Artemisia fukudo essential oil attenuates LPS-induced inflammation by suppressing NF-κB and MAPK activation in RAW 264.7 cells

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In the present study, the chemical constituents of Artemisia fukudo essential oil (AFE) were investigated using GC-MS. The major constituents were α-thujone (40.28%), β-thujone (12.69%), camphor (6.95%) and caryophyllene (6.01%). We also examined the effects of AFE on the production of nitric oxide (NO), prostaglandin E2 (PGE2), tumor necrosis factor-α (TNF-α), interleukin-IL-1β (IL-1β), and IL-6 in lipopolysaccharide (LPS)-activated RAW 264.7 cells. Western blotting and RT-PCR analyses indicated that AFE has potent dose-dependent inhibitory effects on pro-inflammatory cytokines and mediators. We investigated the mechanism by which AFE inhibits NO and PGE2 by examining the level of nuclear factor-κB (NF-κB: p50 and p65) activation within the mitogen-activated protein kinase (MAPK: ERK, JNK and p38) pathway, which is an inflammation induced signal pathway in RAW 264.7 cells. AFE inhibited LPS-induced ERK, JNK and p38 phosphorylation. Furthermore, AFE inhibited the LPS-induced phosphorylation and degradation of IκB-α, which is required for the nuclear translocations of the p50 and p65 NF-κB subunits in RAW 264.7 cells. Our results suggest that AFE might exert an anti-inflammatory effect by inhibiting the expression of pro-inflammatory cytokines. Such an effect is mediated by a blocking of NF-κB activation which consequently inhibits the generation of inflammatory mediators in RAW 264.7 cells. AFE may be useful for treating inflammatory diseases.

Key words: Artemisia fukudo essential (AFE), Chemical composition, inflammation, nuclear factor-κB, MAPK.