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A hypothesis and theoretical model speculating the possible role of therapy mediated neoplastic cell loss in promoting the process of glioblastoma relapse

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Tumor recurrence is considered to be one of the biggest culprits, behind the poor prognosis of glioblastomas. Using published facts on primary glioblastomas, with special reference to cancer stem cells and their recently described heterogeneity, a hypothesis is being proposed which speculates the possible role of therapy mediated neoplastic cell loss in promoting the process of relapse in these tumors. The mechanisms by which such a phenomenon could be functional, has been integrated into a double version theoretical model, which envisages glioblastomas as neoplasms comprising of multiple, differentially regulated and dynamically distinct neoplastic compartments (named as *active* and *back up* compartments in this article) supported by their own complement of cancer stem cells, wherein therapy mediated cell loss, which mainly affects the size of the active compartment, results in abrogating the inhibitory effect of the active compartment on the back up compartment, thereby leading to the activation of the back up compartment. This activation contributes towards tumor recurrence. The possibility of the existence of such a phenomenon could have strong implications on management and prognosis of these tumors. This work aims to provoke discussion and generate new ideas for further research.

Biography

Dr Mrinmay Kumar Mallik obtained his MD in Pathology from PGIMER Chandigarh India in 1996. Although he works as a clinical cytopathologist at the Cytopathology laboratory his main area of interest since 2005 has been the theoretical aspects regarding cancer stem cells specially cancer stem cells in gliomas. After carefully analyzing a large volume of literature concerning cancer stem cells in gliomas he put forward a novel theoretical model which attempts to explain recurrence in glioblastomas. This was published in the Journal of Theoretical Biology in October 2010.