CLINICAL AND PATHOLOGICAL MANIFESTATIONS OF ZINC DEFICIENCY AND ZINC TOXICITY IN GROWING CHICKENS

Thesis for the Degree of M. S.

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CLINICAL AND PATHOLOGICAL MANIFESTATIONS OF ZINC DEFICIENCY AND ZINC TOXICITY IN GROWING CHICKENS

By

CHARLES FRANKLIN HALL

AN ABSTRACT

Submitted to the College of Veterinary Medicine
Michigan State University of Agriculture and
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MASTER OF SCIENCE
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1959

Approved by C. C. Norrell

ABSTRACT

Three purified diets were randomly assigned to nine groups of one-day-old chicks thus providing three lots of chicks on each diet. The basal ration (zinc deficient) contained 16 ppm of zinc; the control ration contained 20 ppm of added zinc, or a total of 36 ppm; and the toxic ration contained 5,000 ppm of added zinc, or a total of 5,016 ppm. Environmental sources of zinc were eliminated by coating cages with plastic and by supplying distilled drinking water in glass waterers.

One lot of chicks on each diet was withdrawn for study when the chicks were 10, 17 and 31 days old, respectively.

Zinc-deficient birds failed to grow well, did not feather properly and developed severe dermatitis, particularly on the feet. Pathological findings included hyperkeratosis, an apparent increase in number of islets of Langerhans in the pancreas accompanied by loss of chromatin in the cells of the islets, an apparent inhibitory effect on deposition of lymphoid tissue, and a decrease in blood lymphocyte levels.

Birds fed toxic levels of zinc failed to grow and were all dead before 31 days of age. Pathological findings included severe fatty change in the liver, hyaline and hydropic changes in the pancreas and differential leukocyte counts characterized by an increase in immature lymphocytes. It is concluded that changes were due to a combination of starvation and toxicity.

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INTRODUCTION

Zinc and other trace minerals are known to be essential elements in the metabolism of farm animals, including poultry. Until recently it was considered that common feedstuffs contained enough available zinc to meet the nutritive requirements of animals.

When parakeratosis of swine was demonstrated to be, at least in part, a zinc deficiency disease, it appeared desirable to study the zinc needs of chickens more closely. This has been done by a number of workers.

The research described in this thesis was undertaken to determine the pathology of experimental zinc deficiency and toxicity in chickens.

REVIEW OF LITERATURE

Zinc - An Essential Trace Mineral

Morrison (12) states that the presence of zinc in animal tissues has long been recognized. The premise that the mineral is essential to life has been supported by the work of many investigators. In 1934 Todd *et al.* (23) demonstrated that rats failed to grow properly and develop normal hair when deficiencies of zinc existed in rations. More recently other investigations have shown zinc to be essential to the proper development of swine (7,8,9,24), chickens (13,14,19,25) and turkeys (22).

The functions of zinc are not completely understood, but McCollum *et al.* (10) and Pensack and Klussendorf (16) cite references indicating that zinc has a profound effect on a number of enzyme systems. Hormone associations are also suggested.

Zinc is abundant in nature. Most forage and pasture plants contain 30-100 ppm of zinc on a dry basis. These levels have been considered to be sufficient to meet the needs of animals raised under normal conditions (5).

Zinc in Swine Nutrition

In 1953 Kernkamp and Ferrin (4) described a dermatosis of swine characterized by parakeratosis. Attempts to transmit the disorder were unsuccessful, and these workers suggested that the condition might be nutritional or metabolic in origin.

Shortly thereafter Tucker and Salmon (24) reported that the feeding of added zinc had a beneficial effect on parakeratotic swine. These (24) and other workers (7,8,9,16) also demonstrated the relationship of high calcium diets to the incidence of the disorder and suggested

that calcium interfered with the availability of zinc in the ration. Hanson and Sorensen (3) have suggested that parakeratosis may be closely related to fatty acid deficiency.

To determine at what levels zinc could be safely added to swine rations, several workers (1,8) studied possible toxic effects of high levels of added zinc. Lewis et al. (8) reported that no toxic effects appeared in pigs being self-fed on a ration containing 1,000 ppm supplemental zinc. Brink et al. (1) reported that diets containing above 2,000 ppm of zinc produced toxicity in weanling pigs characterized by depressed rate and efficiency of gain, lowered feed intake, arthritis, axillary space hemorrhage, gastritis, catarrhal enteritis, mesenteric congestion, and hemorrhages in the ventricles of the brain, lymph nodes and spleen. Toxic levels of zinc frequently produced death within 21 days.

Zinc in Poultry Nutrition

Following the discovery that added zinc in the ration exerted a beneficial effect in the prevention and cure of parakeratosis in swine, many investigators fed diets containing various levels of zinc to poultry.

Mehring et al. (11) found that rations containing up to 814 ppm of zinc produced no apparent adverse effects when fed to chickens. Pensack and Klussendorf (16) fed added zinc to broilers at levels up to 3,000 ppm. Levels above 1367 ppm of added zinc suppressed growth but did not produce anatomical or pathological change. These workers concluded that suppressed growth was due to reduced feed intake rather than to toxicity. Sturkie (21) studied the effects of 2,320 ppm of zinc in the drinking water of six laying hens. During the 20 day period of the trial, water consumption dropped to one half of normal, body weight decreased 0.40

pounds per bird, and production ceased. One bird died during the course of the trial. When water without added zinc was restored, water consumption returned to normal levels immediately, and four of five survivors were laying within ten days.

Pensack and Klussendorf (16) fed White Rock hens for eleven months on diets containing 180 ppm of added zinc. Production, fertility and hatchability in treated pens were comparable to that in untreated pens; and zinc content of eggs was not increased. All treated birds had differential leucocyte counts within normal range.

