

ImmunoTools IT-Box-Cy55M-Award 2013



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Role of CXCR3 Ligands CXCL9 and CXCL10 in Breast Cancer

Both the innate and the acquired immune system are able to suppress tumor growth, the underlying mechanisms are diverse. Common to all, however, is the need for adequate infiltration of the tumor tissue with the appropriate immune effector cells, especially T-lymphocytes and natural killer cells. The two chemokines and CXCR3 ligands CXCL9 and CXCL10 are able to attract such tumor-suppressive lymphocytes in solid tumors via chemotaxis. Accordingly, we and others have already shown that an increased expression of CXCL9 and CXCL10 is associated with an improved therapeutic response and survival in various cancers. But so far only little is known of the expression and regulation of these chemokines in breast cancer.

We investigate the expression of CXCL9 and CXCL10 in breast cancer specimens by immunohistochemistry and ELISA, to correlate the results with the lymphocytic infiltrate the tumor biological characteristics and the clinical data. We want to show that CXCL9 and CXCL10 correlate positively with lymphocytic infiltration and survival in human breast cancer.

Furthermore we want to study the function of CXCL9 and CXCL10 in an immunocompetent mouse model. Therefore we plan a stable transfection of murine breast cancer cells with mCxcl9 and mCxcl10, as well as a stable knockdown of the same genes in the cells. These cells will then be implanted in the mouse model, so that the tumor growth, the lymphocytic infiltration, the immune cell composition in the blood and the regulation of agonistic and antagonistic chemokines in the tumor and in the serum of mice can be measured. Moreover in further experiments we will study the signal-transduction pathways and the interaction with other cytokines. For that mouse model experiments, the **ImmunoTools Box-Cy55M** would be very helpful.

ImmunoTools IT-Box-Cy55M for **Claudia Cerny**
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 IFN γ , rm IL-1alpha, rm IL-1beta, rm IL-2, rm IL-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-1 α / CCL3, rm MIP-1 β /
CCL4, rm MIP3 α / CCL20, rm MIP3 β / CCL19, rm NGF-beta, rm PDGF-AA, rm PDGF-BB,
rm RANTES / CCL5, rm sCD40L / CD154, rm SCF, rm SDF-1 α / CXCL12a, rm SDF-1 β /
CXCL12b, rm TNF α , rm TPO, rm VEGF [DETAILS](#)