Shin-Young Kang et al., J Pharm Sci Emerg Drugs 2018, Volume 6
DOI: 10.4172/2380-9477-C2-009

An iridoid compound K1 isolated from the roots of *Patrinia scabra* attenuates LPS-induced inflammation through suppressing AP-1, NF-κB and STAT1/3 signaling in RAW 264.7 macrophages

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In this study, we isolated an iridoid compound K1 from the Roots Patrinia scabiosaefolia and investigated its anti-inflammatory effects in LPS-induced RAW 264.7 macrophages. K1 reduced LPS-induced nitric oxide (NO) via downregulation of inducible nitric oxide (iNOS) expressions at protein and mRNA levels. And K1 reduced LPS-induced Prostaglandin E2 (PGE2) via downregulation of Cyclooxygenase-2 (COX-2) expressions at protein and mRNA levels. K1 also suppressed the production and mRNA expression of tumor necrosis factor-α (TNF-α), interleukin (IL)-6 and IL-1β. Furthermore, molecular mechanism studies indicated that K1 suppressed LPS-induced transcriptional activity of activator protein-1 (AP-1) as well as the phosphorylation of c-Fos and c-Jun. K1 also reduced transcriptional activities of nuclear factor-kappa B (NF-κB) and phosphorylation of signal transducer and activator of transcription 1/3 (STAT1/3). K1 suppressed the phosphorylation of extracellular signal-regulated kinase (ERK) MAP kinase, but not of c-Jun NH2-terminal kinase (JNK), or p38. Taken together, these results suggest that K1 has anti-inflammatory properties via inhibition of AP-1, NF-κB, and STAT1/3 activation in LPS-induced RAW 264.7 macrophages.

Biography

Shin-Young Kang graduated from Kyung-hee University College of Pharmacy, Department of Pharmaceutical Sciences and is currently a master of biochemistry.

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