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Induction of G1 Arrest and Apoptosis in Human Cancer Cells by Panaxydol

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Abstract : In this study, we focused on the anti-proliferative effects of panaxydol, a C17 polyacetylenic compound derived from Panax ginseng roots, against various human cancer cells. We treated with panaxydol to various cancer cells and panaxydol treatment was found to significantly inhibit the proliferation of human lung cancer cells (A549) and human pancreatic cancer cells (AsPC-1 and MIA PaCa-2), of which AsPC-1 cells were most sensitive to its treatment. DNA flow cytometric analysis indicated that panaxydol blocked cell cycle progression at the G1 phase in A549 cells, which accompanied by a parallel reduction of protein expression of cyclin-dependent kinase (CDK) 2, CDK4, CDK6, cyclin D1 and cyclin E. CDK inhibitors (CDKIs), such as p21CIP1/WAF1 and p27KIP1, were gradually upregulated after panaxydol treatment at the protein levels. Furthermore, panaxydol induced the activation of p53 in A549 cells. In addition, panaxydol also induced apoptosis of AsPC-1 and MIA PaCa-2 cells, as shown by accumulation of subG1 and apoptotic cell populations. Panaxydol triggered the activation of caspase-3, -8, -9 and the cleavage of poly (ADP-ribose) polymerase (PARP). Reduction of mitochondrial transmembrane potential by panaxydol was determined by staining with dihexyloxacarbocyanine iodide. Furthermore, panaxydol suppressed the levels of anti-apoptotic proteins, XIAP and Bcl-2, and increased the levels of proapoptotic proteins, Bax and Bad. In addition, panaxydol inhibited the activation of Akt and extracellular signal-regulated kinase (ERK) and activated the p38 mitogen-activated protein kinase kinase (MAPK). Our results suggest that panaxydol is an anti-tumor compound that causes p53-mediated cell cycle arrest and apoptosis via mitochondrial apoptotic pathway in various cancer cells.

Keywords : apoptosis, cancer, G1 arrest, panaxydol
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