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# Evaluation of ergosterol and its metabolites as LXR agonists and their anticancer potential in colon cancer

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## ABSTRACT

**Purpose:** Aberrant cholesterol homeostasis is a well-recognized hallmark of cancer and implicated in metastasis and chemotherapeutic resistance, the two major causes of cancer associated mortality. Liver X receptors (LXRs) are the key transcription factors that induce cholesterol efflux via enhancing the expression of ABCA1 and ABCG1.

**Methods:** Molecular docking and dynamic simulation studies were done to assess the binding affinity and stability of the receptor ligand complexes. Activation of LXRs was evaluated using the luciferase reporter assay. qRT-PCR and western blotting was done to analyse the mRNA and protein expression of cholesterol homeostasis genes. Flow cytometric analysis was carried out to evaluate the surface expression of ABCA1. The effect of selected sterols on viability of three cancer cell lines and one normal epithelial cell line was assessed using MTT assay.

**Results:** Ergosterol (Erg), ergosta-7,22,24(28)-trien-3 $\beta$ -ol (Erg1), ergosta-5,22,25-trien-3-ol (Erg2), ergosta-5,7,22,24(28)-tetraen-3 $\beta$ -ol (Erg3), and ergosta-7,22-dien-3 $\beta$ -ol (Erg4) displayed good binding affinities and formed stable complexes with both isoforms of LXRs. Treatment with Erg led to 2.5 fold while Erg2 and Erg4 led to 1.7 fold increase in LXR activation. Furthermore, a significant increase in mRNA expression of *NR1H2*, *ABCA1*, *ABCG1* and *ApoE* was observed upon Erg treatment and it also led to a 25 fold increase in cell surface expression of ABCA1. All of the sterol were selectively toxicity toxic towards colorectal cancer cells but not towards normal epithelial cells.

**Conclusion:** Our findings suggests that ergosterol activates LXR $\beta$  and have significant anticancer activity and thus it could be a likely candidate to manage aberrant cholesterol homeostasis associated with colorectal cancer.