Interferon kappa is required for regulation of baseline type I interferon responses in keratinocytes and is dysregulated in cutaneous lupus

Ifn responses could be transferred to fibroblasts by adding supernatants from KCs, and IFN responses, which we confirmed (p < 0.043), showed prominent IFN mRNA was the most increased in CLE (p = 0.001, n = 9, and confirmed by IFN-FK KO delayed and minimized KC responses to exogenous IFNα (p < 0.001). Cutaneous lupus (CLE) is characterized by type I IFN responses, which we confirmed (p< 0.001), baseline type I IFN gene expression, and basal pSTAT1 and 2 activation. Similarly, KO of TYK2, a signal mediator downstream of type I IFN receptors, abolished baseline type I IFN gene expression in KCs (p = 0.043), suppressed IFN mRNA expression (p< 0.001, n = 7 samples). These results were consistent with the clinical observation that treatment with the S1P1 inhibitor FTY720 resulted in rapid repigmentation, yet preserved Trm in skin and in lymph nodes, as well as cytolytic function. Therefore, these results identify TRPM2 as a key player that links oxidative stress to the NLRP3 inflammasome activation and melanocyte autoimmune destruction.

Oxidative stress inducing TRPM2 activate the NLRP3 inflammasome in keratinocytes resulting in melanocyte destruction in vitiligo

Vitiligo is a skin depigmenting autoimmune disease caused by autoreactive CD8+ T cells that kill melanocytes, resulting in patchy depigmentation. Treatment options for IDO, T cell density, and T cell interaction with the endothelial cells. The study raises the possible utility of IDO inhibitors in increasing involution of IH.

Characterization of a conformational epitope on the E1 domain of desmoglein 1 recognized by IgG4 autoantibodies from Fogo Selvagem

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