HILGARDIA

A Journal of Agricultural Science Published by the California Agricultural Experiment Station

VOLUME 8 SEPTEMBER, 1934 NUMBER 9

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VITAMIN-A DEFICIENCY IN TURKEYS

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W. R. HINSHAW[2] AND W. E. LLOYD[3]

LOSSES FROM OBSCURE CAUSES on certain California turkey ranges, where green feed is limited during much of the growing season, suggested the need for studies on vitamin-A deficiency in turkeys. The experiments reported herein were outlined to determine the possible relation of A-avitaminosis[4] to turkey mortality and to obtain information concerning the effect on turkeys of various vitamin-A levels.

A-avitaminosis in chickens has been fully described by Beach(1), Emmett and Peacock(3), Seifried(10), Elvehjem and Neu(4), and many other investigators, so that the pathological changes in chickens are well established. Chicks were included in one experiment as a control on the methods used and as a basis for comparing A-avitaminosis in the two species.

Scott and Hughes(9), using yellow corn as the source of vitamin A, showed that turkeys required more vitamin A than did chickens.

This paper is concerned chiefly with the disease phases of the problem. Because published data on the vitamin-A requirements of turkeys are limited, the results on the comparative value of dehydrated alfalfa-leaf meal for turkeys and chickens are also included. Certain vitamin-A liver storage data collected in connection with the experiments have already been published by Guilbert and Hinshaw.(6)

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[4] Hereafter in the text, the term "A-avitaminosis" will be used interchangeably with "vitamin-A deficiency."

EXPERIMENTAL METHODS

Day-old poults and chicks were used for all the experimental work. The poults were hatched on June 21 and July 5, 1932, from eggs laid by pedigreed Bronze turkey hens from the Experiment Station breeding flock; the chicks on June 29 from eggs of the Station Single Comb White Leghorns. The parent stock of both the poults and the chicks received essentially the same rations, containing adequate vitamin A in the form of yellow corn, dehydrated alfalfa-leaf meal,[5] and freshly cut greens.

The basal ration used throughout was as follows: 25 pounds ground white corn, 25 pounds ground barley, 25 pounds ground wheat, 10 pounds fish scrap, 10 pounds dried milk, 3 pounds bone meal, 2 pounds ground limestone, 0.5 pound salt. Before grinding, the white corn was hand-picked to remove all yellow kernels, amounting to about 1 per cent.

This basal ration was supplemented with alfalfa meal and with finely chopped fresh alfalfa, to supply the various levels of vitamin A. According to feeding tests on rats, the alfalfa meal contained at least 230 Sherman rat units per gram; according to chemical analysis, 130 gamma of carotene per gram. The alfalfa-leaf meal was used as the source of vitamin A because of its availability to the turkey grower, because of its high vitamin-A content, because of the ease with which it could be mixed with the ration, and because of the need for information on the amounts of alfalfa meal required for turkey rations.

In the first experiment, the day-old poults were divided into 6 groups of 42 each. Lot 6 received the basal ration; lot 1 the basal ration plus freshly chopped green alfalfa; and lots 2, 3, 4, and 5 the basal ration plus 8, 4, 2, and 1 per cent of alfalfa meal, respectively. When hatched, the chicks were divided into 6 groups of 42 each and were placed in the pens with the week-old poults. The chicks and poults were reared together throughout the experiment.

In the second experiment the poults were divided into 5 groups of 29 each and were treated identically with those in the first experiment except that the group (lot 5) receiving 1 per cent alfalfa meal was omitted.

The birds were brooded in a long house divided into pens, with outdoor cement sun-porches, where from all indications they obtained a plentiful supply of the antirachitic factor. After being kept in the original pens until 12 weeks old, they were moved to an open-front house, with cement runs. The chicken cockerels were removed from the pens after 16 weeks to prevent fighting and injury to the turkeys.

[5] For the sake of brevity, dehydrated alfalfa-leaf meal may hereafter be referred to in the text as alfalfa meal.

Walnut and fig trees shaded portions of the cement runs, but in most cases could be fenced off so that no leaves fell in the yards. The groups getting green feed were placed in the pens most exposed to falling leaves, and every effort was made to keep the yards free of leaves. For the last 3 months of the experiment, when the trees were shedding more rapidly, the birds were kept indoors.

The birds were started on sand as litter, but rice hulls were substituted after 2 weeks. Since a little water-grass *(Echinochloa crus-galli)* seed was present in the rice hulls, each lot got a limited quantity each time the litter was changed; but, as all pens were treated alike, this procedure presumably did not affect the final results.

Every chick and poult that died was subjected to a careful postmortem examination for fungal, bacterial, and protozoan infections, and for indications of vitamin-A deficiency.

When each experiment was closed, at least 4 birds from each lot were killed for autopsy. As a further check, their livers were tested for vitamin A by the antimony trichloride method. Three turkeys from each lot and three chickens each from lots 4 and 5 were placed in a new group and fed on the basal ration, to determine biologically the amount of vitamin-A storage. The remainder were placed together on a normal ration and later, when fully recovered, were sold for market.

EFFECT OF VITAMIN-A-FREE DIETS

Because the disease manifestations in turkeys receiving a completely deficient vitamin-A ration have not been previously described, they are presented below in some detail. Later they will be compared with observations on the disease in turkeys receiving different levels, but inadequate amounts, of vitamin A for normal growth.

Symptoms, Course, and Mortality.—The symptoms in poults receiving very minute quantities of, or no vitamin A, from the time of hatching were those of an acute-infectio-contagious disease except that fever was absent. In the initial experiment the symptoms were first noticed on the 25th day after hatching; in the second experiment, on the 26th day. The first death occurred on the 30th and 32nd days, respectively, and 100 per cent mortality by the 44th and 40th days, respectively. In contrast, the chicks used for comparison first showed symptoms on the 27th day, the first death occurred on the 34th day, and all were dead by the 56th day.

Daily notes from the first appearance of symptoms were taken on 50 poults. These birds displayed listlessness and an unsteady gait (always the first symptoms observed); they tended to sit with sagging wings,

drooping heads, and closed eyes (fig. 1). Other early symptoms were increased lacrimation, suggested by "foaming" of the lacrimal secretions; swelling of the nictitating membranes; and a slight nasal discharge. In 4 of the 50 poults, symptoms were either not observed or were just appearing at the time of death.

Since the basis for daily individual examination was the discovery of definite abnormalities by flock inspection, such symptoms as swollen nictitating membranes may have been overlooked until the poult was picked up for examination. A milky exudate, though occasionally present early, was usually a later symptom. As the disease advanced, the nictitating membrane often concealed half the eyeball and appeared dry and rough, the surface sprinkled with a finely divided white powdery exudate.

