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P53 INDUCES CELL DEATH BY AUTOPHAGY FOLLOWING IRRADIATION

Abstract

During the gliomas cell lines radiotherapy and chemotherapy, the autophagy has important roles in regulating cell responses to cell stress. Autophagy is an evolutionarily conserved degradative process that is initiated by ROS and misfold proteins following irradiation. As type II cell death way, autophagy will induce cell death by enhancing caspase activity or caspase-independent cell death. Multiple cellular stressors, including activation of the tumour suppressor p53, can effect autophagy. In gliomas, the p53 tumor suppressor is often mutated in human magliant tumors. However, many other tumors retain wild-type (wt) p53 expression, raising the intriguing possibility that they actually benet from it. Recent studies imply a role for p53 in regulation of autophagy, a catabolic pathway by which eukaryotic cells degrade and recycle macromolecules and organelles, particularly under conditions of irradiaton. P53 protein promotes autophagy cell death and protects cell from apoptosis, caspase-independent cell death, by regulating mARF, DRAM, P21, and BCL-2 family members. Therefore recently P53 protein, inducing cell apoptosis by regulating P21 protein, effects the location of autophagy-related protein LC3B. In precious reports, TNF family mainly promotes cell apoptosis, necrosis, and autophagy cell death in gliomas cell lines. P53 regulate TNF family pathway under autophagy, at the same time, influence the sensitivity to the family ligand. In contrast to WT-p53 cell line, P53 mutation protects cell from autophagy cell death. Our aim is that changing P53 gene to enhance glioma cell death under autophagy following irradiation.