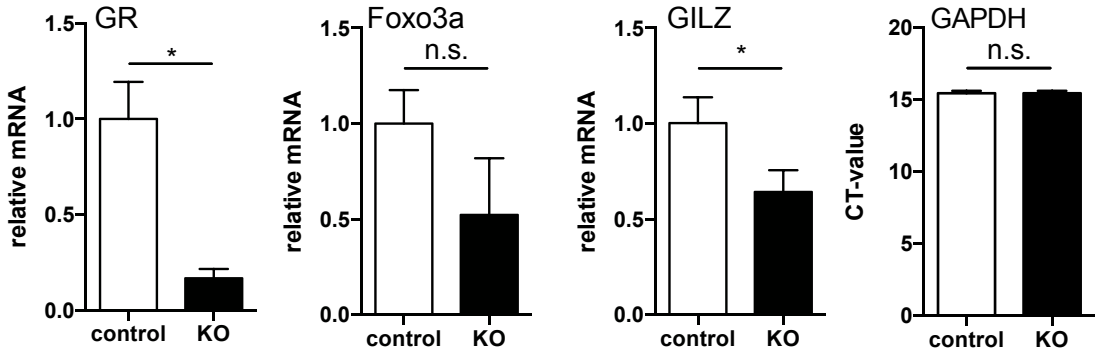
**Supplementary Material**

# Supplementary Figure 1

A

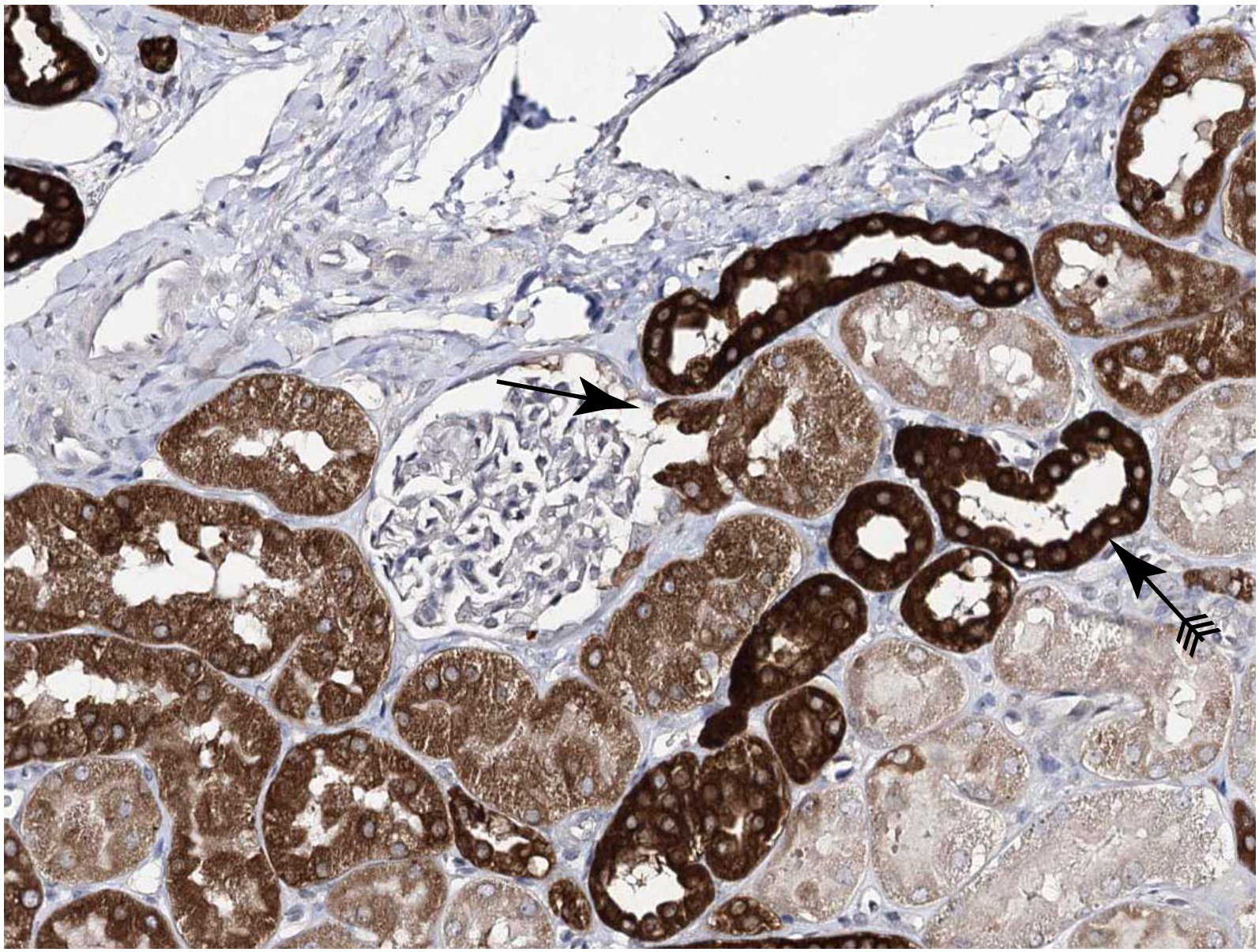


**Supplementary Figure 1**

**MPCR measurements of GR and GR-regulated genes.**

(A) Relative expression of GR and two GR-regulated genes in wild-type and KO mice (n=3), *glucocorticoid receptor* (GR, p=0.01), *forkhead box O3* (Foxo3a, p=0.07) and *glucocorticoid-induced leucine zipper* (GILZ, p=0.02), was verified by rtPCR from total RNA from total kidney cortex. In addition, absolute CT-values are shown for GAPDH to exclude global down-regulation of gene expression (data are expressed as mean ± SD; T-test; *, $p < 0.05$; n.s., not statistically different).