Roberson (17) reports that naturally occurring zinc deficiency has not been observed in chickens. However, the zinc needs of growing chickens have been studied by a number of workers in recent years. Young et al. (25) have presented evidence to show that not more than 40 ppm of added zinc is required for maximum growth and development when the basal diet contains 15 ppm of zinc. Pensack et al. (15) suggest that no more than 20 ppm of zinc is required to meet the needs of the growing chick when purified casein rations are fed. Roberson and Schaible indicate that 30 ppm or more supplemental zinc make a ration independent of that in the basal ingredients. Edwards (2) et al. presented evidence in 1958 to show that a chick's environment and source of water may supply the chick's need for zinc even when purified diets containing no zinc are fed.

Symptoms of zinc deficiency in poultry have been described by several investigators. O'Dell and Savage (13) and O'Dell et al. (14) describe the symptoms of zinc deficiency in chicks as slow growth, shortening and thickening of the long bones, development of frizzled feathers, labored respiration, unsteady gait, keratosis of the skin and

increased packed cell volume.

Supplee et al. (22) report that zinc-deficient turkey poults fail to grow well, have abnormal bone formation and fail to develop normal feathers.

Roberson and Schaible (19) and Roberson (17) report that conditions causing acute zinc deficiency in the growing chick produced depressed growth, poor utilization of feed, failure to feather properly with retention of body down and a ragged condition of wing feathers, dermatitis of feet and a stilted gait.

Young et al. (25) described similar symptoms in young chicks and described a near absence of barbs on all but the tips of the primary wing feathers.

The histopathology of zinc deficiency in chickens has been described by O'Dell et al. (14) and Young et al. (25).

O'Dell et al. (14) found that the epiphyseal cartilage of long bones was reduced considerably in width and that there was less cell division than normal. Maturing cartilage cells were small and appeared less active than normal. The bony collar of the shafts of long bones was thinner, but shaft diameter was greater than in the control birds. Osteoblasts were fewer in number, and the Haversian canals were larger. Hematopoietic activity in Haversian canals was increased.

Lymphoid tissues of the thymus and bursa of Fabricius were atrophic in zinc-deficient chicks.

The skin of deficient chicks showed hyperkeratinization, particularly marked on the wings, legs and feet. Hyperkeratinization of skin extending into the feather follicles resulted in atrophy of follicles and feathers.

Esophageal changes were characterized by parakeratosis. Hyperplasia of pancreatic tissue with an increased number of islets of Langerhans was seen, but many of the islets cells had undergone hydropic degeneration.

Similar bone changes have been described by Young et al. (25).

OBJECTIVES

At the time the studies described in this thesis were initiated no published information was available as to the pathological changes accompanying zinc deficiency and toxicity in chickens.

These studies were initiated to determine what changes, if any, would be produced in selected tissues of growing chickens being fed rations deficient and toxic with respect to zinc levels.

EXPERIMENTAL PROCEDURE

The randomization of chicks to pens or replicates was made in the following manner: one-day-old, White Rock, male chicks received from a commercial hatchery were weighed and distributed into consecutive weight groups, such as 36-38, 39-41, etc., each having a weight range of three grams. The groups with the lowest and highest weights were discarded. An equal number of chicks from each weight group were randomized to each pen. This method minimized the effect of initial weight differences on final results. In these studies 90 chicks were assigned to nine pens of 10 chicks each.

Three purified diets were randomly assigned to the pens in triplicate, thus providing three lots of chicks on each diet. The basal diet (Table 1) contained 16 ppm of zinc and served as the zinc-deficient diet. The second diet (control) contained 20 ppm of added zinc producing a total zinc level of 36 ppm. The third diet (toxic) contained 5,000 ppm of added zinc producing a total zinc level of 5,016 ppm. Added zinc was supplied in the form of zinc sulfate.

Birds were maintained in electrically heated, starting batteries with raised wire floors. To prevent environmental sources of zinc from influencing results, all battery parts were coated with opoxy-resin except the wire floors and dropping pans which were coated with shellac. The galvanized (zinc plated) water troughs were replaced with one-gallon, glass, baby chick founts with plastic bases. Distilled water was used instead of tap water as a source of drinking water.

Three pens of birds, i.e. one replicate of birds on each diet, were withdrawn for study when chicks were 10, 17 and 31 days old. Hereafter, these age groups will be referred to as replicate one,

TABLE 1

COMPOSITION OF THE BASAL DIET

Ingredients	Grams/Kg.
Glucose[1]	627.00
Isolated soybean protein[2]	250.00
Corn oil	30.00
Ground cellulose[3]	30.00
DL-methionine	7.00
Glycine	3.00
$CaHPO_4$	22.00
$CaCO_3$	14.70
K_2HPO_4	5.60
NaCl	6.00
$MgSO_4$	2.50
$FeSO_4 \cdot 7H_2O$	0.333
KI	0.333
$CuSO_4 \cdot 5H_2O$	0.026
$CoCl_2 \cdot 6H_2O$	0.0017
$Na_2MoO_4 \cdot 2H_2O$	0.0083
$ZnSO_4$	0.00
Choline chloride	1.50
Inositel	0.25
Niacin	0.05
CaPantothenate	0.02
a-Tocopherol acetate	0.02
Thiamin HCl	0.01
Riboflavin	0.01
Pyridoxine HCl	0.045
Folic acid	0.004
Menadione	0.0005
Biotin	0.0002
Vitamin B_{12} (1 mg/gm)	0.02
Vitamin A (250,000 IU/gm)	0.02
Vitamin D_3 (200,000 ICU/gm)	0.002

1 - Cerelose, Corn Products Sales Co., 440 New Center Bldg., Detroit, Michigan.

2 - Drackett Assay Protein C-1, The Drackett Products Co., Cincinnati 32, Ohio.

3 - Solka Floc, The Brown Co., Berlin, New Hampshire.

replicate two and replicate three respectively.

Average bird weight, symptoms and external lesions were determined prior to killing the birds. The tissues from one half of each pen sample were pooled for microbiological examination. Selected tissues of remaining birds from each pen were collected for pathological examination. Blood films were prepared from each bird, stained according to the Wright method, and differential leucocyte counts determined. Skin, skeletal muscle, heart, lung, liver, spleen, pancreas, duodenum, kidney, adrenal, testis and brain were collected in 10% isotonic formalin for histopathological examination.

