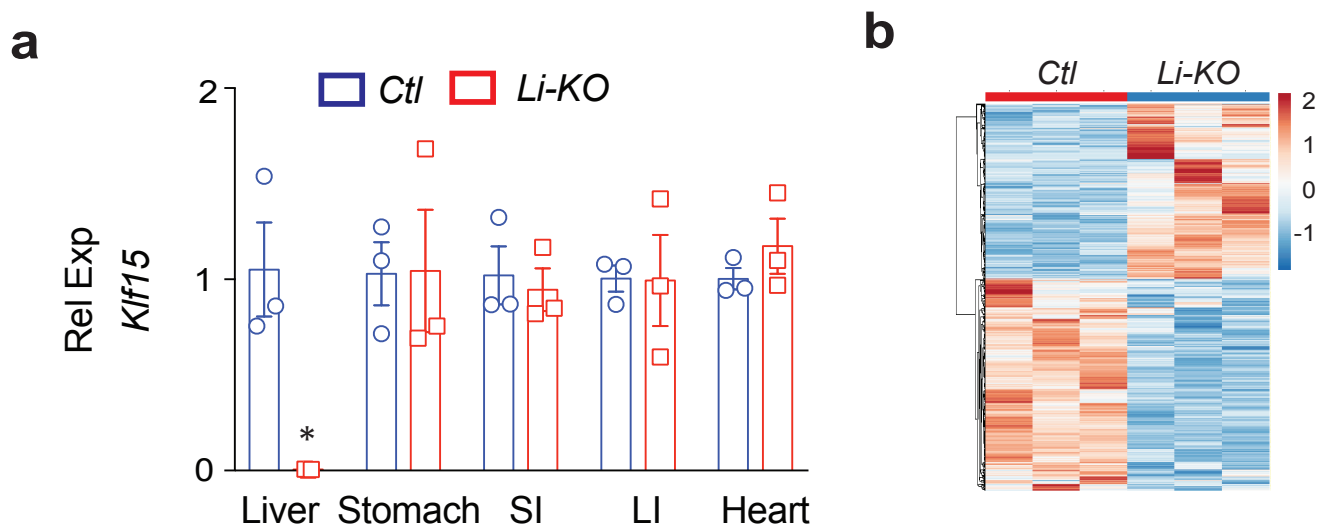


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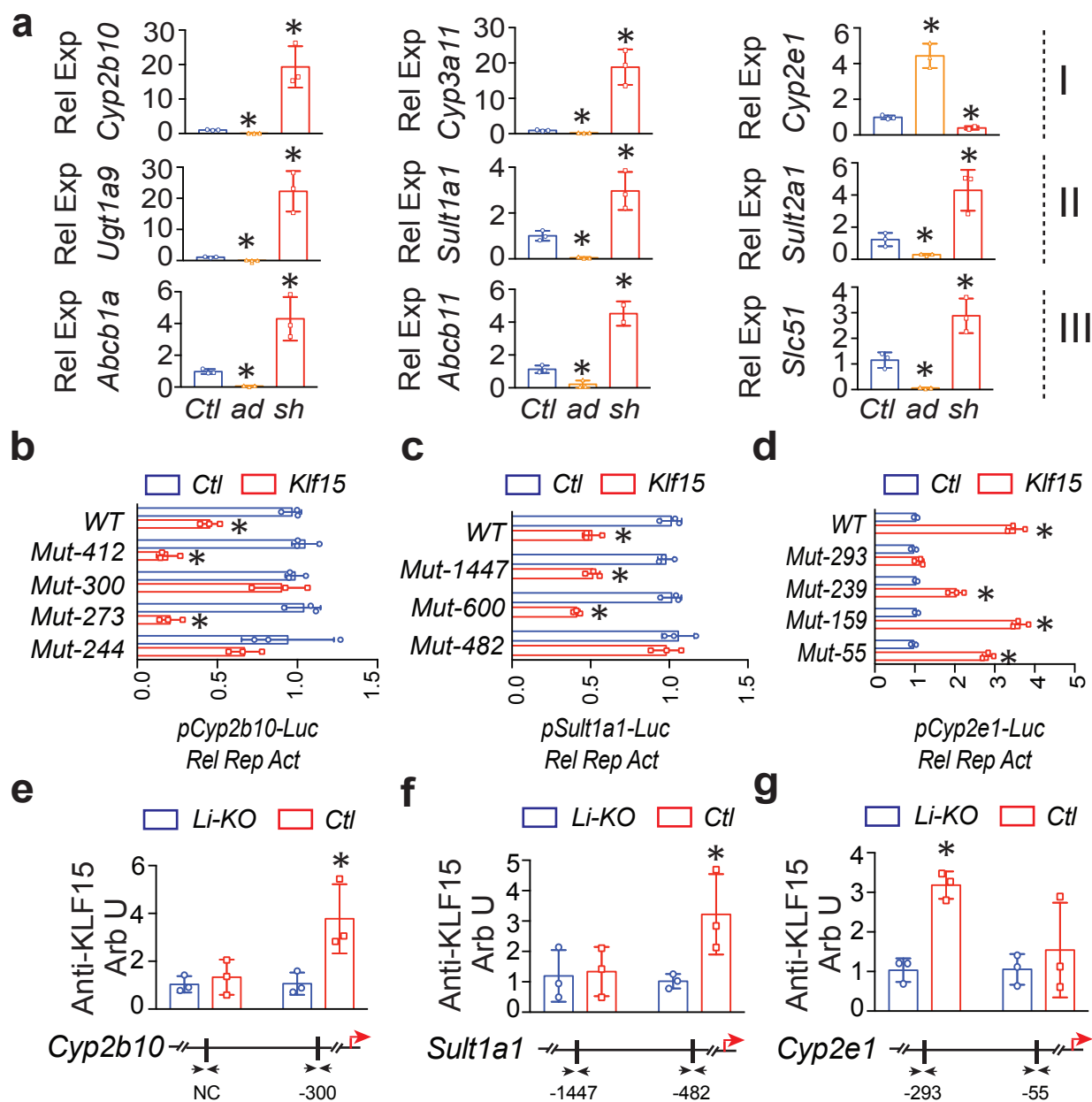
KLF15 regulates endobiotic and xenobiotic metabolism

