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

## Weon-Jong Yoon

Jeju Biodiversity Research Institute (JBRI), Jeju TECHNOPARK (JTP)

In the present study, the chemical constituents of *Artemisia fukudo* essential oil (AFE) were investigated using GC-MS. The major constituents were  $\alpha$ -thujone (40.28%),  $\beta$ -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 E<sub>2</sub> (PGE<sub>2</sub>), tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), interleukin-IL-1 $\beta$ (IL-1 $\beta$ ), 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 PGE<sub>2</sub> by examining the level of nuclear factor- $\kappa$ B (NF- $\kappa$ B: 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 I $\kappa$ B- $\alpha$ , which is required for the nuclear translocations of the p50 and p65 NF- $\kappa$ B 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- $\kappa$ B 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.