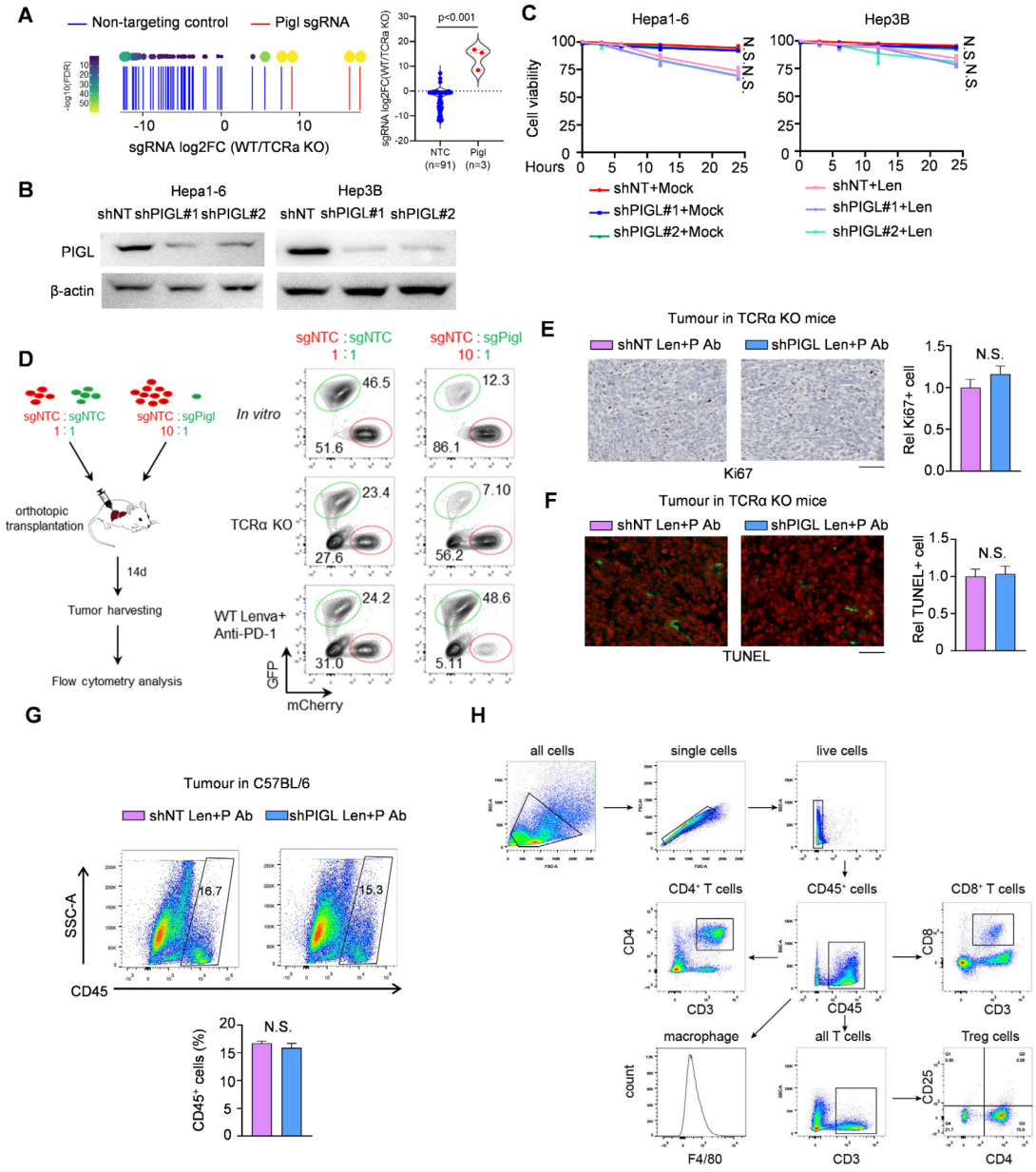
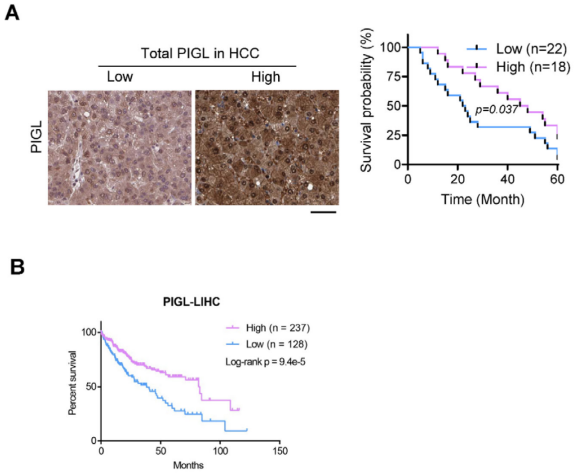
Supplementary Figure and Legends

