Deciphering Tumorigenesis: Pathophysiological Roles of Reactive Oxygen Species in Glioblastoma

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Background. Reactive oxygen species (ROS) play a significant role in activating multiple signaling pathways for cellular proliferation but can be influential in tumorigenesis. We explore how ROS can be used to aid the advances of therapeutic interventions for glioblastoma, a highly aggressive and reoccurring brain cancer.

Methods. Over 30 of the most recent and relevant studies were extensively reviewed to determine the significance and implications of ROS in the pathophysiology of glioblastoma.

Results. ROS are essential for cell signaling. At low levels, ROS have been seen to promote tumorigenesis by inducing cell proliferation, which indirectly causes the tumor microenvironment to become hypoxic, inducing angiogenesis and decreased drug sensitivity. It can also promote an immunosuppressed environment by enhancing recruitment of immunosuppressive cells, facilitating invasiveness of the tumor stem cells. Interestingly, increased levels of ROS induce oxidative stress and cellular distress, leading to DNA damage and ultimately, triggering programmed cell death. However, tumor stem cells can be resistant to the apoptotic cascade through multiple cellular mechanisms. Therefore, therapeutic interventions aim to address these resistance mechanisms and promote tumor cell susceptibility to ROS.

Conclusion. By exploring ROS mechanisms involved in tumorigenesis, innovative therapies could be potentially developed to precisely target glioblastoma stem cells and prolong life expectancy of cancer patients.