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

Objectives :

- Define tubular reabsorption, tubular secretion, transcellular and paracellular transport.
- Identify and describe mechanisms of tubular transport
- Describe tubular reabsorption of sodium and water
- Revise tubulo-glomerular feedback and describe its physiological importance
- Identify and describe mechanism involved in Glucose reabsorption
- Study glucose titration curve in terms of renal threshold, tubular transport maximum, splay, excretion and filtration
- Identify the tubular site and describe how Amino Acids, HCO3-, P04- and Urea are reabsorbed

Contact us : Physiology435@gmail.com

Renal Threshold

When the plasma concentration of the substance is **beyond** "More than" the renal threshold

At this level

the filtered load exceeds the absorptive capacity of the tubules.

the substance begins to appear in urine

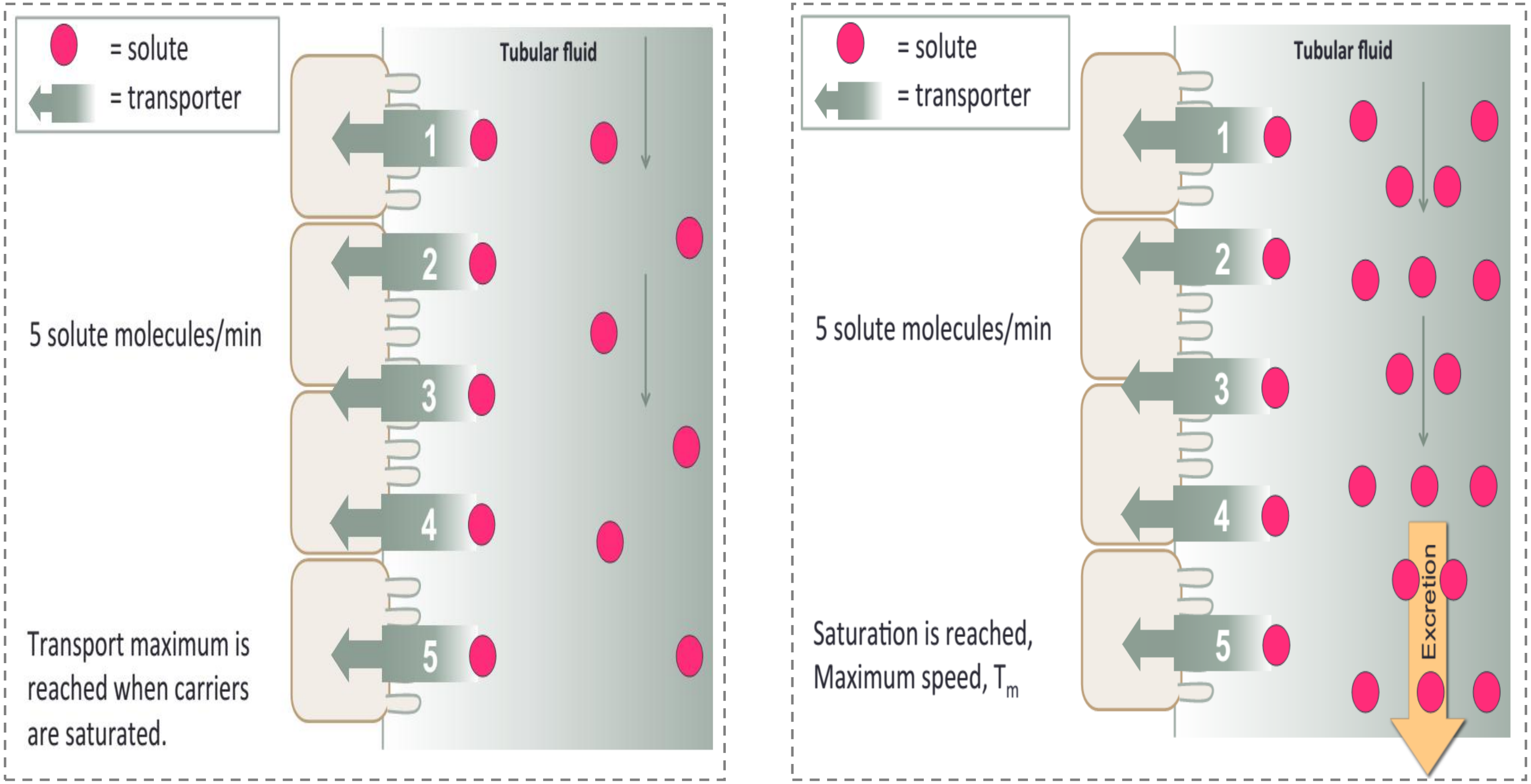
Renal threshold			
HIGH threshold	medium threshold	LOW threshold	NO threshold
- Glucose - Amino acids - vitamins. "Completely Reabsorbed"	- K+ - Urea "Some of the substance will be reabsorbed and the remaining will be excreted in the urine "	- Phosphate - Uric acid "Only small amount will be reabsorbed but the majority of substance will be excreted"	- Creatinine - mannitol - inulin. "Completely excreted"

Tubular transport maximum

- It is the *maximal* amount of a substance (in mg) which can be transported (reabsorbed or secreted) by tubular cells/min.
- Many substances are reabsorbed by carrier mediated transport systems : *glucose, amino acids, organic acids, sulphate, phosphate ions*

Carriers have a maximum transport capacity (T_m) which is due to saturation of the carriers. If (T_m) is exceeded, then the excess substrate enters the urine.

Tubular transport maximum



some substances need carrier proteins to be reabsorbed, when the carriers are fully saturated, In this level we can say that tubular transport reached maximum. The rest will be excreted.

Tubular transport maximum of Glucose

- Glucose is ***Freely filtered*** , so whatever its [plasma] that will be filtered.

- In man for plasma glucose up to 180 mg/dl all will be REABSORPED. Beyond this level of plasma [glucose], it appears in the urine = Renal plasma threshold for glucose.

IF plasma [glucose] = **275** mg/dl

- **275** mg/dl will be **Filtered**
- **180** mg/dl **Reabsorbed**
- **90** mg/dl **Excreted**

- **Linda corner:**

Glucose is filtered across glomerular capillaries and reabsorbed by the epithelial cells of the proximal con- voluted tubule. Glucose reabsorption is a two-step process involving Na+-glucose cotransport across the luminal membrane and facilitated glucose transport across the peritubular membrane. Because there are a limited number of glucose transporters, the mechanism is saturable; that is, it has a transport maximum, or Tm.

