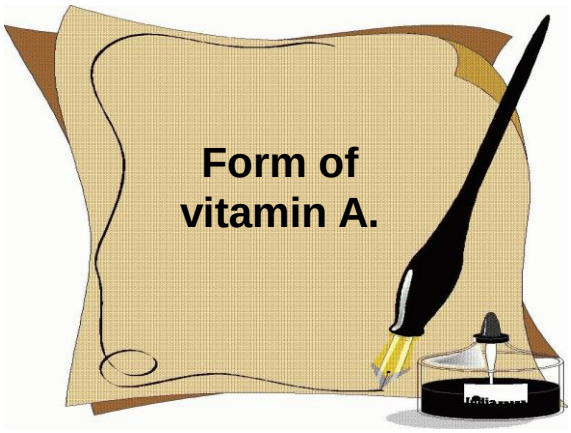

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Form of vitamin A.

Vitamin A1

- ❑Other names: retinol
- ❑ What is the meaning by retinol?
- ❑Retinal: is an unsaturated 20-carbon cyclic alcohol.
- ❑It consists of β-ionone (trimethylcyclohexene) nucleus and an unsaturated side chain.
- ❑Five conjugated double bonds are present in the retinol molecule, including the double bond in the β-ionone ring which is in conjugation with those in side chain.
- ❑Retinol = alcohol form of vitamin A and when alcohol group Replaced by aldehyde group Retinal is formed and alcohol group Replaced by acid group retinic acid is formed.

Vitamin A2

- ❑It has an additional double bond between C-3 and C-4 of the ring.
- ❑It is found in the fresh-water fish.
- ❑It has less activity in mammals and birds than retinol.

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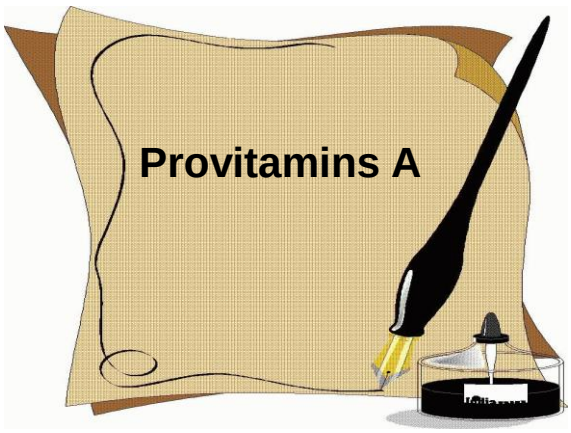

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Provitamin A.

Definition

These compounds which are not themselves vitamins and which function as vitamin A only after undergoing a chemical change. After their ingestion with the diet these compounds are converted in the cells of the intestinal wall to vitamin A.

Members

Carotene — Cryptoxanthine

Carotene

Occur as orange yellow pigments mainly in green leaves .

There are three types :

1 .α- carotene 2 -β-carotene 3- γ-carotene

Vitamin A activity of β-carotene is substantially greater than that of other carotenoids (only one molecule of vitamin A is formed from one molecule of β-carotene .(

α-Carotene and γ-carotene are transformed into retinol with half the efficiency of β-carotene .

Cryptoxanthine

- It is a pigment of yellow maize.
- It has the same provitamine A activity as α-and γ- carotene.

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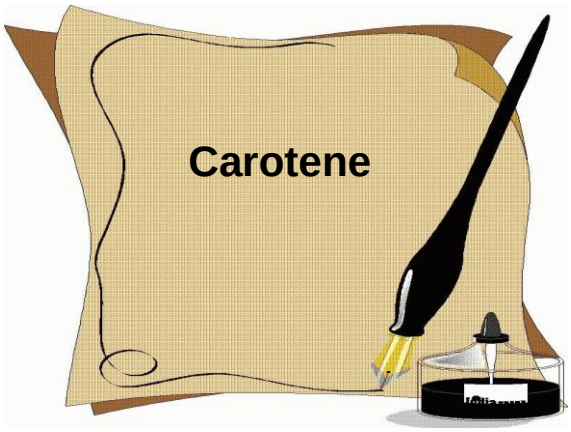

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Carotene

Definition

It is the provitamin A and after their ingestion with the diet these compounds are converted in the cells of the intestinal wall to vitamin A.

Members

α- carotene — β-carotene — γ-carotene

N.B

- Vitamin A activity of β-carotene is substantially greater than that of other carotenoids (only one molecule of vitamin A is formed from one molecule of β-carotene .(
- α-Carotene and γ-carotene are transformed into retinol with half the efficiency of β-carotene .

Conversion of β-carotene

- It takes place in the intestinal mucosa .
- The conversion of β-carotene to retinol needs two enzymes : Carotene- 15,15dioxygenae. And retinaldehyde reductase

Absorption of carotene

- In some species, such as the rat, pig, goat sheep, rabbit, buffalo and dog, the all carotene is cleaved in the intestine .
- In humans, cattle, horses and carp, significant amounts of carotene can be absorbed. The absorbed carotene can be stored in the liver and fatty tissues. Hence, these animals have a yellow body and milk fat .

N.B:The animals do not absorb carotene have white fat .

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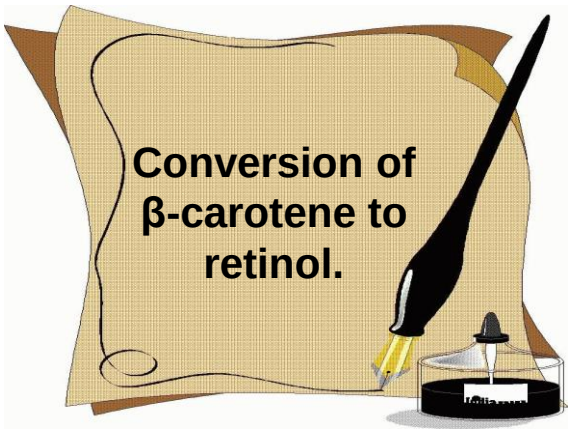

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Conversion of β-carotene to retinol.

Occurrence	❑It takes place in the intestinal mucosa
Enzymes needed	❑The conversion of β-carotene to retinol needs two enzymes: •Carotene- 15, 15-dioxygenae. •Retinaldehyde reductase.
Mechanism	❑Carotene- 15, 15-dioxygenae catalyzes the cleavage of β-carotene at the central double bond to produce two molecules of retinaldehyde and retinaldehyde reductase reduces the retinaldehyde to retinol.
N.B	❑ Enzyme found in many vertebrates. ❑ Enzyme is not present in cat or mink.

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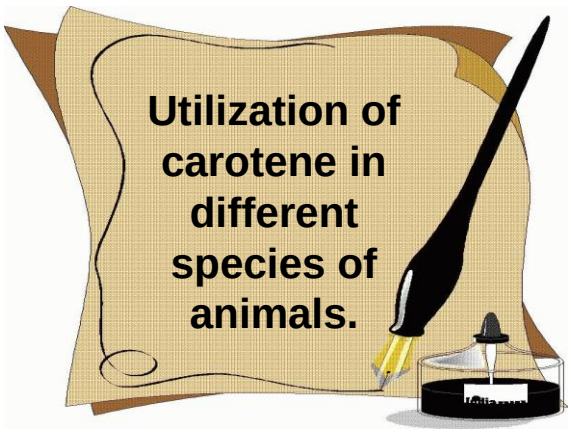

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Utilization of carotene in different species of animals.

In rat, pig, goat, sheep, rabbit and dog

- ❑Almost all of the carotene absorbed into the intestinal mucosa is cleaved to retinol .
- ❑Very little β-carotene is absorbed into circulation .

In rat, pig, goat, sheep, rabbit and dog

- ❑Some carotenoids escape conversion and are taken up intact into the circulation.
- ❑Cattle preferentially absorb carotene.
- ❑Birds absorb mainly xanthophylls (oxycarotenoids) which do not posses vitamin A activity.
- ❑The absorbed carotenoids are transported in the blood plasma, carried to the tissues and contribute to the pigmentation of meat, eggs and milk.
- ❑The animal species capable of absorbing carotene have yellow body and milk fat where as species that do not absorb carotene have white fat and no detectable carotene in blood or milk.

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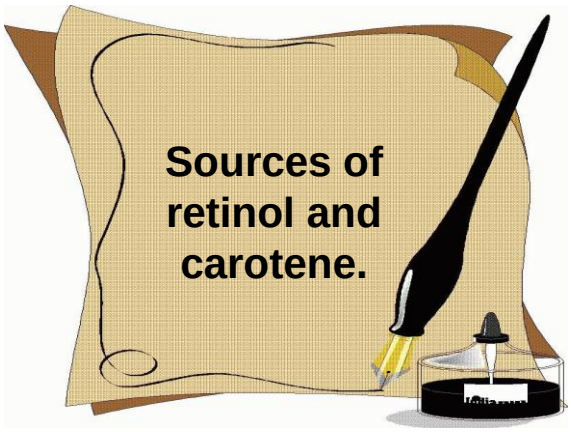

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Sources of retinol and carotene.

Vitamin A

Fish liver oils (vitamin occur largely in esterified form) e.g. cod-fish and shark.

Carotenes

1- Fresh green corps.
2- Hay.
3- Silage products.
4- yellow maize and it's by products.

N.B

- ❑Carotenes in fresh green corps, hay and silage are the most common dietary sources of vitamin A in ruminants.
- ❑The concentration of carotene in green crops fluctuates between 200 and 300 mg/g dry mater.
- ❑The carotene content decreases with age mainly due to an increase in the stalk/leaf ratio.
- ❑The concentration of vitamin A precursors in yellow maize is lower than that found in the dry matter of green crops.

Cryptoxanthine

yellow maize and it's by products.

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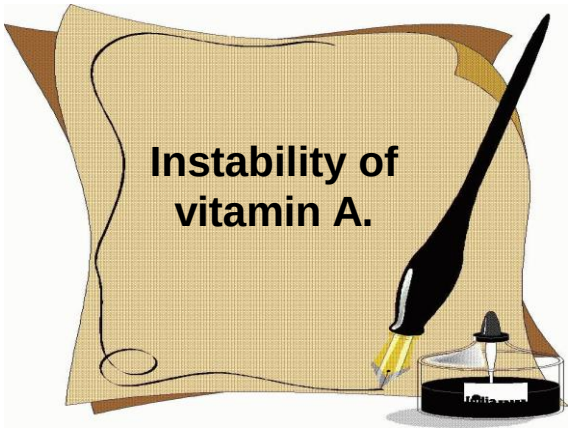

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Instability of vitamin A.

