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论文

利多卡因对LPS诱导巨噬细胞HMGB1释放及转位的影响

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摘要：

目的 观察不同浓度利多卡因对脂多糖(LPS)诱导大鼠腹腔巨噬细胞高迁移率蛋白1（HMGB1）释放及转位的影响。方法 取Wistar大鼠腹腔巨噬细胞置12孔板培养2～3d，分为对照组(C组)、LPS组、利多卡因2mg/L+LPS组(Ll+LPS组)、利多卡因20mg/L +LPS组(L2+LPS组)、利多卡因200mg/L +LPS组(L3+LPS组)，分别于6、12、24、48h酶联免疫吸附试验（ELISA）测培养液中HMGB1蛋白浓度。免疫细胞化学染色法观察HMGB1在巨噬细胞内的转位情况。结果 HMGB1蛋白的释放在LPS组刺激12h开始增多，24h达高峰。与LPS组相比，3个利多卡因处理组HMGB1的释放均有不同程度的减少，以终浓度在20mg/L时最显著（P＜0.05）。同时，免疫细胞化学染色法还观察到利多卡因对HMGB1从细胞核到细胞浆的转位有抑制作用。结论 利多卡因20mg/L可显著抑制LPS诱导大鼠腹腔巨噬细胞HMGB1释放及转位。

关键词： 利多卡因；脂多糖；巨噬细胞；高迁移率族蛋白B1；大鼠，Wistar

Effects of Lidocaine on the release and translocation of HMGB1 in macrophages induced by lipopolysaccharide

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Abstract:

Objective To investigate the effects of Lidocaine on expression and translocation of the high mobility group box-l (HMGB1) in rat peritoneal macrophages induced by lipopolysaccharide(LPS). MethodsPeritoneal macrophages obtained from Wistar rats and incubated in 12-well tissue culture plates for 2-3 days were divided into 5 groups: the control group, LPS group, Lidocaine 2mg/L +LPS treatment group (L1+LPS treatment group), Lidocaine 20mg/L +LPS treatment group (L2+LPS treatment group) and the Lidocaine 200mg/L +LPS treatment group (L3+LPS treatment group). After 6， 12， 24 and 48h treatment， the concentrations of HMGB1 in the cell culture medium were measured by ELISA. The translocation of HMGB1 in rat peritoneal macrophages was observed by cellular immunochemistry. ResultsAfter rat peritoneal macrophages were stimulated by LPS， the LPS group showed that the release of HMGB1 was increased at 12h and reached the peak at 24h. Compared with the LPS group, Lidocaine treatment groups decreased in release of HMGB1 in various degrees especially in the 20mg/L+LPS treatment group（P＜0.05）. Translocation of HMGB1 from the cell nucleus to the cytoplasm in the macrophage was obviously suppressed. ConclusionLidocaine(20mg/L) obviously inhibits the release and translocation of HMGB1 in rat peritoneal macrophages induced by LPS.

Keywords: Lidocaine; Lipopolysaccharide; Macrophages; High mobility group box-I; Rats, Wistar

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