Tubular transport maximum of Glucose

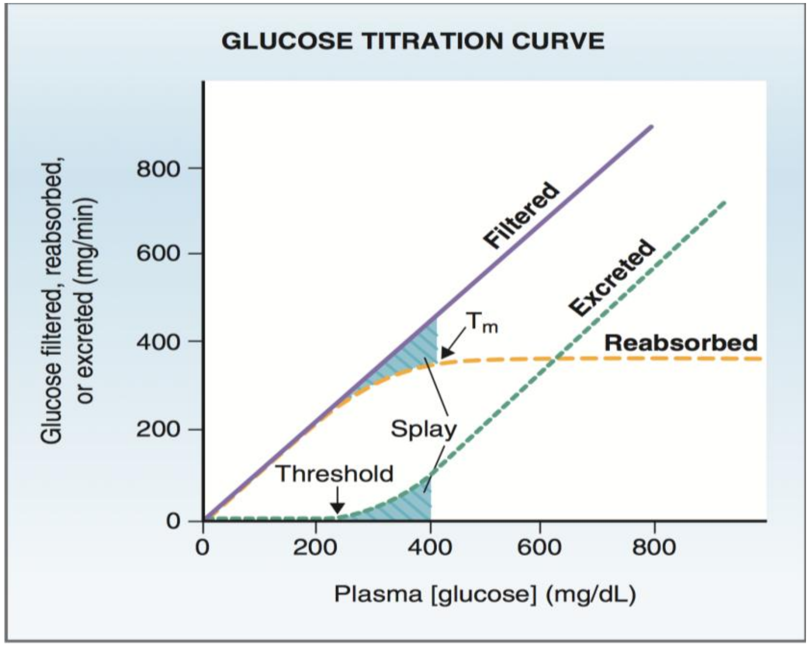
- **Kidney** does NOT regulate glucose *(insulin and glucagon).*
  Normal glucose of 90 mg/dl , So $\mathbf{T_m}$ is set way above any possible level of (non-diabetic) glucose at 360 mg/dl. Thus ensure that all this valuable nutrient is normally reabsorbed. "Glucose is hormonally regulated by pancreas"

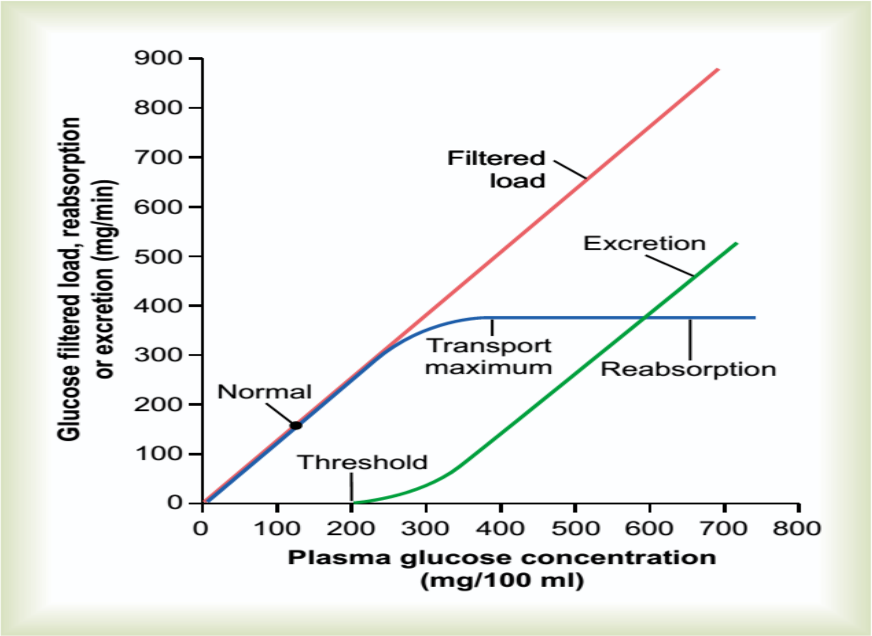
- The appearance of glucose in the urine of diabetic patients
  = glycosuria which is due to **failure of insulin** NOT the kidney.

- **Linda corner:**

Glucosuria :At normal plasma glucose concentrations (70 to 100 mg/dL), all of the filtered glucose is reabsorbed and none is excreted. Under some circumstances, however, glucosuria (excretion or spilling of glucose in the urine) occurs. The causes of glucosuria can be understood by referring again to the glucose titration curve. (1) In uncontrolled diabetes mellitus, lack of insulin causes the plasma concentration of glucose to increase to abnormally high levels. In this condition, the filtered load of glucose exceeds the reabsorptive capacity (i.e., plasma glucose concentration is above the Tm), and glucose is excreted in the urine. (2) During pregnancy, GFR is increased, which increases the fil- tered load of glucose to the extent that it may exceed the reabsorptive capacity. (3) Several congenital ab- normalities of the Na+-glucose cotransporter are associated with decreases in Tm, causing glucose to be excreted in the urine at lower than normal plasma concentrations

EXTRA





- ## Linda corner:

At plasma glucose concentrations less than 200 mg/dL, all of the filtered glucose can be reabsorbed because Na+-glucose cotransporters are plentiful. In this range, the curve for reabsorption is identical to that for filtration; that is, reabsorption equals filtration. The number of carriers is limited, however. At plasma concentrations above 200 mg/dL, the reabsorption curve bends because some of the filtered glucose is not reabsorbed. At plasma concentrations above 350 mg/dL, the carriers are completely saturated and reabsorption levels off at its maximal value, Tm.

- ## Linda corner:

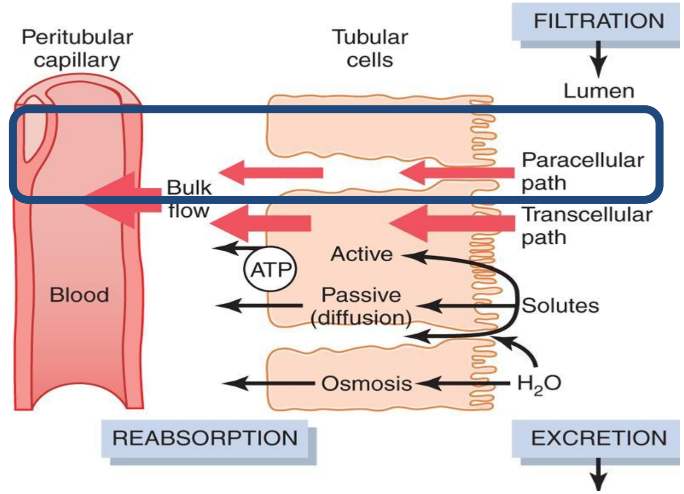
A **glucose titration curve** depicts the relationship between plasma glucose concentration and glucose reabsorption. For comparison, the filtered load of glucose and the excretion rate of glucose are plotted on the same graph. The glucose titration curve is obtained experimentally by infusing glucose and measuring its rate of reabsorption as the plasma con- centration is increased. The titration curve is best understood by examining each relationship separately and then by considering all three relationships together.

