论著

芬太尼对人外周血NF-KB活性的影响

饶 艳 1 , 王焱林 1* , 李建国 1 , 段军民 2

(1. 武汉大学中南医院麻醉科, 湖北 武汉 430071; 2. 武汉癫痫病医院, 湖北 武汉 430035)

收稿日期 2002-11-6 修回日期 网络版发布日期 2008-10-17 接受日期 2003-1-15

摘要 目的 验证芬太尼是否影响免疫炎症反应中的某些因素,如同吗啡。方法 外周血取自7个正常志愿者,实验分为正常对照组、芬太尼(20 μ g · L⁻¹和2 μ g · L⁻¹)组、模型组(脂多糖,LPS组)和治疗组(芬太尼20 μ g · L⁻¹+LPS、芬太尼2 μ g · L⁻¹+LPS)。用流式细胞术检测人外周血中性粒细胞和单核细胞中核因子(NF- μ 8)活性,用ELISA检测血清中肿瘤坏死因子- μ 6(TNF- μ 7)和白介素-6(IL-6)含量。结果 芬太尼组NF- μ 8局活性及TNF- μ 7和IL-6含量与正常对照组比较,均无明显差异(μ 8)0.05)。治疗组(芬太尼20 μ 9 · L⁻¹+LPS、芬太尼2 μ 9 · L⁻¹+LPS)中NF- μ 8的活性分别为81.9%,76.1%(中性粒细胞)和78.6%,72.6%(单核细胞),明显低于模型组88.9%和85.1%(μ 80.01)。TNF- μ 8含量在治疗组(芬太尼20 μ 9 · L⁻¹+LPS、芬太尼2 μ 9 · L⁻¹+LPS)为459和357 μ 9 · L⁻¹,IL-6为796和720 μ 9 · L⁻¹,两者均低于模型组(其中TNF- μ 8为226 μ 9 · L⁻¹,IL-6为1563 μ 9 · L⁻¹)(μ 80.01)。结论 芬太尼对NF- μ 8的活性及TNF- μ 9和IL-6的含量无影响,但可抑制LPS诱导的NF- μ 8的活性及TNF- μ 9和IL-6的含量,且高剂量芬太尼的抑制作用大于低剂量芬太尼的作用。

关键词 <u>芬太尼</u> <u>核因子-κB</u> <u>肿瘤坏死因子</u> <u>白介素-6</u> 分类号 <u>R971.2</u>

Effects of fentanyl on activation nuclear factor-kappaB in human whole blood

RAO Yan¹, WANG Yan-Lin¹, LI Jian-Guo¹, DUAN Jun-Min²

(1. Department of Anesthesiology, Zhongnan Hospital, Wuhan University, Wuhan 430071, China; 2. Wuhan Epilepsy Hospital, Wuhan 430035, China)

Abstract

AIM To clarify if fentanyl affects some factors in immunity and inflammation as morphine did. METHODS Blood samples were collected from 7 healthy volunteers. Each was divided into 6 parts: normal control, fentanyl(20 μg·L⁻¹ or 2 mg·L⁻¹) control, lipopolysaccharide(LPS) alone and fentanyl 20 μg·L⁻¹ or 2 mg·L⁻¹+LPS. The nuclear factor- kappaB(NF-κB) activation in human neutrophils and monocytes was examined by flow cytometric analysis, the plasma tumor necrosis factorα(TNF-α) and interleukin-6(IL-6) concentrations were measured using ELISA. RESULTS The low NF- κB activation (2.8% –4.0%) and TNF-α and IL- 6 production was no significant difference in normal control and fentanyl control(*P*>0.05). The NF-κB activation in treatment groups(fentanyl 20 μg·L⁻¹+LPS, fentanyl 2 mg·L⁻¹+LPS) were 81.9% and 76.1% in neutrophils and 78.6% and 72.6% in monocytes, respectively, which were less than those in LPS alone group (88.9% and 85.1%, *P*<0.01). TNF-α production in treatment groups (fentanyl 20 μg·L⁻¹+LPS, fentanyl 2 mg·L⁻¹+LPS) and LPS group were 459, 357 and 1226 ng·L⁻¹, IL- 6 were 796, 720 and 1563 ng·L⁻¹, respectively. The differences were significant between treatment groups and LPS one(*P*<0.01). CONCLUSION Fentanyl alone has no effect on NF- κB activation and TNF-α and IL-6 production, but attenuates LPS-induced NF- κB activation and TNF-α and IL-6 production.

Key words <u>fentanyl</u> <u>nuclear factor-kappaB</u> <u>tumor necrosis factors</u> <u>interleukin-6</u>

DOI:

扩展功能

本文信息

- ▶ Supporting info
- ▶ <u>PDF</u>(254KB)
- ▶[HTML全文](0KB)
- **▶参考文献**

服务与反馈

- ▶把本文推荐给朋友
- 加入我的书架
- ▶加入引用管理器
- ▶复制索引
- ▶ Email Alert
- ▶文章反馈
- ▶浏览反馈信息

相关信息

▶ <u>本刊中 包含"芬太尼"的</u> 相关文章

▶本文作者相关文章

- · 饶 邦
- 王焱林
- 李建国
- 段军民