عنوان مقاله:

Britannin suppresses MCF-۷ breast cancer cell growth by inducing apoptosis and inhibiting autophagy

محل انتشار:

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خلاصه مقاله:

Objective: Breast cancer is the main reason for cancer-related death in women. Britannin is a sesquiterpene lactone compound derived from Inula aucheriana with anti-tumor properties. We aimed to explore the impacts of britannin on apoptosis and autophagy in MCF-۷ breast cancer cell line.Materials and Methods: The cytotoxic influences of britannin on MCF-۷ cells were estimated by the MTT method. The expression levels of apoptosis-associated genes such as CASP۳, BCL۲, BCL۲L۱, STAT۳, and JAK۲ and transcripts of autophagy markers including ATG۱, ATG۴, ATG۵, ATG۷, ATG۱۲, BECN۱, and MAP۱LC۳A were quantified using quantitative real time-PCR (qRT-PCR). Western blotting method was used to evaluate the amount of caspase ۳, phosphorylated JAK۲, phosphorylated STAT۳, ATG۱, ATG۴, ATG۵, Beclin۱, and LC-III. Results: Treatment of MCF-۷ cells with various concentrations of britannin remarkably hindered the viability of these cells compared to the controls. This compound significantly elevated the expression of pro-apoptotic caspase-۳ but did not influence the levels of anti-apoptotic BCL۲ and BCL۲L۱. Britannin decreased the levels of phosphorylated forms of JAK۲ and STAT۳ proteins causing the blockage of the JAK/STAT pathway. Four autophagy factors expressions, including ATG۴, ATG۵, Beclin۱, and LCIII, were reduced due to the effect of britannin on MCF-۷ cells.Conclusion: Britannin triggered apoptosis in MCF-۷ cells by a mechanism that led to the blockade of the JAK/STAT pathway. Moreover, britannin prohibited autophagy in these cancer cells. This may suggest britannin as an agent for the suppression of breast tumors or as an adjutant for the enhancement of anti-breast cancer drugs effect.

کلمات کلیدی:

Breast Cancer, Britannin, Apoptosis, Autophagy, STAT۳, JAK۲