Poultry Nutrition

Department of Animal Resource Class 4 7.11.2022 **Sixth lecture**

The Vitamins

Vitamins are active substances, essential for life of birds and normal metabolism considered critical to optimum health as well as normal physiological functions such as growth, development, maintenance and reproduction.

Vitamins are regarded as a group of complex essential organic micronutrients that must be supplied in the diet which are added to the feed additional to the existence in the diet ingredients. As most vitamins cannot be synthesized by poultry in sufficient amounts to meet physiological demands, they must be obtained from the diet. Vitamins are present in many feedstuffs in minute amounts and can be absorbed from the diet during the digestive process.

Poultry require supplemental dietary Vitamins since common feed ingredients used in poultry production do not provide adequate quantities to meet minimum requirements. Usually the egg contains vitamins in adequate quantity for the embryo.

Since poultry farming is particularly dynamic and has to respond to changes in animal performance and housing systems, and also to the growing demands from consumer groups, the vitamin supplementation levels should be reviewed on a frequent basis in order to keep the balance between the costs of supplemental vitamins and the risk of losses from vitamin deficiencies and sub-optimal performance. Poultry birds are dependent on their diet for vitamins to a much degree than are ruminants?. This is because a bird has no rumen.

Fat and Water-soluble vitamins with Synonym Names

Vitamin	**Synonym**		**Synonym**
Fat soluble		**Water soluble**	
Vitamin A_1	Retinol	Vitamin B_1	Thiamin ($B_{1)}$
Vitamin A_2	Dehydroretinol	Vitamin B_2	Riboflavin
Vitamin D_1	Ergocalciferol	Vitamin B_3	Niacin, Vitamin pp
Vitamin D_3	Colecalcifirol	Vitamin B_6	Pyridoxol, pyridoxal, pyridoxamine
Vitamin E	Tocopherol	Vitamin B_5	Pantothenic acid
Vitamin K_1	Phylloquinone	Vitamin H	Biotin
Vitamin K_2	Menaquinone	Vitamin B_c, vitamin M	Folacin
Vitamin K_3	Menadione	Vitamin B_{12}	Cobalmin
		Choline	Gossypine
		Vitamin C	Ascorbic acid

Functions of Vitamins

1. Vitamins are involved in over 30 metabolic reactions in cellular metabolism
2. Critical to the efficiency of the Krebs/ Citric acid cycle.
3. To obtain optimum health.
4. To enable the bird to efficiently utilize all other nutrients in the feed.

Classifications of vitamins

Classically, vitamins have been divided into two groups based on their solubility's in fat solvents or in water. These two groups are:

A. Water-soluble vitamins (WSVs)

Vitamins of the B complex and vitamin C are classified as water-soluble. Some of **WSVs** also contain nitrogen, sulfur or cobalt. Their absorption usually occurs via simple diffusion.

Vitamin C is not required in outdoor birds but should be given to the chicken when reared in poultry house under stressed conditions; this is attributed to that the **Vit.C** syntheses in the body under stress conditions is low therefore supplementation in the feed or in water will be necessary. Except for **vit.B_{12}**, **WSVs** are not stored in the body and excesses are rapidly excreted mainly in urine. A continual dietary supply of the **WSVs** is needed to avoid the **WSVs** vitamins participate in catalytic functions or act as control mechanisms of the metabolism, as coenzymes.

Choline is only tentatively classified as one of the B-complex vitamins. Unlike other B-vitamins, choline can be synthesized in the body, is required in large amounts, and apparently functions as a structural constituent rather than as a coenzyme.

B. Fat-soluble vitamins (FSVs)

The fat-soluble group includes the vitamins A, D, E and K. Fat-soluble vitamins are found in feedstuffs in association with lipids. Those vitamins are absorbed along with dietary fats, apparently by mechanisms similar to those involved in fat absorption. **FSVs** consist only of carbon, hydrogen, and oxygen. **FSVs** are stored in appreciable amounts in the animal body. Excesses of **FSVs** are excreted via the bile. Excesses of **FSVs A** and **D** can cause serious problems. **FSVs** vitamins have specific functions in the development and maintenance of tissue structures.

Vitamin sources

Vitamins originated primarily in plant tissues and in bird tissue only as a consequence of consumption of plants. Two of the four **FSVs**, vitamins A and D, differ from the water-soluble B vitamins in that they occur in plant tissue in the form of a provitamin (precursor) of the vitamin, which can be converted into a vitamin in bird's body. No provitamins are known for any water-soluble vitamins.

Vitamin requirements

Chicken depend on food sources for their requirement of vitamins. They are also more susceptible to vitamin deficiencies because:

a. The microorganisms in caeca of these birds do not provide any vitamin synthesis, instead compete with the host for dietary vitamins;

b. Due to higher metabolic rate, chicken have a higher need for vitamins which act as the "spark plugs" of the vital metabolic reactions in the body

c. Intensively kept chicken undergo many stresses which increase their vitamin requirements.

Factors affecting vitamin requirements

1. As the selected and genetically modified strains need more vitamins because they have higher growth

2. Calories in the ration

An increase in carbohydrates increases the need for vitamin B1 and niacin by 10-15% and an increase in fat increases the need for vitamin E and choline by 20-40%

3. Temperature

High temperature leads to a decrease in the effectiveness of most vitamins, especially vitamin C, so it must be added during hot weather

4. Method of rearing

The animal raised in the battery needs a group of vitamin B complex and vitamin K, because the litter contains bacteria that help in bacterial digestion inside the stomach

5. Manufacturing method

Extra heat of conditioning during poultry feed processing decrease in the rate of utilization of vitamins, as well as pressing on high heat leads to the loss of vitamins.

6. Storage

The longer the storage period, the greater the loss in the vitamin, so an antioxidant should be added in the case of long-term storage

7. Infection with intestinal parasites

Infection with coccidiosis and intestinal parasites leads to vitamin damage, especially vitamin A and vitamin K. Therefore, when infected, vitamins must be doubled.

8. Add antibiotics

If the amount of antibiotic is small, it reduces the number of harmful bacteria. But if the antibiotic is put in a large dose, it sterilizes the intestines and kills all the harmful and beneficial bacteria that help in the formation of vitamin B and vitamin K, thus it is necessary to add a biological preparation and double the amount of vitamins.

9. Fungal poisoning

Fungal poisoning leads to a deficiency of vitamin E and vitamin K, so the diet contaminated with fungi must be changed and an antitoxin should be used and an additional amount of these vitamins should be provided.

10. Chemicals

Sulfites in the diet and water destroys vitamins A and B (niacin).

11. Light

Exposure to ultraviolet and infrared rays present in the light affects vitamins, especially vitamin B complex, so the ration should not be exposed to light for a long time, and pharmaceutical companies should also produce these vitamins in dark packages.

The contamination of the feed with **mycotoxins** and **vitamin antagonists** can limit or even block the action of certain vitamins. Any of these factors, ranging from the animals' **genetic background** and **health status** to **management programmes** and the **composition of the diet**, can separately or collectively affect the need for each vitamin.

Vitamin deficiencies or excesses

Classical deficiency signs and ***non-specific parameters*** (e.g. lowered production and reproduction rates) are associated with vitamin deficiencies or excesses. Vitamin nutrition should no longer be considered important only for preventing deficiency signs but also for optimizing animal health, productivity and product quality.

Vitamin supplementation

When supplementing vitamins, several factors have to be taken into consideration.
These factors include:

1) Breed
2) Sex
3) Management practices
4) Development status of the bird
5) Stress
6) Diseases
7) Factors related to the feed as ingredients
    1) Energy level
    2) Processing
    3) Storage
    4) Vitamin sources

Besides production rates, other parameters are also presently evaluated to determine vitamin requirements, such as immunity, animal welfare, carcass characteristics, microbiological analysis, etc.

Supplementation with higher levels than the minimum recommendations will result in higher production performance and carcass quality, and better health and welfare of the birds.

There has, however, been an increase in the vitamin requirements for specific metabolic processes, as the immune responses related to performance expectations of broilers under high stocking densities in commercial rearing conditions.

Poultry vitamin requirements are dynamic and may change with the development of new high performance breeds modifications in nutrition or production systems with improved understanding of vitamin functions. Therefore, vitamin recommendation guidelines need to be updated regularly.

It is now well recognized by the feed industry that the minimum dietary vitamin levels required to prevent clinical deficiencies may not support optimum health, performance and welfare of poultry. The productivity of poultry farming continues to grow through genetic improvement of the breeds and through modifications in nutrition, management and husbandry, which considerably increase the demand for vitamins.

I**ntake** and **utilization of vitamins**

Intake and utilization of vitamins from natural sources is unpredictable owing to differing contents of vitamins and variable vitamin bioavailability in the feedstuffs dependent on:

1. Growing climate
2. Harvesting time of crops,
3. Processing
4. Storage conditions of feed ingredients

The requirement of those vitamins increase in certain conditions like:

1. Transportation of chicks from the hatchery to the poultry house
2. Before and after the vaccination
3. At the infections (illness) of the bird
4. After antibiotic administration in which affect the micro flora also in the intestine
5. When the temperature is high or at high density of birds (both cause stress to the bird)
6. Ate high rate of production (eggs or rapid growth)

Optimum vitamin nutrition OVN

The Optimum Vitamin Nutrition (OVN) concept takes **unreliable and negative factors**, such as the variability in natural vitamin content in feed ingredients, poor environmental conditions and stresses of various origins into account and provides practical recommendations for vitamin supplementation.

