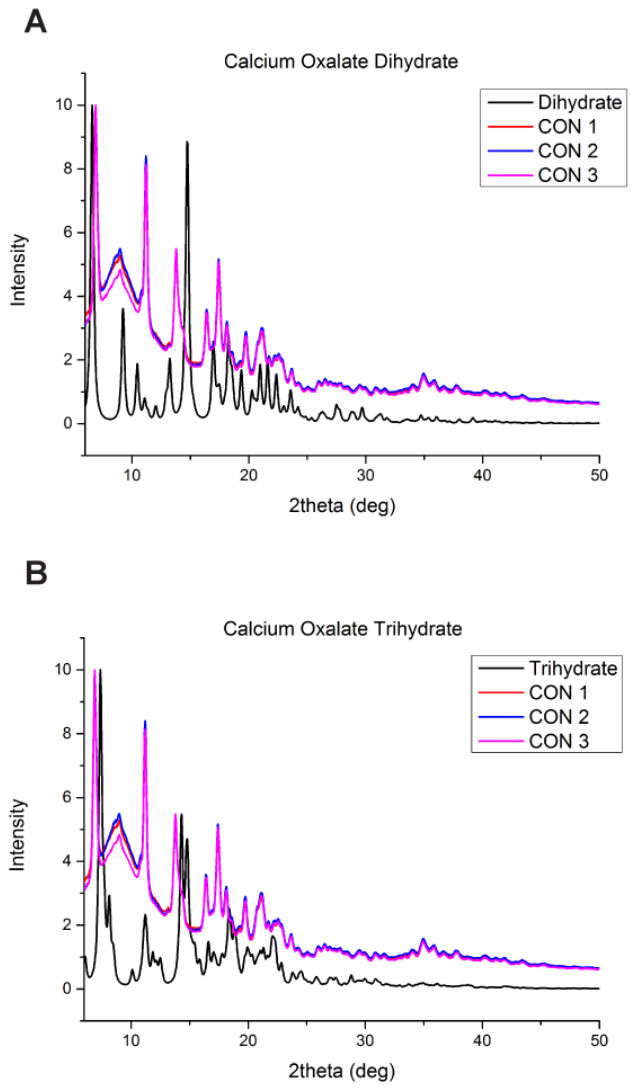
