Early molecular mechanisms of diabetic kidney disease

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Abstract

Diabetic kidney disease is one of the major causes of death in patients with diabetes and the leading cause of end-stage renal disease in the United States. One early sign believed to contribute to diabetic kidney disease is injury to the glomerulus. The glomerulus is a cluster of blood vessels in the kidney that filters excess fluid and waste products out of the blood. However, the molecular mechanisms that lead to irreversible glomerular injury in the setting of diabetes are poorly understood. To gain a clearer picture, researchers evaluated the impact of the diabetic microenvironment on glomerular endothelial cells. Prolonged exposure to diabetic serum affected mitochondria, resulting in cellular dysfunction, but not cell death. It's possible that cells enter an adaptive state of cell dysfunction, inducing the secretion of molecules that promote apoptosis and pathogenic crosstalk with podocytes, epithelial cells that wrap around the glomerular capillaries, the depletion of which is the hallmark of progressive kidney disease. Moving from 2D cultures to 3D co-cultures could offer further insights into these mechanisms and possibly offer clues on how to stop or reverse the progression of diabetic kidney disease.