

Q22 Describe how the kidney handles sodium. (50 marks) What factors influence urinary sodium excretion (50 marks) (Sept 2009)

RENAL SODIUM HANDLING

Kidneys filter approx. 180L/ day, including essential nutrients (glucose and amino acids), electrolytes and organic anions. Up to 99% of filtered Na is reabsorbed along the tubule.

PROXIMAL TUBULE – reabsorbs 65% of filtered sodium. Basolateral Na/K ATPase creates an electrochemical gradient for Na resorption via an Na/organic ion (eg, glucose) cotransporter and an Na/H exchanger.

THIN DESCENDING LIMB OF LOOP OF HENLE – no Na resorption

THICK ASCENDING LIMB OF LOOP OF HENLE – 25% of active Na resorption via Na/K/2Cl cotransporter. Once again a basolateral Na/K ATPase pump sets up an electrochemical gradient. Na is reabsorbed along with K and Cl, and there is passive resorption of further Na, Mg, Cl and K down their concentration gradient through intercellular channels.

EARLY DISTAL TUBULE – 6% of Na resorption through an Na/Cl cotransporter with a basolateral Na/K ATPase pump setting up the electrochemical gradient.

LATE DISTAL TUBULE / CORTICAL COLLECTING DUCT – Remaining 2-3% of sodium reabsorbed via the ENaC channel (under the influence of aldosterone). Electrochemical gradient maintained by basolateral Na/K ATPase pump. Hydrogen and potassium ions excreted into urine.

FACTORS INFLUENCING URINARY SODIUM EXCRETION:

- SYSTEMIC
 - Decreased renal perfusion (eg secondary to hypovolaemia or hypotensing drugs) will reduce GFR and thus sodium excretion
 - Increased systemic sympathetic drive will again constrict the renal arterioles and reduce GFR
- HORMONAL
 - Aldosterone → released from zona glomerulosa of the adrenal cortex to upregulate the activity of the CCD Na/K ATPase and the ENaC channel to increase sodium resorption in exchange for k secretion.
 - Renin → secreted in response to sympathetic stimulation and direct stimulation from renal macula densa. Cleaves angiotensin 1 from angiotensinogen, ATI then converted to AT2 which causes vasoconstriction especially of efferent arteriole, reducing the peritubular capillary hydrostatic pressure to increase the gradient for reabsorption of Na (and water and other electrolytes) in the tubules. AT2 also stimulates the activity of the Na/H exchanger in the proximal tubule and upregulates the Na/K ATPase throughout the tubule system.
 - Atrial natriuretic peptide → secreted from the atria in response to stretching. Opposes Na resorption in the tubules, increases the filtration coefficient at the glomerulus, and inhibits the release of renin
 - Insulin and cortisol → increase Na resorption (minor)