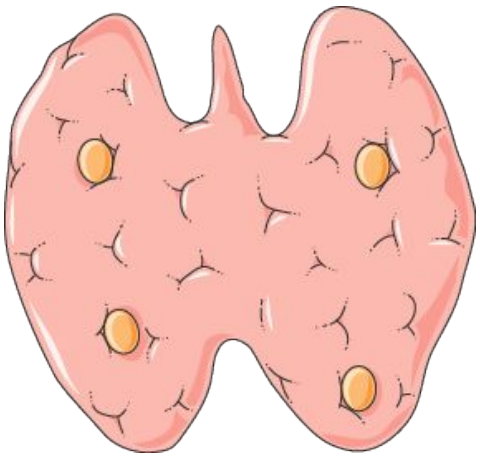
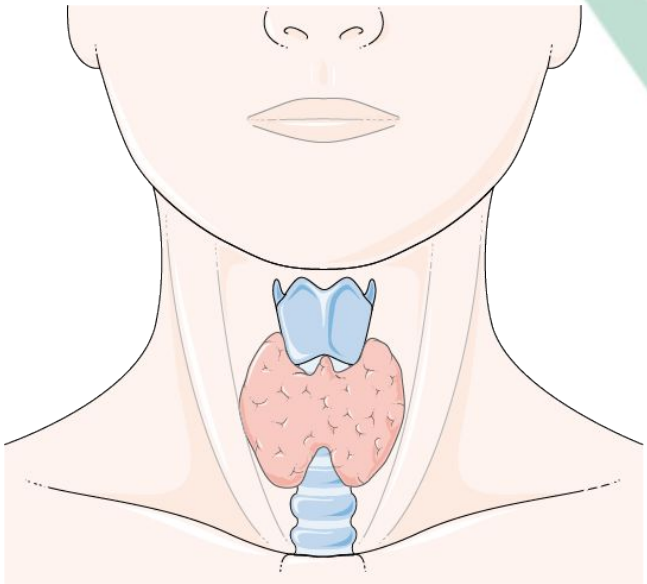
8+9





Calcium Homeostasis & Hypo and Hyperparathyroidism

ENDO Physiology

Editing File

Color Index :

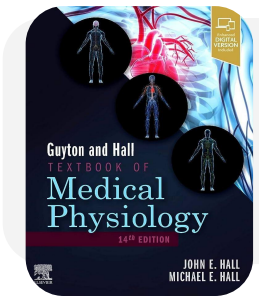
- Main Text
- Important
- Girls Slides
- Boys Slides
- Notes
- Extra

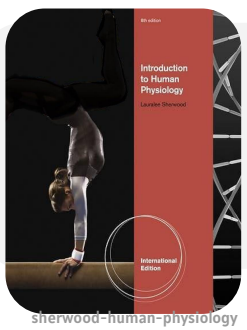
Objectives

- Identify the normal range of dietary calcium and phosphate intake, distribution in the body, and routes of excretion.
- Know the cells of origin for parathyroid hormone
- List the target organs and cell types for parathyroid hormone and describe its effects on each.
- Describe the functions of the osteoblasts and the osteoclasts in bone remodeling.
- Describe the regulation of parathyroid hormone secretion and the role of the calcium-sensing receptor.
- Understand the causes and consequences of: a) over-secretion, and b) under-secretion of parathyroid hormone, as well as its therapeutic use.
- Identify the sources of vitamin D and the organs involved in modifying it to the biologically active I,25(OH2)D3 (I-25 dihydroxycholecalciferol).
- Identify the target organs and cellular mechanisms of action for vitamin D.
- Describe the negative feedback relationship between parathyroid hormone and the biologically active form of vitamin D [I,25(OH2)D3].
- Describe the consequences of vit. D deficiency and vit. D excess.
- Name the stimuli that can promote secretion of calcitonin, and its actions.
- List the functions of calcium, describe calcium metabolism & physiology of bone
- Understand and explain hormonal regulation of calcium metabolism via Parathyroid hormone, Calcitonin, Vitamin D3
- Understand hypo and hyperparathyroidism

Resources

Only ENDO chapters included





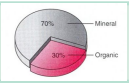
sherwood-human-physiology

This lecture was presented by:
Dr. Abeer Ghumlas - Dr. Khalid Alregaiey

Introduction to bone composition

Female slides

Bone composition



Organic matrix 30%

Components are: (I) Cells (2) Matrix:

I. Cells:

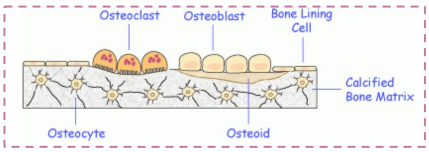
- Osteoblasts: Bone forming cells

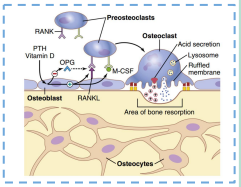
- Osteocytes: Osteoblasts surrounded by calcified matrix

- Osteoclasts: Bone eroding Cell (resorping)

Only in male slides but female Dr explained it

Note that: Remodeling of Bone Bone is continually being deposited by osteoblasts, and it is continually being resorbed where osteoclasts are active



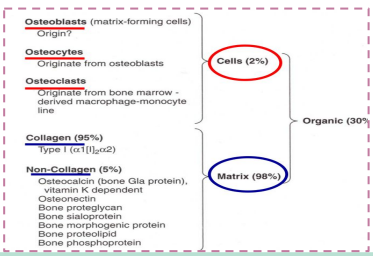


2. Matrix:

- **Provide/Functions:** Tensile force

- Collagen Fibers: 95%

- Ground Substance (ECF, Proteoglycans): 5%



Inorganic matrix (Mineral salts) 70%

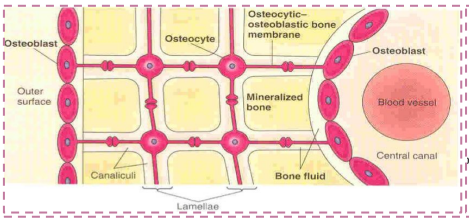
(I) Provide/Functions: Compressional force

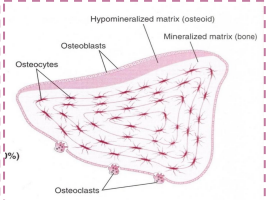
(2) Components:

