**Pemphigus Lesional ELS May Contribute to Persistent Lesions**

Zhou and colleagues detected ectopic lymphoid structures (ELS) that resemble tertiary lymphoid organs in pemphigus vulgaris and pemphigus foliaceous lesions. ELS were characterized by B cells reactive to the desmoglein 3 autoantigen, and were associated with active disease and B cell expansion. B cell differentiation in ELS was supported by the detection of centroblasts, plasmablasts, and plasma cells in these lesions. Chemokines that may induce B cell migration to pemphigus lesions were also detected in ELS. These results provide insight into the complexities of these skin blistering diseases and suggest that targeting B cells in ELS may have therapeutic implications. See pages 275 and 309.

**Coal Tar Alters Cutaneous Microbiome in AD**

Conventional treatments for atopic dermatitis (AD) include corticosteroids, immunosuppressants, and emollients. However, coal tar (CT), which is composed of thousands of chemicals, has been reported to induce AD remission. CT activates the aryl hydrocarbon receptor (AHR), restoring skin barrier function via effects on keratinocyte differentiation and T helper type 2 immune responses. Smits and colleagues reported that CT treatment alters the cutaneous microbiome composition in AD. The lesional microbiome, which is associated with the predominance of *Staphylococcus aureus* in AD, exhibited decreased *Staphylococcus* species abundance and increased *Propionibacterium* species abundance following CT treatment. Mechanistically, CT treatment induced keratinocyte-derived antimicrobial peptide production via AHR signaling. These findings suggest that specifically targeting AHR signaling using more specific compounds may provide efficacious AD therapy. See page 415.

**Spherical Nucleic Acids Target IL-17RA to Treat Psoriasis**

The activation of T helper type 17 signaling underlies psoriasis pathogenesis. Systemic use of monoclonal antibodies that target IL-17 and its receptor (IL-17RA), while being efficacious, is constrained by high cost and the potential for adverse effects. Liu and colleagues demonstrated that topically delivered, liposomal antisense oligonucleotide IL-17RA-targeting spherical nucleic acids (SNA) efficiently cross the epidermal barrier and inhibit IL-17RA gene expression and downstream psoriasis markers in both mouse and human cells. Furthermore, these SNA effectively reduced clinical, histological, and transcriptional signs of psoriasis in an imiquimod-induced mouse model. This new therapeutic approach is currently being studied in clinical trials. See page 435.

**Non-Coding Variant Has Long-Range Enhancer Function in Autoimmune Disease**

Based on previous genome-wide association study reports of the IRF3-TNPO3 risk locus for systemic lupus erythematosus (SLE) and systemic sclerosis (SSc), Thynn and colleagues employed fine-mapping bioinformatics and functional studies to identify an allele-specific functional risk variant (rs13239597) in the TNPO3 promoter region at this locus. These studies provided evidence that the EV11 transcription factor binds to this variant, resulting in chromatin looping and subsequent long-range enhancement of IRF5 expression. This risk allele is associated with both SLE and SSc. Thus, these mechanisms shed light on disease pathogenesis and also highlight potential therapeutic targets in both autoimmune diseases. See pages 277 and 348.

**Generating Useful Outcome Predictions for Mycosis Fungoides Patients**

Mourad and Gniadecki performed a meta-analysis of pooled overall survival data from 10 studies, comprising 6,279 patients, to determine the median survival and best- and worst-case scenarios for the different stages of mycosis fungoides (MF). As expected, these analyses revealed significantly longer survival for early stages compared with later stages. Stage 1B MF patients have an 85.8% chance of survival after 5 years with a best-case survival of 88.8% and a worst-case survival of 82.1%. These data offer useful clinical prognosis information for patients. However, the differences in best-case and worst-case survival suggest that the identification of prognostic markers for each disease stage will offer more personalized information, providing individualized survival outcomes and informing management choices. See pages 281 and 495.