

PATH POWER: THE VITAMIN VAULT



April 2026
ISSUE #1



Fat-soluble v.s. water soluble

Inside The Body:

HOW VITAMINS KEEP YOU HEALTHY

Sources of Vitamins

Functions and Disease



Our parents often tell us to “eat healthy”—to finish our vegetables, drink our milk, and not skip meals. But beyond these familiar reminders lies a deeper question: why does it matter so much? The answer begins with vitamins—tiny, invisible compounds that play massive roles in keeping our bodies functioning.

This magazine explores the fascinating world of vitamins—what they are, how they work, and what happens when our bodies don’t get enough. By understanding the science behind these nutrients, we can better appreciate the importance of a balanced diet and make informed choices about our health. After all, “eating healthy” isn’t just good advice—it’s a foundation for preventing disease and maintaining overall well-being. Also, as a reminder, always consult your physician before taking any vitamin supplements!

YA

Editor-in-Chief

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Vitamins Overview



We get vitamins from our diet since they can't be created by our bodies endogenously. There are two classifications of Vitamins (1):

Fat soluble Vitamins

These vitamins are stored in the body once consumed. They are stored in places like your liver, fatty tissue, and your muscles (2).

Vitamins A, D, E, and K are all fat soluble! (1)

A

D

E

K

Excess > deficiency. More often, patients have an excess amount of this type of vitamin rather than a deficiency due to the fact that it is stored in the body. (3)

Water soluble Vitamins

These vitamins are not stored in our bodies, and any excess that we don't need at the time is expelled through the urine. Therefore, these vitamins must be consumed regularly to ensure that we don't get any deficiencies in our bodies (2).

Vitamin C and the Vitamin B-complex (B1, B2, B3, B5, B6, B7, B9, B12) are all water-soluble! (2)

C

B

Deficiency > excess. More often, patients have a deficiency of this vitamin rather than an excess. due to the fact that they don't stay in the body for very long (3)

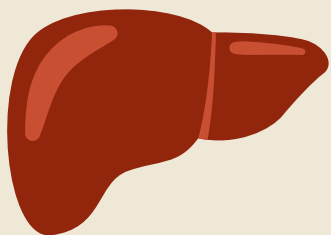
Nutritional Pathology

These disorders arise when there is an excess or a deficiency of nutrients. These deficiencies can either be Primary or Secondary (3)

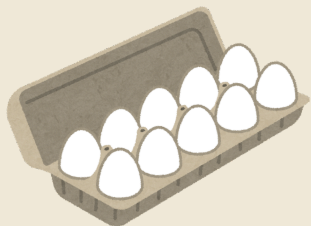
Primary: Due to inadequate dietary intake
Secondary: body has problems with absorption/storage

SOURCES OF VITAMIN A (5, 6)

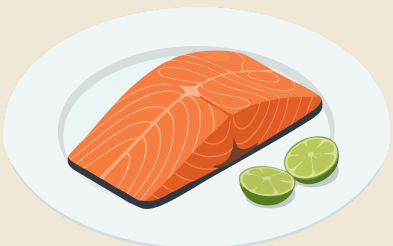
Preformed vitamin A, also known as retinol, is one of the active forms of vitamin A. It is found in animal products (4).



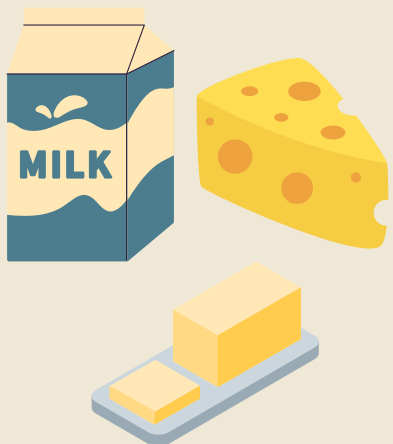
Beef Liver



Eggs



Oily Fish
(i.e salmon)



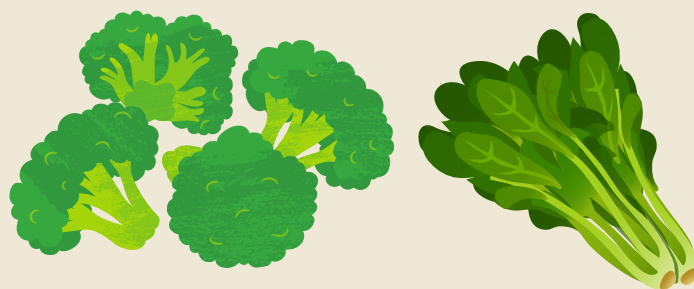
Dairy Products:
Milk
Butter
Cheddar Cheese

Provitamin A, also known as β -carotene, is a precursor to vitamin A. Once consumed, our body converts it into active forms of vitamin A. It is found in many types of colorful vegetables (4).

Yellow and Orange
vegetables like Carrots,
Sweet Potato, and Mangos



Dark leafy green vegetables
like broccoli and spinach



Functions of VITAMIN A



Vitamin A is essential for many physiological processes and has two main functions within the body

Maintains Normal Vision

- Vision depends on vitamin A-derived pigments: rhodopsin in rod cells (for low light) and other pigments in cone cells (for color vision in bright light). One of the active forms of vitamin A is Retinol. Rhodopsin is formed when Retinol → all-trans-retinal → 11-cis-retinal then binds to a protein called opsin (7).
- During dark adaptation, some all-trans-retinal is converted to 11-cis-retinal, but a lot of it is lost, which is why we need constant replenishment of vitamin A (7).

