Vitiligo is maintained by antigen-specific resident memory T cells, which can be targeted to create a durable treatment response

We set out to determine the mechanism by which IFN responses in keratinocytes and is dysregulated in cutaneous lupus

There is evidence of a possible involvement of a host immune response in the involution of inflammatory skin diseases (11). In this study, we utilized mouse models of cutaneous lupus to investigate the role of IFN responses in keratinocytes and in cutaneous lupus.

We explored a possible role for Indoleamine 2,3-deoxegenase (IDO) and the Host Immune Response in Inflammatory Hemangioma

Determinants of the regulatory network of Th17 cells in autoimmune diseases

Vitiligo is an autoimmune disease characterized by the loss of melanocytes resulting in depigmentation. The pathogenesis of vitiligo is complex and involves both genetic and environmental factors. In this study, we used single-cell RNA sequencing (scRNA-seq) to investigate the transcriptional landscape of vitiligo keratinocytes.

We first discovered that TRPM2, a member of transient receptor potential (TRP) protein superfamily, forms a Ca2+ permeable nonselective cation channel, which is activated by reactive oxygen species (ROS) in vitiligo keratinocytes. We further found that Ca2+ influx via TRPM2 leads to the accumulation of damaged, ROS-generating mitochondria and NLRP3/ASC co-location with the mitochondria, and this subsequently activates the NLRP3 inflammasome.

There is evidence of a possible involvement of a host immune response in the involution of inflammatory skin diseases. Here, we show that melanocyte-specific Tr1 are present in both mouse and human vitiligo skin lesions. Functional analysis of Tr1 cells indicates that they sense autoantigen in the skin and secrete alarm signals to recruit recirculating T cells to the skin. This study was the first to utilize melanocyte-specific Tr1 cells to monitor the clinical observation of why blockade of the type I IFN receptor is more effective than targeting IFN in CLE.