

AGRICULTURAL EXPERIMENT STATION

KANSAS STATE AGRICULTURAL COLLEGE
MANHATTAN, KANSAS

THE IMPORTANCE OF VITAMIN A AND VITAMIN C IN THE RATION OF SWINE

Concerning Especially Their Effect on
Growth and Reproduction



PRINTED BY KANSAS STATE PRINTING PLANT
B. P. WALKER, STATE PRINTER
TOPEKA 1928
12-3140

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THE IMPORTANCE OF VITAMIN A AND VITAMIN C IN THE RATION OF SWINE¹

Concerning Especially Their Effect on Growth and Reproduction

J. S. HUGHES, C. E. AUBEL, AND H. F. LIENHARDT

INTRODUCTION

A series of experiments was carried on prior to 1920 at the Kansas Agricultural Experiment Station to study the nutritive value and the deficiencies of corn when used as a ration for growth and reproduction in swine. Much valuable information was secured concerning the manner in which the protein and mineral deficiencies of corn could be supplemented (14). Some results were obtained, however, which were difficult to explain. Some of the pigs failed to grow and reproduce on certain rations on which others had developed normally. This gave rise to the suspicion that an unknown factor which had not been controlled must have caused this variation in the results obtained.

The clue to this possible cause came with the discovery of Steenbock and Boutwell in 1920 (15) that yellow corn contained vitamin A, whereas white corn contained little, if any, of this substance. It had been held previously as a general opinion among swine feeders that there was no difference in the nutritive value of white and yellow corn. Consequently no record was kept of whether or not white or yellow corn was used in these experiments. Mixed white and yellow corn was probably most generally fed. The per cents undoubtedly varied and at times no doubt all white or all yellow corn may have been fed. According to Steenbock and Boutwell's discovery, this variation in the amount of yellow corn in the feed would cause a variation in the amount of vitamin A which the hogs received. This

Acknowledgment.—The authors especially wish to acknowledge the aid and suggestions of Dr. C. W. McCampbell in planning and conducting the experimental work. Credit is also due Mr. H. B. Winchester, who was in charge of the project until the fall of 1922, for the careful and painstaking notes and data he collected, and to Mr. H. W. Marston, who had charge after this time, for his detailed notes on the behavior of the animals and for other data in connection with the experiment.

1. Contribution No. 139 from the Department of Chemistry, No. 88 from the Department of Animal Husbandry, and No. 43 from the Department of Veterinary Medicine. This experiment in its beginning was a cooperative project in animal nutrition between the Departments of Chemistry and Animal Husbandry. Later, when post-mortem and histological studies were found necessary, the cooperation extended to the Department of Veterinary Medicine.

variation in the vitamin A content of the ration suggested a possible explanation of the various results obtained.

Previous to 1920 no definite information was available on the vitamin A requirement of swine. In that year an experiment was planned to study the vitamin A requirement for growth and reproduction in hogs. Subsequently a number of experiments have been reported on the vitamin A requirements of these animals.

Shortly after Steenbock and Boutwell (13) discovered by experiments with rats that yellow corn was a better source of vitamin A than white corn, Morrison, Bohstedt, and Fargo (6) tested the relative value of yellow and white corn in feeding experiments with pigs. They found that if vitamin A was not supplied by some other ingredient of the feed the pigs fed white corn did not gain so well as those receiving yellow corn. They found no difference in the nutritive value of yellow and white corn if vitamin A was provided in the ration. In later tests Morrison, Bohstedt, and Fargo (7) showed the pigs allowed to develop to 142 pounds on an adequate feed before starting on the test would make as good gains during the fattening period on white corn as they would on yellow corn. This was possible because of the storage of vitamin A in the liver and other organs, previous to the test. This storage enabled the pigs to make normal gains during the comparatively short fattening period even though they had no vitamin A.

Following this, Orr and Crichton in 1924 (9) as a result of some well controlled experiments concluded that the requirement of the pig for vitamins A and C during the usual fattening period was so low that there was little possibility of pigs suffering from a lack of either of these vitamins under practical feeding conditions.

The vitamin A requirement of swine was also studied by Rice, Mitchell, and Laible (11). Forty-eight pigs were fed on rations of white corn and tankage and twenty-eight on yellow corn and tankage. Six pigs fed white corn showed pathological symptoms and two of the pigs died before they reached 225 pounds in weight. 411 had been started on feed between the weights of 50 and 70 pounds. In other tests it was shown that when 1 ounce of alfalfa meal, a good source of vitamin A, was fed daily to the pigs receiving white corn, no disturbances resulted. They also found that the reproductive powers of sows kept on a feed of white corn and tankage were gradually undermined. One such sow produced dead litters in the third and fourth gestation periods.

Nelson, Lamb, and Heller (8) found marked nutritional disturb-

ances in pigs farrowed by sows which received a feed low in vitamin A during the gestation and lactation period. These pigs, however, did not develop the eye lesions which characterize avitaminosis A in other animals as in the rat, rabbit, and chicken.

Golding, Zilva, Drummond, and Coward (3), while studying the relation of the fat-soluble factor to rickets and growth in pigs, used a basal ration deficient in vitamin A. At the end of 111 days the pigs receiving this ration low in vitamin A were down in their hind legs, "off their feet." The condition was not such as would justify calling the disease rickets.

They were unable to account for these results, for it was not recognized at that time that in the case of swine the lack of vitamin A will not produce rickets, but will result in a nervous disorder showing some symptoms similar to rickets. With other experimental animals these nervous disorders resulting in symptoms similar to rickets had not been observed. The one outstanding thing that characterized the depletion of vitamin A in the small experimental animals was eye lesions as found by McCollum (4) and further verified by many other investigators. In the case of swine this ophthalmic reaction is scarcely noticeable even in the most advanced stages of avitaminosis A. This important difference in the reaction of rats and swine to the lack of vitamin A complicated the interpretation of the results in the study of rickets in swine when feeds low in vitamin A were used.

Bohstedt and coworkers (1) used such a basal ration low in vitamin A in studying "stiffness" in pigs. In the discussion of their results they did not distinguish clearly between the results produced by a lack of vitamin A and those produced by a lack of vitamin D and minerals. They recognized that vitamin A might be a factor in causing these results, but in just what manner they were not certain, for they state in the general summary that, "There is evidence that vitamin A, perhaps indirectly, plays a part in the prevention of stiffness in pigs."

From this it seems these workers did not recognize that the most characteristic symptom of avitaminosis A in the case of swine is a sort of stiffness or lack of coordination resulting from impairment of the nervous system. They were dealing with at least two distinct kinds of deficiency diseases characterized by stiffness and paralysis, one due to a lack of vitamin A and the other due to a lack of vitamin D or minerals. When both of these factors were present with an adequate supply of minerals "stiffness" did not develop.

So far as the authors are aware, there have been no reports describing the symptoms and lesions of pigs in the advanced stages of avitaminosis A.² It is mainly to supply this information that this work is presented. In addition, results will be presented to show that pigs do not need vitamin C in their feed.

AVITAMINOSIS A IN SWINE

This study of vitamin A in the ration of swine was begun February 24, 1921. Since that time six trials or lots of pigs at different times have been fed a ration deficient in vitamin A. In five of the lots

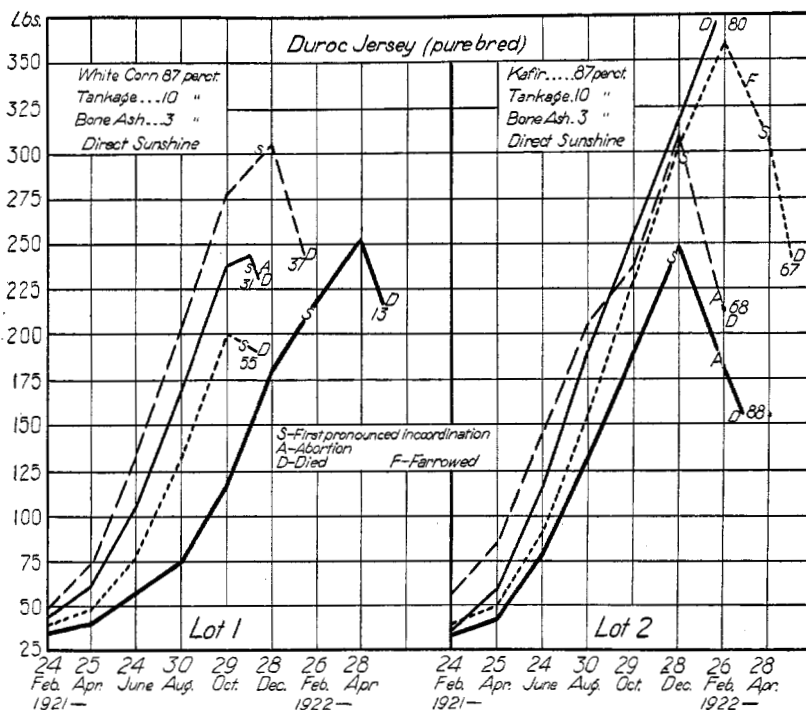


Fig. 1.—Graphs showing rate of growth of the pigs in lots 1 and 2 which received a feed low in vitamin A. These curves show fairly normal growth the first months of the test. Only one of the eight pigs, No. 80, lot 2, died without developing typical nerve disorders.

the pigs were placed on the experimental rations at weaning time, in the other lot, lot 5, pigs weighing from 200 to 225 pounds were placed on the ration. The early history of the pigs used in the tests was known as the animals were from the college herd.

2. Brief statements concerning the work herein reported have been made, however, in recent Director's Reports of the Kansas Agricultural Experiment Station.

All the pigs in the six lots were kept on a diet deficient in vitamin A until they died or until they reached such an advanced stage of the disorder that death would have followed in a short time, when they were killed for post-mortem examination. The only exceptions were three pigs in lot 3 that were given cod-liver oil after they developed the symptoms of avitaminosis A.

The feed used in five of the lots consisted of white corn, 87 per cent, tankage, 10 per cent, and bone ash, 3 per cent. In the other lot, lot 2, kafir replaced the white corn. This combination of feeds furnishes a satisfactory supply of proteins, minerals, and vitamin B.

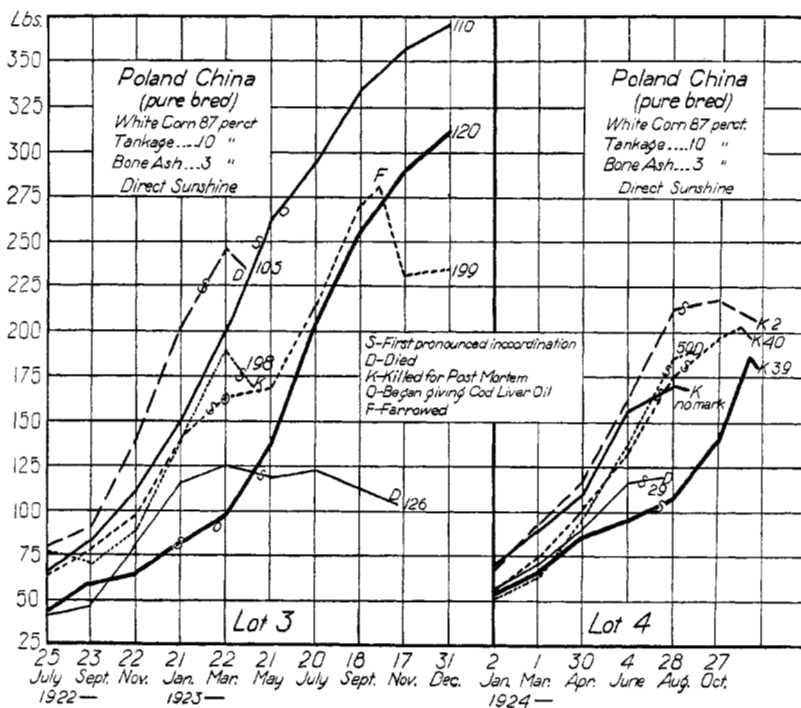


FIG. 2.—Graphs showing rate of growth of the pigs in lots 3 and 4. Three of the pigs, Nos. 110, 120, and 199, were given cod-liver oil after nervous disorders developed. The remaining pig in the same lot, No. 126, began to recover as a result of eating the feces of the three cod-liver-oil-fed pigs. She developed typical symptoms after being removed to another pen.

Vitamin D was supplied by allowing the pigs an outside yard where they received an abundance of direct sunshine. No feed could be obtained from the ground as the floor of the yard was cement. The ration was deficient in only vitamin A and vitamin C. Since pigs do not need vitamin C for normal growth, as will be shown later, the

only food factor known to be essential for normal growth which these pigs did not receive was vitamin A.

