α-Santalol, a terpenoid found in sandalwood oil, has been shown to inhibit cancer cell growth in vitro by inducing apoptosis. This study was performed to investigate the antitumor properties of α-santalol associated with the induction of apoptosis in cultured MCF-7 (estrogen receptor (ER) positive, and wild type p53) and MDA-MB-231 (ER-negative and mutant p53) breast cancer cells. Expression of major proteins examined in the study were determined using standard Western blot protocol and analyzed by LICO- Odyssey infra-red scanner. Total protein levels of survivin were confirmed by survivin ELISA kit. Cell viability was assessed by trypan blue dye exclusion assay, and caspase-3 activity was determined by caspase-3 (active) ELISA kit. Treatment of breast cancer cells for 6 and 9 hour time intervals with α-santalol resulted in statistically significant concentration-dependent downregulation of survivin. pAkt levels were found to be slightly upregulated despite the down regulation of survivin. Pharmacological inhibition of PI3K-Akt pathway, Ly294002, did not provide a significant increase in cell death. Therefore, synergistic/additive increase was not observed.

**CONCLUSIONS**

- Treatment of breast cancer cells with α-santalol resulted in a statistically significant concentration-dependent reduction in survivin levels.
- Expression of pAkt levels were found to be slightly upregulated despite survivin downregulation in both cell lines.
- Treatment with α-santalol in combination with an inhibitor of the PI3K-Akt pathway, Ly294002, did not provide a significant increase in cell death. Therefore, synergistic/additive increase was not observed.
- Survivin downregulation occurred independent of the PI3K-Akt pathway.
- α-Santalol mediated cell death is not regulated through the PI3K-Akt pathway.

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