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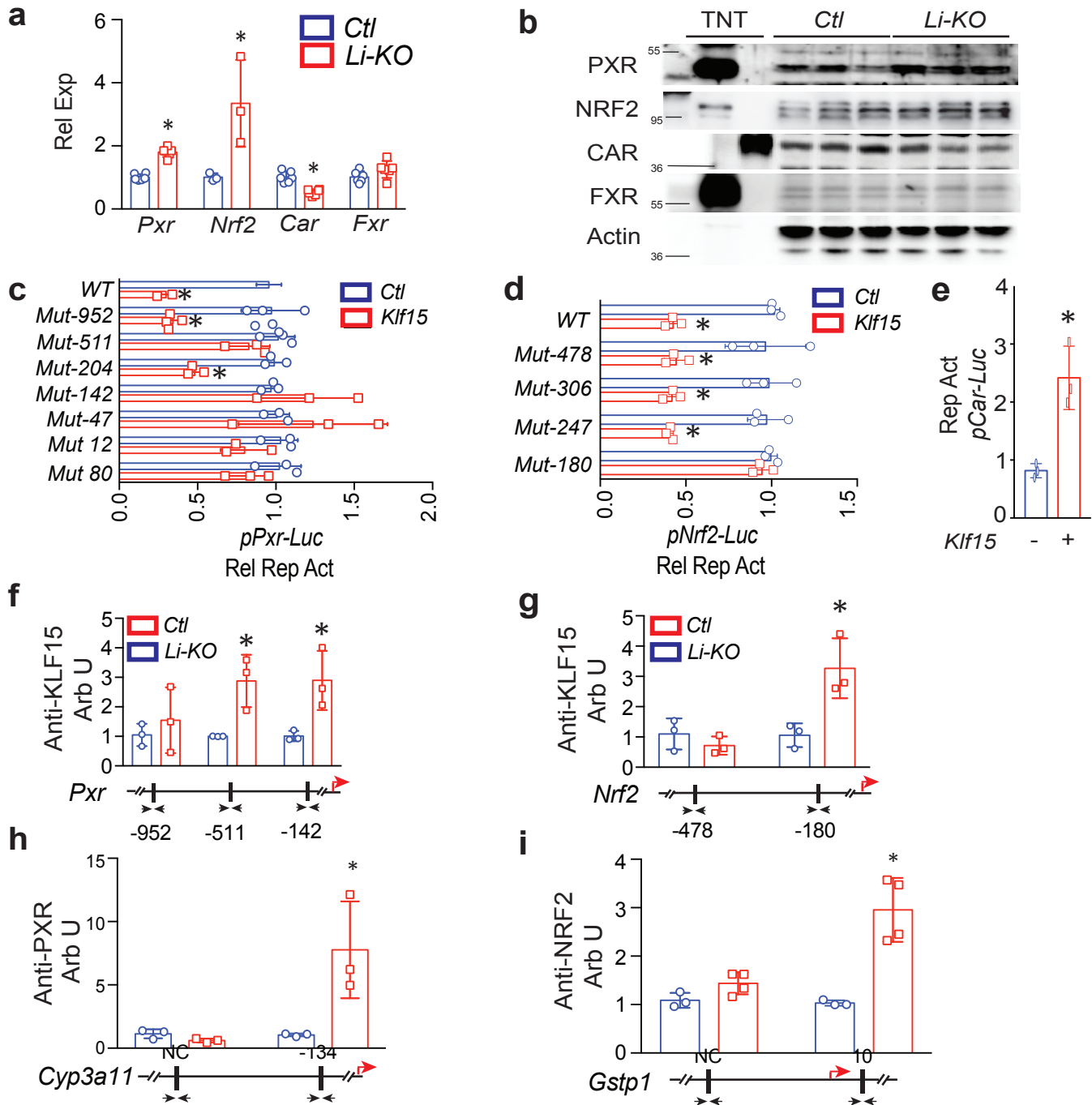
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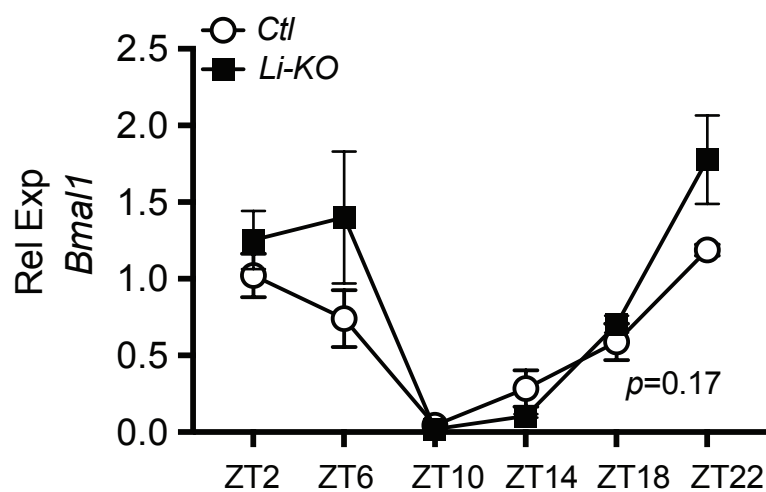
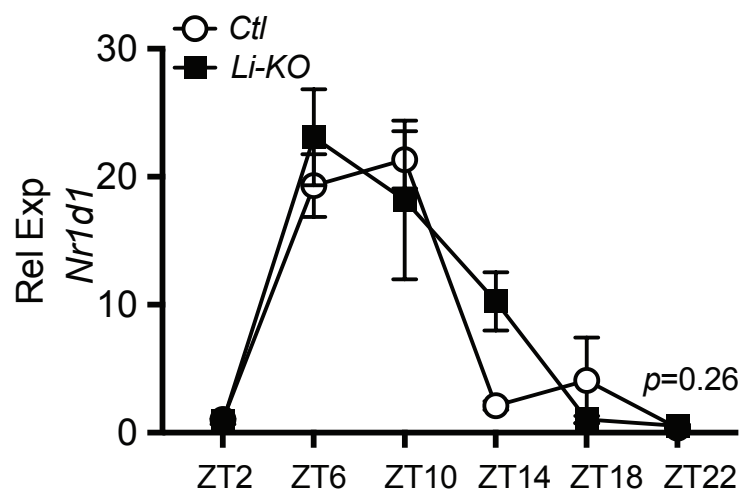
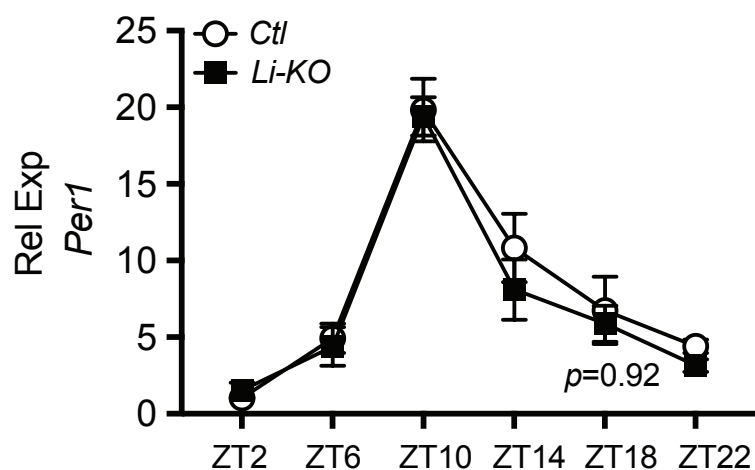
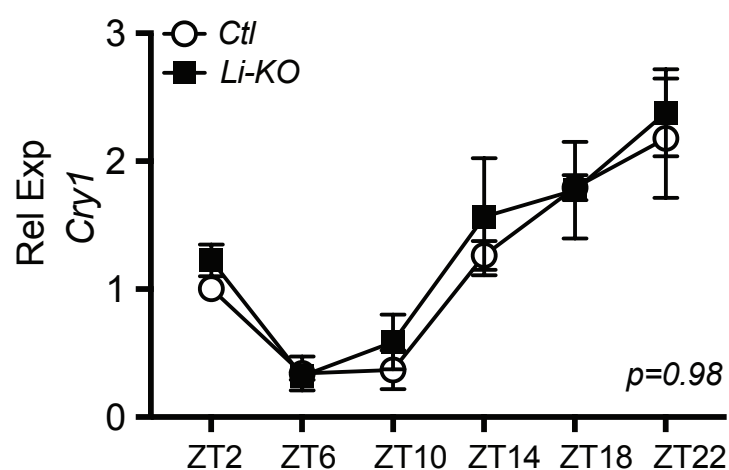
Supplementary Figure 1. Liver-specific *Klf15* deficiency. (a) *Klf15* mRNA expression in multiple organs including liver ($p=0.013$, compared to *Ctl*), stomach ($p=0.97$), small intestine (SI, $p=0.71$), large intestine (LI, $p=0.97$), and heart ($p=0.33$) from *Ctl* and *Li-KO* mice indicated specificity of hepatic knockout ($n=3$ biologically independent animals using two-tailed t test). Error bars are representative of mean $\pm$ SD. (b) Hierarchical heatmap clustering of differentially expressed genes ($p<0.05$) from RNA-seq of *Ctl* and *Li-KO* livers. The p -values are from a one-tailed Fisher's exact test based on a hypergeometric distribution and corrected for multiple-testing using a false-discovery rate (FDR) calculation ($n=3$ biologically independent animals). Normal distribution was assumed for all experimental groups. * indicates $p<0.05$, compared to *Ctl* mice.



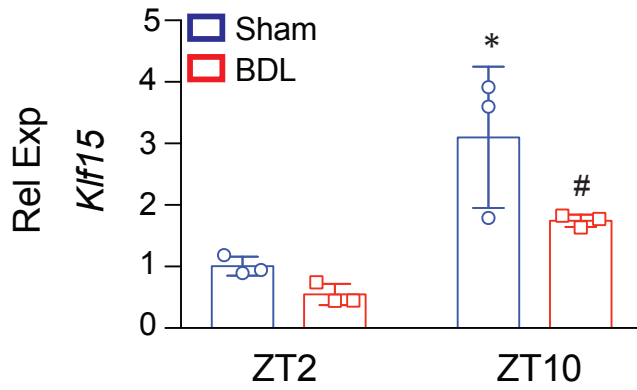
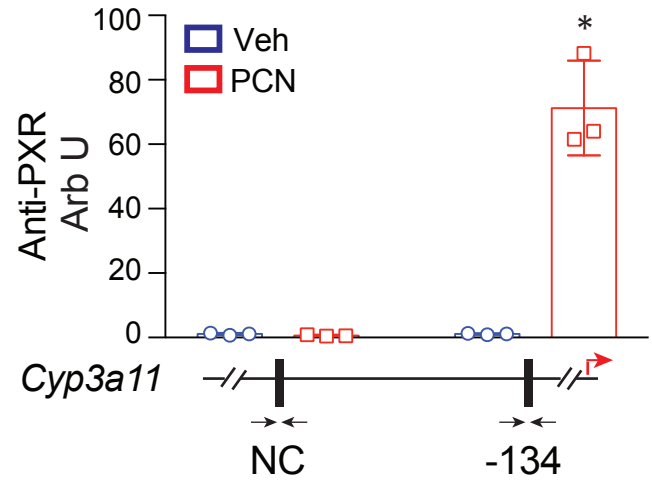
Supplementary Figure 2. KLF15 broadly regulates phase I-III genes. (a) qPCR of phase I-III genes in mouse primary hepatocytes after control (Ctl) infection, or adenoviral (ad) overexpression or shRNA (sh) knockdown of Klf15. *Cyp2b10*, $p=0.0003$ or 0.0062 ; *Cyp3a11*, $p=0.003$ or 0.004 ; *Cyp2e1*, $p=0.001$ or 0.009 ; *Ugt1a9*, $p=0.002$ or 0.005 ; *Sult1a1*, $p=0.002$ or 0.017 ; *Sult2a1*, $p=0.018$ or 0.017 ; *Abcb1a*, $p=0.0005$ or 0.014 ; *Abcb11*, $p=0.008$ or 0.004 ; *Slc51*, $p=0.003$ or 0.015 (compared to Ctl, $n=3$ biologically independent samples using two-tailed t test). (b-d) Luciferase reporter assays of mouse *Cyp2b10*, *Sult1a1*, and *Cyp2e1* luciferase (pCyp2b10-Luc, pSult1a1-Luc, pCyp2e1-Luc) WT and mutated constructs in Hepa1-6 cell line with overexpression of pcDNA3.1 (Ctl) or mouse Klf15. pCyp2b10-Luc: WT ($p=0.0005$), -412 ($p=0.0001$), -300 ($p=0.48$), -273 ($p=0.0004$), -244 ($p=0.2$); pSult1a1-Luc: WT ($p=0.0006$), -1447 ($p=0.0003$), -600 ($p=0.0001$), -482 ($p=0.4$); pCyp2e1-Luc: WT ($p<0.0001$), -293 ($p=0.13$), -239 ($p=0.001$), -159 ($p<0.0001$), -55 ($p<0.0001$). Compared to Ctl, $n=3$ biologically independent samples using two-tailed t test. (e-g) Chromatin immunoprecipitation (ChIP) of KLF15 occupancy on *Cyp2b10* (5'-300 site, $p=0.04$), *Sult1a1* (5'-482 site, $p=0.047$), and *Cyp2e1* (5'-293 site, $p=0.0012$) genes in Ctl and Li-KO mice (compared to Li-KO mice, $n=3$ biologically independent animals using two-tailed t test). Normal distribution was assumed for all experimental groups. * indicates $p < 0.05$, compared with Ctl construct, or Li-KO mice. Error bars are representative of mean $\pm$ SD.



