Supporting Information

SupplementaryMaterials and Methods

Generation of *NCoR1*$^{\Delta hep}$ mice.

NCoR1 floxed (*NCoR1*$^{fl/fl}$) and (*Alb*)-Cre mice (*Alb*-cre Tg/0) were generated as described before using the Cre-loxP system.Briefly, offspring that transmitted the mutated allele, in which the selection marker was excised, and that lost the Flp transgene (*NCoR1*$^{L2/WT}$ mice) were selected, mated with mouse albumin (*Alb*)-Cre mice, and then further intercrossed to generate mutant (*Alb*)-cre Tg/0/-*NCoR1*$^{L2/L2}$ mice, which were termed as *NCoR1*$^{\Delta hep}$ mice. *NCoR1*$^{\Delta hep}$ mice, backcrossed for over 10 generations to C57BL/6J were used in experiments with *NCoR1*$^{fl/fl}$used as controls (Supplementary Fig. 1B-1C).

Animals and experimental procedures

All animal experiments were performed in accordance with the National Institutes of Health guidelines and approved by the animal care and use committee of the Second Military Medical University.Partial hepatectomy (PH) surgeries were performed in 8 to 12-week-old mice by removal of 2/3 or 1/3 of the liver. Livers from 5 mice from each group were collected and the liver-to-body weight ratios were calculated. Part of the liver tissue was fixed in 4% neutral buffered formalin for 48 h. A piece of each liver was frozen in O.C.T, a tissue freezing medium and used to obtain fresh frozen sections. The remaining liver samples were immediately frozen in liquid nitrogen for RNA and protein extraction.Diethylnitrosamine (DEN) induced hepatocarcinogenesis, mice at day 15 of age were injected intraperitoneally with DEN (10 mg/kg, i.p) diluted in saline buffer. GW6471(PPARα antagonist, sigma) was injected intraperitoneally(1mg/kg, i.p). Adeno-associated virus was diluted in PBS and injected through caudal vein(virus titer:10^{11}/ml, 200μl/mouse).Livers were collected at indicated times.

Generation of NCoR1$^{\Delta hep}$ primary hepatocyte.

Primary hepatocytes were generated by rapid isolation from liver from *NCoR1*$^{\Delta hep}$ and *NCoR1*$^{fl/fl}$mice. Mice were anesthetized by isolfluane, and then incisions were made and a catheter inserted into the portal vein. The perfusion pump was run at a rate of 2 ml/minute with Hanks perfusion medium, and then the inferior vena cava (IVC) was immediately cut to remove the blood by 5 minutes perfusion. The pump was stopped and liver digest medium consisting of collagenase IV, and perfusion continued at 3 ml/min for 10 minutes. The liver

was removed and placed in a 100 mm plate filled with cold 20 ml washing medium. The liver was diced with tips and forceps and the cells poured through 100/70μm filters into 50 ml centrifuge tubes, followed by addition of 20 ml cold wash medium to the plate and filter in the same tube. Cells were then centrifuged at 268.32×g for 5 minutes at 4°C and washed with Hanks media. The above steps were repeated two times, and the cells were seeded in collagen-coated plates.

Chromatin immunoprecipitation (ChIP) and ChIP-qPCR

Chromatin immunoprecipitation experiments were performed with the ChiP-IT Express kit (Active Motif) according to manufacturer instructions. Briefly, livers isolated from *NCoR1*$^{fl/fl}$ and *NCoR1*$^{\Delta hep}$ mice were fixed in 1% formaldehyde and protease inhibitor cocktail 1 (PIC1, comprised of 1 μg/ml leupeptin, 1.4 μg/ml pepstatin, 0.2 mg/ml PMSF (Sigma), 1 mM EGTA, 1 mM EDTA) for 10 min with gentle rotation at room temperature for 10 min at room temperature. The cross-linking reaction was quenched with Glycine Stop-Fix Solution. The pellet was resuspended in ice-cold lysis buffer and dounced on ice with ~10 to 15 strokes to aid in nuclei release.Nuclei were released after 30 strokes using a dounce homogenizer and collected after centrifugation. Pellets were resuspended in 6ml homogenization buffer (10mM HEPES, pH 7.6, 25 mM KCl, 1 mM EDTA, 1 mM EGTA, 1M sucrose, 10% glycerol, 0.15 mM spermine, supplemented with PIC1) and layered onto 3 ml of the same buffer. Nuclei were then pelleted at 5031×g for 1 hour (Beckman SW41 rotor) and stored at -80ºC. Nuclear pellets were re-suspended in 0.3 ml nuclear lysis buffer (50 mM Tris pH 7.6, 10 mM EDTA and 1% SDS), and diluted with 0.6 ml immunoprecipitation (IP) dilution buffer (0.01% SDS, 1.1% Triton ×100, 167 mM NaCl, 16.7 mM Tris pH 7.6, 1.2 mM EDTA). For sonication, 0.3 ml (1/3) of nuclear lysate was sonicated for 25-30 cycles 30 seconds on 30 seconds at 4ºC with a BioRuptor twin sonicator (Diagenode). Sonicated chromatin was then further diluted to 1 ml with IP dilution buffer, which is sufficient for three ChIP reactions. The NCoR1 antibody was conjugated to Protein G Dynabeads (Life Technologies) in PBS containing 0.5% BSA overnight at 4ºC with gentle rotation. Sonicated DNA (0.33ml made up to 1ml in IP dilution buffer) was then incubated overnight with Protein G-NCoR1 beads at 4ºC with gentle rotation. For input samples, 10% was used. Samples were then washed twice with wash buffer I (20 mM Tris, pH 7.4, 150 mM NaCl, 0.1% SDS, 1% Triton ×100, 2 mM EDTA), three times with alternate wash buffer III (10 mM Tris, pH 7.4, 250 mM LiCl, 1% NP-40 alternate, 0.7% deoxycholate, 1 mM EDTA), two washes with 0.2% Triton ×100 TE buffer and two final washes with 50 mM NaCl containing TE buffer. Chromatin was recovered and DNA from IP and input was isolated after reverse crosslinking (incubating at 65ºC overnight in 0.3M NaCl and Proteinase K digestion). The DNA was subjected to the QIAquick PCR purification kit (Qiagen) before real-time PCR.NCoR1

ChIP-Seq data have been previously described (GSM647027 and GSM647028). Data were converted to BigWig files and visualized using the IGV browser.

