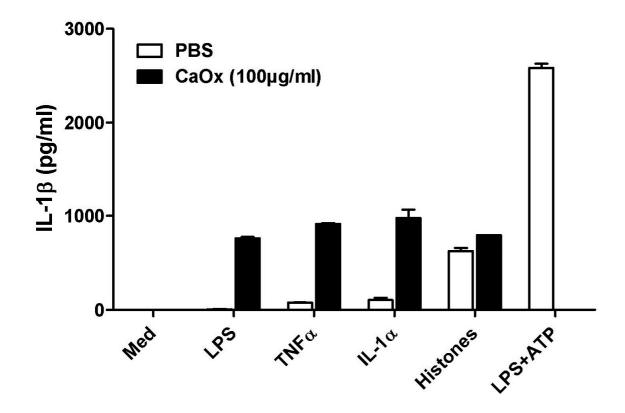
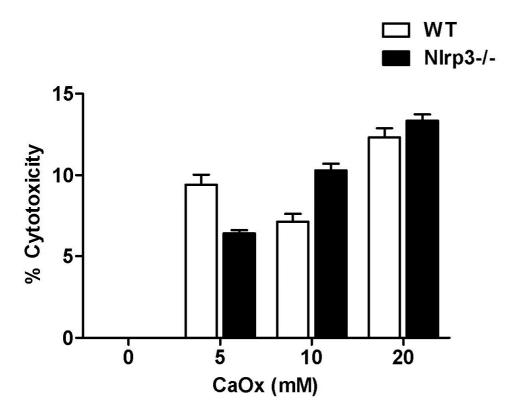


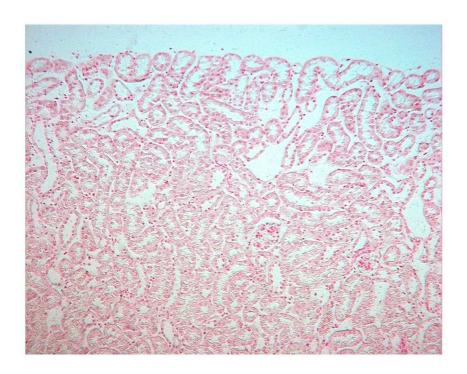
Scanning electron microscopy of the calcium oxalate crystals used for the in-vitro studies. Note that single crystals are of around 1 μm size which can clump to much larger aggregates.



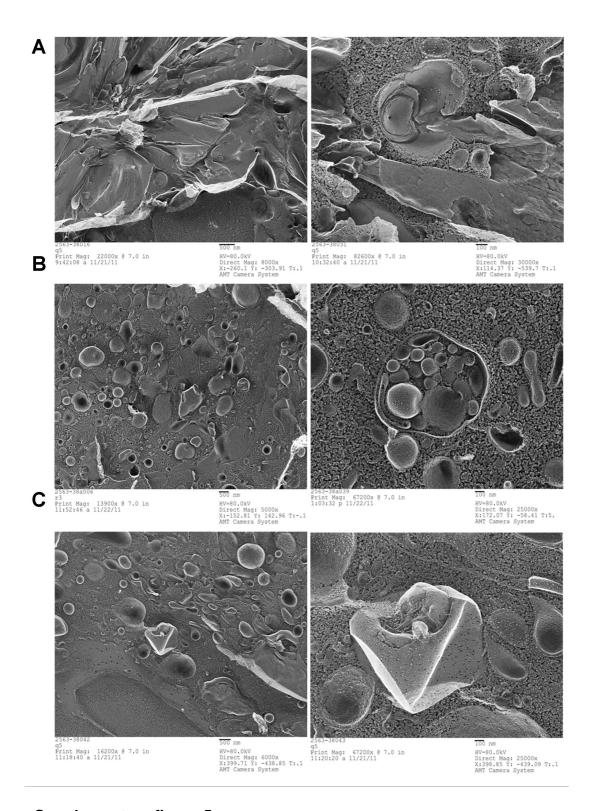
Bone marrow derived dendritic cells were primed with LPS (1 μ g/ml), TNF- α (100ng/ml), IL-1 α (10ng/ml) and histones (50 μ g/ml) for 3h followed by CaOx (100 μ g/ml) stimulation. Six hours after CaOx stimulations the supernatants were collected and IL-1 β levels were measured by ELISA. Data are expressed as mean±SEM from three independent experiments.