Supplementary Figure 3. KLF15 regulates important EXM transcription factors. (a) qPCR analysis of mRNA expression for *Pxr* ($p<0.0001$), *Nrf2* ($p=0.043$), *Car* ($p=0.0002$), and *Fxr* ($p=0.1$) in *Ctl* vs. *Li-KO* mice ($n=6$ or 3 biologically independent animals using two-tailed *t* test). (b) Immunoblotting analysis of protein expression for PXR, NRF2, CAR, and FXR in *Ctl* vs. *Li-KO* mice ($n=3$ biologically independent animals). (c-e) Luciferase reporter assays of mouse *Pxr*, *Nrf2*, and *Car* luciferase (*pPxr-Luc*, *pNrf2-Luc*, *pCar-Luc*) WT and mutated constructs in Hepa1-6 cell line with overexpression of *pcDNA3.1* (*Ctl*) or mouse *Klf15*. *pPxr-Luc*: WT ($p=0.0003$), -952 ($p=0.005$), -511 ($p=0.13$), -204 ($p=0.0006$), -142 ($p=0.28$), -47 ($p=0.46$), +12 ($p=0.102$), +80 ($p=0.15$); *pNrf2-Luc*: WT ($p<0.0001$), -478 ($p=0.02$), -306 ($p=0.003$), -247 ($p=0.001$), -180 ($p=0.28$); *pCar-Luc* ($p=0.0077$). Compare to *Ctl*, $n=3$ biologically independent samples. (f) and (g) ChIP of KLF15 on *Pxr* (5'-511 site, $p=0.02$; 5'-142 site, $p=0.03$) and *Nrf2* (5'-180 site, $p=0.02$) target genes in *Ctl* and *Li-KO* mice (compared to *Li-KO* mice, $n=3$ biologically independent animals using two-tailed *t* test). (h) ChIP of PXR binding at -134 in *Cyp3a11* promoter (5'-134 site, $p=0.04$) and (i) ChIP of NRF2 binding at +10 in *Gstp1* promoter (5'+10 site, $p=0.004$) in *Ctl* vs. *Li-KO* mice (compared to *Li-KO* mice, $n=3$ biologically independent animals using two-tailed *t* test). Normal distribution was assumed for all experimental groups. * indicates $p < 0.05$, compared with *Ctl* construct, or *Ctl* or *Li-KO* mice. Error bars are representative of mean $\pm$ SD.

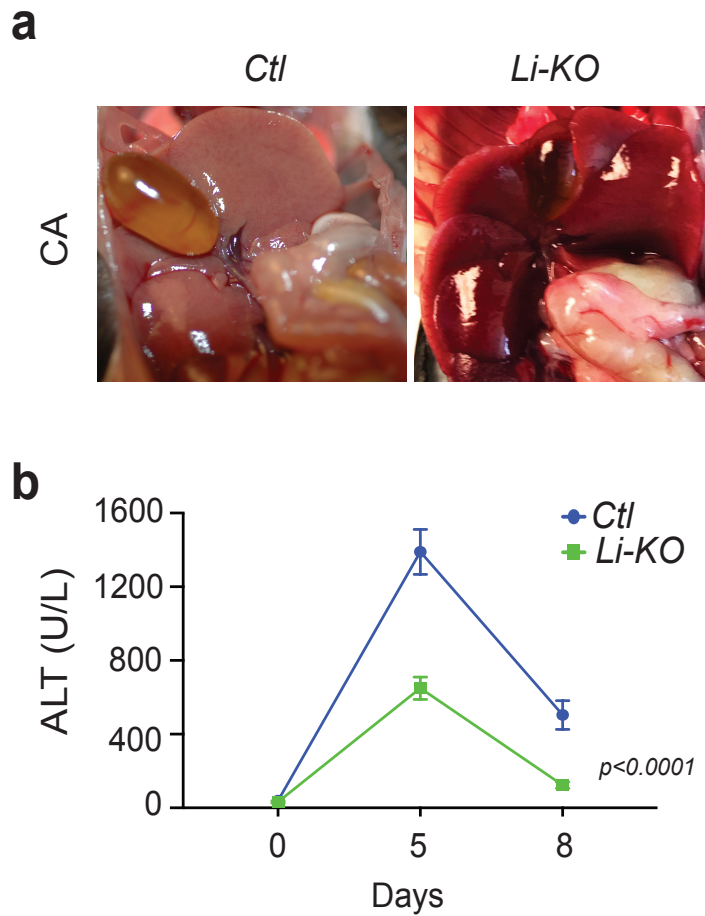
a**b****c****d**

Supplementary Figure 4. KLF15 deficiency has no effect on mRNA expression of known circadian rhythm transcription factors. (a-d) qPCR analysis of *Bmal1*, *Nr1d1*, *Per1*, and *Cry1* mRNA in *Ctl* and *Li-KO* mouse livers ZT2, ZT6, ZT10, ZT14, ZT18, ZT22 (compared to *Ctl* mice, n=3 biologically independent animals). Statistical significance of circadian rhythm for each genotype was analyzed using two-way ANOVA. Error bars are representative of mean $\pm$ SD.

a**b**

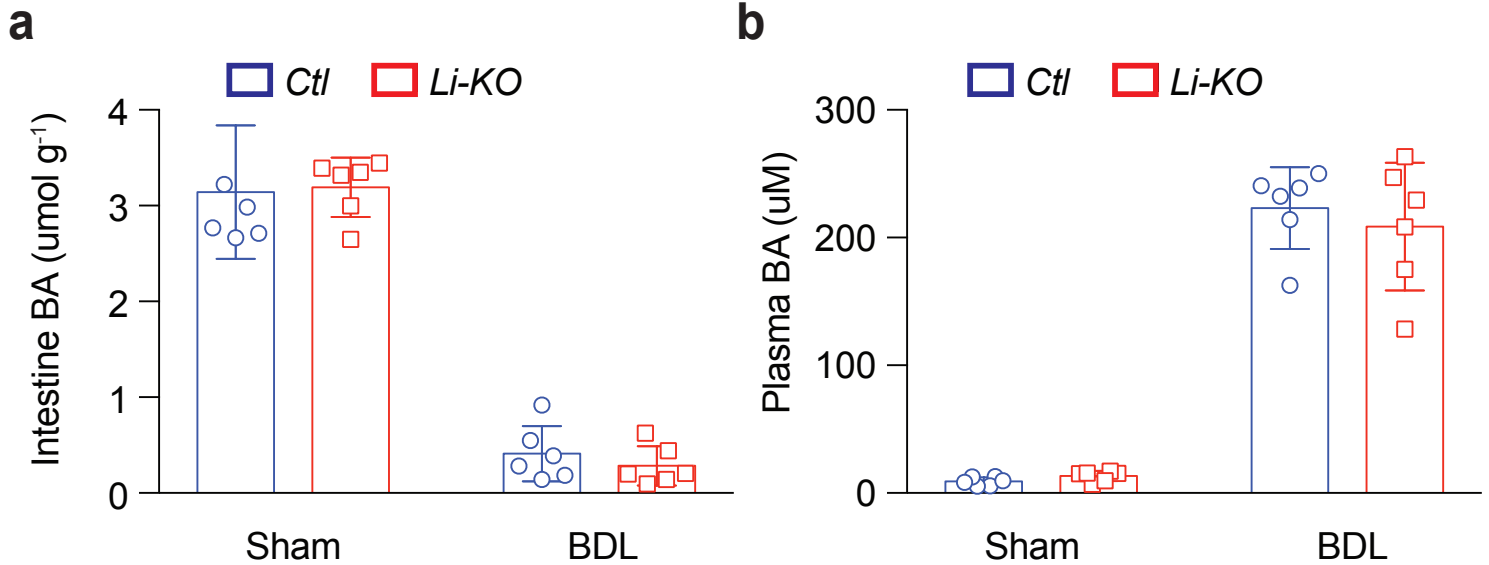
Supplementary Figure 5. The circadian rhythm of *Klf15* was maintained under the post-BDL condition.

(a) qPCR analysis of *Klf15* mRNA expression in sham vs. BDL *Ctl* mice at ZT2 and ZT10. $p=0.035$ or 0.0005 , ZT10 sham or ZT10 BDL compared to ZT2 sham or ZT2 BDL, $n=3$ biologically independent animals using two-tailed t test. (b) ChIP of PXR binding to the motif at -134 in *Cyp3a11* promoter in *Ctl* mice treated with either Veh or PCN (100 mg/kg). $p=0.001$, compared to Veh. $n = 3$ biologically independent animals using two-tailed t test. Normal distribution was assumed for all experimental groups. * indicates $p<0.05$ compared to ZT2 sham or Veh-treated *Ctl* mice. # indicates $p<0.05$ compared to ZT2 BDL mice. Error bars are representative of mean $\pm$ SD.

