Renal system

PHYSIOLOGY

Very important

Extra information

References :

- GUYTON AND HALL 12th edition
- LINDA 5th edition

* Guyton corners, anything that is colored with grey is EXTRA explanation

Renal Transport Process 2 : Tubular secretions

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.

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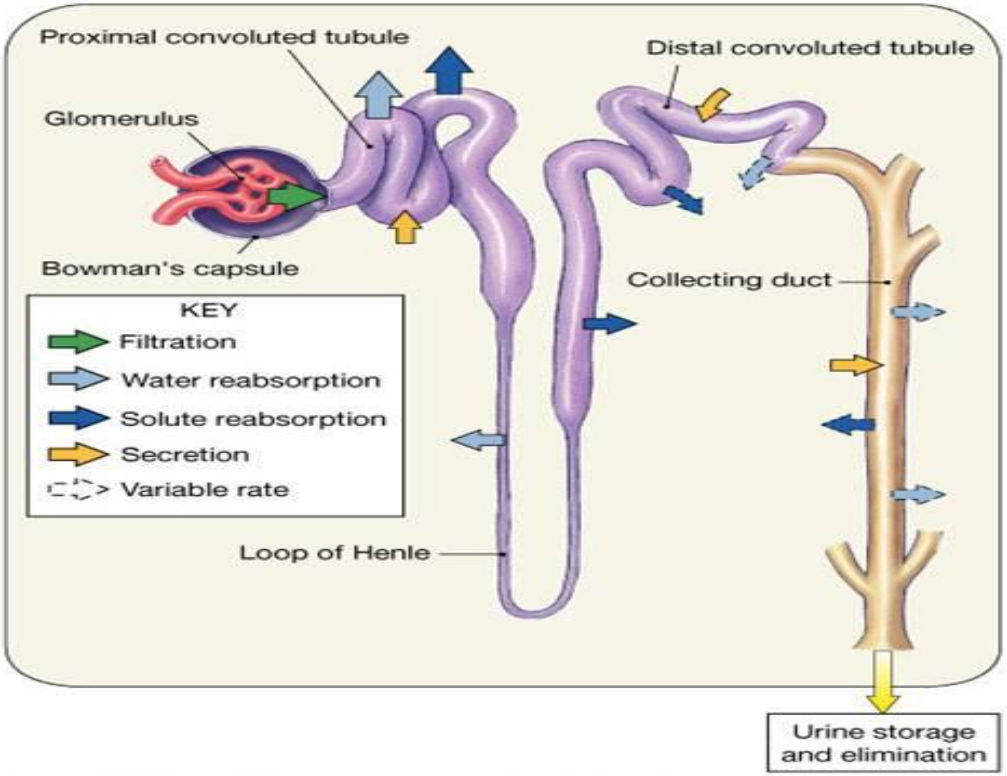
Overview

(reabsorption and secretion)

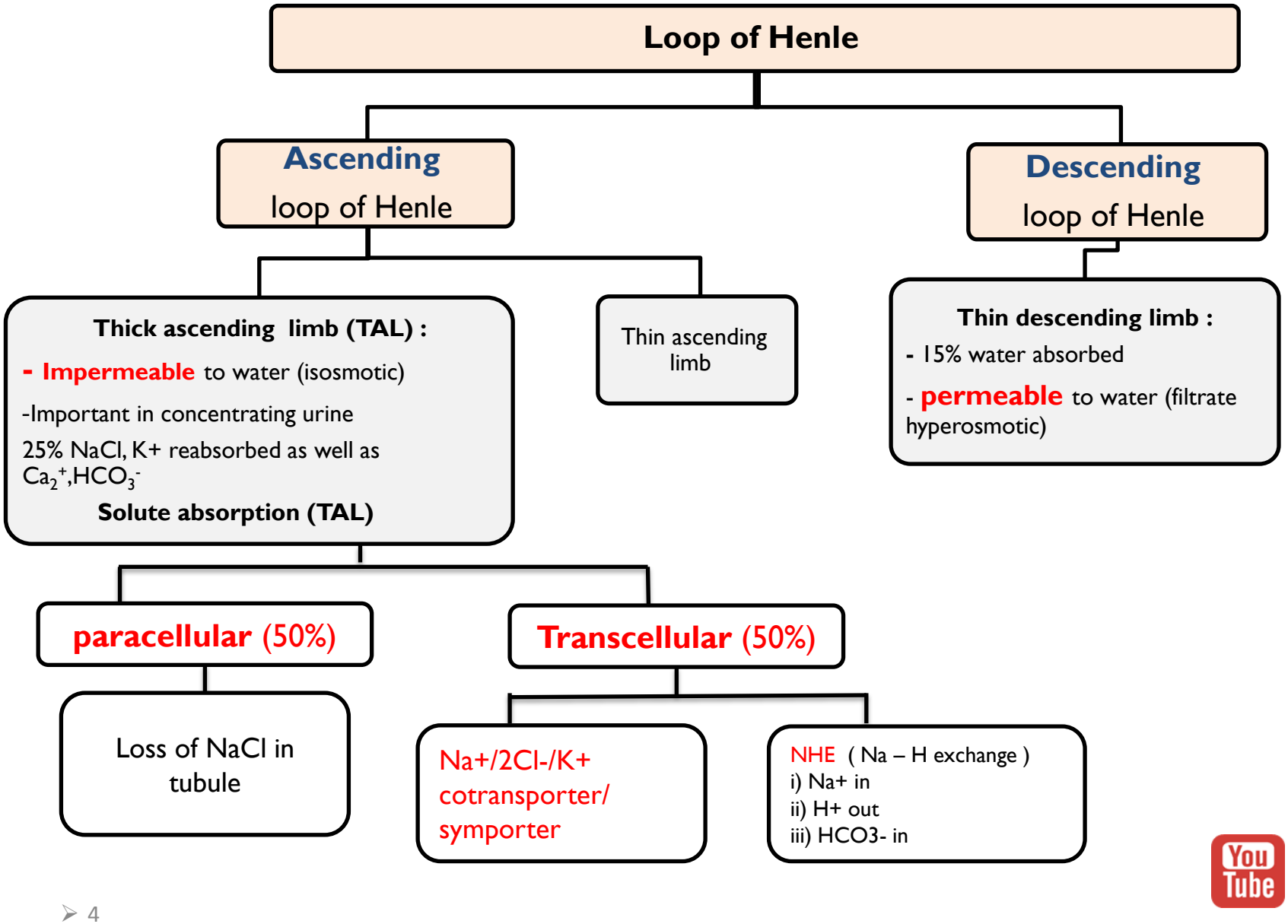
- **Guyton corner :**

The glomerular filtration and tubular reabsorption and tubular secretion are regulated according to the need of the body. page (312).
For many substances, tubular reabsorption plays a much more important role than secretion in determining the final urinary excretion rate. However, tubular secretion accounts for significant amounts of potassium ions, hydrogen ions, and a few other substances that appear in the urine page (323)



Reabsorption	Secretion
Paracellular transcellular	Only transcellular

Overview


Loop of Henle
Ascending loop of Henle
Descending loop of Henle
Thick ascending limb (TAL) :
- Impermeable to water (isosmotic)
-Important in concentrating urine
25% NaCl, K+ reabsorbed as well as Ca_2^+,HCO_3^-
Solute absorption (TAL)
Thin ascending limb
Thin descending limb :
- 15% water absorbed
- permeable to water (filtrate hyperosmotic)
paracellular (50%)
Transcellular (50%)
Loss of NaCl in tubule
Na+/2Cl-/K+ cotransporter/ symporter
NHE (Na – H exchange)
i) Na+ in
ii) H+ out
iii) HCO3- in

