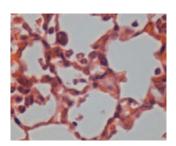


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T. gondii, microbiota, and mucosal immunity

Sara Cohen and Eric Denkers discuss recent progress in elucidating the mechanisms governing inflammatory and protective immune responses following intestinal infection with *Toxoplasma gondii*, an important and prevalent human pathogen. See page 744

Intestinal helminth's effects on systemic immunity

Pankaj Mishra and colleagues discuss current data demonstrating the effect of intestinal helminth infections on systemic immune responses, including allergic and autoimmune inflammation. See page 753

Inflammasomes and *C. rodentium* infection

In companion papers, George Song-Zhao and Sofia Nordlander and their colleagues demonstrate essential roles for NLRP3 and NLRC4 in the intestinal epithelium in innate immunity to infection with *Citrobacter rodentium*. See pages 763 and 775

Retinoic acid inhibits inflammatory Th2 cells

Aya Yokota-Nakatsuma and co-workers present results indicating that retinoic acid inhibits allergic responses to oral antigens by preventing dendritic cells from mesenteric lymph nodes from inducing interleukin-13-producing inflammatory T helper type 2 cells. See page 786

C. jejuni strains and autoimmunity

Ankit Malik and fellow investigators report bacterial strain–specific immune responses against *Campylobacter jejuni* that result in intestinal inflammation or the development of autoantibodies in mouse models. **See page 802**

Primary mouse epithelial cell monolayer culture system

Clara Moon and colleagues describe a new method to generate monolayer cultures of primary mouse intestinal epithelial cells. Using cells from knockout mice, they found that interleukin-17 is a particularly potent inducer of polymeric immunoglobulin receptor expression and immunoglobulin A transcytosis. See page 818

Inflammasome activation by *E. histolytica*

Leanne Mortimer and co-workers demonstrate, for the first time, inflammasome activation in macrophages by invasive *Entamoeba histolytica*, an effect dependent on galactose lectin–mediated cell contact. See page 829

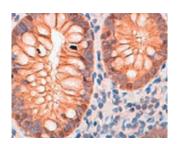
IL-23 drives intestinal tumorigenesis through ILC effects

Using mice overexpressing interleukin-23 (IL-23), Ivan Chan and colleagues show that this cytokine is sufficient to induce rapid *de novo* gut tumorigenesis, independent of carcinogens, through activation of innate lymphoid cells (ILCs). See page 842

Inhibition of McI-1 improves lung injury

Christopher Lucas *et al.* demonstrate that the intracellular protein myeloid cell leukemia 1 (Mcl-1) contributes to lung inflammation by inhibiting neutrophil apoptosis, and that its inhibition results in increased resolution of lung injury while preserving bacterial clearance by macrophages following administration of *Escherichia coli*.

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Plexin C1 mediates lung injury

IL-10 acts on macrophages in

Bofeng Li and colleagues show an essential role for

interleukin-10 (IL-10) signaling in macrophages in

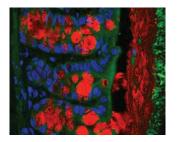
protecting mice from dextran sodium sulfate (DSS)-

induced colitis largely through its effects on nitric oxide

DSS colitis

production. See page 869

Using an experimental lung injury model, Tiago Granja and co-workers found that Plexin C1, the receptor for the guidance protein Semaphorin 7A, is expressed on neutrophils and is important in lung injury, as genetic deletion and functional inhibition of the receptor decreased lung inflammation. See page 879

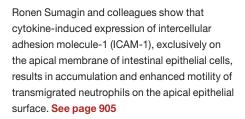


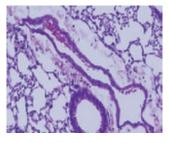
p 939

Inhibition of Peyer's patch lymphocyte egress in Salmonella infection

Olga Schulz and colleagues determined that hypertrophy of Peyer's patches following *Salmonella typhimurium* infection results from interferon-mediated inhibition of lymphocyte egress. **See page 892**







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Tregs and mucosal fungi

Petra Bacher *et al.* observed a substantially expanded population of T regulatory cells (Tregs) following mucosal fungal infection. In addition, they identified a unique capacity of *Aspergillus fumigatus* to selectively generate Treg responses that may prevent allergic responses to this human pathogen.

See page 916

Hypoxic macrophages impair epithelial cell autophagy

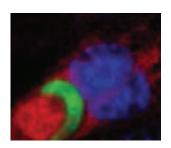
Dolores Ortiz-Masiá and colleagues demonstrate a hypoxia-inducible factor-1-dependent induction of Wnt1 in hypoxic macrophages that undermines autophagy in epithelial cells, as observed in patients with inflammatory bowel disease. See page 929

REG3γ and intestinal inflammation

Linda Loonen and co-workers present evidence that mice deficient in regenerating islet–derived 3-gamma (REG3γ) have impaired mucus distribution and increased mucosal inflammatory responses in the ileum. See page 939

FDC-SP controls IgA production

Sen Hou and colleagues describe previously unreported biological activity of follicular–dendritic cell–secreted protein (FDC-SP) in controlling B-cell immunoglobulin A (IgA) production and identify FDC-SP-deficient mice as a novel mouse model of IgA nephropathy. See page 948



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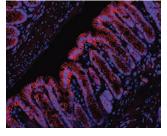
TL1A drives ILC2s in allergic inflammation

François Meylan and colleagues identify costimulation of type 2 innate lymphoid cells (ILC2s) as a novel function of the tumor necrosis factor–family cytokine TL1A that is important for allergic lung disease. See page 958

IFN-γ and *E. falciformis* development

Manuela Schmid and colleagues demonstrate opposing roles for interferon-γ-mediated pathways in the development of the intestinal epithelial cell parasite *Eimeria falciformis* in mouse cecum.

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Altered intestinal microbiome and immune activation in HIV infection

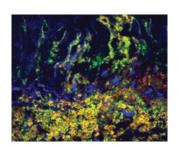
Stephanie Dillon and colleagues show that an altered intestinal mucosal microbiome in HIV-1 infection is associated with mucosal and systemic immune activation and endotoxemia. See page 983

Epithelial Notch-1 regulates mucosal immune responses

Douglas Mathern *et al.* found that epithelial cell Notch-1 expression modulates mucosal chemokine and cytokine secretion as well as FoxP3 and effector T-cell responses. **See page 995**

Soluble CD83 ameliorates colitis

Using a 2, 4-dinitrobenzene sulfonic acid (DNBS)-induced colitis model, Jenny Eckhardt and co-workers observed that CD83, a membrane molecule of mature dendritic cells, was induced on intestinal leukocytes and that soluble CD83 ameliorated colitis development. See page 1006



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