Glioblastoma biology

The biology of glioblastoma is complex and involves genetic, epigenetic, and microenvironmental factors.

Genetic alterations that contribute to the development and progression of glioblastoma include mutations in the tumor suppressor genes TP53 and PTEN, amplification of the epidermal growth factor receptor (EGFR) gene, and loss of heterozygosity on chromosomes 1p and 19q. These alterations can result in the dysregulation of signaling pathways that control cell proliferation, apoptosis, and migration, leading to uncontrolled growth and invasion of tumor cells.

Epigenetic alterations, including DNA methylation and histone modifications, also play a role in glioblastoma biology. These alterations can result in changes in gene expression patterns that promote tumor growth and invasion.

The microenvironment surrounding glioblastomas, which includes blood vessels, immune cells, and other stromal cells, also contributes to the biology of the disease. Glioblastoma cells can induce the formation of abnormal blood vessels to support tumor growth and can evade immune surveillance by suppressing the immune response.

Glioblastomas are also characterized by their heterogeneity, with different regions of the tumor exhibiting different genetic and epigenetic profiles. This heterogeneity can lead to resistance to therapy and the emergence of subpopulations of cells that drive tumor progression and recurrence.

Despite advances in the understanding of glioblastoma biology, there is still much to be learned about this complex disease. Ongoing research aims to identify new therapeutic targets and develop more effective treatments for glioblastoma.