COCCIDIOSIS IN TURKEYS

By

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A THESIS

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INTRODUCTION

Coccidiosis of the domesticated animals is the cause of great loss, and much time and effort has been spent in its study. In poultry this disease has received the greatest attention, primarily because of the ease of handling the smaller animals. However, coccidiosis in turkeys is as little known today as in 1929 when Tyzzer suggested that further researches were necessary for its elucidation.

In recent years it would seem that there was only one method available by which the coccidia could be studied. In this period most investigations have been directed toward finding suitable therapeutic agents for the control of the disease. There have been relatively few attempts made toward a better understanding of the disease. In fact, the emphasis of many has been on the therapeutic agent, often without an understanding of the disease.

In the study of the parasitic diseases two groups of investigators are found, the parasitologist and the clinician. The former is primarily interested in the parasite reaction, the latter in the host reaction, but neither is too interested in the host-parasite reaction relationship. A knowledge of this host-parasite relationship is necessary for the comprehension of parasitic diseases, in fact all infectious diseases.

A complete understanding of a single disease entity in all its complexity is more than a single individual can hope to achieve. However, we will come closer to reaching

this goal if the disease is not submerged in man-made, departmentalized fields of study. In this study of coccidiosis in turkeys, an attempt has been made to study it as a disease. Therefore, in the following pages will be found the subject matter of the parasitologist, pathologist, immunologist and clinician; and because these groups would be directing their interests toward a common end, this is ultimately subject matter for the poultry producer.

HISTORICAL

Coccidiosis in poultry was considered to be caused by a single species of coccidium, Eimeria avium, until Tyzzer (1929) first demonstrated conclusively the multiplicity of species involved in this disease. This investigator was largely concerned with the coccidia of the chicken, and only incidently with those of the turkey. He showed that not only is there marked host-specificity of this parasite, but that there is also an organ specificity. The species occurring in the chicken are specific for the chicken, being incapable of infecting any other host, and are also found in a rather circumscribed area of the intestinal tract of the infected host. Even those forms which may be found in the same location in the intestinal tract evoke, in many instances, a response which is specific for that species. Thus the great contribution of Tyzzer was not only the demonstration of the multiplicity of species which are involved in this disease, but also the marked host and organ specificity of each species.

Of the coccidia which are found in the turkey Tyzzer (1927, 1929) described two species, Eimeria meleagridis and Eimeria meleagrimitis, which were found in the cecum and small intestine respectively. However, two statements made by him at that time indicate that little was known concerning these organisms. These were as follows: "No exact information is available concerning the pathogenicity of these two species of Eimeria, which occur in the turkey", and

"Coccidiosis in the turkey requires further investigation, for not only are we poorly informed as to the tissue reactions concerned, but a great variation in the size of the oocysts in recently obtained material suggests the occurrence in the turkey of forms of Eimeria other than those here described". The status of coccidiosis in the turkey at the time these investigations began was the same as that noted by Tyzzer (1929).

Cole and Hadley (1908) and Cole and Hadley (1910) considered blackhead in turkeys to be caused by a coccidium, Eimeria avium. It was unfortunate that they confused the etiological agents of these two diseases. However, from their descriptions it may be seen that they were dealing with at least three species of coccidia, namely E. meleagridis, E. meleagrimitis and probably E. dispersa as well as Histomonas meleagridis, the latter being considered to be a stage in the life history of the coccidium. A further review of the early work on coccidiosis, before that of Tyzzer (1929) would seem to be out of place here. Previous to 1929 all the numerous species of coccidia now known to occur in birds were assumed to be one, E. avium, with references being made interchangeably to different species of avian hosts, making interpretation difficult as well as doubtful.

Tyzzer's (1929) description of the species of coccidia occuring in the turkey are very meager. E. meleagridis Tyzzer, 1927 occurring in the cecum was first observed by Johnson (1923), but was not at that time regarded as a dis-

tinct species. However, he did observe that oocysts from the chicken would not infect turkeys. In the description of this species Tyzzer (1929) had only scant material available for morphological study. However, he did notice that the surface rather than the glandular epithelium was invaded, and that the parasitized cells showed no marked reactions to infection, but were gradually distended by the parasite which lay superficial to the nucleus. The schizonts were rather large and showed on division a residual mass more than one third the volume of the parent parasite. The merozoites were large and of a rather thick form. Five days were required for the passage of oocysts. He observed no evidence of pathogenicity in this species.

