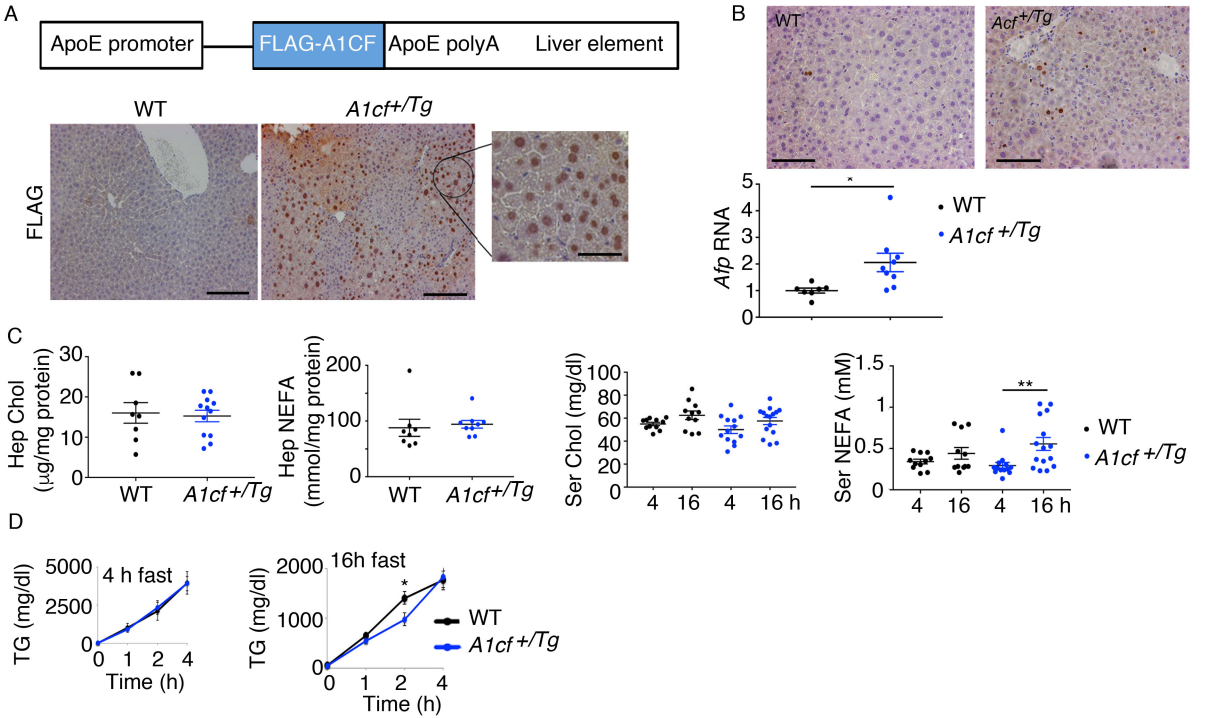
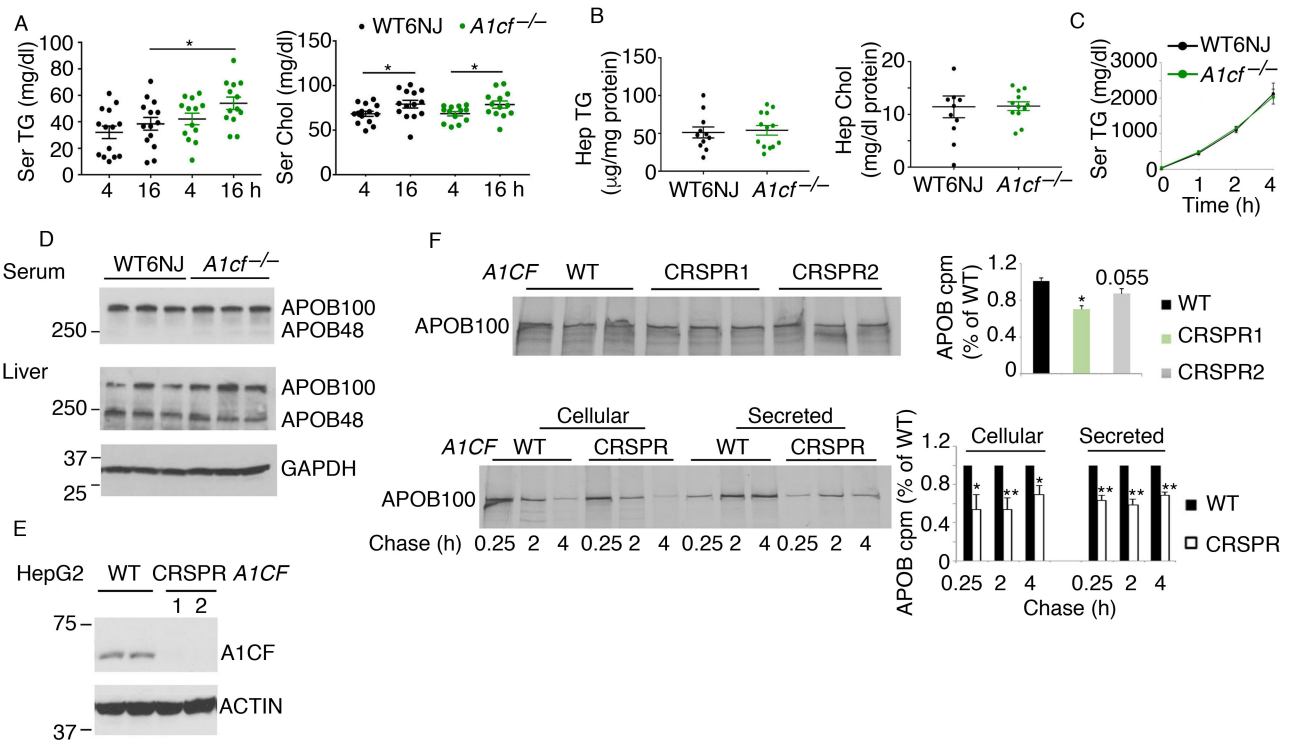