RNA sequencing

Total RNA isolated with TRIzol reagent was treated with RNase-free DNaseI (New England BioLabs) at 37 ºC for 10 min. The Dynabeads mRNA Purification Kit (Life Technologies) was used to isolate mRNA from the total RNA samples. RNA-seq libraries were sequenced as paired-end 100 bp sequence tags using the standard Solexa pipeline.

Cell mitochondria isolation and mitochondrial ROS detection.

The fractionation of intracellular mitochondria was performed with a Cell Mitochondria Isolation Kit (Beyotime Biotechnology, China) according to protocols. $2x10^8$ primary hepatocyte isolated from livers of *NCoR1*$^{fl/fl}$ and *NCoR1*$^{\Delta hep}$ mice and were collected immediately. Density gradient centrifugation was used to separate mitochondrial fractions and supernatants containing cytoplasmic proteins were collected for quality analysis. The remaining pellet (mitochondria) was lysed for further ROS detection and immunoblotting. ROS was determined fluorometrically at an excitation wave length 485 nm and an emission wave length of 538 nm using a fluorescence microplate reader (Synergy HT, USA). Isolated mitochondria (1mg/mL) were suspended in a respiratory medium and loaded with 50 μmol/L H2DCFDA (Sigma) for 15 min at 37°C. Mitochondrial suspensions (0.2 mL) were transferred to a 96-well plate, and the fluorescence was monitored for 30min to calculate the rate of ROS formation. The results were expressed as relative fluorescence units (RFUs).

Analysis of hepatic biochemical parameters

Frozen liver tissues (~100 mg) from mice with PH or DEN treatment were homogenized in 1 mL solution containing 5% NP-40 in water. The samples were slowly heated to 80-100ºC in a water bath for 2-5 min, and then cooled to room temperature. The samples were then centrifuged for 2 min to remove any insoluble material. Samples were detected by commercially available kits, and the results were normalized to protein concentration of the homogenate.

Metabolic measurements

Mitochondrial oxygen consumption rate (OCR) and extracellular acidification rates (ECAR) were measured using a Seahorse XF-96 extracellular flux analyzer, as previously described (Seahorse Bioscience Inc., North Billerica, MA).[26] On the day before the experiment, the sensor cartridge was placed into the calibration buffer supplied by Seahorse Bioscience and incubated at 37°C in a non-CO_2 incubator. Primary hepatocytes were seeded

on Seahorse XF-24 plates at a density of 1 x 10^5cells per well overnight. Cell number were calculated and the viability of cells were observed in order to guarantee the homogeneity of every test. Cells were washed and incubated with assay medium (DMEM without bicarbonate) at 37°C in a non-CO_2 incubator for 1 hour. All media and reagents were adjusted to pH 7.4 on the day of the assay. Analyses were performed both at basal conditions and after injection of the glycolysis inhibitor (2-DG, 5 μM) or mitochondrial poisons (1 μM oligomycin, OLI;1 μM fluorocarbonyl cyanide phenylhydrazone, FCCP; 1 μM rotenone, and 1 μM antimycin A, AA) at the indicated time points.

Cell culture, gene silencing and cell viability detection

The human hepatoma cell lines HCC-LM3 and Huh7 were purchased from Cell Bank of Type Culture Collection of the Chinese Academy of Sciences. Cell lines were routinely cultured in Dulbecco's modified Eagle's medium (4.5 g/L glucose and 2 mM glutamine) (Gibco) supplemented with 10% fetal bovine serum (FBS). For glucose starvation experiments, cells were washed with PBS and cultured in glucose-free DMEM (Gibco) containing 10% FBS. For FBS deprivation experiments, the cells were washed with PBS and cultured in 1% FBS. Stable HCC-LM3 and Huh7 HCC cells silenced for NCoR1 expression were generated using lentiviral constructs expressing sh*NCoR1* and negative control (Genechem Co. Ltd., Shanghai), and maintained with 2 μg/mL puromycin (Sigma-Aldrich). Cell viability under different conditions were detected by a CCK-8 assay or clone formation. Real-time cell viability detection was measured in a specialized device which could detect cells continuously (xCELLigence RTCA, Roche).

Immunohistochemistry and Oil Red O staining

Paraffin-embedded liver sections (5 μm thick) were used for immunohistochemical detection of Ki67, 8-OHdG and Tunnel assays. Tissue microarray slides were deparaffinized and rehydrated in ethanol, and then treated with 0.3% hydrogen peroxide in methanol to block endogenous peroxidase activity. To detect 8-OHdG, the antigen retrieval method was performed using EDTA and EGTA (pH 8.0). To detect Ki67, the antigen retrieval method was performed using 10 mM sodium citrate (pH 6.0). After blocking nonspecific antigens with 2% BSA in PBS for 30 min, the sections were incubated with 1:100 diluted rabbit anti-Ki67 (Cell signaling) or anti-8-OHdG polyclonal antibody (ABCAM) at 4°C overnight. Corresponding secondary antibodies were used at 4°C for one hour, followed by diaminobenzidine (DAB) staining (Dako, Carpinteria, CA). Sections were counterstained with hematoxylin for staining of nuclei. The assessment of immunostaining was performed using the ImageScope software (Media Cybernetics, Inc., Bethesda, MD) according to the staining intensities and the percentage of positively-stained cells. For Tunnel staining, proteinase K digestion was used.

Sections were rinsed with two changes of PBS-Tween 20 for two minutes each. Sections were incubated in 3% H_2O_2 in PBS for 10 minutes to block endogenous peroxidase activity. Sections were incubated in TdT Reaction Buffer for 10 minutes and in TdT Reaction Mixture for 1-2 hours at 37-40°C in a humidified chamber. Then sections were rinsed in stop wash buffer for 10 minutes and nuclei stained with DAPI(blue) and images photographed using a confocal laser scanning microscopy (Leica, Wetzlar, Germany).