Steward (1947) infected two young chickens with E. meleagridis and reported that the chickens became infected and passed oocysts similar to those found in the turkey. Turkeys were subsequently infected with oocysts obtained from the chickens. Due to the general lack of confirmation of this statement, it cannot be accepted at the present time without more adequate investigation.

E. meleagrimitis Tyzzer, 1929 occurs in the small intestine of the turkey. This organism agrees very closely morphologically and in distribution with E. mitis in the chicken. Development occurs throughout the small intestine, although Tyzzer found it more commonly in the posterior portions. The organism did not occur in colonies, but was distributed throughout the epithelium of the villi. They pene-

trated rather deeply into the epithelium, and large numbers of the coccidia were beneath the cell nucleus, which was often pushed toward the surface, the nucleus occasionally being turned with its long axis lying transversely. Some organisms, however, were superficial to the cell nucleus. The invaded cells showed no noticeable change in volume or displacement. Oocysts were passed in the droppings of infected poults six days after infection. Tyzzer presented little evidence for the pathogenicity of this species. Of the large number of turkey poults infected, the death of only two could be attributed to this species.

The above two species were the only ones in turkeys described by Tyzzer. However, he did describe E. dispersa Tyzzer, 1929 from the Bob-white quail (Colinus virginianus virginianus) which was capable of infecting the turkey. This species was differentiated from all others occurring in gallinaceous birds by the absence of a well defined polar inclusion body in the oocyst. It developed in the small intestine, and to a limited extent in the constricted portion of the ceca, with the heaviest infections in quail occurring in the duodenum. The epithelium of the villi rather than the glands was infected by this species, and there appeared to be no characteristic cell reaction. The developing forms occupied all available space, both above and below the host cell nuclei. In addition to the more common type of schizont, producing sausage shaped merozoites arranged in a compact clump with their long axes more or less

parallel, a larger schizont was found, containing more numerous, smaller, ovoidal merozoites. Although the possibility was considered that this represented another species, he concluded that it was a stage in the life cycle of E. dispersa. Four days were required for the passage of oocysts. It was also found that this species possessed less immunizing power in the quail than any of the other species occurring in gallinaceous birds. Repeated infections did not result in a resistance of quail to this species. Tyzzer was unable to determine the pathogenicity of this species in quail. He was able to infect turkeys, and oocysts were discharged five to six days after infection and appeared in the feces for a period of from one to three days. Very light infections were produced in the chicken, which discharged a small number of oocysts seven days after infection. The effects of this species on the turkey and chicken were not studied.

Morehouse (1949) observed that massive infections (100,000 to 600,000 sporulated oocysts) with E. meleagridis resulted in the death of 6.3 percent of 142 infected turkey poults. This author noted that general debility, inappetence and loss of weight followed infection. Peterson (1949) reported a mortality of from 70 to 90 percent in turkey poults infected with E. meleagrimitis.

Hawkins (1949) observed that E. meleagridis, E. meleagrimitis and E. dispersa are frequently present in the turkey, but that only E. meleagrimitis is to be considered a

serious pathogen. The latter form was capable of producing a mortality approaching 100 percent in turkey poults two to three weeks of age. Although the data presented in this paper were limited, there was an indication of a marked resistance in older birds to this species. In addition to a high mortality, there was a marked drop in feed consumption and severe weight losses occurred in infected birds.

Little work has been carried out on the effects of chemotherapeutic agents against coccidiosis in turkeys. Peterson (1949) has determined the minimal prophylactic level of five sulphonamides administered in the water as follows: sulfaquinoxaline (0.005 percent), sulfapyrazine (0.005 percent), sulfachlorodiazine (0.01 percent), sulfamerazine (0.02 percent) and sulfamethazine (0.02 percent). The minimal level for sulfaguanidine in the mash was 0.1 percent. He further demonstrated that test birds showed complete survival when treatment was begun as late as 96 hours after infection and was continued for four days or longer. These compounds were presumably tested against E. meleagrimitis. Morehouse (1949) observed that four inorganic arsenicals, arsenic trioxide, ammonium arsenate, lead arsenate and copper arsenate, were highly effective for the control of E. meleagridis infections, but were toxic at the levels used. He further observed that sodium 4-chlorophenyl-arsonate was effective, and that 3 nitro-4-hydroxyphenyl-arsonic acid showed some value for the control of

E. meleagridis infections. However, none of the arsenicals gave the same high degree of protection as noted in the sulphonamides, such as those mentioned above.

