**CD97 is a critical regulator of acute myeloid leukemia stem cell function**

Despite significant efforts to improve therapies for acute myeloid leukemia (AML), clinical outcomes remain poor. Understanding the mechanisms that regulate the development and maintenance of leukemic stem cells (LSC) is important to reveal new therapeutic opportunities. We have identified CD97, a member of the adhesion class of G-protein coupled receptors (GPCRs), as a frequently upregulated antigen of AML blasts that is a critical regulator of blast function. High levels of CD97 correlate with poor prognosis, and silencing of CD97 reduces disease aggressiveness *in vivo*. These phenotypes are due to CD97’s function to promote proliferation, survival, and the maintenance of the undifferentiated state in leukemic blasts. Collectively, our data credential CD97 as a promising therapeutic target on LSCs in AML.

**Personal Statement**

My main interest in research relies in studying the relationship between cancer and its host. I first completed my undergraduate studies and Master’s degree, with a specialty in Immunology at University Paris VII. During my PhD, under the supervision of Pr. Jose Cohen at the Pitie-Salpetriere (Paris) and Henri Mondor (Creteil) hospitals, I studied graft-versus-host disease (GVHD), a lethal complication following bone marrow transplantation in patients with leukemia. We demonstrated that GVHD in mice could be completely prevented by the administration of regulatory T cells (Treg). I have also acquired broad knowledge in mouse models of autoimmunity, alloreactivity and cancer. In order to improve and expand my knowledge in the field of hematology and cancer, I sought to explore the molecular mechanisms of leukemogenesis. I joined in 2013 the laboratory of Christopher Park, an expert of leukemic stem cells (LSC) biology at Memorial Sloan-Kettering Cancer Center, New York. Based on a high-throughput molecular screening, I have designed a project aiming to study the role of the GPCR CD97 in leukemogenesis. Using murine and human models of AML, we found that CD97, whose expression is highly up-regulated by LSC, was a critical regulator of LSC expansion and function. Thus, it represents a potential regulator of leukemogenesis and a promising target for future therapy.

I now wish to continue my work on the host/cancer interactions in a new laboratory. I am willing to join a dynamic and leading-edge institution such as ImmunoConcept to pursue my work on translational biology.