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## The curry spice curcumin selectively inhibits cancer cells growth in vitro and in preclinical model of glioblastoma.

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### Abstract

Previous studies suggested that curcumin is a potential agent against glioblastomas (GBMs). However, the in vivo efficacy of curcumin in gliomas remains not established. In this work, we examined the mechanisms underlying apoptosis, selectivity, efficacy and safety of curcumin from in vitro (U138MG, U87, U373 and C6 cell lines) and in vivo (C6 implants) models of GBM. In vitro, curcumin markedly inhibited proliferation and migration and induced cell death in liquid and soft agar models of GBM growth. Curcumin effects occurred irrespective of the p53 and PTEN mutational status of the cells. Interestingly, curcumin did not affect viability of primary astrocytes, suggesting that curcumin selectivity targeted transformed cells. In U138MG and C6 cells, curcumin decreased the constitutive activation of PI3K/Akt and NFkappaB survival pathways, down-regulated the antiapoptotic NFkappaB-regulated protein bcl-xl and induced mitochondrial dysfunction as a prelude to apoptosis. Cells developed an early G2/M cell cycle arrest followed by sub-G1 apoptosis and apoptotic bodies formation. Caspase-3 activation occurred in the p53-normal cell type C6, but not in the p53-mutant U138MG. Besides its apoptotic effect, curcumin also synergized with the chemotherapeutics cisplatin and doxorubicin to enhance GBM cells death. In C6-implanted rats, intraperitoneal curcumin (50 mg kg<sup>-1</sup> d<sup>-1</sup>) decreased brain tumors in 9/11 (81.8%) animals against 0/11 (0%) in the vehicle-treated group. Importantly, no evidence of tissue (transaminases, creatinine and alkaline phosphatase), metabolic (cholesterol and glucose), oxidative or hematological toxicity was observed. In summary, data presented here suggest curcumin as a potential agent for therapy of GBMs.

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