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# 尾加压素Ⅱ与中介素在牛磺酸减轻油酸所致大鼠肺



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Title: Role of urotensin Ⅱ and intermedin in taurine alleviating oleic acid-induced acute lung injury in rats

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关键词: 油酸; 急性肺损伤; 尾加压素Ⅱ; 中介素; 牛磺酸

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摘要: 目的 研究牛磺酸对油酸所致急性肺损伤大鼠的干预作用及其通过影响尾加压素Ⅱ(UⅡ)及中介素(intermedin, IMD)合成与分泌发挥肺保护作用的机制。 方法 采用油酸性急性肺损伤大鼠模型。动物分为3组,对照组(C组)、油酸损伤组(A组)、牛磺酸治疗组(T组),分别于0、6、12、24 h 4个时间抽动脉血2 mL并取肺泡灌洗液(BALF),测动脉血氧分压[$p(O_2)$],血浆及BALF中UⅡ、IMD浓度,测定肺湿干质量比(W/D)并进行统计学分析;HE染色、免疫组化检查肺组织中UⅡ与IMD表达水平;电镜观察肺组织超微结构。 结果 T组$p(O_2)$在6、12、24 h时高于A组($P<0.05$);A组肺组织W/D在0 h和6 h高于T组($P<0.05$);A组血浆UⅡ在24 h高于T组($P<0.05$);A、T组BALF UⅡ在0、6 h差异显著($P<0.05$),0 h时T组高于A组($P<0.05$),6 h时A组高于T组($P<0.05$);A组血浆IMD在0 h时高于T组($P<0.05$)。光镜观察:T组肺组织损伤减轻。免疫组化染色结果显示各组大鼠支气管黏膜上皮细胞、平滑肌细胞,肺内血管内皮细胞、平滑肌细胞细胞膜及细胞质均有UⅡ与IMD阳性表达,A组与T组间表达无明显差异。电镜观察: T组较A组损伤减轻,基底膜完整,未见明显胶原增生。 结论 牛磺酸可减轻油酸致大鼠急性肺损伤,其机制可能与抑制UⅡ表达,减轻毛细血管基底膜损伤后肺泡渗出有关。

Abstract: Objective To determine the intervention effect of taurine on oleic acid-

induced acute lung injury (ALI) in rats and investigate its possible mechanisms, especially the role of synthesis and secretion of urotensin Ⅱ (UⅡ) and intermedin (IMD). Methods A total of 54 SD rats, at an age of 9 weeks, weighting 280 to 310 g, were randomly divided into 3 groups, control (*n*=6), the ALI group (*n*=24) and the taurine treatment group (*n*=24). Rat models of ALI were established by tail vein injection of 0.15 mL/kg oleic acid. Taurine solution was given intraperitoneally at a dose of 20 mg/mL, 0.1 mL/kg for 3 consecutive days before oleic acid injection. Blood sample of 2 mL were drawn from the arteries at 0, 6, 12, and 24 h after oleic acid injection, while bronchioalveolar lavage fluid (BALF) was taken at the same time points to measure arterial partial pressure oxygen [$p(O_2)$]. The plasma and BALF contents of UⅡ and IMD, and lung wet-to-dry ratio (W/D) were measured and counted. Immunohistochemical assay was used to detect the expression of UⅡ and IMD in lung tissues. Electron microscopy was used to observe the ultrastructure of lung tissues. Results $p(O_2)$ was significantly higher in the treatment group than in the model group at 6, 12 and 24 h (*P*<0.05). The W/D ratio was obviously higher in the model group than in the treatment group at 0 and 6 h (*P*<0.05). Plasma content of UⅡ was obviously higher in the model group than in the treatment group at 24 h (*P*<0.05). There were significant differences in BALF content of UⅡ between model group and treatment group at 0 and 6 h (*P*<0.05). It was remarkably higher in the treatment group than in the model group at 0 h (*P*<0.05), while higher in the model group than in the treatment group at 6 h (*P*<0.05). But for plasma content of IMD, it was obviously higher in the treatment group than in the model group at 0 h (*P*<0.05). Electron microscopy displayed that the injury of lung tissue was milder in the treatment group than the other groups. Immunohistochemical assay showed there were positive expression of UⅡ and IMD in the bronchial epithelial cells, smooth muscle cells, and pulmonary vascular endothelial cells in the 3 groups, and no significant difference was seen between the model group and treatment group. Electron microscopy indicated the injury was significantly milder in the treatment group than in the model group, with intact basement membrane and without obvious collagen hyperplasia. Conclusion Taurine alleviates the lung injury induced by oleic acid, which might be due to its inhibiting the expression of UⅡ and protecting the capillary basement membrane to reduce plasma protein exudation.

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