

Vitamins

Hofmeister defined vitamins as, these are substances, which are indispensable for the growth and maintenance of the animals, which occur both in animals and plants and are present in only small amounts in food.

In modern sense, vitamins are the substances distinct from major components of food required in minute quantities and whose absence causes specific deficiency disease. Plant can synthesize all the vitamins, which they require as a component of various enzyme systems. Vitamins are organic substances required by animals in very small amounts for regulating various body processes toward normal health, growth, production and reproduction. But this definition ignores the important part that these chemical substances found in plants and their importance generally in the metabolism of all living organisms.

The term vitamins was used by Funk in 1912 for an amine, the active factor from rice polishing which are necessary for existence of life (vital + amine).

There are at least 15 vitamins, which has been accepted as essential food factors and few others have been proposed. The vitamins are divided into two main groups, the water-soluble and the fat-soluble, which are differentiated as:

	Parameters	Fat soluble vitamins	Water soluble vitamins
1.	Solubility	Fat soluble	Water soluble
2.	Chemical nature	Consist only of C,H, and O.	Ring compound except Pantothenic acid (straight chain), contains C, H, O, N, S, P or cobalt Vit. B12 contain cobalt, sulphur containing vitamins are thiamine and biotin.
3.	Functions	Vit. A, D, E, K works as in vision, Calcium absorption, maintenance of genital system and blood clotting respectively	As coenzymes or prosthetic groups of enzymes except Vit. C.
4.	Synthesis	Only Vit. K is synthesized by symbiotic microorganisms Vit. D can be synthesized in the skin upon exposure to sun light	Ruminant synthesized all Vit. B complex with incorporation of cobalt in diet. Niacin can be synthesized from Tryptophane except cat. Vit. C except guinea pig, human.
5.	Absorption	Absorbed from gastro-intestinal tract by passive diffusion	Absorbed from gut directly
6.	Storage	Liver	Not stored except Vit. B12 (liver) and riboflavin (some extent)
7.	Excretion	Excretion in faeces via bile	Excreted in urine
8.	Toxicity	Excess dietary intake causes	Relatively non toxic

		toxicity because they are stored in the body	
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Functions of vitamins:

1. Vitamins are essential for the good health and play important role in the body growth.
2. Vitamin provides resistance against diseases and increases the productivity power of animals.
3. Vitamins are essential constituents of certain enzyme systems, regulate body metabolism and clotting of blood.
4. Vitamins are needed during pregnancy for the development of foetus.
5. Vitamin A is responsible for the proper functioning of vision and Vitamin C keeps the gums in healthy state.

Chemical name of Vitamins: Homework

Vitamin A

Vitamin A was discovered in 1913 by McCollum et al. It has been reported to occur in two different forms, viz. vitamin A₁ or retinol and vitamin A₂ or dehydroxyretinol. It is an unsaturated monohydric alcohol. One international unit of vitamin A is 0.344 µg of pure vitamin A acetate, which is equivalent to 0.3 µg of vitamin A alcohol. One IU of provitamin A activity is equal in activity to 0.6 µg of β-carotene.

Chemical name: Vitamin A, known chemically as retinol, is an unsaturated monohydric alcohol. Vitamin A does not exist as such in plants, but is present as precursors or pro-vitamin in the forms of certain carotenoid which can readily be converted into vitamin A by the animal. At least 80 provitamins are known and these included a,b and g carotenes, cryptoxanthine, which is present in higher plants and mycoxanthin which occur in the blue green algae. Provitamin 13- carotene is the most widely distributed and most active. Vitamin A contains one β-ionone ring and β-carotene contains 2 β-ionone rings. Carotene is converted into vitamin A in the epithelial cell of intestine. Part of carotene is converted into vitamin A in the liver also.

Metabolism: Conversion of β-carotene to vitamin A takes place in intestinal mucosa. In most of animals absorption takes place in the form of vitamin A. Main site of vitamin A absorption is proximal jejunum. Liver is the main storage site of vitamin A (90 percent). In general, carotene digestibility was higher than average during warmer months and lower than average during winter.

Functions: Vitamin A plays following important functions.

1. Growth: Vitamin A is responsible for the normal development of various epithelial tissues in the body. Changes have been demonstrated in the salivary gland, tongue and pharynx,

respiratory tract, the genitourinary tract, eyes and certain glands of internal secretion. The primary change involves atrophy of the epithelium and the formation of stratified keratinizing epithelium.

2. Vision: Vitamin A is helpful in the transmission of light stimuli from the eye to the brain. Each vitamin A molecule is combined with specific type protein called opsin to form a visual pigment. Four such pigments have been identified which are rhodopsin and porpyropsin, which are present in rod and cyanopsin, which are in the cones of the retina. The rods are concerned with vision in dim light while the cones are concerned with bright light and colour vision.

3. Reproduction: Vitamin A plays an important role in male and female reproduction. Lack of vitamin A in the diet causes atrophy of the germinal epithelium resulting in sterility.

4. Skin: Lack of vitamin A in the diet causing keratosis of the skin (dryness and roughness of skin). A keratosis especially of the hair follicles is a prominent feature.

5. Urolithiasis: A condition in which urinary calculi are present is known as urolithiasis. Lack of vitamin A in the diet causing keratinization of epithelial cells in genitourinary tract followed by bacterial invasion and deposition of calcium phosphate precipitate on the site and ultimately calculi is formed.

6. Infection: Vitamin A has been called the anti-infective vitamin. The vitamin does help to establish and maintain a resistance to infection in the body, especially in tissues, which undergoes keratinization in a deficiency of it.

7. Bone development: Vitamin A has a role in the normal development of bone through a control activity of osteoclasts and osteoblasts of the epithelial cartilage.

Requirement

- Growing Cattle :80 IU/ kg body weight
- Dry cow :76 IU/ kg body weight
- Lactating cow: 110 IU/ kg body weigh
- Piglet :500 – 1000 IU / kg feed
- Pig : 2000 – 3000 IU / kg feed
- Poultry
 - Broiler : 1500 IU / kg feed
 - Layer : 4000 IU / kg feed

Factors influencing Vitamin A requirement: Following factors affect vitamin A requirements.

