The antiplatelet activity of Escherichia coli lipopolysaccharide is mediated through a nitric oxide/cyclic GMP pathway.

林建煌;許準榕

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Abstract

In this study, Escherichia coli LPS dose-dependently (100-500 μg/ ml) and time-dependently (10-60 min) inhibited platelet aggregation in human and rabbit platelets stimulated by agonists. LPS also dose-dependently inhibited the intracellular Ca2+ mobilization in human platelets stimulated by collagen. In addition, LPS (200 and 500 μg/ml) significantly increased the formation of cyclic GMP but not cyclic AMP in platelets. LPS (200 μg/ml) significantly increased the production of nitrate within a 10-min incubation period. Furthermore, LPS also dose-dependently inhibited platelet aggregation induced by PDBu (30 nmol/l), a protein kinase C activator. These results indicate that the antiplatelet activity of E. coli LPS may be involved in the activation of a nitric oxide/cyclic GMP pathway in platelets, resulting in inhibition of platelet aggregation. Therefore, LPS-mediated alteration of platelet function may contribute to bleeding diathesis in septicemic and endotoxemic patients.