The pigs were housed in a well-lighted and well-ventilated barn, provided with individual feeding stalls. The feed for each pig was weighed. The amount was changed from time to time so that each

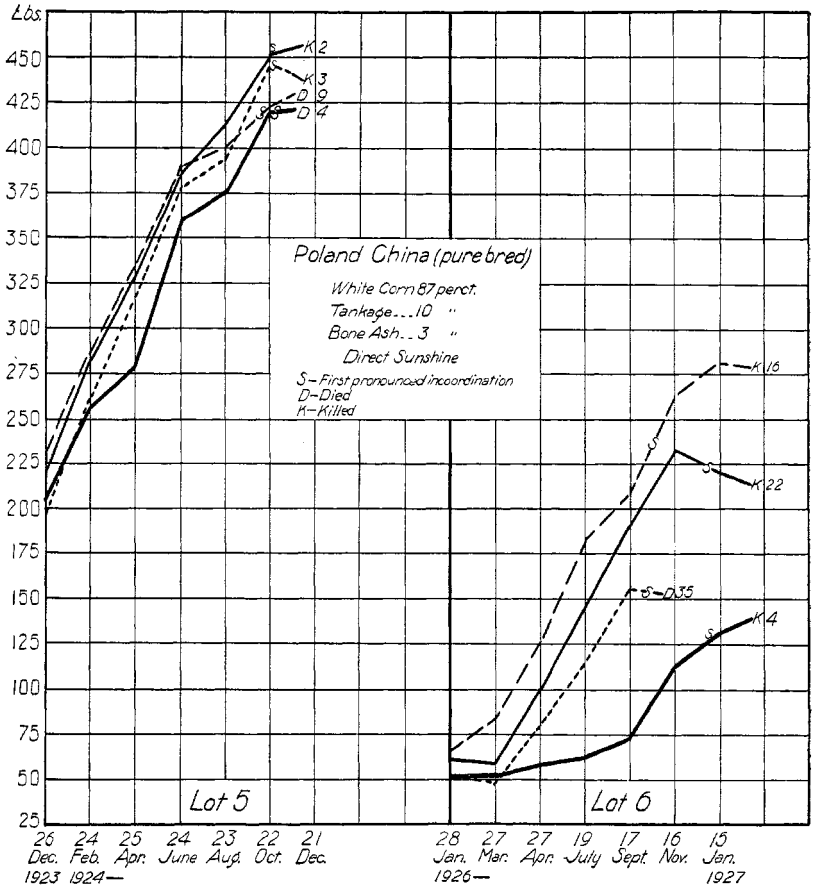


FIG. 3.—Graphs showing rate of growth of the pigs in lots 5 and 6. Lot 5 differs from all other lots in that the pigs were not placed on the experiment until they reached the weight of 200 to 230 pounds. They developed the definite incoordination as soon as the others.

pig was given just the amount of feed it would clean up. Individual weights and measurements were taken every 30 days during each or the six trials. A record of the condition of each pig was kept. This record included both still and motion pictures of the most important developments throughout the test. The blood was analyzed for

calcium and inorganic phosphorus at several stages in the experiment. Post-mortem examinations were made of the pigs that died and of those killed after reaching the advanced stages of the deficiency diseases. Samples of various tissue were taken for histological examination, and samples of the bone and in some cases samples of the brain were taken for chemical analysis.

CLINICAL OBSERVATIONS

THE EFFECT ON GENERAL BEHAVIOR

There was nothing in their behavior during the first four or five months of the experiment to indicate that the pigs were receiving feed deficient in vitamin A. Even the rate of growth, with one or

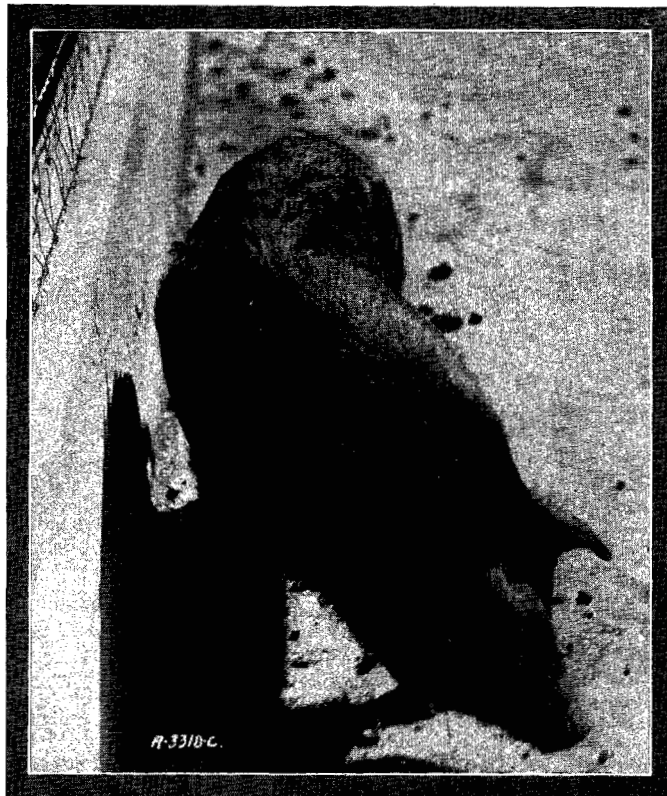


FIG. 4.—Sow No. 13, lot 1. She is one of the 26 pigs receiving a diet deficient in vitamin A. She shows the characteristic curving of back with head drawn to one side, developed in a number of pigs in the early stages of the disorder. Frequently hogs in this condition when excited went around in a circle. When the pigs were placed on feed immediately after weaning, the average time required to develop these symptoms was 219 days.

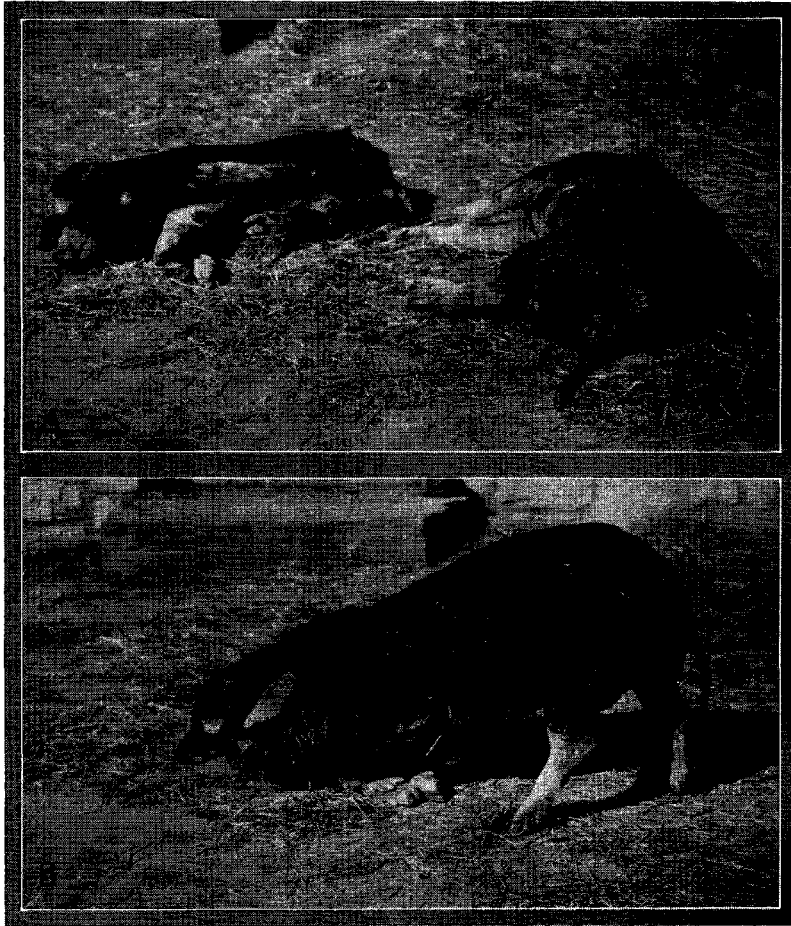


FIG. 5.—(A) Pigs Nos. 4 and 22, lot 6, showing the advanced stages of the nervous disorder, when fed on a diet deficient in vitamin A; namely, white corn, tankage, and bone ash. At the time this picture was taken these pigs had received the ration for a period of 13 months after having been placed on the feed at weaning time. The pig to the left shows the spasms frequently displayed by the pigs on this ration. Observe how the hind feet are drawn up tightly against the body and how the front feet are extended rigidly to the front. During a period of seven years, six lots of pigs, 26 in all, received a ration deficient in vitamin A. In every case striking nerve disorders appeared. This condition was prevented and cured in other lots by the use of vitamin A supplements, such as butter, alfalfa hay, cod-liver oil, and yellow corn. This indicates that the lack of vitamin A in the diet produces an impairment of the nervous system. (B) Pigs Nos. 4 and 22 showing the posture of one when helped to her feet. Most pigs lost the use of their hind quarters first through incoordination. This sow and two others lost the use of their fore quarters first. She could not stand on her fore feet without help.

two exceptions, was uniform and fairly rapid the first eight or ten months. (Figs. 1, 2, and 3.)

Marked restlessness in the pigs was the first indication that they were not normal. While pigs in an adjacent pen, kept under identical conditions with the exception that they received vitamin A, were lying down sleeping, the pigs on the inadequate diet would be up moving around in their lot. In most cases after this restlessness, the pigs developed a mean disposition. This condition was particularly manifested when the pigs were driven from their pens for weighing and during the time it was necessary to hold them to take measurements.

The next noticeable symptom (fig. 4) was a characteristic posture and gait while standing and walking. In many of the cases while standing and walking the pig's back was arched bringing the hind feet much farther forward than normal. There was an indication of soreness or stiffness in walking. The hind feet moved along in short, quick steps. The next symptom was a definite incoördination. In all except two cases this appeared first in the hind quarters. In pig No. 22, lot 6, and pig "No mark," lot 5, this incoördination appeared first in the fore legs. (Fig. 5B.) As this incoordination became more advanced the pigs were unable to get up on their feet without help. (Fig. 5.) In several of the cases they could get up on their front feet and drag themselves about the pen.

Because of this posture the condition was called by some "posterior paralysis." It is felt this term is incorrect as the condition was one of incoordination and not true paralysis. In paralysis there is a loss of the ability to move certain of the muscles. In no case in these trials did the pigs lose the power to move their legs. They did, however, lose the power to control their movements as the condition developed. Furthermore, the pigs would often go into severe spasms. (Figs. 5A and 7.) In most cases these spasms would begin with the pigs stretched out on their sides moving their feet rapidly back and forth as if running. It would usually end with the body stiffened out with all muscles tense. In this condition the head would either be drawn down between the fore legs or as far back as possible. This period was usually followed by very difficult breathing. The entire spasm generally required from one to four minutes. These spasms could usually be brought on by exciting the pigs. This was particularly the case if they had been lying quietly for some time. If they were aroused slowly and allowed to move about the pen for a while, it was more difficult to bring on

the spasms. In the advanced stages of the disorder the spasms developed spontaneously.

In the case of pig No. 67, lot 2, the spasms occurred at the rate of two or three an hour during seven days time. While the pigs were lying still tremors would play back and forth over the body. In a few pigs one or more of the limbs trembled continuously.



FIG. 6.—Sow No. 68, lot 2, showing a small corneal ulcer, the only case of infection of the eyeball developed during the experiment. This is in marked contrast to the condition found in rats receiving a diet deficient in vitamin A for they usually develop corneal ulcers. The pigs in this experiment showed a slight watering of the eyes but in no case developed severe conjunctivitis. However, they did show impaired vision including total blindness in some cases. This is probably due to degeneration of the nervous system.

Since eye lesions seem to be the most characteristic lesion in avitaminosis A in other experimental animals, the condition of the eyes was watched with particular care as the tests proceeded. No cases of severe conjunctivitis developed and in no pig was there an apparent infection involving the tissues surrounding the eye as is so often the case with rats, rabbits, dogs, pigeons, and chickens. Pig No. 68, lot 2, developed a small ulcer on the cornea. (Fig. 6.) Instead of a dryness of the eye resulting from involvement of the tear glands as reported by Mori (5) in case of rats, there was a tendency in some of the pigs to watering of the eyes. This eye watering is common with chickens and has been noticed in dairy cows and dairy calves³ kept on a feed low in vitamin A. In some pigs there was a slight swelling of the tissues around the eye, but in none did it approach the condition observed in other experimental animals.

Quite early in the development of the disorder there appeared an impairment of vision. This probably resulted from some derangement of the nervous system as there was no evidence of any lesion in the eye itself. There was no opacity in either cornea or other tissues or fluids of the eye. The response to varying intensity of light seemed to be very slow. In the advanced stages the iris was open and would not respond to intense direct sunlight. This was tested a number of times by taking the pigs out of the barn into the direct sunshine. Not only the sight, but also the smell and hearing seemed to be impaired. Wolbach and Howe (15) found that rats lost the sense of smell when kept on a feed free from vitamin A.

The appetite remained very good until the last stages of the disorder. Some of the pigs, even though they were unable to stand alone when assisted to arise, would eat if placed before food and held there. Pigs Nos. 37, lot 1, and 67, lot 2, were given water by means of a rubber tube during the last week when they were unable to drink while standing. They would chew and swallow some feed if placed in the mouth. The good appetite no doubt accounts for the steady, even growth during the first eight or ten months of the experiment. In about 75 per cent of the cases the definite incoordination developed before the break in the growth curve. (Figs. 1, 2, and 3.) The number of days before definite incoordination and death occurred in the different animals is shown in table I.

3. Unpublished data, Kansas Agricultural Experiment Station.

TABLE I.—THE APPEARANCE OF FIRST PRONOUNCED INCOORDINATION DISORDER AND THE DEATH OF PIGS KEPT ON FEED DEFICIENT IN VITAMIN A.

