Renal Transport Process 2

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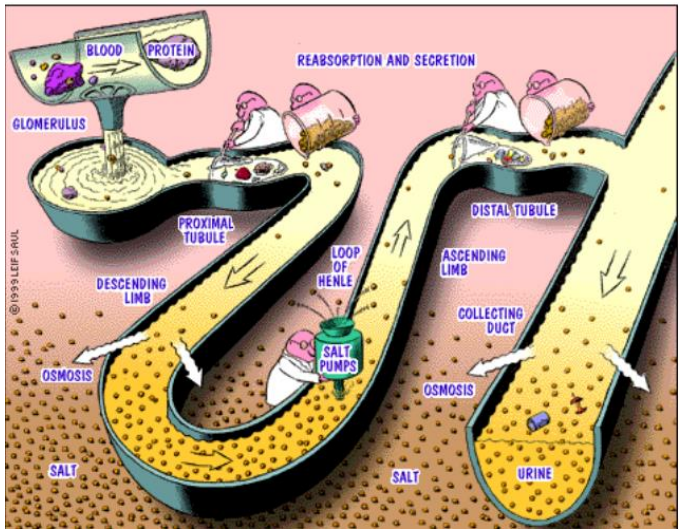


Learning Objectives:

- Describe tubular secretion with PAH transport and K+
- Identify and describe the characteristic of loop of Henle, distal convoluted tubule and collecting ducts for reabsorption and secretion
- Identify the site and describe the influence of aldosterone on reabsorption of Na+ in the late distal tubules.

Loop of Henle

- **Thin descending limb**
  - 15% water absorbed
  - permeable to water (filtrate hyperosmotic)
- **Thick ascending limb (TAL)**
  - Impermeable to water (isosmotic)
  - Important in concentrating urine
  - 25% NaCl, K+ reabsorbed as well as Ca_2^+, HCO_3^-occurs in

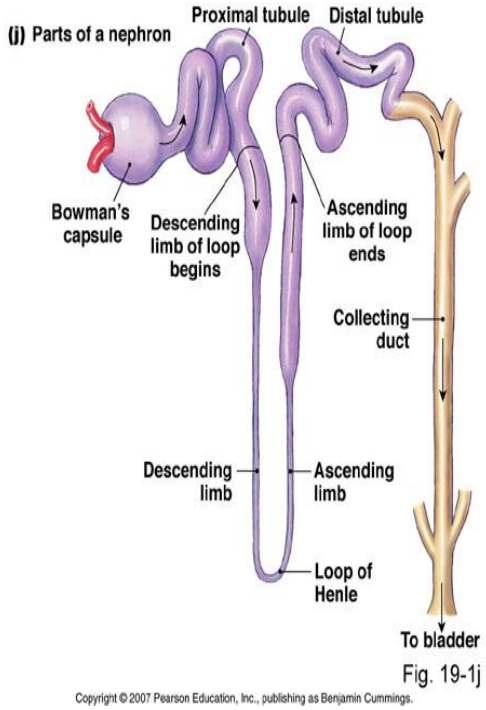


Fig. 19-1j

Loop of Henle

- Solute absorption (TAL):

1) **Transcellular** (50%)

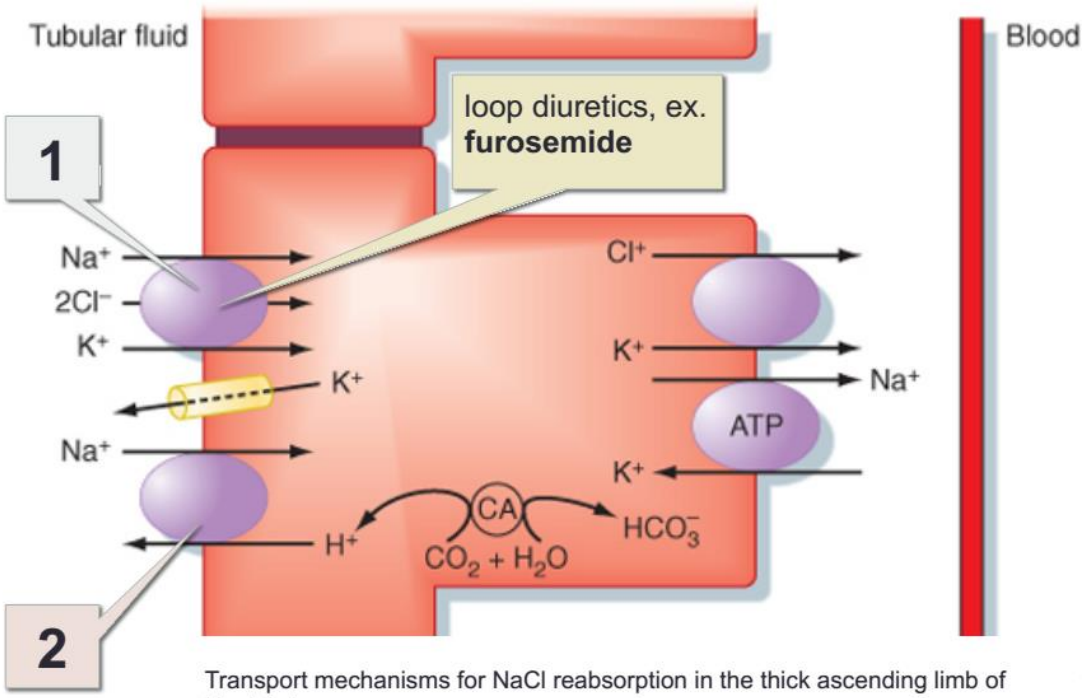
   a) Na+/2Cl-/K+ cotransporter/ symporter

