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

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IFN responses could be transferred to fibroblasts by adding supernatants from KCs, and in KCs via CRISPR/Cas9 abolished KC IFN

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reversal of disease in mice. Based on these data and clinical observations, we propose that with an IL-15 blocking antibody effectively depleted autoreactive Trm and resulted in durable

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exogenous IFN from all concentrations (1, 5, 50ng/ml), IFNKO delayed and minimized KC responses to exogenous IFN-x (p < 0.001). Cutaneous lupus (CLE) is characterized by type I IFN responses, which we confirmed (MX1, OAS1, p < 0.001 for all). Type I IFN family, IFNκ was the most increased in CLE (p < 0.001, n=90), and confirmed by IFN showing prominent IFN in epidermis of active CLE, along with pSTAT1 and pSTAT2 acti-

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vation. In addition, single cell RNA-seq increased baseline pSTAT1 and pSTAT2 activity and higher IFN production compared to KCs from healthy controls. CLE KCs had heightened responses to exogenous IFN4 that could be suppressed to healthy control levels by addition of anti-IFNκ antibody (MX1, p < 0.05). Our data identifies IFN-κ as an important cytokine in the responses in KCs and helps to explain the clinical observations of why blockade of the type I IFN receptor is more effective than targeting IFN-κ in CLE.

Characterization of a conformational epitope on the EC1 domain of desmoglein 1 recognized by IgG4 autoantibodies from Fogo Selvagem

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Vitiligo is an autoimmune disease caused by autoreactive CD8+ T cells that destroy melanocytes resulting in melanocyte destruction in vitiligo

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