For Oil Red O staining, O.C.T tissue blocks from partial hepatectomy and DEN treatment were cut into 50-μm-thick sections. Sections were washed with distilled water, and after rinsing with 70% ethyl alcohol and distilled water. Tissues were then stained with freshly prepared Oil Red O working solution for 20 minutes at room temperature. The working solution of Oil Red O (Sigma Aldrich, St. Louis, MO) was prepared by diluting a stock solution, i.e. 0.5 g of Oil Red O in 100 ml of isopropanol, with distilled water at a ratio of 3:2 (v/v). After rinsing with 70% ethyl alcohol and washing with distilled water for 5 minutes, the tissues were stained with DAPI. Lipid droplets were visualized with a Nikon eclipse 80i microscope with the appropriate filters. Representative images were taken with a Nikon DS-Ri1 digital camera.

Real-time PCR and western blotting

Total RNA was isolated from *NCoR1*$^{fl/fl}$ and *NCoR1*$^{\Delta hep}$ livers using the Trizol method according to the manufacturer's protocols. Real-time PCR analyses were performed using an ABI 7300 Fast Real-Time PCR System (Applied Biosystems, Foster City, CA) and SYBR Green PCR kit (Applied TaKaRa, Otsu, Shiga, Japan). The ΔCt method was used with actin mRNA as an endogenous control for normalization of the results. Primers used in this study are shown in Supplementary table. 2.

Liver tissues were lysed in Triton lysis buffer (20 mM Tris, pH 7.4, 137 mM NaCl, 10% glycerol, 1% Triton X-100, 2 mM EDTA, 1 mM PMSF, 10 mM sodium fluoride, 5 mg/ml of aprotinin, 20 mM leupeptin and 1 mM sodium orthovanadate) and centrifuged at 12 000g for 15 min. Protein concentrations were determined via the BCA assay kit according to manufacturer's protocols. Specific primary antibodies used were as follow: antibody against CyclinD1, CyclinA2, CyclinB1, pRB, C-MYC, P53, PAR4, Caspase-3 (full length and cleaved forms), Caspase-9 (full length and cleaved forms) were purchased from Cell Signaling Technology (USA). Antibodies against ACC1, ACC2, FASN, KAT8, HDAC3 were obtained from Proteintech and β-actin from Santa Cruz Biotechnology (CA). Antibody against NCoR1 was obtained from ABCAM. The dilutions were 1:1000 in 5% BSA. After incubating with the fluorescein-conjugated secondary antibody, the immunocomplexes were detected using an Odyssey fluorescence scanner (Li-Cor, Lincoln, NE).

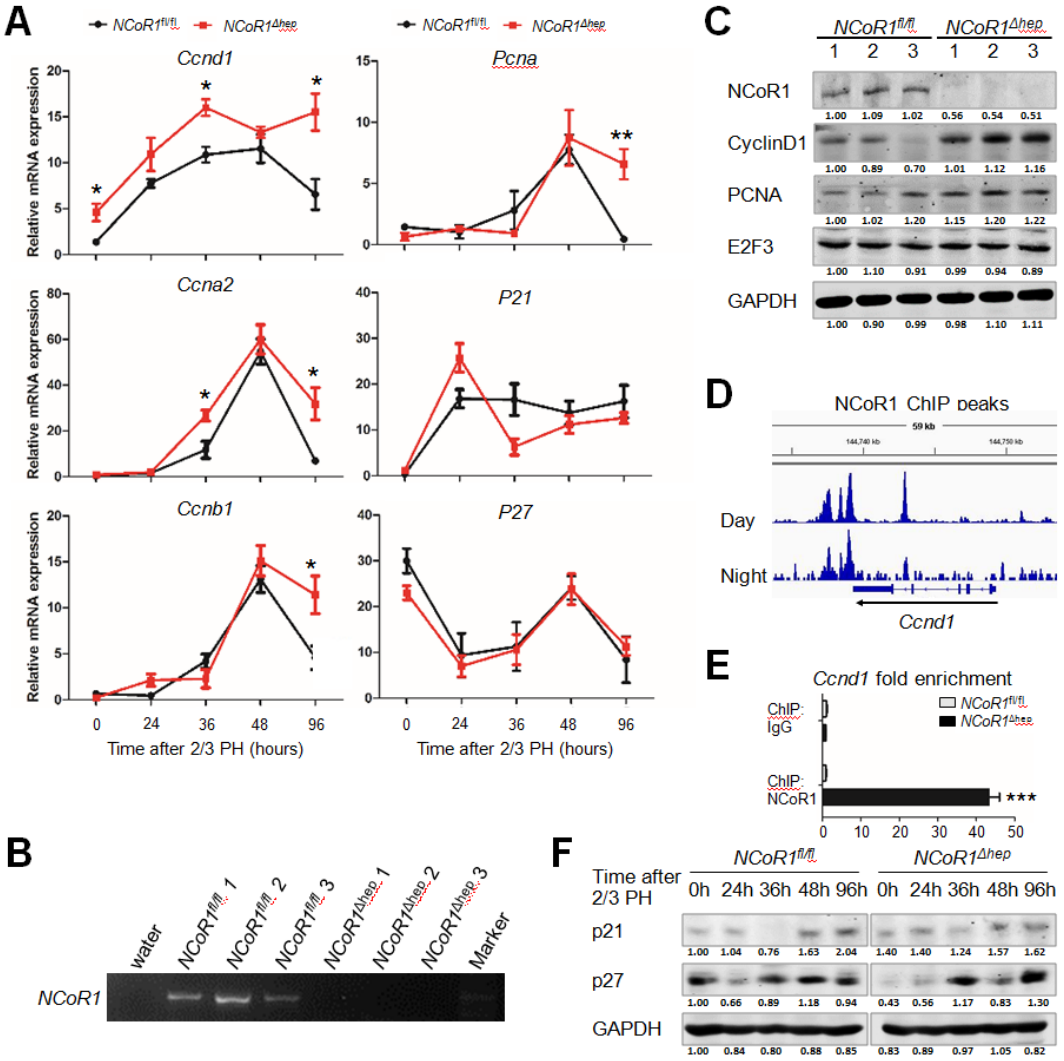
Analysis of hepatic biochemical parameters

