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Oral Communication Abstract
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Depleted HDAC3 attenuates hyperuricemia-induced renal interstitial fibrosis via miR-19b-3p/SF3B3 axis

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Objectives: Dysfunctional histone deacetylases (HDACs) elicit unrestrained fibrosis and damages of organs. With regard to the link between HDACs and fibrosis, this research is practiced to decipher the concrete mechanism of HDAC3 in hyperuricemia (HN)-induced renal interstitial fibrosis (RIF) from microRNA-19b-3p/splicing factor 3b subunit 3 (miR-19b-3p/SF3B3) axis.

Methods: HN model was established on rats to induce RIF by oral administration of adenine and potassium oxalate. Rats were injected with miR-19b-3p- or HDAC3-related sequences to figure out their effects on RIF through detecting 24 h urine protein, uric acid (UA), blood urea nitrogen (BUN) and serum creatinine (Scr) contents in serum, and α-smooth muscle actin (α-SMA), transforming growth factor β1 (TGF-β1) and fibronectin (FN) contents in renal tissues, and observing pathological damages and RIF index of renal tissues. miR-19b-3p and SF3B3 levels in renal tissues were tested, along with their interactions.

Results: Elevated HDAC3 and SF3B3 and reduced miR-19b-3p were displayed in RIF. Suppressed HDAC3 or promoted miR-19b-3p relieved HN-induced RIF, as reflected by their inhibitory effects on 24 h urine protein, UA, BUN, Scr, α-SMA, TGF-β1 and FN contents and RIF index and their ameliorated effects on pathological damages of renal tissues. HDAC3 bound to the promoter of miR-19b-3p to regulate SF3B3. miR-19b-3p depletion abrogated down-regulated HDAC3-induced effects on RIF.

Conclusions: It is delineated that depressed HDAC3 relives HN-induced RIF through restoring miR-19b-3p and knocking down SF3B3, replenishing the references for RIF curing.

Figure 3. Up-regulation of miR-19b-3p improves renal function indexes of hyperuricemia rats

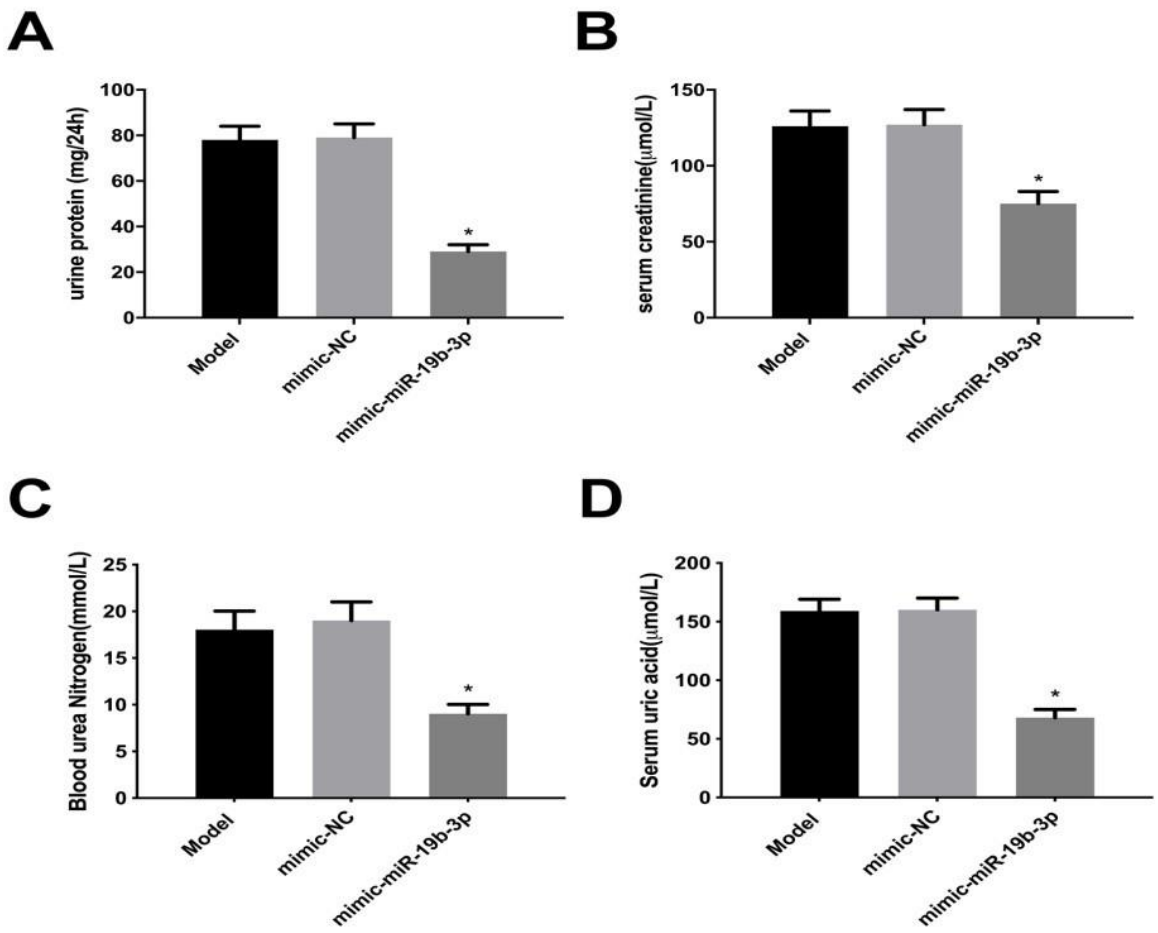

Figure 4. HDAC3 binds to the miR-19b-3p promoter to inhibit its expression.