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Investigations of the Cause and Prevention of Perosis

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FOREWORD

The present methods of poultry production, using the incubator, battery brooder, and other modern devices, are so artificial as compared to nature's methods that they are undoubtedly responsible for the appearance of certain new diseases occurring among chickens in battery brooders. One of these, which was first referred to as "hock joint trouble," "hock disease," or "slipped tendon," is now almost universally called "perosis." The brooder itself is not responsible for the condition, but it undoubtedly aggravates the cause by confining large numbers of chickens in a small space. The nature of the food used to best advantage in this type of feeding is really the primary cause. It has been observed that there is a direct correlation between the amount of mineral present and the incidence of perosis, especialy in the case of the amount of calcium or phosphorus. The more recent observations indicate that a small trace of manganese in some way prevents the occurrence of perosis. Various explanations have been offered for this phenomenon, but no single theory has been universally accepted. Without attempting to select any of these theories, one can safely state that the addition of as little as 50 parts per million of the entire food in the form of maganese will practically assure the producer that perosis will not interfere with the normal development of the chickens.

With the thought in mind that too much mineral increases the disturbance, and that the condition may be remedied by removing some of the excess, or by adding manganese, the following suggestions are made:

1. In view of the variation in the manganese content of feeding stuffs commonly used, the small quantity required, and the fact that too much manganese is harmful, *it does not seem advisable to add manganese to a ration without first making chemical analysis of the ration questioned.*

2. If the ration is known to produce perosis under a certain set of conditions, then it would be advisable to add manganese to that particular ration only after a chemical analysis had been made and the ration found low in manganese. 100 pounds of mash require 6 grams or 1/5 ounce; 1,000 pounds require 57 grams or 2 ounces; 2,000 pounds require ¼ pound or 4 ounces of this material. To assure complete distribution, the manganese sulphate

should be dissolved in a pint of warm water and mixed in a small portion of the feed. This pre-mixed feed is then added to the rest of the ration during the process of mixing.

3. In many cases, after an analysis has been made of the ration which is believed to be producing perosis, it will only be necessary to remove some of the mineral to relieve the condition.

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INVESTIGATIONS OF THE CAUSE AND PREVENTION OF PEROSIS

By

Robert Penquite, V. G. Heller, and R. B. Thompson

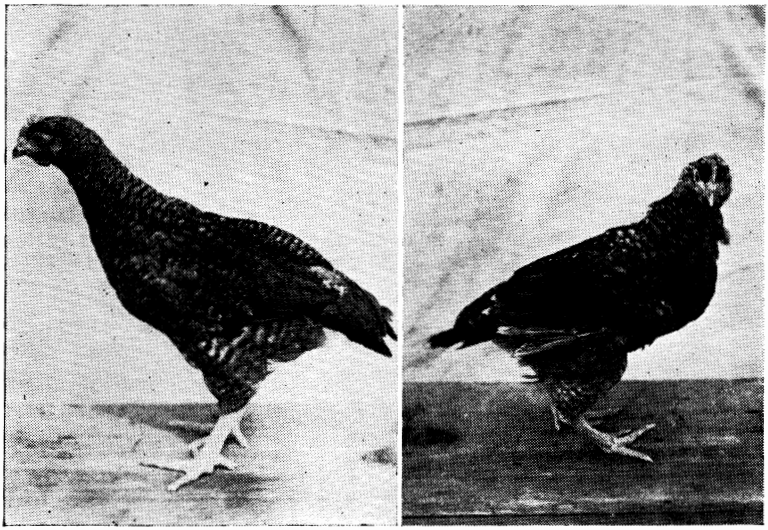
Perosis is a disorder in young, growing chickens resulting in a faulty bone formation. It is first observed as a softening or bending of the bones near the hock. As a result of this malformation of the bones, the tendon which regularly rests in the groove formed by the condyle is pulled or forced to one side and the chicks are soon unable to stand or walk. They are trampled by the other birds and either die or become non-profitable or useless. The condition is illustrated by the accompanying series of photographs and X-ray reproductions (Figures 1 to 4). It will be noted that the hocks are enlarged and that the leg bones are twisted or bent. This fact resulted in the early name, "slipped tendon." As a result of the disarrangement of bones and tendons, an irritation is sometimes started in the joint. This causes swelling and accumulation of lymph, and from this the name "hock disease" originated.

