SYMPTOMS OF NUTRITIONAL DEFICIENCIES IN POULTRY

EXTENSION SERVICE
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SYMPTOMS OF NUTRITIONAL DEFICIENCIES

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Lack of certain nutrients in the diet of poultry results in typical symptoms if such deficiencies are severe and of sufficient duration. These symptoms, in many instances, resemble those of actual disease. The conditions produced by deficiencies in the diet are sometimes called "nutritional diseases." Nutritional diseases are prevented by using rations which are properly formulated and mixed from the best of ingredients. The college formulas, given in bulletin 366-A, "Feeding Chickens," and commercial feeds manufactured by reputable companies are adequate in all respects if fed according to directions. If directions are not carefully followed, if improperly formulated or mixed feeds are used, or if ingredients are of low quality, nutritional deficiency diseases may occur.

No gross changes resulting from a lack of carbohydrates (starches, sugar, fiber), fats, or protein are evident except through laboratory examination. Deficiencies of carbohydrates or fats in the field happen only during starvation. Recently, it has been shown that certain types of carbohydrates are essential for growth and that choline, a fat-like substance, is required for growth and the prevention of perosis. Insufficient protein results in stunting of growth, in reduced production, and in smaller egg size. Insufficient quantities of certain vitamins and minerals produce striking deficiency symptoms, particularly in young birds.

The Vitamins

Practically all of the numerous vitamins are required by poultry, but fortunately many of them are present in sufficient quantities in common feeds to meet the demands of the bird. Of the better-known vitamins, the fowl does not require vitamin C in the ration and can synthesize it in the body, and grains and cereal by-products are sufficiently rich in vitamins B and E to supply plenty of them under practical conditions.

However, some rations in common use have been found to be deficient in vitamins A, D, and G (riboflavin) and possibly several others.

Minerals

Of the many minerals, sufficient quantities of all but calcium, phosphorus, sodium, and chlorine, and often manganese and possibly
iodine, are found in normal cereal ingredients. Meatscrap and fish meal supply phosphorus and much of the calcium required.

Where necessary, additional phosphorus is best supplied through steamed bonemeal. Calcium is very cheaply provided in limestone (95 percent or more calcium carbonate) or oystershell. Sodium and chlorine are provided by common salt (sodium chloride). Manganese is present in variable amounts in wheat shorts, middlings, and bran, but chicks of the heavy breeds reared on wire may need additional amounts in the form of anhydrous manganese sulfate. Iodine is commonly present in appreciable amounts in fish meal and fish oils. Where necessary, iodine may be supplied in trace amounts in the form of potassium iodide. However, the amount of iodine required by poultry is not yet established. Experiments on this problem are in progress.

**Common Deficiency Disorders**

**Nutritional Roup—Vitamin-A Deficiency.**

Vitamin-A deficiency, all too common in Colorado, is evidenced in older birds by a swelling of the tissues around the eye and sinuses, owing to the accumulation of loose, cheesy, white material in the eye. There is no disagreeable odor present as there is in infections of a similar description which are classified as infectious roup. In acute cases, particularly in younger birds, the kidneys are swollen and the ureters are choked with excess urates. In more chronic cases,
small yellowish-white pustules are found in the pharynx and esophagus. A deficiency of vitamin A in breeding rations results in lowered hatchability of eggs and the production of congenitally weak chicks which respond poorly even on the high vitamin-A starting mash.

Young birds show stunting, roughened feathers, incoordination of gait, and frequently xerophthalmia (drying up of eye secretions and accumulation of sticky material around the eye due to secondary bacterial infection). Death usually follows. Turkeys are particularly susceptible. Often they show only delayed growth and incoordination (spasms), followed in a few hours by death.

In both old and young birds insufficient vitamin A results in lowered resistance to disease, particularly colds. Vitamin A in the plant form occurs chiefly as certain carotenes in green feed and as cryptozanthin in yellow corn. Green range, alfalfa leaf meal, excellent quality alfalfa hay, and yellow corn are, therefore, the chief common sources of this important vitamin. The carotene in green feed is readily lost by exposure to sunshine, dampness, air, and heat. Therefore, alfalfa must be properly cured or artificially dehydrated and stored in a cool place.

Vitamin A is present in the animal form in fish oils, particularly from cod liver and sardines. It is quite stable in the oil but becomes oxidized and tends to lose its potency after mixing in the feed. Recent research shows that the rate of loss is moderate and not of importance when feed is not held over 1 month after mixing and when only fresh, high-quality ingredients are used.

