[1]刘红梅,陈新,孙丹云,等.miR-199a-5p在血管紧张素Ⅱ致心肌肥大细胞中的表达变化[J].第三军医大学学报,2014,36(10):1017-1020.
Liu Hongmei,Chen Xin,Sun Danyun,et al.Alteration of miR-199a-5p expression in cardiomyocytes hypertrophy induced by angiotensin Ⅱ in rats[J].J Third Mil Med Univ,2014,36(10):1017-1020.

miR-199a-5p在血管紧张素Ⅱ致心肌肥大细胞中的表达变化

《第三军医大学学报》[ISSN:1000-5404/CN:51-1095/R] 卷: 36 期数: 2014年第10期 页码: 1017-1020 栏目: 论著 出版日期: 2014-05-30

Title: Alteration of miR-199a-5p expression in cardiomyocytes hypertrophy induced by angiotensin Ⅱ in rats

作者: 刘红梅; 陈新; 孙丹云; 唐渊; 李晓辉
第三军医大学药学系药物研究所暨药剂学教研室

Author(s): Liu Hongmei; Chen Xin; Sun Danyun; Tang Yuan; Li Xiaohui
Institute of Materia Medica, College of Pharmacy, Third Military Medical University, Chongqing, 400038, China

关键词: 腹主动脉缩窄; 心肌肥大; 血管紧张素Ⅱ; 心肌细胞; miR-199a-5p

Keywords: transverse aortic coarctation; myocardial hypertrophy; angiotensin Ⅱ; cardiac myocytes; miR-199a-5p

分类号: R363.21; R542.202; R977.6

文献标志码: A

摘要: 目的 研究miR-199a-5p在血管紧张素Ⅱ（Ang Ⅱ）导致肥大心肌细胞中的表达变化。 方法 雄性SD大鼠30只，采用完全随机方法将其分为假手术组、模型组、氯沙坦组[10 mg/（kg·d）]，每组10只。制备腹主动脉缩窄致心肌肥大模型，在术后第28天，检测心脏质量指数和左心室质量指数，HE染色观察左心室心肌细胞肥大情况，qRT-PCR方法测定左心室miR-199a-5p表达。取10只1~3 d SD乳鼠，消化获得心肌细胞，分为对照组、模型组（1×10^{-7} mol/L Ang Ⅱ）和氯沙坦组（1×10^{-7} mol/L Ang Ⅱ+1×10^{-6}mol/L氯沙坦钾），qRT-PCR方法测定心肌细胞miR-199a-5p表达；分别转染miR-199a-5p 模拟物或者抑制物，观察[^{3}H]-亮氨酸的掺入情况。 结果 腹主动脉缩窄大鼠左心室心肌细胞明显肥大，左心室miR-199a-5p表达显著升高（$P<0.05$），Ang Ⅱ受体拮抗剂氯沙坦可显著逆转上述变化。体外实验发现氯沙坦可显著降低Ang Ⅱ刺激致乳鼠心肌细胞所致的miR-199a-5p显著升高（$P<0.05$），采用转染miR-199a-5p抑制物的方法抑制miR-199a-5p表达可显著抑制Ang Ⅱ刺激所致的心肌细胞[^{3}H]-亮氨酸掺入（$P<0.01$）；且转染miR-199a-5p 模拟物增加miR-199a-5p表达可显著增加[^{3}H]-亮氨酸掺入（$P<0.01$）。 结论 miR-199a-5p是介导Ang Ⅱ致心肌细胞肥大的重要调节分子，可能在该信号通路中发挥重要作用。

Abstract: Objective To investigate the alteration of miR-199a-5p expression in cardiomyocyte hypertrophy induced by angiotensin Ⅱ (Ang Ⅱ). Methods The rat model of cardiac hypertrophy was established by transverse aortic coarctation. Thirty SD rats were randomly divided into 3 groups, sham group,

model group, and losartan group (n=10 for each group). The losartan group was injected intraperitoneally with losartan potassium 10 mg/(kg • d). The sham group and model group were treated with the vehicle. On day 28, the cardiac mass index (CMI) and left ventricular mass index (LVMI) were measured. The myocardial tissue morphologic changes were detected by hematoxylin-eosin staining. Quantitative RT-PCR was performed to analyze the expression level of miR-199a-5p. Neonatal rat cardiac myocytes treated by Ang Ⅱ were exposed to losartan, and miR-199a-5p expression level was detected by quantitative RT-PCR. MiR-199a-5p mimic and inhibitor were used to transfect neonatal rat cardiac myocytes, and cardiomyocyte hypertrophy was assessed by [^{3}H]-leucine incorporation. Results The cardiac myocytes were hypertrophic and the expression level of miR-199a-5p was significantly increased in left ventricle induced by transverse aortic coarctation (P<0.05). Ang Ⅱ receptor antagonist losartan significantly reversed the changes. Losartan significantly decreased the expression level of miR-199a-5p in neonatal rat cardiac myocytes induced by Ang Ⅱ (P<0.05). MiR-199a-5p inhibitor significantly decreased [^{3}H]-leucine incorporation in neonatal rat cardiac myocytes induced by Ang Ⅱ (P<0.01), while miR-199a-5p mimic significantly enhanced the incorporation (P<0.01). Conclusion MiR-199a-5p is an important regulator in myocyte hypertrophy induced by Ang Ⅱ, and may play a significant role in myocyte hypertrophy signaling pathways induced by Ang Ⅱ.

参考文献/REFERENCES:

刘红梅, 陈新, 孙丹云, 等. miR-199a-5p在血管紧张素Ⅱ致心肌肥大细胞中的表达变化[J].第三军医大学学报,2014,36(10):1017-1020.

相似文献/REFERENCES:

[1]金丰,李隆贵,耿召华,等.大鼠压力超负荷心肌糖蛋白130表达及贝那普利干预研究[J].第三军医大学学报,2006,28(20):2027.
[2]杨永曜,李隆贵,吴强,等.过氧化物酶体增殖激素型受体对Ang Ⅱ诱导肥厚心肌细胞的影响[J].第三军医大学学报,2006,28(18):1840.
[3]金丰,李隆贵,张钰倩,等.压力超负荷大鼠心肌营养素-1表达对贝那普利干预的反应[J].第三军医大学学报,2006,28(18):1864.
[4]刘伟,何作云,冯兵,等.PDH调控的糖代谢途径对缺氧后复氧的大鼠肥大心肌细胞凋亡的影响[J].第三军医大学学报,2005,27(02):126.