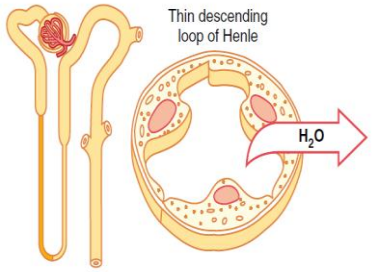
Loop of Henle

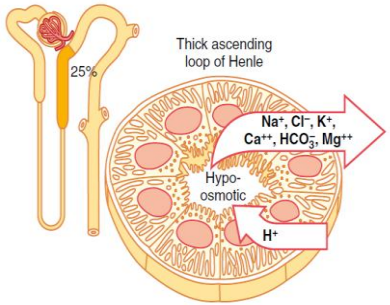
- **Thin descending limb :**
  - 15% water absorbed
  - **permeable** to water (filtrate hyperosmotic)
  to allow simple diffusion
- **Thick ascending limb :**
  - **Impermeable** to water *(isosmotic)*
  - Important in concentrating urine
  - 25% NaCl, K+ reabsorbed as well as Ca_2^+ and HCO_3^-
- **Solute absorption (TAL):**
  - **Transcellular** (50%)

1) Na+/2Cl-/K+ cotransporter/ symporter
2) NHE (Na, H exchange)

- Na+ in
- H+ out
- HCO3- in

  - **Paracellular** (50%)
  Loss of NaCl in tubule





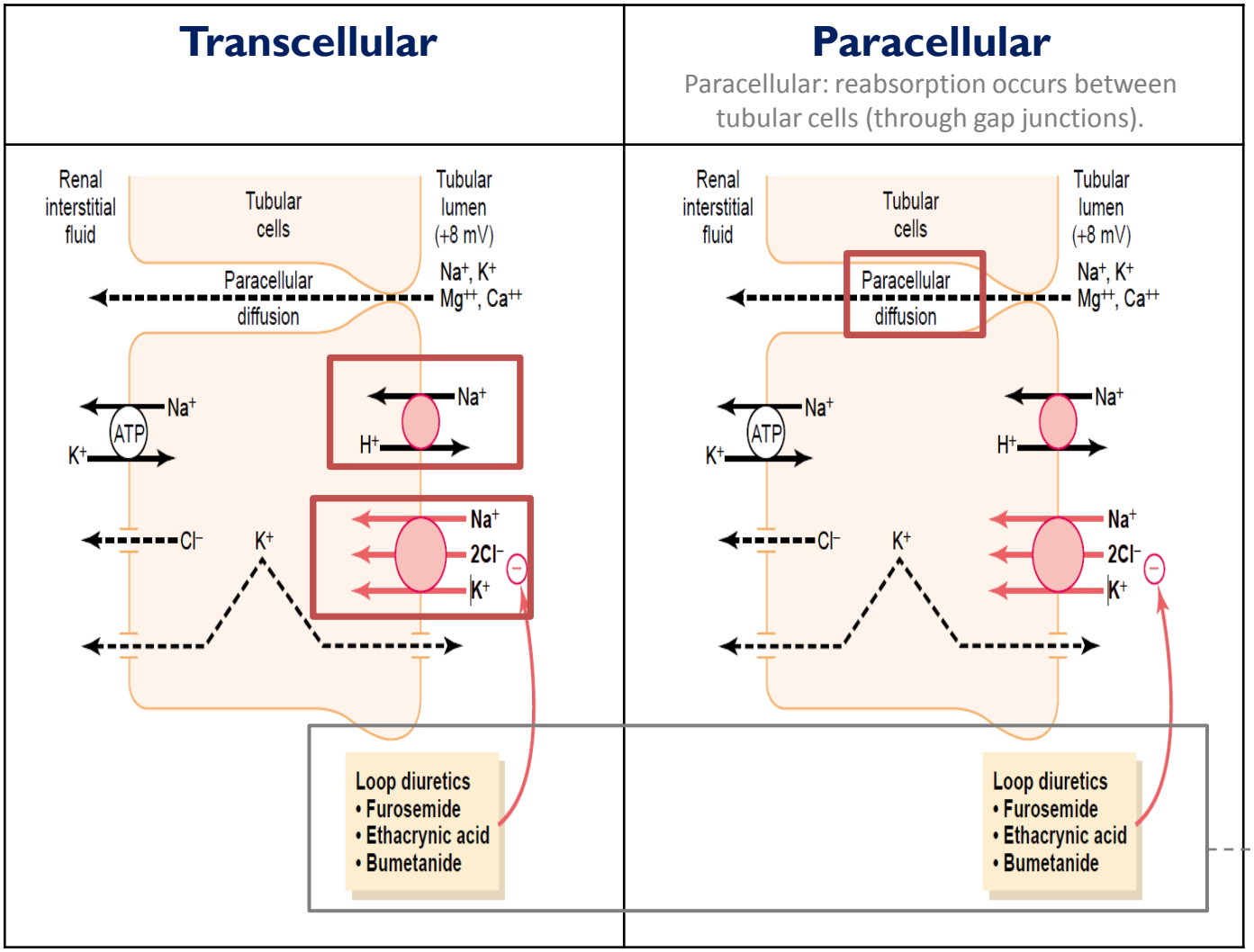
- The thick ascending limb reabsorbs 25% of NaCl and K, but the majority (70%) is reabsorbed in the proximal convoluted tubules. Transcellular is when ions enter the blood circulation after passing through tubular cells.
- Hydrogen secreted through the sodium hydrogen exchanger (NHE) is obtained from the dissociation of carbonic acid in the presence of carbonic anhydrase.

