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Caspase Inhibition Restrains Centchroman Induced Apoptosis in Human Breast Cancer Cells

Manisha Nigam[1], Ramesh Sharma[2] and Anil K. Balapure[3]

[1]Department of Biochemistry, Hemwati Nandan Bahuguna Garhwal University (A Central University), Srinagar (Garhwal), Uttarakhand-246174
[2,3]Tissue and Cell Culture Unit (TCCU), Central Drug Research Institute, Council of Scientific & Industrial Research, Lucknow, India-226001

Aims: Centchroman (CC) has been established as a potent antineoplastic agent in Human Breast Cancer Cells (HBCCs) previously by us (1, 2). This study was done to further elucidate the role of caspases events in its antineoplastic action.

Methodology: MCF-7 and MDA MB-453 human breast cancer cells were used for the study. Tamoxifen (TAM), a widely used antiestrogen was employed as a positive control. MCF-7/MDA MB-453 cells were plated in a 6-welled plate, pretreated with Z-VAD-FMK (30 μM) for 5 h and exposed to CC/TAM. Flow cytometry was performed as reported previously (Nigam et al., 2008). Cells with DNA content less than that of G0/G1-phase cells were considered to be apoptotic (sub-G0/G1) using Cell Quest software.

Key findings: Control, untreated cells of both the types exhibits basal level of cells in sub-G0/G1 (apoptotic) fraction depicting nondescript apoptosis. The exposure of both the cell types to CC/TAM at their respective IC50 doses rapidly increases the apoptotic fraction. However, the pretreatment of ZVAD-FMK significantly but not entirely inhibits apoptosis at IC50 doses for CC in both, MCF-7/MDA MB-453cells.

Significance: Results confirm the involvement Caspase-independent pathways (3) may account for the observed partial inhibition of CC-induced apoptosis by pan caspase-inhibitor ZVAD-FMK.

References:

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