Supplemental Figures and Legends:

Supplemental Figure 1. Dicer-deficiency reduces inflammation and fibrosis in cold-storage transplantation kidneys. The left kidney was collected from Dicer-knockout (KO) or wild-type (WT) mice for 10 hours of cold storage, followed by transplantation into wild-type mice for 7 or 14 days (CST7d, CST14d). The right kidney of donor mice without cold storage transplantation was used as sham control (Sham). **(A)** Representative images of macrophage immunohistochemistry. Scale bar=100μm. **(B)** Representative images of C3 staining. Scale bar=100μm. **(C)** Representative images of α-SMA staining. Scale bar=100μm. **(D)** Representative images of Sirius Red staining. Scale bar=100μm. **(E)** Quantitative analysis of macrophage positive area. **(F)** Quantitative analysis of C3 positive signal. **(G)** Quantitative analysis of α-SMA positive area. **(H)** Quantitative analysis of Sirius Red positive area. Data are expressed as mean±SD (n=4-5). *, $P<0.05$ versus sham control; #, $P<0.05$ versus WT transplant.

Supplemental Figure 2. Taqman real-time PCR analysis of microRNA expression in cold-storage transplantation kidneys

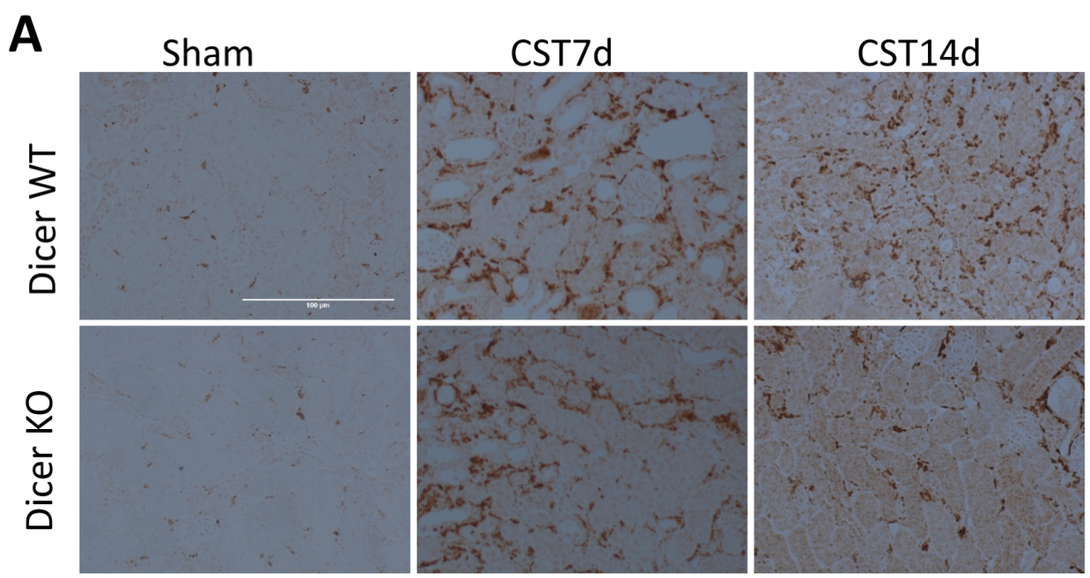
Transplanted kidneys were collected at 1, 7 and 14 days after cold-storage transplantation to isolate RNAs for Taqman real-time PCR analysis of miR-132, miR-874 and miR-185. The right kidney of the donor mice that did not experience cold-storage transplantation was used as sham control for normalization. Data are expressed as mean ± SD (n=5); *, $P<0.05$ versus respective sham control group.

