Effect of lipopolysaccharide on diarrhea and gastrointestinal transit in mice: Roles of nitric oxide and prostaglandin E2

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摘要

Abstract

AIM: To investigate the effect of lipopolysaccharide (LPS)on the diarrheogenic activity, gastrointestinal transit (GIT),and intestinal fluid content and the possible role of nitric oxide (NO) and prostaglandin E2 (PGE2) in gastrointestinal functions of endotoxin-treated mice.METHODS: Diarrheogic activity, GIT, and intestinal fluid content as well as nitric oxide and PGE2 products were measured after intraperitoneal administration of LPS in mice.RESULTS: LPS dose-dependently accumulated abundant fluid into the small intestine, induced diarrhea, but decreased the GIT. Both nitric oxide and PGE2 were found to increase in LPS-treated mice. Western blot analysis indicated that LPS significantly induced the protein expression of inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 in mice intestines. Pretreatment with NG-nitro-L-arginine-methyl ester (L-NAME, a non-selective NOS inhibitor) or indomethacin (an inhibitor of prostaglandin synthesis) significantly attenuated the effects of LPS on the diarrheogenic activity and intestine content, but reversed the GIT.CONCLUSION: The present study suggests that the pathogenesis of LPS treatment may mediate the stimulatory effect of LPS on nitric oxide and PGE2 production and NO/prostaglandin pathway may play an important role on gastrointestinal function.