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D-PENICILLAMINE LOADED NANO LIPID CARRIERS FOR TARGETING GLIOBLASTOMA

B. Mahendran[1], DV Gowda[2], VVS Narayana Reddy[3]

[1]Department of Pharmaceutics, JSS College of Pharmacy, JSS University, Mysuru, India
[2]Department of Pharmaceutics, JSS College of Pharmacy, JSS University, Mysuru, India
[3]Department of Pharmaceutics, JSS College of Pharmacy, Ootacamund, India

Glioblastoma is a lethal cancer with a median survival of about 15 months. It is a well-known fact that most Glioblastoma's are rich in collagens. Specifically, the mesenchymal subtype of Glioblastoma, which has the worst prognosis, is enriched with numerous extracellular matrix proteins such as collagens. While collagen accumulation can be viewed as a downstream effect of mesenchymal differentiation, it was found that the feed forward increases in collagen, extracellular matrix stiffness and biomechanical influences cause enhanced selfrenewal and mesenchymal transition in glioma stem cells with a consequent increase in treatment resistance of these cells. Further, recent studies have also shown that mechanical stress plays an overarching role in the regulation of TAZ signaling. TAZ is a mediator of mesenchymal differentiation in glioma, which relates with collagen stiffness, promotes mesenchymal transition and gliomagenesis via TAZ signaling. D-penicillamine is a known inhibitor of collagen maturation, which acts by increasing the conversion of insoluble to soluble collagen and disrupting the formation of intermolecular bonds. The therapeutic benefit of D-penicillamine has not been extensively evaluated in Glioblastoma especially in the context of glioma stem cells, which represent faithful models of this disease. Directing therapeutic agents such as D-penicillamine towards deeply embedded brain tumours with biophysical barriers such as blood brain barrier and collagen remains a challenge. Hence, in the present study it is proposed to load D-penicillamine into Nano lipid carriers, which can accumulate passively in tumours via leaky tumour vasculature, caused by focused ultrasound. These triggered release strategies disrupt the blood brain barrier, confirming deep penetration in tumours, and overcome the biophysical constraints, which tackle both the challenges (drug delivery and collagen disruption) simultaneously.

naren.mahe@gmail.com