**031**

Development of a mouse model of Pemphigus Vulgaris as a tool to evaluate naïve and antigen-specific tolerance

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Pemphigus Vulgaris (PV), an autoimmune blistering disease caused by autoantibodies mainly directed against desmoglein (Dsg). So far, only broad systemic immunosuppression and B-cell depletion are being applied in PV patients. In this study, we aim at investigating a Dsg-specific CD4+ T cell directed therapy in PV. We apply human leukocyte antigen (HLA)-transgenic mice that express the PV-associated HLA DRB5*02 allele, the human CD4 coreceptor and lack mouse HMC class II (I-Aß-/-). Immunization with recombinant human Dsg3 protein leads to induction of Dsg1-specific CD4+ T and Dsg3-reactive IgG producing B cells. After initial immunization by 74% and 80%, respectively, compared to control ani-

**032**

An advanced biology platform to guide the discovery of a new highly selective JAK1 inhibitor for atopic dermatitis treatment

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Bullous pemphigoid (BP) and mucous membrane pemphigoid (MMP) are blistering skin diseases in which autoantibodies against basal keratinocyte antigens cause loss of cell adhesion to the basement-membrane zone (BMZ). Biopsies usually show BMZ-bound IgG and C3 in direct immunofluorescence (IF), indicating complement activation that results in attraction and activation of inflammatory cells and subsequently dermal-epidermal separa-

**033**

Expansion of BCL2+ lymphocytes in cutaneous graft-versus-host disease is associated with steroid resistance and poor prognosis

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**034**

Characterization of novel TMEM173 mutation causing a lupus- and SAVI-like phenotype, modified by polymorphisms in TMEM173 and IFH1

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**035**

CD4+ resident memory T cells colocalize with CD31b+ dendritic cells in periocular lymphocyte clusters in a murine delayed-type hypersensitivity model

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**036**

Targeted inhibition of complement at the basement-membrane zone in pemphigoid diseases

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S220 Journal of Investigative Dermatology (2019), Volume 139