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13th World Congress on Advances in Oncology

Tumor hypoxia: Impact on gene amplification in glioblastoma

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Pages: 509-515

Abstract:

Gene amplification is frequently found in human glioblastoma but the mechanisms driving amplifications remain to be elucidated. Hypoxia as hallmark of glioblastoma is known to be involved in the induction of fragile sites that are central to gene amplification. We analyzed the potential of hypoxia (pO₂ 0%) and mini hypoxia (pO₂ 5%) to induce fragile sites within a homogeneously staining region (HSR) at 12q14-15 in a glioblastoma cell line (TX3868). Treatment of cells by hypoxia or by mini hypoxia induced double minutes (DMs) and caused breakage of the HSR structure at 12q14-15, suggesting a novel hypoxia inducible fragile site on 12q. Treatment with aphidicolin, a known fragile site inducer, indicates that the hypoxia inducible fragile site is a common fragile site. Reintegration of amplified sequences and occurrence of anaphase-bridge-like structures shows that mini hypoxia and hypoxia are able to initiate amplification processes in human glioblastoma cells. Hypoxia as known tumor microenvironment factor is crucial for the development of amplifications in glioblastoma. The identification and characterization of novel common fragile sites induced by hypoxia will improve the understanding of mechanisms underlying amplifications in glioblastoma.

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