

Abstract Type : Oral

Abstract Submission No. : 1539

Periostin deficiency attenuates kidney fibrosis in diabetic nephropathy via improving pancreatic β -cell dysfunction.

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Objectives: Diabetic nephropathy (DN) is associated with kidney fibrosis. A previous study revealed that periostin (POSTN) contributes to kidney fibrosis. This study examined the role of POSTN in DN.

Methods: POSTN and tenascin C (TNC) concentrations in urine samples from patients with DN were measured. Streptozotocin (STZ) was administered after unilateral nephrectomy (UNXSTZ) to induce DN in both wild-type and Postn-null mice. Four experimental groups were generated: wild-type sham (WT sham), wild-type UNXSTZ (WT STZ), Postn-null sham (KO sham), Postn-null UNXSTZ (KO STZ). After 20 weeks, the molecular expression of fibrosis markers and histological changes were evaluated. Blood glucose levels and urine albumin were periodically measured. As a cell model of DN, we stimulated INS-1 cells with streptozotocin and evaluated the viability of these cells.

Results: The concentrations of POSTN and TNC increased according to the severity of DN in human samples. The KO STZ group had lower urine albumin excretion, glomerular sclerosis, and interstitial fibrosis than the WT STZ group. Also, it had lower expression of fibrosis markers, including TNC. The KO STZ group showed better glucose regulation than the WT STZ model. Furthermore, the KO STZ group preserved pancreatic islet integrity and insulin expression significantly. The anti-POSTN antibody treatment of INS-1 cells with streptozotocin resulted in higher cell viability than treatment with streptozotocin alone.

Conclusions: POSTN has an essential role in both kidney fibrosis and pancreatic β -cell dysfunction in DN. Additionally, there is an association between POSTN and TNC.

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