Loop of Henle

Transcellular

Paracellular

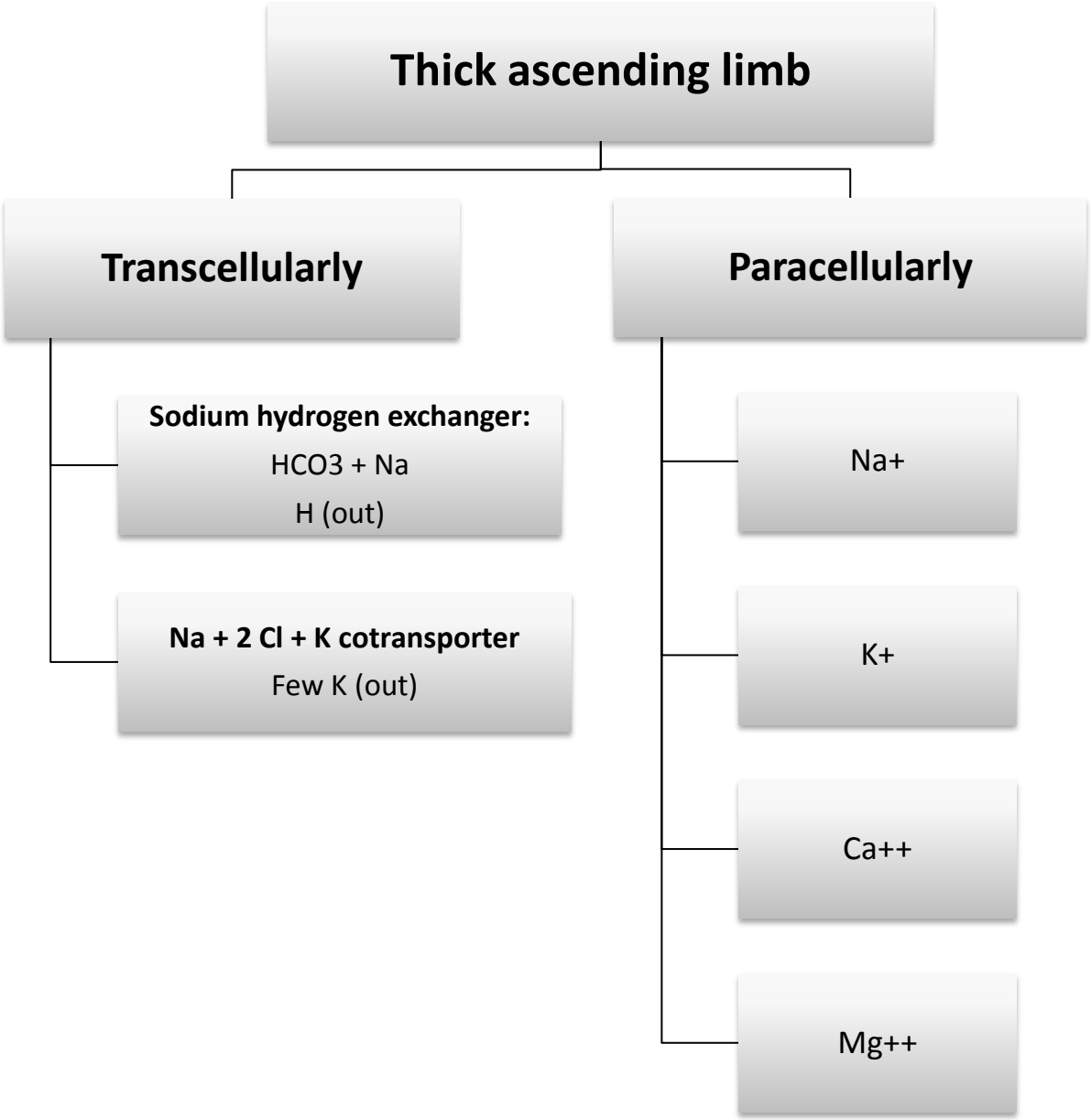
Paracellular: reabsorption occurs between tubular cells (through gap junctions).



Remember from pharmacology *loop diuretics mechanism *

The thick ascending limb of the loop of Henle is the site of action of the powerful *"loop" diuretics furosemide, ethacrynic acid,* and *bumetanide,* all of which inhibit the action of the sodium, 2-chloride, potassium co-transporter.

EXTRA


Thick ascending limb
Transcellularly
Paracellularly
Sodium hydrogen exchanger:
HCO3 + Na
H (out)
Na + 2 Cl + K cotransporter
Few K (out)
Na+
K+
Ca++
Mg++

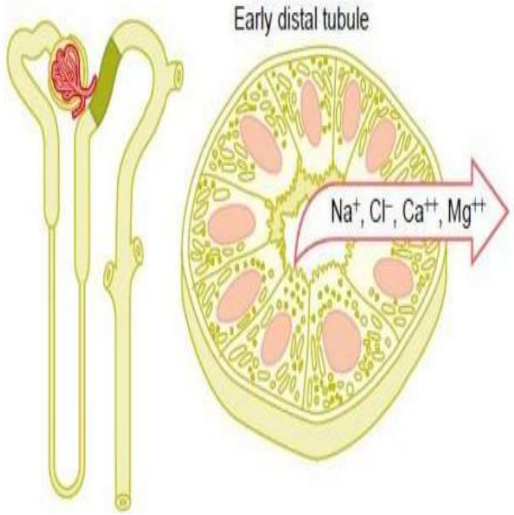
EXTRA

- **Guyton corner :** page (330-331)
- **Solute and Water Transport in the Loop of Henle:**

-The loop of Henle consists of **three functionally distinct segments**: the thin descending segment, the thin ascending segment, and the thick ascending segment.
-The thin segment of the ascending limb has a much **lower reabsorptive** capacity than the thick segment, and the thin descending limb does **not reabsorb** significant amounts of any of these solutes.
An important component of solute reabsorption in the thick ascending limb is **the sodium-potassium ATPase pump**, the reabsorption of other solutes in the thick segment of the ascending loop of Henle is closely linked to the reabsorptive capability of the sodium-potassium ATPase pump, which maintains a low intracellular sodium concentration. The low intracellular sodium concentration in turn provides a favorable gradient for movement of sodium from the tubular fluid into the cell.
In the thick ascending loop, movement of sodium across the luminal membrane is mediated primarily by **a 1-sodium, 2-chloride, 1-potassium co-transporter.**
The thick ascending limb also has a **sodium-hydrogen** counter-transport mechanism in its luminal cell membrane.
There is also significant paracellular reabsorption of cations, such as Mg++, Ca++, Na+, and K+, in the thick ascending limb owing to the slight positive charge of the tubular lumen relative to the interstitial fluid. Although the 1-sodium, 2-chloride, 1-potassium co-transporter moves equal amounts of cations and anions into the cell

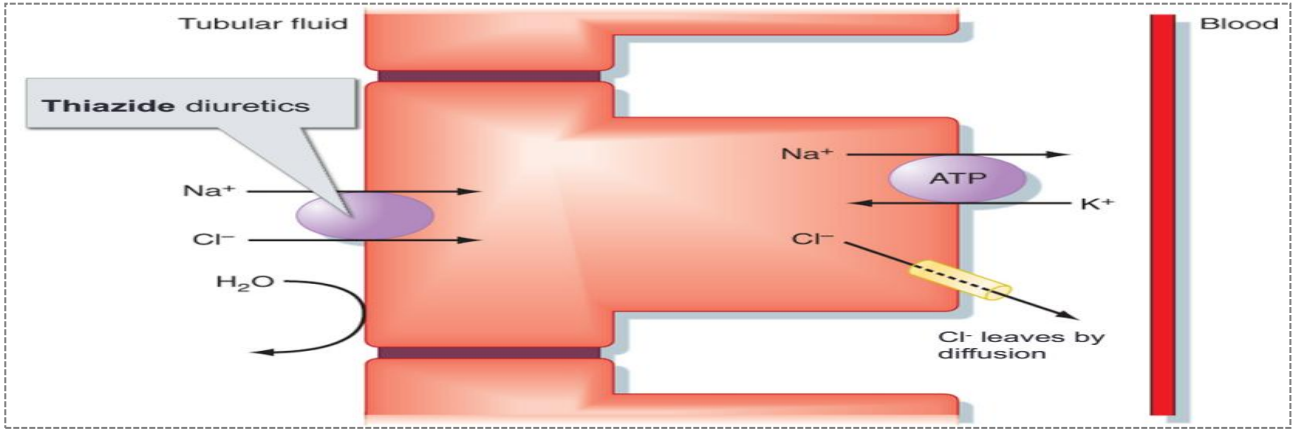
Distal convoluted tubule (DCT) & collecting duct (CD)

- 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)

