

Supplementary Methods

Materials. Chenodeoxycholic acid (CDCA, Cat# C937) and taurocholic acid (TCA, Cat# T4009) were purchased from Sigma-Aldrich (St. Louis, MO, U.S.A.). GW4064 (HY-50108) and tropifexor (HY-107418) were obtained from MCE (Shanghai, China). Recombinant human tissue factor (rh-TF) was purchased from Bersee (Beijing, China). Recombinant human tissue plasminogen activator (rtPA) was obtained from Boehringer Ingelheim (Ingelheim am Rhein, Germany). Phospholipid vesicles (20 mol% phosphatidylserine, 60 mol% phosphatidylcholine, and 20 mol% phosphatidylethanolamine) were obtained from SunLipo NanoTech (Shanghai, China). Boc-Glu-Lys-Lys-AMC substrate (HY-P4319) and human plasma plasmin (HY-108883) were obtained from MedChemExpress (New Jersey, USA). α 2-macroglobulin (SRP6314) was obtained from Sigma (Missouri, USA). The antibody against GAPDH and FXR were obtained from Proteintech Group (Wuhan, China). Antibody against PAI-1 was supplied by Abcam (Cambridge, MA, U.S.A.). Enzyme linked immunosorbent assay (ELISAs) to measure human PAI-1 (E-EL-H2104), human tPA (E-EL-H2106), mouse PAI-1 (E-EL-M3041), and mouse tPA (E-EL-M0917) were obtained from Elabscience (Wuhan, China). ELISAs to measure human α 2-AP (CSB-E09452h), human Plg (CSB-E09177h), mouse α 2-AP (CSB-PA021085GA01HU), mouse Plg (CSB-EL018188MO), and mouse FDP (CSB-E07941m) were obtained from CUSABIO (Wuhan, China). ELISAs to measure culture media (ab197752) were obtained from abcam (Cambridge, UK).

Human sample collection. Plasma samples were collected from patients in our obese cohort at the First Affiliated Hospital of Xi'an Jiaotong University, which was approved by the ethics committee of the hospital. The participants without gastrointestinal diseases, biliary tract diseases, diabetic complications, severe hepatic diseases, or pregnancy were divided into normal weight (NM, $18.5 \leq \text{BMI} < 24 \text{ kg/m}^2$) or obese (OB, $\text{BMI} \geq 28 \text{ kg/m}^2$) groups according to BMI. All individuals signed a written informed consent form before sample collection. Blood obtained by median cubital venipuncture into a 10% volume of sodium citrate (3.8%, w/v) was centrifuged for 20 min at $2000 \times g$, and the plasma was carefully collected from the supernatant fraction. The plasma samples were divided into aliquots, snap-frozen, and stored at -80°C until analysis. The total plasma antigen levels of tPA, PAI-1, α 2-AP, and Plg were measured by ELISA using kits according to the manufacturer's instructions.

Mouse models. All animal protocols were approved by the Institutional Ethics Committee for Animal Experiments of Xi'an Jiaotong University. All the mice were housed in a standard specific-pathogen-free environment. For ascertainment and phenotypic characterization of DVT in diet-induced obese (DIO) mice, 5-week-old male C57BL/6J mice were fed a normal diet (ND) or high fat (Research Diets, catalog 12492) diet (HFD) for 16 weeks. To validate the effects of FXR activation on hepatocyte PAI-1 expression, 12-week-old male C57BL/6J mice fed a ND were orally administered CDCA 50 mg/kg/d or vehicle for 4 days¹; the procedure was replicated with 200

mg/kg/d TCA or vehicle for 4 days^{2,3}. To test the effects of FXR deficiency on plasma fibrinolysis and DVT, *Fxr-null* mice on C57BL/6 background from the Shanghai Model Organisms Center were generated as described previously and compared with littermate controls⁴. To verify the role of FXR activation in alleviating the diet-induced high DVT load, 5-week-old male C57BL/6J mice maintained on a HFD or ND for 12 weeks, designated the DIO model, were generated as described above. Additionally, lean mice were treated with vehicle, and HFD mice were randomly divided into two groups that received vehicle or tropifexor^{5,6} at 0.3 mg/kg/d orally during the last 4 weeks.

Mice with AAV8-TBG-sh*Serpine1*. C57BL/6J mice used for hepatocyte PAI-1 silencing were purchased from Shulaibao company as age-matched lean and DIO mice. DIO mice have been fed a high-fat diet for 14 weeks. Both groups were acquired at 20 weeks of age and were maintained on either standard chow or DIO diet (Research Diets, 12492) upon arrival at the animal facility at Xi'an Jiaotong University. *Fxr-null* mice used for hepatocyte PAI-1 silencing were also purchased from the Shanghai Model Organisms Center. Following a 2 weeks acclimatization period, we injected DIO mice aged 22 weeks or *Fxr-null* mice with an adeno-associated virus (AAV8) expressing short hairpin RNA targeting PAI-1 (AAV8-TBG-sh*Serpine1*, sequences in Table S1) to specifically suppress hepatic PAI-1 production. Control age-matched DIO mice received AAV8-TBG-ZsGreen. IVC stenosis was performed 3 weeks after injection of AAV8 viruses.

FXR liver-specific knockout mice. The hepatocyte-specific *Fxr-null* mice were generated by injecting 10-week-old *Fxr^{fl/fl}* mice (purchased from Shanghai Model Organisms, China) with AAV8-TBG-Cre to achieve liver-restricted FXR deletion. Control age-matched *Fxr^{fl/fl}* received AAV8-TBG-ZsGreen. IVC stenosis was performed 3 weeks after injection of AAV8 viruses.

