Supporting Information

Supporting Methods

Generation of the *Tg(Tp1:mAGFP-gmnn)* and *id2a* mutant lines

The *Tp1:mAGFP-gmnn* construct was generated by first replacing the *EF1* promoter in the *pT2KXIGΔin* vector (1) with the *Tp1* module (multiple RBP-Jκ-binding sites in front of a minimal promoter) and then placing *mAGFP-gmnn* (2) downstream of the *Tp1* element and a 5' beta-globin intron. The final construct together with *Tol2* mRNA was injected into one-cell stage embryos as previously described (3). The left (TAL3100, Plasmid #41274) and the right (TAL3101, Plasmid #41275) TALEN constructs of *id2a* were obtained from Addgene. The *id2a* target sequences are in the first exon, upstream of the helix-loop-helix domain; the spacer region contains a *TfiI* restriction enzyme site. After linearization with *SmaI* digestion, the TALEN mRNA was synthesized using the Ambion mMESSAGE mMACHINE T7 Ultra Transcription Kit (Thermo Fischer Scientific, Waltham, MA). *id2a* TALEN mRNAs were mixed at a 1:1 ratio to a final concentration of 200 ng/ul and subsequently injected into one-cell-stage embryos. Either the adult zebrafish' tail fin or whole embryo at 1-2 dpf was used to obtain genomic DNA for PCR-mediated genotyping. PCR products were sequenced to identify a frameshift mutation. F1 fish containing a 22-bp deletion was selected to establish the *id2a* mutant line.

Genotyping of *tbx2b, smad5,* and *id2a* mutants

For *id2a* genotyping, genomic DNA was amplified with the forward (5'-TCCTGCTGTCAACATGAAGGCA -3') and reverse (5'-AGTCGAGCGCGATCTGCAGG-3') primers, followed by digestion with *TfiI*. The wild-type allele generates two bands of 179 and 87 bp, whereas the *id2a* mutant allele generates a band of 244 bp. *tbx2b* (4) and *smad5* (5) genotyping were performed as previously described.

Heat-shock condition

Tg(hs:dnBmpr1) larvae were heat-shocked at A30h or R8h by transferring them into egg water pre-warmed to 39°C and kept at this temperature for 20 minutes as previously described (6).

Cre/loxP-mediated lineage tracing

Fish carrying the *Tp1:CreERT2* or *fabp10a:CreERT2* transgene were crossed to the reporter line, *Tg(ubb:loxP-GFP-loxP-mCherry)*. Larvae from the crosses were treated with Mtz from 3.5 to 5 dpf and additionally treated with both 5 μM 4-OHT and 10 μM DMH1 (Tocris, Bristol, UK) from A33h to R6h for 9 hours. At R54h, 48 hours after 4-OHT and DMH1 washout, the larvae were harvested and processed for immunostaining to reveal lineage-traced mCherry^{+} cells as previously described (7).

EdU labeling

EdU labeling was performed according to the protocol outlined in the Click-iT EdU Alexa Fluor 647 Imaging Kit (Life Technologies, Grand Island, NY). Larvae were treated with egg water containing 10 mM EdU and 5% DMSO. After a 6-hour EdU treatment, the larvae were harvested for subsequent analysis.

Whole-mount in situ hybridization (WISH) and immunostaining

WISH was performed as previously described (8). cDNA from livers at 5 dpf or R6h was used as a template for PCR to amplify genes-of-interest; PCR products were used to make in situ probes. The primers used for the probe synthesis are listed in Table S2. Whole-mount immunostaining was performed as previously described (9), using the following antibodies: rabbit anti-Prox1 (GTX128354, GeneTex, Irvine, CA), goat anti-Hnf4a (Santa Cruz, Dallas, TX), chicken anti-GFP (Aves Labs, Tigard, OR), mouse anti-Bhmt (1:500; a gift from J. Peng, Zhejiang University, China), mouse anti-Alcam (ZIRC, Eugene, OR), rat anti-mCherry (Allele Biotechnology, San Diego, CA), and Alexa Fluor 488-, 568-, and 647-conjugated secondary antibodies (Life Technologies, Grand Island, NY).

Quantitative RT-PCR (qPCR)

Total RNA was extracted from 100 dissected livers using the RNeasy Mini Kit (Qiagen, Valencia, CA); cDNA was synthesized from the RNA using the SuperScript® III First-Strand Synthesis SuperMix (Life Technologies, Grand Island, NY) according to the kit protocols. qPCR was performed as previously described (10), using the Bio-Rad iQ5 qPCR machine with the iQ™ SYBR Green Supermix (Bio-Rad, Hercules, CA). *eef1b2* was used for normalization. At least three independent experiments were performed. The primers used for qPCR are listed in Table S3.

Mouse LPC cell line and culture condition

The LPC cell line, HSCE1, used in this study was established and maintained as described previously (11). For their differentiation into hepatocytes, HSCE1 cells were cultured in the presence of 1% DMSO and 20 ng/mL mouse Oncostatin M with or without 500 ng/mL recombinant human BMP2 (Peprotech, Rocky Hill, NJ) (n = 4). After 5 days of hepatocyte induction, the cultured cells were harvested for RNA preparation. HSCE1 cells before the induction were used as a negative control. TRIzol reagent (Invitrogen, Carlsbad, CA) was used to extract total RNA; PrimeScript RT reagent kit (Takara, Shiga, Japan) was used to synthesize cDNA, according to the kit protocols. qPCR was conducted with a LightCycler 480 system and Universal Probe Library (Roche Diagnostics,

Indianapolis, IN). The Universal Probe Library Mouse ACTB Gene Assay was used for normalization. The primers used for qPCR are listed in Table S3.

Image acquisition, processing, and statistical analysis

Zeiss LSM700 confocal and Leica M205 FA epifluorescence microscopes were used to obtain image data. Confocal stacks were analyzed using the Zen 2009 software. All figures, labels, arrows, scale bars, and outlines were assembled or drawn using the Adobe Illustrator software. Unpaired two-tailed Student's t-test was used for statistical analysis; $P<0.05$ was considered statistically significant. Quantitative data were shown as means ±SEM.