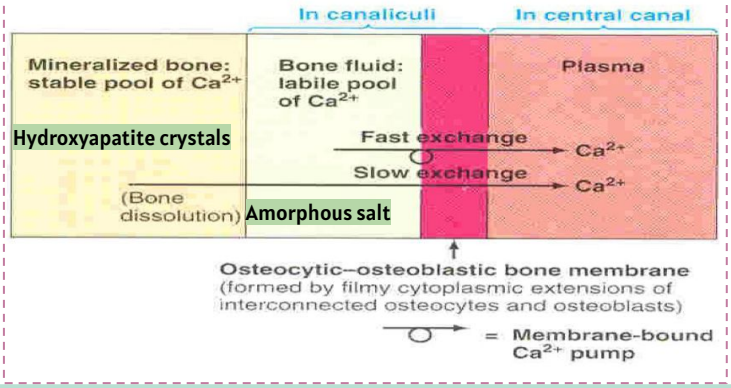
- Salts of Ca++ & PO4- In the form of **Hydroxyapatite crystals** $[Ca_{10}(PO_4)_6(OH)_2]$: 99% of total Ca++ salt

- **Amorphous salts** (0.4-1% of total bone Ca++): A **type of exchangeable calcium**. Play a role in rapid regulation of ionized Ca++ level in ECF. It is always in equilibrium with Ca++ present in ECF.

- Mg, Na, K, Carbonate ions







Male slides

Mechanical Stress (Wolff's Law)

1. States that bone in a healthy person or animal will adapt to the loads under which it is placed. If loading on a particular bone increases, the bone will remodel itself over time to become stronger to resist that sort of loading.

2. For example, the bones of athletes become considerably heavier than those of nonathletes. Also, if a person has one leg in a cast but continues to walk on the opposite leg, the bone of the leg in the cast becomes thin and as much as 30% decalcified within a few weeks, whereas the opposite bone remains thick and normally calcified.

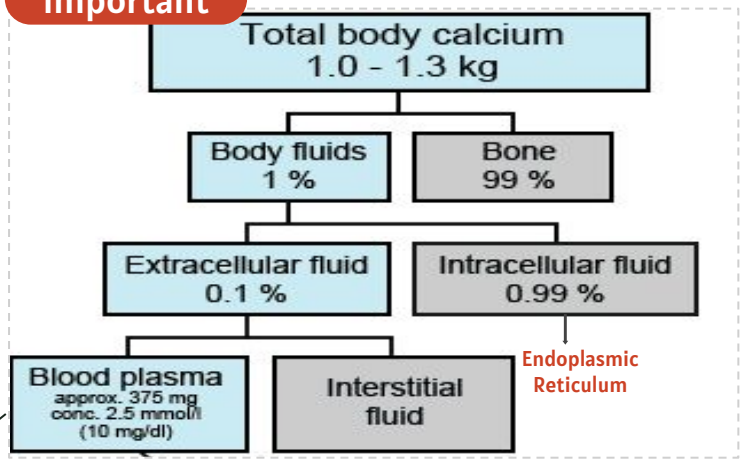
Distribution of Ca++ in the body

TABLE 40.1 Forms of Ca and P_i in Plasma

Ion	mg/dL	Ionized	Protein Bound	Complexed
Ca	8.5–10.2	50%	45%	5%
P_i	3–4.5	84%	10%	6%

Ca^{++} is bound (i.e., complexed) to various anions in plasma, including HCO_3^-, citrate, and SO_4^{2-}. P_i is complexed to various cations, including Na^+ and K^+.
From Koeppen BM, Stanton BA. *Renal Physiology.* 4th ed. Philadelphia: Mosby; 2007.

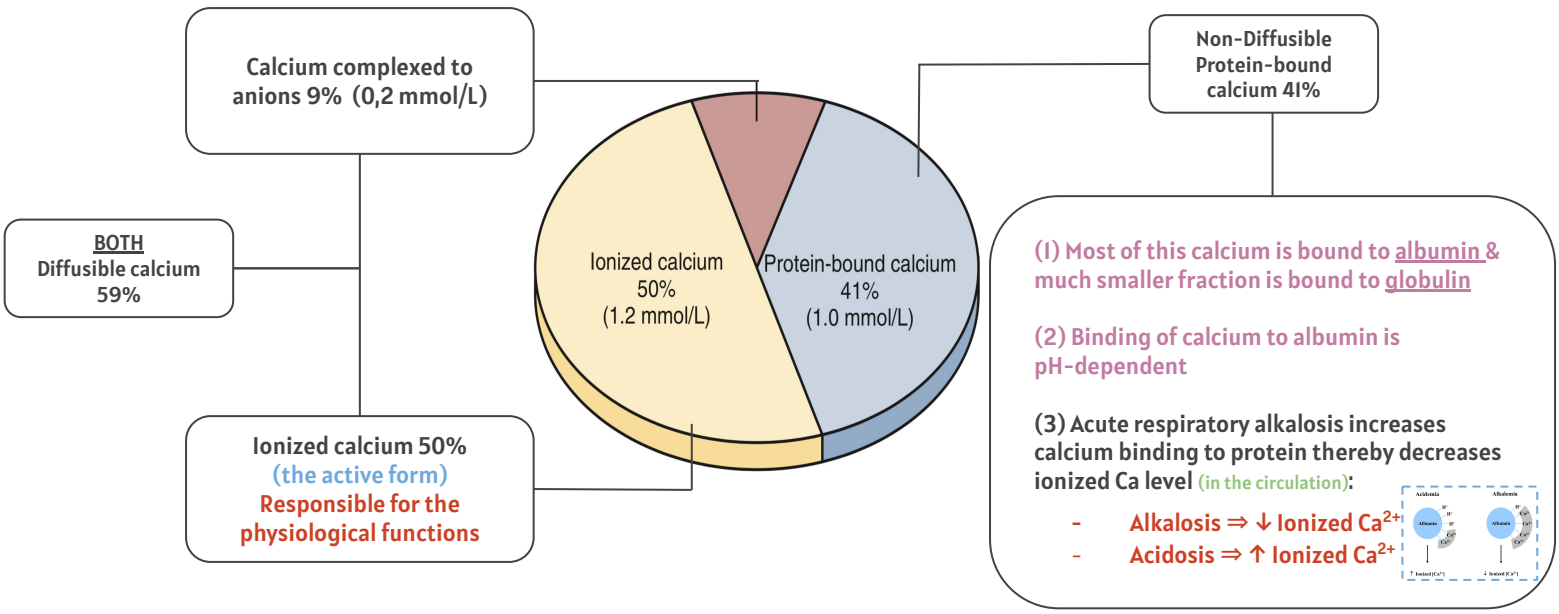
Important



Total plasma Ca++= 9-10.5 mg/dl / 2.4 mM (9.4 mg/dl)

- Diffusible (ionized and complexed) & Non-diffusible (protein bound)
- Ionized calcium is 1.2 mM (50% of total calcium), it is the physiologically active form.

