Aloe emodin induz parada do ciclo celular em G2-M na leucemia promielocítica humana HL-60

Aloe-emodin induced in vitro G2/M arrest of cell cycle in human promyelocytic leukemia HL-60 cells.

Chen HC[1], Hsieh WT, Chang WC, Chung JG.
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Author information

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Department of Pharmcology, China Medical University, 91 Hsueh-Shih Road, Taichung 404, Taiwan, ROC.

Abstract

In this study, we have evaluated the chemopreventive role of aloe-emodin in human promyelocytic leukemia HL-60 cells in vitro by studying the regulation of proliferation, cell cycle and apoptosis. Aloe-emodin inhibited cell proliferation and induced G2/M arrest and apoptosis in HL-60 cells. Investigation of the levels of cyclins B1, E and A by immunoblot analysis showed that cyclin E level was unaffected, whereas cyclin B1 and A levels increased with aloe-emodin in HL-60 cells. Investigation of the levels of cyclin-dependent kinases, Cdk1 and 2, showed increased levels of Cdk1 but the levels of Cdk2 were not effected with aloe-emodin in HL-60 cells. The levels of p27 were increased after HL-60 cells were cotreated with various concentrations of aloe-emodin. The increase of the levels of p27 may be the major factor for aloe-emodin to cause G2/M arrest in these examined cells. Flow cytometric assays and DNA fragmentation gel electrophoresis also confirmed aloe-emodin induced apoptosis in HL-60 cells. The levels of caspase-3 were increased after HL-60 cells were cotreated with 10 microM aloe-emodin for 12, 24, 48, and 72 hours. Taken together, aloe-emodin therefore appears to exert its anticarcinogenesis properties by inhibiting proliferation and inducing cell cycle arrest and apoptosis underwent activation of caspase-3 in human leukemia HL-60 cells.

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