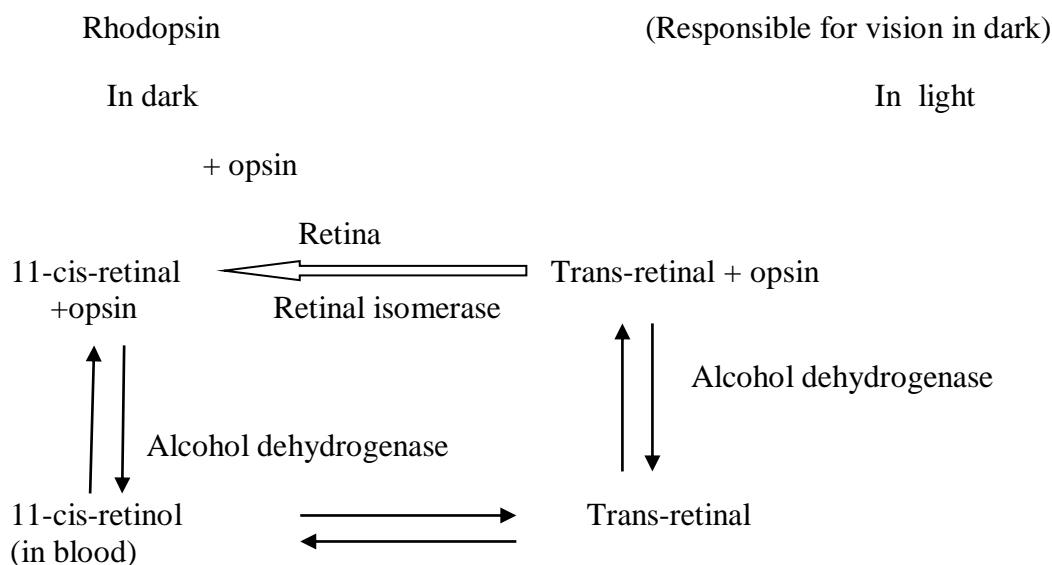
1. Genetic differences
2. Conversion efficiency of carotene to vitamin A
3. Variation in level, type and precursor of vit. A in feed stuff.

4. Destruction of vitamin A in feed through oxidation, peroxidizing effects of rancid fats, length of storage and catalytic effects of trace minerals.
5. Presence of adequate bile in small intestine.

Sources of Vitamin A: In the form of its precursor carotene, vitamin A is found in carrot, yellow maize and green plants. Liver, kidney, buttermilk, cod liver oil and egg yolk are also rich in vitamin A.

Deficiency Symtpoms:

1. Night blindness: Diet deficient in Vitamin A causing impaired rhodopsin formation which makes unable to see in dim light slow, dark adaptation and progress to total blindness.



2. Xerophthalmia: Cattle with prolonged eye symptoms leading to excessive watering, softening and cloudiness of the cornea and development of xerophthalmia, characterized by a drying of the conjunctiva. Constriction of the optic nerve canal may result in blindness in calves.

3. Infertility and abortion: In breeding animals a deficiency may lead to infertility, and in pregnant animals lead to abortion or the production of dead, weak and blind calves. In male there is failure of spermatogenesis.

4. Keratinization of epithelial cell: Vitamin A deficiency causes keratinization of epithelial cell, which results in cold and sinus trouble, diarrhea and formation of calculi in genito-urinary tract and reproductive failure in male and female animals.

5. Disorganized bone growth and irritation of joints are two manifestations of vitamin A deficiencies. In some cases, there is a constriction of the opening through which the optic and auditory nerves pass thereby resulting in blindness and / or deafness.

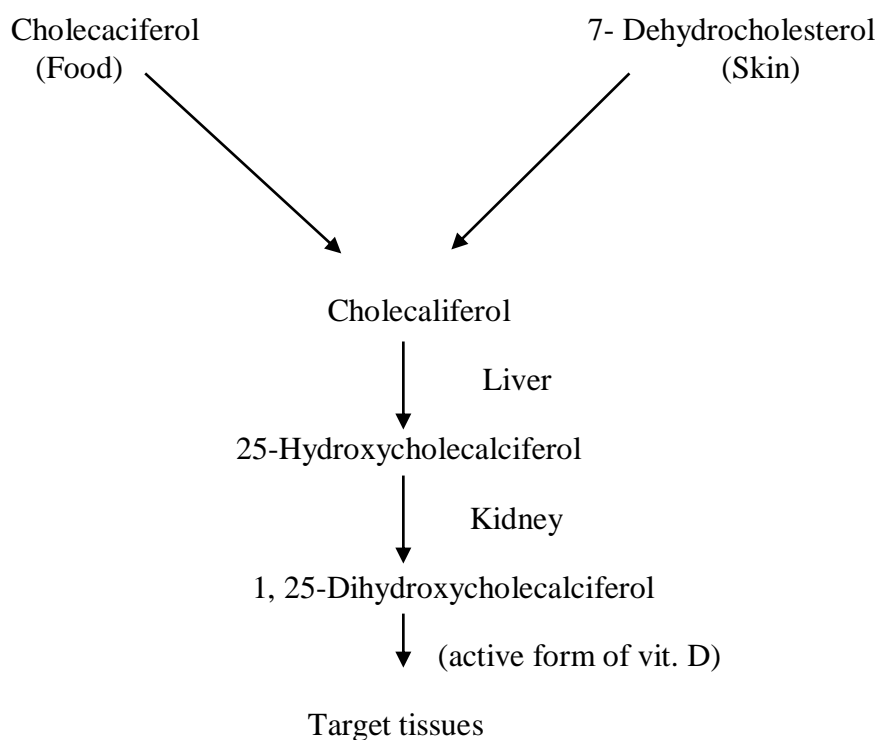
Poultry: Retarded growth, weakness, ruffled plumage and staggering gait are noticed in vitamin A deficiency. In mature birds egg production and hatchability are reduced.

Hypervitaminosis: Excess of vitamin A causes hypervitaminosis in the body resulting in diseases of the nervous system, bone diseases, abnormalities and vomiting. The most characteristic signs of hypervitaminosis are skeletal malformations, spontaneous fracture, internal haemorrhage, degenerative atrophy and fatty infiltration of liver.

