

BioSys/BioISI Research Seminar

Epithelial-Mesenchymal Plasticity in Breast Cancer Metastasis

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Metastatic spread of cancer cells to vital organs is the major cause of death in cancer. Understanding the metastatic process has remained an elusive task and consequently, there is a lack of treatments that target and inhibit metastasis. Despite intense research, our knowledge of how and when cancer cells disseminate are limited. In fact, even the perhaps most fundamental question - why cancer cells metastasize, remains to be answered. Recent data show that epithelial-mesenchymal transition (EMT), a latent developmental process, is re-activated during cancer and chronic inflammation. Cytokines, such as transforming growth factor beta 1 (TGF- β 1) can trigger EMT, whereby cancer cells lose epithelial, and gain migratory properties. Increased levels of TGF- β 1 is detected in plasma of breast cancer patients and at invasive fronts in human breast cancer tissues, and correlates with the presence of lymph node metastasis. Breast cancer cells undergoing EMT also acquire cancer stem cell-like properties. TGF- β 1 signaling towards EMT involves both Smad-dependent, and -independent pathways including Akt and p38 MAPK. Blocking EMT, or its consequences, would be attractive as a novel type of anti-metastatic cancer treatment. Yet, the mechanisms of how EMT is induced and importantly, the functionality of EMT during the different steps of the metastatic cascade are not clear. We have established an interdisciplinary research program to elucidate the role of EMT as a link between cancer and inflammation.

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Faculdade de Ciências da UL, Lisboa (Portugal), Building C1 – room 1.3.15