

Advanced glycation end products and diabetic complications.

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Diabetic complications are major cause of morbidity and mortality in patients with diabetes. While the precise pathogenic mechanism(s) underlying conditions such as diabetic retinopathy, diabetic nephropathy and increased risk of atherosclerosis remain ill-defined, it is clear that hyperglycaemia is a primary factor that initiates and promotes complications. Formation of advanced glycation end products (AGEs) correlate with glycaemic control, and these reactive adducts form on DNA, lipids and proteins where they represent pathophysiological modifications that precipitate dysfunction at a cellular and molecular level. Many of these adducts form rapidly during diabetes and promote progression of a raft of diabetes-related complications. Recent evidence also suggests an important interaction with other pathogenic mechanisms activated within the diabetic milieu. This review outlines the nature of AGE formation in biological systems and highlights accumulative evidence that implicates these adducts in diabetic complications. As more therapeutic agents are developed to inhibit AGE formation or limit their pathogenic influence during chronic diabetes, it is becoming clear that these anti-AGE strategies have an important role to play in the treatment of diabetic patients.

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