   b) NHE

      i) Na+ in

      ii) H+ out

      iii) HCO3- in

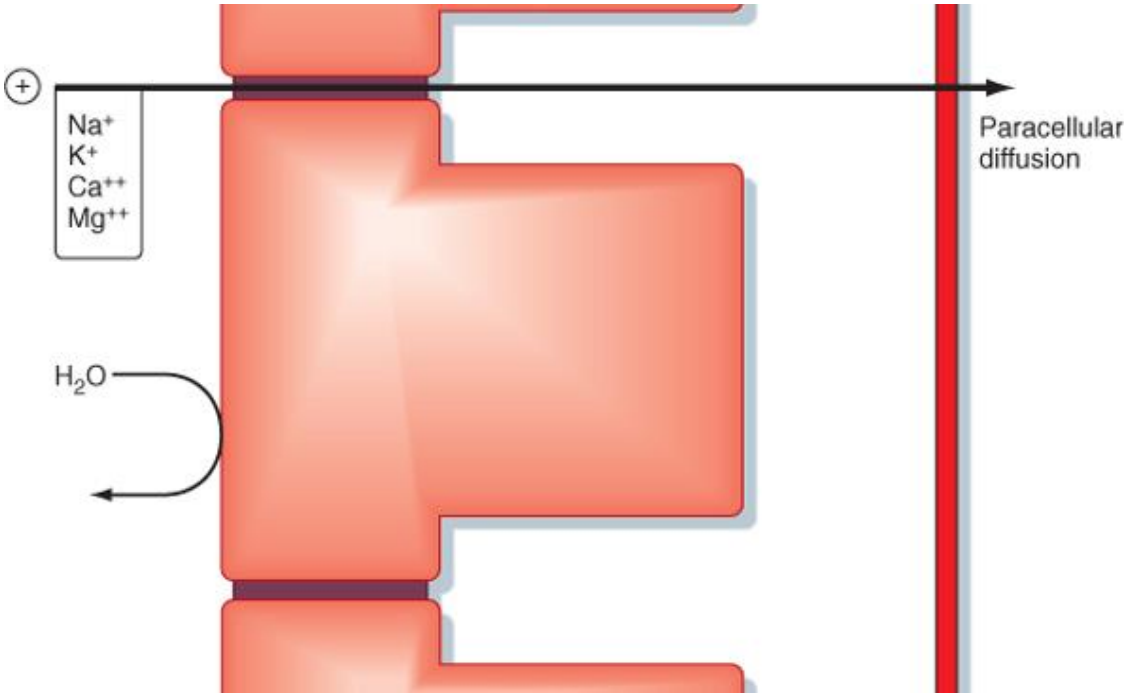


Transport mechanisms for NaCl reabsorption in the thick ascending limb of Henle's loop.

