Expression of Mipul in Response to Myocardial Infarction in Mice

Abstract

Myocardial ischemic preconditioning over- regulated protein 1(Mipu1) was reproduced in our laboratory. Manly Wistar rats were subordinated to left anterior coronary roadway ligation and sham- operation and offered at 1 h, 3 h, 6 h, 12 h, 24 h, 3 d or 5 d after ligation. Expression of Mipu1 mRNA and protein were assessed by Northern blotting, real- time quantitative RT- PCR, In Situ hybridization and Western blotting. Expression of Mipu1 was over- regulated at 3 h and lasted to 12 h with a peak at 6h. Mipu1 mRNA and protein signals express in the endothelium and myocardium in normal and infarcted heart, substantially in infarcted zone. Fluorescent immunocytochemistry showed that Mipu1 protein was localized to the capitals of H9c2 myogenic cells and was up regulated after the cells being exposed toH2O2. These compliances indicates that Mipu1 may play a part in maintaining vascular homeostasis and guarding the myogenic cells from being injured by ischemia-reperfusion or oxidation stress.

Keywords: Mipu1 • Myocardial ischemia • Gene expression • Real- time RT- PCR

Introduction

Acute myocardial infarction (MI) is a leading cause of death and disability in the World. Left ventricular (LV) blowup constantly develops after MI. Myocardial loss as a consequence of infarction initiates a vicious cycle of contractile dysfunction and progressive LV dilation, appertained to as ventricular re modeling. Ventricular re modeling is also affected by the infarct size, which can be limited by opening the infarct-related roadway (i.e., reperfusion and arterial patency) or by the conformation and development of collateral vessels. The development of collateral vessels, nominated angiogenesis after MI, has salutary goods on the infarct mending, and angio genic growth factors are considered to have places in MI. lately, a number of experimental studies have suggested that treatment with angiogenic growth factors can promote the development of collaterals in beast MI models, similar as vascular end othelial growth factor (VEGF), an end othelial cell mitogen that 's allowed to serve in the contract of the conin angiogenesis [1]. During myocardial ischemia or reperfusion numerous genes similar as c- fos, c- jun, junB, Egr- 1, HSP70 can be over- regulated, and some of these myocardial genes have been considered to be involved in the endogenous cardio protection against myocardial ischemia-reperfusion injury. Accoutrements and styles Myocardial infarction was produced by ligation of the left anterior descending coronary roadway (LAD). All protocols involving experimental creatures followed the original institutional guidelines for beast care, which are similar to "Guide for the Care and Use of Laboratory creatures " published by the Institute for Laboratory Animal Research(National Institutes of Health publication No. 85-23, revised 1996). Adult manly Wistar rats importing 350 \pm 500 g were anesthetized using medetomidine, 0.5 mg/kg subcutaneously (s.c.), and ketamine 70 ± 80 mg/kg intra peritoneally. The rats were connected to a respirator through a tracheotomy and the heart was fleetly exterior side through a left thoracotomy and pericardial gash. The coronary roadway was ligated about 3 mm from its origin, the heart was returned

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Discussion

Dion verified that individual zinc cutlet (ZF) sphere recognizes DNA triumvirates with high particularity and affinity. By fusing ZF to suppression or activation disciplines, genes can be widely targeted and switched off and on. The family of Kruppel- suchlike proteins is one of the largest families of zinc- cutlet proteins. These proteins contain two or further C2H2- type zinc- fritters that are separated by a conserved agreement sequence, T/ SGEKPY/ FX. Members of the Kruppel- suchlike zinc cutlet family can serve as activators or repressors of gene recap and regulate embryonic development as well as a variety of physiological processes. lately, studies fastening on C2H2 type zinc cutlet genes have suggested their unique involvement in the regulation of embryogenesis and in a variety of conditions [3].

Conclusions

Our study demonstrates that the expression of Mipu1 mRNA and protein is convinced to be over- regulated after myocardial infarction substantially in the infarcted area and some extent in the remote non infarcted myocardium, suggesting that Mipu1 may be important for the survival and ultimate mending of cardiac structures after experimental myocardial infarction, which is merited to be linked further [4].

Results

The dynamic expression of mRNA of Mipu1

in the course of MI was detected by Northern spot analysis. A appreciatively hybridized band at 3396 bp was easily observed as a single band. Mipu1 was convinced to be over- regulated at 3 h after induction of MI, and achieved to the peak at 6h, and Int.J. Mol. Sci. 2009, 10 498 also decline, at 12 h it still express advanced than control. At after stages (24 h, 3 d, 5 d after MI), Mipu1 mRNA wasn't explosively expressed as compared to the normal heart. Northern spot analysis for Mipu1 mRNA in an infarct heart GAPDH bands are shown below [5]. To examine the change in the position of Mipu1 mRNA in the early phase of MI, aga rose gel electrophoresis was used to show the results of the samples at each time point anatomized by RT- PCR, of which GAPDH was used as the internal control, also quantitative real-time RT-PCR.

Acknowledgement

None

Conflict of Interest

No conflict of interest

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