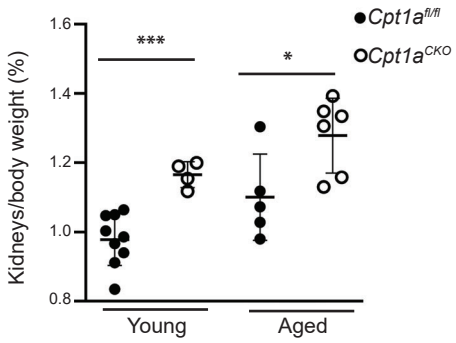
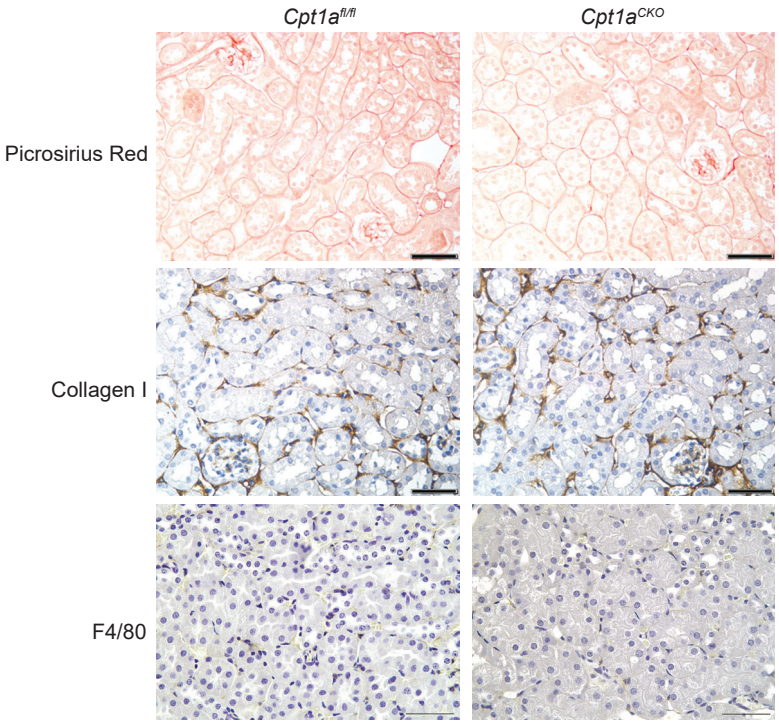
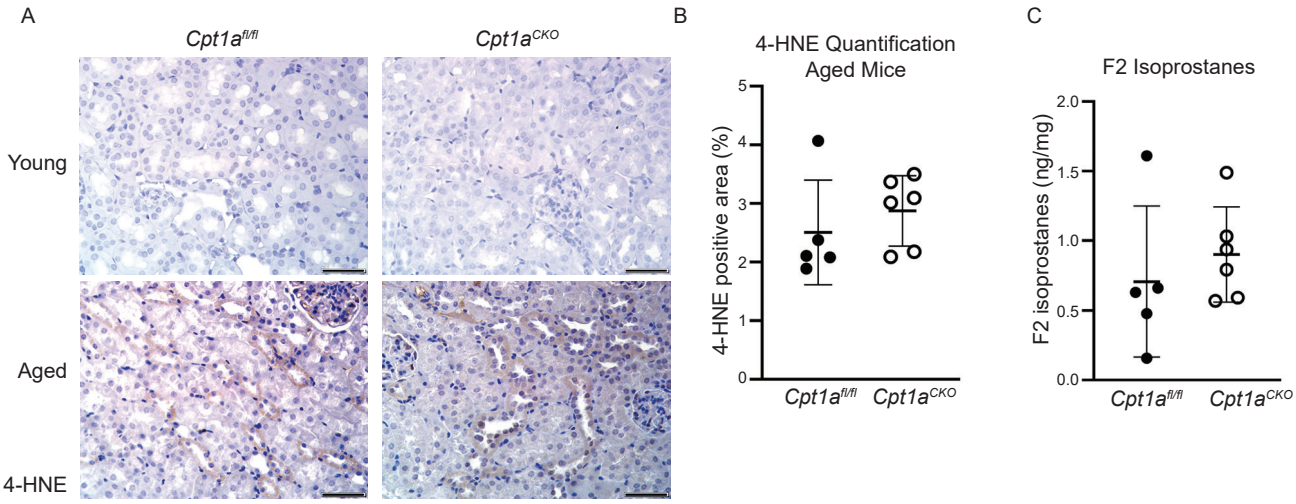