Supporting figures

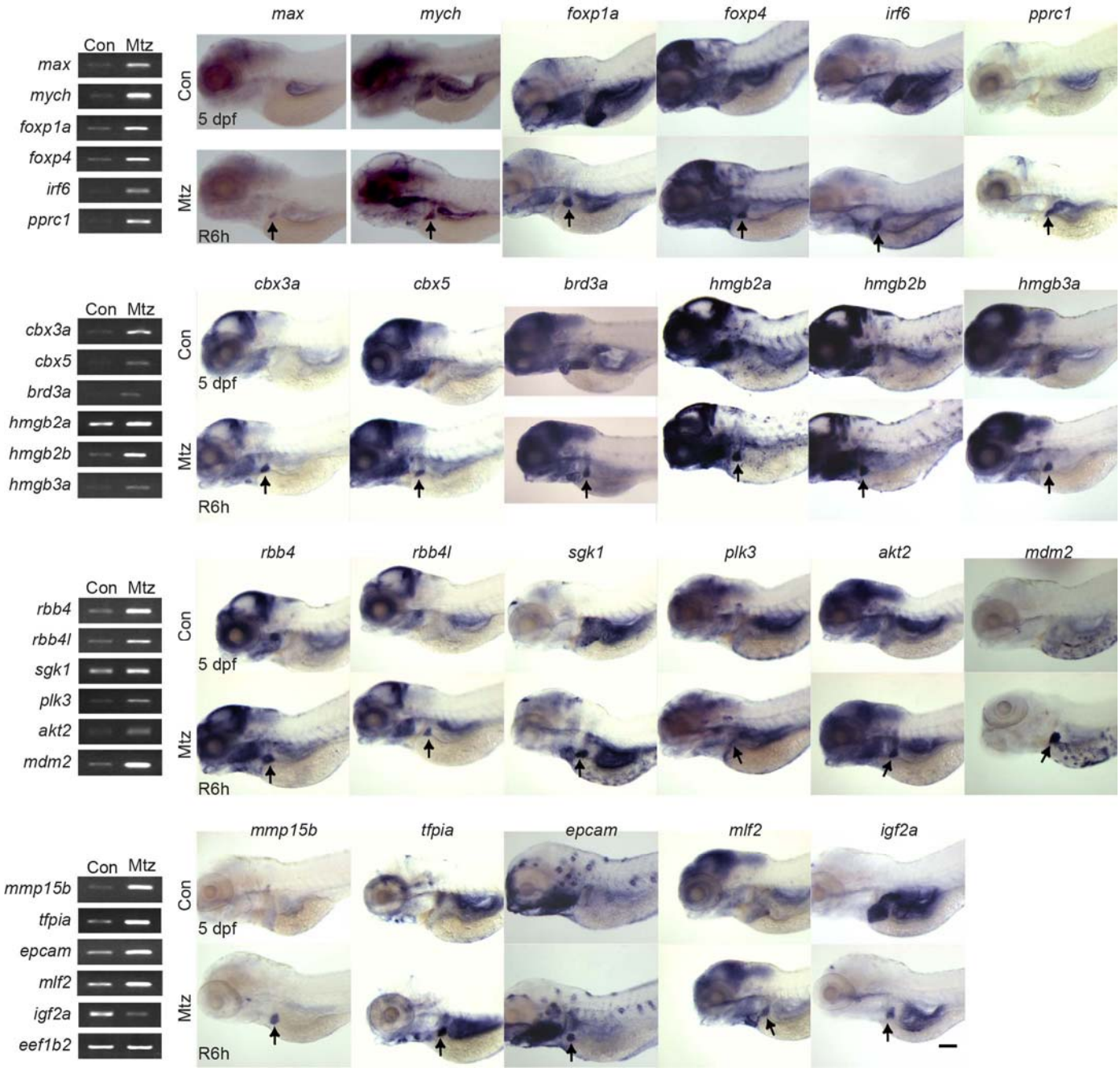

Figure S1. Validation of RNAseq data using RT-PCR and WISH. RT-PCR and WISH were performed to validate genes-of-interest from RNAseq analysis. For RT-PCR, cDNA was prepared from control livers at 5 dpf and from Mtz-treated regenerating livers at R6h. For in situ hybridization, control larvae at 5 dpf and regenerating larvae at R6h were used. Arrows point to regenerating livers. Scale bar: 150 μm.