Symptoms of vitamin-A deficiency are very common in Colorado, particularly in the dry-land areas where chickens are expected to exist on such deficient grains as barley, wheat, and sorghums. Birds under such conditions store up vitamin A in the spring when plenty of green range is available, but these reserves are depleted in the summer, fall, winter, and early spring with resulting loss in production and hatchability of eggs and increase in adult and chick mortality. Strangely enough, Colorado is famous for its alfalfa products, yet suffers from their very lack. The cure is obvious.

Directions for economical, adequate rations are to be found in the bulletin 366-A, "Feeding Chickens." Birds suffering from vitamin-A deficiency usually recover rapidly when they are treated for a week or so by adding 2 percent of standard biologically tested fish oil or when they receive plenty of leafy, green feed and then are kept on an adequate ration.

"Leg Weakness."

Chickens seem to be peculiarly susceptible to many different forms of leg afflictions. One of the most common is fowl paralysis,
an infectious virus disease causing wing or leg paralysis. The following are the most common nutritional forms of leg weakness:

1. **Rickets.**—Rickets may be a result of insufficient vitamin D, calcium, or phosphorus, or of excess calcium. Tendency toward rickets is enhanced by coccidiosis infection, presumably by impairing intestinal absorption. The symptoms vary slightly according to the cause.

   a. **Vitamin-D Rickets.**—In chicks and poults this disorder is evidenced by poor growth, rough feathers, lameness, swelling of the hock and rib joints, crooked breast bones, and leg bone deformities. It is readily distinguished from perosis (enlarged hocks or slipped tendons) by the fact that the leg and breast bones are easily bent or rubbery, and the beaks, particularly at the base, are very soft. If not relieved, the chicken dies. The enlarged joints are due to poor calcification and excessive production of cartilage.

   ![Cornell University Agricultural Experiment Station.](image)
   
   Rickets in a partially grown cockerel. The bird is stiff, and the hock joints are slightly swollen.

   In mature stock the birds with rickets appear unthrifty and underweight and become stiff and lame. Egg production and egg size are reduced, with many thin-shelled and fragile eggs occurring. The breast bone in young laying pullets becomes soft and malformed. Leg bones become thin, brittle, and fragile. The birds eventually lose the use of their legs and die.
The "line-test" for rickets in growing birds. The rachitic bone on the left shows a wide uncalcified area. The normal bone on the right shows a very narrow uncalcified area.

The treatment lies in supplying vitamin D to the birds in the form of direct sunlight or of proper amounts of biologically tested and guaranteed fish oils as described in the bulletin 366-A, "Feeding Chickens." In treating rachitic flocks the amount of fish oil should be doubled or tripled for a week. Recovery is usually rapid, but any deformities usually remain.

Occasionally, rickets results from using an untested fish oil with little or no vitamin-D potency.

b. Low-Calcium-Phosphorus Rickets.—This form of rickets is a result of insufficient calcium or phosphorus or both.

In chicks and pouls, growth is somewhat reduced and the birds are unthrifty. If the deficiency occurs very early, the bones may not calcify, and they remain very soft. If some calcification has occurred previously as in older birds, the bones become very fragile and are easily snapped. Little excess cartilage formation occurs because calcification proceeds in the growing ends of the leg bones, the mineral being taken from the shaft and redeposited in the ends, thus producing fragile bones. The beaks become soft and pliable.

The egg production of laying birds is quickly reduced by calcium deficiency, and the egg shells are thin and fragile. Here, too, the bones tend to become thin-walled and fragile because the bird uses up the mineral to form egg shells.

The difficulty is frequently caused by overfeeding grain to chicks at the expense of mash consumption or by failing to provide limestone grit or oystershell to birds on range on in production. The treatment is obvious. Proper amounts of calcium and phosphorus must be fed. Balanced rations and methods of feeding are described in the bulletin 366-A, "Feeding Chickens." Treatment consists of adding 2 to 5 percent steamed bonemeal to the mash and eliminating grain from the ration for several weeks. Adequate vitamin D should also be assured through the use of direct sunlight or fish oil.
c. **High-Calcium Rickets.**—This type of rickets has not been reported among mature birds, but it causes symptoms in young chicks very similar to the low-calcium form, except that growth may be somewhat depressed. The excess calcium in the diet combines with phosphorus in the digestive tract, rendering the phosphorus unavailable to the bird and, therefore, causing rickets owing to lack of phosphorus. Since manganese absorption is simultaneously reduced under these conditions, perosis may also occur in chicks during the first 6 weeks of life.

Excess calcium in chick mashes is not common. This trouble may be the result of giving chicks limestone grit or oystershell during the first 6 weeks of life.

