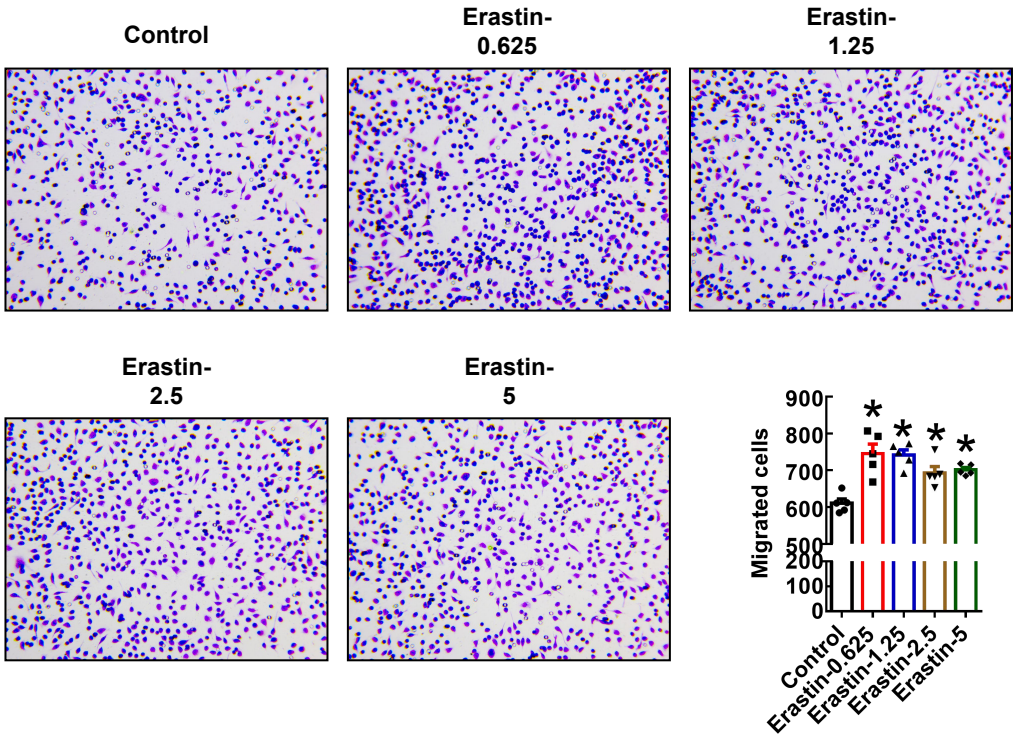
Supplementary Table 1

	AAA	Control	Student's *t* test Or Fisher's exact test *p*-Value
N	7	8	
Demographics			
Age (years)	68.14±5.11	55.75±4.16	0.0799
Male	7(100%)	4(50.0%)	0.0769
Health Status			
Smoking	2(28.6%)	1(12.5%)	0.5692
Coronary Artery Disease	3(42.9%)	0(0%)	0.0769
Congestive Heart Failure	0(0%)	0(0%)	1.0000
Atrial Fibrillation	1(14.3%)	0(0%)	0.4667
Stroke	3(42.9%)	0(0%)	0.0769
Cancer	0(0%)	0(0%)	1.0000
Diabetes Mellitus	1(14.3%)	0(0%)	0.4667
Hypercholesteremia	0(0%)	0(0%)	1.0000
Hypertension	4(57.1%)	1(12.5%)	0.1189
Aneurysm Maximal Transverse Diameter (cm)	4.929±0.822	---	

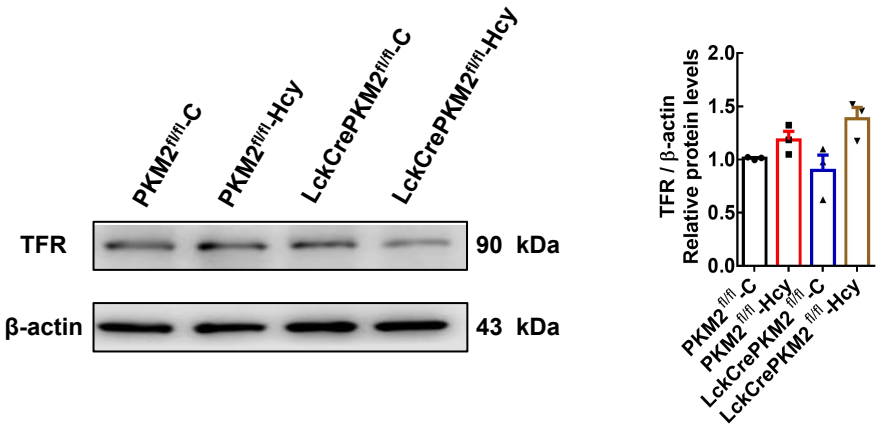
Supplementary Table 2

	Sham	AAA+Vehicle	AAA+GW4869
n	8	8	7
Weight（g）	28.21±0.95	26.76±0.84	27.85±0.93
EVs（μg / μL）	0.1391±0.0019	0.1802±0.0126*	0.1074±0.0051#

