Cudraxanthone D ameliorates the psoriasis-like skin inflammation in an imiquimod-induced mouse model via inhibiting the inflammatory signaling pathways

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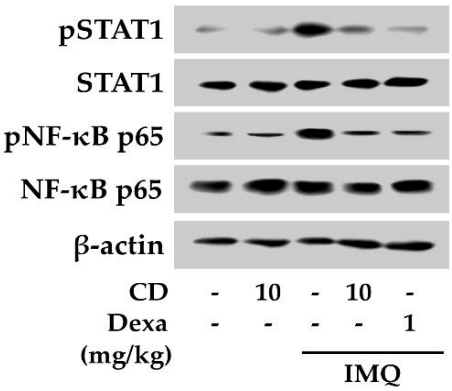
Supplementary material and method

Protein extraction from IMQ-induced mice skin

At the mice sacrificed, mice skin was gathered in RIPA buffer (Biosesang) containing a protease/phosphatase inhibitor mixture (Roche). Next, mice skin was homogenized by a TissueLyser II (Qiagene). The tissue lysates were centrifuged at 1,200 *g* for 15 min at 4°C, and supernatant was collected. The protein quantification assay was performed with the Bradford Protein assay kit (Bio-Rad Laboratories) and loaded onto 10% sodium dodecyl sulfate polyacrylamide gel electrophoresis, and then transferred onto nitrocellulose membranes (Pall Life science). After transference, membranes were observed with Ponceau S stain. Immunodetection was carried out using SuperSignal West Pico Chemiluminescent Substrate (Thermo Fisher Scientific) by G:BOX Chemi XRQ (Syngene).

Figure S3. Effects of CD on the total and phosphorylated form of STAT1 and NF-κB in mouse skin.



After the *in vivo* experiments, the mice were sacrificed, and their skins were obtained. Activation of STAT1 and NF-κB in IMQ-induced skin was detected via Western blot. The loading control was confirmed using β-actin in independent blots from three randomly-selected skin tissue per each group. CD: Cudraxanthone D, Dexa: dexamethasone.