GLA provoca apoptose na leucemia mielógena crônica humana

**Gamma-linolenic acid induces apoptosis and lipid peroxidation in human chronic myelogenous leukemia K562 cells.**

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**Source**

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**Abstract**

Various polyunsaturated fatty acids, especially gamma-linolenic acid (GLA), inhibit the growth of a variety of tumor cells. Some evidence indicates that polyunsaturated fatty acid can kill cells by apoptosis. In the current study, we tested the apoptotic effect of GLA on human chronic myelogenous leukemia K562 cells. GLA induced K562 cell death in a dose-dependent manner. Typical apoptotic nuclei were shown by staining of K562 cells with DNA-binding fluorochrome Hoechst 33342, characterized by chromatin condensation and nuclear fragmentation. Flow cytometric analysis also demonstrated that GLA caused dose-dependent apoptosis of K562 cells. The apoptosis could be inhibited by a pancaspase inhibitor (z-VAD-fmk), suggesting the involvement of caspases. Further, release of cytochrome c, activation of caspase-3 and cleavage of PARP were found in GLA-induced apoptosis. GLA treatment could also elevate lipid peroxidation in K562 cells, and antioxidant alpha-tocopherol could reverse the cytotoxicity of GLA. The saturated fatty acid SA, which did not exhibit significant increase in lipid peroxidation, also did not induce cytotoxicity. Intracellular GSH was also determined, and there was no marked change of GSH levels in cells after incubation with GLA compared with the control. These results demonstrate that GLA could induce apoptosis in K562 cells. Apoptosis is mediated by release of cytochrome c, activation of caspase-3. Lipid peroxidation may play a role in GLA cytotoxicity.

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