of the most widespread health concerns worldwide, characterized by significant morbidity and mortality rates. Research indicates that the incidence and mortality associated with CVD exhibited a gradual upward trend from 1990 to 2019[1]. The pathological mechanisms encompass inflammation, oxidative stress, endothelial dysfunction, disorders of lipid metabolism, and thrombosis. Despite substantial advancements in contemporary medicine, particularly in pharmacological interventions such as antiplatelet agents and lipid-modulating therapies, as well as surgical procedures, there remains a pressing need to investigate innovative therapeutic strategies. This necessity arises from the fact that some patients continue to encounter difficulties, including adverse effects from medications and the recurrence of disease. The significance of traditional Chinese medicine in the prevention and treatment of CVDs is increasingly recognized and has garnered considerable attention. The traditional Chinese medicine *P. notoginseng* is recognized for its ability to activate blood circulation and eliminate blood stasis. In *Yuqiu's Explanation of Medicinals*, it is documented that this herb possesses the effects of harmonizing nutrient-blood and halting bleeding, clearing the veins to alleviate blood stasis, and promoting the convergence of new blood by removing blood stasis. Zhang Xichun asserts that it is particularly effective in the removal of blood stasis. Modern pharmacological research has demonstrated that PNS, the principal component of *P. notoginseng*, comprises over 20 monomeric saponin components, including ginsenoside Rb1, ginsenoside Rg1, and notoginsenoside R1. These components exert influence on the progression of CVDs through various pathways and targets[2]. In recent years, advancements in molecular biology and related technologies have led to a gradual elucidation of the mechanisms underlying the anti-inflammatory, antioxidant, antiplatelet aggregation, and vasoprotective effects of PNS. Consequently, the clinical applications of PNS have been expanding. This paper systematically reviews the pharmacological mechanisms and clinical developments of PNS in the treatment of CVDs in recent years. The objective is to enhance the understanding of the research and to provide a theoretical foundation, as well as to generate new perspectives for clinical treatment.

2 Pharmacological mechanism of PNS

2.1 Antiplatelet aggregation Modern medicine recognizes abnormal platelet aggregation as a significant contributor to the development of CVDs. Research has demonstrated that various cardiovascular conditions, including atherosclerosis, myocardial infarction, and hypertension, are associated with platelet aggregation[3]. PNS has the capacity to inhibit platelet aggregation, adhesion, and release, while simultaneously enhancing hemorheology and reducing the incidence of thrombosis[4]. Through the execution of ischemia-reperfusion experiments utilizing a rat model, Chen Yu *et al.*[5] demonstrated that PNS can inhibit associated proteins and genes via the PI3K/AKT/GSK3 signaling pathway, and subsequently lead to a reduction in platelet activation, thereby enhancing coagulation function. Wang Yilin *et al.*[6] conducted a simulation of an animal thrombosis model, focusing on two primary factors: high blood flow shear stress and arterial endothelial injury. Their findings indicate that PNS can effectively inhibit thrombus formation induced by platelet coagulation under conditions of high shear stress, primarily through the involvement of Piezo1. This mechanism subsequently reduces the binding of platelets to vascular hemophilic factors, thereby decreasing both the activation and aggregation of platelets. Lu Lijuan *et al.*[7] conducted a clinical observation involving 60 patients diagnosed with

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angina pectoris due to coronary artery disease. The study found that PNS effectively regulated plasma endothelin-1 levels, inhibited platelet aggregation, and reduced platelet deposition within blood vessels. These effects contribute to a decrease in the morbidity associated with angina pectoris and facilitate the improvement of clinical symptoms in patients. Shen *et al*. [8] demonstrated that PNS interventions can effectively inhibit thrombin-induced platelet aggregation, thereby enhancing blood flow and reducing the incidence of thrombosis.