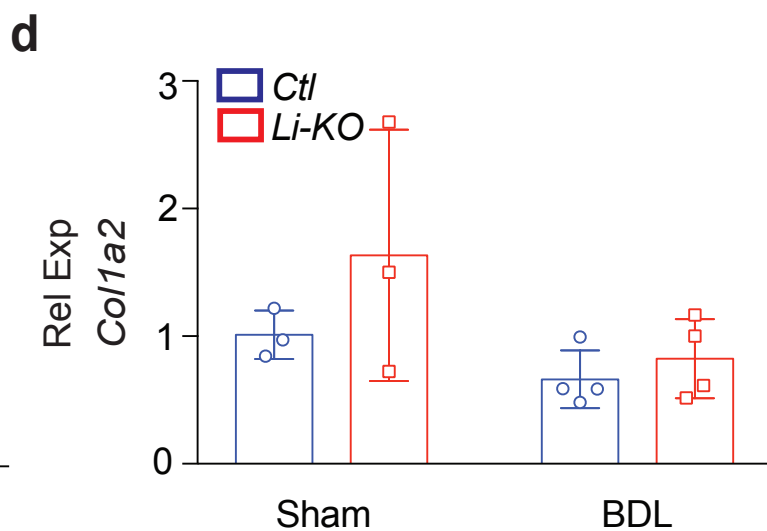
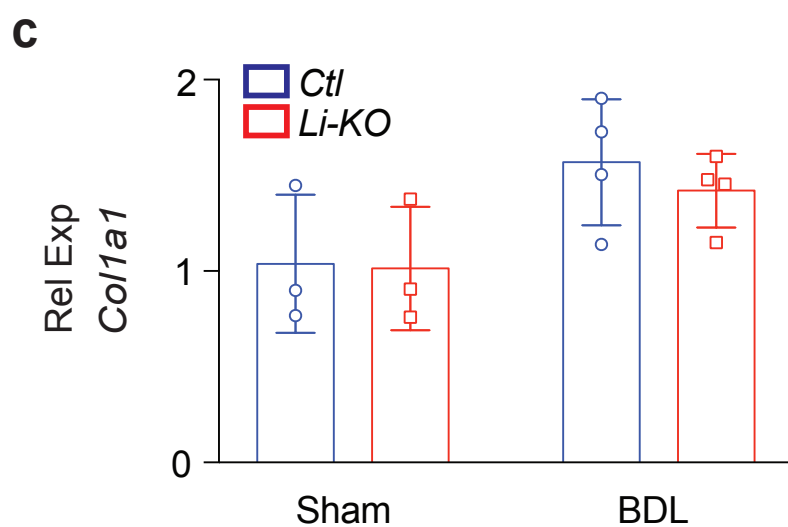
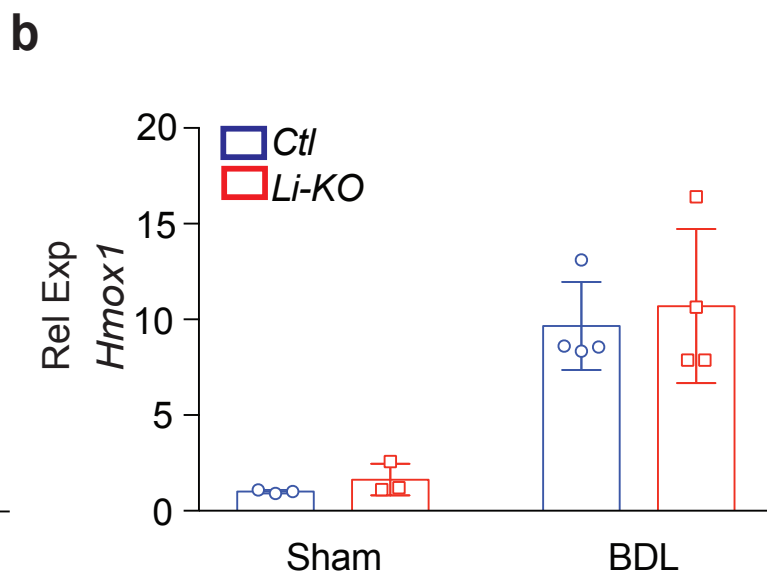
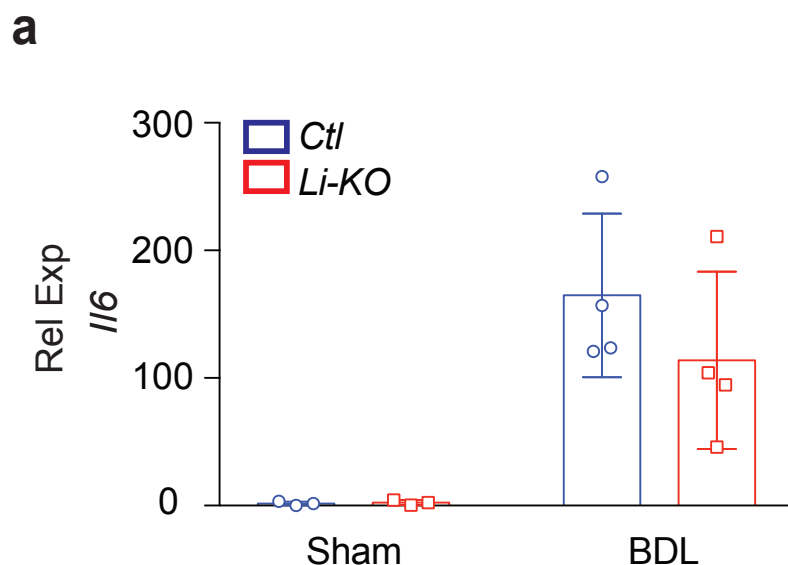


Supplementary Figure 6. Hepatic *Klf15* deficiency resists bile acid diet-induced toxicity.

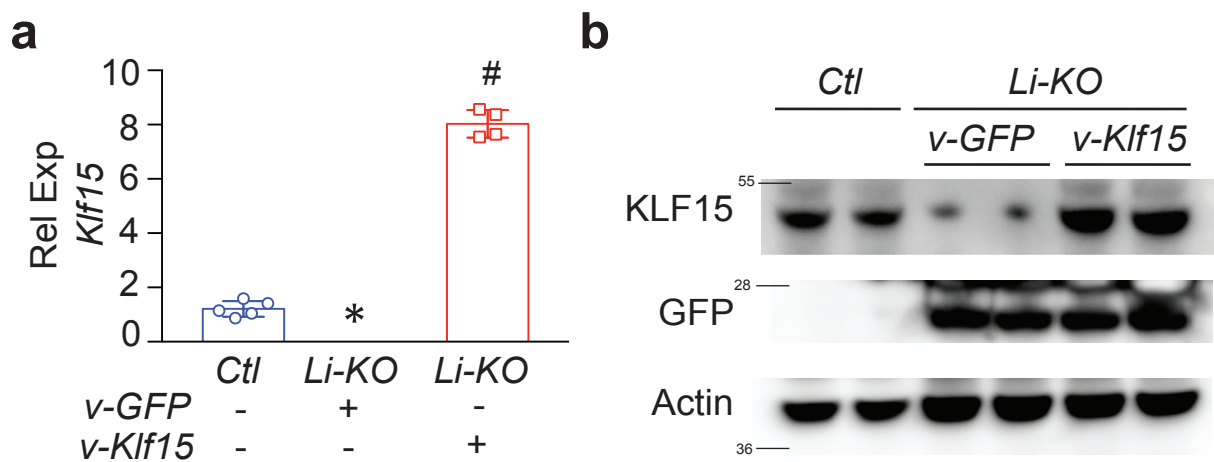
(a) Representative images of gross liver tissue integrity in control (*Ctl*) and *Li-KO* mice five days following a 1% cholic acid (CA) diet (n=5 biologically independent animals). (b) Liver function tests measured ALT blood levels in *Ctl* and *Li-KO* mice consuming a CA diet at 0 , 5 day, and 8 day (compared to *Ctl* mice, n=4 or 5 biologically independent animals using two-way ANOVA). Error bars are representative of mean $\pm$ SD.



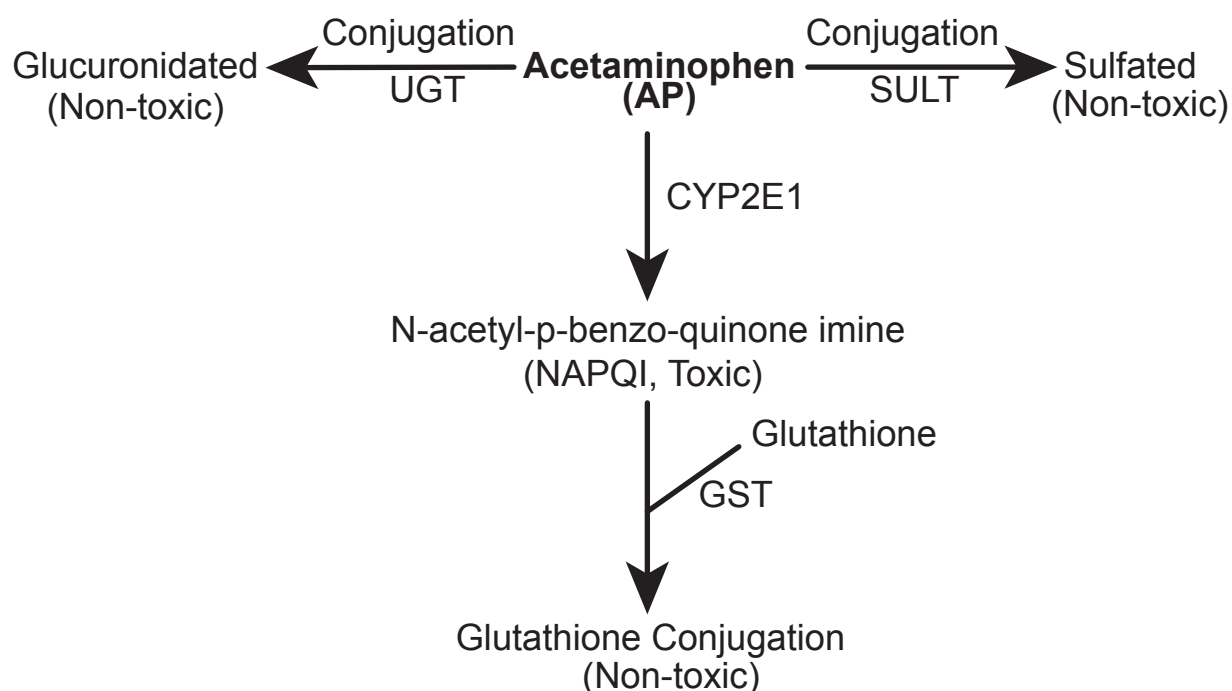
Supplementary Figure 7. Hepatic *Klf15* deficiency has no effect on intestine or blood bile acid fractions under sham or BDL conditions. (a) Intestine BA levels in *Ctl* vs. *Li-KO* mice one day following sham ($p=0.88$, compared to *Ctl*) or BDL surgery ($p=0.4$, compared to *Ctl*). (b) Plasma BA levels in *Ctl* vs. *Li-KO* mice one day following sham ($p=0.08$, compared to *Ctl*) or BDL surgery ($p=0.56$, compared to *Ctl*). $n=6$ biologically independent animals using two-tailed t test. Normal distribution was assumed for all experimental groups. Error bars are representative of mean $\pm$ SD.