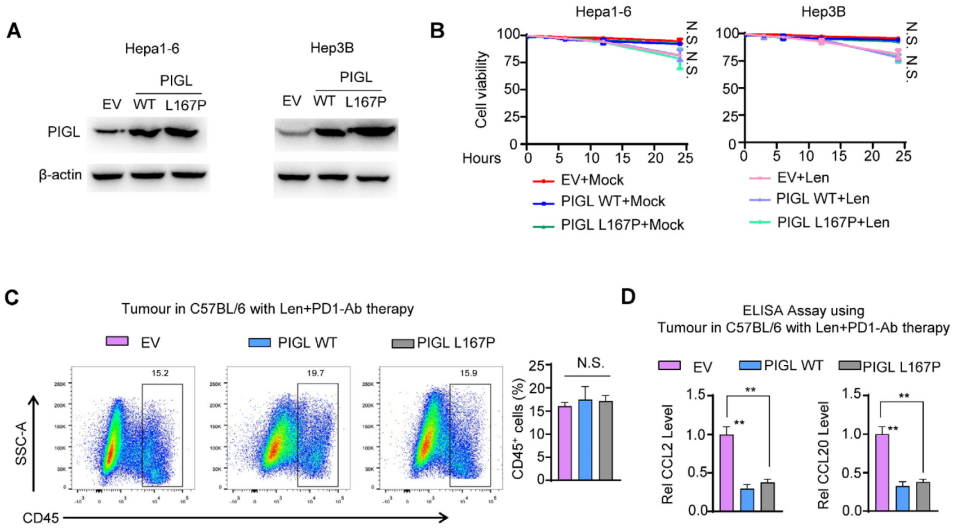


Supplementary Figure 1 referred to Figure 1. (A) Frequency distribution of enrichment or depletion (log2 FC) for indicated sgRNAs (left). Non-targeting control sgRNAs are shown in blue lines; sgRNAs targeting PIGL are shown by the red lines. Quantification and cooperation are shown in the violin plot (right). (B) PIGL depletion in Hepa1-6 cells or Hep3B cells was confirmed by WB using indicated antibodies. (C) In Hepa1-6 with or without PIGL under Lenvatinib treatment for indicated time points, then *in vitro* Cell viability was tested by CCK8 assay. (D) Diagram of the competing growth assay *in vivo* (left). Cell fractions within the tumour of indicated treatments were tested using flow cytometry (right). (E-F) In Hepa1-6 cell formed solid tumours in TCRα mice, the *in vivo* cell proliferation (E) and cell death (F) were measured by Ki67 and TUNEL, respectively. Scale bar, 200 μm. (G) The proportions of $CD45^+$ cells in shNT or shPIGL Hepa1-6 cells formed

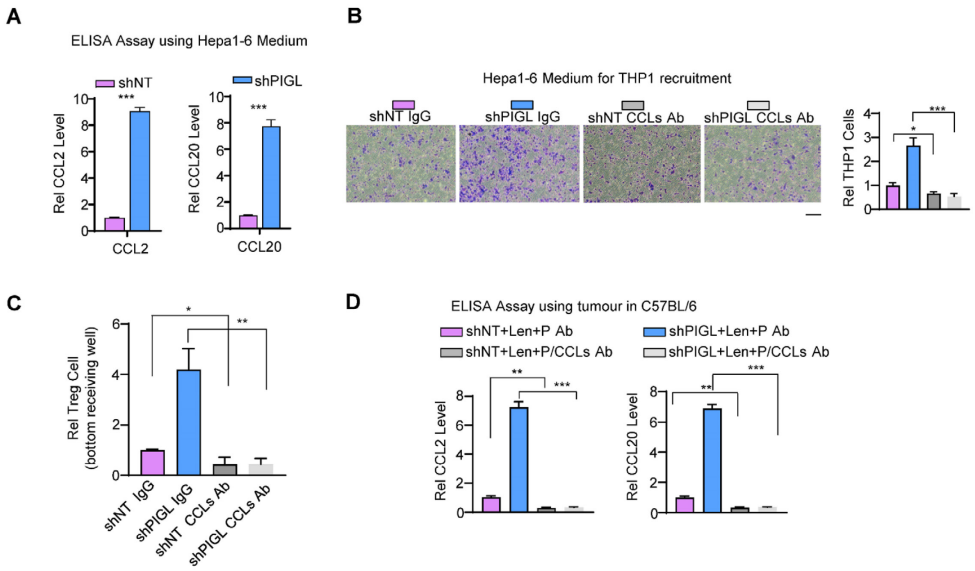
tumours upon lenvatinib plus PD-1 antibody treatment were tested by FCM. (H) Gating strategy to analyze the population of $CD45^+$ cells, $CD4^+$ T cells, CD8+ T cells, Treg cells or macrophages in mouse tumours. C, two-way ANOVA; E-G, two-tailed Student's t-test. (N.S., not significant).)