2.2 Anti-oxidative stress Vascular endothelial dysfunction is a critical factor in the pathogenesis of CVD. Oxidative stress serves as a significant mechanism contributing to endothelial injury, which may result in endothelial cell apoptosis due to the excessive production of reactive oxygen species (ROS)[9]. The excessive accumulation of ROS leads to oxidative stress, which subsequently impairs the function of cardiac vascular endothelial cells. Xuan Nan *et al*. [10] demonstrated that PNS can exert antioxidant effects and enhance cardiomyocyte function by upregulating the expression of miR-16 and further inhibiting the activity of downstream targets of the TXNIP protein, as evidenced by animal studies. Recent pharmacological studies have demonstrated that PNS can effectively inhibit oxidative stress and inflammatory responses, thereby providing protection to the vascular endothelium. Furthermore, in cases of endothelial dysfunction induced by oxygen-glucose deprivation/reoxygenation (OGD/R), PNS has been shown to alleviate damage to vascular endothelial function through the activation of the PPARα/Nrf2 signaling pathway[11]. An important pathological alteration following oxidative stress injury in vascular endothelial cells is cellular autophagy. Research has demonstrated that H_2O_2 can stimulate cellular autophagy, with Beclin1 identified as a crucial gene involved in this process. Furthermore, PNS have been shown to decrease the expression of Beclin1 at both the mRNA and protein levels. This reduction is associated with the ability of PNS to mitigate oxidative stress injury induced by H_2O_2, thereby further modulating cellular autophagy and providing protection to endothelial cells[12]. Wang Linlin *et al*. [13] demonstrated that PNS can inhibit the production of ROS and decrease the generation of free radicals by activating the nuclear factor E_2-related factor 2 (Nrf2) pathway, thereby mitigating oxidative stress-related damage. Zhang Wei *et al*. [14] demonstrated that PNS can protect the damaged myocardium in rats with coronary heart disease by upregulating the Nrf2/HO-1 signaling pathway. This mechanism effectively regulates oxidative stress levels in the body, enhances myocardial function, and safeguards cardiac performance. Wan Jingzhi *et al*. [15] established a model of SH-SY5Y cell injury induced by H_2O_2 and discovered that Sirt1 and Bcl-2 effectively inhibited oxidative stress and reduced apoptosis. Furthermore, it was observed that PNS enhanced the protective effects on endothelial cells through the Sirt1/Foxo3a and Bcl-2/Bax signaling pathways.

2.3 Regulation of lipid metabolism Atherosclerosis serves as the primary pathological foundation for CVD, and its progression is closely linked to disorders in lipid metabolism. Niu Meilan *et al*. [16] employed PNS to treat a rat model subjected to a high-fat diet. Their findings demonstrated that PNS can effectively regulate lipid levels in atherosclerotic rats, leading to a reduction in serum total cholesterol (TC) and triglyceride (TG) levels. This suggests that PNS may mitigate vascular damage induced by elevated plasma cholesterol and subsequently decrease the progression of atherosclerosis. Gao Lei *et al*. [17] identified a significant association between sortilin protein and the development of atherosclerosis, highlighting its correlation with cholesterol metabolism. Their findings indicate that PNS can modulate the expression of sortilin and ABCA1 via the ERK signaling pathway, thereby influencing cholesterol metabolism. Zhu Junfeng *et al*. [18] developed an apolipoprotein E knockout (ApoE-/-) mouse model and conducted intervention experiments using PNS. The results demonstrated a significant reduction in the serum levels of TC, TG, and LDL-C in the mice treated with PNS. These findings suggest that PNS exerts lipid-regulating and plaque-stabilizing effects, thereby effectively modulating lipid metabolism and attenuating plaque formation.

