

ImmunoTools IT-Box-Cy55M-Award 2013



Cristina Riccadonna

PhD Supervisor: Dr. Paul R. Walker

Immunobiology of Brain Tumours
Geneva University Hospitals and University of Geneva
rue Gabrielle-Perret-Gentil 4
1211 Geneva 14, Switzerland

Cytotoxic T cell interactions with glioma and how these are impacted by different cell mediators

Malignant glioma is an aggressive brain tumour. It is infiltrated by immune cells, including cytotoxic T lymphocytes (CTLs). The majority of glioma cells are differentiated, whereas a small fraction of cells (glioma initiating stem-like cells), is de-differentiated, reported to be chemoresistant, and is likely to be the cause of recurrence. The prognosis of glioma patients remains very poor, despite treatment (surgical resection, radiotherapy and chemotherapy).

I am currently investigating if T cell immunotherapy, an attractive future treatment for glioma, can synergize with chemotherapy. In particular, I am interested in understanding if and how sensitivity of glioma and glioma stem-like cells to CTL killing can be modulated by chemotherapeutic drugs and by immune mediators.

In order to model glioma *in vitro*, I am currently using the murine astrocytoma GL261 cell line. GL261 cells grow as adherent cultures and are differentiated cells. GL261 neurospheres (which are enriched in glioma initiating stem-like cells) are de-differentiated from adherent cultures, grow in suspension and required serum-free medium supplied with growth factors (EGF and FGF-b). The neurospheres express CXCR4 and I will be investigating the consequences of its ligation with SDF-1 α (CXCL12a) and SDF-1 β (CXCL12b).

I will test the effect that different chemotherapeutic drugs can have on CTL recognition and CTL killing of glioma and glioma stem-like cells. I will also use TNF α and IFN γ to investigate the effect of these two immune mediators on the sensitivity of glioma and glioma stem-like cells to CTL-induced cell death.

ImmunoTools IT-Box-Cy55M for Cristina Riccadonna

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](#)