Uniform techniques of tissue collection were employed to allow for accurate appraisal of any tissue change noted. Skin was collected from an interdigital space on the foot. Skeletal muscle was collected from the superficial pectoral muscle and was trimmed on a longitudinal plane. Heart was collected and trimmed in cross section at the mid-ventricular level. Lung was trimmed in cross section at mid-level antero-posteriorly. Liver and spleen were trimmed in cross section. Pancreas and duodenum were collected together and trimmed in cross section at a level midway down the duodenal loop. A triad of tissue consisting of kidney, adrenal and testis was removed and in a plane that would allow for a single imbedding procedure. Brain was removed in toto and trimmed on a sagittal plane.

Following fixation, dehydration, imbedding and sectioning, tissues were stained with hematoxylin and eosin. Selected tissues were also stained with Sudan IV for fat and with Mallory's aniline blue.

RESULTS

Livability and Growth

Livability during the course of the experiment is given on Table 2.

Average growth rates for birds are given on Table 3.

In replicate one (10 days old), 10 control birds averaged 75.2 grams; 10 zinc-deficient birds averaged 66.4 grams; and 8 surviving zinc-toxic birds averaged 38.5 grams.

In replicate two (17 days old), 10 control birds averaged 166.1 grams; 10 zinc-deficient birds averaged 94.1 grams; and 6 surviving zinc-toxic birds averaged 49.2 grams.

In replicate three (31 days old), 8 surviving control birds averaged 300.5 grams; and 9 surviving zinc-deficient birds averaged 143.1 grams. No survivors were on the toxic ration at the time samples were taken.

External Symptoms and Lesions

All control birds on each replicate appeared normal when examined.

Zinc-deficient birds appeared to be normal in replicate one except for slight growth depression.

When replicate two of zinc-deficient chicks was examined, stunting was readily observable. Feathering was not proceeding at a normal rate, and the bodies of the birds were still primarily covered with down. Wing feathers which were present were juvenile and ragged. No evidence of skin change was noted.

Zinc-deficient birds of replicate three had symptoms and external lesions as described for replicate two, but the changes were more pronounced (Figure 1). In addition the shanks of birds in this group

TABLE 2

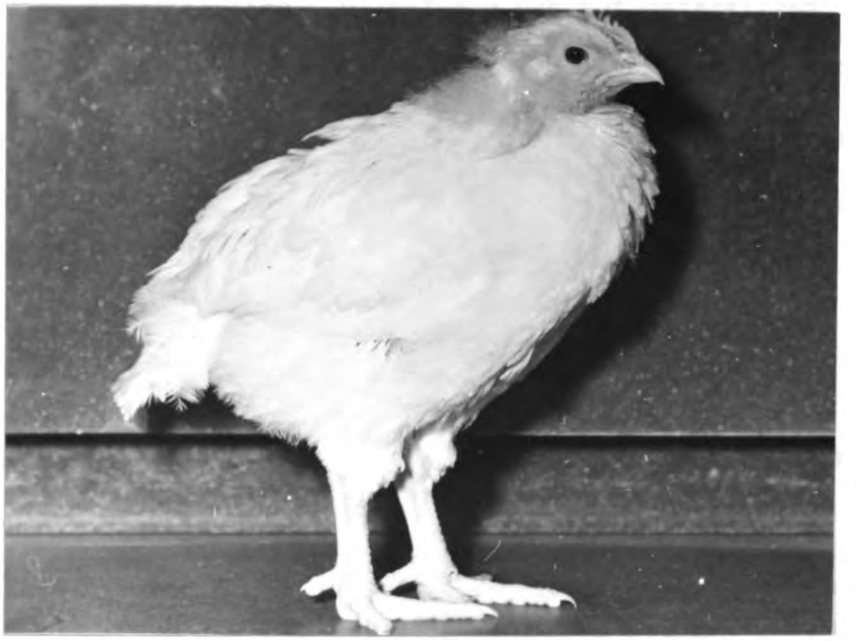
LIVABILITY

Treatment	Surviving Chicks of 10 Started		
	Replicate one (10 days old)	Replicate two (17 days old)	Replicate three (31 days old)
Control	10	10	8
Zinc deficient	10	10	9
Zinc toxic	8	6	0

TABLE 3

GROWTH

Treatment	Average Weight in Grams		
	Replicate one (10 days old)	Replicate two (17 days old)	Replicate three (31 days old)
Control	75.2	166.1	300.5
Zinc deficient	66.4	94.1	143.1
Zinc toxic	38.5	49.2	____



A

B

Figure 1*: A, The normal chicken; B, The zinc-deficient chicken.

*Courtesy of P. J. Schaible and R. N. Roberson.

showed bluish discoloration. Dermatosis was not generalized, but the feet of the chicks had severe encrustations dorsally (Figure 2) and ventrally (Figure 3). The skin was cracked in some of the birds, and open bleeding was observed. Gross bone deformities as described by O'Dell et al. (14) and Young et al. (25) were not observed.

Zinc-toxic birds in replicate one were stunted and weak. The skeletal muscles appeared pale and grayish.

When replicate two of zinc-toxic birds was examined, stunting was very severe. Chicks were extremely weak and had difficulty standing.

All birds in replicate three of zinc-toxic birds were dead prior to the sampling period.

No gross internal lesions were noted in any of the birds examined. Size of organs varied between treatment groups, but this was considered proportionate to weight differences of birds.

Hematology

Blood films were prepared from birds which were killed for histopathological examination. The results of differential leucocyte counts are summarized in Table 4.

No attempt was made to classify lymphocytes as to size. The relative numbers of large and small lymphocytes was considered to be in normal range except in blood films prepared from birds fed toxic levels of zinc. In these instances, a marked increase in numbers of large lymphocytes was seen.

All differential counts were considered to be in normal range except for replicate one birds fed toxic levels of zinc and replicate three chicks fed deficient levels of zinc. In each instance the lymphocyte count was low and the heterophil count high.

A

B

Figure 2*: A, Foot from a normal chicken (dorsal view). B, Foot from a zinc-deficient chicken with hyperkeratosis (dorsal view).

*Courtesy of P. J. Schaible and R. N. Roberson.