2.4 Inhibition of myocardial fibrosis Myocardial fibrosis is a prevalent pathological characteristic observed in various CVDs. This condition can result in heightened myocardial stiffness, damage diastolic function, and ultimately cardiac insufficiency. Yang Zhenni *et al*. [19] demonstrated that PNS effectively reduced the production of neutrophil extracellular traps (NETs), and subsequently inhibited collagen fiber deposition in the myocardial tissue of ISO mice following modeling. Furthermore, PNS mitigated the effects on the alignment of myocardial tissue, thereby mitigating damage to myocardial cells and providing protection against myocardial fibrosis. Li Shucheng *et al*. [20] found that PNS can mitigate the damage to myocardial fibers by decreasing plasma concentrations of natriuretic peptide and angiotensin II, thereby protecting cardiomyocytes and effectively hindering the progression of myocardial remodeling. Yang Hui *et al*. [21] conducted a study on rats with chronic heart failure, administering PNS treatment. The results demonstrated that PNS significantly improved the LVFS and LVEF levels in the rats. Additionally, there was a notable reduction in the ratio of perivascular collagen area to vascular lumen area (PVCA/LA), indicating a decrease in myocardial fibrosis. The findings suggest that PNS exerts its effects by inhibiting myocardial fibrosis via the Sirt1/Foxo1 signaling pathway, thereby promoting the recovery of cardiac function and alleviating symptoms associated with heart failure.

2.5 Suppression of inflammatory response Tang *et al*. [22] demonstrated that PNS can promote the protection of vascular endothelial cells and significantly diminish local inflammatory responses. Similarly, Yan Xiaoqing *et al*. [23] provided evidence that PNS can effectively inhibit inflammatory reactions, thereby mitigating myocardial damage and alleviating lesions in cardiac tissue.

Xiao Yuxue *et al.* [24] demonstrated that PNS significantly reduced the levels of inflammatory cytokines, specifically IL-1β, IL-6, and TNF-α, in the serum of mice subjected to myocardial ischemic reperfusion injury (MIRI). Furthermore, PNS inhibited the release of these inflammatory factors, modulated the myocardial inflammatory response, protected cardiomyocytes, and preserved the damaged myocardium. Yu Yanqiao *et al.* [25] demonstrated that PNS effectively reduced the levels of inflammatory factors, including TNF-α and IL-6. Additionally, PNS decreased inflammatory responses, inhibited intimal thickening, and further regulated the progression of coronary atherosclerosis. Based on bioinformatics analysis, Zhang Qi *et al.* [26] demonstrated that PNS can utilize various miRNAs to modulate inflammatory responses, mitigate cardiomyocyte injury, and preserve endothelial function. Gao Ruimin *et al.* [27] employed PNS to treat rats with heart failure. The findings indicated that the LVEF level in rats administered PNS exhibited a significant increase, while the levels of inflammatory cytokines, including TNF-α and IL-6, were notably reduced. These results suggest that PNS may effectively inhibit the production of inflammatory cytokines in the serum of rats with heart failure, thereby mitigating apoptosis in cardiac myocytes.

2.6 Inhibition of cardiomyocyte apoptosis Autophagy plays a crucial role in maintaining homeostasis within the intracellular environment. Wang Piao *et al.* [28] demonstrated that the inhibition of excessive autophagy can mitigate damage resulting from myocardial ischemia and reperfusion. Furthermore, it was found that PNS can inhibit the binding of lysosomes and autophagosomes within the autophagy pathway by upregulating the expression of P62 proteins in cardiac muscle tissues, while simultaneously downregulating the expression of Beclin-1 and LC3II proteins, thereby providing protection for the myocardium. Meanwhile, PNS has the capacity to inhibit autophagic flux by activating the PI3K/Akt/mTOR signaling pathway[29], thereby reducing cardiomyocyte death during the reperfusion phase. Dong Yan *et al.* [30] demonstrated that PNS significantly inhibited apoptosis and autophagy in vascular endothelial cells by upregulating the BCL2A1 protein and downregulating the Beclin1 protein, thereby providing an effective treatment for blood stasis in coronary heart disease. Li Hongzheng *et al.* [31] employed PNS to treat ApoE-/- mice subjected to a high-fat diet. Their findings indicated that the diameter of the cingulum increased while the peak flow rate decreased in the mice receiving PNS treatment. Furthermore, the experiments demonstrated that PNS effectively regulated the transcription and phosphorylation of JNK and c-Jun, inhibiting the JNK/c-Jun signaling pathway. This inhibition resulted in anti-apoptotic effects, which may contribute to the attenuation of aortic valve calcification. Wang Lihong *et al.* [32] demonstrated that PNS has the capacity to inhibit mitochondrial autophagy, regulate selective mitochondrial autophagy, mitigate excessive autophagic damage to cardiomyocytes, and decrease cardiomyocyte apoptosis via the HIF-1α/BNIP3 mitochondrial autophagy signaling pathway. Chen Shaoxian *et al.* [33] demonstrated that PNS preserves the stability of the mitochondrial membrane potential via the MIF signaling pathway, thereby mitigating cardiomyocyte apoptosis.

