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Potential role of TIGAR in OSCC: tumorigenesis and survival

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Abstract

Despite the improvement of treatment modalities, OSCC remains a prevalent disease in India and 50% of OSCC patients die within 5 years of disease detection. One of the major reasons for treatment failure is imbalances of metabolic profile in cancer. TIGAR (TP53-induced glycolysis and apoptosis regulator), is a p53-inducible protein that functions as fructose-2, 6-bisphosphatase and fructose-1, 6-bisphosphatase, reducing the glycolytic rate and promoting the Pentose Phosphate Pathway. Consistent with increased activation of the PPP, cells expressing TIGAR have higher NADPH levels and a concomitantly enhanced ability to regulate levels of cellular ROS and thus reduce oxidative stress. Apart from the metabolic function of TIGAR, it has another role in the survival of cancer. Recently found that TIGAR is upregulated in some cancer, the main focus of our proposed study is to find out the potential role of TIGAR in tumorigenesis and survival of OSCC. For this we are using OSCC cell line as well as primary OSCC sample, some our recent finding suggesting that TIGAR is upregulated in OSCC cell line as well as patient sample compared their respective control, and also found that TIGAR knockdown decreases cell viability and drug sensitivity. Our main focus is how TIGAR regulated OSCC cell survival and what mechanism behind this, for this we will use RNA-Seq in TIGAR KD cell and TIGAR OE cell and found that how many pathway or which gene is affecting when targeting TIGAR. So overall our findings prove that TIGAR has some role in the survival of OSCC.

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