

论著

心房钠尿肽对肺泡 II 型上皮细胞的保护作用

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摘要 目的: 探讨心房钠尿肽(ANP)对脂多糖(LPS)引起肺泡 II 型上皮细胞(AT-II)损伤的治疗作用。方法: 分离培养 AT-II, 以 LPS 复制大鼠 AT-II 损伤模型, 分别给予 10-6、10-7、10-8 mol/L 等不同剂量的 ANP 进行治疗, 通过观察 4 h、12 h、24 h 等时点细胞培养上清液中 LDH、MDA、AKP、总磷脂(TPL)水平及细胞培养上清液的表面张力(ST)变化, 研究 ANP 对 LPS 引起的 AT-II 损伤的治疗作用。结果: 在不同剂量、不同时间条件下, 各 ANP 组细胞培养上清液 LDH、AKP 活性及 MDA 含量均明显低于 LPS 组, 呈明显的剂量依赖性和时间依赖性, 以高剂量组(10-6)和 12 h 时点疗效最佳。以 12 h 时点为例, 细胞培养上清液中 AKP 活性为: control (43.5±10.4) U/L, LPS (98.1±16.4) U/L, LPS+ANP(10-6) (46.4±10.5) U/L, LPS+ANP(10-7) (60.7±9.5) U/L, LPS+ANP(10-8) (91.3±13.9) U/L。LPS 组细胞培养上清液中 TPL 含量明显低于对照组, ST 水平明显高于对照组, 不同剂量、不同时间点的各 ANP 组细胞培养上清液中 TPL 含量均不同程度地高于对应的 LPS 组, 各 ANP 组细胞培养上清液中的 ST 水平均低于对应的 LPS 组。结论: ANP 可显著减轻 LPS 引起的 AT-II 损伤, 促进肺表面活性物质(PS)的合成、分泌, 且该作用有明显的剂量依赖性和时间依赖性。

关键词 [心钠素](#) [呼吸窘迫综合征](#) [肺泡](#)

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Protective effect of atrial natriuretic peptide on alveolar type II cells

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Abstract

AIM: To study the protective effect of atrial natriuretic peptide (ANP) on alveolar type II cells (AT-II) damaged by lipopolysaccharide (LPS).
METHODS: AT-II were placed in a 6 well cell culture cluster (0.5×10⁶ cells/cm²) and divided into 3 groups: (1) control group (n=6), the medium consisted of RPMI-1640 without FBS. (2) LPS group (n=6), the medium consisted of RPMI-1640 without FBS supplemented with LPS (1 mg/L). (3) ANP group (n=6), the medium consisted of RPMI-1640 without FBS supplemented with LPS (1 mg/L) and ANP (10-8, 10-7, 10-6 mol/L). After 4, 12 and 24 h, the cell culture mediums of control group, LPS group and ANP (10-7 mol/L) group were collected, and those of the ANP (10-6, 10-8 mol/L) group were collected after 12 h. Alkaline phosphatase (AKP), lactate dehydrogenase(LDH), malondialdehyde(MDA), total phospholipids (TPL) and surface tension (ST) in the medium of every group were examined.
RESULTS: AT-II were characterized by AKP staining. The contents of LDH, AKP and MDA in the medium of every ANP group were lower than those in the corresponding LPS group. The TPL content in the medium of every ANP group was higher than that in the corresponding LPS group, and the change of ST of the medium was opposite to that of TPL. The effect at 12 h was the most significant, for example, at 12 h, the activities of AKP in the mediums were: control (43.5±10.4) U/L, LPS (98.1±16.4) U/L, LPS+ANP (10-6) (46.4±10.5) U/L, LPS+ANP(10-7) (60.7±9.5) U/L, LPS+ANP(10-8) (91.3±13.9) U/L.
CONCLUSION: ANP protects the AT-II from being damaged by LPS and promotes the secretion of pulmonary surfactants.

Key words [Atrial natriuretic factor](#) [Respiratory distress syndrome](#) [Pulmonary alveoli](#)

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