A

B

Figure 3*: A, Foot from a normal chicken (ventral view). B, Foot from a zinc-deficient chicken with hyperkeratosis (ventral view).

*Courtesy of P. J. Schaible and R. N. Roberson.

TABLE 4

SUMMARY OF DIFFERENTIAL LEUCOCYTE COUNTS[1]

Treatment	Lympho-cytes	Hetero-phils	Mono-cytes	Eosino-phils	Baso-phils	Myelo-cytes
Replicate one (10 days old)						
Control	71.6	21.0	4.6	1.6	1.2	___
Zinc deficient	66.4	25.2	4.6	0.8	2.6	___
Zinc toxic	47.5	46.0	3.5	___	2.0	1.0
Replicate two (17 days old)						
Control	66.4	26.8	2.8	___	4.0	___
Zinc deficient	57.4	35.2	2.6	1.0	3.6	0.2
Zinc toxic	66.6	21.6	3.3	2.0	5.6	0.6
Replicate three (31 days old)						
Control	67.0	26.8	3.4	0.4	2.2	0.2
Zinc deficient	47.0	45.2	3.2	0.2	4.0	0.4
Zinc toxic[2]	___	___	___	___	___	___

1 Expressed in numbers of cells per 100 counted
2 No survivors

Microbiological Findings

Intestinal contents from each replicate of control and treatment groups yielded coliform and Proteus organisms. These organisms were not considered to be pathogenic to the chicks.

Pooled tissues, consisting of heart, lung, liver and spleen, failed to yield micro-organisms when inoculated on bovine blood-enriched tryptose agar and incubated for 48 hours at 37.5 C. This was uniformly true for each treatment group of each replicate.

Histopathological Findings

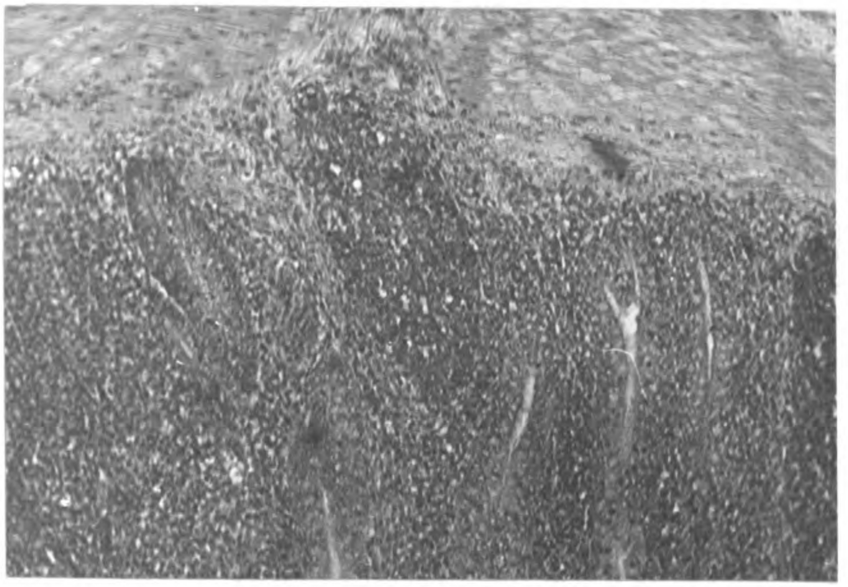
General Findings:

Lymphocytic accumulations, diffuse and follicular (Figure 4), were observed in assorted tissues of all chicks examined. Accumulations were most evident in the tissues from control birds of replicate three. Numerous mitotic figures were present in affected areas. On the other hand, few accumulations were seen in the tissues from zinc-deficient chicks of replicate three.

Skin:

The skin from control birds appeared to be normal. The epidermis was 4-7 cells thick and was covered by a thin layer of keratin (Figure 5).

The skin from zinc-deficient birds was thickened. The epidermis, in some sections, was increased in thickness up to 20 layers of cells. The layer of keratin was also thickened up to five times normal. Nuclei were not present in the keratinized layer; but in some sections, the line of demarcation between the stratum granulosum and the stratum corneum was indistinct, many of the outer cells of the stratum granulosum appearing to be undergoing keratinization. These changes are illustrated in Figure 5.



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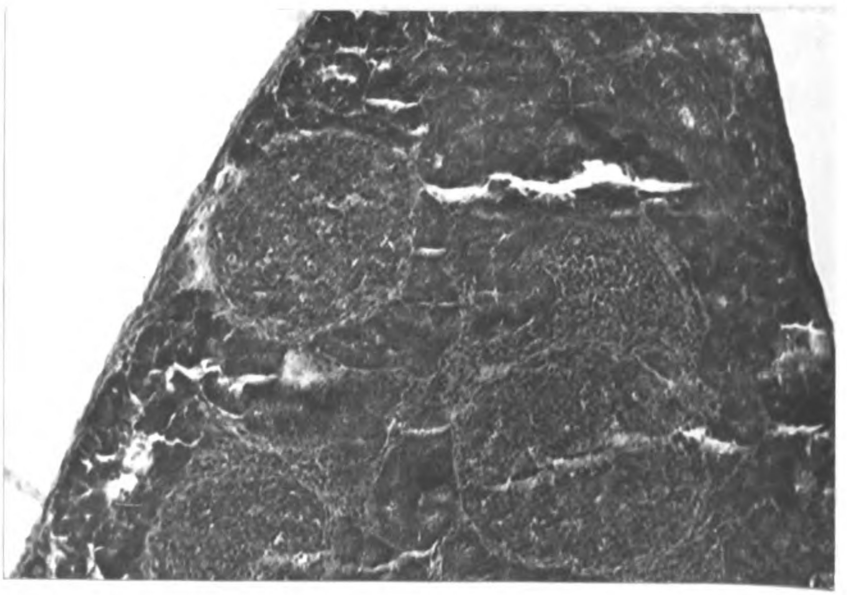

