■ CANCER

Oncogenic CMV

A viral protein from cytomegalovirus (CMV) may have protumorigenic effects (*J. Clin. Invest.* doi:10.1172/JCI42563).

CMV is broadly present in humans. It can infect several tissues, including gastrointestinal cells. Previous evidence suggested that latent CMV might disrupt normal growth and induce transformation *in vitro*. Gerold Bongers *et al.* developed a mouse engineered to express US28, a CMV protein resembling human cytokine receptors, in intestinal epithelial cells.

Using this model, the authors recreated the phenotypic changes that might arise from persistent CMV infection, enabling them to isolate the molecular events driving such changes. US28 increased the proliferation of intestinal cells, eventually leading to the development of tumors in some mice. Interestingly, the presence of the viral protein also exacerbated cancer development in conditions mimicking human colitis. The effects of US28 correlated with alterations in the Wnt signaling pathway, a known perpetrator of intestinal tumorigenesis, and probably involve the engagement of inflammatory cytokines.

This study raises awareness of the potentially harmful effects of immunosuppressive therapies on individuals with cancer-prone colitis and sheds light on the silent contribution of ubiquitous CMV to carcinogenesis.—*VA*

■ NEUROSCIENCE

Tau turnover

The microtubule-binding protein tau accumulates in many neurodegenerative conditions, leading to neuronal dysfunction. Sang-Won Min *et al.* now report that acetylation of tau enhances its stability by reducing its degradation by the proteasome (*Neuron* **67**, 953–966).

The researchers found that tau acetylation is increased in humans with Alzheimer's disease and in mice expressing human tau. The acetyltransferase p300 seems to be involved in this acetylation, because drugs that block this enzyme reduced tau acetylation in culture. The expression of the deacetylase enzyme sirtuin-1 (SIRT1) decreases with aging, and SIRT1-deficient mice had increased levels of acetylated tau. Drugs that inhibit SIRT1 activity decreased the proteasomal degradation of tau.

Modulating tau acetylation may be a unique mechanism to affect tau accumulation, but it is unclear whether reducing acetylation would ameliorate cognitive deficits in diseases in which tau accumulates.—*EC*

■ METABOLISM

Macrophage meet-up

Macrophages accumulate in the adipose tissue of obese individuals, where they contribute to insulin resistance, but the mechanisms that lead to this accumulation have been unclear. Aliki Kosteli *et al.* now implicate lipolysis as a key factor in recruiting macrophages to adipose tissue (*J. Clin. Invest.* **120**, 3466–3479).

Macrophage numbers in adipose tissue decrease after weight loss. The new wrinkle of this study is that, soon after weight loss or fasting in mice, macrophages in fat paradoxically increased. At these early time points, lipolysis was triggered, causing macrophage recruitment. The recruited macrophages accumulated lipid and dampened lipolysis.

As discussed by the authors, the concept that macrophages buffer lipid accumulation in adipose tissue is reminiscent of their role in atherosclerosis, where an adaptive role of macrophages in clearing lipid from the vessel wall becomes maladaptive under high lipid burden. The identity of the lipolysis-associated signals that lead to macrophage accumulation remains to be established.—*MB*

■ IMMUNOLOGY

Optimus (T_H2) prime

Two reports suggest that dendritic cells (DCs) are key players in orchestrating a T helper type 2 (T_H2) immune response.

Basophils can promote T_H2 responses to allergens and helminth infection by processing and presenting antigens and producing interleukin-4 (IL-4). Although DCs are antigen-presenting cells, they are not a major source of IL-4 and are dispensable during T_H2 priming.

Hamida Hammad *et al.* (*J. Exp. Med.*, **207**, 2097–2111) now report that depletion of basophils only partly reduces T_H2 responses to house dust mite allergen. They suggest that a small population of inflammatory DCs are a source of IL-4 and are crucial for T_H2 polarization. Alexander Phythian-Adams *et al.* (*J. Exp. Med.*, **207**, 2089–2096) in turn found that DCs are necessary and sufficient to induce a T_H2 response to helminth infection. In both studies, depletion of DCs impaired T_H2 responses and IL-4 production.

These studies do not rule out a role for basophils in amplifying the immune response, but they do suggest that it may be premature to rule out the contribution of DCs and other innate cell types during T_H2 priming.—*KDS*

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New from NPG

Non-muscle myosin IIA is a functional entry receptor for herpes simplex virus-1.
Arii, J. *et al. Nature* **466**, 859–862.
Nonmuscle myosin heavy chain IIA functions as an entry receptor for herpes simplex virus-1 by interacting with glycoprotein B, another known receptor for this virus.

Angiocrine factors from Akt-activated endothelial cells balance self-renewal and differentiation of haematopoietic stem cells.
Kobayashi, H. *et al. Nat. Cell Biol.* doi:10.1038/ncb2108 (24 October).
Akt activation in endothelial cells upregulates factors that support expansion of hematopoietic stem and progenitor cells with long-term repopulation capacity.

A multi-stage genome-wide association study of bladder cancer identifies multiple susceptibility loci.
Rothman, N. *et al. Nat. Genet.*, doi:10.1038/ng.687 (24 October).
Three new regions associated with bladder cancer map to chromosomes 22q13.1, 19q12 and 2q37.1, according to this genomic analysis.

Efficient CNS gene delivery by intravenous injection.
Louboutin, J.-P. *et al. Nat. Methods* doi:10.1038/nmeth.1518 (17 October).
Intravenous injection of recombinant SV40-derived viral vectors, particularly with mannitol pretreatment, results in extensive expression of multiple transgenes throughout the brain.

IL-37 is a fundamental inhibitor of innate immunity.
Nold, M.F. *et al. Nat. Immunol.* doi:10.1038/ni.1944 (10 October).
In a model of sepsis, interleukin-37 interacts with Smad3 to function as a suppressor of inflammatory responses.

Wild-type and mutant SOD1 share an aberrant conformation and a common pathogenic pathway in ALS.
Bosco, D.A. *et al. Nat. Neurosci.* doi:10.1038/nn.2660 (17 October).
Mutations in superoxide dismutase (SOD) cause familial amyotrophic lateral sclerosis (ALS). Oxidized wild-type SOD and mutant SOD share a conformational epitope absent from wild-type SOD1, indicating that oxidized SOD could be pathogenic in sporadic forms of ALS.