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Immunomodulatory and anti-inflammatory activities of *Chrysanthemum indicum* extract

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Objectives

Chrysanthemum indicum Linné (Compositae) is a traditional herbal medicine used for treatment of various diseases. In the present study, we determined the ability of Chrysanthemum indicum extract (CIE) to inhibit skin inflammation following exposure to the well-characterized protein kinase C activator and tumor promoter, 12-O-tetradecanoyl-phorbol-13-acetate (TPA).

Materials and Methods

○ Materials
- The flowers of C. indicum as a dried herb were obtained from the Oriental Drug Store (Omniherb Co.) in Korea.
- Specific pathogen - free 5-week-old male C57BL/6J mice were purchased from Dae Han Biolink Co. in Korea.
- TPA and indomethacin were from Sigma Chemical Co. (St Louis, MO, USA). All other chemicals and reagents were of the highest commercial grade available.

○ Methods
- The mouse model of acute inflammation employed here was a slight modification of a previously described procedure (Stanley, et al., 1991).
- The effect of CIE on chronic skin inflammation was evaluated by a slight modification of a previous described procedure (Burke, 2001).

Results and Discussion

CIE ameliorated the inflammatory phenotype, leading to substantial reductions in skin thickness and tissue weight, inflammatory cytokine production, neutrophil-mediated myeloperoxidase activity, and various histopathological indicators. CIE was also effective at reducing inflammatory damage induced by chronic TPA exposure. These results demonstrate that CIE is an effective anti-inflammatory agent in murine phorbol ester - induced dermatitis, and suggest that the compound may have therapeutic potential in a variety of immune-related cutaneous diseases.

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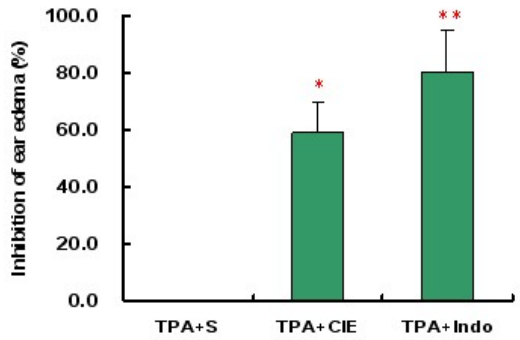

Figure 1. Effect of CIE on TPA-induced ear edema in the acute mouse model. Mice were treated with a topical application of TPA as has been described in Materials and Methods. Ear edema was measured at 6 h after TPA treatment. Each point represented the mean of the difference between ear thickness before and after challenge with S.E.M. (N=10, **P*<0.01 compared to vehiclel, ***P*<0.01 compared to TPA alone as determined by the Student's *t*-test.)

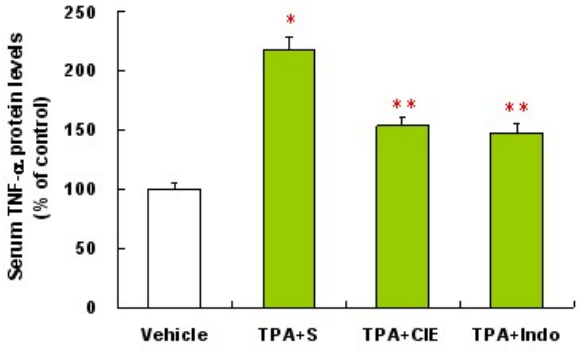

Figure 2. Effect of CIE on TPA-induced tumor necrosis factor (TNF)-α production. TNF-α level was measured at 6 h after TPA treatment. The data are shown as mean percent inhibition with S.E.M. (N=10, **P*<0.01 compared to control, ***P*<0.05 compared to TPA alone as determined by the Student's *t*-test.)

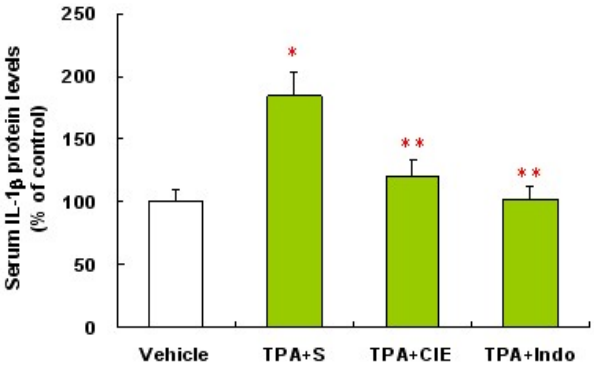

Figure 3. Effect of CIE on TPA-induced interleukin-1β (IL-1β) production. IL-1*β* level was measured at 6 h after TPA treatment. The data are shown as mean percent inhibition with S.E.M. (N=10, **P*<0.01 compared to control, ***P*<0.05 compared to TPA alone as determined by the Student's *t*-test.)