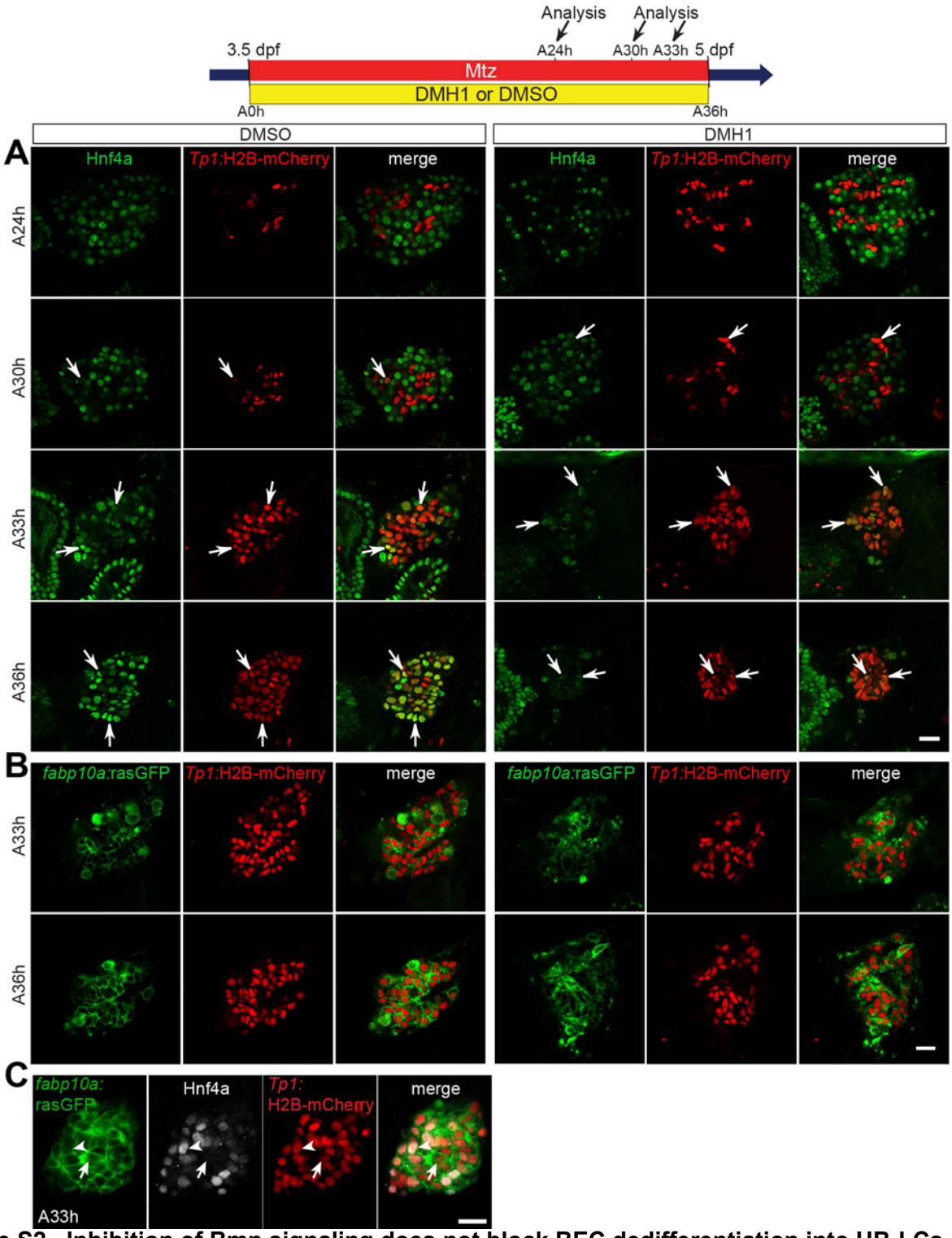

Figure S2. Inhibition of Bmp signaling does not block BEC dedifferentiation into HB-LCs. (A) Single-optical section images showing Hnf4a (green) and *Tp1:*H2B-mCherry (red) expression in ablating livers of DMH1- or DMSO-treated larvae at A24h, A30h, A33h and A36h. Arrows point to Hnf4a^{+}/H2B-mCherry^{+} cells. (B) Single-optical section images showing *fabp10a:*rasGFP (green) and *Tp1:*H2B-mCherry (red) expression in ablating livers at A33h and A36h. (C) Single-optical section images showing the expression of *fabp10a:*rasGFP (green), Hnf4a (gray), and *Tp1:*H2B-mCherry (red) in ablating livers at A33h. Arrows point to rasGFP^{+}/Hnf4a^{+}/H2B-mCherry^{+} cells; arrowheads point to rasGFP^{+}/Hnf4a^{-}/H2B-mCherry^{+} cells. Scale bars: 20 μm.

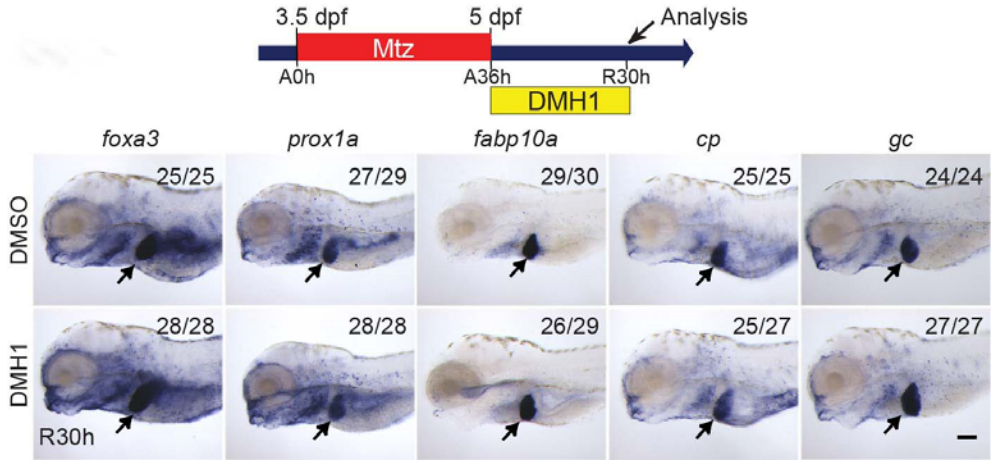

Figure S3. Inhibition of Bmp signaling after hepatocyte ablation does not affect HB-LC differentiation into hepatocytes. WISH images showing the expression of *fabp10a*, *foxa3*, *prox1a*, *cp* and *gc* in regenerating livers (arrows) at R30h. Numbers in the upper right corner indicate the proportion of larvae exhibiting the representative expression shown. Scale bar, 150 μm.

