

## Apoptotic Induction Ability of Harmalol and Its Binding: Biochemical and Biophysical Perspectives

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**Abstract :** Harmalol administration caused remarkable reduction in proliferation of HepG<sub>2</sub> cells with GI<sub>50</sub> of 14.2 mM, without showing much cytotoxicity in embryonic liver cell line, WRL-68. Data from circular dichroism and differential scanning calorimetric analysis of harmalol-CT DNA complex shows conformational changes with prominent CD perturbation and stabilization of CT DNA by 8 °C. Binding constant and stoichiometry was also calculated using the above biophysical techniques. Further, dose dependent apoptotic induction ability of harmalol was studied in HepG<sub>2</sub> cells using different biochemical assays. Generation of ROS, DNA damage, changes in cellular external and ultramorphology, alteration of membrane, formation of comet tail, decreased mitochondrial membrane potential and a significant increase in Sub G<sub>0</sub>/G<sub>1</sub> population made the cancer cell, HepG<sub>2</sub>, prone to apoptosis. Up regulation of p53 and caspase 3 further indicated the apoptotic role of harmalol.

**Keywords :** apoptosis, beta carboline alkaloid, comet assay, cytotoxicity, ROS

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