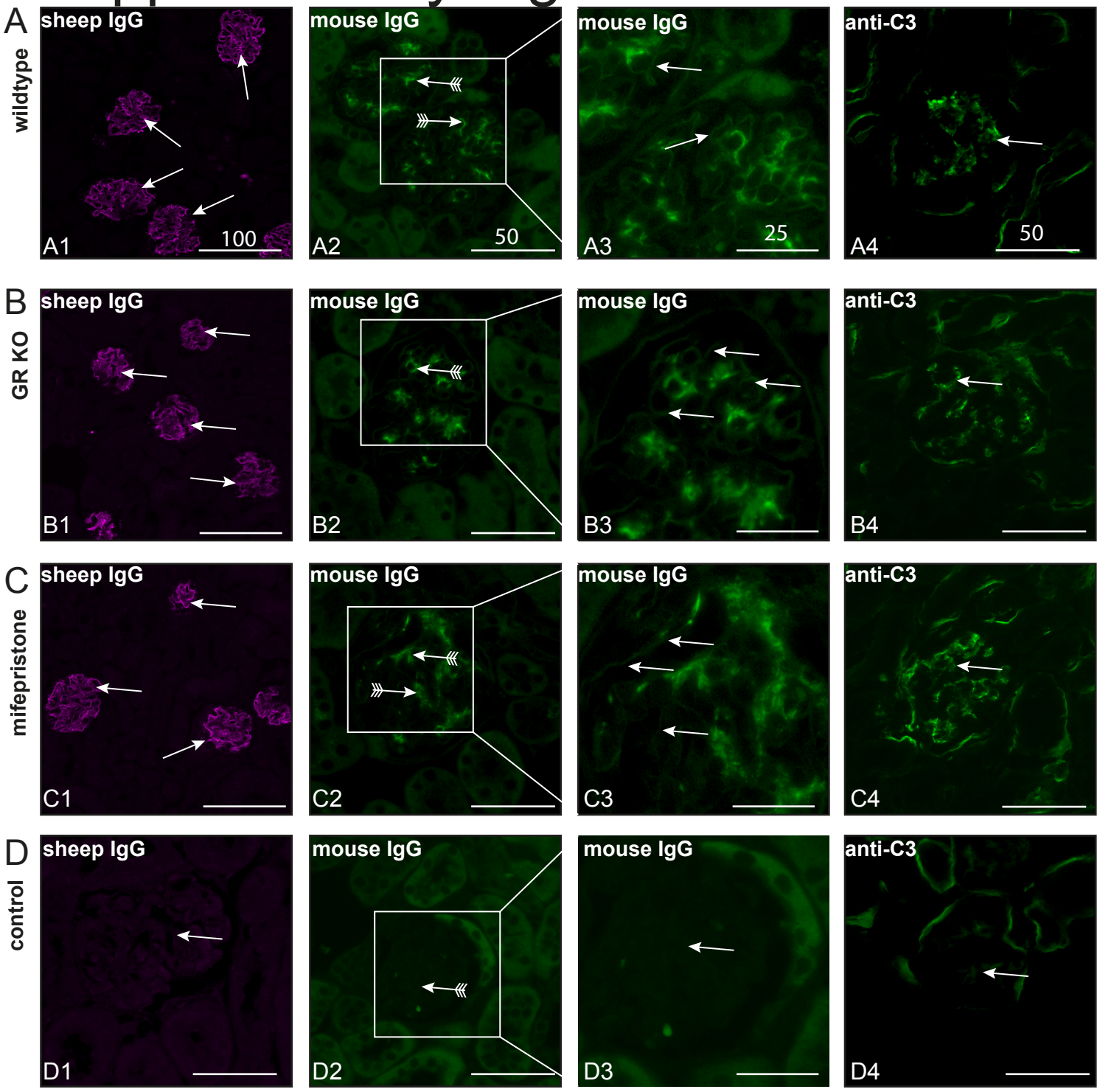
# Supplementary Figure 2



**Supplementary Figure 2**

Hydroxysteroid(11-beta)dehydrogenase 2 (HSD11b2) is expressed in highest amounts within the distal tubule and collecting ducts (arrow with tails). Expression is observed also within the proximal tubule (arrow). The glomerulus and tubulo-interstitium including blood vessels is negative (as detected by immunohistochemical staining using ab CAB032443, as well as on mRNA level as published on www.humanproteinatlas.com).