Total conc of calcium in ECF=10 mg/dl ,5m mEq/L ,2.5mmol/L



Phosphate

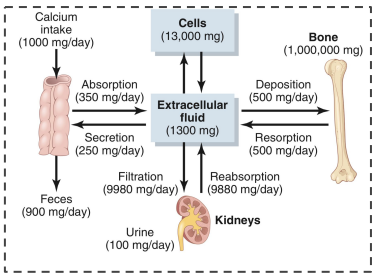
- Approximately 85 % of the body's phosphate is stored in bones,14-15 % is in the cells. Only Less than 1% is in the extracellular fluid. Although extracellular fluid phosphate con. is not nearly as well regulated as calcium concentration, phosphate serves several important functions and is controlled by many of the same factors that regulate calcium.

- Phosphorous is an essential mineral necessary for ATP and cAMP second messenger systems
- Phosphate plasma concentration is around 4 mg/dL
- Forms:
  - Ionized (diffusible) around 50% of total
  - un-ionized (non-diffusible) protein- bound (50%)

Calcium

Female slides

Calcium

Physiological importance	- Calcium salts in bone provide **structural integrity** of the skeleton . - Calcium ions in extracellular and cellular fluids is essential to normal function for the biochemical processes: - Neuromuscular excitability - Hormonal secretion - Enzymatic regulation - Blood coagulation - Second messenger
Source	- Milk, dairy products, Fish  
Daily Requirement	- Infants & adults: 12.5 -25 mmol/day - Pregnancy, lactation ,after menopause: 25-35 mmol/day
Absorption	- Duodenum: active transport - Small intestine: concentration gradient
Metabolism	1000 - 350= 650 (Read the explanation) Explanation: The usual rates of intake are approximately **1000 mg/day** each for calcium and phosphorus, about the amounts in 1 liter of milk. Normally, divalent cations such as calcium ions are poorly absorbed from the intestines. However, (as discussed later), vitamin D promotes calcium absorption by the intestines, and about **35% (350 mg/day) of the ingested calcium is usually absorbed**; the remaining calcium in the intestine is **excreted in the feces (650 mg/day)**. An additional **250 mg/day** of calcium enters the intestines via secreted gastrointestinal juices and sloughed mucosal cells. **Thus, about 90% (900 mg/day) of the daily intake of calcium is excreted in the feces + excretion (urine 100) equals the intake (1000).** Note: Both male and female slides 
Regulation	- Changes in Plasma Concentrations of Free Calcium -Nonhormonal Mechanisms Can Rapidly Buffer Small Changes in Plasma Concentrations of Free Calcium - Hormonal Mechanisms Provide High-Capacity, Long-Term Regulation of Plasma Calcium and Phosphate Concentrations • [Ca2+] < 9-10.5 mg/dl → Tetany + seizures + excitation of the nervous system • [Ca2+] > 9-10.5 mg/dl → Renal stones + depression of the nervous system

Regulated by

Phosphate	Hormones
• Ca is tightly regulated with Phosphorus in the body.	• Vitamin D (Diet and sun) • Parathyroid hormone (Parathyroid gland) • Calcitonin Thyroid gland

Hormones that regulate Ca++

Note that each hormone will be discussed in details in the following slides

Table view summary

table 7.11 Summary of Hormones that Regulate Ca^{2+}

	PTH	Vitamin D	Calcitonin
Stimulus for secretion	↓ serum [Ca^{2+}]	↓ serum [Ca^{2+}] ↑ PTH ↓ serum [phosphate]	↑ serum [Ca^{2+}]
Action on			
Bone	↑ resorption	↑ resorption	↓ resorption
Kidney	↓ P reabsorption (↑ urinary cAMP)	↑ P reabsorption	
	↑ Ca^{2+} reabsorption	↑ Ca^{2+} reabsorption	
Intestine	↑ Ca^{2+} absorption (via activation of vitamin D)	↑ Ca^{2+} absorption (calbindin D-28K)	
		↑ P absorption	
Overall effect on			
Serum [Ca^{2+}]	↑	↑	↓
Serum [phosphate]	↓	↑	

cAMP = cyclic adenosine monophosphate. See Table 7.1 for other abbreviation.

Diagram view summary

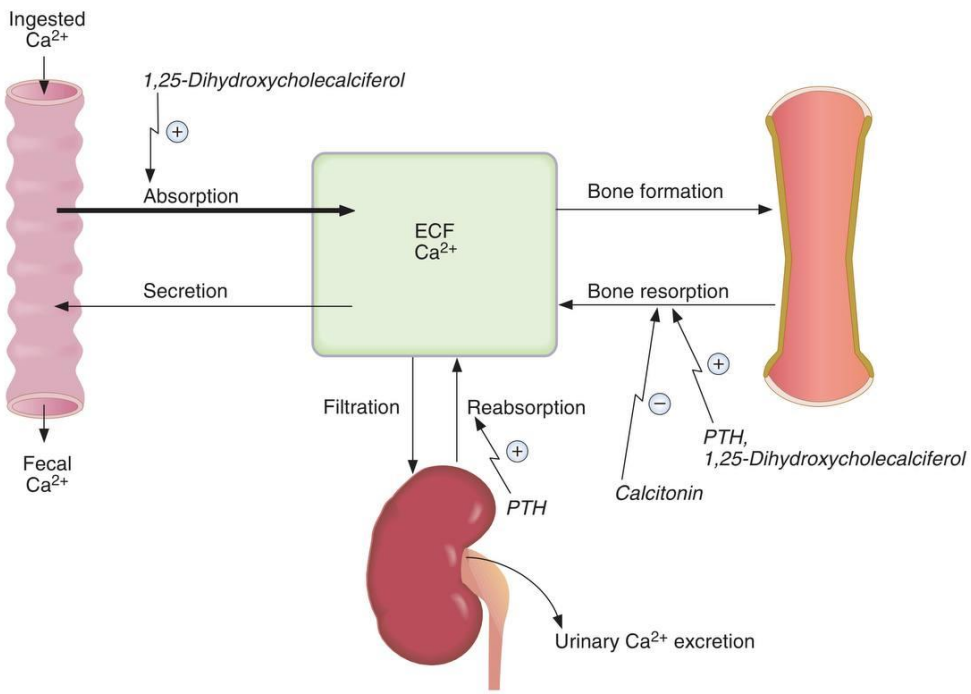


FIGURE 7.13. Hormonal regulation of Ca^{2+} metabolism. ECF = extracellular fluid; PTH = parathyroid hormone.

