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Adenosine monophosphate-activated protein kinase in diabetic kidney disease

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Diabetic kidney disease is the leading cause of end-stage renal disease, and is a growing global health problem. Despite developments in pharmacological strategies to modulate diabetes, diabetic kidney disease remains a major microvascular complication in many patients with diabetes and its pathogenesis has not yet fully elucidated. A peculiarity of this disease is the accumulation of glucose and lipids in renal cells, resulting in oxidative and endoplasmic reticulum stress, intracellular hypoxia, and inflammation, eventually leading to glomerulosclerosis and interstitial fibrosis. In an attempt to unravel the core of this disease entity, a growing body of evidence shows that dysregulation of 5′ adenosine monophosphate-activated protein kinase (AMPK) in relevant tissues is crucial to the development of diabetes. AMPK is metabolic master switch that regulates downstream signals based n shifts in the surrounding energy reservoir: the net effect of AMPK activation is an increased cellular energy level via the inhibition of anabolic energy-consuming pathways, as well as the simulation of catabolic, energy-producing pathways. AMPK plays a major role in glucose hemostasis by modulating glucose transport in peripheral tissues and lipid metabolism via the regulation of fatty acid oxidation and cholesterol synthesis, of which disturbed action has been elucidated in diabetic kidney disease. Targeting this enzyme may ameliorate some pathologic features of this disease proven to improve glucose and lipid homeostasis in diabetic experimental models, as well as demonstrating mitochondrial biogenesis and antitumor activity. The aims of this talk are to give an overview on the role of AMPK in the pathogenesis in diabetic kidney disease and to summarize the emerging therapeutic agents in the management of diabetic kidney disease, especially with a new insight of functional foods as a novel AMPK activator.