DVT model of IVC stenosis in mice. Mice were anesthetized with 5% isoflurane. Once adequate anesthesia was confirmed by loss of reflex and reduced respiratory rate, mice were placed supine on a warming pad. The abdomen was shaved, disinfected with iodine, and draped with sterile towels. A midline laparotomy (~1 cm) was performed to expose the abdominal cavity. The intestines were gently exteriorized onto saline-moistened sterile gauze and covered to prevent desiccation. The abdominal wall was retracted to expose the inferior vena cava (IVC). All branches of the IVC within 1 cm caudal to the left renal vein were ligated with 7-0 silk sutures. The IVC was carefully separated from the abdominal aorta below the left renal vein using blunt dissection. A 7-0 silk suture was passed beneath the IVC and aorta at this level. A 30G curved blunt needle was placed alongside the IVC, and the suture was tied around both the IVC and the needle. After three secure knots were made, the needle was gently withdrawn, resulting in approximately 90% reduction in IVC blood flow. Care was taken to avoid inclusion or compression of the abdominal aorta. The time of ligation was recorded. The intestines were returned to the abdominal cavity. The abdominal musculature and skin were closed in layers. Mice received 0.5 mL of glucose-saline subcutaneously for fluid support and were recovered in a warming chamber, then returned to clean cages after full recovery. After 24

hours, mice were euthanized. The abdomen was reopened, and the IVC was exposed. Thrombi were carefully isolated, weighed, and measured for length.

Real-time PCR analysis. Total RNA was extracted from cultured cells or tissue samples using an RNA Easy Fast Kit (TIANGEN, Cat# DP451) according to the manufacturer's instructions. The purified RNA was reverse-transcribed to cDNA using an Evo M-MLV RT Kit with gDNA Clean for qPCR (Accurate Biology, #AG11728, Changsha, China), and quantitative PCR was performed using a SYBR Green Premix Pro Taq HS qPCR Kit (Accurate Biology, #AG11701) with a CFX96 Real-Time System (Bio-Rad). The real-time PCR primer sequences are listed in Supplementary Table S2. The relative abundances of the involved genes were calculated by normalization to the level of GAPDH.

Immunoblotting. Cultured cells were lysed in RIPA buffer (Cybrdi) supplemented with protease inhibitor (Roche Diagnostics). The protein concentration was determined using a BCA protein assay reagent kit (Thermo Fisher Scientific). Equal amounts of protein were loaded onto 8–10% Bis-Tris precast gels for electrophoresis and were electro-transferred onto PVDF membranes (Roche Life Science). After blocking for 1 h at room temperature, the membranes were sequentially incubated with primary antibodies at 4 °C overnight and secondary antibodies at room temperature for 1 h. The immunoreactive bands were visualized using ECL substrate in a ChampChemi imaging system (SINSAGE). The target protein levels were normalized to those of GAPDH.

Immunohistochemistry analysis. Adipose tissues and vessels were fixed with 4% paraformaldehyde solution, embedded in paraffin blocks and processed by immunohistochemistry. The tissue sections were deparaffinized and rehydrated using a graded ethanol series, rinsed three times in phosphate-buffered saline and incubated with 10% normal goat serum for 30 min to block nonspecific antibody binding. After washing, the samples were incubated with primary antibodies against PAI-1 (Abcam, ab182973, 1:100). The sections were then washed in PBS three times and incubated with secondary antibodies. The sections were stained with DAB according to the manufacturer's protocol, mounted on slides, and photographed using a digital microscope camera. The immunohistochemistry sample images were quantified using ImageJ.

Platelet Aggregation Assay. Mice were anesthetized via intraperitoneal injection of sodium pentobarbital (1%, 60 mg/kg). Blood samples (0.8–1 mL per animal) were collected into anticoagulant-treated tubes and processed by sequential centrifugation: first at 160 ×g for 10 min to obtain platelet-rich plasma (PRP), followed by a second centrifugation of the remaining sample at 2000×g for 10 min to yield platelet-poor plasma (PPP). The platelet count in the PRP was adjusted to 5×10^{11} /L using the PPP. ADP solution (150 μmol/L) was prepared by reconstituting lyophilized ADP powder with 0.5 mL of diluent according to the manufacturer's protocol. Aggregation was initiated by mixing 295 μL of adjusted PRP with 5 μL of ADP solution (final ADP concentration: 2.5 μmol/L), and kinetic parameters were monitored at a controlled temperature (37 °C) using

optical turbidimetry.

Plasmin generation (PG) assay. To each reaction well, 10 μ L of trigger solution (containing 0.5 pM TF, 4 μ M phospholipid, and 0.31 μ g/mL rtPA at final concentrations) was added. For the calibration wells, 10 μ L of α 2-macroglobulin-plasmin was introduced. Subsequently, 40 μ L of diluted plasma was dispensed into all the wells. The reaction was initiated by adding 10 μ L of substrate/calcium solution (0.5 mM Boc-Glu-Lys-Lys-AMC substrate, 16.6 mM calcium chloride, and 60 mg/mL bovine serum albumin in 20 mM HEPES buffer, pH 7.3). The plate was mixed by shaking for 10 seconds. The fluorescence signals (excitation/emission: 390/460 nm) were monitored every 60 seconds for 1 hour using a Biotek fluorometer equipped with a dispenser.

Ch-IP assay. Based on predicted FXR binding sites within the PAI-1 promoter identified using the JASPAR database, four pairs of amplification primers were designed, including a distal control primer set (Ch-IP1). AML12 mouse hepatocytes were transfected with either an empty vector (NC) or FXR overexpression plasmid using Lipofectamine™ 2000. At 36 hours post-transfection, cells were crosslinked with 1% formaldehyde (10 min, RT), quenched with glycine, and processed using the Thermo Fisher Ch-IP kit (#26156). Chromatin was fragmented via MNase digestion (0.5 μ L, 37°C, 15 min), followed by lysis buffer washes. Diluted chromatin samples (500 μ L) were immunoprecipitated overnight at 4°C with 10 μ L anti-FXR antibody (CST #72105) or 2 μ g Normal Rabbit IgG (negative control), then incubated with Protein A/G beads (1 h). After sequential stringent washes (Buffers 1-3), bound complexes were eluted (150 μ L Elution Buffer, 65°C, 40 min), reverse-crosslinked with NaCl/proteinase K (65°C, 1.5 h), and DNA was column-purified using kit buffers. Purified DNA was analyzed by qPCR using the designed primer sets to assess FXR enrichment at target sites versus the distal control (Ch-IP1).

