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J Neurosurg. 2011 Jul 1. [Epub ahead of print]**Antitumor effect of aspirin in glioblastoma cells by modulation of  $\beta$ -catenin/T-cell factor-mediated transcriptional activity.**

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**Abstract**

**Object** The goal in this study was to investigate the antitumor effect of aspirin in glioblastoma cells and the molecular mechanism involved in its antineoplastic activities. **Methods** The authors used the 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide method, flow cytometry, the annexin V method, and Transwell cell invasion test to detect the proliferation and invasive activity of U87 and A172 glioma cells before and after being treated with aspirin. To determine the effects of aspirin on  $\beta$ -catenin/T-cell factor (TCF) transcription activity, reporter constructs containing 3 repeats of the wild-type (TOPflash) or mutant (FOPflash) TCF-binding sites were used. Reverse transcriptase polymerase chain reaction and Western blot analyses were used to detect the expression of multiple  $\beta$ -catenin/TCF target genes following aspirin treatment. **Results** The transcriptional activity of the  $\beta$ -catenin/TCF complex was strongly inhibited by aspirin. Increasing the concentration of aspirin resulted in decreased expression of c-myc, cyclin D1, and fra-1 mRNA and protein in U87 and A172 cells in a dose-dependent manner. Aspirin inhibited glioma cell proliferation and invasive ability, and induced apoptotic cell death. **Conclusions** The results suggest that aspirin is a potent antitumor agent, and that it exerts its antineoplastic action by inhibition of the  $\beta$ -catenin/TCF signaling pathway in glioma cells.

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