Differentiation and Cell Growth

- Vitamin A plays a significant role in the differentiation and cell growth of mucus-secreting epithelium (7)

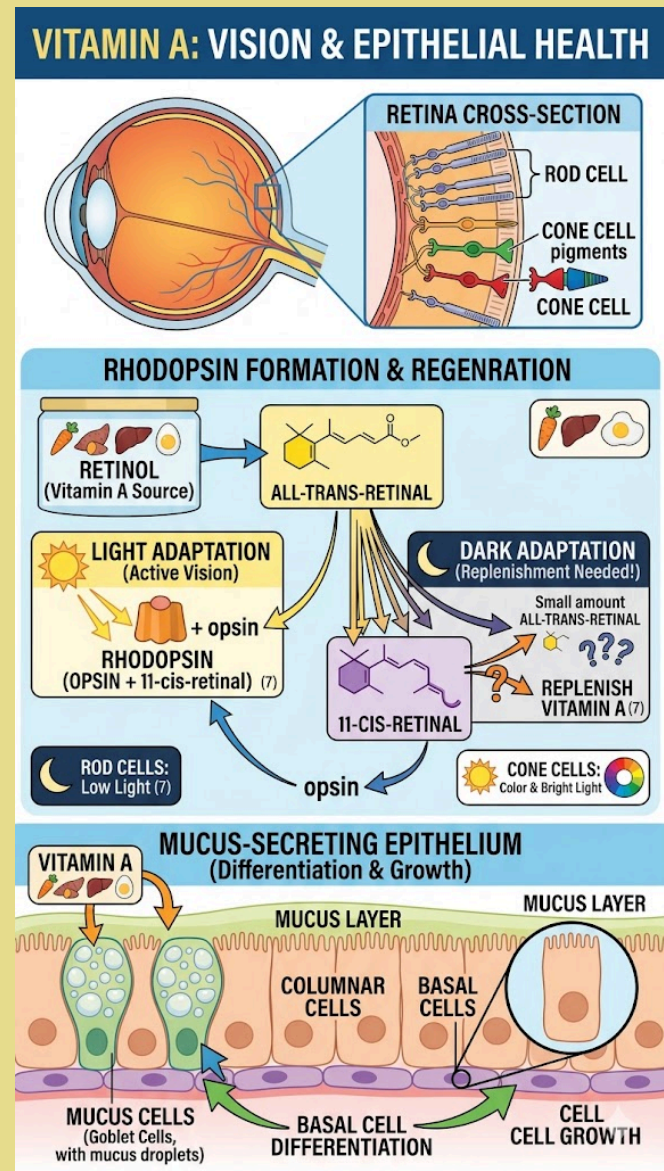
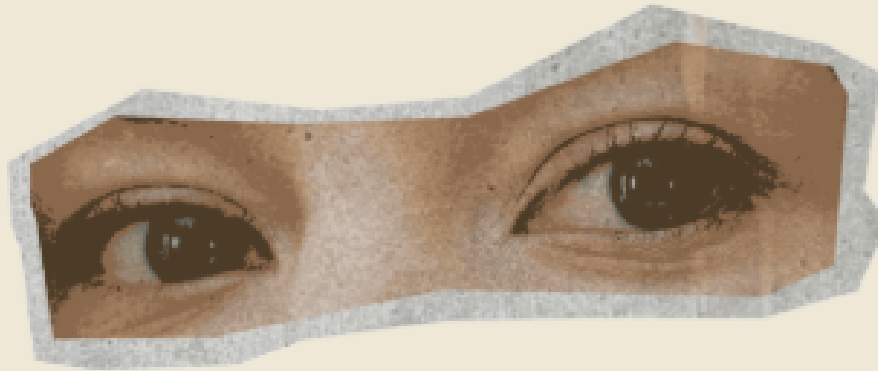


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Vitamin A Deficiency: Night Blindness & Xerophthalmia



Vitamin A Deficiency is the Leading cause of blindness worldwide. Although it is a very preventable disease, unfortunately, it causes irreversible eye blindness (8).

Night Blindness - Early Stage vitamin A deficiency

Early on, Vitamin A deficiency causes night blindness due to the poor regeneration of rhodopsin and other pigments (7, 8)

Xerophthalmia- Later Stage vitamin A deficiency

1. Dryness in the eyes, specifically the conjunctiva and cornea. This occurs due to squamous metaplasia of the epithelial cells in both the conjunctiva and tear ducts. The mucus-secreting epithelium is replaced with keratinized epithelium.
2. Bitot spots. Keratin debris accumulates in the eyes, presenting as small, opaque sheets.
3. Keratomalacia. This is the softening of the Cornea. Leads to an increased vulnerability to ulceration and other bacterial infections.
4. In addition to the ocular epithelium, the lining of the upper respiratory passage and urinary tract is also replaced by keratinized epithelium. In the renal system, this can cause pelvic keratinization, in which keratin debris causes bladder stones.