# Supplementary Figure 3

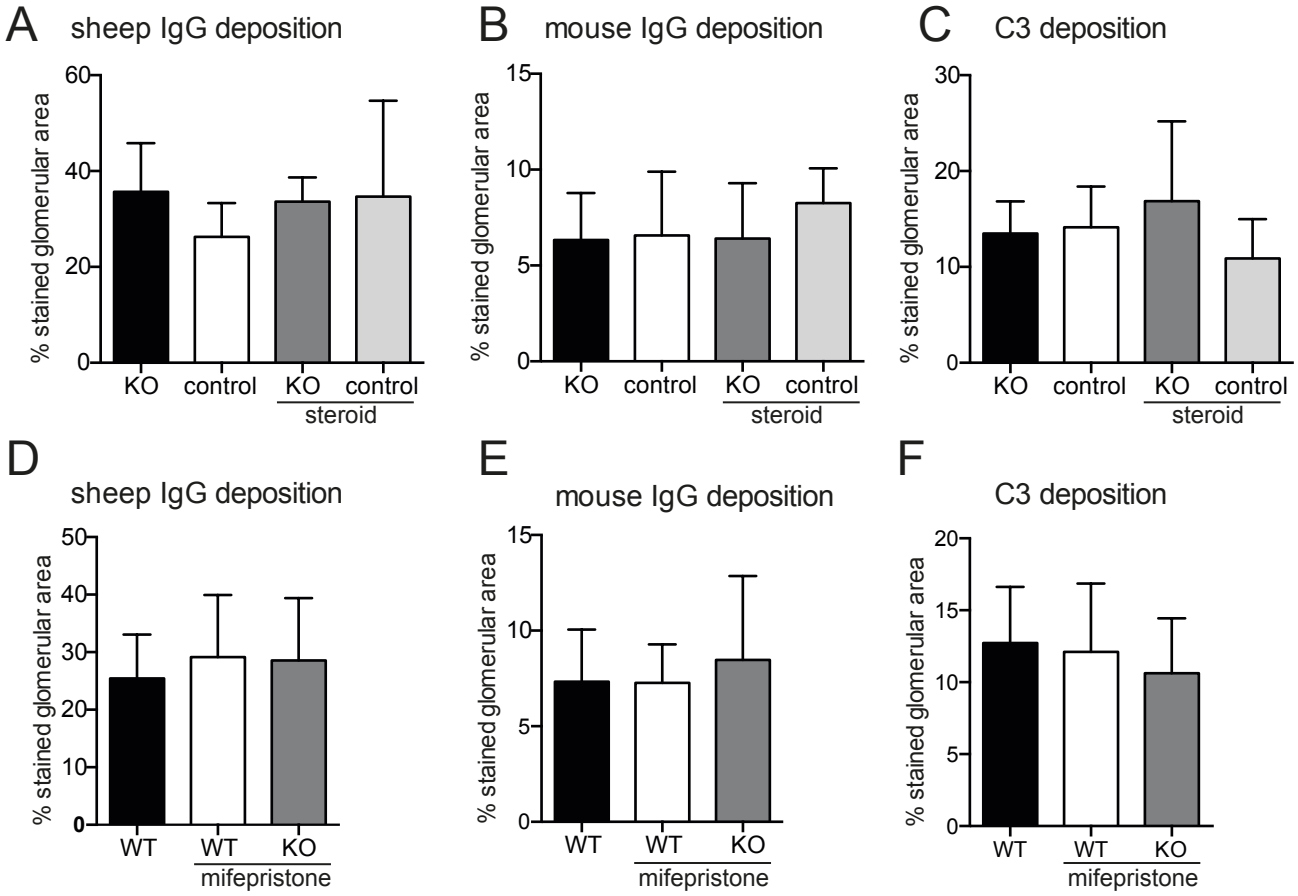


**Supplementary Figure 3**

**Analysis of glomerular IgG/C3 deposition.**

(A-D) Representative photographs of kidney sections from nephritic mice (wildtype, KO and mifepristone-treated, A-C respectively) stained for sheep IgG, mouse IgG and complement factor C3 at day 14 upon NTN induction. As control kidney sections from non-injected mice were used (D1-D4). While sheep IgG deposition occurred in a linear pattern in all groups (arrows in A1-C1), mouse IgG deposition also showed a granular deposition (arrows with tails in A1-C3). Anti-complement C3 deposition is shown in A4-C4. In comparison no sheep, mouse or C3 glomerular deposition could be detected in control mice. (scale bars: 100 µm in A1-D1; 50 µm in A2-D2 and A4-D4; 25 µm in A3-D3).

