# Mechanism of cytotoxic activity of chalcone derivatives against K562 leukemia cell lines

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| **Title** | Mechanism of cytotoxic activity of chalcone derivatives against K562 leukemia cell lines |
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| **Abstract** | Two chalcone derivatives i.e. (E)-1-(4-aminophenyl)-3-(2,3dimethoxyphenyl)-prop-2-en-1-one (Compound-1), and (E)-1-(4-aminophenyl)-3-phenylprop-2-en-1-one) (Compound-2),has been proven to have potential cytotoxic activity. The aim of this study was toevaluate the effect of these compounds on PI3K/Akt signalling pathway in K562 celllines. After incubation with the tested compounds, AKT, caspase-3, STAT3 and cyclinD1 concentrations were measured using ELISA. Furthermore, cell cycle was analysedusing flowcytometry. Imatinib and isotretinoin were used as positive control, whereascell culture without treatment was used as negative control. The AKT concentration aftertreatment with Compound-1 and -2 was significantly lower than that control, imatiniband isotretinoin ($p<0.05$). The apoptotic indices after treatment with Compound-1 and-2 were significantly higher than control, however they were lower than imatinib andisotretinoin ($p<0.05$). The caspase-3 concentration after treatment with Compound-1 at5 and 10 ÃŽÂ¼g/mL and Compound-2 at 10 ÃŽÂ¼g/mL was significantly higher than that controland imatinib, however it was lower than isotretinoin ($p<0.05$). The STAT3 concentrationafter treatment with Compound-1 and -2 was significantly lower than that control andisotretinoin at 50 ÃŽÂ¼g/mL ($p<0.05$) and similar with imatinib ($p>0.05$). The cyclin D1concentration after treatment with Compound-1 and -2 was significantly lower than thatcontrol, imatinib and isotretinoin ($p<0.05$). In addition, Compound-1 and -2 arrested G0/G1 and G2/M phase in K562 cell lines, with comparable results to imatinib and isotretinoin.In conclusion, the mechanism of cytotoxic activity of Compound-1 and -2 are through thePI3K/Akt signalling pathway inhibition, apoptosis induction by upregulation of apoptoticmarkers, and inhibition of cell cycle progression by regulating cell cycle-related factors. |
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