Aloe-emodin modulates PKC isozymes, inhibits proliferation, and induces apoptosis in U-373MG glioma cells.

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Abstract

Aloe-emodin (1,8-dihydroy-3-[hydroxymethyl]-anthraquinone) purified from Aloe vera leaves has been reported to have antitumor activity. The objectives of our research were to determine how aloe-emodin regulates the cell cycle, cell proliferation and protein kinase C (PKC) during glioma growth and development. To establish the cell cycle effects of aloe-emodin on brain cells [transformed glia cell line (SVG) and human glioma U-373MG cell line (U-373MG)], cells were treated with either dimethylsulfoxide (DMSO; control) or aloe-emodin (40 microM). Results from flow cytometry demonstrated that aloe-emodin delayed the number of cells entering and exiting DNA synthesis (S) phase in both SVG and U-373MG cells indicating that aloe-emodin may inhibit S phase progression. Assessment of cell viability demonstrated that SVG and U-373MG glioma cell were highly sensitive to aloe-emodin. The aloe-emodin-induced decreased proliferation was sustained at 48-96 h. A PKC activity assay was quantified to establish the role of PKC in aloe-emodin’s mode of action. Exposure of SVG and U-373MG glioma cells to aloe-emodin suppressed PKC activity and reduced the protein content of most of the PKC isozymes. We determined that cancer growth inhibition by aloe-emodin was due to apoptosis (i.e., programmed cell death). Taken together, these results support the hypothesis that aloe-emodin represents a novel antitumor chemotherapeutic drug.

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