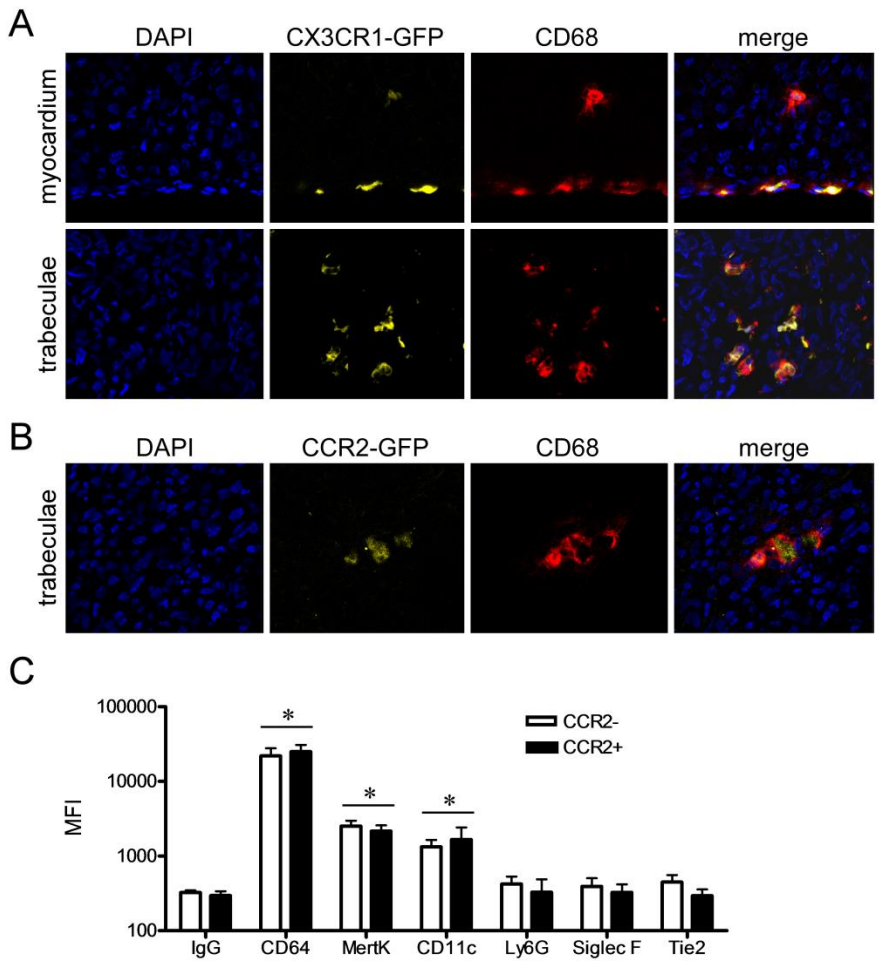
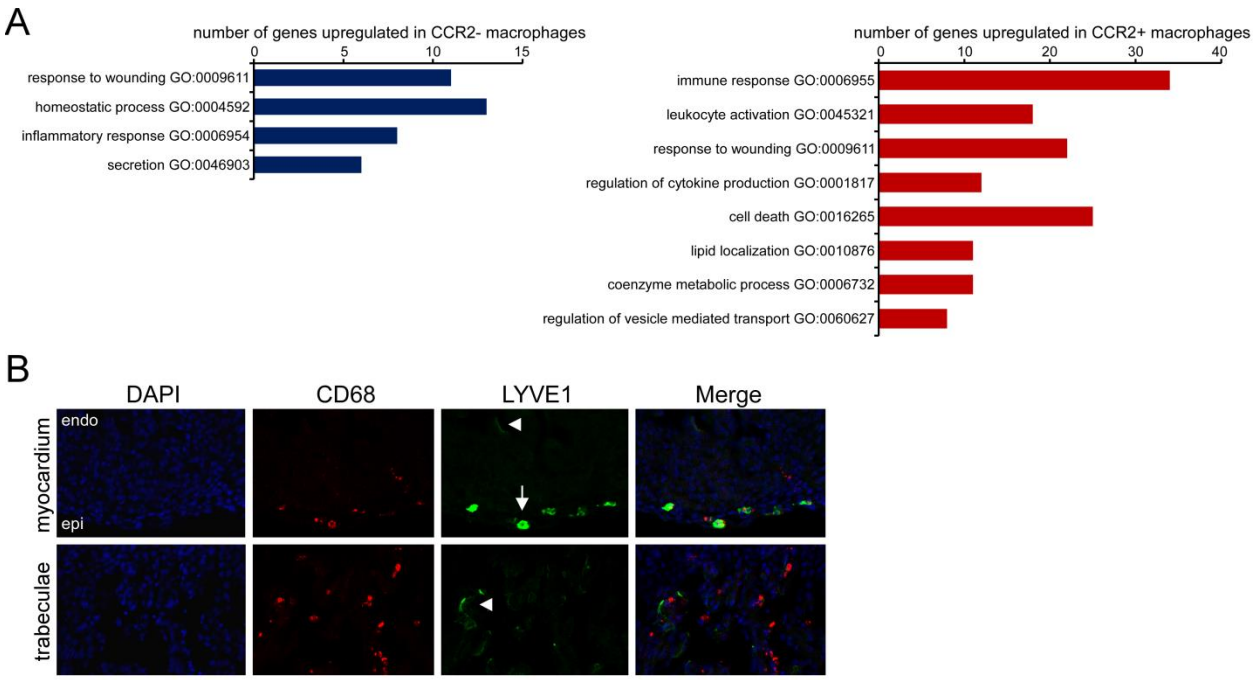
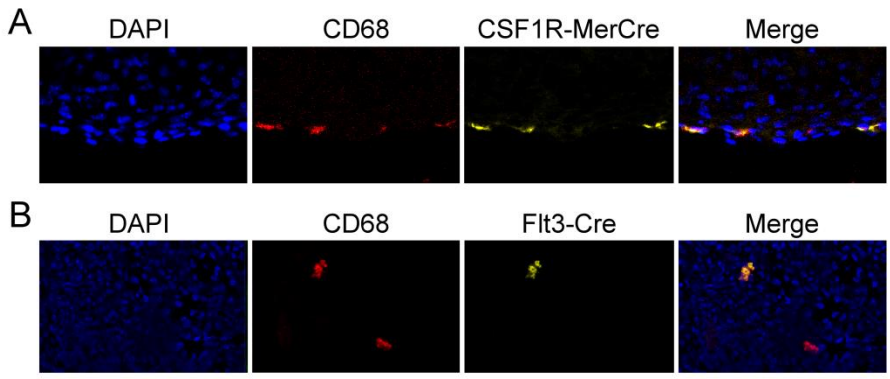


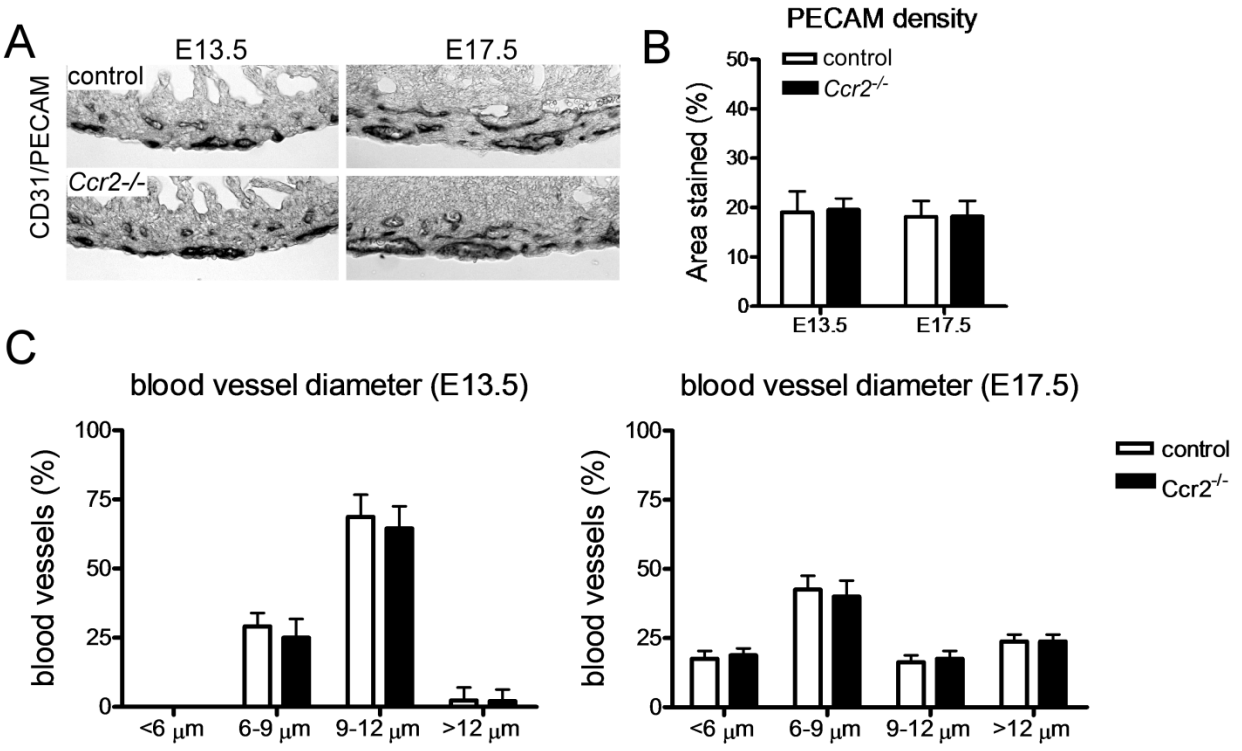
Online Figure I. A, High magnification images demonstrating that the macrophage reporter CX3CR1-GFP (yellow) labels CD68+ macrophages (red) within the myocardium and endocardial trabeculae. **B**, Immunostaining revealing that the CCR2-GFP reporter (yellow) labels CD68+ macrophages (red) within the endocardial trabeculae. 40X magnification, DAPI (blue). Immunostaining performed at E14.5. **C**, Quantification of flow cytometry derived mean florescent intensity (MFI) showing statistically indistinguishable