生物化学与生物物理进展
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# Toll样受体抗体抑制脂多糖激活巨噬细胞的吞噬活性

# The Inhibition on Phagocytosis of Lipopolysaccharide-stimulated Macrophages by Polyclone Antibody of TLR2

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英文关键词：lipopolysaccharide(LPS)　TLR2　antibody　macrophage　TRAIL　TNFα

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| 作者 | 单位 |
|---|---|
| 王梁华 | 第二军医大学基础医学部卫生毒理学教研室，上海 200433 |
| 冯煜 | 第二军医大学基础医学部卫生毒理学教研室，上海 200433 |
| 钟山 | 第二军医大学基础医学部卫生毒理学教研室，上海 200433 |
| 朱玉平 | 第二军医大学基础医学部卫生毒理学教研室，上海 200433 |
| 娄永华 | 第二军医大学基础医学部卫生毒理学教研室，上海 200433 |
| 焦炳华 | 第二军医大学基础医学部卫生毒理学教研室，上海 200433 |




中文摘要：

应用脂多糖(LPS)激活巨噬细胞后其吞噬能力大大增强，以此为模型发现TLR2的多抗能部分抑制LPS激活巨噬细胞吞噬金葡萄球菌的能力，也能部分阻断对U937细胞的吞噬活性．TRAIL或TNFα多克隆抗体同样能起到类似作用．实验还发现低浓度血清培养亦在一定程度上抑制LPS激活巨噬细胞的吞噬作用．结果证实TLR2能介导LPS的功能，而某些血清因子参与了介导LPS的信号转导过程，提示LPS激活巨噬细胞吞噬能力的提高与诱导表达TRAIL等细胞因子有关．

英文摘要：

The phagocytosis to *Staphylococcus aureus* (SA) and cultivated U937 cells by macrophages derived from PBMC was enhanced significantly under lipopolysaccharide(LPS) stimulation. The present experiment showed that the application polyclone antibodies to TLR2 suppressed partly the phagocytosis of macrophages in this model. The LPS-enhanced phagocytosis could be further blocked partly by polyclone antibodies to TRAIL or TNFα. Cells cultivated in a lower serum concentration(1%) was also shown to be a decreased phagocytosis. The results confirmed that TLR2 was a LPS receptor which mediated its signal transduction, while some serum factors participated in the binding of LPS to its membrane receptors. It further suggested that LPS induced effectors such as TRAIL and TNFα might be the key mediators involved in the mechanisms of LPS-enhanced phagocytosis.

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