- Retinal is readily oxidizable owing to the presence of the conjugated double bond system .
- Retinol is particularly unstable on exposure to light or heat, especially in the presence of heavy metal ions and water .
- Esterified vitamin A is more stable than retinol .
- Trace metals present in feed mixtures and hydroperoxides of fatty acids present in the lipid fraction of animal feeds have a deleterious effect on vitamin A stability, the unsaturated easily oxidized fats being the most destructive .

N.B :

- A stable preparation of vitamin A intensively used in animal feeding involves coating.
- In this process the vit. A is embedded, together with an antioxidant such as ethoxyquine or BHT, in a gelatin-carbohydrate matrix to form a beadle.
- In the gastrointestinal tract the beadle softens disintegrates as the matrix is digested and releases the vitamin A which is thus fully biologically available .
- This form of vitamin A preparation when added to feed mixtures, results in less than 3-4% loss of vitamin A per month on storage of animal feeds under low moisture conditions .
- Pelleting feeds destabilizes the gelatin beadle some what and results in greater loss of vitamin A on storage .

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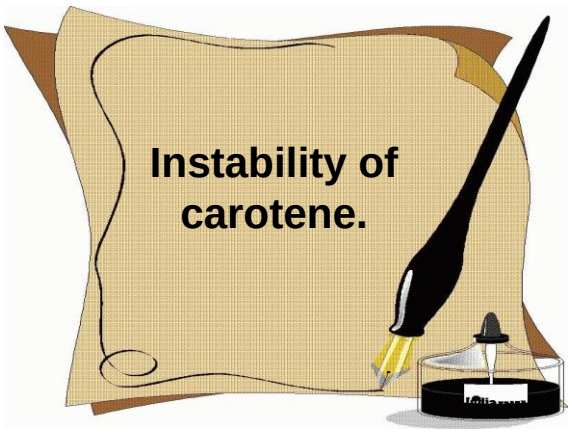

١٥

Instability of carotene.

- ❑Carotene is readily oxidizable owing to the presence of the conjugated double bond system .
- ❑Carotenoids in plant material are subject to enzymatic oxidation through the lipoxygenase system, and to non-enzymatic processes accelerated by direct sunlight.
- ❑Slow drying of forages in the sun-for hay preparation may cause an up to 80% destruction of carotene.
- ❑High-temperature short-term heat treatment (artificial dehydration of green crops) deactivates the lipoxygenase and enables conservation of the carotene.
- ❑Carotene is usually well preserved in silage, with total losses of about 10% typical of ensiling processes. Wilting prior to ensiling causes grater carotene losses of up to 30%.

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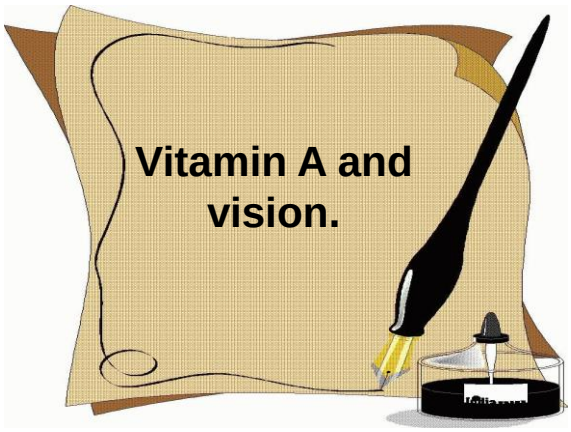

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Vitamin A and vision.

Function

- ❑Rhodopsin formation which is essential for transmission of light stimuli to the brain .
- ❑It is essential for healthy of the epithelium of the eye.

Mechanisms

<u>Formation of rhodopsin andtransmission of light stimuli to the brain .</u>

- ❑11-Cis-Retinal is combined with the protein opsin in both rods and cones in the light sensitive pigments.
- ❑ This pigment is then bleached by light isomerizing the cis-retinal to trans-retinal which is bound less strongly to the visual pigment, and trans-retinal is accordingly released.
- ❑The isomerization of cis-retinal triggers a never impulse, and the energy derived from this reaction is transferred to the brain via the optic nerve and the seeing of color is registered.
- ❑In the dark, the pigment is regenerated; this requires that transretinal is isomerized to the cis form, which occurs following reduction to retinol.

<u>healthy of the epithelium of the eye</u>

- ❑ It prevent keratinization of corneal epithelium.

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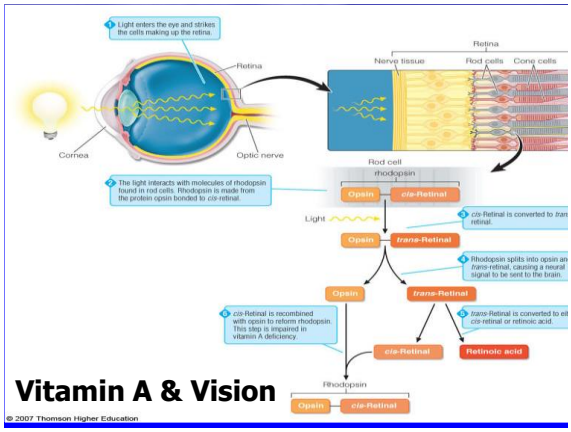

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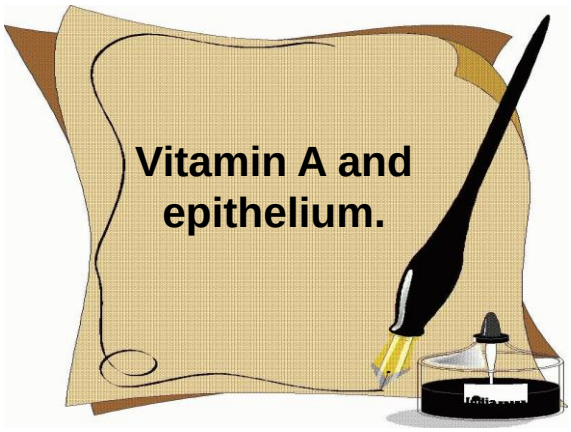

٢٠

Vitamin A and epithelium.

Function

- ❑Vitamin A is required form maintenance of epithelial cells lining of all those canals and cavities of the body which communicate with the external air such as the alimentary, respiratory and the genitor-urinary treat as well as the corneal epithelium and the soft tissues around the eyes.
- ❑It is essential for differentiation of mucus secreting cells.

Mechanisms

Stabilizing the membrane

- ❑Vitamin A penetrates lipoprotein membranes and at optimum levels, may act as a cross-linkage agent between lipid and protein, thus stabilizing the membrane.

differentiation of mucus secreting cells

- ❑Maintaining cell differentiation for mucus-secretion through two possible mechanisms:
  - •Action of the cell nucleus, by altering expression of genetic information.
  - •Extranuclear process, for example, the synthesis of cell surface glycoprotein.

Vitamin A is necessary for the formation of large molecules containing glucosamine. These are the mucopolysaccharides occurring in almost all tissues of mammal organisms but principally in the mucus-secreting epithelial and in the extra cellular matrix of cartilage, mainly as chondroitin sulfate.

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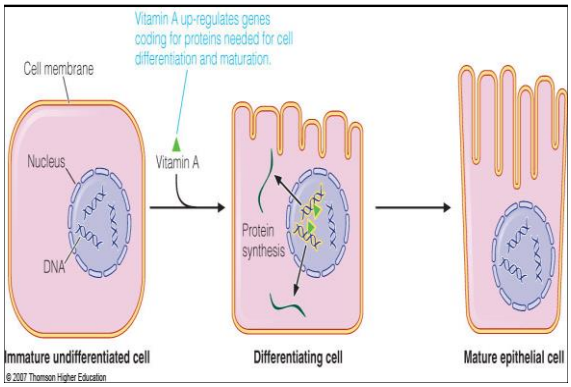
Vitamin A & Cell Differentiation



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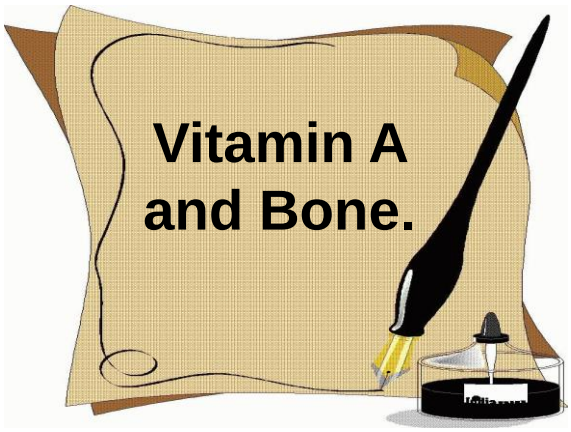

٢٣

Vitamin A and bone.

Function

- ❑Vitamin A has a role in the normal development of bone .

Mechanisms

- ❑ Vitamin A control exercised over the activity of osteoclasts and osteoblasts of the epithelial cartilage.
- ❑Vitamin A has a role on the biosynthesis of mucopolysaccharides (glycoproteins) which are major constituents of organic matter in bones.

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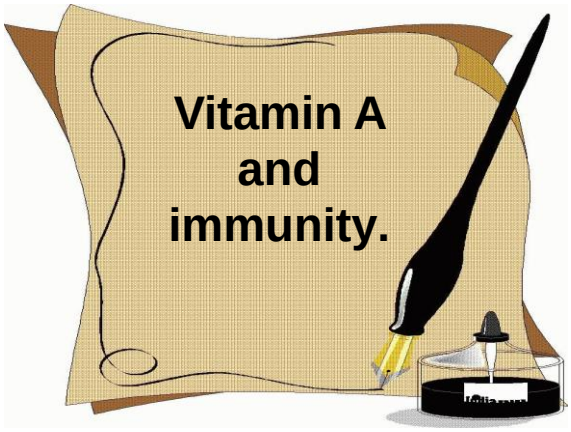

٢٥

Vitamin A and immunity.

Function

- Vitamin A is required for the normal functioning of the immune system.

Mechanisms

- Animals deficient in vitamin A will show increased frequency and severity of bacterial, protozoal and viral infections as well as other disease conditions.
- Vitamin A is required for the normal functioning of the immune system. Retinol and its derivatives are required for the maintenance of the skin and mucosal cells that function as a barrier against infection, and are also required for the development of white blood cells that play a critical role in mounding an immune response. For example, the activation of T-cell lymphocytes requires the binding of the RAR receptor to retinoic acid. A deficiency in vitamin A can cause the mucosal membranes to atrophy, decreasing resistance to infection, and can increase the severity of infection. As such, vitamin A deficiency can be regarded as a nutritionally acquired immunodeficiency disease.
- Part of disease resistance, as a function of vitamin A is related to normal functioning of the cilia and normal functioning of the adrenal gland for production of corticosteroids needed to combat diseases.
- Vitamin A deficiency causing a reduced immune response.