Supplementing feed with vitamins in accordance to OVN is the **safest** and most **economical way** to maintain a high level of welfare and health, achieve good performance and achieve high product quality

Variables to be analyzed

Besides the production rates there are also important variables to be analyzed as:

1. Immunology,
2. Interaction between vitamins and other nutrients
3. Meat quality and food safety,
4. Performance under stress conditions
5. The effect of supplementation of the parents diet on chick quality

Example on vitamin antagonism

Increase of Thiamine (**B1**) intake cause pyridoxine (B6) deficiency

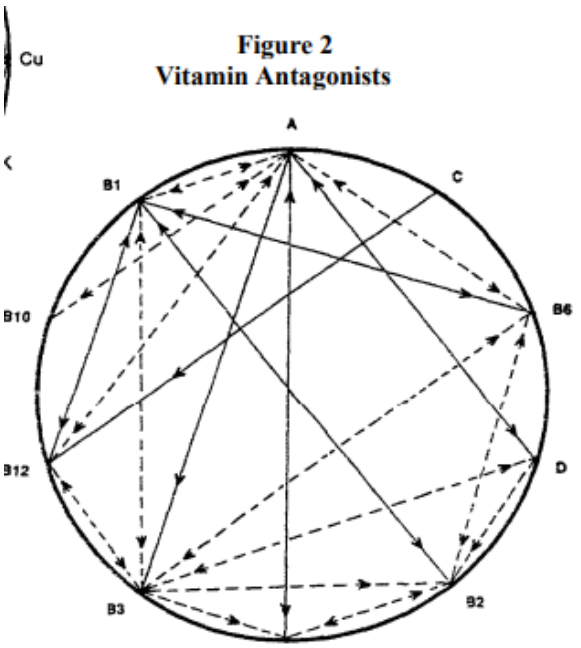
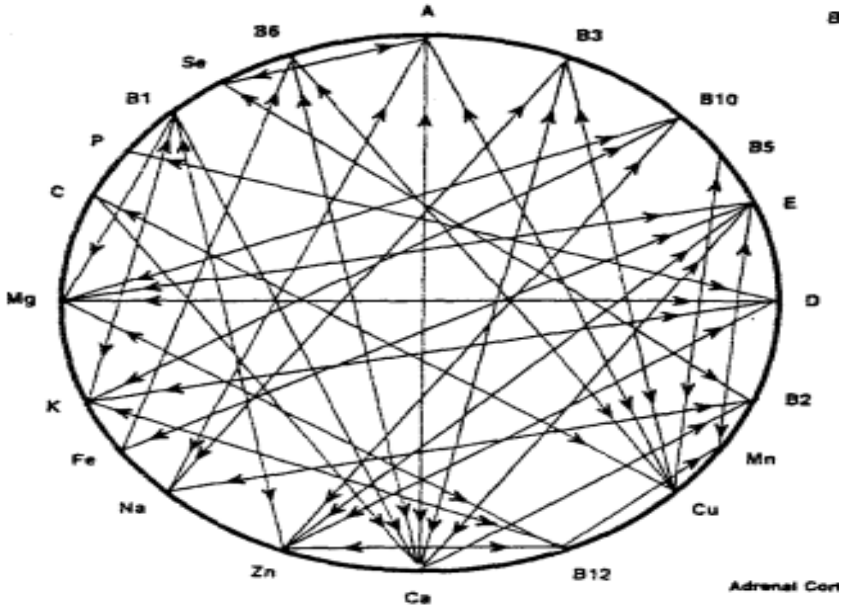


Figure 2
Vitamin Antagonists

Figure 3
Vitamin-Mineral Antagonists



Besides deficiency signs and/or weight gain and feed conversion, new parameters are being evaluated to determine the vitamin requirements of broilers. These include immune response, animal welfare, and quality of the final product (meat and eggs). The goal is to improve the aspect and nutritional value of the product, and lengthen its shelf-life. Higher vitamin levels have been used in the diet of broilers to compensate for variations in intake, bioavailability of the vitamins in the diet, anti-nutritional factors of the feedstuffs, stress (e.g. temperature, stocking density, management practices, diseases), etc. These are some of the other factors that can prevent the minimum requirements of the birds from being met.

Relation of vitamins to other nutrients

The amino acid Tryptophan can be converted into niacin (Vit B3) in bird's body, thus the importance of this amino acid should be considered in poultry diet.

FACTORS RESULTING IN INADEQUATE DIETARY VITAMIN INTAKES

1. Agronomic Effects and Harvesting Conditions
2. Processing and Storage Effects
3. Reduced Feed Intake
4. Vitamin Variability and Insufficient Analysis
5. Vitamin Bioavailability
6. Computerized Least-Cost Feed Formulations

A. Agronomic Effects and Harvesting Conditions

Vitamin levels will vary in feed ingredients because of crop location, fertilization, plant genetics, plant disease and weather. Harvesting conditions often play a major role in the vitamin content of many feedstuffs. Vitamin content of corn is drastically reduced when harvest months are not conducive to full ripening. In one study, vitamin E activity in blight corn was 59% lower than in sound corn, and activity of the vitamin in lightweight corn averaged 21% lower than in sound corn. It is reported that the rate of oxidation of natural tocopherol was higher in high moisture corn than in low-moisture corn due to increased peroxidation of the lipid.

B. Processing and Storage Effects

Many vitamins are delicate substances that can suffer loss of activity due to unfavorable circumstances encountered during processing or storage of premixes and feeds. Stress factors for vitamins include humidity, pressure (pelleting), friction (abrasion), heat, light, oxidation-reduction, rancidity, trace minerals, pH and interactions with other vitamins, carriers, enzymes and feed additives. Humidity is the primary factor that can decrease the stability of vitamins in premixes and feedstuffs. Humidity augments the negative effects exerted by choline chloride, trace elements and other chemical reactions that are not found in dry feed. Corn is often dried rapidly under high temperatures, resulting in losses of vitamin E activity and other heat-sensitive vitamins. When corn was artificially dried for 40 minutes at 88°C, losses of -tocopherol averaged 19%, and when corn was dried for 54 minutes at 107°C, losses averaged 41%. After 3 months of storage, the vitamin A retention was 88% under low temperature and low humidity, 86% under high temperature and low humidity and 2% under high temperature and high humidity. They concluded that humidity was significantly more stressful than temperature. Vitamins that undergo friction or are mixed and stored with minerals are subject to loss of potency. Friction is an important factor because it erodes the coating that protects several vitamins and reduces vitamin crystals to a smaller particle size. Hazards to vitamins from minerals are abrasion and direct destruction by certain trace elements, particularly copper, zinc and iron; manganese and selenium are the least reactive. Some vitamins are destroyed by light. Riboflavin is stable to most factors involved in processing; however, it is readily destroyed by either visible or ultraviolet light. VitaminB6, vitamin C and folacin can also be destroyed by light. While pelleting generally improves the value of energy and protein carriers in a feed, this is not true for most vitamins. During pelleting of feeds, four elements destructive for a number of vitamins are applied in combined action: friction, heat, pressure and humidity. Increasing the pelleting temperature or conditioning time generally enhances redox reactions and destroys

vitamins. It is reported that vitamins A, D, C and thiamin are most likely to show stability problems in pelleted feeds. Feed manufacturers have increased pelleting temperatures for all animal feeds in order to control Salmonella organisms and increase digestibility and are using steam pelleting, pre pelleting conditioners and feed expanders, which lead to increased vitamin degradation.

C. Reduced Feed Intake

When feed intake is reduced, vitamin allowances should be adjusted to ensure adequate vitamin intake for optimum performance. Restricting feed intake practices and/or improved feed conversion will decrease dietary intake of all nutrients, including vitamins. Restricted feeding of broiler breeders and turkey breeder hens may result in marginal vitamin intake if diets are not adequately fortified. Reduced feed intake may also result from stress and disease. Use of high-energy feeds such as fats to provide diets with greater nutrient density for higher animal performance requires a higher vitamin concentration in feeds. Poultry species provided diets ad libitum consume quantities sufficient to meet energy requirements. Thus, vitamin fortification must be increased for high-energy diets because animals will consume less total feed. Feed consumption was compared in broilers receiving metabolizable energy ranging from 2,800 to 3,550 kcal/kg of feed. Feed and vitamin consumption were each 19.1% lower in broilers consuming the diet with greater energy density compared to those consuming the lowest energy diet. Ambient temperature also has an important influence on diet consumption, as animals consume greater quantities during cold temperatures and reduced amounts as a result of heat stress. Vitamins, as well as other nutrients, must therefore be adjusted to reflect changing dietary consumption.