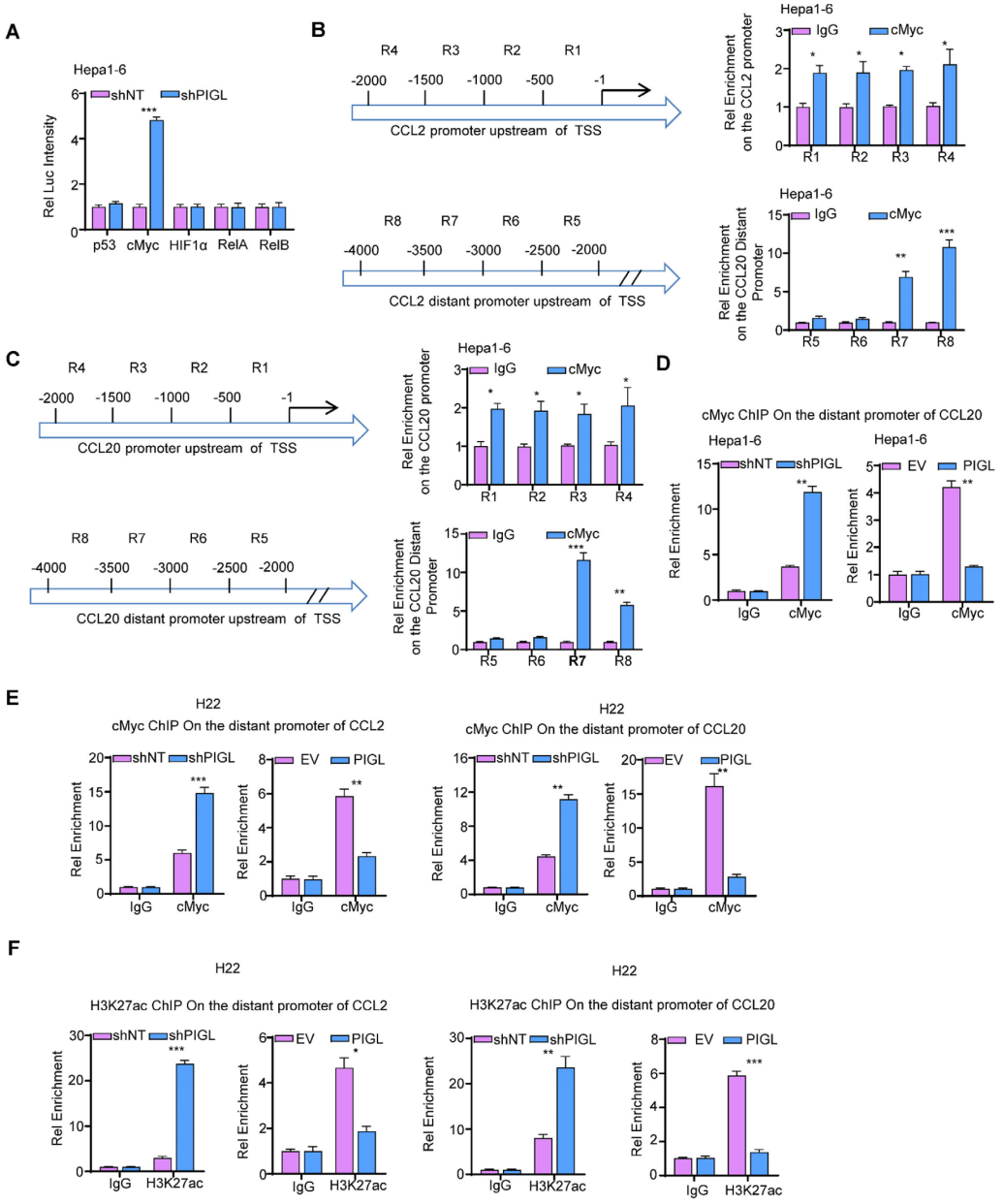
Supplementary Figure 2 referred to Figure 2. (A). The survival curves were plotted using the best cutoff of the IHC score, which separated HCC patients into two groups according to total PIGL IHC intensities. (B) PIGL mRNA information was extracted in TCGA-LIHC database. With beat separation, survival curves were plotted by grouping liver HCC (LIHC) patients into high or low intensity according to PIGL mRNA levels. A-B, two-way ANOVA.



Supplementary Figure 3 referred to Figure 3. (A). WT- or L167P-PIGL overexpression in Hepa1-6 cells or Hep3B cells was confirmed by WB using indicated antibodies. EV, empty vector. (B) In Hepa1-6 and Hep3B with or without WT- or L167P-PIGL overexpression under Lenvatinib treatment for indicated time points, then *in vitro* cell viability was tested by CCK8 assay. (C) Upon lenvatinib plus PD-1 antibody treatment, the proportions of $CD45^+$ cells in the whole tumours formed by indicated Hepa1-6 cells were tested by FCM. (D) Using solid tumours in Figure 3B, ELISA was performed using ELISA kit corresponding to the test of soluble and secreted CCL2 or CCL20. B, two-way ANOVA; C-D, two-tailed Student's t-test. (N.S., not significant; $^{**}p < 0.01$).