Frozen liver tissues (~100 mg) from mice with PH or DEN treatment were homogenized in 1 mL solution containing 5% NP-40 in water. The samples were slowly heated to 80-100ºCin a water bath for 2-5 min, and then cooled to room temperature. The samples were then centrifuged for 2 min to remove any insoluble material. TG content was determined using a commercially available kit (BioVision, Milpitas, CA, USA) following the manufacturers' instructions. NADPH and GSH assays were performed using enzymatic cycling methods. NADP+/NADPH and GSSG/GSH ratios were measured from liver tissue lysates using commercially available kits according to manufacturer's protocols (BioVision, Milpitas, CA, USA). ATP level was measured using the ATP lite assay (BioVision, Milpitas, CA, USA). 8-OHdG Check is a competitive enzyme-linked immunosorbent assay (ELISA) using monoclonal antibody that is highly specific for DNA damage (Stressmarq Biosciences Inc, Canada). These results were also normalized to protein concentration of the homogenate. Liver tissues from at least 5 mice per group were used for measurements.

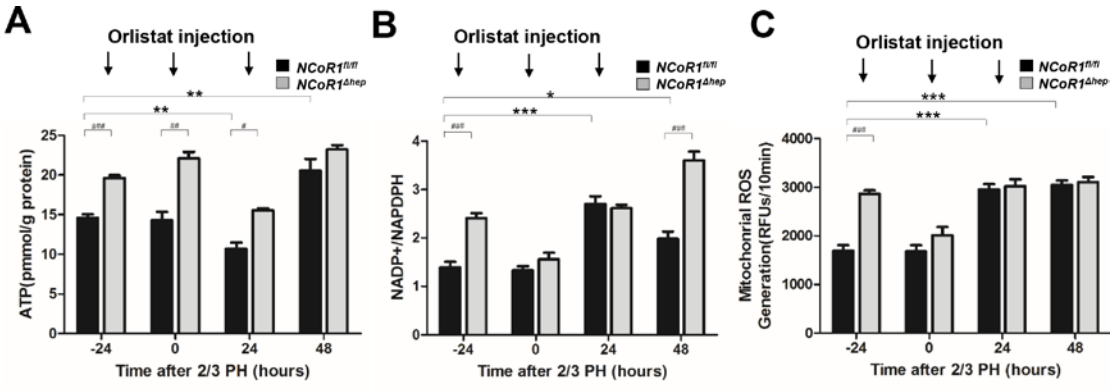
Analysis of serum parameters

For serum biochemical analysis, total blood was withdrawn after Enucleation of the eye under isolfluane anesthesia, allowed to clot at room temperature and centrifuged for 5 min at 1341.6×g in a microcentrifuge. Serum was stored at -20ºC. AST, ALT and total bilirubin were measured in an Analysis Laboratory for serum of animals (Mindrid, China).

