

SUPPLEMENTAL FIGURES

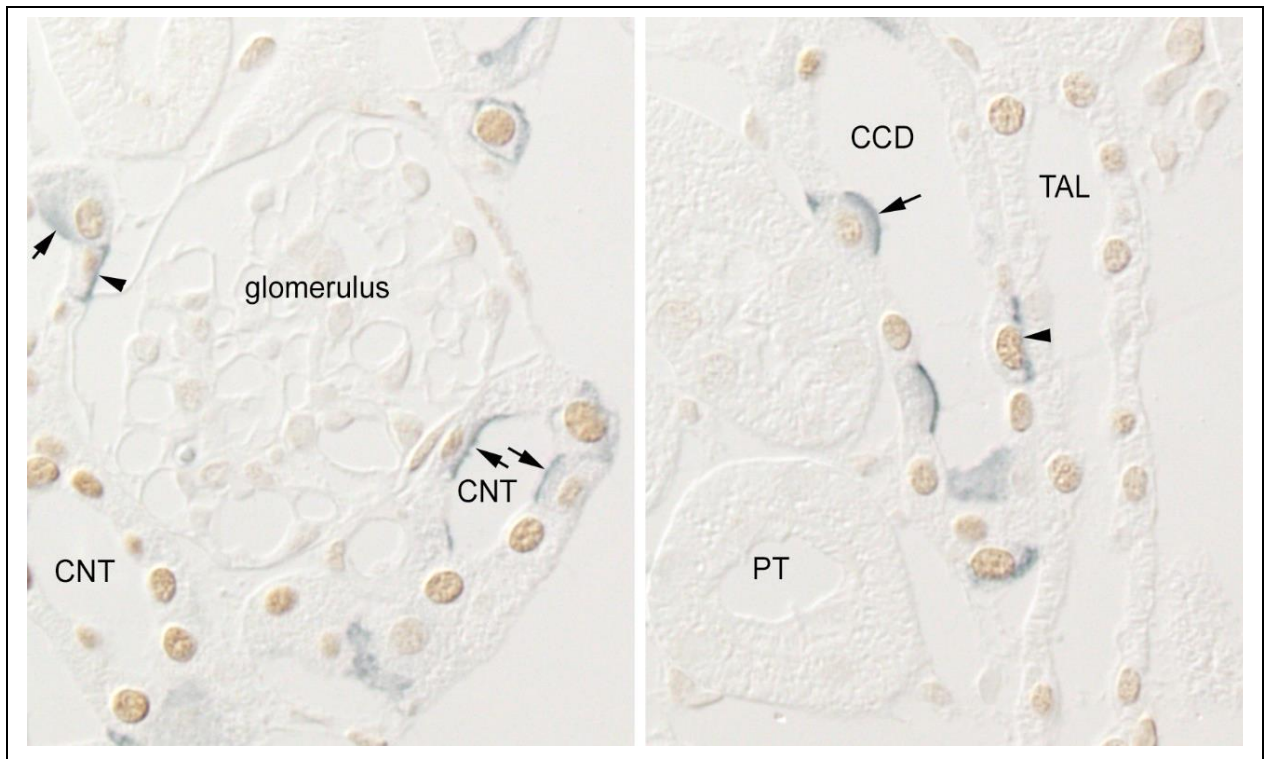
Table of Contents:

Supplemental Figure 1, page 2. MR labeling in the cortex of NaCl-deficient wild type (floxed MR) mice.

Supplemental Figure 2, page 3: Current voltage relationship of single channel recordings from CCD principal cells taken from aldosterone-treated IC MR null and wild type littermates.