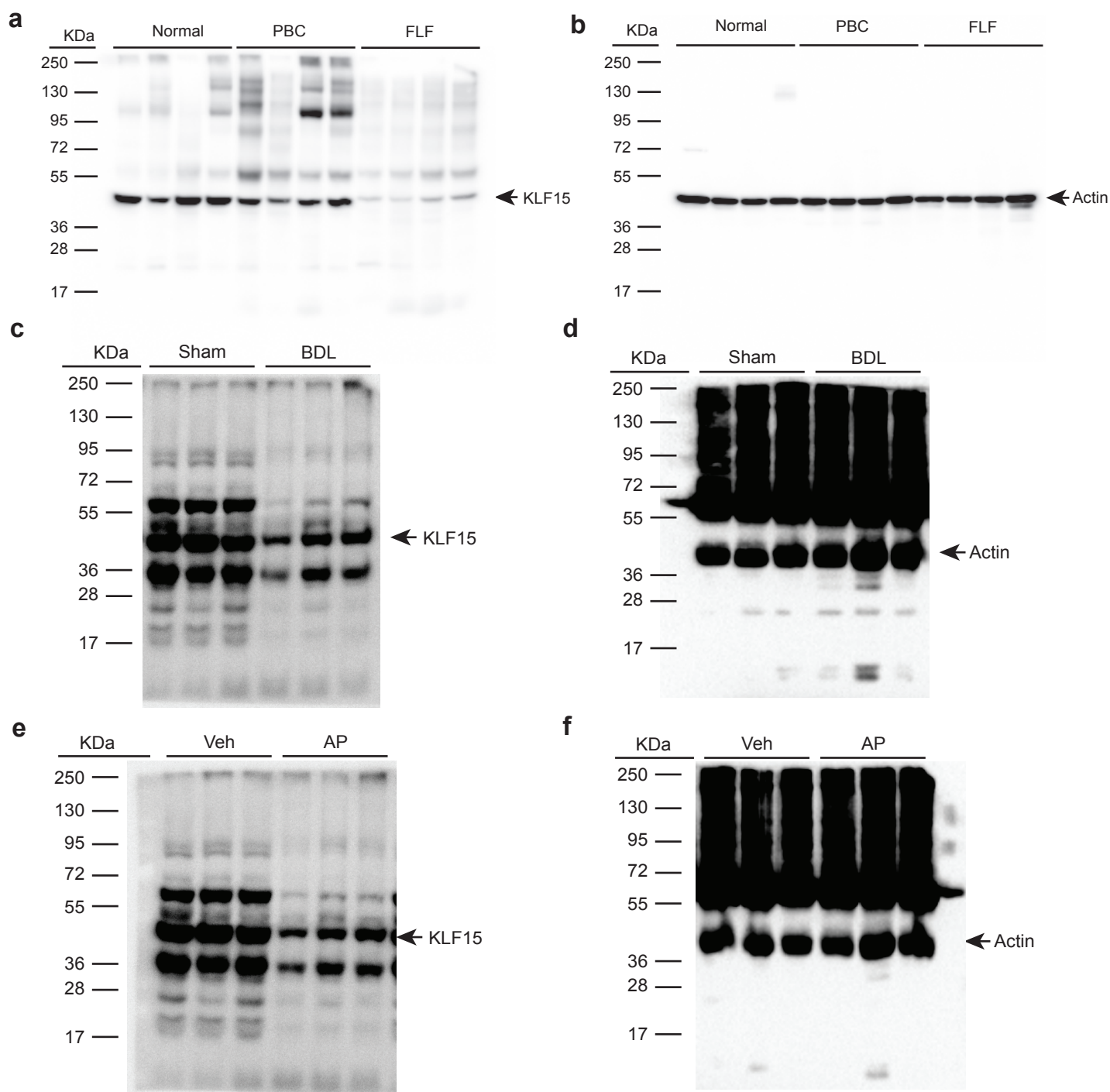
Supplementary Figure 8. Hepatic *Klf15* deficiency has no effect on inflammatory, oxidative and fibrotic markers at the early post-BDL stage. (a-d) qPCR analysis of mRNA expressions of inflammatory, oxidative, and fibrotic markers *Il6*, *Hmox1*, *Col1a1* and *Col1a2* in *Ctl* and *Li-KO* mouse livers one day following sham (*Il6*, $p=0.64$; *Hmox1*, $p=0.26$; *Col1a1*, $p=0.93$; *Col1a2*, $p=0.34$, compared to *Ctl*) or BDL surgery (*Il6*, $p=0.32$; *Hmox1*, $p=0.67$; *Col1a1*, $p=0.47$; *Col1a2*, $p=0.43$, compared to *Ctl*). $n=3$ or 4 biologically independent animals using two-tailed t test. Normal distribution was assumed for all experimental groups. Error bars are representative of mean $\pm$ SD.



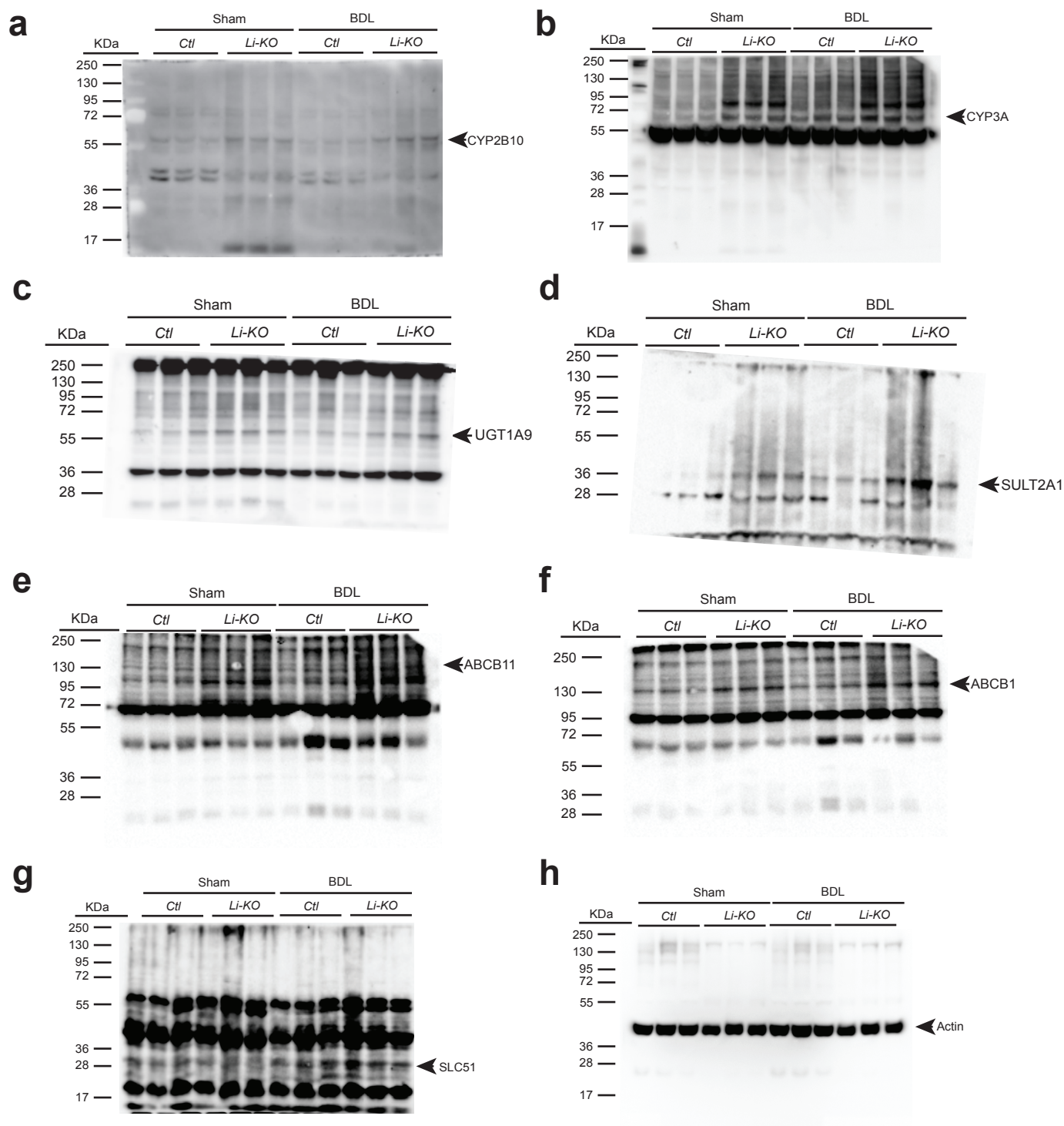
Supplementary Figure 9. Confirmation of viral re-expression of KLF15 in *Li-KO* mouse liver. (a) qPCR analysis of *Klf15* mRNA expression in *Ctl* and *Li-KO* mice with either *v-GFP* ($p < 0.0001$, compared to *Ctl*) or *v-Klf15* ($p < 0.0001$, compared to *Ctl* or *Li-KO* with *v-GFP*) overexpression. $n = 4$ or 5 biologically independent animals using two-tailed t test. Normal distribution was assumed for all experimental groups. * indicates $p < 0.05$ compared to *Ctl* mice. # indicates $p < 0.05$ compared to *v-GFP*-infected *Li-KO* mice. (b) Immunoblotting analysis of KLF15 protein expression in *Ctl* and *Li-KO* mice with either *v-GFP* or *v-Klf15* overexpression. One representative of five independent repeats. Bar graphs representative of mean $\pm$ SD.



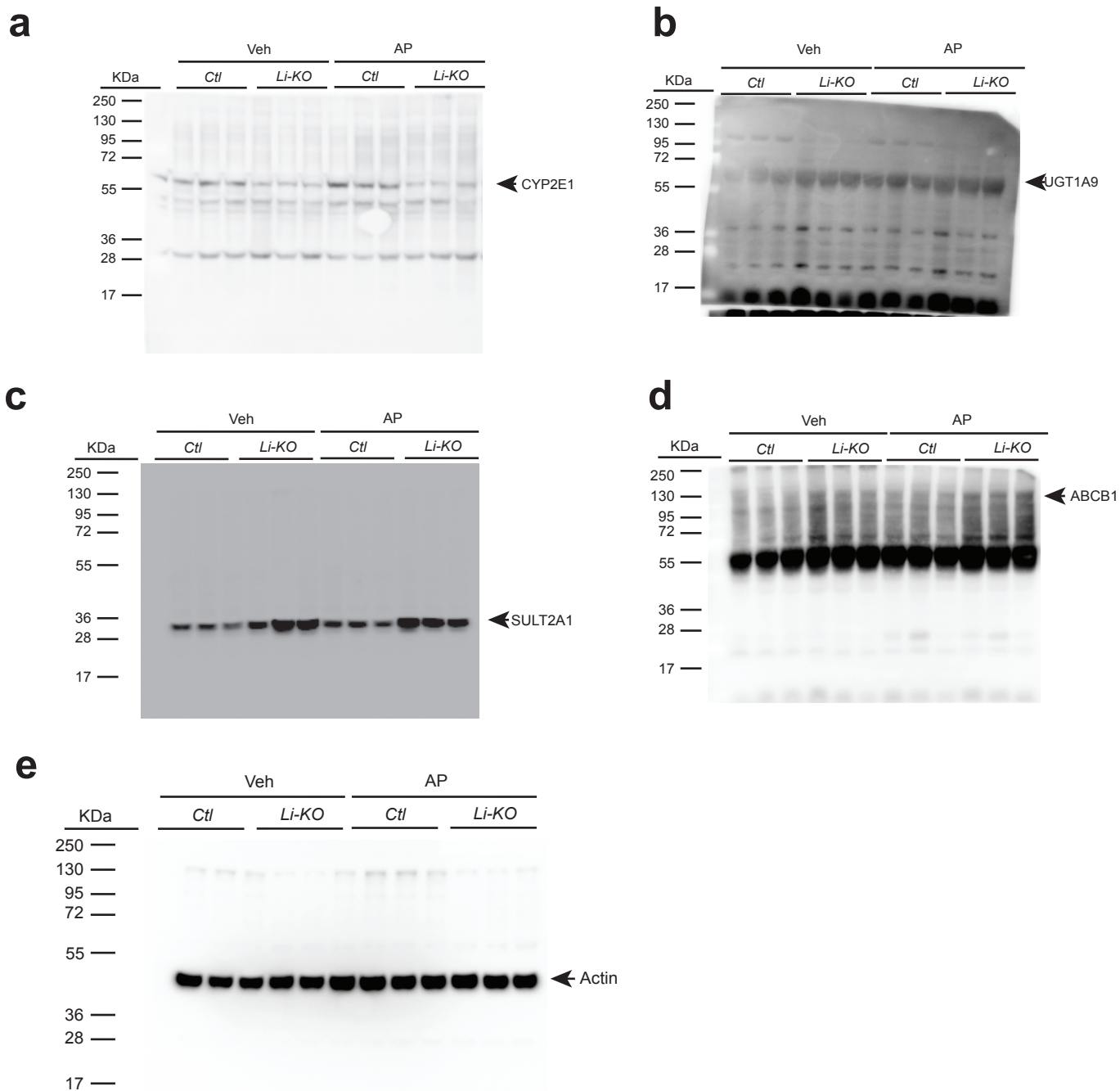
Supplementary Figure 10. EXM biotransformation of acetaminophen. Acetaminophen (AP) is primarily conjugated by phase II enzymes (UGT and SULT) into nontoxic glucuronidated and sulfated molecules. CYP2E1 hydroxylates excessive AP to a reactive and toxic quinone form, N-acetyl-p-benzoquinone imine (NAPQI), which are conjugated and detoxified by phase II enzymes, GSTs. The conjugated metabolites are eliminated in urine.



