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Caffeine inhibits cell proliferation and regulates PKA/GSK3 β pathways in U87MG human glioma cells.

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Abstract

Caffeine is the most commonly ingested methylxanthine and has anti-cancer effects in several types of cancer. In this study, we examined the anti-cancer effects of caffeine on gliomas, both in vitro and in vivo. In vitro, caffeine treatment reduced glioma cell proliferation through G(0)/G(1)-phase cell cycle arrest by suppressing Rb phosphorylation. In addition, caffeine induced apoptosis through caspase-3 activation and poly(ADP-ribose) polymerase (PARP) cleavage. Caffeine also phosphorylated serine 9 of glycogen synthase kinase 3 beta (GSK3 β). Pretreatment with H89, a pharmacological inhibitor of protein kinase A (PKA), was able to antagonize caffeine-induced GSK3 β (ser9) phosphorylation, suggesting that the mechanism might involve a cAMP-dependent PKA-dependent pathway. In vivo, caffeine-treated tumors exhibited reduced proliferation and increased apoptosis compared with vehicle-treated tumors. These results suggest that caffeine induces cell cycle arrest and caspase-dependent cell death in glioma cells, supporting its potential use in chemotherapeutic options for malignant gliomas.

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