Vitamin-D

McCullum in 1922 discovered this vitamin as an antirachitic factor. For nutritional purposes the two most important vitamin D are D₂ (ergocalciferol) and D₃ (Cholecalciferol). Ergocalciferol is produced from ergosterol, which occurs in plants while cholecalciferol is derived from 7dehydrocholesterol. Ultraviolet light is the main power, which converts provitamin into vitamin D. One IU of vitamin D is defined as the activity of 0.025µg crystalline vitamin D₃. The sulfate derivative of vit. D present in milk is a water-soluble form of vitamin. Vit. D₃ is more stable than D₂.

Metabolism



About 50 percent of the dietary vitamin is found in the chylomicrons leaving the digestive tract in the lymph; most of this vitamin finds its way to the liver with the remnants of the chylomicrons. Vitamin D synthesized in the liver diffuses into the blood and picked up by a specific vitamin D bind protein which transports it to the liver, although some may remain free and be deposited in fat and muscle. Dietary vitamins D₂ and D₃ are absorbed through the small intestine and are transported in the blood to the liver where they are converted into 25hydroxycholecalciferol, which is converted into active form as 1, 25-

dihydroxycholecalciferol in kidney and reached to target tissues by blood circulation. Vitamin D transported by protein called transcalciferin or vitamin D binding protein (DBP). Excretion of absorbed vitamin D and its metabolites occurs primarily in faeces with the addition of bile salts.

Functions:

1. Vitamin D plays an important role for the absorption of calcium and phosphorus from gastrointestinal tract, which accounts for the antirachitic properties of vitamin D.
2. It helps in the reabsorption of phosphorus from the kidney tubules.
3. Addition of vitamin D reduces oxidation of citric acid and a high citrate concentration is found in kidney, bone and blood but not in liver.
4. It increases the activity of the enzyme phytase in the intestine.
5. It also stimulates release of calcium rather than up take of calcium by kidney mitochondria.
6. It stimulates incorporation of phosphorus into phospholipids of intestinal mucosa.
7. It promotes normal development of bone, mobilization of calcium from bone to extracellular fluid compartment and biosynthesis of collagen.

Requirements: Animals and humans do not have a nutritional requirement for vitamin-D. Factors, which influence dietary vitamin D requirements, are:

- (i) Amount and ratio of dietary calcium and phosphorus.
- (ii) Availability of phosphorus and calcium
- (iii) Species
- (iv) Physiological factors.

- Lactating cow :30 IU/ kg body weight
- Piglet :100-200 IU / kg feed
- Pig : 200 – 400 IU / kg feed
- Poultry
 - Broiler : 200 ICU / kg feed
 - Layer : 600 ICU / kg feed

Sources of vitamin D: As vitamin D it is present in cod liver oil, kidneys, lungs, egg yolk, liver, milk, fish oil and sun dried grasses. The vitamin is synthesized by the action of ultraviolet rays on the skin of the animals. Heating destroys the rachitogenic activity.

Deficiency Symptoms: Deficiency of vitamin D impairs the following functions.

- (a) Failure of calcium salt deposition in the cartilage matrix.

- (b) Failure of cartilage cells to mature, leading to their accumulation rather than destruction.
- (c) Condensation of proliferating cartilage cells.
- (d) Elongation, swelling and degeneration of proliferative cartilage.
- (e) Abnormal pattern of invasion of cartilage by capillaries.

In young animal vitamin D deficiency results in rickets and retarded growth. Ricket includes skeletal deformities characterized by enlarged junction between bone and cartilages, curvature of the bones, tendency to drag hind legs, beaded ribs, deformed thorax and weakening of muscular tissue and susceptibility to infection. In adult animals vitamin D deficiency causes osteomalacia, where there is reabsorption of bone calcium already laid down.

In poultry a deficiency of vitamin D causes the bone and beak to become soft and rubbery, growth is usually retarded and the legs may become bowed (rubbery legs), ruffled feathers. Egg production is reduced and egg quality deteriorates.

In swine, deficiency of vitamin D causes poor growth, stiffness, lameness and stilted gait, softness of bones, bone deformities, unthriftiness, enlargement and erosion of joints.

Vitamin-E (Tocopherol)

In 1936 Evans and Sure discovered it as an important factor in reproduction of rats. After absorption from the wall of the gastrointestinal tract the vitamin is mainly stored in the liver and to a certain extent in various organs and tissues of the body. The vitamin can pass through the placenta and mother's milk to its offsprings. Vitamin E is a group name, which includes a number of closely related active compounds. Eight naturally occurring forms of the vitamins are known and they can be divided into two groups.

1. Four saturated vitamins that is a, b, g and d tocopherols
2. Four unsaturated vitamins that is a, b, g and d tocotrienols,

α -tocopherol is the most biologically active and most widely distributed. Selenium and vitamin E are interrelated. Both are needed by animals and both have metabolic roles in the body in addition to an antioxidant effect.

Metabolism: It acts as a biological antioxidant with glutathione peroxidase enzyme, which contains selenium. It protects cells against oxidative damage caused by free radicals. Free radicals are scavenged by vitamin E and glutathione peroxidase destroys any peroxide formed before they can damage the cell. Vitamin E is also helpful in development and function of the immune system. Absorption of Vit.E either in free alcohol or esters is facilitated by bile and pancreatic lipase. Most of Vit.E is absorbed as alcohol. Tocopherol passes through placental membranes and mammary gland. Less than 2 percent of dietary Vit.E is transferred from feed to milk. Vit. E is stored through out all body tissues, with highest storage in liver. In the plasma, the vitamin is transported in the low density lipoprotein (LDL) fraction and concentrates in the cell membrane. Highest concentrations are found in the adipose tissue,

other organs and tissues which contain the vitamin include liver, heart, skeletal muscle and adrenal gland.

