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Cellular senescence in glioma

Rafał Chojak^{1 2}, Jawad Fares^{1 2}, Edgar Petrosyan^{1 2}, Maciej S Lesniak^{3 4}

Affiliations

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Abstract

Introduction: Glioma is the most common primary brain tumor and is often associated with treatment resistance and poor prognosis. Standard treatment typically involves radiotherapy and temozolomide-based chemotherapy, both of which induce cellular senescence—a tumor suppression mechanism.

Discussion: Gliomas employ various mechanisms to bypass or escape senescence and remain in a proliferative state. Importantly, senescent cells remain viable and secrete a large number of factors collectively known as the senescence-associated secretory phenotype (SASP) that, paradoxically, also have pro-tumorigenic effects. Furthermore, senescent cells may represent one form of tumor dormancy and play a role in glioma recurrence and progression.

Conclusion: In this article, we delineate an overview of senescence in the context of gliomas, including the mechanisms that lead to senescence induction, bypass, and escape. Furthermore, we examine the role of senescent cells in the tumor microenvironment and their role in tumor progression and recurrence. Additionally, we highlight potential therapeutic opportunities for targeting senescence in glioma.

Keywords: Glioblastoma; Neuro-oncology; Oncogene-induced senescence; One-two punch; Replicative senescence; Senolytics; Therapy-induced senescence.

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