Lot number and date placed on experiment.	Pig No.	Number of days before—		Remarks.
		First symptoms.	Death.	
Lot 1 2-21-'21	55	287	293	No typical lesions. Lungs congested. Lungs congested. Septicemia. Fetuses retained in uterus 81 days after farrowing time.
	31	287	295	
	37	285	345	
	13	356	(a) 434	
Lot 2 2-21-'21	67	361	458	Post-mortem decomposition. Septicemia. Gangrene in uterus. Died 64 days after aborting nine dead pigs. Had shown no signs of disorder. Ate full feed in morning, found dead at 2 p. m. Due to farrow in nine days. Ten fetuses in uterus. Unable to stand last 60 days. No typical lesions.
	68	304	370	
	80	357	357	
	88	301	386	
Lot 3 7-25-'22	105	214	266	Post-mortem decomposition. Recovered on cod-liver oil. Recovered on cod-liver oil. No typical lesions. Abscesses in both large and small intestines. Recovered on cod-liver oil.
	110	285	
	120	180	
	128	280	476	
	108	251	(a) 274	
	199	225	
Lot 4 1- 1-'24	2	266	(a) 356	No typical lesions. Congestion of the lungs. No typical lesions. Post-mortem decomposition. No typical lesions.
	29	187	226	
	39	232	(a) 356	
	40	260	(a) 350	
	50	232	273	
	No mark	232	(a) 260	
Lot 5 12-26-'24	2	298	335	Post-mortem decomposition. Ulcers of stomach. Inflammation of intestines. No lesions. No lesions.
	3	299	341	
	4	299	(a) 346	
Lot 6 1-28-'26	9	293	(a) 352	No characteristic lesions. No characteristic lesions. No characteristic lesions. No characteristic lesions.
	4	312	(a) 403	
	16	252	276	
	22	312	(a) 403	
	35	264	(a) 403	

(a) Killed.

Ration.—The ration used consisted of white corn, 87 per cent, tankage, 10 per cent, and bone ash, 3 per cent, except lot 2, which received blackhull kafir instead of white corn. All lots had access to direct sunlight.

The daily notes kept by Mr. Winchester and later by Mr. Marston give a very vivid picture of the condition of the pigs as the deficiency disease developed. The lots referred to in these notes received a ration low in vitamin A. This ration consisted of white corn, 87 per cent, tankage, 10 per cent, and bone ash, 3 per cent, except lot 2, in which blackhull kafir was fed in place of white corn. The notes for a few days as recorded by Mr. Winchester follow:

JANUARY 13, 1922.

8:30 a. m. Pig No. 37, lot I, unable to rise on hind legs. Sits on haunches. Has not eaten evening or morning feed.

5:30 p. m. Animal lying prone on right side. Has frequent convulsions or

spasms of about 1 to 2 minutes duration. Paws air frantically, then holds legs out stiff. Head drawn between fore legs. Top line has convex shape.

Pig No. 68, lot 2, drags herself around by fore legs unable to get on all feet at once. Pig No. 88 down on right side showing same symptoms as pig No. 37, lot 1.

5:30 p. m. Pig No. 68 walking around but stiff and stilted. Pig No. 88 still down with spasms lasting about 2 minutes occurring about every 15 to 20 minutes for about an hour. Most of the spasms have occurred at meal times up to and including this date.

JANUARY 14, 1922.

Pig No. 37, lot 1, still on side. Has not eaten in 48 hours. Spasms not so frequent but animal apparently has lost control of legs from standpoint of being able to rise. During spasms she quivers perceptibly and holds fore legs out stiff. Does not move hind legs very much.

Pig No. 68, lot 2, still able to rise on feet with effort. Gets up usually like a cow, hindquarters first.

Pig No. 88, same lot, rarely moves unless disturbed. In spasms observed at this time she folds left leg up to body and holds right leg stiff and pawing with it. Her top line is convex in shape with tail drawn up and held back.

JANUARY 19, 1922.

The feeder reported that pig No. 37 was found at 6 a. m. over by doorway leading to outside lot. This is a distance of about 15 feet and she probably got over by pulling herself with her fore legs. Both animals, pigs Nos. 37 and 88, were watered by means of a hose and a bottle. They were also given small amounts of feed. Pig No. 88 can eat hers alone, but No. 37 has to have hers poured into her mouth and seems unable to pick her feed off the floor like No. 88. They are being watered and fed small amounts of feed twice daily. Pig No. 68 still moves around in a wobbly manner and carries herself half-moon shaped.

The pigs in other lots appear to be O. K.

JANUARY 21, 1922.

Pig No. 37 able to stand on all four feet if helped up. Does not care to eat feed out of trough or off floor, but will eat if her upper lip is raised and feed inserted. Pig No. 88 still down although she eats heartily when offered feed.

Pig No. 68 appears to be in fairly good shape although she still walks a trifle stiff. A rather noticeable thing about this pig is that her disposition improves as her condition improves and *vice versa*. All other lots seem to be in good shape.

FEBRUARY 1, 1922.

Pig No. 37 aborted seven pigs during previous night, being 86 days after breeding. Pigs turned over to pathological department. Pig No. 88 had a spell this morning in which she moaned, had difficulty in getting her breath, stiffened out and gave all indications as though she might die at any time. However, at 5:30 p. m. she appeared to have come out of it O. K.

FEBRUARY 7, 1922.

Pig No. 88 shows no improvement. Pig No. 68 walks around but acts nervous and uncertain how to handle her hind feet. She stands with her hind feet well toward the front legs and picks them up and acts as though she is trying to find a safe footing for them.

FEBRUARY 14, 1922.

Pig No. 68 a trifle weaker to-day. Pig No. 88 about the same.

FEBRUARY 15, 1922.

Pigs Nos. 68 and 88 show no apparent changes, except that they seem to be getting weaker. Pig No. 13, lot 1, when being turned out of her stall this morning wheezed, coughed, and seemed to have a hard time breathing. She fell and started bleeding at mouth. After a few minutes rest she recovered sufficiently to move out into the outside lot,

FEBRUARY 19, 1922.

All pigs measured to-day. Pig No. 88 has a sort of matting over eyelids, which is similar to eye that No. 37, lot 1, showed. Pig No. 68 has a foamy discharge at her nostrils.

The following notes are taken from Mr. Marston's records:

MARCH 2, 1923.

Pig No. 105, lot 3, has been very irritable the last few days, but otherwise seems to be all right. This morning she staggered somewhat when she first got up, but was soon able to walk straight.

MARCH 7, 1923.

Pig No. 199, lot 3, when roused this morning about 10 o'clock fell on her side, drew her hind feet up as close to the body as possible, stretched her front legs out in front of her, threw her head back and seemed to cease breathing. Every muscle in her body was tense. Then she relaxed slowly, got up and walked away with only a stiffness of the hind legs showing that anything was wrong.

Moved pig No. 105 from the west end of the nutrition barn to the east end. When brought out she traveled sideways seeming to be unable to make her hind end coordinate with the front end of her body. She is still very touchy and wild-eyed.

MARCH 12, 1923.

The condition of pig No. 105, lot 3, is not greatly changed. The partial paralysis in the hindquarters is not so pronounced but the unsteadiness in that region is more regular. Her eyeballs are protruding and the eyes are bloodshot, giving a horrible expression to the face.

MARCH 15, 1923.

Pig No. 199, lot 3, paralyzed in the hind quarters. She is able to move her hind legs but cannot get them under her to get up. She did not eat this morning. Her eye is dull, she is very listless and does not try to move very much.

Pig No. 120, lot 3, is able to walk about but staggers when she moves. She did not eat this morning. Her eye is dull. Her back is badly swayed when she stands still. She is somewhat restless. While under observation she fell on her side and kicked all four legs as fast as possible for about a minute. She seemed to have difficulty in breathing during this time. Presently she lay still and breathed heavily. When she tried to get up she fell and went through a similar thing again, except that it was not of such long duration this time. When she finally got her feet under her and was able to get up she had a hard time keeping her balance. She stood with legs wide apart trembling violently and breathing heavily. When she moved her hind legs were very stiff. This occurred several times during the day.

Similar notes were made each day during the seven years these trials were in progress, when there was anything of interest to record.



FIG. 7.—Sow No. 68, lot 2, the day after aborting nine dead pigs (fig. 8). She had been unable to stand for two weeks prior to the abortion and during the time showed frequent spasms as illustrated in this picture. She showed no mammary gland development or other indications of pregnancy.

THE EFFECT ON REPRODUCTION

Lots 1 and 2 were started at the same time, and the gilts therein did so well during the first months of the experiment that it was decided to determine their ability to reproduce. Five of the gilts, Nos. 13 and 37, lot 1, and 67, 80, and 68, lot 2, were bred. The other three gilts would not breed, although they seemed to be in heat most of the time, judging from the swollen condition of the vulva. None of the five gilts bred produced living pigs.

Gilt No. 37, lot 1, aborted six dead pigs 86 days after breeding. These pigs varied in weigh from 315 to 85 grams. The two smaller pigs were very dark in color and seemed to be partly resorbed as though they had been dead for some time before abortion.

Gilt No. 68, lot 2, aborted nine pigs 87 days after breeding. (Figs. 7 and 8.) These pigs seemed to be well formed but varied in weight from 340 to 62 grams. The smallest, pig as in those from gilt No. 37, was dried up and mostly resorbed with just the skin and bone remaining. Two other pigs showed decomposition of the hind quarters.

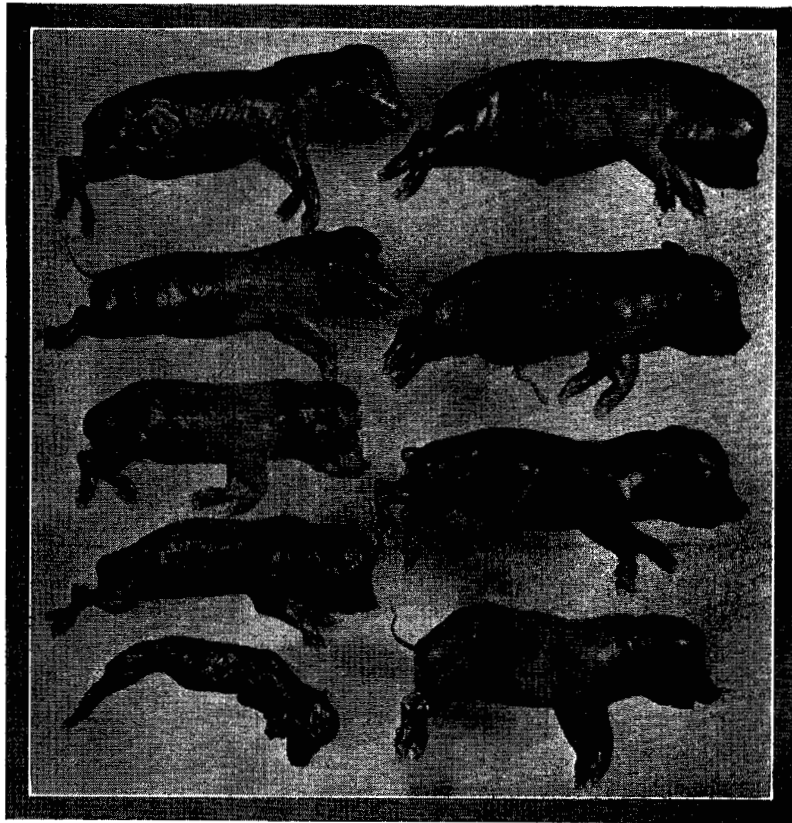


FIG. 8.—The nine dead pigs aborted by sow No. 68, lot 2, 27 days before she was due to farrow. No living pigs were farrowed by sows on the vitamin A deficient diet. In no case were organisms for contagious abortion found.

Gilt No. 67, lot 2, gave birth at normal time to 10 dead pigs. (Figs. 9 and 10.) This sow manifested none of the normal signs of approaching farrow, as milk in the udder, swelling of genitals, and sinking of hip muscles. She had farrowed four dead pigs by the time she was observed at the evening feed. She farrowed four more about

one hour apart. Shortly after 10:30 p. m. she voided what appeared to be the entire afterbirth. Consequently she was observed no more that night. While under observation the next morning at 8:30 she farrowed two more pigs. Her ten pigs were well formed and normal in size, but were all born dead.

Gilt No. 80, lot 2, died nine days before she was due to farrow. On post-mortem examination 10 apparently normal fetuses were found in the uterus.

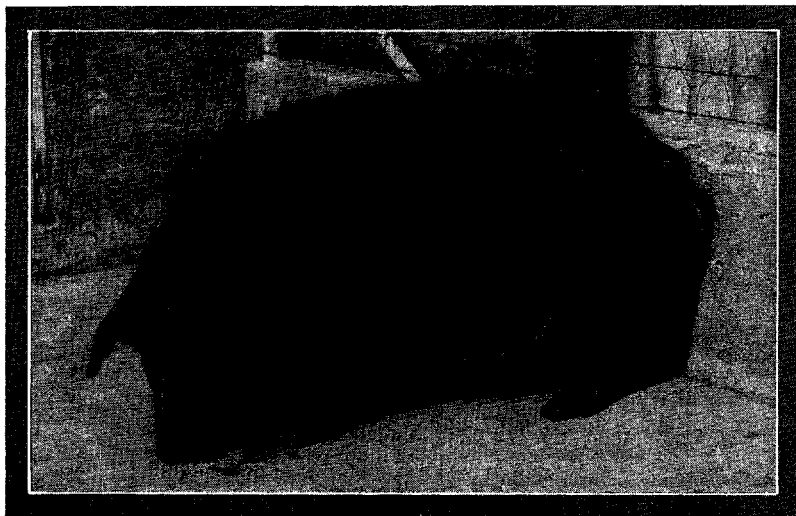


Fig. 9.—Sow No. 67, lot 2, the day following the farrowing of ten full-time dead pigs (fig. 10). She had been on the vitamin A deficiency diet for 396 days. Notice fine appearance of sow. She developed the incoordination characterizing avitaminosis A in pigs, 80 days after farrowing.

