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The nonsteroidal anti-inflammatory drug celecoxib suppresses the growth and induces apoptosis of human glioblastoma cells via the NF- κ B pathway.

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Abstract

Gliomas are devastating primary tumors of the central nervous system and tend to recur even after standard therapy. Celecoxib, the selective COX-2 nonsteroidal anti-inflammatory drug, has anti-neoplastic activity against several malignancies. Accumulating evidence suggests that several COX-2-independent mechanisms may also be involved in the anti-tumor effects of celecoxib. Deregulation of the NF- κ B signaling pathway contributes to enhanced glioma cell survival, proliferation, and chemoresistance. In this study, we examined the efficacy of celecoxib in suppressing the growth of glioblastoma cell lines. We observed that treatment with celecoxib significantly reduced the proliferation of a variety of GBM cell lines in a dose-dependent manner and also induced apoptosis, which was evident from enhanced caspase-3 and 8 activity, PARP cleavage, and TUNEL positive cells. Celecoxib treatment significantly down-regulated TNF- α induced NF- κ B nuclear translocation, NF- κ B DNA binding activity, and NF- κ B-dependent reporter gene expression in U373 and T98G cells in a dose-dependent manner. Furthermore, celecoxib suppressed I κ B α degradation and phosphorylation and reduced IKK activity in a dose-dependent manner. This study provides evidence that celecoxib suppresses the growth of GBM cell lines partly by inhibiting the NF- κ B signaling pathway.

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