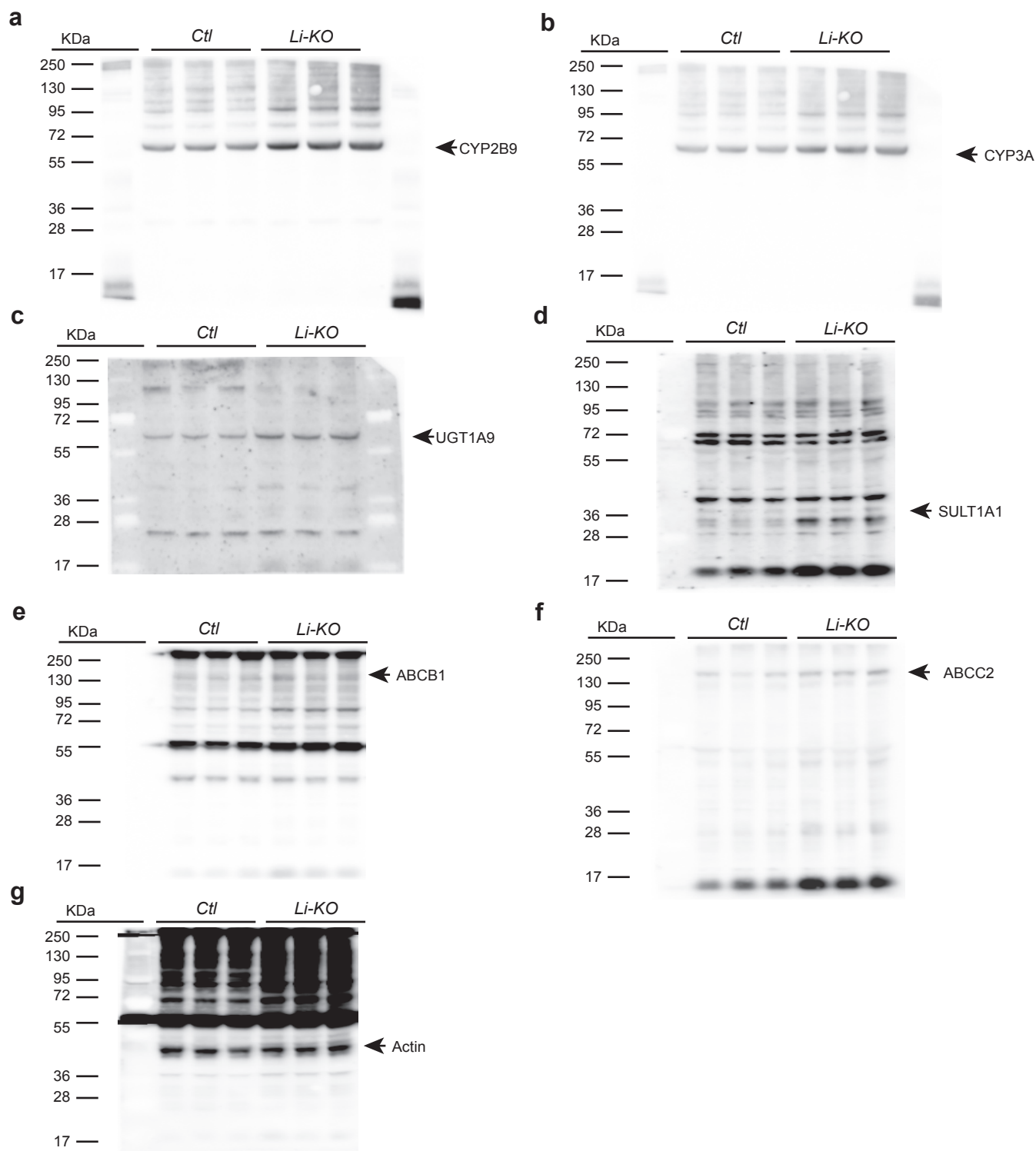
Supplementary Figure 11. Immunoblots of mouse liver KLF15 expression. (a) and (b) Immunoblots of KLF15 and actin protein expression in human liver tissue samples from normal liver donors or patients with PBC or FLF (n=4 biologically independent samples). (c) and (d) Immunoblots of KLF15 and actin protein expression in mouse livers one day following sham vs. BDL (n=3 biologically independent animals). (e) and (f) Immunoblots of KLF15 and actin protein expression in mouse livers one day following Veh vs. AP (750 mg/kg) treatment (n=3 biologically independent animals).



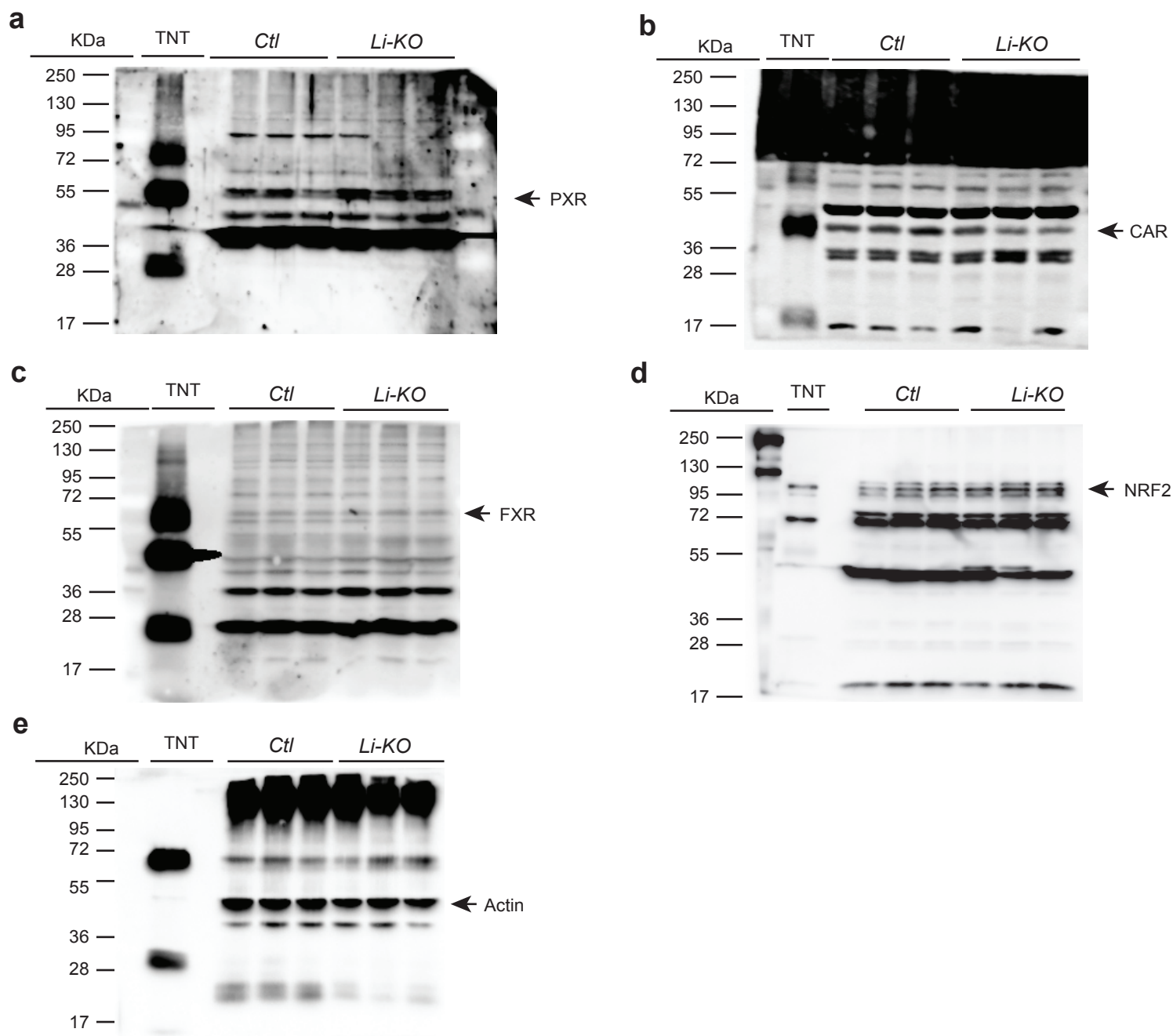
Supplementary Figure 12. Immunoblots of mouse liver CYP2B10, CYP3A, UGT1A9, SULT2A1, ABCB11, ABCB1, and SLC51 expression. (a-h) Immunoblots of CYP2B10, CYP3A, UGT1A9, SULT2A1, ABCB11, ABCB1, SLC51, and actin protein expressions in mouse livers one day following sham vs. BDL (n=3 biologically independent animals).