D. Vitamin Variability and Insufficient Analysis

Tables of feed composition demonstrate the lack of complete vitamin information, with vitamin levels varying widely within a given feedstuff. Variability of vitamin content within ingredients is generally large and difficult to quantify and anticipate. It is well recognized that vitamin levels shown in tables of vitamin composition of feedstuffs represent average values and that actual vitamin content of each feedstuff varies over a fairly wide range. Feed table averages are often of little value in predicting individual content of feedstuffs or bioavailability of vitamins. Vitamin E content of 42 varieties of corn varied from 11.1 to 36.4 IU per kg, a 3.3 fold difference. For 65 samples of corn, biotin varied between 0.012 and 0.072 ppm, a 6 fold difference.

E. Vitamin Bioavailability

Even accurate feedstuff analyses of vitamin concentrations do not provide bioavailability data needed for certain vitamins. Bound forms of vitamins in natural ingredients often are unavailable to animals. Bioavailability of choline, niacin and vitamin B is adequate in some feeds but limited or variable in others. For example, bioavailability of choline is 100% in corn but varies from 60 to 75% in soybean meal; that of niacin is 100% in soybean meal but zero in wheat and sorghum and varies from 0 to 30% in corn; that of vitamin B is 65% in soybean meal and varies from 45 to 56% in corn. For alfalfa meal, corn, cottonseed meal and soybean meal, bioavailability of biotin is estimated at 100%. However, biotin availability is variable for other feedstuffs, for example, 20 to 50% in barley, 62% in corn gluten meal, 30% in fish meal, 20 to 60% in sorghum, 32% in oats and 0 to 62% in wheat. Many of the earlier established requirements for poultry relied heavily on purified feed ingredients.

F. Computerized Least-Cost Feed Formulations

Vitamins are not usually entered as specifications in computerized feed formulations. Therefore, vitamin-rich feedstuffs—such as alfalfa, distiller's solubles or grains; brewer's grains; fermentation products; and meat, milk and fish by-products—are often excluded or reduced when least-cost feed formulations are computed. The resulting least-cost diet consisting of a grain and soybean meal is usually lower in vitamins than a more complex one containing more costly vitamin-rich feeds.

FACTORS AFFECTING VITAMIN REQUIREMENTS AND VITAMIN UTILIZATION

A. Physiological make-up and production function

Vitamin needs of poultry depend greatly on their physiological make-up, age, health, and nutritional status and function, such as producing meat or eggs. Breeder hens have higher vitamin requirements for optimum hatchability, since vitamin requirements for egg production are generally less than that for egg hatchability. Higher levels of vitamins A, D and E are needed in breeder hen diets than in feeds for rapidly growing broilers. Selection for faster growth rate may allow animals to reach much higher weights at much younger ages with less feed consumed. It's concluded that since genetic potential has improved at the rate of 0.8% feed conversion yearly and most of the NRC vitamin requirement data is 20 to 40 years old, vitamin requirements determined several decades ago may not apply to today's poultry.

B. Disease or Adverse Environmental Conditions

Results of research showed that as degree of stress increased, bird performance declined. Furthermore, although the highest level of vitamins did not completely overcome the detrimental effect of stress.

Intensified production increases stress and subclinical disease level conditions because of higher densities of animals in confined areas. Stress and disease conditions in animals increase the basic requirement for certain vitamins. A number of studies indicate that nutrient levels that are adequate for growth and egg production may not be adequate for normal immunity and for maximizing the animal's resistance to disease. Higher than recommended levels of vitamin A to layer chickens under heat stress was beneficial to laying performance and immune function. High levels of vitamin E maintained antibody production in chicks. Vitamin E stimulates live turkeys' immune responses (via lymphocytes). Diseases or parasites affecting the gastrointestinal tract will reduce intestinal absorption of vitamins, both from dietary sources and those synthesized by microorganisms. If they cause diarrhea or vomiting this will also decrease intestinal absorption and increase needs. Vitamin A deficiency is often seen in heavily parasitized animals that supposedly were receiving an adequate amount of the vitamin. Mycotoxins are known to cause digestive disturbances such as vomiting and diarrhea as well as internal bleeding, and interfere with absorption of dietary vitamins A, D, E and K. In broiler chickens moldy corn (mycotoxins) has been associated with deficiencies of vitamins D (rickets) and vitamin E (encephalomalacia) in spite of the fact that these vitamins were supplemented at levels regarded as satisfactory. Mortality from fowl typhoid (Salmonella gallinarum) was reduced in chicks fed vitamin levels greater than normal. Vitamin E supplementation at a high level decreased chick mortality due to Escherichia coli challenge from 40 to 5%.

Coccidiosis produces a triple stress on vitamin K requirements as follows:

(1) Coccidiosis reduces feed intake, thereby reducing vitamin K intake;

(2) Coccidiosis injures the intestinal tract and reduces absorption of the vitamin; and

(3) Treatment with sulfaquinoxaline or other coccidiostats causes an increased requirement for vitamin K.

The current NRC (1994) vitamin K requirement for growing chicks is 0.5 mg per kg of diet. However, it's concluded that as much as 8 mg of vitamin K per kg of diet was needed for chicks with coccidiosis. A number of important aspects of footpad dermatitis in turkeys are summarized. While wet litter is a leading cause, and will increase both the severity and incidence, biotin insufficiency can be a primary contributing factor. Biotin is active in skin formation and maintenance, and a deficiency causes abnormal keratinization and cornification of the epidermis, leading to low tensile strength and more skin lacerations. Perhaps more importantly, biotin deficiency can slow the process of wound healing. Ironically, the biotin requirement is highest for turkeys, the specie that suffers the greatest from footpad dermatitis. Although several factors influence the actual biotin requirement, two conclusions were drawn: biotin is most effective when fed at high levels early in the life of the poultry, and 250-400 mg biotin/ton diet hinders footpad dermatitis.

C. Heat Stress

The decreased nutrient intake by poultry at high temperatures also has repercussions on the intake of vitamins and metabolism such as for vitamin A, E and C which play important roles in performance and immune function. Supplementation of these vitamins is helpful for maintaining performance and immune function of heat stressed birds. High dietary vitamin E provided to broilers reduced negative effects of heat stress (32°C). Vitamins A (15,000 mg/kg) + E (250 mg/kg) reduced heat stress related decreases in broilers. Both vitamins E (65 mg/kg) and C (1000 mg/kg) enhanced in vitro lymphocyte proliferative responses of heat stressed hens. High dietary supplemental vitamin E (250 mg/kg) is beneficial to egg production at high temperatures. In addition, a recent study with broilers under heat stress conditions found increased levels of vitamin E (100-200 IU/kg) to improve live performance of birds, as well as the digestibility of crude protein, ether extract and gross energy. Optimum responses in growth, feed efficiency and/or livability in broilers under heat stress seem to occur with supplements of vitamin C. For laying hens under stress, there are improvements in livability, food intake, egg production and egg quality with vitamin C concentrations in the range of 250-400 mg/kg. Production responses in poultry confirm that dietary supplementation with vitamin C generally only benefits birds under stress. Significant improvements in live performance, digestibility of nutrients and immunity measurements can occur in poultry raised under heat stress and supplemented with 200 ppm vitamin C. And when maintained at cold temperatures (7oC), laying hens benefited from 250 ppm vitamin C with a reduction in N excretion and an increase in N retention.

D. Vitamin Antagonists

Vitamin antagonists (antimetabolites) interfere with the activity of various vitamins. The antagonist could cleave the metabolite molecule and render it inactive, as occurs with thiaminase and thiamin; it could complex with the metabolite, with similar results, as happens between avidin and biotin; or by reason of structural similarity it could occupy reaction sites and thereby deny them to the metabolite, as with dicumarol and vitamin K. The presence of vitamin antagonists in animal diets should be considered in adjusting vitamin allowances, as most vitamins have antagonists that reduce their utilization. Some common antagonists are as follows:

1. Thiaminase, found in raw fish and some feedstuffs, is a thiamin antagonist.

2. Dicumarol, found in certain plants, interferes with blood clotting by blocking the action of vitamin K.

3. Avidin, found in raw egg white, and streptavidin, from Streptomyces molds, are biotin antimetabolites.

4. Rancid fats inactivate biotin and destroy vitamins A, D, and E and possibly others.

5. Mycotoxins increase requirements for fat-soluble and other vitamins (e.g. biotin, folic acid and possibly others).

E. Use of Antimicrobial Drugs

Some antimicrobial drugs will increase vitamin needs of animals by altering intestinal microflora and inhibiting synthesis of certain vitamins. Certain sulfonamides may increase requirements of biotin, folacin, and vitamin K and possibly others when intestinal synthesis is reduced.

F. Levels of Other Nutrients in the Diet

Level of fat in the diet affects absorption of the fat-soluble vitamins A, D, E and K, as well as the requirement for vitamin E and possibly other vitamins. Fat-soluble vitamins may fail to be absorbed if digestion of fat is impaired. Polyunsaturated fatty acids (PUFA) and oxidized sources of fats can influence individual vitamin allowances. For example, high dietary PUFA increases the vitamin E requirements by 3 IU per g of PUFA. Many interrelationships of vitamins with other nutrients exist and, therefore, affect requirements. For example, prominent interrelationships exist for vitamin E with selenium, vitamin D with calcium and phosphorus, choline with methionine, and for niacin with tryptophan.