MATERIALS AND METHODS

Source and handling of materials

The coccidia used in this study were obtained from all sections of the United States. Vials of 2.5 percent potassium dichromate solution were sent to poultry pathologists who were cooperating in this study. When they received turkeys which were infected with coccidia they placed the contents of the affected areas into the vials and immediately forwarded them, along with pertinent data concerning the infection, to the Department of Bacteriology and Public Health, Michigan State College, East Lansing, Michigan. When this material was received in the laboratory it was centrifuged, washed and suspended in fresh 2.5 percent potassium dichromate, placed in petri dishes to a depth of five or six millimeters and maintained at room temperature for one or two weeks. It was later found that sporulation was facilitated if the material was placed in a 125 ml. flask and connected to an aquarium aspirator.

As soon as possible after the receipt of infective material, usually within a month, the oocysts were administered to coccidia free turkey poults. The birds were from two to three weeks of age when used for this purpose, and four to five poults were used for each strain received. They were killed five, six and seven days after infection. When killed early on the fifth day following infection pure cultures of E. meleagridis occurring in the

cecum could be obtained. Birds killed on the sixth and seventh days following infection with unknown cultures might contain all four species occurring in the turkey. On these days material from the duodenum, small intestine and cecum was removed separately, washed and placed in flasks and the oocysts sporulated. The lesions occurring in the small intestine and cecum were noted.

In the material which established infections in the small intestine it was noted that two distinct types of lesions were produced. One of the samples received from Massachusetts was a pure culture. At first it was designated as #532 and later proved to be a pure culture of E. meleagrimitis. With this material most of the work on this species was carried out. A second sample, also from Massachusetts, likewise produced a distinctive type of lesion, and was at first designated as #583. On examination this appeared to be a mixed culture, so single oocysts were isolated by means of a Chambers micromanipulator, administered to coccidia free poults, and the oocysts obtained in this manner proved to be those of E. dispersa. Most of the work with this species has been carried out with this material. A third species was isolated by administering unidentified oocysts to turkeys which had been immunized against E. dispersa, E. meleagrimitis and E. meleagridis. The birds became infected and the oocysts proved to be a new species which was named E. gallopavonis.

Rearing Birds and Infection Experiments

The most difficult thing to control in studies of this type is accidental infection of the experimental birds. With the facilities available it was not possible to maintain all the birds in the same building, and have any assurance that they would remain free of infection. Therefore, the birds were kept in three separate buildings, and they were cared for by different individuals. The feed and equipment for each group was maintained separately.

Bronze turkeys were used throughout the course of this study, and they were obtained as day old poults. During the spring of 1949 fifty birds were received each week for eight weeks. Two hundred poults used for screening samples were handled in a similar manner in 1948. These were immediately placed in brooders in the basement of the veterinary hospital where turkeys and chickens had not been kept for over a year. They were fed a 26 percent protein commercial (Arcady) turkey mash in which was incorporated 0.2 percent sulfaguanidine to prevent any possibility of infection with coccidia. No birds from this area were ever found to be infected with coccidia. Slightly abnormal losses occurred in one lot of poults and they were found to have Newcastle disease (We wish to thank Dr. C. H. Cunningham for making the virus isolation). Following this, all the poults were vaccinated when received and losses ceased.

When the poults were two weeks of age they were removed to another building for infection experiments. Those birds

to be infected with E. meleagrimitis were placed in batteries in the basement of the Bacteriology Annex, those infected with E. dispersa were placed in batteries on the second floor of the same building, and E. meleagridis infections were carried out on the second floor of the diseased poultry building. All birds were kept for two days on unmedicated mash before they were infected.

All infections were oral and consisted of administering known numbers of oocysts, predetermined by a dilution count. As far as possible, infections were carried out between the hours of 8:00 and 12:00 A.M. and the exact time of infection was noted. For pathological examinations and life history studies the birds were killed at six and twelve hour intervals, up to eight days after infection. A complete post-mortem examination was made, including a microscopic examination of all parts of the intestinal tract. A number of specimens were submitted to the Poultry Pathology Clinic for Salmonella pullorum examinations, but all proved negative.

