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Early alterations in renal intercellular junctions in diabetes

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Repensive treatments. Usually this complication is diagnosed at final stages. There is scant information on the initial damage of the kidney in these patients. The aim of this study was to identify the initial alterations on the renal mechanisms involved in the handling of sodium and glucose in streptozotocin-induced diabetes in rats. We focused on claudins, proteins of vascular and epithelial tight junctions (TJs). These junctions regulate the absorption of sodium and water at the proximal tubule. In this segment all filtered glucose is reabsorbed as well as 60 to 70% of filtered sodium. Hyperglycemia induced decrease of claudin 5 (vascular) and claudin 2 (epithelial) in kidney of diabetic rats. Increased nitration of tyrosine residues was identified that might contribute to dysfunction of this protein and to increased excretion of sodium and polyuria. Sodium/ glucose transporters (SGLT 1 and 2) and GLUT 1 were increased at the proximal tubule. These alterations were associated to oxidative stress, that was more severe in proximal than in distal tubules or glomeruli. We identified early damage in kidney that provides some clues on the deleterious factors leading to progressive renal failure in diabetes.

Biography

Jose L Reyes graduated at Medical School of National University of Mexico and Pediatric Nephrologist (Hospital Infantil, Mexico). He got PhD degree at Center for Research and Advanced Studies, Mexico, Postdoctoral training at Children's Hospital (Los Angeles CA). He has published 90 articles and authored 20 chapters. He was an invited Professor at Albert Einstein College of Medicine, NY, University of Laussane, Switzerland, University of Paris VI and at University of Nice, France. He was the former President of the Mexican Board of Nephrology and former Head of the Department of Pharmacology and Toxicology and of the Department of Physiology, at the Center for Research and Advanced Studies, Mexico and currently he is the full Professor at this institution.

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