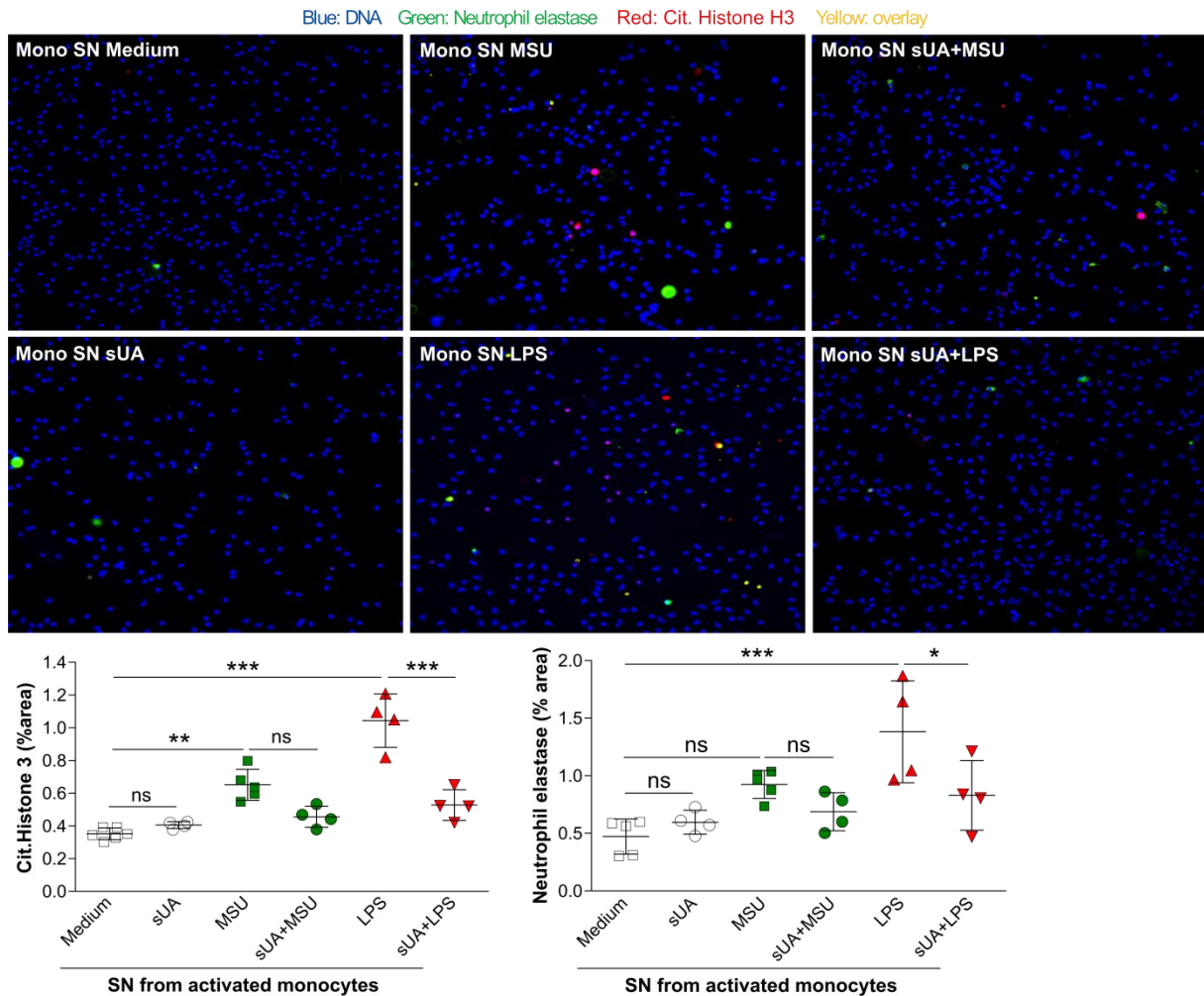
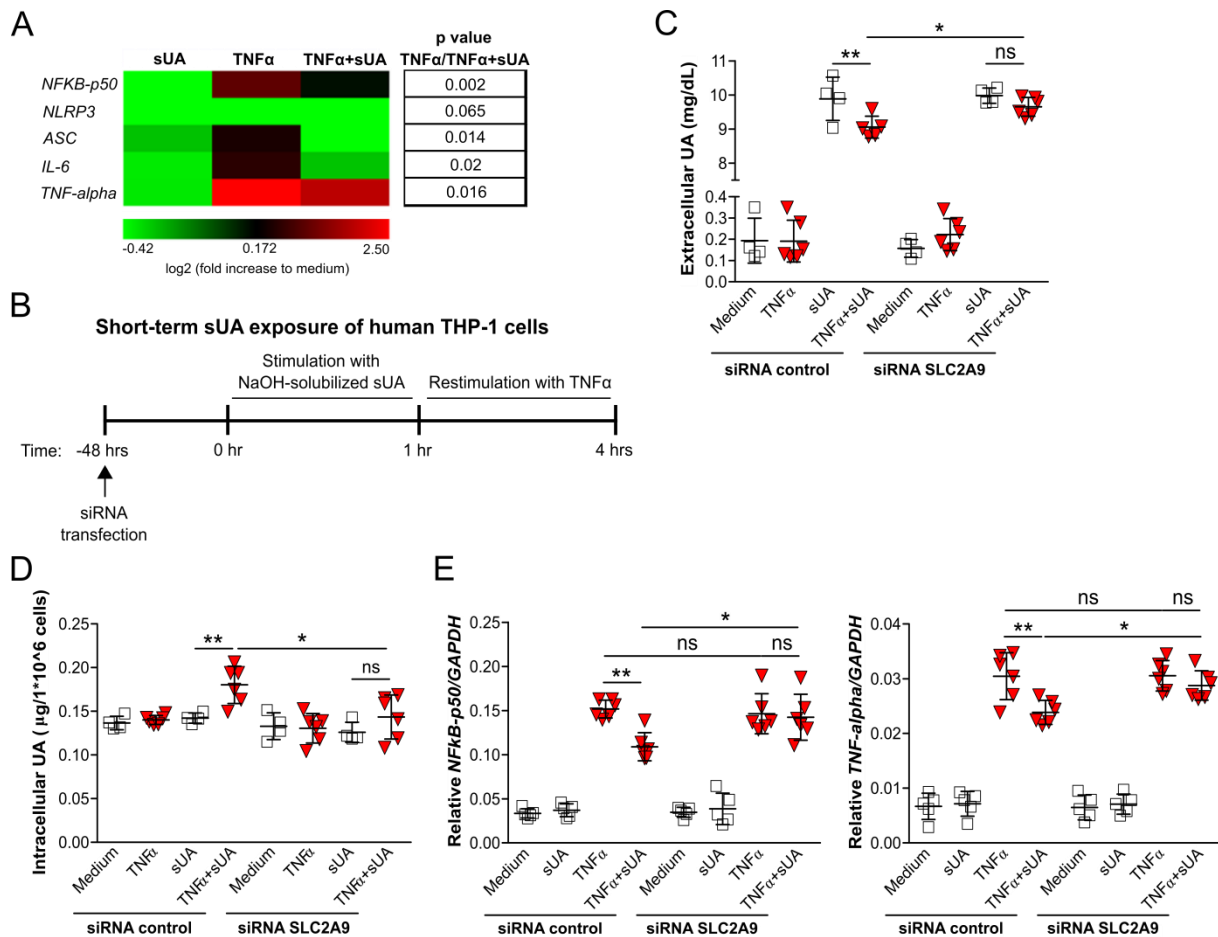