EARLY STUDIES OF PEROSIS

This condition has been prevalent in chickens for a long time, but general interest in the subject did not begin until about 1930. Some persons believed that wire floors in brooders might possibly have been responsible for the injury; while others confused the malady with rickets, because the legs appeared to be bent as they are in rachitic conditions. The first theory was disproved by investigations at the Oklahoma Agricultural Experiment Station. Trials were conducted using three different types of floors in the battery brooder, with results as shown in Table I. (See page 10.) The belief that the chicks were suffering from a rachitic condition led to the inclusion in the ration of those constituents used in the cure of rickets: calcium, phosphorus, vitamin D as cod liver oil, sunshine, ultra violet light, and irradiated foods; but none of these singly or in combination relieved or prevented the condition. In fact, in the majority of the cases, the more minerals used the more aggravated the condition became.

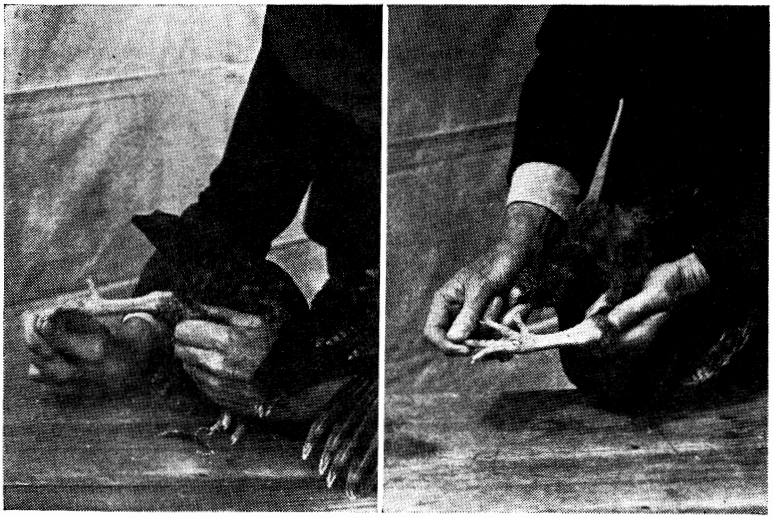
Calcium and Phosphorus in Relation to Perosis

In many of the growing mashes used, bone meal was added in rather generous amounts to guard against rickets and to furnish ample minerals for body requirements. Analyses of these rations indicated the presence of large amounts of phosphorus, and for several years a number of investigators were, therefore, of the opinion that an excessive amount of phosphorus might be responsible for the condition. Hunter and Funk (1930) observed a large number of perosis birds on rations containing large amounts of meat meal, meat and bone meal, or bone meal. Hunter, Dutcher and Knandel (1931) produced the condition experimentally by adding to the ration mineral mixtures including bone meal, sodium phosphate, and calcium carbonate. They noted that decreased amounts of these minerals reduced the number of cases of perosis. Payne, Hughes and Leinhardt (1932), studying the factors involved in the malformation of bones in young chicks, found that a mixture of chemically pure calcium phosphate and calcium carbonate produced the same effects as an equivalent amount of bone meal.



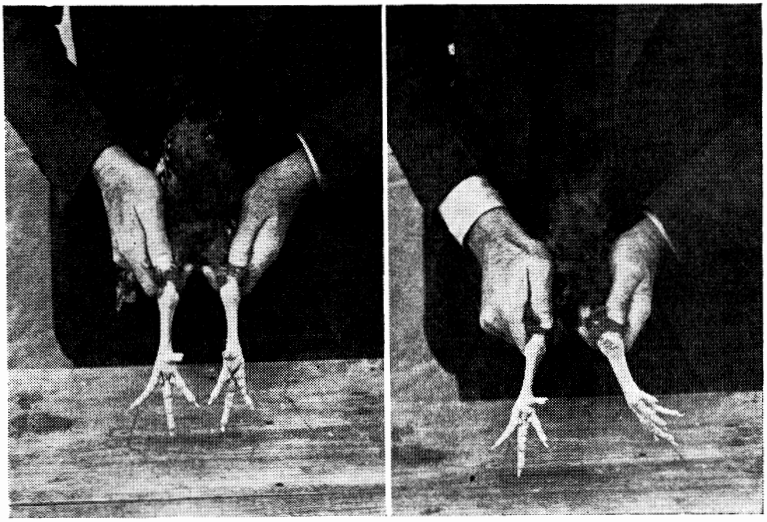
Normal Perosis

Figure 1. A comparison of the outward appearance of a normal chick (left) with one affected by perosis (right).



