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## Deletion of the Gene for Adiponectin Accelerates Age-Related Kidney Injury

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**Objectives :** Aging causes renal fibrosis, and aging related renal changes are characterized by oxidative stress. However, the role of adiponectin in aging process has not been elucidated. The present study was aimed to investigate the role of adiponectin in renal fibrosis in aging.

**Methods :** We used male 2 and 12 months old C57BL/6 (wild type, WT) mice and adiponectin knock out (APN-/-) mice. The fluorescent probe 2',7'-dichlorofluorescein diacetate was used to measure intracellular levels of reactive oxygen species (ROS). The protein expression of transforming growth factor β (TGF- β), Smad-2/3, Smad-4, Smad-6, α smooth muscle actin (α-SMA), collagen IV, pro-apoptoic Bax and anti-apoptotic protein Bcl-2, phosphorylated AMP-activated protein kinase (p-AMPK) was determined by semiquantitative immunoblotting. For the in vitro experiments, human proximal tubular epithelial (HK2) cells were treated with TGF- β with or without pretreatment of adiponectin.

**Results :** 12 month old APN-/- mice exhibited decreased body weight, increased albuminuria and kidney to body weight ratio compared to 12 months WT mice. Fibrosis markers such as α smooth muscle actin and collagen IV were increased. The protein expression of TGFβ, Smad-2/3, and Smad-4 was increased, while inhibitory Smad-6 decreased in 12 months APN-/- mice compared to WT mice. ROS generation was also increased. Apoptosis marker such as Bax expression was increased while Bcl-2 expression was decreased in 12 months APN-/- mice compared WT mice. Phosphorylation of AMP-activated protein kinase (AMPK) was decreased in 12 months APN-/- mice compared to WT mice. TGFβ treatment showed decreased AMPK phosphorylation in HK2 cells. Pre-treatment of adiponectin attenuated fibrosis markers, apoptosis marker expression and ROS generation.

**Conclusions :** Our results suggest that adiponectin plays a role in the pathogenesis of progressive kidney injury associated with aging process.

**Keywords :** Aging, adiponectin, fibrosis, Oxidative stress, apoptosis