A組、藥物化學暨藥理學組

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An indole-3-carbinol derivative induces apoptosis and autophagy

in AML cells

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Recent studies showed that phosphoinositide 3-kinase (PI3K)/Akt signaling is frequently activated in acute myeloid leukemia (AML) patient blasts and strongly contributes to proliferation, survival and drug resistance of these cells. The current therapeutic regimen for AML involves traditional chemotherapeutics which unfortunately have poor efficacy and high toxicity. Indole-3-carbinol (I3C) suppressed constitutive NF-κB activation in mononuclear cells derived from bone marrow of acute myelogenous leukemia patients, and this correlated with inhibition of cell growth. In this study, A9M · an I3C derivative which was evaluated the cytotoxic effect in AML cells (HL-60 and THP-1). This compound inhibited cell growth of HL-60 and THP-1 cells with IC₅₀ of 2.4 · 2.5 μ M, and flow cytometry analysis indicated it induced apoptosis. In addition, it downregulated Akt phosphorylation and induced dose-dependent increases in the proteolytic cleavage of PARP, caspase-3 and caspase-9, and the expression of mTOR and LC3B-II of THP-1 cells.