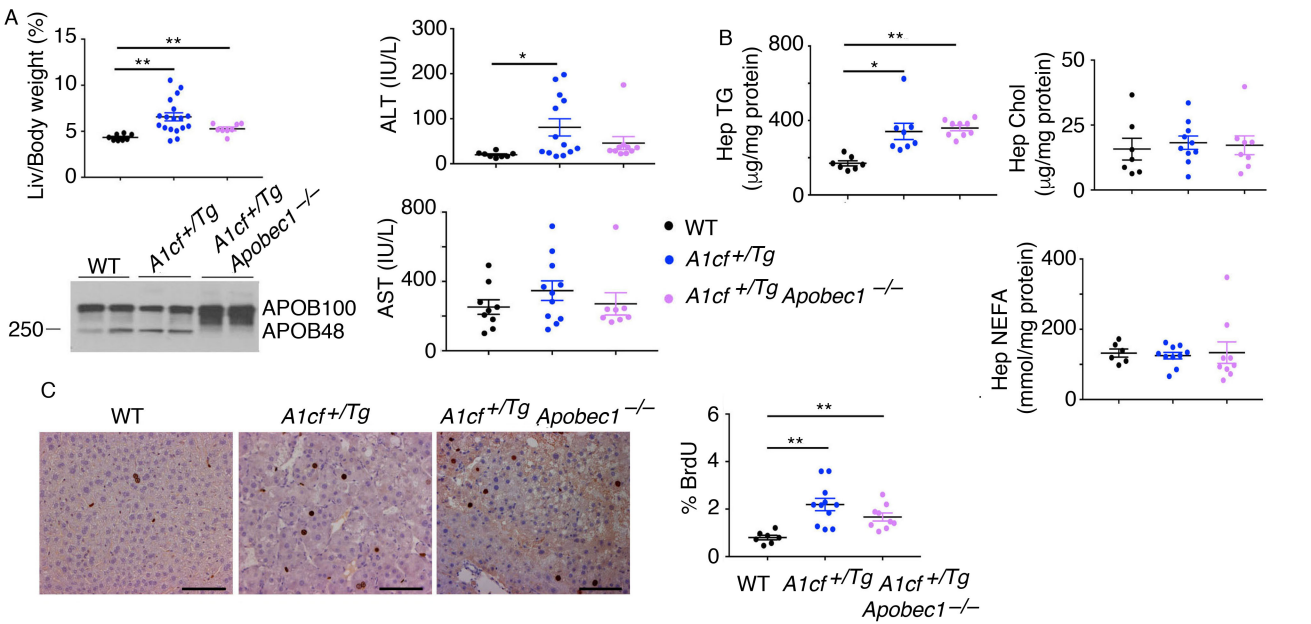
Supplemental Figure 1

Supplemental Figure 1. Baseline characterization of 12-week-old chow fed *A1cf* $^{+/Tg}$ mice. (**A**) Diagram of the ApoE-A1cf transgenic construct. *A1cf* transgene is expressed as a N-terminal FLAG-tagged protein. A1CF transgene is detectable by immunohistochemistry using anti-FLAG antibody and appears highly expressed in nuclear compartment of hepatocytes. Scale bars: main panel: 100 μm; inset: 50 μm. (**B**) Representative images of proliferative hepatocytes labeled with BrdU in *A1cf* $^{+/Tg}$ mice (related to Figure 1B) Scale bars: 50 μm. Hepatic *Afp* RNA expression in *A1cf* $^{+/Tg}$ mice evaluated by QPCR (n=7-9 animals per genotype). Data are mean ± SE. * $P < 0.05$ (unpaired t-test). (**C**) Hepatic cholesterol and NEFA content were determined biochemically and normalized to hepatic protein. Values are means ± SE from 8-12 mice per genotype. Biochemical determination of serum cholesterol and NEFA in chow-fed *A1cf* $^{+/Tg}$ mice after a 4 or 16 h fast; n=11-15 animals per genotype and fasting condition. Data are means ± SE, ** $P < 0.01$ (unpaired t-test). (**D**) Plasma VLDL-TG secretion rate in *A1cf* $^{+/Tg}$ mice fasted for 4 or 16 h and injected with Pluronic F-127. Each point represents mean ± SE (n=2-6 mice per genotype) * $P < 0.05$ (unpaired t-test).