Gilt No. 13 (fig. 11), lot 1, was killed 81 days after she was due to farrow at which time six partly resorbed fetuses were found in the uterus. (Fig. 12.) It appeared that at some previous time they had been well developed. Although this sow showed heavy with pig, there was no evidence of development of the mammary glands or teats. The whole mechanism controlling birth was entirely upset seemingly by the lack of vitamin A in the diet.

In every case the dead pigs and the sows were tested for the organisms known to produce abortion in swine. All the tests were negative. It seems only reasonable to conclude that the death of the fetuses in the uterus was due to lack of vitamin A in the feed of the mothers.

In none of the lots of later tests fed a diet low in vitamin A was the question of reproduction studied. Most of the gilts receiving the ration were irregular in their heat periods. The periods were more frequent than normal and of longer duration. The vulva of some of the gilts in the later stages of the disorder remained swollen as

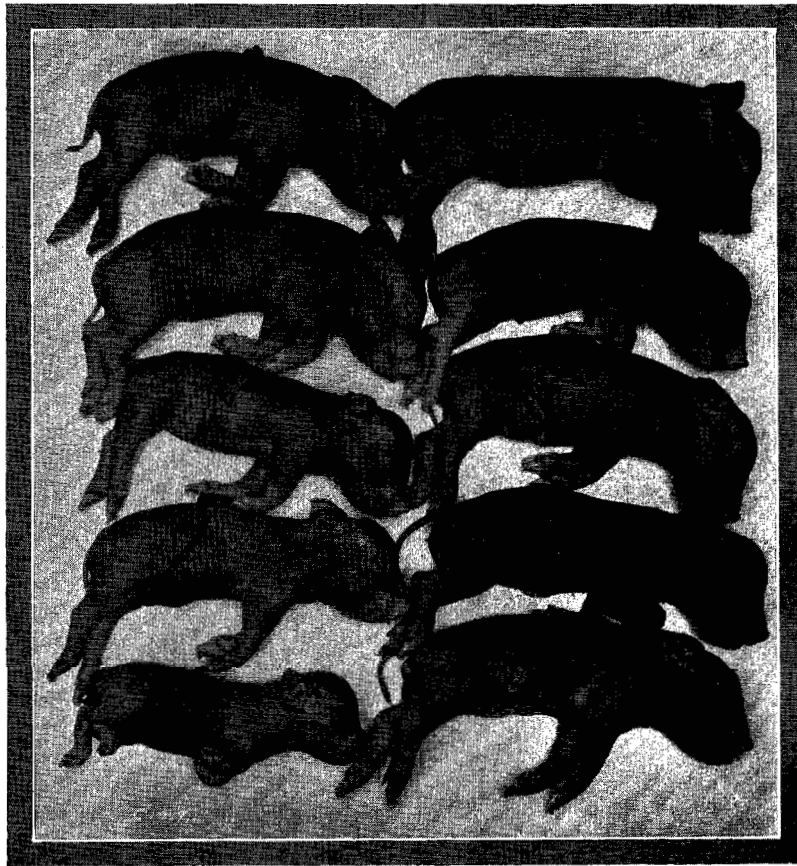


FIG. 10.—The ten full-time dead pigs farrowed by sow No. 67, lot 2 (fig. 9).
 The pigs appeared normal in development.

though in continuous œstrum. This was true particularly of gilts Nos. 4 and 22, lot 6. (Fig. 5.) The ovaries of these sows on examination showed the unusual condition of containing at the same time, ripe Graffian follicles, freshly ruptured follicles, and both new and old *corpora lutea*. (Fig. 13.) These findings are interesting in view of the results obtained by Evans and Bishop (2) in

their study of the relation of nutrition to fertility. They reported conditions as follows:

The disturbance of the œstrus from fat soluble vitamin A deficiency is highly characteristic, resembling no other nutritional upset known to us. It consists in the prolongation of the œstrus desquamative change in the vaginal epithelium, the smear consisting chiefly, if not exclusively, of cornified cells, which in normal animals characterize the actual period of estrus and ovulation only, but which, in the case of animals showing vitamin A deficiency, occur throughout the entire period of acute deficiency . . . In this respect the ill effects of vitamin A deprivation are entirely different from those resulting from the withdrawal of vitamin B, which has as its sequel complete cessation of ovarian function. Animals submitted to the degree of vitamin A deficiency giving the above test, however, continue to ovulate and to form *corpora lutea* irregularly.



FIG. 11.—Sow No. 13, lot 1, the day she was killed for post-mortem examination. She had received a vitamin A deficiency ration 356 days before showing the pronounced incoördination. This was three days after she was due to farrow. She was killed 81 days after her farrowing time, at which time six partly absorbed fetuses were found in the uterus (fig. 12). She appeared "piggy" but showed no development of the mammary glands or teats.

From the foregoing it would seem, therefore, that a further study of the effect of vitamin A on reproduction might afford additional valuable data.

CHEMICAL ANALYSIS

A complete feed analysis was made of each new lot of material used in mixing the feeds for these experiments. As the results of these analyses have no special interest in connection with the phase of the work being discussed in this bulletin, they are omitted.

The blood was analyzed a number of times for calcium and in-

organic phosphorus for indications of rickets. In no case was there a significant difference between the blood of normal pigs and those receiving the feed low in vitamin A. These findings together with the normal condition of the bones in all cases show quite clearly that



FIG. 12.—The six fetuses removed from sow No. 13, lot 1 (fig. 11), on post-mortem examination 81 days after she was due to farrow. These pigs indicated that at some previous time they had been well-developed, but were now in the advanced stages of resorption.

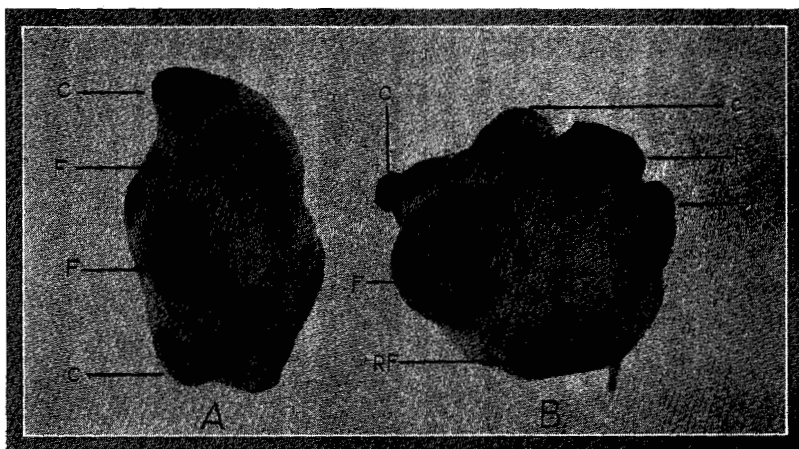


FIG. 13.—The ovaries of sow No. 22, lot 6. This sow appeared to be in heat continuously for six weeks previous to post-mortem examination. The ovaries were abnormal in that they contained at the same time recently ruptured follicles (RF), ripe follicles (F), and both new and old corpus lutea (C).

the condition under consideration was not complicated by rickets. This is in accord with the well-established fact that rickets does not develop when animals receive a diet containing a well-balanced mineral mixture and have access to direct sunshine as did the pigs in this experiment. Table II gives the results of one analysis.

TABLE II.—CHEMICAL ANALYSIS OF BLOOD FROM PIGS OF LOT 4 IN THE ADVANCED STAGES OF AVITAMINOSIS A AND FROM NORMAL PIGS.

Pig No.	Milligrams per 100cc. of plasma.				
	Inorganic phosphorus.	Calcium.	Uric acid.	Urea N.	Total nonprotein nitrogen.
No mark	5.0	9.6	1.1	10.5	14.0
39	4.6	9.8	1.2	10.5	13.2
40	4.9	11.0	1.5	7.4	14.3
2	6.2	10.7	1.2	8.5	17.6
50	5.7	9.5	1.5	8.2	16.3
Average	5.2	10.1	1.3	9.0	15.0

NORMAL PIGS.

1	6.3	10.7	1.2	12.4	18.7
3	5.1	9.8	1.2	20.0
26	4.6	9.0	1.2	8.3	16.2
30	4.9	12.5	1.0	13.0	26.7
Average	5.1	10.7	1.1	11.2	20.8

The blood was also analyzed for the more common nonprotein nitrogen compounds. The blood from the affected pigs did not differ appreciably from that of the normal ones. The results for one such determination are also found in table II.

The hemoglobin was found to be normal.

POST-MORTEM FINDINGS

No macroscopic lesions were found that could be considered characteristic of avitaminosis A. Of microscopic lesions, only those of the nervous system, found in several of the subjects, could be considered characteristic. The other lesions observed were attributed to secondary causes, such as infection brought on by a lowered resistance resulting from the lack of vitamin A. Of the lesions resulting, those involving the respiratory tract were the most common. The digestive tract was also frequently involved. In many cases where the pigs were killed for post-mortem examination before the

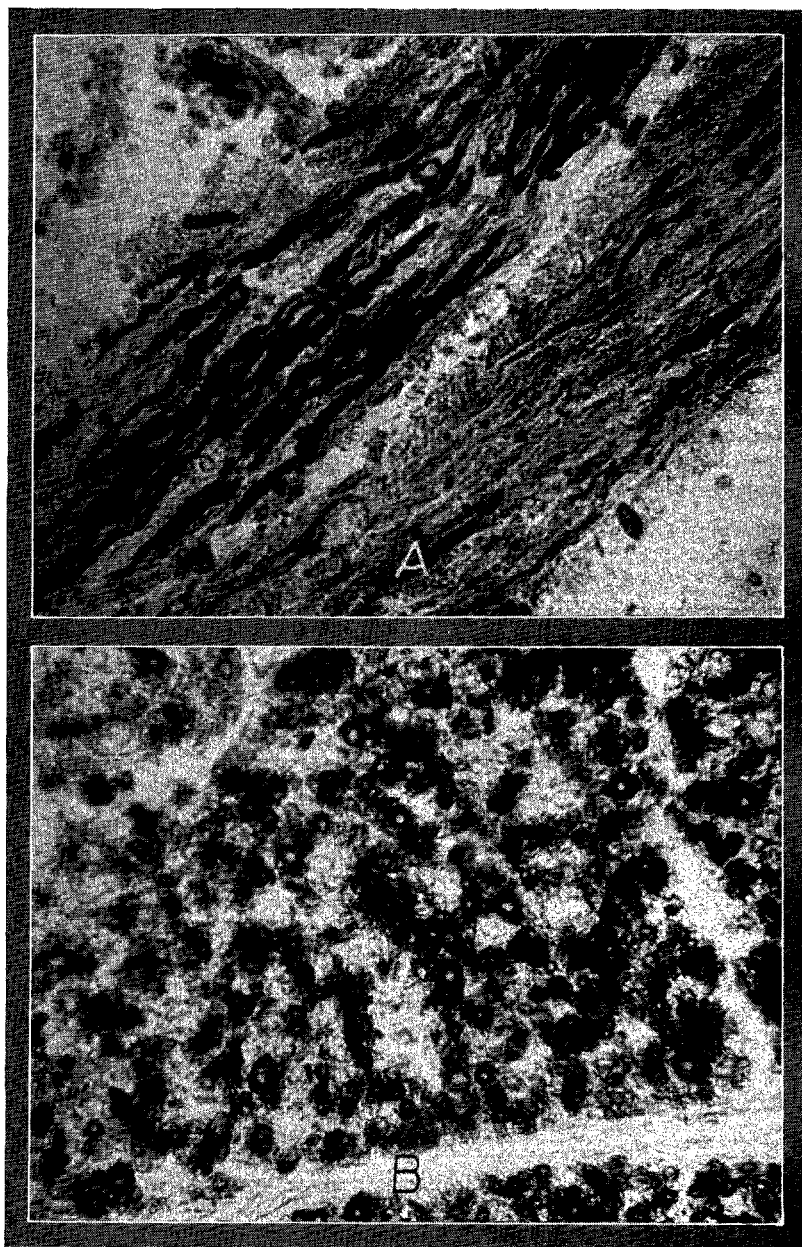


FIG. 14.—Sections of nerves from sow No. 16, lot 6, on a feed low in vitamin A. Magnification approximately 500 diameters. Stained by Marchi method. Blackened areas indicate degeneration. (A) Optic thalamus. (B) Optic nerve.

