Supplemental Figure 1. Human Hepatocytes express HTR1A receptor.

(A-B) Immunohistochemical analysis of HTR1A in liver sections from non-diseased subjects (A) and patients with Primary Biliary Cirrhosis (PBC) (B). (C-D) Representative pictures of the same samples exposed to non-immune anti-sera as negative control.

Supplemental Figure 2. Hepatocytes do not express tph1 nor tph2.

(A-B) 2% agarose gel of QRT PCR amplicons demonstrates expression of tryptophan hydroylase (tph) -1 (A) and -2 (B) in 603B cholangiocytes (Chol), primary Hepatocytes (Hep) and Negative Controls (i.e. dWater)

Supplemental Figure 3. Mice with functional inactivation of TPH2 (TPH2KI) displayed higher levels of KRT19, AE1/AE3 and AFP.

Immunohistochemical analysis of KRT19 (A), AE1/AE3 (B) and AFP (C) in liver sections from WT and TPH2KI after Sham or BDL. Representative pictures are displayed at Original magnification of 10X.

Supplemental Figure 4. Reduced TPH2 activity and biliary 5HT content induced up-regulation of transcripts encoding liver progenitor markers.

(A) Whole liver tissues from TPH2KI (black bars) and WT (grey bars) animals 2 weeks after BDL surgery were analyzed by QRT PCR to assess changes in markers of immature cholangiocytes (krt19, krt7, nestin) and immature hepatocytes (krt7 and afp). Data are normalized to WT-BDL values and displayed as mean±SEM. differences between groups are evaluated by two-tail Student t-test. $^{*}P<0.05$, $^{**}P<0.001$.

Supplemental Figure 5. Alteration of IL6 expression in TPH2KI mice

(A) Whole liver tissues from TPH2KI (black bars) and WT (grey bars) mice 2 weeks after BDL were analyzed by QRT PCR to assess changes in the cytokine, interleukin 6 (IL6). Data are normalized to WT-BDL values and displayed as mean±SEM. Differences between groups are evaluated by two-tail Student t-test. $^{*}P<0.05$.