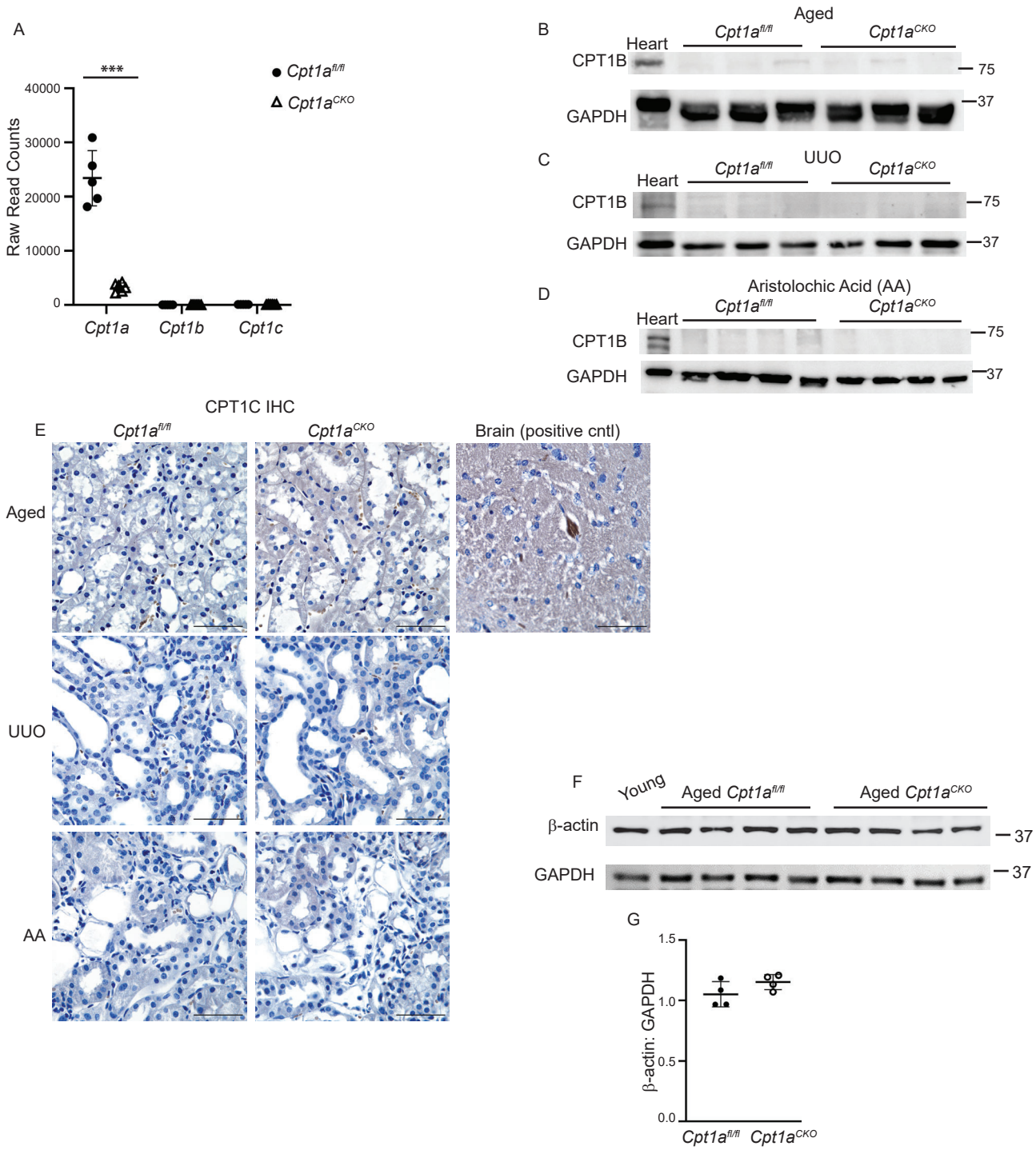
Supplemental Figure S1. Supplemental Figure S1. Cpt1aCKO mice have larger kidneys. Kidney weights expressed as a percentage of body weight in young and aged Cpt1afl/fl and Cpt1aCKO mice. Means are shown +/- SD with * = $p<0.05$ and ** = $p<0.01$.