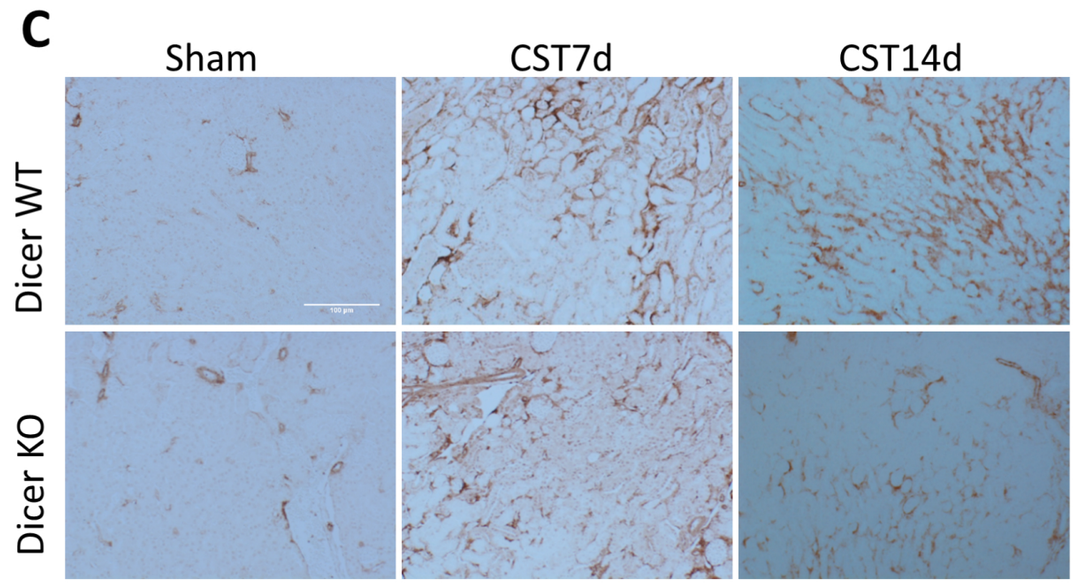
Supplemental Figure 3. Overexpression of NDUFA4 suppresses miR-147-induced mitochondrial dysfunction and cell death.

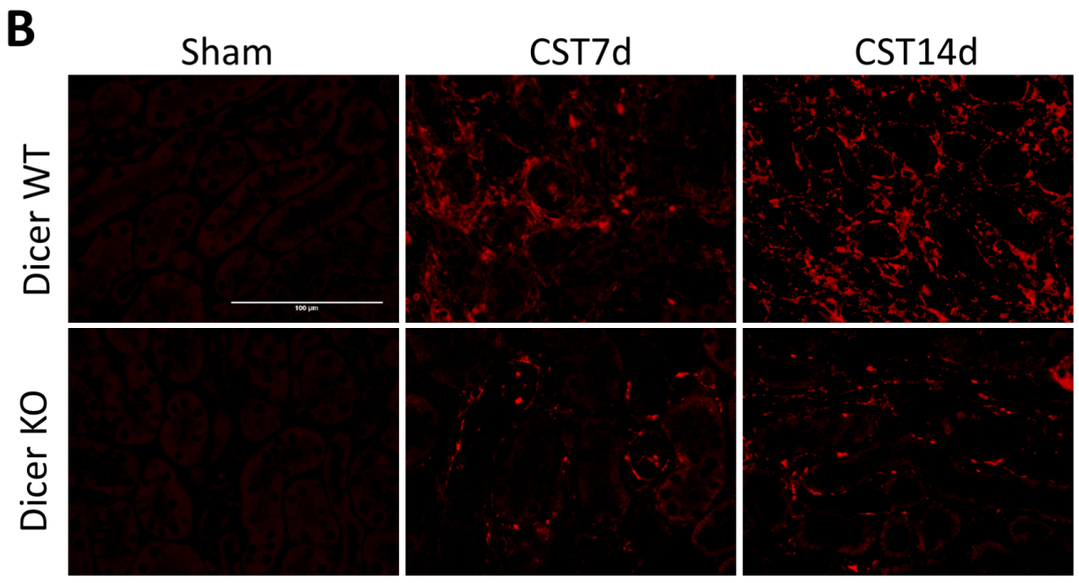
RPTCs with NDUFA4-pcDNA3.1 or empty pcDNA3.1 were transfected with miR-147 mimic or

