





JOINT TRANSNATIONAL CALL 2017: "Translational Research on Rare Cancers"

PARTNER REQUEST/COLLABORATION OFFER

If you would like to have your profile published on the TRANSCAN-2 website, "Looking for a research partner" webpage, please fill out this form and send it to 

If you have any questions about this form, please do not 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-2 Website:**

YES



SEARCH FOR A COLLABORATOR

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

Project proposal

Project title (draft):

Short description of the project in preparation and of the consortium; description of the areas of expertise needed (Max. 2000 words):



OFFER FOR COLLABORATION

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

Short description of the areas of interest and expertise (Max. 2000 words):

Our team is devoted to discovery and validation of novel checkpoints of the immune response to cancer. We are especially interested on the role and function of regulatory T cells (Treg) at preventing an efficient immune response to tumors. Our current work focus on the role of members of the TNFR family on the biology and function of Treg in the tumors. Our preferred model is the NSG mouse strain reconstituted with a human immune system and bearing human tumors. Using this model, we have validated a novel combination of treatment for breast cancer that could be applied to humans (Burlion et al, A novel combination of cyclophosphamide and anti-ICOS mAb prevents tumor growth by affecting regulatory T cells in mice with a human immune system. Preprint on *Biorxiv* (2017) doi:10.1101/191031.). We have access and have used mass cytometry to precisely decipher the quantity and the quality of human immune cells infiltrating the tumor. Our goal would be to have access to primary human tumors with matched PBMC to respect the autologous nature of the immune response to tumors in vivo. In this model, the xenograft vs host disease that develops in PBMC-grafted NSG mice may act as an “adjuvant” to activate T cells in vivo and reveal the role of Treg and/or checkpoints molecules to enhance or prevent an efficient immune response. Isolating cells and pathways that prevent anti-tumoral immunity in vivo with human cells and human tumors may open new avenues for personalized medicine.

