IMCB Invited Speaker

Speaker : **Dr. Frederic Saltel**
Principal Investigator, INSERM U1053,
Université Bordeaux Segalen, France

Date : 22 July 2013 (Monday)

Time : 11:00AM - 12:00PM

Venue : IMCB Seminar Room 3-46, Level 3, Proteos, Biopolis

Host : Dr. Frederic Bard

Seminar :

Physiological type I collagen organization induces the formation of a novel class of linear invadosomes

Invadosomes are F-actin structures capable of degrading the extracellular matrix through the activation of matrix metalloproteases. Invadosomes is a global term including podosomes found in normal cells and invadopodia observed in cancer cells. As fibrillar type I collagen promotes pro-matrix metalloproteinase 2 activation by membrane type 1 matrix metalloproteinase, we aimed at investigating the functional relationships between collagen I organization and invadosome induction. We found that fibrillar collagen I induced linear F-actin structures, distributed along the fibrils, on endothelial cells, macrophages, fibroblasts, and tumor cells. These structures share features with conventional invadosomes, as they express cortactin and N-WASP and accumulate the scaffold protein Tks5, which proved essential for their formation. On the basis of their ability to degrade extracellular matrix elements and their original architecture, we named these structures "linear invadosomes." Interestingly, podosomes or invadopodia were replaced by linear invadosomes upon contact of the cells with fibrillar collagen I. However, linear invadosomes clearly differ from classical invadosomes, as they do not contain paxillin, vinculin, and β1/β3 integrins. Using knockout mouse embryonic fibroblasts, blocking antibody and RGD peptide, we demonstrate that linear invadosome formation and activity are independent of β1 and β3 integrins. This study demonstrates the existence a new type of invadosomes specifically induced by physiological type I collagen organization.

About the Speaker :

I have done my PhD in the Ecole normale supérieur (ENS) de Lyon in the Pierre Jurdic's laboratory. My Thesis subject was the dynamic of the actin cytoskeleton during osteoclast differentiation and resorption (2001-2004). I was fascinated by actin, microscopy and the relationship between cells and the extracellular matrix. So, I decided to join the Bernhard Werhle-Haller's laboratory, to work on integrin to try to understand how the alpha v Beta3 integrin can be activated, to form cluster and focal adhesion. During this first post-doc I obtained a EMBO fellowship. In 2007, I joined the E. Génot group at Bordeaux (France) to work on podosomes in endothelial cells. And after my recruitment as researcher, since 2010, I started to create my own group in collaboration with the Dr. Violaine Moreau with the objective to understand mechanisms involved in cell invasion during liver cancer progression.

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