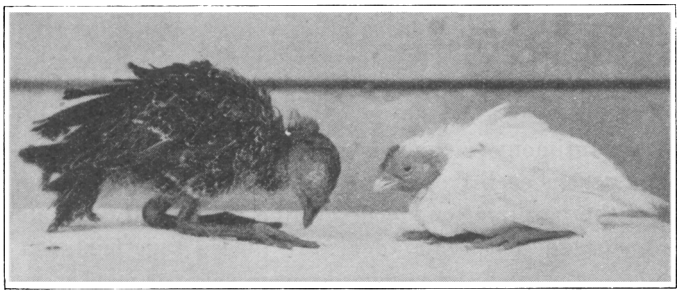

Fig. 1. A five-weeks-old poult and a six-weeks-old chick. Both received the basal ration from time of hatching and were showing typical symptoms of A-avitaminosis for the respective species.

Early in the morning, many poults were found with their eyes "glued" shut. Eye changes were observed in 46 of the 50 poults during the course of the disease. In some, as the disease advanced, the secretion tended to increase rather than decrease and to appear milky. Little or no caseated whitish-yellow pus formed in the eyes or sinuses, probably because the disease was acute.

In only 1 poult were pustules in the mouth, common in more chronic cases, observed before death. Slight nasal discharges were noted in 22 cases, and marked nasal catarrh appeared in 6. In these, the poults tended to sneeze and cough, though such symptoms were not general. The sick poults continued to eat and drink until just before death. When symptoms first appeared, it was very difficult to keep water fountains clear of food deposited there because of the difficulty in swallowing. No similar trouble was experienced in the control pens or in pens that failed to show symptoms.

Rectal temperatures continued within the normal range, 105.0° to 107.0° F, until just before death, when they took a marked drop to as low as 101.4°.

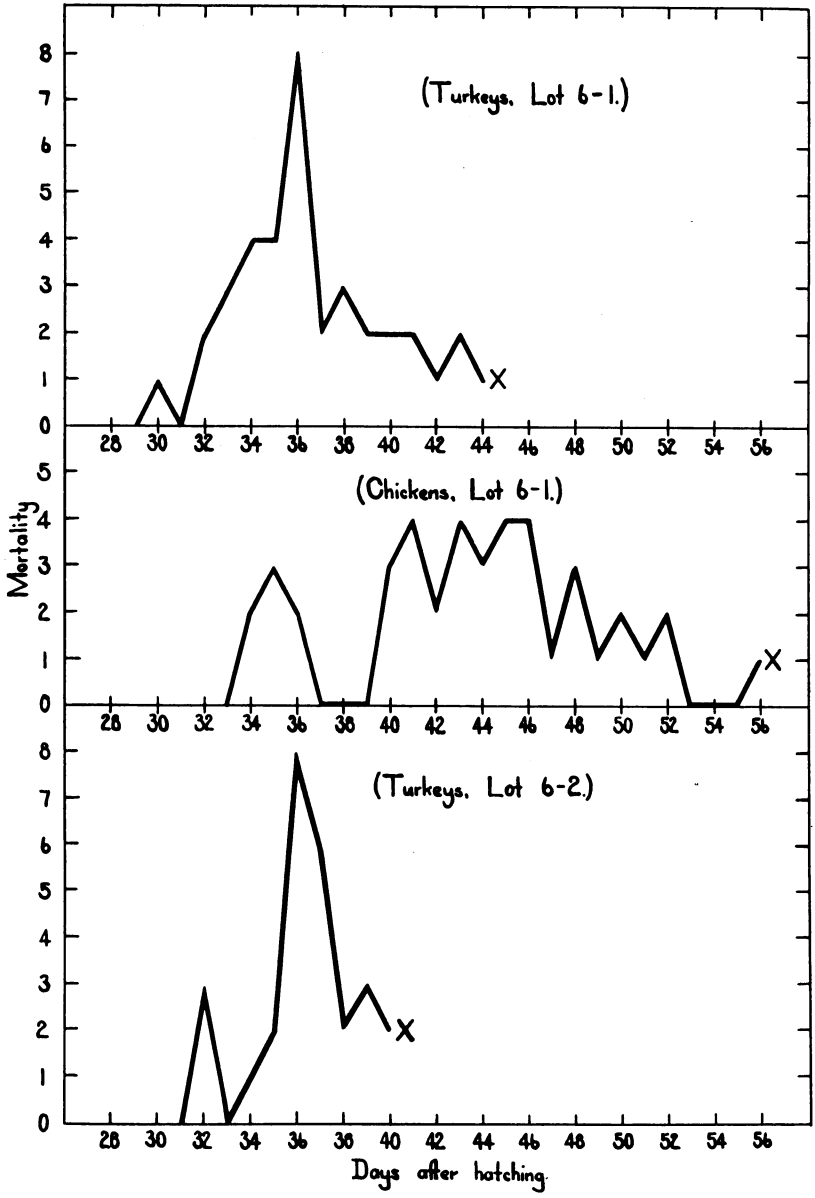

Fig. 2. Comparative mortality rates for the chicks and poults receiving the basal ration. No losses from A-avitaminosis occurred before the 30th day, and 100 per cent mortality was reached at the time indicated by *X*.

Figure 2 shows the mortality curves of the two pens of poults and the one pen of chicks from the 28th day until all the birds were dead. Among the chicks there was no mortality until losses from A-avitaminosis began. Five turkeys in one pen and 2 in the other died from undiagnosed causes before definite lesions of A-avitaminosis were observed. In each case the turkey mortality curve resembled that of an acute infection, with most deaths occurring on the 4th and 6th day after symptoms first appeared.

TABLE 1

DEATHS IN LOT 6 (BASAL RATION) DISTRIBUTED BY DAYS FROM FIRST APPEARANCE OF SYMPTOMS IN THE FLOCK TO TIME OF DEATH

Number of days to death after first appearance of symptoms	Turkeys		Chickens	
	Number of deaths	Per cent of total deaths	Number of deaths	Per cent of total deaths
1	7	12.96	0	0.00
2	4	7.41	0	0.00
3	14	25.93	0	0.00
4	11	20.37	2	4.76
5	4	7.41	2	4.76
6	5	9.26	2	4.76
7	7	12.96	3	7.14
8	0	0.00	5	11.90
9	1	1.85	3	7.14
10	1	1.85	5	11.90
11	0	0.00	5	11.90
12	0	0.00	4	9.52
13	0	0.00	4	9.52
14	0	0.00	1	2.38
15	0	0.00	2	4.76
16	0	0.00	3	7.14
17	0	0.00	1	2.38

Of 5 poults observed while dying, 4 lay on their sides in a comatose condition and, except for gasping and slightly increased movement of the legs, did not struggle. The fifth struggled and flapped its wings until it finally gasped and died.

The symptoms, the course of disease, and the rate of mortality in the chicks receiving the basal ration were more chronic than in the turkeys on the same ration. The chicks, like the turkeys, became dull and listless. When disturbed, however, they were nervous and excited, a condition not observed in the turkeys.

Records on the course of the disease were kept on 54 poults and 42 chicks. Table 1 gives the distribution of deaths according to the time after appearance of first symptoms. As shown by this table and by figure 2, the course of the disease was much more prolonged in the chicks.

Gross Pathology.—Each bird that died was carefully examined for

gross pathological changes, fungi, coccidia, and worm eggs. Bacteriologic examinations were made on 20 poults and 18 chicks, with results that will be reported under another heading.

The types and principal location of the gross lesions are given in table 2. Most of the birds were in fair flesh at the time of autopsy, and a few that died early in the experiment were in good condition.