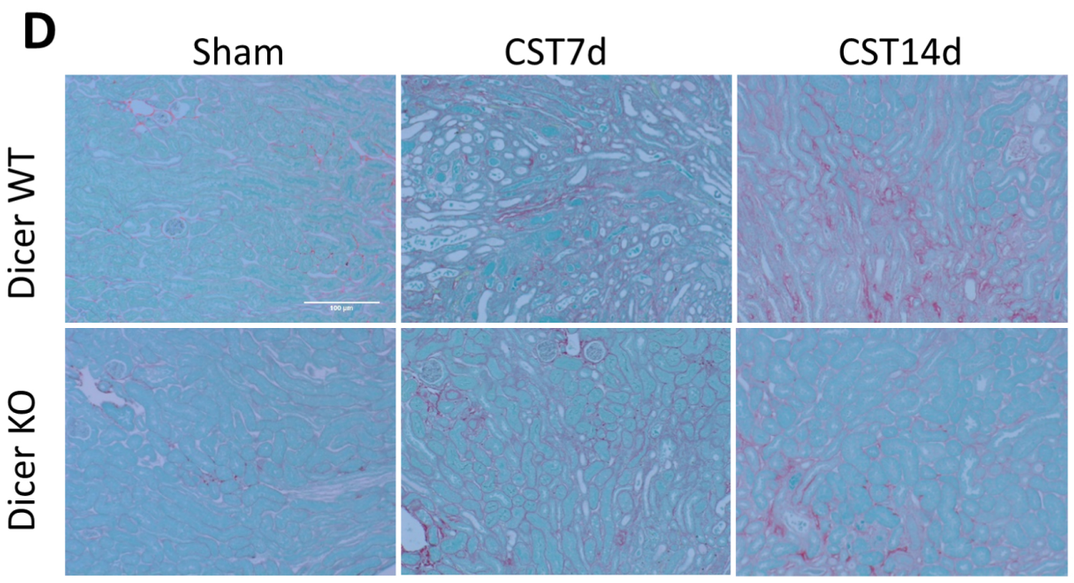
negative control (NC) oligo. **(A)** Representative cell morphology. Scale bar=100 μm. **(B)** The percentage of apoptosis evaluated morphologically. **(C)** Measurement of oxygen consumption rate (OCR) using an XF24 Extracellular Flux Analyzer **(D)** Flow cytometry analysis of mitochondrial membrane potential after JC-1 staining. **(E)** Cellular ATP. Quantitative data are expressed as mean±SD (n=4-10); *, $P<0.05$ versus respective NC group; #, $P<0.05$ versus miR-147 mimic group with pcDNA 3.1.

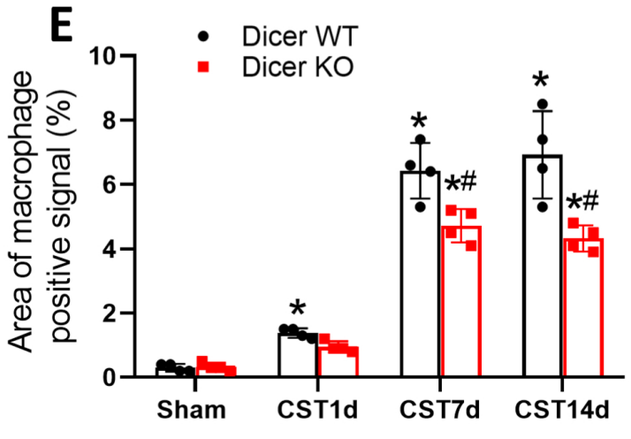
Supplemental figure 1. Dicer-deficiency reduces inflammation and fibrosis in cold-storage transplantation kidneys