Functions:

1. Vitamin E acts as an antioxidant at cellular level. Thus for an example it prevent the oxidation of unsaturated fatty acids mostly present in all cell wall components. a-tocopherol is an excellent natural antioxidant.
2. It participates in normal tissue respiration possible by the way of cytochrome reductase system and to protect the lipid structure of mitochondria from oxidative destruction.
3. It aids the normal phosphorylation of creatine phosphate, ATP-which is a high phosphate energy compound in the body.
4. It also involved in the synthesiS of ascorbic acid, ubiquinone (Co-enzyme) and the metabolism of nucleic acid.
5. a-tocopherol exerting a unique influence on structural component of membrane phospholipid.
6. It stimulates the formation of prostoglandin E from arachidonic acid while synthetic vito E had no effect.
7. Vito E inhibits platelet aggregation so help in blood clotting.
8. Relationship of vit E with toxic elements

Both vitamin E and selenium provide protection against toxicity with three groups of heavy metals.

(i) Cadmium and Mercury- Selenium is highly effective in altering toxicities, vit. E has little influence.

(ii) Silver and Arsenic- Vito E is highly effective and selenium at higher doses.

(iii) Lead- Vito E is highly effective in altering toxicity produced by lead.

Requirements: Vit. E requirement of normal animals and humans is approximately 30 ppm of diet. Requirement of vit E is dependent on dietary levels of polyunsaturated fatty acids (PUFA), antioxidants, and sulphur containing amino acids and selenium.

- Lactating cow : 2.5 IU/ kg body weight
- Piglet : 5- 10 IU / kg feed
- Pig : 20 - 30 IU / kg feed
- Poultry
 - Broiler : 5 - 10 IU / kg feed
 - Layer : 5 IU / kg feed

Sources: Vitamin E is widely distributed in foods. Green fodder is good sources of a-tocopherol. Cereal grains are also good sources. Animal products are relatively poor sources of the vitamin. One IU of vitamin E is defined as the specific activity of 1 mg synthetic a-tocopherol acetate.

Deficiency symptoms: The vit E deficiency symptoms in farm animals are muscle degeneration (myopathy). Nutritional myopathy (muscular dystrophy) in cattle affects the skeletal muscles, which is manifested by difficulty in standing, trembling and staggering gait. The animals are unable to rise and weakness of the neck muscles prevents them from raising their heads, also known as white muscle disease. Nutritional myopathy in lambs is called stiff lamb disease (**white muscle disease**).

In pigs vitamin E deficiency diseases are myopathy and cardiac disease known as **mulberry heart disease** (haemorrhagic lesions within the heart that gives characteristic 'mulberry' appearance) and hepatitis dietetica (toxic liver dystrophy).

In poultry, Vit. E deficiency causes following diseases.

1. Exudative diathesis: It is characterised by edema, blackening of affected part, apathy and inappetance.

2. Nutritional encephalomalacia (Crazy chick disease): It is characterised by ataxia, head retraction and cycling with legs.

3. Muscular dystrophy

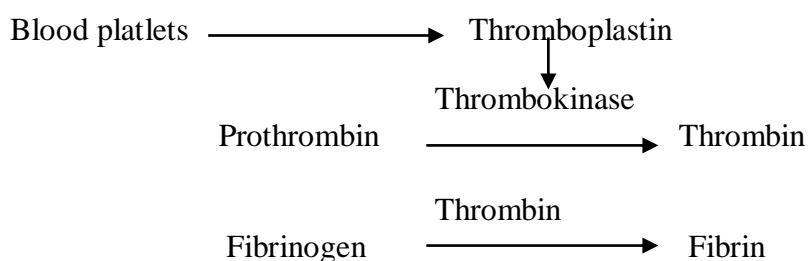
* Vitamin E is non toxic even at higher doses.

Vitamin-K

Vitamin-K was identified in 1935 by Henrik Dam to be an essential factor in the prevention of haemorrhagic symptoms produced in chicks. The new fat-soluble vitamin was designated as vitamin K for the Danish word Koagulation. Vitamin K is synthesized in the body of ruminants by the action of rumen microbes. Bile juice assists in the absorption of this vitamin from the intestine. In dogs there is microbial synthesis in the intestine. The important naturally occurring compounds are vitamin K₁ (Phylloquinone) and vitamin K₂ (Prenylmenaquinone). Vitamin K₃ (Menadione) is a synthetic compound, which is about 3.3 times as potent, biologically, as the naturally occurring vitamin K₂.

Metabolism: Like all fat-soluble vitamins, Vitamin K is absorbed in association with dietary fats and requires the presence of bile salts and pancreatic juice for adequate uptake from the alimentary canal. Vit. K is stored in the liver. Most of the ingested vitamin appears in the chylomicrons entering the lymph. The synthetic form is absorbed directly in the hepatic portal vein and carried to the liver, where it is activated and then released along with the naturally occurring forms of vitamin K. These are carried in the LDL to target sites.

Functions: The vitamin K is necessary for the formation of prothrombin, which is important intermediate of the blood clotting process. Prothrombin must bind to calcium ions before it can be activated as thrombin. If the supply of vitamin K is inadequate, the prothrombin molecule is deficient in a carboxyglutamic acid, a specific amino acid responsible for calcium binding. Thrombin converts the protein fibrinogen in blood plasma into fibrin, which holds blood clots together. There are four blood clotting proteins, which are dependent on vitamin K for their synthesis.



The important biochemical function has been found to be involved in electron transport and in bacteria, oxidative phosphorylation.

Requirements: The daily requirement for most species varies in a range of 2-200 µg vitamin K /Kg body weight. This requirement can be altered by age, sex, strain, antivitamin K factor, disease condition.

Animal	Requirement (mg/kg)
Beef cattle, Dairy cattle, Horse, Goat, Sheep	Microbial synthesis
Swine	0.5
Fish	0.5-1
Chicken	0.5-1

Sources of Vitamin K: Green and leafy fodder, Lucerne, Cabbage, soyabean, liver, fish meal and egg yolks are good source of vitamin K.

Deficiency symptoms:

1. Ruminants and pigs: Under normal conditions vitamin K deficiency have not been reported, but deficiency symptoms, occur when spoiled sweet clover forage is fed. When sweet clover hay undergoes spoilage with certain molds, the coumarin is converted to dicumarol, (Dicumarol passes through the placenta in pregnant animals and new born animals may become affected immediately after birth) an anti-vitamin K and lowers the prothrombin content of the blood and at the same time a bleeding syndrome develops throughout the animal body. This disease is called sweet clover poisoning or bleeding diseases or haemorrhagic sweet clover disease. Prolongation the prothrombin time in absence of liver disease indicates vitamin K deficiency. Initial signs may be stiffness and lameness from bleeding into the muscles and articulations. Haematomas, epistaxis or gastrointestinal bleeding may be observed.