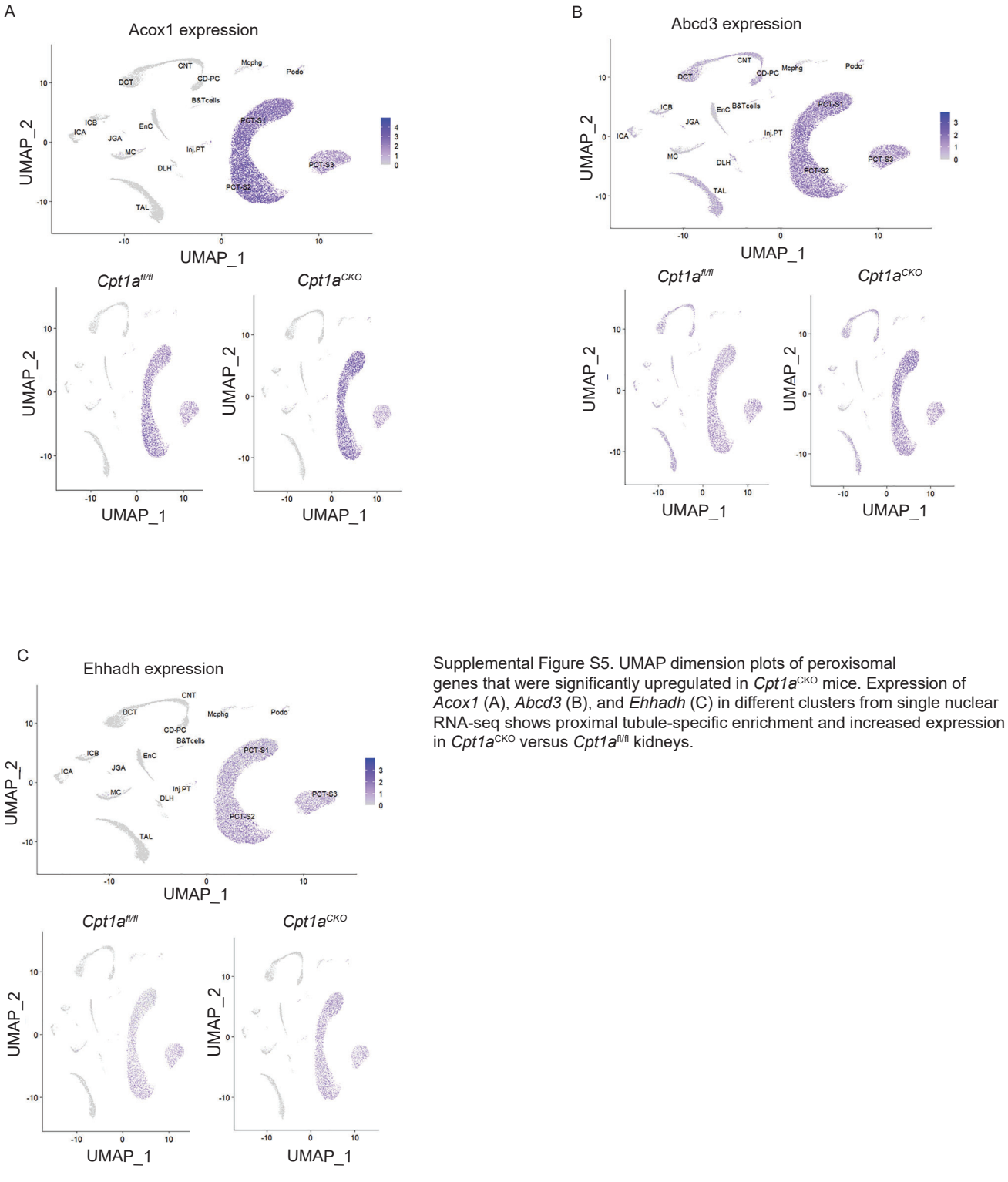
Supplemental Figure S2. Supplemental Figure S2. No difference in fibrosis or inflammation in young *Cpt1a*^{CKO} mice compared to floxed controls. Picrosirius Red staining, IHC of collagen I and F4/80 on kidney tissue of young *Cpt1a*$^{fl/fl}$ and *Cpt1a*^{CKO} mice. Scale bars = 50μM.



