

Ginkgo biloba Extract Inhibits Endotoxin-Induced Human Aortic Smooth Muscle Cell Proliferation via Suppression of Toll-Like Receptor 4 Expression and NADPH Oxidase Activation.

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摘要

Abstract

Toll-like receptor 4 (TLR4) initiates the inflammatory response in blood vessels in reaction to immune stimuli such as lipopolysaccharide (LPS) produced by gram-negative bacteria. LPS-induced proliferation and functional perturbation in vascular smooth muscle cells play important roles during atherogenesis. Ginkgo biloba extract is an antiatherothrombotic Chinese herbal medicine with anti-inflammatory properties. The effects of *G. biloba* extract on LPS-induced proliferation and TLR4 expression and the underlying mechanisms for these actions, in human aortic smooth muscle cells (HASMCs), were examined in vitro. LPS-induced proliferation was mediated by the expression of TLR4 in HASMCs. LPS increased the expression of TLR4 in HASMCs, and this effect was mediated by the activation of nicotinamide adenine dinucleotide phosphate (NADPH) oxidase, phosphorylation of intracellular mitogen-activated protein kinases (MAPKs), and increases in the cytoplasmic level of HuR and TLR4 mRNA stability. *G. biloba* extract inhibited LPS-induced HASMC proliferation and decreased the expression of TLR4 by inhibiting LPS-induced NADPH oxidase activation, mRNA stabilization, and MAPK signaling pathways. These results suggest that LPS-induced TLR4 expression contributes to HASMC proliferation and that *G. biloba* inhibits LPS-stimulated proliferation of HASMCs by decreasing TLR4 expression.