Important

Hormones that regulate Ca++

Click here to see Graphs Explanations !!

Vitamin D

Vit. D metabolism

"Look at the Pic"

Vitamin D3 (cholecalciferol) is a prohormone that must undergo two successive hydroxylation reactions to become the active form known as 1,25- dihydroxyvitamin D or calcitriol

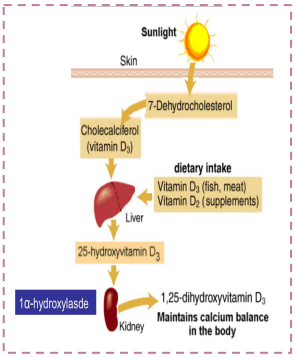
1- Sunlight / Ultraviolet B light : (7-Dehydrocholesterol > Vit D3 aka; cholecalciferol) inactive

2-Dietary intake: Vit D3 (fish, meat). Vit D2(supplements)

3- stored in the liver

4- The liver take up the Vit D forms and convert it to 25-hydroxyvitamin D3 By 25α-hydroxylase (Feedback control limits concentration)

5- Kidneys take up 25-hydroxyvitamin D3 and convert it into 1-25 dihydroxyvitamin D3 (active form) by the enzyme 1α-hydroxylase (this enzyme is activated by PTH) (Under the feedback control of (PTH))



Stimulus for secretion

reduced in calcium level

Site of action

Bone, kidney and Intestinal tract

Function (actions)

Vitamin D in the form of **1,25 Dihydroxycholecalciferol** increases calcium blood level by:

Female slides

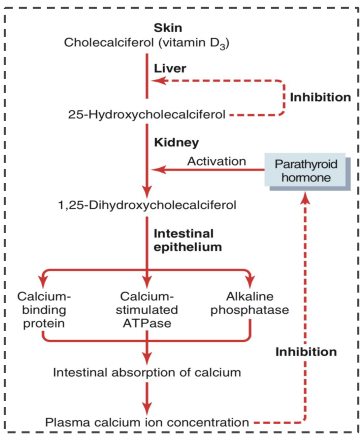
On bone & Its Relation to PTH.	On intestine	On kidney	On immunity
(1) Vitamin D in smaller quantities : promotes bone calcification (by↑calcium and phosphate absorption from the intestine and enhances the mineralization of bone) (2) The administration of extreme quantities of vitamin D causes absorption of bone: - By facilitating PTH action on bones. - Number & activity of osteoclasts.	Has a potent effect to increase calcium and phosphate absorption. (increases synthesis of calcium binding proteins (thats why its imp when taking vit. D supp. to take it with a Ca source/dairy) and related facilitated transport)	Slight effect ↑calcium and phosphate reabsorption.	Stimulates differentiation of immune cells

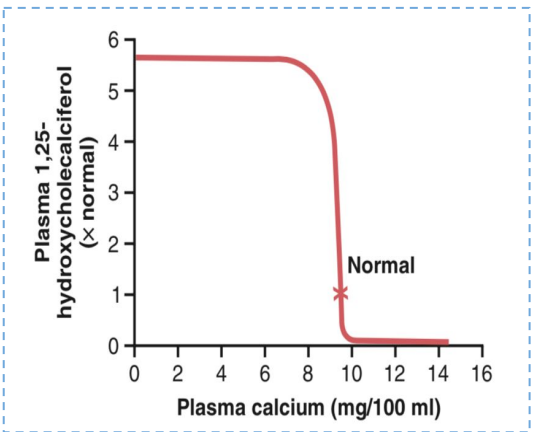
Control of Vit. D

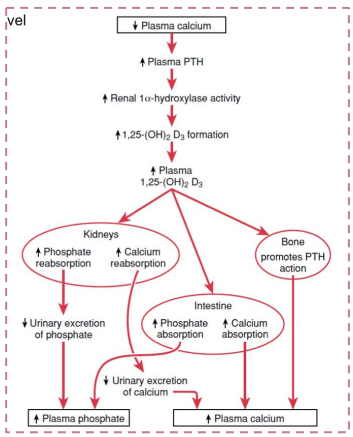
They all stimulate renal 1,alpha hydroxylase:

1- low Ca++ ions 2- prolactin 3- PTH

Pictures & Diagrams





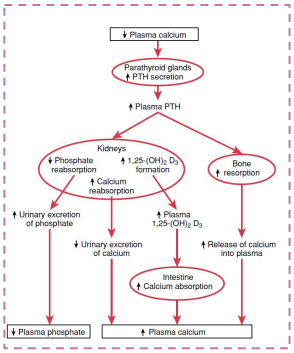
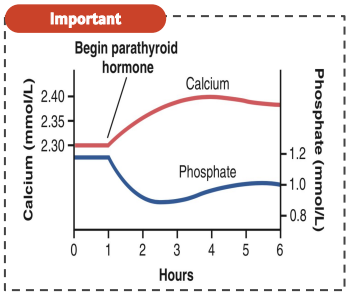
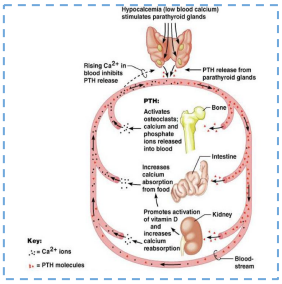
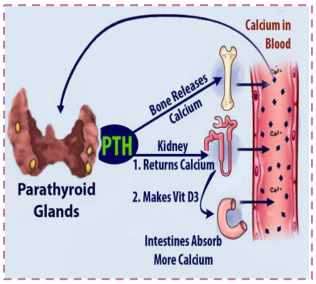


Important

Hormones that regulate Ca++

Click here to see Graphs Explanations !!

