

Novel Strategies for Detecting and Treating Podocyte Injury in Diabetic Nephropathy

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Diabetes is a multi-factorial disorder, characterized by chronic elevation of glucose in the blood. Chronic exposure to high glucose (HG) is a leading cause of cell damage, which is responsible for diabetes' numerous complications. Since diabetes is an energy metabolism dysfunction disease and mitochondria are the cells' power generators, it is believed that these organelles play a central role in the pathogenesis of diabetic complications. Consequently, finding new therapies to restore mitochondrial function is essential. Our study investigated if supplementation with beta nicotinic amide mononucleotide (NMN) or with a combination of NMN and cyclodextrin (MBCD) is effective in reducing podocyte injury in diabetic nephropathy. By utilizing advanced microscopy methods, this study indicated that human podocytes which have been exposed to HG conditions and were treated with these pharmacological agents displayed improved mitochondrial function in vitro. Treatment with NMN alone or with the combination of NMN and MBCD reduced co-localization with lipid droplets, partially restored mitochondrial volume (via confocal microscopy), improved altered mitochondrial shapes (via STED), reversed glycolysis and restored oxidative phosphorylation (via FLIM). The present study provides experimental evidence that NMN alone or in combination with MBCD attenuate mitochondrial changes and metabolic reprogramming in podocytes, which could be a potential therapeutic strategy for treating diabetic nephropathy.

Awards Won:

Fourth Award of \$500