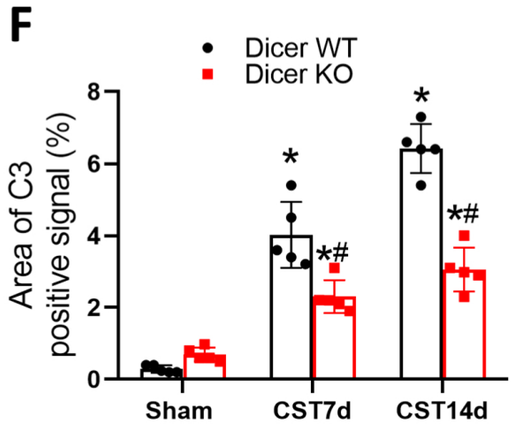


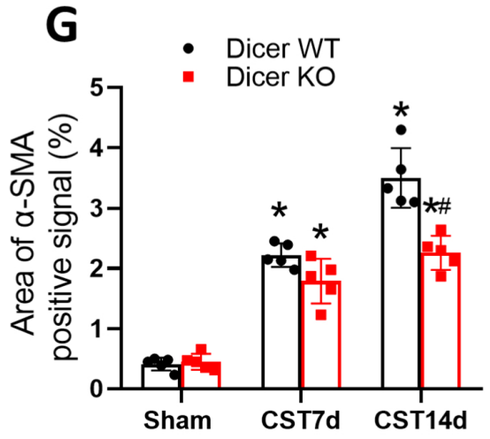


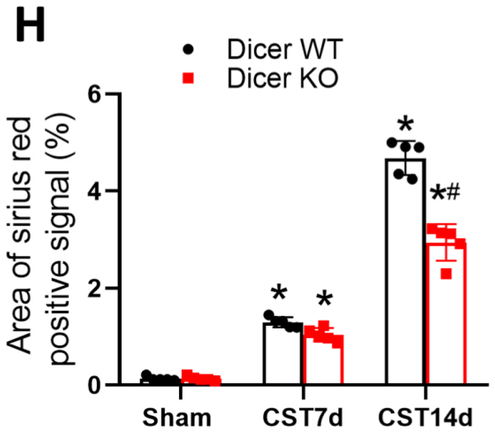




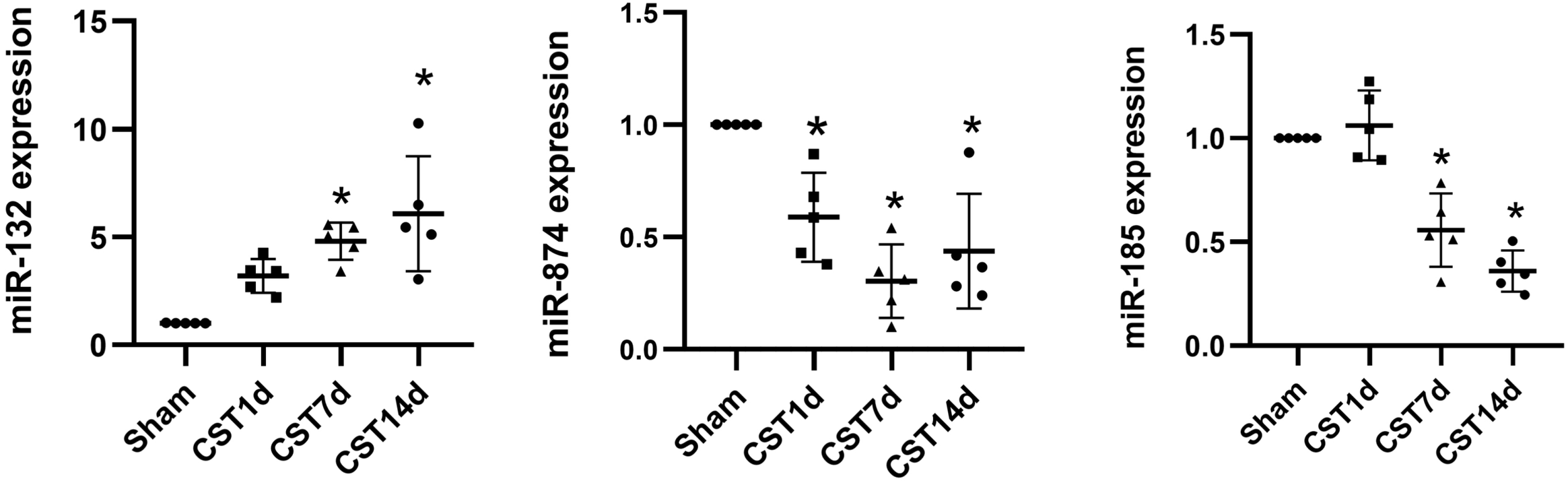




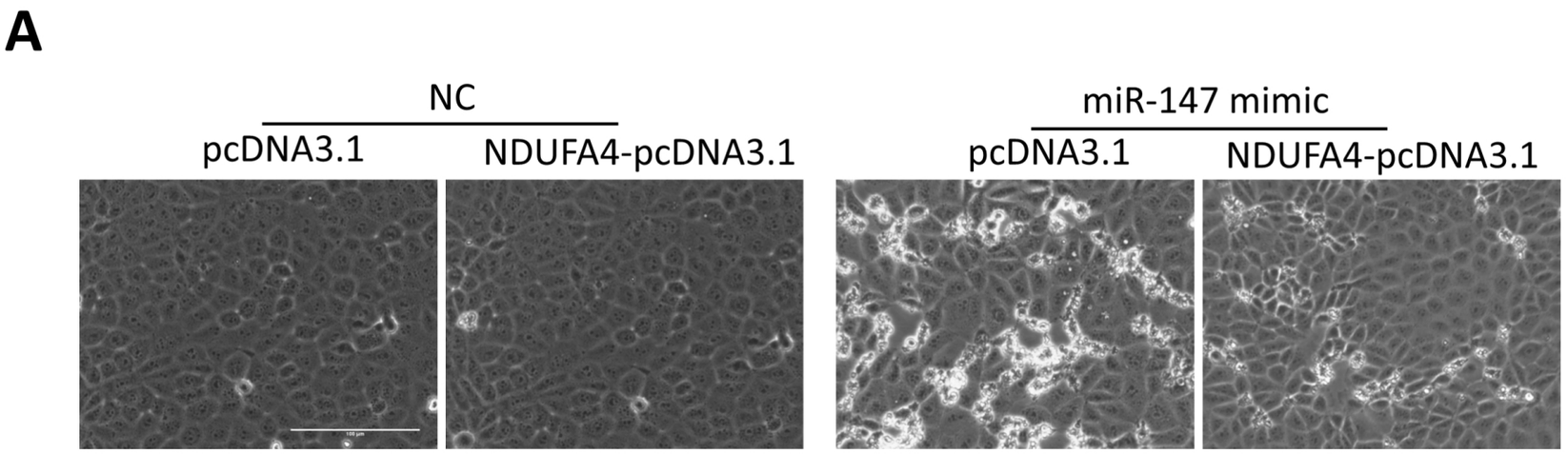