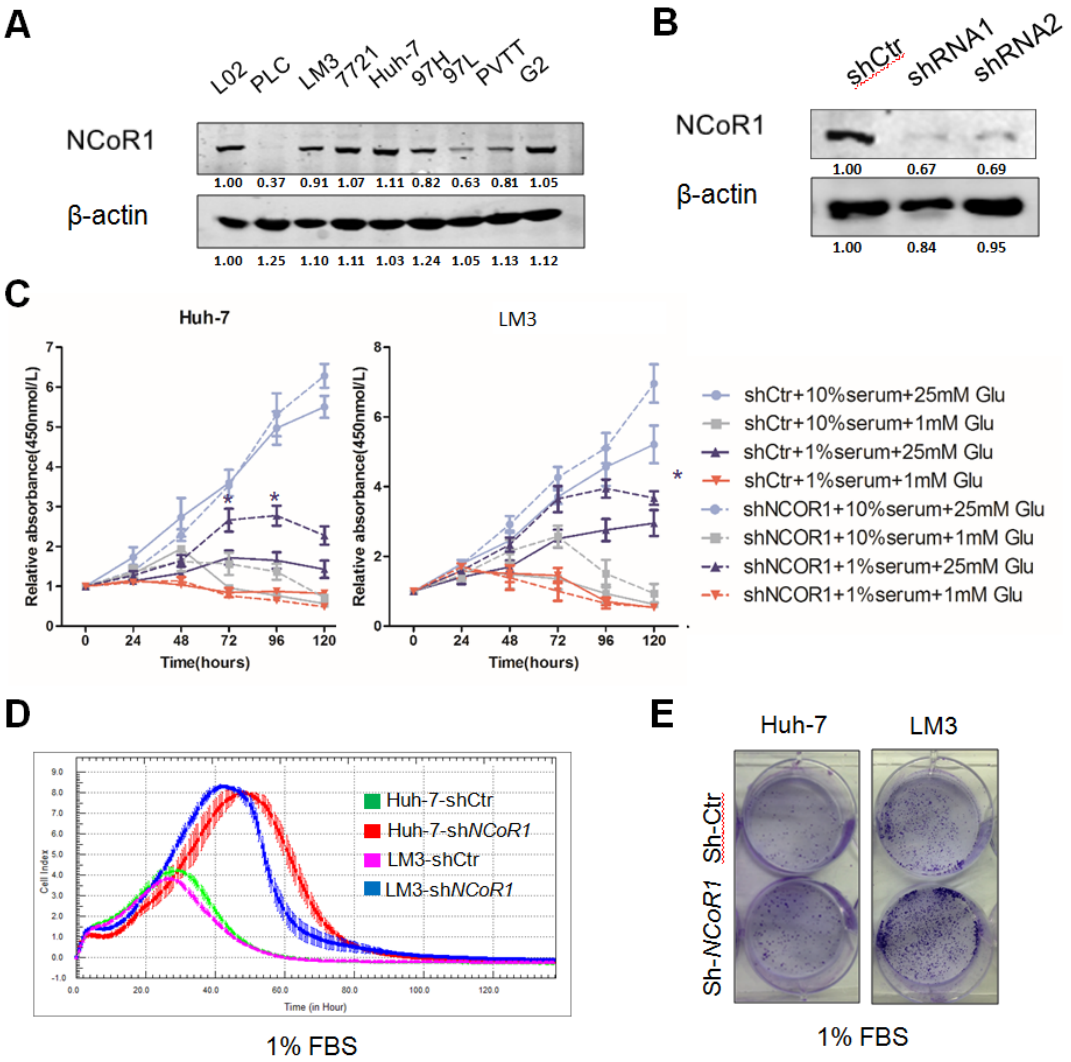
Supplementary Figures



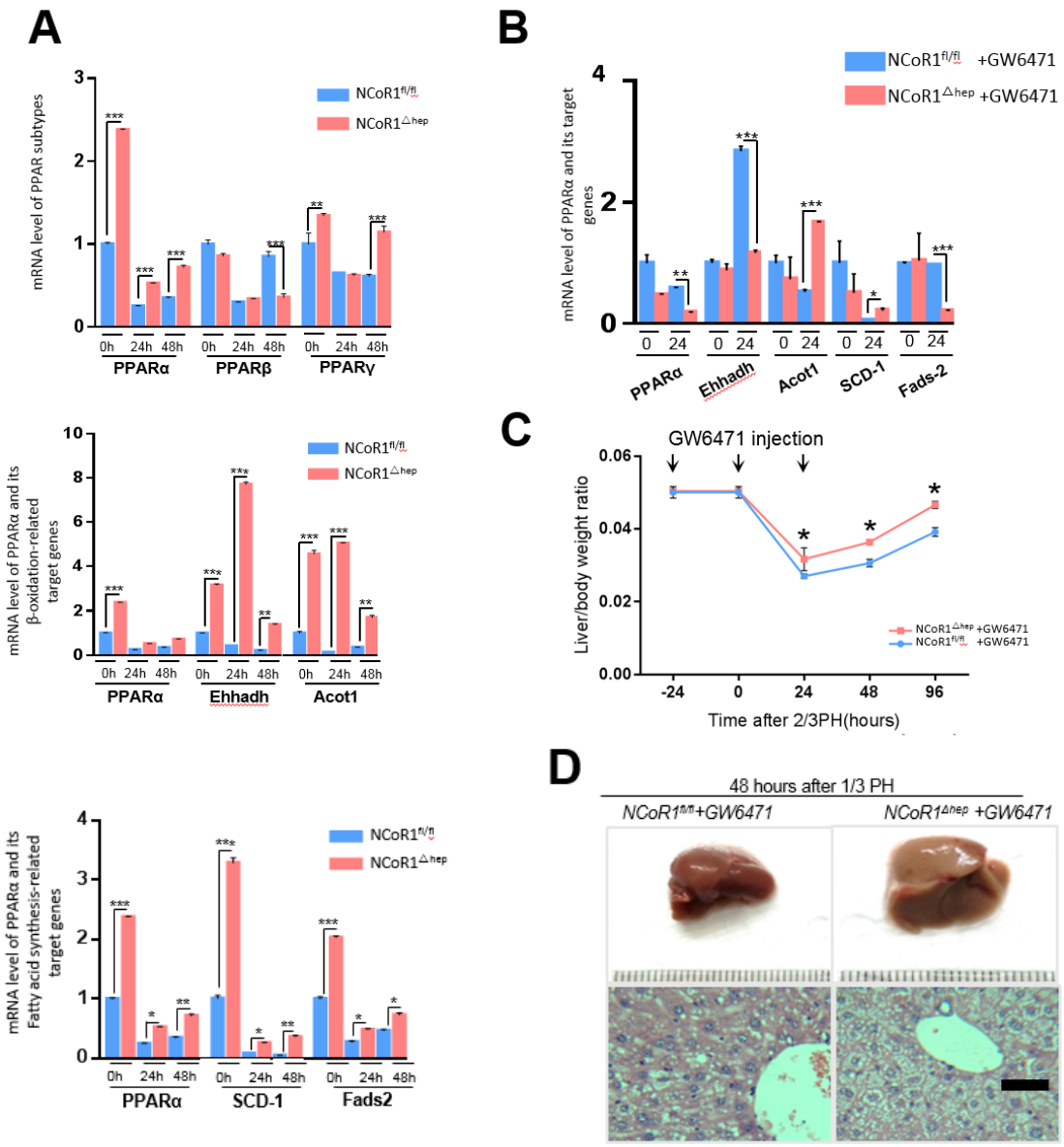
Supplementary Figure 1 |(**A**) qPCR analysis of *Ccnd1, Ccna2, Ccnb1, Pcna, P21* and *P27* mRNA in the liver of *NCoR1*$^{fl/fl}$ and *NCoR1*$^{\Delta hep}$ livers mice after 2/3 PH. Data are presented as mean ± SEM (n=4). The different degrees of significance were indicated as follows in the graphs: *P<0.05; **P<0.01 *NCoR1*$^{\Delta hep}$ mice compared to *NCoR1*$^{fl/fl}$ mice. (**B**)Agarose gel electrophoresis for the qPCR product for mRNA detection of *NCoR1* in *NCoR1*$^{fl/fl}$ and *NCoR1*$^{\Delta hep}$ mice (**C**) Western blot expression of protein level of NCoR1, CyclinD1, PCNA and E2F3 in *NCoR1*$^{fl/fl}$ and*NCoR1*$^{\Delta hep}$ mice. (**D**) NCoR1 binding site from ChIP-seq results in *Ccnd1.* (**E**) ChIP-qPCR result using anti-NCoR1 antibody and anti-IgG antibody for *Ccnd1.* (**F**) Western blot expression of protein level of P21 and p27 after 2/3 PH in *NCoR1*$^{fl/fl}$ and *NCoR1*$^{\Delta hep}$ mice. (Two Way ANOVA plus Student's t test for **A;** Student's t test for **E**).

