ABSTRACT BOOK ABSTRACTS



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ATOPIC ECZEMA/DERMATITIS

## SYNERGISTIC EFFECTS ON KOREAN RED GINSENG EXTRACT ON CONVENTIONAL THERAPEUTIC MEDICINES IN A MURINE MODEL OF ATOPIC DERMATITIS

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Introduction : Korean red ginseng (KRG) has been shown to possess diverse biological effects, including anti-inflammatory and anti-allergic. KRG also exerts therapeutic effects against atopic dermatitis (AD) by reducing clinical and systemic inflammation. However, in the actual clinical situation, the use of KRG as an adjunct to the treatment of AD is more common than the use of KRG alone in the treatment of AD.

Objective : In this study, we identified the synergistic effects on KRG extract on conventional therapeutic medicines in a mouse model of AD.

Materials and Methods : Ninety NC/Nga mice were randomly divided to 18 groups. AD-like skin lesions were induced by percutaneous challenge with 2,4,6-trinitro- 1-chrolobenzene (TNCB) on the ears and backs of NC/Nga mice. The group was divided into two big groups, one group taking oral medication and the other applying topical agent. For systemic feeding group, KRG extract, evening primrose oil, cyclosporine, and hydroxyzine were administered orally by a gastric tube. For topical group, topical desonide, tacrolimus hydrate 0.03%, diphenhydramine were applied. The effects of KRG and the other agents were assessed by measuring the clinical severity score, ear thickness, extent of transepidermal water loss (TEWL), total systemic immunoglobulin E (IgE) and interleukin (IL)-31 levels, histologic changes of cutaneous lesions, and mRNA expression levels of tumor necrosis factor (TNF)- $\alpha$ , interferon (IFN)- $\gamma$ , thymic stromal lymphopoietin (TSLP), and CD1a.

Results : Clinical severity score, ear thickness, TEWL, serologic result and histologic changes were all improved.

Conclusion : KRG exerts synergistic effects on conventional therapeutic against AD by inhibiting the T helper 2 (Th2) mediated inflammation. KRG also reduces clinical and systemic inflammation synergistically in our murine model of AD.





