

论著

内毒素性急性肺损伤大鼠内源性H₂S/CSE体系的变化

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摘要 目的: 观察内毒素性急性肺损伤 (ALI) 大鼠内源性硫化氢/胱硫醚- γ -裂解酶 (H₂S/CSE) 体系、IL-1 β 和 IL-10 的动态变化。

方法: 健康雄性SD大鼠共80只, 随机分为 I (对照) 组; II (LPS 1 h) 组; III (LPS 3 h) 组; IV (LPS 6 h) 组; V (LPS 9 h) 组; VI (LPS 12 h) 组。给予LPS复制内毒素性ALI大鼠模型, 分别于1、3、6、9、12 h处死, 观察光镜和电镜下肺组织形态学改变, 检测肺系数、肺湿/干重比、血浆中H₂S含量、肺组织CSE活性、血清中IL-1 β 和IL-10的动态变化。

结果: (1)LPS 1 h组, 光镜和电镜下肺组织形态学无明显改变, 肺系数、肺湿/干重比、血浆中H₂S的含量和肺组织CSE活性与对照组比较无明显变化, 血清中IL-1 β 和IL-10含量明显高于对照组 (IL-1 β , $P < 0.05$; IL-10, $P < 0.01$)。 (2)LPS 3、6、9、12 h组, 光镜和电镜下肺组织明显受损, 超微结构明显改变, 肺系数和肺湿/干重比明显高于对照组 ($P < 0.05$ 或 $P < 0.01$), 血浆中H₂S的含量和肺组织CSE活性明显低于对照组 ($P < 0.05$ 或 $P < 0.01$), 血清中IL-1 β 和IL-10的含量明显高于对照组 ($P < 0.01$)。

结论: 内源性H₂S/CSE体系、IL- β 和IL-10参与内毒素性ALI的病理生理过程。

关键词 [脂多糖类](#); [急性肺损伤](#); [硫化氢](#); [白细胞介素1](#); [白细胞介素10](#)

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Change of endogenous hydrogen sulfide/cystathionine- γ -lyase system in acute lung injury induced by LPS in rats

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Abstract

AIM: To observe the changes of endogenous hydrogen sulfide/cystathionine- γ -lyase (H₂S/CSE) system while acute lung injury induced by LPS in rats.
METHODS: Eighty rats were randomly divided into six groups (n=8): I, control group; II, LPS 1 h group; III, LPS 3 h group; IV, LPS 6 h group; V, LPS 9 h group; VI, LPS 12 h group. The ALI model of rats was prepared with LPS. The rats were respectively killed at 1, 3, 6, 9 or 12 h after administration of LPS. The morphological changes of lung tissues were observed by light and electron microscope. The lung coefficient and the wet-to-dry weight ratio were measured. The contents of IL-1 β and IL-10 in serum, the H₂S level in plasma and the CSE activity in lung tissue were respectively detected.
RESULTS: (1) In LPS 1 h group, the morphology, the lung coefficient, the wet-to-dry weight ratio, the H₂S level and the CSE activity showed no changes compared with the control group. The contents of IL-1 β and IL-10 were increased compared with the control group (IL-1 β , $P < 0.05$; IL-10, $P < 0.01$). (2) In LPS 3 h, 6 h, 9 h and 12 h groups, compared with the control group, the lung tissues were significantly damaged, the lung coefficient and the wet-to-dry weight ratio were significantly increased respectively (LPS 3 h, $P < 0.05$; LPS 6 h, 9 h, 12 h, $P < 0.01$). The contents of IL-1 β and IL-10 in serum were markedly increased ($P < 0.01$). The H₂S level in plasma and the CSE activity in lung tissue were significantly decreased ($P < 0.01$).
CONCLUSION: The changes of inflammatory cytokines may be the pathological foundation of the ALI induced by LPS and the endogenous hydrogen sulfide/cystathionine- γ -lyase system is possibly involved in the formation of the ALI.

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