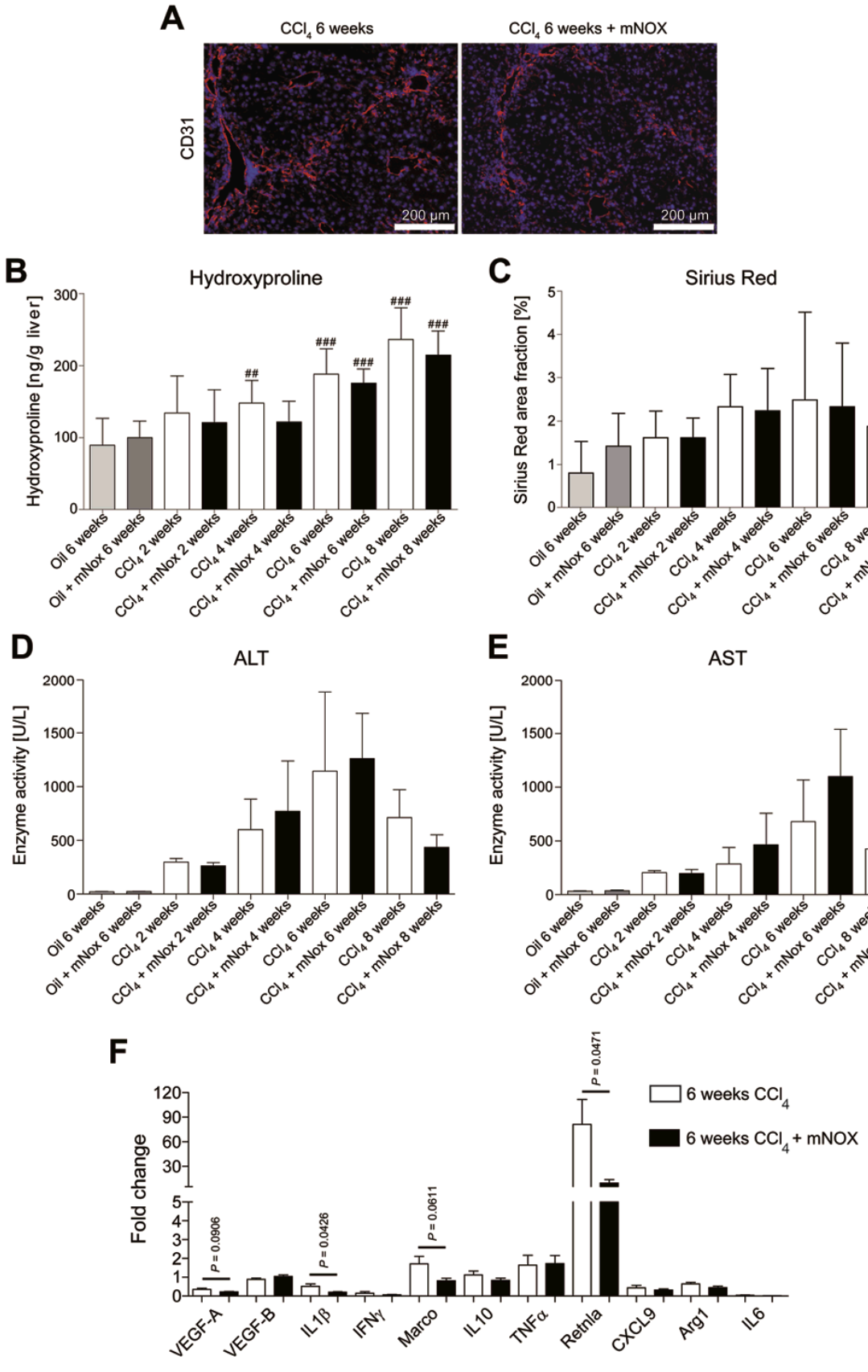
Supplementary Figure Legends

Supplementary Figure 1: Quantification of proteins and genes related to hepatic inflammation, angiogenesis and fibrosis. Chronic toxic liver injury of c57BL/6 wildtype mice was induced by 6 weeks of carbon tetrachloride (CCl_4) treatment twice weekly. Another group of animals additionally received thrice weekly subcutaneous injections of mNOX-E36 (anti-CCL2 agent). (A) Representative microscopy images of CD31 immunofluorescence. (B-C) Quantification of collagen deposition in chronically injured and mNOX-E36 treated livers by determination of hydroxproline content (B) or Sirius Red area fraction (C). (D-E) Serum ALT (D) and AST (E) activity representing the injury in CCl_4 or CCl_4 & mNOX-E36 treated livers in comparison to healthy control livers. (F) Hepatic gene expression profiling of inflammation, angiogenesis and fibrosis associated genes. Data are shown as mean ± SD. ### < 0.001 and ## < 0.01 for the comparison of CCl_4 or CCl_4 + mNOX-E36 vs. the corresponding control groups (i.e. 6 weeks oil or 6 weeks oil + mNOX-E36) (n=30 mice; unpaired Student's *t* test).

