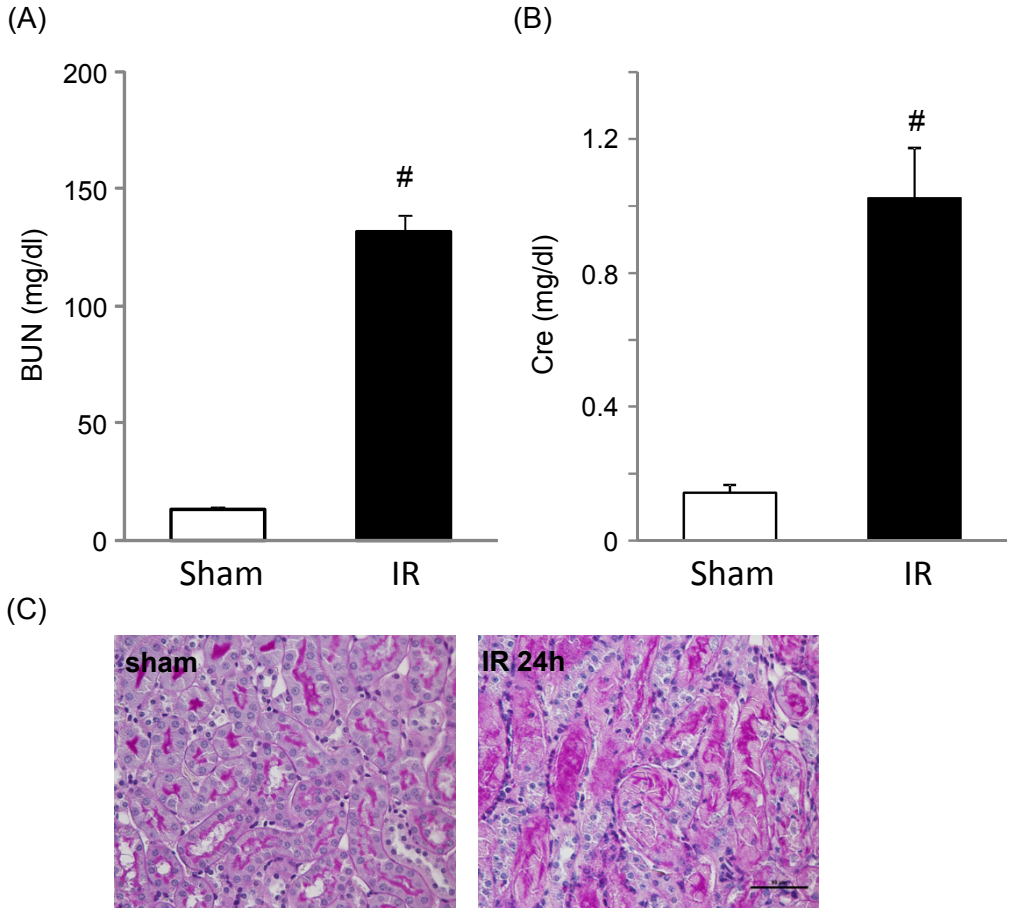
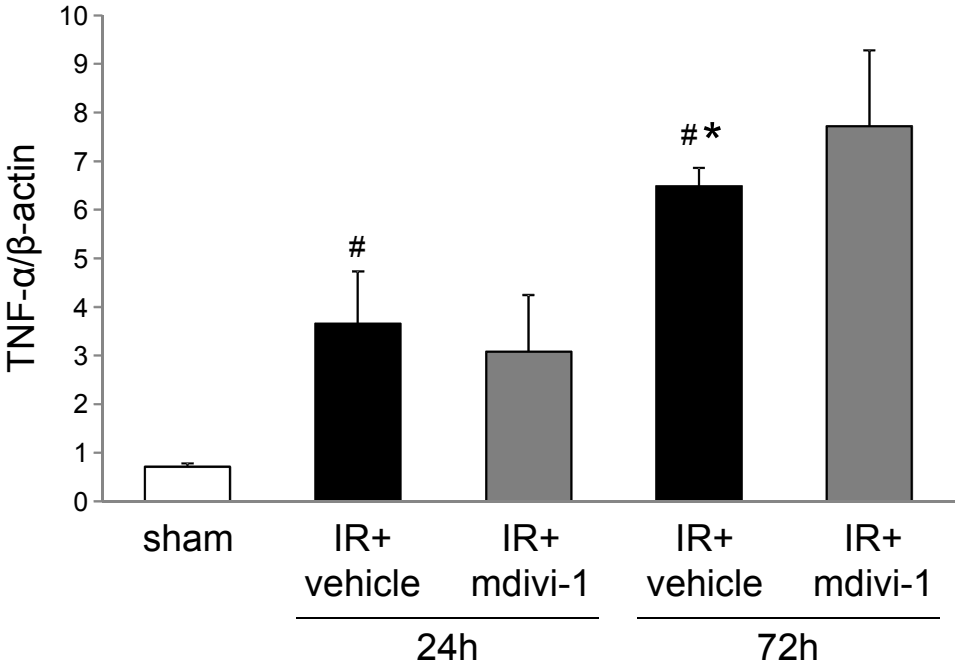
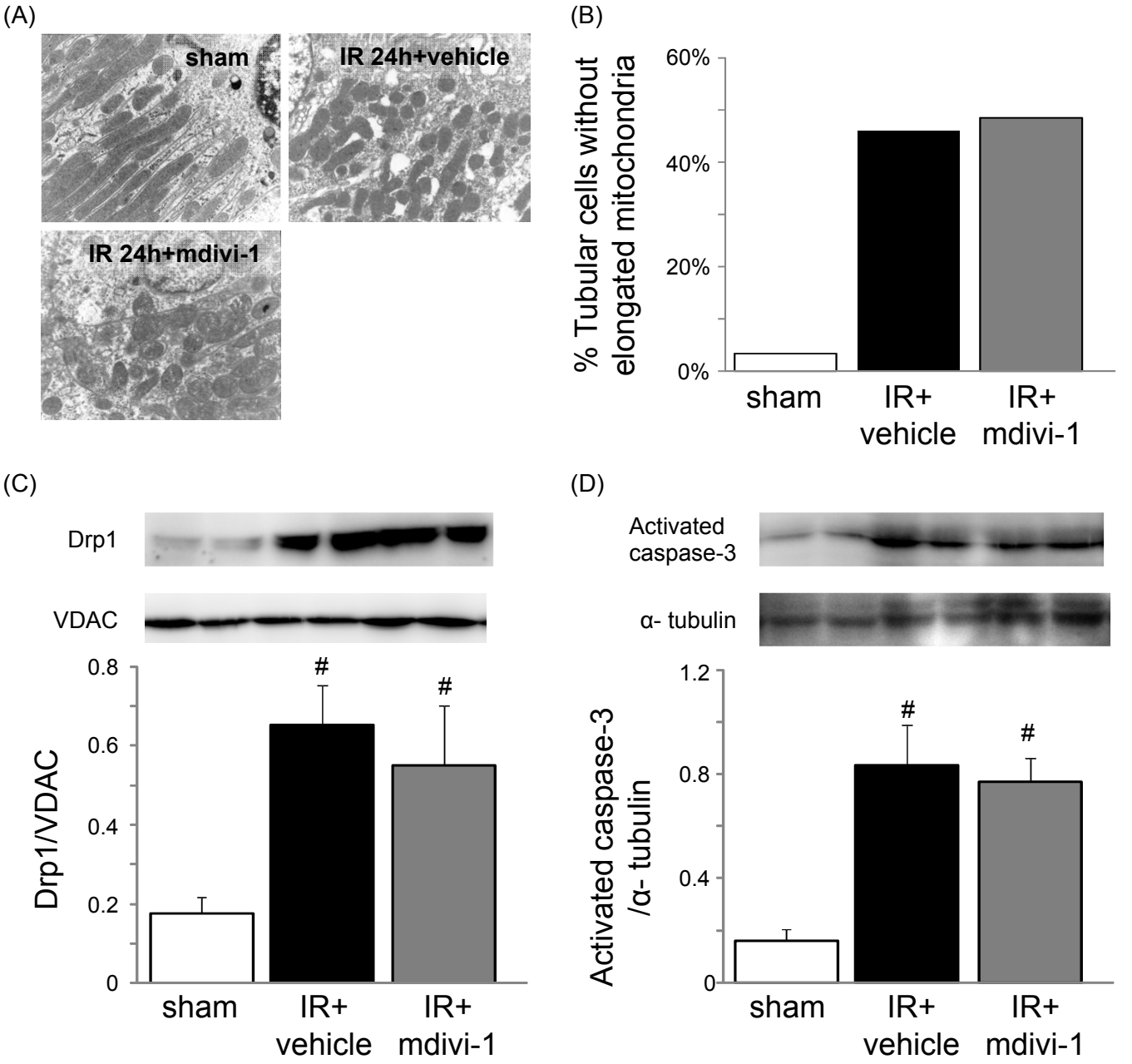


Supplemental Figure 1. Renal dysfunction and pathological changes induced by renal ischemia reperfusion injury.
BUN (A) and plasma creatinine (Cre) (B) at 24 h after renal ischemia reperfusion (IR) injury was significantly increased compared with the sham-operated mice (n=7 in each group). # $p < .05$ versus sham.
(C) Renal tubular necrosis caused by IR is shown in Periodic acid–Schiff (PAS) staining. Original magnification: ×400. Bar = 50 μm.



Supplemental Figure 2. TNF-α expression in the heart induced by renal ischemia reperfusion injury. Renal ischemia reperfusion increased cardiac TNF-α expression but mdivi-1 did not suppress this response (n=6 per group). # $p < .05$ versus sham. * $p < .05$ versus IR+vehicle 24 h.



Supplemental Figure 3. Effect of Drp1 inhibitor mdivi-1 on ischemic renal mitochondria and renal cell apoptosis.
Mitochondrial fragmentation in the kidney was observed 24 hours after renal ischemia reperfusion (A and B). Increased Drp1 expression in the mitochondrial fraction (C) and activated caspase-3 expression (D) were also observed. Treatment of mdivi-1 did not attenuated these renal injury. # $p < .05$ versus sham.