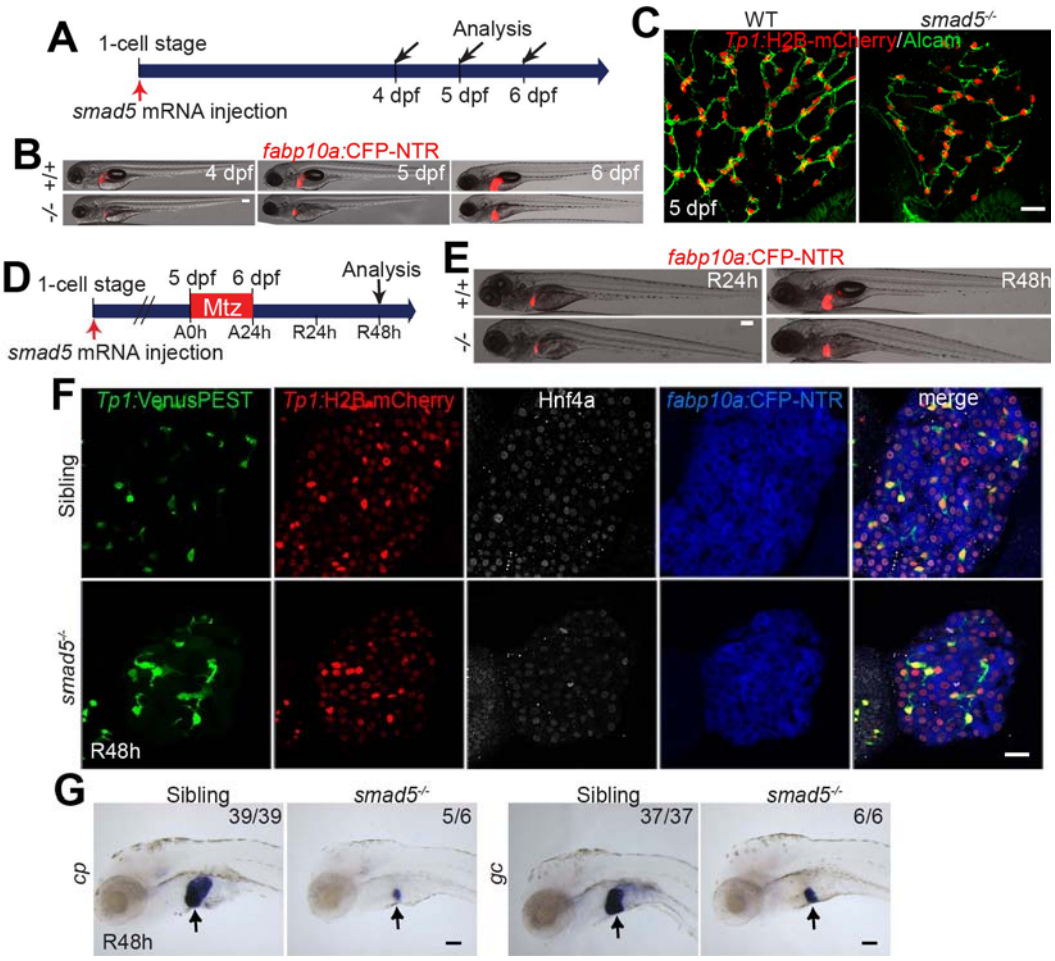

Figure S4. HB-LC differentiation defect observed in *smad5* mutants at R24h recovers at R48h. (A) Experimental scheme illustrating the stages of *smad5* mRNA injection (red arrow) and analysis (arrows) for B and C. (B) Epifluorescence images showing hepatic *fabp10a:*CFP-NTR expression (red) in *smad5*$^{-/-}$ mutants and wild-type siblings at 4, 5, and 6 dpf. (C) Confocal projection images showing Alcam (green) and *Tp1:*H2B-mCherry (red) expression in the livers of *smad5*$^{-/-}$ mutants and wild-type siblings at 5 dpf. (D) Experimental scheme illustrating the stages of *smad5* mRNA injection and Mtz treatment and analysis for E-G. (E) Epifluorescence images showing *fabp10a:*CFP-NTR expression (red) in the regenerating livers of *smad5*$^{-/-}$ mutants and wild-type siblings at R24h and R48h. (F) Single-optical section images showing the expression of Hnf4a (gray), *Tp1:*H2B-mCherry (red), *Tp1:*VenusPEST (green), and *fabp10a:*CFP-NTR (blue) in the regenerating livers of *smad5*$^{-/-}$ mutants and wild-type siblings at R48h. (G) WISH images showing *cp* and *gc* expression in the regenerating livers (arrows) of *smad5*$^{-/-}$ mutants and wild-type siblings at R48h. Scale bars: 150 (B, E, G), 20 (C, F) μm.