TABLE 2

DISTRIBUTION OF PRINCIPAL LESIONS IN TURKEYS AND CHICKENS IN LOT 6 (BASAL RATION)

Type and location of lesions	Turkeys*		Chickens†	
	Number	Per cent	Number	Per cent
Xerophthalmia	52	81.25	42	100.00
Sinusitis	29	45.31	13	30.95
Pustules in mouth	21	32.81	34	80.95
Pustules in upper esophagus	24	37.50	23	54.76
Pustules in crop	39	60.94	32	76.19
Pustules in lower esophagus	25	39.06	17	40.48
Enlarged proventriculus	2	3.13	11	26.19
Catarrhal to caseous exudate in bursa of Fabricus	44	68.75	37	88.10
Excessive urates in kidneys	6	9.38	25	59.52
Excessive urates in ureters	1	1.56	25	59.52
Urates in abdominal cavity	0	0.00	10	23.81
Urates in thoracic cavity	2	3.13	8	19.05

* Total turkeys observed, 64.
† Total chickens observed, 42.

As shown in table 2, the lesions were similarly located in the two species but with one exception (sinusitis) were somewhat more prevalent in the chick. This was especially true of urate deposits, which are considered of diagnostic value in A-avitaminosis of chickens. In only 6 of the 64 poults examined were abnormal urate deposits observed in the kidneys, and in only 1 were the ureters abnormally distended. In contrast, 25 of the 42 chicks had enlarged kidneys and ureters distended with urates.

The bursa of Fabricus is not mentioned, in the A-avitaminosis literature available, as an organ commonly affected in the disease of young birds. In these experiments, however, 44 of the 64 turkeys and 37 of the 42 chicks exhibited lesions in it. Most commonly, the bursa contained a deposit of a flaky white urate-like substance in varying quantities, which often distended it to two or three times its normal size (fig. 3). In other cases, the lesions varied from a thick mucoid exudate to a mixture of white flaky and mucoid deposits. In most instances the mucous membranes appeared dry but were not greatly involved.

To determine the incidence of such deposits in normal birds, between 200 and 300 young turkeys known to have been receiving adequate

amounts of vitamin A have been examined. In no instance has a similar deposit been observed in the bursa.

Pustules, containing a whitish-yellow caseous exudate, in the glands of the upper digestive tract, were much more pronounced in chicks than in poults. In the latter, the glandular portions of the crops showed lesions more consistently than the other divisions, but in most instances ex-

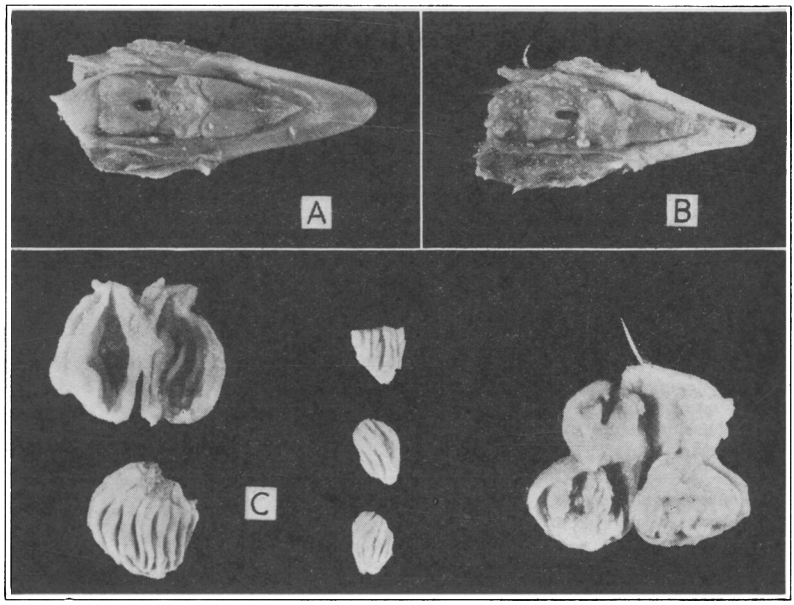

Fig. 3. *A*, Floor of mouth and pharyngeal region of a 40-day-old turkey that died from A-avitaminosis.

B, Same of a 45-day-old chick. Note the large numbers of pustules in *B* as compared with *A*. These specimens are typical for the two species.

C. Sagittal sections of bursas of Fabricus and caseous plugs characteristic of A-avitaminosis in young turkeys and chickens. The left bursa was from a poult; the right, from a chick. The middle specimens are typical caseous plugs from bursas taken from chicks.

hibited only swelling. Seldom were more than five or six pustules observed in the mouth or esophagus of the poults, whereas in the chicks the mucous membranes were frequently studded with lesions. Figure 3 shows the caseous deposits in the bursa of Fabricus and the swollen mouth glands of a chick and a poult.

The infraorbital and orbital sinuses were commonly involved in both species, but more severely in the poults. In the acute cases where few or no nasal discharges were noticed, the mucous membranes of the sinuses were inflamed and contained little or no exudate. In birds that lingered

longer before dying, a deposit of whitish-yellow flaky exudate was often found.

The proventriculus was enlarged in 11 chicks and 2 turkeys. In these cases, the glands were swollen and filled with a milky exudate, but no caseation appeared.

Intestinal changes were not consistent. Slight lack of tone and catarrhal inflammation of the duodenum were seen in a few of the turkeys and chickens. In some of the chicks the cloacae were filled with a granular-to-flaky urate deposit.

A few coccidia were demonstrated in cecal scrapings from 1 poult, and tapeworms in 1 chick. Otherwise, the birds were free of all parasites, and no fungi were found in any crop or esophagus.

Bacteriologic Findings.—Seedings on turkey meat infusion agar were made with the bone marrow and with either liver tissue or heart blood from each of 20 poults and 18 chicks. These cultures were incubated for at least 72 hours at 37° C.

Escherichia coli was isolated in pure culture from the bone marrow of 1 turkey and from the heart or liver of 7 turkeys and 1 chick. *Pseudomonas aeruginosa* was isolated from the bone marrow of 1 chick and from the heart or liver of 11 chicks and 1 poult.

EFFECT OF LOW VITAMIN-A LEVELS

The effect of low vitamin-A levels on the health of birds has probably more practical importance than has the effect of completely deficient vitamin-A rations. Certain field evidence indicates that hypovitaminosis (border-line cases) not showing typical recognized lesions may cause considerable losses. Lots 1 to 5 were included in order to ascertain the manifestations of hypovitaminosis in turkeys. Chickens were used in one trial, as mentioned previously, for the purpose of comparison. All pens were held intact for 28 to 30 weeks.

Since no clinically recognized cases of A-avitaminosis occurred in any chickens receiving more than 1 per cent of alfalfa meal, such groups can be eliminated. No mortality from A-avitaminosis occurred in the chickens receiving 1 per cent of alfalfa meal, although a few clinical cases developed towards the end of the experiment. Being fairly typical of cases of A-avitaminosis already described, these will not be included in this paper.

