Abstract Submission No. : 9088
May 27(Fri), 16:50-18:50 R&D in Nephrology

C01 inhibits the development of kidney fibrosis

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We demonstrate that the C01 protects against kidney injury and progression to CKD. In renal ischemia-reperfusion injury (IRI), C01 pre-treatment preserved kidney function and significantly reduced in tubular damage, apoptosis, and inflammatory cell infiltration. Compared with 5/6 nephrectomy (Nx) control rats, kidney function was protected, and fibrosis was attenuated in CHP-treated 5/6 Nx rats. CHP also improved kidney injury in a unilateral ureteral obstruction (UUO) model with prophylactic and therapeutic treatment regimens. Moreover, CHP plus zinc aggravates diabetic nephropathy by inhibiting inflammation and oxidative stress in KKAy mice. To explore the direct binding of C01 with CL1, the cellular thermal shift assay (CETSA) was applied. C01-treated cells exhibited enhanced thermal stability of CL1. CL1 knockdown abolished NRF2 expression level. Our data support the efficacy of C01 treatment in inhibiting the development of kidney fibrosis and its target protein CL1 was identified using CETSA, a label-free method. C01, which has an excellent safety record, may hence represent a novel approach to manage CKD.