A Tubulin Binding Peptide Targets Glioma Cells Disrupting Their Microtubules, Blocking Migration, and Inducing Apoptosis

Despite aggressive treatment regimes, glioma remains a largely fatal disease. Current treatment limitations are attributed to the precarious locations within the brain where such tumors grow, their highly infiltrative nature precluding complete resection and lack of specificity among agents capable of attenuating their growth. Here, we show that in vitro, glioma cells of diverse origins internalize a peptide encompassing a tubulin-binding site (TBS) on the neurofilament light protein. The internalized peptide disrupts the microtubule network, inhibits migration and proliferation, and leads to apoptosis. Using an intracerebral transplant model, we show that most, if not all, of these responses to peptide exposure also occur in vivo. Notably, a single intratumor injection significantly attenuates tumor growth, while neither peptide uptake nor downstream consequences are observed elsewhere in the host nervous system. Such preferential uptake suggests that the peptide may have potential as a primary or supplementary glioblastoma treatment modality by exploiting its autonomous microtubule-disrupting activity or engaging its capacity to selectively target glioma cells with other cell-disrupting cargos.
Liens

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