Parathyroid hormone (PTH) (It is essential for life)

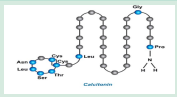
Source	Secreted by chief (Principale) cells of parathyroid gland		
General	- Polypeptide hormone: (84 aa) - Molecular Weight: 9500 daltons - Half Life: 10 min		
Stimulus for secretion:	Rapid response to reduced calcium (minutes)		
MOA	Acts via 2nd messenger mechanism utilizing cAMP Operates in tissues via GPCR		
Site of action	Primary target : Bone, Kidney and Intestine		
Function (actions)	On bone	On kidney	On intestine
	(1) Existing osteoclasts activated (2) new osteoclasts formed (Days to weeks) to digest bone and release calcium and phosphate. (2) Existing osteocytes stimulated (minutes to hours) to transport calcium – calcium pumps (3) Stimulated indirectly by osteoblasts: osteoblasts express RANKL which binds to RANK on osteoclasts leading to its activation.) (4) Increases Calcium and Phosphate Absorption from the Bone (5) Depression of Osteoblastic activity	(1) ↓phosphate reabsorption from the proximal convoluted tubules (phosphaturic action). Which leads to: ↑Phosphate excretion in the urine ↓ plasma phosphate concentration (2) ↑ Ca++ & Mg ions reabsorption from the distal convoluted tubules, collection ducts and ascending loop of Henle. (3) Decreases excretion of calcium by kidneys important to prevent bone deterioration (4) ↑ Formation of 1,25 vit D3 in the kidney and Produces most active form of Vitamin D3 in the kidney (1,25-dihydroxy-cholecalciferol)	(1) ↑ absorption of calcium effect manifested via Vitamin D3 and phosphate indirectly through stimulating formation of 1,25 – (OH) 2 -D3 in kidney
Abnormalities	(1) Hypoparathyroidism (2) Hyperparathyroidism		
Pictures & Diagrams		Important  Parathyroid hormone infusion during the first 5 h		

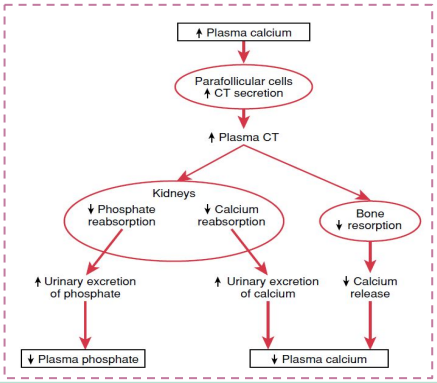
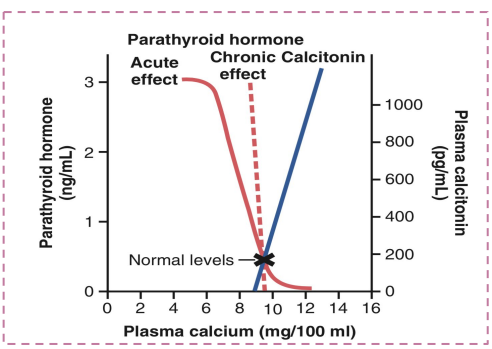
Important

Hormones that regulate Ca++

Click here to see Graphs Explanations !!

Calcitonin



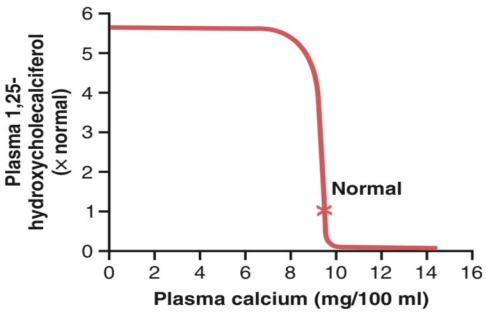
Source	Secreted by the parafollicular cells (C cells) of the thyroid gland.	
General	- Nature: 32 amino acid peptide.	
Stimulus for secretion:	Increased plasma calcium concentration	
MOA	Decrease blood Ca++ level very rapidly within minutes. Opposite effect to PTH	
Site of action	Bone and Kidney	
Function (actions)	On bone	On kidney
	(I) ↑Ca++ deposition of bone (Effect to decrease calcium is not permanent) (2) Inhibits Bone resorption by I. inhibition of osteoclasts. 2. ↓ formation of osteoclasts. - Effect to decrease calcium is transitory - Causes reduced bone turnover	Weak effect in kidney and intestines (I) ↓↓ Ca++ reabsorption (2) ↑↑ Ca++ excretion (in addition to phosphate)
Abnormalities	(I) Osteomalacia (2) Osteoporosis	
Pictures & Diagrams		

Important

Extra

Hormones that regulate Ca++

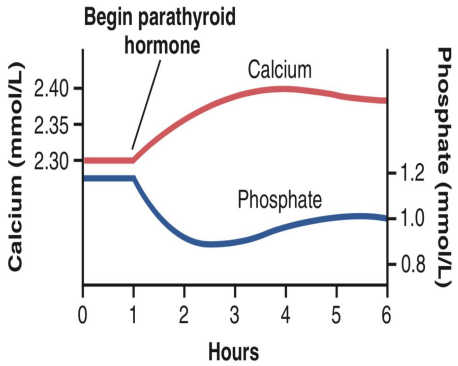
Graphs explanations



This graphs demonstrates that plasma concentration of I,25-dihydroxycholecalciferol is inversely affected by the concentration of calcium in the plasma. There are two reasons for this effect: **First**, the calcium ion has a slight effect in preventing conversion of 25-hydroxycholecalciferol to I,25-dihydroxycholecalciferol. **Second**, and even more important, the rate of PTH secretion is greatly suppressed when plasma calcium ion concentration rises above 9 to I0 mg/I00 ml. Therefore, at calcium concentrations below this level, PTH promotes conversion of 25-hydroxycholecalciferol to I,25-dihydroxycholecalciferol in the kidneys.

At higher calcium concentrations, when PTH is suppressed, 25-hydroxycholecalciferol is converted to a different compound (24,25-dihydroxycholecalciferol) that has almost no vitamin D effect.

When plasma calcium concentration is already too high, formation of I,25-dihydroxycholecalciferol is greatly depressed. Lack of I,25-dihydroxycholecalciferol, in turn, decreases absorption of calcium from the intes- tines, bones, and renal tubules, thus causing the calcium ion concentration to fall back toward its normal level.