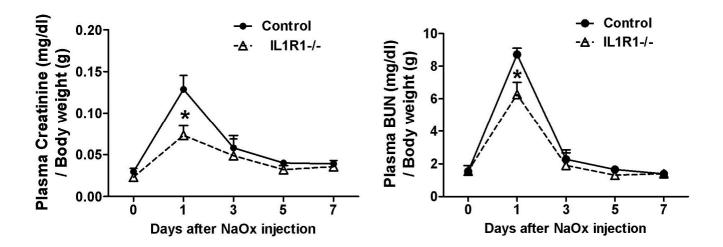
Primary tubular epithelial cell isolated from wildtype (WT) or *Nlrp3*-deficient mice were cultured with increasing concentrations of CaOx crystals as indicated. Lactate dehydrogenase (LDH) concentrations were measured in the supernatants after 18h of stimulation and data are expressed as mean±SEM percentage of positive control, which killed all the cells.



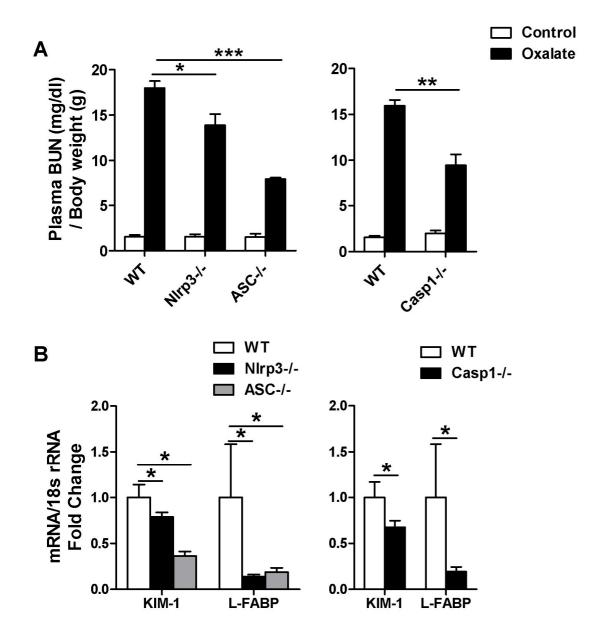
Representative Pizzolatto staining of a normal C57BL/6 mouse kidney (without NaOx injection). Original image magnification: x100.



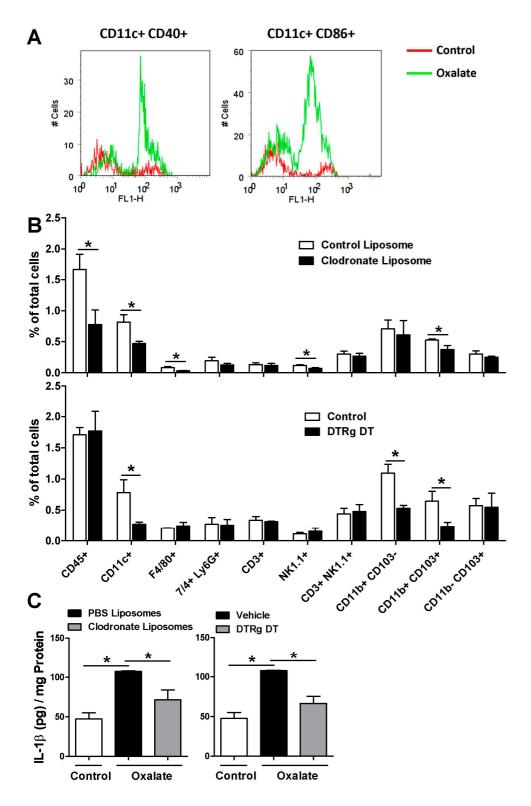
Quick-freeze deep-etch electron microscopy of 1.5 mm bread slices of non-fixed kidney illustrates bulky CaOx deposits within the tubular lumen and tubular epithelial cells often disrupting the luminal membrane of the cells (A). Inside tubular epithelial cell cytoplasm; irregularly-shaped lysosome-like organelles appear to be processing crystal material, which most likely represent CaOx crystals (B). Occasionally, diamond-shaped CaOx crystals are seen also within the pits of cross-sectioned brush boarder villi (C).



