

Internal Seminar

Mechanism of the formation of high mannose N-glycans on *trans*-Golgi enzymes in advanced prostate cancer cells: Shifting of the Golgi targeting site of glycosyltransferases and α -mannosidase IA from giantin to GM130-GRASP65

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There is a pressing need for biomarkers that can distinguish indolent from aggressive prostate cancer to prevent over-treatment of patients with indolent tumour. Recently, we have found that defected giantin in aggressive prostatic cancer cells is responsible for the alteration of Golgi targeting of glycosyltransferases and mucin O-glycans. But, its effect on N-glycans is not clear. Defective giantin in androgen-independent prostate cancer cells results in a shift of Golgi targeting of glycosyltransferases and α -mannosidase IA from giantin to GM130-GRASP65. Consequently, *trans*-Golgi enzymes and cell surface glycoproteins acquire high mannose N-glycans, which are absent in cells with functional giantin. *In situ* proximity ligation assays of Golgi localization of α -mannosidase IA at giantin versus GM130-GRASP65 site, and absence or presence of N-glycans terminated with a 3-mannose on *trans*-Golgi glycosyltransferases may be useful for distinguishing indolent from aggressive prostate cancer cells.

Tuesday, Apr 17th 2018

03:00 PM (Tea/Coffee at 02:30 PM)

Seminar Hall, TIFR-H