Rhizoma Polygoni Cuspidati (Huzhang) is the dried rhizome and root of Polygonum cuspidatum, being a Chinese herbal drug, recorded first in the book Miscellaneous Records of Famous Physicians (Ming Yi Bie Lu). It is a drug of bitter taste and cold nature, entering Liver (Gan), Lung (Fei), and Gallbladder (Dan) meridians, has actions of dispelling dampness, alleviating jaundice, clearing heat, subsiding toxin, activating blood, and removing stasis. Modern pharmacological study showed that it has obvious antibacterial, anti-inflammatory, diuretic, purgative, and menstrual restoration effects. As one of its main ingredients, polydatin (PD, also be named resveratrol) is a kind of glycoside, which exists widely in the plants like grape, earthnut, giant knotweed, black falsehellebore, sickle senna, etc. In human body, PD is hydrolyzed in intestine by glycosidase to resveratrol to display its pharmacological action. Resveratrol is the aglycone of PD; it possesses the pharmacological actions similar to those of PD but is unstable in light and heat. Both are proved to have multiple biological actions, such as liver protection, anti-inflammation, antitumor, and anti-pathogenic microbe, and are applied to prevent/treat cardiovascular diseases. Particularly, its cardiovascular pharmacological actions, such as cardio-myocyte (CM) protection, vascular smooth muscle dilation, platelet aggregation, thrombosis, and atherosclerosis prevention, have received great attention from scholars of related fields in latest years, the current progress in these aspects were reviewed.

CM Protecting and Myocardial Contraction Enhancing Actions

CMs are very sensitive to the injuries of ischemia, anoxemia, lipo-polysaccharide (LPS). Some cardio-toxic drugs, such as adriamycin, and various internal/external factors could cause cardiac changes, such as marked myocardial contraction depression, arrhythmia and myocardial stunning, as well as the ultrastructural abnormality of CMs.

Using Langendorff's technique to prepare rat's heart in vitro, Zhang, et al observed the protective action of PD on the ischemia/reperfusion (I/R) injured model. The results showed that before ischemia administering of PD could obviously reduce the injured cardiac infarcted area and improve myocardial ultrastructure after I/R injury, its mechanism was considered to be related with PD's effects of enhancing the ATP sensitive potassium channel in CMs and mitochondrial membrane and suppressing the opening of mitochondrial permeability transition pore. Zhao, et al observed the effect of PD on...
adriamycin injured myocardial ultra-structure of rat, and they discovered that PD could significantly reduce the toxicity of adriamycin on CMs, showing evident protective action. Adopting the myocardial infarction canine model established by coronary left anterior descending branch ligation, Zhang, et al (6) observed the effect of PD on acute myocardial infarction. They found that PD injection showed apparent protection on CMs, it could markedly reduce the severity of ischemia, diminish the ischemic and infarcted area, lower the activities of serum lactate dehydrogenase and creatine kinase, thus to alleviate the ischemic injury of CMs.

An extra-corporeal experiment conducted by Zhao, et al (7) for exploring the impact of LPS on β-adrenergic receptor (β-AR) and the preventing/treating effect of PD on it showed that LPS could directly induce the decrease of β-AR number and downregulate its affinity in CMs; while PD could reverse these changes, it may be one of the important mechanisms of PD for improving myocardial contraction. Another experiment conducted by Xue, et al (8) observed the influence of PD on myocardial function and ultrastructure of LPS infected rats. The results showed that LPS could induce obvious lowering of myocardial contraction, even cause shock and death, mitochondrial injury, and myocytolysis may be the important path and factor for inducing myocardial injury and contraction incapacity, while PD could alleviate these unfavorable changes to protect CMs by regulating protein kinase C activity and protecting the ultrastructure of myocardial fibers.

The research of Gao, et al (9) discovered that PD could improve the ventricular remodeling in mouse model established by isoproterenol, which showed reduction of ventricular collagen content, CMs' hypertrophy, and heart mass index. The improvement may be realized by the effect of PD in depressing blood pressure, inhibiting rennin-angiotensin-aldosterone system activation, and lowering levels of tumor necrosis factor-α (TNF-α) and endothelin-1 (ET-1).

The above researches indicated that myocardial I/R, adriamycin, LPS, etc., could induce different degrees of calcium overload, ultrastructural abnormality, decreased number and activity of β-AR, and incidence of ventricular remodeling, leading to myocardial injury and contractive power reduction. The chief mechanism of PD in protecting CMs and enhancing myocardial contraction might be its effective intervention on these ring joints.

Vascular Dilation and Microcirculation Promotion

Vascular smooth muscle (VSM) plays an important role in regulating blood pressure and microcirculatory perfusion flow. In the case of shock happening, the regulatory contraction of VSM would cause deficiency of tissue perfusion flow, leading to tissue and organ ischemia/anoxia and serial secondary pathological changes.

The study of Huang, et al (10) found that PD possesses marked vascular dilative function, showing strong dilation on coronary, cerebral, pulmonary, intestinal, and hepatic vessels, and it could enlarge the caliber of contracted small vessels, increase pulse pressure, and sweep away stasis blood cells to restore capillary blood flow. Other studies carried out by Liu, et al (11) and Jin, et al., indicated that these benefitting effects might be provided by the capability of PD in lowering the intracellular calcium concentration and pH value to reduce angiastosis; in the meantime, the vascular K+ATP channel activating action of PD was capable of improving the microcirculation of shocked animals by dilating arterioles through inducing smooth muscle hyperpolarization. Besides, PD might enhance vascular activity through promoting extra-cellular sodium ion influx to cause cell depolarization.

Wang, et al (12,13) observed the effect of PD on the activity of protein kinase C (PKC) in vascular smooth muscle cells (VSMCs) under different conditions (ischemia/anoxia, impacts of burning serum, and LPS), respectively, to explore its antishock mechanism. They referred that the protective action of PD on VSMC was made available by regulating PKC activity, which might be one of the molecular mechanism of PD's antishock action.

Thrombosis Prevention

Thrombosis is a very complex pathophysiological process; its important pathogenetic factors are the mutual intercellular reactions among platelet, white blood cell, endothelial cell, etc. PD could intervene thrombosis process through multipath, which provides experimental base for developing natural drugs associated with preventing/treating cardiocerebral