ImmunoTools IT-Box-Cy55M-Award 2013

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The immunobiology of therapeutic, p53-specific T cell antigen receptor (TCR) gene transfer

Adoptive T cell therapy (ACT) has been shown to be a promising approach to target cancer. However, tumor immune escape mechanisms often suppress an effective antitumor adaptive immune response. Here, the plasticity of tumor cells or the ability to quickly adapt to the environment helps the tumor cells to overcome the antitumor immune response, which results in the outgrowth of the tumor. To date, targeted therapies against tumors and immunotherapy have recently proven clinical synergistic effect when combined together, but only a better understanding of the molecular mechanisms regulating tumor evasion from the immune system will allow the development of more effective cancer treatments.

In order to enhance the antitumor response of our optimized high-affinity p53-specific single-chain T cell receptor, which targets the broadly expressed tumor-associated antigen p53(264-272) we try to weaken the immunosuppressive tumor microenvironment. Therefore we are working with an immunocompetent mouse tumor model, to dissect the role of different immunosuppressive cell compartments such as myeloid derived suppressor cells (MDSC) or T regulatory cells (Tregs), tumor-associated neutrophils (TAN) and macrophages (TAM) on tumor growth.

Having deeper insight into processes within the tumor micro milieu will allow targeting simultaneously several immune escape mechanisms, resulting in more effective anti-tumor responses. Therefore, the ImmunoTools cytokines from the *IT-Box-Cy55M* will give us the unique opportunity to screen tumor samples and shed light on still unclear molecular pathways that are crucial for tumor growth and survival. We are particularly interested in cytokines, which attract for example MDSC or affect T cell function in positive or negative way like IL-4/5, MIP-1 or TNFα.

ImmunoTools *IT-Box-Cy55M* for **Jutta Petschenka**
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