The results with turkeys will be discussed collectively for the two trials, from the standpoint of the relation to other diseases and of A-avitaminosis manifestations.

Influence on Other Disease Conditions.—Table 3 summarizes the probable causes of death for both trials. A-avitaminosis in each case was diagnosed by observing symptoms before death and by autopsy findings. With few exceptions, the birds were not killed, but allowed to die, before autopsy. Other diseases and pathological changes are included because in this phase of the experiment the complicating factors are probably as important as A-avitiminosis. All pens had the same oppor-

TABLE 3

DISTRIBUTION OF DEATHS IN TURKEYS ACCORDING TO CAUSE

Probable cause of death	Lot 1 (Basal ration plus green alfalfa)	Lot 2 (Basal ration plus 8 per cent dehydrated alfalfa-leaf meal)	Lot 3 (Basal ration plus 4 per cent dehydrated alfalfa-leaf meal)	Lot 4 (Basal ration plus 2 per cent dehydrated alfalfa-leaf meal)	Lot 5 (Basal ration plus 1 per cent dehydrated alfalfa-leaf meal)	Lot 6 (Basal ration only)
A-avitaminosis	0	0	11	17	11	64
A-avitaminosis and coccidiosis	0	0	6	15	29	1
A-avitaminosis, coccidiosis, and mycosis of crop	0	0	1	9	1	0
A-avitaminosis and mycosis of crop	0	0	3	6	0	0
Coccidiosis	3	4	1	2	0	0
Paralysis plus coccidiosis	4	4	0	0	0	0
Paralysis of very young poults without coccidiosis	0	3	4	1	0	0
Enteritis	23	2	4	3	0	0
Miscellaneous	6	5	3	4	1	6
Total mortality	36	18	33	57	42	71
*Per cent mortality**	*50.70*	*25.35*	*46.48*	*80.28*	*100*	*100*

* Based on 71 turkeys used in each lot except in lot 5, where 42 were used.

tunity for spread of infections; and, according to the evidence, in both coccidiosis and moniliasis, vitamin-A deficiency may be a factor in the mortality. Proof of this observation, however, lies in artificially producing the diseases in question so as to have equal distribution in all pens.

The excessive mortality caused by enteritis in the pens receiving green alfalfa (lot 1) occurred during the first 6 weeks and was greatest during the first 3 weeks. This loss has not been accounted for, although there is some field evidence that very young poults apparently do not tolerate freshly chopped young alfalfa leaves given earlier than the second week after hatching.

Part of the mortality not associated with gross lesions of A-avitaminosis is traceable to the fact that the experiments were conducted at the end of the regular breeding season; late-hatched poults in the hot interior valleys of California often suffer considerable infant mortality.

Coccidiosis did not appear in either experiment until the poults were about 8 weeks old.

Moniliasis of the crop was an important factor in lots 3 and 4 of the second experiment, in which no lot 5 was included. Other research work by one of the authors (Hinshaw[7]) has shown that moniliasis in turkeys has most economic importance in flocks suffering from other debilitating conditions.

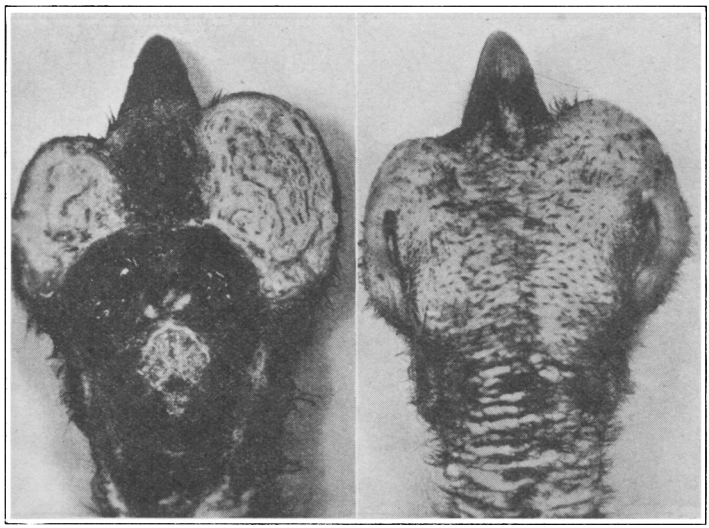

Fig. 4. An extreme case of sinusitis in a turkey hen suffering from A-avitaminosis after being fed for 8 months on a ration containing a low level of vitamin-A. The picture on the left is a sagittal section of the head shown on the right. Note the massive accumulation of the whitish-yellow caseous exudate typical of sinusitis as associated with A-avitaminosis in turkeys.

Tapeworms *(Choanotaenia infundibulum)* were somewhat more common in the chickens than in the turkeys killed for autopsy at the close of the experiment; and they were most prevalent in the chickens receiving a 1 per cent level of alfalfa meal.

Sinusitis (swellhead) of unknown etiology is prevalent in many turkey flocks. Field observations have sometimes suggested vitamin-A deficiency as a contributing factor; this possibility was further confirmed in a feeding trial conducted in 1931. Two lots of 38 Bronze turkeys each were started June 24, 1931, on the following "all-in-one" mash as a basal ration: 25 pounds yellow corn, 25 pounds wheat, 25 pounds barley, 15 pounds fish scraps, 10 pounds dried milk, 5 pounds bone meal, 2 pounds limestone, 0.5 pound sodium chloride.

Both lots were cared for identically except that one had daily access to

fresh greens (lawn clippings or freshly chopped alfalfa) whereas the other had 5 pounds of sun-cured alfalfa meal (in this instance, leaf and stem meal) added to the basal ration and received no fresh greens. Both lots appeared normal for about 4 months; then the one receiving the alfalfa meal became unthrifty. The green-feed lot made normal gains until the termination of the experiment on March 9, 1932. The total mortality in the two groups was 78.94 and 20.51 per cent, respectively.

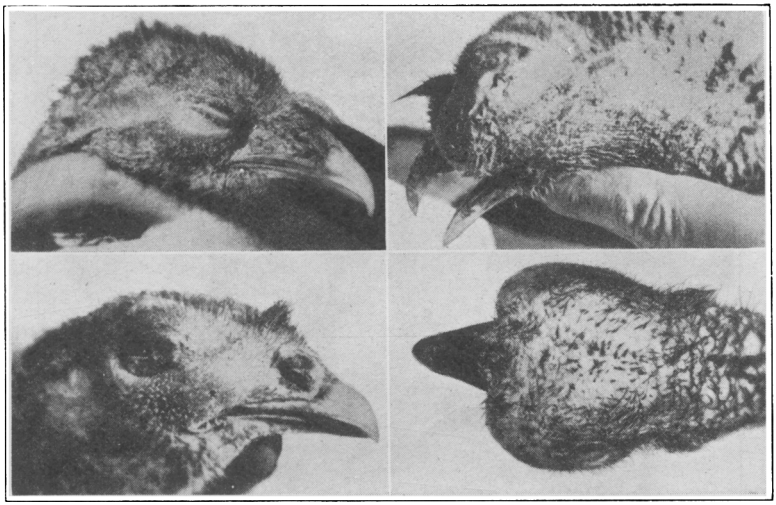

Fig. 5.—Four turkey heads photographed a few days before the birds died from A-avitaminosis. These illustrate the various degrees of ophthalmia and sinusitis seen in the advanced stages of the more chronic form of A-avitaminosis, caused by a continuously low vitamin-A diet.