3 Progress in clinical research

As the primary active component of the traditional Chinese medicine *P. notoginseng*, PNS has gained significant attention in recent years for its clinical application in the treatment of CVDs. PNS has exhibited notable efficacy and safety in managing conditions such as coronary heart disease, hypertension, myocardial infarction, heart failure, and other related disorders. Kuang Rongren *et al.* [34] conducted a randomized study involving 70 patients diagnosed with paroxysmal atrial fibrillation, who were equally divided into a treatment group and a control group, with 35 patients in each group. The treatment group received a combination of PNS and amiodarone, while the control group was administered amiodarone alone. After 6 months of treatment, the maintenance rate of sinus rhythm in the treatment group was found to be 85.71% (30 patients), which was significantly higher than 60.00% (21 patients) observed in the control group. These results indicate that the combination of amiodarone and PNS is more effective in maintaining normal sinus rhythm compared to amiodarone treatment alone. Jiang Xiaoping *et al.* [35] conducted a study involving 99 patients diagnosed with atherosclerotic plaques or exhibiting carotid artery intima-media thickness of ≥0.9 mm. The participants were allocated into three groups: the PNS group, the fluvastatin group, and the PNS combined with fluvastatin group, with each group comprising 33 individuals. After a treatment period of 12 weeks, the levels of CK and TG in the PNS combined with fluvastatin group were found to be significantly lower than those in both the PNS group and the fluvastatin group, with the differences reaching statistical significance ($P < 0.05$). These findings suggest that the combination of PNS and fluvastatin may be more effective in managing atherosclerotic plaques and reducing carotid intima-media thickness.

4 Clinical safety and adverse reactions

The overall safety of PNS is favorable, characterized by a lower incidence of clinical adverse reactions. Xueshuantong, which incorporates PNS, the extract of the traditional Chinese medicine *P. notoginseng* as its primary component, has been reported to occasionally induce allergic reactions during clinical application, including symptoms such as flushing of the head and face, as well as mild skin rashes[36-37]. Another injectable formulation of Xueshuantong (lyophilized), which contains PNS as its primary component, is associated with several common adverse reactions, including dry throat, dizziness, panic, and rash[38]. Generally, the adverse reactions associated with PNS are predominantly localized skin reactions[39]. In clinical practice, it is essential to inquire

about patients' allergy histories and monitor their reactions closely. Adverse reactions should be addressed in a timely manner.

5 Conclusions

As a fundamental component of the traditional Chinese medicine *P. notoginseng*, PNS exhibits distinct advantages in the prevention and treatment of CVDs. Its pharmacological effects include antiplatelet aggregation, antioxidative stress, and the regulation of lipid metabolism through multiple pathways and targets. Despite its potential, the clinical application of PNS encounters several challenges, including a limited number of clinical trials and a deficiency of long-term clinical data. Future research must further validate its efficacy and safety, and conduct an in-depth exploration of its role. Additionally, the development of innovative formulations that integrate modern technology is essential to generate new strategies for the broader application of PNS in the treatment of CVDs. In conclusion, PNS is a significant paradigm within the modernization research of traditional Chinese medicine, demonstrating considerable potential in the prevention and treatment of CVDs. Furthermore, the integration of basic research with clinical applications will facilitate the advancement of PNS in the treatment of CVDs.

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