

Advanced glycation endproducts (AGEs) induce oxidant stress in the gingiva: a potential mechanism underlying accelerated periodontal disease associated with diabetes.

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We hypothesized that one mechanism underlying advanced periodontal disease in diabetes may involve oxidant stress in the gingiva, induced by the effects of Advanced Glycation Endproducts (AGEs), the irreversible products of non-enzymatic glycation and oxidation of proteins and lipids which accumulate in diabetic plasma and tissue. Infusion of AGE albumin, a prototypic ligand, into mice resulted in increased generation of thiobarbituric acid reactive substances (TBARS) compared with infusion of non-glycated albumin in the gingiva, as well as in the lung, kidney and brain. Pretreatment of the animals with the antioxidants probucol or N-acetylcysteine (NAC) prevented the generation of TBARS in the gingiva. Affinity-purified antibody to AGEs demonstrated increased immunoreactivity for AGEs in the vasculature and connective tissues of the gingiva in streptozotocin-induced diabetic mice compared to non-diabetic controls. Increased immunoreactivity for AGEs was also demonstrated in the gingiva of diabetic humans compared with non-diabetic individuals via immunohistochemistry and ELISA. Consistent with these data, immunohistochemistry for heme oxygenase-1, a marker of enhanced oxidant stress, was increased in the gingival vasculature of diabetic mice and humans compared with non-diabetic controls. These data suggest that AGEs present in diabetic gingiva may be associated with a state of enhanced oxidant stress, a potential mechanism for accelerated tissue injury.

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