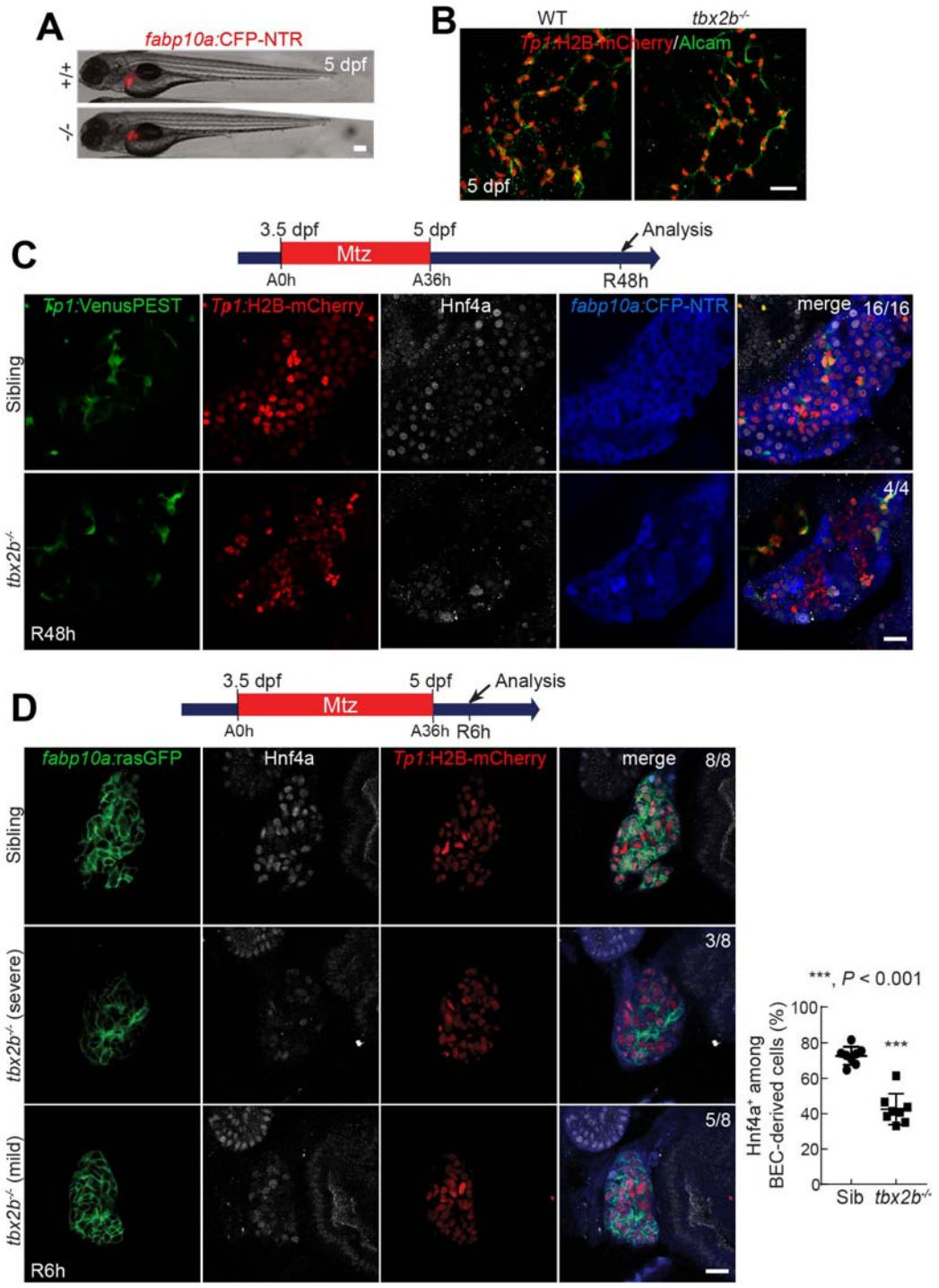

Figure S5. ***tbx2b*** **mutants still exhibit a defect in HB-LC differentiation into hepatocytes at R48h**. (A) Epifluorescence images showing hepatic *fabp10a:*CFP-NTR expression (red) in *tbx2b*$^{-/-}$ mutants and wild-type siblings at 5 dpf. (B) Confocal projection images showing Alcam (green) and *Tp1:*H2B-mCherry (red) expression in the uninjured liver of *tbx2b*$^{-/-}$ mutants and wild-type siblings at 5 dpf. (C) Single-optical section images showing the expression of *Tp1:*VenusPEST (green), *Tp1:*H2B-mCherry (red), *fabp10a:*CFP-NTR (blue), and Hnf4a (gray) in the regenerating livers of *tbx2b*$^{-/-}$ mutants and their siblings at R48h. (D) Single-optical section images showing the expression of *fabp10a:*rasGFP (green), *Tp1:*H2B-mCherry (red), and Hnf4a (gray) in the regenerating livers of *tbx2b*$^{-/-}$ mutants and their siblings at R6h. Quantification of the percentage of Hnf4a^{+} cells among H2B-mCherry^{+} BEC-derived cells is shown. Numbers in the upper right corner indicate the proportion of larvae exhibiting the representative phenotype shown. Scale bars: 150 (A), 20 (B-D) μm.