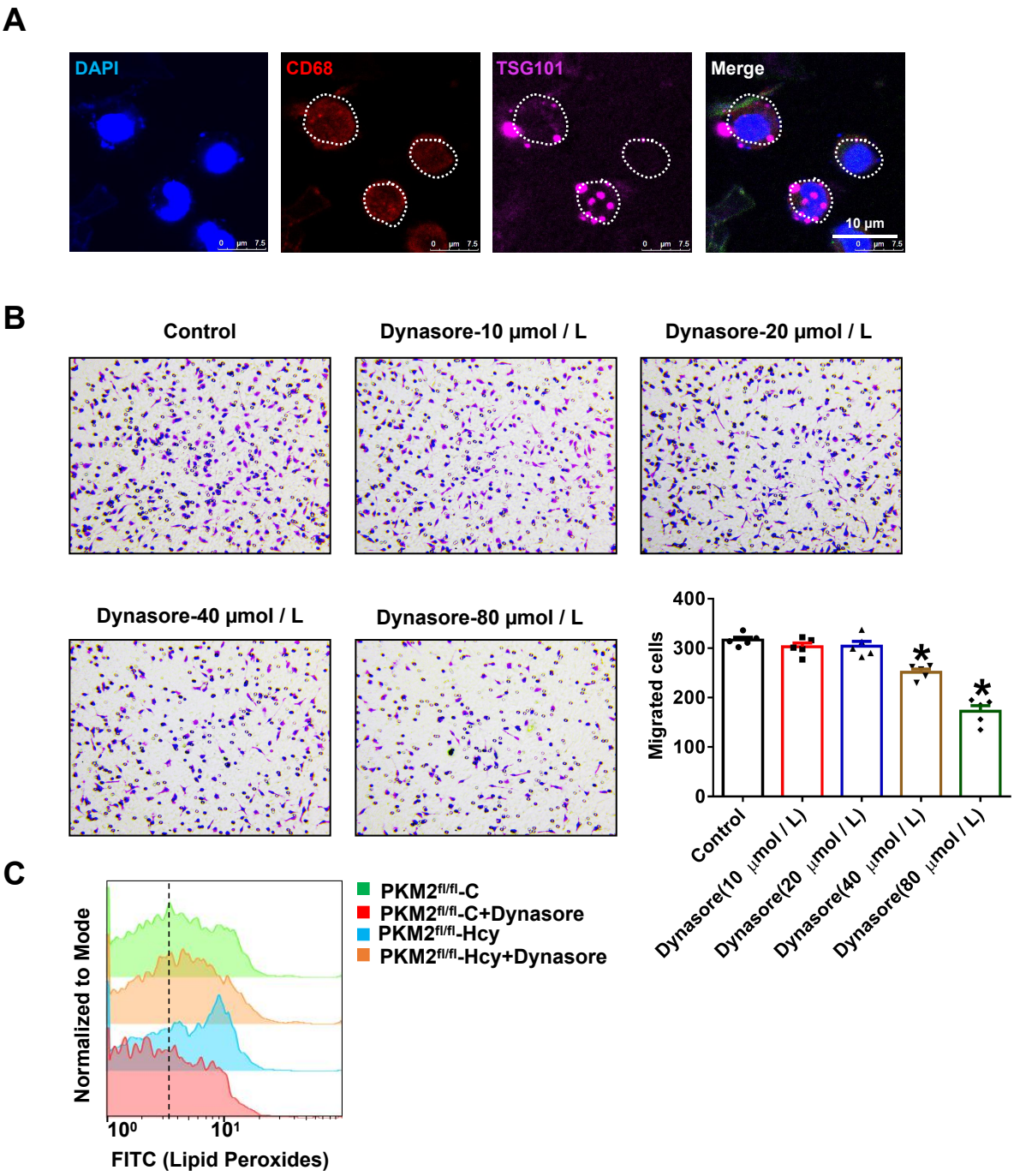
Supplementary Figure 1



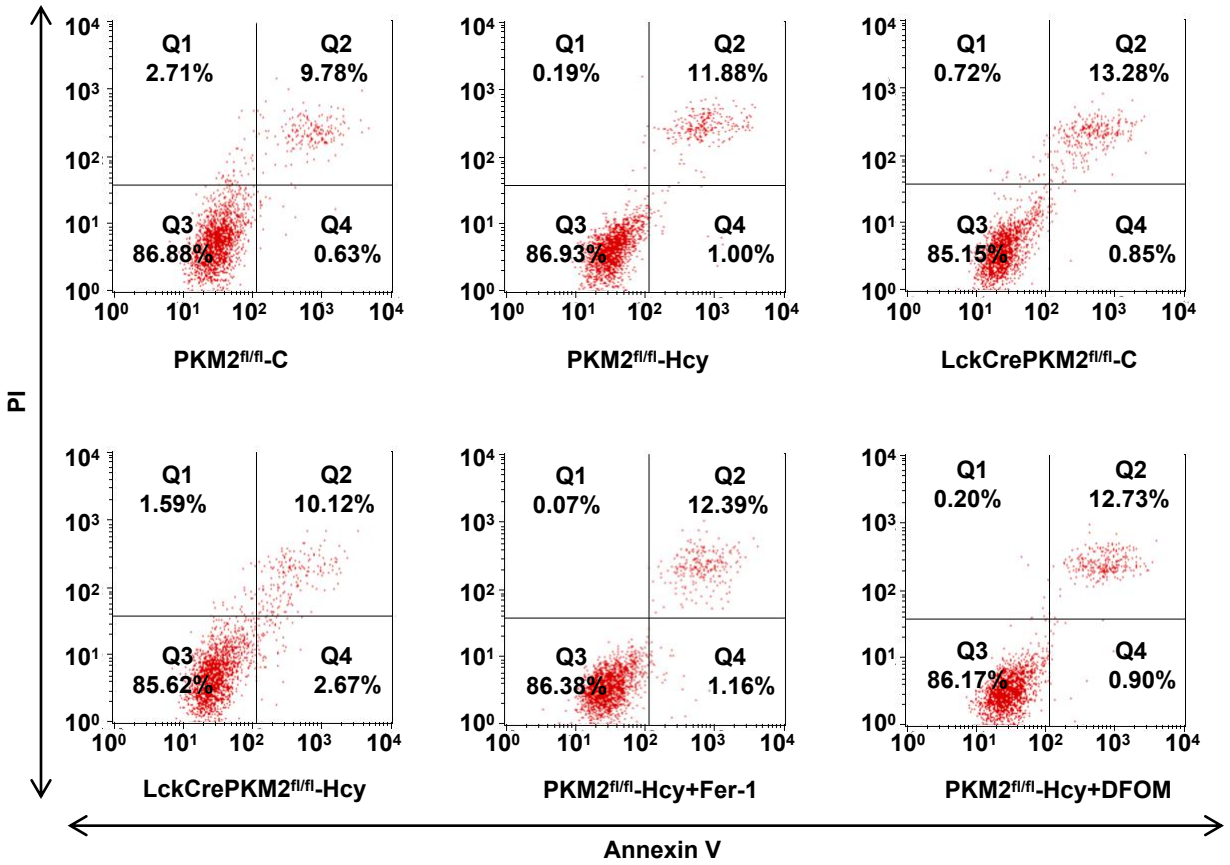
Supplementary Figure 2

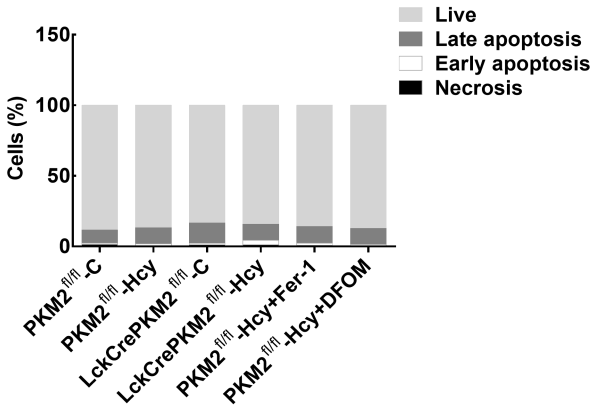


Supplementary Figure 3



Supplementary Figure 4





Supplementary Data

Supplementary Table 1. Baseline characteristics of AAA patients and control subjects.

Baseline characteristics of AAA patients and control subjects. The data were compared using Student's *t* test or Fisher's exact test.

Supplementary Table 2. EVs were inhibited by GW4869 treatment.

Infrarenal abdominal aortas of 10-week-old C57BL/6J mice were treated with elastase for 2 weeks to induce AAA. Mice were injected intraperitoneally once daily with GW4869 (1.25 mg/kg, dissolved in saline with 2.5% dimethylsulfoxide) (AAA+GW4869) or saline with 2.5% dimethylsulfoxide (AAA+vehicle) 7 days prior to elastase-induced AAA for a total of 21 injections. The numbers of experimental mice, weights and plasma EV concentrations in the three groups are shown in this table. The data are presented as the mean±SEM. *$p<0.05$, compared with the sham group. #$p<0.05$, compared with the AAA+vehicle group. The data were compared using one-way ANOVA followed by Tukey's multiple comparison test.