Tubular transport maximum of Glucose

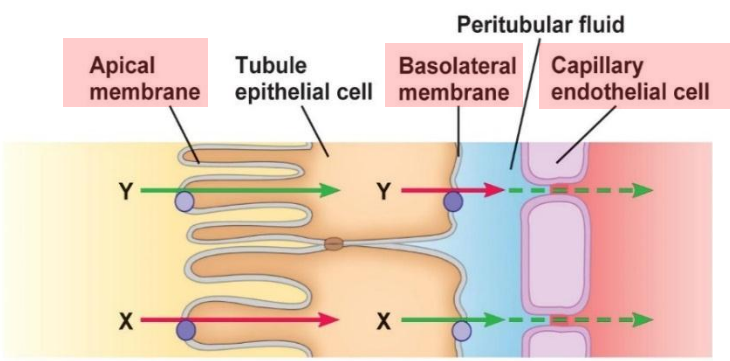
- For Amino acids, $\mathbf{T_m}$ also very high → no urinary excretion occurs.
  "Reabsorption will occur"
- Kidney does regulate some substances by means of the $\mathbf{T_m}$ mechanism e.*g* sulphate and phosphate ions.
- This is because $\mathbf{T_m}$ is set at a level whereby the normal plasma causes saturation, so any ↑ above the normal level will be *excreted*, therefore achieving its plasma regulation.
- Also subject to PTH regulation for phosphate, [PTH ↓ reabsorption].
  *PTH = parathyroid hormone

Tubular Reabsorption

- **Transported substances move through three membranes :**
  - **Luminal** "Apical" membranes of tubule cells.
  - **Basolateral** membranes of tubule cells
  - **Endothelium** of peritubular capillaries



- **Ca^{2+}, Mg^{2+}, K+**, and some **Na+** can be reabsorbed via *paracellular pathway*. "بين الفراغات"



(a) Active solute reabsorption

- paracellular means that the pathway is between cells.
- Transcellular means through cell.
- Paracellular:
- Passive diffusion – Down gradient– Cross tight junction.
- Transcellular:
  Active Transportation of solutes Cross a cell.

Tubular Reabsorption

Tubular reabsorption

- All organic nutrients are reabsorbed
- Water and ion reabsorption is hormonally controlled
- Reabsorption may be an active (requiring ATP) or passive process

Examples :

- **ADH** Reabsorb water (retention of water).
- **Aldosterone** tends to promote Na^+ and water retention, and lower plasma K^+ concentration.

Filtration, Excretion, and Reabsorption of Water, Electrolyte, and Solute by the Kidneys

Substance	Measure	Filtered*	Excreted	Reabsorbed	% Filtered Load Reabsorbed
Water	L/day	180	1.5	178.5	99.2
Na^+	mEq/day	25,200	150	25,050	99.4
K^+	mEq/day	720	100	620	86.1
Ca^{++}	mEq/day	540	10	530	98.2
HCO_3^-	mEq/day	4320	2	4318	99.9+
Cl^-	mEq/day	18,000	150	17,850	99.2
Glucose	mmol/day	800	0	800	100.0
Urea	g/day	56	28	28	50.0

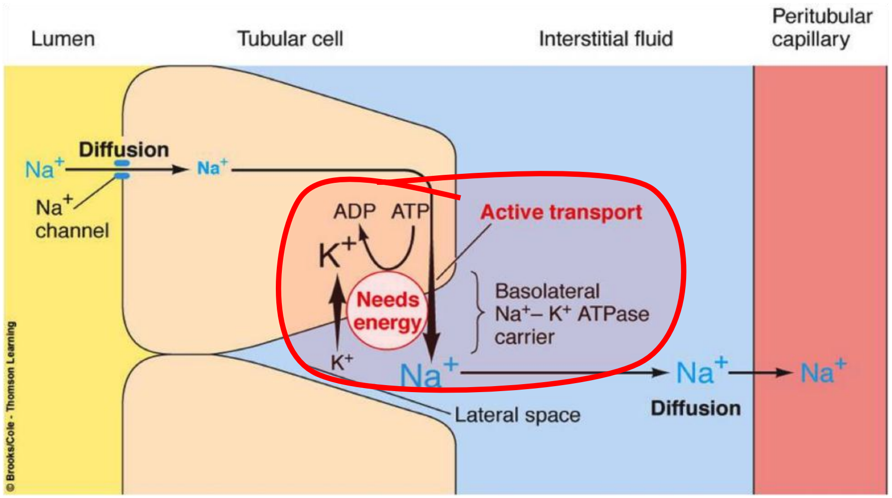
the table shows the ability of tubules to reabsorb and if the substrate has high or low threshold.

$$\left\{ \frac{\text{Reabsorbed}}{\text{Filtered}} \times 100 \right\}$$

Sodium Reabsorption: Primary Active Transport

- **Sodium reabsorption** is almost always by <u>active</u> transport "Requires ATP"
- **Na^+** enters the tubule cells at the luminal membrane.
- Is actively transported out of the tubules by a Na^+-K^+ ATPase pump

k+ enter <u>inside</u> the cell
Na+ come <u>outside</u> of the cell



Sodium Reabsorption: Primary Active Transport

25,000 mEq/day Na+
179 L/day water
Reabsorped daily by renal tubules

One of the main functions of nephron

Other important solutes are linked either directly or indirectly to reabsorption of Na^+.

أي أثناء عملية إعادة امتصاص الصوديوم
ترتبط معه جزيئات أخرى مثل الجلوكوز

Water

And Solute handling

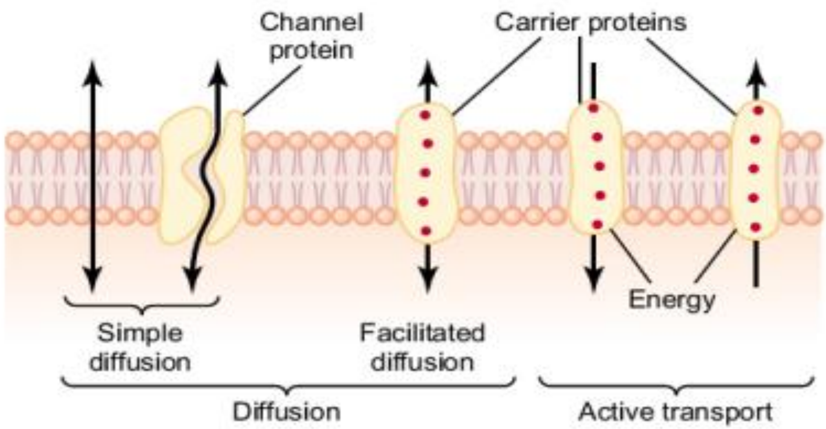
Mechanisms of Tubular Absorption & secretion

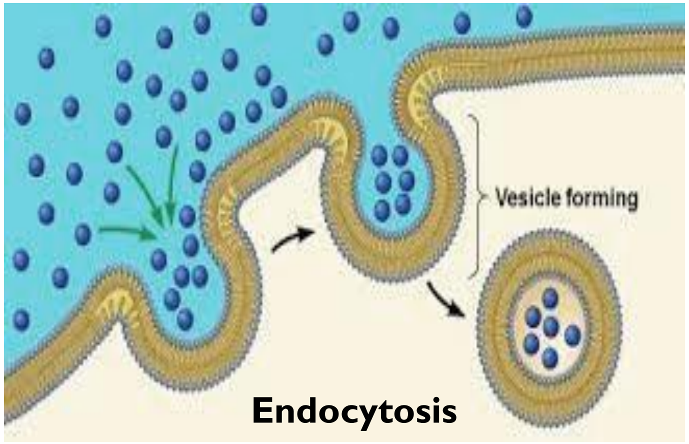
Down chemical/electrical gradient

- Passive diffusion
- Facilitated diffusion

Against chemical/electrical gradient (needs energy)

- Active transport
- Endocytosis





Endocytosis is a form of active transport in which a cell transports molecules (such as proteins) into the cell (endo- + cytosis) by engulfing them in an energy-using process.