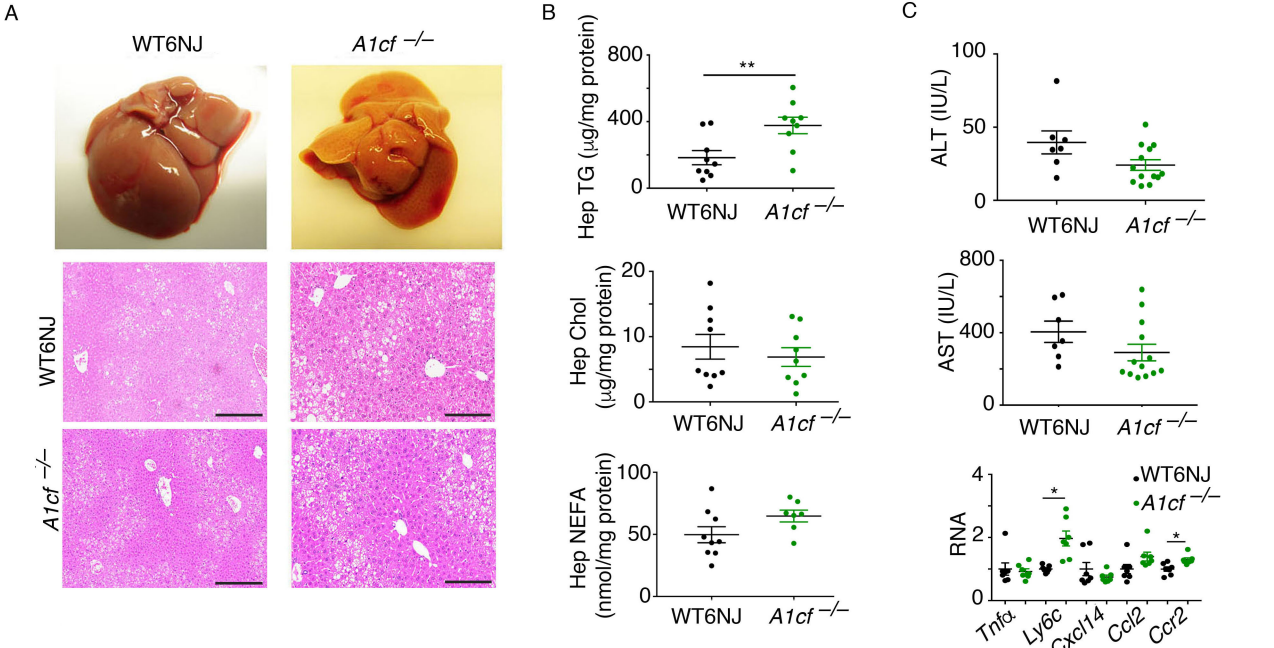
Supplemental Figure 2

Supplemental Figure 2. *A1cf* $^{-/-}$ mice show no alteration of APOB and VLDL secretion. (**A**) Serum TG and cholesterol in chow-fed *A1cf* $^{-/-}$ mice after a 4 or 16 h fast, n=13-14 animals per genotype and fasting condition. Data are means ± SE, * $P < 0.05$ (unpaired t-test). (**B**) Hepatic TG and cholesterol contents normalized to hepatic protein. Values are means ± SE from 11-13 mice per genotype. (**C**) Serum TG secretion rate in *A1cf* $^{-/-}$ mice following Pluronic F-127 injection after an overnight fast. Each point represents mean ± SE (n=6 per genotype). (**D**) Western blot analysis of serum and hepatic APOB after a 4h fast. GAPDH was used as loading control. (**E**) Western blot validation of A1CF knockout in two separate HepG2 CRSPR *A1CF* clones. ACTIN was used as loading control. (**F**) APOB synthesis in 2 separate HepG2 CRISPR *A1CF* clones. Following 15 min incubation with ^{35}S-methionine, extracts were separated on a 4-15% SDS-PAGE. After autoradiography radiolabeled APOB was quantitated by determination of count (cpm) in APOB protein band and expressed as % of WT CPM. Data represent mean ± SE (n=3 per clones). Cellular and secreted APOB was evaluated after pulse chase analysis. Autoradiograph is a representative image of two independent experiments, each performed with two separate HepG2 CRSPR *A1CF* clones. Quantitation of cellular and secreted APOB100 is shown in the graphical representation. Values for the HepG2 CRSPR *A1CF* represent mean ± SE (2 separate clones/ 2 independent experiments) * $P < 0.05$; ** $P < 0.01$ (unpaired t-test).



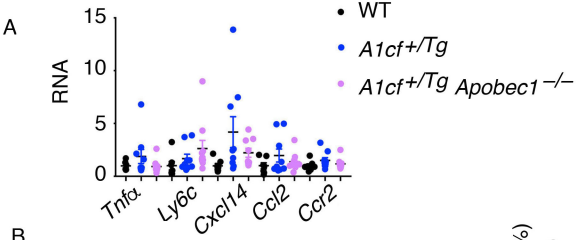
Supplemental Figure 3

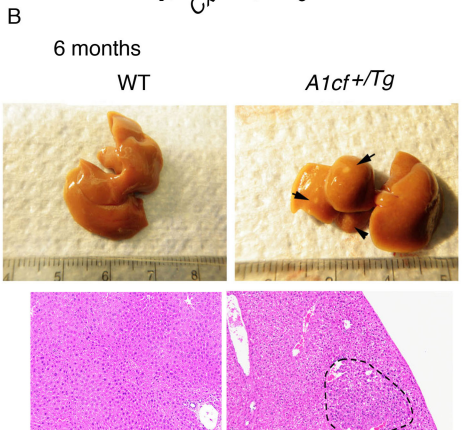
Supplemental Figure 3. Characterization of chow-fed 12 month-old *A1cf* $^{+/Tg}$ mice. (**A**) Liver to body weight ratio of aged *A1cf* $^{+/Tg}$ (n=14) and *A1cf* $^{+/Tg}$ *Apobec1*$^{-/-}$ (n=10), represented as mean ± SE, ** $P < 0.01$ (Unpaired t-test for individual comparison with WT control). Significance between means of the 3 groups was determined using one-way ANOVA, P value = 0.0021). Plasma ALT and AST in aged *A1cf* $^{+/Tg}$ and *A1cf* $^{+/Tg}$ *Apobec1* $^{-/-}$ mice (n=7-13) as mean ± SE * $P < 0.05$ (Unpaired t-test). Significance between the 3 groups was determined using one-way ANOVA, P value = 0.0425. Lower panel, Western blot of APOB isoforms (B100 and B48) in serum of aged *A1cf* $^{+/Tg}$ and *A1cf* $^{+/Tg}$ *Apobec1* $^{-/-}$ mice. (**B**) Hepatic lipid in aged mice (n=7-10) represented as mean ± SE, * $P < 0.05$; ** $P < 0.01$ (Unpaired t-test for individual comparison with WT). Significance between the 3 groups was evaluated using one-way ANOVA, $P = 0.0002$). (**C**) Hepatocytes stained with BrdU in aged *A1cf* $^{+/Tg}$ and *A1cf* $^{+/Tg}$ *Apobec1* $^{-/-}$ livers and proliferative index expressed as percent of BrdU-positive hepatocytes. Data represent means ± SE (n=7-10) ** $P < 0.01$ (unpaired t-test for individual comparison). One-way ANOVA was used to determined significance between all groups, $P = 0.0006$). Scale bars: 50 μm.

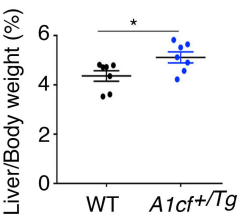


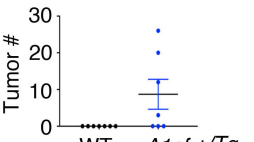
Supplemental Figure 4

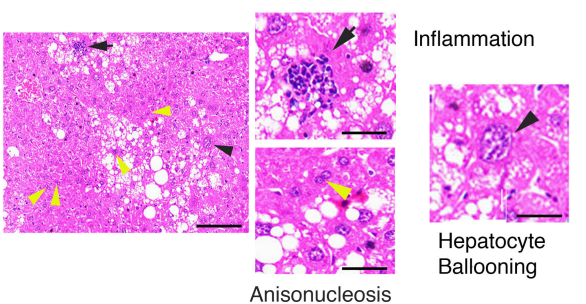
Supplemental Figure 4. Chow-fed aged *A1cf* $^{-/-}$ do not develop spontaneous tumors. (**A**) Liver gross views and H&E sections from aged *A1cf* $^{-/-}$ mice. Scale bars: 100 μm (left) and 50 μm (right). (**B**) Hepatic lipid represented as mean ± SE, ** $P < 0.01$ (n=9) (Unpaired t-test). (**C**) Plasma ALT and AST in aged *A1cf* $^{-/-}$ mice shown as mean ± SE (n=7-13). QPCR determination of Inflammatory markers RNA expression in aged *A1cf* $^{-/-}$ liver represented as mean ± SE, * $P < 0.05$ (n=7) (Unpaired t-test).





