ImmunoTools *special* Award 2014

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The role of type I interferons in the intestinal immune system during fungal infections

The pathogenic fungus *Candida albicans* is a human commensal which colonizes the skin and mucosal surfaces. However, in immunocompromised patients (e.g. AIDS or cancer patients), *C. albicans* can cause severe life-threatening systemic infections, which reach high mortality rates up to 40 %. During the host response to *C. albicans* infections, innate immune cells (e.g. phagocytes) are significantly involved in defence mechanisms by executing anti-fungal effector functions (e.g. cytokine and chemokine production).

One important group of cytokines are type I interferons (IFNs), which are well-known for the induction of the anti-viral state during viral infections, but can also have beneficial and detrimental effects on the host during bacterial infections. However, much less is known about the role of type I IFN signaling during *C. albicans* infections. Interestingly, our group has shown that type I IFNs are detrimental for the host by promoting renal immunopathology and fungal persistence in different murine infection models with *Candida* spp. (Mayer O *et al.*, 2012; Bourgeois C *et al.*, 2011). In contrast, also beneficial effects of type I IFNs have been reported (Biondo C *et al.*, 2011; del Fresno C *et al.*, 2013) which let suggest that these cytokines might function in a highly context-dependent manner during fungal infections.

The mucosal immune system, especially in the intestine, represents a highly complex network of diverse innate and adaptive immune cells to maintain intestinal homeostasis and to defend invading microorganisms. Since *C. albicans* adheres to the mucosal epithelium and penetrates this barrier in immunocompromised patients,

the first goal of this study is the establishment of an intestinal infection model to study fungal-host interactions at mucosal surfaces. Further, by using *Ifnar1*$^{-/-}$ mice, which are deficient for type I IFN signaling, we want to determine the role of these cytokines in the specialized microenvironments of the intestine during fungal infections.

Hence, this particular ImmunoTools Box with its diverse cell surface markers would be a huge benefit for our study. Thereby, we want to decipher whether the loss of type I IFN signaling affects e.g. the cellular composition of the lamina propria or Peyer's patches, the epithelial integrity, the activation/maturation of distinct subsets of dendritic cells or the recruitment of neutrophils/monocytes and adaptive immune cells during intestinal *C. albicans* infections.

ImmunoTools *special* AWARD for
Michael Riedelberger includes 25 reagents

FITC - conjugated anti-mouse CD3e, CD4, CD25, CD45RC, CD45R, CD62L, CD117, CD134, a/b TCR, g/d TCR,

PE - conjugated anti-mouse CD11b, CD34, Gr-1, a/b TCR, g/d TCR,

APC - conjugated anti-mouse CD4, CD8, CD19, CD45, CD62L, NK-cells,

recombinant mouse cytokines: rm Flt3L / CD135, rm G-CSF, rm GM-CSF, rm IL-4

DETAILS more AWARDS