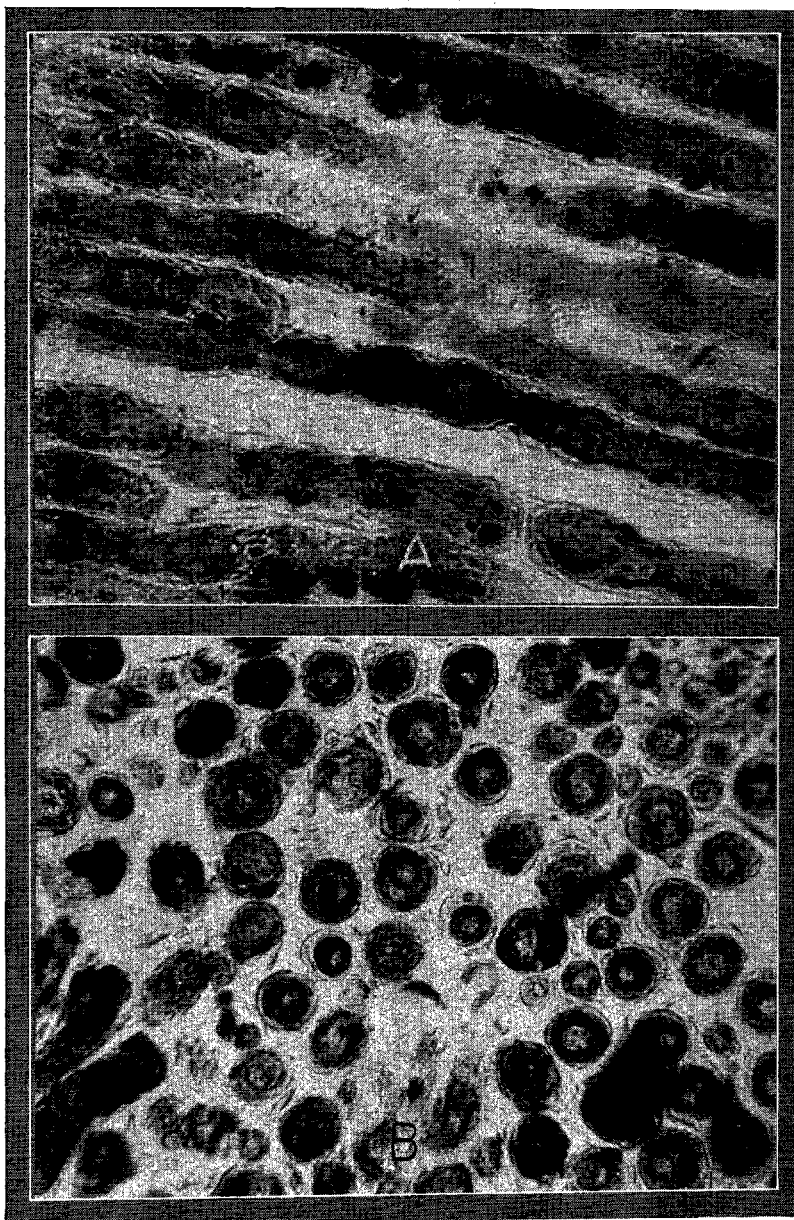


FIG. 15.—Sections of brachial plexus from sow No. 22, lot 6, on a feed low in vitamin A. Magnification approximately 500 diameters. Stained by Marchi method. Blackened areas indicate degeneration. (A) Longitudinal section. (B) Cross section.

secondary disturbances advanced far, no important lesions were found.

As the clinical symptoms were such as to indicate an impairment of the nervous system, histological examination was confined mainly to this tissue. Osmic acid was used as the stain. Tissues were fixed in Mueller's fluid which mordanted the myelin substance so as to prohibit the ability of myelin substance staining in the presence of

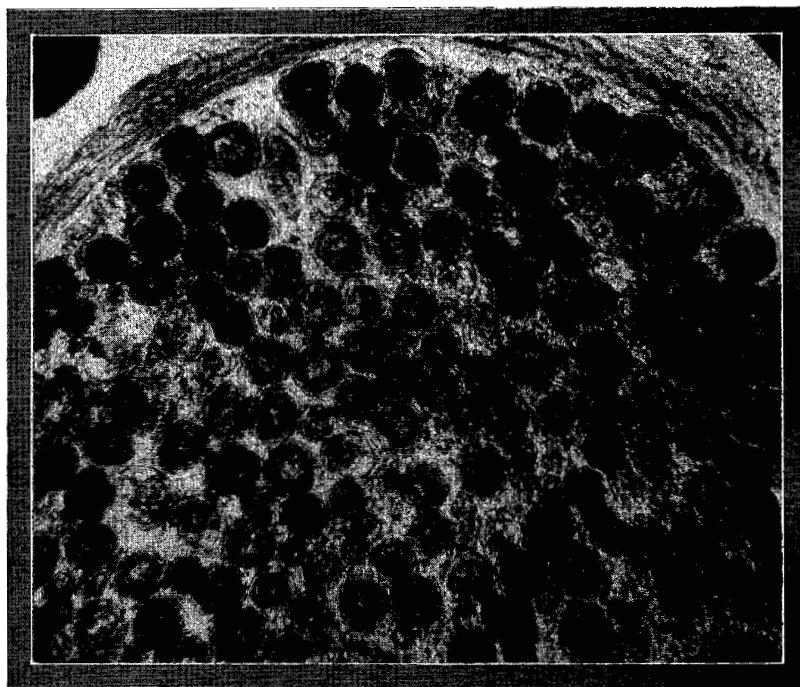


FIG. 16.—Cross section of sciatic nerve from sow No. 22, lot 6, showing blackened areas of degeneration. Stained by Marchi method.

osmic acid. Following the period of fixation (three weeks) the tissues were placed in equal parts of Mueller's fluid and 1 per cent aqueous solution of osmic acid for another period of from three to four weeks. Control nerves were examined by the same method and in no instance did they show suspicious staining characteristics. Osmic acid, the active principle in this staining process, stains fat black, while it has no effect on the myelin substance properly mordanted by the chrome salts in the fixative.

In a number of cases definite degeneration of the nerve bundles

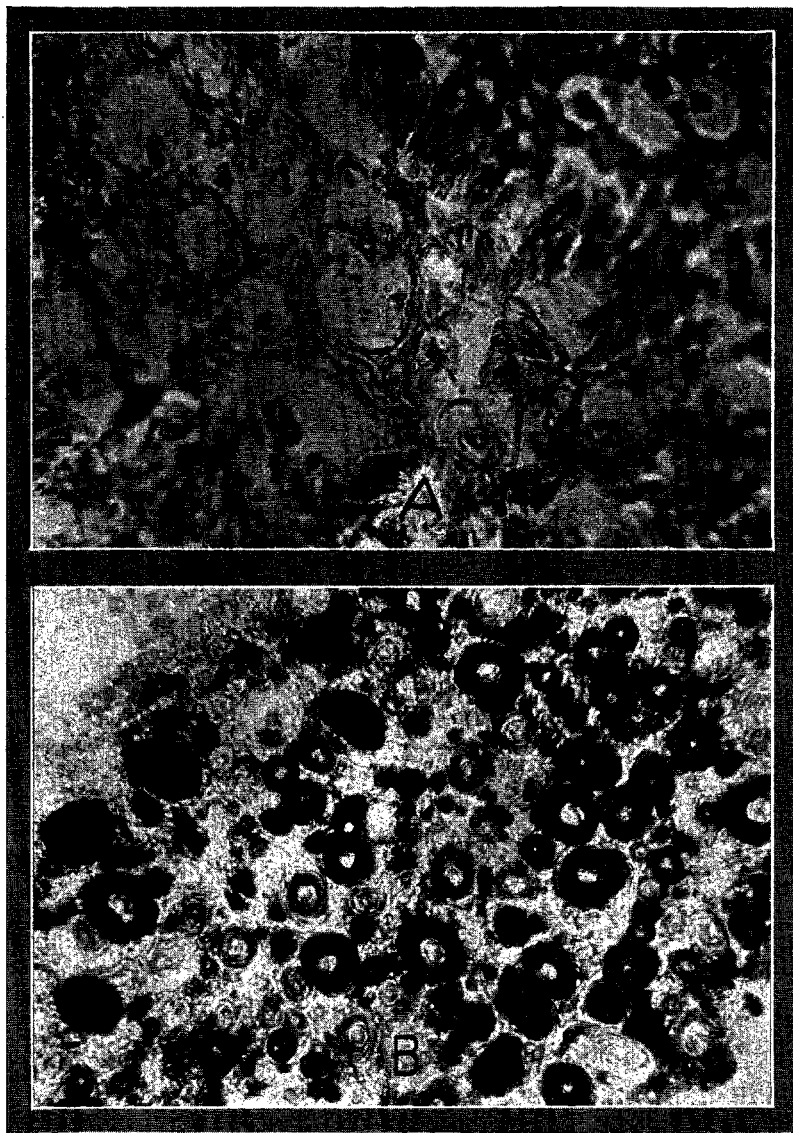


FIG. 17.—Cross sections of spinal cord magnification approximately 500 diameters. Stained by Marchi method. (A) From normal cord. (B) From sow No. 22, lot 6, on a feed low in vitamin A. Blackened area indicates degeneration.

was found. The degenerated bundles were found in the optic thalamus, optic, femoral, and sciatic nerves, and in certain areas of the spinal cord. Figs. 14, 15, 16, and 17 show the blackened areas indicative of fatty degeneration of the nervous tissue mentioned. Although the symptoms indicated cerebral impairment no lesions were found in this tissue. These nerve lesions, together with the clinical observations, furnish proof that the lack of vitamin A in pigs results in degeneration of nervous tissue. *This is the first case reported, in so far as the authors are aware, that lack of vitamin A, directly or indirectly, causes such a degeneration of the nervous system.*

Wolbach and Howe (15) in a systematic histological study of the lesions resulting from the lack of vitamin A in rats, found no lesions in the nervous system. Some simple chemical analyses of the brains of the affected pigs in which moisture, protein, ash, ether extract, organic and inorganic phosphorus were determined, revealed no significant variation from the results obtained from brains of normal pigs. No attempt was made to study the various lipins of the brains.

RESULTS OBTAINED WHEN THE BASAL DIET WAS SUPPLEMENTED WITH VITAMIN A

During the seven years these six trials were made with the basal ration low in its vitamin A content, other lots of pigs were carried along in which all the conditions were identical, with the exception that the basal ration was supplemented with vitamin A. Butter, alfalfa hay, sprouted oats, and cod-liver oil were used as sources of vitamin A. The first year, lots 7 and 8, which were conducted simultaneously with lots 1 and 2, received 5 per cent of commercial alfalfa meal as a vitamin A supplement. Lot 7 (fig. 18A) received the same basal ration as lot 1 which had as its basis white corn, and lot 8 (fig. 19) the same as lot 2 which had kafir as its basis.

The addition of 5 per cent commercial alfalfa meal, although it greatly improved the ration, left it still inadequate. This is shown by the fact that one sow (No. 66) in lot 8 and two sows (Nos. 64 and 2) in lot 7 finally developed symptoms of a nervous breakdown similar to the symptoms shown by the gilts which received the basal ration only. The onset of the disorder was much delayed, averaging 507 days for the sows receiving the 5 per cent alfalfa as against an average of 219 days for the ones receiving the basal ration.

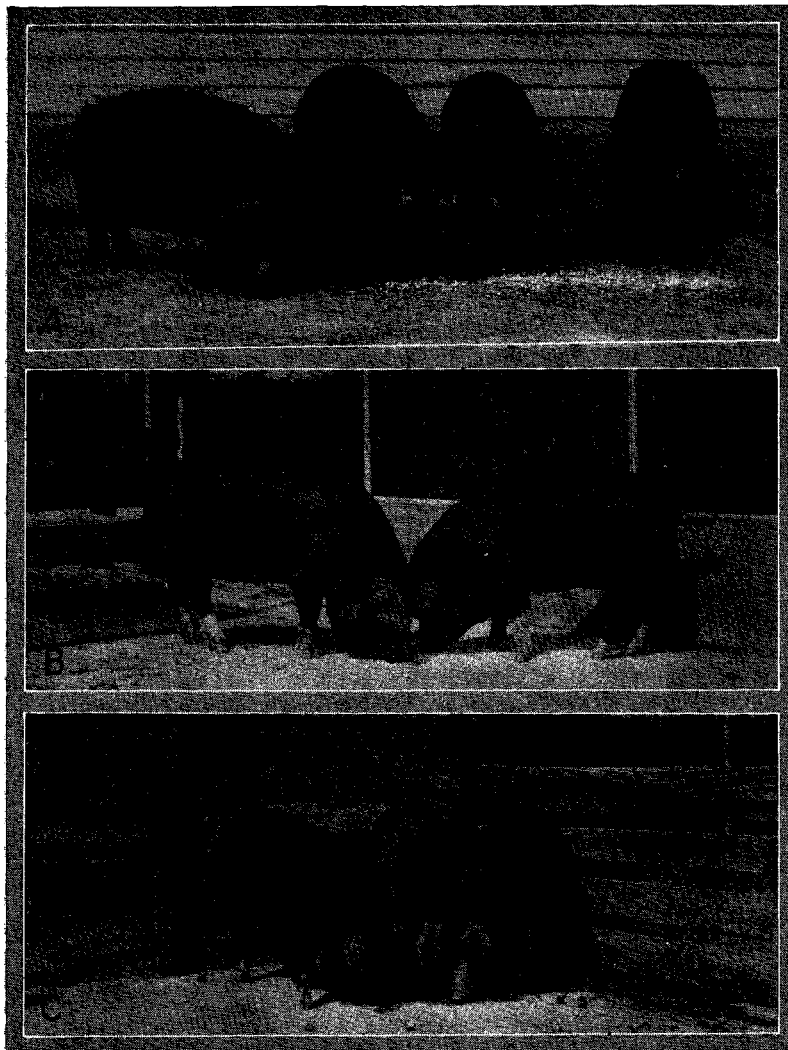


FIG. 18.—Pigs receiving the basal ration, white corn, tankage, and bone ash supplemented with vitamin A supplements. Compare the condition of these hogs shown in figures 4, 5, and 7. (A) The pigs in lot 7, one year after being on a diet in which 5 per cent commercial alfalfa meal furnished the source of vitamin A. This 5 per cent alfalfa meal did not furnish complete protection as one sow developed the nerve disorder 65 days later. (B) The pigs in lot 9, receiving 5 per cent butter as a source of vitamin A. (C) The pigs in lot 11, receiving prime alfalfa meal as a source of vitamin A.

