

# ImmunoTools *special* Award 2014



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## **Neutrophil phenotype, migration and potential collaboration with B- and T cells during *Plasmodium falciparum* infection**

The aim of this study is to characterize neutrophil phenotype and migration patterns during *Plasmodium falciparum* infection (*ex vivo*) and to investigate a possible collaboration and/or influence on B- and T cells (*in vitro*).

Malaria caused by protozoan *Plasmodium* parasites remains one of the most widespread human infectious diseases. Children under the age of five in malaria endemic areas in Sub-Saharan Africa are at the highest risk of suffering from the disease, due to inadequate immunity that develops slowly after repeated exposure to the parasite. Understanding how this immunity is developed is important for vaccine development.

Neutrophils have lately been acknowledged as versatile cells and their function in the immune system seem to be more complex than previously thought. However, the role of neutrophils during malaria infection remains poorly understood. Neutrophils are the most abundant cell type of all white blood cells in humans. They are phagocytic cells that can ingest cell debris and kill microorganisms by the release of reactive oxygen species and antimicrobial peptides. They usually are the first cell type to arrive at an infected site, thus having the potential to influence the following adaptive immune response, by interacting with other immune cells and secreting cytokines. Proteins known to be produced by neutrophils have been detected in increased levels in patients with severe malaria, indicating an active role of neutrophils during infection. Recent studies have also shown that neutrophils can produce large amounts of cytokines crucial for B-cell survival, maturation and differentiation.

In addition, under certain circumstances, neutrophils seem to be able to acquire a DC-like phenotype with up-regulated MHC class II expression and co-stimulatory molecules, thus having the potential to function as an antigen presenting cell that could induce T-cell proliferation. In this study, we aim to investigate neutrophil phenotype characteristics in acutely infected malaria patients and investigate the migration patterns of neutrophils against chemotactic stimuli as well as cytokine release upon lipopolysaccharide (LPS) stimulation. Since neutrophils are a source of B-cell stimulating factors, we wish to explore a possible influence of neutrophil secreted factors on B-cell subtypes as well as their antibody production when co-

cultured with *P. falciparum* parasites. In addition, we also aim to investigate co-stimulatory molecules and MHC class II expression on neutrophils from the malaria infected patients and perform T-cell proliferation assays. The **ImmunoTools** special award would greatly help us in the set-up and optimization of the *in vitro* stimulation assays and flow cytometry analyses that are needed to address the role of neutrophils in the immune response to malaria parasites.

**ImmunoTools special** AWARD for **Stéphanie Boström** includes 25 reagents  
**FITC** - conjugated anti-human CD19, CD20, CD21, CD27, CD38, CD80, CD86, HLA-DR,

**PE** - conjugated anti-human CD4, CD19, CD20, CD21, CD27, CD38,

**PerCP** - conjugated anti-human CD3, CD20,

**APC** - conjugated anti-human CD8, CD10, CD19, CD21, CD27, CD38

recombinant human cytokines: rh BAFF, rh G-CSF, rh GM-CSF, rh IFN-g, rh IL-8

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