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Cytotoxic effects of Doxorubicin on human leukemia Jurkat cells: Drug interactions with quercetin

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The cytotoxic effect of the anticancer drug doxorubicin (DOX) in a human leukemia Jurkat T cell line was determined by flow cytometric assays of cell cycle, apoptosis/necrosis, oxidative status and mitochondrial Ca^{2+} level. 18-h and 45-h DOX-exposure induced apoptosis with IC50=951 nM, and $IC50_1$=135 nM/$IC50_2$=1.92 M (bimodal), respectively, which was accompanied by significant oxidative stress generation (IC50=620 nM). The addition of the flavonoid quercetin (QC) (10 μM) resulted in a significant decrease of cell viability for DOX levels <100 nM. DOX induced cell cycle arrest displaying a trimodal distribution, so that low, intermediate and high doses of DOX specifically produced G2/M, S and G0/G1 blockage with IC50 of 49 nM, 464 nM and 1866 nM, respectively. QC (15 μM) exerted strong antioxidant effects, reducing DOX-induced oxidative stress and apoptosis (IC50=2119 nM and 4897 nM, repectively). However, cell cycle arrest induced by low and moderate doses of DOX was maintained in the presence of QC levels <25 μM. DOX induced substantial mitochondrial depolarization within 4-h in a dose-dependent manner (IC50=0.200 nM). A 15-min exposure to DOX induced an immediate decrease of mitochondrial Ca^{2+} level IC50=18.3 μM and the addition of QC (15 μM) amplified this effect for a concentration of DOX <20 μM but resulted in an increase of mitochondrial Ca^{2+} for higher concentrations.

Biography

Vlad Cosoreanu is an undergraduate student of Medicine at Carol Davila University of Medicine and Pharmacy in Bucharest and currently doing research in the Department of Biophysics.

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