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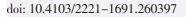






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Original Article





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Methanolic extract of *Abrus precatorius* promotes breast cancer MDA–MB–231 cell death by inducing cell cycle arrest at G_0/G_1 and upregulating Bax

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ABSTRACT

Objective: To determine the anti-proliferative activity of *Abrus precatorius* (*A. precatorius*) leaf extracts and their effect on cell death.

Methods: *A. precatorius* leaves were extracted successively with hexane, ethyl acetate and methanol by Soxhlet extraction. Aqueous extract was prepared by decoction at 50 $^{\circ}$ C. Extracts of *A. precatorius* leaves were used to treat selected cancer and normal cell lines for 72 h. Furthermore, 3-(4,5-dimethyl thiazol-2-yl) 2,5-diphenyl tetrazolium bromide assay was performed to determine cell viability. Analysis of cell cycle arrest, apoptosis assay and apoptosis protein expressions were determined by flow cytometry.

Results: Methanolic extract of *A. precatorius* leaves showed the lowest IC₅₀ on MDA-MB-231 cells at (26.40±5.40) μ g/mL. Flow cytometry analysis revealed that cell arrest occurred at G₀/G₁ phase and the apoptosis assay showed the occurrence of early apoptosis at 48 h in MDA-MB-231 cells treated with methanolic extract of *A. precatorius* leaves. Methanolic extract of *A. precatorius* leaves induced apoptosis by upregulation of Bax, p53 and caspase-3 and downregulation of Bcl-2.

Conclusions: Methanolic extract of *A. precatorius* leaves promotes MDA-MB-231 cell death by inducing cell cycle arrest and apoptosis possibly *via* the mitochondrial-related pathway.

1. Introduction

Apoptosis is a characterized form of cell death and is mostly studied. Known commonly as programmed cell death, apoptosis is the packaging of dying cells into fragments that are easily consumed and eliminated by phagocytes without disturbing the normal function of surrounding tissues[1]. Equilibrium between cell death and cell proliferation is important to avoid disruption of the cellular balance. Excessive apoptosis or deficient apoptosis is the cause of many clinical diseases including cancer^[2]. Apoptosis can be initiated through two separate pathways, the intrinsic or mitochondrial pathway and the extrinsic or death receptor pathway. In cancer management, apoptosis has become an important tool as a target by potent apoptosis-inducing agents, including both chemical and biological^[3].

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