(7,8)

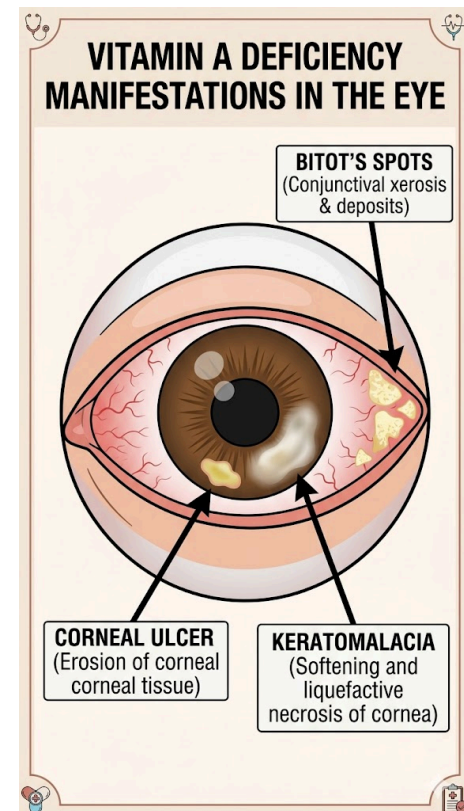
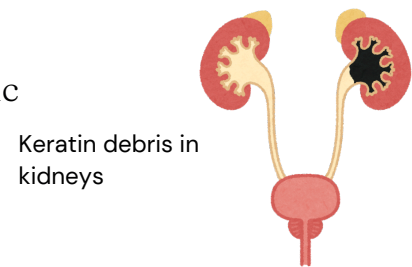


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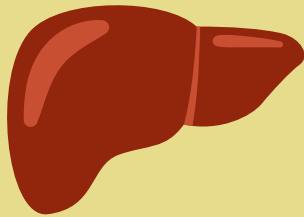


Keratin debris in kidneys

SOURCES OF *VITAMIN D* (9)



Egg Yolks



Beef Liver



Oily Fish
(i.e salmon)

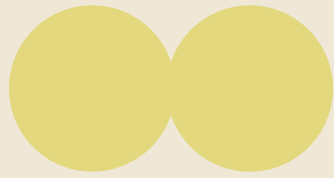


Fortified Milk
and Cereal



UV rays from
the Sun*

* UV is converted into Vitamin D3
all other food sources are Vitamin D2



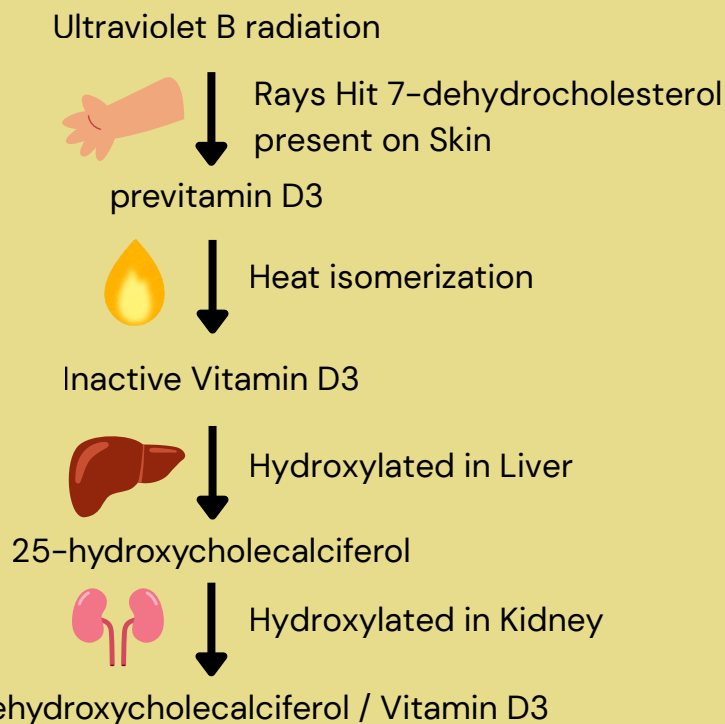
Functions of **VITAMIN D**

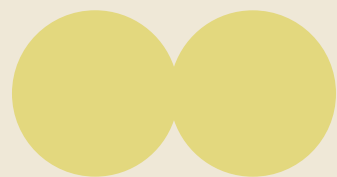


How do we get vitamin D from sunlight?

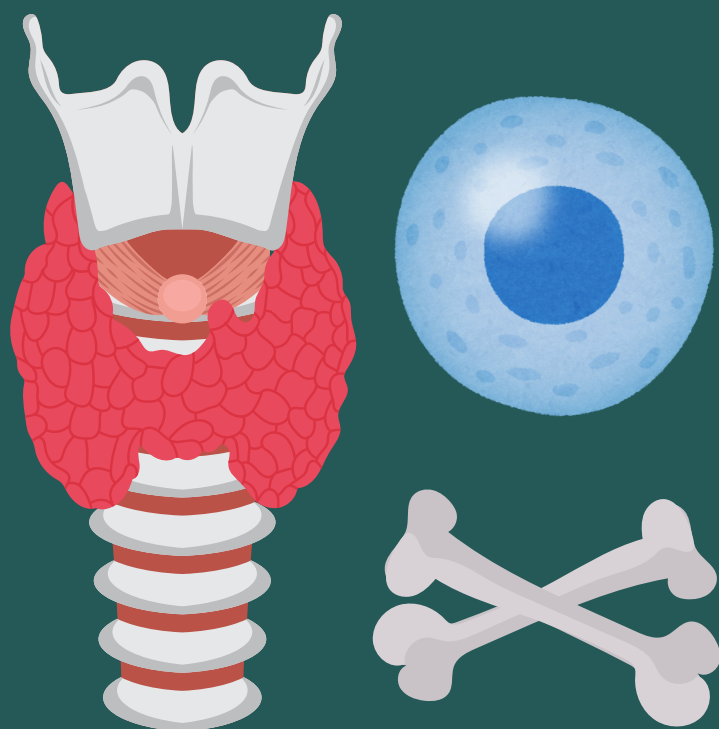
One way we can get vitamin D is from UV rays. Ultraviolet B (UVB) radiation converts 7-dehydrocholesterol in the skin to previtamin D₃. This previtamin D₃ is converted to vitamin D₃ through heat isomerization. (10) At this moment in time, the vitamin D₃ is still inactive. It moves to the liver via the blood, specifically via a carrier protein. When it reaches the liver, it gets hydroxylated into 25-hydroxycholecalciferol, also known as 25(OH)D₃. 25(OH)D₃ produced goes on to the kidneys, where it is hydroxylated again into 1,25-dehydroxycholecalciferol, or 1,25(OH)₂D₃. This is the active form of Vitamin D₃, also known as cholecalciferol (3, 11).