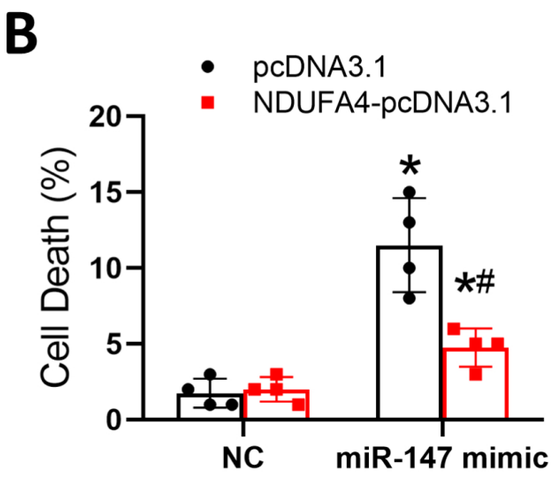


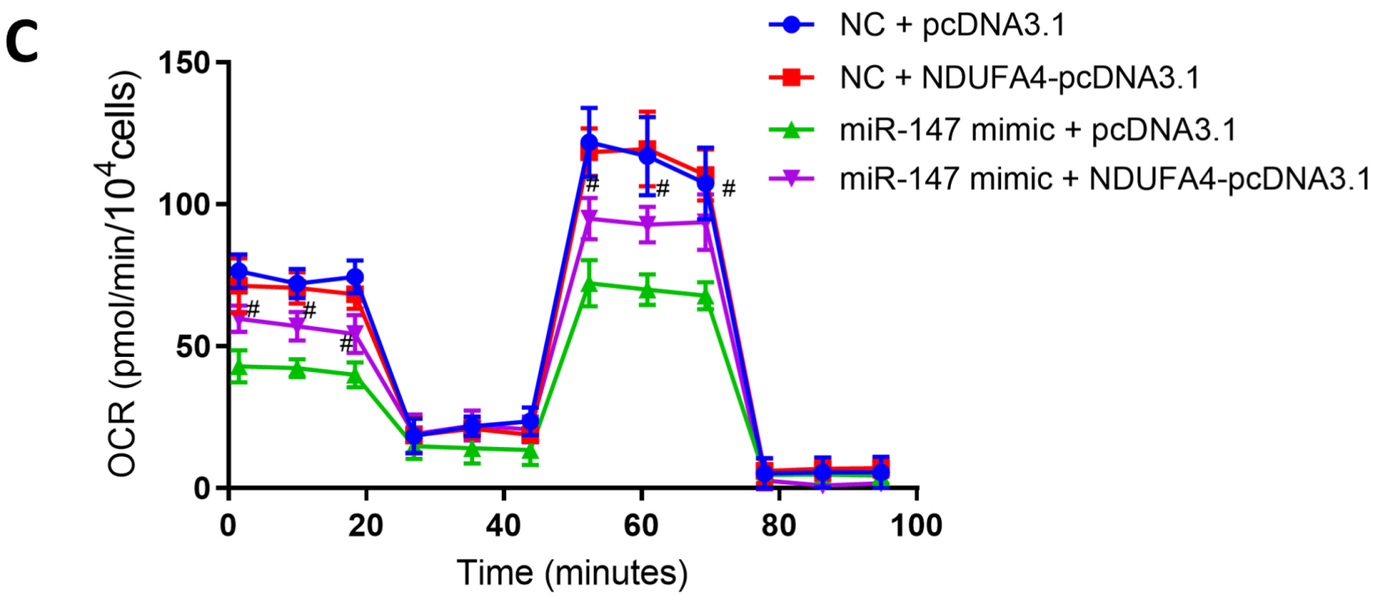
Supplemental Figure 2. Taqman real-time PCR analysis of microRNA expression in cold-storage transplantation kidneys

