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BASTION – FROM BASIC TO TRANSLATIONAL RESEARCH IN ONCOLOGY

Report on the visit of Piotr Religa in the Department of Internal Medicine, Warsaw Medical University within 7PR21/BASTION/WP1 (Twinning)

From September 2014 to November 2014 I visited the Department of Internal Medicine,Angiology and Hypertension, the Medical University of Warsaw. This visit was coordinated under the twinning agreement between the Medical University of Warsaw and the Karolinska Institutet WP1 (Task 1.7). During my stay in Warsaw, I conducted research projects in the field of experimental medicine together with a research team led by Prof. Zbigniew Gaciong and Dr Grzegorz Placha (Fig. 1).

Figure 1 – Dr Piotr Religa in the research laboratory of the Department of Internal Medicine, Angiology and Hypertension, MUW.

Our research was aimed to establish the collaboration to study the role of cytomegalovirus in oncology by using equipment and facilities that are available at the Medical University ofWarsaw.

Our primary goal is to use new approaches and technologies to study the relationship between cytomegalovirus (CMV) and progression of colon cancer. The rationale for this research is that CMV has been shown to be a common virus that occurs in 98% of human population and is associated with cancer progression. The mechanism by which CMV affects progression of cancer is still not evaluated in details. We plan to analyze the clinical samples obtained from colon cancer patients, toidentify CMV-related proteins in tumors and to explain the mechanism of CMV-dependent tumor progression.

During my twinning in MUW 2014, we established animal models of colon cancer and glioblastoma that will be useful for our future research.

In order to analyze the effect of CMV infection on tumor growth and formation of metastasis, we established ananimal model of tumor implantation. We selected CT26 syngeneic mouse colon cancer cell line. Thistype of cancer gives metastasis to lungs, brain and liver in a way that mimic human colon cancer. Wehave chosen syngeneic models in order to avoid the artificial influence of the immune response to the xenograft.

In our model we use green fluorescent protein (GFP) and lacZ labeling of CT26 cancer cells. These techniqueallows us to easily identify tumor cells in blood, bone marrow and metastasis to various organs (Fig. 2).

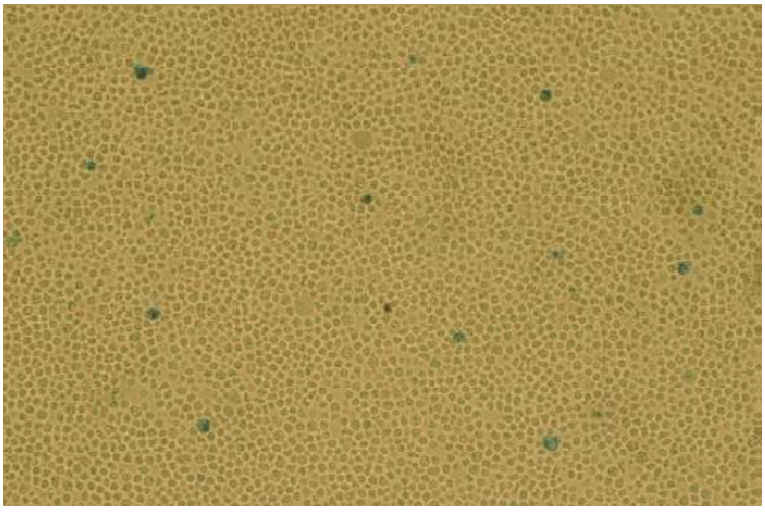

Figure 2. CT26 cells with beta-galactosidase activity disseminated to bone marrow.

We have also established a murine model of CMV infection that let us study the effect of CMV viruson tumor growth and spreading. Previously, we used those animal models

to study the effects ofC/EBPbeta on tumor metastasis. Briefly, we implanted 2.5 x 10^6 of CT26 cells subcutaneously to control mice and micepreviously infected with CMV virus (4 days before CT26 cells implantation). The third experimentalgroup constituted mice implanted with CMV-infected CT26 cells. Tumors were established at sites ofimplantation during 2 weeks. Thereafter, the primary tumors were surgically cut off. Two monthslater tumors recurrence at implantation sites and metastasis occurred. Both recurrent tumors andmetastases in lungs, liver and brain were assessed.

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