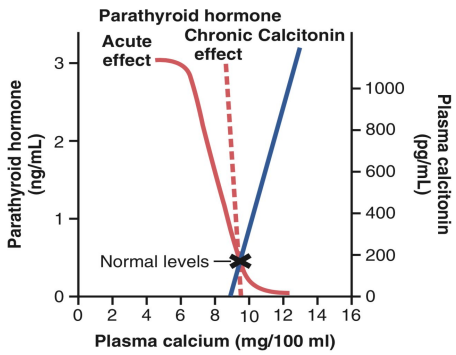
This graph shows the approximate effects on the blood calcium and phosphate concentrations caused by suddenly infusing PTH into an animal and continuing this infusion for several hours.

Note that at the onset of infusion, the calcium ion concentration begins to rise and reaches a plateau in about 4 hours. However, the phosphate concentration falls more rapidly than the calcium rises and reaches a depressed level within I or 2 hours.

The rise in calcium concentration is caused mainly by two effects:

(I) an effect of PTH to increase calcium and phosphate absorption from the bone, and
(2) a rapid effect of PTH to decrease excretion of calcium by the kidneys.

The decline in phosphate concentration is caused by a strong effect of PTH to increase renal phosphate excretion, an effect that is usually great enough to override increased phosphate absorption from the bone.



This graph shows the approximate relation between plasma calcium concentration and plasma PTH concentration.

The solid red curve shows the acute effect when the calcium concentration is changed over a period of a few hours and illustrates that even small decreases in calcium concentration from the normal value can double or triple the plasma PTH.

The approximate chronic effect when calcium ion concentration changes over a period of many weeks, thus allowing time for the glands to hypertrophy greatly, is shown by the dashed red line, which demonstrates that a decrease of only a fraction of a milligram per deciliter in plasma calcium concentration can double PTH secretion.

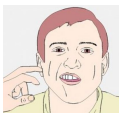
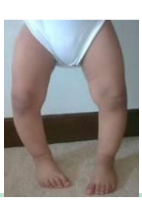
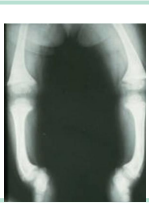
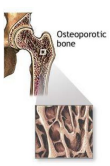
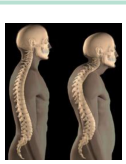
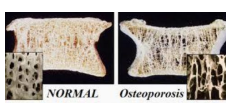
This is the basis of the body's extremely potent feedback system for long-term control of plasma calcium ion concentration.

Abnormalities

Female slides

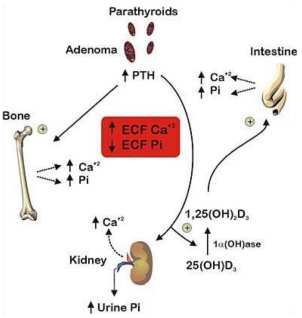
Whole slide in female slides except blue

1-Rickets 2-Osteomalacia 3- Osteoporosis 4-Hypo/Hyperparathyroidism

Bone disorders	Rickets (in children)	Osteomalacia (Adult Rickets) (rare)	Osteoporosis
Features:	-Low plasma calcium and phosphate. -Weak bones. -Tetany. -Occurs in spring? (because vit. D level decreases in winter & by spring storage levels decrease)  **-Positive Chvostek's sign is facial nerve irritability / spasms elicited by tapping the nerve.** (Hypocalcemia stimulate muscle contraction) -Normal formation of the collagen matrix BUT Incomplete mineralization (poor calcification), leading to Soft Bones (clinically: bone deformity).	- Serious deficiencies of both vitamin D & calcium occasionally occur as a result of steatorrhea (failure to absorb fat). - Almost never proceeds to the stage of tetany but often is a cause of severe bone disability. - Demineralization (poor calcification) of preexisting bones leads to more susceptibility to fractures	-The most common bone disease in adults, especially in old age. -Results from equal loss of both organic bone matrix and minerals resulting in loss of total bone mass and strength. **The cause of the diminished bone:** - The osteoblastic activity in the bone is usually less than normal so the rate of bone osteoid deposition is depressed. - Excess osteoclastic activity.
Causes:	Lack of vitamin D leading to calcium/phosphate deficiency in ECF.	Poor absorption of vitamin D and calcium.	(I) Lack of physical stress (2) Malnutrition (3) Lack of vitamin C (4) Postmenopausal lack of estrogen (5) Old age (6) Cushing's syndrome
Treatment:	Supplying adequate calcium and phosphate in the diet and, administering large amounts of vitamin D.	-	-
—	**-Early stages:** a- No tetany b- (PTH stimulate osteoblastic absorption of bone) c- ECF Calcium level is normal **-When the bones finally become exhausted of calcium,** Calcium level falls rapidly. **-Blood level of calcium falls below 7 mg/dl:** a-signs of tetany: (positive Chvostek's sign) b-Death: tetanic respiratory spasm	**Renal Rickets:** A type of Osteomalacia due to prolonged kidney disease. -Failure of the damaged kidney to form.	**Osteoporosis symptoms:** -Hip fracture -Kyphosis (hunched posture)
Pics:	 	-	  

Hyperparathyroidism (PTH Excess)

- -The disorder is characterized by hypercalcemia, hypercalciuria, hypophosphatemia, and hyperphosphaturia.
- -Parathyroid hormone causes phosphaturia and a decrease in serum phosphate.
- -Plasma Calcium rises and it is also secreted in the urine.
- -Renal stones made of calcium phosphate can result.
- -Most serious complication is the deposition of calcium in the kidney tubules resulting in impaired renal function.



Causes of Hypercalcemia / Hyperparathyroidism

Primary Hyperparathyroidism	Secondary (normo- or hypocalcemic) (compensatory) Hyperparathyroidism
•Adenoma (90%) •Multiple gland enlargement (10%) • Familial hyperparathyroidism • Carcinoma (<1%) • Familial benign hypercalcemia (FBH)	(due to ↓ Ca2+ in ECF) •Vitamin D deficiency •Low calcium diet •Pregnancy •Lactation •Rickets •Osteomalacia •Chronic renal failure ↓ 1,25(OH) – D3 synthesis
Affects approximately 100,000 patients a year (in the US). Prevalence: 0.1 to 0.3% of the general population. More common in women (1:500) than in men (1:2000). Patients with single adenoma (~90%): minimally invasive surgery.	

Common Causes

Male slides

1- PTH mediated:

- Primary hyperparathyroidism

2- Non-PTH mediated

- Vitamin D intoxication, granulomatous disorders, osteolytic bone metastases, malignancy.
- Parathyroid hormone–related peptide (PTHrP): certain tumors secrete high levels of PTHrP, which causes hypercalcemia of malignancy.

