SIgN Immunology Seminar

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The regulatory role of B effector cells in infectious disease models

Host
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Date
Friday
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Time
11am – 12pm

Venue
SIgN Seminar Room
Immunos Building
Level 4
Biopolis

B cells are essential for humoral immunity, but the role that they have in regulating CD4+ T cell responses remains controversial. Recent data showed that transient B cell depletion influences induction, maintenance and reactivation of CD4+ T cells, mainly by antibody independent regulatory (Breg)and effector (Be) B cells. These findings revoked new interest in the many roles of B cells in both infectious and autoimmune diseases. Similar to T helper 1/2 cell dichotomy, B effector cells can be stimulated in vitro by IL-12 and IFN-g to Be1 cells, or by IL-4 to Be2 cells with a specific cytokine profile. To further study the influence of B cell in vivo, we established conditional ($Mb1^{Cretg}IL\text{-}4Ra^{-/lox}$) Balb/c mice, where IL-4 responsiveness was impaired only in B cells. IImpairing Be2 differentiation, resulted in dramatic changes in health and disease for Type 1 and Type2 infectious disease models. In cutanous leishmaniasis (Type1 protective), infected with L. major, non-healer mice converted to healer mice in the absence of IL-4-responsive B cells. The contrary was observed during Schistosoma mansoni infection, causing bilharzia, where resistant wild type mice (Type2 protective) became hypersusceptible to the disease, in the absence of IL-4 responsive B cells. This was caused by dramatic modulation of T cell helper cytokine responses early during disease, strongly suggesting that B effector cells tipping the in vivo T cell dichotomy, thereby crucially influencing the out come of the infectious disease.