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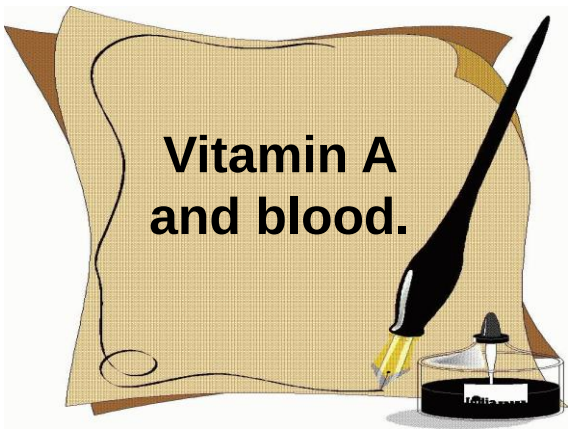

٢٧

Vitamin A and blood.

Function

- ❑Vitamin A has a role in the production of rdd blood cells and function of Hb.

Mechanisms

- ❑Vitamin A is also involved in the production of red blood cells, which are derived from stem cells that are dependent upon retinoids for their proper differentiation.
- ❑Vitamin A appears to facilitate the mobilisation of iron stores to developing red blood cells, where it is incorporated into the oxygen carrier haemoglobin).

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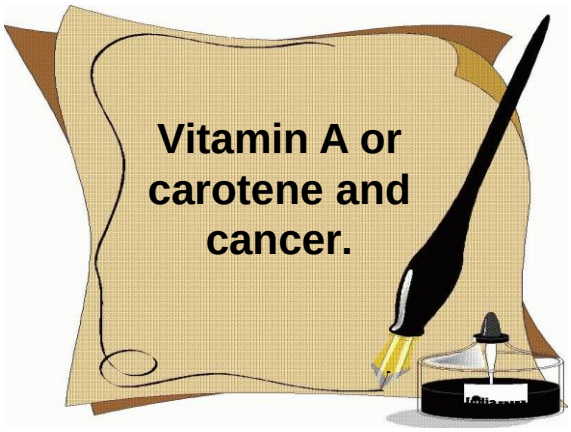

٢٩

Vitamin A or carotene and cancer.

Function

- ❑Vitamin A intake has a complex relationship with cancer prevention.

Mechanisms

- ❑Vitamin A intake has a complex relationship with cancer prevention: while small doses of vitamin A or beta-carotene appear to help prevent cancer, higher doses seem to have the reverse effect. The anti-cancer effects of beta-carotene appear to stem from its anti-oxidative ability to scavenge for reactive oxygen species, as well as through its conversion to vitamin A, which can improve immune function in addition to eliciting an anti-proliferative effect through the RAR and RXR receptors, thereby acting to block certain carcinogenic processes and inhibit tumour cell growth.However, an excessive intake of beta-carotene appears to have carcinogen effects, possibly through its promotion of the eccentric (or asymmetric) pathway of beta-carotene cleavage, which produces breakdown products that might lead to the destruction of retinoic acid through the activation of the P450 enzyme, which in turn could decrease retinoid signalling leading to enhanced cell proliferation. Therefore dosage seems to be an important factor in beta-carotene action.

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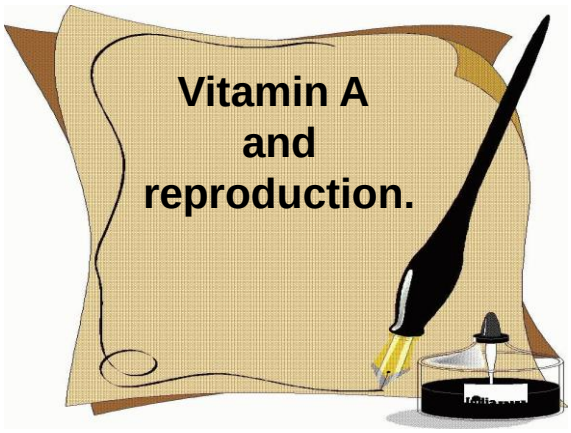

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Vitamin A and reproduction.

Function

- In most livestock, the absence of vitamin A in the ration will dramatically reduce reproductive ability.

Mechanisms

- Vitamin A is required form maintenance of epithelial cells lining of the genitor-urinary treat.
- It prevents keratinization of the mucus membrane of the reproductive system in male and female.

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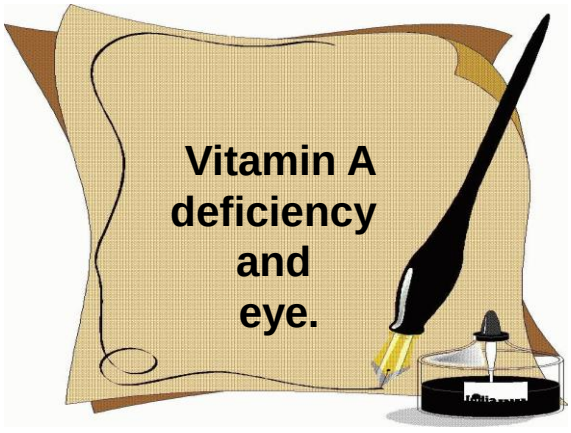

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Vitamin A deficiency and eye.

- **Xerophthalmia**
  - ❑Xerophthalmia 2- Copious lacrimation
  - ❑3- Night blindness
- **Copious lacrimation**
  - ❑diarrhea
  - ❑ coccidosis in birds
- **Night blindness**
  - ❑ Cold
  - ❑Pneumonia
- **N.B**
  - ❑Keratinization of corneal epithelium occurring in vitamin A deficiency is an important cause of vision failure which is termed xerophthalmia.
  - ❑A watery discharge from the nostrils and eyes results from vitamin A deficiency in chicks and calves and the eyelids are often stuck together.

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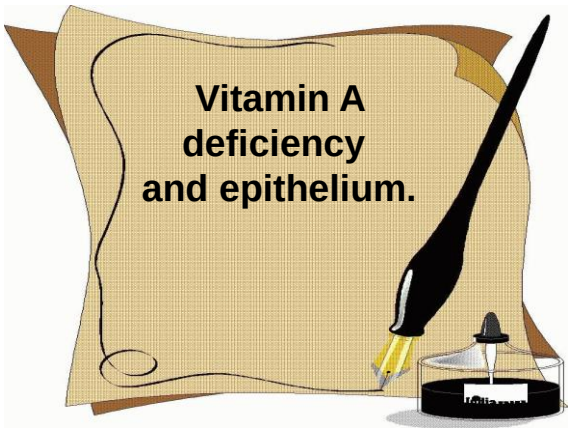

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The normal mucus-secreting cells of epithelium in various location of the body replaced by a stratified keratinized epithelium. There are many non infective problems due to keratinization of epithelium.

- **In the mouth**
  - ❑Loss of appetite due to infiltration of keratin into the pores of the test buds.
  - ❑Creamy white pustules were observed in the roof of the mouth and in esophagus in vitamin A-deficient hens.
- **In the intestine**
  - ❑diarrhea
  - ❑ coccidosis in birds
- **In the respiratory system**
  - ❑ Cold
  - ❑Pneumonia
- **In the Urinary tract**
  - ❑ Formation of stones.
- **In the eye**
  - ❑Keratinization of corneal epithelium occurring in vitamin A deficiency is an important cause of vision failure which is termed xerophthalmia.
  - ❑A watery discharge from the nostrils and eyes results from vitamin A deficiency in chicks and calves and the eyelids are often stuck together.

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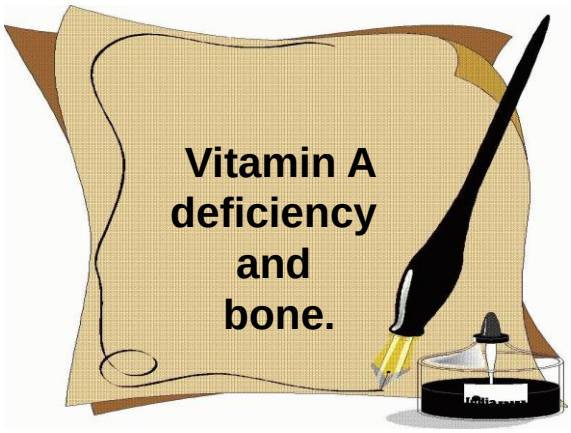

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Vitamin A deficiency and bone.

Direct effect

- ❑Disorganized bone growth.
- ❑Irritation of the joints.

Indirect effect

- ❑In some cases, there is a constriction of the openings through which the optic and auditory nerves pass, thereby resulting in blindness and or deafness.
- ❑Bone changes may also be responsible for the muscle incoordination and other nervous symptoms shown by vitamin A-deficient cattle, sheep and swine. These changes may be involved in the increase in cerebrospinal fluid pressure shown to be characteristics of the deficiency.

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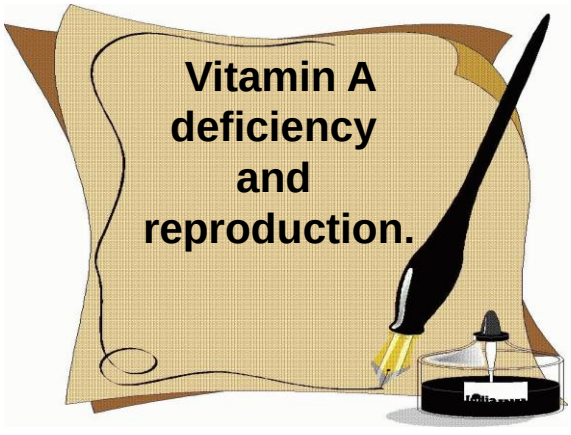

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Vitamin A deficiency and reproduction.

In male
- ❑Vitamin A deficiency in the male results in a decline in sexual activity and failure of spermatogenesis.

In female
- ❑The resorption of the fetus.
- ❑Abortion.
- ❑Birth of dead offspring.
- ❑Retained placenta may be a characteristic of vitamin A deficiency in some species

In rats
- ❑Degeneration of germinal epithelium and seminiferous tubules.
- ❑Cessation of spermatogenesis in vitamin A-deficient rats is not prevented by retinoic acid.

