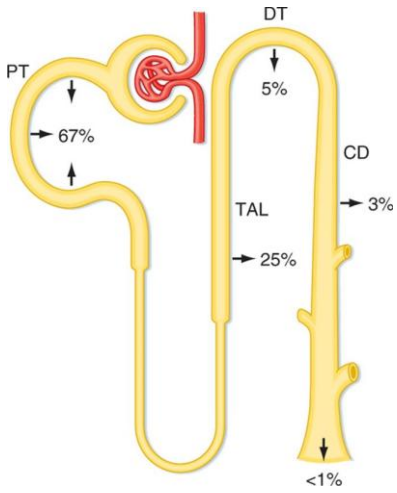
Renal Handling of sodium and potassium ions



1- Renal handling of Sodium ions :

- Normal Na^+ level (ECF) is 135 - 145 meq/L
- Na^+ is freely filtered and is actively transported out of all portions of the tubule except thin descending limb of loop of Henle.
- Na^+ is transported by the activity of Na^+ /k^+ ATP ase pump at the basolateral membrane from inside of the tubular cells to the interstitial fluid.
- Normally about 99% of the filtered Na^+ is reabsorbed
- Na^+ Transport occurs in different tubular segments:
  - PCT (~ 65%): Counter transport with H+ , cotransport with amino acid , glucose , lactate ,phosphate and paracellular diffusion.
  - Thick Loop of Henle (25%): Na- K -2Cl cotransport ,Counter transport with H^+.
  - Early D.C.T (5%): cotransport with Cl^-
  - The late DCT and collecting duct(3%) by ENaC
- **Na^+ reabsorption is regulated by** :
  - Angiotensin II : ↑ Na reabsorption by stimulating Na-K ATP ase Pump and Na-H counter transporter.
  - Aldosteron stimulate Na^+ reabsorption in the late DCT and CD through ENaC
  - Atrial Natriuretic Peptide (ANP) inhibits Na reabsorption in the late DCT , CD through ENaC.

Note :

stimuli for rennin secretion :

- low Na delivery to DCT (macula densa)
- low pressure in the afferent arteriole (low ECF volume)
- sympathetic stimulation

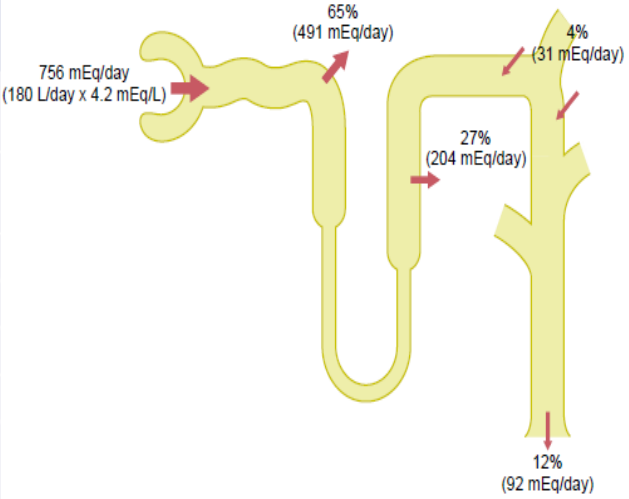
2- Regulation of K^+ ion:

i. Regulation of internal potassium distribution :

- Normal K^+ level (ECF) is 3.5-5 mEq/L
- < 3.5 meq/l is hypokalemia
- > 5 meq/l is hyperkalemia
- 98 % of the total body K is intracellular and only 2 % in the extracellular fluid

- Control of K ions distribution between the extracellular and intracellular compartments also plays an important role in potassium homeostasis

Factors That Shift K^+ Into Cells (Decrease Extracellular $[K^+]$)	Factors That Shift K^+ Out of Cells (Increase Extracellular $[K^+]$)
Insulin	Insulin deficiency (diabetes mellitus)
Aldosterone	Aldosterone deficiency (Addison's disease)
β-adrenergic stimulation	β-adrenergic blockade
Alkalosis	Acidosis
	Cell lysis
	Strenuous exercise
	Increased extracellular fluid osmolarity



ii. Renal Handling of K^+:

- Easily filtered and reabsorbed :
  - PCT (~ 65%) : paracellular passive reabsorption
  - Thick ascending limb:(25-30%) Na- K -2Cl Cotransporter
  - D.C.T & C.D are the most important sites for regulating potassium excretion by the principal cells and intercalated where K^+ can be reabsorbed or secreted according to the body needs.
- **Regulation of K^+ secretion**

1- Extracellular fluid potassium concentration: (Hyperkalemia) stimulates the Na-K ATPase pump →↑ Intracellular K ion concentration→ ↑tubular K secretion while hypokalemia →↓K secretion .

2- Aldosterone ↑ Na-k ATP ase pump activity and number of K^+ channels in the luminal membrane .

3- Tubular flow rate : ↑ flow rate of the tubular fluid through the distal portions of the nephron →↑ K^+secretion, because with rapid flow , tubular K^+ concentration cannot rise enough →↑ the driving force for potassium diffusion across the luminal membrane

4- ↑H ion concentration (acidosis) →↓K excretion : K is reabsorbed by H,K-ATPase in collecting cells in exchange for H^+ : ↑H^+ secretion →↓K^+ excretion .