JOINT TRANSNATIONAL CALL 2015:

"Immunology and Immunotherapy of Cancer: Strengthening the Translational Aspects"

PARTNER REQUEST/COLLABORATION OFFER

If you would like to have your profile published on our "Search for a research partner" webpage, please fill out this form and send it to ✉
If you have any questions about this form, please do no hesitate to contact us at ✉

Note: Fields marked with a * are mandatory

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***I agree with the publication of my contact data and of this form on the TRANSCAN Website:**

☑ YES

SEARCH FOR A COLLABORATOR

IF YOU ARE LOOKING FOR A PARTNER IN YOUR SUGGESTED PROPOSAL, PLEASE SPECIFICY ALSO THE NEEDED EXPERTISE

Project proposal	
Project title	
Provide a short project description about the project and the consortium (Max. 450 words)	

OFFER FOR COLLABORATION

IF YOU PROPOSE YOURSELF AS A PARTNER IN A CONSORTIUM, PLEASE DETAIL YOUR EXPERTISE

Type of partner (Research institution, university, etc.)	Research institution in medical university
Provide a short description about the expertise (Max. 200 words)	
In a FP7 EU project OCTIPS (www.octips.eu), which I coordinate, we aimed at defining molecular mechanisms of the recurrent and resistant high grade serous ovarian cancer. We compared gene expression profiles of paired primary and recurrent tumors (by RNA seq) from the same patient (70 pairs). We found that the most overwhelming difference in the gene expression cross all samples are not from the epithelial tumor cells, but from cells specifically involved in the adaptive immune reaction, including numerous genes (over 200 with fold change >5, and cstatistics >75%) specific for such as immature B cells, activating B cells, TFH, Treg, and genes coding for the proteins constituting the antibodies. The tumors can be clustered into immune active type and immune silent one, which has the impact on the interval of disease recurrence. This raises the question, what and how such a difference in the host immune reaction has been caused. There are two aspects which can be considered: 1. The whole exome sequencing of the 70 pairs tumors can give us the information of mutation profiles and their difference in the immune active and immune silent tumors, which may lead to the identification of tumor specific antigen (DNA from most of the samples are available); 2. We established tumor cell lines from	

about 20 patients with high grade serous ovarian cancer. If we can define the immune reactive type of these tumors (either by RNA seq of the tumor samples or/and with specific protein expression), it will be highly interesting to investigate the difference of tumor cell lines in regard of mutation and gene expression profiles to define the possible tumor specific antigens.

We are interested in participating consortium working on the Transcan-2, Aim 1: Identification and validation of shared or personalized mutated human tumor antigenic targets.