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IN BRIEF

T HELPER CELLS

Differential glycosylation of T_H1, T_H2 and T_H-17 effector cells selectively regulates susceptibility to cell death.

Toscano, M. A. *et al. Nature Immunol.* 24 June 2007 (doi:10.1038/ni1482)

T-cell differentiation is accompanied by a programmed remodelling of cell-surface glycans, such that each effector-cell type has a unique glycosylation 'signature'. This study shows that differential glycosylation of T helper 1 (T_H1), T_H2 and T_H17 cells regulates their susceptibility to cell death induced by the glycan-binding protein galectin-1. Whereas T_H1 and T_H17 cells were shown to be susceptible to galectin-1-induced cell death, T_H2 cells were protected, owing to differential sialylation of cell-surface glycoproteins. Accordingly, compared with wild-type mice, galectin-1-deficient mice showed greater T_H1- and T_H17-cell responses and higher susceptibility to experimental autoimmune encephalomyelitis. Together with emerging evidence that galectin-1 contributes to the immunosuppressive activity of regulatory T cells, this study provides support for a key role of galectin-1 in the regulation of T-cell homeostasis.

INNATE IMMUNITY

The inflammasome mediates UVB-induced activation and secretion of interleukin-1β by keratinocytes.

Feldmeyer, L. *et al. Curr. Biol.* **17**, 1140–1145 (2007)

The precursor proteins pro-interleukin-1α (pro-IL-1α) and pro-IL-1β can be produced by human keratinocytes and are activated and released in response to UV irradiation. How the maturation and secretion of IL-1 in keratinocytes is regulated, however, is unknown. Previous work suggested that keratinocytes can express proteins that belong to a multiprotein innate immune complex known as the inflammasome, and that these proteins are responsible for pro-IL-1β maturation and secretion. Here, Feldmeyer *et al.* found that keratinocytes do express inflammasome proteins together with pro-IL-1α, pro-IL-1β and IL-18. The activation of the IL-1β-converting enzyme caspase-1 requires the enhancement of intracellular free Ca^{2+} by UVB irradiation. Caspase-1 then activates pro-IL-1β, resulting in the secretion of IL-1β and other inflammasome components. The presence of a pro-IL-1β-processing inflammasome in keratinocytes suggests that these are important immunocompetent cells.

T CELLS

Bim/Bcl-2 balance is critical for maintaining naive and memory T cell homeostasis.

Wojciechowski, S. *et al. J. Exp. Med.* **204**, 1665–1675 (2007)

Wojciechowski *et al.* examined the roles of the anti-apoptotic molecule B-cell lymphoma 2 (BCL-2) and the pro-apoptotic BCL-2 family member BIM (BCL-2-interacting mediator of cell death) in controlling naive and memory T-cell homeostasis. *Bim*$^{+/-}$*Bcl2*$^{-/-}$ mice had substantially fewer naive T cells than control mice, and normal numbers were largely restored by the loss of the remaining *Bim* allele. Thymectomy experiments showed that BCL-2 is required for the maintenance of peripheral $CD8^+$ (but not $CD4^+$) naive T-cell survival by antagonizing BIM. Lymphocytic choriomeningitis virus infection did not seem to affect the ability of *Bim*$^{+/-}$*Bcl2*$^{-/-}$ mice to generate normal numbers of memory T cells, although lymphopenia-driven proliferation probably caused these cells to accumulate. Together, these results indicate that homeostasis of naive and memory T cells depends on a balance between BIM and BCL-2.