

1: [Diabetes](#). 2002 Nov; 51(11):3274-82.



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**Reduction of the accumulation of advanced glycation end products by ACE inhibition in experimental diabetic nephropathy.**

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The effect of ACE inhibition on the formation of advanced glycation end products (AGEs) and oxidative stress was explored. Streptozocin-induced diabetic animals were randomized to no treatment, the ACE inhibitor ramipril (3 mg/l), or the AGE formation inhibitor aminoguanidine (1 g/l) and followed for 12 weeks. Control groups were followed concurrently. Renal AGE accumulation, as determined by immunohistochemistry and both serum and renal fluorescence, were increased in diabetic animals. This was attenuated by both ramipril and aminoguanidine to a similar degree. Nitrotyrosine, a marker of protein oxidation, also followed a similar pattern. The receptor for AGEs, gene expression of the membrane-bound NADPH oxidase subunit gp91phox, and nuclear transcription factor-kappaB were all increased by diabetes but remained unaffected by either treatment regimen. Two other AGE receptors, AGE R2 and AGE R3, remained unchanged for the duration of the study. The present study has identified a relationship between the renin-angiotensin system and the accumulation of AGEs in experimental diabetic nephropathy that may be linked through oxidative stress

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