IN BRIEF

IMMUNITY

Lipid accumulation and dendritic cell dysfunction in cancer

Herber, D. L. *et al. Nature Med.* 11 Jul 2010 (doi:10.1038/nm.2172)

The authors report that dendritic cells from humans and mice with tumours show lipid accumulation, which is caused by an increased uptake of extracellular lipids owing to the upregulation of scavenger receptor A. Dendritic cells with accumulated lipids showed impaired antigen presentation and T cell stimulation. An inhibitor of acetyl-coA carboxylase normalized the levels of lipids in dendritic cells and restored their functional activity. These results indicate that immune responses against cancer cells can be modified by altering lipid levels in dendritic cells.

THERAPY

Enhancing tumor-specific uptake of the anticancer drug cisplatin with a copper chelator

Ishida, S. *et al. Cancer Cell* **17**, 574–583 (2010)

Hanahan and colleagues showed that the expression of the copper transporter *CTR1* is associated with responses to platinum-based therapies in human ovarian tumours. In a mouse model of cervical cancer, combined treatment using cisplatin and a copper chelator selectively increased the formation of cisplatin–DNA adducts in tumour tissues and improved therapeutic efficacy. The chelator also enhanced cisplatin-mediated cytotoxicity of human ovarian and cervical cancer cells *in vitro*. This study therefore suggests that copper chelators might be used to improve platinum-based chemotherapy.

LYMPHOMA

ATM-deficient thymic lymphoma is associated with aberrant *tcrd* rearrangement and gene amplification

Zha, S. *et al. J. Exp. Med.* **207**, 1369–1380 (2010)

Ataxia telangiectasia-mutated (ATM) deficiency predisposes to T cell lymphomas that have recurrent chromosome 14 translocations involving the T cell receptor-α/δ (*TCRA/D*) locus. In addition to this known translocation, Alt and colleagues identified recurrent translocations and amplifications involving a centromeric region of chromosome 14 that extends to the 5′ boundary of *Tcra/d* in mice with ATM-deficient thymic lymphomas. These translocations and amplifications are associated with V(D)J recombination-initiated breaks in the *Tcrd* locus, thus revealing new mechanisms that are involved in ATM-deficient T cell lymphomagenesis.

ANGIOGENESIS

New mechanism for notch signaling to endothelium at a distance by Delta-like 4 incorporation into exosomes

Sheldon, H. *et al. Blood* 17 Jun 2010 (doi:10.1182/blood-2009-08-239228)

Delta-like 4 (DLL4) is a Notch ligand that regulates the differentiation of endothelial cells during angiogenesis. Sheldon and colleagues show that DLL4 can be incorporated into exosomes *in vivo* and in cancer cell lines that overexpress DLL4. The exosomes transfer DLL4 from the tumour cells to endothelial cells to promote angiogenic branching.