Biol Pharm Bull. 2004 Dec;27(12):1970-5.

Evaluation of cell death caused by triterpene glycosides and phenolic substances from Cimicifuga racemosa extract in human MCF-7 breast cancer cells.

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Abstract

We previously reported that the antiproliferative effect of an isopropanolic-aqueous extract of black cohosh (iCR) on **MCF-7** estrogen-responsive **breast cancer cell** line was due to the induction of apoptosis. Here we address the question to what extent apoptosis induction can be ascribed to one of the two major fractions of iCR, the **triterpene glycosides** (TTG) or the cinnamic acid esters (CAE). Furthermore, as black cohosh is routinely administered orally, we studied whether its pharmacological effects would withstand simulated liver metabolism. The antiproliferative activity of TTG and CAE as well as of rat liver microsomal S9 fraction-pretreated iCR on **MCF-7 cells** were investigated by WST-1 assay. The features of **cell death** induced were tested for apoptosis by flow cytometry (light scatter characteristics, Annexin V binding). Irrespective of S9-pretreatment, 72 h iCR treatment induced a dose-dependent down regulation of **cell** proliferation with the same IC50 of 55.3 microg/ml dry residue which corresponds to 19.3 microg/ml TTG and 2.7 microg/ml CAE. The degree of apoptotic **MCF-7 cells** was also comparable. Both, isolated TTG and CAE fractions inhibited **cell** growth, the IC50 being 59.3 microg/ml and 26.1 microg/ml, respectively. Interestingly, whereas IC50 and apoptosis induction correspond well for the whole extract, TTG and CAE fractions induced apoptosis at concentrations (25 and 5 microg/ml) well below those required for significant growth inhibition. Observation of this study firstly showed that the **cell death** induced by iCR withstood a metabolic activation system. In addition, TTG and CAE compounds significantly contributed to its apoptotic effect, CAE being the more potent inhibitor of proliferation and apoptosis inducer.

PMID: 15577215 [PubMed - indexed for MEDLINE] Free full text