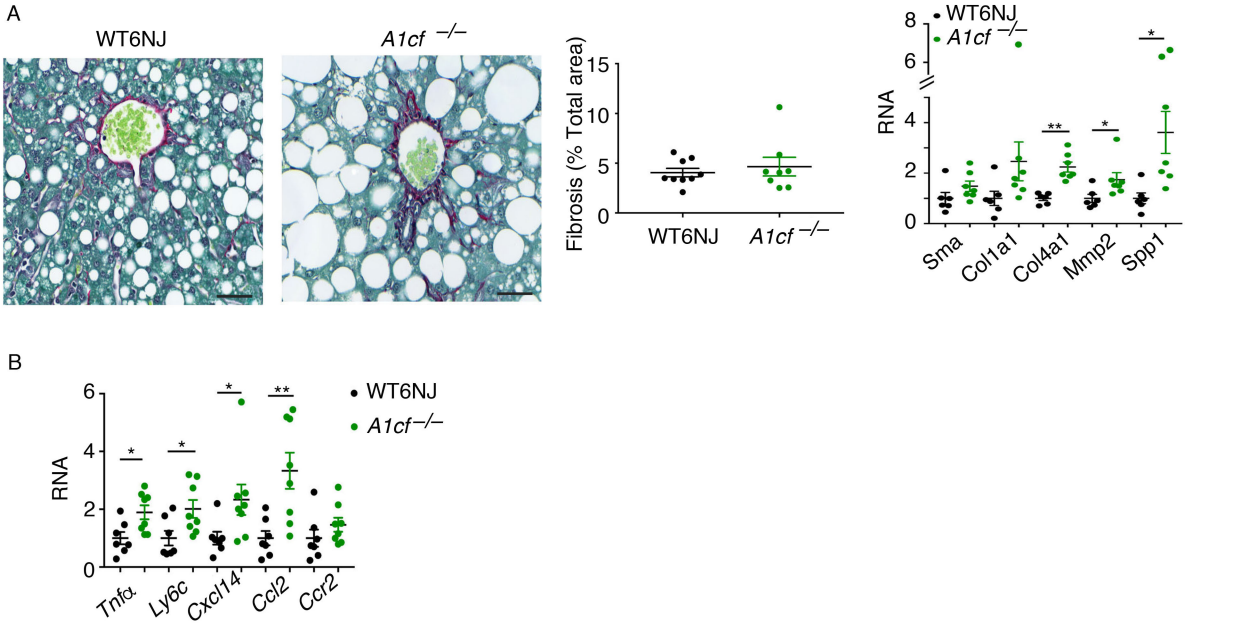






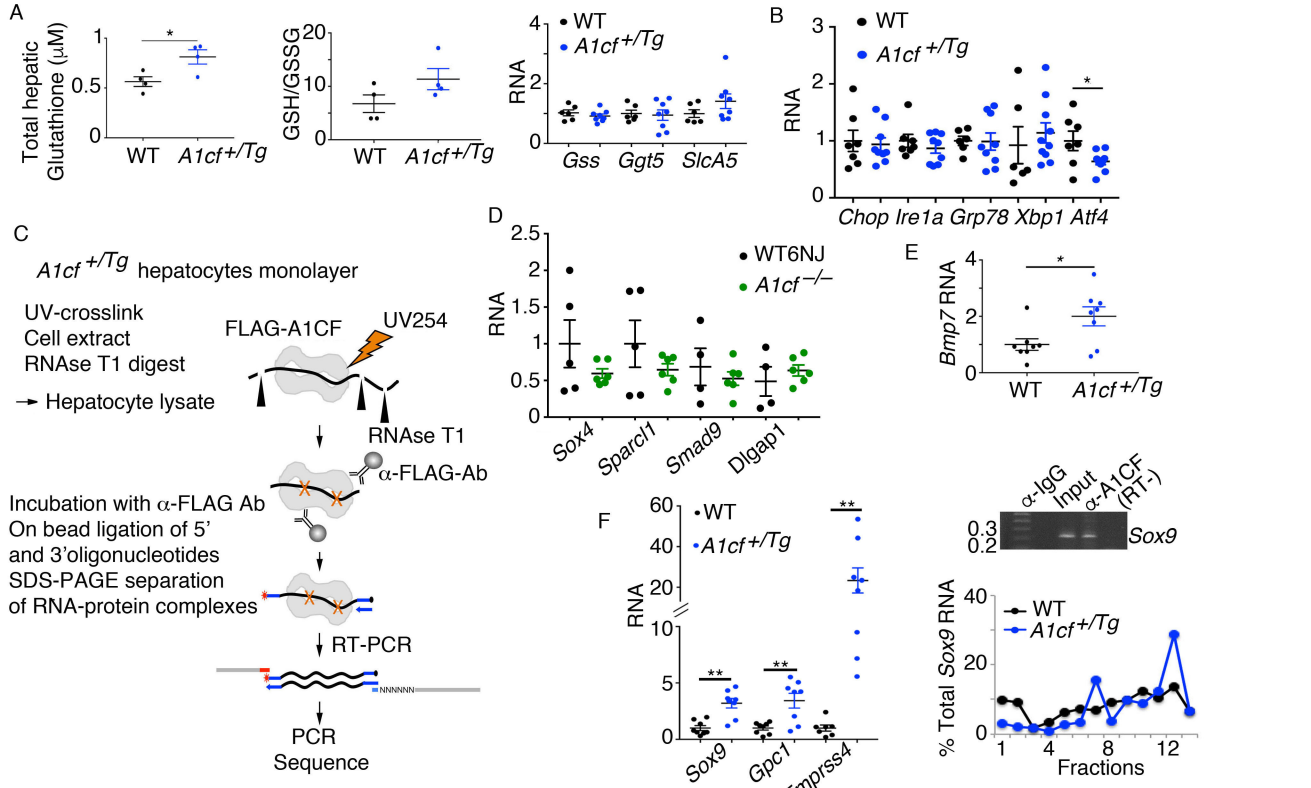
Supplemental Figure 5

Supplemental Figure 5. (**A**) QPCR evaluation of Inflammatory markers RNA expression in aged *A1cf* $^{+/Tg}$ and *A1cf* $^{+/Tg}$ *Apobec1* $^{-/-}$ (n=7) as mean ± SE. (**B**) Livers from 6-month-old chow-fed *A1cf* $^{+/Tg}$ and H&E staining of liver sections. The arrows indicate macroscopic tumors (dashed area). Scale bars: 100 μm. Liver to whole body weight ratio as mean ± SE (n=7) * $P < 0.05$ (Unpaired t-test). Tumor burden evaluated as number of macroscopically detectable tumors. Histological features of livers from 6-month-old chow-fed *A1cf* $^{+/Tg}$ mice. Black arrowheads: focal inflammation and hepatocyte ballooning. Yellow arrowheads: anisonucleosis. Scale bars: 50 μm.