expression of CD64, MertK, and CD11c on the surface of CCR2- and CCR2+ embryonic macrophages. $*$ denotes $p<0.05$ compared to IgG control.



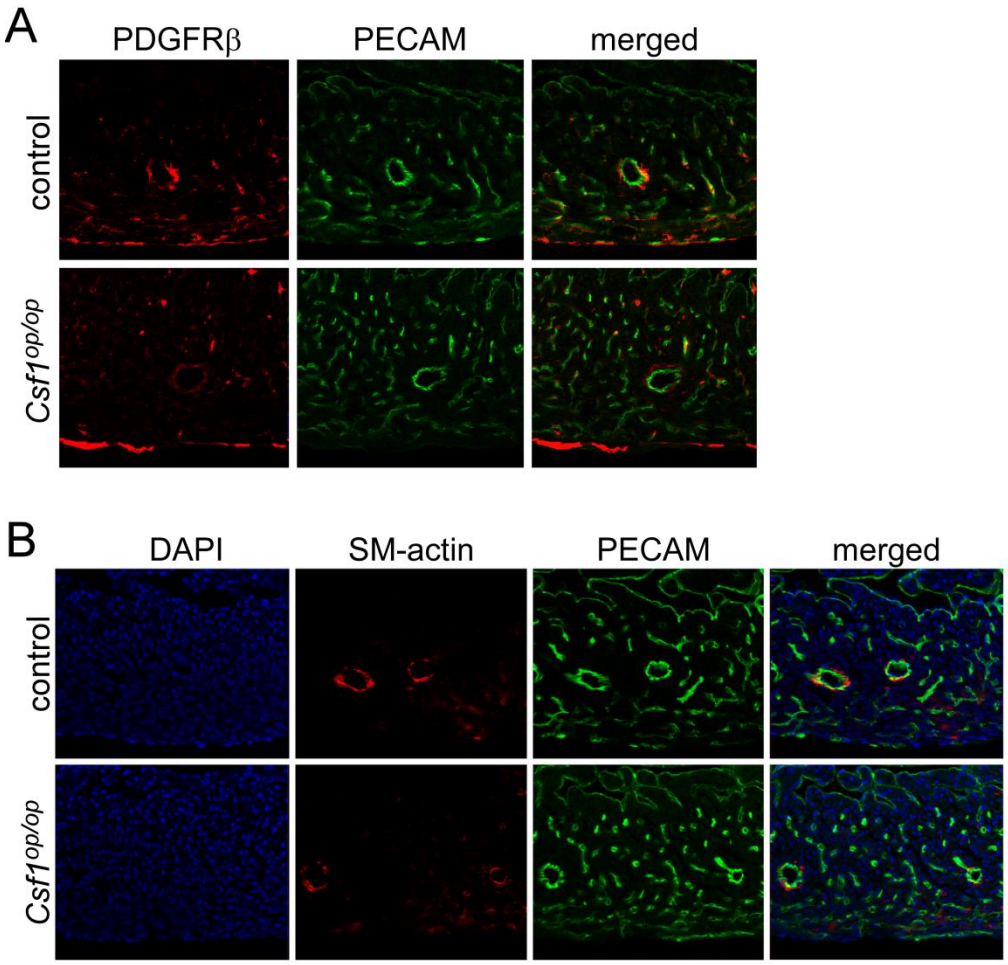
Online Figure II. A, GO pathway analysis highlighting molecular pathways that are differentially represented in embryonic CCR2- and CCR2- cardiac macrophages. **B**, Immunostaining for CD68 (red) and LYVE1 (green) demonstrating that only CCR2- macrophages located in the myocardium and not CCR2+ macrophages located within the trabecular myocardium express LYVE1 (arrow). Arrowheads denote LYVE1+ lymphatic endothelial cells.



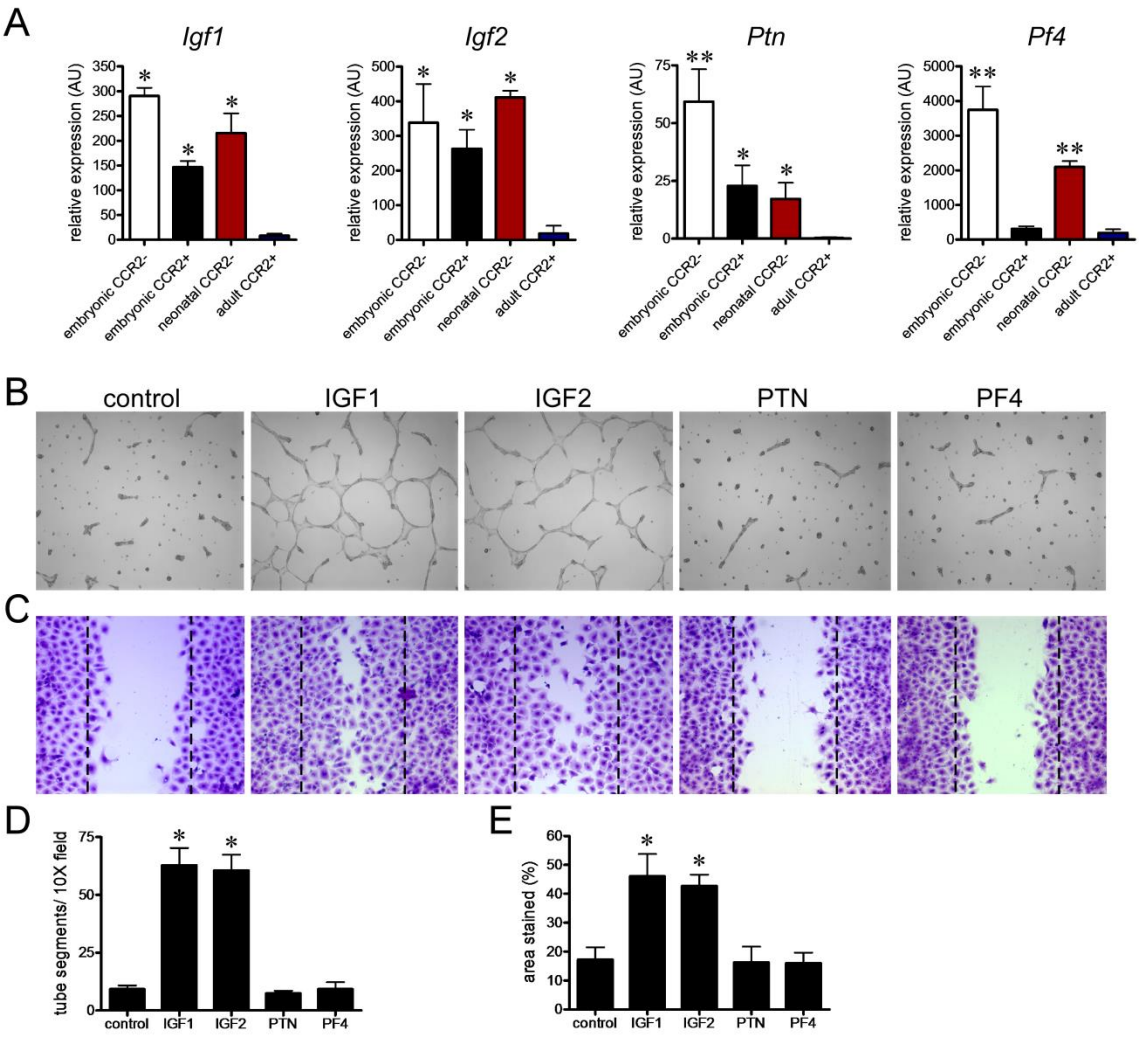
