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Supplemental Information

The Biomarker TCONS_00016233 Drives Septic AKI by Targeting the miR-22-3p/AIFM1 Signaling Axis

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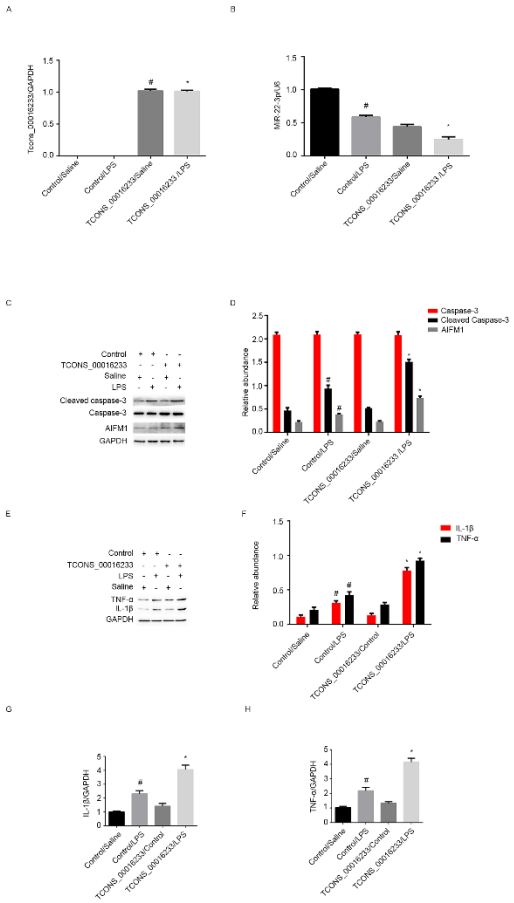
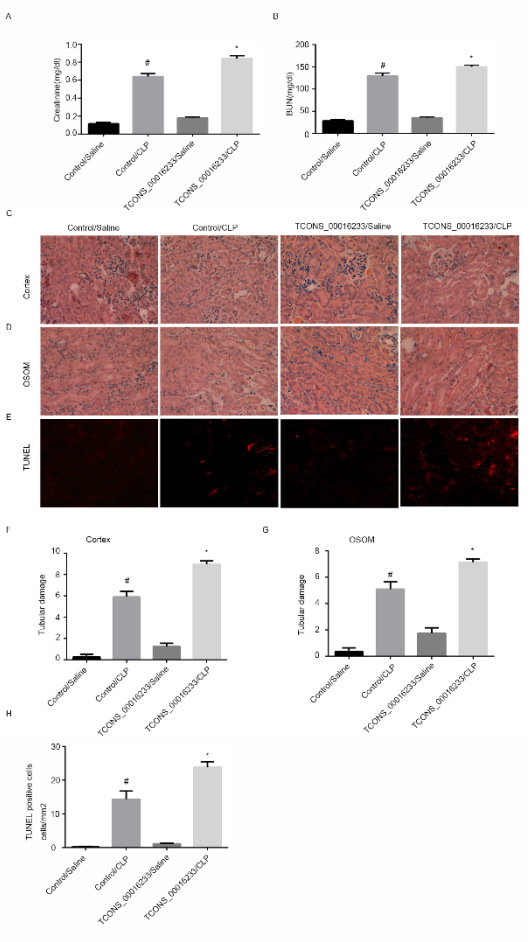
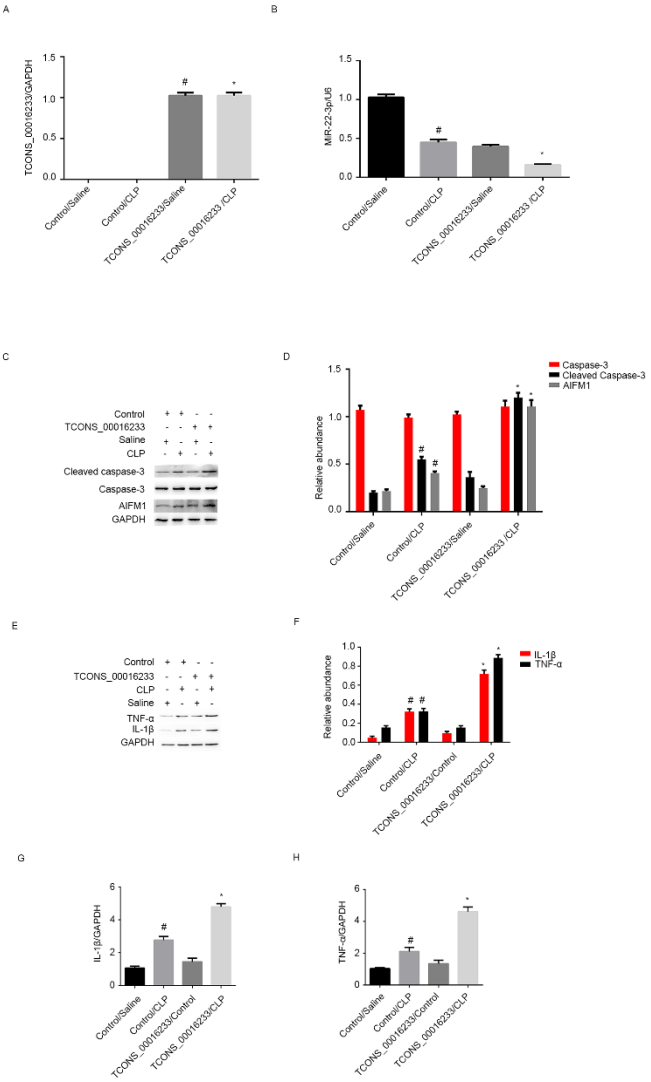


