

*Corydalis turtschaninovii*의 근경에서 분리한 pseudocoptisine의 항염증 효과 및
기전 연구

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**Anti-inflammatory effects and its molecular mechanism of pseudocoptisine
isolated from tuber of *Corydalis turtschaninovii***

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Objectives

In this study, we have made attempt to screen anti-inflammatory effects of pseudocoptisine, one of the active compound isolated from the tuber of *Corydalis turtschaninovii*, and understand the molecular mechanism using an *in vitro* model system of mouse macrophage Raw 264.7 cells.

Materials and Methods

○ Materials

Pseudocoptisine isolated from the tuber of *Corydalis turtschaninovii* (Papaveraceae) and were determined by HPLC to be > 97 % pure. The Raw 264.7 macrophage cell line was obtained from the Korea Cell Line Bank (Seoul, Korea).

○ Methods

To determine the effect of pseudocoptisine on NO production, nitrite levels in culture media were determined using the Griess reagent assay. Levels of PGE₂, TNF- α , IL-6 in culture media were quantified using R&D System EIAs kits. Western blot and reverse transcriptase polymerase chain reaction (RT-PCR) were examined to determine inflammation-related protein and mRNA levels. Electrophoretic mobility shift assay (EMSA) and luciferase assay were performed to measure the effect of pseudocoptisine on DNA binding and transcriptional activity of nuclear factor- κ B (NF- κ B).

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Results

We have demonstrated that pseudocoptisine can potentially suppress LPS-induced NO, PGE₂, TNF- α and IL-6 productions, iNOS and COX-2 protein and mRNA levels, and TNF- α and IL-6 mRNA levels by inhibiting the binding activity and transcriptional activity of transcription factor NF- κ B. And pretreatment of pseudocoptisine prevented I κ Ba phosphorylation and degradation. Moreover, pseudocoptisine suppressed phosphorylation of ERK and p38 but not JNK. These results suggest that the anti-inflammatory properties of pseudocoptisine might be due to the inhibition of NF- κ B activation through I κ Ba and MAPK dependent signalling pathway and it lead to reduction of pro-inflammatory expression such as iNOS, COX-2, TNF- α and IL-6.

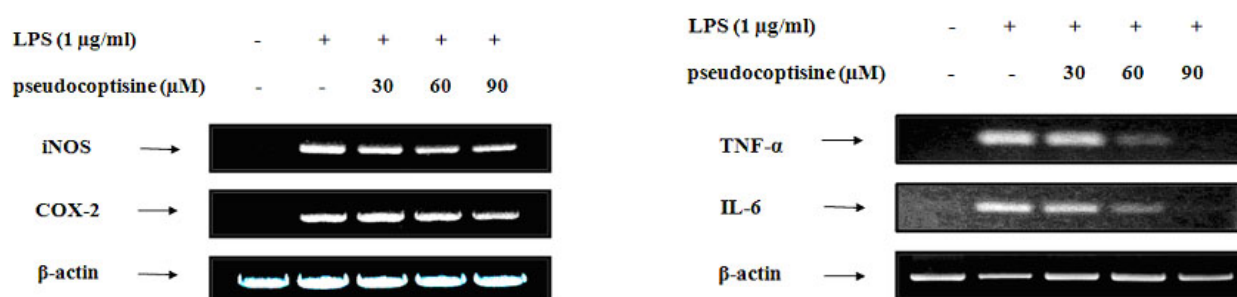


Fig 1. Effect of pseudocoptisine on iNOS, COX-2, TNF- α and IL-6 levels in LPS-stimulated Raw 264.7 cells.

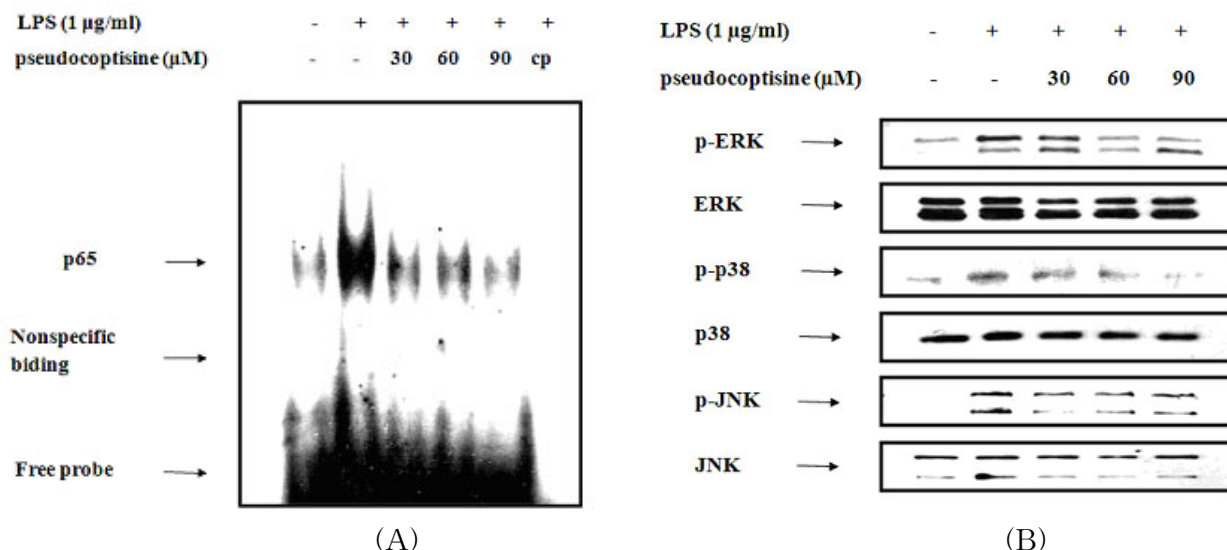


Fig 2. Effect of pseudocoptisine on NF- κ B DNA binding activity (A) and MAPKs phosphorylation (B) in LPS-stimulated Raw 264.7 cells.