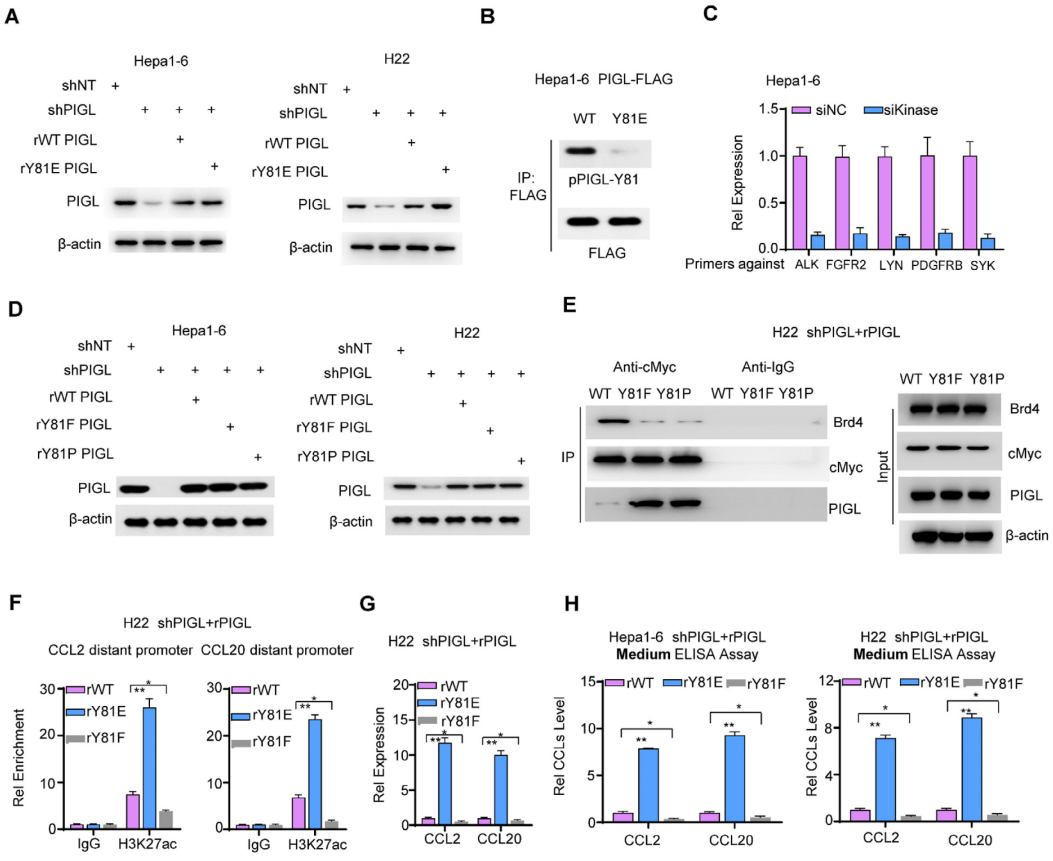


Supplementary Figure 4 referred to Figure 4. (A). In the medium from Hepa1-6 cells with or without PIGL, ELISA was performed using ELISA kit corresponding to the test of soluble and secreted CCL2 or CCL20. (B) With or without CCL2/CCL20 antibody (CCLs Ab) neutralization, the effect of conditioned medium (CM) from indicated genetically manipulated Hepa1-6 on THP1 cells recruitment was detected by transwell migration assay. Scale bar, 200 μm. (C) Treg cells in the bottom wells are quantified in the migration assay with indicated conditions. (D) Using solid tumours in Figure 4C, ELISA was performed. A-D, two-tailed Student's t-test. (*p < 0.05; **p < 0.01; ***p < 0.001).



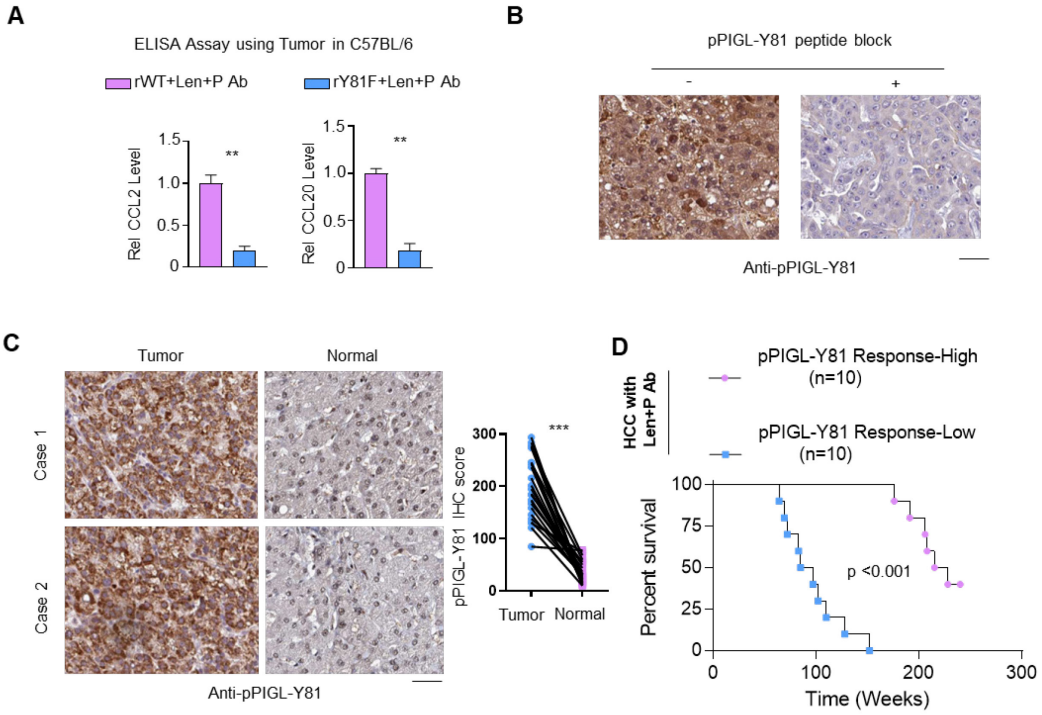
Supplementary Figure 5 referred to Figure 5. (A). Dual luciferase reporter assay. The five major transcriptional factors, p53, cMyc, HIF1α or RelA/B were co-transfected with pGL3-Response

element and Renilla inner control plasmids into Hepa1-6 cells with or without PIGL depletion. (B-C) The 4000 base pairs in the upstream of transcriptional start site of CCL2 gene (B, Left) or CCL20 gene (C, Left) was shown and ChIP-qPCR assay was performed using antibodies against cMyc. IgG was used as a blank control for ChIP assay. Data was collected and analyzed (B and C, Right). (D) ChIP-qPCR assay was performed to test whether cMyc targeted the promoter of CCL20 gene, using antibodies against cMyc in indicated Hepa1-6 cells with PIGL depletion or overexpression. (E-F) ChIP-qPCR assay was performed to test whether cMyc (E) or H3K27ac (F) targeted the promoters of CCL2 and CCL20, using antibodies against cMyc (E) or H3K27ac (F) in indicated H22 cells with PIGL depletion or overexpression. A-F, paired two-tailed Student's t-test. (*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$).