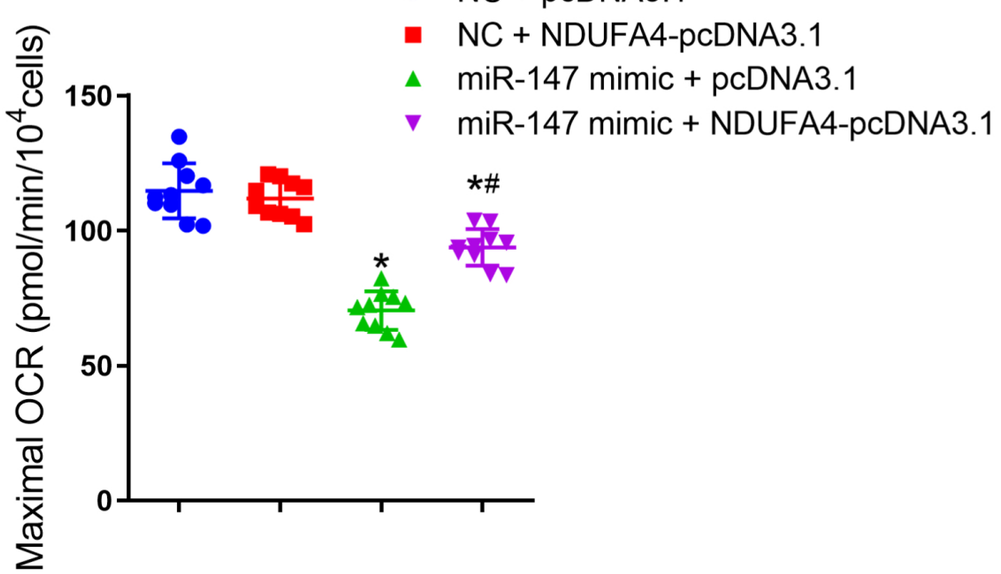


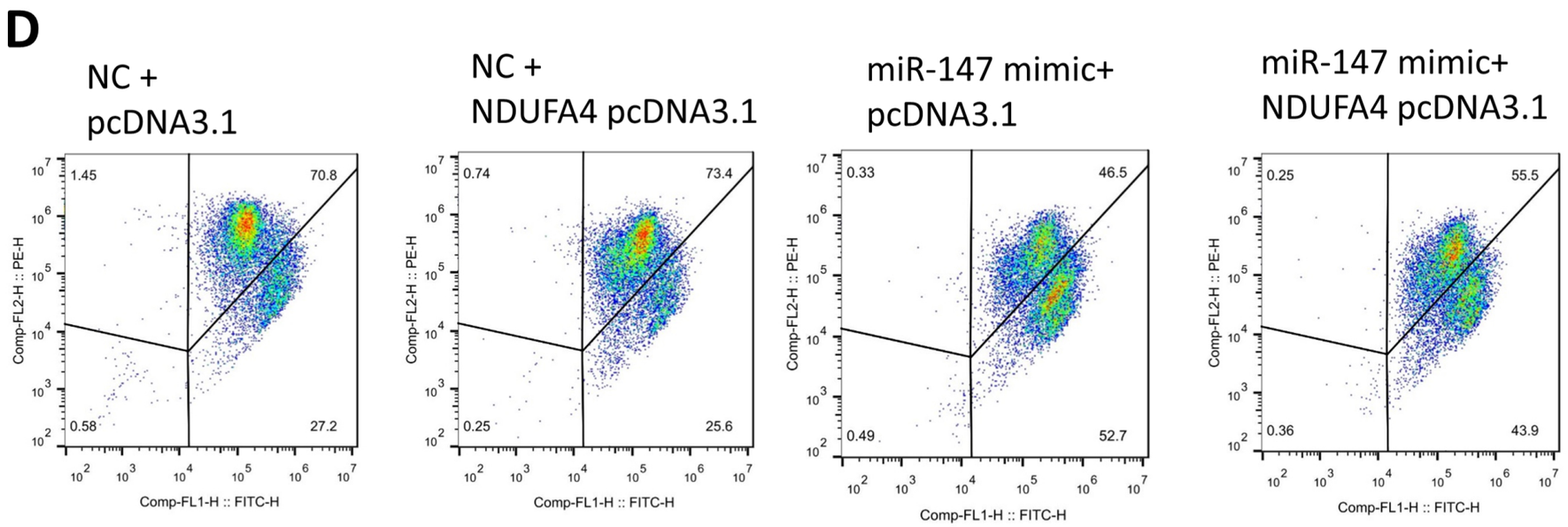
Supplemental Figure 3. Overexpression of NDUFA4 suppresses miR-147-induced mitochondrial dysfunction and cell death.











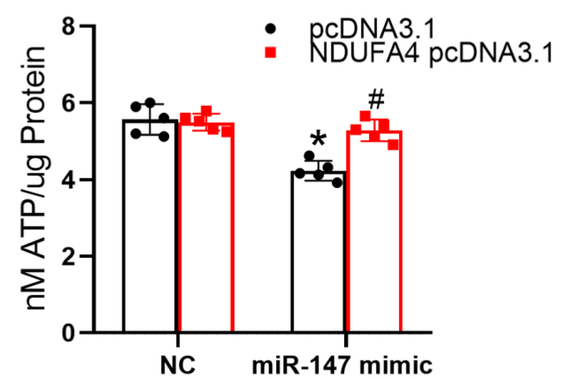


TABLE 1. CLINICAL DATA OF DGF AND NON-DGF PATIENTS EXAMINED

PARAMETER	DGF (n=5)	Control (n=5)	P Value
Donor information			
Donor age, (year)	48.8 ± 7.28	/	
Donor Sex, F/M	M (4) F (1)	/	
Donor source, living/deceased	Deceased	/	
Donor last creatinine, mg/dL	1.0 ± 0.14	/	
Donor last BUN mg/dl	19.40 ± 1.38		
Clinical indexes	M (4) F (1)	M (4) F (1)	
Sex	39.6 ±9.44	50.6±10.48	0.1251
Age (year)	80%	20%	
Hypertension	All negative	All negative	
Diabetes	Chronic glomerulonephritis	/	
Primary cause for ESRD	DGF(5)	para-cancer renal tissue	
Diagnosis	10 (6; 14)	during surgery	
Time of Biopsy, days after transplantation			
Immunosuppression			
Calcineurin inhibitors	100%	0%	
Mycophenolic acid	100%	0%	
mTOR inhibitors	20%	0%	
Steroids	100%	0%	
Laboratory data			
BUN (mg/dl)*	55.44 ± 20.37	13.93 ± 2.52	0.0001*
Creatinine (mg/dl)*	4.57 ± 0.98	0.76 ± 0.09	0.0006*

(2-tailed student's t-test for comparison). *, statistically significant difference between DGF and non-DGF group.