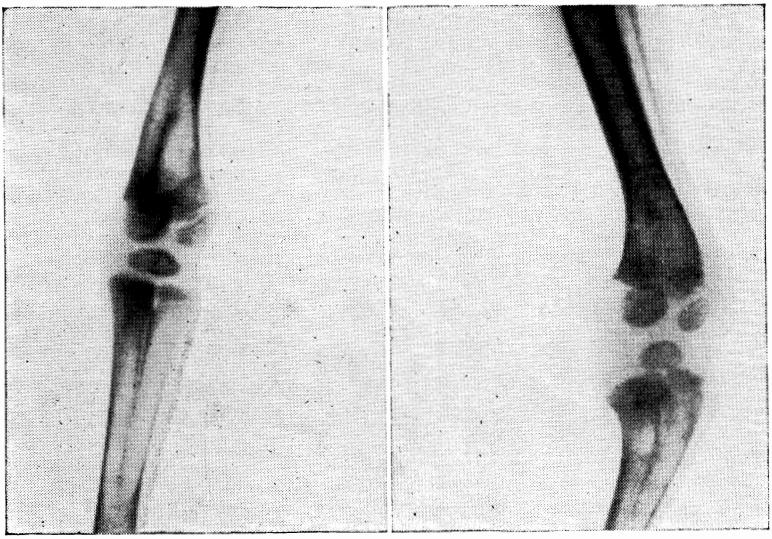
Normal Perosis

Figure 2. The same condition shown in a side view.



Normal **Perosis**

Figure 3. The legs of these chickens as they appear from the rear.



Normal **Perosis**

Figure 4. X-ray skiagraph of normal bones and of bones twisted and deformed by perosis.

Card (1930) cautioned poultrymen not to feed rations containing excessive amounts of minerals. Herner and Robinson (1932), Parkhurst and McMurry (1933), Hunter, Dutcher and Knandel (1931), Schaible, Moore and Conolly (1933), Solun and Arsenjaw (1934), and Milby (1933) all concluded that the factor involved in perosis was excessive mineral feeding, and that phosphorus was one of the chief offenders. Milby (1934) later stated that "the incidence of slipped tendons increased as the phosphorus increased in the ration." Insko, Sowell and Lyons (1934) concluded that the amount of phosphorus fed might be one of the causative factors. Hammond (1936) in his studies observed a high correlation between perosis and phosphorus. Maw (1937), Milne (1936) and Branion (1937) also noted that the addition of minerals caused a marked increase in perosis. Wilgus, Norris and Heuser (1935) made the observation that chemically pure primary sodium phosphate produced more severe cases of perosis than steamed bone meal or tricalcium phosphate.

Other investigations, however, tended to throw doubt on the belief that phosphorus and calcium were factors in the incidence of perosis. Payne, Hughes and Leinhardt (1932) found no association between the inorganic calcium and phosphorus in the blood while attempting to correlate blood mineral analysis and the incidence of perosis. Blood phosphorus studies made at the Oklahoma Agricultural Experiment Station about the same time indicated that there were changes in certain fractions of the blood; but the changes were not great enough to warrant the conclusion that phosphorus alone was responsible for the difficulty. The work of Herner and Robinson (1932), Wilcke (1932), Heller and Penquite (1937), Milby (1935), Branion (1937) and Holmes, Pigott and Moore (1933) indicated that there was no abnormal calcification of the leg bones of birds affected with perosis.

Development of a Perosis-Producing Ration

In the early days of the investigation the difficulty of the study was increased because the occurrence of the malady appeared to be more or less a matter of chance. To study the causes it was necessary to know conditions which would produce perosis in a majority of the chickens. Rations for that purpose were developed gradually; and, finally, Hunter and Funk (1930) published a formula which was satisfactory. Since that time rations have been developed by numerous workers: Titus (1932), Sherwood and Couch (1936), Clifcorn, Elvehjem and Hart (1938), and Heller and Penquite (1936). These rations would produce perosis in 40 to 100 percent of the chicks involved. Modifications of Hunter's and Funk's 1930 rations were made at the Oklahoma Agricultural Experiment Station until a mixture was reported which would produce faulty bone formation in 76 to 100 percent of the chicks involved.

This basic or control perosis-producing diet was designated by number CK324.

Ration Number CK324	Percent (by weight)
Yellow corn (ground)	66.0
Wheat gray shorts	5.0
Alfalfa leaf meal	3.0
Dried buttermilk	15.0
Meat and bone scraps	5.0
Bone meal	5.0
Cod liver oil	.25
Salt (sodium chloride)	.75

The analysis of the ration is as follows:

	Percent (by weight)
Moisture	11.10
Protein	15.30
Ether-soluble extract	4.43
Crude fiber	3.41
Ash	9.36
Nitrogen-free extract	56.60
Calcium	2.22
Phosphorus	1.28
Magnesium	.271
Manganese	.00089

This ration is to all appearances a well-balanced mixture. The analysis indicates that the protein is adequate, and that the carbohydrate and fat composition are normal. The alfalfa leaf meal is an excellent vitamin carrier, and the yellow corn is well supplied with vitamin A. To this mixture is added a fortified cod-liver oil which furnishes ample vitamin A and D. The cereals furnish the B complex. The large amount of dried buttermilk is an adequate source of riboflavin. Both meat scraps and bone meal furnish an abundance of calcium and phosphorus. Yet, as shown in Table I, from 76 to 100 percent of the chicks fed on this ration developed either bent legs or true perosis.