- **Linda corner:**

- The distal tubule and collecting duct constitute the **terminal nephron,** and together they reabsorb about 8% of the filtered Na+. Like the thick ascending limb, reabsorption in the terminal nephron is **load-dependent,** with considerable capacity to reabsorb extra Na+ that may be delivered from the proximal tubule. The mechanism of Na+ transport in the early distal tubule differs from that of the late distal tubule and collecting duct.
- **The early distal tubule** reabsorbs **5%** of the filtered Na+. At the cellular level, the mechanism is an **Na+-Cl− cotransporter** in the luminal membrane, the energy for which derives from the Na+ gradient. There is net reabsorption of Na+ and Cl− in the early distal tubule, which is explained as follows: Both ions enter the cell on the Na+-Cl− cotransporter; Na+ then is extruded from the cell into the blood by the Na+-K+ ATPase, and Cl− diffuses out of the cell through Cl− channels in the basolateral membrane.
- The Na+-Cl− cotransporter of the early distal tubule differs from the Na+-K+-2Cl− cotransporter of the thick ascending limb in the following respects: It transports two ions (not three), it is electroneutral (not electro genic), and it is inhibited by a different class of diuretics, the **thiazide diuretics** (e.g.chlorothiazide, hydrochlorothiazide, metolazone). Like the loop diuretics, the thiazides are organic acids, which are anions at physiologic pH. Thiazide diuretics bind to the Cl− site of the Na+-Cl− cotransporter and prevent it from cycling, thus inhibiting NaCl reabsorption in the early distal tubule.
- The early distal tubule is **impermeable to water.** Thus, it reabsorbs solute but leaves water behind, which then dilutes the tubular fluid. For this reason, the early distal tubule is called the ***cortical*** **diluting segment** ("cortical" because distal tubules are in the kidney cortex). Recall that the tubular fluid entering the early distal tubule is *already* dilute (compared with blood) because of the function of the thick ascending limb; the early distal tubule *further* dilutes it.

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

Late DCT

Principle cells:

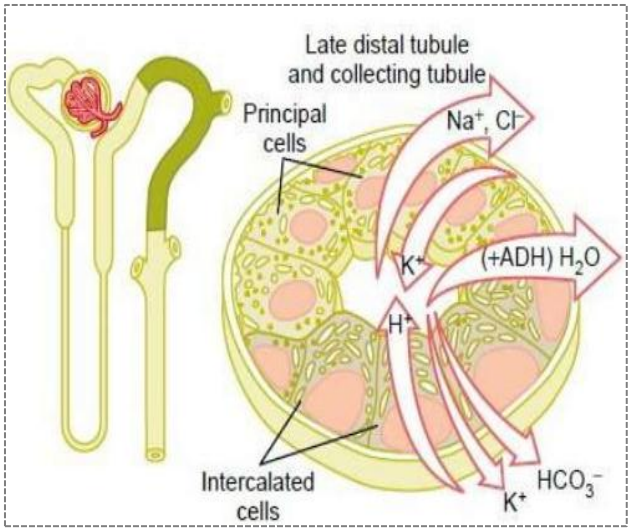
- Reabsorb Na^+, Na+ diffuses via selective channels.
- Reabsorb water
- Secrete K^+

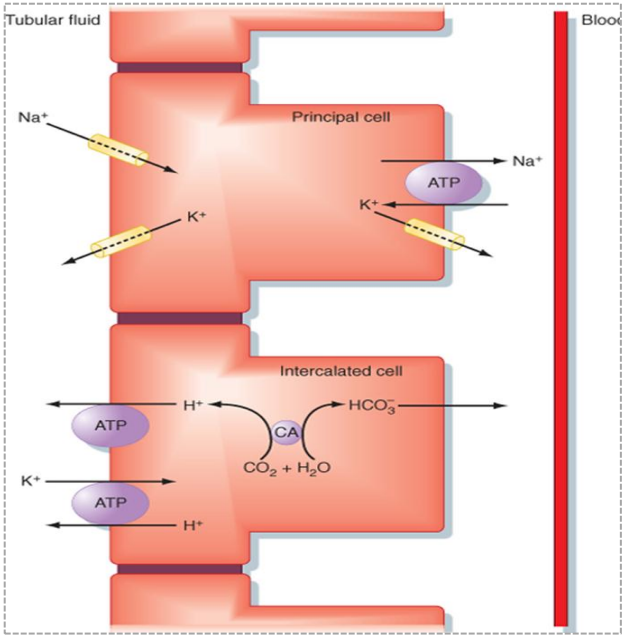
Intercalated cells:

- Secrete H+
- Reabsorb $HCO3^-$
- Reabsorb K^+

Aldosterone:

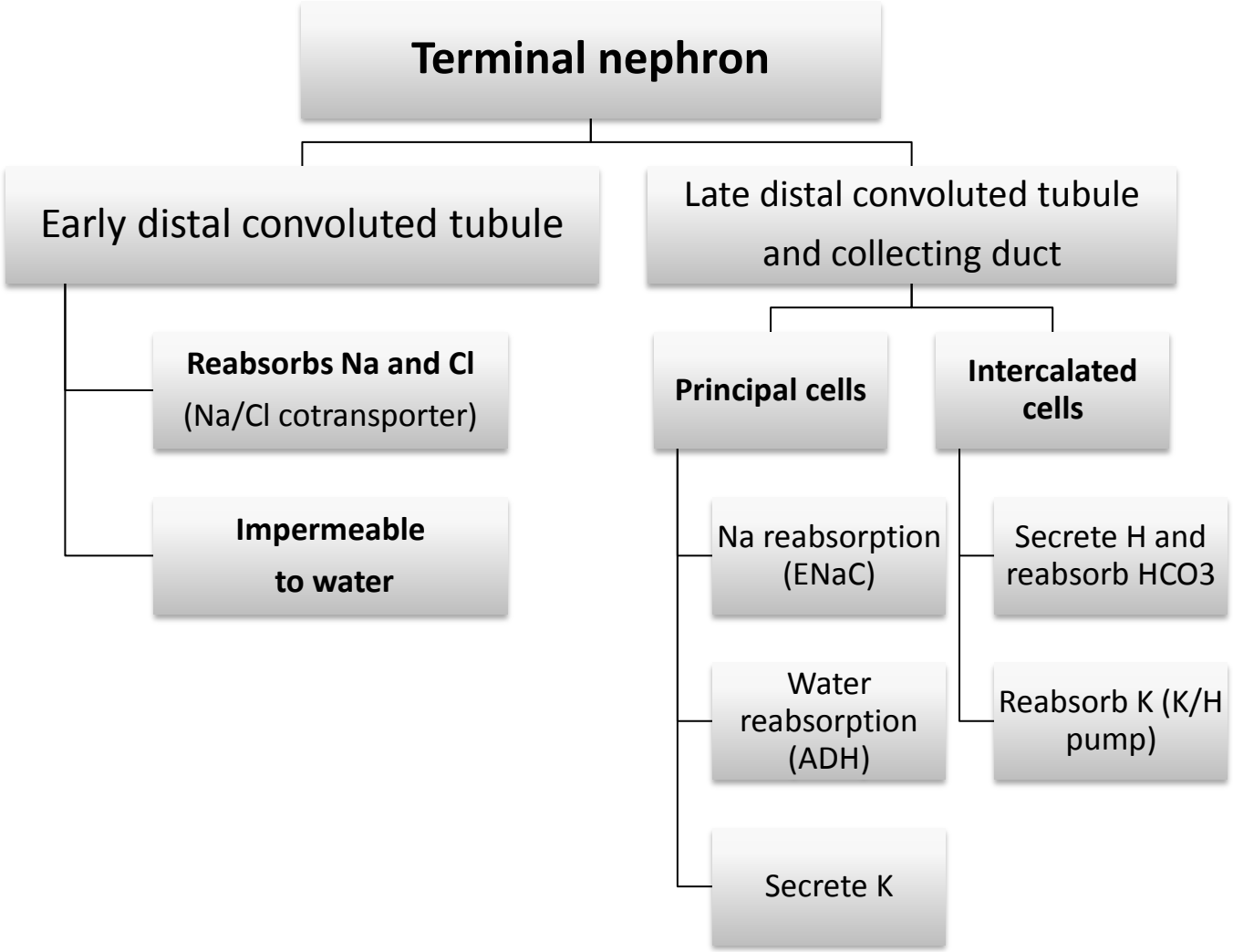
- ↑Na reabsorption by principle cells,
- ↑K+ secretion