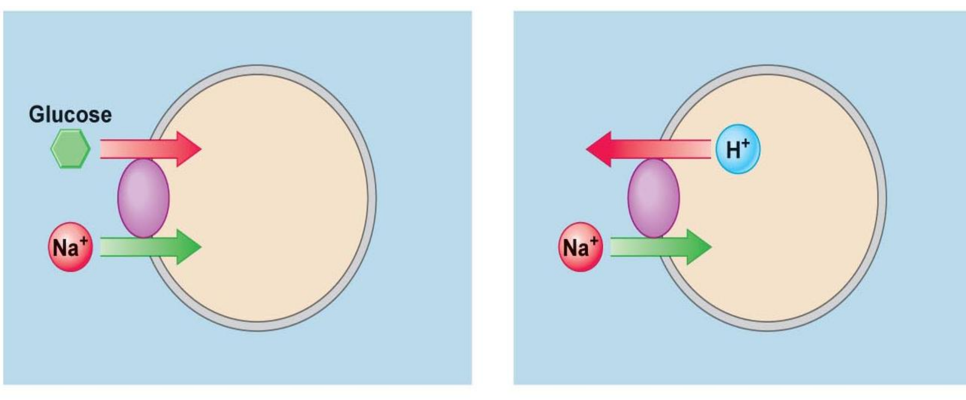
Proximal Convoluted Tubule (PCT) Reabsorption

"for better understanding"

- ## Guyton corner :

The high capacity of the proximal tubule for reabsorption results from its special cellular characteristics, as shown in **Figure 28-6**. The proximal tubule epithelial cells are highly metabolic and have large numbers of mitochondria to support powerful active transport processes. In addition, the proximal tubular cells have an extensive brush border on the luminal (apical) side of the membrane, as well as an extensive labyrinth of intercellular and basal channels, all of which together provide an extensive membrane surface area on the luminal and basolateral sides of the epithelium for rapid transport of sodium ions and other substances.

The extensive membrane surface of the epithelial brush border is also loaded with protein carrier molecules that transport a large fraction of the sodium ions across the luminal membrane linked by way of the *co-transport* mechanism with multiple organic nutrients such as amino acids and glucose. Additional sodium is transported from the tubular lumen into the cell by *counter-transport* mechanisms that reabsorb sodium while secreting other substances into the tubular lumen, especially hydrogen ions. 13th edition, P.343



(a) Cotransport (b) Countertransport

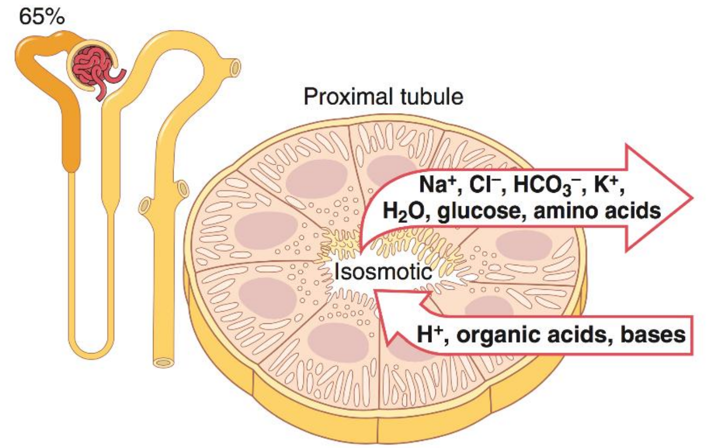
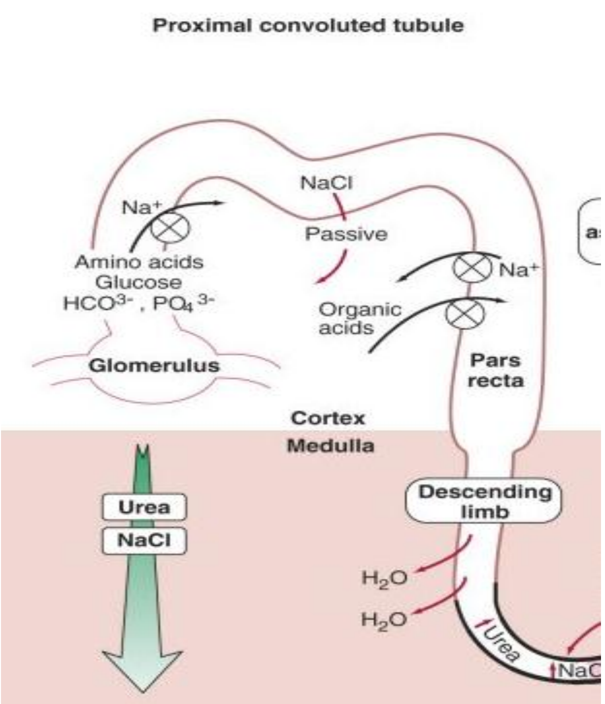


Figure 28-6

PCT Reabsorption

Na^+ Reabsorption

- The epithelium is *leaky*, and it's permeable to ions & water.
- **70%** of Na+, Cl-, K+ & water is absorbed passively (with Na+).

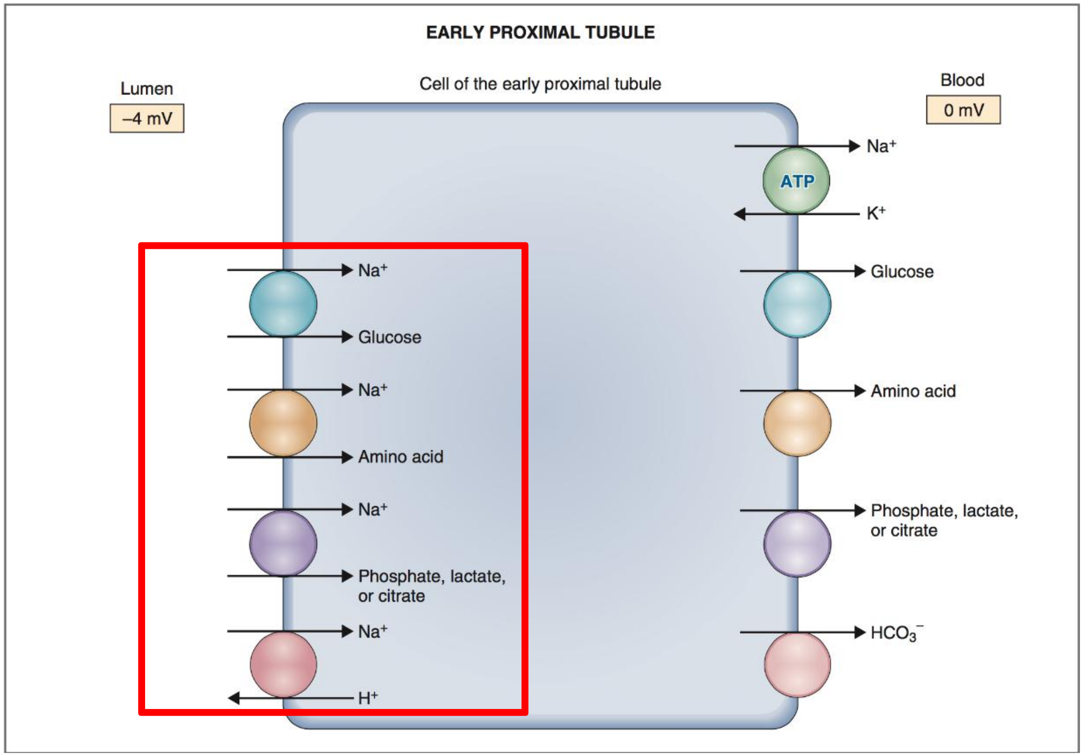


- **Linda corner:**

The proximal convoluted tubule consists of an early proximal convoluted tubule and a late proximal convoluted tubule.
The mechanisms for Na+ reabsorption in the early and late proximal tubules are different, as reflected in the anions and other solutes that accompany Na+. In the early proximal tubule, Na+ is reabsorbed primarily with HCO3− and organic solutes such as glucose and amino acids. In the late proximal tubule, Na+ is reabsorbed primarily with Cl−, but without organic solutes. *5th edition, p.271*