Loop of Henle

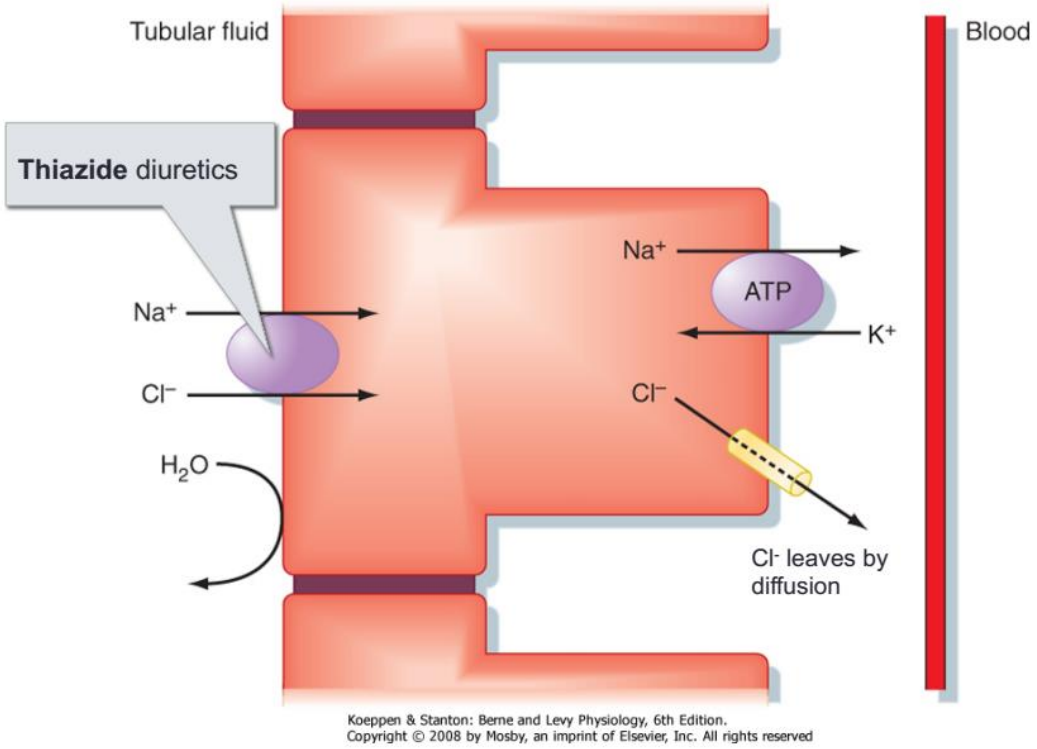
2) **Paracellular** (50%)

- Loss of NaCl in tubule



Distal convoluted tubule (DCT) & collecting duct (CD)

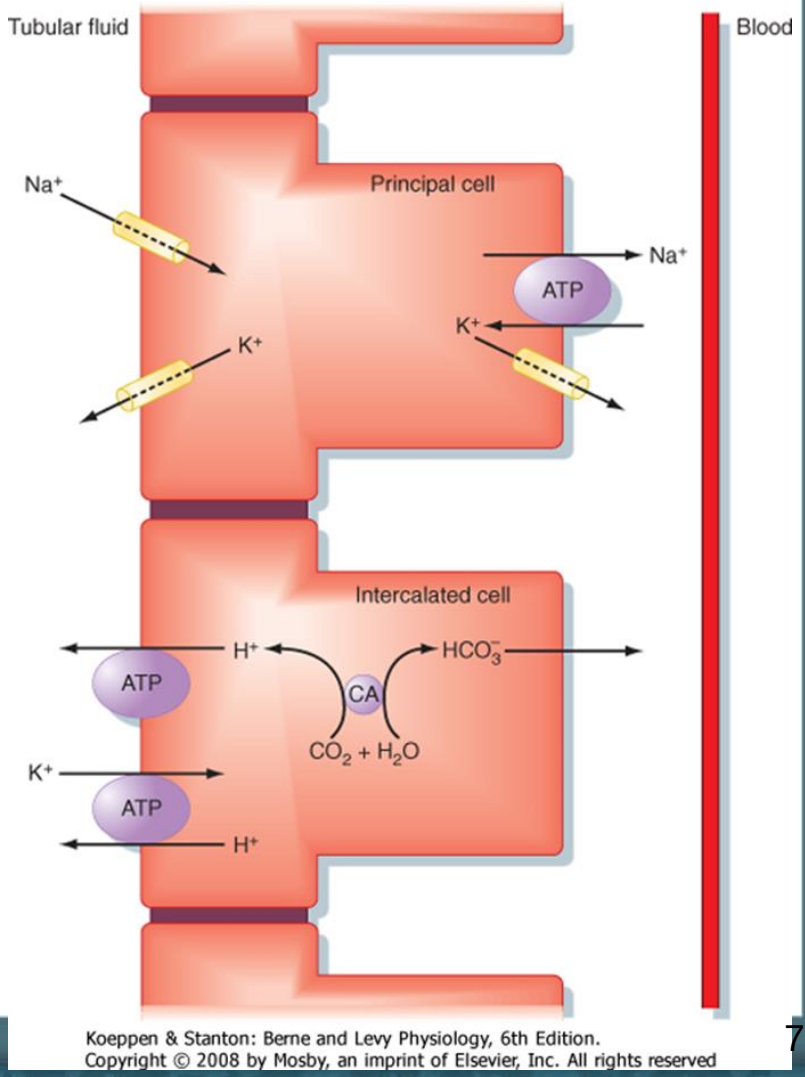
- 7% NaCl
- 8 – 15 % water reabsorbed (needs ADH)
- Some K^+, H^+ secreted **into** tubule
- **Early DCT**:
- Reabsorbs Na^+, Cl^-
- (impermeable to water)



