



Protein glycation: a firm link to endothelial cell dysfunction.

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The advanced glycation end products (AGEs) are a heterogeneous class of molecules, including the following main subgroups: bis(lysyl)imidazolium cross-links, hydroimidazolones, 3-deoxyglucosone derivatives, and monolysyl adducts. AGEs are increased in diabetes, renal failure, and aging. Microvascular lesions correlate with the accumulation of AGEs, as demonstrated in diabetic retinopathy or renal glomerulosclerosis. On endothelial cells, ligation of receptor for AGE (RAGE) by AGEs induces the expression of cell adhesion molecules, tissue factor, cytokines such as interleukin-6, and monocyte chemoattractant protein-1. A chief means by which AGEs via RAGE exert their effects is by generation of reactive oxygen species, at least in part via stimulation of NADPH oxidase. Diabetes-associated vascular dysfunction in vivo can be prevented by blockade of RAGE. Thus, agents that limit AGE formation, increase the catabolism of these species, or antagonize their binding to RAGE may provide new targets for vascular protection in diabetes.

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