ImmunoTools IT-Box-Cy55M-Award 2013

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Characterisation of sepsis-induced Hepatic Stellate Cell activation

Sepsis and its sequel are increasingly recognized as one of the capital problems of patients hospitalized at intensive care units (ICUs). During systemic infection the acute and post-acute dysfunction of the liver is moving into the international focus. First results obtained in the host institution applied for provide evidence of early stages of liver fibrosis in long term sepsis survivors (mice). In polymicrobial peritonitis long lasting changes in both tissue architecture and functions are well investigated. Pioneering results of mice suffering from pneumonia-induced sepsis encouraged me to extend our all knowledge on the pathomechanisms of the second leading cause of sepsis beyond peritonitis.

Hepatic fibrosis always relates to an activation of hepatic stellate cells (HSC). These cells located in the space of Disse are able to transform during activation from a quiescent to a myofibroblast-like cell type with the ability of collagen production. That's why HSC play such an important role in the course of increasing extracellular matrix components which is the main criteria for fibrosis. Quiescent HSC are activated by several stimuli such as inflammatory cytokines (TNF-α, ET-1, IL-6) over the course of infection but also by cell specific growth factors like PDGF, FGF and EGF. Nearly all of them are involved in host response towards sepsis but most of the time these are only seen as isolated markers for the damage of the growth factor - associated cells (platelets, fibroblasts) disregarding their influence on other cells. But the specific processes of activation of hepatic stellate cells during different sepsis related causes (peritonitis, pneumonia) are mostly unexplored.

Therefore I aim to understand the sepsis-associated specific activation mechanisms of primarily isolated HSC by stimulating them with serum of sepsis-treated mice which are suffering either from peritonitis or from pneumonia.

To comprehend the outcome of these treatments I would like measure certain cytokines and growth-factors playing a role in HSC activation by ELISA and Cytometric Bead Array. With the help of the ImmunoTools IT-Box-Cy55M I'll be able to treat the isolated cells with the ascertained concentration of these stimuli to characterize the influence of the cell-signalling molecules on HSC activation in the course of sepsis.

ImmunoTools IT-Box-Cy55M for Benjamin Giszas

includes 55 recombinant mouse 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