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POSTER

# Sodium citrate induces apoptosis in HepG2 cell lines

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## Abstract

**PURPOSES:** Cancer cells were observed to increase glucose uptake and fermentation of glucose to lactate to to synthesis rapidly ATP for cell growth, survival and proliferation. Thus, inhibition of glycolysis might be useful in antitumor treatment. This phenomenon occurred even with fully functioning mitochondria, and known as Warberg effect. Sodium citrate, an inhibitor of Warberg effect, was reported to antiproliferate many cancer cells line. However, sodium citrate has not been studied in Hepatocellular Carcinoma cells line yet. Here we aimed to investigate the effect of sodium citrate in HepG2 cells line.

**MATERIAL AND METHODS:** HepG2 cell lines was treated with sodium citrate at different concentrations. Viable cells were determined by Alamar Blue. The apoptosis induced-cells was detected by Annexin V with FCM technique. Disintegrated nuclei and DNA fragmentation was analyzed. The activity of caspase-3 was also tested.

**RESULTS:** We observed that the IC50 value of sodium citrate on HepG2 is at 10mM. FCM analysis showed that sodium citrate induced apoptosis in HepG2 cell line in dose-dependent manner. At 10mM sodium citrate, the caspase-3/7 was observed to be activated in time-dependent manner. Sodium citrate also induced nuclei disintergated in HepG2. DNA fragmentation was observed when HepG2 cells were treated with 10mM sodium citrate.

**CONCLUSIONS:** We have shown that sodium citrate possesses the antiproliferative ability on HepG2 at IC50 10mM. Sodidum citrate induces apoptosis cells in hepatocellular carcinoma HepG2 by capases-3 activation. More investigation of glycolysis inhibition of sodium citrate on HepG2 should be performed in animals

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Apoptosis, caspase-3/7, glycolysis, HepG2, sodium citrate

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## References



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