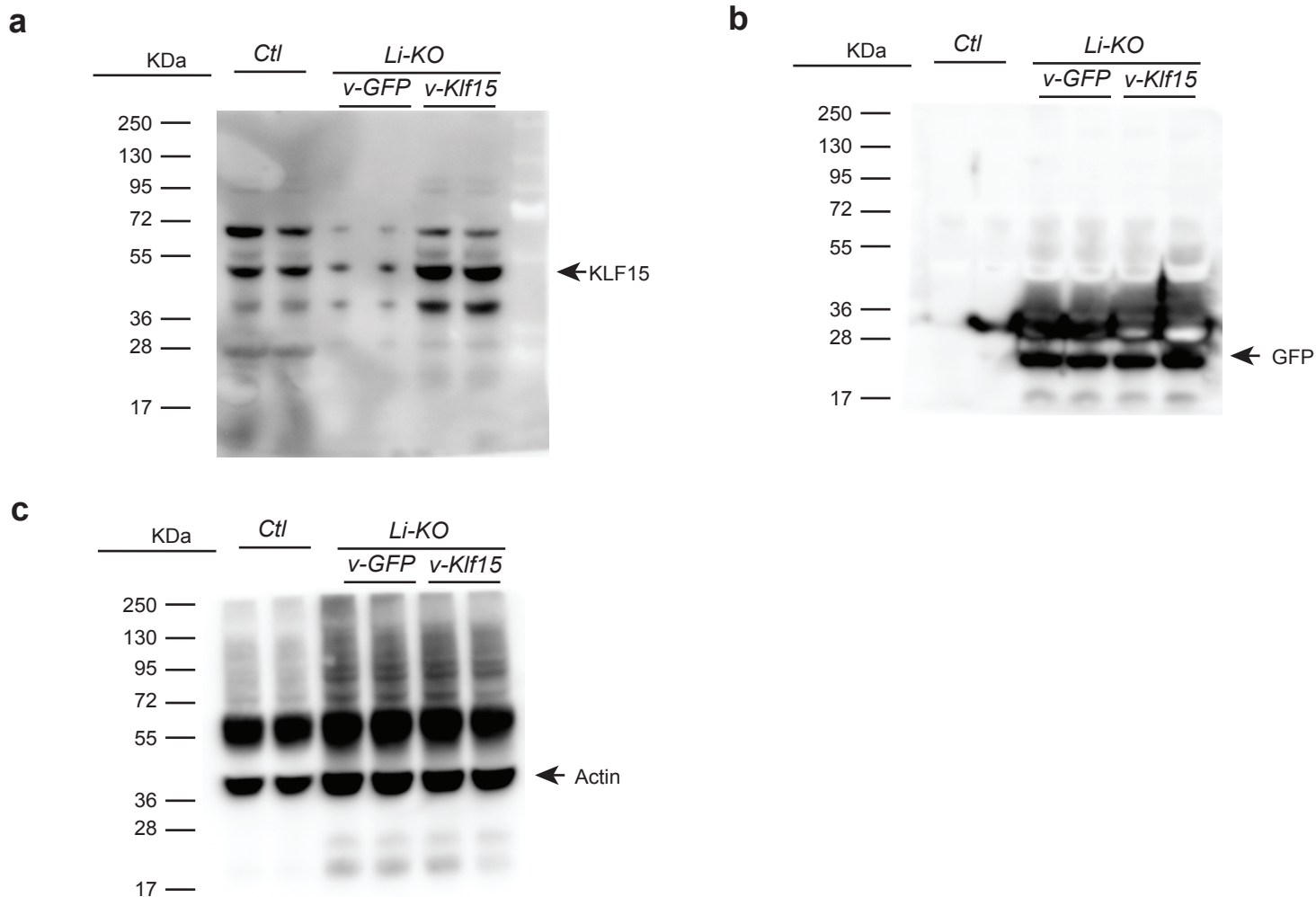
Supplementary Figure 13. Immunoblots of mouse liver CYP2E1, UGT1A9, SULT2A1, and ABCB1 expression. (a-e) Immunoblots of CYP2E1, UGT1A9, SULT2A1, ABCB1, and actin protein expression in mouse livers one day following Veh vs. AP (750 mg/kg, n=3 biologically independent animals).



Supplementary Figure 14. Immunoblots of mouse liver CYP2B9, CYP3A, UGT1A9, SULT1A1, ABCB1, and ABCC2 expression. (a-g) Immunoblots of CYP2B9, CYP3A, UGT1A9, SULT1A1, ABCB1, ABCC2, and actin protein expression in *Ctl* vs. *Li-KO* mice (n=3 biologically independent animals).



Supplementary Figure 15. Immunoblots of mouse liver PXR, NRF2, CAR, and FXR expression. (a-e) Immunoblots of PXR, NRF2, CAR, FXR, and actin protein expression in *Ctl* vs. *Li-KO* mice (n=3 biologically independent animals).



Supplementary Figure 16. Immunoblots of mouse liver KLF15 overexpression.
(a-c) Immunoblots of KLF15, GFP, and actin protein expressions in *Ctl* and *Li-KO* with *v-GFP* or *v-Klf15* overexpression mice. One representative of five independent repeats.

Supplementary Table 1. Primer sequences and probes used for RT-qPCR analysis.

Target	Forward primer	Reverse primer	Probe
<i>hKLF15</i>	caaaagcagccacctaag	tcagagcgcgagaacctc	64
<i>h18S</i>	ccgattggatgggttagtgag	agttcgaccgtcttctcagc	81
<i>Klf15</i>	acagggcgagaagcccttt	catctgagcgggaaaacct	64
<i>18S</i>	aaacggctaccacatccaag	tacagggcctcgaaagagtc	74
<i>Cyp2b9</i>	agcgccaccctccactat	caatctcctttggactttctctg	15
<i>Cyp2b10</i>	tggagctgttctatcagacctt	agccagagaagagctcaaaca	19
<i>Cyp3a11</i>	tgaatatgaaacttgctctcactaaaa	cctgtctgcttaatttcagaggt	12
<i>Ugt1a9</i>	cctgtgatgcctaacatggtc	ggcgttgacataggcttca	16
<i>Sult1a1</i>	caatggagcaactgcagaac	catgatctcactcatccaggtagta	64
<i>Sult2a1</i>	ccaagctgaagccttcgata	aacaacaggaacataaacatcagtgt	2
<i>Abcb11</i>	agtggctgcttttggtggt	cagcgctgagcaaacataag	10
<i>Abcb1a</i>	gggcatttacttcaaacttgta	tttacaagcttcatttctaattcaa	18
<i>Abcc2</i>	caaatccaattctctacatatgcac	gcctgcagtggttgatca	92
<i>Slc51</i>	gctgcccacctctcatactt	gaagaaggcgactggaaagg	18
<i>Slco1a1</i>	ggaaaaccttggaatcactaaaga	ggagcacactcgtaagactgaa	84
<i>Slc10a</i>	tgaagggggacatgaacct	gtagatgtataagaggagaggcatca	78
<i>Cyp2e1</i>	ccaccagcacaactctgagata	cccaataacctgtcaatttctt	92
<i>Cyp2d22</i>	tcagaacgcaccggtaaag	tgctctttgggttctttgg	78
<i>Gstp1</i>	tgtcacctcatctacaccaac	ggacagcagggtctcaaaag	105
<i>Cyp2f2</i>	atcctggaagaaggcagctt	ggataaacacggggtcaaag	67
<i>Cyp2j5</i>	agctgctgcagttactagatgaga	catgggaagccattgtagagt	40
<i>Cyp2u1</i>	aaacttttattccttttgggatagg	catcaggaataactccatcttgg	108
<i>Abcc3</i>	tccgaaactacgcaccagat	ctcatttgcattttgcaagg	79
<i>Pxr</i>	acttcgcaaagtcatactact	aaaagtggcccccttcag	93
<i>Nrf2</i>	catgatggacttggagttgc	cctccaaaggatgtcaatcaa	3
<i>Car</i>	aaatgttggcatgaggaaaga	ctgattcagttgcaaagatgct	98
<i>Fxr</i>	gggatgttggctgaatgtatg	cacattttccttagccgtttag	47
<i>Bmal1</i>	aagtacgcctccccctga	tgtctggagtccctccattt	80
<i>Nr1d1</i>	aggagctgggcctattcac	cggttcttcagcaccagag	1
<i>Per1</i>	aatatgtcaccatggacaccag	ggctgggggagtgaaagac	1
<i>Cry1</i>	ggcagagcagtaactgatacga	tgactttcccaccaactca	52
<i>Il6</i>	gctaccaaactggatataatcagga	ccaggtagctatggtactccagaa	6
<i>Hmox1</i>	aggctaagaccgccttct	tgtgttctctgtcagcatca	17
<i>Col1a1</i>	catgttcagctttgtggacct	gcagctgacttcagggatgt	15
<i>Col1a2</i>	gcagggtcacctactctgtcct	cttgccccattcatttgtct	46

Supplementary Table 2. Primer sequences used for cloning.

Gene promoter	Luciferase Reporter Plasmid	Forward primer	Reverse primer	5' start position	3' end position	Length (bp)	5' restriction enzyme	3' restriction enzyme
<i>Cyp2e1</i>	<i>pGL3-Cyp2e1</i>	ttaggtaccCAGAAGTGAGATTCCTGTTCT	cgatctcgagTGCCGATGGGGCTCCACTCT	-523	17	540	Kpn I	Xho I
<i>Sult1a1</i>	<i>pGL3-Sult1a1</i>	cgatctcgagCGAACGCCCACCCACAAG	gctattcgaaCGGTGCTCAGTGGTAGGG	-1445	31	1476	Xho I	Hind III
<i>Cyp2b10</i>	<i>pGL3-Cyp2b10</i>	cgatctcgagGCCAAATGTAATCACG	gctattcgaaCGGGACTTTGGGTGTCC	-498	100	598	Xho I	Hind III
<i>Pxr</i>	<i>pGL3-Pxr</i>	cgatggtaccGGGAGTGACTCAGGTCCTCT	cgatctcgagCTGCACATGGAGATCTCAGC	-1040	172	1212	Kpn I	Xho I
<i>Nrf2</i>	<i>pGL3-Nrf2</i>	cgatctcgagTGCTGGGCTCTGGGTGGATG	gctattcgaaGGCTACTTGCAGCAGAGGTGAGG	-901	56	957	Xho I	Hind III
<i>Car</i>	<i>pGL3-Car</i>	ttaggtaccGAGCCTGGAGGGTTTGTGTTG	cgatctcgagCCTACCTGTTTCCCTTAGGC	-1029	209	1238	Kpn I	Xho I

Supplementary Table 3. Oligo sequences used for site-directed mutagenesis.