TESTS WITH SUPPLEMENTS TO THE PEROSIS-PRODUCING RATION CK324

With the basic perosis-producing ration available as a check, various vitamin and mineral supplements were added to it in an effort to discover a preventive factor. The various supplements used, and their effects, are listed in Table I.

Methods Used in Studies

In conducting the tests, eggs were selected from the Oklahoma Agricultural Experiment Station flock, set in incubators, and the chicks individually wing-banded when hatched. Chicks were divided into lots of 25 to 100 and placed in battery brooders maintained at fairly uniform temperatures. Every precaution was taken to avoid variations in management of lots. The feed was carefully mixed in a 500-pound mixer in 100-pound lots, then transferred to closed cans. Birds in all trials were fed twice daily in feed troughs of the type found on the majority of battery brooders. All trays and feeders were kept clean, and water containers were washed daily. Once each week thoroughout the experiment all chicks were individually weighed, the amount of feed eaten by each lot of birds was weighed, and the chicks carefully examined.

TABLE I.—Percent of Chicks Showing Bent Legs and True Perosis When Fed Perosis-producing Ration CK324 With Various Supplements.*

No. Chicks	Supplements	Bent	True Perosis	Total Defective
		Percent	Percent	Percent
100	Control	37	58	95
100	2% Na_2CO_3	24	47	71
100	1% Na_2SiO_2	13	75	88
100	1% H_3PO_4	0	74	74
100	8% Fiber	6	87	93
50	Control	12	64	76
50	Alcoholic extract of wheat embryo	40	48	88
50	Alcoholic extracted wheat embryo	20	48	68
50	Control	4	79	83
50	Water extract of wheat embryo	16	76	92
50	Water extract of rice bran	18	0	18
50	10% of autoclaved yeast	17	46	63
50	10% of yeast	4	79	83
35	Control	4	96	100
35	Water extract of wheat embryo	8	92	100
35	Water extract of rice bran	17	20	37
35	Water extract of alfalfa flour	12	72	84
35	Autoclaved yeast	13	71	84
35	Ash of wheat embryo	12	72	84
40	Control	8	92	100
40	Minus bone meal	9	63	72
40	Rice bran tea	8	4	12
40	Ash of rice bran	29	68	97
40	Cottonseed meal replacing meat scraps	20	72	92
40	15% of rice bran	26	44	70
40	10% of meat scraps replacing bone meal	23	43	66
25	Control	16	60	76
25	Water extract of wheat embryo	16	52	68
25	Water extract of wheat bran	0	100	100
25	Water extract of wheat gray shorts	8	56	64
25	Water extract of rice bran	0	12	12
25	Ash of 70 pounds of rice bran	0	0	0
25	Ash of 70 pounds of wheat gray shorts	20	4	24
25	Ash of 70 pounds of wheat bran	8	48	56
25	Plus .02% $MnCO_3$	4	0	4
25	Plus .02% $MnCl_2$	0	0	0
25	Plus 1% of $MnCO_3$	0	0	52% died
25	Low protein	16	8	24
25	Casein replacing bone meal and meat and bone scraps	0	0	0
25	Control	8	76	84
25	2% $CaCO_3$	0	4	4
25	Minus bone meal	4	4	8
25	Plus 4% bone meal	4	84	88

* "Control" is in each case basic perosis-producing diet number CK324, page 9.

TABLE I.—(Continued.)

No. Chicks	Supplements	Bent	True Perosis	Total Defective
		Percent	Percent	Percent
25	Control	6	70	76
25	Birds on tin tray	36	54	90
25	Birds on sand tray	33	41	74
25	Minus bone meal+5% calcium lactate	0	16	16
25	Minus bone meal+5% Ca_2 $(PO_4)_2$	0	80	80
25	Control	13	58	71
25	Plus .02% $CoCl_3$	4	83	87
25	Plus .03% ferric lactate	12	52	74
25	Minus meat meal and bone meal + dehydrated meat	0	0	0
25	Minus bone meal+calcium glucinate	13	66	79
25	Minus bone meal and meat scraps + 5% casein	0	0	0

* "Control" is in each case basic perosis-producing diet number CK324, page 9.

The sodium carbonate was added to make the mixture more alkaline and to overcome any acidity from the protein supplements. The phosphorus was added to acidify the mixture and as an added source of phosphate. The fiber was added to the basal diet to increase the bulk of the feces in the hope that it would aid assimilation.