Supplementary Figure 6 referred to Figure 6. (A) Restored expression of WT- or Y81E-PIGL in Hepa1-6 and H22 cells was confirmed by WB using indicated antibodies. (B) FLAG tagged WT- or Y81E-PIGL were transiently expressed in Hepa1-6 cells and enriched for WB using indicated antibodies. (C) siRNAs targeting five TKs coding genes were shown to be transiently transfected into Hepa1-6 cells. Primers against these five genes were used to perform qRT-PCR. TK, tyrosine kinase. (D) Restored expression of WT-, Y81F- or Y81P-PIGL in Hepa1-6 and H22 cells was

confirmed by WB using indicated antibodies. (E) Using H22 cells with restored expression of WT-, Y81F- or Y81P-PIGL, Co-IP was performed to test the association of cMyc with BRD4 and PIGL. (F-G) In indicated H22 cells, ChIP–qPCR using an H3K27ac antibody was performed to test H3K27ac enrichment at the distant promoters of CCL2/20 (F). Readout gene expression was detected using qRT–PCR (G). (H) In the medium from Hepa1-6 and H22 cells with restored WT-, Y81E- or Y81F-PIGL, ELISA was performed using ELISA kit corresponding to the test of soluble and secreted CCL2 or CCL20. C and F-H, two-tailed Student's t-test. (*$p < 0.05$; **$p < 0.01$).



Supplementary Figure 7 referred to Figure 7. (A). In the solid tumours in Figure 7A, ELISA was performed using ELISA kit corresponding to the test of soluble and secreted CCL2 or CCL20. (B) The specificity of antibody against pPIGL-Y81 phosphorylation in tissue level was tested by incubating indicated peptide to block the antigen. Scale bars: 200 μm. (C) pPIGL-Y81 phosphorylation in 25 paired HCC tumour and adjacent normal tissues was tested by an antibody specially against pPIGL-Y81 phosphorylation. Scale bar, 200 μm. (D) pPIGL-Y81 phosphorylation level was calculated in Figure 7E. With median separation, survival curve was plotted by grouping HCC patients into high or low intensity according to the changed levels of pPIGL-Y81 phosphorylation before and after Lenvatinib. A, two-tailed Student's t-test. C, two-way ANOVA.

Supplementary materials and methods

Cell lines, antibodies and reagents

Cell lines: Hepa1-6, H22, Hep3B, THP-1 and HEK293T were obtained from the SIBCB (Institute of Biochemistry and Cell Biology, SIBS, CAS, China) cell collection or American Type Culture Collection. The cells were authenticated using the short tandem repeat (STR) method. Hepa1-6, H22, Hep3B and HEK293T Cells were cultured in DMEM supplemented with 10% FBS and penicillin-streptomycin (100U/mL) and incubated in 37℃ with 5% CO2. THP-1 cells were cultured using RPMI-1640 supplemented with 10% FBS, penicillin-streptomycin (100U/mL) and 2-mercaptoethanol (0.05 mM). Mycoplasma were detected at least twice a month; only mycoplasma-free cells were used for further experiments.

Reagents: Lipofectamine 3000 was obtained from Invitrogen (Carlsbad, CA, USA). Cell Counting Kit-8 (HY-K0301), DAPI (D9542), Lenvatinib (E7080); and FGFR2 inhibitor Alofanib (HY-17601) were purchased from Merck or MedChemExpress. TUNEL FITC Apoptosis Detection Kit (A111-02) was purchase from Vazyme (Nanjing, China). RIPA lysis buffer, puromycin and hygromycin were purchased from Merck/Millipore (Darmstadt, Germany). *InVivo*MAb anti-mouse/human/rat CCL2 (BE0185) and RecombiMAb anti-mouse PD-1 (CP162) and anti-human PD-1 (#BE0193) were purchased from BioXCell (Lebanon, NH, USA). And Monoclonal Rat anti-Mouse CCL20/MIP-3-Alpha Antibody (clone 4N5F7, LS-C745268-MSS10-25) was purchased from LSBio (Seattle, WA, USA).

The primary antibodies used were as follows: mouse monoclonal antibodies against HA, Flag, GST (700775, 1:5000 for WB), His (700158, 1:5000 for WB), β-actin (a5316) and tubulin (ab3201) were purchased from Sigma (St. Louis, MO, USA). Rabbit polyclonal antibodies against PIGL (NBP1-86471, 1:1000 for WB, 1: 50 for IHC), Ki-67 Antibody (MA5-14520, 1:200 for IHC), cMyc (MA1-980, 1:2000 for WB, 1:100 for ChIP), RelB (PA5-27679, 1:2000 for WB), BRD4 (PA5-85662, 1:3000 for WB, 1:200 for ChIP or IP), H3K9ac (MA5-11195, 1:200 for WB), H3K14ac (MA5-24668, 1:200 for WB), H3K27ac (MA5-23516, 1:200 for ChIP), FGFR2 (MA5-32629, 1:4000 for WB), Importin α1/KPNA2 (MA5-17016, 1:1000 for WB) and Importin β1 (MA3-070, 1:2000 for WB) were purchased from Thermo Fisher Scientific (Shanghai, China) or Novus (Shanghai, China). Antibody against phosphorylation of PIGL-Y81 was made in Abclonal (Wuhan, China). Lamin B1 (D4Q4Z) Rabbit mAb (12586S) was from Cell Signaling Technology.

