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Abstract

Radioresistance (inherent or acquired) remains a major obstacle affecting the clinical outcome of radiotherapy for laryngeal carcinoma. Results from our laboratory and other groups suggest that aberrant glycosylation contributes to cancer acquired radioresistance. However, the role of glycosylation in inherent radioresistance of laryngeal carcinoma has not been fully uncovered. In this study, we investigated the glycan profiling of the inherent radioresistant (Hep-2max) and radiosensitive (Hep-2min) cell lines using lectin microarray analysis. The results revealed that the radioresistant cell line Hep-2max presented higher core 1-type O-glycans than the sensitive one. Further analysis of the O-glycan regulation by benzyl- α -GalNAc application in Hep-2max cells showed partial inhibition of the O-glycan biosynthesis and increased radiosensitivity. In addition, core 1 β 1, 3-galactosyltransferase (C1GALT1) overexpression in Hep-2min cells enhanced cell

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