2. Poultry: The symptom of vitamin K deficiency in chicks is a delayed clotting time of the blood; birds are easily injured and may bleed to death. Chicks show anaemia, which in part may be due to loss of blood or to the development of a hypoplastic bone marrow.

3. Human: It is uncommon in humans because wide distribution of vitamin K in plant and animal tissue and microflora of gut synthesize the menaquinones. Newborn infants may suffer because (i) Placenta is a relatively poor organ for maternal-foetal transmission of lipids. (ii) Sterile gastrointestinal tract. (iii) Breast and cow's milk are poor source of vitamin K.

Factors which causes vit. K deficiency:

1. Increase control feeding with less pasture and alfalfa meal.
2. Feeding of solvent extracted soyabean meal and other seed meal and better quality fishmeal.
3. Haemorrhagic gastric ulcers which occurs frequently.
4. Mycotoxins and molds present in the feed.
5. Any antimetabolites (antivitamin K) in the feed.
6. Use of sulfa drugs and different type of antibiotics.
7. Use of slatted floors which reduce the opportunity for coprophagy.

Vitamin K toxicity: Toxic effects mainly related with haematological and circulatory disturbances. The natural vitamin K phyloquinone and menaquinone are non-toxic at very high dose level. Synthetic menadione shows toxic effects at higher level i.e. anaemia, hemoglobinuria and urobilinuria.

Water soluble vitamins: The vitamins of the B complex and vitamin C comprise the water soluble group. Vitamin C is the only member of the water soluble groups that is not a member of the B family and its functions and characteristics are different from the B complex vitamins.

Vitamin B Complex: The vitamins included under this group are water soluble and most of them a component of coenzymes. Ruminants are able to synthesis all the vitamin of B group in the rumen through rumen microorganism. In preruminant calves and mono gastric animals B-complex vitamin should be supplied in the daily ration.

Thiamin (Vitamin B1): Thiamin is considered to be the oldest vitamin. Deficiency of this causes beri-beri in man which is earliest documented deficiency disorder. In 1890 Eijkman, a Dutch investigator, seen polynerritis in chicken that was fed boiled polish rice. Jansen and Donath (1926) succeeded in crystallizing vitamin B in pure form. In 1936 R.R. Williams determined the chemical structure of thiamin. 1 IU of thiamin is the activity of 3 µg thiamine hydrochloride. Thiamin is a complex nitrogenous base containing a pyrimidine ring joined to a thiazole ring. Because of the presence of hydroxyl group at the end of the side chain, thiamine can form esters. The main form of thiamine diphosphate ester (TDP) formerly known as thiamine pyrophosphate (TPP), although thiamine monophosphate and thiamine triphosphate are also occure.

Metabolism: Thiamine is absorbed in duodenum. Ruminants can also absorb free thiamin from the rumen. The horse can also absorb from the caecum. The mechanism of thiamin absorption is both active absorption and simple diffusion. At high levels of intake, most absorption is passive. Absorption may be inhibited by alcohol and by the presence of thiaminases, which are found in some fishes. On absorption, thiamine is phosphorylated to thiamine pyrophosphate (TPP), especially in the liver. The major tissues which contain thiamine are the skeletal muscle, heart, liver, kidney and brain. Thiamin is most poorly stored of all vitamins, is mainly retained in organs with a high metabolic activity. Absorbed thiamine is mainly excreted through urine, faeces and sweat.

Functions: Thiamine diphosphate is a coenzyme involved in the oxidative decarboxylation of pyruvic acid to acetyl coenzyme A, the oxidative decarboxylation of α-ketoglutarate to succinyl coenzyme A in TCA cycle the pentose phosphate pathway. Thiamine involved in the synthesis of the amino acid valine in bacteria, yeast and plants.

Deficiency symptoms: Deficiency of thiamin in human causes beri-beri disease, which is characterised by numbness of the legs, later with pain in muscles, severe exhaustion, finally emaciation and paralysis. The patients have difficulty in breathing, there is an abnormal enlargement of the right side of the heart and decrease in the rate of the heart beat. The most characteristics feature of the disease is the so-called pheripheral neuritis. This is often accompanied by contraction of the feet and severe weakness of the wrists. The brain may be affected under these conditions.

Thiamine deficiency in chick causes poor appetites and consequently emaciated followed by polyneuritis, which is characterised by nerve degeneration paralysis. On thiamin deficient

animals, there is an accumulation of pyruvic acid and its reduction product, lactic acid, in their tissues, which leads to muscular weakness. Ruminants since microbial synthesis occur in the rumen of cattle, sheep and goat and in the caecum of horses are unlikely to show thiamin deficiency. Raw fish contains thiaminase, which destroys the activity of thiamin of food with which the raw fish is mixed. Heat treatment or cooking destroyed the activity of thiaminase. Microbes of gastrointestinal tract of man, pig, poultry, cat and dogs are having thiaminase activity. Due to this reason the thiaminase deficiency occurs.

Sources: Egg yolk, liver, heart, all living cells of the body, milk, meat, green grasses, cereal grains and yeast are rich sources of this vitamin.

Riboflavin (Vitamin B2): It was found in a coenzyme before it was discovered in free form. The discovery of riboflavin goes back to 1929 when Norris and his associates found out an unknown vitamin as a cause of leg paralysis among chicks. In 1934 Gyorgy isolated it from B complex. Independently, Kuhn and Karrer et al. (1935) synthesized it. Riboflavin consists of a dimethyl-isoalloxazine nucleus combined with ribitol. It is yellow, crystalline compound and soluble in water. It is stable in heat, acid and neutral solution but destroyed by alkali. It is unstable to ultraviolet light.

Absorption and metabolism of Riboflavin: Riboflavin is readily absorbed from the small intestine. In the plasma it is carried in association with albumin, which carries both the free vitamin and co-enzyme forms. In the tissues, riboflavin is converted into co-enzymes flavin mononucleotide (FMN) and flavin adenine dinucleotide, which constitute the active groups in a number of flavoproteins.

