■ IMMUNOLOGY

## ILCs in the margins

Innate lymphoid cells (ILCs) are found in the spleen, where they promote antibody production by marginal zone B cells, according to a recent study in *Nature Immunology* (**15**, 354–364, 2014).

ILCs are commonly found at barrier surfaces where, in response to environmental stimuli, they provide an important source of cytokines that prime the development and function of immune cells. Although ILCs have been shown to support antibody production at mucosal surfaces, whether these cells also function in the spleen is not clear.

Andrea Cerutti and his colleagues now report that in humans, ILCs are found within the marginal zone of the spleen and exhibit a phenotype similar to mucosal ILCs. These ILCs interact with and receive survival signals from stromal cells within the spleen that help promote their function. ILCs are positioned near B cells and promote the survival and differentiation of marginal zone B cells and plasma cells via B cell–activating factor (BAFF), CD40 ligand and the Notch ligand delta-like ligand 1 (DLL1). In addition, ILCs help recruit neutrophils to the spleen, which, in turn, promotes antibody production. In the absence of splenic ILCs, T cell–independent antigen antibody production is impaired. These findings suggest ILCs provide crosstalk between stromal cells and different arms of the immune system to support antibody production. —*KDS*

■ ADULT NEUROGENESIS

## Making new neurons

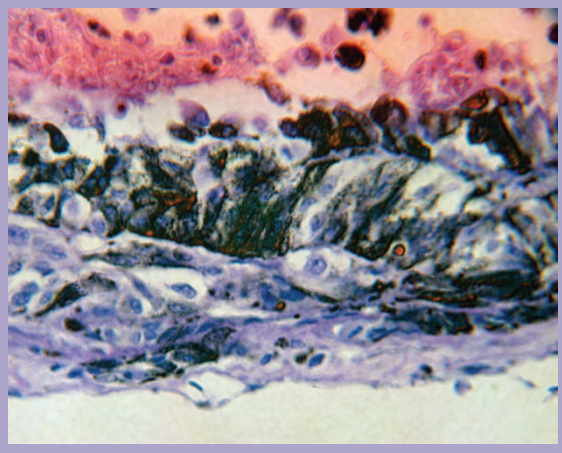
The generation of neurons from precursor cells occurs in the hippocampus throughout the lifespan of humans, but the extent to which this occurs in other brain structures in adult primate species has ignited much controversy**.** Now, Aurélie Ernst and her colleagues report that the adult human striatum is capable of generating new interneurons (*Cell* **156**, 1072–1083, 2014).

The researchers obtained postmortem tissue from individuals who had received a thymidine analog as part of cancer treatment; cells incorporating the analog are assumed to have divided during the course of the treatment. They found that some of the dividing cells in the striatum also expressed neuronal markers. They then carried out carbon-14 dating and cell nuclei sorting techniques in postmortem tissue. This determined that striatal interneurons tended to be the striatal neuron subtype that indicated they were generated sometime after the birth of the individuals, and that the rate of neuronal formation of this type tended to be much lower in patients with the neurodegenerative disorder Huntington's disease, which affects this brain region.

The extent to which these late-forming neurons contribute to striatal function or to regeneration after injury in humans remains an open question. —*EC*

METASTASES

## Lighting the way to melanoma metastasis



Dr. Cecil H. Fox / Science Source

**A recent study (*Nature* 507, 109–113, 2014) has found that ultraviolet (UV) radiation promotes melanoma progression by creating an inflammatory microenvironment that fosters metastatic spread.**

**UV exposure is known to underscore the mutational profile of melanoma cells. However, using a genetically engineered mouse model of melanoma, the authors observed that UV radiation–induced damage of normal keratinocytes caused an inflammatory response that has more effect on metastatic progression than on primary tumor growth.**

**The authors unravel a role for the innate immune system in sensing this UV-induced DNA damage. Irradiated keratinocytes release the nuclear protein HMGB1, which serves as a chemoattractant for neutrophils. In turn, neutrophils secrete factors that promote angiogenesis and angiotropic growth of melanoma cells, contributing to their spread.**

**The relevance of the findings to human disease, as well as their therapeutic application, needs further exploration, but the authors were able to show that ulcerated tumors, or those with evident neutrophil infiltration, bear a higher risk of metastasis. This suggests that the metastasis mechanism uncovered in the study plays a role in the context of UV-induced skin tumorigenesis in humans. —*VA***

■ MICROBIOTA

## Dysbiosis as a diagnostic

A recent study analyzing the intestinal microbiota in new-onset cases of Crohn's disease provides insights into patterns of dysbiosis associated with Crohn's and a means with which to diagnosis the disease.

Several studies have reported that Crohn's disease is associated with an alteration in the composition of the intestinal microbiota. However, several factors, including small sample size, the use of patients with established disease and reliance on sampling from a single site, has made physicians question their relevance to clinical application.

Ramnik J. Xavier and his colleagues (*Cell Host Microbe* **15**, 382–392, 2014) assessed a large group of treatment-naive, new-onset cases of Crohn's disease. By assessing both the intestinal mucosal and lumen-associated microbiota, they found that there was a reduction in species richness and alterations in the abundance of several bacteria in comparison to microbiota from healthy individuals. By assessing both fecal and tissue samples, they found that these changes in the microbiota were not well-reflected in the stool samples. With receiver operating characteristic (ROC) analysis, samples from the ileum, and to a lesser extent, rectal biopsies, could be used to diagnose disease. Dysbiosis also correlated with disease severity and was exacerbated with antibiotic usage. Taken together, these findings suggest future studies are warranted to examine whether dysbiosis can be used to diagnose disease at an early stage. —*KDS*