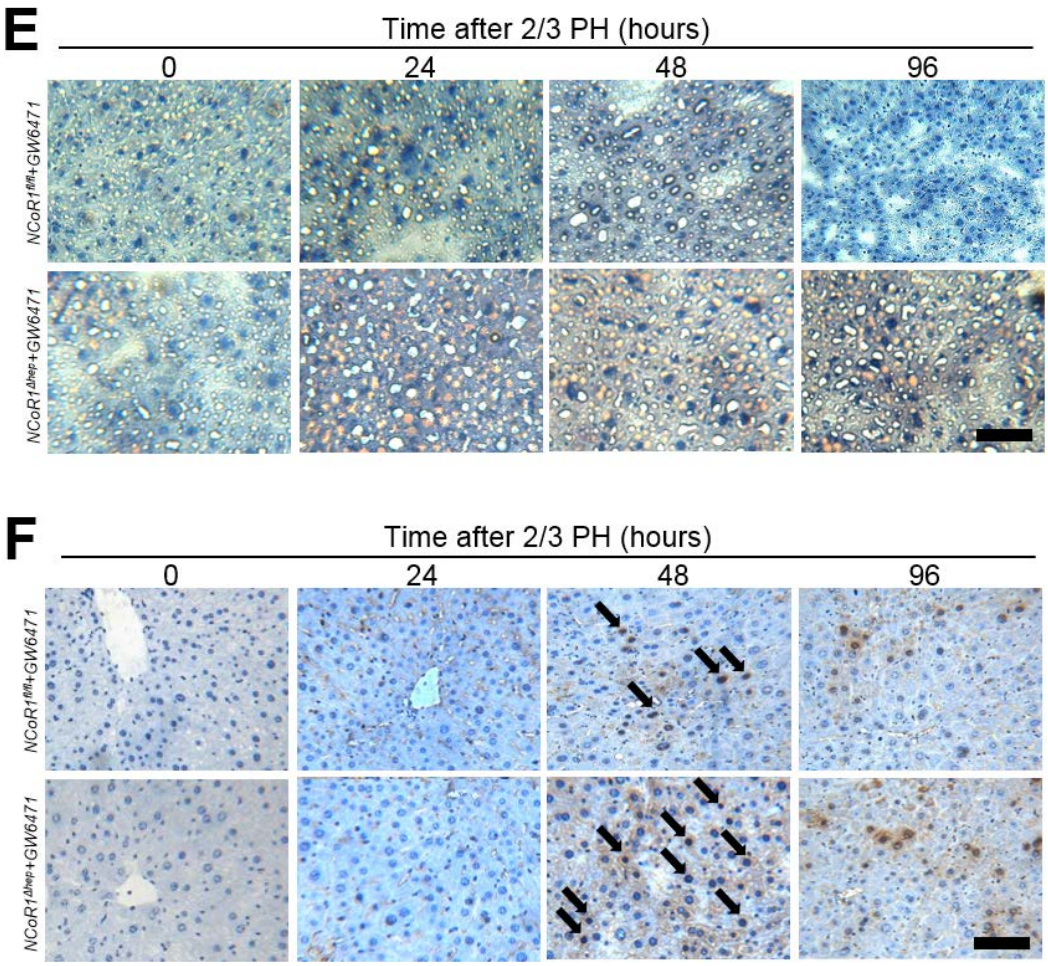


Supplementary Figure 2 |Biochemical detection of intracellular ATP levels(**A**), NADP+/NADPH ratios(**B**) and mitochondrial ROS(**C**) in *NCoR1*$^{fl/fl}$ and *NCoR1*$^{\Delta hep}$ mice pretreated with orlistat at the indicated times after 2/3 PH.Data are presented as mean ± SEM (n=4). The different degrees of significance were indicated as follows in the graphs: **P*<0.05; ***P*<0.01; ****P*<0.001, *NCoR1*$^{fl/fl}$ mice treated with orlistat at -24 hours compared with *NCoR1*$^{fl/fl}$ mice treated with orlistat at indicated times. $^{\#}P$<0.05; $^{\#\#}P$<0.01, $^{\#\#\#}P$<0.001, *NCoR1*$^{\Delta hep}$ mice treated with orlistat compared to *NCoR1*$^{fl/fl}$ mice treated with orlistat at indicated times. (Student's t test for**A, B, C**).