Supplementary Figure 1. Erastin promotes macrophage migration.

Transwell migration assays of peritoneal macrophages in the upper chamber with MCP-1 (20 ng/mL) added to the lower chamber with or without erastin (0.625-5 μmol/L). Representative images of crystal violet staining were captured at 48 hours after incubation to indicate migrated cells and quantification of migrated cells. Cells were counted from 5 random microscope fields for each sample in 5 independent experiments. The data are presented as the mean±SEM. *$p<0.05$, compared with the control. The data were compared using one-way ANOVA followed by Tukey's multiple comparison test.

Supplementary Figure 2. The protein expression level of TFR.

Protein expression (relative to β-actin) of transferrin receptor (TFR) in RAW264.7 cells was measured via Western blot after treatment with different EVs for 48 hours.

Supplementary Figure 3. Dynasore blocks EV internalization.

(A) Alexa-647-TSG101-labeled exosomes were added to macrophage medium and

incubated for 12 hours. Representative immunofluorescence staining of CD68 (red), DAPI (blue), and Alexa-647 (purple) in macrophages is shown here. Detectable purple staining was observed in the cytoplasm of macrophages. The white dotted line indicates the macrophage border. (B) Transwell migration assays of peritoneal macrophages in the upper chamber moving toward MCP-1 (20 ng/mL) in the lower chamber with or without Dynasore (10, 20, 40, and 80 μmol/L). Representative images of crystal violet staining were captured at 48 hours after incubation to indicate migrated cells and the quantification of migrated cells. Cells were counted from 5 random microscopic fields for each sample in 5 independent experiments. (C) Measurement of the lipid peroxidation levels in RAW264.7 cells by detecting oxidized BODIPY-C11 (emission: 590 nm) through flow cytometry of BODIPY 581/591 C11. The data are presented as the mean±SEM. *p<0.05, compared with the control. The data were compared using one-way ANOVA followed by Tukey's multiple comparison test.

Supplementary Figure 4. Qualification of cell death.

RAW264.7 cells were pretreated with or without the lipid peroxidation inhibitor Fer-1 (5 μmol/L) or the iron chelating agent DFOM (10 μmol/L) and then cultured with equal concentrations (10 μg/mL) of EVs from the PKM2$^{fl/fl}$-C, PKM2$^{fl/fl}$-Hcy, LckCrePKM2$^{fl/fl}$-C or LckCrePKM2$^{fl/fl}$-Hcy T lymphocytes. After 24 hours, RAW264.7 cells were double stained with Annexin V-FITC/PI and analyzed by flow cytometry to quantify cell death. Representative density maps of Annexin V-FITC/PI staining for apoptosis are shown in the upper panels. A dot plot of Annexin V-FITC vs. PI showed four separate clusters: viable cells (lower left quadrant Q3), early apoptotic cells (lower right quadrant Q4), late apoptotic cells (upper right quadrant Q2), and necrotic cells (upper left quadrant Q1). The lower panel shows the rates of apoptosis and necrosis obtained by double staining with Annexin V-FITC/PI (four duplicates for each experiment).

Supplementary movies 1-4. Single-cell migration movies.

T lymphocytes from the PKM2$^{fl/fl}$ or LckCrePKM2$^{fl/fl}$ mice were isolated and then treated with or without Hcy (100 mmol/L) *in vitro*. After 48 hours, EVs were isolated from cell culture supernatants. Equal concentrations (10 μg/mL) of EVs from the PKM2$^{fl/fl}$-C, PKM2$^{fl/fl}$-Hcy, LckCrePKM2$^{fl/fl}$-C or LckCrePKM2$^{fl/fl}$-Hcy T lymphocytes cultured with RAW264.7 cells for an

additional 24 hours. Migration of RAW264.7 cells by MCP-1 was observed by the cell dynamic visualization system TAXIScan-FL. The cells at the start line on the edge of the channel were aligned, MCP-1 (2 ng/mL) was added as a chemokine on the other edge of the channel, and then, time-lapse image recording was performed (taking an image every 30 seconds for 3 hours). The movies recorded single cell migration for 3 hours. Supplementary Movies 1-4: RAW264.7 cells stimulated by EVs from the $PKM2^{fl/fl}$-C, $PKM2^{fl/fl}$-Hcy, $LckCrePKM2^{fl/fl}$-C or $LckCrePKM2^{fl/fl}$-Hcy T lymphocytes, respectively.