PCT Na+ Reabsorption

Early stage



- **Linda corner:**

The *cotransport* mechanisms in the luminal membrane of the early proximal tubule are Na+-glucose (SGLT), Na+-amino acid, Na+-phosphate, Na+-lactate, and Na+-citrate. In each case, Na+ moves into the cell and down its electrochemical gradient coupled to glucose, amino acid, phosphate, lactate, or citrate, which move into the cell against their electrochemical gradients. 5th edition, p.272

PCT Na+ Reabsorption

Early stage

Early PCT reabsorption

Exchange:

Na⁺/H⁺ exchanger (the transporter)

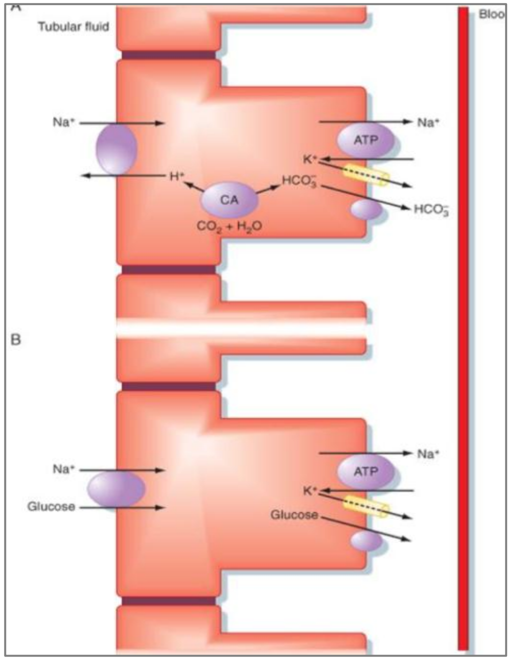
takes up Na^+ for H^+.

Which eventually Causes *reabsorption of HCO3⁻*

Symport:

Symporters (or cotransporters) are:

- Na^+ - glucose
- Na^+ - amino acid
- Na^+ - Pi
- Na^+ - lactate

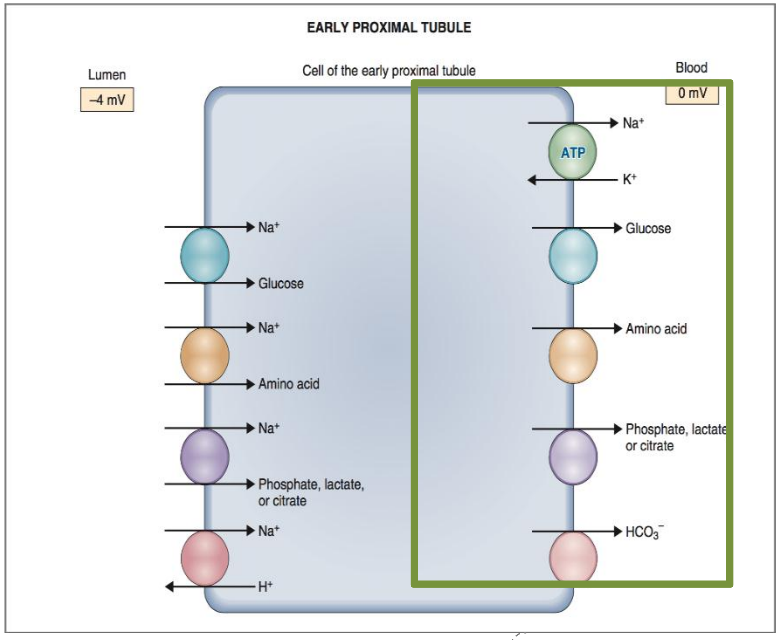


- **Symporters :** these carrier that transport Na passively Can take along with the Na some substrates as glucose and AA against their concentration gradient . So this active transport Is called secondary active Because it utilized Na energy to move Glucose and AA.
- **Symport "another definition":** a transport mechanism that moves two compounds simultaneously across a cell membrane in the same direction

Glucose Reabsorption

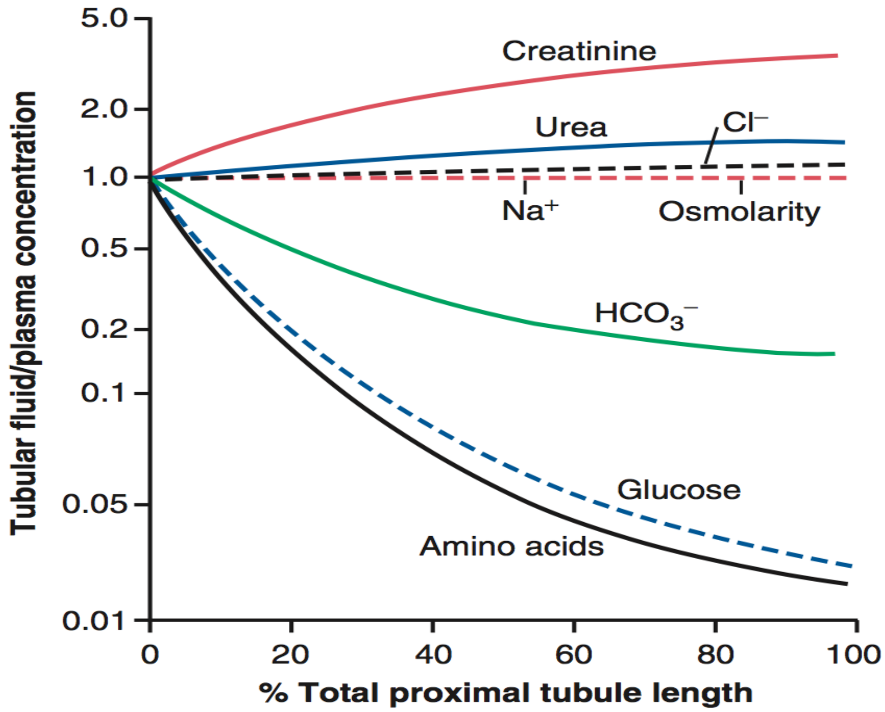
Early stage

- **From tubular lumen to tubular cell:**
  Sodium co-transporter (Carrier-mediated secondary active transport). Uphill transport of glucose driven by electro-chemical gradient of sodium, which is maintained by Na-K pump presents in basolateral cell membrane.