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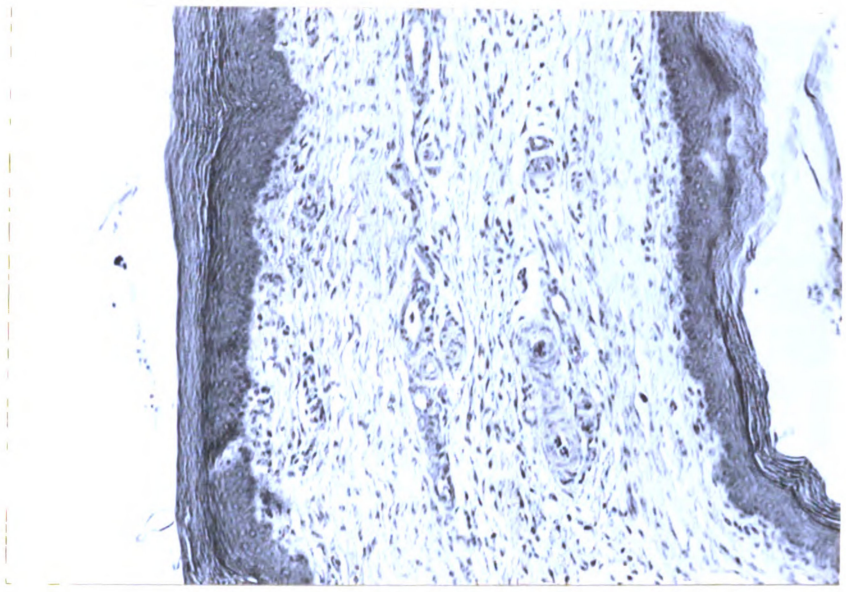
Figure 4: A, Diffuse lymphocytic accumulations in the small intestines. B, Follicular lymphocytic accumulations in the pancreas. X 150, H. & E. stain.



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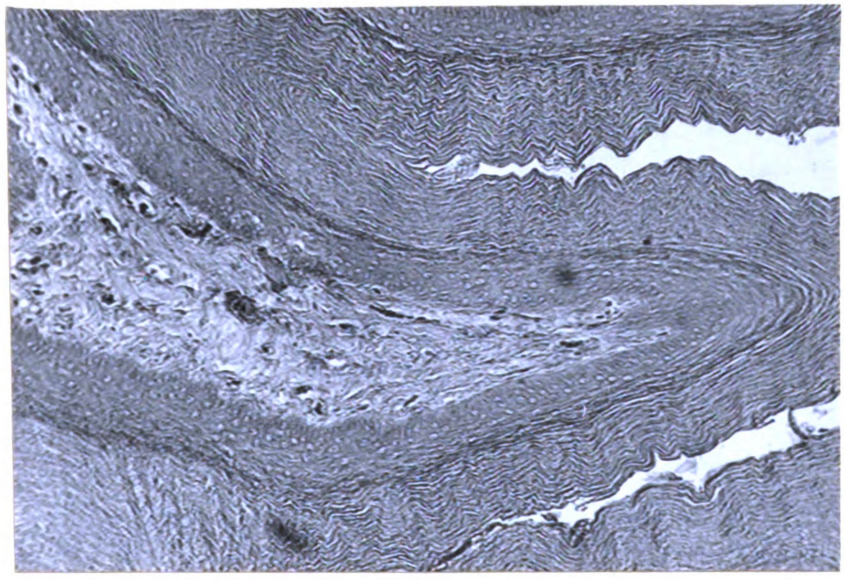


B

Figure 5: A, Skin from a normal chicken. B, Skin from a zinc-deficient chicken with hyperkeratosis. X 150, H. & E. stain.

The skin of birds fed toxic levels of zinc appeared normal.

Skeletal muscle:

The fibers of skeletal muscle from control birds were normal in appearance. In bundles, individual fibers were rather indistinct; and the nuclei were pale and rather sparsely distributed (Figure 6).

Skeletal muscle from zinc-deficient birds had more distinct fibers and nuclei, but individual fibers appeared somewhat smaller in cross section than those of the controls.

The skeletal muscle fibers in birds fed toxic levels of zinc were very small in cross section. The nuclei were quite numerous and deeply basophilic. The individual fibers were quite distinct. The fibers did not appear degenerative (Figure 6).

Cardiac muscle:

Myocardial change, characterized by the presence of diffuse and focal aggregates of heterophils and lymphocytes, was seen in individual birds from each treatment group. The reaction was not consistently found in any treatment group, however. Since bacterial pathogens were not isolated from pooled tissues, including heart, these findings are of unknown significance.

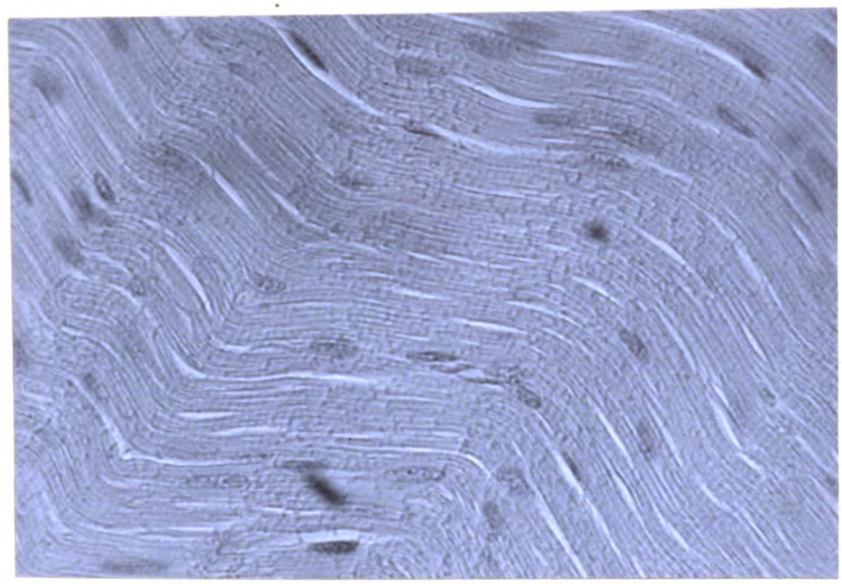
Lung:

Hemorrhage, considered to be due to extraction, was seen in the lungs of all birds. Lymphocytic accumulations, mild to marked, were present about primary bronchi in all chicks.