Simple Diagrammatic Depiction





Functions of VITAMIN D Cont.



Vitamin D, also known as the anti-rachitic vitamin, is required for calcium homeostasis.

How is calcium homeostasis achieved?

When blood calcium levels fall below normal, parathyroid hormone (PTH) is released from the parathyroid glands. PTH acts on osteoblasts to increase the expression of RANKL (bone metabolism protein), which activates osteoclasts and promotes bone resorption, releasing calcium into the bloodstream (12). PTH also acts on the kidneys to increase calcium reabsorption and decrease phosphate reabsorption, resulting in increased phosphate excretion in the urine. Additionally, PTH stimulates the kidneys to activate vitamin D, which enhances calcium absorption in the small intestine, particularly in the duodenum. As blood calcium levels rise, calcium and active vitamin D provide negative feedback to reduce PTH secretion, preventing excessive increases in calcium levels (7,13).

REGULATION OF BLOOD CALCIUM (PTH AND VITAMIN D)

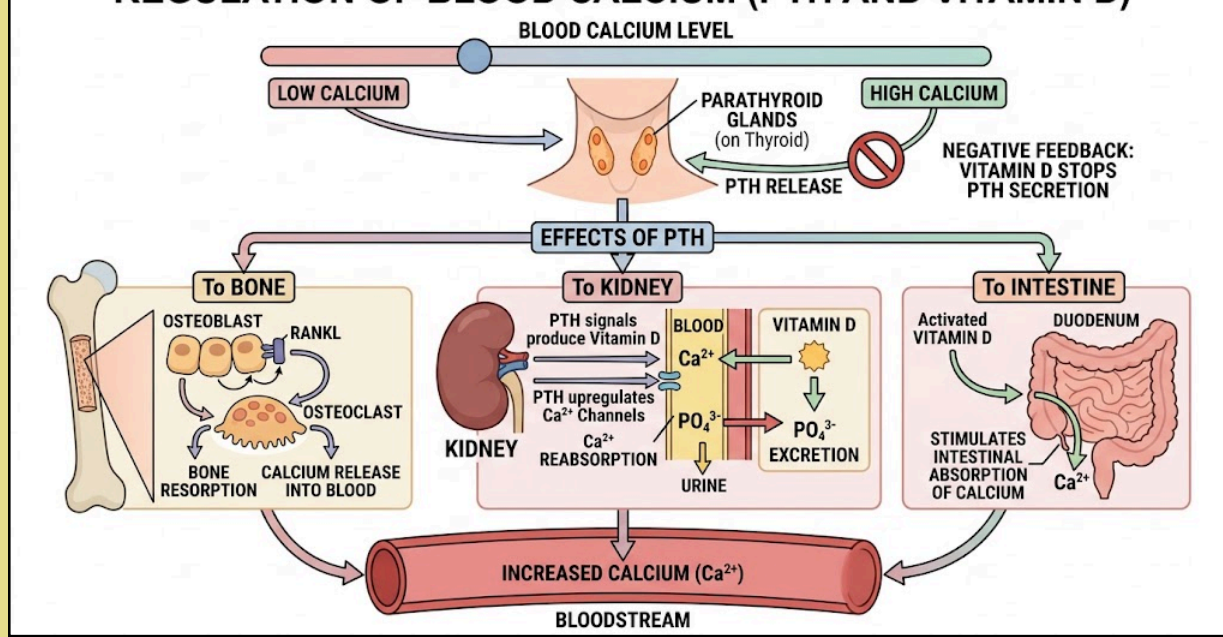
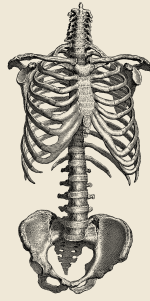


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Vitamin D Deficiency



How is the PTH pathway effected when this is an absence of vitamin D?

In vitamin D deficiency, reduced calcium absorption from the intestine leads to hypocalcemia, which stimulates the release of PTH. PTH attempts to restore calcium levels by activating vitamin D in the kidneys, increasing calcium reabsorption in the kidneys, and promoting bone resorption to release calcium into the bloodstream. At the same time, PTH increases phosphate excretion in the urine, leading to low phosphate levels. Osteoblastic effects also decrease phosphate absorption in the intestine and kidneys. Although normal levels of calcium persist, since there is not enough vitamin D to reduce PTH production, phosphate levels remain low and therefore impair the mineralization of bone (7).

