

Fig. S1 Activated AXL attenuates hepatocytes apoptosis during OGD/R *in vitro*. (A-B) WB analysis of the relative protein expression of p-AXL and cleaved-Caspase-3 in primary mouse hepatocytes of control group and treatment group (as shown in the figure) after OGD/R and densitometric quantification. (C-D) Flow cytometry assay showing the apoptosis level of primary mouse hepatocytes and corresponding statistical chart. (E) TUNEL staining for the level of apoptosis in primary mouse hepatocytes. Scale bar = 100 μ m. Data are shown in mean \pm SD. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$; p-AXL, phosphorylated AXL; rmGas6, recombinant growth arrest-specific protein 6; OGD/R, oxygen-glucose deprivation/reoxygenation; TUNEL, Terminal deoxynucleotidyl transferase-mediated nick end labeling.

Fig. S2 (A) Establishment of ALD model and the liver I/R model was conducted. **(B)** Pictures of liver tissues in Pair and ALD mice. **(C)** WB analysis of the relative protein expression of p-AXL in primary hepatocytes with or without EtOH treatment. rmGas6, recombinant growth arrest-specific protein 6; ALD, alcoholic liver disease; p-AXL, phosphorylated AXL; EtOH, ethyl alcohol.

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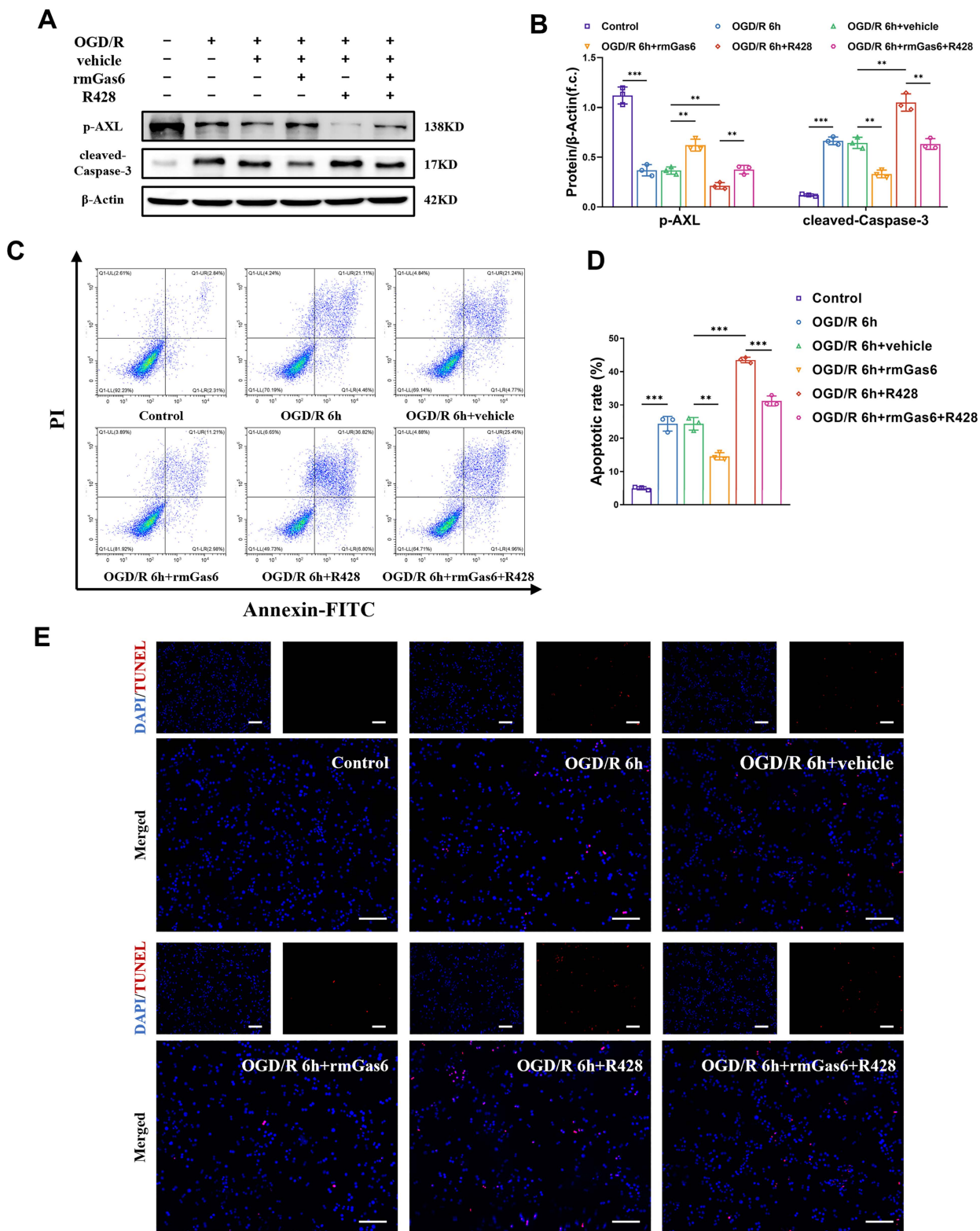
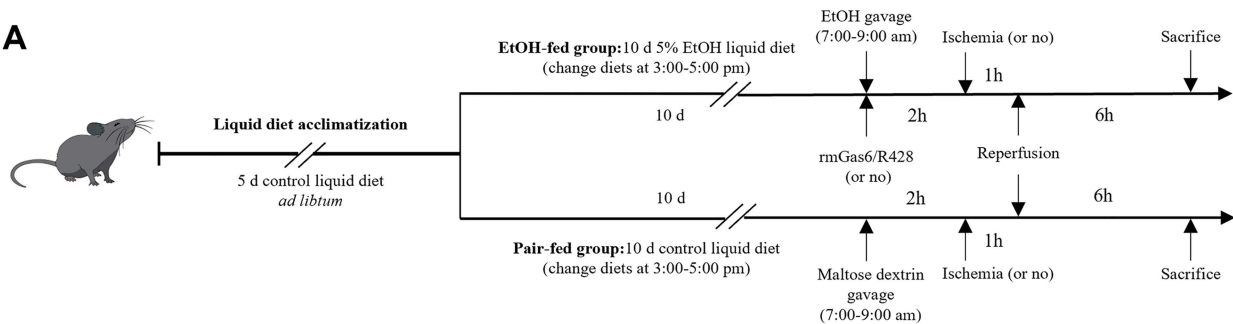
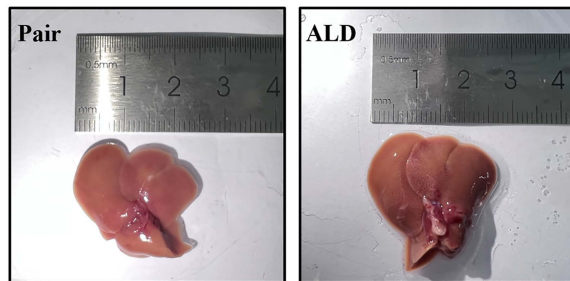


Fig. S2

A



B



C