Dual luciferase reporter gene experimental procedure. The software predicted that there were four possible binding sites for FXR in the mouse PAI-1 promoter region, so five promoter fragments, PAI1-1, PAI1-2, PAI1-3, PAI1-4 and PAI1-5, were amplified by PCR using mouse genomic DNA. The PAI-1 core promoter fragment was inserted into the luciferase reporter vector (pGL3-Basic luciferase reporter vector), and the pGL3-basci plasmid was constructed, containing PAI1 -1, PAI1-2, PAI1-3, PAI1-4, PAI1-5 promoter sequences and empty vector. The experiment was divided into two groups: NC+DMSO group and NC+CDCA group; both groups received 5 plasmids for transfection, and 3 replicate wells were set up for transfection; the experiment was repeated on AML12 and 293T cell lines respectively. Resuscitate AML12 and 293T cells respectively, place them in a 37°C incubator with 5% CO₂, pass them through a 24-well plate, and perform plasmid transfection when the cell density is 70%; Transfect luciferase reporter gene plasmid into AML12 and 293T cell lines, that is, take 2 μ L of plasmid mother liquor (0.5 μ g/ μ L) and dissolve it into 250 μ L of Opti-MEM medium as Liquid A. Take 4 μ L of Lipofectamine™ 2000 and dissolve it into Opti-MEM medium as Liquid B. After mixing the Liquid B for 5 min, mix Liquid A with Liquid B,

and then add it into the liquid B after 20 min of resting. After 5 minutes of mixing of liquid B, liquid A and liquid B are mixed and allowed to stand for 20 minutes before being added to the cell culture plate. The above operation is the amount per well, the amount of medium is 500 μ L. 4h after transfection, switch to cell growth medium and add CDCA (25 μ M) for 24h stimulation. After 48h of transfection, the samples were collected for dual fluorescence detection. Discard the medium, wash with PBS twice, and add 100 μ L/well of PLB for 15 min at room temperature. Add 100 μ L/well of PLB, shake gently for 15 min at room temperature and collect the cell lysate→20 μ L of cell lysate was added to the luminescence plate and the background value was read by the bioluminescence detector for 2s→Add 100 μ L/sample of LAR II working solution, mix quickly and read by bioluminescence detector for 2s→Add 100 μ L/sample of Stop & Glo® Reagent, mix quickly, and read for 2s on the luminescence detector → Organize and analyze data.

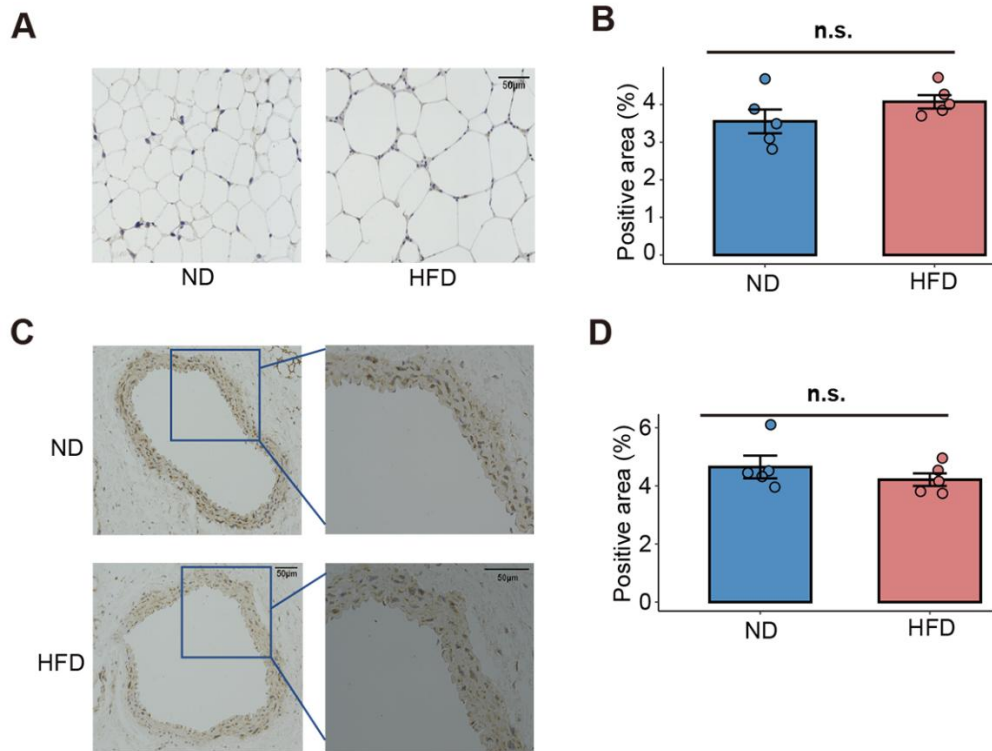
ScRNA-seq and snRNA-seq Data Processing. Data preprocessing for scRNA-seq and snRNA-seq was carried out using the “Scater” R package. Outlier detection was performed based on three key metrics: library size, number of expressed genes, and mitochondrial gene proportion. Cells with values exceeding or falling below the median absolute deviation (MAD) for each metric were excluded. Principal component analysis (PCA) was conducted using the “runPCA” function from “Scater”, followed by outlier identification using the “mvoutlier” R package. Genes not expressed in at least three cells were excluded from further analysis by creating a Seurat object. For normalization and detection of highly variable genes, the “SCTransform” R package was utilized. If batch correction was needed at the sample level, the Seurat functions “NormalizeData” (log2 transformation), “FindVariableFeatures”, and “ScaleData” were combined with the Harmony R package for integration. Clustering and UMAP visualization were performed according to the Seurat workflow⁷.