Vitamin D Deficiency Overview

Vitamin D deficiency leads to Rickets in children and Osteomalacia in adults. These may result from diets deficient in vitamin D, but are more likely to be caused by a lack of adequate sunlight. These diseases are more common in northern latitudes because of a lack of exposure to sunlight in those areas. (7)

Rickets and Osteomalacia: the lack of vitamin D in adults affects bone remodeling. Newly placed osteoid-matrix, placed by osteoblasts, is badly mineralized. Leading to an excess in unmineralized matrix.

Rickets

In children, vitamin D deficiency has much more lasting effects.

- Failure of endochondral ossification; Overgrowth of epiphyseal cartilage due to inadequate calcification and failure of the cartilage cells to mature (3, 7).
- Loss of structural rigidity leads to skeletal deformations
 - Excess unmineralized bone leads to frontal bossing (more prominent forehead) and a square-shaped skull
 - Overgrowth of cartilage at the rib joints forms a “rachitic rosary”
 - Weakened ribs are pulled inward, causing pigeon breast deformity (protruding sternum)
 - These issues also affect the spine, pelvis, and legs, leading to bowed legs and lumbar lordosis (curve of the lower spine) (3, 7)

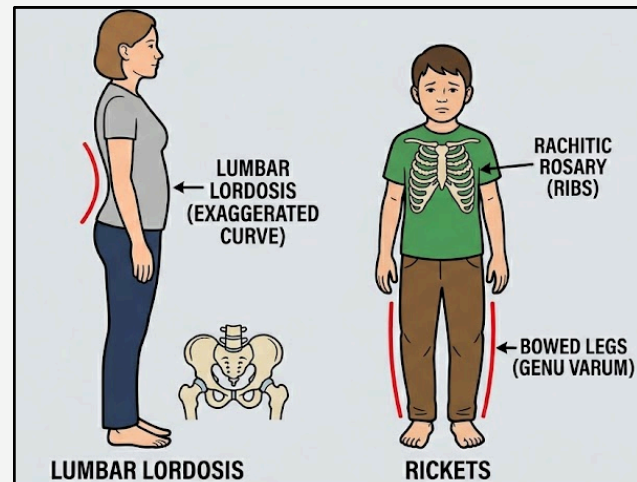


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Osteomalacia

This leads to bones that are vulnerable to fractures. Much less severe than rickets, since this happens after epiphyseal closure (3,7)



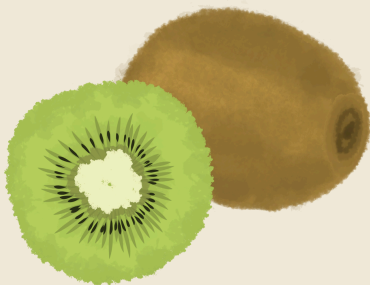
SOURCES OF VITAMIN C (14)



Citrus Fruits



Red and Green
Peppers



Kiwi

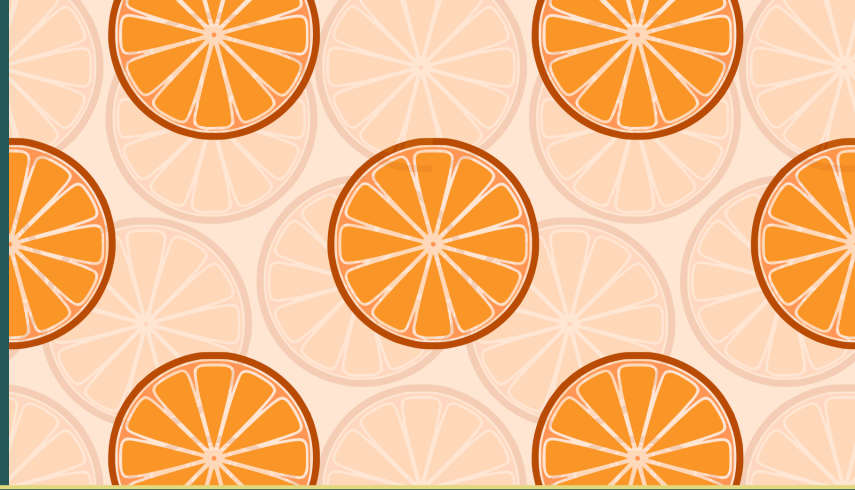


Broccoli



Orange Juice
Grapefruit Juice
Tomato Juice

FUNCTIONS OF VITAMIN C



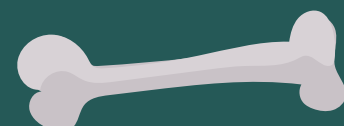
MAIN FUNCTION

Vitamin C is very important in connective tissue healing and the formation of collagen. It is a cofactor for prolyl hydroxylase and lysyl hydroxylase. These enzymes catalyze the hydroxylation of proline and lysine residues of procollagen. This allows proper folding of stable collagen triple-helix formation (15).

