



Advanced glycation end products: sparking the development of diabetic vascular injury.

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Advanced glycation end products (AGEs) are proteins or lipids that become glycated after exposure to sugars. AGEs are prevalent in the diabetic vasculature and contribute to the development of atherosclerosis. The presence and accumulation of AGEs in many different cell types affect extracellular and intracellular structure and function. AGEs contribute to a variety of microvascular and macrovascular complications through the formation of cross-links between molecules in the basement membrane of the extracellular matrix and by engaging the receptor for advanced glycation end products (RAGE). Activation of RAGE by AGEs causes upregulation of the transcription factor nuclear factor-kappaB and its target genes. Soluble AGEs activate monocytes, and AGEs in the basement membrane inhibit monocyte migration. AGE-bound RAGE increases endothelial permeability to macromolecules. AGEs block nitric oxide activity in the endothelium and cause the production of reactive oxygen species. Because of the emerging evidence about the adverse effects of AGEs on the vasculature of patients with diabetes, a number of different therapies to inhibit AGEs are under investigation.

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