

ImmunoTools IT-Box-139 Award 2013



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The Role of Innate Lymphoid Cells in Intestinal Inflammation

A breakdown in intestinal homeostasis due to dysregulated immune responses against intestinal bacteria is thought to underlie the pathogenesis of inflammatory bowel disease (IBD) in genetically susceptible individuals. Approximately 1 in 1000 people suffer from IBD currently and disease incidence is rising, indicating the need for a better understanding of the immune pathways mediating this chronic inflammatory condition. Crohn's disease (CD) and ulcerative colitis (UC), the two main forms of IBD, are highly debilitating relapsing-remitting conditions characterized by recurrent abdominal pain, diarrhea, intestinal bleeding and malnutrition. No curative treatments are available and despite the availability of immunosuppressive agents, the rate of surgical intervention remains high.

IL-23 driven immune responses are crucially involved in the pathogenesis of IBD. While early interest focused mainly on IL-23-driven T cell responses, innate lymphoid cells (ILCs) were subsequently shown to play pathological roles in two mouse models of innate-driven colitis.

However, the potential of ILCs to contribute to disease pathogenesis in a lymphocyte-replete setting as is usually found in humans is not well understood. While ILCs are an extremely rare population of immune cells, they may act as amplifiers of developing immune responses, bridging initial responder cells and adaptive immune cells.

This project aims to investigate three main areas of ILC biology by means of *ex vivo* and *in vitro* analyses of human ILC populations:

- (1) Signals (cytokines, TLR agonists) that regulate ILC function and plasticity
- (2) Functional effects of ILC-stromal interactions
- (3) Functional effects of ILC-adaptive interactions

Using flow cytometry, ILCs are phenotypically defined as Lineage-CD45⁺CD127⁺ cells (lineage markers include CD1a, CD3, CD4, CD8, CD14, CD16, CD11c, CD19, CD20, HLA-DR). Distinct ILC subsets can then be identified on the basis of the expression of NK cell receptors such as CD56. **ImmunoTools** antibodies would

greatly aid in these flow cytometry-based *ex vivo* and *in vitro* examinations of human intestinal ILC populations.

ImmunoTools *IT-Box-139.3* for **Anna-Lena Schaupp** includes 100 antibodies

FITC - conjugated anti-human CD1a, CD2, CD3, CD4, CD5, CD6, CD7, CD8, CD9, CD11a, CD11b, CD14, CD15, CD16, CD18, CD19, CD21, CD25, CD29, CD36, CD41a, CD43, CD45, CD45RA, CD46, CD52, CD53, CD54, CD58, CD62p, CD63, CD69, CD71, CD80, CD86, CD95, CD235a, HLA-ABC, HLA-DR, IL-6, Control-IgG1, Control-IgG2a, Control-IgG2b, Annexin V

PE - conjugated anti-human CD2, CD3, CD4, CD8, CD11b, CD14, CD15, CD18, CD19, CD20, CD21, CD22, CD27, CD33, CD34, CD37, CD38, CD40, CD42b, CD45, CD45RB, CD50, CD72, CD95, CD105, CD147, CD177, Control-IgG1, Control-IgG2a, Control-IgG2b, Annexin V

PE/Dy647 -tandem conjugated anti-human CD45

APC -conjugated anti-human CD3, CD4, CD7, CD8, CD10, CD11c, CD14, CD16, CD19, CD27, CD37, CD40, CD44, CD56, CD59, CD61, CD62L, CD62P, CD69, IL-6, Control-IgG1, Control-IgG2a, Control-IgG2b, Annexin V

[DETAILS](#)

plus CD45RB FITC, CD45RO FITC