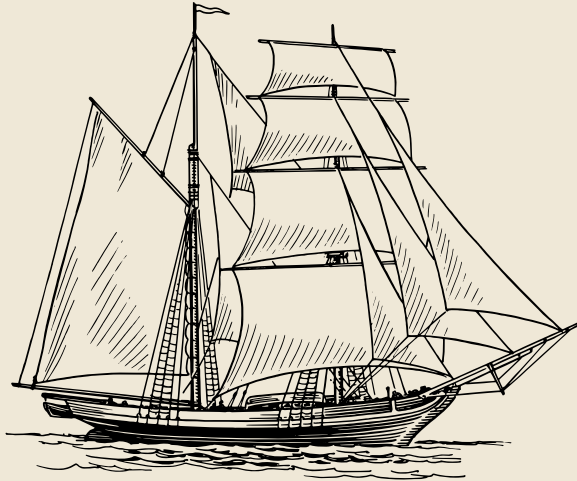


ADDITIONAL FUNCTIONS

- Vitamin C is an antioxidant, as it destroys reactive oxygen species responsible for cell apoptosis during the inflammatory phase (15).
- Vitamin C facilitates iron uptake in the gastrointestinal tract (8).
- important for the formation of the intercellular ground substance of bone and dentin formation (15).



Vitamin C deficiency: **SCURVY**



Vitamin C deficiency Overview

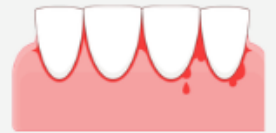
- Scurvy is caused by vitamin C deficiency. The lack of vitamin C causes impaired collagen formation, which leads to defective collagen all over the body
- Defective collagen surrounding blood vessels can lead to Hemorrhages, bruising, and poor wound healing in many areas of the body, such as in the skin, joints, and teeth
- Children with scurvy experience musculoskeletal signs and symptoms from poor bone formation due to inadequate synthesis of osteoid. Most complications occur in the joints.
- Scurvy is rapidly reversed in days after receiving vitamin C supplementation if treated early

(3,8)

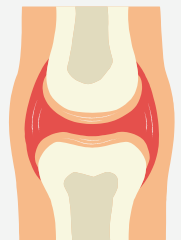
Fun Facts!

- In the 1800s, sailors were called “limeys” because they used to consume lots of citrus, particularly limes, when travelling on long voyages (3).
- Most animals can synthesize their own Vitamin C, but unfortunately, we can't so that is why we need to eat a balanced diet! (3)

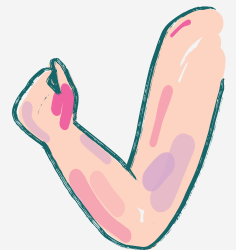
Common Symptoms



bleeding gums



poor joint healing

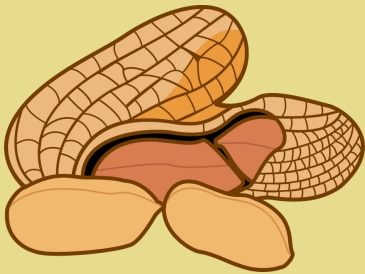


Easy bleeding and bruising in skin

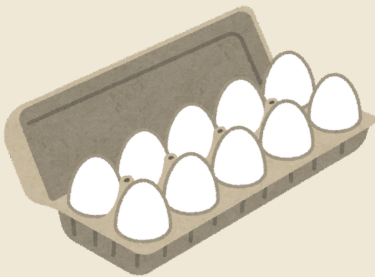
SOURCES OF *VITAMIN B9* (16)



Dark Leafy
Vegetables



Peanuts



Eggs

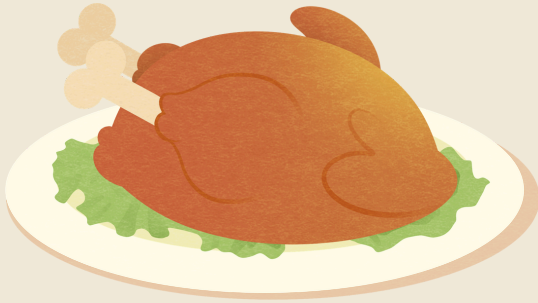


Beans



Whole grains

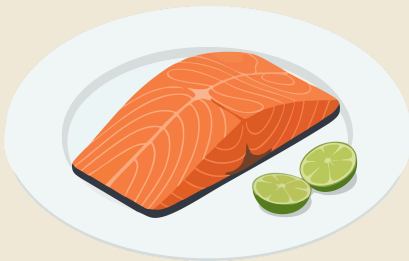
SOURCES OF VITAMIN B12 (17)



Chicken



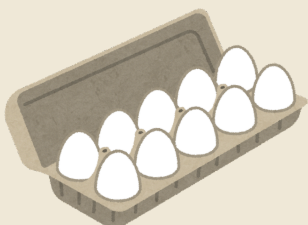
Red meat



Fish



Milk



Eggs

FUNCTIONS OF VITAMIN B



The vitamins B9 and B12 share some similar functions, but each have distinct their own distinct functions

VITAMIN B9

- Essential for rapid cell division during fetal development (18)

VITAMIN B12

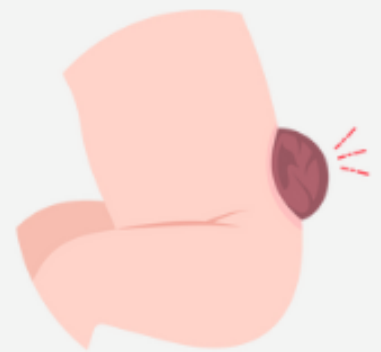
- Essential for the formation of DNA (3, 18, 19)
- Required for red blood cell formation (3, 18, 19)
- Neurological function (maintaining the nervous system and myelin sheath) (8)
- Epithelial cell maturation (8)

Vitamin B9 deficiency: ***SPINA BIFIDA***



Neural Tube defects

Neural tube defects, specifically Spina bifida. Caused by a folate deficiency during the first few weeks of pregnancy. There are many types of spina bifida; we will focus on myelomeningocele. It is a more serious type of spina bifida. The spinal canal is open throughout many vertebrae in the lower back. The spinal cord's protective covering and spinal nerve push through this opening at birth, forming a sac. This causes important tissues and nerves to be exposed, making the child more susceptible to infections, and causes loss of movement in the legs. Also causes bowel and bladder problems (20).



