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苓桂术甘汤对急性心肌梗死后心室重构模型大鼠**NF-κB**的影响

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中文摘要：目的：观察苓桂术甘汤对急性心肌梗死(acute myocardial infarction,AMI)后心室重构模型大鼠心肌组织核因子-κB(nuclear factor-κB,NF-κB)及NF-κB mRNA表达,血清NF-κB含量的影响,探讨苓桂术甘汤干预AMI后心室重构(ventricular remodeling,VR)的作用机制。 方法：采用冠状动脉结扎法复制心室重构大鼠模型,造模2周后将模型大鼠随机分为模型组,卡托普利4.4 mg·kg^{-1}组,苓桂术甘汤(按生药量计)低、中、高剂量(2.1,4.2,8.4 g·kg^{-1})组,另设假手术组,分别ig给药,连续给药4周,采用Western blot,RT-PCR及ELISA技术检测各组大鼠心肌组织NF-κB,NF-κB mRNA表达,血清NF-κB含量。 结果：假手术组,模型组,苓桂术甘汤低、中、高剂量组,卡托普利组的心肌组织NF-κB相对表达量(NF-κB/β-actin)分别为:0.190±0.011,0.772±0.026,0.366±0.059,0.295±0.033,0.235±0.013,0.341±0.023;NF-κB mRNA相对表达量分别为:1.000,26.875,6.574,4.340,1.194,5.540;血清NF-κB含量分别为(125.85±14.76),(196.98±17.79),(163.89±20.08),(131.73±10.47),(141.93±10.32),(133.93±9.27)ng·L^{-1}。模型组与假手术组比心肌组织NF-κB,NF-κB mRNA表达、血清NF-κB含量均显著升高(P<0.01);苓桂术甘汤各剂量组及卡托普利组能够显著抑制模型大鼠心肌组织NF-κB,NF-κB mRNA表达、降低模型大鼠血清NF-κB含量,与模型组比有显著性差异(P<0.01或P<0.05)。 结论：苓桂术甘汤干预AMI后VR的机制与其抑制NF-κB有关。

中文关键词：苓桂术甘汤 心肌梗死 心室重构 核因子-κB

Effects of Linggui Zhugan Decoction on NF-κB in Rats with Ventricular Remodeling after Acute Myocardial Infarction

Abstract:Objective: To study the mechanism of Linggui Zhugan decoction (LGZGD)interference on ventricular remodeling (VR) in rats after acute myocardial infarction (AMI), through observing the expression of nuclear factor-κB(NF-κB)and NF-κB mRNA of myocardial tissue and the content of NF-κB in ventricular remodeling rats with AMI. **Method:** AMI model was produced by ligation of coronary artery. 2 weeks after modeling,rats were randomly classified into model, captopril, and low (2.1 g·kg^{-1}), middle(4.2 g·kg^{-1}) and high (8.4 g·kg^{-1})dosage of LGZGD group. Control group and the other 5 groups were administered medications intragastrically for 4 consecutive weeks. The content of NF-κB in myocardial tissue and serum was detected by ELISA and the expression of NF-κB was assayed by RT-PCR and the expression of NF-κB mRNA by Western blot. **Result:** For sham-operation, model, low, middle and large dosage of LGZGD group and captopril group, the content of NF-κB of myocardial tissue was 0.190±0.011,0.772±0.026,0.366±0.059,0.295±0.033,0.235±0.013,0.341±0.023.The relative expression of NF-κB mRNA were 1.000, 26.875, 6.574, 4.340, 1.194, 5.540.The content of serum NF-κB were (125.85±14.76),(196.98±17.79),(163.89±20.08),(131.73±10.47),(141.93±10.33),(133.93±9.27)ng·L^{-1}.The content of NF-κB in serum and the expression of NF-κB, NF-κB mRNA of myocardial tissue of model group increased significantly compared with control group (P<0.01). Low, middle and high dosage of LGZGD and captopril group could decrease the content of NF-κB in serum and inhibited the expression of NF-κB,NF-κB mRNA in myocardial tissue of model rats significantly compared with model group (P<0.01 or P<0.05). **Conclusion:** The mechanism of LGZGD interference VR of post-AMI seems to be related to the inhibition of NF-κB.

keywords:LGZGD myocardial infarction ventricular remodeling nuclear factor-κB

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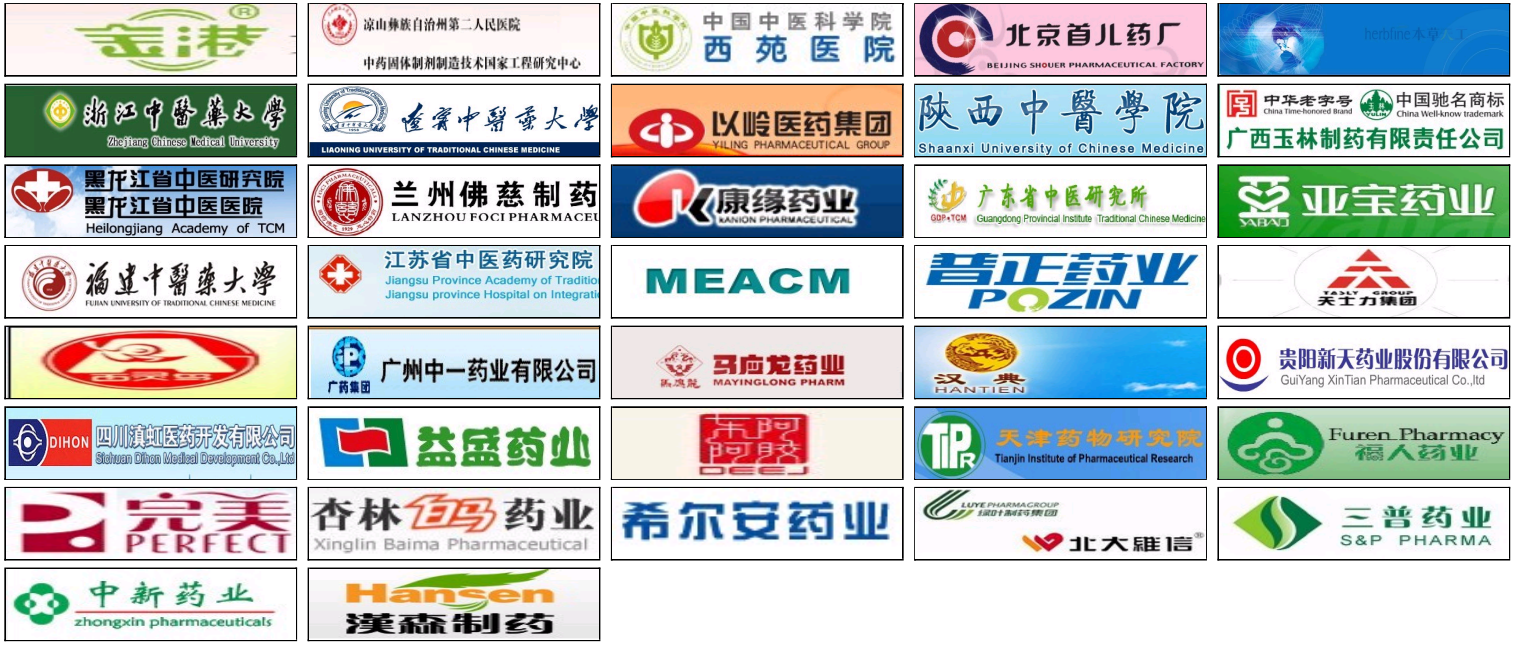

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