RNA-seq and Transcriptome Microarray Data Processing. For RNA-seq and microarray data, preprocessing was first performed to ensure normalization. Differential gene expression (DEG) analysis was conducted to identify significant differences between experimental groups. DEG analysis was performed using either the “DESeq2” R package (for RNA-seq) or the “limma” R package (for microarray data), depending on the dataset type and experimental design. Genes with an adjusted p-value < 0.05 and a fold change (FC) > 1.5 were considered differentially expressed. Subsequent functional enrichment analysis was performed to assess the biological relevance of the identified differentially expressed genes.

Reference:

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191 Steatohepatitis and Fibrosis Is Associated With the Antioxidative Gene Expression Profile in
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194 evolutionarily conserved hepatic macrophage niches. *Cell.* 2022;185(2):379-396.e338.



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199 **Figure S1. PAI-1 is expressed across vessels and adipose tissue in mice.** Tissue sections
 200 were subjected to IHC with PAI-1 antibodies and compared between mice fed a ND or HFD:
 201 (A) representative IHC image of adipose tissue and (B) the difference in the percentage of
 202 PAI-1-positive area per total area; (C) representative IHC image of vessels and (D) the
 203 difference of percentage in the PAI-1-positive area per total area (N=5). The data are
 204 represented as the mean $\pm$ SEMs, n.s. $P > 0.05$. vs. ND group.

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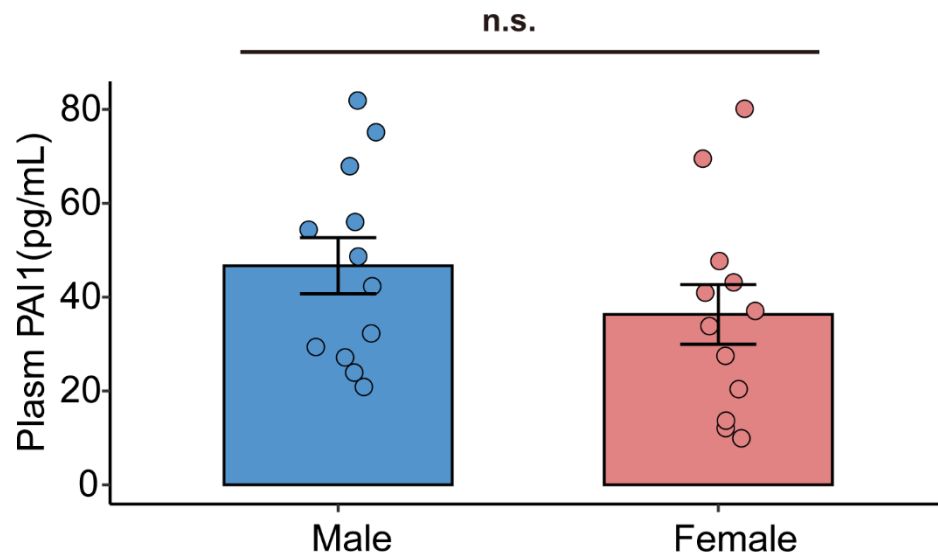


Figure S2. Sex-based differences in plasma PAI-1 levels in humans (N = 12). The data are represented as the mean $\pm$ SEMs, n.s. $P > 0.05$. vs. Male group.

A HFD mice

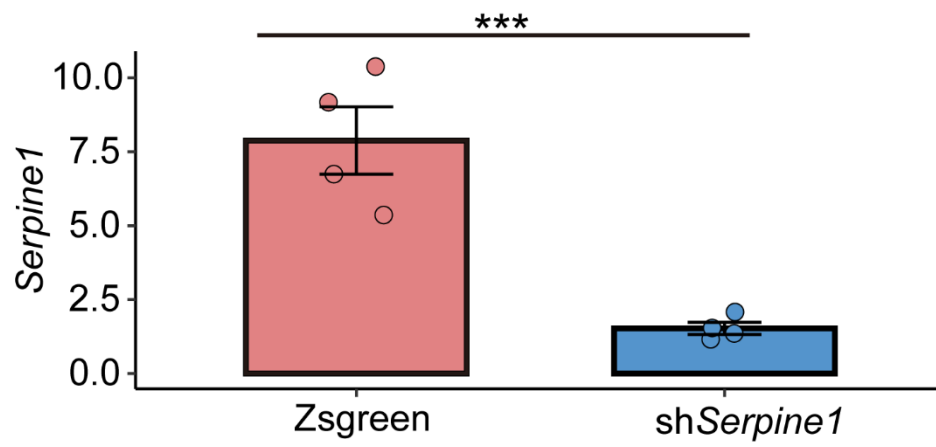


Figure S3. Administration of AAV8-TBG-sh*Serpine1* significantly suppressed hepatic PAI-1 expression in HFD mice (N = 4). The data are represented as the mean $\pm$ SEMs, *** P < 0.001. vs. HFD with AAV8-TBG-Zsgreen.