Sow No. 66, lot 7, developed a bad case of prolapse of the rectum shortly after the nervous symptoms developed. When this condition became more pronounced a surgical operation was performed. She recovered from the operation but her incoördination grew steadily worse. She died two weeks later.

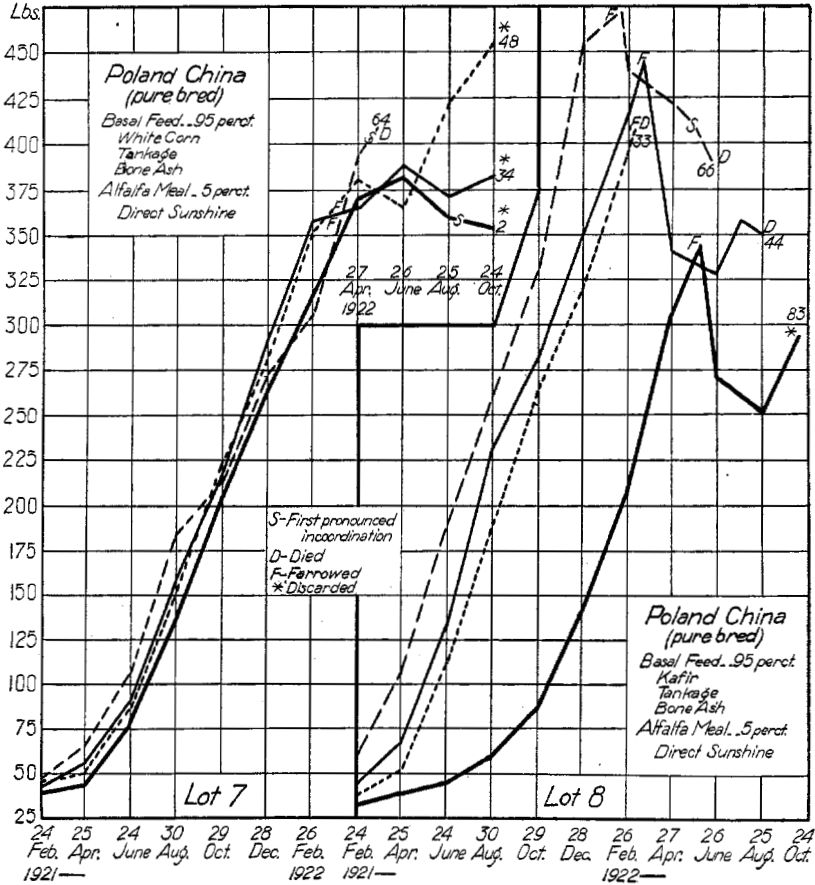


Fig. 19.—Graph showing rate of growth of lots 7 and 8, which received 5 per cent commercial alfalfa meal as a source of vitamin A. This amount of alfalfa did not furnish complete protection as sows Nos. 64 and 2 in lot 7 and No. 66 in lot 8 developed incoördination on an average of 507 days.

Sow No. 2, lot 7, developed incoördination 24 days before the experiment was discontinued. In an attempt to cure the disorder she was given cod-liver oil and sprouted oats to supply vitamins A and C. At this time it was not known definitely that hogs did not need vitamin C. When the cod-liver oil and sprouted oats were

added to the diet she was in a very bad condition, was unable to stand, and most of the time seemed quite lifeless. An improvement was evident about 10 days after vitamins A and C had been added. She was bright, had a normal appetite, and could get up on her fore feet enough to drag herself around. She was kept on this diet for about three months but never was able to control the movement of her hind legs so she could stand without being helped. She was strong enough but lacked control of the muscles of the hind legs. The failure of this sow to recover completely when given vitamin A is what might be expected considering the advanced stage to which the disorder had developed before vitamin A was given.

In lot 8 three of the sows died before the experiment ended. Only one of these showed incoördination. Sow No. 44 died as a result of sunstroke. The door to her inside pen was accidentally closed when she was in the outside pen where she was exposed to the sun. When she was found at noon her condition was critical and she died shortly after being moved into the shade.

Sow No. 33 was found the next morning after farrowing, with a prolapsed uterus. The entire organ was protruding and it was necessary to operate. She died a few hours later. No doubt the inadequate diet was the indirect cause of this condition.

Sow No. 64 developed the typical incoördination after she had been on feed 494 days. She died 40 days after the first sign of nervousness and two days after she was due to farrow.

The reproductive record of the sows receiving the alfalfa meal was an improvement over the sows not receiving this vitamin A supplement. It was, however, far from satisfactory. Six of the eight sows farrowed 46 living and seven dead pigs; one died two days after she was due to farrow and one did not reproduce.

While this experiment was in progress the question arose as to why the addition of 5 per cent alfalfa meal had not rendered the ration adequate. Whether it was due to an insufficient supply of vitamin A in the 5 per cent alfalfa or due to a lack of vitamin C could not be determined from the results. The nervous condition was unlike anything encountered in the parallel experiments with rats on a vitamin A free diet, or anything reported in the work of others. It resembled to some degree the stiffness and soreness observed in guinea pigs in advanced stages of scurvy, but these gilts showed no lesions resembling scurvy.

More definite information in regard to the relation of vitamins A and C to this disorder was thought desirable, consequently three new

experimental lots were started. These experiments were conducted at the same time as lot 3 on the basal ration. One lot, lot 9 (fig. 18B and fig. 20), received 5 per cent butter as a source of vitamin A in addition to the basal diet of white corn, tankage, and bone ash. This

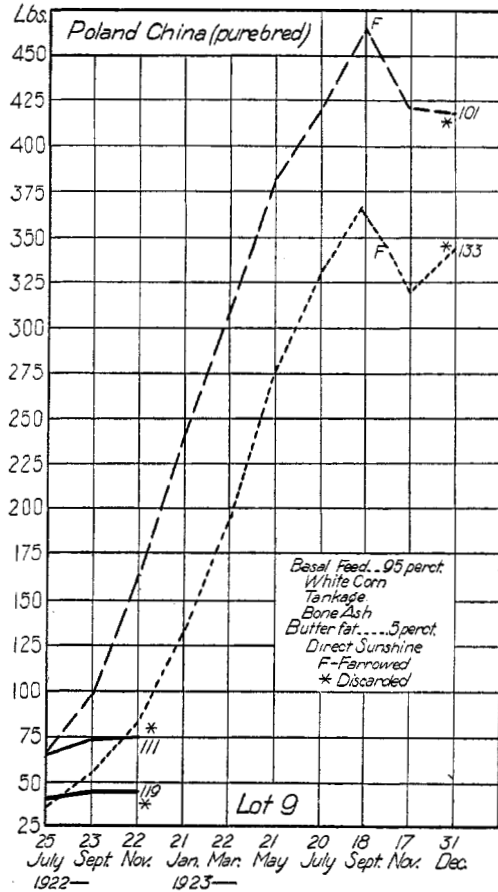


FIG. 20.—Graphs showing the rate of growth and farrowing record of the sows in lot 9 receiving 5 per cent butter. Pigs Nos. 111 and 119 became unthrifty shortly after being placed on experiment and were discarded. post-mortem examination showed necrotic enteritis.

addition of butter would supply vitamin A without adding any vitamin C. A second lot, lot 10 (fig. 23, first generation), received sprouted oats in addition to the basal ration. The sprouted oats would supply both vitamins A and C.

The oats were sprouted by soaking one pound of the grain in water until they began to germinate. The grain was then placed in a flat tin pan with perforated bottom. The pans were 12 inches in diameter. When the sprouts were two to three inches long they were fed to the pigs. One panful a day was given to four pigs, each pig getting one-fourth of a panful in its individual feeding pen at the time the basal ration was fed.

A third lot, lot 11 (fig. 18C and fig. 24, first generation), received 10 per cent home-made alfalfa meal. The commercial meal used in the previous experiment was of unknown origin. In the preparation of the meal for this experiment, the alfalfa was cut in the bud stage so there would be a high proportion of leaves to stems. It was collected before it was dry enough for the leaves to shake off and placed for curing in a thin layer on the barn floor. When it was thoroughly dry it was ground into a fine meal. Ten pounds of this meal was mixed with 90 pounds of the basal ration which had previously been mixed.

In order to secure more proof that the lack of vitamin A was causing this disorder, three of the pigs in lot 3, Nos. 128, 199, and 110, were given cod-liver oil as a corrective measure after they had developed nervous disorders. Two of these, Nos. 128 and 110 (fig. 21) made a complete recovery. Number 199 showed marked improvement but always showed some incoördination in her movements.

Two pigs in lot 9, Nos. 111, and 119, and one in lot 11, No. 113, did not do well after they were placed on the experiment. They did not grow and they were unthrifty. It was evident this did not result from any inadequacy of the diet, for its onset was too immediate. The pigs were removed from the experiment and found to be suffering from necrotic enteritis.

The remaining two pigs in lot 9 (figs 18B and 20), receiving butter as a vitamin A supplement, developed normally and farrowed seven live and eight dead pigs, all well developed. The pigs farrowed alive were all dead at the end of 48 hours. The remaining three pigs in lot 11 (figs. 18C and 22), which received 10 per cent alfalfa meal, farrowed normal pigs but the mortality was high.

Three of the pigs in lot 10 receiving the sprouted oats developed exactly the same symptoms—cross disposition, staggering gait, incoördination, and spasms—as the pigs receiving the basal ration. The beginning of the disorder was, however, somewhat delayed.

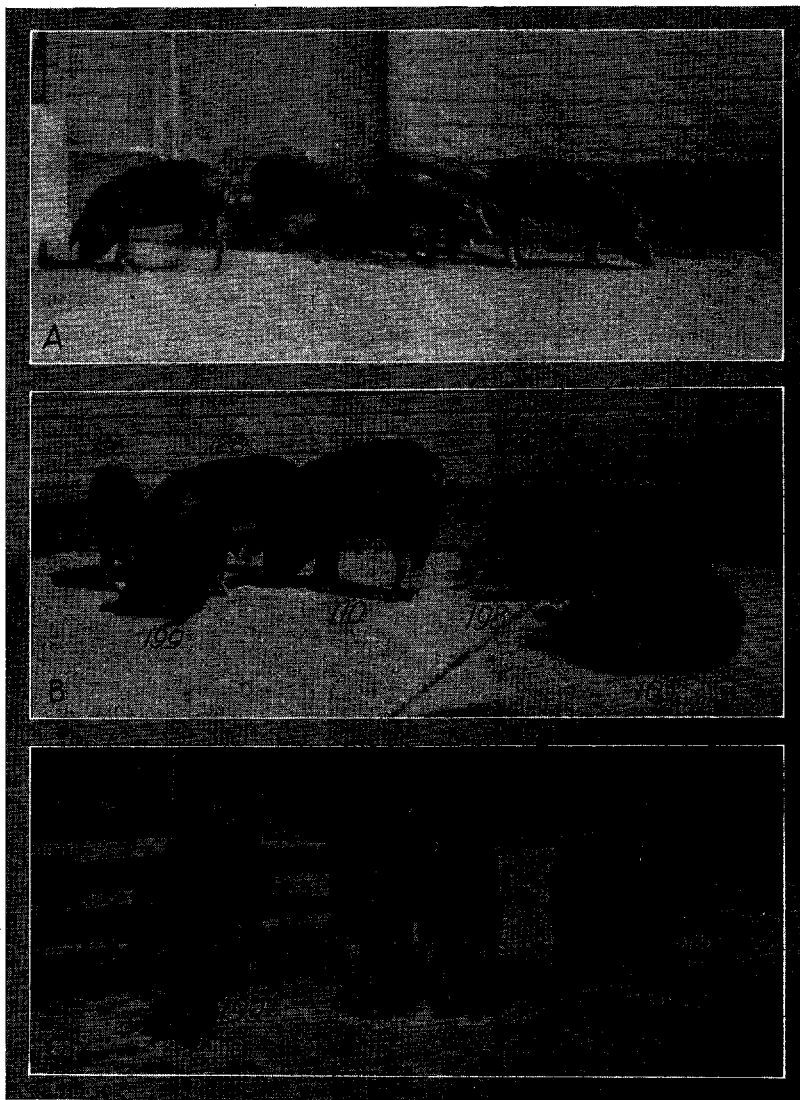


FIG. 21.—Pictures showing the progress of the disorder in lot 3, which were started on the basal ration of white corn, tankage, and bone ash. (A) The pigs at the beginning of the experiment. (B) The pigs 265 days later. Nos. 128, 199, and 110 had developed advanced stages of nerve disorder and had been given cod-liver oil as a curative agent. Nos. 128 and 110 had apparently entirely recovered; No. 199 had not as yet recovered the use of her hind legs; Nos. 198 and 105 were killed for post-mortem examination; No. 126 developed the disorder later. (C) The pigs 100 days later or one year after the experiment began. At this time sow No. 199 was able to stand but still showed marked incoördination. No. 126 did not develop the disorder because of the vitamin A received from the feces of the pigs receiving cod-liver oil. She developed the disorder shortly after her removal from the lot.

The average number of days before distinct incoördination developed in the case of the three pigs receiving the sprouted oats was 330 days, while in the case of the 28 pigs receiving the basal rations alone in the six trials, it was 219.