Relation of Maganese to Perosis

At one time it was thought that a vitamin of the B complex might be lacking. To test this theory, water extracts of several potent sources, such as wheat bran, alfalfa, and rice bran were added to the ration. Yeast, autoclaved yeast, and yeast extracts were also included. Of these fractions, none save the water extract of rice bran proved to be beneficial; and it proved to be preventive. The problem then became one of determining what portion of the rice bran was responsible for the prevention of the disorder. Heating the extract did not injure its potency, therefore it was not a thermolabile constituent. At about this time, Sherwood and Couch (1936) of the Texas Experiment Station reported that wheat gray shorts likewise had some preclusive properties; but tests at this station failed to confirm these findings and the tests using rice bran extract therefore were continued. Considerable amounts of rice bran were ashed and the ash added to the basal diet. The ash of rice bran also proved to be a deterrent, indicating that the beneficial portion of the bran was not a vitamin but some mineral which was missing from the ration.

Analysis of the ash of rice bran showed little difference from other cereals save the presence of considerable manganese. The possibility of its being responsible was not apparent until a chance discovery by Wilgus (1936) and his associates at Cornell University was revealed. They had used one sample of a technical grade of mono-calcium phosphate which proved to be a preventive rather than a causative factor. A spectroscopic examination of this salt showed the presence of considerable manganese. This one discovery opened the way for many investigations and demonstrated why the manganese found in rice bran has proved so effective. Since the original report of Wilgus, Norris and Heuser (1936), the effectiveness of manganese in controlling perosis in chicks has been confirmed by Heller and Penquite (1937), Gallup and Norris (1937), Wiese, Elvehjem, Hart and Halpin (1938),

Lyons, Insko and Martin (1939), and Schaible, Bandemer and Davidson (1938). The results of Schaible and associates (1938), obtained through subcutaneous injections of maganese, demonstrated that perosis is linked directly to a deficiency of this element in the tissues of the chick and that exceedingly small amounts are sufficient to check the condition. Their analyses of about five hundred samples of feedstuffs commonly used in chick rations clearly demonstrated that meat scraps, fish meal, dried milk, and bone meal were deficient in manganese, and that manganese was concentrated in the brans of corn, wheat, oats, and rice. Rice hulls or rice bran were found to contain exceedingly high amounts of manganese, which accounts for the beneficial results obtained at this station with a water extract of rice bran.

Further investigations at the Oklahoma Agricultural Experiment Station have proved that only a small amount of manganese is necessary and that large amounts are toxic, although the tolerance of poultry for manganese, as suggested by Wilgus (1939), appears to be relatively high. Through studies of laboratory animals by Gallup and Norris (1937 and 1939), Insko and Martin (1938), Schaible, Bandemer and Davidson (1938), Van der Hoorn, Branion and Graham (1938), Heller and Penquite (1937), and Caskey and Norris (1938), much evidence has accumulated indicating that the quantity of manganese required to protect chicks from perosis ranges from 30 parts per million to 100 parts per million.

Susceptibility of Various Breeds of Chickens to Perosis

Investigators have called attention to the fact that Leghorns are less susceptible to perosis than are other breeds. Branion (1937) noted among chicks from the same hatch, brooded in the same battery room and fed the same ration, that almost every Jersey Black Giant chick and about one-half of the Barred Plymouth Rock chicks were afflicted, while White Leghorn chicks showed little or no evidence of the condition. Gallup and Norris (1939), Serfontein and Payne (1934) and Heller and Penquite (1937) have all reported that differences in availability of manganese strongly influence requirements in all breeds of chickens.

DEVELOPMENT OF CASEIN RATION

In experiments at the Oklahoma Agricultural Experiment Station, bone meal and meat scraps were removed from the basic ration and a protein low in mineral matter obtained from dehydrated meat or casein was substituted. No perosis resulted even though the manganese was low. Perosis did result, however, when calcium phosphate, calcium carbonate and sodium phosphate, or any combination of the salts, were added to make the calcium phosphate high in the ration. In other words, as long as the bone meal and meat scraps were present in large amounts in the ration, perosis was likely to be prevalent. When bone meal was removed, and calcium or phosphorus salts either singly or in combination were added, the incidence of perosis was not greatly reduced.

In order to still further reduce the ash content, the meat scraps were removed and casein added to keep the protein at the same level. The results have been surprising. The ration has produced no perosis, but, on the contrary, has proved to be the most satisfactory broiler ration ever used at this station. The average growth of the chicks fed this ration has been most rapid, and the mortality has been surprisingly low.

The extraordinary feature of this ration, designated as number CQ324, is the fact that no calcium or phosphorus salts are added. Not only has it been used with success in repeated trials at this station, but commercial producers have on repeated occasions praised the results secured.

