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ERK1/2介导去甲肾上腺素对内皮祖细胞的功能调节

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摘要：

目的 探讨去甲肾上腺素(NE)对内皮祖细胞(EPCs)增殖和迁移能力的调节作用及其分子机制。 方法 将培养的健康成人外周血EPCs用不同浓度的NE、肾上腺素能受体拮抗剂或MAPK信号通道阻滞剂干预,检测EPCs的增殖和迁移能力,以及ERK1/2信号通路的激活情况。结果 NE浓度依赖性地(0.01、0.1、1、10 μmol/L)促进EPCs增殖,与对照组比较 EPCs分别增加(48.3±23.3)%、(70.5±35.6)%、(82.4±14.9)%和(100.3±48.1)%。α受体拮抗剂酚妥拉明、选择性β2肾上腺能受体拮抗剂I127、JNK抑制剂SP600125和ERK1/2 抑制剂A6355能够阻断NE的促增殖作用；而β1受体拮抗剂美托洛尔和p38抑制剂PD169318不能阻断NE的刺激效应。10 μmol/L NE促进EPCs的迁移(P<0.05),10 μmol/L酚妥拉明和10 μmol/L I127能够阻断这种作用,但美托洛尔不能。NE能够浓度依赖性(0.1、1、10 μmol/L)地激活EPCs 内的ERK1/2,酚妥拉明和I127能够阻断ERK1/2激活(P<0.05),而美托洛尔不能。结论 NE可能通过α和β2肾上腺能受体激活ERK1/2促进EPCs的迁移和增殖。

关键词：内皮祖细胞 去甲肾上腺素 α受体拮抗剂 β受体拮抗剂 细胞增殖 细胞迁移 ERK1/2

ERK1/2 pathway-mediated norepinephrine regulates function of endothelial progenitor cells

Fulltext

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Abstract:

Objective To analyze the effects of norepinephrine(NE) on the proliferation and migration of endothelial progenitor cells (EPCs) and the related mechanism. Methods NE,adrenoceptor antagonist, and MAPK signal pathway blocker of various concentrations were used to treat peripheral EPCs derived from healthy adults. The proliferation potential, migration capacity and activation of ERK1/2 were assessed after different treatments. Results NE increased the proliferation potential of EPCs in a dose-dependent manner. The number of EPCs increased by (48.3±23.3)%, (70.5±35.6)%, (82.4±14.9)% and (100.3±48.1)% after treatment with NE at 0.01 μmol/L, 0.1 μmol/L, 1 μmol/L and 10 μmol/L, respectively. Addition of alpha adrenoceptor antagonist phentolamine, selective beta 2 adrenoceptor antagonist I127, JNK blocker SP600125 and ERK1/2 blocker A6355 could block the above effects of NE, and beta 1 adrenoceptor antagonist metoprolol and p38 blocker PD169318 failed to block the effects of NE. NE at 10 μmol/L significantly promoted the migration of EPCs (P<0.05). These effects could be blocked by addition of phentolamine (10 μmol/L) and I127 (10 μmol/L), but not by addition of metoprolol. NE(0.1, 1 and 10 μmol/L) activated ERK1/2 pathway in a dose-dependent manner, which could also be blocked by phentolamine and I127, but not by metoprolol. Conclusion NE can increase the proliferation potential and migration capacity of EPCs via activating ERK1/2 pathway with alpha and beta 2 adrenoceptor.

Keywords:endothelial progenitor cells norepinephrine alpha adrenoceptor antagonist beat adrenoceptor

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