# Supplementary Figure 4

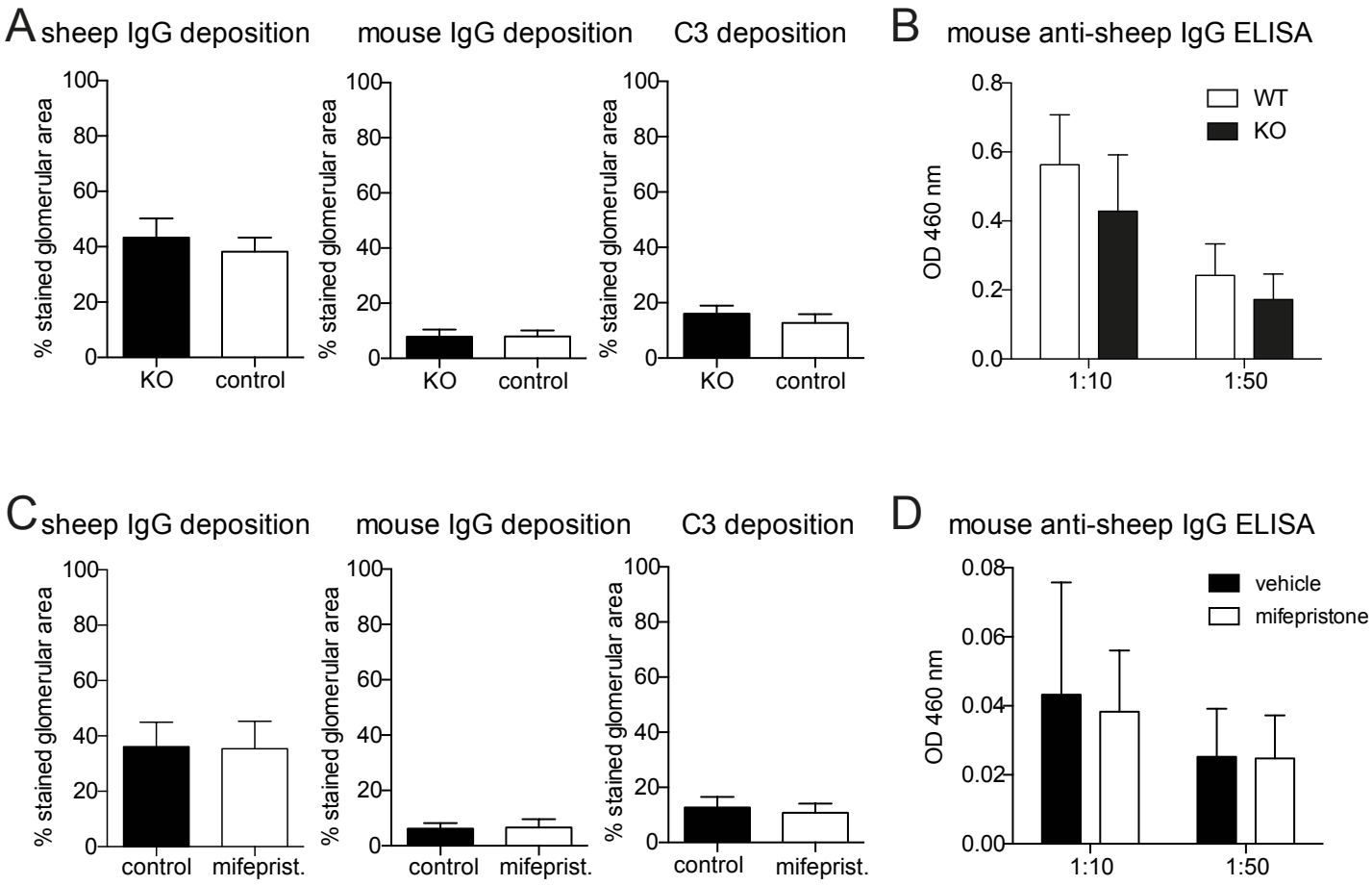


**Supplementary Figure 4**

**Quantification of glomerular IgG/C3 deposition**

(A-C) Quantification of glomerular sheep-IgG (A), mouse IgG (B) and complement C3 (C) of KO and control mice treated with or without steroids from the experiments displayed in Figure 3. (D-F) Quantification of glomerular sheep-IgG (A), mouse IgG (B) and complement C3 (C) of KO and WT mice treated with or without mifepristone from the experiment displayed in Figure 6. (data are expressed as mean ± SD; T-test; no significant statistical differences).