Distal convoluted tubule (DCT) & collecting duct (CD)

Late DCT:

- **Principle cells:**
  - reabsorb Na^+, Na+ diffuses via selective channels
  - Reabsorb water
  - secrete K^+
- **Intercalated cells:**
  - secrete H+
  - reabsorb HCO3-
  - reabsorb K^+
- **Aldosterone**:↑Na reabsorption by principle cells, ↑K+ secretion



Transport of potassium

- Most abundant cation in the body
- 3,500-4,000 mmol in blood.
- 98 % is intracellular, [150mM]
  - Regulates intracellular function such as Cell volume, Acid/base status, cell growth & division
- 2% K extra-cellular [3.5-5mM]
  - This regulates membrane potentials in excitable cells and diffusion potentials in transporting epithelia.

- K^+ Intake 80-120 mmol/day
- Tissue damage leading to cell lysis increases plasma $[K^+]$
- Both extracellular $[K^+]$ and total body potassium are tightly regulated.

HOW?

INTERNAL DISTRIBUTION

(This regulates extracellular $[K^+]$)

RENAL K^+ EXCRETION

(This regulates total body potassium)

Internal potassium distribution

- Potassium content of average meal is 30-40mmol. This is rapidly absorbed.
- Renal elimination is slow. It can take up to six hours eliminate this load.
- If nothing happened then this absorbed load would cause Plasma [K^+] to rise by ~ 2-5mmol which is potentially lethal.
- Buffering of the load occurs by increased intracellular uptake via Na^+/K^+ pump into Skeletal Muscle, Liver, Bone RBCs etc.
- Loss of K^+ from exercising muscle can seriously increase plasma K^+ ,trained athletes show accelerated uptake after exercise

Renal excretion of potassium

- 90-95% of Dietary K excreted via the kidneys
- 5-10% in Sweat & Feces (This is unregulated and may become significant in diarrheas)
- In normal individual intake is matched by excretion and potassium balance is maintained.
- Filtered load of potassium ~ 720 mmol/day
- **Bulk reabsorbed by proximal tubule and loop of Henle.**