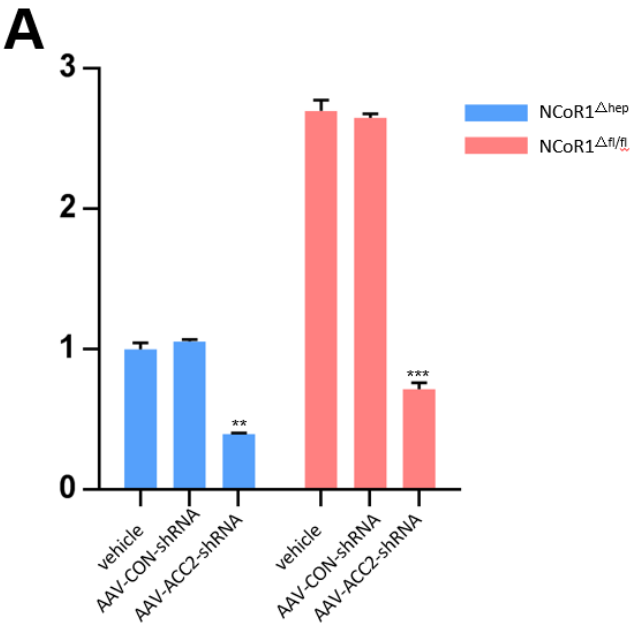


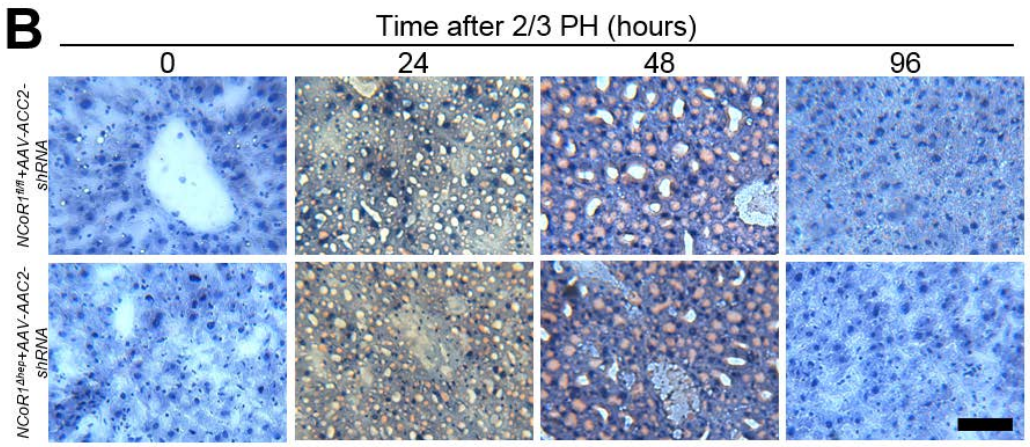
Supplementary Figure 3 |(A) Western blot expression of protein level of NCoR1 in different hepatoma cell lines. (**B**) Western blot expression of protein level of NCoR1 after stable NCoR1-knock down by using lentivirus-based small hairpin RNA. (**C**) HCC-LM3 cells and Huh7 cells were transfected with lentivirus-based small hairpin RNA targeted to NCoR1 (shNCoR1), which established stable NCoR1-knockdown cell lines. Cells with shCtr (negative control) and shNCoR1 were maintained in medium with different glucose concentration (1 mM or 25 mM) and different FBS concentration (1% or 10%) for 120 hours and the cell viabilities were detected by CCK-8 assay at indicated times. Data are presented as mean ± SEM (n=4). The different degrees of significance were indicated as follows in the graphs: **P*<0.05; ***P*<0.01, sh*NCoR1* compared to shCtr at indicated condition. (**D**) Continuous measurement of cell viability of shCtr and shNCoR1 of HCC-LM3 cells and Huh7 cells in 1% FBS medium. (**E**) Plate colony assay cell viability of shCtr and shNCoR1 of HCC-LM3 cells and Huh7 cells in 1% FBS medium for 1 week. (Two Way ANOVA plus Student's t test for **C**).

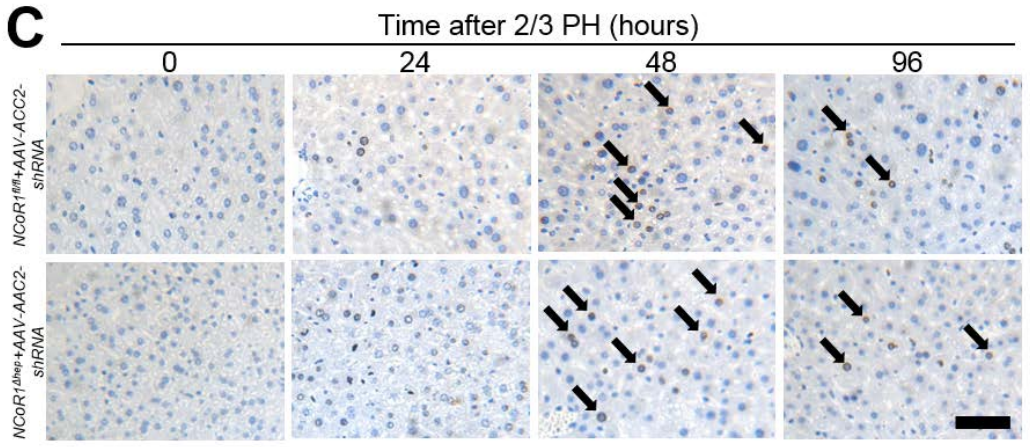

A
mRNA level of PPAR subtypes
NCoR1fl/fl
NCoR1Δhep
0h 24h 48h
PPARα
PPARβ
PPARγ
mRNA level of PPARα and its β-oxidation-related target genes
Ehhadh
Acot1
mRNA level of PPARα and its Fatty acid synthesis-related target genes
SCD-1
Fads2
B
mRNA level of PPARα and its target genes
NCoR1fl/fl +GW6471
NCoR1Δhep +GW6471
0 24
PPARα
Ehhadh
Acot1
SCD-1
Fads-2
C
GW6471 injection
Liver/body weight ratio
Time after 2/3PH(hours)
D
48 hours after 1/3 PH
NCoR1fl/fl+GW6471
NCoR1Δhep +GW6471



Supplementary Figure 4 | The blockade of PPARα does not affect the process of Liver regeneration in NCoR1fl/fl or NCoR1Δhep mice after 2/3 partial hepatectomy. (A) mRNA expression change of PPARs and the target genes of PPARα in the process of liver regeneration were detected by qPCR, Data are presented as mean ± SEM (n=4). *P<0.05; **P<0.01; ***P<0.001. (B) mRNA expression change of PPARα and its target genes in the process of liver regeneration after being blocked by its antagonist GW6471 were detected by qPCR, Data are presented as mean ± SEM (n=4). *P<0.05; **P<0.01; ***P<0.001. (C) Liver weight to body weight ratio analysis in NCoR1fl/fl and NCoR1Δhep mice after being administered with GW6471(1mg/kg/i.p.) over a time course from 0 to 96 hours after 2/3 PH. Data are presented as mean ± SEM (n=4). *P<0.05; **P<0.01, NCoR1Δhep mice compared to NCoR1fl/fl mice at the indicated times. (D) Morphological changes of livers of NCoR1fl/fl mice and NCoR1Δhep mice after being administered with GW6471(1mg/kg/i.p.) at the 48 hours after 2/3 PH as determined by H&E staining. scale bar: 50 μm. (E) Analysis of the lipids by Oil Red O staining performed with frozen liver sections. scale bar: 50μm. (F) Immunohistochemical analysis of Ki67 in paraffin tissues from liver of NCoR1fl/fl and NCoR1Δhep mice at the same time. scale bar: 50μm . (Student's t test for A, B, C).


