

Glioblastoma invasion mechanism

Contains an intricate network of interactions and [signaling pathways](#) with the [extracellular matrix](#). Among these related [molecules](#), [TGF- \$\beta\$](#) , the [ECM](#), [Akt](#), and [microRNAs](#) are most significant in terms of cellular procedures related to [Glioblastoma motility](#) and [invasion](#). [Musashi-1 \(MSI1\)](#), a neural [RNA-binding protein](#) (RBP), regulates Glioblastoma motility and invasion, maintains stem cell populations in Glioblastoma, and promotes drug-resistant Glioblastoma phenotypes by stimulating necessary oncogenic signaling pathways through binding and regulating mRNA stability. Importantly, these necessary oncogenic signaling pathways have a close connection with TGF- β , ECM, and Akt. Thus, it appears promising to find MSI-specific inhibitors or RNA interference-based treatments to prevent the actions of these molecules despite using RBPs, which are known as hard therapeutic targets.¹⁾.

¹⁾

Liu X, Chen JY, Chien Y, Yang YP, Chen MT, Lin LT. Overview of the Molecular Mechanisms of Migration and Invasion in Glioblastoma Multiforme. J Chin Med Assoc. 2021 May 21. doi: 10.1097/JCMA.0000000000000552. Epub ahead of print. PMID: 34029218.

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