Vitamin deficiency symposiums

Deficiency diseases or syndromes will result if vitamins are absent from the diet or improperly absorbed or utilized. Deficiency of one or more vitamins can lead to multiple metabolic disorders, resulting in decreased productivity, delayed growth, reproductive problems and/or decreased immunity.

Vitamin deficiencies are most commonly due to inadvertent omission of a vitamin premix from the birds' diet. Multiple signs are therefore seen, although in general, problems with deficiencies of the

B vitamins appear first. Because there are some stores of fat-soluble vitamins in the body, it often takes longer for these deficiencies to affect the bird.

Treatment and prevention rely on an adequate dietary supply, usually microencapsulated in gelatin or starch along with an antioxidant. Vitamin destruction in feeds is a factor of time, temperature, and humidity. For most feeds, vitamin efficiency is little affected over 2-month storage within mixed feed.

Optimum vitamin nutrition of laying hens

In particular, B vitamins are required for efficient nutrient utilization, and together with vitamin A are important to support the hens' metabolic activity for maintenance and high laying performance. Furthermore, both vitamins C and E improve the birds' resistance to stress, and help sustain health and longevity. Specific benefits related to superior egg quality can be achieved if supra-nutritional levels of vitamin E are added to the feed of laying hens. And finally, considerable vitamin D activity is required in order to support an adequate skeletal development and to avoid leg problems of various origins.

The optimum vitamin supplementation levels are given in the table below.

Vitamins (added to air-dried feed)	Replacement pullets	Laying hens
Vitamin A (IU/kg)	7 000–10 000	8 000–12 000
Vitamin D3 (IU/kg)	1 500–2 500	2 500–3 500[1]
Vitamin E (mg/kg)	20–30	15–30[2]
Vitamin K3 (mg/kg)	1–3	2–3
Vitamin B1 (mg/kg)	1.0–2.5	1.0–2.5
Vitamin B2 (mg/kg)	4–7	4–7
Vitamin B6 (mg/kg)	2.5–5.0	3.0–5.0
Vitamin B12 (mg/kg)	0.015–0.025	0.015–0.025
Niacin (mg/kg)	25–40	20–50
Pantothenic acid (mg/kg)	9–11	8–10
Folic acid (mg/kg)	0.8–1.2	0.5–1.0
Biotin (mg/kg)	0.10–0.15	0.10–0.15
Vitamin C (mg/kg)	100–150	100–200
Hy•D® (25-OH D3) (mg/kg)	0.069[3]	0.069[3]
Choline (mg/kg)	200–400	300–500

[2] Under heat stress conditions: 200 mg/kg

[3] Local legal limits of total dietary vitamin D activity need to be observed

Broiler vitamin supplementation

Broiler performance has improved enormously in recent decades. As a result, nutrition patterns have also changed, Based on the genetic progress of the birds. Broilers with higher genetic potential for growth, with more than 20% improvement in feed conversion (higher weight gain in a short period) and a 87% increase in daily weight gain (from 26.8 g/day in 1970 to 50 g/day in 2000). As for most nutrients, the vitamin requirements for broilers have probably undergone very few modifications in the past 30-40 years, as the nutrient levels required for maintenance are practically fixed and the composition of muscles/tissues are resistant to changes. Modern breeds have a higher growth and production rate, and have higher nutritional requirements to express their genetic potential.
Proper vitamin supplementation levels are needed. The Optimum Vitamin Nutrition (OVN) concept is a useful tool for supplying the correct amount of vitamins to broiler diets. Literature shows a large variation in vitamin levels used in commercial supplements for broilers. It is for this reason that there is great interest in new studies to determine the levels that provide the best economic return, without interfering with the production performance of birds. Recent studies have shown vitamin levels above minimum requirements may optimize genetic potential and improve immune status. Vitamin requirements of today's broiler breeds are related to optimum performance, final quality of the product, and financial return.

For read only****

Vitamin A

Figure 1 shows a normal chick together with a chick of the same age deficient in vitamin A. Vitamin A is needed by chicks for growth and by adult birds to maintain good health, production, and hatchability. A severe vitamin A deficiency gives birds the appearance of having a form of chronic respiratory disease, a cold, or other such diagnosis. There is a discharge from one or both nostrils, swelling of the face around the eyes, and the eyelids may be glued together by a discharge from them; yellowish white, round, cheesy patches about the size of a pinhead may be seen in the mouth and throat. These same symptoms are also caused by some common poultry virus or disease. It is important for those of you that raise turkeys to note that twice as much vitamin A is required by this bird compared to a chicken.

Vitamin A Deficiency

Adult birds, depending on liver storage, could be fed a vitamin A-deficient diet for 2–5 mo before signs of deficiency develop. Eventually birds become emaciated and weak with ruffled feathers. Egg production drops markedly, hatchability decreases, and embryonic mortality with incubated eggs increases. As egg production declines, there will likely be atretic follicles in the ovary, some of which show signs of hemorrhage. A watery discharge from the eyes may also be noted. As the deficiency continues, milky white, cheesy material accumulates in the eyes, making it impossible for birds to see (xerophthalmia). The eye, in many cases, may be destroyed.

The first lesion usually noted in adult birds is in the mucous glands of the alimentary tract. The normal epithelium is replaced by a stratified squamous, keratinized layer. This blocks the ducts of the mucous glands, resulting in necrotic secretions. Small, white pustules may be found in the nasal

passages, mouth, esophagus, and pharynx, and may extend into the crop. Breakdown of the mucous membrane may allow pathogenic microorganisms to invade these tissues and cause secondary infections.

Depending on the quantity of vitamin A passed on from the breeder hen, day-old chicks reared on a vitamin A-deficient diet may show signs within 7 days. However, chicks with a good reserve of maternal vitamin A may not show signs of a deficiency for up to 7 wk. Gross signs in chicks include anorexia, growth retardation, drowsiness, weakness, incoordination, emaciation, and ruffled feathers. If the deficiency is severe, the chicks may become ataxic, as with vitamin E deficiency (see Vitamin E Deficiency). The yellow pigment in the shanks and beaks is usually lost, and the comb and wattles are pale. A cheesy material may be noted in the eyes, but xerophthalmia is seldom seen because chicks usually die before the eyes become affected. Infection may play a role in many of the deaths noted with acute vitamin A deficiency.

Young chicks with a chronic vitamin A deficiency may also show pustules in the mucous membrane of the esophagus that may also affect the respiratory tract. Kidneys may be pale and the tubules distended due to the uric acid deposits. In extreme cases, the ureters may be filled with urates. Blood levels of uric acid can rise from a normal level of ~5 mg to as high as 40 mg/100 mL of blood. Vitamin A deficiency does not interfere with uric acid metabolism but does prevent normal excretion of uric acid from the kidney. Histologic findings include atrophy of the cytoplasm and a loss of the cilia in the columnar, ciliated epithelium.

While vitamin A-deficient chicks can be ataxic, similar to those with vitamin E deficiency, no gross lesions are found in the brain of vitamin A-deficient chicks as compared with degeneration of the Purkinje cells in the cerebellum of vitamin E-deficient chicks (see Vitamin E Deficiency). Also, the livers of ataxic vitamin A-deficient chicks contain little or no vitamin A.

Because stabilized dry, vitamin A supplements are almost universally used, it is unlikely that a deficiency will be encountered. However, if a deficiency does develop due to inadvertent omission of the vitamin A supplement or poor mixing, up to 2 times the normally recommended level should be fed for ~2 wk. The dry, stabilized forms of vitamin A are the feed supplements of choice. Forms that can be administered through the drinking water are available and usually result in fast recovery than medication of the feed.

Vitamin D

Figure 8 shows a chick that is Vitamin D: the weak legs, ruffled feathers, and a general unthrifty condition are typical. In this particular individual even the beak is soft and out of shape, which is not, however, always a symptom accompanying leg weakness. These symptoms normally show up between the 4th and 11th weeks of brooding. Be aware that these same symptoms can be attributed to what is known as perosis or slipped tendon in which the legs are bowed or twisted in spite of the fact that the bones are well calcified and hard. Vitamin D is sometimes called the sunshine vitamin. This vitamin is necessary to prevent rickets in growing chicks and to prevent a condition often known as egg paralysis that accompanies the production of soft shelled eggs in laying birds. When consideration is taken to supplement vitamin D, the calcium and mineral content of the ration must also be considered. A consultation with your vet would be very appropriate in this instance. Most commercial feeds contain the supplements to prevent this condition. Sources for Vitamin D are fish liver oils, such as cod liver oil and sardine oil. This can be drizzled on their daily ration for several days with good results but as mentioned above, is a longer dosage is needed a consult with your vet would be appropriate. Let me point out that direct contact with the ultra-violet rays of the sun

enables chickens to produce within themselves the necessary vitamin D, enabling them to make the best use of their feed. Birds kept in an enclosed barn; coop or other places that do not have direct sunlight available are the birds that might require this vitamin supplement.
Vitamin D - Expressed in ICU, which are based on the activity of D3 because birds do not use D2. (Turkeys are especially sensitive!)