Functions: Riboflavin is required as part of many enzymes essential to utilization of carbohydrate, protein and fats. Riboflavin in the form of flavin mononucleotides (FMN) and Flavin adenine dinucleotide (FAD) act as the prosthetic group of several enzymes involved in biological oxidation-reduction reaction. Both FMN and FAD act as electron and hydrogen donors and acceptors, which allow them to play a critical role in many oxidation-reduction reactions of metabolic pathways, passing electron to the electron transport chain.

Deficiency symptoms: Deficiency of riboflavin in human produces a cheilosis (severe dermatitis and fissures at the corner of the mouth), angular stomatitis, glossitis and seborrheic dermatitis.

Chick: Deficiency causes slow growth and develops "curled toe paralysis" a specific symptom, caused by peripheral nerve degeneration, in which the chicks walk on their hocks with toe curled inwards. In breeding hen causes poor hatchability, embryonic abnormalities, including characteristic "clubbed down" condition in which the down feathers continue to grow inside the follicle, resulting in a coiled feather.

Sources: It is widely distributed throughout the plant and animal kingdom with very rich sources in anaerobic fermenting bacteria. Milk, liver, kidney and heart are good sources of vitamin.

Niacin (Nicotinamide): American scientist Elvehjem and his associates in 1937 discovered this vitamin as a cure for black tongue disease in dogs and pellagra in human being. It is not destroyed by heat, acid, alkali or by oxidation.

Absorption and metabolism of niacin: There is a rapid absorption of dietary niacin, both by active and passive mechanisms. Once the niacin has been converted to NAD or NADP, it is trapped within the cells and can not diffuse out. NAD and NADP act as hydrogen acceptors in oxidation reactions forming NADH and NADPH.

Functions: Its function in the animal body as coenzyme such as: Diphosphopyridinenucleotide (DPN) or coenzyme I or Nicotinamide adenine dinucleotide (NAD) and Triphosphopyridinenucleotide (TPN) or coenzyme II or NADP (Nicotinamide adenine dinucleotide phosphate (NADP)).

The primary action of these two coenzymes is to remove hydrogen from substrate as a part of dehydrogenase enzymes and transfer hydrogen and/ or electrons to the next coenzyme in the chain or to another substrate which then become reduced.

Deficiency symptoms:

Poultry: The deficiency of vitamin causes "black tongue" characterized by inflammation of the mouth and the upper part of the oesophagus. In chick deficiency produces enlargement of the tibiotarsal joint, a bowing of the legs, poor feathering and slight dermatitis.

Swine: In swine niacin deficiency is known as "pig pellagra" the disease is characterized by poor growth, poor hair and skin condition, occasional vomiting and diarrhoea.

Sources: Nicotinic acid can be synthesized from tryptophan in the body tissues. Niacin is found abundantly in yeast, meat, liver and poultry, groundnut and sunflower meal, milk, tomatoes and varieties of leafy green vegetables. Milk and eggs are almost devoid of the vitamin although they contain the precursor tryptophan. Pyridoxine (Vitamin-B6): Pyridoxine was first defined by Gyorgy (1934) as a part of vitamin B-complex, which is responsible for specific dermatitis in rats. The dermatitis is characterized by scaliness around the peripheral part of the body such as paws and mouth. The vitamin exists in three forms (pyridoxine, pyridoxal and pyridoxamine) which are interconvertible in the body tissues. The amine and aldehyde derivatives are less stable than pyridoxine and are destroyed by heat.

Absorption and metabolism of vitamin B6: The vitamin has to be released from its phosphorylated forms prior to absorption. Once in its free form, absorption is rapid. The liver and muscles are the main sites for pyridoxal phosphate in the body; once it is phosphorylated, the vitamin is trapped in the cell. Pyridoxal is involved in many biological reactions particularly those associated with amino acid metabolism like decarboxylation and transamination etc.

Functions: The active compound pyridoxal phosphate plays an essential role as a coenzyme in the reaction by which a cell transforms nutrient amino acids into mixture of amino acids

and other nitrogenous compounds required for its own metabolism. These reactions involve the activities of transaminases and decarboxylases.

Deficiency symptoms:

Chicks: Pyridoxine deficiency causes acute convulsion, flatter on the pen, usually start kicking, a characteristic posture with wings slightly spread and head resting on ground and generally die.

Pigs: Deficiency of vitamin causes anorexia, roughness of hair coat, fatty infiltration of the liver, goose step type of gait and convulsions:

Pantothenic Acid: Williams and his associates discovered pantothenic acid in 1933 which was derived from Greek word "pantos" means found every where. Pantothenic acid is a dipeptide derivative and the two components of pantothenic acid are dihydroxydimethylbutyric acid and the amino acid, α -alanine.

Functions: Pantothenic acid is the prosthetic group of coenzyme A, an important coenzyme involved in many reversible acetylation reactions in carbohydrate, amino acid and fat metabolism with synthesis of steroids. Coenzyme A may act as an acetyl donor and acetyl acceptor. The vitamin involved in the formation of citrate oxaloacetate in the T.C.A. cycle.

Requirement: For growth and reproduction, the majority of species have a dietary requirement between 5 and 15 mg/ kg. For egg production by chickens the vitamin requirement is very low (2.2 mg/kg) compared to requirement of 10 mg/kg for growth and reproduction.

Sources: Egg yolk, kidney, liver and yeast, groundnuts, pea skimmed milk, sweet potatoes and molasses are the good source of this vitamin.

Deficiency symptoms: It causes severe degeneration of myelin sheath of nervous tissues and affects steroid hormones of adrenal gland.

Poultry: Retarded growth, dermatitis, fatty liver condition, severe edema and subcutaneous haemorrhage are the common symptoms.

Swine: Deficiency symptoms are retarded growth rate and bloody diarrhea. Goose stepping gait, a typical nerve disease that is characterized by movement of hind leg become stiff and jerky, exaggerated legs.

Folic Acid (Folacin): The name folic acid was proposed by Mitchell et al. (1941) for a compound isolated from spinach and shown to be necessary for growth of streptococcus faecalis R. The same pteroyl glutamic acid compound was later on called as folic acid. It contains three distinct components i.e. p-amino benzoic acid (PABA) and pteridine nucleus.

