**056**
In vivo tracking of antigen-specific skin-resident memory CD4+ T cells
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Background: The efficacy and autologous role of whole blood injection (AWBI) in treating chronic spontaneous urticaria (CSU) is unclear, which may be explained by seeking appropriate biomarkers to predict the response of the affected patients. Objective: To explore the possible mechanism of AWBI in treating CSU by investigating the correlation between IgE, D-dimer, anti-FcεRI IgG and basophil FcεRI expression and the clinical symptoms of CSU treated with AWBI Methods: Eighty patients with autologous serum skin test (ASST)-positive CSU were enrolled and randomly divided into AWBI treated group (receiving AWBI and antihistamine) and control group (only with antihistamine). Urticaria activity score (UAS) and psychological burden were compared and analyzed. Levels of plasma total IgE, D-dimer, and anti-FcεRI IgG of 30 AWBI group patients before and after treatment were compared with those of 25 healthy controls: The basophil FcεRI expression in the peripheral venous blood of the patients was also analyzed. Results: A better clinical response was observed in the AWBI treated group than in controls. ASST+ CSU patients had higher concentrations of baseline plasma IgE, D-dimer and anti-FcεRI IgG as compared to health controls. IgE and D-dimer were differentially expressed between AWBI responders and non-responders, displaying good diagnostic value in predicting therapeutic response to AWBI. Basophil FcεRI expression was significantly higher in AWBI responders, with obvious decline during AWBI treatment. Conclusion: This study supported the effectiveness of AWBI in CSU, with changes of plasma total IgE and D-dimer as potential predictors of treatment response. A possible mechanism of AWBI in treating CSU is through the reduced expression of basophil FcεRI.

**057**
Levels of plasma total IgE and D-dimer and basophil FcεRI expressions: potential predictors of response to autologous whole blood injection in chronic spontaneous urticaria
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Objective: To investigate the correlation between plasma total IgE, D-dimer and basophil FcεRI expression in chronic spontaneous urticaria (CSU) patients and the clinical symptoms of CSU. Methods: Eighty-two patients with CSU were included in this study. The concentrations of plasma total IgE, D-dimer and basophil FcεRI expression were measured by ELISA. The clinical symptoms were assessed by UAS. The relationship between plasma total IgE, D-dimer and basophil FcεRI expression and the clinical symptoms of CSU were compared and analyzed. Results: The plasma total IgE and D-dimer levels were significantly higher in non-responders than in responders, and these two parameters were significantly negatively correlated with the UAS. The percentage of basophil FcεRI expression was significantly lower in non-responders than in responders. Conclusion: Plasma total IgE and D-dimer are potential predictors of treatment response to AWBI. Basophil FcεRI expression is a potential predictor of non-responders. These results provide insights into the mechanism of AWBI in treating CSU.

**065**
CDK7 inhibitor suppresses psoriasis inflammation via inhibiting glycolysis to modulate Th17/Treg balance
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Background: Psoriasis is a chronic inflammatory disease characterized by hyper-activated Th17 and suppressive Treg cells, but the mechanism of Th17/Treg cells imbalance is still unclear. Cyclin-dependent kinase 7 (CDK7) which is known as a cell cycle regulator has been reported an anti-inflammatory effect in immune-cells. Here we firstly found CD4+ T cells of psoriasis patients expressed higher levels of CDK7 along with an increased glycolysis levels than those in healthy controls. The chemical inhibitor of CDK7 called THZ1 restricted glycolytic metabolism in CD4+ T cells of psoriasis patients as well as typical glycolysis related genes. More importantly, THZ1 could suppress Th17 cell differentiation and promote Treg cell differentiation even under Th17 polarizing condition in vitro. Intraperitoneal injection of THZ1 in mice exhibited an alleviated epidermal hyperplasia and alleviated inflammation caused by imiquimod (IMQ) treatment. THZ1-treated IMQ mice had significantly lower ratio of Th17 cells and increased ratio of Treg cells. In conclusion, THZ1 could be a potential drug for psoriasis treatment. Objective: To determine the effect of THZ1 on Th17/Treg balance. Methods: The expression of CDK7 and related genes were measured by qPCR. The differentiation of Th17 and Treg cells were assessed with flow cytometry. The proliferation of Th17 and Treg cells were assessed by MTT assay. Results: CDK7 inhibitor THZ1 may serve as an immuno-modulator for psoriasis therapy in the future.

**067**
Development of allergen-specific Foxp3+RORγt+Treg cells during allergen-specific immunotherapy
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Background: Allergen-specific immunotherapy (ASIT) is an effective treatment that can induce clinical and immunological improvement in atopic dermatitis. The mechanism of ASIT therapy is to induce the allergen-specific regulatory T (Treg) cells. Objective: To validate the hypothesis that allergen-specific immunotherapy is effective for psoriasis and to identify the regulatory T cells. Methods: We measured the development of skin-resident memory CD4+ T (TRM) cells and Treg cells in psoriasis patients treated with ASIT. Results: The expression levels of Th1, Th2 and Th17-related genes were significantly decreased after ASIT. Treg cells were isolated from peripheral blood mononuclear cells before and after 3, 6, 12 months of ASIT. Treg cells were isolated from peripheral blood mononuclear cells after 3, 6, 12 months of ASIT. The percentage of Treg cells was significantly increased from 3 months through 12 months of ASIT. The percentage of Treg cells was significantly increased from 3 months through 12 months of ASIT. Conclusion: The development of skin-resident memory CD4+ T cells (TRM) cells and allergen-specific Treg cells are increased as a result of allergen-specific immunotherapy.