# Supplementary Figure 5

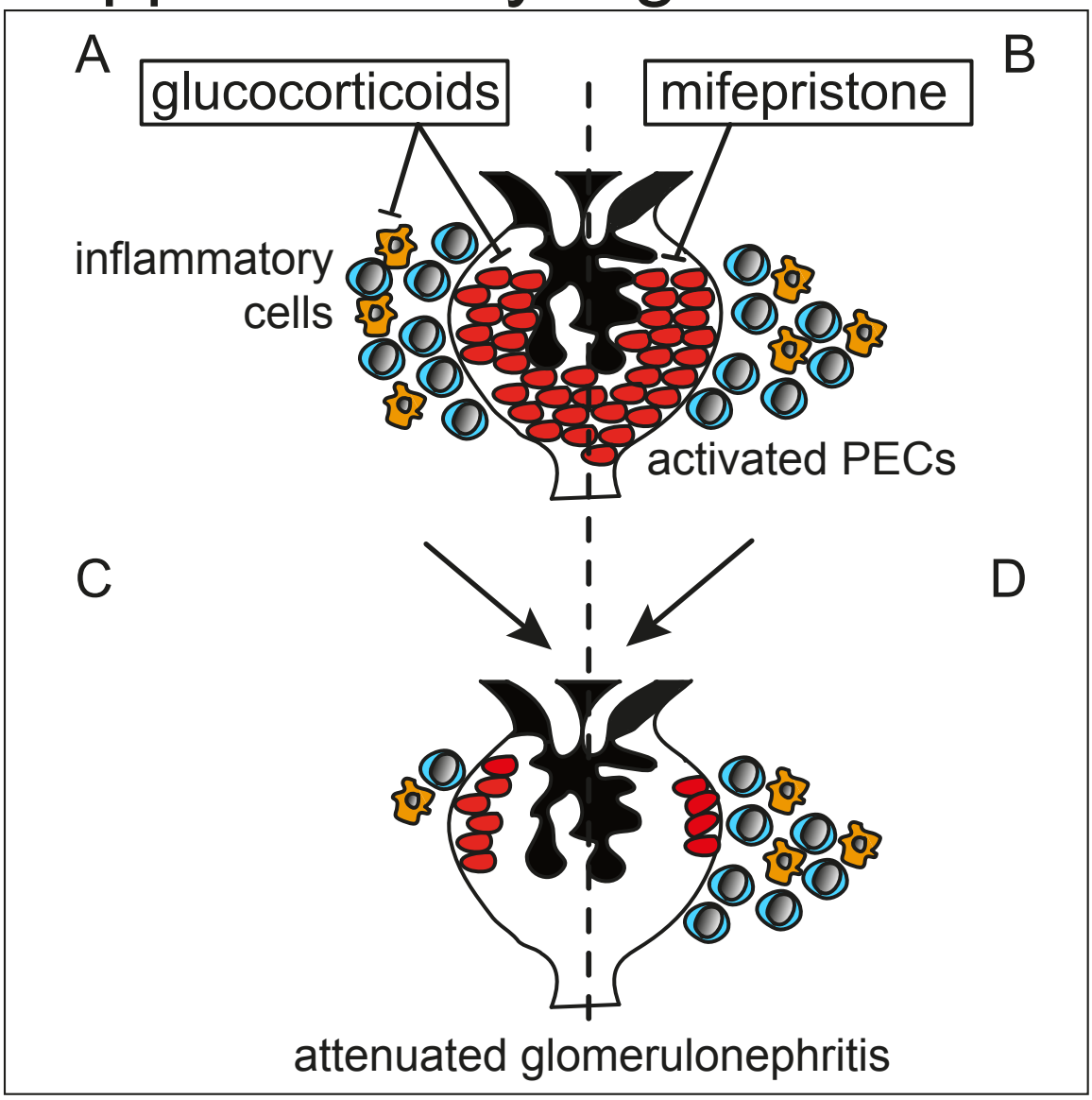


**Supplementary Figure 5**

**Immune response in KO and mifepristone-treated mice vs. control**

(A-D) In separate experiments the immune response of KO and mifepristone-treated mice vs. control was tested using glomerular sheep-IgG (A), mouse IgG (B) and complement C3 deposition as surrogate markers. Anti-sheep mouse ELISA results are shown in B and D (data are expressed as mean ± SD; T-test; none reach statistical significance). (A-B) Comparison of Pax8CreGR$^{flox/flox}$ vs. wildtype littermates (C-D) and of mifepristone-treated vs. vehicle-treated littermates.

# Supplementary Figure 6



**Supplementary Figure 6**

**Summary of experimental findings.**

(A-B) Inflammatory cells as well as activated parietal epithelial cells (PECs) participate in rapid progressive glomerulonephritis (RPGN). High-dose glucocorticoids inhibit both inflammatory cells and PECs (A, C). In addition, our findings show that mifepristone inhibited PECs directly (B). While high-dose prednisolone exerted immune-suppressive effects (C), no direct effects of mifepristone were observed on immune cells (in D).