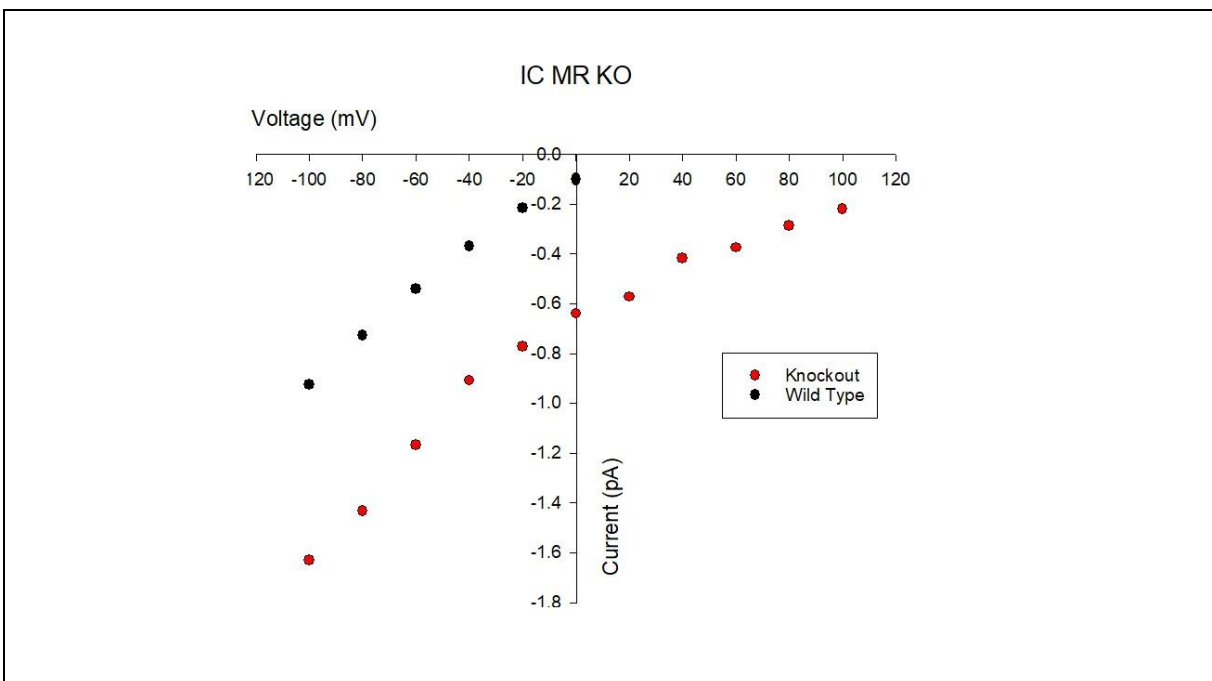
Supplemental Figure 3, page 4. Abundance of the 80 and 30 kD fragments of α ENaC taken from aldosterone-treated IC MR null and wild type littermates.

Supplemental Figure 1: MR labeling in the cortex of NaCl-deficient wild type (floxed) mice. These figures shows MR (brown) and pendrin/AE1 (blue) immunolabel label in cortical sections from NaCl-deficient (Treatment #1) wild type (floxed MR) mice. The left panel shows that MR label is very weak or absent in the glomerulus, but prominent in the CNT. Apical pendrin label (arrows) and basolateral AE1 immunolabel (arrowheads) are seen in the CNT but not in the glomerulus. The right panel shows no detectable MR label in the proximal tubule (PT), although clear MR label is seen in the thick ascending limb (TAL) and the CCD. Apical pendrin label (arrows) and basolateral AE1 label (arrowheads) are seen in the CCD, but not in PT or TAL.



Supplemental Figure 2: Current voltage relationship of single channel recordings from CCD principal cells taken from aldosterone-treated IC MR null and wild type littermates.

This figure shows the current from individual events in the single channel records displayed in Figure 2 C (*Treatment 4*). It shows that currents inwardly rectify, which is characteristic of epithelial sodium channels (ENaC). The difference in the two I-V relationships is because wild type mice have greater ENaC activity (and greater intracellular Na^+), which shifts the I-V relationship to the left, whereas the IC MR null mice have reduced ENaC activity (and lower intracellular Na^+), which shifts the I-V relationship to the right. This implies that the wild type mice have a relatively depolarized apical membrane potential as compared to the IC MR null mice.



Supplemental Figure 3: Abundance of the 80 and 30 kD fragments of α ENaC taken from aldosterone-treated IC MR null and wild type littermates. This figure shows an immunoblot of kidney lysates from aldosterone-treated IC MR null and wild type littermates probed for α ENaC, using an antibody described previously that recognizes amino acids 2-21 of the mouse α ENaC sequence. The 85 and 30 kD band density obtained with this antibody were combined with the 85 kD band densitometry obtained in separate mice that employed an antibody that recognizes amino acids 46 to 68 or the rat α ENaC sequence. These values are displayed in Figure 2E.

