

[本期目录](#) | [下期目录](#) | [过刊浏览](#) | [高级检索](#)[\[打印本页\]](#) [\[关闭\]](#)**论文****乌司他丁对脂多糖致小鼠急性肺损伤的保护作用以及与诱导型一氧化氮合酶和c-Jun表达的关系**谭正怀·余凌虹·魏怀玲·刘耕陶<sup>1</sup>

1. 中国医学科学院、中国协和医科大学 药物研究所, 北京 100050; 2. 四川省中药研究所, 四川 成都 610041

**摘要:**

目的探讨乌司他丁对脂多糖(LPS)致小鼠急性肺损伤的作用及其机制。方法小鼠腹腔注射乌司他丁(50和100  $\text{ku} \cdot \text{kg}^{-1}$ )或等体积生理盐水30 min后, 分别静脉注射LPS 15  $\text{mg} \cdot \text{kg}^{-1}$ 或等体积生理盐水, 于注射LPS后不同时间检测有关各项指标。ELISA法测定血清和肺组织中TNF $\alpha$ 水平, RT-PCR法测定TNF $\alpha$  mRNA和iNOS mRNA的表达。Western blotting法检测c-Fos, c-Jun及iNOS等蛋白表达。结果乌司他丁100  $\text{ku} \cdot \text{kg}^{-1}$ 能显著降低LPS引起的小鼠的肺指数、肺组织及血清中NO水平的增加, 下调肺组织c-Jun蛋白表达量和iNOS mRNA及其蛋白的表达量, 而对小鼠的血清和肺组织冲洗液中TNF $\alpha$ 含量以及肺组织MDA无明显影响。结论乌司他丁对LPS引起的小鼠肺损伤有保护作用, 该作用与其抑制c-Jun蛋白和iNOS mRNA的表达有关。

关键词: 脂多糖 急性肺损伤 乌司他丁 iNOS c-Jun

**Protective action of ulinastatin against lipopolysaccharides-induced acute lung injury in mice and the relation of it to iNOS and c-Jun expressions**

TAN Zheng-huai; YU Ling-hong; WEI Huai-ling; LIU Geng-tao

**Abstract:**

**Aim** To study the protective action of ulinastatin against lipopolysaccharide (LPS)-induced acute lung injury in mice and the mechanism of its action. **Methods** Mice were intraperitoneally injected with ulinastatin (50 and 100  $\text{ku} \cdot \text{kg}^{-1}$ ) or saline at a period of 12 h, separately, 30 min after the last injection of ulinastatin, except normal control, all mice of other groups were injected a dose of LPS 15  $\text{mg} \cdot \text{kg}^{-1}$  via tail vein. The levels of TNF $\alpha$  in serum and lung were measured by ELISA. The expression of TNF $\alpha$  mRNA and iNOS mRNA in lung was assayed by RT-PCR. The expression of c-Fos and c-Jun protein in lung was measured by Western blotting method. And the  $\text{NO}_2^-/\text{NO}_3^-$  level in serum and MDA in lung were measured with kits. **Results** The levels of  $\text{NO}_2^-/\text{NO}_3^-$  and TNF $\alpha$  in serum, MDA and TNF $\alpha$  in lung all increased after iv injection of LPS. The expressions of TNF $\alpha$  mRNA, iNOS mRNA, c-Fos and c-Jun in lung of LPS-injected mice were enhanced. Pretreatment with ulinastatin 100  $\text{ku} \cdot \text{kg}^{-1}$  decreased the levels of  $\text{NO}_2^-/\text{NO}_3^-$  in serum and lung, reduced the index of lung, and inhibited the expressions of iNOS mRNA and c-Jun in lung induced by LPS in mice, while ulinastatin showed no effect on TNF $\alpha$  level in serum and lung. **Conclusion** Ulinastatin protected mice from acute lung injury induced by lipopolysaccharides via inhibiting the activation of c-Jun and iNOS mRNA expression.

Keywords: acute lung injury ulinastatin iNOS c-Jun lipopolysaccharides

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作者简介:

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