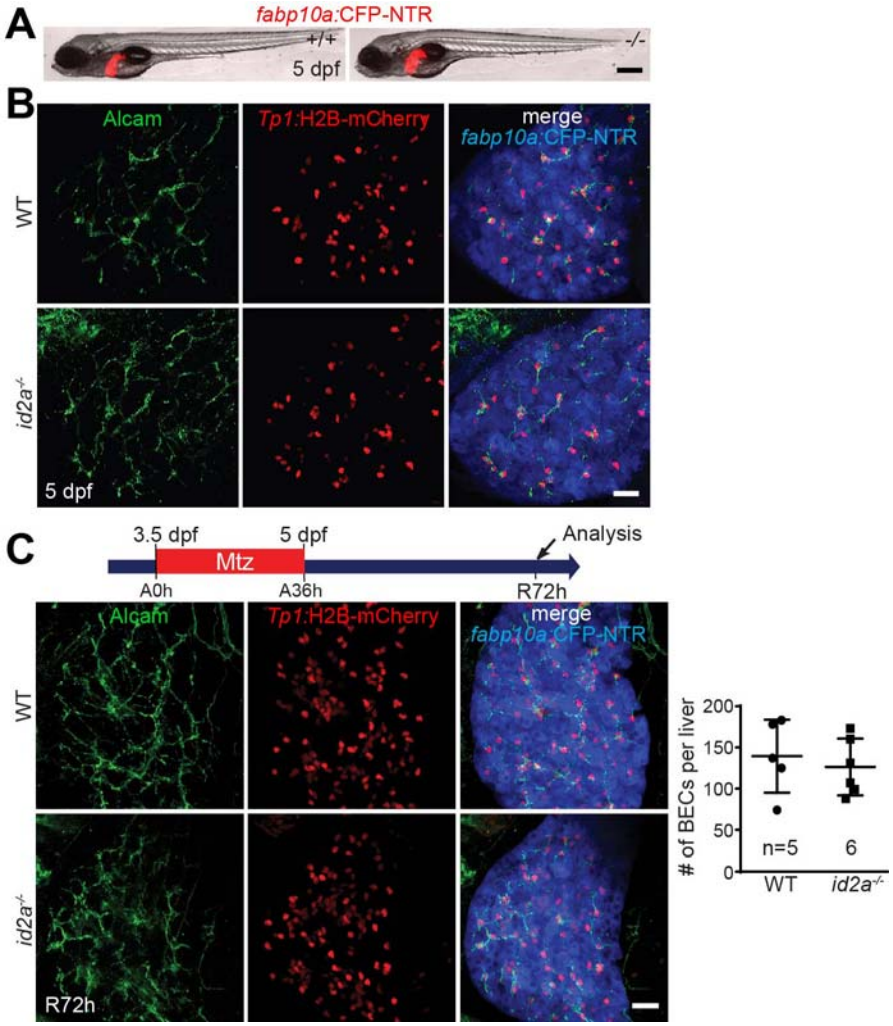

Figure S6. The excess BEC phenotype observed in *id2a* mutants at R30h recovers at R72h. (A) Epifluorescence images showing *fabp10a:*CFP-NTR expression (red) in the uninjured livers of *id2a*$^{-/-}$ mutants and wild-type siblings at 5 dpf. (B) Confocal projection images showing the expression of *fabp10a:*CFP-NTR (blue), Alcam (green), and *Tp1:*H2B-mCherry (red) in the uninjured livers of *id2a*$^{-/-}$ mutants and wild-type siblings at 5 dpf. (C) Confocal projection images showing the expression of Alcam (green), *Tp1:*H2B-mCherry (red), and *fabp10a:*CFP-NTR (blue) in the regenerating livers of *id2a*$^{-/-}$ mutants and wild-type siblings at R72h. Quantification of the total numbers of BECs (Alcam^{+}/H2B-mCherry^{+}) per liver is shown. Scale bars: 300 (A), 20 (B, C) μm; error bars: ±SEM.

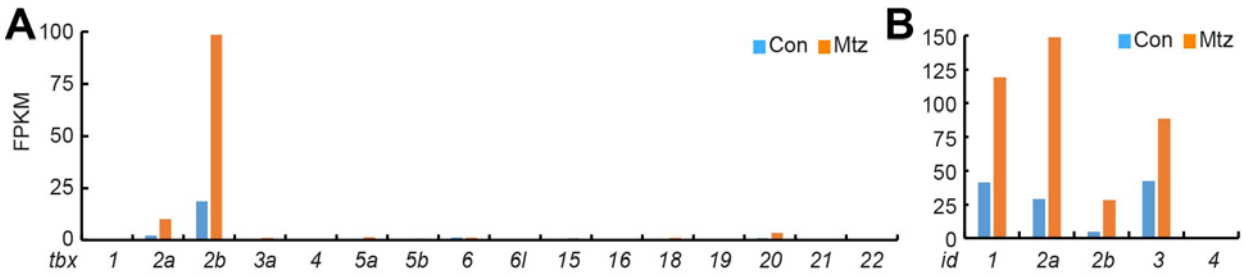

Figure S7. Expression levels of *tbx* and *id* genes in regenerating livers at R6h. (A, B) Graphs showing the expression levels of 16 *tbx* (A) and 5 *id* (B) genes in control and regenerating livers at R6h. FPKM stands for fragments per kilobase of exon per million fragments mapped.

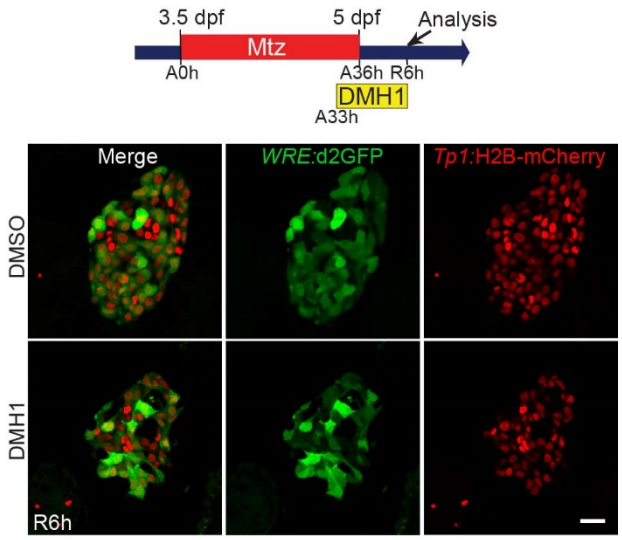

Figure S8. Inhibition of Bmp signaling does not affect Wnt activity in regenerating livers at R6h. Single-optical section images showing *WRE:*d2GFP (green) and *Tp1:*H2B-mCherry (red) expression in regenerating livers at R6h. The Wnt reporter line, *Tg(WRE:d2GFP)*, was used to reveal hepatic Wnt activity. Scale bar: 20 μm.

Supporting tables

Table S1. Transgenic and mutant lines used in this study

Names used in this study	Official names (ZFIN database)	Allele #	Ref
Tg(Tp1:VenusPEST)	*Tg(EPV.Tp1-Mmu.Hbb:Venus-Mmu.Odc1)*	*s940*	(12)
Tg(Tp1:H2B-mCherry)	*Tg(EPV.Tp1-Mmu.Hbb:hist2h2l-mCherry)*	*s939*	(12)
Tg(ubb:loxP-GFP-loxP-mCherry)	*Tg(-3.5ubb:LOXP-EGFP-LOXP-mCherry)*	*cz1701*	(13)
Tg(Tp1:CreERT2)	*Tg(EPV.Tp1-Mmu.Hbb:Cre-ERT2,cryaa:mCherry)*	*s959*	(14)
Tg(fabp10a:CreERT2)	*Tg(fabp10a:Cre-ERT2,cryaa:ECFP)*	*pt602*	(7)
Tg(fabp10a:rasGFP)	*Tg(-2.8fabp10a:CAAX-EGFP)*	*s942*	(15)
Tg(fabp10a:CFP-NTR)	*Tg(fabp10a:CFP-NTR)*	*s931*	(7)
Tg(hs:dnBmpr1)	*Tg(hsp70l:dnXla.Bmpr1a-GFP)*	*w30*	(16)
Tg(Tp1:mAGFP-gmnn)	*Tg(EPV.Tp1-Mmu.Hbb:mAGFP-gmnn)*	*s707*	this study
Tg(WRE:d2GFP)	*Tg(OTM:d2EGFP)*	*kyu1*	(17)
smad5	*smad5*	*m169*	(5)
tbx2b	*tbx2b*	*c144*	(4)
id2a	*id2a*	*pt661*	this study

