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Molecular Basis for Renal Tubular Acidosis

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The kidney plays a critical role in acid-base homeostasis by the reabsorption of filtered bicarbonate and the generation of new bicarbonate. In the proximal tubule, HCO_3^- reabsorption occurs via the transcellular coupling of the apical Na^+/H^+exchanger with the basolateral $Na^+(HCO_3^-)$ cotransporter (NBC), which plays a critical role in mediating electrogenic bicarbonate efflux. In the collecting duct, acid secretion is primarily mediated by a vacuolar H^+-ATPase and H^+-K^+-ATPase located in the apical plasma membranes of type-A intercalated cells. Basolateral bicarbonate efflux is mediated by the anion exchanger AE1 in this cell type. In type-B intercalated cells, the vacuolar H^+-ATPase is located on the basolateral membrane. This cell type is thought to mediate bicarbonate secretion in the collecting duct via an apical anion exchanger pendrin. Renal ammonia excretion is the predominant component of renal net acid excretion. In the proximal tubule, the apical Na^+/H^+ exchanger (NHE3) facilitates the reabsorption of Na^+ in exchange for H^+, contributing indirectly to NH_4^+ secretion. In the thick ascending limb of Henle, luminal ammonium ion is reabsorbed through the apical Na^+-K^+-$2Cl^-$ cotransporter and basolateral NHE4 for the accumulation of ammonium ions in the medulla. Also, in the cells, ammonium ion is dissociated into ammonia and hydrogen ions, and ammonia is transported to the interstitium by diffusion. The collecting duct is a major site for renal ammonia secretion, involving parallel H^+ secretion and NH_3 secretion. Renal tubular acidosis (RTA) is a disorder characterized by an inability of the kidneys to properly acidify urine or reabsorb bicarbonate, leading to systemic acidosis. There are several types of RTA, each associated with different molecular mechanisms affecting various parts of the renal tubules. Each type of RTA presents unique challenges in managing electrolyte imbalances and systemic acidosis.

Keywords: acid-base, H+ secretion, HCO3- reabsorption, Renal tubular acidosis, electrolyte imbalance