2. **Perosis—(Slipped Tendons or Enlarged-Hocks Disease).**—Perosis occurs in young chicks and poults between 3 and 6 weeks of age, although a few birds occasionally are afflicted with it when hatched, or contract it earlier.

It is usually characterized by enlargement of the hock joint, followed frequently by twisting or rotation of the shank bone and slipping of the tendon. Such birds are permanently crippled. It is much more common in heavy breeds and in chicks or poults reared in batteries or on wire floors. Male birds appear to be more susceptible than females.

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Perosis (enlarged hocks or slipped tendons). The chick on the left has enlarged hocks and twisted shanks. The one on the right has slipped tendons. Note that the toes curl outward.
Perosis is differentiated further from rickets by the fact that the bones are fully calcified; are difficult to bend or break, and by the fact that recovery is rare.

This disorder is the result of a true nutritional deficiency in that it is due to a lack of manganese and of choline, a fat-like substance found especially in soybean oilmeal. Excessive amounts of calcium (such as limestone) or of calcium and phosphorus (such as bone in bonemeal or meatscrap) will greatly aggravate perosis because the excess of these elements in the digestive tract of the bird combines with manganese, rendering it insoluble and unavailable to the bird. The result is a manganese deficiency, although the ration itself may be relatively rich in manganese. Iron salts, even in small amounts, may similarly render dietary manganese unavailable. Gravel or grit from native stone rich in iron (red) should be avoided.

The prevention of perosis lies in keeping the amount of limestone or oystershell and bone-carrying supplements, such as steamed bonemeal and meatscrap, at a level in the chick ration sufficient to supply adequate but not excessive calcium and phosphorus. Soybean oilmeal is an ideal substitute for part of the meatscrap because it is low in calcium and phosphorus, high in protein value, and rich in

Comparison of perosis and curled-toe paralysis. Note the normal hocks and inward curling of the toes of the chick on the right and the enlarged hocks and twisted shanks of the bird on the left.
choline. Another essential step is the use of feedstuffs rich in manganese, such as wheat shorts, middlings, bran, and oats. Since these feeds vary in manganese content according to the soil on which they were grown, and since heavy breeds, particularly on wire floors, have a higher requirement for this element than Leghorns, the addition of 4 ounces of 90- to 95- percent anhydrous manganese-sulfate powder per ton of starter mashes for chicks and poults is recommended where any manganese deficiency may be possible. Natural manganese-carbonate is not recommended because it cannot be assimilated by the bird.

3. Nutritional or "Curled-Toe" Paralysis.—This affliction appears most commonly in chicks between 3 and 6 weeks old. It is characterized by a tendency to walk on the hocks with the toes curled inward. Usually the birds thus affected remain active and recover spontaneously, but some may grow progressively worse with eventual sprawling and permanent crippling.

These symptoms are caused by a deficiency of riboflavin (vitamin G) in the starting ration and are readily prevented or cured by feeding an adequate ration rich in milk products and alfalfa leaf meal. Since this vitamin is also essential for growth, flocks in which this "paralysis" occurs may not be growing normally.

4. Nutritional Encephalomalacia — "Crazy-Chick Disease."—This condition occurs in chicks from 2 to 8 weeks of age. The chicks show symptoms varying from droopiness and stupor to more severe cases of muscular incoordination, sitting on the abdomen, retraction of the head, and somersaults (cart wheels), particularly when lying on their sides. Mortality varies up to about 50 percent. The main lesions are confined to the brain, with enlargement and edema (swelling) of the cerebellum, associated with slight grayish discoloration and small hemorrhages.

Recent information indicates the disease is caused by destruction of some necessary nutritional factor by animal fats in the diet. Presumably this destruction takes place when fats oxidize. Rancid
fats appear to speed up the reaction. There is good evidence that vitamin E or some closely related compound is the factor destroyed. The use of freshly milled cereal by-products and of meatscrap, fish meal, and fish oil low in free-fatty acid would be a precautionary measure against outbreaks of encephalomalacia.

Until more is known on this problem, it is suggested that a flock of chicks suffering from this disorder be placed on fresh, green range, or on the following all-mash formula until they are 8 weeks old, when the regular feeding schedule can be resumed.

- Freshly ground yellow corn (not kiln dried)........ 20 pounds
- Freshly ground wheat (not kiln dried) ............ 20 pounds
- Freshly pulverized oats (not kiln dried) .......... 20 pounds
- Alfalfa leaf meal, preferably dehydrated .......... 7½ pounds
- Soybean oilmeal ........................................ 25 pounds
- Dried buttermilk ........................................ 5 pounds
- Steamed bonemeal .................................... 2½ pounds
- Manganese-sulfate ................................... ½ ounce
- Reenforced fish oil (400 units of D per gram) ... ¼ pound

Freshly cracked, naturally dried corn, oats, and wheat may be fed as a scratch grain in every other feeder if desired.