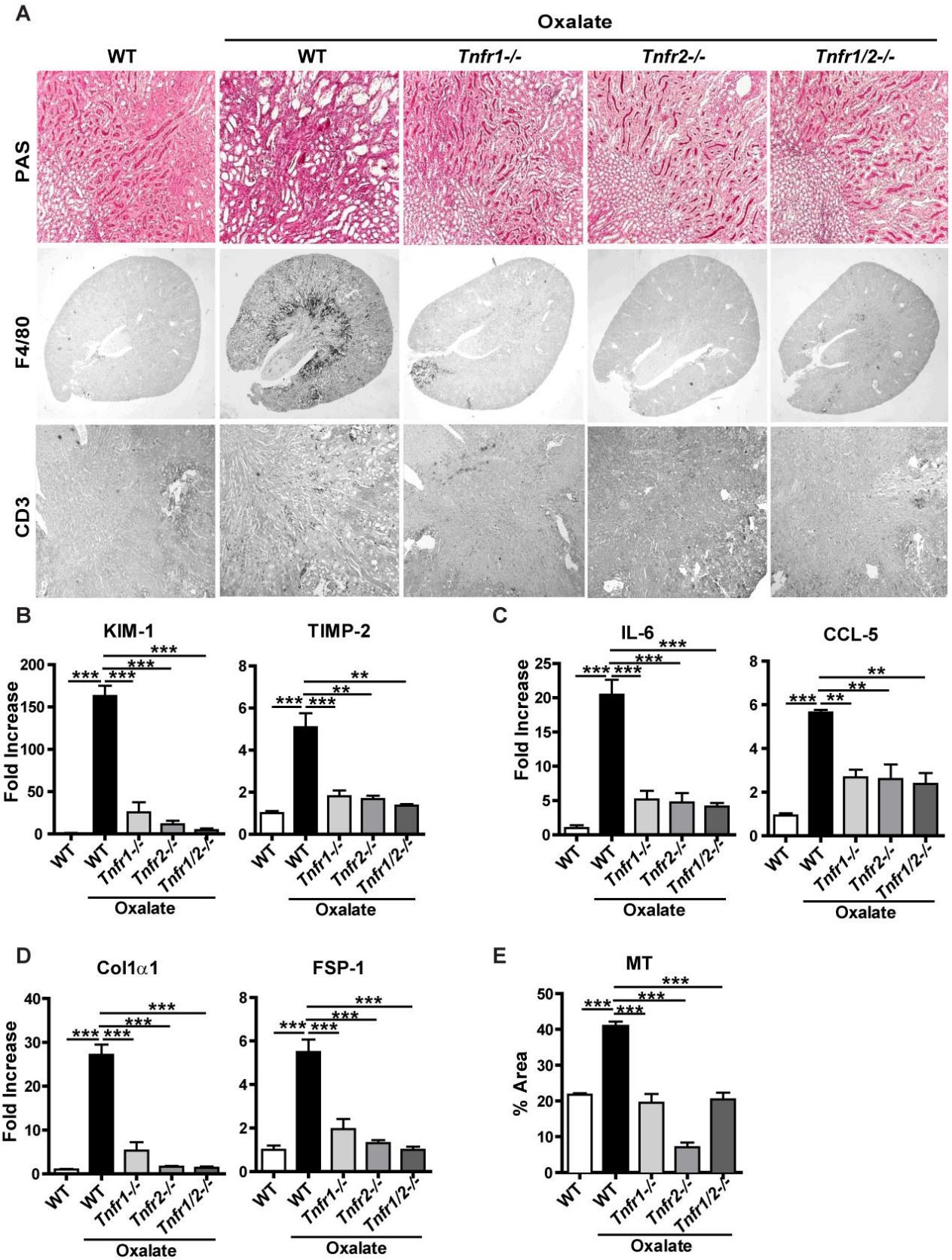
## Supplementary figure 1