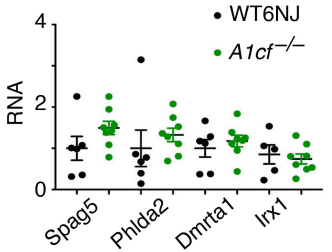
Supplemental Figure 6

Supplemental Figure 6. TFF-fed *A1cf* $^{-/-}$ mice exhibit similar fibrotic injury to WT controls but activation of inflammatory response. (**A**) Representative images of Sirius red staining of livers from *A1cf* $^{-/-}$ mice fed a TFF diet for 6 months. Scale bars: 20 μm. Sirius red-positive area was expressed as percent total area (n=8-9 animals per genotype) and represented as mean ± SE (n=8-9). Relative expression of fibrogenic genes evaluated by QPCR. Data represent mean ± SE (n=6-7 per genotype). Unpaired t-test was used to determine significance between *A1cf* $^{-/-}$ and WT6NJ RNA samples. * $P < 0.05$, ** $P < 0.01$. (**B**) Expression of inflammatory markers determined by QPCR. Data represent mean ± SE (n=6 -8 per genotype), * $P < 0.05$; ** $P < 0.01$ (Unpaired t-test).



Supplemental Figure 7

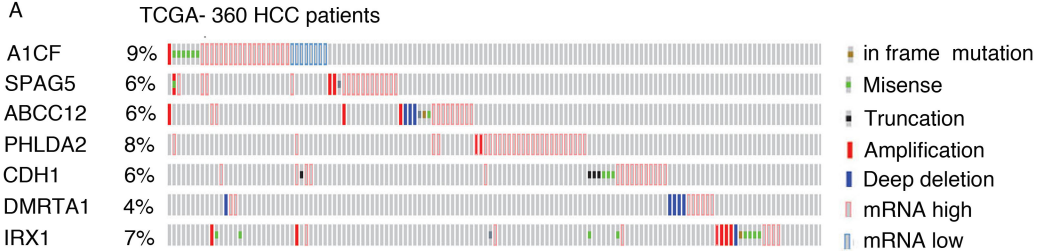
Supplemental Figure 7. Oxidative stress characterization of young *A1cf* $^{+/Tg}$ mice. (**A**) Hepatic glutathione levels in WT and *A1cf* $^{+/Tg}$ liver. Levels of glutathione are represented both as total glutathione and as reduced to oxidized glutathione ratio. Data represent mean ± SE (n=4); * P< 0.05 (unpaired t-test). QPCR expression of RNAs involved in glutathione synthesis (*Gss*, *Slc1A5*) and transport (*Ggt5*) (6-8 animals per genotype). Data represent mean ± SE (unpaired t-test). (**B**) QPCR expression of ER-stress response related genes (6-9 animals per genotype). Data are mean ± SE, * P < 0.05 (unpaired t-test). (**C**) Schematic of RNA-CLIP method as described in Methods. (**D**) QPCR expression of A1CF RNA targets differentially expressed in 12-week-old *A1cf* $^{+/Tg}$ liver (see figure 9) in age-matched *A1cf* $^{-/-}$ livers. Data represent mean ± SE, (n= 5-6). (**E**) QPCR expression of *Bmp7* RNA in liver from young *A1cf* $^{+/Tg}$ mice, represented as mean ± SE (n=8); * P < 0.05 (unpaired t-test). (**F**) Left, Increased expression of RNAs involved in organogenesis in 12-week-old chow-fed *A1cf* $^{+/Tg}$ liver. QPCR from 7-8 animals per genotype. Data represent mean ± SE, * P < 0.05; ** P < 0.01 (unpaired t-test). Right top panel, representative image of 3 independent co-immunoprecipitations (IP) of A1CF and *Sox9* RNA from whole liver extract. Control immunoprecipitation with IgG was performed to confirm the specificity of anti-A1CF antibody. *Sox9* RNA was detected only in input fraction (total RNA before IP) and in the A1CF-specific IP. Right lower panel, polysomal distribution of *Sox9* RNA from sucrose gradient-fractionated hepatic cytoplasmic extracts isolated from *A1cf* $^{+/Tg}$. *Sox9* RNA in each fraction was analyzed by QPCR. Data are means of 2 separate isolations per genotype.



Supplemental Figure 8

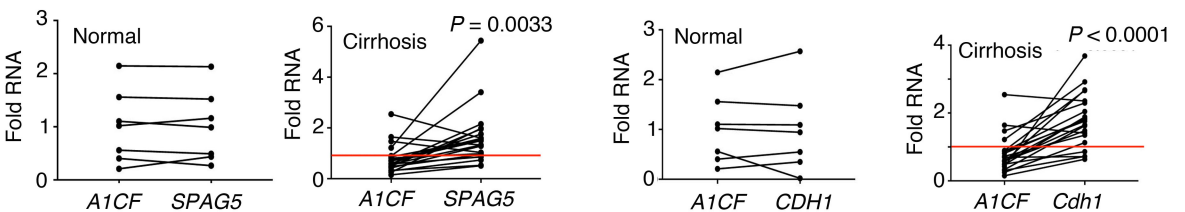
Supplemental Figure 8. Gain/loss of function effect on differentially expressed A1CF RNA targets identified in 12 month-old *A1cf* $^{+/Tg}$ mice. Expression of A1CF RNA targets identified by RNA-Seq in aged *A1cf* $^{+/Tg}$ mice (Figure 12) was compared by QPCR in the aged *A1cf* $^{-/-}$ liver. Data represent mean ± SE, n= 5-8 animals per genotype (unpaired t-test).

A



A1CF vs SPAG5 5/360 co-occurence p-value 0.031
A1CF vs CDH1 5/360 co-occurence p-value 0.054

B



Supplemental Figure 9

Supplemental Figure 9. Expression of A1CF RNA targets in human HCC. (**A**) Oncoprint representing distribution of genetic alterations of A1CF RNA targets differentially expressed in 12-month-old *A1cf* $^{+/Tg}$ mice in 366 patients with liver hepatocellular carcinoma (HCC) from TCGA database (cbioportal.org). Genetic alterations are indicated to the right. The percentage associated with each gene indicates the number of patients with a genetic alteration of the given gene over the total number of profiled patients. Below the oncoprint are indicated two A1CF RNA-CLIP targets (*SPAG5*, *CDH1*) whose genetic alteration significantly co-occurs with A1CF alteration. (**B**) Comparison of expression of *A1CF*, *SPAG5* and *CDH1* RNAs in 21 cirrhotic human liver samples. Expression of individual RNAs by QPCR was evaluated in relation to 7 normal human livers. Fold expression in normal tissue is indicated as 1 (red line). Fold expression of *SPAG5* and *CDH1* RNAs was then separately compared to *A1CF* RNA among categories. *P* values are indicated on top of each graph (unpaired t-test).

