

Supplemental information list

Supplemental Figures

- **Fig. S1. (related to Fig.1) Pathway analysis of proteomics of different liver disease models and validation studies of NANOG target genes identified by NANOG ChIP-seq.**
- **Fig. S2. (related to Fig.2) Validation of reconstituted bone-marrow-derived cells and Tlr4- and Nanog-dependency of mouse TICs isolated from liver tumor model.**
- **Fig. S3. (related to Fig.3) TLR4 stimulation transactivates NANOG through TAK1 and TBK1-mediated phosphorylation of E2F1 at serines 337 and 332.**
- **Fig. S4. (related to Fig.4) Silencing of Tlr4 or Nanog promotes basal levels of oxygen consumption rate.**
- **Fig. S5.(related to Fig.5) NANOG cooperates PPARs to promote FAO of TICs.**
- **Fig. S6. (related to Fig.6) Silencing Nanog promotes glutaminolysis pathway, ATP production and glucose flux in TICs judged by metabolomics analysis, qRT-PCR and stable isotope experiments.**
- **Fig. S7. (related to Fig.7) Restoration of OXPHOS and/or suppression of FAO reduce the tumor growth and drug resistance.**

Supplemental Experimental Procedures

Supplemental Discussion

Supplemental References

Supplemental Tables

- **Table S1. 48 Signature proteins in all group analysis.**
- **Table S2. Average scores of liver histology in HCV Core and/or NS5A Tg mice fed the ethanol or Western diet (WD) for 12 months.**
- **Table S3. List of members of each clusters in Chip-seq clusters 1 to 4**