中国现代应用药学
Chinese Journal of Modern Applied Pharmacy
追踪在新药研发的一线
关注于药学应用的前沿

高夏青,薛凌.罗格列酮对兔心肌缺血再灌注损伤保护机制的研究[J].中国现代应用药学,2014,31(3):265-270

罗格列酮对兔心肌缺血再灌注损伤保护机制的研究

Study on the Protective Mechanisms of Rosiglitazone on Myocardial Ischemia Reperfusion Injury in Rabbits

投稿时间： 2013-08-06 最后修改时间： 2013-11-29

DOI：

中文关键词： 罗格列酮 PPAR-γ 心肌缺血再灌注损伤 炎症反应 氧化应激 心肌梗死面积

英文关键词：rosiglitazone PPAR-γ ischemia-reperfusion injury inflammation oxidative stress myocardial infarction size

基金项目:辽宁省科技厅项目(2011225015)

作者	单位	E-mail
高夏青	辽宁医学院，辽宁 锦州 121000	gaoxiaqing@ymail.com
薛凌*	辽宁医学院附属第三医院，辽宁 锦州 121000	xueling27@sohu.com

中文摘要:

目的 通过研究过氧化物酶体增殖物激活受体γ(PPAR-γ)激动剂对心肌缺血-再灌注损伤兔模型梗死面积及组织病理学改变的影响，探讨PPAR-γ介导心脏保护作用的可能机制。方法 新西兰白兔40只随机分为5组，包括：假手术组、模型组、罗格列酮低剂量组、罗格列酮高剂量组及罗格列酮高剂量+GW9622组。假手术组只开胸穿线不结扎心脏，其余组兔冠状动脉左前降支结扎30 min，再灌注120 min。建立心肌缺血再灌注模型后1 h分别检测血清一氧化氮(NO)、超氧化物歧化酶(T-SOD)浓度；ELISA法检测血清炎症因子白介素-6(IL-6)浓度；术后取兔心脏，硝基四氮唑蓝(NBT)染色后评价心肌梗死面积；做HE染色切片及电镜片观察组织及超微结构改变。结果 与模型组和罗格列酮高剂量+GW9622组相比，罗格列酮低剂量组、罗格列酮高剂量组血清NO含量降低，IL-6浓度显著降低，血清T-SOD浓度显著升高(P<0.01)；心肌梗死面积明显下降，心肌病理结构改变较轻，心肌细胞超微结构损坏破坏程度也明显减轻。结论 罗格列酮通过提高血清T-SOD水平减轻氧化应激，抑制炎症因子IL-6的生成减轻炎症反应，起到减轻心肌损伤，降低心肌梗死面积的保护作用。

英文摘要:

OBJECTIVE To explore the mechanisms and protective effect of peroxisome proliferators-activated receptor-γ (PPAR-γ) agonist on myocardial ischemia reperfusion(I/R) injury rabbits by observation the size of myocardial infarction and the histopathological changes. METHODS Forty New Zealand white rabbits were randomly divided into five groups(n=8): sham group, model group, low dose of rosiglitazone group, high dose of rosiglitazone group and high dose of rosiglitazone+GW9622 group. Thoracotomy and threading of the coronary artery without ligation was performed in sham group, whereas the coronary artery was ligated for 30 min, under 120 min

reperfusion in othe groups. Plasma concentration of NO, T-SOD and serum inflammatory cytokines IL-6 were tested after 1 h of I/R. The heart were harvested after I/R for pathological and ultrastructure analysis, and the areas of myocardial infarction were assessed. RESULTS Compared with model group and high dose of rosiglitazone+GW9622 group, in low dose of rosiglitazone group and high dose of rosiglitazon group, the plasma concentration of NO and IL-6 decreased, T-SOD significantly increased; the size of myocardial infarct decreased, and myocardial pathological and ultrastructure changes significantly improved. CONCLUSION The results indicate that PPAR-γ agonist of rosiglitazone can attenuate myocardial ischemia reperfusion injury, improve myocardial pathological and ultrastructure, reduce infarct size by mitigating oxidative stress and reducing the inflammatory response.