

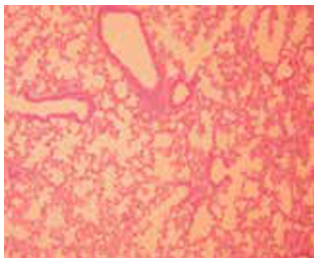
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## Neural-immune receptor cross-talk in the intestine

Bakri Assas and colleagues discuss cross-talk between neural and immune receptors in controlling immune homeostasis in the intestine. They propose a mechanism by which Gram-negative bacterial signaling via Toll-like receptor 4 can activate the sensory vanilloid receptor 1 via intracellular signaling, thereby inducing the release of anti-inflammatory calcitonin gene-related peptide to maintain mucosal homeostasis. [See page 1283](#)

## Regulatory T cells promote Th17 responses to *C. rodentium*

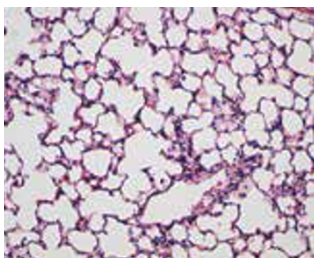
Zuobai Wang *et al.* demonstrate that CD4<sup>+</sup> Foxp3<sup>+</sup> regulatory T cells promote T helper type 17 cell (Th17) responses to intestinal infection with the mouse pathogen *Citrobacter rodentium*. [See page 1290](#)



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## Malaria suppresses *Salmonella*-induced intestinal inflammation

Jason Mooney and colleagues found that interleukin-10 production during malaria infection suppressed inflammatory responses to intestinal infection with nontyphoidal *Salmonella* infection. [See page 1302](#)



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## NOD2 signaling inhibits NF-κB signaling

Tomohiro Watanabe and colleagues show that activation of nucleotide-binding oligomerization domain 2 (NOD2) results in increased expression of interferon regulatory factor 4 and binding to tumor necrosis factor receptor-associated factor 6 (TRAF6) and receptor interacting serine-threonine kinase (RICK), resulting in inhibition of Lys63-linked polyubiquitination of TRAF6 and RICK and thus to downregulation of nuclear factor-κB (NF-κB) activation. [See page 1312](#)

## cHMOS regulates signaling in the immature intestine

Yingying He and co-workers demonstrate that human milk oligosaccharides from colostrum (cHMOS) can directly modulate the signaling pathways of the immature mucosa. [See page 1326](#)

## Claudin-2 regulates intestinal homeostasis

Rizwan Ahmad and colleagues identified a critical role of claudin-2 in intestinal homeostasis: regulating epithelial permeability, inflammation, and proliferation. [See page 1340](#)

## α4 Integrins in colitis

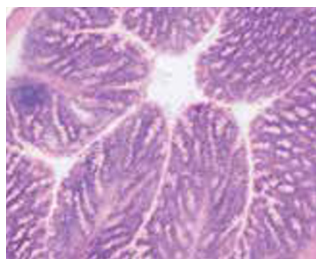
Elvira Kurmaeva *et al.* demonstrate a direct role for T cell-associated α<sub>4</sub>β<sub>7</sub>, but not α<sub>4</sub>β<sub>1</sub>, integrins during initiation and perpetuation of chronic colitis in mice. They found that anti-α<sub>4</sub> (natalizumab) treatment reduced mucosal CD4 T-cell accumulation in patients with Crohn's disease. [See page 1354](#)

## IL-16 and *S. aureus* pneumonia

Danielle Ahn *et al.* provide evidence that the production of interleukin-16 (IL-16) by CD4<sup>+</sup> T cells contributes to immune pathology and inhibits bacterial clearance in *Staphylococcus aureus* pneumonia. [See page 1366](#)