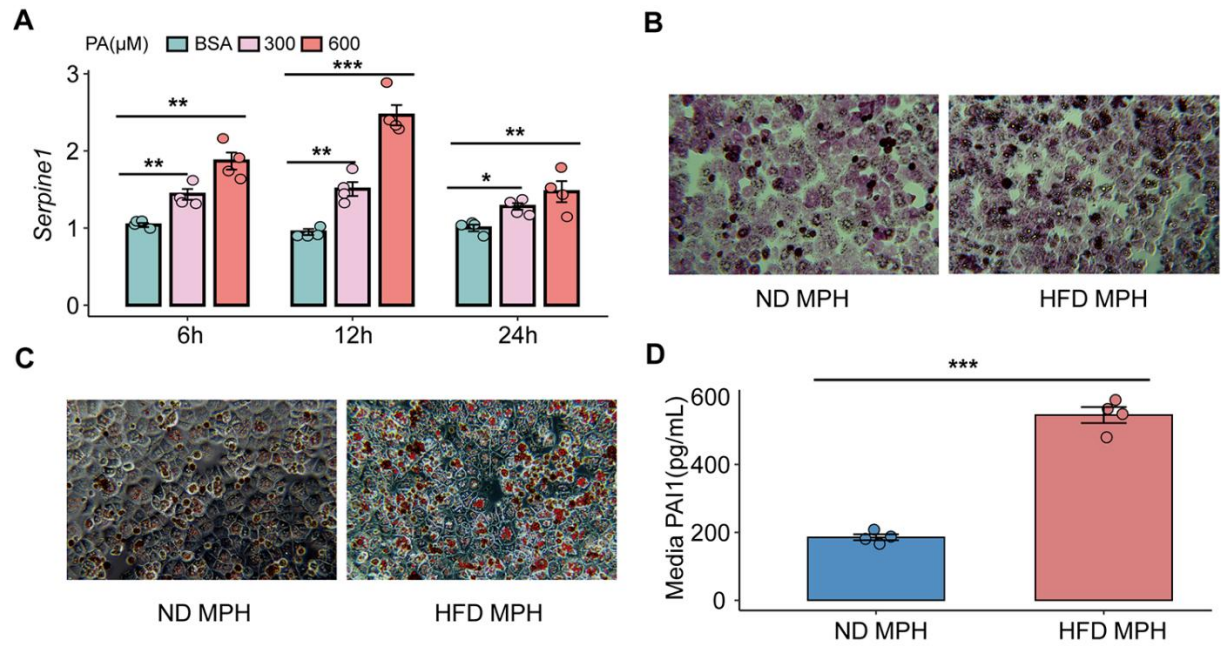


Figure S4. Hepatocytes secrete elevated levels of PAI-1 under obesity conditions. (A)

MPHs from 10-week-old were treated with different time points and concentrations of palmitic acid (N = 4). (B-D) MPHs isolated from HFD and ND mice (N = 4): (B) glycogen and (C) Oil Red O staining; (D) PAI-1 levels in culture medium after 12-hour incubation. The data are represented as the mean $\pm$ SEMs, * P < 0.05; ** P < 0.01; *** P < 0.001. vs. BSA or ND MPH.

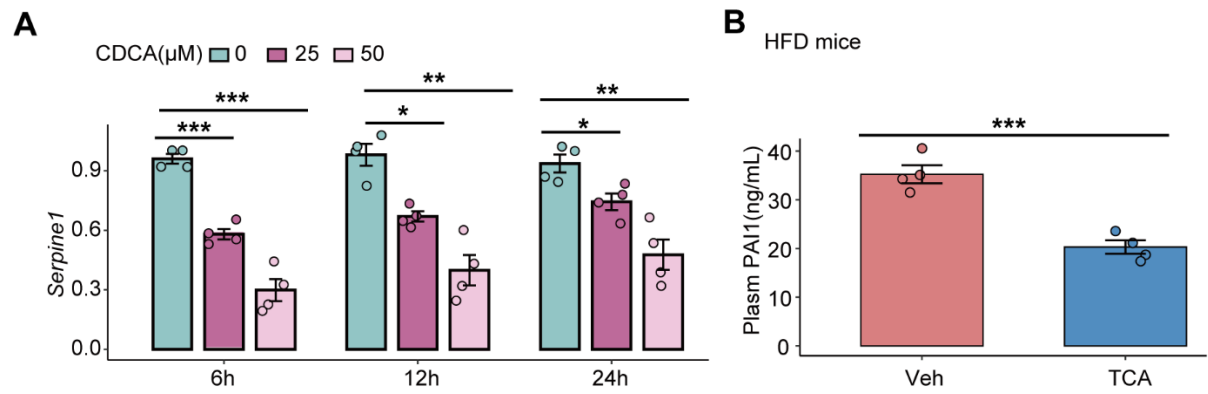


Figure S5. Effects of reagents on the expression of *Serpine1* in MPHs and plasma PAI-1 in HFD mice. (A) MPHs treated for different durations and with different concentrations of CDCA (N = 4). (B) Plasma PAI-1 levels in HFD mice with TCA or vehicle administration (N = 4). The data are represented as the mean $\pm$ SEMs, * P < 0.05; ** P < 0.01; *** P < 0.001. vs. the lowest concentration of CDCA or Veh group.

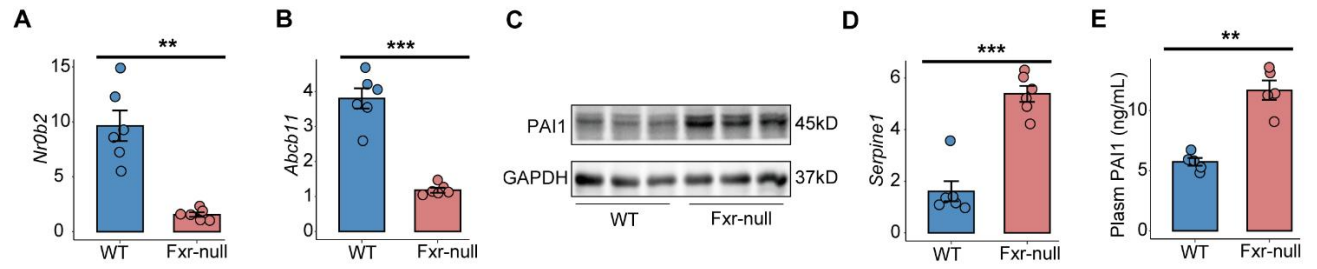


Figure S6. PAI-1 expression in liver and plasma PAI-1 levels in *Fxr-null* mice. (A) Liver *Nr0b2* mRNA (N=6); (B) Liver *Abcb11* mRNA (N=6); (C) Liver PAI-1 protein; (D) Liver *Serpine1* mRNA (N=6); (E) Plasma PAI-1 levels (N=6). The data are represented as the mean $\pm$ SEMs, ** P < 0.01; *** P < 0.001. vs. WT group.

