

**Immunopharmacological Analysis of Allergic Inflammation and
an Approach for Development of New anti-Allergic Agents**

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Research in the past decade has provided much evidence regarding the onset mechanism and therapy of immuno-inflammatory diseases including allergic asthma, rhinitis and atopic dermatitis. Recently, allergic disease is understood to be caused by Th2 polarized immunity. Helper T cell of type 1 variety (Th1) secrete IFN- γ , IL-2 and lymphotoxin, while those of the type 2 variety (Th2) cells secrete IL-4, -5 and -13. Th1 cells enhance cellular immune responses: prototype is the tuberculin (PPD) skin test response. Th2 cells favor humoral antibody production including IgE antibody associated with allergic condition. Recent studies demonstrated that most allergic diseases are associated with active T-cell immune response against invaded allergen that is skewed towards the Th2 phenotype in the contrast to a Th1-skewed immunity.

From above background, we investigated the reasons why immune response shifted to Th2-skewed immunity; in another word what is a promoting factor for allergy. We focused on the genetics and environmental conditions. Our data indicate the importance of some gene polymorphism of Th2 cytokine and lipid mediator related molecules. Moreover, our data showed the importance of environmental conditions including period and kind of antigen exposure, the air pollution and foods especially lipids. Otherwise, we tried to develop a new anti-allergic remedy which is able to modify an imbalance of Th1 and Th2 immunity. Our recent data concerning the onset mechanism of allergy and remedy will be presented.