Supplemental Fig. 1: Hyperuricemia does not affect kidney function. Alb-creERT2;*Glut9*^{lox/lox} mice and *Glut9*^{lox/lox} control mice were injected intraperitoneally with tamoxifen. Both groups were fed a standard chow diet enriched with inosine for 22 days. **(A)** Periodic acid-Schiff staining of kidney section from healthy mice **(A)** and mice with HU **(A')** on day 22 (n = 5 mice per group). **(B)** Intrarenal mRNA expression levels of the kidney injury marker *KIM-1*, the inflammation marker *Tnfα*, and the fibrosis marker *Fibronectin 1* on day 22. Data are mean ± SD. Ns, not significant.



Supplemental Fig. 2: Soluble uric acid inhibits the release of inflammatory cytokines by activated monocytes to induce NET release. Human blood CD14⁺ monocytes and neutrophils were isolated from healthy individuals. CD14⁺ monocytes were pre-incubated with or without sUA (10 mg/dL) and then stimulated with MSU crystals or LPS or left untreated (medium). Supernatants (SN) from activated monocytes were collected and added to the neutrophil culture for 3 hours. NETs were stained with anti-neutrophil elastase antibody (green), anti-citrullinated Histone H3 antibody (cit. Histone H3, red) and DAPI (for DNA release, blue), and the % area of cit. Histone H3 and NE were quantified using the software ImageJ (n = 4-6; one-way ANOVA). Data are mean \pm SD. * p<0.05; ** p<0.01; *** p<0.001; ns, not significant.



Supplemental Fig. 3: Soluble uric acid modulates TNF α -activated monocytes by intracellular uptake via SLC2A9/GLUT9. (A) Human THP-1 cells were pre-incubated with 10 mg/dL sUA prior to stimulation with TNF- α for 4 hours, and mRNA expression levels of the inflammatory genes *NFKB-p50*, *NLRP3*, *ASC*, *IL-6* and *TNF-alpha* determined via RT-PCR, and illustrated as heat map (n = 6 per group; Student's *t*-test, p value of TNF- α vs. TNF- α +sUA). (B to E) Knockdown of SLC2A9 using specific siRNA (siRNA SLC2A9) or scrambled siRNA (siRNA control) in human THP-1 cells. After transfection, THP-1 cells were pre-incubated with 10 mg/dL sUA prior to stimulation with TNF- α for 4 hours (B). (C and D) The concentration of extracellular sUA (C) and intracellular sUA levels (D) were determined using an UA assay kit (n = 4-6 per group; two-way ANOVA). (E) mRNA expression levels of the inflammatory genes *NFKB-p50* and *TNF-alpha* were determined via RT-PCR (n = 5-6 per group; two-way ANOVA). Data are mean \pm SD. * p<0.05; ** p<0.01; ns, not significant.