- Principal and intercalated cells are both hormonally regulated.
- The intercalated cells reabsorb K by the K/H pump (active transport).

EXTRA


Terminal nephron
Early distal convoluted tubule
Late distal convoluted tubule and collecting duct
Reabsorbs Na and Cl (Na/Cl cotransporter)
Impermeable to water
Principal cells
Intercalated cells
Na reabsorption (ENaC)
Water reabsorption (ADH)
Secrete K
Secrete H and reabsorb HCO3
Reabsorb K (K/H pump)

EXTRA

- **Guyton corner :**

- The thick segment of the ascending limb of the loop of Henle empties into the distal tubule. The first portion of the distal tubule forms the macula densa. The next part of the distal tubule is highly convoluted and has many of the same reabsorptive characteristics of the thick segment of the ascending limb of the loop of Henle. That is, it avidly reabsorbs most of the ions, including sodium, potassium, and chloride, but is virtually impermeable to water and urea. For this reason, it is referred to as the *diluting segment* because it also dilutes the tubular fluid.

- Approximately 5 percent of the filtered load of sodium chloride is reabsorbed in the ***early distal tubule.*** The *sodium-chloride co-transporter* moves sodium chloride from the tubular lumen into the cell, and the sodium-potassium ATPase pump transports sodium out of the cell across the basolateral membrane. Chloride diffuses out of the cell into the renal interstitial fluid through chloride channels in the basolateral membrane.

- The *thiazide diuretics,* which are widely used to treat disorders such as hypertension and heart failure, inhibit the sodium-chloride co-transporter.

- The second half of the distal tubule and the subsequent cortical collecting tubule have similar functional characteristics. Anatomically, they are composed of two distinct cell types, the *principal cells* and *intercalated cells*. **The principal cells** reabsorb sodium and water from the lumen and secrete potassium ions into the lumen. **The intercalated cells** reabsorb potassium ions and secrete hydrogen ions into the tubular lumen.

- ***Principal Cells Reabsorb Sodium and Secrete Potassium***. Sodium *reabsorption* and potassium *secretion* by the principal cells *depend on the activity of a sodium-potassium ATPase pump* in each cell's basolateral membrane. This pump maintains a low sodium concentration inside the cell and, therefore, favors sodium diffusion into the cell through special channels.
- The secretion of potassium by these cells from the blood into the tubular lumen involves two steps: (1) Potassium enters the cell because of the sodium-potassium ATPase pump, which maintains a high intracellular potassium concentration, and then (2) once in the cell, potassium diffuses down its concentration gradient across the luminal membrane into the tubular fluid.

- ***Intercalated Cells Secrete Hydrogen and Reabsorb Bicarbonate and Potassium Ions***. Hydrogen ion secretion by the intercalated cells is mediated by a hydrogen- ATPase transporter. Hydrogen is generated in this cell by the action of carbonic anhydrase on water and carbon dioxide to form carbonic acid, which then dissociates into hydrogen ions and bicarbonate ions. The hydrogen ions are then secreted into the tubular lumen, and for each hydrogen ion secreted, a bicarbonate ion becomes available for reabsorption across the basolateral membrane.

EXTRA

- **Linda corner:**

- Anatomically and functionally, the late distal tubule and collecting duct are similar and can be discussed together. There are two major cell types interspersed along these segments: the **principal cells** and the **α-intercalated cells.** The principal cells are involved in Na+ reabsorption, K+ secretion, and water reabsorption; the α-intercalated cells are involved in K+ reabsorption and H+ secretion. The late distal tubule and collecting duct reabsorb only **3%** of the filtered Na+. Quantitatively, this amount is small when compared with the amounts reabsorbed in the proximal tubule, the thick ascending limb, and even the early distal tubule. The late distal tubule and collecting duct, however, are the last segments of the nephron to influence the amount of Na+ that is to be excreted (i.e., they make the fine adjustments of Na+ reabsorption).
- Rather than the coupled trans- port mechanisms seen in other nephron segments, the luminal membrane of the principal cells contains **Na+ channels** (epithelial Na+ channels, or **ENaC**). Na+ diffuses through these channels down its electrochemical gradient, from the lumen into the cell. Na+ then is extruded from the cell via the Na+-K+ ATPase in the basolateral membrane. The anion that accompanies Na+ is mainly Cl−, although the transport mechanism for Cl− has not been elucidated.
- Given the critical role of the late distal tubule and collecting duct in the fine adjustments to Na+ excretion, it should not be surprising that Na+ reabsorption in these segments is hormonally regulated. **Aldosterone** is a steroid hormone that acts directly on the principal cells to **increase Na+ reabsorption.** Aldosterone is secreted by the zona glomerulosa of the adrenal cortex, is delivered to the principal cells via the circulation, and diffuses into the cells across the basolateral cell membrane. In the cell, the hormone is transferred to the nucleus, where it directs the synthesis of specific messenger RNAs (mRNAs). These mRNAs then direct the synthesis of new proteins that are involved in Na+ reabsorption by the principal cells. The aldosterone- induced proteins include the luminal membrane **Na+ channel** itself, the Na+-K+ ATPase, and enzymes of the citric acid cycle (e.g., citrate synthase).
- Na+ reabsorption by the principal cells is inhibited by the **K+-sparing diuretics** (e.g., amiloride, triamterene, spironolactone). **Spironolactone,** a steroid and aldosterone-antagonist, prevents aldosterone from entering the nucleus of the principal cells and therefore blocks the synthesis of mRNAs and new proteins. **Amiloride** and **triamterene** bind to the luminal membrane Na+ channels and inhibit the aldosterone-induced increase in Na+ reabsorption. The K+-sparing diuretics produce only mild diuresis because they inhibit such a small percentage of the total Na+ reabsorption. However, as the name suggests, their main use is in combination with other diuretics to inhibit K+ secretion by the principal cells, as discussed in the section on K+ handling.
- **Water reabsorption** by the late distal tubule and collecting duct is variable, as described later in this chapter. Water permeability of the principal cells is controlled by **ADH,** which is secreted by the posterior lobe of the pituitary gland according to the body's need for water. When ADH levels are low or absent, the water permeability of the principal cells is low, and little, if any, water is reabsorbed along with NaCl. When ADH levels are high, aquaporin 2 (AQP2) channels are inserted in the luminal membranes of the principal cells, turning on their water permeability; thus, in the presence of ADH water is reabsorbed along with NaCl.

