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Cigarette smoke exacerbates kidney injury in diabetic rats

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Objectives: Chronic kidney disease (CKD) has become epidemic worldwide. Epidemiological studies have demonstrated that cigarette smoke promotes CKD including diabetic kidney injury. However, the mechanism of cigarette smoke in progression of CKD is incompletely understood, in part because basic studies using animal models are lacking. The present study aimed to establish experimental model for the mechanistic study of renotoxic effect of cigarette smoke.

Methods: Experimental diabetes was induced by a single intravenous injection of streptozotocin (STZ, 60 mg/kg) to 7-week-old Sprague Dawley rats. Four weeks after the induction of diabetes, rats were exposed to cigarette smoke (200 mg/L), 4 hours daily for 4 weeks.

Results: Cigarette smoke did not affect plasma glucose, HbA1c, and free fatty acid in both control and diabetic rats. Cigarette smoke, however, significantly increased diabetes-induced glomerular hypertrophy and urinary KIM-1 and NGAL excretion in diabetic rats. Cigarette smoke promoted macrophage infiltration as indicated by CD68-positive staining area in diabetic rats but not in control. The progression of diabetes-induced kidney fibrosis was also accelerated by cigarette smoke, presented by increased tubulointerstitial collagen accumulation. As expected, cigarette smoke increased oxidative stress in both control and diabetic rats.

Conclusions: These data demonstrated that 4 weeks exposure of cigarette smoke accelerated the progression of kidney injury in diabetic rats