In rabbits
- ❑Reduction in fertility and an increased incidence of abortion in pregnant does.

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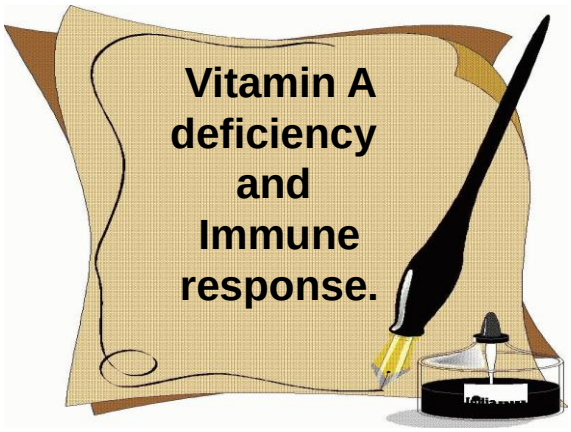

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Vitamin A deficiency and immune response of animals and birds.

Effect
- ❑Vitamin A deficiency causing a reduced immune response.

Examples
- ❑Animals deficient in vitamin A will show increased frequency and severity of bacterial, protozoal and viral infections as well as other disease conditions.
- ❑Part of disease resistance, as a function of vitamin A is related to maintenance of mucus membranes and normal functioning of the adrenal gland for production of corticosteroids needed to combat diseases.

N.B:

- ❑Vitamin A-deficient chicks showed rapid loss of lymphocyts.
- ❑Vit A-deficient rats showed atrophy of the thymus and spleen and reduced response to diphtheria and tetanus toxodis.
- ❑Vitamin A is instrumental in curing ring warm infestation in cattle.
- ❑Supplementation with vitamin A improved the health of animals infected with round warms.

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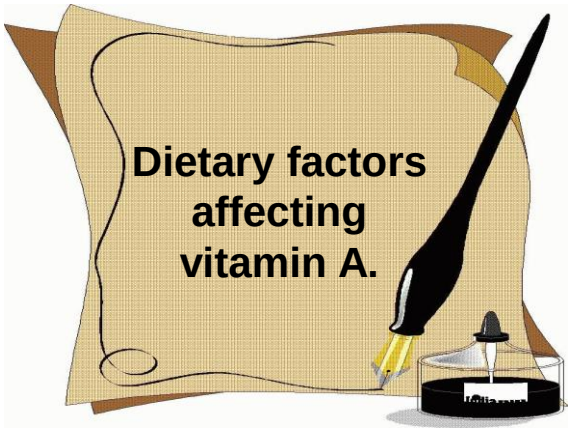

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Vitamin A deficiency and immune response of animals and birds.

Protein

- ❑Adequate dietary protein is required in order to synthesize the proteins connected with both the absorption and transport of carotenes and retinol such as:
  - Pancreatic retinyl hydrolase.
  - Retinal binding protein (RBP).
  - Carotens 15, 15 dioxygenase.
- ❑On the other hand, the vitamin A requirements increases with the supply of diets high in protein or energy which accelerate the growth rate.

Uncompleted…

Fat

- ❑Small amounts of fat are usually contained in animal diets are necessary for micellar absorption of vitamin A or carotene from the intestinal lumen.

Zinc

- ❑Zinc may limit the synthesis of RBP because of its general role in protein synthesis.

Vitamin E

- ❑Small amounts of fat are usually contained in animal diets are necessary for micellar absorption of vitamin A or carotene from the intestinal lumen. .

Mineral oils

- ❑Small amounts of fat are usually contained in animal diets are necessary for micellar absorption of vitamin A or carotene from the intestinal lumen. .

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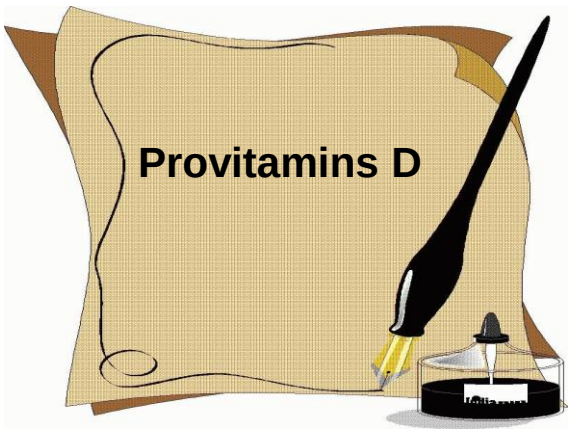

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Provitamin D

Definition

These compounds which are not themselves vitamins and which function as vitamin D only after undergoing a chemical change. The precursors have no biological activity until the β-ring is opened between the 9-and 10- . .position of the steroids

Members

- Ergosterol
- 7-Dehydrocholesteral

Ergosterol

- ❑Provitamin D2, occurs commonly in plants and is transformed into vitamin D2 during sun-drying of harvested forages.
- ❑1 Kg of hay contains between 800 and 1700 IU.
- ❑Yeast is rich in ergosterol and its irradiation results in a potent source of vitamin D.

7-dehydrocholesterol-

- ❑ Irradiated 7-dehydrocholesterol is used as a feed additive for poultry diets
- ❑D3 are formed by ultraviolet irradiation

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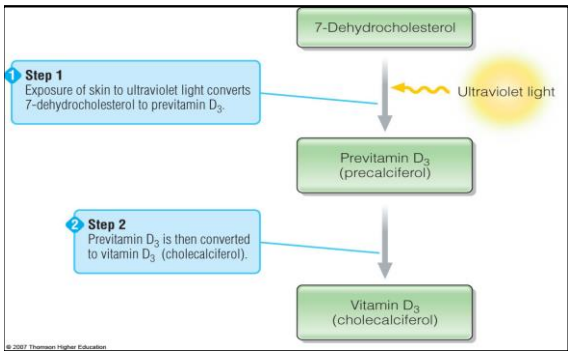
Synthesis of Vitamin D



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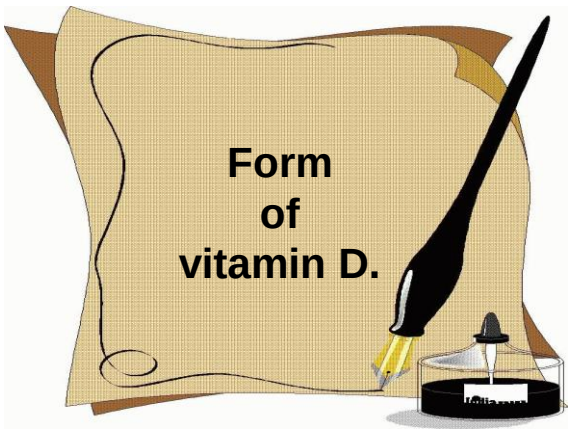

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Form of vitamin D.

- **Vitamin D2** — (Vitamin D2) - found in plants.
- **Vitamin D3** — (Vitamin D3) - found in animalsPhotochemical conversion in skin:7—dehydrocholesterol converted into cholecalciferol.

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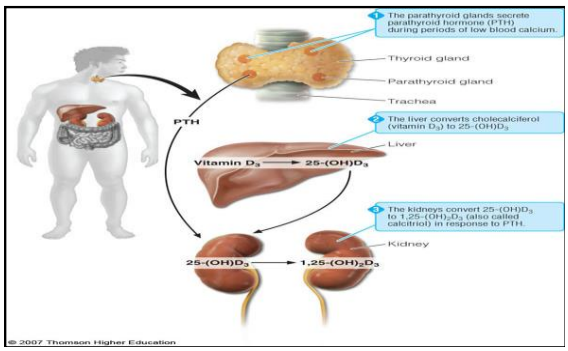
Activation of Vitamin D



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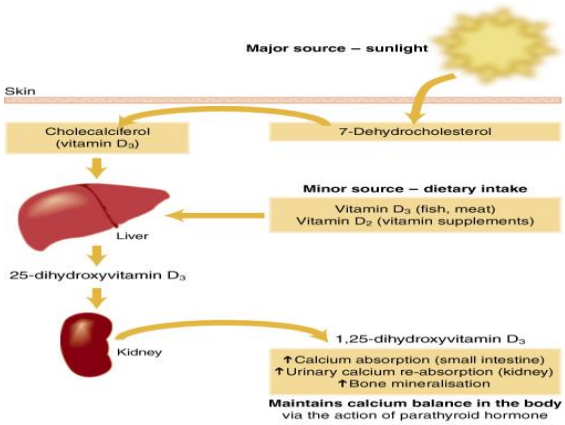

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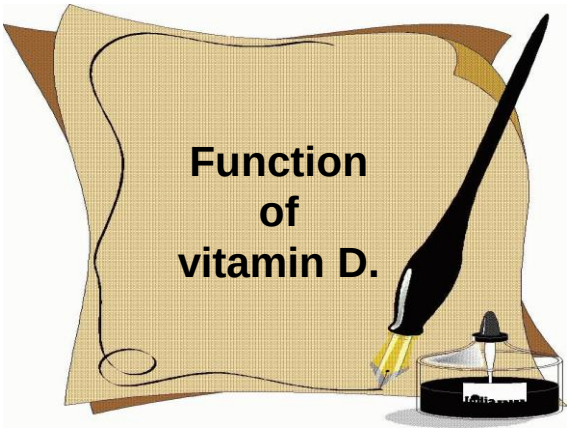

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Function of vitamin D.

The general function of vitamin D is to elevate plasma calcium and phosphorus to level that will support normal mineralization of bone as well as other body function .This function occurs through three effects :

- **Intestinal effect** → Vitamin D stimulates active transport of calcium and phosphorus across intestinal epithelium.
- **Bone effect** → Vitamin D brings about mineralization of bone matrix
- **Kidney effect** → Vitamin D improves calcium reabsorption in the distal renal tubules. Form of vitamin D = 1.25 (OH)2 D3.