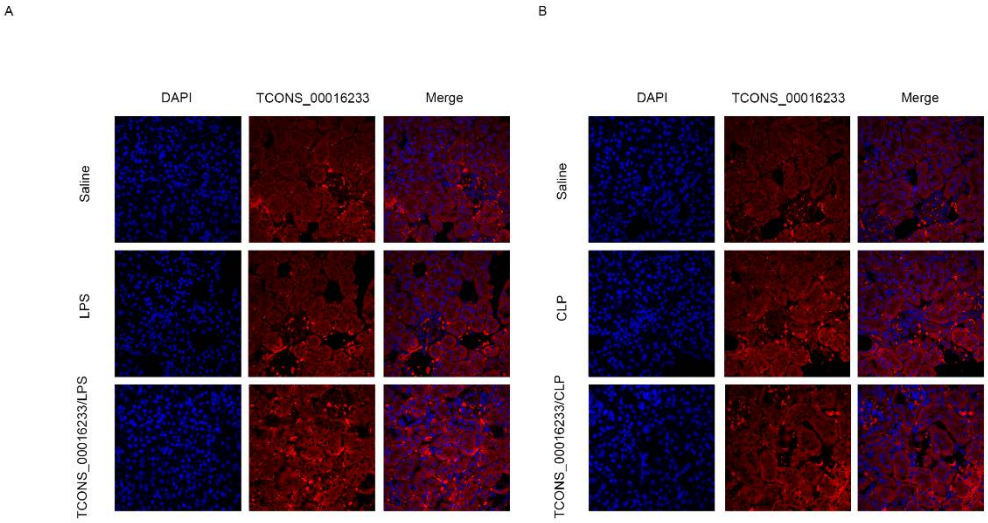
Supplementary figure1 . overexpression of TCONS_00016233 attenuated LPS-induced mice AKI via targeting miR-22-3p/AIFM1 axis. C57BL/6J mice was pretreated with the TCONS_00016233 plasmid via tail vein for 12h, and then injected with 10mg/kg LPS for24 hor saline as control. (A&B) RT-qPCR analysis of TCONS_00016233 and miR-22-3p.(C) Immunoblot analysis of cleaved caspase3, caspase3, and AIFM1. (D) Densitometric analysis of Immunoblot bands. (E) Immunoblot analysis of TNFα, IL-1β, and GAPDH. (F) Densitometric analysis of Immunoblot bands. (G&H) RT-qPCR analysis of the expression levels of IL-1β and TNFα. Data are expressed as mean ± SD (n=6). # *P<0.05*Control with LPS group vs. Saline group, * *P<0.05* TCONS_00016233 plasmid with LPS group vs.Control with LPS group.