Renal K^+ Transport mechanisms

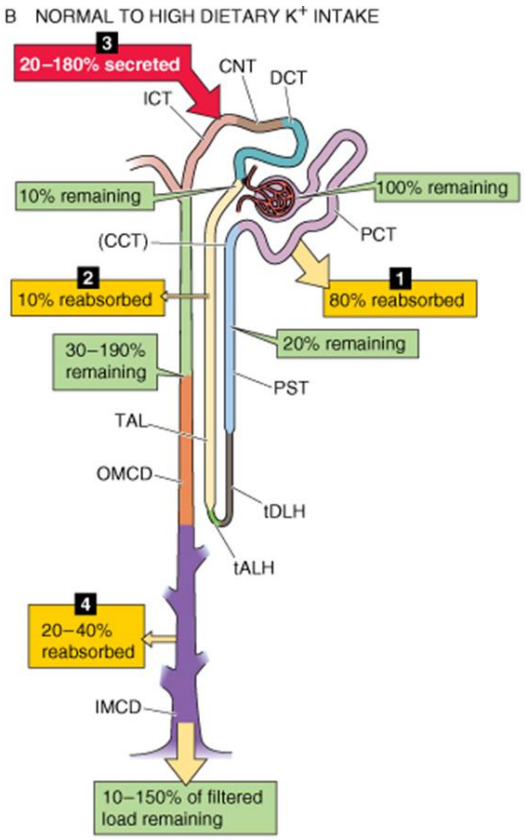
- Cell membrane transporters

  - Na^-K ATPase, H^-K ATPase

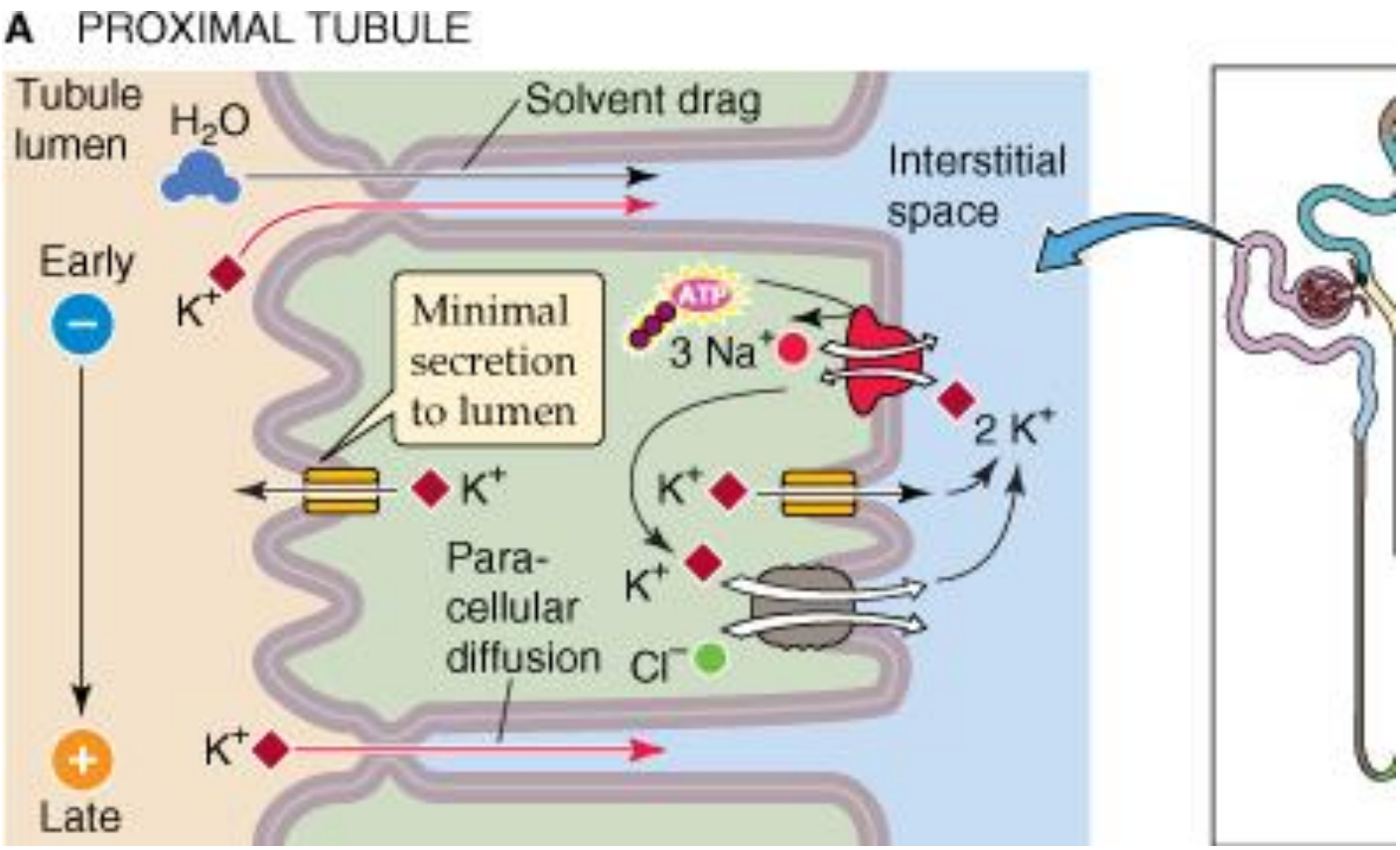
  - K^+ channels, K:Cl cotransport