Ration Number CQ324

	Percent (by weight)
Yellow corn meal	71.00
Wheat gray shorts	5.00
Alfalfa leaf meal	3.00
Dried buttermilk	15.00
Casein (crude commercial)	5.00
Cod liver oil	.25
Salt (sodium chloride)	.75

The analysis of the ration is as follows:

	Percent (by weight)
Moisture	11.19
Protein	16.27
Ether-soluble extract (fat)	4.09
Fiber	3.43
Ash	4.55
Nitrogen-free extract	58.47
Calcium	.414
Phosphorus	.467
Magnesium	.235
Manganese	.00084

TESTS WITH SUPPLEMENTS TO THE CASEIN RATION

The casein ration, although producing such splendid growth and physical condition, was nevertheless very similar to that which had been found to produce perosis (Ration CK324; see page 9). Therefore it was reasoned that the former procedure might be reversed, and an attempt made to find the cause of the bone disorders by adding supplements to a ration known to prevent perosis (Ration CQ324) rather than by adding them to one which was known to produce it.

TABLE II. The Occurrence of Perosis and Growth Response Resulting From the Use of Various Supplements to the Casein Ration CQ324.

Lot No.	PERCENTAGE OF: Calcium	PERCENTAGE OF: Phosphorus	Manganese (grams per 100 lbs. of feed)	Percent of chicks with bent legs	Percent of true Perosis	Total gains in 6 weeks (grams)
1	Control ration CQ324			0.0	6.6	456
2	.5			6.6	20.0	293
2M	.5		9.46	0.0	0.0	410
3	1.0			7.1	21.4	226
3M	1.0		9.46	0.0	0.0	250
4		.5		7.1	0.0	275
4M		.5	9.46	0.0	0.0	333
5		1.0		0.0	0.0	182
5M		1.0	9.46	0.0	0.0	160
6	1.0	.5		0.0	66.6	350
6M	1.0	.5	9.46	0.0	0.0	405
6MM	1.0	.5	18.92	0.0	0.0	430
7	1.0	*Glycerol phosphate		9.1	54.5	206
8	1.0	1.0		0.0	45.1	264
8M	1.0	1.0	9.46	0.0	0.0	286

M—Manganese added.
MM—Twice the above amount (9.46 grams) added. Total added, 18.92 grams.
*—Introduced subcutaneously.

To test this hypothesis, various supplements were added to this casein ration CQ324 and fed to groups of chicks. The supplements fed and the reactions of the chicks to them are given in Table II. Inspection of the results shown in Table II, which have been confirmed in other trials, demonstrates again that the addition of minerals is productive of defective bone formation, although the picture is not identical with that presented in previous trials. In these later trials, the addition of calcium was always associated with increased perosis, while an increase of both calcium and phosphorus hastened the incidence of perosis. It is interesting to note that introduction of phosphorus subcutaneously, using a hypodermic needle, was just as injurious as was phosphorus fed in the ration. This points to the fact that calcium as well as phosphorus is associated with perosis. Wilgus and Patton (1939) have recently called attention to this association of calcium and phosphorus in producing perosis. They state that "excessive calcium in the diet is apparently essential for the stimulation of perosis."

At this time it was considered advisable to make a study of the chemical composition of the blood of groups of chickens to ascertain if any changes in the mineral balance might explain the occurrence of these conditions. Blood was taken by heart puncture from five groups of chickens reported in Table II when they had reached six weeks of age. The phosphorus partitions were studied by the method of Youngburg and Youngburg (1930); and the inorganic calcium of the serum was studied by the method of Roe and Kahn (1928). The data for three separate analyses of the blood of these chicks have been averaged, and the results are presented in Table III.

TABLE III. Variations in the Calcium and Phosphorus Content of the Blood of Five Lots of Chickens Reported in Table II.
(Mg per 100 ml of blood)

*Ration	Lot No.	Total Whole Blood	Total Plasma	Total Cells	Liquid Plasma	Liquid Cells	Inorganic Plasma	Inorganic Cells	Calcium Serum
**Control	1	119.1	20.7	288.6	18.6	23.6	8.2	9.5	13.9
Control+ 1.0% Ca	3	107.8	17.6	265.5	17.2	21.3	3.1	6.6	16.7
Control+ 1.0% P.	5	113.7	21.7	286.6	17.8	22.8	10.0	11.1	11.9
Control+ 1.0% P. + .5% Ca.	6	114.8	20.5	274.6	18.2	21.7	8.0	9.2	13.8
Control+ 1.0% Ca. + .5% P. + manganese	6MM	116.5	20.5	278.2	12.5	20.9	8.8		16.4

*See Table II.
**Control fed ration CQ324, page 13.

The information to be gained from this table suggests a correlation between the presence of calcium and the defective bone formation. It will be noted that there are no significant changes in the total phosphorus of the whole blood, the cells, and the plasma, save in the case of the chicks in lot number 3. In that lot, where the chicks were receiving one percent calcium supplement, the phosphorus is materially reduced.