Table 1. Pathological analysis of 12 month-old *A1cf* $^{+/Tg}$ livers.

	WT	*A1cf* $^{+/Tg}$	*A1cf* $^{+/Tg}$ *Apobec1*$^{-/-}$	WT6NJ	*A1cf* $^{-/-}$
# Animals	7	13	10	7	10
No lesion	7	1	0	7	10
Non dysplastic lesions	0	1	5	0	0
Dysplastic lesions	0	7	4	0	0
HCC	0	4	1	0	0

Table 2. Pathological analysis of livers of 6 month-old TFF fed *A1cf* $^{+/Tg}$.

	WT	*A1cf* $^{+/Tg}$	WT6NJ	*A1cf* $^{-/-}$
Animals	8	14	9	12
No lesion	8	5	9	12
Non Dysplastic lesions	0	2	0	0
Dysplastic lesions	0	7	0	0

Table 3. Distribution of patients by A1CF expression category in uninvolved and tumor tissue.

A1CF expression	Number of patients	
Category	Uninvolved	Tumor
1	39	36
2	45	36
3	23	30
4	20	35
Total	127	137

Supplemental Table S1. 3'UTR sequences of A1CF RNA targets identified by RNA-CLIP and differentially expressed in *A1cf* $^{+/Tg}$ mice.

Gene	Length (nt)	A	U	A and U	% AU	AUUUA	Poly(U) (5U)	Poly(U) > 6U
Sox4	2807	819	858	1677	59.74	4	>20	14 (+)
Sox9	2247	644	788	1432	63.73	6	>20	13 (+)
Sparcl1	441	151	138	289	65.53	0	0	0
Dlgap1	3179	976	920	1896	59.64	12	11 (+)	4 (+)
Smad9	3767	1105	1038	2143	56.9	8	9 (+)	3 (+)
Dram1	1937	536	557	1093	56.43	6	6 (+)	2 (+)
Phlda2	268	57	73	130	48.51	1	2 (+)	2 (+)
Cdh1	1647	446	490	936	56.83	2	8 (+)	4 (+)
Irx1	402	108	113	221	54.9	0	2 (+)	1 (+)
Dmrta1	2490	827	813	1640	65.9	6	10 (+)	1 (+)
Abcc12	377	95	85	180	47.74	0	0	0
Spag5	258	83	67	150	58.14	2	0	0

(+) represents one poly(U) motif with the indicated number of uridines

Supplemental Table S2. Clinical and laboratory data for all hepatocellular cancer patients.

Parameter	**N (%) / mean (±SD)**
Gender	
Female	51 (37%)
Male	86 (63%)
Age at diagnosis (year)	62.70 (±14.3)
Body mass index (kg/m^2)	27.71 (±5.7)
Obese (body mass index >30)	
No	91 (72%)
Yes	35 (28%)
Race	
Caucasian	96 (71%)
African American	4 (3%)
Asian	8 (6%)
Hispanic	2 (1.5%)
Unknown	25 (18.5%)
Underlying liver disease	
HCV hepatitis	27 (20%)
Alcoholic hepatitis	19 (14%)
NAFLD	19 (14%)
HBV hepatitis	13 (9%)
Alcoholic hepatitis and HCV hepatitis	2 (1%)
HBV and HCV hepatitis	1 (1%)
Other/Unknown	56 (41%)
Total follow-up (week)	193.3 (±184.6)
Survival by the end of follow-up	
Alive	17 (12%)
Dead	120 (88%)
Recurrence follow-up (week)	111.9 (±139.7)
Recurrence	
No	33 (30%)
Yes	77 (70%)
Eastern cooperative oncology group (ECOG) performance status	
0	77 (60%)
1	40 (31%)
2	10 (8%)
3	2 (1%)
Barcelona clinic liver cancer (BCLC) stage	
0	7 (5%)
1	46 (34%)
2	18 (13%)

3	60 (44%)
4	5 (4%)
Tumor, Node, Metastasis (TNM) stage	
1	90 (75%)
2	8 (7%)
3	14 (11%)
4	8 (7%)
Tumor number	
1	91 (67%)
2	17 (13%)
3	9 (7%)
>3	18 (13%)
Extent of liver fibrosis	
0	45 (34%)
1	5 (4%)
2	9 (7%)
3	4 (3%)
4	70 (52%)
Alpha Fetoprotein (ng/mL)	23486.18 (±184510.10)
Alkaline phosphatase (IU/L)	263.29 (±346.1)
Aspartate aminotransferase (IU/L)	84.39 (±84.1)
Alanine transaminase (IU/L)	79.23 (±83.2)
Sodium (mEq/L)	139.08 (±3.6)
Prothrombin time (seconds)	1.09 (±0.2)
White blood cell (count/μL)	6442.86 (±2990.8)
Hemoglobin (g/dL)	13.26 (±1.7)
Platelet (count/μL)	212.00 (±130)
Total bilirubin (mg/dL)	1.20 (±1)
Creatinine (mg/dL)	1.13 (±1)
Albumin (g/dL)	3.71 (±0.6)
Child-Pugh score	5.83 (±1.4)
Model for end-stage liver disease (MELD) score	9.42 (±3.72)
SD: standard deviation; HCV: hepatitis C virus; HBV: hepatitis B virus; NAFLD: non-alcoholic fatty liver disease; IU: international unit; mEq: milliequivalent.	

Supplemental Table S3. Multivariable Cox proportional-hazard model for overall survival of hepatocellular cancer patients.

Prognostic factor	subgroups	Hazard ratio	P value
Eastern cooperative oncology group (ECOG) performance status	0	Reference	
	1	1.45	.2
	2	5.38	<.001
Alkaline phosphatase (IU/L)	per 100-unit increments	1.05	<.001
White blood cell (count per μL)	per 1000-unit increments	1.09	.09
Tumor number	1	Reference	
	2	1.17	.66
	3	3.38	.02
	>3	1.04	.88
TNM stage	1	Reference	
	2	0.53	.35
	3	2.70	.003
	4	2.17	.06
Total A1CF expression	quartile 1	Reference	
	quartile 2	1.55	.14
	quartile 3	1.66	.05
	quartile 4	2.01	.05

TNM: tumor, node, metastasis; A1CF: APOBEC1 complementation factor

Supplemental Table S4. Clinical and laboratory data for hepatocellular cancer patients with underlying NAFLD.