Liver:

No significant changes were observed in the livers of control birds.

No changes were noted in the livers of zinc-deficient chicks from



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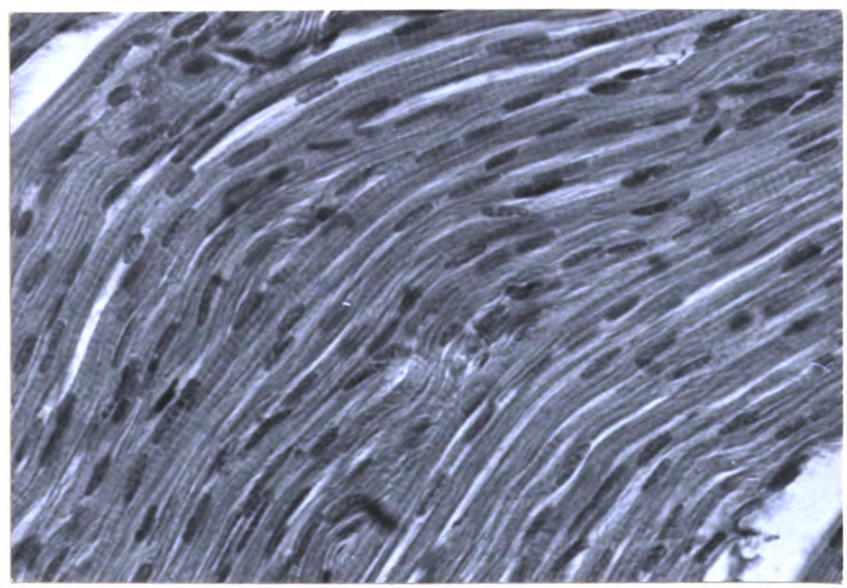


B

Figure 6: A, Skeletal muscle from a normal chicken. B, Skeletal muscle from a zinc-toxic chicken. Note small individualized fibers with numerous deep staining nuclei. X 650, H. & E. stain.

replicates one and two. Slight fatty changes were noted in one of the birds on replicate three (Figure 7).

Marked fatty change, confirmed by Sudan IV stain, was observed in the livers of birds fed toxic levels of zinc. Fat was uniformly dispersed throughout liver tissue (Figure 7).

Spleen:

The spleens of control birds appeared more congested than did the spleens of the zinc-deficient and zinc-toxic chicks. Spleens of the zinc-toxic chicks were very small and had little blood present.

Pancreas:

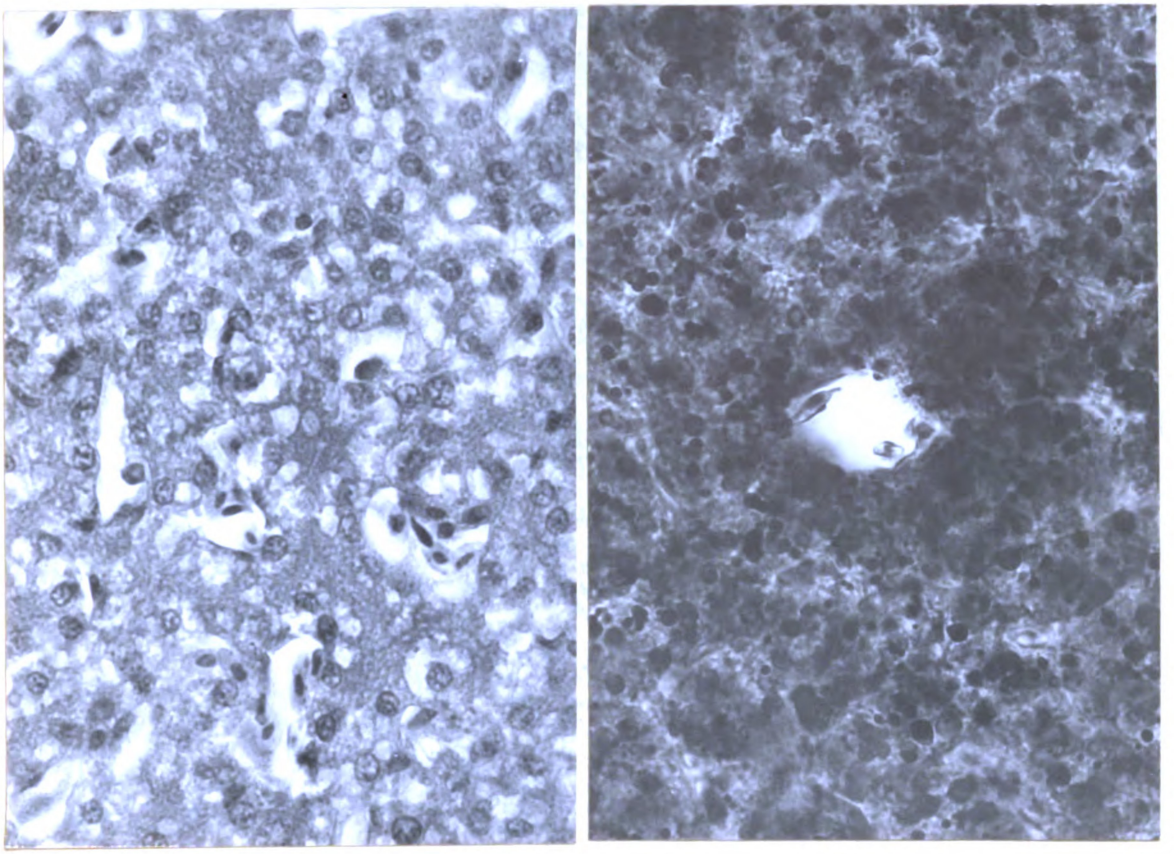
Pancreatic tissue was considered normal in control birds other than for lympho-follicular reactions which were marked in one bird from replicate three. Normal pancreatic tissue is shown in Figure 8.