## HIV targeting of Th17 cells in the female reproductive tract

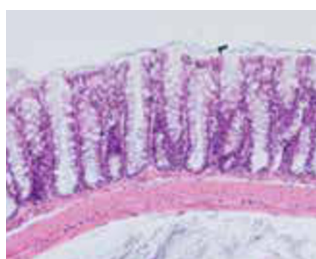
Marta Rodriguez-Garcia and colleagues describe the phenotype of T helper type 17 (Th17) cells in the female reproductive tract and demonstrate increased susceptibility to HIV infection *in vitro* of cells expressing CCR5 and CD90. [See page 1375](#)



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## DCs and lung injury in the elderly

Sangeetha Prakash and co-workers observed that monocyte-derived dendritic cells (DCs) from elderly, but not young, subjects produced tumor necrosis factor- $\alpha$  that was able to induce cytokines and chemokines and alter the barrier function of primary bronchial epithelial cells. [See page 1386](#)



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## T-cell IL-4 drives Th2 cells in food allergy

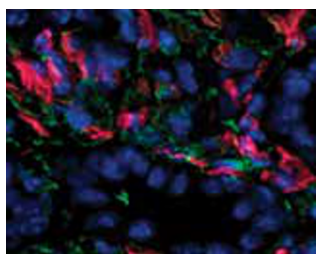
Derek Chu *et al.* show that the priming of T helper type 2 (Th2) cell responses in the intestine to initiate food allergy in mice is dependent on interleukin-4 (IL-4) produced by the T cells themselves and not by other innate sources and is driven by OX40L expressed by dendritic cells. [See page 1395](#)

## Vitamin D and Crohn's disease

Serge Dionne and colleagues report a differential effect of vitamin D on Toll-like receptor- and nucleotide-binding oligomerization domain 2-induced cytokine production by mononuclear phagocytes from patients with Crohn's disease. [See page 1405](#)

## LXR $\beta$ protects against colitis in mice

Tomas Jakobsson and co-workers found that the oxysterol receptor LXR $\beta$  protected against dextran sodium sulfate- and 2,4,6-trinitrobenzene sulfonic acid-induced colitis in mice. [See page 1416](#)



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## SHIP1 promotes mucosal T-cell survival

Mi-Young Park and colleagues demonstrate that SHIP1 is important for the survival of protective T cells at mucosal surfaces, thus preventing abnormal myeloid cell activation and inflammation. [See page 1429](#)

## Foxp3<sup>+</sup> Tregs induce epithelial cell proliferation in the lung

Using an experimental model of lung injury, Jason Mock *et al.* obtained evidence that CD4<sup>+</sup> Foxp3<sup>+</sup> regulatory T cells (Tregs) contribute to epithelial cell proliferation and epithelial repair. [See page 1440](#)

## CHOP regulates epithelial cell proliferation

Nadine Waldschmitt and colleagues show a role for the unfolded protein response target gene C/EBP homologous protein (CHOP) in regulating epithelial cell proliferation. They found that overexpression results in poor epithelial repair in dextran sodium sulfate-induced colitis and experimental wounding. [See page 1452](#)

## SIRT1 in control of colitis

Roberta Caruso and colleagues combined analyses of human inflammatory bowel disease and mouse models to identify a role for SIRT1, an NAD-dependent deacetylase, in the control of intestinal inflammation. [See page 1467](#)

## Caspase-11 protects against DSS colitis

Dieter Demon and co-workers demonstrate that caspase-11 is expressed in the colonic mucosa and protects against dextran sodium sulfate-induced (DSS) colitis, through a mechanism that is independent of inflammasome activation of interleukin-1 $\beta$  and -18. [See page 1480](#)

## Blocking TL1A/DR3 inhibits intestinal fibrosis

David Shih *et al.* report evidence of a direct role for TL1A, a product of the inflammatory bowel disease-susceptibility gene *TNFSF15*, in mediating colitis-induced intestinal fibrosis in a mouse model. They observed that inhibition of TL1A/DR3 signaling *in vivo* could block the fibrotic response. [See page 1492](#)