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Cinnamic acid prevents diabetic pathophysiological complication and inhibits non-enzymatic glycation by binding to subdomain IIA of HSA: A mechanistic approach

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Under hyperglycaemic conditions, non-enzymatic glycation of proteins gives rise to advanced glycation end products (AGEs). The AGEs thus formed generate free radicals, which foster the development of diabetes and its associated complications. Inhibition of glycation is expected to play a role in controlling diabetes. Several plant derived antioxidants including cinnamic acid (CA) are known for limiting AGE formation; however, the mechanism involved is poorly understood. Therefore, we aimed to investigate the possible mechanism of inhibition of AGEs formation by CA through various experimental approaches. Glycation of HSA was achieved by incubating the reaction mixture with glucose for 30 days at 37 °C. The protein samples were tested for levels of free lysine and thiol group, carbonyl content and reactive oxygen species (ROS). Interaction between CA and HSA was also studied through various biophysical techniques. Thermodynamic studies showed strong exothermic interaction between CA and HSA. The positive value of TΔS° and negative value of ΔH° indicates that HSA-CA complex is mainly stabilized by hydrophobic interaction and hydrogen bond. Further, molecular docking reveals that CA binds to HSA subdomain IIA (Sudlow's site I) with a binding energy of -7.0 kcal mol-1, nearly the same as obtained in isothermal titration calorimetry (ITC) and fluorescence spectroscopy. Results of various spectroscopic techniques along with molecular docking and examination of many biomarkers highlights the role of CA in preventing disease progression.

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