  - Na:K:2Cl cotransport

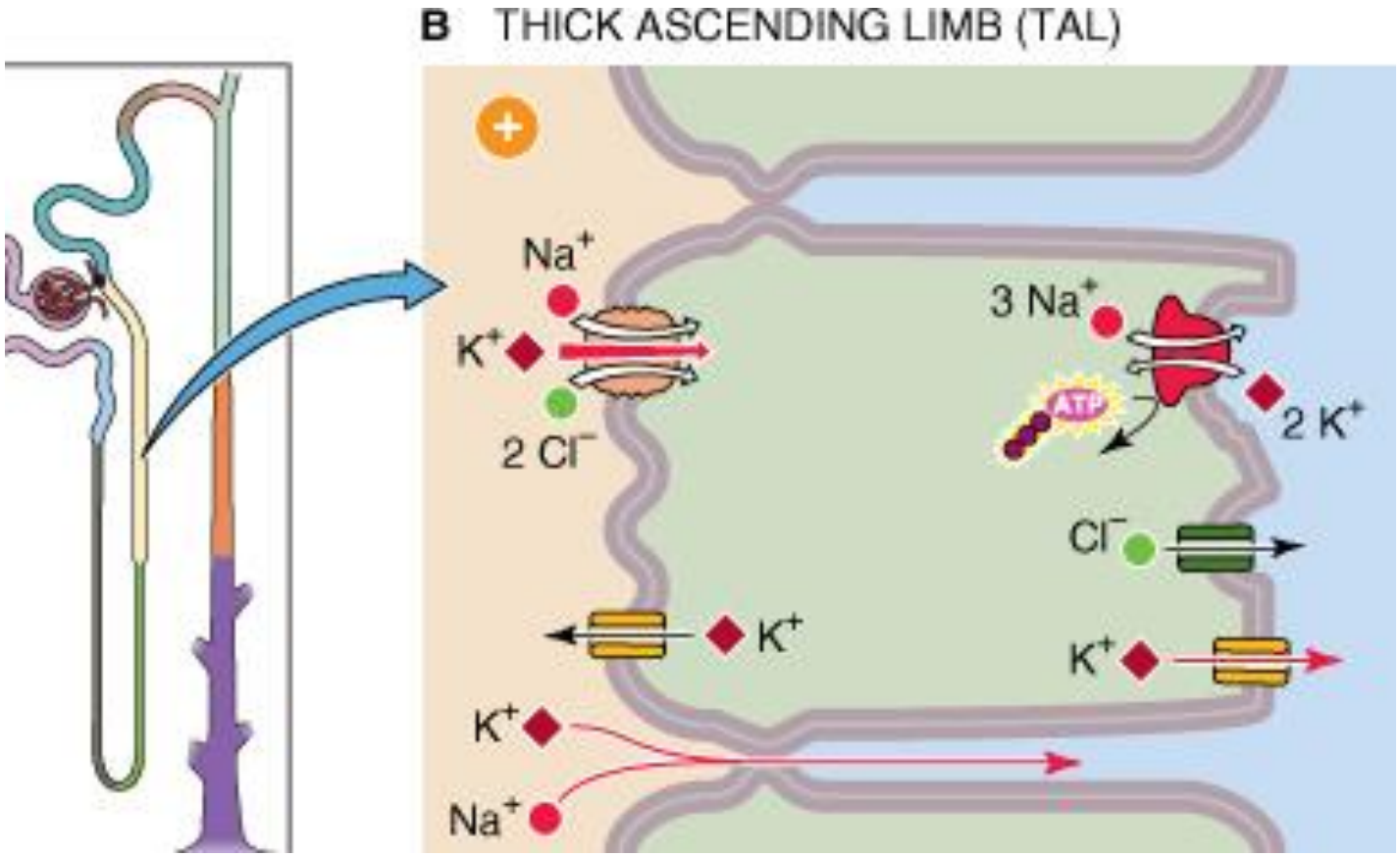
- **K^+ is Reabsorbed** in PT, TAL & intercalated cell in CCD
- **K^+ Secreted** in late distal tubule and in principal cells of late DT & CCD