5. **Vitamin-B4 Deficiency.**—A peculiar, stiff-legged, stilted walk or "goose-step" apparently differentiates a paralysis resulting from a lack of vitamin B4 from other forms. Occasional cases of what is believed to be a deficiency of this vitamin are observed in the field. Since this vitamin is destroyed by heating the ration, it is possible that some birds with an hereditarily high requirement for it may develop these symptoms if the cereal portion of the ration was derived from heated grains or if the feed is very stale.

There is no clear-cut evidence as yet to distinguish entirely between this deficiency and nutritional encephalomalacia. The treatment suggested in case of an outbreak is the same as for the latter difficulty.

**Crooked Breast Bones.**

The occurrence of crooked breast bones in chicks may be a result of rickets, as discussed under that title.
This deformity in chickens and turkeys is apparently governed by heredity, nutrition, and environment. There is evidence that there is an hereditary susceptibility to it. A deficiency of vitamin D, calcium, or phosphorus may result in crooked breast bones. Similarly, early roosting on narrow or sharp-edged perches will aggravate the disorder.

The obvious remedy lies in eliminating all crooked-breasted birds from the breeding flock, in supplying adequate vitamin D, calcium, and phosphorus, and in using flat or slightly tilted perches that are at least 2 inches wide for chickens and 3 to 4 inches wide for turkeys.

Heavily producing pullets are apparently quite susceptible to this deformity, owing to the demands of egg-shell formation and of a still-growing body. Special care must be taken to see that they have plenty of vitamin D and calcium.

**Dermatosis (Dermatitis) or “Pellagra.”**

Dermatosis is an abnormal condition of the skin of chicks and poults. It shows up as scabby or crusty lesions at the corners of the mouth and eyes, on the bottoms of the feet and toes, and sometimes around the vent. It is caused by a lack of certain factors in the vitamin-G complex and, therefore, is associated with poor growth. These
factors are usually supplied by using sufficient dried-milk products and green feed in the ration.

In chicks, dermatosis is caused by the lack of a vitamin designated by California workers as the "filtrate factor" and by the Cornell workers as the "antidermatosis factor." It has recently been identified by the California workers as panthothenic acid. Feeds apparently vary in their content of this vitamin, and occasional cases are seen, particularly where the brooder houses are overheated and improperly ventilated. Cane molasses, as well as the dried-milk products and green feed, is a good source of the vitamin.

In poults, this disorder is caused by insufficient riboflavin (vitamin G). Again, it seems probable that this or a similar dermatosis is aggravated by overheating and poor ventilation.

The prevention and treatment consist of feeding diets with adequate amounts of milk products and green feed and of providing plenty of fresh air and moderate brooding temperatures, preferably allowing the birds to run out and feed in a cool room or sunporch. In the case of afflicted chicks it may be desirable to add 5 percent of blackstrap cane molasses to the starting ration until the symptoms clear up. Turkeys should be fed a normal complete ration fortified with 5 percent of dried buttermilk or liquid milk until the lesions disappear.

**Gizzard Erosions.**

Occasionally poultrymen find birds in which the gizzard lining is eroded and rough, particularly in the forward end. The eroded spots are usually dark brownish in color. This condition appears to differ from a similar difficulty where the gizzard lining is thickened and possibly loosened.

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"Gizzard erosions."
Recent studies indicate that both the erosions and the loosened lining are caused by faulty diet. Green feed, wheat bran, oats, and soybean oilmeal appear to be highly preventive in the case of the erosions, and use of these ingredients in the ration tends to control or prevent any serious lesions. Since growth is apparently not greatly affected, this disorder is not of great concern except that it may indicate improper nutrition. The swollen lining of gizzards appears to be prevented where coarse, ground feeds are used. Insoluble grit is also helpful.

Gizzard erosions have been found in apparently healthy day-old chicks in many parts of the country. The cause still remains to be satisfactorily explained.

**Hemorrhages.**

Rarely and under unusual conditions a deficiency of vitamin K may appear in chickens. This factor is essential for normal clotting power of blood. In its absence extensive bleeding occurs from small wounds and bruises. This vitamin is present in green feed and other feeds, and a deficiency in practical rations is not commonly recognized. Green feed is the basic source of this vitamin.