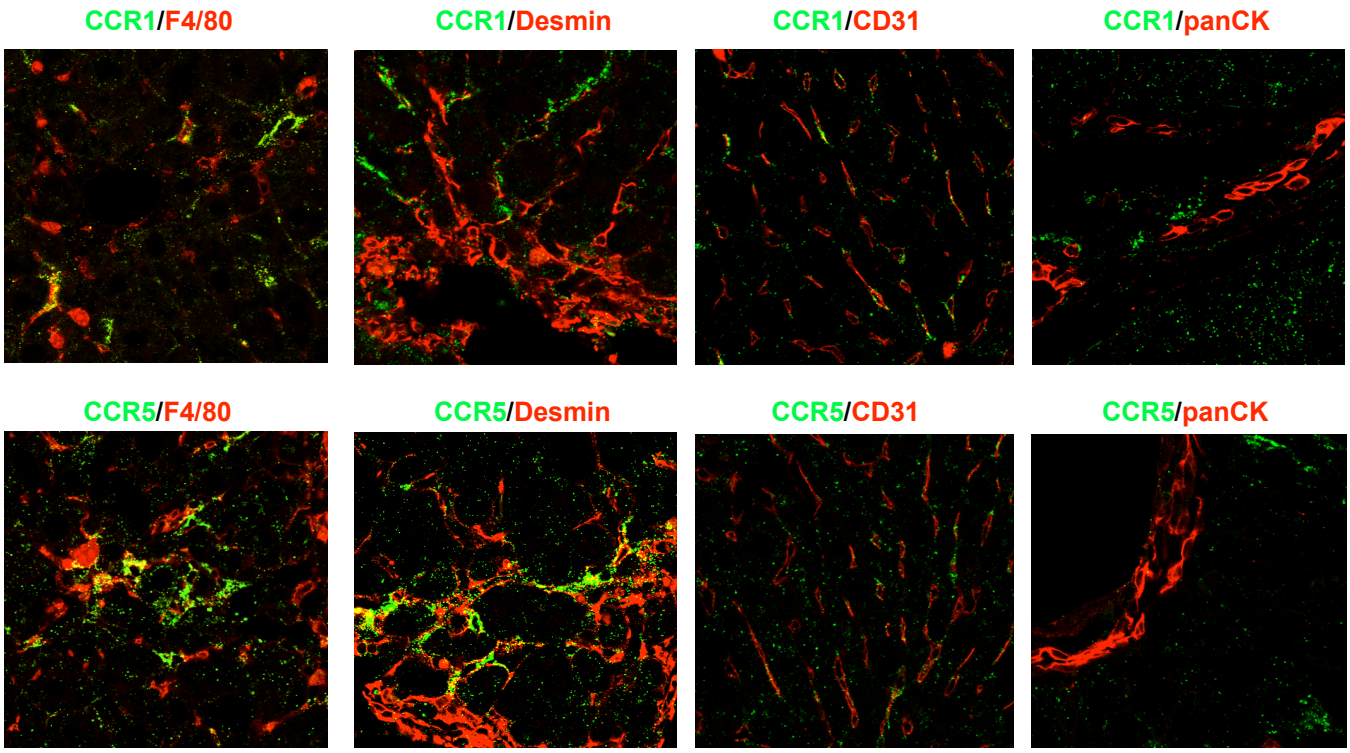
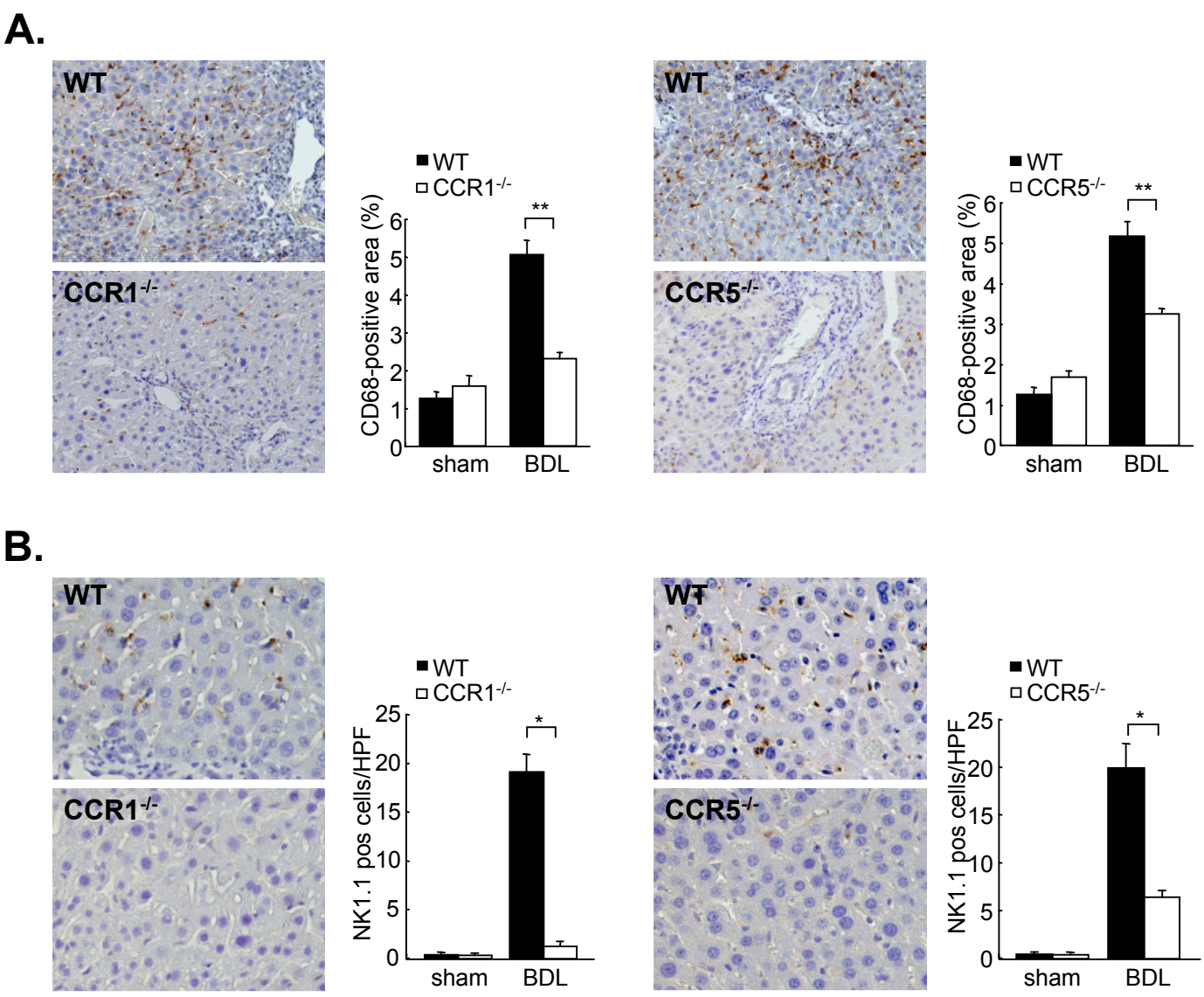
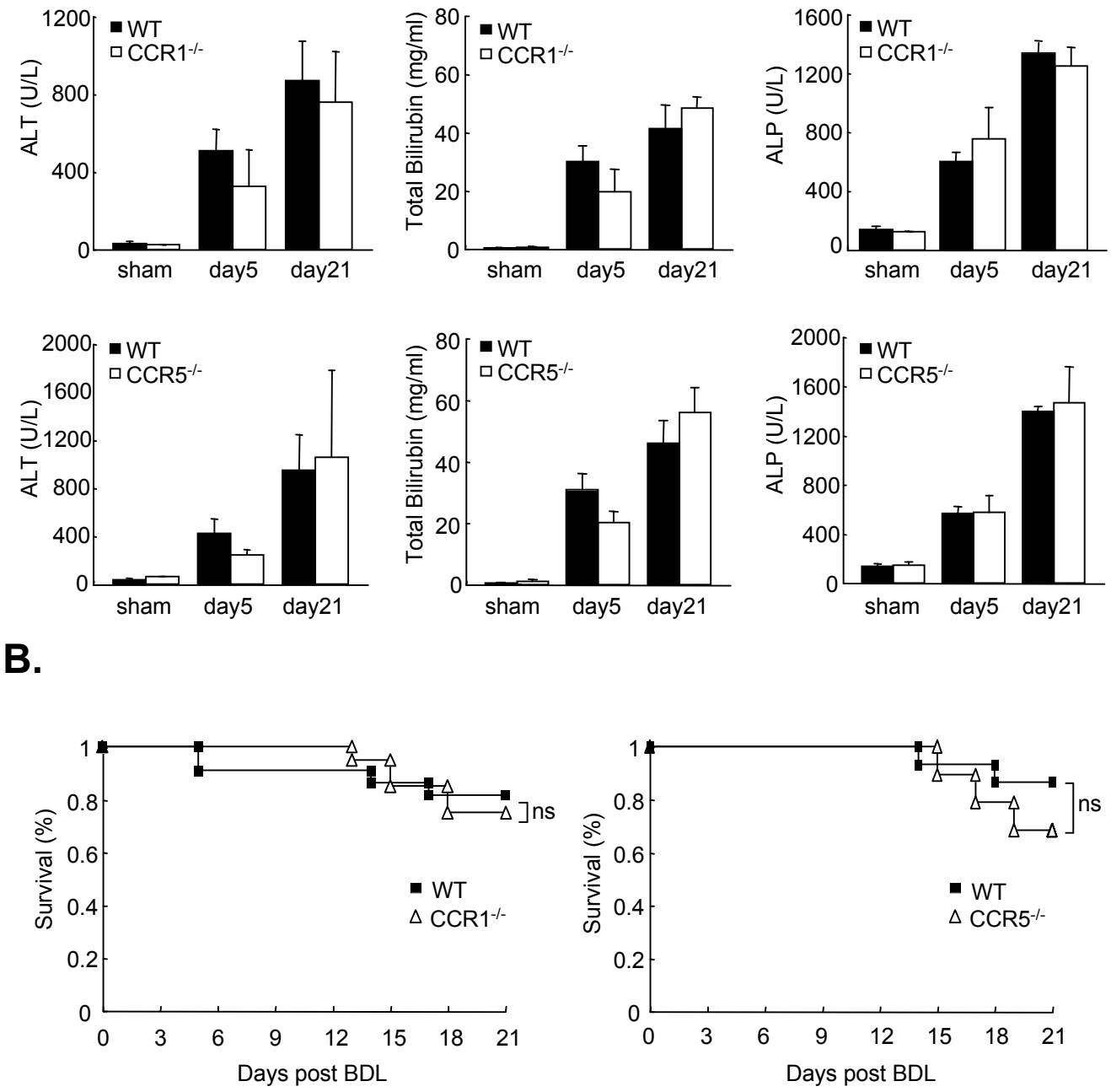


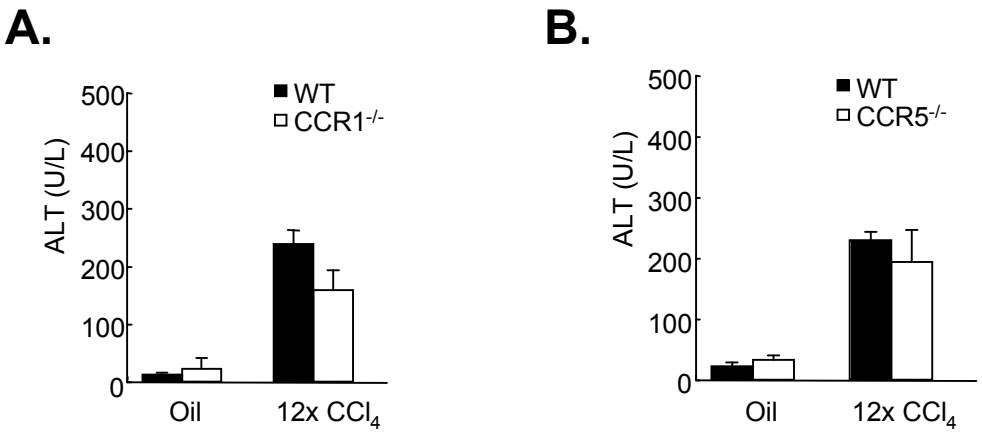
Supplementary Figure 1. Expression of CCR1 and CCR5 in CCl_4-induced liver fibrosis. Mice underwent 12 injections of CCl_4 or oil. Livers were stained for CCR1 (green fluorescence, upper panel), CCR5 (green fluorescence, lower panel), F4/80, desmin, CD31 or panCK (all red fluorescence) followed by confocal microscopy.



