

**Breakers of advanced glycation end products restore large artery properties in experimental diabetes.**

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Glucose and other reducing sugars react with proteins by a nonenzymatic, posttranslational modification process called nonenzymatic glycation. The formation of advanced glycation end products (AGEs) on connective tissue and matrix components accounts largely for the increase in collagen crosslinking that accompanies normal aging and which occurs at an accelerated rate in diabetes, leading to an increase in arterial stiffness. A new class of AGE crosslink "breakers" reacts with and cleaves these covalent, AGE-derived protein crosslinks. Treatment of rats with streptozotocin-induced diabetes with the AGE-breaker ALT-711 for 1-3 weeks reversed the diabetes-induced increase of large artery stiffness as measured by systemic arterial compliance, aortic impedance, and carotid artery compliance and distensibility. These findings will have considerable implications for the treatment of patients with diabetes-related complications and aging.

PMID: 9539789 [PubMed - indexed for MEDLINE]