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Role of THIK-1 in glioblastoma multiforme progression

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Glioblastoma multiforme (GBM) [World Health Organization (WHO) grade IV astrocytoma] is the most malignant form of brain tumors, carrying a poor prognosis and high rate of recurrence. During the course of the disease, microglia and brain macrophages are both recruited by the tumor microenvironment via the release of several chemoattractants and contribute considerably in several aspects of glioma development and therapeutic resistance. Microglia are the resident mononuclear macrophages of the CNS and are heterogeneously distributed in non-overlapping regions throughout the brain and spinal cord. In the healthy adult CNS, microglia exhibit a 'resting'phenotype, characterized by small cellular bodies from which thin ramified processes are extended. The transition from the 'resting'to the 'activated'state under pathological conditions, such as inflammation or disease, implies not only functional but also morphological alterations. Advances in our understanding of microglial physiology and in our understanding of the complex interactions between microglia and tumor cells in GBM can elucidate their role in glioma progression and indicate potentially interesting druggable targets. Here, we plan on investigating the two-pore domain potassium channel THIK-1 (Tandem-pore domain Halothane-Inhibited K+ channel; Knck13) as such a target. THIK-1 is expressed almost exclusively by microglia in the brain, and plays a key role in regulating microglia ramification, processes baseline motility and release of interleukin-1ß. Considering that the standard treatment for GBM is surgery followed by adjuvant radiotherapy and chemotherapy, we aim to look at the curative perspectives of THIK-1 under the scope of brachytherapy, which is a form of internal radiation using stereotactic techniques.

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