Myelomeningocele, a type of spina bifida affecting newborns

Megaloblastic anemia

Individuals with a deficiency in vitamin B9 (folate) often develop megaloblastic anemia. This condition is characterized by a reduced number of red blood cells, while the cells that are present are macrocytic (larger than normal) and normochromic (normal in color). The underlying cause is impaired DNA synthesis, which disrupts normal cell division. As a result, cells are unable to divide properly and instead grow larger, leading to the formation of these abnormally large red blood cells (3,8).

Fun Fact!

Initiatives in 1998 led to the fortification of foods with folic acid, which halved the prevalence of this disease amongst newborns (3). The neural tube develops often before an XX individual even realizes she is pregnant, so menstruating XX individuals must get the B9 that they need to keep the child healthy .



Vitamin B12 Deficiency



Vitamin B12 deficiency can occur in two different ways:

Decreased intake (primary disease)

It is very rare to obtain a B12 deficiency from decreased intake. However, since B12 comes from foods of animal origin, typically, vegetarians can suffer from this disease. Older people and alcoholics can also suffer from this disease (8).

Impaired absorption (secondary disease)

- Intrinsic factor deficiency. Intrinsic factor is a glycoprotein produced by parietal cells in the stomach, essential for the absorption of vitamin B12 (3, 21).
- Pernicious anemia: an autoimmune T-cell and antibody-mediated condition in which antibodies attack cells in the mucosal lining of your stomach and nerve cells (gastric atrophy). Causes lack of intrinsic factor and achlorhydria (no stomach acid to help with breakdown, which causes bad absorption of B12) (22).
- Other inferences with absorption, taking a protein pump inhibitor medicine, being an alcoholic, and having a diffuse intestinal disease such as Crohn's disease (8)

Clinical Signs of Vitamin B12 deficiency

Megaloblastic anemia

Individuals with a deficiency in vitamin B9 (folate) often develop megaloblastic anemia. This condition is characterized by a reduced number of red blood cells, while the cells that are present are macrocytic (larger than normal) and normochromic (normal in color). The underlying cause is impaired DNA synthesis, which disrupts normal cell division. As a result, cells are unable to divide properly and instead grow larger, leading to the formation of these abnormally large red blood cells (3,8).

Spinal Cord Degeneration

Spinal cord degeneration can occur from B12 deficiency. Diffuse demyelination and axonal loss, particularly in the white matter of the spinal cord, causes Paresthesia (tingling), and numbness of toes and fingers; ataxia (loss of bodily movements), and later on paraplegia (8)



Other symptoms

Atrophy and ulceration of oral mucosa caused by poorly developed epithelium (3)



Test Your Knowledge

1.
Which of the following best describes fat-soluble vitamins?
A. Rapidly excreted in urine
B. Stored in liver and adipose tissue
C. Only obtained from plant sources
D. Rarely absorbed into tissues
2.
Which of the following vitamins is water-soluble?
A. Vitamin A
B. Vitamin D
C. Vitamin C
D. Vitamin K
3.
A primary nutritional deficiency is best defined as:
A. A defect in nutrient absorption
B. Excess nutrient intake
C. Inadequate dietary intake
D. Genetic enzyme deficiency
4.
Which form of vitamin A can be used immediately by the body?
A. β -carotene
B. Retinol
C. Retinoic acid
D. Retinal
5.
Night blindness in vitamin A deficiency is primarily due to:
A. Loss of cone cells
B. Decreased melanin production
C. Impaired rhodopsin regeneration
D. Increased intraocular pressure
6.
Which of the following is a hallmark of xerophthalmia?
A. Increased tear production
B. Keratinization of ocular epithelium
C. Retinal detachment
D. Increased vascularization
7.
Bitot spots are caused by:
A. Calcium deposits
B. Bacterial infection
C. Keratin debris accumulation
D. Lipid accumulation
8.
Vitamin D is essential for:
A. DNA replication
B. Collagen synthesis
C. Calcium homeostasis
D. Neurotransmitter release
9.
Parathyroid hormone (PTH) increases blood calcium by:
A. Inhibiting osteoclasts
B. Increasing phosphate reabsorption
C. Stimulating bone resorption
D. Decreasing vitamin D activation
10.
Where is vitamin D activated to its final form?
A. Skin
B. Liver
C. Kidneys
D. Small intestine

