Figure S1. Renal pathological and blood biochemical indicators in mice with sepsis-induced AKI. (A) Representative hematoxylin and eosin staining of morphological changes in kidney tissue from control and sepsis-induced AKI mice. Scale bar represents 50 μm. Automated biochemical analysis of serum (B) BUN and (C) CREA from control and sepsis-induced AKI mice. Automated biochemical analysis of the levels of (D) LDH, (E) ALT and (F) AST from the control and sepsis-induced AKI mice. **P<0.01. BUN, urea nitrogen; CREA, creatinine; AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; AKI, acute kidney injury.

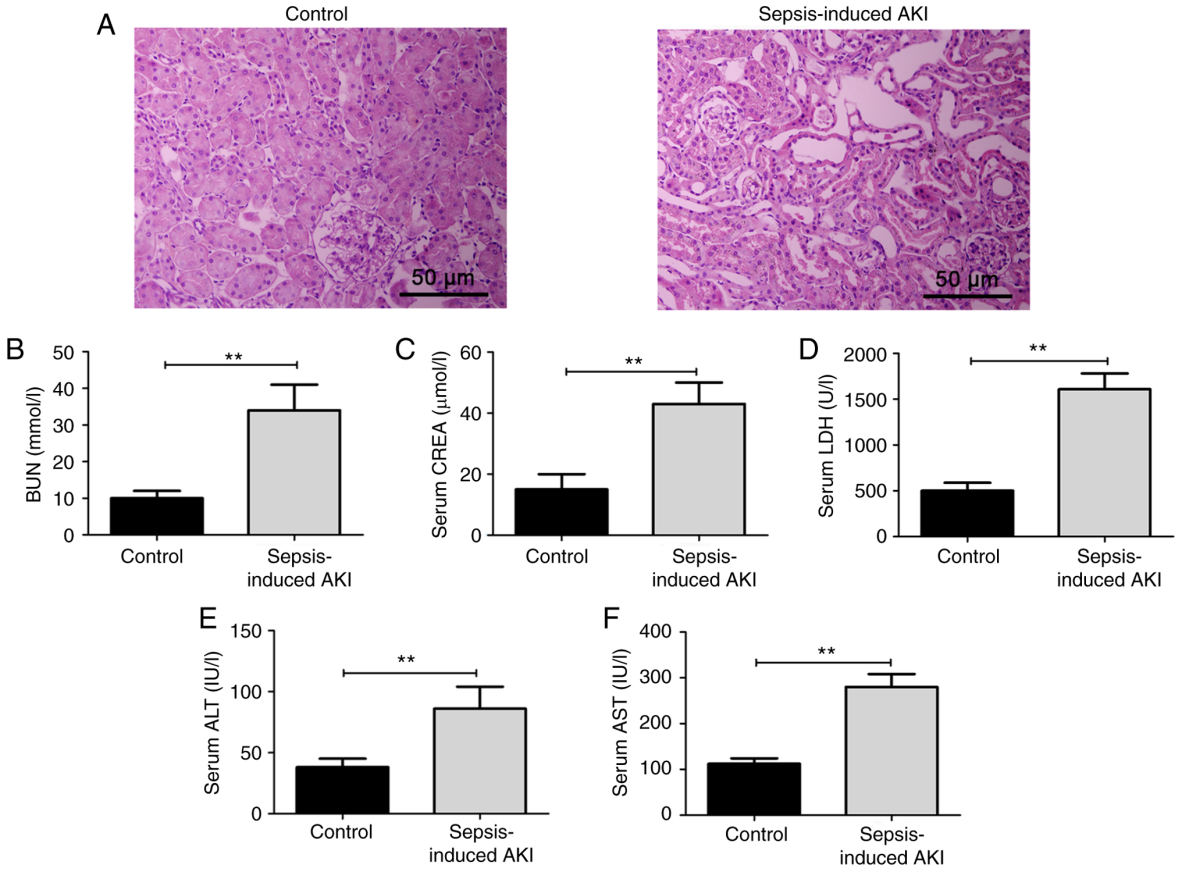

Figure S2. Suppression of miR-155 does not affect the activity of NF-κB in LPS-stimulated HK-2 cells. (A) Western blot analysis of the expression of p-IκBα, cytosolic p50 (c-p50) and p65 (c-p65) in the HK-2 cells from the control, LPS, LPS+miR-155 inhibitor-NC and LPS+miR-155 inhibitor groups. (B) Western blot analysis of the expression of nuclear p50 (n-p50) and p65 (n-p65) in the HK-2 cells from control, LPS, LPS+miR-155 inhibitor-NC and LPS+miR-155 inhibitor groups. (C) EMSA analysis of the activity of the NF-κB pathway in HK-2 cells from control, LPS, LPS+miR-155 inhibitor-NC and LPS+miR-155 inhibitor groups. **P<0.01. LPS, lipopolysaccharide; NF, nuclear factor; NC, negative control; miR, microRNA.