- **From tubular cell to peritubular capillary:**
  Facilitated diffusion (Carrier-mediated passive transport)

Finally, all substances move from tubular cell into blood capillary by facilitated diffusion (down their concentration gradient) EXCEPT for Na+ & K+, they have to move against their concentration gradient, so they need "Active Transport"



- **Guyton corner : Figure 28-7** summarizes the changes in concentrations of various solutes along the proximal tubule. Although the *amount* of sodium in the tubular fluid decreases markedly along the proximal tubule, the *con- centration* of sodium (and the total osmolarity) remains relatively constant because water permeability of the proximal tubules is so great that water reabsorption keeps pace with sodium reabsorption. Certain organic solutes, such as glucose, amino acids, and bicarbonate, are much more avidly reabsorbed than is water, and thus their con- centrations decrease markedly along the length of the proximal tubule. Other organic solutes that are less per- meant and not actively reabsorbed, such as creatinine, increase their concentration along the proximal tubule. The total solute concentration, as reflected by osmolarity, remains essentially the same all along the proximal tubule because of the extremely high permeability of this part of the nephron to water. 13th edititon, p.354

PCT Na+ Reabsorption

Late PCT

Late Proximal Convoluted Tubule

- **Na+ Reabsorbed mainly with Cl^- , Why ?**
  - Due to different transport mechanisms in late PCT.
  - Lack of organic molecules.

movement of Cl:

- A- it can be 2ry active because it move with Na transcellularly. If you wonder why it's not dragged along with solute in early PCT? the answer is because Cl are only uptaked by carriers in later part of PCT
- it can move through the tight junction. The reason is because the water has been reabsorbed in early PCT so the concentration of Cl will be higher than in interstitium. It will diffuse downhill to interstitium.

PCT Na+ Reabsorption

Late PCT

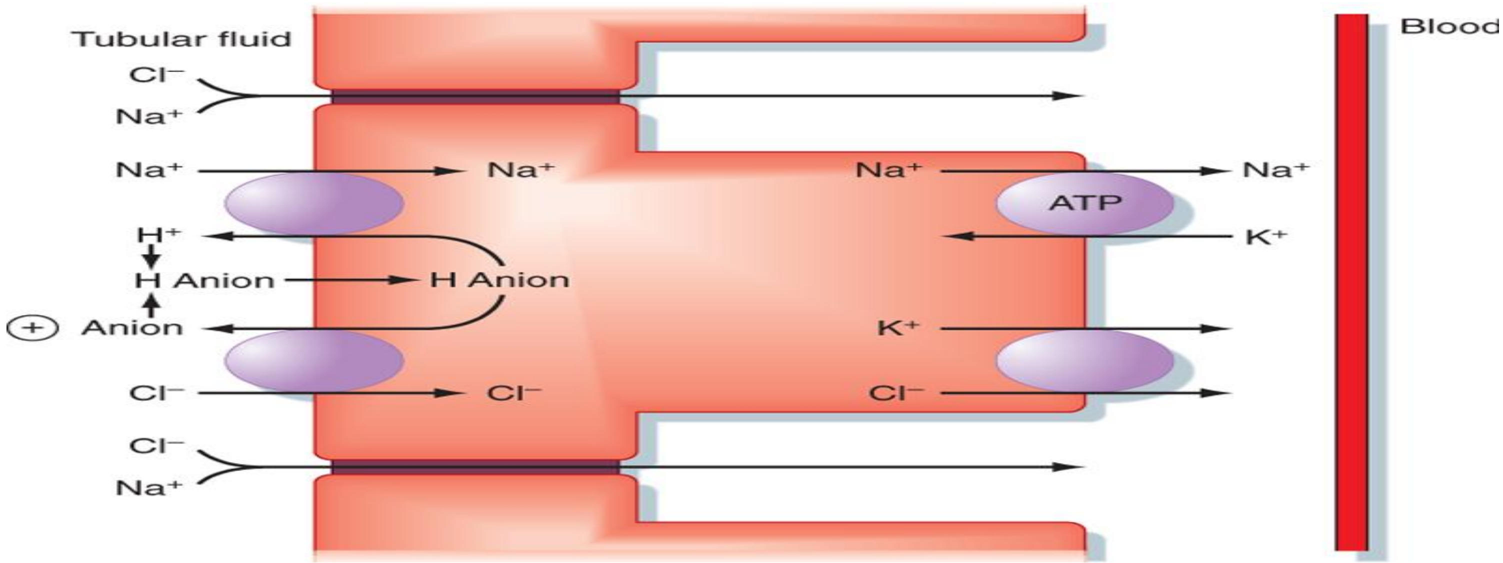
Paracellular

- passive diffusion With Cl^-
- driven by high [Cl-] in tubule
- 140mEq/L in the tubule lumen and 105 mEq/L in interstitium
- This conc. gradient favors diffusion of Cl- from the tubular lumen a cross the tight junction into the lateral intercellular space.

Transcellular

- Na+ entry using NHE & 1 or 2 Cl^- -anion antiporters
  - Na+-H+ exchanger

PCT Na+ Reabsorption



- **Guyton corner :**

Renal tubular cells, like other epithelial cells, are held together by tight junctions.Lateral intercellular spaces lie behind the tight junctions and separate the epithelial cells of the tubule. Solutes can be reabsorbed or secreted across the cells through the transcellular pathway or between the cells by moving across the tight junctions and intercellular spaces by way of the paracellular pathway. Sodium is a substance that moves through both routes, although most of the sodium is transported through the transcellular pathway. In some nephron segments, especially the proximal tubule, water is also reabsorbed across the paracellular pathway, and substances dissolved in the water, especially potassium, magnesium, and chloride ions, are carried with the reabsorbed fluid between the cells.

PCT Na+ Reabsorption

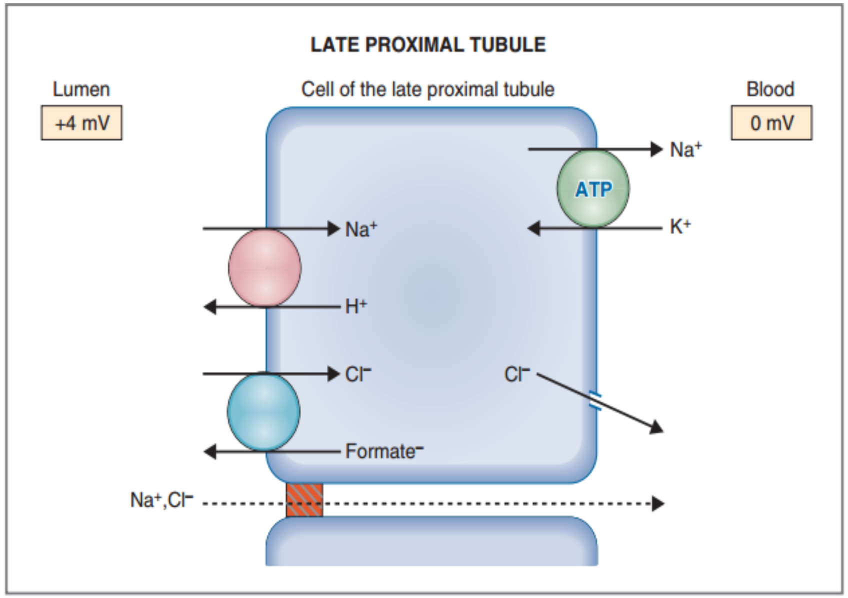


Figure 6–21 Cellular mechanisms of Na+ reabsorption in the late proximal tubule. The trans-epithelial potential difference is +4 mV. ATP, Adenosine triphosphate.

