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Insulin resistance in diabetic nephropathy and its effect on metabolism and inflammation

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Diabetic nephropathy (DN) is one of the major complications of diabetic patients and is the leading cause of end-stage renal disease. Kidneys are major sites of deregulated glucose production in diabetic patients. It is therefore crucial to investigate the mechanisms involved in their pathogenesis. We analyzed the expression of the insulin receptor (IR) in renal cortex and proximal tubules from diabetic rats and humans. Surprisingly, in the kidney of both human type 2 diabetic patients and in a type 1 diabetic rat model, a significant reduction in the protein levels of IR and a consequent increase of PEPCK is produced. Thus, expression of IR protein in proximal tubules from type 1 and type 2 diabetic kidney indicates that this is a common regulatory mechanism which is altered in DN, triggering enhanced gluconeogenesis. Moreover, we detected activation of the GSK3β kinase and overexpression of muscle glycogen synthase (MGS) and glycogen deposition in the diabetic kidney from rat and human, that correlates with induced-cell death in a model of human renal tubule cells. This differential expression suggests the participation of MGS in the renal metabolic changes associated that also are induce the increased levels of inflammation markers (MCP-1, ICAM-1) and activation of NFk-B. Hence, altered expression of IR in human diabetic kidney could be one of the main triggers for enhanced inflammation and gluconeogenesis and the consequent kidney dysfunction in DN.

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