The secondary antibodies goat anti-mouse IgG HRP (G-21040) and goat anti-rabbit HRP (G-21234) were purchased from Thermo Fisher Scientific (Shanghai, China). Goat anti-rabbit Alexa Fluor® 488 (ab150077) and goat anti-mouse Alexa Fluor® 555 (ab150118) were purchased from Abcam (Britain).

IP and immunoblotting (IB) analyses were performed as follows. Notably, antibodies for IB were diluted 1:1000-1:5000; for IP or chromatin IP, we used 2-10 μg for each sample; for IF or IHC, the antibodies were diluted 1:100 or 1:200. Extraction of total proteins using a modified RIPA lysis buffer was followed by IP and IB using the indicated antibodies. Protein levels were quantified using a Pierce™ BCA Protein Assay Kit (23228, Thermo Fisher Scientific).
siRNAs targeting mice ALK (AM16708), FGFR2 (4392420), LYN (4390824), PDGFRB (1299001) and SYK (4392420) were purchased from Thermo Fisher Scientific (Shanghai, China).

Transfection

The cells were planted in 60mm or 100mm petri dishes and transfected when the cell density is approximately 70%. Plasmids were transfected into indicated cells using the Lipo3000TM transfection reagent (Life Technologies, USA) while siRNA were transfected by Lipofectamine™ RNAiMAX Transfection Reagent (Life Technologies, USA). The ratio of plasmid to transfection reagent is 1:3 (m/m).

Lentivirus packaging and infection

The lentivirus was packaged in HEK293T cells that transfected using polyethylenimine with psPAX2 (addgene: #12260), pMDG.2 (addgene: #12259) and the target plasmids at a ratio of 3:1:4. All plasmids used for transfection were endotoxin-free. The 48hrs and 72hrs medium were collected, filtered and concentrated (7,5000g, 120 min, 4 °C). Hepa1-6 or Hep3B cells were infected for 12-16 hours with lentivirus in the presence of polybrene and the overexpression or knockdown efficiency was evaluated by western blotting. The constructed stable cell lines were amplified and saved for future experiments.

Western Blot Analysis

The cells were lysed with RIPA buffer and the protein concentration was measured by using the BCA assay. Equal amounts of protein were subjected to 10% SDS-PAGE for electrophoresis and transferred to a polyvinylidene difluoride membrane (Millipore, Billerica, MA). The membrane was blocked with 5% fat-free milk at room temperature for 1 hour and then incubated with indicated primary antibodies overnight at 4°C. HRP conjugated secondary antibodies were added and incubated for 2 hrs at room temperature. Immunoreactive bands were visualized with enhanced chemiluminescence (Thermo Fisher Scientific, Waltham, MA) by using the LAS 4000 Imaging system (Fujifilm, Tokyo, Japan).

RNA extraction and Quantitative RT-PCR (qRT-PCR)

Total RNA was extracted with an RNA high-purity total RNA rapid extraction kit (QIAGEN). cDNA was prepared using a GoScript reverse transcription system (Promega). qRT-PCR was performed using SYBR green PCR premixture (Promega) under the following conditions: 5 mins at 95^0C followed by 38 cycles at 95^0C for 30 s, 60^0C for 40 s, and 72^0C for 1 min using an ABI 7500 fast system. Data were analyzed using the $2^{(-\Delta\Delta Ct)}$ method and normalized to the expression of the control gene (*ACTB*) for each experiment. Data represent the mean ± SD of three independent experiments. The sequences of primer pairs used for qRT-PCR were listed in the Supplementary Table 1.

Cell viability Assay

Cell counting kit-8 (CCK8) assay was used to determine the cell viability. Briefly, cells in logarithmic growth phase were inoculated into 96 well plates (8×10^3 cells/150 μl). After drug treatment, cells were incubated with 10% (v/v) CCK-8 reagent (Dojindo, Japan) at 37 °C for 1 h, and the absorbance was measured at 450 nm using a microplate reader (BioTek, Vermont, USA) at the indicated time points (0, 3, 6, 12 or 24 hours).

Plasmids construction and Mutagenesis

DNA constructs: cDNAs of mouse or human PIGL, cMyc and BRD4 were amplified by PCR from total RNAs of Hepa1-6 cells or Hep3B cells. The total RNAs were extracted and isolated using Trizol reagent (Invitrogen, Carlsbad, CA, USA). Reverse transcription of RNAs was performed using the reverse transcription kit (Promega, Beijing, CHN). pCDH-3'SFB, pCDH-3'flag, pET28a-5'His or pET28a-3'GST was used as a backbone. Constructions were performed using a ClonExpress® Ultra One Step Cloning Kit according to the manual (C115, Vazyme, Nanjing, China). PIGL L167P mutation contains T500C and G501T was generated using the QuikChange site-directed mutagenesis kit (Stratagene, La Jolla, CA, USA). The reporter plasmids of p53, c-Myc, HIF1a, RelA and RelB were kindly given by Weiwei Yang. shRNA constructs were generated using a pGIPZ backbone digested by XhoI and EcoRI and target sequences were inserted using the T4 DNA Ligase (M0202V, NEB). All the PCR primers and target sequences are shown in the supplementary table1.