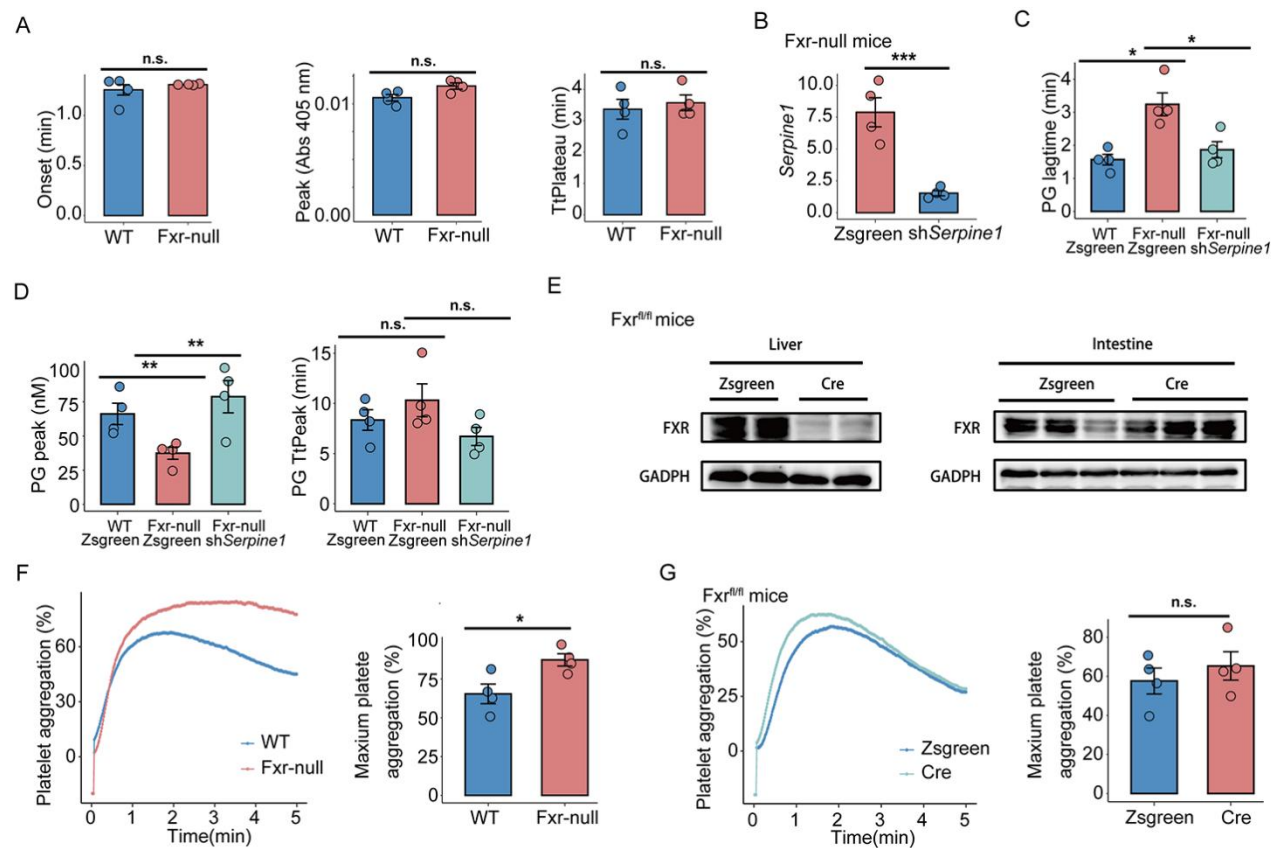


Figure S7. Hepatic FXR reduces thrombosis by suppressing PAI-1 expression. (A) The parameters of plasma fibrin formation and lysis test: Onset, Peak, and TtPlateau (N = 4). (B) Liver *Serpine1* mRNA levels in *Fxr-null* mice injected with AAV8-TBG-sh*Serpine1* or AAV8-TBG-Zsgreen (N = 4). (C-D) The parameters of PG assays (N = 4): (C) PG lagtime; (D) PG peak and PG TtPeak. (E) PAI-1 protein levels in the liver and intestine of *Fxr^{fl/fl}* injected with AAV8-TBG-Zsgreen or AAV8-TBG-Cre. (F) ADP-induced platelet aggregation curves and maximum platelet aggregation rate in *Fxr-null* mice and wild-type mice (N = 4). (G) Platelet aggregation curves and maximum platelet aggregation rates in *Fxr^{fl/fl}* mice with AAV8-TBG-Cre or AAV8-TBG-Zsgreen under ADP induction (N = 4). The data are represented as the mean $\pm$ SEMs, n.s. $P > 0.05$; * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$. vs. WT group or *Fxr-null* mice and *Fxr^{fl/fl}* mice with AAV8-TBG-Zsgreen.

