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Suppressoin of NF-κB signaling pathways by ergolide in HeLa cells

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In previous study, we investigated the mechanism of suppression of inducible nitric oxide synthase (iNOS) and cyclo-oxygenase-2 (COX-2) by ergolide, sesquiterpene lactone from *Inula Britannica*. In this study, the suppression of iNOS and COX-2 by ergolide might be attributed to selective inhibition of NF-κB signaling pathways. Here, we investigated the suppression mechanism of NF-κB signaling pathways by ergolide in TPA-stimulated HeLa cells. We showed that ergolide inhibited NF-κB promoter activity. This effect was accompanied by the parallel reduction of NF-κB DNA binding activity as well as nuclear translocation of subunit p65 of NF-κ B. In addition, ergolide decreased the degradation of IκB and phosphorylation of IκB in TPA-stimulated HeLa cells. Ergolide also inhibited protein levels of phospho-IKK in TPA stimulated-HeLa cells. These results indicate that the inhibition of NF-κB signaling pathways may be associated with the inhibition of IKK activity. Taken together, the results suggest that suppression of NF-κB signaling pathways is responsible for the anti-inflammatory activity of ergolide through inhibition of IKK activity, which play important roles in inflammatory signaling pathways

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Why is β-ketoacyl-ACP synthase II (FabF) is toxic in *E. coli* fatty acid biosynthesis ?

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In the type II system, there are two elongation enzymes in *E. coli*, FabB is well-known to its ability to elongate *cis*-3-decenoly-ACP (C10:1) in unsaturated fatty acid synthesis, whereas FabF is important for the thermal regulation of fatty acid composition by its ability to elongate palmitoleic acid to vaccenic acid, based on their genetic mutation anaylsis. Radiochemical enzyme assay was performed using myristoyl-ACP as a substrate, which is known for general substrate of FabB and FabF. The specific activity of FabB (139.7 ± 3.9 pmole/min/mg) was about 10-fold less than that of FabF (2032 ± 39.4 pmole/min/mg). Both FabF and FabB made triacetic acid lactone (TAL) from malonyl-ACP, and FabF was 10-fold more active than FabB. Within the cell, FabF could effectively convert malonyl-ACP to TAL instead of fatty acid biosynthesis. It could be an explanation why FabF is toxic when it is overexpressed. These data demonstrated that FabF can function in unsaturated fatty acid synthesis like FabB, and in the regulation of malonyl-CoA level by conversion malonyl-ACP to TAL.

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Purification and Partial Characterization of a Lectin with Potent Immunomodulatory Activity from the Mushroom *Fomitella fraxinea*

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