Involvement of nuclear factor-kB in lipoteichoic acid-induced cyclooxygenase-2 expression in RAW 264.7 macrophages

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摘要

Abstract

We have investigated the role of protein kinase C (PKC) and nuclear factor-kappaB (NF-kappaB) in cyclooxygenase-2 (COX-2) expression caused by Staphylococcus aureus lipoteichoic acid in RAW 264.7 macrophages. A phosphatidylcholine-phospholipase C (PC-PLC) inhibitor (D-609) and a phosphatidylinositol-phospholipase C (PI-PLC) inhibitor (U-73122) attenuated lipoteichoic acid-induced COX-2 expression, while a phosphatidate phosphohydrolase inhibitor (propranolol) had no effect. Two PKC inhibitors (Go 6976 and Ro 31-8220) and the NF-kappaB inhibitor, pyrrolidine dithiocarbamate (PDTC), also attenuated lipoteichoic acid-induced COX-2 expression. Lipoteichoic acid resulted in a decrease in PKC activity in the cytosol and an increase in PKC activity in membranes. The lipoteichoic acid-induced translocation of p65 NF-kappaB from the cytosol to the nucleus was inhibited by D-609, U-73122, Go 6976, Ro 31-8220, and PDTC, but not by propranolol. The results suggested that lipoteichoic acid might have activated PC-PLC and PI-PLC to induce PKC activation, which in turn initiated NF-kappaB activation, and finally induced COX-2 expression in RAW 264.7 macrophages.