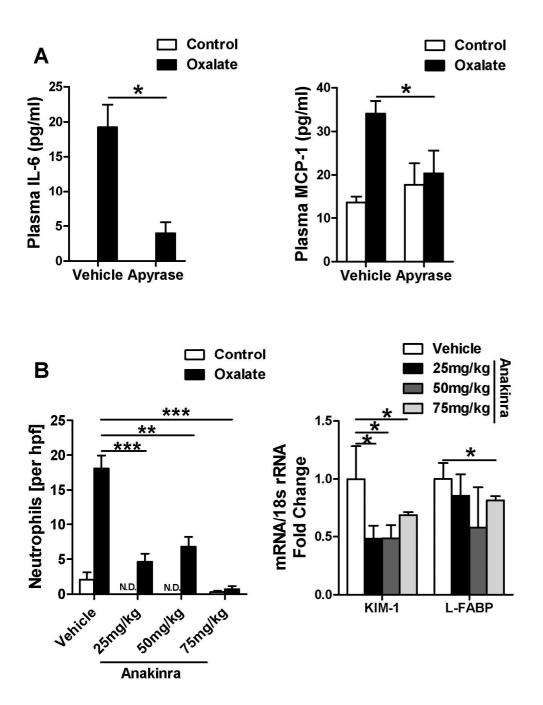
Oxalate nephropathy was induced in C57Bl6 and *Il-1r1*-deficient mice. Plasma creatinine and BUN levels were determined as described in the methods. Data are expressed as means± SEM from 5-6 mice in each group. Note that the model is only transient. Both parameters peak at 24h upon NaOx exposure and reaching baseline levels at day 3-5. *Il-1r1*-deficient mice were protected from the transient renal function impairment.



A: Plasma BUN levels of wildtype control mice and *Nlrp3*-, *Asc*-, or *casp-1*-deficient mice with oxalate nephropathy. Data are means ± SEM from 5-6 mice in each group. * p<0.05, ** p<0.01, *** p<0.001 versus wild type control mice. Healthy control mice are illustrated in white bars. B: Renal mRNA expression of KIM-1 and L-FABP in wild type, *Nlrp3*-, *Asc*-, or *casp-1*-deficient mice with oxalate nephropathy. * p<0.05 versus wild type mice.



A: Flow cytometry of CD11c/CD40+ and CD11c/CD86+ leukocytes from kidney taken 24h after exposure to NaOx or control. The image displays representative mean peak fluorescence plots out of 5-6 mice in each group. B: Flow cytometry of renal leukocytes and mononuclear phagocytes populations after injections of clodronate liposomes in WT and DT injection in CD11c DTR mice. C: The IL-1β levels were measured in the homogenates of kidneys after clodronate and DT injections by ELISA. Data are means ± SEM from 5-6 mice in each group. * p<0.05, versus vehicle-treated control mice.



A: Plasma levels of IL-6 and MCP-1 of mice with oxalate nephropathy injected either with apyrase or vehicle. B: Renal neutrophil counts were determined by immunostaining and quantified per high power field (hpf) and renal mRNA expression of KIM-1 and L-FABP from the kidneys of mice with oxalate nephropathy treated with different doses of anakinra or vehicle. Data are means \pm SEM from 5-6 mice in each group. * p<0.05 versus vehicle-treated control mice. Healthy control mice are illustrated in white bars. N.D. = not done.