IN BRIEF

IMMUNE TOLERANCE

Limiting T cell access protects the fetus

Previous studies have described several mechanisms of feto-maternal tolerance that minimize the activation of naive T cells specific for fetal or placental antigens. In this study, the authors found that re-activation of memory T cells with known fetal or placental specificity in early pregnancy did not result in fetal rejection, suggesting the existence of additional tolerance mechanisms. Unlike myometrial stromal cells, decidual stromal cells (DSCs) — which encapsulate the fetus and placenta — were shown to express only very low levels of the T cell chemoattractants CCL5, CXCL9 and CXCL10 under inflammatory conditions *in vivo*, thereby preventing T cell accumulation in the decidua. This specific reduction in chemokine expression by DSCs was due to repressive histone modifications in the promoters of the encoding genes. Thus, limiting effector T cell accumulation within the decidua is an additional mechanism of feto-maternal tolerance.

ORIGINAL RESEARCH PAPER Nancy, P. *et al.* Chemokine gene silencing in decidual stromal cells limits T cell access to the maternal-fetal interface. *Science* **336**, 1317–1321 (2012)

MUCOSAL IMMUNOLOGY

Milk fat can inflame the gut

The increase in the incidence of chronic inflammatory disorders over the past half century is thought to be due to changes in lifestyle. This study shows that consumption of a diet high in saturated (milk-derived) fat results in the specific outgrowth of a low-abundance, sulphite-reducing pathobiont, *Bilophila wadsworthia*, in the gut. This was associated with a T helper 1-type immune response and an increase in disease in colitis-susceptible ($Il10^{-/-}$) mice but not wild-type mice. The milk-fat diet promoted the conjugation of bile acids with taurine, resulting in an increased availability of organic sulphur and thus growth of *B. wadsworthia*. Indeed, administration of taurocholic acid mimicked the effect of the milk-fat diet on *B. wadsworthia* growth and colitis in $Il10^{-/-}$ mice. So, dietary fats, by affecting bile acid composition, can result in microbiota dysbiosis and intestinal inflammation in genetically susceptible individuals.

ORIGINAL RESEARCH PAPER Devkota, S. *et al.* Dietary-fat-induced taurocholic acid promotes pathobiont expansion and colitis in $Il10^{-/-}$ mice. *Nature* 13 Jun 2012 (doi:10.1038/nature11225)

DENDRITIC CELLS

Distinguishing classical dendritic cells

Two research groups, headed by Murphy and Nussenzweig, have identified the zinc-finger transcription factor ZBTB46 (also known as BTBD4) as being selectively expressed by classical dendritic cells (cDCs) and their committed progenitors. ZBTB46 was not expressed by monocytes, macrophages, plasmacytoid DCs or other immune cell lineages. Although ZBTB46 is not required for cDC development, it enforces cDC lineage restriction by repressing the expression of alternative myeloid growth factor receptors. Furthermore, the generation of mice in which cDCs were specifically depleted showed that these cells are only partially required for the initiation of immunity against infection and tumours. These mice, and others generated by the two groups, will be useful tools in helping to delineate the exact functions of cDCs in the immune system.

ORIGINAL RESEARCH PAPERS Satpathy, A. T. *et al. Zbtb46* expression distinguishes classical dendritic cells and their committed progenitors from other immune lineages. *J. Exp. Med.* **209**, 1135–1152 (2012) | Meredith, M. M. *et al.* Expression of the zinc finger transcription factor zDC (Zbtb46, Btbd4) defines the classical dendritic cell lineage. *J. Exp. Med.* **209**, 1153–1165 (2012)

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