Gene	Binding Protein	Mutant name	5' start position of binding motif	Forward oligo	Reverse oligo
<i>Klf15</i>	PXR	Mut-800	-800	GAAGGAAGGTCTGTCAACCATGTCCTGGGTATTAAG	CTTAATACCCAGGACATGGTGACAGACCTTCCTTC
<i>Klf15</i>	PXR	Mut-271	-271	GTCCCACGTGGTCTATCCTTCGGGACTCTGCG	CGCAGAGTCCCGAAGGATAGACCACGTGGGAC
<i>Klf15</i>	PXR	Mut-202	-202	CATCGGAGGACCCAGCTAAGATCCACGAAGAG	CTCTTCGTGGATCTTAGCTGGGTCCTCCGATG
<i>Klf15</i>	PXR	Mut 93	92	CCAGGCGACGCGGAGCATAGGTGGGAGACGCGC	GCGCGTCTCCACCTATGCACCGCGTCGCCTGG
<i>Cyp2e1</i>	KLF15	Mut-293	-293	GGGAAGACATTCCCCTGACCCCAGGACTGACCTATG	CATAGGTCAGTCCTGGGGTCAGGGGAATGTCTTCCC
<i>Cyp2e1</i>	KLF15	Mut-239	-239	CTACATGTACTCACCAGTGACCTGACAGGTGCCAG	CTGGCACCTGTCAGGTCAGTGGTGAGTACATGTAG
<i>Cyp2e1</i>	KLF15	Mut-159	-159	GATGCAACTTCTAAGCTGCAATGGCTCCAAGG	CCTTGAGCCATTGCAGCTTAGAAGTTGCATC
<i>Cyp2e1</i>	KLF15	Mut-55	-55	GTTATTGGCTGATGAGCTGACCTCCTTCTCAACGAC	GTCGTTGAGAAGGAGGTCAGCTCATCAGCCAATAAC
<i>Sult1a1</i>	KLF15	Mut-1447	-1447	CGAACGCCCATGAACAAGAAAAAAGATG	CATCTTTTTTCTTGTTCATGGGCGTTTCG
<i>Sult1a1</i>	KLF15	Mut-600	-600	CTTGCTTTTGCAGATCATGAAATCTTTCTAGGG	CCCTAGAAAGATTTTCA7GATCTGCAAAGACAAG
<i>Sult1a1</i>	KLF15	Mut-482	-482	CACACTCCCATGAACATGACTCCAACCTCTGTTGC	GCAACAGAGTTGGAGTCATGTTTATGGGAGTGTG
<i>Cyp2b10</i>	KLF15	Mut-412	-412	GACATGTAAATATGACCCAGACATGTAAG	CTTACATGTCTGGGTCATATTTACATGTC
<i>Cyp2b10</i>	KLF15	Mut-300	-300	GCGTACAGCATGACCGCAGTCTCTTGTC	GACAAGAGACTGCGGTCATGCTGTACGC
<i>Cyp2b10</i>	KLF15	Mut-273	-273	CTTGTCCTCAGTAAGTTCACAGACAAAACCTGAG	CTCAGTTTTGTCTGTGAACCTACTGGGACAAG
<i>Cyp2b10</i>	KLF15	Mut-244	-244	GAGCTGACAAGGGCATGACCACCTCCATAAAAC	GTTTTATGGAGGTGGTCATGCCCTTGTGAGCTC
<i>Pxr</i>	KLF15	Mut-952	-952	CTACCACAACTGGTCATTTAACTGGCTTCC	GGAAGCCAGTTTAAATGACCAGTTTGTGGTAG
<i>Pxr</i>	KLF15	Mut-511	-511	CAGGGCCAAGGACATGACGATGATGTCCTGC	GCAGGACATCATCGTCATGTCCTTGGCCCTG
<i>Pxr</i>	KLF15	Mut-204	-204	CTAGAGGCAAAATCATGATCAGAGAGGCAAAC	GTTTGCCTCTCTGA7CATGATTTTGCCTCTAG
<i>Pxr</i>	KLF15	Mut-142	-142	CTTAAACACACTTCCCAGACCTTACATGTTTTAC	GTAAACATGTAAGGTC7GGGAAGTGTGTTTTAAG
<i>Pxr</i>	KLF15	Mut-47	-47	CATTGCTAGCTCGGTCAGTGGACTTGGGAC	GTCCCAAGTCCAGTGACCGAGCTAGCAATG
<i>Pxr</i>	KLF15	Mut 12	12	CTACATTGGACCTGGGTCATACTGTGGGCAAG	CTTGCCACAGTATGACCCAGGTCCAATGTAG
<i>Pxr</i>	KLF15	Mut 80	80	CTAAGGTTAGCTGAGGTCAAGAATCGGGGATAG	CTATCCCCGATTCTTGACCTCAGCTAACCTTAG
<i>Nrf2</i>	KLF15	Mut-478	-478	CTCTAGCAAGGGGTCAGTGTCTACAGAGGAC	GTCCTCTGTAGGACAGTGACCCCTTGCTAGAG
<i>Nrf2</i>	KLF15	Mut-306	-306	CTCAGGTCCATGCCCAAACCTCGGGCTCTTGC	GCAAGAGCCCCGAGG7TTGGGCATGGACCTGAG
<i>Nrf2</i>	KLF15	Mut-247	-247	CCTCCCCTTGCCCAAACCTAGGCCTTTGCGG	CCGCAAAGGCCTAGG7TTGGGCAAGGGGAGG
<i>Nrf2</i>	KLF15	Mut-180	-180	CCAAGCTCTTGCCCAAACCTTACCCCGCCTC	GAGGCGGGGTAAGGG7TTGGGCAAGAGCTTGG

Supplementary Table 4. Primer sequences used for ChIP assay.

Gene	Protein	Antibody Information	Forward primer	Reverse primer	5' starting position	3' ending position	Length (bp)	Binding motif
<i>Klf15</i>	PXR	Santa Cruz Cat #: sc-25381, 200 ug/ml	GGTTTGAAGCCAGGCAGTGTA	GAAACACGTGAGCAGAGAGT	-675	-606	75	NC
<i>Klf15</i>	PXR	Santa Cruz Cat #: sc-25381, 200 ug/ml	CCAGGCGACGCGGAG	TGATCCCCAAGGGAGGACC	81	151	71	GAGTCCAGG
<i>Cyp3a11</i>	PXR	Santa Cruz Cat #: sc-25381, 200 ug/ml	CCACACAGAATGTTAGCTCAAAG	TCTCTTCTCTAGGCAGTTCGTAT	-177	-105	73	ATGAACTATAC
<i>Cyp3a11</i>	PXR	Santa Cruz Cat #: sc-25381, 200 ug/ml	GGGGGACTTTTGGGATAGCAT	GGTTGCCTGTCTGTCCTATGT	-4194	-4102	93	NC
<i>Cyp2e1</i>	KLF15	Abcam Cat #: ab2647, 0.5 mg/ml	GACCAGTGCCATGGGGAAG	TGTAGGAATACCTCACCAATTTCAT	-321	-258	64	CACCC
<i>Cyp2e1</i>	KLF15	Abcam Cat #: ab2647, 0.5 mg/ml	AAGGGAGATGAGTGGTTATTGGC	AAACAAGGATTTTATAACGTGTCGT	-86	-17	70	NC
<i>Sult1a1</i>	KLF15	Abcam Cat #: ab2647, 0.5 mg/ml	GAACGCCCACCCACAAGAAAAA	CCAGCATGTCAAGGGCTAAGAT	-1454	-1372	83	CACCC
<i>Sult1a1</i>	KLF15	Abcam Cat #: ab2647, 0.5 mg/ml	AGACTCACACTCCCATGAACAC	GATGGGAAGGAGCAAGGTCAA	-501	-430	72	CACCC
<i>Cyp2b10</i>	KLF15	Abcam Cat #: ab2647, 0.5 mg/ml	ACACATCCGTACATTCATCTCAG	TACTGGGACAAGAGACTGCG	-356	-277	80	CACCC
<i>Cyp2b10</i>	KLF15	Abcam Cat #: ab2647, 0.5 mg/ml	GAACCCAGGATCACCAAGTCCA	CAGCGATTGGATGTTGGAGGG	-924	-855	70	NC
<i>Pxr</i>	KLF15	Abcam Cat #: ab2647, 0.5 mg/ml	AGTGACTCAGGTCCTCTCTG	GGGCTTTATTTGGAAGCCAG	-1010	-922	89	NC
<i>Pxr</i>	KLF15	Abcam Cat #: ab2647, 0.5 mg/ml	CCCTTGTCAGGCTTCCCAGAT	TCTGGGGTAGCAGGACATCATC	-555	-484	71	CACCC
<i>Pxr</i>	KLF15	Abcam Cat #: ab2647, 0.5 mg/ml	GGCCCAGAATTACTTAGAGCA	TGTCTTCACTGTAAACATGTAAGG	-182	-113	70	CCCCTCCC
<i>Nrf2</i>	KLF15	Abcam Cat #: ab2647, 0.5 mg/ml	GCTCTGGGTGGATGAAGTGATAC	CTCCACTTCTCCCACGTGCC	-895	-825	71	GGGTG
<i>Nrf2</i>	KLF15	Abcam Cat #: ab2647, 0.5 mg/ml	GTACGCGATTCCAAGCTCT	CAAGAGGCAAGGGCCAGA	-199	-123	77	CCCCGCCCC
<i>Gstp1</i>	NRF2	Proteintech Cat#: 16396-1-AP, 70 ug/150 ul	GGTGGCCTACGCGTGC	CTGCTGGGTGCGGATTCA	-11	52	63	TCACTCT
<i>Gstp1</i>	NRF2	Proteintech Cat#: 16396-1-AP, 70 ug/150 ul	GAATTCATTACAGGGCAGCAGG	GGGAGACACCCAAAGATGTACC	-2977	-2897	81	NC