Answer Key

1. B
2. C
3. C
4. B
5. C
6. B
7. C
8. C
9. C
10. C

Test Your Knowledge

11. Vitamin D deficiency in children leads to:
 - A. Osteoporosis
 - B. Osteomalacia
 - C. Rickets
 - D. Scurvy
12. The "rachitic rosary" is caused by:
 - A. Bone fractures
 - B. Overgrowth of cartilage at rib joints
 - C. Calcium deposits in arteries
 - D. Muscle wasting
13. Which of the following best describes osteomalacia?
 - A. Excess bone formation
 - B. Poor mineralization of bone matrix
 - C. Increased cartilage formation
 - D. Bone infection
14. Vitamin C is a cofactor for enzymes involved in:
 - A. DNA replication
 - B. Lipid metabolism
 - C. Collagen synthesis
 - D. Glycolysis
15. Scurvy results primarily from:
 - A. Impaired iron absorption
 - B. Defective collagen formation
 - C. Excess oxidative stress
 - D. Decreased calcium levels
16. Which symptom is most associated with vitamin C deficiency?
 - A. Night blindness
 - B. Poor wound healing
 - C. Bone deformities
 - D. Neurological deficits
17. Vitamin B9 (folate) is essential for:
 - A. Lipid digestion
 - B. DNA synthesis
 - C. Calcium absorption
 - D. Vision
18. A deficiency in vitamin B9 during pregnancy can cause:
 - A. Osteomalacia
 - B. Scurvy
 - C. Spina bifida
 - D. Pernicious anemia
19. Vitamin B12 absorption requires:
 - A. Bile salts
 - B. Intrinsic factor
 - C. Vitamin D
 - D. Pancreatic enzymes
20. Megaloblastic anemia in vitamin B12 deficiency is characterized by:
 - A. Small, pale RBCs
 - B. Large, normal-colored RBCs
 - C. Increased platelet count
 - D. Decreased hemoglobin only

Answer Key

11. C
12. B
13. B
14. C
15. B
16. B
17. B
18. C
19. B
20. B

Case Study

A 68-year-old XY individual presents to his physician with complaints of fatigue, numbness and tingling in his hands and feet, and difficulty maintaining balance while walking. He also reports a sore tongue and occasional mouth ulcers.

His diet consists mainly of tea, toast, and small portions of vegetables, with very little meat or dairy. He has a history of chronic gastritis and has been taking proton pump inhibitors for several years.

On physical examination:

Pale skin
Smooth, inflamed tongue (glossitis)
Decreased vibration sensation in the lower limbs

Laboratory findings:

Low red blood cell count
Enlarged red blood cells (macrocytic), but normal hemoglobin concentration per cell (normochromic)
Further testing reveals the presence of antibodies against gastric parietal cells.



Questions (Answer in full sentences)

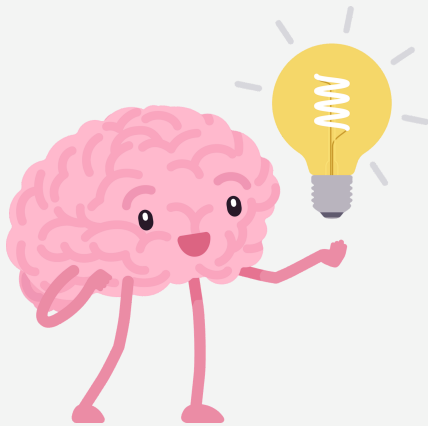
1. What is the most likely diagnosis?
2. What vitamin deficiency is responsible for this condition?
3. Is this a primary or secondary nutritional deficiency? Explain why.
4. What is the role of intrinsic factor in this condition?
5. Why does this deficiency lead to megaloblastic anemia?
6. What explains the patient's neurological symptoms?
7. How does chronic use of proton pump inhibitors contribute to this condition?
8. Why are vegetarians or individuals with low animal product intake at higher risk for this deficiency?
9. What is one effective treatment for this condition?
10. What could happen if this condition is left untreated long-term?



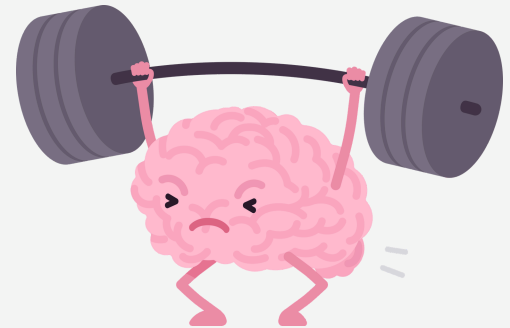
Case Study

Answer Key

1. Pernicious anemia
2. Vitamin B12 deficiency
3. Secondary nutritional deficiency
4. Intrinsic factor enables vitamin B12 absorption in the ileum
5. Impaired DNA synthesis leads to enlarged (megaloblastic) red blood cells
6. Demyelination of neurons due to vitamin B12 deficiency
7. Reduced stomach acid impairs vitamin B12 release and absorption
8. Vitamin B12 is primarily found in animal products
9. Vitamin B12 injections (intramuscular)
10. Permanent neurological damage and worsening anemia



Mnemonics



1. Fat-soluble vitamins

Mnemonic: "ADEK stays"

A, D, E, K = fat-soluble

"stays" → stored in the body (liver, fat)

3. Rickets features

Mnemonic: "BRaVe Pigeon"

B = Bowed legs

R = Rachitic rosary

V = Vertebral issues (lordosis)

Pigeon = Pigeon chest

2. Water-soluble vitamins

Mnemonic: "B and C = Be Cleared"

B-complex + C

"cleared" → excreted in urine → need regular intake

4. Vitamin C deficiency (Scurvy)

Mnemonic: "4 H's of Scurvy"

Hemorrhage

Hematoma (bruising)

Hair/skin issues

Healing impaired

5. Vitamin A deficiency symptoms (eye-related)

Mnemonic: "NB-KK" (Night, Bitot, Keratin, Keratomalacia)

N = Night blindness

B = Bitot spots

K = Keratinization (xerophthalmia)

K = Keratomalacia



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