Nephro-2013
OMICS Group Conferences
Accelerating Scientific Discovery
2nd International Conference on
Nephrology & Therapeutics
July 29-31, 2013 Embassy Suites Las Vegas, USA

Renal cell injury in diabetes

Samy L Habib
South Texas Veterans Health Care System, USA

Abstract

Diabetes is the leading cause of end-stage renal failure in most developed countries. Although vascular and glomerular injuries have been considered the main features of diabetic kidney diseases, tubular atrophy is also found. Proximal tubular functional and structural changes correlate better with diabetic nephropathy progression, and may be key to kidney dysfunction development in diabetes. Diabetes induces early signs of tubular dysfunction and diabetic kidneys are particularly prone to acute tubular necrosis in diverse clinical situations. Alterations in renal structure may occur that are not specific to nephropathy but reflect a consequence of long-standing diabetes/hyperglycemia. Renal hypertrophy, matrix protein accumulation and tubulointerstitial fibrosis are major pathological features of diabetic nephropathy (DN) that eventuate in renal failure. Hyperglycemia and high concentration of glucose increase matrix protein expression but the pathogenic mechanisms are not fully understood. We have previously reported that inactivation of tuberin resulting in activation of the mammalian target of rapamycin (mTOR) pathway and enhance matrix protein accumulation in cultured proximal tubular cells exposed to high glucose and in kidney cortex of rats with type1 diabetes. In this report, we show that kidney sections of diabetic patient express higher levels of phospho-tuberin (inactive form of tuberin) and that associated with increase in mTOR activation measured by phosphorylation level of p70S6K. Inactivation of tuberin and activation of mTOR lead to accumulate cell matrix proteins (fibronectin and collagen IV) mainly in tubular epithelial cells of the kidney of diabetic patient. In addition, the morphologic changes in kidney sections of diabetic patient showed tubular thickening, glomerular and tubular hypertrophy. These data suggest that alterations in tubular cells structure including tubular thickening and hypertrophy are major mediators of the fibrotic process in diabetic nephropathy.

Biography

Samy Habib has completed his Ph.D at Roswell Park Cancer Center, Buffalo, NY and trained at University of California, Irvine, University of Texas, Austin, TX and University of Texas Health Science Center, San Antonio (UTHSCSA), TX. He has published more than 35 papers in reputed journals and serving as an editorial board member and Editor of 7 journals. Dr. Habib is Associate Professor at UTHSCSA and holds Research Scientist position at the South Texas Veterans Health Care System at Audie Murphy VA Hospital. He was a recipient of several research grant awards from American Diabetes Association, American Heart Association, National Kidney Foundation, New Investigator Award and Merit Review Award from Veterans Affairs, and Pilot Research Award from NIH/NIDDK. He has recently received the Excellent of Performance Award from the VA. Dr. Habib has been a regular member of the Kidney Cancer Study Section of the Medical Research Program, and Tuberous Sclerosis Complex Research Program (TSCRP) study section, Department of Defense.