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Abstract
Glioblastoma multiforme remains one of the most devastating human malignancies because of its high infiltrative capacity. This study aimed to investigate the effects of silibinin on human glioblastoma U87MG cells. The microculture tetrazolium test, bromodeoxyuridine cell proliferation assay, cell-based nuclear factor kappa B (NF-[kappa]B) activation assessment, cathepsin B activity assay, gelatin zymography, and quantitative real-time reverse transcription-PCR were performed to appraise the effects of silibinin on the metabolic activity, DNA synthesis, NF-[kappa]B phosphorylation, cathepsin B activity, and gelatinolytic activity of U87 cells. Silibinin inhibited metabolic activity, cell proliferation, NF-[kappa]B activation, cathepsin B enzymatic levels, and gelatinase B activity in U87 cells. In addition, an expressive decrease in mRNA levels of matrix metalloproteinase-9, cathepsin B, urokinase plasminogen activator receptor, urokinase plasminogen activator, and intercellular adhesion molecule 1 coupled with a significant induction in transcriptional levels of stefin A was observed. Altogether, these issues show for the first time that silibinin treatment could trammel invasive features of a highly invasive human glioma cell line, U87, through suppression of NF-[kappa]B-mediated stimulation of matrix metalloproteinase-9. Furthermore, silibinin might cripple the activation of gelatinase B by cramping transcriptional and enzymatic activities of cathepsin B in U87 cells.

PMID:20166242