ImmunoTools IT-Box-Cy55M-Award 2013

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The Role of Tec Family Kinases in the Innate Immune Response to Human Fungal Pathogens

The human fungal pathogen *Candida albicans* (Ca) represents the 4th-most frequent cause of nosocomial bloodstream infections with mortality rates of about 40%. Impaired immunity such as in HIV or transplantation patients constitutes a major risk factor for invasive infections with Ca. To mount a protective immune response against Ca, the innate immune system must rapidly sense and respond to invading pathogens. Through the stimulation of specific pattern recognition receptors (PRRs), immune cells trigger signal transduction pathways that orchestrate immune response activation, cytokine production and other effector functions. One group of intracelullar signalling molecules involved in transducing stimulatory signals is the Tec kinase family of non-receptor tyrosine kinases (Bmx, Btk, Itk, Rlk and Tec). They are preferentially expressed in cells of the haematopoietic system, including innate immune cells. Deficiencies in Tec family kinases cause several immunological defects in humans and mice, including defects in B cell and T cell function. However, very little is known about the role and function of Tec family kinases in innate immune cells and nothing is known about their possible role in fungal-host interactions.

We have some recent unpublished data indicating that Tec Family Kinases are indeed involved in the innate immune response to human fungal pathogens, leading to drastic changes in the release of cytokines from innate immune cells as well as T cells. Furthermore, we do have evidence for changed singalling patterns in immune cells upon Tec Kinase-deficiency. Hence, this particular IT-Box would help us to decipher for the very first time the role of Tec Kinases during cytokine signalling and would help to understand the specific role of these kinases during the course of fungal infection

ImmunoTools IT-Box-Cy55M for Florian Zwolanek includes 55 recombinant cytokines

rm EGF, rm Eotaxin / CCL11, rm FGF-a / FGF-1, rm FGF-b / FGF-2, rm FGF-8, rm Flt3L / CD135, rm G-CSF, rm GM-CSF, rm GRO-a / CXCL1, rm GRO-b / CXCL2, rm IFNgamma, rm IL-1alpha, rm IL-1beta, rm IL-2, rmIL-3, rm IL-4, rm IL-5, rm IL-6, rm IL-7, rm IL-9, rm IL-10, rm IL-11, rm IL-13, rm IL-15, rm IL-16, rm IL-17A, rm IL-17C, rm IL-17F, rm IL-19, rm IL-20, rm IL-21, rm IL-22, rm IL-25 / IL-17E, rm IL-27, rm IL-31, rm IL-33, rm IP-10 / CXCL10, rm LIF, rm MCP1 / CCL2, rm M-CSF, rm MIP-1α/ CCL3, rm MIP-1β / CCL4, rm MIP3α / CCL20, rm MIP3β / CCL19, rm NGF-beta, rm PDGF-AA, rm PDGF-BB, rm RANTES / CCL5, rm sCD40L / CD154, rm SCF, rm SDF-1α / CXCL12a, rm SDF-1β / CXCL12b, rm TNFα, rm TPO, rm VEGF DETAILS