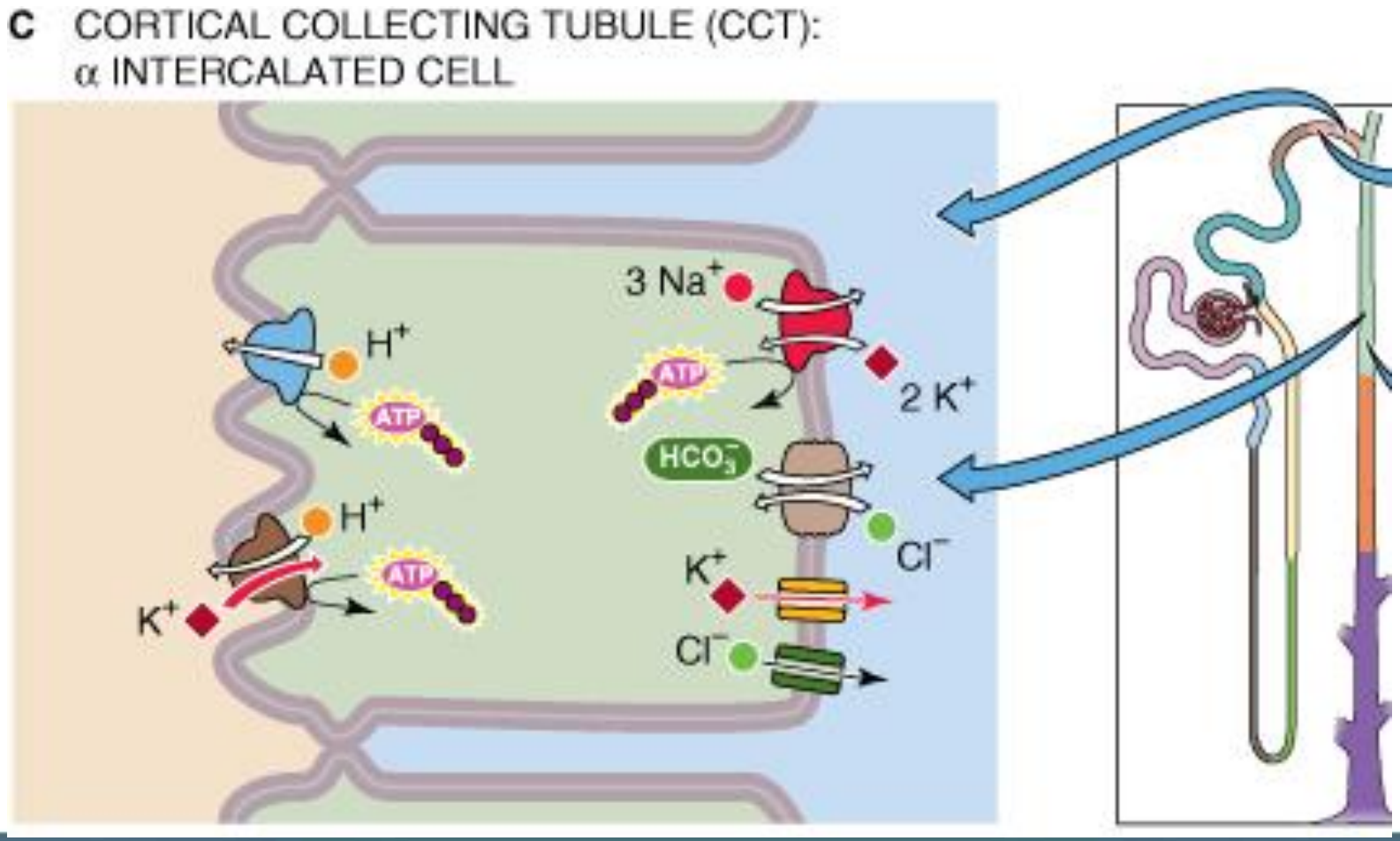
- **Proximal Tubule:** K^+ is absorbed by intercellular solvent drag whereby fluid movement driven by Na^+ absorption entrains K^+ ions