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.

- Potassium is important for the proper functioning of the heart.

EXTRA

- **Linda corner:**

- The maintenance of potassium (K+) balance is essential for the normal function of excitable tissues (e.g., nerve, skeletal muscle, cardiac muscle). the K+ concentration gradient across excitable cell membranes sets the resting membrane potential. Also, that changes in resting membrane potential alter excitability by opening or closing gates on the Na+ channels, which are responsible for the upstroke of the action potential. Changes in either intracellular or extracellular K+ concentration alter the resting membrane potential and, as a consequence, alter the excitability of these tissues.
- Most of the **total body K+** is located in the ICF: 98% of the total K+ content is in the intracellular compartment and 2% is in the extracellular compartment. A consequence of this distribution is that the intracellular K+ concentration (150 mEq/L) is much higher than the extracellular concentration (4.5 mEq/L). This large concentration gradient for K+ is maintained by the Na+- K+ ATPase that is present in all cell membranes.
- One challenge to maintaining the low extracellular K+ concentration is the large amount of K+ present in the intracellular compartment. A small shift of K+ into or out of the cells can produce a large change in the extracellular K+ concentration. The distribution of K+ across cell membranes is called **internal K+ balance.** Hormones, drugs, and various pathologic states alter this distribution and, as a consequence, can alter the extracellular K+ concentration.
- Another challenge to maintaining the low extracellular K+ concentration is the variation in dietary K+ intake in humans: Dietary K+ can vary from as low as 50 mEq/day to as high as 150 mEq/day. To maintain K+ balance, urinary excretion of K+ must be equal to K+ intake. Thus, on a daily basis, urinary excretion of K+ must be capable of varying from 50 to 150 mEq/day. The renal mechanisms that allow for this variability are called **external K+ balance.**
- Internal K+ balance is the distribution of K+ across cell membranes. To reemphasize, most K+ is present inside the cells and even small K+ shifts across cell membranes can cause large changes in K+ concentrations in ECF and blood. A shift of K+ out of cells produces an increase in the blood K+ concentration called **hyperkalemia.** A shift of K+ into cells produces a decrease in the blood K+ concentration called **hypokalemia.**

Transport of potassium

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)

In addition to internal distribution and renal K excretion, the balance between K intake and excretion plays a major role in regulating potassium levels.

Internal potassium distribution

Potassium content of average meal is 30-40 mmol.

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 ~ 2-5 mmol 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

- 2-5mmol increase in potassium plasma concentration is lethal **why?** Due to arrhythmias
- **Doctor Mona** mentioned that Na/K pumps differ; not all pumps allow 3 Na out and two K in. In fact, the Na/K pump we're talking about in this slide is a one-to-one pump.

- **Guyton corner :**

After ingestion of a normal meal, extracellular fluid potassium concentration would rise to a lethal level if the ingested potassium did not rapidly move into the cells. For example, absorption of 40 mEq of potassium (the amount contained in a meal rich in vegetables and fruit) into an extracellular fluid volume of 14 liters would raise plasma potassium concentration by about 2.9 mEq/L if all the potassium remained in the extracellular compartment. Fortunately, most of the ingested potassium rapidly moves into the cells until the kidneys can eliminate the excess

EXTRA

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

factors that can influence the distribution of potassium between the intracellular and extracellular compartments

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*.

- (The second point) This explains why diarrhea causes hypokalemia and eventually, alkalosis.
- Potassium reabsorption in the loop of Henle takes place in the thick ascending limb.

Renal K+ Transport mechanisms

Cell membrane transporters :

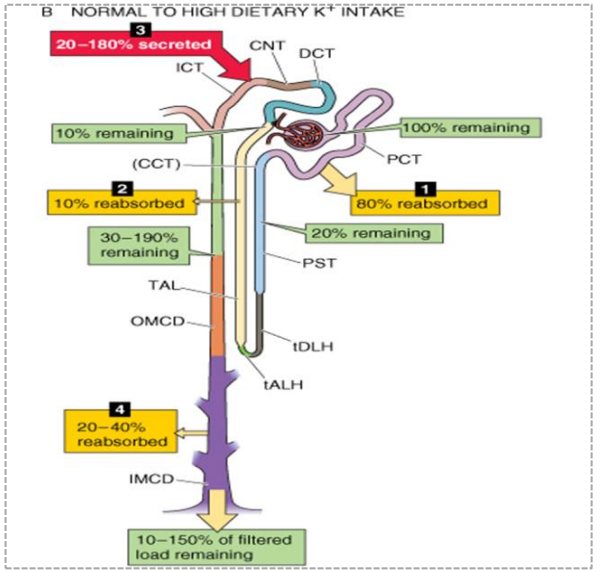
- Na-K ATPase, H-K ATPase
- K+ channels, K:Cl cotransport
- Na:K:2Cl cotransport

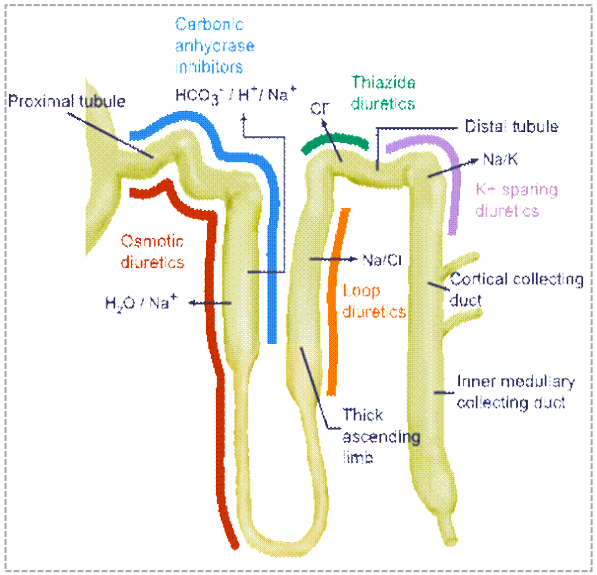
K+ is Reabsorbed in :

- PT (Proximal tubules)
- TAL (Ascending limb of loop of Henle)
- Intercalated cell in CCD (cortical collecting duct)

K+ Secreted in :