The importance of calcium and vitamin D3

The process of calcium uptake, deposition and excretion is regulated by vitamin D3 and its metabolites. Vitamin D3 is absorbed from the intestine in association with fats and requires the presence of bile salts for absorption. The first transformation occurs in the liver, where vitamin D3 is hydroxylated to become 25-hydroxyvitamin D3 (25-OH D3). This vitamin D3 metabolite is then transported to the kidney where it is converted to the most active hormonal compound 1,25-dihydroxyvitamin D3 (1,25-(OH)2 D3). The production of 1,25-(OH)2 D3 is tightly regulated by parathyroid hormone (PTH) in response to serum calcium. If plasma calcium is low, PTH secretion is induced, which stimulates the hydroxylation of 25-OH D3 to 1, 25-(OH)2 D3. This compound will increase calcium absorption in the intestine, mobilize calcium from the bones and reduce calcium excretion via the kidney. If plasma calcium is high, first PTH secretion and then 1,25-(OH)2 D3 production are suppressed, which result in a reduction of calcium absorption in the gut as well as calcium resorption from the bones and an increase in calcium excretion. Therefore it is of utmost importance for an optimum egg shell quality to optimize calcium supply and secure sufficient vitamin D3 activity available to the laying hen.

Despite adequate fortification of layer feeds with vitamin D3, clinical signs of vitamin D3 deficiency such as rickets or cagelayer- fatigue can frequently be observed in laying hens kept under commercial conditions. Such disorders indicate insufficient utilization of the dietary **vitamin D3.**

Vitamin D_3 Deficiency

Abnormal development of the bones is discussed under calcium and phosphorus imbalances (see Calcium and Phosphorus Imbalances) and manganese deficiency (see Manganese Deficiency). Vitamin D_3 is required for the normal absorption and metabolism of calcium and phosphorus. A deficiency can result in rickets in young growing chickens or in osteoporosis and poor eggshell quality in laying hens, even though the diet may be well supplied with calcium and phosphorus.

Laying hens fed a vitamin D_3-deficient diet show loss of egg production within 2–3 wk, and depending on the degree of deficiency, shell quality deteriorates almost instantly. Using a corn-soybean meal diet with no supplemental vitamin D_3, shell weight decreases dramatically by about 150 mg/day within 7 days. The less obvious decline in shell quality with suboptimal supplements is more difficult to diagnose, especially because it is very difficult to assay vitamin D_3 in complete feeds.

There is a significant increase in plasma $1,25(OH)_2D_3$ of birds producing good versus poor eggshells. Feeding purified $1,25(OH)_2D_3$ improves the shell quality of these inferior layers, suggesting a potential inherent problem with metabolism of cholecalciferol.

Retarded growth and severe leg weakness are the first signs noted when chicks are deficient in vitamin D_3. Beaks and claws become soft and pliable. Chicks may have trouble walking and will

take a few steps before squatting on their hocks. While resting, they often sway from side to side, suggesting loss of equilibrium. Feathering is usually poor, and an abnormal banding of feathers may be seen in colored breeds. With chronic vitamin D_3 deficiency, marked skeletal disorders are noted. The spinal column may bend downward and the sternum may deviate to one side. These structural changes reduce the size of the thorax, with subsequent crowding of the internal organs. A characteristic finding in chicks is a beading of the ribs at the junction of the spinal column along with a downward and posterior bending. Poor calcification can also be seen at the epiphysis of the tibia and femur. By dipping the split bone in a silver nitrate solution and allowing it to stand under an incandescent light for a few minutes, the calcified areas are easily distinguished from the areas of cartilage. Adding synthetic $1,25(OH)_2D_3$ to the diet of susceptible chicks reduces the incidence of this condition. Although the response is variable, results suggest that some leg abnormalities may be a consequence of inefficient metabolism of cholecalciferol.

In the laying hen, signs of gross pathology are usually confined to the bones and parathyroid glands. Bones are soft and easily broken, and the ribs may become beaded. The ribs may also show spontaneous fractures in the sternovertebral region. Histologic examination shows deficiency of calcification in the long bones, with excess of osteoid tissue and parathyroid enlargement.

Enough vitamin D is added to commercial diets to provide 3 times the normally recommended level for a period of ~3 wk. Dry, stabilized forms of vitamin D_3 are recommended to treat deficiencies. In cases of severe mycotoxicosis, a water-miscible form of vitamin D is administered in the drinking water to provide the amount normally supplied in the diet.

Vitamin E

Figure 2 shows a chick that is deficient in Vitamin E. Vitamin E seems to be essential for normal reproduction in hatching. Young chicks grown on a vitamin E deficient diet from the time of hatching develop a condition of imbalance and loss of muscular control about the third week. These chicks are found staggering around the pen or lying on their sides. Vitamin E improves immune function by inhibiting the production of immunosuppressant prostaglandins. The most concentrated source of Vitamin E is wheat germ oil and will undoubtedly prove beneficial in increasing hatchability. As an example, there has been a linear decrease in vitamin E intake in the past 20 years, of 0.8%/year/kg weight gain, considering 20 IU vitamin E/kg feed and a feed conversion of 2.0 in 1987 and 1.7 today.

Vitamin E Deficiency

The 3 main disorders seen in chicks deficient in vitamin E are encephalomalacia, exudative diathesis, and muscular dystrophy. The occurrence of these conditions depends on various dietary and environmental factors.

Encephalomalacia is seen in commercial flocks if diets are low in vitamin E, if an antioxidant is either omitted or is not present in sufficient quantities, or if the diet contains a reasonably high level of an unstable, unsaturated fat. For exudative diathesis to occur, the diet must be deficient in both vitamin E and selenium. Signs of muscular dystrophy are rare in chicks, as the diet must be deficient in both sulfur amino acids and vitamin E. Because the sulfur amino acids are necessary for growth, a deficiency severe enough to induce muscular dystrophy is unlikely to occur under commercial conditions. Signs of exudative diathesis and muscular dystrophy can be reversed in chicks by supplementing the diet with liberal amounts of vitamin E, if the deficiency is not too advanced. Encephalomalacia may respond to vitamin E supplementation, depending on the extent of the damage to the cerebellum.

The classical sign of encephalomalacia is ataxia. The results from hemorrhage and edema within the molecular and granular layers of the cerebellum, with pyknosis and eventual disappearance of the Purkinje cells and separation of the molecular and granular layers of the cerebellar folia. Due to its inherently low level of vitamin E, the cerebellum is particularly susceptible to lipid peroxidation. In prevention of encephalomalacia, vitamin E functions as a biologic antioxidant. The quantitative need for vitamin E for this function depends on the amount of linoleic acid and polyunsaturated fatty acids in the diet. Over prolonged periods, antioxidants have been shown to prevent encephalomalacia in chicks when added to diets with very low levels of vitamin E or in chicks fed vitamin E-depleted purified diets. Chicks hatched from breeders that are given additional dietary vitamin E are also less susceptible to lipid peroxidation in the brain. The fact that antioxidants can help prevent encephalomalacia, but fail to prevent exudative diathesis or muscular dystrophy in chicks, strongly suggests that vitamin E is acting as an antioxidant. Exudative diathesis results in a severe edema caused by a marked increase in capillary permeability. Electrophoretic patterns of the blood show a decrease in albumin levels, whereas exudative fluids contained a protein pattern similar to that of normal blood plasma.

Vitamin E deficiency accompanied by sulfur amino acid deficiency results in severe muscular dystrophy in chicks by ~4 wk of age. This condition is characterized by degeneration of the muscle fibers, usually in the breast but sometimes also in the leg muscles. Histologic examination shows Zenker's degeneration, with perivascular infiltration and marked accumulation of infiltrated eosinophils, lymphocytes, and histocytes. Accumulation of these cells in dystrophic tissue results in an increase in lysosomal enzymes, which appear to function in the breakdown and removal of the products of dystrophic degeneration. Initial studies involving the effects of dietary vitamin E on muscular dystrophy showed that the addition of selenium at 1–5 mg/kg diet reduced the incidence of muscular dystrophy in chicks receiving a vitamin E-deficient diet that was low in methionine and cysteine, but did not completely prevent the disease. However, selenium was completely effective in preventing muscular dystrophy in chicks when the diet contained a low level of vitamin E, which alone had been shown to have no effect on the disease.

Studies with chicks on the interrelationships between antioxidants, linoleic acid, selenium, and sulfur amino acids have shown that selenium and vitamin E play supportive roles in several processes, one of which involves cysteine metabolism and its role in the prevention of muscular dystrophy in the chicken. Glutathione peroxidase is soluble and is located in the aqueous portions of the cell, while vitamin E is located mainly in the hydrophobic environments of membranes and in lipid storage cells. The overlapping manner in which vitamin E and selenium function in the cellular antioxidant system suggest that they spare one another in the prevention of deficiency signs.