The delay in the appearance of the disorder was no doubt due to the vitamin A furnished by the sprouted oats and not to the vitamin C, as pigs do not need vitamin C. It was not known at that time that sprouting grain had to be kept under conditions so as to develop a good green color in order to be a good source of



FIG. 22.—The normal litter of pigs produced by the sow in lot 11, which received 10 per cent prime alfalfa meal as a source of vitamin A. None of the sows receiving diet deficient in vitamin A farrowed living pigs. See figures 7, 8, 9, 10, 11, and 12.

vitamin A. The oats used in this experiment were grown in a well-lighted room so that the sprouts looked green, but the green color was confined to their tips. Beneath their surface, owing to the crowded condition of the sprouts shutting out the light, they were white. For this reason the sprouted oats contained little vitamin A. As the development of vitamin C in sprouting grain does not depend on the action of light, it was a source of vitamin C.

One of the pigs in lot 10 receiving sprouted oats, No. 116, was killed and examined. She had developed the extreme stages of incoördination, yet no significant macroscopic lesions were observed. The other two which developed the nervous symptoms were given cod-liver oil as a corrective agent. Sow No. 99 responded to the addition of vitamin A and in two weeks time regained an apparently normal condition with complete coördination. The condition of

No. 96 was greatly improved, but she did not gain control of her hind legs. Her recovery was much like No. 2, lot 7.

The reproductive performance of No. 96 was of unusual interest. On May 24 before she had developed signs of incoördination she was bred. Sixteen days later, June 9, she developed such a degree of incoordination that she could scarcely get up. She could stagger along but could not control her steps. On the same day she appeared to be in heat. Four days later she recovered sufficiently so that she could get on her feet but walked with a very unsteady gait. The heat period continued so she was rebred on June 13. Her condition did not change much until July 7 when she was unable to get up without help. Two days later she was having the spasms and trembling spells. At this time one ounce of cod-liver oil was added to her feed each day. But July 13 the spasms and trembling condition had stopped and her appetite was better. A month later, August 14, she was lively and could move her front legs, but could not get up on her hind legs nor stand without help. She remained in this condition until October 10, at which time she farrowed nine pigs, seven living and two dead. One of the farrowed dead pigs was badly decomposed, apparently dead some time. All the pigs were normally developed in every way excepting their eyes. In no case were the eyes normal. The lids were malformed and were stuck tight to the eyeball so that it was difficult to separate them. When the lids were pulled back the eyeball itself was slightly opaque. None of the pigs could see. Although these pigs were quite lively at first, they were all dead at the end of 43 hours. This is a case in which eye lesions developed in fetuses apparently because of the insufficient amount of vitamin A received by the mother during the first part of the gestation period, although she received an abundance of this vitamin during the last part of her pregnancy. This sow never regained the use of her hind legs and in January abscesses formed there, apparently the result of bruises from the cement floor. The infection spread throughout her body and a post-mortem examination, February 10, showed large abscesses in the lungs and all of the evidences of pyemia.

The remaining sow, No. 132, in the lot receiving the sprouted oats, developed no nervous symptoms but died about 40 days after she farrowed her second litter of pigs. Death was due to a ruptured heart, the cause of which was not determined.

VITAMIN C NOT ESSENTIAL IN FEED FOR SWINE

During the first three years of this experiment seven lots of pigs, lots 1, 2, 3, 7, 8, 9, and 11, were kept on feeds containing very little, if any, vitamin C without any of the pigs showing the characteristic symptoms of scurvy. Although this indicated that pigs do not need vitamin C, it did not furnish conclusive proof, as the pigs in all the lots had been kept under normal conditions until weaning time, when they were placed on the experiment. Their mothers received vitamin C during the gestation and lactation period.

In order to secure definite information in regard to the vitamin C requirement of pigs, it was decided to continue the pigs farrowed by the sows in lot 11 until they themselves had farrowed and weaned pigs. These sows received no vitamin C excepting a slight trace in alfalfa meal. If no scurvy developed during the three generations on this vitamin C free diet, it would furnish positive proof that pigs do not require vitamin C for development and reproduction.

As a check lot the pigs farrowed by the same sows in lot 10, which had recovered from nervous disorder by the use of cod-liver oil as a source of vitamin A, were continued to the third generation. These pigs continued to receive sprouted oats as a source of vitamin C. Cod-liver oil was continued in this lot as a source of vitamin A and in order that each of these lots might be kept as nearly alike as possible the pigs from lot 11 which were continued on the experiment were given a like amount of cod-liver oil. This, of course, added no vitamin C to their feed.

Four gilts were continued in each of two lots, which were designated as lot 10, second generation (fig. 23), and lot 11, second generation (fig. 24). These lots were continued until the second generation pigs were three years and five months old. No apparent abnormalities were shown by any of the eight sows during the entire experiment.

Three of the sows in each lot farrowed in the spring of 1926 and each of the four sows in each lot farrowed during the spring and early summer of 1927. During the first farrowing period the three sows receiving no vitamin C, lot 11, second generation, farrowed 23 living and four dead pigs. The three in the lot receiving vitamin C, lot 10, second generation, farrowed 23 living and three dead pigs. During the second farrowing period the four sows receiving no vitamin C (fig. 25) farrowed 31 living and no dead pigs, while the four sows receiving vitamin A (fig. 26) farrowed 32 living and two dead pigs.

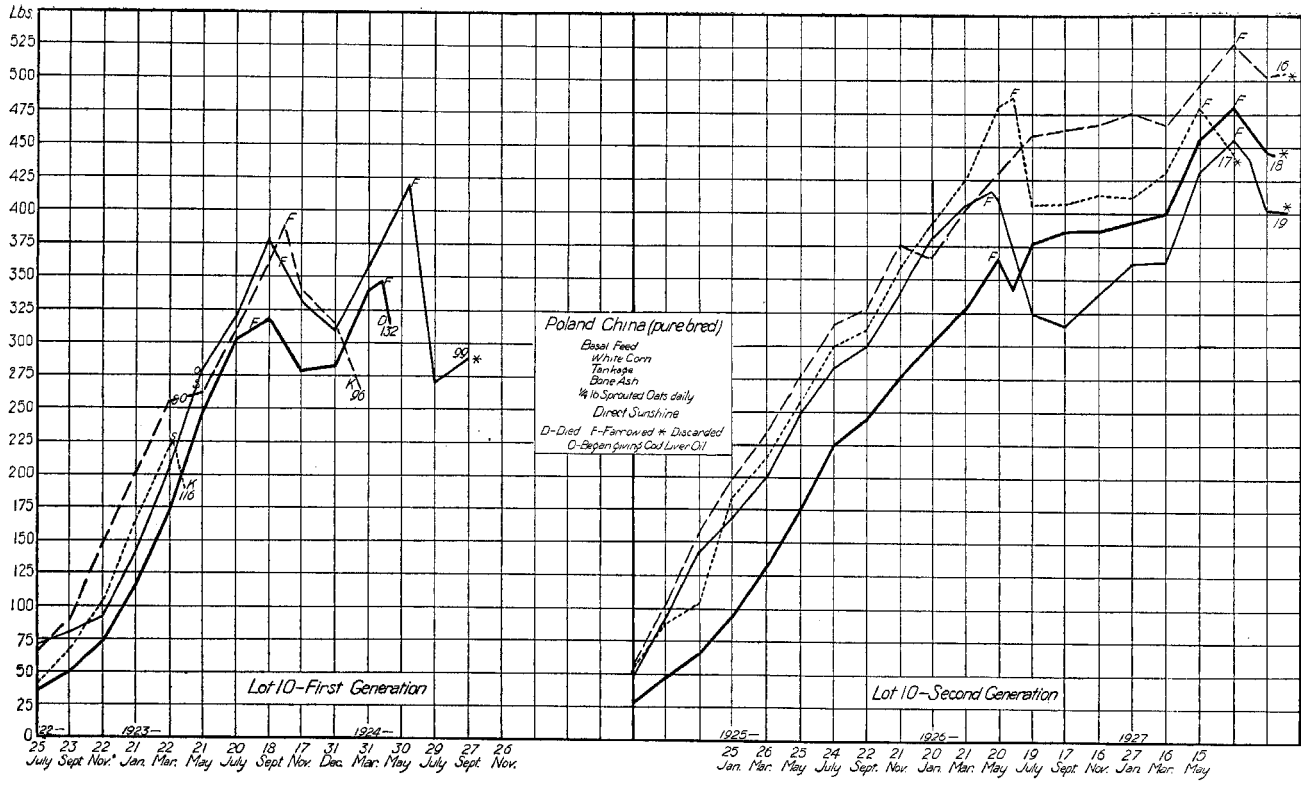


Fig. 23.—Graphs showing rate of growth and reproduction record of two generations of swine on a feed containing sprouted oats as a source of vitamin C. Compare with figure 24.

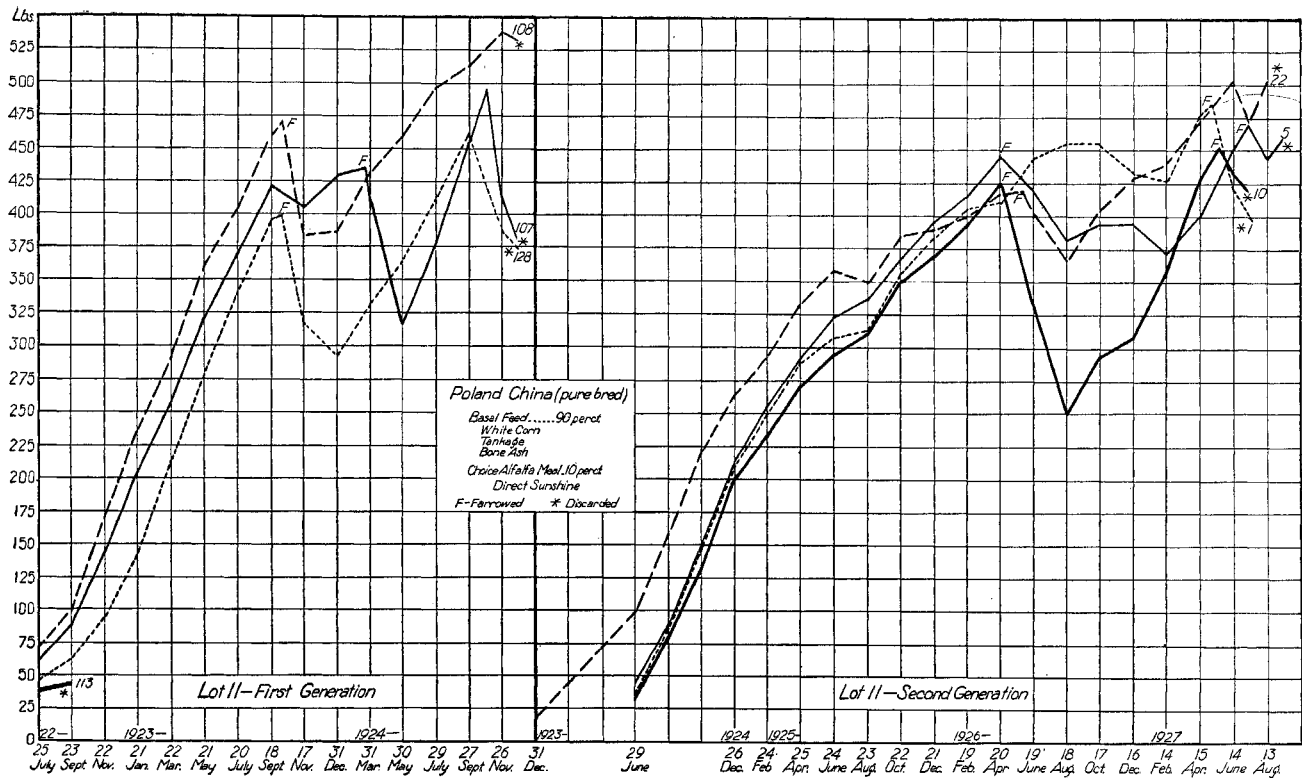


FIG. 24.—Graphs showing rate of growth and reproduction record of two generations of swine on a feed deficient in vitamin C. No symptoms of scurvy developed during the five years these hogs were on experiment, which indicates that the normal growth of hogs is not interfered with by a lack of vitamin C.

Of the 31 live pigs farrowed by the sows receiving no vitamin C, 21 lived until weaning time; of the 32 live pigs farrowed by sows receiving vitamin C, 18 lived until weaning time. The results are presented in table III.

TABLE III.—DATA ON THE REPRODUCTION OF SOWS RECEIVING VITAMIN C IN THE DIET IN COMPARISON TO THOSE RECEIVING NO VITAMIN C.

	Number of sows.	Number of pigs farrowed.		Weaned.
		Living.	Dead.	
First Farrowing:				
Sows receiving no vitamin C.....	3	23	4	
Sows receiving vitamin C.....	3	23	3	
Second Farrowing:				
Sows receiving no vitamin C.....	4	31	0	21
Sows receiving vitamin C.....	4	32	2	18

From the results of this test it is seen that the absence of vitamin C in no way affected the growth or reproduction of these hogs. Lot 11, receiving the basal ration—white corn, tankage, bone ash, plus 10 per cent alfalfa meal—was started with four gilts which had been weaned July 25, 1922. The daughters of these sows with their second litters of pigs were discarded in August, 1927. During a period of a little over five years these three generations were kept on a feed practically free from vitamin C. The growth and behavior in every way equaled those of the group receiving vitamin C.

Throughout all these experiments on vitamins A and C the mortality of the young pigs during the first days of life was very high. An experiment is now under way which has for its purpose a study of the relation of various food factors, other than vitamin A, to this high infant mortality in pigs.