Absorption and metabolism of folate: Most folate in the diet is in the bound form and for optimal absorption glutamates have to be removed to produce the monoglutamate. Most folate is stored in the liver, which is therefore also a good dietary source of folate. Once taken

up by target cells, polyglutamates are formed. These are trapped within the cell and are used as co-enzyme tetrahydrofolic acid. Polyglutamate forms are digested via hydrolysis to pteroyl monoglutamate prior to transport across the intestinal mucosa. The enzyme responsible for the hydrolysis of pteroyl polyglutamate is a "α-carboxy peptidase" known as folate conjugase. Pteroyl polyglutamate is absorbed predominately in the duodenum and jejunum by an active process involving sodium.

Functions: The active form of the vitamin is tetrahydrofolic acid (FH₄), Folic acid is carrier for the single carbon groups, may be either formyl (-CHO), formate (H. COOH), or hydroxymethyl (-CH₂OH). These are metabolically interconvertible in a reaction catalyzed by a NADP dependent hydroxymethyl dehydrogenase. Folic acids are important in the biosynthesis of purine and pyrimidines and in certain methylation reactions. Folic acid is involved in the interconversion of glycine to serine, methylation of ethanolamine to choline and homocysteine to methionine. Folic acid is needed to maintain immune system.

Sources: Fresh leafy green vegetables, cauliflower, cereals and extracted oilseeds meal are rich sources of folic acid.

Deficiency symptoms: Glossitis, gastrointestinal disturbances, diarrhoea and reduced erythropoiesis are common deficiency symptoms. In chicks, poor growth, poor feathering, depigmentation, anaemic appearance and perosis develops. In pigs, macrocytic anaemia, lipopenia, megaloblastic arrest etc. develops.

Biotin: Biotin was first described as the factor protective against "egg white injury". Egg white contains avidine which combined with biotin and prevent its absorption from the intestine. Chemically, biotin is 2-keto-3, 4-imidazolido-2-tetrahydro-thiophene-n-valeric acid. Rat fed large amount of raw egg white developed an eczema-like dermatitis, paralysis of the hind legs, and characteristics alopecia around the eyes, termed spectacle eye. This vitamin has been known by a variety of names including bios factor, vitamin-H, coenzyme R and egg white injury protection factor.

Functions: Biotin serves as the prosthetic group of several enzymes which catalyze fixation of carbon dioxide into organic linkage. Enzymes containing biotin include acetyl coenzyme A carboxylase, propionyl coenzyme A carboxylase and methylmalonyl transcarboxylase. The acetyl coenzyme A carboxylase is required for the initial stage of fatty acid synthesis. In biological systems, it function as the coenzyme for carboxylases, enzyme which catalyse carbon dioxide fixation or carboxylation and also appear to be necessary for synthesis of dicarboxylic acids. Specific biotin dependent reactions in carbohydrate metabolism are 1. Carboxylation of pyruvic acid to oxaloacetic acid 2. Conversion of malic acid to pyruvic acid 3. Interconversion of succinic acid and propionic acid 4. Conversion of oxalosuccinic acid to α-ketoglutaric acid.

Biotin enzymes are important in protein synthesis, amino acid deamination, purine synthesis and nucleic acid metabolism.

Requirements: Biotin is synthesized by many different microorganisms and certain fungi.

Sources: Yeast, milk, cereals and vegetables state, pea nuts and eggs.

Deficiency symptoms: Biotin deficiency is more prevalent in swine and poultry than farm animals. Biotin deficiency could be introduced by giving animal avidin, a protein in raw egg white, which combined with biotin and prevent its absorption from the intestine. The deficiency symptoms are retarded growth and development, falling of hairs (alopecia) and dermatitis characterized by dryness, roughness and brownish exudates, ulceration of skin, transverse of soles and tops of hooves. Recently it has been demonstrated that biotin deficiency is the chief cause of fatty liver and kidney syndrome (FUG) which is characterized by a lethargic with death. Reduced growth rate, disturbed and broken feathering, dermatitis, leg and weak deformities are observed in poultry.

Choline: It is discovered as an essential part of the phospholipid lecithin. The requirement of the choline is met through two ways (1) By ration (2) By the process of transmethylation. Choline can be synthesized in the liver from methionine.

Functions: 1. Choline is a metabolic essential for building and maintaining cell structure. As a phospholipid it is a structural part of lecithin (phosphatidylcholine), certain plasmalogens and the sphingomyelins.

2. It plays an essential role in fat metabolism in the liver. It prevents abnormal accumulation of fat (fatty liver) by promoting its transport as lecithin or by increasing the utilization of fatty acids in the liver itself. Thus choline is referred as lipotropic factor.

3. It is essential for the formation of acetylcholine, which is important in the transmission of nerve impulses.

4. Choline is as a source of three labile methyl groups for formation of methionine by the transmethylation reactions.

Deficiency symptoms: Deficiency of choline causes a deficiency of phospholipid in tissues, which are generally concerned with the transportation and oxidation of fatty acid in the liver. As a consequence, fat accumulates in the liver causing fatty liver. So choline is also concerned with the prevention of perosis or slipped tendon in chicks.

Sources: Natural fat is a good source of choline. Green leafy materials, yeast, egg yolk and cereals are rich sources.

Vitamin-B12 (Cyanocobalmin): Rickes and his associates (1948) isolated this vitamin from the liver as pinkish crystalline substance and name it B12. It was also known as animal protein (APF) and antipernicious anaemia factor (APA). In patients suffering from pernicious anaemia the absorption of B12 from the gastro intestinal tracts is impaired owing to the absence of a specific glycoprotein termed the "Intrinsic factor" normally secreted in the gastric juice. Vitamin B12 has been isolated in several different biologically active forms. Cyanocobalamin, the principle form of the vitamin contains a cyanide group attached to the central cobalt. The cyanide ion may be replaced by a variety of anions e.g., hydroxyl

(Hydroxy cobalamin) or nitrite (nitro cobalamin). The biological actions of these derivatives appear to be similar to that of cobalamin. Vitamin B12 was the last vitamin to be discovered and the most potent of the vitamin. Vitamin B12 is unique that it is synthesized in nature only by the microorganisms.