Table S2. Sequences of primers used for in situ probe synthesis

Gene	Primer	Nucleotide sequence (5' to 3')
pprc1	forward	CTAGCATTGTTATCAAGACCGTTG
pprc1	reverse	GCCTTTTTGTATCCTCACTGCTAT
tfpia	forward	CAGGTTTTACTTTGACATCGACAC
tfpia	reverse	TCTTTATCCGTATTTGCTTTCTCC
hmgb3a	forward	GCTTATGCCTATTTCGTTCAGACT
hmgb3a	reverse	CTATTCGTCGTCGTCATATTCGT
hmgb2b	forward	GGTGAAAGGAGACGTGAACAA
hmgb2b	reverse	TCTTCCTCATCATCTTCCTCGT
hmgb2a	forward	GTAAAGATCCAAATAAGCCCAGAG
hmgb2a	reverse	CTCATCATCGTCATCATCAGCTT
cbx3a	forward	CAGGCAAGTCAAAGAAGGAAGTT
cbx3a	reverse	GCTCATCCTCAGGACAAGAATG
cbx5	forward	ATGGGAAAGAAGAGCCAGAAC
cbx5	reverse	CTGTGGCACTCTTCTCCTTCTTG
brd3a	forward	TTTCAACACAATGTTCACAAACTG
brd3a	reverse	TTGACAGCATTTCTTTAAGGATGA
foxp4	forward	CCAGCCGTAGAGTGAAAGTAGAGT
foxp4	reverse	GTAGATTCAGAATGTGTTGCTGCT
foxp1a	forward	TAAAACTCAGACTCTCAACCACCA
foxp1a	reverse	TGTTCGAGTGAACCAGTTGTAGAT
tbx2b	forward	ATAAATATCAGCCCAGGTTCCATA
tbx2b	reverse	GGAGAGAAGCTGTCTTTACTACCG
id2a	forward	TCCTGCTGTCAACATGAAGGCA
id2a	reverse	TAATACGACTCACTATAGGGCCAGTTCCCAGGTCCTGTGTGT
mlf2	forward	TTTCGTTACTTGAATGATGTGGAT
mlf2	reverse	ATAATCTAAACTGCGGGCAGTAGG
irf6	forward	AGTATCAGGAAGGAGTGGATGAAC
irf6	reverse	GAACAGCTCCTCTTGGTTCACTAT

igf2a	forward	GGATGATTACCATGTATTCTGTGC
igf2a	reverse	GTTTGCTCCTCATCTTGGATTTT
akt2	forward	ACAGCAATTCTGAAAGAGAGGAGT
akt2	reverse	GCCAAAGTCTGTGATTTTAATGTG
sgk1	forward	ACAAGATGTGGAGCTAATGAACAG
sgk1	reverse	GCAGGCCATATAACATTTCATACA
plk3	forward	GAATCCACCTTTTGAAACCTTAGA
plk3	reverse	AAGACAAGCAGCTGTTAAGAACCT
mych	forward	GTATCTGAACTTTTAATGGAGGACAC
mych	reverse	TAATACGACTCACTATAGGGATCTGCAGACCTCGCTGGGAGT
max	forward	CAACGATGATATCG AGGTCGACAG
max	reverse	TAATACGACTCACTATAGGGTGAAACAGGCCATTGCTGTGACTC
mdm2	forward	TACAGATTCAGACTCTCGCTCATC
mdm2	reverse	GAGTTTCTTTTCGAAGGTTGTGTT
rbb4	forward	AATCATGAAGGAGAGGTTAACAGG
rbb4	reverse	GAACCTGGAAGATTTCATCTTTGT
rbb4l	forward	TGGATCAGTTAGTGGAAAGATTGA
rbb4l	reverse	GACCTGGAAGATTTCATCCTTATG
smad5	forward	AAACAGAAAGAAGTGTGCATCAAC
smad5	reverse	ACCTCTCCTCCAACATAGTACAGG
epcam	forward	CACAATGTGCTTGTAAAACAATGA
epcam	reverse	TAGATCAGGACATTCTCCATTGAA
mmp15b	forward	AGGTAATGATCTGTTCCTGGTAGC
mmp15b	reverse	GAAGAAGTAAGTGAAGCCAGAAGG
samd4	forward	ACCTTCAAAGTGCCCTCGT
samd4	reverse	GTCTAACGGTGTGGGGTCTG
smad1	forward	AAGGCCCTAGAGTGCTGTGA
smad1	reverse	GGACTCCTTTTCCGATGTGA
smad9	forward	GCACAGCTTCCCAAACTCTC
smad9	reverse	CCGTGCAGGTGGATTTCTAT

Underlined are T7 primer sequences.

