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# 丹酚酸B盐对心肌肥厚大鼠心肌基膜型胶原酶活性及其调节因子的影响

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中文摘要：目的: 探讨丹酚酸B盐(SA-B)影响纤维化心脏基膜型基质金属蛋白酶及其抑制物的抗心肌纤维化作用机制。 方法 :腹主动脉不完全结扎法制备大鼠心肌肥厚模型。大鼠随机分为假手术组、模型组、丹酚酸B盐低、中、高剂量组(10,20,40 mg·kg$^{-1}$·d$^{-1}$)和卡托普利组(40 mg·kg$^{-1}$·d$^{-1}$)。HE和天狼星红胶原染色法观察左心室组织病理和胶原沉积改变;Western印迹法分析心肌组织Ⅳ型胶原,MMP-2/9及TIMP-2蛋白表达;明胶酶谱法测定MMP-2/9活性。 结果 :SA-B干预组较模型组大鼠心肌炎性减轻,胶原沉积减少,心肌组织Hyp含量显著下降,纤维化程度明显减轻;模型大鼠心肌组织Ⅳ型胶原,MMP-2/9,TIMP-2蛋白表达以及MMP-2活性明显升高,SA-B干预组降低Ⅳ型胶原,MMP-2/9和TIMP-2 蛋白表达,降低MMP-2 活性,中、高剂量组作用更为明显。 结论 :SA-B抗心肌纤维化的作用机制之一是抑制TIMP-2的表达,降低基底膜胶原酶的表达及活性,减轻正常基底膜胶原的破坏。

中文关键词：丹酚酸B盐 心肌纤维化 Ⅳ型胶原 基膜型基质金属蛋白酶-2/9 活性

## Effects of salviandic acid B(SA-B) on activity of basement membrane-type collagenase and impact of regulatory factors in rats with cardiac hypertrophy

**Abstract:**Objective : To observe the effect of salviandic acid B(SA-B) on MMP-2/9 and TIMP-2 of fibrotic cardiac tissues in rats and explore the action mechanism of SA-B anti-fibrosis of heart. Method : Ventricular remodeling model was induced by abdominal aortic banding(AAB)in rats.Rats were randomly divided into 6 groups: normal, model, SA-B high, SA-B middle, SA-B low and captopril control group. Histological changes of heart were observed with hemotoxylin and eosin (H&E)staining and Sirius red staining. Hydroxyproline (Hyp) content in heart tissue was measured by hydrolysis method. Expression of heart tissue collagen Ⅳ, MMP-2/9 and TIMP-2 were analyzed with Western blot. The activities of heart tissue MMP-2 were determined with gelatin zymography substrate degradation method. Result : SA-B treated groups had lower heart inflammation and lower heart Hyp content; decreased Collagen deposit and alleviated cardiac fibrosis. SA-B treated groups obviously decreased the expression of Collagen Ⅳ, MMP-2/9 and TIMP-2. The activity of MMP-2 was decreased in treated SA-B treated groups. Conclusion : The mechanism of SA-B action against cardiac fibrosis may be related to down-regulating the expression of TIMP -2 and the activity of MMP-2/9, thus protect the normal basal membrane.

**keywords:**salviandic acid B cardiac fibrosis collagen Ⅳ metalloproteases-2/9 activity

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