Supplemental Figure S3. No difference in 4-HNE or F2 isoprostanes between genotypes. 4-HNE staining was done on aged kidney tissues and quantified using ImageJ (A, B). Urine F2 isoprostanes were measured from aged mice and reported normalized to creatinine (C). Scale bars = 50μM.



Supplemental Figure 4. Supplemental Figure S4. *Cpt1a*^{CKO} kidneys do not have compensatory upregulation of *Cpt1b* and *Cpt1c*. (A) Transcript levels of *Cpt1a*, *Cpt1b*, and *Cpt1c* from RNAseq are shown for each genotype. There was only a statistical difference between genotypes in *Cpt1a* levels, means +/- SD shown, with p value calculated using the DESeq2 package. Immunoblots for CPT1B on kidney tissue lysates from aged (B), UUO-injured (C) or AAN-injured (D) mice with heart as a positive control. (E) Immunohistochemistry for CPT1C with brain as positive control. As heart does not express β-actin, we used GAPDH for loading control and show that GAPDH and β-actin have no significant differences as loading controls when CPT1A is deleted (F, G).



Supplemental Figure S5. UMAP dimension plots of peroxisomal genes that were significantly upregulated in *Cpt1a*^{CKO} mice. Expression of *Acox1* (A), *Abcd3* (B), and *Ehhadh* (C) in different clusters from single nuclear RNA-seq shows proximal tubule-specific enrichment and increased expression in *Cpt1a*^{CKO} versus *Cpt1a*$^{fl/fl}$ kidneys.

Table. List of primers used in the study

Gene name	Forward	Reverse
Acox1	5'-AGGGAATTTGGCATCGCAGA-3’	5’-CATGCCCAAGTGAAGGTCCA-3’
Actin	5'-GGGATGTTTGCTCCAACCAA-3’	5’-GCGCTTTTGACTCAGGATTTAA-3’
aSMA	5'-CAGGGAGTAATGGTTGGAAT-3’	5’-TCTCAAACATAATCTGGGTCA-3’
Col1a2	5'-GGAGGGAACGGTCCACGAT-3’	5’-GAGTCCGCGTATCCACAA-3’
D17 del	5'-GAACCAAACTGAACGCCTAAAC-3’	5’-TGGGCTTTTGGTAGTCATAGGT-3’
FABP1	5'-ATGAAGGCAATAGGTCTGCCC-3’	5’-CGATTTCTGACACCCCCTTGA3’
GAPDH	5’-AGGTCGGTGTGAACGGATTTG-3’	5’-TGTAGACCATGTAGTTGAGGTCA-3’
Havcr1 (Kim-1)	5’-AAACCAGAGATTCCCACACG-3’	5’-GTCGTGGGTCTTCCTGTAGC-3’
Hmgcs2	5'-CAGTGGAAGCAAGCTGGAAAC-3'	5’-TCTTGCAAAAGGGTGTGTGG-3’
IL1b	5'-CCCAAAAGATGAAGGGCTGC-3’	5’-TGATGTGCTGCTGCGAGATT-3’
IL6	5'-CTCTGCAAGAGACTTCCATCCA-3’	5’-AGTCTCCTCTCCGGACTTGT-3’
mtND1	5'-TAGAACGCAAAATCTTAGGG-3’	5’-TGCTAGTGTGAGTGATAGGG-3’
PDK4	5'-GCTGCTGGACTTTGGTTCAGA-3’	5'-GGATATTGGCCAGGCGGAC-3’