Table S3. Sequences of primers used for qPCR

Gene	Forward primer sequence (5' to 3')	Reverse primer sequence (5' to 3')
pprc1	TGCTACGGTTGAGCTGATTG	AGGCAGTGGTCTTGCCTCTA
tfpia	CAAGGCAATGAAGGACAGGT	TTGGCTTCCTCTTCCTCTGA
hmgb3a	TTGAAGACATGGCCAAACAA	ACAGTTAGCCATTGGCATCC
hmgb2b	TATGCGTTCTTCGTGCAGAC	TCTTTGCTGCTCTGCTTTGA
hmgb2a	CCTCTTACGCCTTCTTCGTG	GGTGAAAGCTTGGACCACAT
cbx3a	AGGTAATGGACCAGCGAGTG	CGCTTCATCAGAGTCCTTCC
cbx5	AAGAAGAGCCAGAACCGTGA	TTTTTCCTCTTGCTGGTGCT
brd3a	GGGTGCGGAACAGTGTATCT	TTCTTTGGTTGCCTGTTTCC
foxp4	GGTTTTCCTGAGGGTCTTCC	CGTTGCTGTGTTTCTCCTGA
foxp1a	ACTGGCTCTCCTCTGGTGAA	TTGGTGTCTGGACTCTGCTG
tbx2b	GCCAAAGGCTTCAGAGACAC	TCGTCTTTCTTCTCCGCAAT
id2a	GAAGGCAATAAGCCCAGTGA	GTCAGGGGTGTTCTGGATGT
MLF2	ATGGATCCGTTTGCTCTCAC	ATCGTTTGACGCGTCTCTCT
irf6	GCTGGTCTGGCTGGATAGAG	CCGTCTCGTATGTGGGAACT
igf2a	GACTCTCTGTGGCGGAGAAC	GGTTTGCTCCTCATCTTGGA
akt2	AGAACACACGGCATCCTTTC	GTCTTCATGGTGGCCTCATT
sgk1	GTGCTTTGGGTTACCTGCAT	CTGCAGTGGCTTGTTCAAAA
plk3	CCGGGAGGTCGTACTGTAAA	GATGCCTTTGTTGTGGAGGT
mych	CTCCGACATAGACACGCAGA	TGCTGCTGGATTTCAAAGTG
max	TCGAGGTCGACAGTGATGAA	TTGGTGTACAGGCTGCTGTC
mdm2	CCTCCTCTTCCTCGACACTG	GGGTCTCTTCCTGACTGCTG
rbb4	GTTCATTCTGGCCACAGGTT	CCCATGGTTCATTTGGATTC
rbb4l	GTGTTGGGCACACACACTTC	TTCCAGGACAAACCATAGCC
smad5	CGGCTCCAAACAGAAAGAAG	GTCTTGCCCCATCTGTTCAT
epcam	CTTGTTTGTTGTGGCATTGG	TTGACGCACCAGCATACTTC
mmp15b	GAGCCGCAGATCAGGACTAC	TCAGAACCACATCCACCTCA
hnf4a	GCCGACACTACAGAGCATCA	TGGTAGGTTGAGGGATGGAG
eef1b2	CCCTCTCAGGCTGATATTGC	TAAGCTGCAAGCCTCTCCTC
G6pc	TCTGTCCCGGATCTACCTTG	GAAAGTTTCAGCCACAGCAA
Tdo2	GGGGATCCTCAGGCTATCAT	AATCCACAAAAACCTTGTACCTG
Tat	GGAGGAGGTCGCTTCCTATT	GCCACTCGTCAGAATGACATC

Supporting references

1. Urasaki A, Morvan G, Kawakami K. Functional dissection of the Tol2 transposable element identified the minimal cis-sequence and a highly repetitive sequence in the subterminal region essential for transposition. Genetics 2006;174:639-649.
2. Sugiyama M, Sakaue-Sawano A, Iimura T, Fukami K, Kitaguchi T, Kawakami K, Okamoto H, et al. Illuminating cell-cycle progression in the developing zebrafish embryo. Proc Natl Acad Sci U S A 2009;106:20812-20817.
3. Kwan KM, Fujimoto E, Grabher C, Mangum BD, Hardy ME, Campbell DS, Parant JM, et al. The Tol2kit: a multisite gateway-based construction kit for Tol2 transposon transgenesis constructs. Dev Dyn 2007;236:3088-3099.
4. Snelson CD, Santhakumar K, Halpern ME, Gamse JT. Tbx2b is required for the development of the parapineal organ. Development 2008;135:1693-1702.
5. **Kramer C, Mayr T, Nowak M**, Schumacher J, Runke G, Bauer H, Wagner DS, et al. Maternally supplied Smad5 is required for ventral specification in zebrafish embryos prior to zygotic Bmp signaling. Dev Biol 2002;250:263-279.
6. **Shin D, Shin CH**, Tucker J, Ober EA, Rentzsch F, Poss KD, Hammerschmidt M, et al. Bmp and Fgf signaling are essential for liver specification in zebrafish. Development 2007;134:2041-2050.
7. Choi TY, Ninov N, Stainier DY, Shin D. Extensive conversion of hepatic biliary epithelial cells to hepatocytes after near total loss of hepatocytes in zebrafish. Gastroenterology 2014;146:776-788.
8. Alexander J, Stainier DY, Yelon D. Screening mosaic F1 females for mutations affecting zebrafish heart induction and patterning. Dev Genet 1998;22:288-299.
9. Choi TY, Khaliq M, Ko S, So J, Shin D. Hepatocyte-specific ablation in zebrafish to study biliary-driven liver regeneration. J Vis Exp 2015:e52785.
10. de Groh ED, Swanhart LM, Cosentino CC, Jackson RL, Dai W, Kitchens CA, Day BW, et al. Inhibition of histone deacetylase expands the renal progenitor cell population. J Am Soc Nephrol 2010;21:794-802.
11. **Okabe M, Tsukahara Y, Tanaka M**, Suzuki K, Saito S, Kamiya Y, Tsujimura T, et al. Potential hepatic stem cells reside in EpCAM(+) cells of normal and injured mouse liver. Development 2009;136:1951-1960.
12. Ninov N, Borius M, Stainier DYR. Different levels of Notch signaling regulate quiescence, renewal and differentiation in pancreatic endocrine progenitors. Development 2012;139:1557-1567.
13. Mosimann C, Kaufman CK, Li P, Pugach EK, Tamplin OJ, Zon LI. Ubiquitous transgene expression and Cre-based recombination driven by the ubiquitin promoter in zebrafish. Development 2011;138:169-177.
14. Ninov N, Hesselson D, Gut P, Zhou A, Fidelin K, Stainier DYR. Metabolic Regulation of Cellular Plasticity in the Pancreas. Current Biology 2013;23:1242-1250.
15. Cheung ID, Bagnat M, Ma TP, Datta A, Evason K, Moore JC, Lawson ND, et al. Regulation of intrahepatic biliary duct morphogenesis by Claudin 15-like b. Developmental Biology 2012;361:68-78.
16. Pyati UJ, Cooper MS, Davidson AJ, Nechiporuk A, Kimelman D. Sustained Bmp signaling is essential for cloaca development in zebrafish. Development 2006;133:2275-2284.
17. Shimizu N, Kawakami K, Ishitani T. Visualization and exploration of Tcf/Lef function using a highly responsive Wnt/beta-catenin signaling-reporter transgenic zebrafish. Developmental Biology 2012;370:71-85.

Author names in bold designate shared co-first authorship.