Apparently the sun-cured alfalfa meal used was of low vitamin content, for by December 18 a few typical cases of xerophthalmia had appeared among the turkeys receiving this supplement. Before the close of the experiment more than one-half of the turkeys in this lot developed sinusitis, in its early stages indistinguishable from swellhead (fig. 4). Typical lesions of A-avitaminosis were found in the birds that died. In contrast, the other group remained normal and showed no indication of A-avitaminosis on postmortem examination of representative birds.

In the 1932–33 experiments, individual cases of sinusitis indistinguishable in the early stages from swellhead were seen, but they were less severe than in 1931–32 (fig. 5).

Comparison of A-Avitaminosis Manifestations in the Different Lots.—The disease in the turkeys of lot 6, which received no vitamin A, was so acute that in many instances manifestations of A-avitaminosis did not

TABLE 4

SUMMARY OF MORBIDITY AND MORTALITY IN THE TURKEYS ASSOCIATED WITH A-AVITAMINOSIS

Lot and ration		Cases of A-avitaminosis		Deaths associated with A-avitaminosis		Days to death from first symptoms	
		Total number	Per cent of total birds*	Total number	Per cent of total birds*	First death	Last death
Lot 3 (Basal ration plus 4 per cent of dehydrated alfalfa-leaf meal)	First trial	7	16.67	7	16.67	63	32 alive at termination
	Second trial	16	55.17	14	48.28	42	6 alive at termination
Lot 4 (Basal ration plus 2 per cent of dehydrated alfalfa-leaf meal)	First trial	28	66.67	25	59.52	43	11 alive at termination
	Second trial	23	79.31	22	75.86	34	5 alive at termination
Lot 5 (Basal ration plus 1 per cent of dehydrated alfalfa-leaf meal)†		41	97.62	41	97.62	47	147‡
Lot 6 (Basal ration)	First trial	38	90.48	38	90.48	30	44‡
	Second trial	27	93.10	27	93.10	32	40‡

* Based on 42 birds in first trial and 29 birds in second trial.

† Only one trial with lot 5.

‡ All died.

TABLE 5

SUMMARY OF DISTRIBUTION OF GROSS LESIONS IN THE TURKEYS

Location of lesions	Lot 3 (Basal ration plus 4 per cent of dehydrated alfalfa-leaf meal)		Lot 4 (Basal ration plus 2 per cent of dehydrated alfalfa-leaf meal)		Lot 5 (Basal ration plus 1 per cent of dehydrated alfalfa-leaf meal)		Lot 6 (Basal ration only)	
	Birds affected	Per cent of total	Birds affected	Per cent of total	Birds affected	Per cent of total	Birds affected	Per cent of total
Eyes	15	71.43	31	68.89	37	90.24	52	80.00
Sinuses of head	3	14.29	8	17.78	5	12.20	29	44.62
Mouth	4	19.05	13	28.89	31	75.61	21	32.31
Upper esophagus	5	23.81	16	35.56	31	75.61	24	36.92
Crop	2	9.52	9	20.00	26	63.41	39	60.00
Lower esophagus	6	28.57	15	33.33	29	70.73	25	38.46
Kidneys	4*	19.05	0	0.00	0	0.00	6	9.23
Bursa of Fabricus	8	38.10	16	35.56	37	90.24	44	67.69
Trachea	2	9.52	5	11.11	25	60.98	0	0.00

* Very slight indication of excessive urates.

appear. The longer the birds suffering from inadequate supplies of vitamin A lived, the more typical the disease was of that seen in chickens. This was especially true regarding the external symptoms and lesions.

Table 4 summarizes the data collected when the first recognizable symptoms appeared, and the average time of death after appearance of first symptoms. Lot 6 is included for comparative purposes.

The total period for each trial was between 210 and 220 days. Apparently the amount of vitamin A in the ration and the time of the first

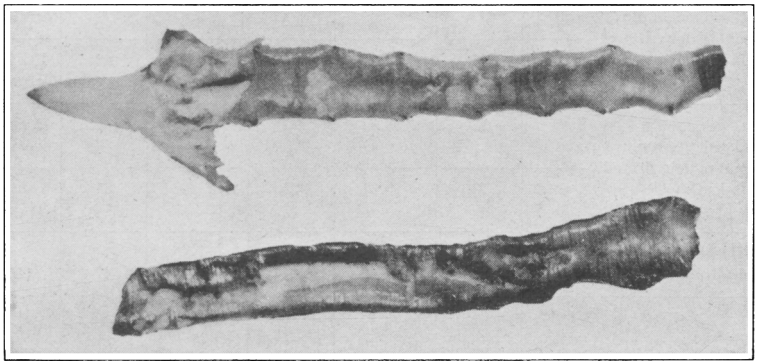

Fig. 6. Trachea of turkey No. 1094, lot 5 (basal ration plus 1 per cent of dehydrated alfalfa-leaf meal). Died from A-avitaminosis at the age of 71 days. Note the caseous plugs at the superior laryngeal opening and the tube-like casts, posterior. Both were common types of lesions found in the more chronic cases in the low-level vitamin-A groups.

death associated with A-avitaminosis were not correlated; but frequently the lack of vitamin A was not the primary cause of death, although lesions were found on autopsy. On the other hand, the course of the disease, the total number of cases of A-avitaminosis, and the percentage mortality were directly related to the vitamin-A level in the rations.

A marked individual variation in susceptibility of the turkeys to A-avitaminosis was noted in lots 3, 4, and 5. This may be attributed to the selective ability of certain birds to obtain more vitamin A than others, or to natural resistance. The most notable example of this individual variation occurred in lot 5: a male, D 1037, lived 147 days (46 days longer than its penmates) and held its own weight for over a month after the others were dead.

Distribution of Lesions.—Table 5 summarizes the distribution of gross lesions in the turkeys suffering from A-avitaminosis. These data were secured only from cases that died or were killed in coma.