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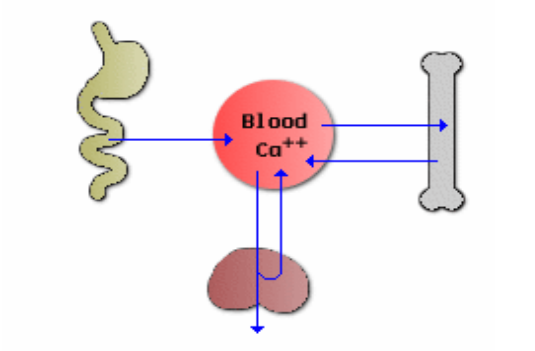

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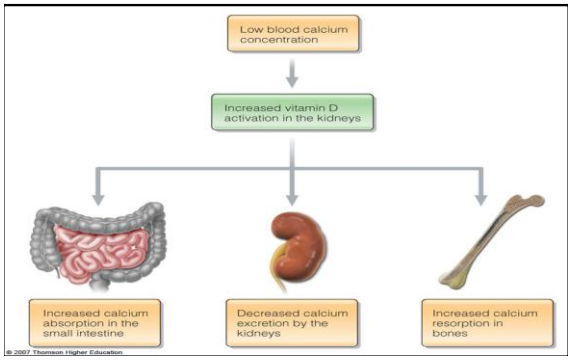
Vitamin D & Calcium Homeostasis



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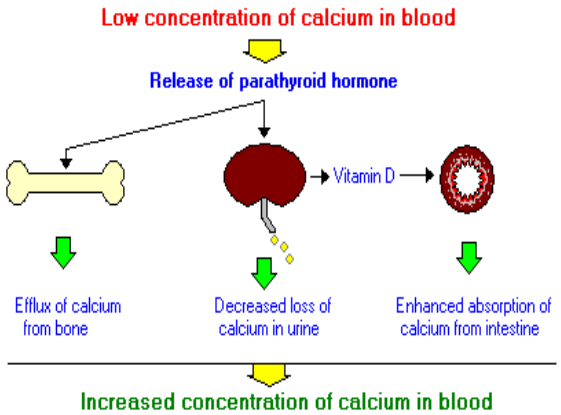

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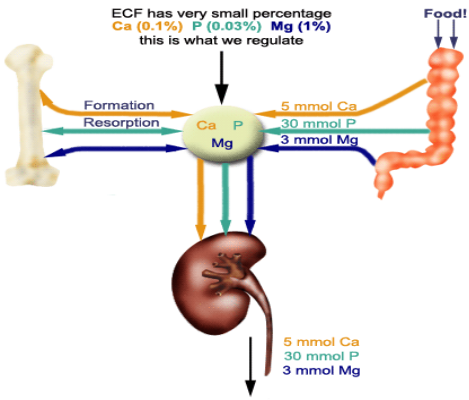

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Intestinal effect.

Effect

Vitamin D stimulates active transport of calcium and phosphorus across intestinal epithelium.

Mechanism

❑ (OH) D3 is transferred to the nucleus of the intestinal cell where it interacts with the chromatin material. In response to the 1.25 (OH)2 D3 specific RNAS are elaborated by the nucleus and when these are translated into specific protein by ribosome, leading to enhancement of calcium and phosphorus absorption occur.

❑ In intestine, 1.25 (OH)2 D3 promotes synthesis of calcium binding protein which stimulate calcium and phosphorus absorption two kinds of mucosal proteins are dependent on vitamin D:

- Calcium binding protein = CaBp
- Intestinal membrane calcium binding protein = IMCBp.

❑ It is proposed that the primary non genomic mechanism by which 1.25- (OH)2 D3 regulates calcium transport across the luminal membrane of the enterocyte involves inducing a specific alteration in membrane phosphatidylcholin content and structure, which leads to an increase in membrane fluidity and thereby to an increase in calcium rate.

N.B :Phosphate is transported against an electrochemical potential gradient involving sodium in response o 1.25 – (OH)2 D3.

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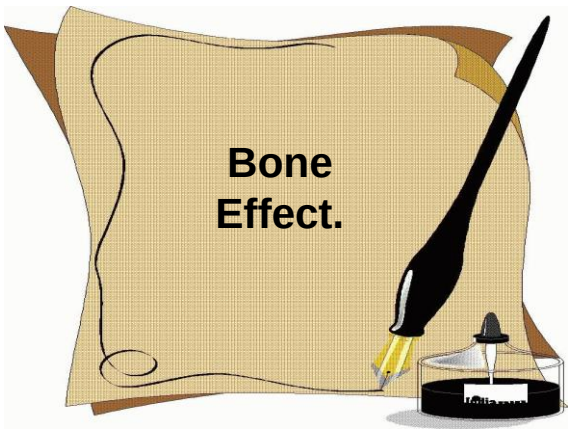

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Bone effect.

Effect

Vitamin D brings about mineralization of bone matrix.

Mechanism

- ❑(OH)2 brings about mineralization of bone matrix.
- ❑1.25 (OH)2 D3 is localized in the nuclei of bone cells. Also there is some indication that 24, 25- (OH)2 D3 and possibly 25-oH D3 may have unique actions on bone.
- ❑The 24, 25 (OH)2 D3 appears to be accumulated in bone, where it promotes normal development.
- ❑Vitamin D plays another role in bone, that is, in mobilization of calcium from bone to the extracellular fluid compartment this function is shared by PTH.
- ❑Vitamin D has a role in the biosynthesis of collagen in preparation for mineralization.

N.B: A vitamin D deficiency causes inadequate cross-linking of collagen as a result of low lysyl oxidase activity, which is involved in a condensation reaction for the collagen cross-linking. This may be a direct effect of vitamin D or a result of mineral changes in blood, it is not considered a major function of vitamin D.

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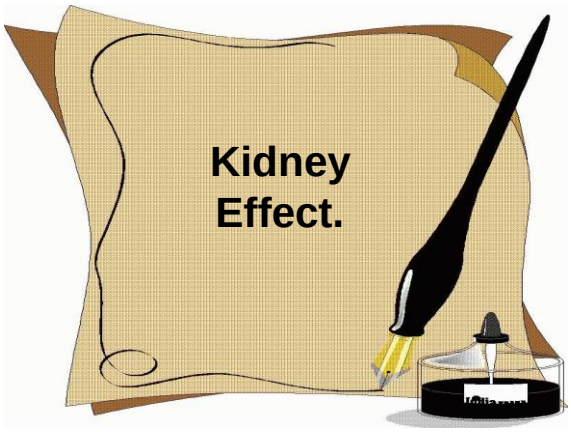

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Kidney effect.

Effect

Vitamin D improves calcium reabsorption in the distal renal tubules.

Mechanism

❑25-hydroxy-vit D3 converted into1, 25-dihydroxy-vit D3 which improves calcium reabsorption in the distal renal tubules .

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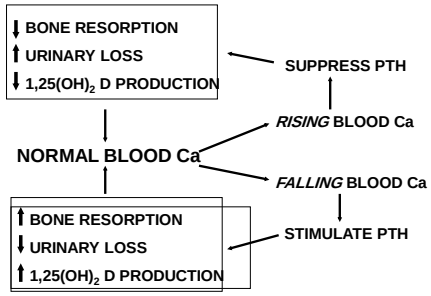
LOOPS CALCIUM, PTH, AND VITAMIN D FEEDBACK



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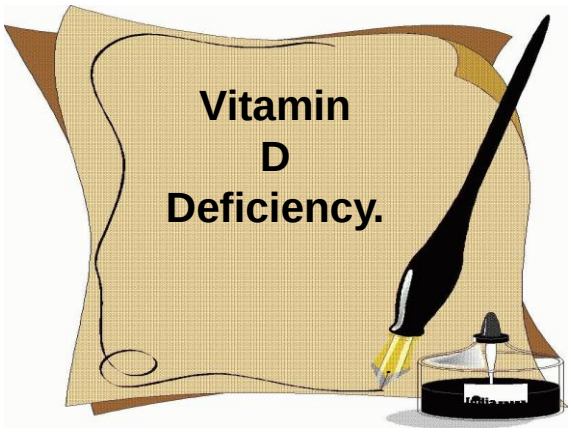

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Vitamin D deficiency.

The typical signs of vitamin D deficiency are

- ❑Rickets in young animals.
- ❑Osteomalacia in adult animals.

clinical symptoms differ in different species

- ❑In calves as swelling occurs in the metacarpal and metatarsal bones. Bending of the forelegs.
- ❑Arching of the forelegs arching of the back.
- ❑In chicks the joints become enlarged and the beak becomes soft and rubbery and can be easily been (plate in atlas).
- ❑The most characteristic internal signs of vitamin D deficiency in chicks are:
- ❑A beading of the ribs at their junction with the spinal column.
- ❑Crooked backbone (plate in atlas).
- ❑In laying hens, the same signs are observed and the eggs become thin-shelled, hatchability is reduced and finally egg production decreased.

N.B:

- ❑Rickets represents a disturbance of the mineral metabolism in such as way that the calcification of the growing bones does not take place normally (Ca and P are not deposited in bone)
- ❑Ostemoalacia softening of bone, represents a decrease in the mineral contents of adult bone.

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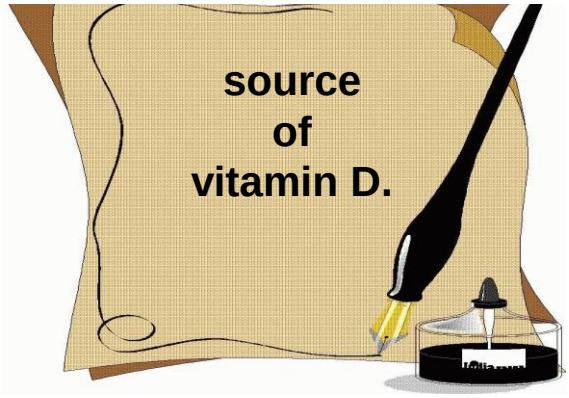

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Sources of provitamin and vitamin.

Provitamins

- Provitamins D2 and D3 are both widely distributed in nature.

Vitamins

- ❑ The distribution of vitamin D2 and D3 in food is very limited.
- ❑ The best sources of vitamin D3 are:
- ❑ Fish liver and fish liver oils.
- ❑ Among animal products, milk and egg yolks contain the vitamin, but the amount present depends on the season in which they are produced a mount of the vitamin supplied in the diet of cows or hens.
- ❑ Average of 260 IU in 100/ gm of egg.
- ❑ Average of 1-4 IU in 100/gm of milk.

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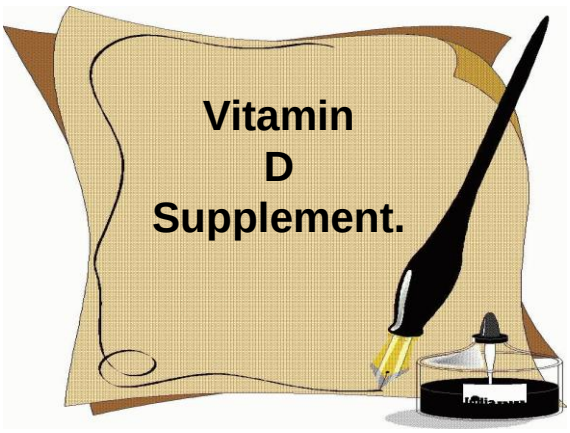

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Vitamin D supplement.

