

Advanced glycation end products in kidney transplant patients: a putative role in the development of chronic renal transplant dysfunction.

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Chronic renal transplant dysfunction is one of the leading causes of graft failure in kidney transplantation. A complex interplay of both alloantigen-related and alloantigen-unrelated risk factors is believed to underlie its development. We propose that advanced glycation end products (AGEs) are involved in the development of chronic renal transplant dysfunction. AGE formation is associated with different alloantigen-unrelated risk factors for chronic renal transplant dysfunction, such as recipient age, diabetes, proteinuria, hypertension, and hyperlipidemia. In vitro studies have shown that AGEs induce the expression of various mediators associated with chronic renal transplant dysfunction. Furthermore, AGE-induced renal damage has been found in multiple experimental studies. This renal damage shows similarity to the damage found in chronic renal transplant dysfunction. Together, several lines of evidence support a role of AGEs in the development of chronic renal transplant dysfunction and suggest that preventive therapy with AGE inhibitors may be helpful in preserving renal function in transplant recipients.

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