Absorption and metabolism of vitamin B12: Ingested vitamin B12 has to be combined with intrinsic factor produced by the stomach before it can be absorbed. Vitamin is absorbed from terminal ileum leaving the intrinsic factor behind, in the absence of intrinsic factor there is only minimal absorption of the vitamin by passive diffusion. The metabolic role of vitamin B12 is associated with availability of tetrahydrofolate and metabolism of some fatty acids.

Functions: 1. There is a nutritional inter-relationship with folic acid, B12 vitamin and the metabolism of one-carbon compound.

2. Cobamide coenzyme plays an important role in the transformation of methyl malonyl coenzyme A to succinyl coenzyme A in the metabolism of propionic acid in ruminants.

3. Vitamin B12 and folic acid are required for the formation of DNA whereas vitamin B12 alone being necessary for the synthesis of RNA.

4. Folic acid and B12 are essential for the maturation of R.B.C. Vitamin B12 is necessary in reduction of one carbon compounds of formate and formaldehyde, it participates with folacin in biosynthesis of labile methyl groups. Formation of labile methyl groups is necessary for biosynthesis of purine and pyrimidine bases.

Sources: The origin of Vitamin B12 in nature appears to be microbial synthesis. Foods of animal origin are good sources i.e. meat, kidney, liver, milk, egg, fish, root nodules of certain legumes contain small quantities of Vitamin B12. It is mostly deficient in grains and fodder crops.

Deficiency symptoms:

Human: Vitamin B12 deficiency causes pernicious anaemia.

Poultry: In poultry, poor growth, poor feathering and kidney damage may occur. In adults hatchability goes down.

Pig: Young pig shows poor growth, shows incoordination of the hind legs. Adult pigs show dermatitis, a rough coat, and suboptimal growth.

Ruminants: It has been shown that when the vitamin is deficient, propionic acid can not be metabolized adequately when sufficient cobalt is present in the diet, the sufficient amount of vitamin B12 can be synthesized by the rumen microorganism to meet the animal needs.

Vitamin-C (Ascorbic Acid): The name of vitamin C became quite popular in 18th century when it was found that sailors developed a disease called scurvy after they had been at sea for a period of 4 to 5 months. A British Naval Surgeon Dr. Lind in 1747 told that scurvy disease can be prevented by feeding the juice of citrus fruits in human beings. On this basis it was

discovered as an Anti- scurvy factor. It was first isolated in 1932 by American scientist King and Hungarian scientist S. Gyorgy. Vitamin C is chemically known as L- ascorbic acid and has the following formula.

The vitamin is colourless, crystalline, water soluble compound having acidic and strong reducing properties. In some species it is synthesized from glucose, via glucuronic acid and gulonic acid lactone; the enzyme L-gulonolactone oxidase is required for the synthesis. Glycoascorbic acid acts as an antimetabolite for vitamin C. This enzyme is absent in guinea pig, human, bats, certain birds and fishes. Glycoascorbic acid acts as antimetabolites for vitamin-Co One LV. is the activity of 0.05 mg of ascorbic acid.

Absorption and metabolism: Both forms of vitamin are readily absorbed by active transport and passive diffusion. Vitamin C is absorbed from the small intestine and excreted via urine. There is particularly no storage of this vitamin and secreted in the milk of lactating animals.

Functions:

1. Vitamin C is essential for collagen formation.
2. It aids for the conversion of folic acid to its active form tetrahydrofolic acid.
3. Vitamin C is also involved in the hydroxylation of proline, lysine and aniline, which are important for normal physiology of the animals.
4. It aids iron to stay in reduced state, which is very important for the body and have stimulatory effect on phagocytic activity of leucocytes.
5. It participates in the synthesis of steroid hormones by the adrenal cortex.
6. It involves in the metabolism of lipids, as blood cholesterol level appear to fall with the administration of ascorbic acid and rise due to deficiency of this vitamin.
7. It aids for the conversion of tryptophan to serotonin.
8. It is an antioxidant and used in canning of certain fruits to prevent the oxidative changes which cause darkening.

Sources: Citrus fruits and juices, lemon, tomato, green vegetables, milk, body tissues and plasma are good sources. Dry roughages and concentrates are deficient in ascorbic acid. Guava and Aonla are very rich in vitamin C.

Deficiency symptoms: Man, monkey and guinea pig suffer with severe deficiency of vitamin C is called scurvy. The disease is characterized by weakness, swollen and tenderness of joints, delayed healing of wounds, spongy haemorrhagic friable gum, loose teeth and small haemorrhage which may appear anywhere throughout the body, particularly near the bone and joints and under the skin and mucous membrane due to increase fragility of the blood

capillaries. Resistance to infection is reduced. Symptoms of oedema, emaciation and diarrhoea are also appeared.

Unidentified vitamins: The discovery of vitamins is of course a very great research in the field of nutrition. If all the vitamins discovered so far are included in the ration of poultry, even then they may lack in proper development. When these birds are offered fish meal, penicillium mycelium meal, molasses, green leaves and dried skimmed milk. They have a better growth and development. It clearly indicates that these substances contain certain nutrients which are still to be identified. Hence they were called as unidentified vitamins or growth factors. Certain factors which appear to be of some significance in poultry nutrition are the grass factor, whey factor and fish factor. The evidence for these has been obtained from growth responses in feeding trials and from hatchability studies.

Hypervitaminosis: It is the name given to pathological conditions resulting from an overdose of vitamins. In natural conditions, it is unlikely to occur, until the synthetic vitamins are not added in the ration. Clinical sign of hypervitaminosis A in young chicks include loss of appetite, poor growth, diarrhoea, encrustation around the mouth and reddening of the eyelids. In pigs rough coat, Scaly skin, hyperirritability, haemorrhages, periodic tremors and even death. Excessive intakes of vitamin D cause abnormally high levels of calcium and phosphorus in the blood, which result in the deposition of calcium and phosphorus in the blood, which result in the deposition of calcium salts in the arteries and organs. Hypervitaminosis K showed a depression in growth and anaemia as toxic symptoms.