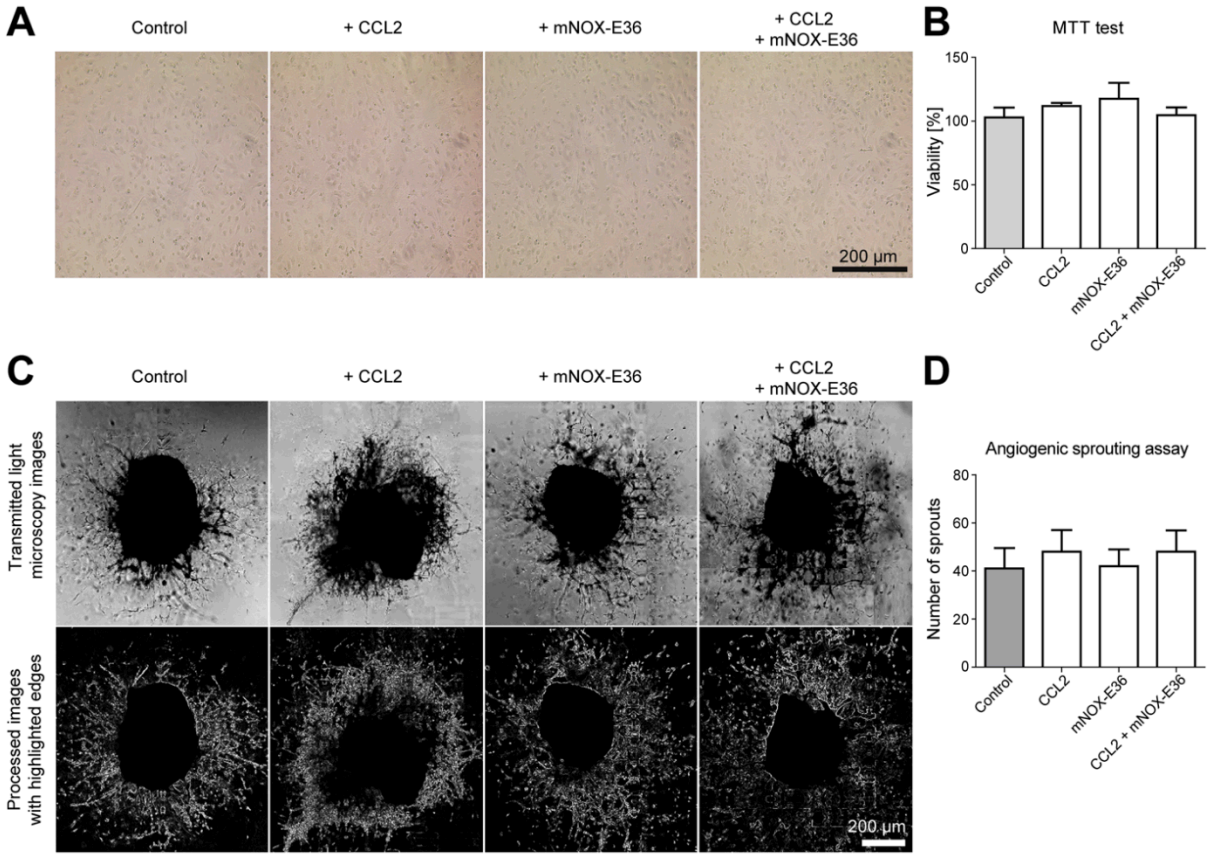
Supplementary Figure 2: Effects of CCL2 and mNOX-E36 on cell viability and sprouting activity of primary murine endothelial cells *in vitro*. (A) Endothelial cells were isolated from murine livers and cultured with VEGF containing medium, followed by stimulation with CCL2, mNOX-E36 or CCL2 + mNOX-E36 for 24 h or were left unstimulated. (B) Quantification of cell viability by MTT test. Cells were pooled from n=3 c57BL/6 wild-type mice. (C) Aortic rings were isolated from the thoracic aorta of c57BL/6 wildtype mice, embedded in Matrigel and cultured with VEGF containing medium, followed by stimulation with CCL2, mNOX-E36 or CCL2 & mNOX-E36 for 10 days or were left unstimulated. (D) Angiogenic sprouting was not affected by CCL2, mNOX-E36 or CCL2 & mNOX-E36 (n=3 aortic rings per condition; unpaired Student's *t* test).

Supplementary Figure 3: Vascular branching analysis for quantifying areas of sprouting angiogensis in a model of cholestatic hepatic injury. (A) Ex vivo high-resolution µCT-based quantification of the mean total number of branching points in livers with a chronic cholestatic injury caused by bile duct ligation (BDL) 21 days ago and sham control animals. All analyses were performed by quantifying the total amount of branching points in 5 representative blood vessels for each, the central and the portal vein system. Data are shown as mean ± SD; ***P < 0.001, (unpaired Student's t test). (B) Ex vivo µCT-based quantification of the percentage of branching points per increasing order (1^{st} to 11^{th} branching order) for livers of sham control and mice suffering from a BDL 21 days ago. ***P < 0.001 (unpaired Student's *t* test).

Supplement Figure 1



Supplement Figure 2



Supplement Figure 3