The distribution of lesions was not markedly different in birds receiving various levels of vitamin A. Attention is again called to the infrequent occurrence of kidney lesions. A smaller percentage of bursa-of-

Fabricus lesions might be expected in lots 3 and 4, for these birds were older when the losses started, the bursal opening had often closed, and the bursa itself was more or less atrophied. Tracheitis, ranging from a

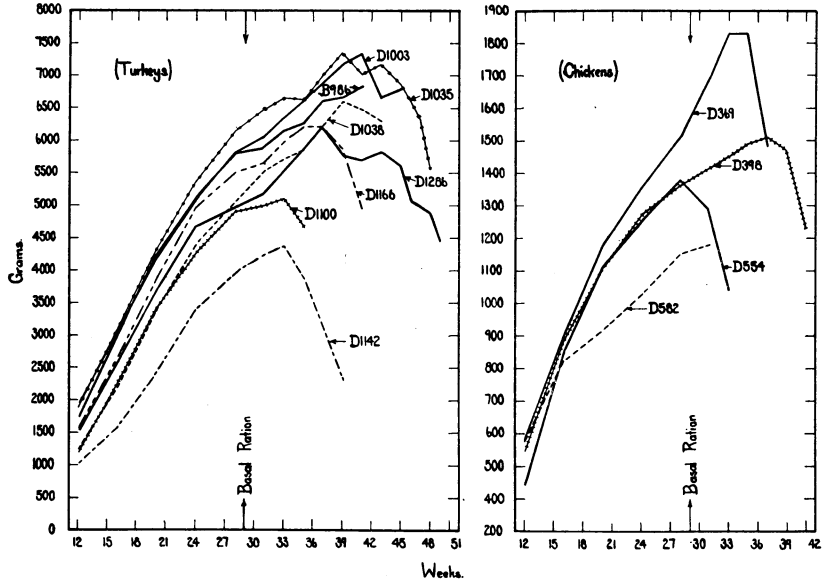

Fig. 7. Growth rates of representative turkeys and chickens fed on a vitamin-A-free diet after first receiving various levels of vitamin A for 29 weeks. The original rations for the different birds were as follows: Turkeys D 1100 and D 1142, basal plus 2 per cent of dehydrated alfalfa-leaf meal; B 986 and D 1166, basal plus 4 per cent of dehydrated alfalfa-leaf meal; D 1003 and D 1035, basal plus 8 per cent of dehydrated alfalfa-leaf meal; D 1038 and D 1286, basal plus fresh greens; chickens D 554 and D 582, basal plus 1 per cent of dehydrated alfalfa-leaf meal; and D 369 and D 398, basal plus 2 per cent of dehydrated alfalfa-leaf meal.

catarrhal to a croupous type, was prevalent in many cases, especially in lot 5. Figure 6 shows a typical case of the latter type.

Urate deposits on the heart or viscera were so uncommon that they were not included in the table.

EFFECT OF REMOVAL OF VITAMIN A FROM THE RATIONS

The phase of the experiments discussed in the following section was conducted to ascertain the length of time required to deplete the body storage of vitamin A, and the disease manifestations under such conditions.

Three representative turkeys from each of the pens of lots 1, 2, 3, and 4, and three representative chickens from each of lots 3 and 4 were placed in the same pen on January 11, 1933, with the basal ration as their sole diet. Daily observations were made for symptoms of A-avitaminosis, and weights were taken of each individual, first at two weeks'

intervals, later at weekly intervals, until death. The growth data for these birds are given in figure 7.

The time of first symptoms and the survival time were directly correlated with the vitamin-A storage, as indicated both by the amount of alfalfa meal in the original rations and by the number of antimony tri-

TABLE 6

RELATION BETWEEN VITAMIN-A STORAGE IN THE LIVERS OF REPRESENTATIVE TURKEYS AND CHICKENS KILLED AT 30 WEEKS OF AGE AND THE SURVIVAL TIME OF PENMATES PLACED ON THE BASAL RATION*

Lot numbers and original ration	Vitamin-A units per gram of liver at 30 weeks	Days after placing on basal ration to		
		First clinical symptoms	First death	Last death
Lot 1, turkeys (basal ration plus green alfalfa)	100	96	†	140
Lot 2, turkeys (basal ration plus 8 per cent of dehydrated alfalfa-leaf meal)	65	87	103‡	135
Lot 3, turkeys (basal ration plus 4 per cent of dehydrated alfalfa-leaf meal)	30	56	90	¶
Lot 4, turkeys (basal ration plus 2 per cent of dehydrated alfalfa-leaf meal)	‡	33	53	86
Lot 4, chickens (basal ration plus 2 per cent of dehydrated alfalfa-leaf meal)	36	56	60	85
Lot 5, chickens (basal ration plus 1 per cent of dehydrated alfalfa-leaf meal)	4	14	24	37

* Table data from work published elsewhere. (6)

† Two birds killed to determine the antimony trichloride blue value at the time of first clinical symptoms.

‡ One bird killed on the 96th day which probably would not have lived to the 107th day.

¶ Remaining birds at 90 days were in advanced stages of deficiency and were autopsied.

chloride blue units in liver extracts in a representative number of penmates killed before this group was placed on the basal ration. Table 6 summarizes these data.

Considerable individual variation was noted, being especially marked in the lot 4 group of turkeys. There was a difference of 33 days in the time of the first and the last death in this lot. The last survivor lived as long as did one of the chickens receiving an equal supply of alfalfa in the original ration, but the chickens on an original 2 per cent level were more comparable to the turkeys on the 4 per cent level.

Figure 8 shows the typical attitude of a turkey that developed A-avitaminosis after being placed on a vitamin-A-free diet. Figure 9 shows the type of lesions seen in the upper esophagus in such cases.

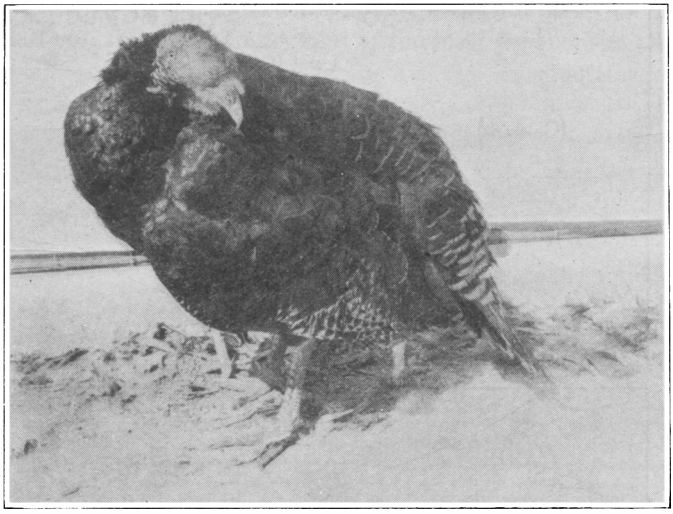

Fig. 8. Turkey hen No. 1166, taken 87 days after transfer from a ration containing a 4 per cent level of dehydrated alfalfa-leaf meal to the basal ration. The bird died 3 days later.

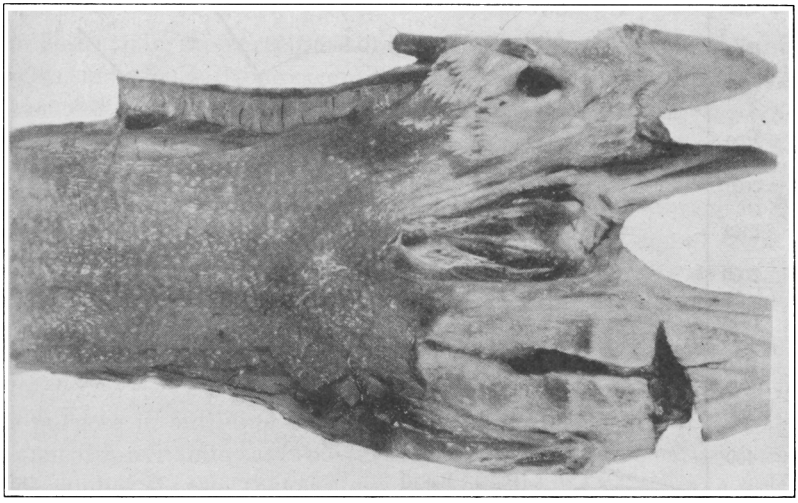

Fig. 9. Portion of head and esophagus of turkey hen No. 1166 (fig. 8) laid open to show the pustular lesions in this part.