Only stabilized fat should be used in feeds. Adequate levels of stabilized vitamin E should be used in conjunction with a commercial antioxidant and up to 0.3 ppm selenium. Signs of exudative diathesis and muscular dystrophy due to vitamin E deficiency can be reversed if treatment is begun early by administering vitamin E PO or through the feed. Oral administration of a single dose of 300 IU of vitamin E per bird usually causes remission.

Vitamin K

Figure 3 shows a deficiency of vitamin K. This is not visible to the naked eye. The picture taken shows us the research taken to find the cause of death. Today this is extremely rare. Vitamin K is known to preserve the clotting power of blood. In the absence of this vitamin, chicks bleed to death from any injury causing a rupture of the blood vessel walls.

Vitamin K Deficiency

Impairment of blood coagulation is the major clinical sign of vitamin K deficiency. With a severe deficiency, subcutaneous and internal hemorrhages can prove fatal. Vitamin K deficiency results in a reduction in prothrombin content of the blood, and in the young chick, plasma levels are as low as 2% of normal. Because the prothrombin content of newly hatched chicks is only ~40% that of adult birds, young chicks are readily affected by a vitamin K-deficient diet. A carryover of vitamin K from the dam to eggs, and subsequently to hatched chicks, has been demonstrated, so breeder diets should be well fortified. Hemorrhagic syndrome in day-old chicks has been attributed to a deficiency of vitamin K in the diet of the breeder hens. Gross deficiency of vitamin K results in such a prolonged blood clotting time that severely deficient chicks may bleed to death from a slight bruise or other injury. Borderline deficiencies often cause small hemorrhagic blemishes. Hemorrhages may appear on the breast, legs, wings, in the abdominal cavity, and on the surface of the intestine. Chicks are anemic, which may be due in part to loss of blood but also to the development of hypoplastic bone marrow. Although blood-clotting time is a fairly good measure of vitamin K deficiency, a more accurate measure is obtained by determining the prothrombin time. Prothrombin times in severely deficient chicks may be extended from a normal of 17–20 sec to 5–6 min or longer. No major heart lesions are seen in vitamin K-deficient chicks such as those that occur in pigs.

A vitamin K deficiency in poultry may be related to low dietary levels of the vitamin, low levels in the maternal diet, degree of intestinal synthesis, extent of coprophagy, or the presence of sulfur drugs and other feed additives in the diet. Chicks with coccidiosis can have severe damage to their intestinal wall and can bleed excessively. Antimicrobial agents can suppress intestinal synthesis of vitamin K, rendering the bird completely dependent on the diet for its supply of the vitamin. In poultry, there is little intestinal synthesis due to the short digestive tract. Synthesis of vitamin K does occur in the bacteria resident in the bird's digestive tract; however, such vitamin K remains inside the bacterial cell, so the only benefit to the bird arises from the bacterial cell digestion or coprophagy.

The inclusion of menadione at 1–4 mg/ton of feed is an effective and common practice to prevent vitamin K deficiency. If signs of deficiency are seen, the level should be doubled. A number of stress factors (eg, coccidiosis and other intestinal parasitic diseases) increase the requirements for vitamin K. Dicumarol, sulfaquinoxaline, and warfarin are antimetabolites of vitamin K.

Vitamin B_1 (Thiamin)

The one that is of interest to us is vitamin B1 (thiamin). Figure 7 shows how a bird’s neck is affected by a deficiency of this vitamin. A deficiency of this vitamin involves the nervous system and young birds are most likely to be affected. Affected birds show weakness, particularly of the legs, with loss of weight, in coordination and jerky movements of the neck and legs.

Thiamine Deficiency

Polyneuritis in birds represents the later stages of a thiamine deficiency, probably caused by buildup of the intermediates of carbohydrate metabolism. Because the brain's immediate source of energy results from the degradation of glucose, it is dependent on biochemical reactions involving thiamine. In the initial stages of deficiency, lethargy and head tremors may be noted. A marked decrease in appetite is seen in birds fed a thiamine-deficient diet. Poultry are also susceptible to neuromuscular problems, resulting in impaired digestion, general weakness, star-gazing, and frequent convulsions.

Polyneuritis may be seen in mature birds ~3 wk after they are fed a thiamine-deficient diet. As the deficiency progresses, birds may sit on flexed legs and draw back their heads in a star-gazing

position. Retraction of the head is due to paralysis of the anterior neck muscles. Soon after this stage, chickens lose the ability to stand or sit upright and topple to the floor, where they may lie with heads still retracted. Thiamine deficiency may also lead to a decrease in body temperature and respiratory rate. Testicular degeneration may be noted, and the heart may show slight atrophy. Birds consuming a thiamine-deficient diet soon show severe anorexia. They lose all interest in feed and will not resume eating unless given thiamine. If a severe deficiency has developed, thiamine must be force-fed or injected to induce the chickens to resume eating.

Thiamine deficiency is most common when poorly processed fish meals are used, because they contain thiaminase enzyme. In such situations, adding extra thiamine may be ineffective. In regular diets, deficiency is prevented by supplements of thiamine at 4 mg/kg.

Vitamin B2 (Riboflavin)

Figure 6 shows a chick that has been fed a diet deficient in Vitamin riboflavin. A deficiency in this vitamin results in nutritional leg paralysis in growing chicks due to degeneration in the nerve tissue and results also in reduced egg production and lowered hatchability in mature hens. In addition to the curled toes, chicks affected with this vitamin deficiency will walk on their hocks and appear unable to stand on their feet. It is interesting to note that adult hens deficient in vitamin B2, riboflavin, pass this deficiency through the egg which in turn affects the embryonic growth and condition of the chicks at hatching. Sources for Vitamin B2 are milk products (many breeders add yogurt to their feed during egg lying season), yeast and alfalfa. Note: Yogurt has also been proven to aid in digestion and replace enzymes sometimes lacking in the stomach.

Riboflavin Deficiency

Many tissues may be affected by riboflavin deficiency, although the epithelium and the myelin sheaths of some of the main nerves are major targets. Changes in the sciatic nerves produce “curled-toe” paralysis in growing chickens. Egg production is affected, and riboflavin-deficient eggs do not hatch. When chicks are fed a diet deficient in riboflavin, their appetite is fairly good but they grow slowly, become weak and emaciated, and develop diarrhea between the first and second weeks. Deficient chicks are reluctant to move unless forced and then frequently walk on their hocks with the aid of their wings. The leg muscles are atrophied and flabby, and the skin is dry and harsh. In advanced stages of deficiency, the chicks lie prostrate with their legs extended, sometimes in opposite directions. The characteristic sign of riboflavin deficiency is a marked enlargement of the sciatic and brachial nerve sheaths; sciatic nerves usually show the most pronounced effects. Histologic examination of the affected nerves shows degenerative changes in the myelin sheaths that, when severe, pinch the nerve. This produces a permanent stimulus, which causes the curled-toe paralysis.

Signs of riboflavin deficiency in the hen are decreased egg production, increased embryonic mortality, and an increase in size and fat content of the liver. Hatchability declines within 2 wk when hens are fed a riboflavin-deficient diet, but returns to near normal when riboflavin is restored. Affected embryos are dwarfed and show characteristically defective “clubbed” down. The nervous system of these embryos shows degenerative changes much like those described in riboflavin-deficient chicks.

Signs of riboflavin deficiency first appear at 10 days of incubation, when embryos become hypoglycemic and accumulate intermediates of fatty acid oxidation. Although flavin-dependent enzymes are depressed with riboflavin deficiency, the main effect seems to be impaired fatty acid oxidation, which is a critical function in the developing embryo. An autosomal recessive trait blocks the formation of the riboflavin-binding protein needed for transport of riboflavin to the egg. While the adults appear normal, their eggs fail to hatch regardless of dietary riboflavin content. As eggs become deficient in riboflavin, the egg albumen loses its characteristic yellow color. In fact, albumen color score has been used to assess riboflavin status of birds.

Chicks receiving diets only partially deficient in riboflavin may recover spontaneously, indicating that the requirement rapidly decreases with age. A 100-μg dose should be sufficient for treatment of riboflavin-deficient chicks, followed by incorporation of an adequate level in the diet. However, when the curled-toe deformity is longstanding, irreparable damage occurs in the sciatic nerve, and the administration of riboflavin is no longer curative.

Most diets contain up to 10 mg riboflavin/kg. Treatment can be given as two 100 μg doses for chicks or poults, followed by an adequate amount of riboflavin in feed.

Niacin B3 (Nicotinic Acid or Vitamin pp**) Deficiency**

There is considerable evidence that poultry—even chick and turkey embryos—can synthesize niacin, but at a rate that is too slow for optimal growth. It has been claimed that a marked deficiency of niacin cannot occur in chickens unless there is a deficiency of tryptophan, a niacin precursor.

Niacin deficiency is characterized by severe metabolic disorders in the skin and digestive organs. The first signs are usually loss of appetite, retarded growth, general weakness, and diarrhea. There is conflicting evidence as to whether broilers respond, in terms of growth and feed utilization, to niacin supplementation. However, it has been clearly established that chicks do have a requirement for niacin. Deficiency produces enlargement of the tibiotarsal joint, bowing of the legs, poor feathering, and dermatitis on the head and feet.

