Psoriasis patient serum biomarkers may be useful for separating response to therapy endotypes

Psoriasis is a chronic inflammatory skin disease with a high burden of disease. The psoriasis Center of Research Translation (CSR) level and FcγRIIb-dependent blockade of immunoglobulin class-switch is crucial to prevent pemphigus onset in desmoglein 3-specific B cell receptor knock-in mouse

Pemphigus is an autoimmune bullous disease caused by anti-desmoglein 1 (Dsg1) IgG. We previously isolated pathogenic anti-Dsg1 IgG from a clinical sample. We produced a transgenic mouse model, gld(-/-)AK23 KI mice, in which all Dsg1-specific B cells and Abs are deleted. gld(-/-)AK23 KI mice were crossed with FcγRIIb−/− mice. There was no difference in amount of serum total IgG between FcγRIIb+/+ and FcγRIIb−/− WT mice at the age from 6 to 16 weeks. However, FcγRIIb−/− AK23 MII mice spontaneously produced anti-Dsg1 IgG from early age to 6 weeks and developed PV phenotype. The results demonstrated that pathogenic autoantibody production is prevented at CSR level and FcγRIIb crucially maintains this tolerogenic condition to avoid autoimmunity.

IL-27 induces IL-15 production to facilitate T cell survival in allergic contact dermatitis

IL-27 downregulates IL-23-induced IL-17A expression in psoriasis. The expression of IL-27 in psoriasis was determined using immunofluorescence staining. The expression of IL-17A in T cells was determined using enzyme-linked immunosorbent assay. Imiquimod (IMQ)-induced psoriasis model was used to determine the effect of IL-27 on the IL-17A production from T cells. In conclusion, IL-27 plays an anti-inflammatory role in psoriasis by suppressing IL-17A expression.