### 013

**Semaphorin4D drives CD8+ T cells skin trafficking in oral lichen planus via CXC9 and CXCL10 upregulation in oral keratinocytes**

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**Abstract**

Chemokine-mediated trafficking of CD8+ T cells is essential for T-cell-mediated immune responses. The chemokines CXCL9 and CXCL10 are known to be involved in the trafficking of CD8+ T cells. However, the exact role of these chemokines in oral lichen planus (OLP) remains unclear. In this study, we investigated the role of CXCL9 and CXCL10 in OLP skin disease by analyzing their expression in OLP tissues and blood. We found that CXCL9 and CXCL10 were significantly upregulated in OLP tissues and blood samples compared to control samples. Furthermore, we observed that the expression of CXCL9 and CXCL10 was positively correlated with Sema4D levels in OLP tissues and blood. These results suggest that CXCL9 and CXCL10 may play a crucial role in the recruitment of CD8+ T cells in OLP skin disease.

**Keywords**

Chemokine, CXCL9, CXCL10, CD8+ T cells, OLP

### 014

**A novel role of sensory nerves to establish contact hypersensitivity by promoting cutaneous dendritic cell functions via PACAP**

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**Abstract**

Contact hypersensitivity (CHS) is a type of delayed-type hypersensitivity reaction to a previously introduced antigen. Sensory nerves play a critical role in initiating and regulating CHS responses. In this study, we investigated the role of sensory nerves in the establishment of CHS responses. We found that sensory nerves were involved in the initiation of CHS responses, as shown by reduced CHS responses in mice with sensory denervation. These results suggest that sensory nerves play a crucial role in the establishment of CHS responses.

**Keywords**

Contact hypersensitivity, Sensory nerves, PACAP, Dendritic cells

### 015

**Effects of constant light exposure on contact hypersensitivity in mice**

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**Abstract**

Contact hypersensitivity (CHS) is a type of delayed-type hypersensitivity reaction to a previously introduced antigen. Sensory nerves play a critical role in initiating and regulating CHS responses. In this study, we investigated the role of sensory nerves in the establishment of CHS responses. We found that sensory nerves were involved in the initiation of CHS responses, as shown by reduced CHS responses in mice with sensory denervation. These results suggest that sensory nerves play a crucial role in the establishment of CHS responses.

**Keywords**

Contact hypersensitivity, Sensory nerves, PACAP, Dendritic cells

### 016

**Hyperactivation of Nrf2 contributes to keratinocyte hyperplasia in psoriasis**

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**Abstract**

Psoriasis is an inflammatory skin disease characterized by keratinocyte hyperproliferation of epidermis. Although hyperproliferation-associated keratins including K6, K16 and K17 are considered to be the hallmarks of psoriasis, the underlying mechanism accounting for overexpression of these keratins remains unclear. Nuclear factor erythroid-derived 2 related factor 2 (Nrf2), a transcription factor known for its role in cytoprotection against oxidative stress, has recently been shown to regulate cell proliferation. Therefore, we investigated whether Nrf2 regulates keratinocyte proliferation via hyperproliferation-associated keratins.

**Keywords**

Nrf2, Keratinocytes, Hyperproliferation, Psoriasis

### 017

**The associations of HLA class I and II alleles in bullous pemphigoid**

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**Abstract**

Bullous pemphigoid (BP) is a chronic autoimmune disease that affects the skin. The associations of HLA class I and II alleles with BP have been extensively investigated. In this study, we investigated the associations of HLA class I and II alleles with BP development in a Chinese population. We found that certain HLA class I and II alleles were associated with BP development. These results suggest that HLA class I and II alleles may play a role in the development of BP.

**Keywords**

Bullous pemphigoid, HLA, Associations

### 018

**Intradermal administration of nor epinephrine (NE) biases topical immunization with dinitrofluorobenzene (DNFB) away from IFNγ and II-22 and IL-4 responses toward an IL-17A response**

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**Abstract**

We have previously reported preliminary results showing that exposure of primary dermal dendritic cells (DCs) to NE biased the T-cell response away from pro-inflammatory Th1 and Th2 responses toward a TH17 response. We hypothesized that the ability of DCs to present antigens-specific signals to naive T-cells involved in the initiation of Th17 helper cells. To assess NE effects on immune responses in vivo, naive BALB/c mice were injected at 2 sites on the mid-dorsum, each with 1 µg of NE in phosphate buffered saline (PBS). Mice were then immunized 15 min later with DNFB by application of 100 µl of 1% DNFB in acetone-oil (4:1) at each site injected. Control mice were treated identically except that PBS without NE was injected at the site of immunization. Additional groups of control mice were injected with NE or PBS followed by thymic immunization with acetone-oil alone. Mice were euthanized 3 days after immunization, draining lymph nodes obtained and CD4+ T cells isolated by negative selection using magnetic antibody techniques. CD4+ T cells were cultured with anti-CD3 and anti-CD28 antibodies. Supernatants were harvested at 72 hrs and cytokine content assessed by ELISA. CD4+ T cells from mice immunized with NE-injected sites had a statistically significant increase in IL-17A release along with significant decreases in IL-4, IL-22, and IFNγ production. While the location of action of NE in this type of in vivo experiment is unknown, these results demonstrate that administration of NE into skin biases the induction phase of immunity to DNFB toward an IL-17A response. These results may suggest a role for the stress hormone NE in the regulation of immune responses and immune- mediated disorders in vivo.