Immunohistochemical (IHC) analyses

Tissue sections from paraffin-embedded human HCC patients' specimens were stained with antibodies against PIGL, pPIGL-Y81 or indicated antibodies. We quantitatively scored the tissue

sections according to the percentage of positive cells and staining intensity. We rated the intensity of staining on a scale of 0-3 points: 0, negative; 1, weak; 2, moderate; and 3, strong. We assigned the following proportion scores: X indicates that X% of the tumour cells were stained ($0 \leq X \leq 100$). The score (H-score) was obtained using the following formula: 3 × the percentage of strongly stained signal + 2 × the percentage of moderately stained signal + 1 × the percentage of weakly stained signal, with a range of 0 to 300 points. Scores were compared with overall survival, which was defined as the time from the date of diagnosis to death or the last known date of follow-up. We compared the survival durations of 40 HCC patients in Figure 2F and 112 HCC patients in Figure 7D, all of whom received appropriate therapy, with low (0–125 staining) versus high (125.1–300 staining) total/nuclei PIGL or PIGL Y81 phosphorylation levels.

Transwell Migration Assay for Treg cells

The $CD4^+CD25^+$ Treg cells were isolated and sorted from mouse splenocytes by Fluorescence-activated Cell Sorting (FACS). THP1 cells were cultured in a 60 mm petri dish. Cells were counted using a hemocytometer with typan blue staining. Transwell upper chambers were placed in a 24 well plate, and 50,000 cell suspensions were seeded in the upper chamber with 3-μm pores (#3402, Corning, USA). 600 μl of Hepa1-6 cell supernatant was added in the bottom receiving cell. After 36 hrs of culture in the incubator, the upper chamber removed and fixed in 4% paraformaldehyde solution for 30 mins and stained with crystal violet at room temperature for 10 mins. Then the upper layer of cells that did not pass through the ependyma were gently wiped off with cotton swabs and washed gently with PBS. Under the microscope, five high power fields were randomly selected to count the number of cells passing through the membrane, and the average value was taken as the number of cells passing through the chamber in each group, so as to evaluate the migration ability of cells. For Treg cells migration rate quantification, we also enumerated the cells in the bottom receiving wells respectively from three replicates.

Co-Immunoprecipitation

Anti-Flag M2 affinity gel (#a2220, Merk, USA) was used to incubate with flag-tagged cell lysate (cytosol or nuclei) at 4 °C for 4 hrs. Then the beads were washed with protein lysis buffer and resuspended in 40 μl of 1×loading buffer. Next, purified flag-tagged protein and its associated complexes were boiled in 95°C for 8 mins. The proteins were then used for immunoblot detection with indicated antibodies.

Chromatin immune-precipitation (ChIP) assay

For ChIP experiments, cells were fixed in 1% formaldehyde for 10 min for pull-down of cMyc and H3K27ac. The cells were rotated in cold lysis buffer (10 mM Tris-HCl pH 7.4, 10 mM NaCl, 3 mM MgCl2, 0.5% NP40, supplemented with freshly prepared PMSF and protease inhibitor cocktail) for 30 min. Then, the nuclear pellets were resuspended in RIPA buffer (300 mM NaCl, 3 mM EDTA, 1% NP40, 0.5% sodium deoxycholate, 0.1% SDS, 100 μg/ml BSA, 50 mM Tris-HCl pH 7.5) and sonicated with a Covaris S220 to yield DNA fragments of approximately 200–500 bp. A ChIP-grade antibody was incubated with 30 μl of protein A/G Dynabeads (10003D, Thermo Fisher) for 4 hours. Then, the DNA fragments were co-immunoprecipitated with the specific antibody-conjugated protein A/G beads at 4°C overnight. The DNA was purified with a MinElute PCR Purification Kit (QIAGEN, 28004). The promoter or enhancer regions of CCL2 and CCL20 were amplified and quantified by qRT-PCR using SYBR Green Mix (Takara, RR820A) on an ABI 7500 Fast system. For PCR, 0.02 ng of the immunoprecipitated DNA and 2 ng of the total DNA were used in a 20 μl reaction. The results from each immunoprecipitation were normalized to the respective inputs. The primers are listed in Supplemental Table 1.

TCGA, GTEx and CPTAC Database analysis

For the gene expression analysis, TCGA datasets of patients with liver cancer (TCGA-LIHC) and GTEx normal samples were used (https://xenabrowser.net/datapages/). To compare PIGL mRNA expression difference between tumour and normal tissues, the TCGA-BLCA datasets and the GTEx dataset were combined and batch biases were corrected and data were normalized before performing the unpaired two-tailed Student t-test. The Kaplan-Meier plot was done based on PIGL mRNA expression and clinical data. Patients were divided into high (n = 237, 65%) and low (n = 128, 35%) groups according to PIGL mRNA expression in the primary tumours and the Log-rank test was used for comparing the survival distributions of two groups. PIGL protein expression was analyzed using the CPTAC-HCC cohort[1], the normalized data was extracted and compared using Student t-test.