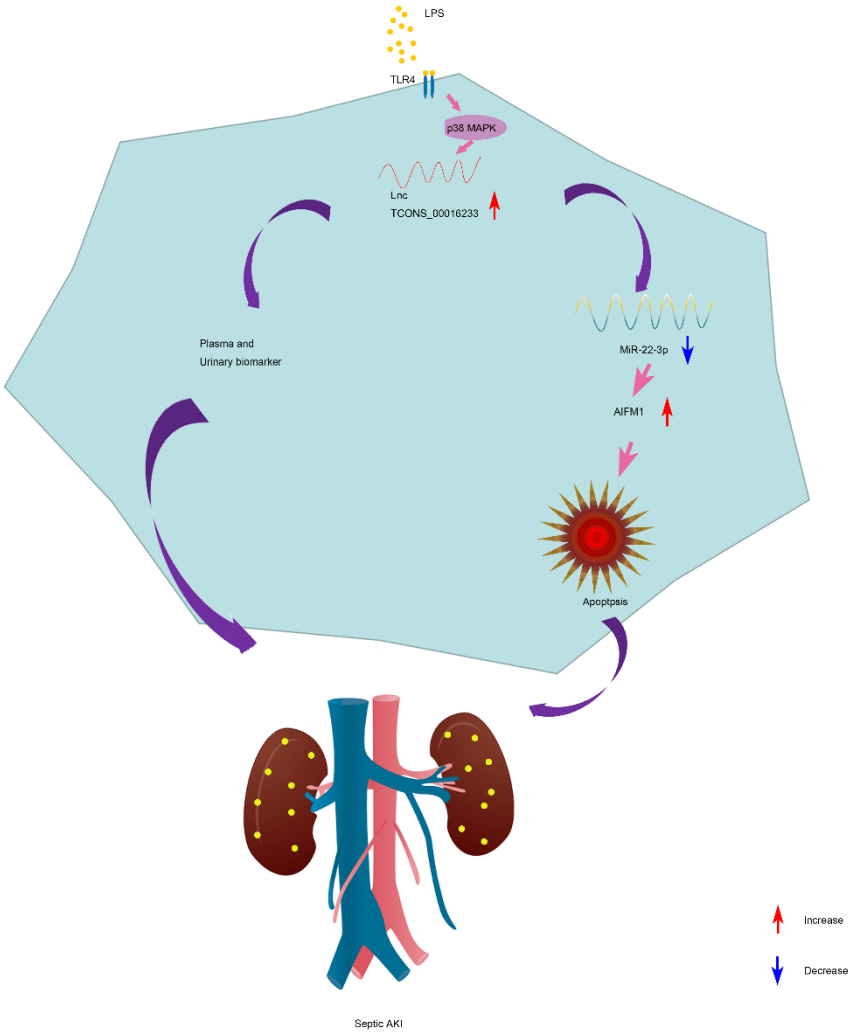
Supplementary figure2. CLP-induced AKI was aggravated by the overexpression of TCONS_00016233 in male C57BL/6 mice.C57BL/6J mice was pretreated with the TCONS_00016233 plasmid via tail vein for 12h, and then subjected to the CLP for 18 hours, saline as control. Blood serum were obtained to detection of nitrogen (BUN) (A) and creatinine (B) concentration. (C&D) The sections of kidney were stained with Hematoxylin and eosin (HE) and TUNEL(E). Tubular damage scores of kidney cortex(F)and OSOM(G). (H) Conntion of TUNEL positive cells. Data are expressed as mean ± SD (n=6). # *P<0.05*Control with CLP group vs. Sham group, * *P<0.05* TCONS_00016233 plasmid with CLP group vs. Control with CLP group. Original magnification, x200.



Supplementary figure3. Overexpression of TCONS_00016233 ameliorated CLP-induced mice AKI via targeting miR-22-3p/AIFM1 axis. C57BL/6J mice was pretreated with the TCONS_00016233plasmid via tail vein for 12h, and then subjected to the CLP for 18 hours, saline as control. (A&B) RT-qPCR analysis of TCONS_00016233 and miR-22-3p.(C) Immunoblot analysis of cleaved caspase3, caspase3, and AIFM1. (D) Densitometric analysis of Immunoblot bands. (E) Immunoblot analysis of TNFα, IL-1β, and GAPDH. (F) Densitometric analysis of Immunoblot bands. (G&H) RT-qPCR analysis of the expression levels of IL-1β and TNFα. Data are expressed as mean ± SD (n=6). # *P<0.05*Control with CLP group vs. Sham group, * *P<0.05* TCONS_00016233 plasmid with CLP group vs.Control with CLP group.



Supplementary figure4. Overexpression of TCONS_00016233 mainly expressed in the tubular cells of kidney in AKI model induced by the LPS and CLP treatment. C57BL/6J mice was pretreated with the TCONS_00016233plasmid via tail vein for 12h, and then subjected to the LPS or CLP for 24 or 18 hours, respectively, saline as control. (A&B) The FISH detection of TCONS_00016233.



Supplementary figure5. The role and molecular mechanism of TCONS_00016233 in Septic-induced AKI. The expression of lncRNA TCONS_00016233 was considered an early diagnosis marker of septic AKI, and induced by LPS treatment via TLR4/p38MAPK signal pathway. Mechanistically, TCONS_00016233 sponged miR-22-3p to upregulate AIFM1 expression and consequently increased the renal cell apoptosis during septic AKI.