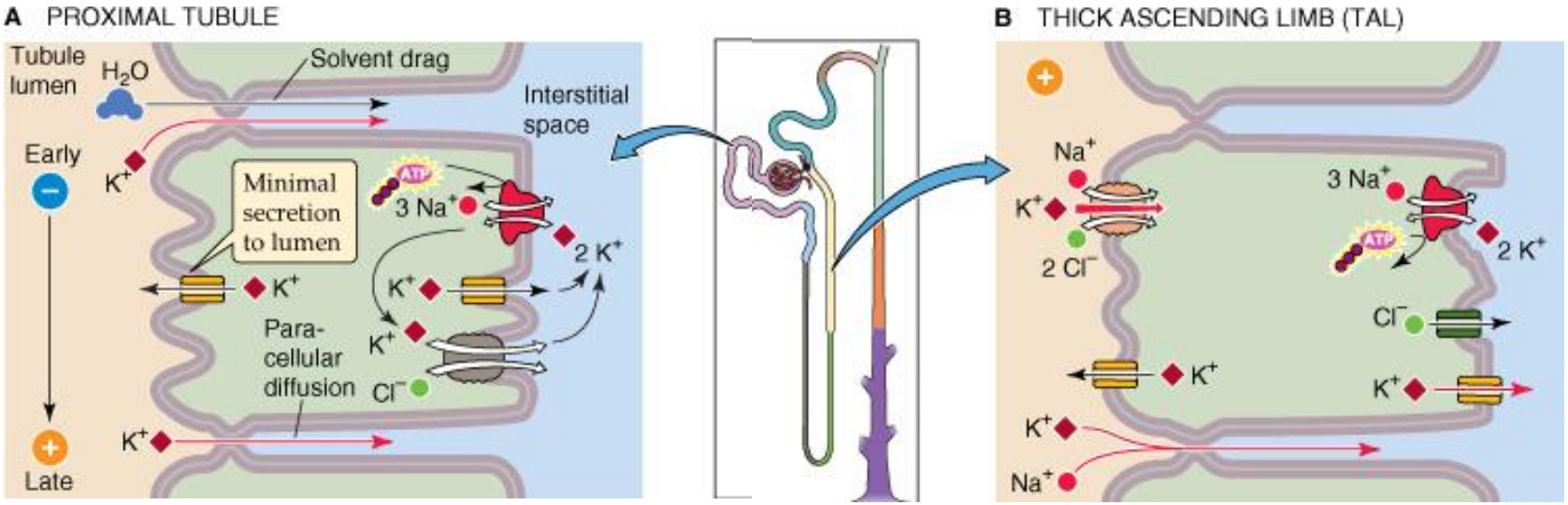
- Late distal tubule
- principal cells of late DT (Distal Tubules)
- CCD (cortical collecting duct)



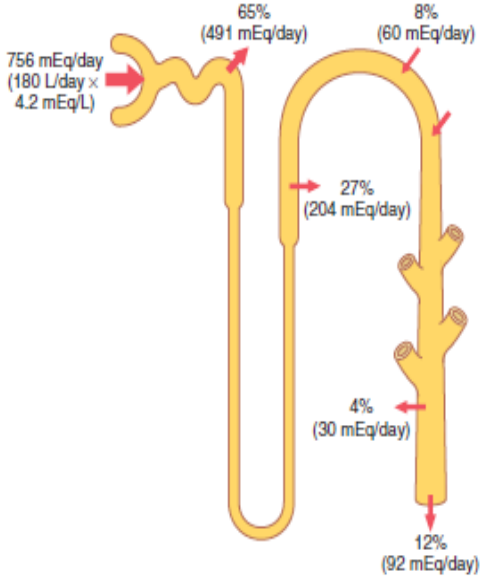


Renal K+ Transport mechanisms

- **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*



- **Guyton corner :** the tubular handling of potassium under normal conditions. About 65 percent of the filtered potassium is reabsorbed in the proximal tubule. Another 25 to 30 percent of the filtered potassium is reabsorbed in the loop of Henle, especially in the thick ascending part where potassium is actively co-transported along with sodium and chloride. In both the proximal tubule and the loop of Henle, a relatively constant fraction of the filtered potassium load is reabsorbed. Changes in potassium reabsorption in these segments can influence potassium excretion, but most of the day-to-day variation of potassium excretion is not due to changes in reabsorption in the proximal tubule or loop of Henle. There is also some potassium reabsorption in the collecting tubules and collecting ducts; the amount reabsorbed in these parts of the nephron varies depending on the potassium intake.



- **Guyton corner :** the basic cellular mechanisms of potassium secretion by the principal cells. Secretion of potassium from the blood into the tubular lumen is a two-step process, beginning with uptake from the interstitium into the cell by the sodium-potassium ATPase pump in the basolateral cell membrane; this pump moves sodium out of the cell into the interstitium and at the same time moves potassium to the interior of the cell. The second step of the process is passive diffusion of potassium from the interior of the cell into the tubular fluid. The sodium-potassium ATPase pump creates a high intracellular potassium concentration, which provides the driving force for passive diffusion of potassium from the cell into the tubular lumen. The luminal membrane of the principal cells is highly permeable to potassium because there are two types of special channels that allow potassium ions to rapidly diffuse across the membrane: (1) the *renal outer medullary potassium (ROMK) channels,* and (2) high conductance *"big" potassium (BK) channels.* Both types of potassium channels are required for efficient renal potassium excretion, and their abundance in the luminal membrane is increased during high potassium intake.

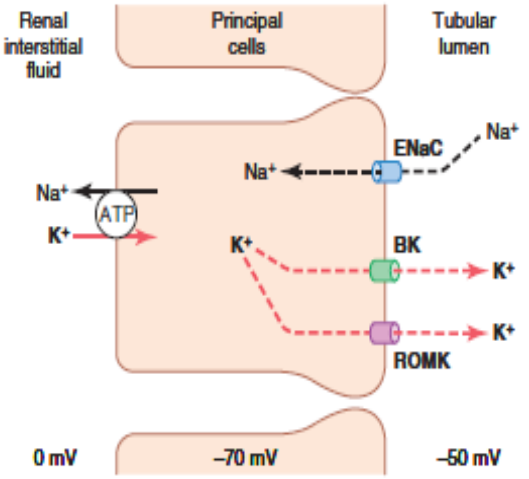
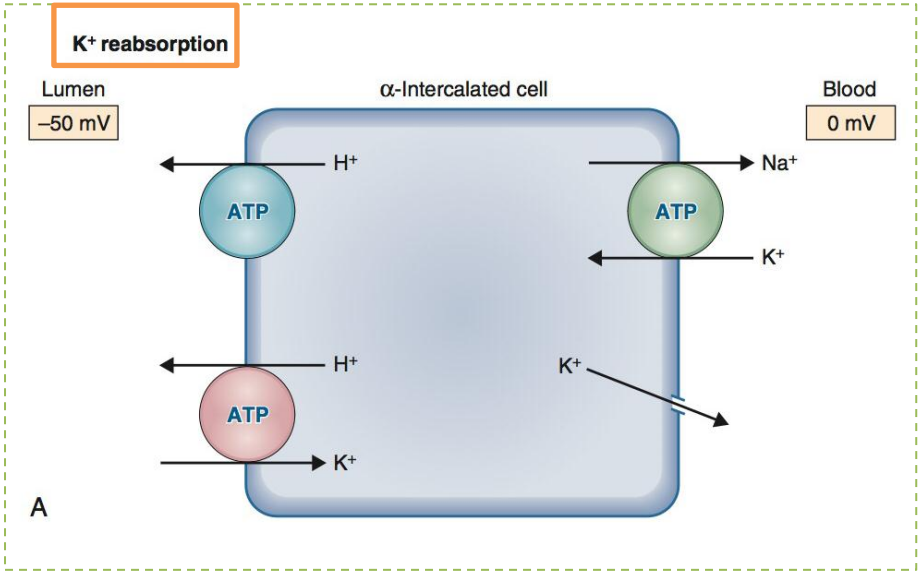


Figure 30-3. Mechanisms of potassium secretion and sodium reabsorption by the principal cells of the late distal and collecting tubules. BK, "big" potassium channel; ENaC, epithelial sodium channel; ROMK, renal outer medullary potassium channel.