The most interesting change is to be noted in the case of the inorganic phosphorus of both plasma and cells, where the blood of chicks receiving the calcium supplement contained only 40 percent as much inorganic phosphorus as did the blood of the chicks fed the basal ration.

The group which received one percent calcium and one-half percent phosphorus and contained 60 percent perosis birds had a lower total phosphorus. The addition of a trace of manganese produced a slightly higher level of total phosphorus, but the change in the phosphorus level was not sufficient to predict the function of manganese in this connection. The fact that calcium is just as productive of perosis as phosphorus is confirmed by similar statements in the recent work of Wilgus and Patton (1939).

SUMMARY

1. A ration was developed that was effective in producing perosis in 76 to 100 percent of the chicks used.
2. A drinking solution consisting of the water extract of rice bran prevented perosis in nearly 100 percent of the chicks involved.
3. Ether, alcohol, and water-extracts of the components of the basal ration, when added to the basal ration, did not prevent the incidence of perosis.
4. Water extracts of wheat bran, wheat shorts, wheat embryo, and alfalfa did not alleviate the condition.
5. The growth of the chicks consuming water extracts of rice bran was increased, disproving the observation often made in the past that the most rapidly growing chicks were more susceptible to perosis.
6. The chicks consuming water extracts of rice bran were larger, smoother, and possessed better colored shanks than the control group.
7. The type of wire floors used in battery brooders did not increase the occurrence of perosis.
8. The ash of 70 pounds of rice bran added to 100 pounds of the basic perosis-producing diet prevented the occurrence of perosis.
9. A correlation seems to exist between the manganese content of the rations and supplements used and their corrective properties.
10. The exact quantity of manganese required in chick diets varies so greatly, depending on chickens used and amounts of minerals in the ration, that more work will need to be done to determine all the causative factors.
11. It is obvious that the chickens used, and the quantity of calcium and phosphorus, influenced the amounts of manganese required to prevent perosis.
12. Manganese was not the only preventive of perosis, since a ration containing approximately the same quantity of manganese as the perosis-producing diet has produced practically no perosis.
13. The introduction of phosphorus subcutaneously was just as productive of perosis as when given orally.
14. Calcium was associated with perosis to a much greater extent than was phosphorus.
15. The birds receiving a calcium supplement had only 40 percent as much inorganic phosphorus present in plasma and cells as determined by blood analysis.
16. Manganese fed at a level of 4,800 parts per million decreased the viability and rate of growth of the chicks.

LITERATURE CITED

1. Branion, H. D. Minerals in Poultry Nutrition.
Scientific Agriculture 18:217, 1938.
2. Card, L. E. Mistake to Feed Extra Minerals to Chicks.
Ill. Agr. Exp. Sta. Annual
Rept. 113, 1928-1929.
3. Caskey, C. E., and Norris, L. C. Further Studies on the Role of Manganese in Poultry Nutrition.
Poul. Sci., 17:433, 1938. (Abstract.)
4. Clifcorn, L. E., Elvehjem, C. A., and Hart, E. B. The Development of a Ration for the Study of Perosis in Chicks.
Poul. Sci., 17:28, 1938.
5. Gallup, W. D. and Norris, L. C. Studies of the Perosis-Preventing Properties of Manganese.
J. Biol. Chem., 119:XXXVI, 1937. (Proceedings.)
6. Gallup, W. D. and Norris, L. C. The Amount of Manganese Required to Prevent Perosis in the Chick.
Poul. Sci., 18:76, 1939.
7. Heller, V. G., and Penquite, R. Factors Producing and Preventing Perosis in Chickens.
Poul. Sci., 16:243, 1937.
8. Herner, M. C. and Robinson, A. D. A Study of Leg-bone Deformities in Growing Chicks.
I. Deformities Produced by High Mineral Rations.
Poul. Sci., 11:283, 1932.
9. Hunter, J. E., and Funk, E. M. The Production of "Slipped Tendon" in Chicks by Experimental Feeding.
Proc. 22nd Poul. Sci. Assoc., 45, 1930.
10. Hunter, J. E., Dutcher, R. A., and Knandel, H. C. Further Studies on the Production of Experimental "Slipped Tendons" or "Hock Disease" in Chicks.
Poul. Sci., 10:392, 1931. (Abstract.)
11. Hammond, J. C. Inorganic Phosphorus and Perosis.
Poul. Sci., 14:302, 1935. (Abstract.)
12. Holmes, A. D., Pigott, M. G., and Moore, W. B. Mineral Content of Tibiae from Chicks with Slipped Tendon.
Poul. Sci., 12:356, 1933.
13. Insko, W. M., Jr., Sowell, D. F., and Lyons, M. Is Phosphorus a Causative Factor in the Production of Slipped Tendon?
Poul. Sci., 13:370, 1934.
14. Insko, W. M., Jr., Lyons, M., and Martin, J. H. The Quantity Requirement of the Growing Chick for Manganese.
J. Nutrition, 15:621, 1938.
15. Lyons, M., Insko, W. M., Jr., and Martin, J. H. The Effect of Intraperitoneal Injections of Manganese, Zinc, Aluminum, and Iron Salts on the Occurrence of Slipped Tendon in Chicks.
Poul. Sci., 17:12, 1938.
16. Maw, W. A. Minerals in Poultry Feeding, 2, Bone Char in the Chick Ration as Affecting Perosis.
Unpublished Data 1937. Quoted by Branion, H. D.
Minerals in Poultry Nutrition,
Scientific Agriculture, 18:241, 1938.
17. Milby, T. T. A Statistical Analysis of Some Experiments on Slipped Tendons.
Poul. Sci., 12:352, 1933.

