Abstract

Apoptosis is a physiological process regulated by a delicate balance between pro and anti-apoptotic factors, which plays a critical role in cell growth. Defects in its balance may lead to a selective advantage in cell survival that promotes neoplasia and malignancy. NFkB enhances cell proliferation thus, the use of selective inhibitors induces apoptosis. The aim of the present study was to characterise the cell line PL104 obtained in this laboratory from bone marrow cells of a young patient with an atypical acute myeloid leukaemia (AML) and to evaluate its susceptibility against the chemotherapeutic agents Doxorubicine (DOX), Vincristine (VCR) Gemcitabine and the inhibitors of NFkB, CAPE and MG-132. PL104 phenotype, evaluated by flow cytometry, showed expression of the following markers: CD19, CD20, CD22, CD38, CD45, CD79a, HLA-DR and lambda chain plus aberrant expression of CD71. The effect of these compounds on cell proliferation was evaluated resulting in 5.2 ± 0.7%, 42.8 ± 3.6%, 2.4 ± 0.4%, 0.7 ± 0.2% and 0.7 ± 0.3% (p < 0.001) of cell growth for DOX (4 µM), VCR (10 µM), Gemcitabine (0.04 µM), CAPE (360 µM) and MG-132 (4 µM) respectively after 24 h of treatment. Apoptosis assessed by acridine orange and ethidium bromide staining after 24 h showed 28.9 ± 1.9%, 47 ± 2.7%, 77.1 ± 5.7% and 92.8 ± 2.9% (p < 0.001) for DOX (1.5 µM), VCR (1 µM), CAPE (360 µM) and MG-132 (4 µM) respectively versus 6.63 ± 0.2% for untreated cells, data confirmed by Annexin-V test. It can be concluded that the cell line PL104, originated from an AML, corresponds to B lymphocytes significantly susceptible to the chemotherapeutic agents and inhibitors tested. Therefore, NFkB implication in controlling these cells’ survival has been demonstrated.

Keywords

acute myeloid leukemia * apoptosis * nuclear transcription factor kB * doxorubicine * vincristine * gemcitabine * caffeic acid phenylethyl ester * MG-132