There was an apparent increase in the number of islets of Langerhans in zinc-deficient birds. Little chromatin material was present in the cells of the islets (Figure 8).

The pancreatic tissue of birds on toxic levels of zinc appeared quite degenerative. Outlines of acini were hazy as cells appeared individualized. Cellular swelling, hydropic change and some hyaline degeneration were noted. Few distinguishable islets of Langerhans were seen (Figure 8).

Duodenum:

Duodenal lymphocytic accumulations were observed in all groups of chicks. Although primarily diffuse, occasional follicular accumulations were seen. These findings appeared most severe in the control birds in replicate three and least severe in the zinc-deficient birds in replicate three.



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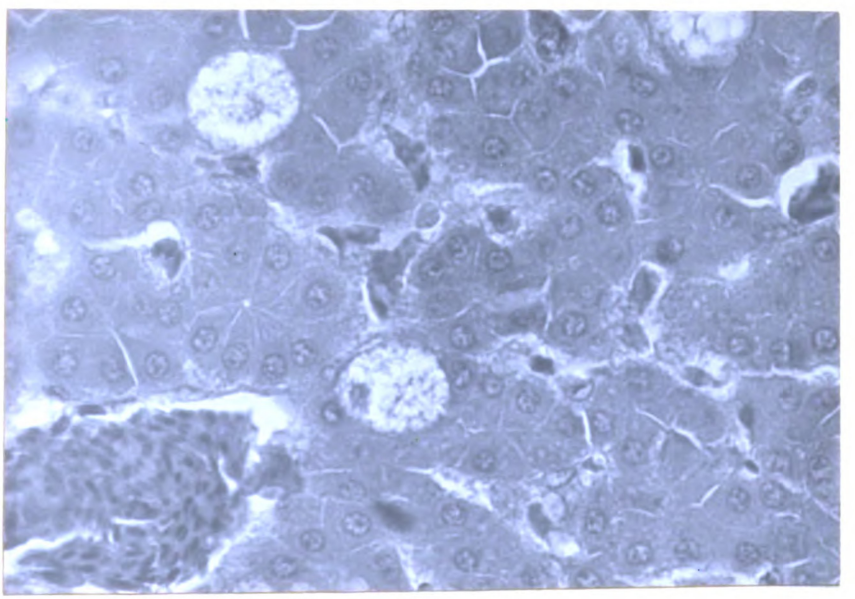
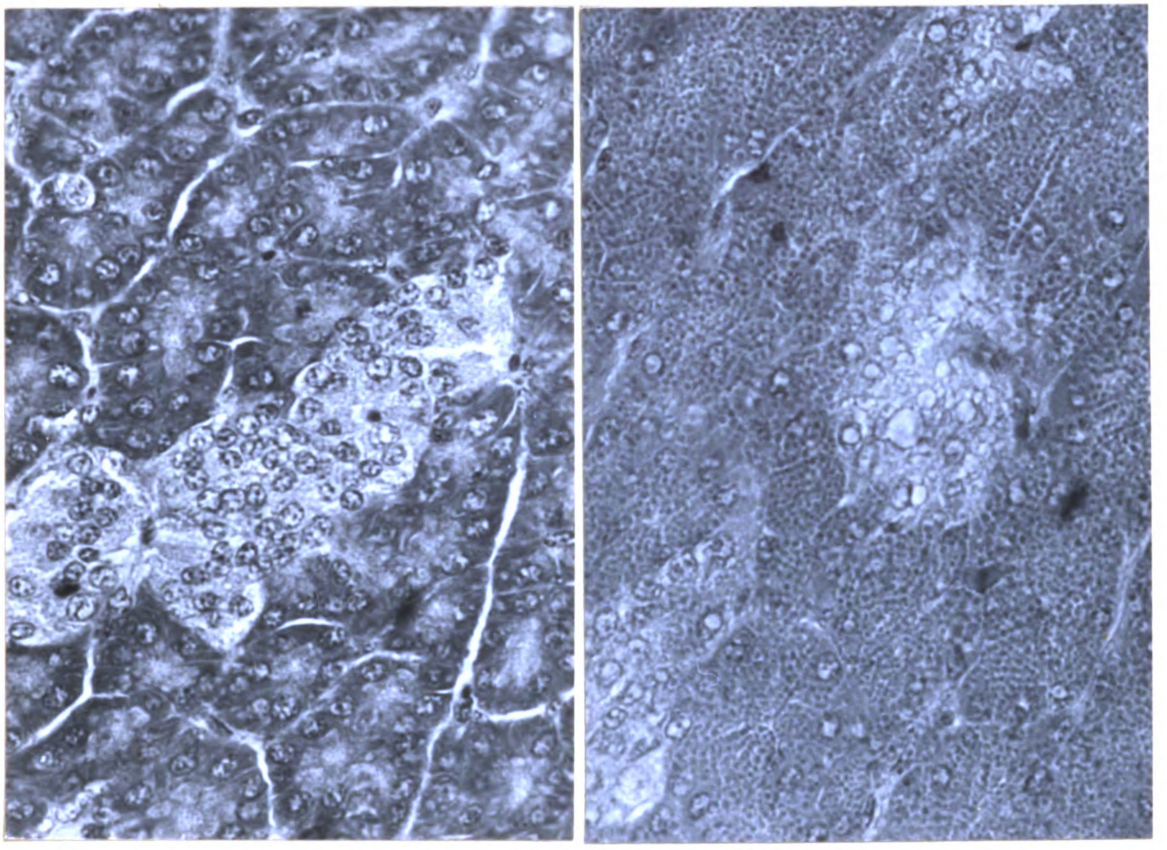


C

Figure 7: A, Liver from a zinc-toxic chicken showing fatty change. X 650, H. & E. stain. B, Liver from a zinc-toxic chicken showing fatty change. X 650, Sudan IV stain. C, Liver from a zinc-deficient chicken containing vacuoles, but essentially normal. X 650, H. & E. stain.



