BioISI Research Seminar

Tumor cell signaling and the role of alternative spliced Rac1b in colon cancer

Peter Jordan, PhD
BioISI - FCUL

The development of cancer is closely associated to an accumulation of mutations in genes that control cell proliferation and survival and many of these genes operate by activating signal transduction pathways. The inhibition of such pathways in a given cancer type is a promising therapeutic strategy to develop more specific treatment options but requires detailed pathway characterization. In this seminar I will present our work on Rac1b signaling in colorectal cancer (CRC).

Rac1b is an alternative splicing variant of the signaling GTPase Rac1, which is overexpressed in a specific subtype of colorectal cancer that is characterized by the presence of mutation in the oncogene BRAF. This subtype represents about 10-15% of all CRC cases and arises from serrated adenomas. Rac1b overexpression is required for cancer cell survival through stimulation of the transcription factor NFkB but how its overexpression is induced remains unclear. Current data from our lab suggest that increased Rac1b levels are not a consequence of mutated BRAF signaling or of a mutation in the RAC1 gene, but rather are triggered through signals from an inflammatory tumor cell microenvironment. We are attempting to reconstruct these inflammatory conditions in vitro and dissect how such signals change alternative splicing decisions in colon cells, including the overexpression of Rac1b.

Host: **Margarida Gama - Carvalho**
BioISI GER

When: **3rd February 2016, 12h00**
Where: **FFCUL - Auditorium**

Faculdade de Ciências da Universidade de Lisboa -Campo Grande, Lisboa (Portugal)

BioISI **http://www.fc.ul.pt/en/unidade/bioisi**
Contact: **bioisinfo@fc.ul.pt**