DISCUSSION AND SUMMARY

During the seven years prior to the beginning of these experiments in 1921, the senior author had conducted numerous vitamin experiments with pigeons, rats, and guinea pigs and so was familiar with the symptoms resulting from the lack of vitamins in these animals. There was nothing in his experience or in the numerous experiments reported in the literature up to that time that suggested pigs would develop such striking nervous disorders as were observed as a result of the lack of vitamin A. So when these symptoms were first observed in lot 1 and lot 2 it was thought that some factor other

than the inadequacy of the diet was the cause. When members of the Department of Veterinary Medicine examined the pigs showing the nervous disorder, they called attention to the fact that many cases of a seemingly similar disorder were occurring on the farms throughout the state. At that time there were a number of patients at the veterinary hospital showing the same symptoms which were being studied to determine the cause of the disorder. The veterinarians thought at first that some bacterial infection was responsible. However, no progress was made in transmitting the disease to normal pigs or in isolating any organisms. A histological examination from diseased pigs revealed a degeneration of certain nerve fibers.

A careful comparison of the symptoms of the pigs in the nutrition experiment, and those sent in by farmers for the purpose of studying the cause of this disorder, led to the belief that the two lots of pigs were suffering from the same disorder. The condition at that time was called posterior paralysis. From that time on, the departments of Chemistry, Animal Husbandry, and Veterinary Medicine, coöperated in the study. The results obtained in treating the patients from the farm herds and those developed in the foregoing experiment showed clearly that this particular nervous disorder was due to a lack of vitamin A.

There are many conditions that can produce nervous symptoms. The lack of vitamin B, particularly the vitamin F fraction (12), causes nervous disorders. Certain bacterial toxins as well as many drugs produce nervous symptoms of one kind or another. Animals suffering from rickets usually manifest more or less nervous symptoms. In the latter there is always a faulty calcification of the bones. Bohstedt and coworkers (1) found in a number of their experimental pigs bones so weakened that the vertebræ slipped out of place and pinched the spinal cord. This resulted in posterior paralysis. As before mentioned the authors are convinced these workers were dealing with two deficiency diseases, avitaminosis A and rickets, each of which is characterized by nervous symptoms, and, judging from results reported in the present work, it appears their experiments were not continued a sufficient length of time for the typical symptoms of avitaminosis A to develop. Even when the pigs were started on the experiment at weaning time it was found that it required an average of 219 days for the first distinct sign of incoördination.

A comparison of the symptoms of avitaminosis A in the pigs with the symptoms manifested by the rat and other experimental animals is interesting. The most characteristic symptom of avita-

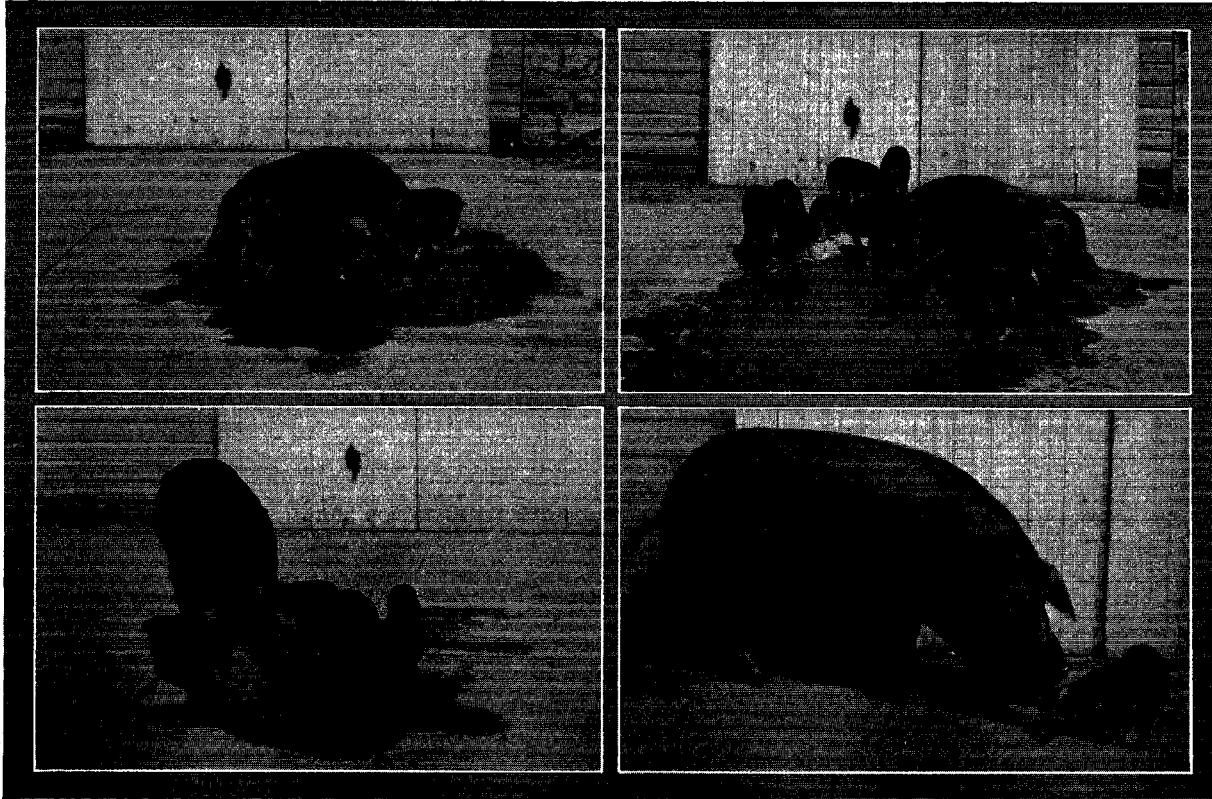


FIG. 25.—Second generation sows and third generation pigs kept on a feed deficient in vitamin C. The first generation was placed on experiment five years previously. Observation on the normal growth and reproduction of three generations of hogs on a ration deficient in vitamin C over a period of five years, shows that hogs do not need vitamin C in the ration.

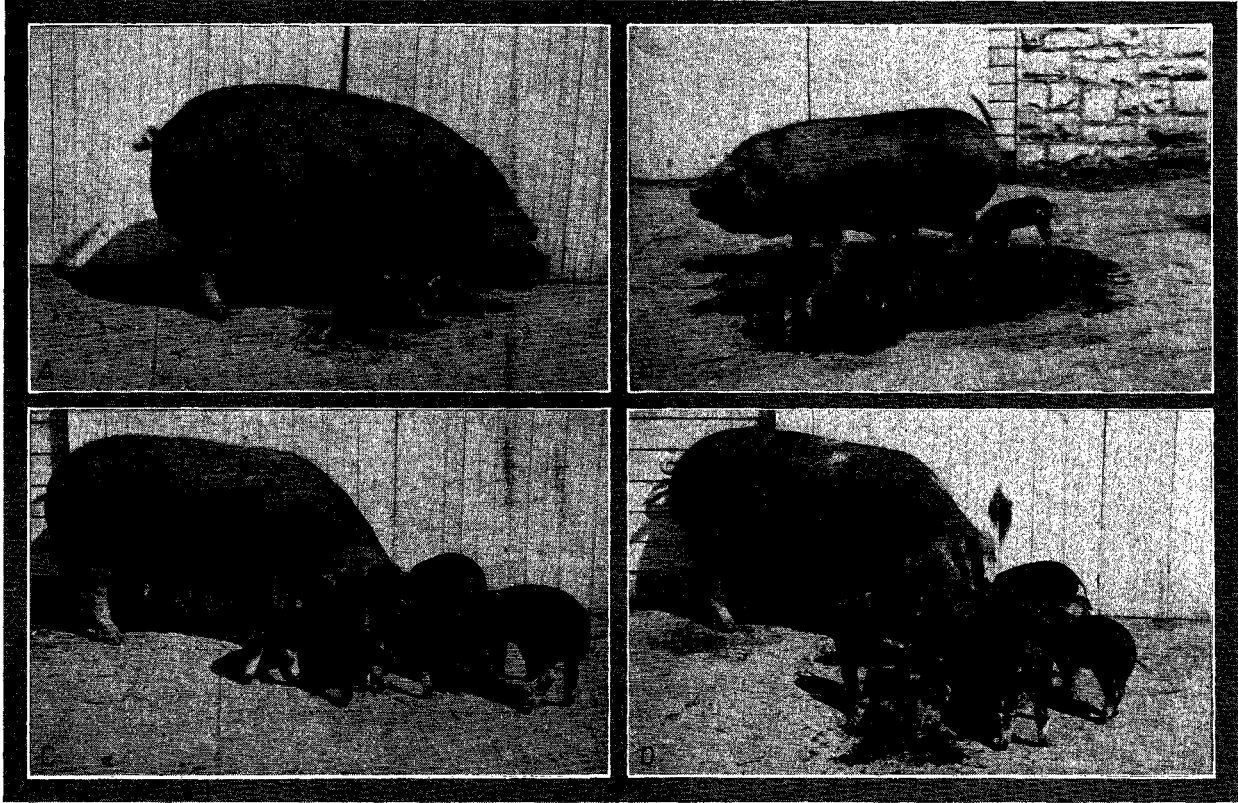


FIG. 26.—Second generation sows and third generation pigs kept on a ration containing vitamin C. The first generation was placed on experiment five years previously. The hogs receiving vitamin C grew and reproduced no better than those without vitamin C.

minosis A in the rat is ophthalmia. This is so constant that many workers use it as a criterion by which to determine whether or not a rat, is suffering from avitaminosis A. In fact ophthalmia and avitaminosis A are used synonymously. The nervous symptoms in rats are so slight that they are usually overlooked. McCollum and Simmonds (4) in fact do not mention the nerves as being involved in this disorder. Rats in the last stages of the disease do, however, show a decided unsteadiness of gait which does not seem to be caused by muscular weakness, but to some impairment of the nervous system.

Avitaminosis A in pigs is characterized primarily by nervous symptoms, while eye lesions play a minor part. Only one small corneal ulcer was observed in the 32 pigs which in these experiments developed advanced stages of avitaminosis A. The difference in the symptoms in this disorder manifested by rats and pigs are quantitative, not qualitative, differences. They both show nerve disorders and eye lesions. In the rat the eye lesions characterize the disorder, in the pigs the nervous symptoms constitute the outstanding characteristic.

Chickens seem to have a place midway between rats and pigs so far as the relative severity of these two symptoms is concerned. Under ordinary laboratory conditions mature chickens when kept on a feed otherwise adequate but lacking in vitamin A will develop characteristic eye lesions accompanied by bacterial infection. This is commonly called roup. In a very small per cent of cases where the onset of the eye lesion is delayed, nervous symptoms are manifested. Day-old chicks started on a vitamin A free feed and kept in a carefully sterilized pen develop few eye lesions.⁴ Under these conditions striking nervous symptoms appear which are similar to those developed by pigs—the same unsteady gait, incoördination, trembling of the legs, and spasms.

Nelson, Lamb, and Heller (8) found that rabbits, like chickens, show both the symptoms of nerve degeneration and marked eye lesions in the advanced stages of avitaminosis A.

This general occurrence of nervous symptoms resulting from avitaminosis A in experimental animals suggests that in some cases the lack of vitamin A may be a contributing cause to nervous disorders in people.⁵

4. Unpublished data, Kansas Agricultural Experiment Station.

5. A moving picture showing these striking nervous symptoms in pigs resulting from the lack of vitamin A was shown accompanying a lecture given by the senior author before the Sigma Xi Society at the Mayo Foundation, Rochester, Minn. Nerve specialists, following the viewing of this picture, suggested a possible explanation in the experiment of baffling cases of certain human nerve disorders which had been observed in the clinic.

These experiments have not only furnished valuable information from a purely scientific point of view in regard to the relation of avitaminosis A to certain degeneration of the nerves, but they have also furnished information of great practical importance to the farmer. They have solved the problem of a certain type of nervous disease, posterior paralysis, which is of considerable economic importance throughout the section of the country where but little yellow corn is raised.

The experiments have also focused the attention of the swine grower on the importance of including in the ration of hogs some source of vitamin A when white corn or the sorghums make up the grain portion of the feed. Alfalfa hay is a good practical source of vitamin A. No attempt has been made to determine the optimum amount needed. Ten per cent of good alfalfa meal gave complete protection. The quantitative requirement of hogs for vitamin A for growth and reproduction will be studied as soon as the factors causing the high farrowing loss of vitamin A fed pigs is determined.

The experiments show that hogs do not need vitamin C in their diet for growth and reproduction. No attempt was made to determine whether or not the hogs synthesized vitamin C as was found by Parsons (10) to be the case with rats.

CONCLUSIONS

1. The lack of vitamin A in the diet of pigs results in a degeneration of the nervous system, characterized in the advanced stages by striking nervous symptoms such as impaired vision, extreme incoordination, and spasms.
2. Histological examination of the nerves of the pigs with these nervous symptoms shows definite degeneration of some nerve bundles. This degeneration was observed in portions of the spinal cord, optic, sciatic, and femoral nerves.
3. Eye lesions are of minor importance in extreme avitaminosis A in swine.
4. Gilts with avitaminosis A showed irregularity in the oestrus cycle. It occurred more frequently and was of longer duration.
5. Gilts bred prior to the onset of the nervous symptoms either aborted or farrowed dead pigs.
6. Swine do not need vitamin C in their feed for growth and reproduction.

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