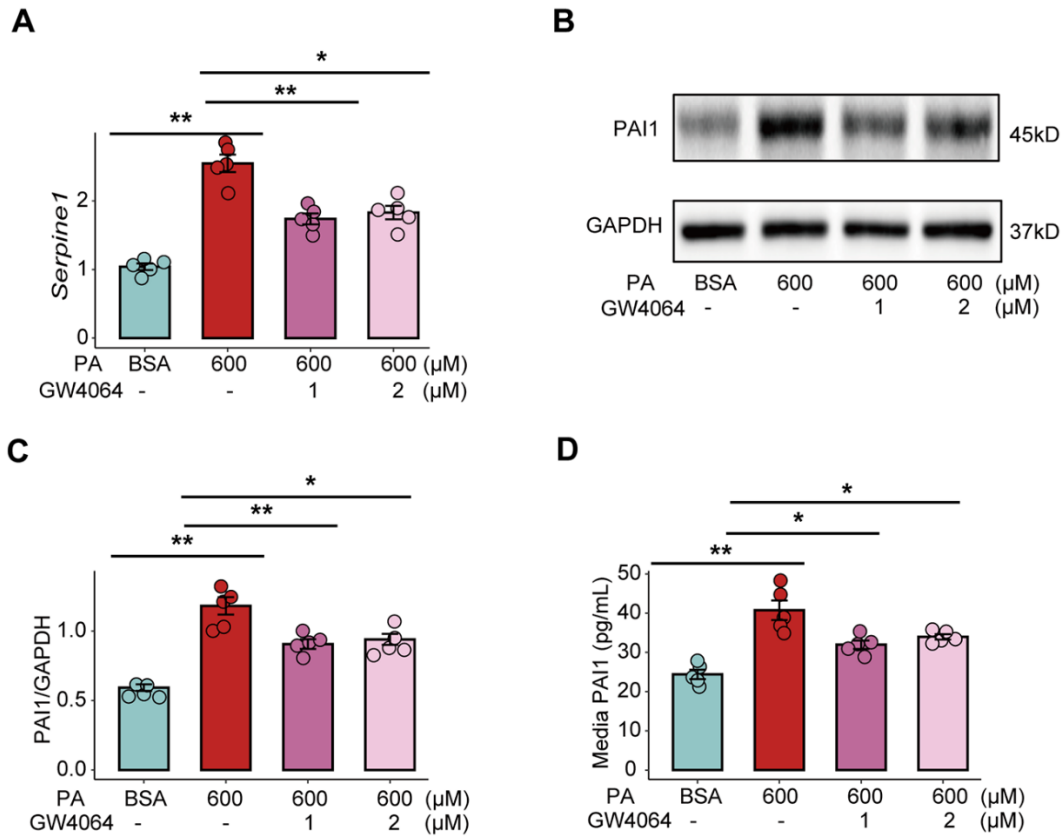


Figure S8. FXR agonists improve obesity-induced PAI-1 expression in hepatocytes.

MPHs were treated with palmitic acid for 6 hours and subsequently coincubated with FXR agonists (GW4064) for the next 6 hours: (A) *Serpine1* mRNA (N=5); (B-C) PAI-1 protein levels (N=5); (D) PAI-1 concentration in medium (N=5). The data are represented as the mean $\pm$ SEMs, * P < 0.05; ** P < 0.01. vs. BSA group.

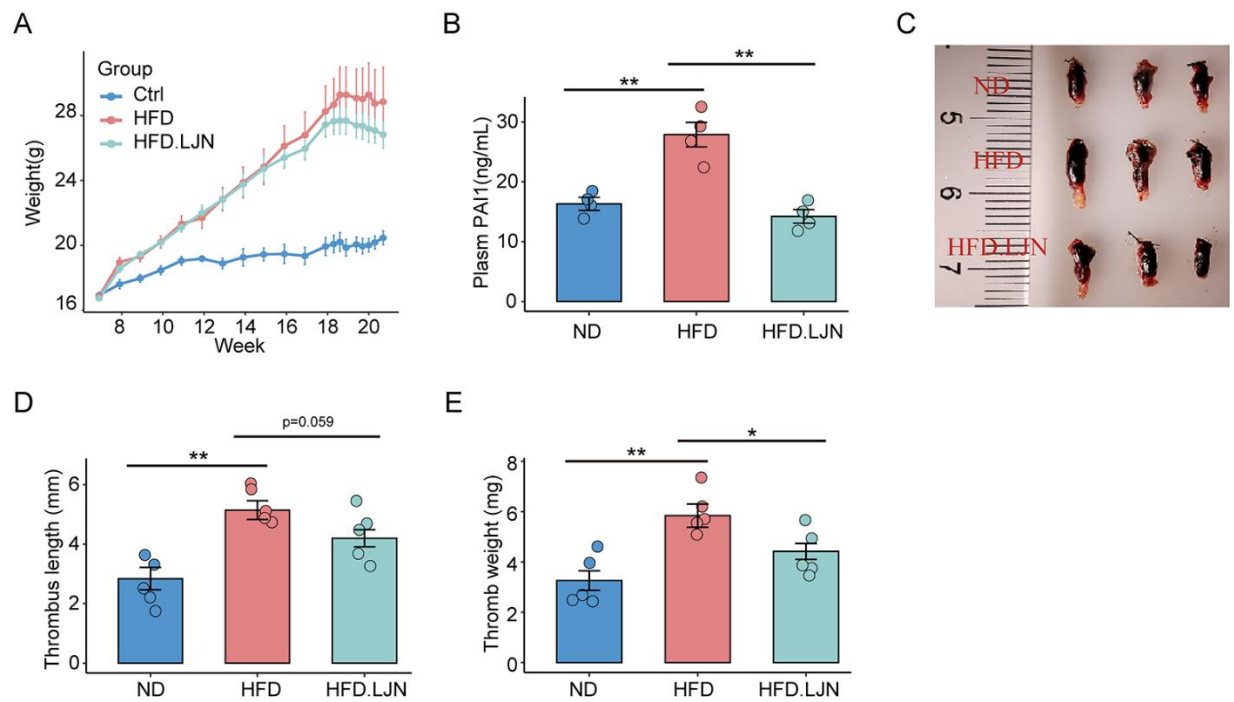


Figure S9. FXR agonists alleviate obesity-associated PAI-1 elevation and thrombotic phenotype in female mice. (A) Body weight curve of female mice. (B) Plasma PAI-1 levels in female mice (N = 4). (C-D) Thrombus weight and length in response to LJN452 treatments (N = 5). The data are represented as the mean $\pm$ SEMs, * P < 0.05; ** P < 0.01. vs. HFD female mice.

