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Urine concentration and key transporters of renal tubules

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Body water homeostasis is mainly established by the kidney function, such as tubular reabsorption of water and sodium through water channel proteins (aquaporins: AQPs) and sodium-cotransporters. As water can slowly diffuse through biomembranes composed of lipid bilayers, all membranes exhibit some degree of water permeability. Nonetheless, the plasma membranes of the renal tubular epithelia have distinctly high water permeability for water transport. Water reabsorption in the kidney tubule depends on the driving force (i.e., high interstitial osmolality/tonicity) and osmotic equilibration of water across the tubular epithelia (i.e., high osmotic water permeability of the membrane). The majority of fluid filtered in the glomerulus is constitutively reabsorbed in the proximal tubules and descending thin limbs. The subsequent renal tubular segments, i.e., ascending thin limbs, thick ascending limbs, and distal convoluted tubules, are relatively water-impermeable and hence the tubular fluid could be delivered into the connecting tubules and collecting ducts. Kidney collecting duct is an important renal tubular segment for regulation of body water homeostasis and urine concentration. Water reabsorption in the collecting duct principal cells is controlled by vasopressin, a peptide hormone which induces the osmotic water transport across the collecting duct epithelia through regulation of water channel proteins aquaporin-2 (AQP2) and aquaporin-3 (AQP3). In particular, vasopressin induces both intracellular translocation of AQP2-bearing vesicles to the apical plasma membrane and transcription of Aqp2 gene to increase AQP2 protein abundance. The signaling pathways, including AQP2 phosphorylation, RhoA phosphorylation, intracellular calcium mobilization, and actin depolymerization, play a key role in the translocation of AQP2. This session will summarize recent data demonstrating the underlying molecular mechanisms for the homeostasis of water balance in the body.