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基础医学

NAD对AngII诱导的心肌成纤维细胞I型胶原mRNA表达的影响

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摘要：

目的 探讨烟酰胺腺嘌呤二核苷酸(NAD) 对血管紧张素II（AngII)诱导的大鼠心肌成纤维细胞I型胶原mRNA表达的作用。方法 提取新生Wistar大乳鼠原代心肌成纤维细胞，传代培养，采用2~4代细胞。实验分为空白对照组，AngII (0.01、 0.1、 1μmol／L)组，NAD(250μmol／L)组, AngII(1μmol／L)+ NAD (250μmol／L)组，FAM组，Sirt1-siRNA+AngII(1μmol／L)组，Sirt1-siRNA 组，Sirt1-siRNA+ NAD（250μmol／L）组，Sirt1-siRNA+Ang II(1mol／L)+NAD（250μmol／L）组。刺激24h后，提取总RNA, Real time RT-PCR法分别检测SIRT1和I型胶原mRNA的表达。结果 心肌成纤维细胞I型胶原mRNA的表达随着AngⅡ浓度升高明显增加(P＜0.05)，呈浓度依赖性。与空白对照组比较，NAD（250μmol／L）组SIRT1 mRNA表达增高(P＜0.05)。与AngⅡ（1μmol／L）组比较，AngII (1μmol／L)+ NAD(250μmol／L)组I型胶原mRNA的表达明显减少(P＜0.01)。相对于FAM组，Sirt1-siRNA转染后各组SIRT1 mRNA的表达减少(P＜0.05)，而心肌成纤维细胞I型胶原mRNA表达增多(P＜0.05)。结论 NAD可以抑制心肌成纤维细胞 I型胶原mRNA的表达，SIRT1影响I型胶原mRNA的表达，它们对Ang II诱导的心肌纤维化有保护作用。

关键词： 烟酰胺腺嘌呤二核苷酸; I型胶原；血管紧张素II; 纤维化；心肌重构；SIRT1

Effects of NAD on the mRNA expression of collagen type I induced by Angiotensin II in cardiac fibroblasts

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Abstract:

Objective To explore the effect of NAD on the mRNA expression of collagen type I induced by angiotensin II in cardiac fibroblasts in vitro. Methods Neonatal rat cardiac fibroblasts were isolated from wistar rats and then cultured. The generation 2-4 were used for the experiment and treated as follows: control group, Ang II (0.01， 0.1， 1μmol／L) group，NAD (250μmol／L)group, Ang II (1μmol／L)+ NAD (250μmol／L)group, FAM group, Sirt1-siRNA+AngII (1μmol／L) group, Sirt1-siRNA group, Sirt1-siRNA+NAD（250μmol／L）group and Sirt1-siRNA+ Ang II (1μmol／L)+NAD（250μmol／L）group. 24 hours later, the mRNA expressions of SIRT1 and collagen I were measured by real time reverse transcription-polymerase chain reaction(Real time RT-PCR). Results After treatment with Ang II for 24 hours，the mRNA expression of collagen I was enhanced in a Ang II dose-dependent manner(P＜0.05). In contrast with control group, the mRNA expression of SIRT1 was enhanced after being treated with NAD（250μmol／L）(P＜0.05) and the mRNA expression of collagen I in AngII (1μmol／L)+ NAD (250μmol／L) group was attenuated compared with Ang II (1μmol／L) group (P＜0.01). Compared with FAM group, SIRT1 mRNA expression was attenuated(P＜0.05) and collagen I mRNA expression was increased (P＜0.05) in the other groups. Conclusion The results suggest that NAD and SIRT1 may be beneficial to cardiac fibrosis by attenuating the mRNA expression of collagen type I induced by Ang II in cardiac fibroblatsts.

Keywords: Nicotinamide adenine dinucleotide; Collagen I; Angiotensin II; Fibrosis; Cardiac remodeling; SIRT1

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