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Britannin induces apoptosis through targeting AMPK in human breast cancer

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Nowadays, there is an urgent need for novel drugs with improved efficiency against tumor cells. Of note, induction of cell death pathways with regard to molecular targets is considered as a promising treatment method in cancerous cells. AMP-activated protein kinase (AMPK) which is involved in suppression of cell growth and induction of apoptosis is emerged as an attractive target molecule for cancer treatment. Recently, naturally derived products such as britannin had drawn growing attention as an agent in cancer therapy. Britannin was extracted from Inula aucheriana by using chromatography methods. Human breast cancer cell line (MCF-7) and normal breast cancer cell line (MCF10A) were applied to investigation of anticancer properties of britannin. Cytotoxic effects of britannin were examined by MTT assay. Apoptosis induction was evaluated by flow cytometry and also, activity of caspase3 was assessed by colorimetric assay. Furthermore, intracellular changes in protein expression of Bax, Bcl2 and AMPK were analyzed by western blotting method. The viability of MCF-7 and MCF10A cells inhibited after 24h treatment by britannin with the IC50 values 15±2.3μM and 80±5.4μM, respectively. Apoptosis induction in MCF-7 cells was confirmed by annexin V-FITC/PI staining and caspase3 activation. In addition, britannin decreased the expression of Bcl2 (anti-apoptotic protein) and increased the expression of Bax (pro-apoptotic protein) in MCF-7 cells. Moreover, britannin increased phosphorylated active form of AMPK in MCF-7 cells. Taken together, the data shows that the cytotoxic effect of britannin on the cancerous cells is significantly higher than on normal cells. Furthermore, results demonstrate that britannin induces mitochondrial-apoptosis through activation of AMPK in breast cancer cells and may potentially serve as an adjuvant agent for the treatment of human breast cancer.

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