A
3
2
1
0
NCoR1Δhep
NCoR1Δfl/fl
**
***
vehicle
AAV-CON-shRNA
AAV-ACC2-shRNA
vehicle
AAV-CON-shRNA
AAV-ACC2-shRNA


B
Time after 2/3 PH (hours)
0
24
48
96
NCoR1fl/fl+AAV-ACC2-shRNA
NCoR1Δhep+AAV-AAC2-shRNA


C
Time after 2/3 PH (hours)
0
24
48
96
NCoR1fl/fl+AAV-ACC2-shRNA
NCoR1Δhep+AAV-AAC2-shRNA

Supplementary Figure 5|(A) mRNA expression change of ACC2 after the injection of AAV-ACC2-shRNA and AAV-CON-shRNA in livers of both mice groups were detected by qPCR, Data are presented as mean ± SEM (n=4). *P<0.05; **P<0.01; ***P<0.001.(B) Analysis of the lipids by Oil Red O staining performed with frozen liver sections. scale bar: 50μm. (F) Immunohistochemical analysis of Ki67 in paraffin tissues from liver of NCoR1fl/fl and NCoR1Δhep mice at the same time. scale bar: 50μm . (Student's t test for A).

Supplementary Tables

Supplementary Table 1. Enhanced de novo FAS, glucose flux, mitochondrial function following PH in NCoR1Δhep mice.

Parameters	WT	NCoR1 hep-/-	*P*
Glucose (U/L)	3.90±1.49	2.82±0.94	<0.05
TG (mmol/L)	1.21±0.43	1.58±0.73	
TC (mmol/L)	2.45±0.37	2.49±0.43	
ALT (U/L)	76.75±14.03	93.50±45.52	
AST (U/L)	208.00±46.53	488.62±248.30	<0.05
TBA (μmol/L)	5.50±4.02	11.88±5.50	<0.05
Alb (g/L)	32.50±3.16	30.50±3.49	

Abbreviations: TC: total cholesterol; TG: Triglyceride; Alb: Albumin; ALT: Alanine Aminotrasferase; AST: Aspartate Aminotransferase; TBA Total Bilirubin. Data are mean +/- SEM (3-5 animals/condition). Student's test was used.

Supplementary Table 2. Primer used for PCR

Primer	Sequence
Mouse Ncor1 forward	TGGACAGAAGAAGAAATGGAAG
Mouse Ncor1 reverse	GGCAGAAACAGTGGAAGCAACA
Mouse Ccnd1 forward	AAGCATGCACAGACCTTTGTGG
Mouse Ccnd1 reverse	TTCAGGCCTTGCATCGCAGC
Mouse Ccna2 forward	TGATGCTTGTCAAATGCTCAGC
Mouse Ccna2 reverse	AGGTCCTCCTGTACTGCTCAT
Mouse Ccnb1 forward	CTGCACTTCCTCCGTAGAGC
Mouse Ccnb1 reverse	AAA ATGCACCATGTCGTAGTCC
Mouse Pcna forward	AGGGTTGGTAGTTGTCGCTG
Mouse Pcna reverse	ATTCACCCGACGGCATCTTT
Mouse Cdkn1a forward	GCTGTCTTGCACTCTGGTGT
Mouse Cdkn1a reverse	CTGCGCTTGGAGTGATAGAA
Mouse Cdkn1b forward	CAGGCGGTGCCTTTAATTGG
Mouse Cdkn1b reverse	TTCGGGGAACCGTCTGAAAC
Mouse Acaca forward	CATGTCATTGTTCCTGGTCAAAC
Mouse Acaca reverse	AAGAGCCTCCTCTTAGCCACCTC
Mouse Acacb forward	AGAACTCCAAACCAACTGGACAG
Mouse Acacb reverse	CTGTCTCTTGATGTGTGCCTGC
Mouse Glut2 forward	GCCCAGCAGTTCTCAGGAAT
Mouse Glut2 reverse	ACATGCCAATCATCCCGGTT
Mouse Glut4 forward	CTACTCAGGGCTAACATCAGGGC
Mouse Glut4 reverse	AGCATAGACTCCAAGCCCAGCAC
Mouse Fasn forward	AGATGGAAGGCTGGGCTCTA
Mouse Fasn reverse	GAAGCGTCTCGGGATCTCTG
Mouse G6pdx forward	GCTTGGACCGCCATTTTGTC
Mouse G6pdx reverse	GGCTGGAAGGGAGGTGATTC
Mouse Gsst forward	TGTACCTGGATCTGCTGTCG
Mouse Gsst reverse	CTTCTCCGAAGGCCCGTATG
Mouse Pkm1 forward	TGCTGAAGGCAGTGATGTGGCC
Mouse Pkm1 reverse	TGGAGTGACTGGAGGCTCGCAC
Mouse Pkm2 forward	ACCAGCGACCCCACAGAAGCTGC
Mouse Pkm2 reverse	CACGGCATCCTTACACAGCAC
Mouse PPARα forward	CTATAATTTGCTGTGGAGATCGGC
Mouse PPARα reverse	GGATGGTTGCTCTGCAGGT
Mouse PPARβ forward	TCCATCGTCAACAAAGACGGG
Mouse PPARβreverse	ACTTGGGCTCAATGATGTCAC
Mouse PPARγ forward	TCGCTGATGCACTGCCTATG
Mouse PPARγreverse	GAGAGGTCCACAGAGCTGATT
Mouse Ehhadh forward	CGGTCAATGCCATCAGTCCAA
Mouse Ehhadhreverse	TGCTCCACAGATCACTATGGC
Mouse Acot-1 forward	CGATGACCTCCCCAAGAACAT
Mouse Acot-1reverse	CTTTTACCTCGGGGTGGCT

Mouse SCD-1 forward	TTCTTGCGATACACTCTGGTGC
Mouse SCD-1reverse	CGGGATTGAATGTTCTTGTCGT
Mouse Fads2 forward	AAGGGAGGTAACCAGGGAGAG
Mouse Fads2reverse	CCGCTGGGACCATTTGGTAA
Primers were produced in Invitrogen (Shanghai. China)	