Supplementary Table 1

Primers for constructs：

UNI-CMV-musPIGL-F
GTGGAATTCGCTAGCGGATCC atggaattggtgggtttctt
UNI-CMV-musPIGL-R
ATGTCGACCTCGAGTGCGGCCGC tcagaggaaccgcagtgagt
UNI-CMV-homoPIGL-F
GTGGAATTCGCTAGCGGATCC atggaagcaatgtggctcctg
UNI-CMV-homoPIGL-R
ATGTCGACCTCGAGTGCGGCCGC tcagaggaagctcagtgagtt

Sequence for knocking down mus musculus PIGL: #1, 5'-TGTCAATAATCATTACACG; #2, 5'-TTGTGGTAAAGACAACTTC
Sequence for knocking down homo sapiens PIGL: #1, 5'-AATCATTACACTGGAGAGT; #2, 5'-TTAGGTAACTTCCCTTCTG
Sequence for shRNA control: 5’- CTCGCTTGGGCGAGAGTAA-3’.

Sequences of Primers for qRT-PCR

Mus-PIGL-F 5'- CCGGACGATGAAGCCATGTT
Mus-PIGL-R 5'- CCGGAAAGTCCCTCTTGTCAATA

Mus-CCL2-F 5’- TAAAAACCTGGATCGGAACCAAA
Mus-CCL2-R 5’- GCATTAGCTTCAGATTTACGGGT

Mus-CCL20-F5’- ACTGTTGCCTCTCGTACATACA
Mus-CCL20-R 5’- GAGGAGGTTCACAGCCCTTTT

Mus-ALK-F 5’- GGAGCCGCTCAGTTATTCG
Mus-ALK-R 5’- AGCAGGTCTCGGGCATAGA

Mus-FGFR2-F 5’- GCCTCTCGAACAGTATTCTCCT
Mus-FGFR2-R 5’- ACAGGGTTCATAAGGCATGGG

Mus-PDGFRB-F 5’- AGGAGTGATACCAGCTTTAGTCC
Mus-PDGFRB-R 5’- CCGAGCAGGTCAGAACAAAGG

Mus-LYN-F 5’- ATCCAACGTCCAATAAACAGCA
Mus-LYN-R 5’- ATAAGGCCACCACAATGTCAC

Mus-SYK-F 5’- CTACCTGCTACGCCAGAGC
Mus-SYK-R 5’- TTCCCTCTCGATGGTGTAGTG

Mus-ACTB-F 5’-TGGACTCTGTTCGCTCAGGT
Mus-ACTB-R 5’TGCCTCCTTCCGTACCACAT

Sequences of Primers for ChIP-qPCR

Mus-CCL2-1-F 5'- GACTTACTGGGGGTCCTTTC
Mus-CCL2-1-R 5'- CTGCATGGTGGTGGAGGAAG

Mus-CCL2-2-F 5’- AGGGCAGAGAGCTACCAGGA
Mus-CCL2-2-R 5’- GAAAGGACCCCCAGTAAGTC

Mus-CCL2-3-F 5’- GTGTTACCTATGGGTAATTA
Mus-CCL2-3-R 5’- TCCTGGTAGCTCTCTGCCCT

Mus-CCL2-4-F 5’- GAGCTGTGGGCATCTATGAA
Mus-CCL2-4-R 5’- TAATTACCCATAGGTAACAC

Mus-CCL2-5-F 5’- GCATCTGGAGCTCACATTCC
Mus-CCL2-5-R 5’- TTCATAGATGCCCACAGCTC

Mus-CCL2-6-F 5’- GGAGGATGACCAGGGACCAA
Mus-CCL2-6-R 5’- GGAATGTGAGCTCCAGATGC

Mus-CCL2-7-F 5’- TTTCATGAAAATGGAGCTGG
Mus-CCL2-7-R 5’- TTGGTCCCTGGTCATCCTCC

Mus-CCL2-8-F 5’- CACGAATTTCCCTTGGATGA
Mus-CCL2-8-R 5’- CCAGCTCCATTTTCATGAAA

Mus-CCL20-1-F 5'- ACATCCTGAGGAATTAACAG
Mus-CCL20-1-R 5'- GAGGACAGAAGTCTCCTGTA

Mus-CCL20-2-F 5’- GACACCTCTGTCATGTTGTT
Mus-CCL20-2-R 5’- CTGTTAATTCCTCAGGATGT

Mus-CCL20-3-F 5’- GATACAGTTGCTTGGTGTGTA
Mus-CCL20-3-R 5’- AACAACATGACAGAGGTGTC

Mus-CCL20-4-F 5’- GTGGGGCAGAGCCTAGAAAGA
Mus-CCL20-4-R 5’- TACACACCAAGCAACTGTATC

Mus-CCL20-5-F 5’- GGTAAGTTTATTGCTTAGCA
Mus-CCL20-5-R 5’- TCTTTCTAGGCTCTGCCCCAC

Mus-CCL20-6-F 5’- GTCGGTCAACACTGTACTCA
Mus-CCL20-6-R 5’- TGCTAAGCAATAAACTTACC

Mus-CCL20-7-F 5'- TAAATGGAATTGTGTGTGTG
Mus-CCL20-7-R 5'- ATACCCAGTAATGGGATTGC

Mus-CCL20-8-F 5'- TATTCTTTTTGATGGCATTGT
Mus-CCL20-8-R 5'- CACACACACAATTCCATTTA

Reference

1 Gao, Q. *et al.* Integrated Proteogenomic Characterization of HBV-Related Hepatocellular Carcinoma. *Cell* **179**, 561-577 e522, doi:10.1016/j.cell.2019.08.052 (2019).