MINIMUM VITAMIN-A REQUIREMENTS

Figure 10 presents the growth data for the 1932–33 experiments. According to these data and table 3, the turkeys required at least 8 per cent of alfalfa meal to prevent A-avitaminosis. Furthermore, according to the data on chickens, turkeys require considerably more vitamin A for

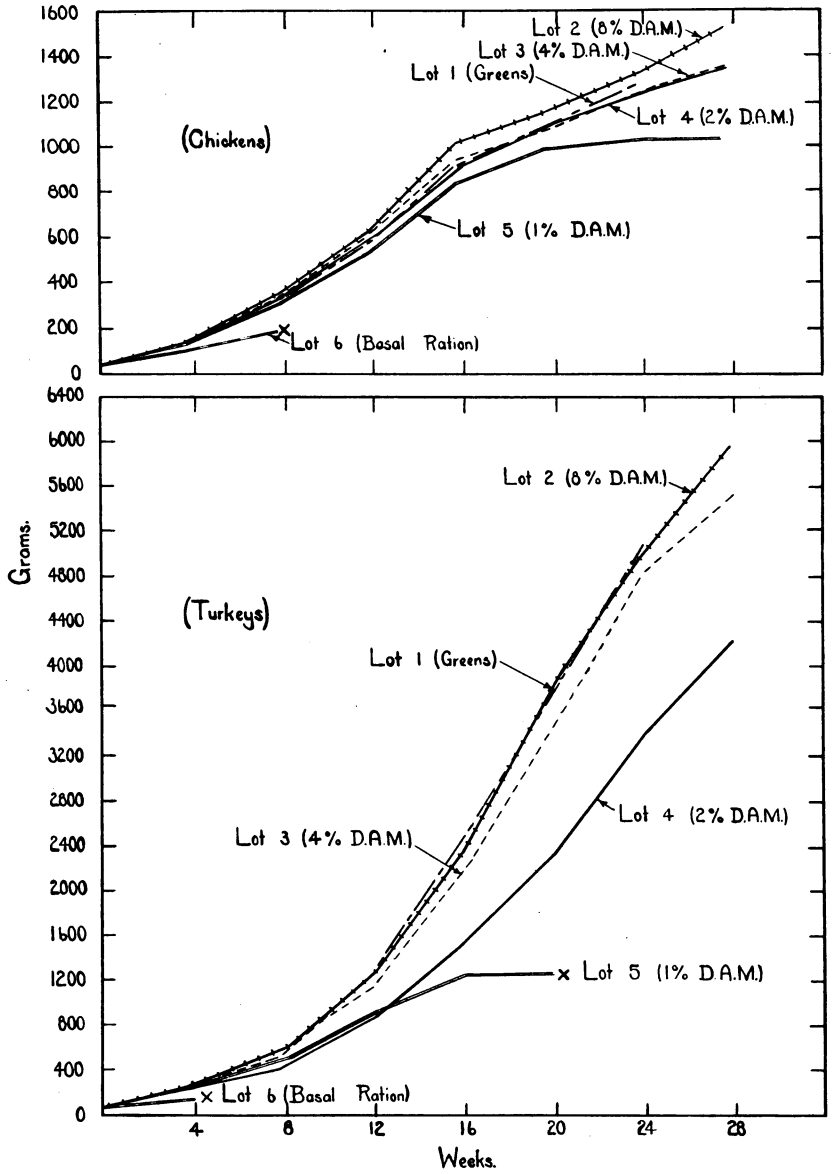

Fig. 10. Growth rates of chickens and turkeys, lots 1–6, inclusive. (The abbreviation "D.A.M." refers to dehydrated alfalfa-leaf meal.)

normal growth. These conclusions are further substantiated by liver analysis (table 6).

The amounts of freshly cut alfalfa consumed by the pens receiving their vitamin A from this source were recorded. In the first trial, includ-

ing both turkeys and chickens, the average consumption in terms of dried alfalfa meal[6] was 11.13 per cent of the total feed consumed. In the second trial, including only turkeys, the average consumption was 9.51 per cent of the total ration.

Table 7 summarizes the estimated consumption by 4-week growing periods. Differences in the two trials probably arise from varying consumption in the individual pens rather than from the influence of the chickens in the first trial.

TABLE 7

PERCENTAGE OF FRESH ALFALFA FED LOT 1 CONVERTED TO PERCENTAGE OF DRIED ALFALFA, IN WHOLE RATION*

	Growth periods						Average for total period
	0–4 weeks	5–8 weeks	9–12 weeks	13–16 weeks	17–20 weeks	21–24 weeks	
Trial 1, chickens and turkeys	2.61	9.83	12.38	10.84	11.20	12.27	11.13
Trial 2, turkeys only	3.01	8.00	8.38	10.04	10.17	10.36	9.51

* Calculated on the basis of fresh alfalfa of the age used, containing 75 per cent moisture.

In both trials, the percentages of dried alfalfa in terms of the total feed consumption with one exception increased with the age of the birds. The amounts consumed further indicated that a ration containing 8 per cent of a good grade of alfalfa meal, like that used in lot 2, approaches the optimum amount for normal growth of turkeys when further vitamin A is not supplied. The data obtained on vitamin-A storage in the livers of these birds (table 6) indicated that they had stored slightly more vitamin A than those receiving 8 per cent of alfalfa meal. The growth rates of the two groups were, however, almost identical (fig. 10).

DISCUSSION

The purpose in outlining these studies was to determine the possible relation of A-avitaminosis to obscure losses in range-reared turkey flocks. These studies showed that certain disease manifestations of A-avitaminosis in turkeys were more obscure than those in chickens under the same conditions. Since a careful search for A-avitaminosis lesions was often required when specimens were brought to the laboratory after death, routine autopsy might have failed to detect the lesions in many instances. For this reason, a diagnostician should inquire carefully into the feeding history and should examine the sinuses, the eyes, and the

[6] Based on unpublished data by H. R. Guilbert, who found that freshly cut alfalfa, of the approximate maturity of that used, contains about 75 per cent moisture.

upper digestive tract for evidences of the disease. Uremia, as indicated by urate deposits on the viscera or by enlarged kidneys and ureters with excessive urate deposits, was seldom seen, even in the more chronic cases, and cannot be considered diagnostic. The bursa of Fabricus, on the other hand, if found thickened and containing a white flaky deposit between the folds, suggests A-avitaminosis.

Scott[7] called attention to hemorrhagic enteritis as a common lesion in the poults of his low-vitamin-diet groups that died from A-avitaminosis. A recheck of our autopsy reports shows that only three specimens of 70 examined (all of which were uncomplicated with coccidiosis or other parasites) were suffering from mild hemorrhagic enteritis. About half of the poults had irregular areas of congestion in the upper third of the intestine, but catarrhal enteritis was a common manifestation.

