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Analysis of convergent effects of doxorubicin, quercetin and menadione in treatments of human leukemia Jurkat T cells

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Given the major diversity of cancer forms, development of treatments with specific functions is of great importance. In this *in vitro* study, leukemia Jurkat cells interactions with antitumoral antibiotic Doxorubicin (DOX) were investigated, and their modulation by flavonoid Quercetin (QC) and Menadione (MD). Cell cycle, apoptosis/necrosis and oxidative status were assessed by flow cytometry. DOX acts on malignant cells via cell-cycle arrest, mitochondrial depolarization and oxidative stress. Cellular viability dose-dependently decreased after 18 h and 48 h exposure to DOX (IC50=571 nM and 93 nM, respectively), while the sub-G0 cell fraction increased (IC50=385 nM and 128 nM, respectively). Also, 0.1 μM and 1 μM DOX arrested the cell cycle in G2/M (63% cells) and S phase (70%), respectively. In 18 h treatments, the 15 μM equimolar QC/MD combination produced by itself 52% cell death rate, by oxidative stress generation and apoptosis induction, and enhanced DOX cytotoxicity, dramatically decreasing the viable cell fraction (IC50=1.25 μM). 15 μM QC with 7.5 μM MD produced 69% viable cells; association with DOX exhibited additive cytotoxicity (IC50=2.26 μM). 48 h exposures further enhanced the effects. Addition of QC/MD up to 7.5 μM and 2.5 μM, respectively, to the 0.1 μM and 1 μM DOX-plan, increased the S-cell fraction, with higher levels progressively increasing the G0/G1 cell fraction. Therefore, QC/MD combination appears to be a good addition to DOX-treatments.

Biography

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