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Adiponectin may improve diabetic nephropathy

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Diabetic nephropathy is a major microvascular complication. However, there are no entirely effective remedies to prevent the progression of diabetic nephropathy. Adiponectin has been shown to exert largely beneficial effects on insulin resistance and atherosclerosis. Recently, it has been reported that plasma adiponectin levels are inversely correlated with the degree of albuminuria in obese patients, and that adiponectin-deficient mice exhibit albuminuria and podocyte dysfunction. This suggests that adiponectin may play a protective role in improving kidney disease. However, the effects of adiponectin in diabetic nephropathy are still unknown. To investigate the effects of adiponectin in diabetic nephropathy, we used an adenovirus to chronically overexpress adiponectin (Ad-Adipo) in streptozotocin (STZ) induced diabetic nephropathy rats. At 10 weeks of STZ injection, diabetic rats were injected with Ad-Adipo or Ad-lacZ. At 2 weeks of adenovirus injection, fasting glucose levels was similar in two group, whereas the degree of proteinuria significantly reduced by 24.3% ($P<0.05$) in Ad-Adipo rats compared with Ad-lacZ rats, suggesting the improvement of early stage diabetic nephropathy in hyperadiponectinemia animals. Nephrin, a protein found in podocytes, is crucial for maintaining the integrity of the intact glomerular filtration barrier. Nephrin mRNA expression level was significantly increased by 68.0% ($P<0.01$) in the renal cortex of Ad-Adipo rats. Furthermore, endothelial dysfunction correlates with progression of diabetic nephropathy. ET-1 and PAI-1 mRNA expression levels were significantly decreased by 33.4% ($p<0.01$) and 34.7% ($P<0.01$) in the renal cortex of Ad-Adipo rats, respectively, whereas eNOS mRNA expression level was significantly increased by 117.2% in the renal cortex of Ad-Adipo rats ($P<0.01$). In conclusion, we have shown that chronic hyperadiponectinemia inhibits the progression of glomerulo-endothelial dysfunction in early stage diabetic nephropathy. The mechanism whereby adiponectin decreases proteinuria involves an increase of nephrin expression, and an improvement of the endothelial dysfunction due to decreases of ET-1 and PAI-1, and an increase of eNOS expression in the renal cortex. Thus, chronic overexpression of adiponectin has beneficial effects on early stage diabetic nephropathy.

Biography

Hiroaki Satoh, is Associate Professor of the Department of Nephrology, Hypertension, Diabetology, Endocrinology, and Metabolism at Fukushima Medical University. He received his MD from Akita University and his PhD from University of Tokyo Graduate School of Medicine. Before coming to Fukushima Medical University, he was a resident in Internal Medicine at the University of Tokyo Hospital, a graduate student at University of Tokyo Graduate School of Medicine, a research in Jerrold M Olefsky's lab at University of California, San Diego. He has been on the Fukushima Medical University faculty since 2004.

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