3- Dehydration.

4- Immobility.

5- Medications :

- Lithium, thiazide diuretics.

Hyperparathyroidism (PTH Excess)

Clinical Manifestations

- Nausea, vomiting.
- Anorexia, weight loss, constipation, abdominal pain, peptic ulcer & decreased appetite.
- Lethargy and Fatigue.
- Confusion, stupor, coma.
- Impaired concentration and memory Depression & anxiety.
- Reduced neuromuscular excitability and muscle weakness.
- Easy fatigability and muscle weakness more common in hyperparathyroidism. than other hypercalcemic conditions.
- Cardiac arrhythmias.
- Vascular calcification.
- Shortening of the QT interval.
- Hypercalcemia (↑Ca2+), Hypercalciuria.
- Hypophosphatemia (↓PO-4), Hyperphosphaturia.
- Demineralization of bone forming multiple bone cysts (osteitis fibrosa cystica).
- Broken bones.
- ↑ Alkaline phosphatase.
- CNS depressed and peripheral nervous system depressed.
- Muscle weakness.
- Depressed relaxation of the heart during systole.
- Calcium containing stones in kidney.
- Parathyroid poisoning: Precipitation of calcium in soft tissues occur when Ca2+ > 17 mg/dl → lead to death.

Treatment

Male slides

Severe hypercalcemia:

- **Indications for therapy :**
  a- Symptoms of hypercalcemia.
  b- Plasma [Ca] > 14 mg/dl.
- **Principles of therapy:**
  a- Expand ECF volume.
  b- Increase urinary calcium excretion.
  c- Decrease bone resorption.
- **NS bolus to restore volume; then 100 – 200 ml/hr.**
- **Bisphosphonates (onset 24-48 hrs).**
- **Calcitonin 4 – 8 IU q6-8 hrs (onset immediate, resistance develops in 24-48 hrs).**
- **Surgery for adenoma.**

Hypoparathyroidism

Causes of Hypocalcemia:

- Hypoparathyroidism:
  -Injury to the parathyroid glands (surgery).
  -Autoimmune.
  -Magnesium deficiency.
- PTH resistance (pseudohypoparathyroidism): Normal PTH levels but deficient receptors.
- Vitamin D deficiency or resistance.
- Lack of Iα hydroxylase, no vit D3 activation.
- Other: renal failure, pancreatitis, tumor lysis.

Hypocalcemia Signs:

- Paraesthesia (tingling sensation) around mouth, fingers and toes.
- Muscle cramps, carpopedal spasms.
- **Tetany** can be overt or latent.
- Neuromuscular excitability.
- **Laryngospasm**, stridor and apneas (neonates).
- Seizures – focal or generalised.
- Cardiac rhythm disturbances, delayed cardiac repolarization (prolonged QT interval).
- Chvostek's sign (facial muscle twitch): Tapping the facial nerve as it emerge from the parotid gland in front of the ear → causes contraction of facial muscles.
- Trousseau's sign (carpal spasm): Arresting (stopping) blood flow to the forearm for few minutes (e.g., by sphygmomanometer) → causes flexion at the wrist, thumb and metacarpophalangeal joints.
- Latent hypocalcemia.

Chvostek's sign:

It refers to an abnormal reaction to the stimulation of the facial nerve. When the facial nerve is tapped at the angle of the jaw (i.e. masseter muscle), the facial muscles on the same side of the face will contract momentarily (typically a twitch of the nose or lips) because of hypocalcemia.

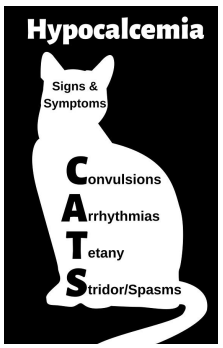
Click here!

Trousseau's sign:

To elicit the sign, a blood pressure cuff is placed around the arm and inflated to a pressure greater than the systolic blood pressure and held in place for 3 min. This will occlude the brachial artery. In the absence of blood flow, the patient's hypocalcemia and subsequent neuromuscular irritability will induce spasm of the muscles of the hand and forearm. The wrist and metacarpophalangeal **joints flex**.

Hypocalcemia Symptoms

- **Tingling** in the lips, fingers, and toes
- Dry hair, brittle nails, and dry, coarse skin
- **Muscle cramps** and pain in the face, hands, legs, and feet
- Cataracts on the eyes
- Malformations of the teeth, including weakened tooth enamel
- Loss of memory
- Headaches
- neuromuscular irritability, numbness, cramps, anxiety
- Tetany, carpopedal spasms
- Severe Hypocalcemia and hyperphosphatemia
- **convulsions**, stridor, dystonia and depression.

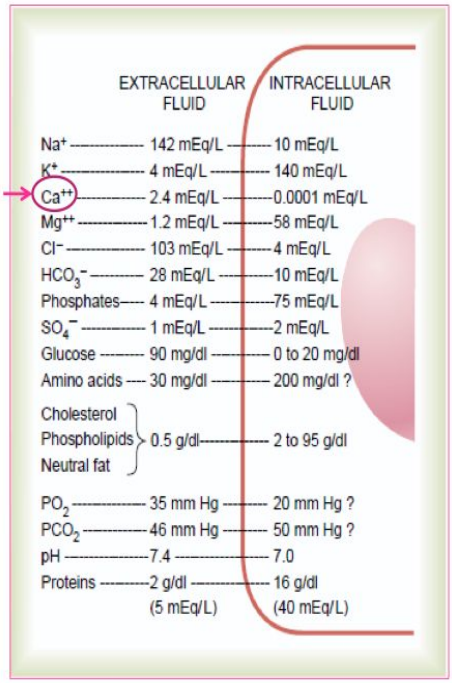


Treatment Calcium carbonate and vitamin D supplements

Pictures were in the lec

Inorganic Constituents of Bone

Constituent	% of Total Body Content Present in Bone
Calcium	99
Phosphate	86
Carbonate	80
Magnesium	50
Sodium	35
Water	9