Niacin deficiency in chicks can also result in "black tongue." At ~2 wk of age, the tongue, oral cavity, and esophagus become distinctly inflamed. In the niacin-deficient hen, weight loss, reduced egg production, and a marked decrease in hatchability can result. Turkeys, ducks, pheasants, and goslings are much more severely affected by niacin deficiency than are chickens. Their apparently higher requirements are likely related to their less efficient conversion of tryptophan to niacin. Ducks and turkeys with a niacin deficiency show a severe bowing of the legs and an enlargement of the hock joint. The main difference between the leg seen in niacin deficiency and perosis seen in manganese and choline deficiency is that with niacin deficiency the Achilles tendon seldom slips from its condyles.

Niacin deficiency in chickens may be prevented by feeding a diet that contains ≥30 mg niacin/kg; however, many nutritionists recommend 2–2.5 times as much. An allowance of 55–70 mg/kg of feed appears to be satisfactory for ducks, geese, and turkeys. Ample niacin should be provided in poultry diets so that the birds do not have to synthesize it from tryptophan.

Vitamin B5 (PANTOTHENIC ACID) – (CHICK DERMATITIS)

In figure 5 shows a chick with this deficiency. A chick affected with this vitamin deficiency is shown with scabby lesions on the margin of the eyelids, at the corners of the mandibles of the beak, and on the feet. These lesions sometimes also appear around the vent. Evidently the first symptom is the chick sitting back on its hocks and is not willing to move around from the discomfort and

weakness. Sources for this vitamin are milk by-products, liver meal, yeast, alfalfa meal, green grass or cane molasses. Rations that provide a fairly liberal amount of the products will provide adequate amounts of this vitamin. Most commercial feed contains Enough milk byproduct to provide our chicks with the correct percentage needed for good health. Panothenic acid in adult birds causes reduced reproduction, and the hatchlings are weak.

Pantothenic Acid Deficiency

Pantothenic acid is the prosthetic group of coenzyme A, an important coenzyme involved in many reversible acetylation reactions in carbohydrate, fat, and amino acid metabolism. Signs of deficiency relate to general avian metabolism.

The major lesions of pantothenic acid deficiency involve the nervous system, the adrenal cortex, and the skin. Deficiency may result in reduced egg production; however, a marked drop in hatchability is usually noted prior to this event. Embryos from hens with pantothenic acid deficiency can have subcutaneous hemorrhages and severe edema, with most other mortality showing up during the later part of the incubation period. In chicks, the first signs are reduced growth and feed consumption; poor feather growth, with feathers becoming ruffled and brittle; and a rapidly developing dermatitis. Corners of the beak and the area below the beak are usually the worst affected, but the condition is also noted on the feet. In severe cases, the skin of the feet may cornify, and wart-like lumps occur on the balls of the feet. The foot problem often leads to bacterial infection.

Liver concentration of pantothenic acid is reduced during a deficiency, with the liver becoming atrophied. A faint to dirty yellow color may be noted. Nerve fibers of the spinal cord may show myelin degeneration. Panthothenic acid-deficient chicks show lymphoid cell necrosis in the bursa of Fabricius and thymus, together with lymphocytic paucity in the spleen. The foot condition in chicks and the poor feathering are difficult to differentiate from signs of a biotin deficiency. In a pantothenic acid deficiency, dermatitis of the feet is usually noted first on the toes; in contrast, a biotin deficiency primarily affects the foot pads and is usually more severe. Ducks do not show the usual signs noted for chickens and turkeys, except for in retarded growth, but mortality can be quite high.

Most poultry diets contain supplements of calcium pantothenate. Periodically, growing chickens fed practical diets develop a scaly condition of the skin, the exact cause of which is not known. Treatment with both calcium pantothenate (2 g) and riboflavin (0.5 g) in the drinking water (50 gal [190 L]) for a few days has been successful in some instances.

Vitamin (B_6) Pyridoxine, Pyridoxol, pyridoxal, pyridoxamine **Deficiency**

A vitamin B_6 deficiency causes retarded growth, dermatitis, and anemia. Because a major role of the vitamin is protein metabolism, deficiency can result in reduced nitrogen retention. Dietary protein is not well utilized and thus nitrogen excretion increases. Increased iron levels and decreased copper levels are noted in the serum, and iron utilization appears to be markedly decreased. The resulting anemia is likely due to a disturbance in the synthesis of protoporphyrins. Anemia is often noted in ducks but is seldom seen in chickens and turkeys. Young chicks may show nervous movements of the legs when walking and often undergo spasmodic convulsions, leading to death. During convulsions, the chicks may run about aimlessly, flapping their wings and falling with jerking motions. The greater intensity of activity, resulting from pyridine deficiency, distinguishes these signs from those of encephalomalacia. Gizzard erosion has been noted in vitamin B_6-deficient chicks. It can be prevented by inclusion of 1% taurocholic acid in the diet,

leading to the speculation that pyridoxine is involved in taurine synthesis and is important for gizzard integrity. In pyridoxine deficiency, collagen maturation is incomplete, suggesting that this vitamin is essential for integrity of the connective tissue matrix. A chronic deficiency can result in perosis, with one leg usually being crippled and one or both middle toes bent inward at the first joint.

In adult birds, pyridoxine deficiency results in reduced appetite, leading to reduced egg production and a decline in hatchability. Severe deficiency can cause rapid involution of the ovary, oviduct, comb, and wattles, and of the testis in cockerels. Feed consumption in B_6-deficient hens and cockerels declines sharply. Although a partial molt is observed in some hens, normal egg production returns within 2 wk following provision of a normal dietary level of pyridoxine. Deficiency can be prevented by adding 3–4 mg pyridoxine/kg feed.

Vitamin B_{12} (Cobalmin**) Deficiency**

Vitamin B_{12} is an essential part of several enzyme systems, with most reactions involving the transfer or synthesis of methyl groups. While the most important function of vitamin B_{12} is in the metabolism of nucleic acids and proteins, it also functions in carbohydrate and fat metabolism.

In growing chickens, a deficiency of vitamin B_{12} results in reduced weight gain and feed intake, along with poor feathering and nervous disorders. While deficiency may lead to perosis, this is probably a secondary effect due to a dietary deficiency of methionine, choline, or betaine as sources of methyl groups. Vitamin B_{12} may alleviate perosis due to its effect on the synthesis of methyl groups. Other signs reported in poultry are anemia, gizzard erosion, and fatty infiltration of the heart, liver, and kidneys. Laying hens appear to be able to maintain body weight and egg production; however, egg size is reduced. In breeders, hatchability can be markedly reduced, although several months may be needed for signs of deficiency to appear. Changes noted in embryos from B_{12}-deficient breeders include a general hemorrhagic condition, fatty liver, fewer myelinated fibers in the spinal cord, and high incidence of embryo deaths at 17 days incubation.

Deficiency of vitamin B_{12} is highly unlikely, especially for birds grown on litter or where animal-based ingredients are used. Treatment involves feeding up to 20 µg/g feed for 1–2 wk.

Choline (Gossypine **) Deficiency**

In addition to poor growth, the classic sign of choline deficiency in chicks and poults is perosis. Perosis is first characterized by pinpoint hemorrhages and a slight puffiness about the hock joint, followed by an apparent flattening of the tibiometatarsal joint caused by a rotation of the metatarsus. The metatarsus continues to twist and may become bent or bowed so that it is out of alignment with the tibia. When this condition exists, the leg cannot adequately support the weight of the bird. The articular cartilage is displaced, and the Achilles tendon slips from its condyles. Perosis is not a specific deficiency sign; it appears with several nutrient deficiencies.

Although choline deficiency readily develops in chicks fed diets low in choline, a deficiency in laying hens is not easily produced. Eggs contain ~12–13 mg of choline/g of dried whole egg. A large egg contains ~170 mg of choline, found almost entirely in the phospholipids. Thus, there appears to be a considerable need for choline to produce an egg. In spite of this, producing a marked choline deficiency in laying hens has been difficult even when highly purified diets essentially devoid of choline were provided for a prolonged period. The choline content of eggs was not lowered, suggesting synthesis by the bird.

Diets that contain appreciable quantities of soybean meal, wheat bran, and wheat shorts are unlikely to be deficient in choline. Soybean meal is a good source of choline, and wheat byproducts are good sources of betaine, which can perform the methyl-donor function of choline. Other good sources of choline are distiller's grains, fishmeal, liver meal, meat meals, distiller's solubles, and yeast. A number of commercial choline supplements are available, and choline is routinely added to most poultry feed.

Folic Acid (Folacin, B_c, vitamin M) **Deficiency**

A folacin deficiency results in a macrocytic (megaloblastic) anemia and leukopenia. Tissues with a rapid turnover, such as epithelial linings, GI tract, epidermis, and bone marrow, as well as cell growth and tissue regeneration, are principally affected.

