

IL-17 deficiency suppresses the streptozotocin-induced diabetic nephropathy through the regulation of autophagy in mice

Ju-Young Jung

Chungnam National University, Republic of Korea

Diabetic nephropathy (DN) is one of the most important medical complications of diabetes mellitus. Autophagy is an important mediator of pathological response and plays a critical role in inflammation during the progression of diabetic nephropathy. Interleukin (IL)-17A favorably modulates inflammatory disorders including DN. In this study, we examined whether IL-17A deficiency affected the autophagy process in the kidneys of mice with streptozotocin (STZ)-induced DN. The autophagic response of IL-17A to STZ-induced nephrotoxicity was evaluated by analyzing STZ induced functional and histological renal injury in IL-17A knockout (KO) mice. IL-17A KO STZ-treated mice developed more severe nephropathy than STZ-treated wild-type (WT) mice, with increased glomerular damage and renal interstitial fibrosis at 12 weeks. IL-17A deficiency also increased the up-regulation of proinflammatory cytokines and fibrotic gene expression after STZ treatment. Meanwhile, autophagy associated proteins were induced in STZ-treated WT mice. However, IL-17A KO STZ-treated mice displayed a significant decrease in protein expression. Especially, the levels of LC3 and ATG7, which play crucial roles in autophagosome formation, were notably decreased in the IL-17A KO STZ-treated mice compared with their WT counterparts. IL-17 deficiency aggravates of STZ-induced DN via attenuation of autophagic response. Our study demonstrated that IL-17A mediates STZ-induced renal damage and represents a potential therapeutic target in DN.

Biography

Ju-Young Jung has his expertise in evaluation and passion in improving the kidney and prostate disease. He is interested in animal welfare and health. He experienced in research, evaluation, and teaching and administration since 2006 at College of Veterinary Medicine in Chungnam National University.

jyung@cnu.ac.kr

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