- **Linda corner:**

In contrast to the early proximal tubule, the late proximal tubule reabsorbs primarily NaCl **(Fig. 6-21).** The high tubular fluid Cl− concentration is the driving force for this reabsorption, for which there are both cellular and paracellular (between cells) components.The cellular Component of NaCl reabsorption is explained as follows: The luminal membrane of late proximal cells contains two exchange mechanisms, including the familiar Na+-H+ exchanger and a Cl−-formate−anion exchanger, which is driven by the high tubular fluid Cl − concentration. The combined function of the two exchangers is to transport NaCl from the lumen into the cell. Na+ then is extruded into blood by the Na+-K+ATPase, and Cl−moves into blood by diffusion.The paracellular component also depends on the high tubular fluid Cl−concentration. The tight junctions between cells of the proximal tubule are, in fact, not tight: They are quite permeable to small solutes, such as NaCl, and to water. Thus, the Cl − concentration gradient drives Cl− diffusion between the cells, from lumen to blood. This Cl−diffusion stablishes a Cl−diffusion potential, making the lumen positive with respect to blood. Na+reabsorption follows, driven by the lumen-positive potential difference. Like the cellular route, the net result of the paracellular route is reabsorption of NaCl.

Urea Reabsorption

Normal plasma level of urea 2.5-6.5 mM/L (15-39 mg/100ml).

Mechanism of urea reabsorption:

- About 40-70% of filtered load of urea is reabsorbed in:
  - Second half of PCT.
  - Medullary CT and CD *(ADH dependent)*.

- Due to water reabsorption in the first half of PCT, the conc. of urea is increased in the second half and urea is reabsorbed by simple diffusion *(downhill)*.

فقد الماء زاد من تركيز اليوريا مما أدى إلى سهولة انتقاله إلى الدم (passive diffusion)

- **Guyton corner :**

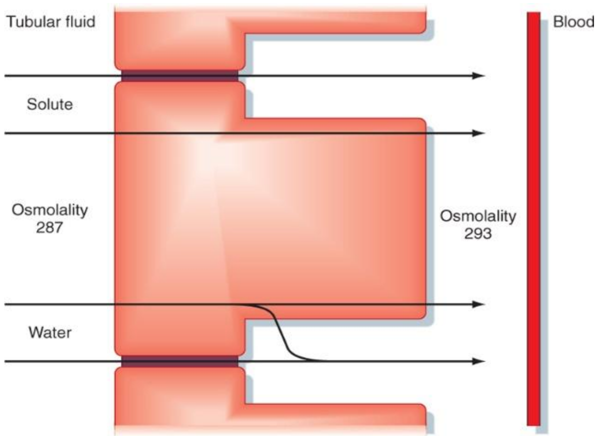
Urea is also passively reabsorbed from the tubule, but to a much lesser extent than chloride ions. As water is reabsorbed from the tubules (by osmosis coupled to sodium reabsorption), urea concentration in the tubular lumen increases. This increase creates a concentration gradient favoring the reabsorption of urea.

- **Linda corner:**

Urea is freely filtered across the glomerular capillaries, and the concentration in the initial filtrate is identical to that in blood (i.e., initially, there is no concentration difference or driving force for urea reabsorption). However, as water is reabsorbed along the nephron, the urea concentration in tubular fluid increases, creating a driving force for passive urea reabsorption. Therefore, urea reabsorption generally follows the same pattern as water reabsorption—the greater the water reabsorption, the greater the urea reabsorption and the lower the urea excretion.In the proximal tubule,50% of the filtered urea is reabsorbed by simple diffusion. As water is reabsorbed in the proximal tubule, urea lags slightly behind, causing the urea concentration in the tubular lumen to become slightly higher than the urea concentration in blood; this concentration difference then drives passive urea reabsorption. At the end of the proximal tubule, 50% of the filtered urea has been reabsorbed; thus, 50% remains in the lumen.

Water reabsorption

- **PCT** cells *permeable* to water.
- **PCT** Reabsorbs 67% of filtered water.
- **Transtubular Passive (osmosis)** due to osmotic active substances that are absorbed, e.g. *Na+, glucose, HCO3-, Cl-*
  ⇒ ↓ tubule osmolality.
  ↑ intracellular space osmolality.



- **Guyton corner :**

When solutes are transported out of the tubule by either primary or secondary active transport, their concentrations tend to decrease inside the tubule while increasing in the renal interstitium. This phenomenon creates a concentration difference that causes osmosis of water in the same direction that the solutes are transported, from the tubular lumen to the renal interstitium. Some parts of the renal tubule, especially the proximal tubule, are highly permeable to water, and water reabsorption occurs so rapidly that there is only a small concentration gradient for Solutes across the tubular membrane .A large part of the osmotic flow of water in the proximal tubules occurs through the so-called tight junctions between the epithelial cells, as well as through the cells themselves. The reason for this Situation, as already discussed, is that the junctions between the cells are not as tight as their name would imply and permit significant diffusion of water and small ions. This condition is especially true in the proximal tubules, which have a high permeability for water and a smaller but significant permeability to most ions, such as sodium, chloride, potassium, calcium, and magnesium.

- **Linda corner:** Isosmotic reabsorption is a hallmark of proximal tubular function: Solute and water reabsorption are coupled and are proportional to each other. Thus, if 67% of the filtered solute is reabsorbed by the proximal tubule, then 67% of the filtered water also will be reabsorbed. What solutes are included in the general term “solute”?The major cation is Na +, with its accompanying anions HCO3−(early proximal tubule) and Cl−(late proximal tubule). Minor anions are phosphate, lactate, and citrate. Other solutes are glucose and amino acids. Quantitatively, however, most of the solute reabsorbed by the proximal tubule is NaCl and NaHCO3.

Water Reabsorption

Solvent drag of K+, Ca2+

↓

Carried with water "reabsorbed"

↓

Accumulation of fluid and solutes within the lateral intercellular space

↓

Increase in hydrostatic pressure

↓

This forces fluid and solutes into the capillaries

↓

Water reabsorption follows solutes

The proximal tubule reabsorption is isosmotic

it's important to know that The proximal tubule reabsorption is isosmotic. Meaning that there are equal amount of solute and water reabsorbed.

Water Reabsorption, Cont..