Online Figure III. A, High magnification images demonstrating labeling of CD68+ macrophages (red) within the myocardium by the CSF1R-MerCre/Rosa26-tdtomato (yellow) reporter. Tamoxifen gavage performed by E7.5 and embryonic hearts harvested and stained at E14.5. **B**, Immunostaining revealing that the Flt3-Cre/Rosa26tdtomato reporter (yellow) labels CD68+ macrophages (red) within the endocardial trabeculae. 40X magnification, DAPI (blue). Immunostaining performed at E14.5.



Online Figure IV. Normal coronary development in Ccr2$^{-/-}$ hearts. A, PECAM/CD31 immunostaining showing normal coronary vascular plexus density and patterning at E13.5 and E17.5. **B**, Indistinguishable coronary vascular density in control and Ccr2$^{-/-}$ embryonic hearts. **C**, Quantitative analysis of coronary vessel diameter demonstrating that CCR2 is dispensable for coronary remodeling. n=5.



Online Figure V. Macrophage deficiency does not disrupt epicardial development. A, PGGFRβ (red) and PECAM/CD31 (green) immunostaining demonstrating indistinguishable distribution of WT1+ epicardial and epicardial-derived cells in E16.5 control and *Csf1*$^{op/op}$ hearts. **B**, Smooth muscle actin (red) and PECAM/CD31 (green) immunostaining revealing similar extent and patterning of smooth muscle cells in E16.5 control and *Csf1*$^{op/op}$ hearts.



Online Figure VI. Candidate cardiac macrophage-derived pro-angiogenic growth factors. A, Quantitative RT-PCR analysis showing the relative expression of *Igf1*, *Igf2*, *Ptn*, and *Pf4* in embryonic CCR2-, embryonic CCR2+, neonatal CCR2- and adult CCR2+ cardiac macrophages. * $p<0.05$ compared to adult CCR2+ cardiac macrophages. **B**, Matrigel angiogenesis assays demonstrating that only IGF1 and IGF2 treatment results in capillary plexus formation in cultured coronary endothelial cells. **C**, Scratch assays revealing that only IGF1 and IGF2 treatment stimulates coronary endothelial cell migration. **D-E**, Quantitative analysis of Matrigel angiogenesis (d) and scratch assays (e). * $p<0.05$ compared to vehicle control.