- ❑The need for supplementing the diet of ruminants with vitamin D is not as great as for poultry.
- ❑Ruminants can receive adequate amounts of vitamin D from irradiation or from sun-cured hays.
- ❑In animals that do not have direct access to sunlight confined farm birds, the endogenous vitamin D production does not meet the requirement and their diets must be supplemented with vitamin D concentrates.
- ❑The use of vitamin D and its metabolites for milk fever. This disease and metabolic bone disorders respond to very small doses of synthetic 1, (OH)D3 and 1.25 (OH)2 D3.
- ❑Exposure of animals to sunlight for a short time during the day is sufficient to convert provitamin D3 present in the skin to vitamin D3 which eliminates the need for a dietary source.
- ❑Vitamin D2 is almost as effective as vitamin D3 for mammals, but vitamin D2 is virtually inactive in birds, thus poultry feed must be fortified with vitamin D of animal origin or synthetic vitamin D3.

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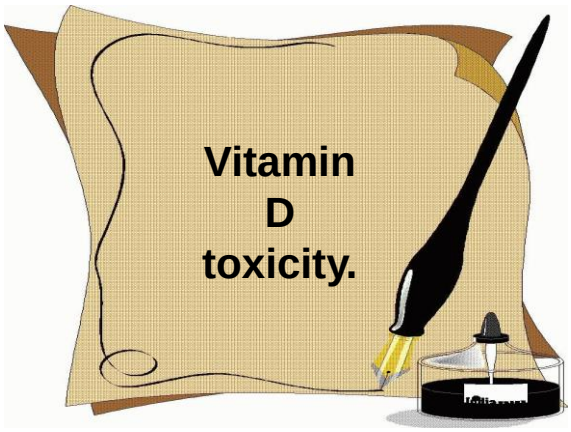

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Vitamin D Toxicity.

Safe feeding levels

- ❑A few days - 25,000 IU/kg feed
- ❑60 days - 2,200 IU/kg feed

Symptoms of toxicity

- ❑ Loss of appetite
- ❑ Weight loss
- ❑ Reduced rumination
- ❑ Depression
- ❑ Widespread calcification of soft tissue
- ❑Kidneys, heart, pancreas, lymph glands, lung alveoli
- ❑ Inflammation
- ❑ Demineralization of skeletal system.

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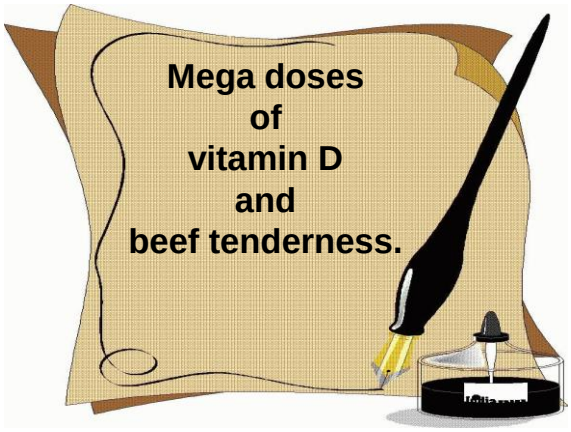

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Feeding Mega Doses of Vitamin D and Beef Tenderness.

- ❑ Feed cattle 5 to 7 million IU/d of Vit D3 8 to 10 days prior to harvest improves tenderness of skeletal muscle.
- ❑ Response not consistent in all experiments.
- ❑ Less response with increased aging of beef.
- ❑ Accumulation of vitamin D and metabolites in tissues.
- ❑ Reduced feed intake.
- ❑ Loss of body weight.
- ❑Feeding cattle125 ug of 25-hydroxy Vit D3 for 4 days prior to harvest may be alternative.
- ❑ Response not consistent.

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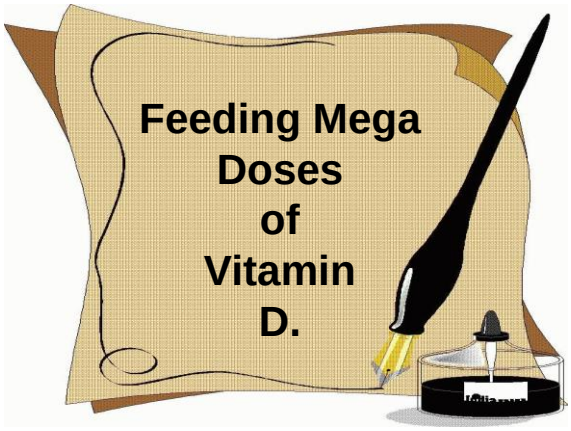

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Feeding Mega Doses of Vitamin D .

- **Prevent milk fever**
  - ❑20 million IU/d starting 3 to 5 days before calving.
  - ❑continuing through the fist day postpartum؛
- **Improve tenderness of beef**
  - Increased blood and muscle Ca increases activity of calpains, enzymes in muscle that degrade muscle myofibrils.
- **Observations**
  - ❑ Blood Ca increased.
  - ❑ Increased degradation of myofibrils.
  - ❑ Reduced force to shear muscle.

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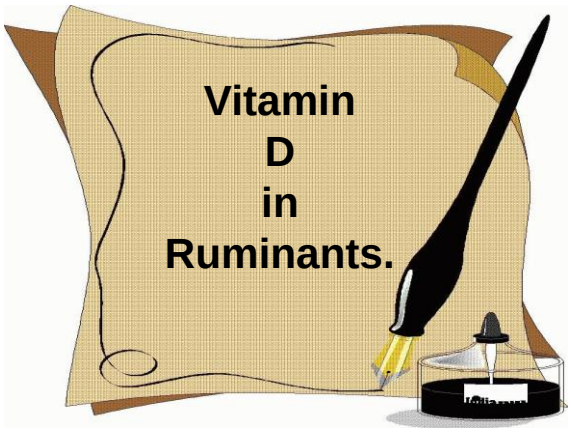

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Vitamin D - Ruminants.

- **Deficiency**
  - ❑Rickets in calves
  - ❑ Swollen and stiff joints
  - ❑ Reduced feed intake
  - ❑ Tetany
  - ❑ Weak bones
- **Vitamin D can be toxic**
  - ❑ High blood Ca.
  - ❑ Calcification of soft tissues.
  - ❑ Loss of appetite.
- **Storage**
  - Vitamin D not stored in the body in any quantity

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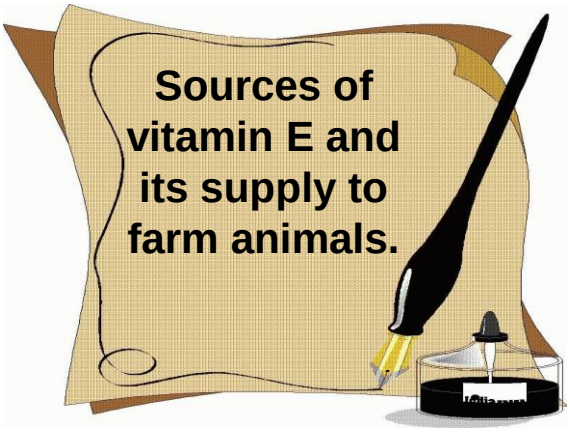

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Sources of vitamin E and its supply to farm animals.

- ❑α- Tocopherol is practically the only tocopherol present in green plants and in animal products.
- ❑The content is quite high in green plants but low in feedstuffs of animal origin.
- ❑The germs of cereal, particularly the germ oil as well as other plant oils are rich sources of tocopherols.

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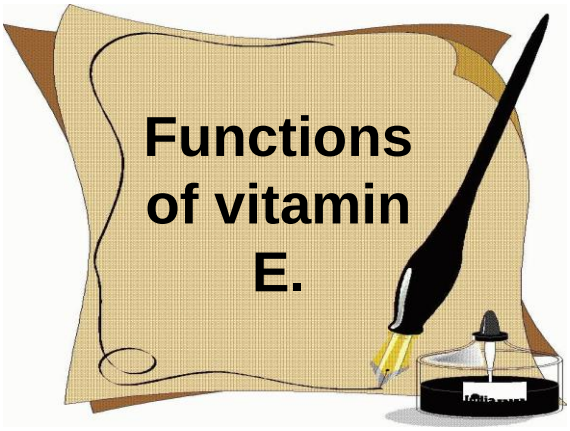

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Functions of vitamin E.

1. Vitamin E as a biological antioxidant.
2. Membrane structure and prostaglandin synthesis.
3. Blood clotting.
4. Disease resistance.
5. Electron transport and deoxyribonucleic acid.
6. Relationship to toxic Elements.
7. Other functions .

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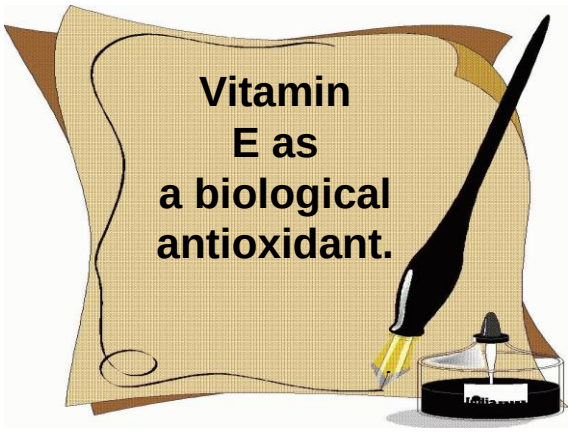

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Vitamin E as a biological antioxidant.

Function
- ❑Vitamin E is an intercellular and intracellular antioxidant .

Mechanisms
- ❑Vitamin E reacts or functions as a chain-braking antioxidant, thereby neutralizing free radicals and preventing oxidation of lipids within membran.
- ❑it prevents oxidation of unsaturated lipid materials within cells, thus protecting fast within the cell membrane from breaking down.

Absence of vitamin E as antioxidants.
- Destroys structural integrity of the cells and causes:
- ❑Metabolic derangements.
- ❑Morphological damage of the muscles.

