ImmunoTools IT-Box-139 Award 2012

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The Immunomodulatory Role of Endogenous Glucocorticoids in Ovarian Cancer

Since December 2008, I have been working as PhD student in the junior research group "tumor progression and immune escape" at the University of Würzburg, Germany. This enabled me to explore various mechanisms which allow tumor cells to escape from cancer immunosurveillance. A special focus in our group lies on classical and non-classical members of the TGF-beta superfamily which are abundant in many malignancies. As TGF-beta displays stronger immune-inhibitory functions than any other known cytokine, its role in tumor immune escape is well-established. Other members of this group have, however, not been explored in greater detail. Further projects deal with the putative role of miRNAs in TGF-beta signaling in immune cells and the immunological characteristics of tumor-initiating cells. In my PhD project, I am analyzing the potential immunomodulatory role of endogenous glucocorticoids in ovarian cancer. This project is based upon the observation that tumor-infiltrating myeloid-derived suppressor cells (MDSC) and/or tumor-associated macrophages (TAM) express the enzyme 11Beta-Hydroxysteroid dehydrogenase I (11β-HSD1). 11β-HSD1 converts biologically inactive cortisone into active cortisol. The presumed endogenous activation of glucocorticoids is corroborated by high levels of endogenous cortisol in ascitic fluid and tumor exudates from ovarian cancer patients. Considering that cortisol has strong suppressive effects on all kinds of immune cells, the activation of endogenous glucocorticoids by MDSC or TAM may constitute a new immunosuppressive mechanism in ovarian cancer. To test this, a spontaneous mouse model for ovarian cancer is combined with adoptive transfer of immune cells from glucocorticoid-receptor knock-out mice. Likewise, pharmacological inhibitors of 11β-HSD1 and various immune stimuli shall be tested.

My project postulates a new immunosuppressive mechanism for tumor-infiltrating myeloid-derived suppressor cells (MDSC) or tumor-associated macrophages (TAM) in ovarian cancer. I believe that the ImmunoTools antibodies from the IT-Box-139 will enable me to optimize the immunophenotyping from the tumor microenvironment - which is an essential part of my project. Techniques for the dissociation of tumor tissue have been established. Thus, infiltrating immune cells can be characterized by flow cytometry, using the antibodies from IT-Box-139. In fact, we are already using many antibodies from ImmunoTools and have generally obtained good results with these reagents.

ImmunoTools IT-Box-139 for Ahmed Adel Seida include 100 antibodies

FITC - conjugated anti-human CD1a, CD3, CD4, CD5, CD6, CD7, CD8, CD14, CD15, CD16, CD19, CD21, CD25, CD29, CD35, CD36, CD41a, CD42b, CD45, CD45RA, CD45RB, CD45RO, CD49d, CD53, CD57, CD61, CD63, CD80, CD86, HLA-DR, IL-6, Control-IgG1, Control-IgG2a, Control-IgG2b, Annexin V

PE - conjugated anti-human CD3, CD4, CD8, CD11b, CD15, CD14, CD18, CD19, CD20, CD21, CD22, CD31, CD33, CD38, CD40, CD45, CD45RB, CD50, CD52, CD56, CD58, CD62p, CD72, CD95, CD105, CD147, CD177, CD235a, HLA-ABC, IL-6, Control-IgG1, Control-IgG2a, Control-IgG2b, Annexin V

PE/Dy647 -tandem conjugated anti-human CD3, CD4, CD8, CD14, CD19, CD20, CD25, CD54

APC -conjugated anti-human CD2, CD3, CD4, CD8, CD10, CD11a, CD11c, CD14, CD16, CD27, CD37, CD42b, CD44, CD45, CD59, CD62L, CD69, CD71, IL-6, Control-IgG1, Control-IgG2a, Control-IgG2b, Annexin V

DETAILS