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Xanthotoxin, a furanocoumarin compound expresses anti-inflammatory effects through suppression of iNOS, COX-2, TNF-α, and IL-6 via AP-1, NF-κB, and JAK-STAT inactivation in RAW 264.7 cells

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Xanthotoxin has been reported to possess skin-protective and anti-oxidative properties. However, anti-inflammatory property has not been studied to date. Therefore, we investigated the role that xanthotoxin plays on anti-inflammatory activity as well as its underlying molecular mechanisms in lipopolysaccharide (LPS)-induced RAW 264.7 macrophages. In LPS-induced macrophages, xanthotoxin was found to inhibit nitric oxide (NO), prostaglandin E_2 (PGE_2), tumor necrosis factor (TNF-α), and interleukin-6 (IL-6) in a concentration-dependent manner. It also suppressed the LPS-induced inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2) expression at the protein level and iNOS, COX-2, TNF-α, and IL-6 at the mRNA level. Molecular mechanism shows that xanthotoxin attenuated the LPS-induced transcriptional and DNA-binding activity of activator protein-1 (AP-1), and this was associated with a decrease in the phosphorylation of c-Fos instead of c-Jun. It played a suppressive effect on the transcriptional and DNA-binding activity of nuclear transcription factor kappa-B (NF-κB) through the inhibiting of p65 nuclear translocation. In addition, the LPS-induced phosphorylation of extracellular signal-regulated kinase (ERK) 1/2 and p38 mitogen-activated protein kinase (MAPK) was found to be suppressed by xanthotoxin. Taken together, these results indicate that xanthotoxin decreased NO, PGE_2, TNF-α, and IL-6 production through downregulation of NF-κB, AP-1, and JAK/STAT signaling pathways in LPS-induced RAW 264.7 macrophages.

Biography

Seung-Bin Lee is a student at Kyung Hee University in South Korea and has been intensively studied on screening anti-inflammatory effect among various natural product derived compounds. In an idea to alleviate these tendency, he has been investigated the underlying molecular mechanism of several drugs which elicit significant decrease of inflammatory endpoints such as nitric oxide, prostaglandin E_2, and pro-inflammtory cytokines.

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