Altered *N*-glycosylation in Progeny From Irradiated Colorectal Cancer Cells Contribute to Acquisition of EMT-Like Phenotype

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INTRODUCTION: Colorectal cancer (CRC) represents the third most commonly diagnosed cancer in males and the second most commonly diagnosed cancer in females. CRC is a leading cause of cancer-related mortality, and it accounts for 8.5% of all cancer-related deaths. In rectal cancer, radiotherapy has been explored for improving the local control and survival of locally advanced disease. Recently, we demonstrated that transgenerational effects induced by radiation increase the malignant features on the progeny derived from irradiated parental HT-29 colorectal cancer cells. **OBJECTIVES:** The aim of the present study is to investigate the changes in *N*-glycans in these radioresistant progenies and its relationship with acquisition of EMT-Like Phenotype. **METHODS:** Irradiated human colorectal adenocarcinoma cell line HT-29 (HTB-38TM) was used as model of EMT-acquisition. β1,6-branched and α2,6-sialylated complex-type *N*-glycans were detected using the lectins L-PHA (*Phaseolus vulgaris L*) and SNA (*Sambucus nigra*) respectively. Cell migration was examined by transwell assay followed by crystal violet staining. **RESULTS:** Our preliminary results have shown that the progeny of HT-29 colorectal cancer cells displayed both mesenchymal-like features and increased expression of β1,6-branched and α2,6-sialylated *N*-glycans. In addition, pharmacological inhibition of α-mannosidase II enzyme by swainsonine, thereby blocking the formation of complex-type *N*-glycans, have inhibited cell migration in HT-29 progenies. **CONCLUSION:** Additionally, our in vitro results have suggested that the biosynthesis of *N*-glycans appears to be a potential therapeutic target to inhibit malignant phenotype displayed by these radioresistant progenies.

Keywords: *N*-glycosylation, radioresistance, EMT

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