Poultry seem more susceptible to folacin deficiency than other farm animals. Deficiency results in poor feathering, slow growth, an anemic appearance, and perosis. As anemia develops, the comb becomes waxy white and pale mucous membranes in the mouth are noted. Elevated erythrocyte phosphoribosylpyrophosphate concentration can be used as a diagnostic tool in folate-deficient chicks. There may also be damage to liver parenchyma and depleted glycogen reserves. While turkey poults show some of the same signs as chickens, mortality is usually higher and the birds develop a spastic type of cervical paralysis that results in the neck becoming stiff and extended.

The abnormal feather condition in chickens leads to weak and brittle shafts. Depigmentation develops in colored feathers due to a deficiency of the vitamin. While a folacin deficiency can result in reduced egg production, the main sign noted with breeders is a marked decrease in hatchability associated with an increase in embryonic mortality, usually during the last few days of incubation. Embryos have deformed beaks and bending of the tibiotarsus. While birds may exhibit perosis, the lesions seen differ histologically from those that develop due to choline or manganese deficiency. Abnormal structure of the hyaline cartilage and retardation of ossification are noted with folacin deficiency. Increasing the protein content of the diet has been shown to increase the severity of perosis in chicks receiving diets low in folic acid, as there is an increased folacin demand for uric acid synthesis.

Signs of folic acid deficiency in poultry can be prevented by ensuring diets contain supplements of up to 1 mg/kg.

Vitamin (H) Biotin Deficiency

Biotin deficiency results in dermatitis of the feet and the skin around the beak and eyes similar to that described for pantothenic acid (*see* p 2533). Perosis and footpad dermatitis are also characteristic signs. While signs of classic biotin deficiency are rare, occurrence of fatty liver and kidney syndrome (FLKS) is important to commercial poultry producers. FLKS was first described in Denmark in 1958, but was not a major concern until the late 1960s, especially in Europe and Australia. Chicks ~3 wk of age become lethargic and unable to stand, then die within hours. Mortality is usually quite low at 1–2% but can reach 20–30%. Postmortem examination reveals pale liver and kidney with accumulation of fat.

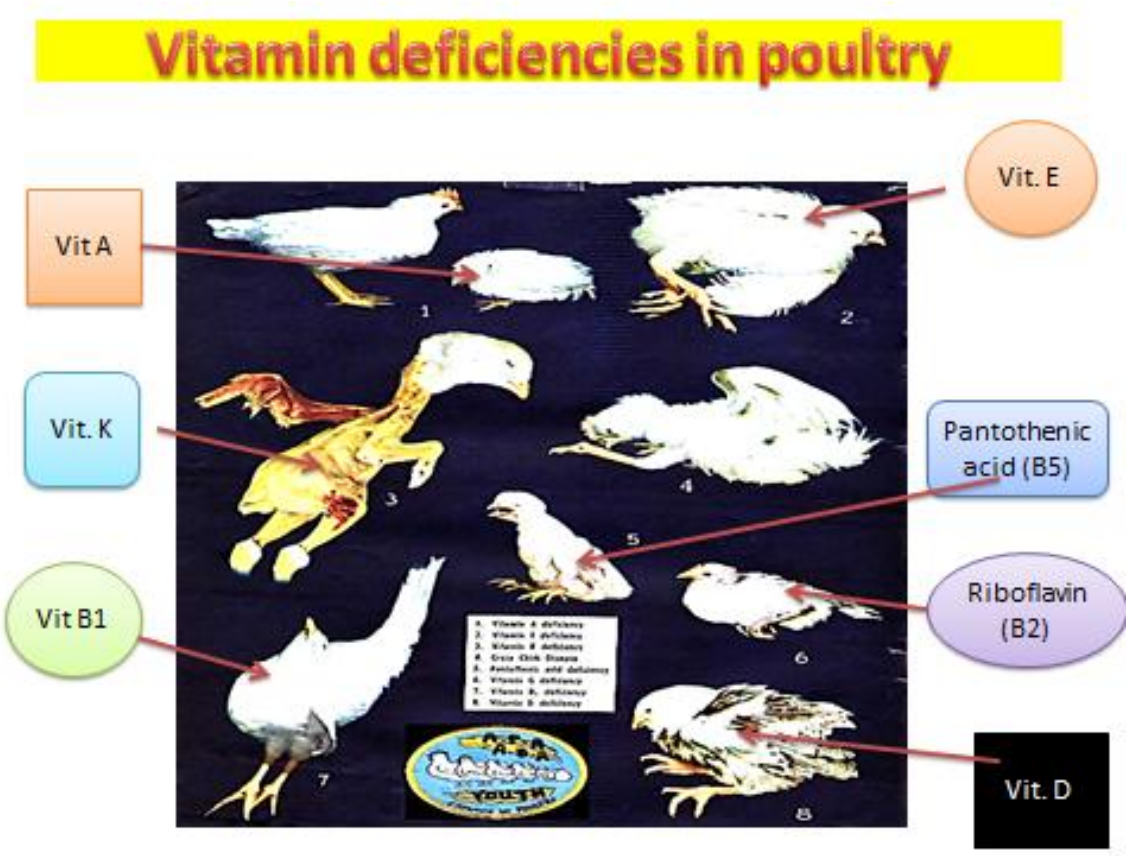
The condition was usually confined to wheat-fed birds and was most problematic in low-fat, high-energy diets. High vitamin supplementation in general corrected the problem, and biotin was isolated as the causative agent. It is now known that biotin in wheat has exceptionally low availability. The trigger of high-energy diets led to investigation of biotin in carbohydrate metabolism. Chicks with FLKS are invariably hypoglycemic, emphasizing the importance of biotin in 2 key enzymes: pyruvate carboxylase and acetyl Co-A carboxylase. Acetyl Co-A carboxylase

appears to preferentially sequester biotin, such that with low biotin availability and need for high de novo fat synthesis (high energy, low-fat diet), pyruvate carboxylase activity is severely compromised. Even with this imbalance, birds are able to grow. However, with a concurrent deprivation in feed intake or increased demand for glucose, hypoglycemia develops, leading to adipose catabolism and the characteristic accumulation of fat in both liver and kidney. Birds with FLKS rarely show signs of classic biotin deficiency.

Plasma biotin levels <100 ng/100 mL has been reported as sign of deficiency. However, recent evidence suggests that plasma biotin levels are quite insensitive to the birds' biotin status, and that biotin levels in the liver or kidney are more useful indicators. Plasma pyruvic carboxylase is positively correlated with dietary biotin concentration, and levels plateau much later than does the growth response to biotin.

Embryos are also sensitive to biotin status. Congenital perosis, ataxia, and characteristic skeletal deformities may be seen in embryos and newly hatched chicks when hens are fed a deficient diet. Embryonic deformities include a shortened tibiotarsus that is bent posteriorly, a much shortened tarsometatarsus, shortening of the bones of the wing and skull, and shortening and bending of the anterior end of the scapula. Syndactyly—an extensive webbing between the third and fourth toes—in biotin-deficient embryos has been noted. Such embryos are chondrodystrophic and characterized by reduced size, parrot beak, crooked tibia, and shortened or twisted tarsometatarsus.

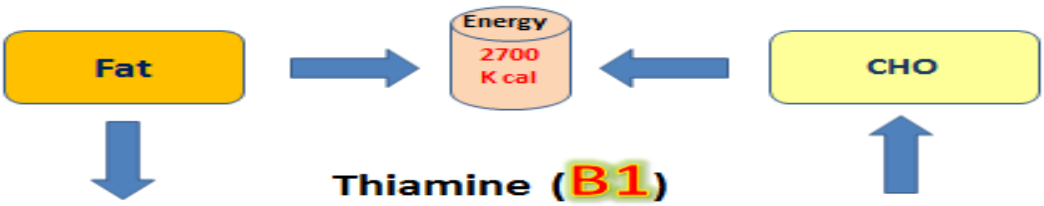
A number of factors increase biotin requirements, including oxidative racidity of feed fat, competition by intestinal microorganisms, and lack of carryover into the newly hatched chick or poult. It is good practice to add 150–200 mg biotin/ton feed, especially when wheat or wheat byproducts are used.



Vitamins are organic substances required by the body in minute amounts for normal growth, health and production.

Effect of feed composition on vitamin requirements

Rations contain high level of carbohydrates
increase
thiamine (vit. B1) requirement



Effect of feed composition on vitamin requirements

Pantothenic acid B3
can be synthesized
from
the amino acid **Tryptophan**

Relation of vitamins to other nutrients

The amino acid **Tryptophan** can be converted into **niacin** (Vit B5) in bird's body

thus

the importance of this amino acid should be considered in poultry diet

Resources

1) Vitamins in Animal Nutrition. By (Lee Russell McDowell).
2) Feeds and Nutrition. By (M. E. Ensminger, J. E. oldfield and W. W. Heinemann)
3) (http://www.thepoultrysite.com/publications/1/egg-quality-handbook/3/optimum-vitamin-nutrition-of-laying-hens)

(http://www.worldpoultry.net/Broilers/Nutrition/2010/8/Broiler-diets-demand-balanced-vitamin-supplementation-WP007802W/