Parameter	**N (%) / mean (±SD)**
Gender	
Female	5 (26%)
Male	14 (74%)
Age at diagnosis (year)	65.71 (±10.5)
Race	
Caucasian	17 (89%)
Unknown	2 (11%)
Body mass index (kg/m^2)	34.01 (±4.7)
Obese (body mass index >30)	
No	2 (11%)
Yes	16 (89%)
Total follow-up (week)	188.64 (±146.8)
Survival	
Alive	3 (16%)
Dead	16 (84%)
Recurrence follow-up (week)	85.0 (±84.6)
Recurrence	
No	5 (28%)
Yes	13 (72%)
NAFLD: non-alcoholic fatty liver disease; SD: standard deviation	

Supplemental Table S5. Multivariable Cox proportional-hazard model for overall survival of hepatocellular cancer patients with underlying NAFLD.

Prognostic factor	subgroups	Hazard ratio	P value
Time to the first recurrence (week)	per 1-unit increments	0.99	.03
Recurrence	No	Reference	
	Yes	1.41	.7
Total A1CF expression	Quartile 1	Reference	
	Quartile 2	2.0	.5
	Quartile 3	8.10	.01
	Quartile 4	8.64	.002

NAFLD: non-alcoholic fatty liver disease; A1CF: APOBEC1 complementation factor

Supplemental Table S6. Demographic descriptors for Hong Kong patient cohort.

cDNA	Sex/Age	Diagnosis
306N	F/46	CA colon to liver, Metastatic adenocarcinoma
209N	M/43	CA colon to liver, Metastatic adenocarcinoma
215N	F/44	CA colon to liver, Metastatic adenocarcinoma
422N	F/31	CA colon to liver, Metastatic adenocarcinoma
227N	F/48	CA colon to liver, Metastatic adenocarcinoma
279N	M/69	Autopsy. COD: Ruptured abdominal aortic aneurysm. Sever coronary atherosclerosis with acute myocardial infraction
335N	M/53	CA colon to liver, Metastatic adenocarcinoma
C441	F/51	Explanted liver, High grade dysplastic nodule, macroregenerative nodules; HBV-associated cirrhosis, chronic cholecystitis; cholelithiasis.
C440	M/40	Explanted liver, HBV-associated cirrhosis, fatty change with Mallory bodies
C456	M/49	Explanted liver, Active HBV-associated cirrhosis
C231	M/39	Explanted liver, Fibrosing cholestatic hepatitis
C453		Explanted liver, HBV-associated cirrhosis, moderate activity, large regenerative nodules, chronic cholecystitis, cholelithiasis
C452	M/49	Explanted liver; HBV-associated cirrhosis with severe activity, macroregenerative nodules
ch281	M/55	Explanted liver. Cirrhosis in keeping with chronic Hep B
ch253	M/49	Explanted liver, chronic Hep B virus associated cirrhosis with acute exacerbation: Cholestasis
ch229	M/47	Explanted liver, Chronic hepatitis in a background of cirrhosis (Scheuer grade 2, Stage 4), regenerate nodules formation
ch279	M/40	Explanted liver. Chronic Hepatitis, grade 3 Stage 4 cirrhosis.
ch261	M/52	Explanted liver, Cirrhosis, compatible with HBV-related
ch277	M/43	Explanted liver. Chronic Hep B cirrhosis: mild haemosiderosis
ch329	F/55	Explanted liver. Cirrhosis. Portal vein thrombus.
ch263	M/57	Explanted liver, Submassive hepatic necrosis: chronic Hep B with bridging fibrosis
ch361	M/55	Explanted liver. Cirrhosis and Hep B with siderosis
ch246	M/61	Explanted liver, Hep B associated cirrhosis
ch230	M/58	Explanted liver. Irregular cirrhosis with no malignancy

ch295	M/46	Explanted liver. Hep B associated mild chronic Hepatitis with cirrhosis. Chronic cholecystitis
ch297	F/61	Explanted liver. Chronic Hep B with septal fibrosis & minimal activity; portal vein thrombosis
ch294	M/49	Explanted liver, HBV-associated cirrhosis, with mild activity, grade 2-3 siderosis. Caroli's disease
ch225	M/49	Explanted liver, alcoholic cirrhosis, liver flukes

Supplemental Table S7. Antibodies.

Antibodies	Species	Manufacturer	Catalog #	WB	IH
A1CF	Rabbit	*		1:3000	1:2000
FLAG	Rabbit	ThermoScientific	PA1-984B	1:1000	1:200
CIDEA	Rabbit	Millipore	ABC350	1:600	1:100
β-CATENIN	Mouse	BD Biosciences	610154	1:2000	1:600
CDH1	Mouse	Abcam	76055		1:250
CYCLIN D1	Rabbit	Abcam	134175		1:100
HSP70	Rabbit	Abcam	2787		1:200
GPC3	Mouse	ThermoScientific	1G12		1:100
P62	Rabbit	Millipore	EP396		1:200
APOB	Rabbit	**		1:4000	
CD36	Goat	R&D	AF2519	1:7000	
MOGAT1	Rabbit	***		1:1000	
MOGAT2	Rabbit	Invitrogen	PA5-42625	1:1000	
ACTIN	Rabbit	Sigma	A2066	1:2000	
GAPDH	Rabbit	Santa Cruz Biotechnology	Sc-25778	1:1000	
STAT3	Mouse	Cell Signaling	9139S	1:2000	
PSTAT3	Rabbit	Cell Signaling	9131S	1:1000	

* (67); ** (68)*** (69)

Supplemental Table S8. Quantitative PCR primers.

