OFFICIAL ABSTRACT and CERTIFICATION

The Effects of Global Knockdown of Cytochrome C Oxidase Assembly Protein (Sco2) in Diabetic Kidney Disease

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The kidneys are mitochondrially rich, highly metabolic organs that require vast amounts of ATP for their normal function. However, in diabetes, the ATP production decreases in the mitochondria. Type 2 diabetes, characterized by insulin resistance, has been the leading cause of Chronic Kidney Disease(CKD) with about 35% of diabetic people aged 20 years or older who acquire CKD.1 Because the kidney requires large amounts of energy to function, the proper maintenance of mitochondria structure and function is crucial. Synthesis of Cytochrome C Oxidase (Sco2) has been identified as an essential protein for maintaining the function of mitochondria and for the assembly of cytochrome c oxidase, a key component of the mitochondrial respiratory chain. Although emerging evidence on the importance of Sco2 has been recently found, little is known about the role of Sco2 and the effects of the global knockdown of Sco2 in various kidney cells especially in a diabetic model. In this study, processed tissues were obtained and stained for different cell markers in various kidney cells (podocytes, endothelial cells, and tubular cells) by using immunofluorescence staining. Glomerular volume and mesangial expansion were qualitatively analyzed following Periodic-Acid Schiff's Staining. In this study, no significant difference was observed in tubular damage between the different genotype. Conversely, in the setting of diabetes and Sco2 mutation, podocyte injury seems to decrease and the Sco2 mutation seems to be a protective mutation. This is seen by podocyte marker staining and the albumin analysis. Finally, after staining for endothelial cell markers, there was a significant increase observed between wild type and the diabetic genotype and trend of an increase between the WT and the Sco2 knockdown genotype. Collectively, these data suggest that in a diabetic condition, the Sco2 mutation has no negative effect on tubules, a protective effect on podocytes, and a negative effect on endothelial cells.

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