

# Anti-inflammatory effect of spilanthol from *Spilanthes acmella* on murine macrophage by down-regulating LPS-induced inflammatory mediators

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## Abstract

*Spilanthes acmella* (Paracress), a common spice, has been administered as a traditional folk medicine for years to cure toothaches, stammering, and stomatitis. Previous studies have demonstrated its diuretic, antibacterial, and anti-inflammatory activities. However, the active compounds contributing to the anti-inflammatory effect have seldom been addressed. This study isolates the active compound, spilanthol, by a bioactivity-guided approach and indicates significant anti-inflammatory activity on lipopolysaccharide-activated murine macrophage model, RAW 264.7. The anti-inflammatory mechanism of paracress is also investigated. Extracts of *S. acmella* are obtained by extraction with 85% ethanol, followed by liquid partition against hexane, chloroform, ethyl acetate, and butanol. The ethyl acetate extract exhibits a stronger free radical scavenging capacity than other fractions do, as determined by DPPH and ABTS radical scavenging assays. The chloroform extract significantly inhibits nitric oxide production ( $p < 0.01$ ) and is selected for further fractionation to yield the active compound, spilanthol. The diminished levels of LPS-induced inducible nitric oxide synthase (iNOS) and cyclooxygenase (COX-2) mRNA and protein expression support the postulation that spilanthol inhibits proinflammatory mediator production at the transcriptional and translational levels. Additionally, the LPS-stimulated IL-1 $\beta$ , IL-6, and TNF- $\alpha$  productions are dose-dependently reduced by spilanthol. The LPS-induced phosphorylation of cytoplasmic inhibitor-kappaB and the nuclear NF-kappaB DNA

binding activity are both restrained by spilanthol. Results of this study suggest that spilanthol, isolated from *S. acmella*, attenuates the LPS-induced inflammatory responses in murine RAW 264.7 macrophages partly due to the inactivation of NF-kappaB, which negatively regulates the production of proinflammatory mediators.