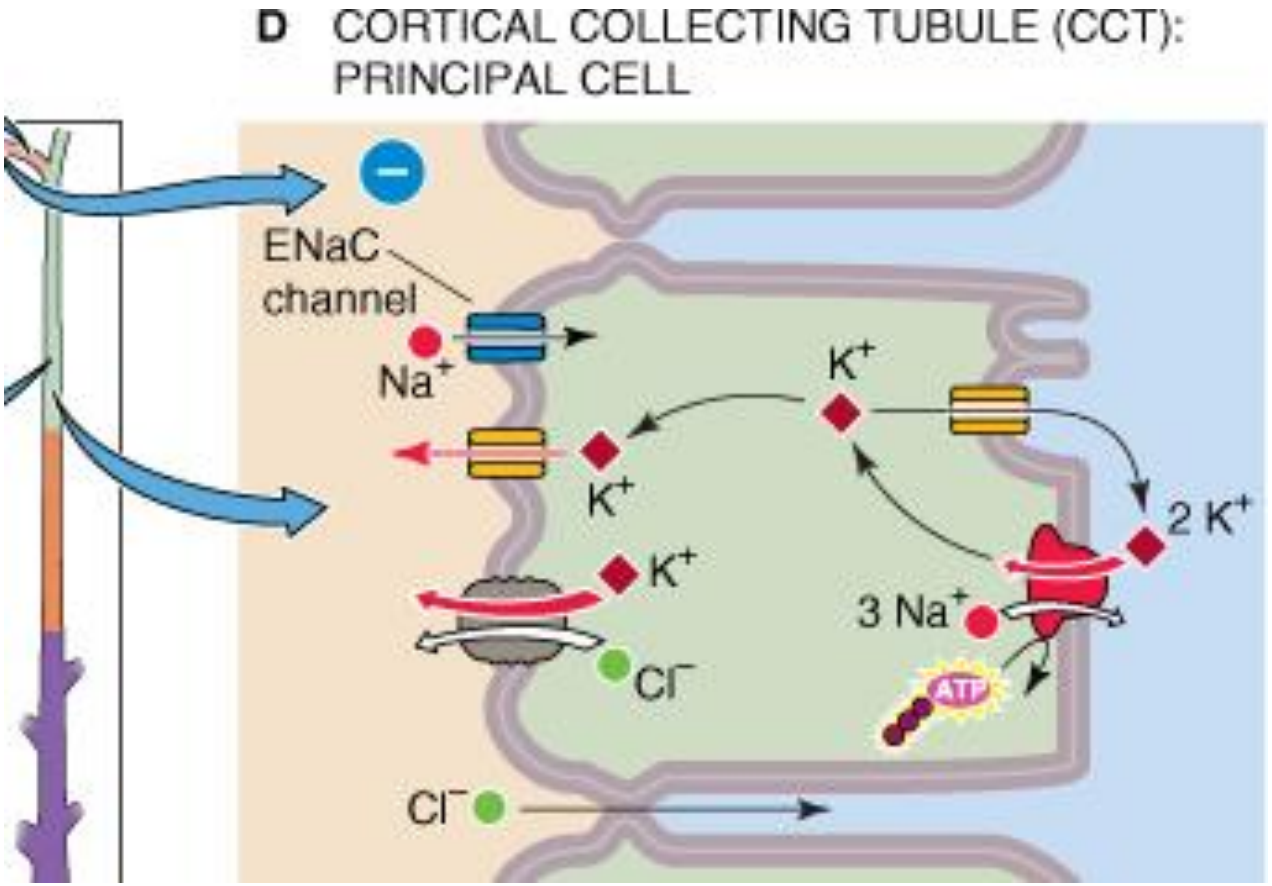
- **TAL:** Na:K:2Cl in luminal membrane
- K:Cl co-transport in baso-lateral membrane



CD: K reabsorption is by the intercalated cells via a luminal H-K ATPase.



- **CD:** K+ secretion in the principal cells (via luminal K channels and basolateral Na-K ATPase).



Factors affecting potassium secretion

Peritubular factors:

1.Hyperkalemia: increase K in tubular cells, increase chemical gradient of K between tubular cell and tubular lumen which lead to increase in the secretion and excretion of K.

2.Hyper-aldosteronism: increase aldosterone increase secretion and excretion of K.

3.Alkalosis: increase H-K exchange at baso-lateral membrane then increase secretion and excretion of K.

Factors affecting potassium secretion

Luminal factors:

1.Diuresis: increase volume of urine and decrease conc of K in lumen which causes secretion via chemical gradient. (increase secretion and excretion)

2.Increased urinary excretion of Na: increase in Na-K exchange at luminal membrane causes an increase in secretion and excretion of K.

3.Increased urinary excretion of bicarbonate, phosphate, sulphate and ketone acids: increase negativeness of lumen then increase electrochemical gradient between cell and lumen causes secretion and excretion of K.

NaCl Transport along the Nephron

Segment	Percentage of Filtrate Reabsorbed	Mechanism of Na^+ Entry across the Apical Membrane	Major Regulatory Hormones
Proximal tubule	67%	Na^+-H^+ antiporter, Na^+ symporter with amino acids and organic solutes, $1Na^+$-$1H^+$-$2Cl^-$-anion antiporter, paracellular	Angiotensin II Norepinephrine Epinephrine Dopamine
Loop of Henle	25%	$1Na^+$-$1K^+$-$2Cl^-$ symporter	Aldosterone Angiotensin II
Distal tubule	≈5%	NaCl symporter (early) Na^+ channels (late)	Aldosterone Angiotensin II
Collecting duct	≈3%	Na^+ channels	Aldosterone, ANP, BNP, urodilatin, uroguanylin, guanylin, angiotensin II

Water Transport along the Nephron

Segment	Percentage of Filtrate Reabsorbed	Mechanism of Water Reabsorption	Hormones That Regulate Water Permeability
Proximal tubule	67%	Passive	None
Loop of Henle	15%	Descending thin limb only; passive	None
Distal tubule	0%	No water reabsorption	None
Late distal tubule and collecting duct	≈8%-17%	Passive	ADH, ANP, BNP*

References

- Guyton and Hall Textbook of physiology
  - Chapter 27


Thanks