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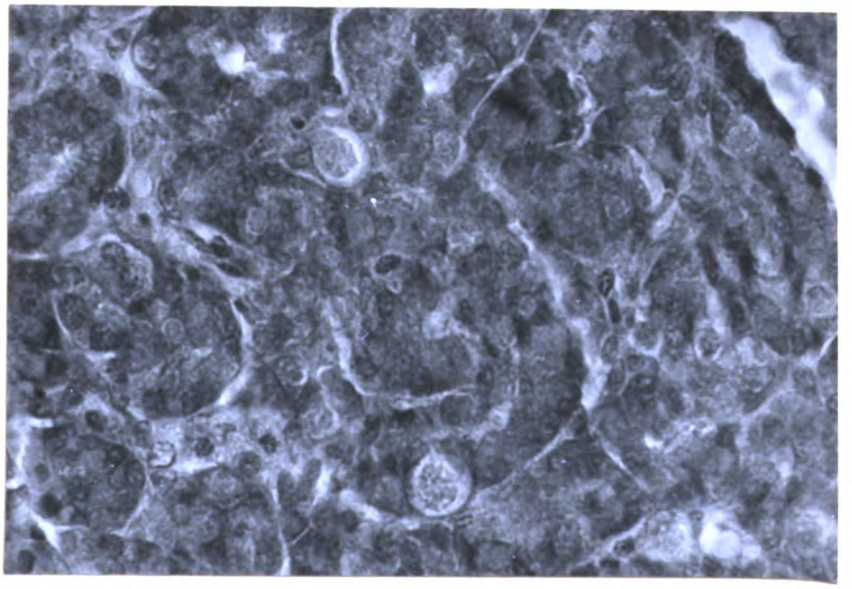

C

Figure 8: A, Pancreas from a normal chicken. B, Pancreas from a zinc-deficient chicken showing loss of chromatin within the cells of the islets of Langerhans. C, Pancreas from a zinc-toxic chicken showing hyaline change. X 650, H. & E. stain.

Kidney, Adrenal, Testis and Brain:

No significant lesions were noted in any treatment group.

DISCUSSION

Zinc Deficiency

Suppression of growth, ragged feathering and dermatosis were observed in chicks fed zinc-deficient diets. These changes were also noted by O'Dell and Savage (13), O'Dell et al. (14), Roberson and Schaible (19), and Roberson (17). The bone changes described by these workers were not observed in this study.

Differential leucocyte counts of birds on zinc-deficient diets were normal in replicates one and two. The drop in lymphocyte count in replicate three could be due to (a) some factor completely unrelated to the zinc deficiency, or (b) the direct effects of zinc deficiency. O'Dell et al. (14) report that lymphoid tissues of the thymus and bursa of Fabricius were atrophic in chicks on zinc-deficient diets. These tissues were not examined in this study, but other tissues, i.e. liver, pancreas, spleen, intestines and lungs from birds on replicate three, revealed less diffuse and follicular lymphocytic accumulations than did tissues from control birds on replicate three. These findings suggest that the normal or pathological presence or absence of lymphocytes in blood and other tissues may be influenced by zinc deficiency.

The microscopic skin changes observed in this study were more severe than those described by O'Dell et al. (14). This is most likely due to the fact that skin from the foot was examined in this study, and it would be expected that trauma would aggravate lesions. Changes observed were typical of hyperkeratosis, whereas zinc deficiency in swine is characterized by parakeratosis (4).

Skeletal muscle fibers were somewhat smaller in cross section than those of controls, but this was considered to be due to suppressed growth.

Pancreatic changes were characterized by an apparent increase in the number of islets of Langerhans and loss of chromatin within the cells of the islets. These changes are similar to those seen by O'Dell et al. (14).

Zinc Toxicity

Severe growth depression and mortality were produced in this study by 5,016 ppm of zinc. Pensack and Klussendorf (16), and Roberson (17), have reported that poor growth is characteristic in chicks when zinc levels in the ration are 3,000 ppm and above. Pensack and Klussendorf (16) suggested that the poor growth at such levels was due to reduced feed intake rather than to direct toxic effects. These workers did not observe any gross anatomical or pathological changes when levels up to 3,000 ppm of zinc were added to the ration.

This suggests the possibility that chick mortality observed in this study may have been due to starvation rather than to direct toxic effects.

The lesions in birds fed high levels of zinc were most apparent in the liver and pancreas. The severe fatty change seen in the liver reaffirms the thought that starvation may have influenced losses. Hyaline and hydropic changes were in evidence in the pancreas, and few islets of Langerhans were distinguishable. That these and the liver changes may have been due to toxicity seems plausible when it is remembered that liver and pancreas concentrations of zinc increase more markedly than do concentrations in other tissues when zinc content of diet is increased (8). Kidney tissue is saturated at a relatively low concentration of dietary zinc (8) and may explain why kidney changes were not observed. Likewise, the lymphocytic shift to more immature forms in the birds on high levels

of zinc suggests that a toxic effect might be present.

Individual muscle fibers did not appear degenerate but were small in cross section, individualized and had numerous deeply staining nuclei.

It is considered that changes seen are due to a combination of starvation and toxicity.

SUMMARY

White Rock, male, broiler chicks were maintained on zinc-deficient (16 ppm), zinc-adequate (36 ppm), and zinc-toxic (5,016 ppm), diets for periods of 10, 17 and 31 days. Environmental sources of zinc were eliminated by coating cages with plastic and by supplying distilled water in glass waterers for drinking purposes.

Symptoms of zinc deficiency were impaired growth rate, poor feathering and dermatitis. Differential leucocyte counts revealed a drop in numbers of lymphocytes. Microscopic manifestations were hyperkeratosis of the skin, an apparent increased number of islets of Langerhans in the pancreas accompanied by loss of chromatin in the cells of the islets, and reduced amounts of lymphoid tissue.

Zinc toxicity was characterized by poor growth and complete mortality within 31 days. Lesions observed in birds were fatty changes in the liver, and hydropic and hyaline changes in the pancreas. Differential leucocyte counts revealed a predominance of immature cells in the lymphocytic series. These manifestations are considered to be due to the combined effects of toxicity and starvation.

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Thesis

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Zinc deficiency and zinc
toxicity in growing chickens.