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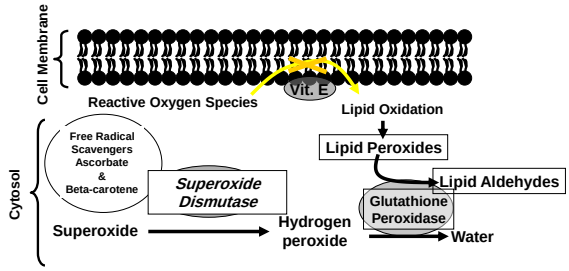
Cellular Antioxidant Defense Mechanisms



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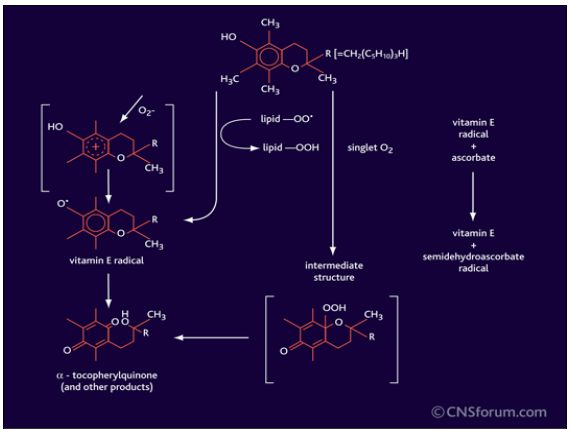

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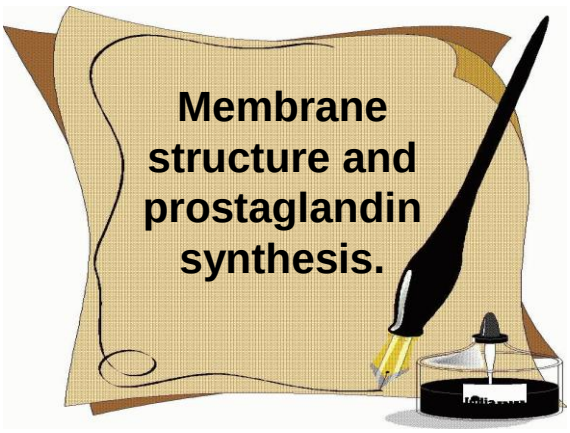

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Membrane structure and prostaglandin synthesis.

Membrane structure

- ❑α-Tocopherol may be involved in the formation of structural components of biological membranes.
- ❑It is reported that-tocopherol stimulated the incorporation of C14 from linoleic acid into arachidonic acid in fibroblast phospholipids.

Prostaglandin synthesis

It was found that α-tocopherol exerted a pronounced stimulatory influence on formation of prostaglandin E from arachidonic, while a synthetic antioxidant had no effect.

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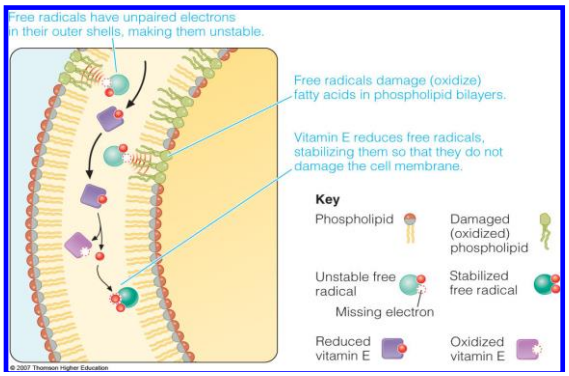
Cell Membrane Structure



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Blood clotting.

- ❑Vitamin E is an inhibitor of platelet aggregation.
- ❑It may play a role by inhibiting peroxidation of arachidonic acid, which is required for formation of prostaglandins involved in platelet aggregation.

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Disease resistance.

- **Function**
  - ❑Vitamin E has a role in disease resistance.
- **Mechanisms**
  - ❑Vitamin and selenium play in protecting leukocytes and macrophages during phagocytosis.
  - ❑Vitamin E and selenium may help these cells to survive the toxic products that are produced in order to effectively kill ingested bacterial.
- **Examples**
  - ❑Mice fed vitamin E-deficient diets are unable to produce a vigorous humoral response and this decreased immune reactivity undoubtedly contributes to increased susceptibility to bacterial infections associated with vitamin E-deficiencies.

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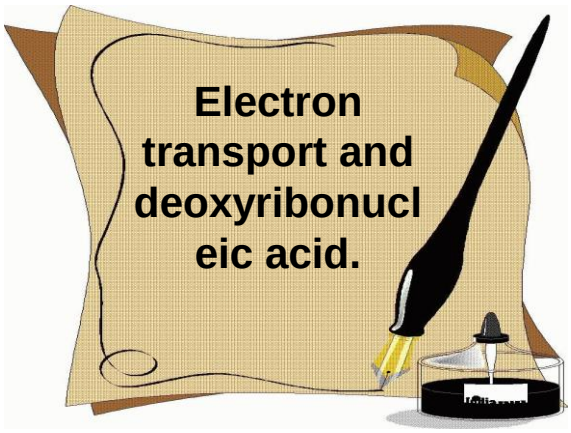

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Electron transport and deoxyribonucleic acid.

- **Electron transport** — ❑Vitamin E act as a cofactor in the cytochrome reductase portion of the nicotinamid-adenine dinucleotids (NAD) oxidase and the succinate oxidase systems.
- **Deoxyribonucleic acidbiosynthesis.** — Vitamin E also appears to regulate the biosynthesis of deoxyribonucleic acid (DNA) within cells:

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Vitamin E as a biological antioxidant.

Function	❑Both vitamin E and selenium provide protection against toxicity with three classes of heavy metals.
First class	Consists of metals like cadmium, mercury: ❑ Selenium is highly effective. ❑ Vitamin E is little influence.
First class	Consists of metals like sliver, arsenic: ❑ Selenium is effective at high level. ❑ Vitamin E is highly effective.
First class	Consists of metals like lead: ❑ Selenium is little effective. ❑ Vitamin E is effective.

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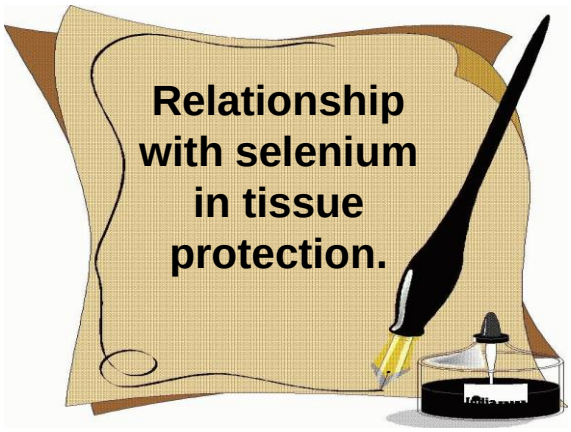

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Relationship with selenium in tissue protection.

Relationship	There is a close working relationship between vitamin E and selenium. ❑Selenium has a sparing effect on vitamin E. ❑Selenium delays onset of deficiency syndromes. ❑Vitamin E and sulfur amino acids partially protect against or delay onset of several forms of selenium deficiency syndromes. N.B: There are three lines of defense against tissue breakdown.
First line	It now appears that vitamin E in cellular and subcellular membranes against peroxidation of vital phospholipids.
Second line	Selenium, as part of the enzyme glutathione peroxidase that destroys these peroxides before they have an opportunity to cause damage to membranes.
Third line	Sulfur amino acids are precursors of glutathione peroxidae.

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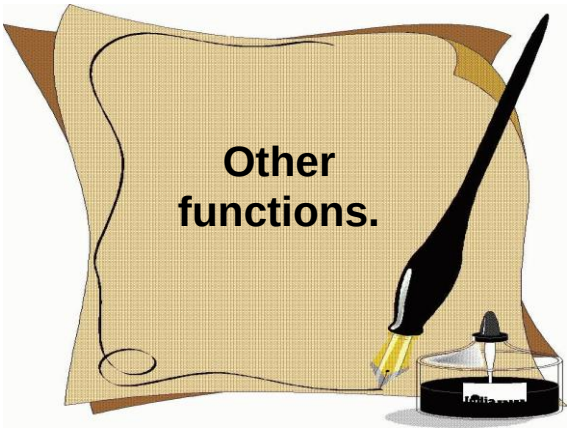

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Other functions.

- ❑ Normal phosphorylation reactions, especially of high-energy phosphate compounds such as creatine phosphate and adenosine triphosphate.
- ❑ In synthesis of ascorbic acid.
- ❑ In synthesis of ubiquinone.
- ❑ In sulfur amino acid metabolism.
- ❑ Vitamin E is reported to have a role in vitamin B12 metabolism.

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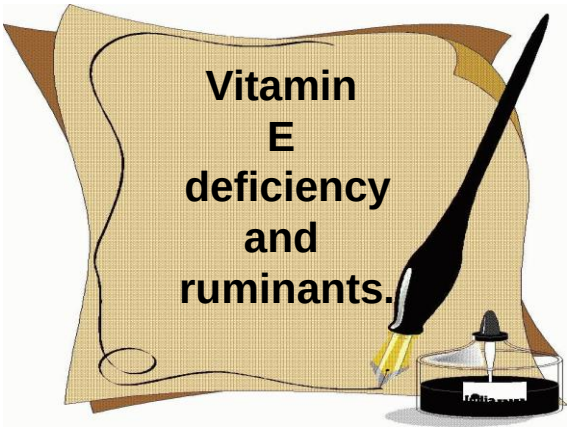

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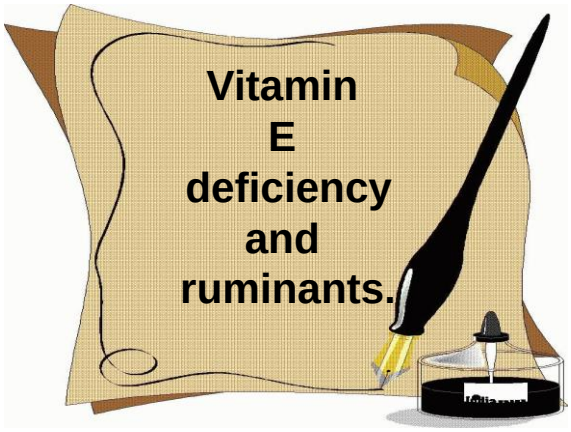

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White muscle disease.

