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A3 Adenosine receptor agonist (IB-MECA) induced apoptosis in OVCAR3 ovary cancer cell line

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Background and Aims: A3 Adenosine receptor has shown several physiological and pathological activities, including cell proliferation and apoptosis in various cancer cell lines. This study is designed to investigate apoptotic pathway of A3 adenosine receptor in OVCAR3 human ovary cancer cells.
Methods: MTT viability and BrdU incorporation tests were used to study the cell proliferation effect of IB-MECA. Annexin V-FITC staining, mitochondrial membrane potential (ΔΨM), caspase3 activity, Bcl-2 and Bax protein expression were used to detect apoptosis.
Results: The MTT and BrdU incorporation assay revealed that IB-MECA reduced cells proliferation in a dose dependent manner in the OCAR3 cell line. IB-MECA at (10-100 □M) induced apoptosis. The activity of caspase 3 was also increased. Expression of Bcl-2 was decreased in response to IB-MECA, while the expression of Bax protein was increased. The results showed a significant loss of ΔΨM, in a dose-dependent manner.
Conclusions: This study introduces a possible mechanism through A3 adenosine receptor activation. IB-MECA inhibited ovary cancer cells proliferation and induced apoptosis. Apoptosis determined by increased in early apoptotic population. Loss of MMP, activation of caspase3 and down-regulation of Bcl-2 expression indicated mitochondrial signaling pathway that involved in the apoptosis.

Keywords: Ovary cancer; A3 Adenosine receptor; Apoptosis