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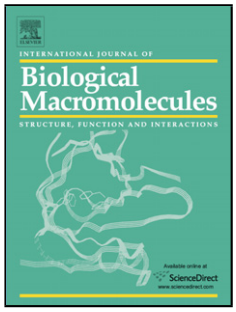

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Fructose-human serum albumin interaction undergoes numerous biophysical and biochemical changes before forming AGEs and aggregates

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HIGHLIGHTS

- Fructose modification brings about structural perturbations in HSA
- Fructose forms fluorescent and non-fluorescent AGEs in HSA
- Fructosylated-HSA-AGEs may undergo amyloid aggregation
- Aggregates accumulation in liver may set the stage for liver hepatopathy

Abstract

Fructose is a reducing and highly lipogenic sugar that has unique metabolic effects in the liver. Non-enzymatic fructosylation of proteins generates advanced glycation end products (AGEs). Human serum albumin (HSA) may undergo fructosylation vis-à-vis AGEs formation. High fructose consumption may lead to structurally altered and functionally compromised fructosylated-HSA-AGEs, which can cause damage to hepatocytes resulting in hepatic macro- and microvesicular steatosis. In this study,