

The kidneys are able to secrete different concentrations of urine depending on the body's needs

Renin-angiotensin-aldosterone system RAAS involved in regulating  $\text{Na}^+$

Renin converts Angiotensinogen to Angiotensin I

Angiotensin I converted to Angiotensin II by ACE in lungs

Angiotensin II

↳ Vasoconstrictor

Induces thirst

Stimulates the release of vasopressin

Stimulates the release of aldosterone

Aldosterone increases  $\text{Na}^+$  reabsorption in distal tubules  
↓ lumen → Interstitium → Peritubular capillary

RAAS is a long-term mechanism for increasing blood volume



Interstitial fluid via osmosis

Up tubular lumen into surrounding interstitium. Impermeable to water

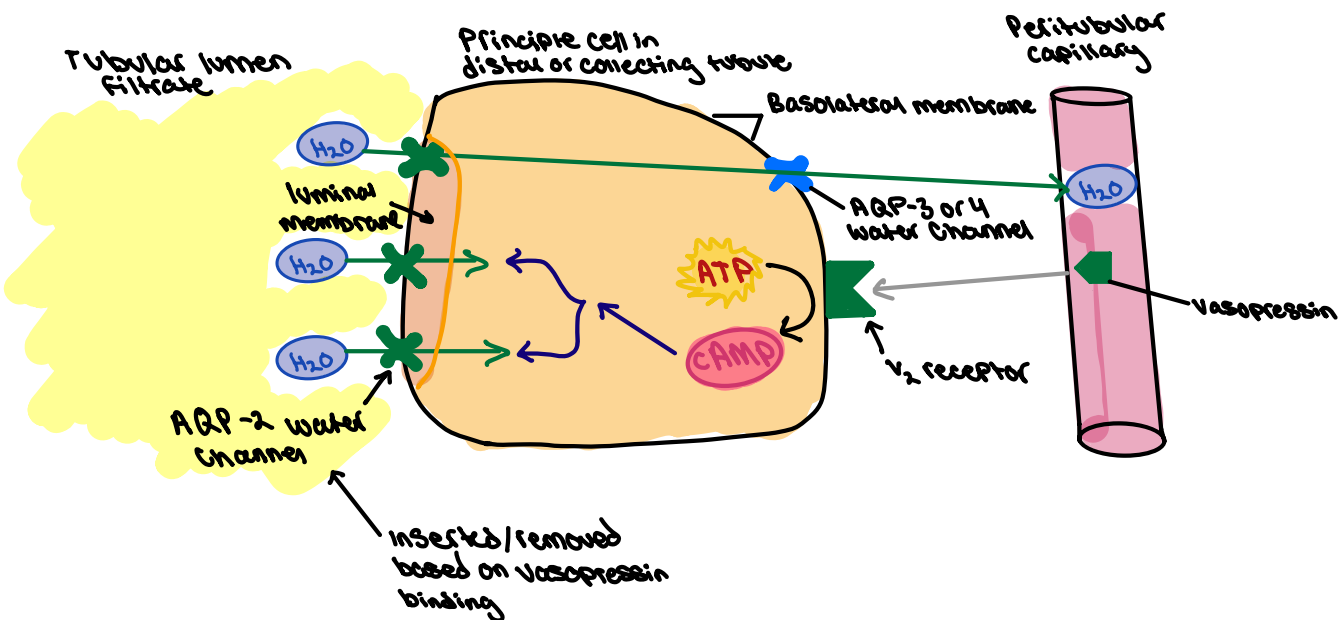
Vasopressin: Produced in the hypothalamus and stored in the posterior pituitary

- Its release signals the distal tubule and collecting duct to absorb water

Dehydration Vasopressin secretion increases (less water elimination)

Excess hydration Vasopressin secretion decreases (more water elimination)

Mechanical action of Vasopressin + Water reabsorption



Aldosterone: Increases Na<sup>+</sup> reabsorption

Increases K<sup>+</sup> secretion

Aldosterone secretion by adrenals is directly increased by high  $K^+$  (hyperkalemia) and indirectly increased via RAAS by low  $Na^+$  (hyponatremia)

The point of the hypertonic medulla isn't to change tonicity of the urine flowing along the lumen of the LOH as it dips down into the medulla, but rather to provide a gradient for water to leave the tubule lumen once it has entered the distal tubule and collecting duct, under influence of ADH.