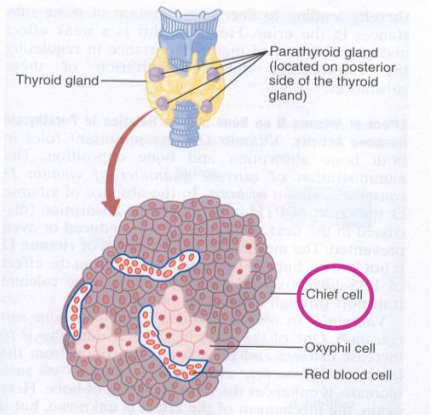
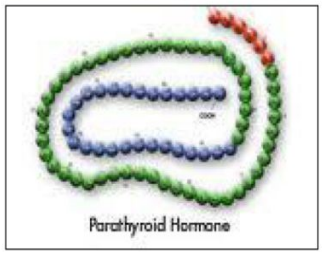
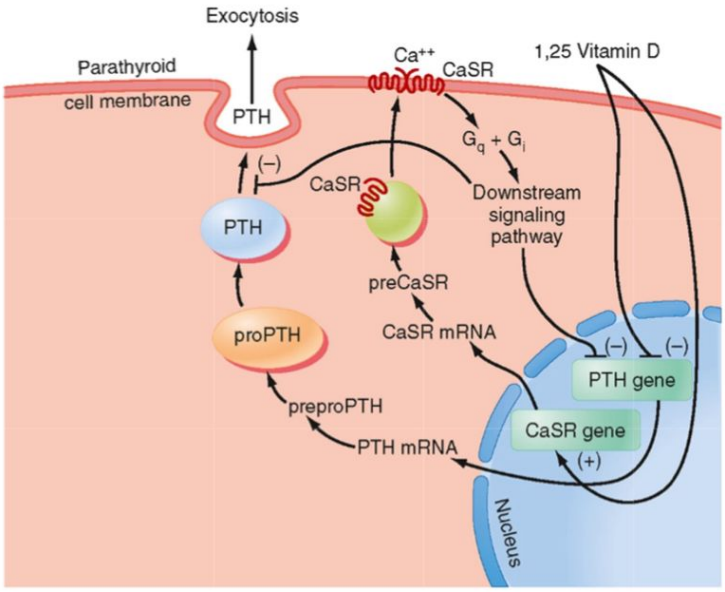
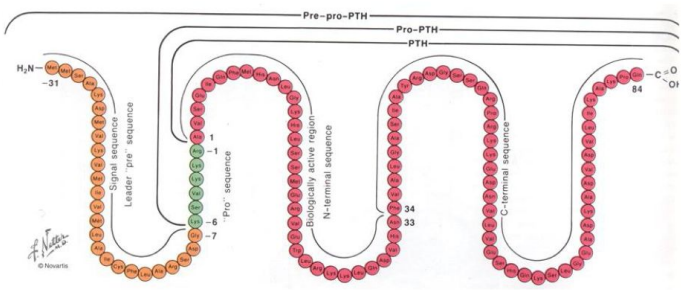


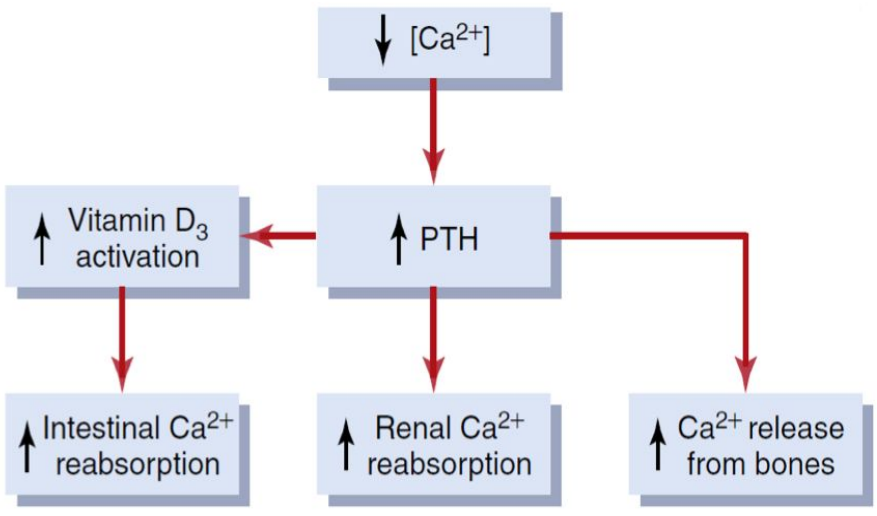
TABLE 36.1 Body Content and Tissue Distribution of Calcium and Phosphorus in a Healthy Adult

	Calcium	Phosphorus
Total Body Content	1,300 g	600 g
Relative Tissue Distribution (% of total body content)		
Bones and teeth	99%	86%
Extracellular fluid	0.1%	0.08%
Intracellular fluid	1.0%	14%









MCQs:

QI: Which of the following may lead to a positive Chvostek's and Trousseau's sign?

A- Hyperthyroidism | B- Hypothyroidism | C- Hyperparathyroidism | D- Hypoparathyroidism

Q2: Which plasma findings are indicative of low plasma calcium levels?

A- Increased PTH | B- Increased Phosphate | C- Increased TH | D- Increased Calcitonin

Q3: A 41 year old woman has hypocalcemia, hyperphosphatemia and decreased urinary phosphate excretion. What is the most likely diagnosis?

A- Vit. D intoxication | B- Primary hyperparathyroidism | C- Hypoparathyroidism | D- Vit. D deficiency

Q4: Which of the following is the physiologically active form of Calcium?

A- Albumin Bound | B- Free form | C- Phosphate Bound | D- Serum carbonate Bound

Q5: If blood calcium levels increase, which one of the following is the main physiological response to reduce it?

A- Decreased calcitonin release | B- Increased activity of Vit. D | C- Decreased PTH release | D- Increased Cortisol

Q6: What causes stimulation of osteoclastic activity?

A- Low levels of 1,25 Vit. D | B- Calcitonin | C- Estrogen | D- PTH

Answers: Q1: D | Q2: D | Q3: C | Q4: B | Q5: C | Q6: D

SAQs:

- QI: Enumerate the forms of calcium in ECF
- Q2: Mention all the actions of PTH on bones
- Q3: List three causes of Hypercalcemia & three manifestations of hypercalcemia

A1: (1) Free form (2) Complexed to anions (3) Bound to plasma proteins

A2: (1) Stimulates the Formation of new osteoclasts (2) Activation of existing osteoclasts (3) Increases Calcium and Phosphate Absorption from the Bone (4) Depression of Osteoblastic activity

A3: Causes: (1) Hyperparathyroidism (2) Vit. D toxicity (3) Thiazides Diuretics Manifestations: (1) Neural depression (2) Kidney stones (3) Cardiac arrhythmias

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