[1]成彩联,郑振达,石成钢,等.晚期糖基化终产物对足细胞内肾素-血管紧张素系统的影响[J].第三军医大学学报,2013,35(17):1779-1782.
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晚期糖基化终产物对足细胞内肾素-血管紧张素系统的影响

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Title: Effect of advanced glycation end products on renin-angiotensin system in podocytes

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关键词: 晚期糖基化终产物; 磷酸肌醇3激酶; 足细胞; 肾素-血管紧张素系统

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摘要: 目的 观察晚期糖基化终产物（advanced glycation end products, AGEs）对足细胞内肾素-血管紧张素系统（renin-angiotensin system, RAS）的影响及作用机制。方法 不同浓度的AGEs干预小鼠足细胞24 h，分别检测肾素(renin)、血管紧张素原(renin-angiotensin system, AGT)、血管紧张素Ⅱ 1型、2型受体(AT1R、AT2R)的表达，血管紧张素转换酶(angiotensin-converting enzyme, ACE)的活性和血管紧张素Ⅱ(angiotensin Ⅱ, Ang Ⅱ)的浓度，观察蛋白激酶B(Akt)的磷酸化，然后分别加入磷酸肌醇3激酶抑制剂LY294002、losartan、captopril和chymastatin，观察足细胞粘附性的变化。 结果 与对照组相比，AGEs（80 μg/mL）明显上调AGT和AT1R的表达[(183.0±19.0)% *vs* 100%，(179.0±17.0)% *vs* 100%，*P*<0.05]，裂解液中ACE活性明显增加[（142.8±10.3）U/μg *vs* （85.0±9.2）U/μg，*P*<0.05]，细胞上清中Ang Ⅱ的浓度明显增加[(11.2±0.8) pg/mL *vs* (7.0±0.7) pg/mL，*P*<0.05]；Akt的磷酸化上调100%(*P*<0.05)，而LY294002可减轻AGEs介导的足细胞内RAS的激活；与AGEs组相比，LY294002可改善AGEs介导的足细胞粘附性的下降[（82±13）% *vs* (40±12)%；(78±14)% *vs*(42±13)%，*P*<0.05]。 结论 AGEs可能通过磷酸肌醇3激酶途径激活足细胞内的RAS，降低足细胞粘附性。

Abstract: Objective To investigate the effect and mechanism of advanced glycation end products (AGEs) on the components of renin-angiotensin system (RAS) in the podocytes. Methods Immortalized mouse podocytes were exposed to

various concentrations of AGEs for 24 h. The expression levels of renin, angiotensinogen (AGT) and angiotensin Ⅱ type 1 and 2 receptors (AT1R and AT2R), the level of angiotensin Ⅱ (Ang Ⅱ), and the activity of angiotensin-converting enzyme (ACE) were assayed. The levels of Akt and phosphorylated Akt were examined by Western blotting. Cell adhesion was measured in the podocytes pretreated with phosphoinositide 3-kinase (PI3-K) inhibitor LY294002, losartan, captopril and chymostatin, respectively. Results Treatment with AGEs resulted in significant increase in the expression of AGT and AT1R. Moreover, ACE activity and Ang Ⅱ level increased significantly. However, there was no significant change in renin and AT2R expression. AGEs increased the phosphorylation of Akt by 100%. When the podocytes were pretreated with LY294002 (10 μmol/L), the AGEs-induced increase in AGT and AT1R expression reduced remarkably. Likewise, ACE activity and Ang Ⅱ level decreased significantly, and the reduced podocyte adhesive capacity induced by AGEs was improved significantly. Conclusions AGEs activate the RAS *via* PI3-K/Akt-dependent pathway, and lead to a decrease in podocyte adhesion.

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