- **Guyton corner :**

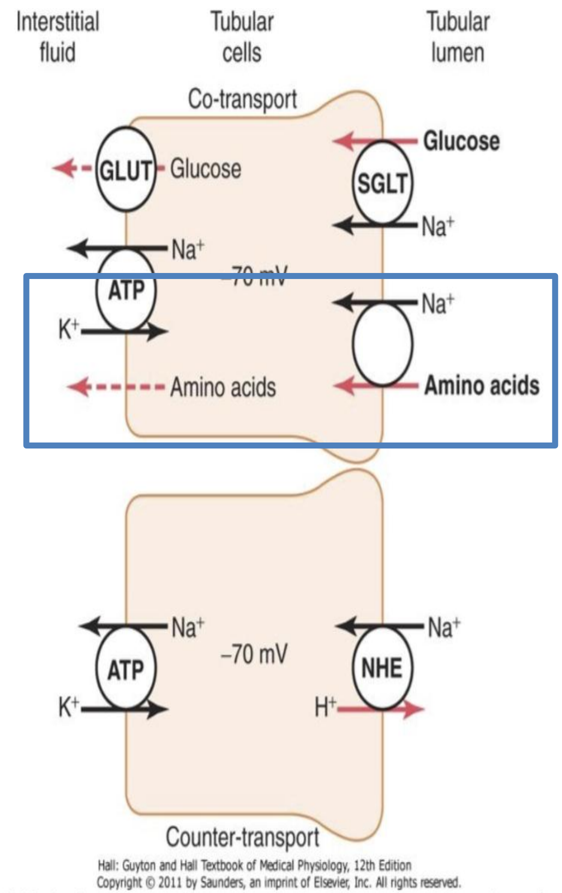
As water moves across the tight junctions by osmosis, it can also carry with it some of the solutes, a process referred to as solvent drag.

- **Tubular Fluid Remains Isosmotic in the Proximal Tubule:**

As fluid flows through the **proximal tubule**, solutes and water are reabsorbed in equal proportions, so little change in osmolarity occurs; thus, the **proximal tubule** fluid remains **isosmotic** to the plasma, with an osmolarity of about 300 mOsm/L. As fluid passes down the descending loop of Henle, water is reabsorbed by osmosis and the tubular fluid reaches equilibrium with the surrounding interstitial fluid of the renal medulla, which is very hypertonic—about two to four times the osmolarity of the original glomerular filtrate. Therefore, the tubular fluid becomes more concentrated as it flows into the inner medulla.

Protein Reabsorption

- Peptide hormones, small proteins & amino acids are reabsorbed in **PCT**.
- **Has a maximum capacity:**
  if **too much** protein is filtered → Proteinuria.
- Peptide hormones, small proteinuria & amino acids undergo Endocytosis into the PCT, **either** intact **or** after being partially degraded by enzymes.
- Once protein is inside the cell, enzymes digest them into amino acids, which leave the cell to the blood.



Organic Ion / Cation **Secretion**

Organic Ion/Cation Secretion

Endogenous Compounds:

- End products of metabolism
- Bile Salts
- Creatinine
- Catecholamine :

(Adrenaline , Noradrenaline)

Exogenous Compounds:

- Penicillin

-NSAIDs

(e.g. Ibuprofen)

- Morphine

Organic Ion / Cation Secretion, Cont..

- **Guyton corner :**

The proximal tubule is also an important site for secretion of organic acids and bases such as bile salts, oxalate, urate, and catecholamines. Many of these substances are the end products of metabolism and must be rapidly removed from the body. The secretion of these substances into the proximal tubule plus filtration into the proximal tubule by the glomerular capillaries and the almost total lack of reabsorption by the tubules, all combined, contribute to rapid excretion in the urine.

In addition to the waste products of metabolism, the kidneys secrete many potentially harmful drugs or toxins directly through the tubular cells into the tubules and rapidly clear these substances from the blood. In the case of certain drugs, such as penicillin and salicylates, the rapid clearance by the kidneys creates a problem in maintaining a therapeutically effective drug concentration.

HCO_3^- Reabsorption

- The renal tubules are *poorly- permeable* to HCO_3^- . **However**, it is still reabsorbed **but** in the form of CO_2 (to which the tubules are highly permeable)

- **This occurs through the following steps:**

H^+ is formed inside the cells then secreted into the tubular fluid

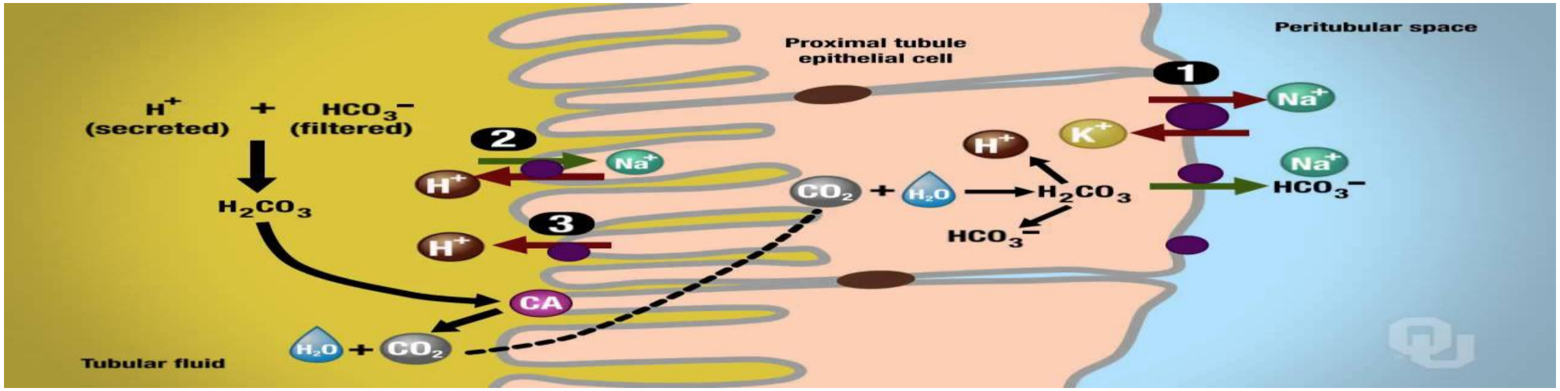
→ H^+ Combines with HCO_3^- in the tubular fluid forming H_2CO_3

→ By activity of Carbonic Anhydrase enzyme (C.A.) in the **Tubular cells**, H_2CO_3 dissociates into CO_2 & H_2O

→ CO_2 Diffuses into the cells Where it combines with H_2O (By activity of **intracellular** C.A), Forming H_2CO_3 which dissociates into HCO_3^- & H^+

→ HCO_3^- passively diffuses into the interstitial fluid (then to the blood) while H^+ is secreted into the tubular fluid to help more reabsorption of HCO_3^-

EXTRA



H_2CO_3 Dissociates 2 times :

- The First time: Is In the Tubular Fluid with the help of Extracellular C.A. Into CO_2 and H_2O.
- The Second time: Is in the Tubular Cell into HCO_3^- & H^+

Na - H exchanger: this process happen to reabsorb HCO3 is the following step :

1- in the cell we will find H2O & CO2 due to the passive diffusion of them. Then the enzyme CA will fuse them together to form H2CO3 which is unstable it dissolve easily into H and HCO3

2- H+ is moved inside the tubule through a counter-transport with Na actively (2ry active transport)

3- the purpose of transporting H+ inside tubule is to destroy the filtered HCO3 before it get excreted in urine. It will dissolve to form H2CO3 then into CO2 and water.

4- CO2 enter to cell to form bicarbonate and excrete it to blood with facilitated diffusion.

HCO_3^- Reabsorption, Cont..

Factors Affecting HCO_3^- Reabsorption:

- Arterial P_{CO2}
- Plasma K^+
- Plasma Cl^-
- Plasma Aldosterone

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