18. Milby, T. T. Factors Influencing a Malformation of the Leg Bones of Growing Chickens.
Iowa Agr. Exp. Sta. Res. Bul., 172, 1934.

19. Milby, T. T. A Method for Determining the Ash Content of the Leg Bones of Chicks with Slipped Tendon.
Poul. Sci., 14:247, 1935.

20. Milne, H. I. Untersuchungen Uber Huhnerperosis (Studies of Perosis in Chicks).
Proc. 6th World's Poultry Congress, V. 3. 431, 1936.

21. Parkhurst, R. T., and McMurry, M. R. Factors in the Development of Deforming Leg Weakness in Chickens.
Jour. Agr. Sci., 23:311, 1933.

22. Payne, L. F., Hughes, J. S., and Leinhardt, H. F. The Etiological Factors Involved in the Malformation of Bones in Young Chickens.
Poul. Sci., 11:158, 1932.

23. Roe, J. H., and Kahn, B. S. The Colorimetric Determination of Blood Calcium.
J. Biol. Chem., 81, p. 1, 1928.

24. Schaible, P. J., Moore, J. M., and Conolly, R. A. Factors Influencing the Incidence of Perosis in Barred Rock Chicks.
Poul. Sci., 12:324, 1933. (Abstract.)

25. Schaible, P. J., Bandemer, S. L., and Davidson, J. A. The Manganese Content of Feedstuffs and Its Relation to Poultry Nutrition.
Mich. Agr. Exp. Sta. Tech. Bul., 159, 1938.

26. Serfontein, P. J., and Payne, L. F. Inheritance of Abnormal Anatomical Condition in the Tibial Metatarsal Joints.
Poul. Sci., 13:61, 1934.

27. Sherwood, R. M., and Couch, J. R. Wheat Gray Shorts for the Prevention of Slipped Tendons in Battery Brooded Chicks.
Texas Agr. Exp. Sta. Bul., 525, 1936.

28. Solun, A., and Arsenjaw, A. Foot Disorders in Battery Reared Chicks.
Bredermann's Zentralblatt, 6, 1934.

29. Titus, H. W. Perosis, or Deforming Leg Weakness in the Chicken.
Poul. Sci., 11:117, 1932.

30. Van Der Hoorn, R., Branion, H. D., and Graham, W. R., Jr. Studies in the Nutrition of the Chick. III. A Maintenance Factor Present in Wheat Germ and the Effect of the Addition of a Small Amount of Manganese Dioxide to the Diet.
Poul. Sci., 17:185, 1938.

31. Wilcke, H. L. Variation in Body Weight, Bone Ash, Plasma Calcium, and Inorganic Phosphorus of Chicks Affected with so-called "Slipped Tendons" or Rickets.
Thesis Iowa State College (Quoted by Milby), 1932.

32. Wilgus, H. S., Jr., and Patton, A. R. Factors Affecting Manganese Utilization in the Chicken.
Jour. Nutr., 18, 1938.

33. Wilgus, H. S., Jr., Norris, L. C., and Heuser, G. F. The Role of Certain Inorganic Elements in the Cause and Prevention of Perosis.
Science, 84, 252, 1936.

34. Wilgus, H. S., Jr., Norris, L. C. and Heuser, G. F. The Role of Manganese in Poultry Nutrition.
Proc. of Seventh World's Poultry Congress, 171, 1939.

35. Weise, A. C., Elvehjem, C. A., Hart, E. B., and Halpin, J. G. Studies on the Prevention of Perosis in the Chick.
Poul. Sci., 17:33, 1938.

36. Youngburg, G. E., and Youngburg, M. V. A System of Blood Phosphorus Analysis.
J. Lab. and Clin. Med., 16, 1930.