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Heparanase Expression Soils the Microenvironment for Tumor Growth by Enhancing Notch Signaling and Suppressing Antitumor Immunity

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Heparanase expression has been found up-regulated in almost all types of lung cancers. To find out the implications of heparanase on lung cancer development, we examined the pathology of Lewis Lung Carcinoma (LLC2) cells growth after inoculation into the heparanase over expression mice (Hpa-tg) along with wild type (WT) control. Inoculation of the cells into the tail vein led to advanced lung colonization in the Hpa-tg mice. Injection of LLC2 cells into the right flank resulted in significantly faster growth and bigger tumors in the Hpa-tg mice, which were mainly, contributed by the higher proliferation of injected LLC2 cells. Higher proliferation of LLC2 cells, cultured in the conditioned medium of Hpa-MEF cells, indicates that stromal cells over expressing heparanase may release molecules stimulating LLC2 proliferation. This was further suggested by immuno-staining of enhanced Notch 1 signalling in the tumor. FACS analysis of the tumor-derived cells revealed fewer infiltrated neutrophils, but substantially higher percentage of IL-35+ cells in the Hpa-tg tumor. Further analysis of the cell population in the immune organs, spleen and inguinal lymph node revealed an overall suppressed immunity. Our findings suggest that heparanase expression in the host promoted LLC2 tumor growth through suppressing the anti-tumor immune response and the results support that application of heparanase inhibitors may strengthen the effects of current treatments for lung cancers.

Biography:

Tahira Batool completed her PhD in Medical Biochemistry, Uppsala university, Sweden. She is a highly motivated biologist with 11 years' experience of studying and research in different branches of Biology. She has wide range of expertise in driving scientific projects with interdisciplinary background. She is presently looking for an opportunity to apply her skills and knowledge for the benefit of patients.