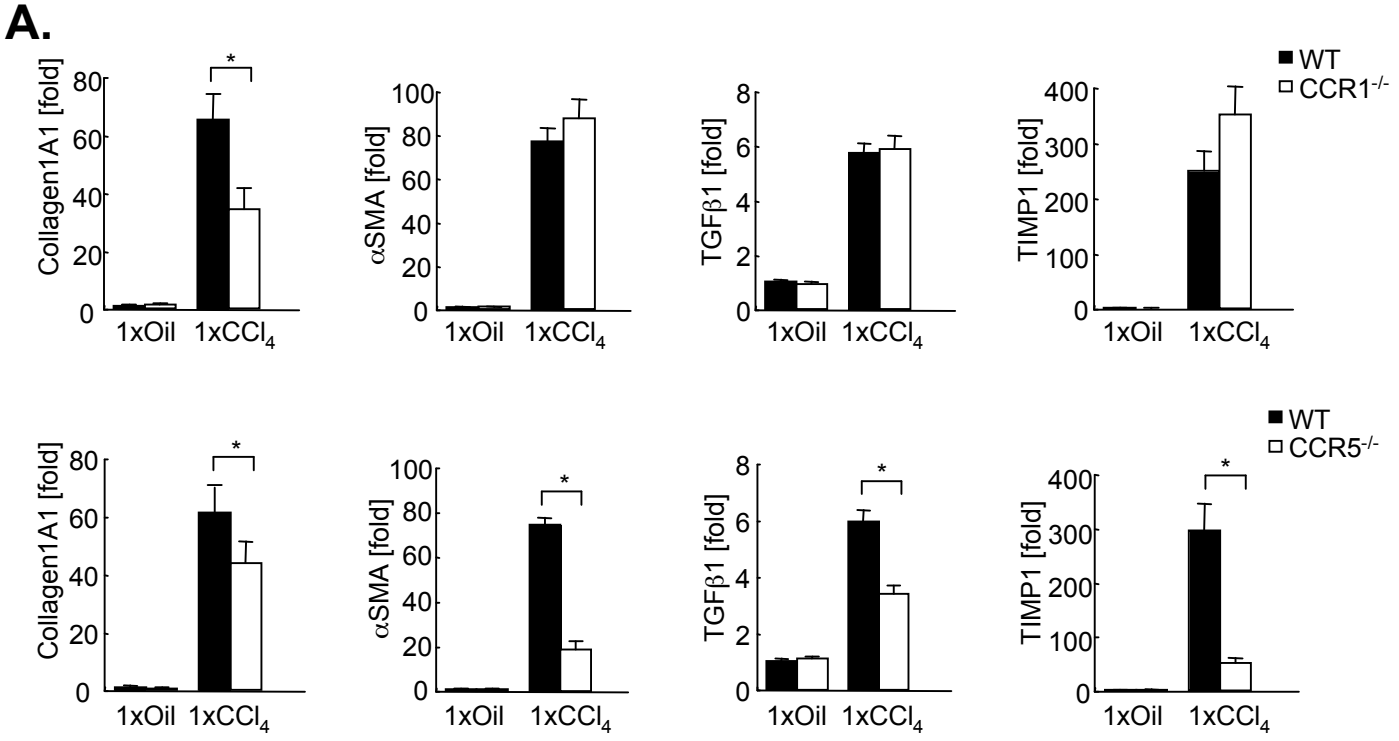
Supplementary Figure 2. Inflammatory cell recruitment in CCR5- and CCR1-deficient mice after BDL. A. Wild-type, CCR1$^{-/-}$ and CCR5$^{-/-}$ mice underwent BDL for 21 day followed by immunohistochemistry for CD68 (A) or NK1.1 (B). *p<0.05,**p<0.01



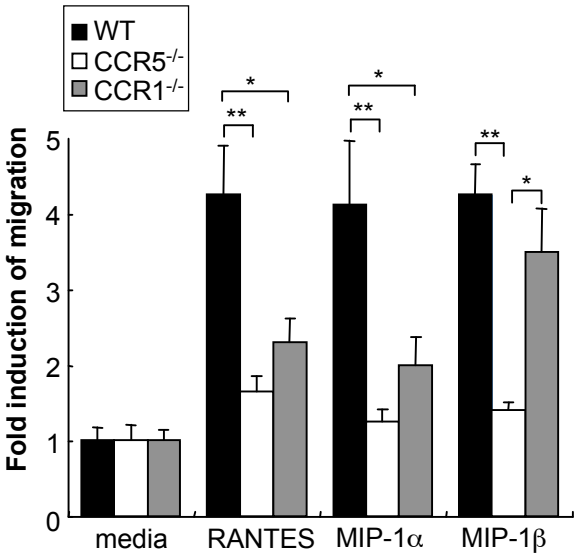
Supplementary Figure 3. CCR1- and CCR5-deficient mice do not display increased liver injury or decreased survival after BDL. A. CCR1-deficient (n=4) and wild-type mice (n=5) as well as CCR1-deficient (n=6) and wild-type controls (n=6) underwent BDL for 5 or 21 days followed by measurement of serum ALT, total bilirubin and alkaline phosphatase (ALP). B. Survival of $CCR1^{-/-}$ (n=15) and wild-type mice (n=22), as well as $CCR5^{-/-}$ (n=19) and wild-type mice (n=15) after BDL.



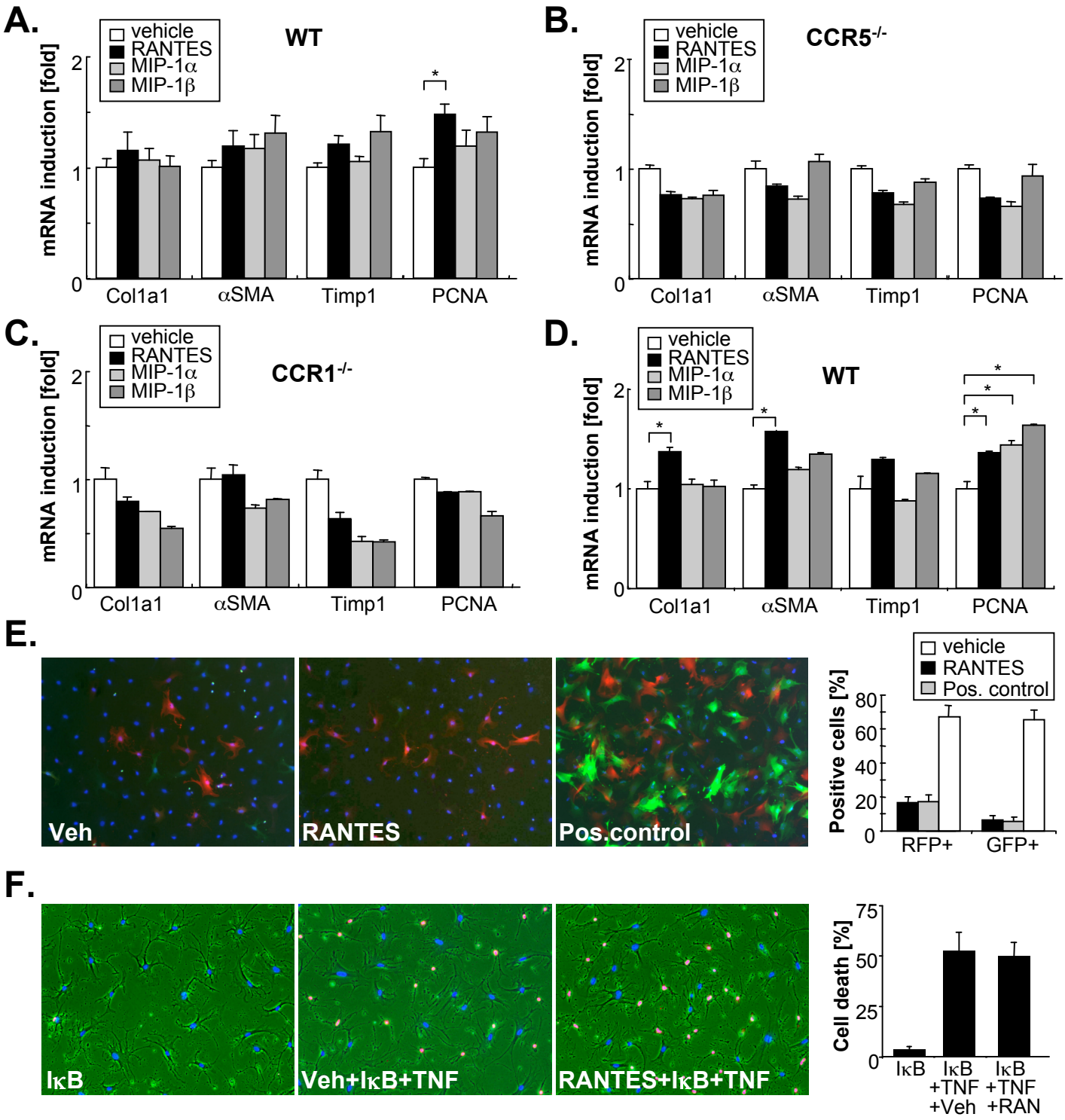