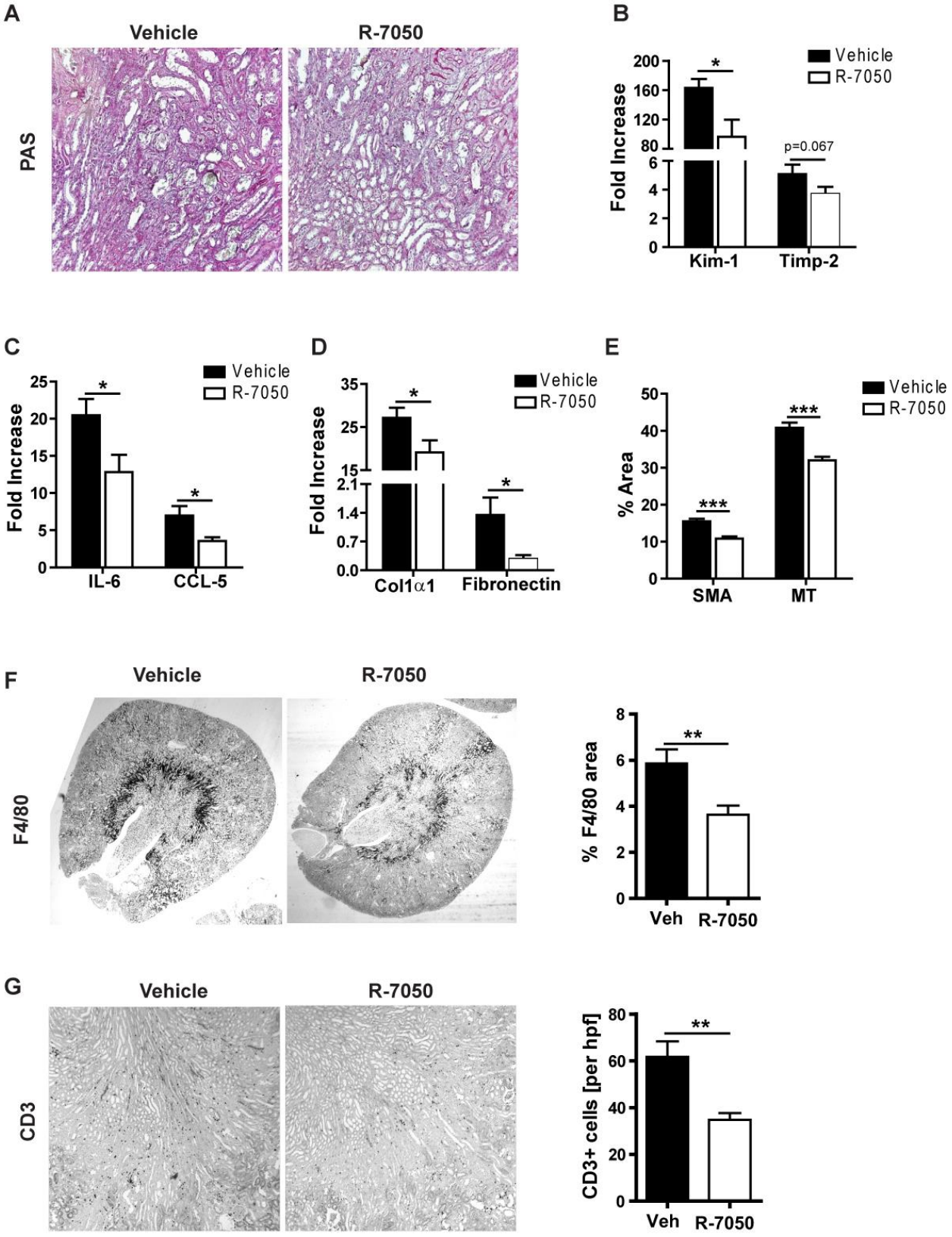

**Supplementary figure 1. X-ray diffraction analysis of mouse kidneys.** C57BL/6 male mice were fed either high oxalate or control diet for 14 days and kidneys were analyzed by X-ray powder diffraction. Note that the crystals deposited in the kidneys were neither calcium oxalate dihydrate nor calcium oxalate trihydrate. CON: Calcium oxalate nephropathy.

Supplementary figure 2




**Supplementary figure 2. *Tnfr1*-, *Tnfr2*-, and *Tnfr1/2*-deficient mice are protected from hyperoxaluria-related CKD.** C57BL/6 wild type, *Tnfr1*-, *Tnfr2*-, and *Tnfr1/2*-deficient mice were fed either high oxalate or control diet for 14 days. (A) Tubular injury was analyzed using PAS staining. Immune cell infiltration was analysed using F4/80 and CD3 staining. B-D: Gene expression analysis for (B) kidney injury markers KIM-1 and TIMP-2, (C) inflammation markers IL-6 and CCL-5, and (D) profibrotic markers collagen1α1 and FSP-1 in the kidney tissue. (E) Quantification of Masson Trichrome staining for fibrosis in the kidney tissue. Data are means ± SEM from 6-7 mice in each group. **p<0.01, ***p<0.001.

**Supplementary figure 3**




**Supplementary figure 3. The TNFR antagonist R-7050 protects hyperoxaluric mice from chronic kidney disease.** C57BL/6 male mice were fed high oxalate diet for 14 days with or without R-7050 (12mg/kg *i.p.* every alternate day) treatment. (A) Tubular injury was analyzed using PAS staining. B-D: Gene expression analysis for (B) kidney injury markers KIM-1 and TIMP-2, (C) inflammation markers IL-6 and CCL-5, and (D) profibrotic markers Cal1α1 and fibronectin in the kidney tissue. E-G: Quantification of immunostaining for (E) SMA and Masson Trichrome staining for fibrosis, (F) F4/80 positive macrophage area, (G) CD3 positive cells per high power field in the kidney tissue. Data are means ± SEM from 6-8 mice in each group. *p<0.05, **p<0.01, ***p<0.001 versus vehicle group.