RNA	Forward primer	Reverse Primer
Abcc12	5'-GAGGAGTTCAGGAGCTCAAGCA-3'	5'-AGACCCTGTATGGAGGAGGTGAT-3'
αfp	5'-TCATGTATGCCCCAGCCATT-3'	5'-CAGCATGCCAGAACGACCTT-3'
Apob	5'-AATATAATCGGAGAAGCAGGACCTA-3'	5'-TCCCGAAGTTGACATCAAACC-3'
αSma	5'-CCAGAGCAAGAGAGGGATCCT-3'	5'-TGTCGTCCCAGTTGGTGATG-3'
Atf4	5'-CGAGTTAAGCACATTCCTGGAATC-3'	5'-TTCGCTGTTCAGGAAGCTCAT-3'
Bmp7	5'-CCTTCATGGTGGCCTTCTTC-3'	5'-CCCCGTGGACCGGATACTA-3'
Cbr3	5'-AGAGGAAAGCGGACAGGATTC-3'	5'-TCGCCATGTCGGTCTTCA-3'
Ccr2	5'-TCCACGGCATACTATCAACATCTC-3'	5'-GGCCCCTTCATCAAGCTCTT-3'
Cdh1	5'-ACCCCCTTACGACTCTCTGTTG-3'	5'-CAGGCTAGCGGCTTCAGAAC-3'
Cdh24	5'-CGTACAGCCATCCCCAACAT-3'	5'-GCTTGAATCACCACCAAGAACTC-3'
Cdk1	5'-GGACGAGAACGGCTTGGAT-3'	5'-ATTCGTTTGGCAGGATCATAGAC-3'
Cd36	5'-TCTTCCAGCCAATGCCTTTG-3'	5'-TGGAGATTACTTTTTCAGTGCAGAA-3'
Chka	5'-GGTCACTTGGGCCAAAACTC-3'	5'-CGCCGGCTCGGGATA-3'
Chop	5'-CCACCACACCTGAAAGCAGAA-3'	5'-TGAAAGGCAGGGACTCA-3'
Cidea	5'-CCAGAGTCACCTTCGACCTATACA-3'	5'-CATCGTGGCTTTGACATTGAGA-3'
Col1a1	5'-CACGGCTGTGTGCGATGA-3'	5'-TCGCCCTCCCGTCTTTG-3'
Col4a1	5'-CCAGGATGCAACGGTACAAA-3'	5'-AACGTGGCCGAGAATTTCAC-3'
Cxcl14	5'-GCACTGCCTGCACCCTAAG-3'	5'-TCGTTCCAGGCATTGTACCA-3'
Dlgap1	5'-GCAGCCGATGACGACTTTG-3'	5'-GTCCGGAGGAGGCAGGATAG-3'
Dmrta1	5'-CTGGTTCCAGCATTGCCTTT-3'	5'-ATCATCCCTGGAAACGCATAA-3'
Dram1	5'-GCCATCTCCGCTGTTTCGT-3'	5'-GGATTCCATTCCAGCTTGGTT-3'
Gapdh	5'-TGTGTCCGTCGTGGATCTGA-3'	5'-CCTGCTTCACCACCTTCTTGA-3'
Ggt5	5'-GGTTCCCGTGTGCCCTTTATC-3'	5'-GCCGCAGGATGCTGTTGT-3'
Gpc1	5'-GGACATCACCAAGCCAGACA-3'	5'-CATAGGCGCCACGTAAACG-3'
Gpx3	5'-TCACCTGGCCGCCTCTT-3'	5'-AAAGTTCCAGCGGATGTCATG-3'
Grp78	5'-ACCCCGAGAACACGGTCTT-3'	5'-GCTGCACCGAAGGGTCATT-3'
Gss	5'-CATTGCCCAGACCGTGTTC-3'	5'-TGCGCCGCACTGGAA-3'
Gstm3	5'-TGCTGCAGTCCCGATTTTG-3'	5'-TTCTCAGGGATGGCCTTCAA-3'
Il19	5'-GACATGCGCCTCATAGAAAAGA-3'	5'-ACAGGATGGTGACATTTTTAAAGGT-3'
Jak3	5'-TGCTGCCCTGAACCTAACATC-3'	5'-TTCGCACCACGATCAAATTG-3'
Kif20a	5'-ACCTAGCCATCAGCATAAGAGACA-3'	5'-CACGTAAGGATTGCCGTTCTG-3'
Klf4	5'-ACCTATACCAAGAGTTCTCATCTCAA3'	5'-CCGTCCCAGTCACAGTGGTAA-3'
Ly6c	5'-GGATTCTGCATTGCTCAAAACA-3'	5'-CTGACGGGTCTTTAGTTTCCTTCT-3'
Mcm2	5'-GTTCAGCGTCATGCGGAGTAT-3'	5'-TCTCGCCGGAAGGAGAGATA-3'
Mcm4	5'-CCAACCCAGTCCCTTCGAA-3'	5'-TCGCCTCTACGTCTCCGATT-3'
Mcm6	5'-TGTCAGCGCCCATCATGT-3'	5'-CATTGCATTCATCCACCAGAA-3'
Mmp2	5'-CTATGTCCACTGTGGGTGGAAA-3'	5'-TTGTTGCCCAGGAAAGTGAAG-3'
Mogat1	5'-ACCACAAATCCTGCGAAAGG-3'	5'-TGTTGAGTGGCGCGAACTC-3'
Mogat2	5'-CTGATGATGCTGACTGTGTGGTT-3'	5'-CCAGCCCCCCAGACATG-3'

Pbrm1	5’-GGTGTTACAGCGACTCCTTAGCA-3’	5’-CCGATACCGATTACTTTCAACATTT-3’
Phlda2	5’-ACCGCCTGCGCCTCTT-3’	5’-TGGAGTGGAAAAAACAGCTCCTT-3’
Rb1	5’-TCCACCAGGCCTCCTACCT-3’	5’-GAATCCGTAAGGGTGAACTAGAAAAC-3’
Sall2	5’-TCCACCTTCGTTCCCACACT-3’	5’-CCGAGTTGTGAAACGGTTACC-3’
Scd2	5’-GTACCGCTGGCACATCAACTT-3’	5’-ACACTCTCTTCCGGTCGTAAGC-3’
Slc1a5	5’-GCCAGCAAGATTGTGGAGATG-3’	5’-TGTATTTGCCGAGGCTGATG-3’
Smad9	5’-AAAACACCAGGAGGCACATTG-3’	5’-ACCTCGCCCCCAACGTA-3’
Sox4	5’-GGCTAGGCAAACGCTGGAA-3’	5’-TCCTGGATGAACGGAATCTTG-3’
Sox9	5’-CGGCTCCAGCAAGAACAAG-3’	5’-TGCGCCCACACCATGA-3’
Spag5	5’-TTGCAGCAGGACTGGACATC-3’	5’-TCGAGACCAACTCAGCAAAGC-3’
Sparcl1	5’-CTCGCTTCTTTGAGGAGTGTGA-3’	5’-TGGCCCCATTCCTTCAAG-3’
Spp1	5’-TTTCACTCCAATCGTCCCTACA-3’	5’-TCAGTCCATAAGCCAAGCTATCAC-3’
Tmp1	5’-CATGGAAAGCCTCTGTGGATATG-3’	5’-GATGTGCAAATTTCCGTTCCTT-3’
Tmprss4	5’-CCTGCAGACAGATGGGCTATG-3’	5’-GGTTCTGATCTGGACGGATCTC-3’
Tnf α	5’-TACCTTGTCTACTCCCAGGTTCTCT-3’	5’-GTGTGGGTGAGGAGCACGTA-3’
Xbp1	5’-ATCAGCTTTTACGGGAGAAAACTC-3’	5’-CCATTCCCAAGCGTGTTCTT-3’
Xpo4	5’-GCAGTGCCTTGCCCAGTT-3’	5’-AATCAACCTGGGATCCTTCGT-3’