Synopsis of Original Research Paper

Development of a new skin inflammation model useful for the evaluation of stimulating and protective effects of cosmetics on the skin of atopic patients

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Stimulating activity for skin, including that of patients with atopic dermatitis, is one of the most important issues for the development of cosmetics. I have recently proven that antigen-specific T cells have potential to induce allergic inflammation in various tissues including skin, without assistance of IgE/mast cell-dependent pathway. Here, I aimed to establish a new mouse model of skin inflammation that enable to evaluate the stimulating and protective effects of cosmetics on atopic patients' skin. Hairless mice in which antigen-specific T cell receptor (TCR) is expressed on all peripheral T cells is generated by mating BALB/c-background Hr mice with cloned mice that we recently established from mite-specific T cells. Th1 and Th2 cells were differentiated from splenic $CD4^+$ T cells of ovalbumin (OVA)-specific TCR-transgenic mice by *in vitro* stimulation culture. BALB/c mice were adoptively transferred with the antigen-specific Th1 and Th2 cells, then the back skin was challenged with topical application of OVA solution. In addition to the inflammatory features observed in the histological specimens, accumulation of eosinophils and antigen-specific Th2 cells, evaluated by the elevation of eosinophil peroxidase activity and by flow cytometry, respectively, in the skin was induced by antigen challenge, despite the absence of antigen-specific IgE. Neither antigen-specific Th1 nor naive $CD4^+$ T cells induced eosinophil accumulation, although Th1 cells by themselves migrated into the skin. The accumulation of eosinophils and Th2 cells in the skin was suppressed by administration of dexamethasone and FK506, indicating an essential role for Th2 cells in the eosinophil recruitment. Based on the rapid development of atopic dermatitis-like pathological features and its responsiveness to therapeutic drugs prescribed, the usefulness of our skin inflammation model for the evaluation of stimulating and protective effects of cosmetics on the skin of atopic patients was indicated.