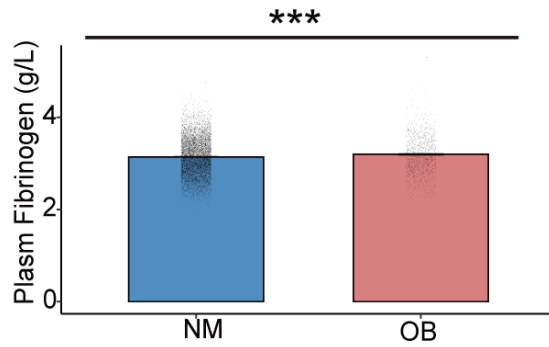
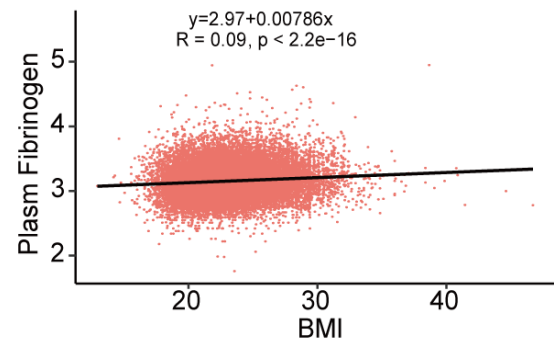
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Figure S10. Obesity and fibrinogen in the Xifei prospective cohort. (A) Fibrinogen levels in obese vs. healthy individuals. (B) Correlation analysis between BMI and fibrinogen. The data are represented as the mean $\pm$ SEMs, *** $P < 0.001$. vs. NM group.

Supplementary Tables

Table S1. Ch-IP primer sequences and corresponding amplified sequences of the PAI-1 promoter.

Ch-IP	Primer Sequence (5'—3')	Amplified sequence of the PAI-1 promoter region
Ch-IP1	Forward	CCGTCCCGGAACTTGCTCTATAG
		CCGTCCCGGAACTTGCTCTATAGACCAACTGAC
	Reverse	TTCCAACCTCAGAGATCTGCCTGCCTTCATTAATAA ATAATTTAAACCAAGGCTCAAAAGGATTAAGTTA
		TAAGTCTCCGTCTATCTGCCTCAG TGCTGGATGGGGGACCACATAGCTGCAATCCCA ACATCTGGAAGGCTGAGGCAGATAGACGGAGA GTT (Located at -5725 bp to -5557 bp of the predicted PAI-1 promoter)
Ch-IP2	Forward	ACTGGTTCTTGCTCCTTGAC
		ACTGGTTCTTGCTCCTTGACAGAGCTTTCTGTG
	Reverse	GTAACCTCTGTTCTCATTAGTGGAAGAGGCTGG TCCAATCCAGCCATCAGCACCCACCCAGTACA CCTCAAAACCCAGCCGACAAAGGCTATTGACAC
		AAAGAGCGAGCCCTCAGGGCACCAGGAGAGTC TGGCCCATGTGGGGAGTCAGACATGCTTCACA GCAGG (Located at -793 bp to -592 bp of the predicted PAI-1 promoter)
Ch-IP3	Forward	TGCCTTCTGTGTCTTCTCAAG
		TGCCTTCTGTGTCTTCTCAAGGAAGTAAACACAG GAGTGTGGGCTGGTAAAGTGGCTCAGTGGGTAG AACACGTGCCACCGGAAACCGATGACCTGAGT
	Reverse	TCAAACACTCTGAATCCCGCATGATGGGAGGAG AGAACCGGTCCCTGAAAGCTGTCCCTGACCT
		CCACACACAAAACATGGCACA (Located at +13 bp to +196 bp of the predicted PAI-1 promoter)
Ch-IP4	Forward	GGCAGGCAAGATGGCTCAGATG
		GGCAGGCAAGATGGCTCAGATGGTAATAAGCAC TACTGAGAAAGTCTATTGATGACCTTAGTTTCG
	Reverse	ATTCCCAGAAACCACACAATGGAAGGGAAGAA CCAATGGGGGTTGCCTCTGACTTGACATACATAC ATGATGGGGACAC (Located at +246 bp to +390 bp of the predicted PAI-1 promoter)

269 The yellow-highlighted region indicates the PAI-1 promoter area predicted by the JASPAR database for
 270 FXR binding.

Table S2. JASPAR database prediction of FXR binding to the PAI-1 promoter.

Matrix ID	Name	Score	Sequence ID	Predicted sequence	Predict sequence position
MA1110.2	Nr1H4	12.199442	PAI-1	TCTGACCTCC	+168bp~+177bp
MA1110.2	Nr1H4	11.230725	PAI-1	GATGACCTGA	+99bp~+108bp
MA1110.2	Nr1H4	11.143495	PAI-1	GATGACCTTA	+297bp ~+306bp
MA1110.2	Nr1H4	7.999789	PAI-1	GGTAACCTCT	-761bp ~-752bp

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Table S3. The sequence of AAV8-TBG-sh*Serpine1*.

Species	Gene	Sequence of AAV8-TBG-sh <i>Serpine1</i>
Mouse	<i>Serpine1</i>	GCTATGGGATTCAAAGTCAAT

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Table S4. The real-time PCR primer sequences.

Species	Gene	Primer Sequence (5'—3')	
Mouse	<i>Gapdh</i>	Forward	AGCAAGGACACTGAGCAAGA
		Reverse	GGGTGCAGCGAACTTTATTGA
	<i>Serpine1</i>	Forward	AGCTTTGTGAAGGAGGACCG
		Reverse	CTGATGGGCTGTGTGGGATT
	<i>Nr1h4</i>	Forward	CCTCGGCTCCTTCTCAGTTG
		Reverse	TTCCGGCACAAATCCTGACA
	<i>Nr0b2</i>	Forward	GCACGATCCTCTTCAACCCA
		Reverse	CAGAAGGGTGCCTGGAATGT
	<i>Abcb11</i>	Forward	GAGTTGGTGTGGCTGTCCTTATC
		Reverse	GTGCAGTCAAACCATCCGATTTC

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