The results obtained with the Carr-Price[2] antimony trichloride color reaction, utilized in a part of these studies, suggested that it might be an aid in diagnosing borderline cases of A-avitaminosis. The results reported by Guilbert and Hinshaw[6] indicate a direct relation of vitamin-A storage in turkey livers to the age of the bird. Experiments now under way may yield further data on vitamin-A storage at different ages, which will serve as criteria for diagnosing borderline cases of vitamin-A deficiency.

Vitamin A is generally recognized as important in disease prevention, especially in diseases of the head and respiratory organs (Green[5], Turner and Loew[11]). As already noted, at least two important infectious diseases appeared in the 1932–33 trials—coccidiosis and moniliasis. Neither disease occurred among the chickens in the same pens, nor in the turkeys until after the poults and chicks fed on the basal ration had all died. In lots 3, 4, and 5, according to the evidence obtained, inadequate levels of vitamin A may have influenced the severity of the two diseases, since the turkeys in lots 1 and 2 had equal opportunity to contract the same infections.

The reason for the chickens' not developing coccidiosis was that chicken types of coccidia were not present (as determined by frequent fecal examinations) until a few weeks before the close of the experiment. Although chickens are susceptible to moniliasis (Jungherr[8], Hinshaw[7]), they did not contract the disease, presumably because they received a more adequate diet.

Several outbreaks of sinusitis indistinguishable from the disease commonly called swellhead have been observed in connection with vitamin-A deficiency and have responded to cod-liver oil treatment. On the other

[7] Scott, H. M., in personal interview, August, 1934.

hand, numerous outbreaks of equal severity have occurred on ranches where the turkeys have had ample vitamin A in the form of alfalfa meal and fresh greens.

The experimental and field evidence that in certain outbreaks swellhead is definitely related to vitamin-A deficiency, however, suggests the need for more research on this subject. The available knowledge of this relation emphasizes, furthermore, the need of eliminating the possibility of vitamin-A deficiency in all outbreaks of swellhead.

In the 1932–33 experiments, generalized bacterial infections were uncommon. *Escherichia coli* predominated in the turkeys that yielded organisms from the livers, heart bloods, and bone marrow, while *Pseudomonas aeroginosa* was most often isolated from the chickens. This latter observation is especially interesting because Green[5] reported spontaneous *Ps. aeroginosa* infection of rabbits suffering from experimental A-avitaminosis, but not in the controls.

Elvehjem and Neu[4] produced symptoms in White Leghorn chicks with their basal ration somewhat sooner than was possible with the basal ration used in these experiments. This difference may have resulted partly from a slight amount of vitamin A in the latter basal ration, from the small amount of water-grass seed in the rice hulls used as litter, or from a greater reserve storage in the chicks at the time of hatching. Since the poults were kept in the same pens with the chicks, the differing results obtained with the two species are considered comparable.

The results reported indicate that the severity of coccidiosis and moniliasis was influenced by the amounts of vitamin A received by the various groups. Thus they emphasize the importance of making complete autopsy examinations in such experiments, because the practical results of A-avitaminosis are produced not only by the disease itself, but by its effect on existing parasites, infective organisms, and other debilitating conditions.

SUMMARY AND CONCLUSIONS

A comparative study of A-avitaminosis in Bronze turkeys and White Leghorn chickens is reported.

Poults fed a vitamin-A-free ration from the time of hatching, developed symptoms of A-avitaminosis in 25 days in one trial, and in 26 in a second trial. Chicks kept as penmates to the poults in one of these trials began to show symptoms on the 27th day. The disease was much more acute among the poults than among the chicks, the first death among the former occurring on the 30th and 32nd days, respectively, with 100 per cent mortality by the 44th and 40th days, respectively. The first death among the chicks occurred on the 34th day; the last, on the 56th.

In the poults, symptoms resembled those of an infectious disease. Xerophthalmia was the principal differential symptom noted in them as well as in the chicks; but caseated pus seldom collected in the eyes, as in older fowls. Marked nervousness was noted in the chicks but not in the poults.

Lesions in the poults were confined to mucous membranes of the head, the upper digestive tract, the respiratory tract, and the bursa of Fabricus. The lesions, though typical of those described in chickens, were never equally numerous and were confined principally to the crop and lower esophagus. Tracheitis was an occasional manifestation.

The bursa of Fabricus, not previously reported as a seat of lesions, was affected in 44 of 64 poults and in 37 of 42 chicks examined. A white, flaky, urate-like deposit between the thickened bursal folds was the most common manifestation.

Deposits of urates in kidneys and ureters, considered pathognomonic in diagnosis of A-avitaminosis in chicks, seldom occurred in the poults.

In the turkeys receiving various levels of dehydrated alfalfa-leaf meal as the source of vitamin A, the percentage mortality associated with A-avitaminosis varied inversely as the amount of alfalfa in the ration. It was 97.62 per cent, 66.19 per cent, and 54.93 per cent, respectively, for the lots receiving 1 per cent, 2 per cent, and 4 per cent levels of dehydrated alfalfa-leaf meal. No mortality associated with or caused by A-avitaminosis occurred in turkeys receiving either 8 per cent of dehydrated alfalfa-leaf meal or freshly cut alfalfa as the sole source of vitamin A.

Symptoms and autopsy findings in the turkeys dying in the low-level vitamin-A groups, though complicated with coccidiosis and moniliasis, did not differ essentially from those in the pens receiving no vitamin A. As in the vitamin-A-free groups a deposit of urates in the kidneys and ureters was not a common manifestation. The mortality associated with moniliasis and coccidiosis in the different lots indicated a possible relation between the severity of the diseases and the vitamin-A level of the rations.

Sinusitis, in some respects resembling a disease commonly called swell-head, was prevalent in the 1931–32 experiments and occasionally observed in 1932–33. This fact suggests the need of eliminating A-avitaminosis as a possible factor in all field outbreaks of this disease.

The survival time of representative turkeys and chickens, placed in a pen at the end of 30 weeks and given the basal ration, varied directly as the amount of vitamin A received before the transfer—from 24 days in chickens getting a 1 per cent level of dehydrated alfalfa-leaf meal in the

original ration, to 140 days for a turkey fed freshly cut alfalfa ad lib in the original ration.

Marked individual resistance was noted throughout the experiment. One poult receiving 1 per cent of dehydrated alfalfa-leaf meal lived a total of 147 days, or 46 days after its last penmate had died.

Bronze turkeys were found to require a ration including 8 per cent of dehydrated alfalfa-leaf meal (containing approximately 130 gamma of carotene per gram) for normal growth to 30 weeks of age. White Leghorn chickens kept as penmates to the turkeys made normal gains and showed no evidence of A-avitaminosis on as low as a 4 per cent level of the alfalfa meal.

ACKNOWLEDGMENTS

The writers wish to acknowledge their appreciation to H. R. Guilbert for furnishing the carotene and biological assay data on the alfalfa meal used in the experiments; and to T. J. Taylor and C. E. Davies for their aid in making field observations and in keeping careful records throughout the experiments.

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