Supplementary Figure 4. CCR1- and CCR5-deficient mice do not display increased liver injury after CCl_4. A. CCR1-deficient (n=5) and wild-type mice (n=5) were treated with 12 CCl_4-injections followed by measurement of serum ALT levels. B. CCR5-deficient (n=5) and wild-type controls (n=5) were treated with 12 CCl_4-injections followed by measurement of serum ALT levels.



Supplementary Figure 5. mRNA expression of fibrosis related genes in CCR1- and CCR5-deficient mice after CCl_4 treatment. A-B. CCR1-deficient mice (n=4) and isogenic wildtype controls (n=4) as well as CCR5-deficient mice (n=4) and wild-type littermates (n=4) were treated with 1 injection of CCl_4 followed by quantitative real-time PCR for Col1a1, αSMA, TGFβ1 and TIMP1.*$p<0.05$



Supplementary Figure 6. CCR5 and CCR1 induce Kupffer cell migration. A. Kupffer cells from wild-type, CCR5- and CCR1-deficient mice were placed in a Boyden chamber and migration through an 8 micron filter was determined after stimulation with RANTES, MIP-1α or MIP-1β (all 50 ng/ml). * $p<0.05$, ** $p<0.01$



Supplementary Figure 7. CCR5 and CCR1 do not induce HSC activation and do not prevent cell death. A-D. HSCs were isolated from wild-type, CCR5- and CCR1-deficient mice. HSCs were stimulated with RANTES, MIP-1α or MIP-1β (all 100 ng/ml) or vehicle (0.1% BSA) for 24 hours (A-C) or 5 days (D). Following RNA extraction and reverse transcription, collagen α1(I), αSMA, TIMP1 and PCNA mRNA levels were measured by quantitative real-time PCR. E. HSCs were isolated from double-transgenic mice expressing GFP under the collagen α1(I) promoter and RFP under the αSMA promoter, and treated for five days with either vehicle (0.1% BSA) or RANTES (100 ng/ml). GFP and RFP expression were evaluated by fluorescent microscopy and quantified. F. Culture-activated HSCs were infected with AdIκBsr, followed by pretreatment with RANTES (100 ng/ml) or vehicle for 12h, and treatment with TNFα (30 ng/ml) for 8h. Cell death was determined by propidium iodide staining, and normalization to nuclei stained by cell-permeable Hoechst. * $p<0.05$