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“CELL PLASTICITY IN CANCER”

(Faculty Host: Dr. Jim Uniacke)



Wed. Feb. 15th, 2017
SSC 2315 @ 10:30 am

Tumours contain populations of cells with stem cell like properties, and these phenotypically plastic cells are responsible for cancer recurrence and metastatic potential. Stem cell-like populations are regulated by dynamic niches, characterized by specific growth factors and extracellular matrices, as well as biophysical features such low oxygen tensions. Moreover, a growing body of evidence suggests that cancer cells co-opt stem cell-associated regulatory networks in order to sustain plasticity. For example, we have discovered that an embryonic-associated protein called Nodal maintains stem cell phenotypes in cancer, and that it promotes classical hallmarks of cancer such as angiogenesis, chemotherapy resistance invasion and metastasis. We have also found that biophysical features of a growing tumour, in particular hypoxia, can promote tumour cell plasticity by up-regulating embryonic proteins like Nodal via a combinatorial mechanism involving alterations in bivalent histone modifications as well as selective mRNA translation. Finally, we have determined that these complex interactions can be confounded by genetic variability leading to the expression of unique proteoforms. By studying the mechanisms by which cancer cells acquire and sustain phenotypic plasticity, we may uncover novel targets for the prediction and prevention of tumour progression.

**“A GREAT OPPORTUNITY TO HEAR LEADING RESEARCHERS IN THE SCIENTIFIC
COMMUNITY DISCUSS THEIR WORK”**

*** ALL WELCOME TO ATTEND ***

*** COFFEE, TEA AND TIMBITS ***