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Preferential recognition of advanced glycation end products by serum antibodies in diabetes mellitus

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This study analyses the detrimental effect of non-enzymatic glycation on human serum albumin (HSA) leading to the production of advanced glycation end products (AGEs). HSA (20 μM) incubated with glucose (400 mg/dL) formed AGEs confirmed by scanning electron microscopy. DNA-damage in subjects with diabetes mellitus was assessed with comet assay. Antibodies against *in vitro* formed AGEs were evaluated in the sera of diabetic patients by enzyme-linked immune-sorbent assay, Molecular docking to demonstrate affinity of native and glycosylated HSA with IgG. Low-grade systemic inflammation was quantified with IL-4, IL-6, TNF-α and NF-κβ in serum and mRNA expression. The SEM showed the formation of aggregates in glycated-HSA. Serum auto-antibodies from diabetes patients with chronic kidney disease (CKD) showed appreciably high recognition of glycated-HSA compared to native HSA. Comet showed severe DNA damage in subjects with CKD compared to healthy. Molecular docking showed less affinity of glycosylated-HSA with IgG compared to native-HSA. Serum IL-4, IL-6 and TNF-α were found significantly higher in subjects with CKD compared to T2DM and healthy. mRNA expression of IL-4, IL-6 and NF-κβ are also found significantly higher in CKD. The non-enzymatic glycation-induced damage to the HSA and generate neo-epitopes that possess immunogenic response and low-grade systemic inflammation.

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