Other names	Nutritional muscular dystrophy and WMD.
Occurrence	Young ruminants
Causes	Selenium deficiency which influenced by vitamin E. status.
Development	❑ Extrauterine ❑ Interauterine
Symptoms	❑Generalized weakness ❑Affected animal having difficulty standing and Stiffness. ❑In calves, the tongue musculature may be affected and prevent suckling. ❑Deterioration of muscles. ❑Death occurs from heart failure as a result of severe damage of heart muscles
In lambs	In lambs, the WMD is also known as stiff lamb disease which characterized by: ❑A gradual swelling of the muscles, particularly in the lumbar is and rear thigh regions. ❑Chronic cardiac muscle degeneration is also found in the lamb.

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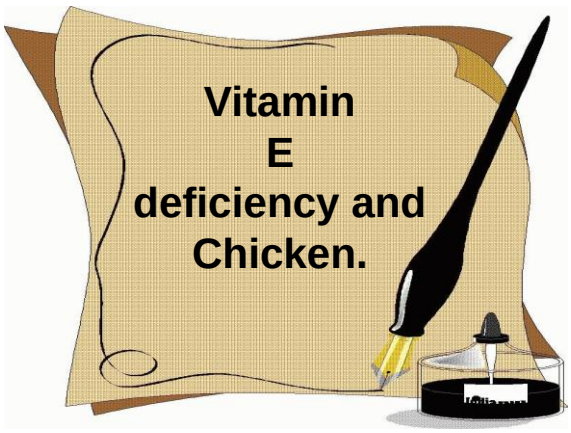

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Chickens show three distinct vitamin E deficiency diseases.

- Encephalomalcia.
- Exudative diathesis
- Nutritional muscular dystrophy

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Encephalomalcia.

- Other names: Crazy chick disease.
- Occurrence: Young chickens
- Causes: Vitamin E deficient diets containing certain polyunsaturated fatty acids such as linoleic acid.
- Symptoms:
  - Nervous syndrome characterized by
  - ❑ ataxia.
  - ❑Lack ofcoordination.
  - ❑Involuntary movements of the legs.
  - ❑Head twisting.
  - The cerebellum is softened and swollen because of hemorrhage and edema.

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Exudative diathesis.

- ❑It is characterized by severe edema produced by a marked increase in capillary permeability.
- ❑Exudative diathesis not affected by the levels of antioxidants or cystine in the diet.
- ❑This disease can be largely prevented by selenium and vitamin E.

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Nutritional muscular Dystrophy.

- **Causes**: The muscular dystrophy developed on diets low in vitamin E, sulfur containing amino acids and some times selenium and high in playunsaturated fatty acids.
- **Symptoms**: This condition in chicks is characterized by degeneration of muscle fibers (myopathy), particularly those of the breast and legs.
- **Prevention**: The disease is prevented in chicks by supplement of either vitamin E or cysteine, in ducks only by vitamin E, while in turkeys selenium is the primary factor responsible for the prevention of muscular dystrophy.
- **N.B**: Similar signs of muscular dystrophy occur in vitamin-E-deficient ducks and turkeys.
  In turkeys it is characterized by lesions in the muscular wall of the gizzard.

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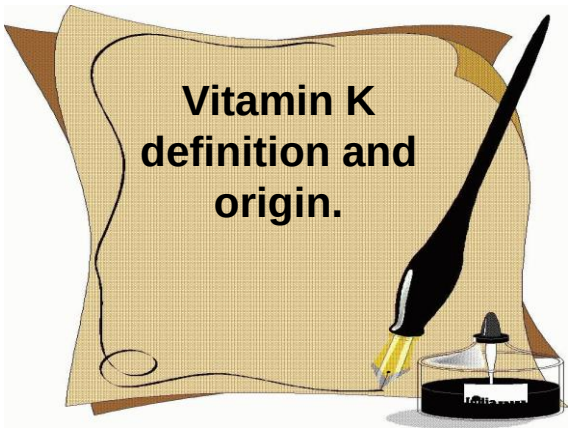

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Vitamin K

- **Other names**:
  - ❑Vitamin K1 = Phylloquinone
  - ❑Vitamin K2 =Menaquinone
  - ❑Vitamin K3 = Menadione
- **Origin**:
  - ❑ Extracted from plant material was named K1.
  - ❑ Form material that had undergo bacterial fermentation was named K2.
  - ❑ It is the synthetic form = K3.

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Supply and source of vitamin K to form animals.

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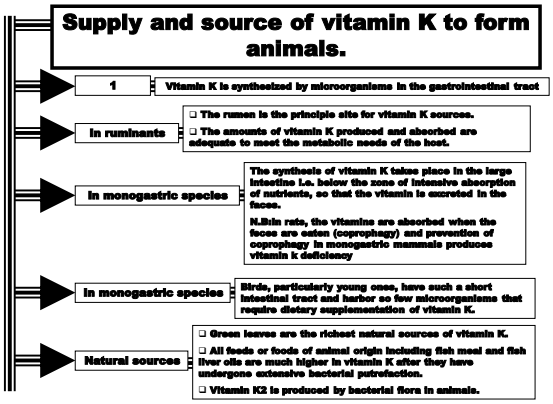
Supply and source of vitamin K to form animals.
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Vitamin K is synthesized by microorganisms in the gastrointestinal tract
In ruminants
❑ The rumen is the principle site for vitamin K sources.
❑ The amounts of vitamin K produced and absorbed are adequate to meet the metabolic needs of the host.
In monogastric species
The synthesis of vitamin K takes place in the large intestine i.e. below the zone of intensive absorption of nutrients, so that the vitamin is excreted in the feces.
N.Bin rats, the vitamins are absorbed when the feces are eaten (coprophagy) and prevention of coprophagy in monogastric mammals produces vitamin k deficiency
In monogastric species
Birds, particularly young ones, have such a short intestinal tract and harbor so few microorganisms that require dietary supplementation of vitamin K.
Natural sources
❑ Green leaves are the richest natural sources of vitamin K.
❑ All feeds or foods of animal origin including fish meal and fish liver oils are much higher in vitamin K after they have undergone extensive bacterial putrefaction.
❑ Vitamin K2 is produced by bacterial flora in animals.

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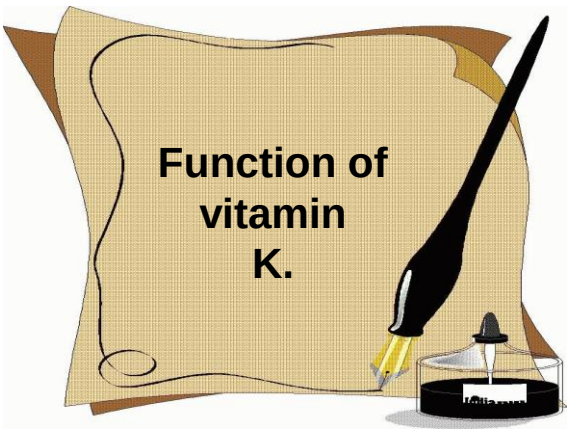
Function of vitamin K.

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Function of vitamin K.

Function

Vitamin K is required for normal blood clotting.

Mode of action

- ❑ The colorless protein mainly responsible for coagulation is fibrin.
- ❑ Fibrin is formed from its soluble precursor fibrinogen.
- ❑This transformation is catalyzed by an enzyme called thrombin.
- ❑ Thrombin is formed from prothrombin.
- ❑ Vitamin K is required for:
  - ▪The synthesis of the following three additional factors involved in the conversion of prothrombin to thrombin.
  - ▪The synthesis of prothrombin in the liver.
- ❑Fibrinogen ⟶ fibrin + peptide .

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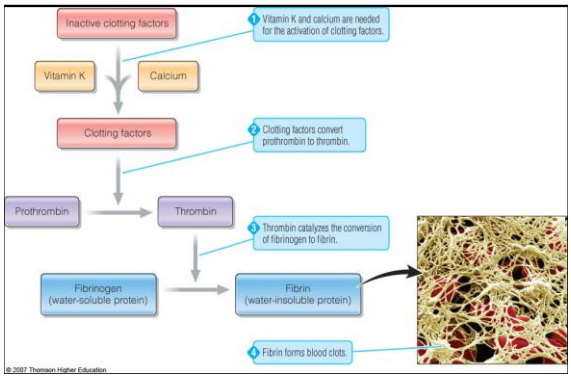
Functions of Vitamin K in Coagulation



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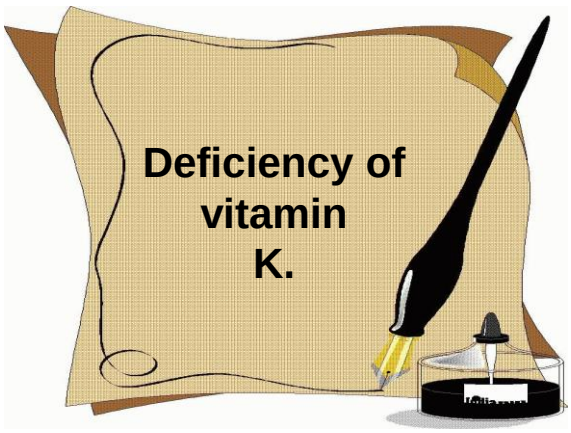

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Deficiency of vitamin K.

Symptoms

Symptoms of vitamin K deficiency occur in practice only in intensively reared young chicks:

- ❑Subcutaneous hemorrhages on the breast, legs, and wings or on the surface of the intestine.
- ❑Some deficient chicks may bleed to death from a slight bruise or other injury.

N.B: Adult hens deficient in vitamin k produce eggs low in this vitamin and chicks hatched from such eggs are deficient and may bleed to death from slight injury.

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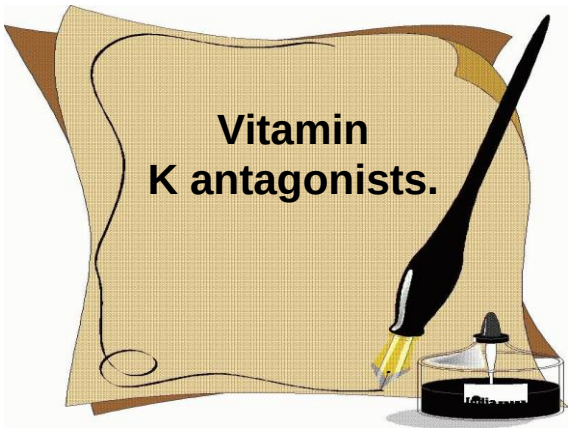

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Vitamin K Antagonists.

Dicumarol

Dicumarol:

- ❑- Found in certain plants.
- ❑- Inters with blood clotting by blocking the action of vitamin K.

Mode of action

- ❑By reason of structural similarly it could occupy reaction sites and there by deny them to the metabolite. e.g., dicumarol and vitamin K.

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