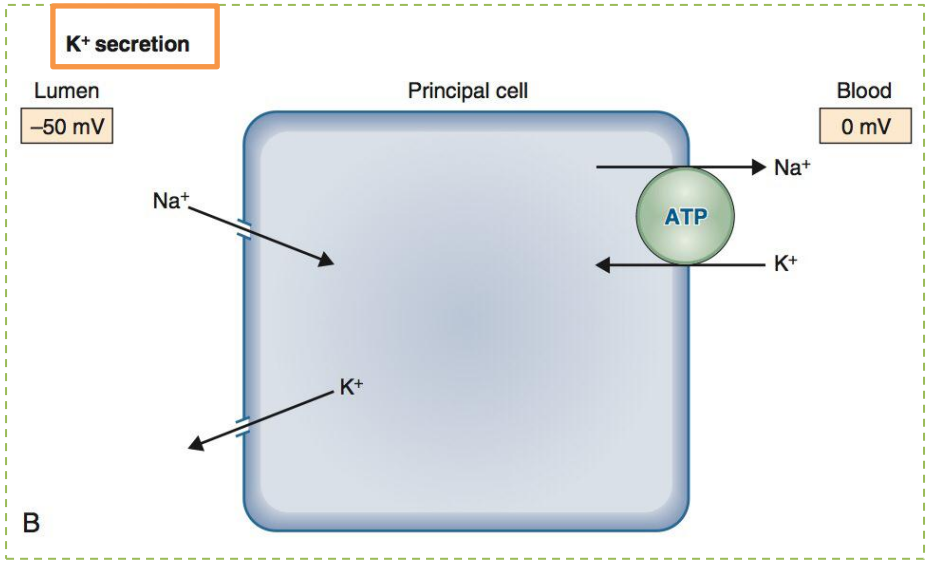
Renal K+ Transport mechanisms

(Late distal tubule and collecting duct)



When a person is on a low K+ diet

K+ reabsorption is by the intercalated cells via a luminal H-K ATPase (primary active transport)



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

The factors that alter K secretion is that the magnitude of K secretion is determined by the size

Factors affecting potassium secretion *(peritubular factors + luminal factors)*

Peritubular factors *(change inside tubular cells.)*

(All Increase the secretion and excretion of potassium)

Hyper-kalemia	Hyper-aldosteronism	Alkalosis
Increase K in tubular cell and increase chemical grdient of K between tubular cell and tubular lumen which lead to increase in the secretion and excretion of K.	Increase aldosterone increase in the secretion and excretion of K.	Increase H-K exchange at basolateral membrane then increase in the secretion and excretion of K.
	First, aldosterone induces the synthesis of more luminal membrane Na+ channels, which increases Na+ entry into the cell and provides more Na+ to the Na+-K+ ATPase. As more Na+ is pumped out of the cell, more K+ must be simultaneously pumped into the cell. Second, aldosterone increases the quantity of Na+-K+ ATPase, further increasing the amount of K+ pumped into the cell. Together, the two effects raise the intra- cellular K+ concentration, which increases the driving force for K+ secretion from the cell into the lumen. Finally, as a separate effect, aldosterone increases the number of K+ channels in the luminal membrane, which coordinates with the increased driving force to increase K+ secretion	In alkalosis, there is a deficit of H+ in the ECF. H+ leaves the cells to aid in buffering, and K+ enters the cells to maintain electroneutrality. The increased intracellular K+ concentration increases the driving force for K+ secretion, causing hypokalemia

Luminal factors

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

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

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.

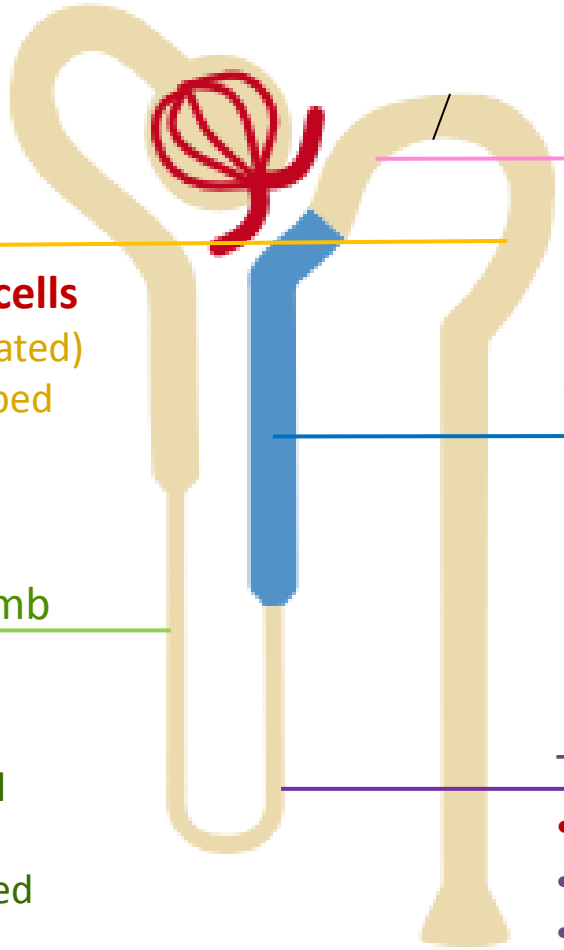
NaCl Transport along the Nephron

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 ducts	≈ 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 20 and collecting duct	≈ 8% - 17%	Passive	ADH, ANP, BNP*

Based on pages (276-279) of Linda S. Costanzo physiology (5th edition)



Late Distal Convoluted Tubule & Collecting Duct

- **Principal and intercalated cells**
- Permeable to H2O (ADH-regulated)
- Na, H2O, HCO3, K are reabsorbed
- K and H are secreted

Thin Descending Limb

- **Hyperosmotic**
- Permeable to H_20
- Permeable to NaCl and urea
- Solutes are secreted

Early Distal Convoluted Tubule

- **Cortical diluting segment**
- Impermeable to H_2O
- Na/Cl reabsorption

Thick Ascending Limb

- **Isosmotic**
- **Load dependent**
- Na, Cl, K, HCO3, Ca, Mg are reabsorbed
- H and K are secreted

Thin Ascending Limb

- **Hyposmotic**
- impermeable to H_20
- Permeable to NaCl

physiology team

Physiology Leaders :

Khawla Alammari
Meshal Alhazmy

Girls team :

- Alhanouf Aljlaoud
- Afnan Almalki
- Asrar Batarfi
- Amal alomran
- Alanoud AlOmair
- Deema AlFaris
- Elham Alzahrani
- Hissah almuzini
- Johara Almalki
- Jwaher alHarbi
- Lojain alsiwat
- Munerah AlOmari
- Munira Alhussaini
- Malak alshareef
- Mai Alageel
- Nojood alhaidri
- Nora Alsomali
- Noura Alkharraz
- Noura alTawil
- Norah Alakeel
- Reem AlAgeel
- Renad alkhtani
- Raghad alnafisah
- Ruba Alselaimy
- Reema Allhuidan
- Razan Alsabti
- Wadha Alotaibi
- Nurah Alqahtani

Boys team :

- Abdullah Aljaafar
- Omar Alotaibi
- Abdulrahman Albarakah
- Adel Alshehri
- Abdulaziz Alghanaym
- Abdulmajeed Alotaibi
- Khalil Alduraibi
- Hassan Albeladi
- Omar Alshehri
- Saleh Alshawi
- Abdulaziz Alhammad
- Faisal Alabdulatif
- Abdulnasser Alwabel
- Saad Almutairy