SIgN Immunology Seminar



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Primary and secondary B cell activation

Host
Dr Katja Fink
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Date
Thursday 17 December 2015

Time
11am – 12pm

Venue
SIgN Seminar Room
Immunos Building
Level 4
Biopolis

Antibody-mediated immune responses rely on the activation of the B cell antigen receptor (BCR). Since many years, our group studies the molecular details of how the ligated BCR connects to its intracellular effector proteins and how they orchestrate downstream signaling cascades. Several positive and negative regulators of BCR signaling were identified such as SLP65 or NTAL, respectively. More recently, we identified the ubiquitously expressed adaptor Cbl-interacting protein of 85 kDa (CIN85) as a novel regulator of BCR-induced NF-kB activation and marker for X-linked antibody deficiency in humans. Another research focus of our lab assesses the molecular details that facilitate B cell memory responses upon antigen recall. This phenomenon provides the fundamental basis of successful vaccinations. We detected a signal amplification module within IgG- and IgE-BCR classes that are expressed on memory B cells. Targeted disruption of the underlying signaling motif, named immunoglobulin tail tyrosine (ITT), in the mouse IgG1-BCR resulted in severely compromised IgG1 memory responses. Our data provide a first mechanism as to how B cells remember their cognate antigen, and consequently, why memory B cell responses are less dependent on T cell help than primary antibody responses.