In the routine infection experiments the poults were kept in the same cages, which were cleaned daily. However, in the experiments for testing the development of immunity the birds were moved daily to a clean cage.

The pheasants and Hungarian partridges were obtained from the State Game Farm, Mason, Michigan through the courtesy of the Michigan Department of Conservation. The Bob-white quail were purchased from a licensed quail farm near Kalamazoo, Michigan. These birds were maintained mostly on a scratch feed of cracked corn, oats and barley.

Technical procedures

Counting of oocysts was carried out as described by Dunlap, Hawkins and Nelson (1949). Counts were made in all cases from composite fecal samples of four or five poults. In counts of E. dispersa and E. meleagrimitis small intestinal droppings were used, for E. meleagridis cecal droppings were utilized.

The measurement of all oocysts was made under oil immersion with a 91X objective and a Leitz filar micrometer.

For histological examination, unopened sections of the duodenum, the jejunum near the yolk stalk, the ileum near the tip of the ceca, the dilated portion of each cecum, and the rectum posterior to the ceca were placed in Bouin's fixative. These were fixed for 24 hours, washed, dehydrated in alcohol and infiltrated with paraffin ("Tissuemat") in the Autotechnicon. The tissues were embedded in paraffin in the usual manner. Sections were cut 7 microns thick, and stained in both haematoxylin and eosin, and Heidenhain's hematoxylin. The former staining was carried out in the Autotechnicon, and the latter was done in staining dishes, using the 24 hour procedure. The only difference from the standard technique was that the tissues were differentiated with a saturated solution of picric acid.

Smears were made of the intestinal mucosa, dried, fixed in absolute methyl alcohol and stained with Giemsa.

Drawings were made with a camera lucida and all are

drawn to the same scale. Photographs were made with a Bausch and Lomb H camera, and were taken at various magnifications.

Eimeria dispersa Tyzzer, 1929

SOURCE

This species was originally described by Tyzzer (1929) from the Bob-white quail. All the material used in this study was obtained from turkeys. This species has been recovered from material received from Connecticut, Iowa, Massachusetts, Michigan, Minnesota, Missouri, Nebraska, North Dakota, New Jersey, Oregon, and Utah. Two strains, one obtained from Harrison County Missouri through the courtesy of Dr. G. N. Snoeyenbos have been studied more thoroughly than the others.

HOSTS

In addition to describing this species from the Bob-white quail (*Colinus virginianus virginianus*) Tyzzer (1929) was able readily to transmit this infection to the turkey, and occasionally he obtained a light infection in the chicken. In addition, he observed natural infections in the pheasant which he believed to be caused by *E. dispersa*, although he doubted that he could transmit the quail strain to the pheasant.

Four mature Hungarian partridges (*Perdix perdix perdix*) were obtained from the Mason Game Farm of the Michigan Department of Conservation. Oocyst counts were made on these birds for two weeks prior to infection, and although they were already infected it was at a level of less than 100 oocysts per gram of feces. They were infected with 100,000 sporulated oocysts of a turkey strain of *E. dispersa*. Six

days later oocysts of this species were found in the feces and they reached a peak of 286,400 oocysts per gram of feces eight days after infection, after which the numbers declined rapidly until by the eleventh day they had practically disappeared. Reinfection three weeks after the original infection showed that the partridges were completely resistant to further infection with this species. This is the first time that this host has been recorded for E. dispersa. The course of this infection is illustrated in Figures 1 and 2.

OOCYST

The oocysts of the turkey strain of E. dispersa were passed in the feces of infected turkeys, Hungarian partridges and Bob-white quail late on the fifth day of infection or on the sixth day. Tyzzer (1929) observed that the quail strain of E. dispersa had a prepatent period of four days in quail, five days in the turkey and pheasant and seven days in the chicken.

The oocysts passed in the feces measured 21.8 to 31.1 microns in length (average 26.07 microns) (see Figure 5) and 17.7 to 23.9 microns in width (average 21.04 microns). These dimensions are based on the measurement of 100 oocysts. The oocyst is broadly ovoidal, with a length breadth ratio of 1.23 (see Figure 6). The cyst wall does not present the double contoured appearance which is observed in the oocysts of the other species occurring in the turkey. In contrast, the cyst wall has only an inner dark refraction line (see Plate V-A, B, C and D).

FIGURE 1.

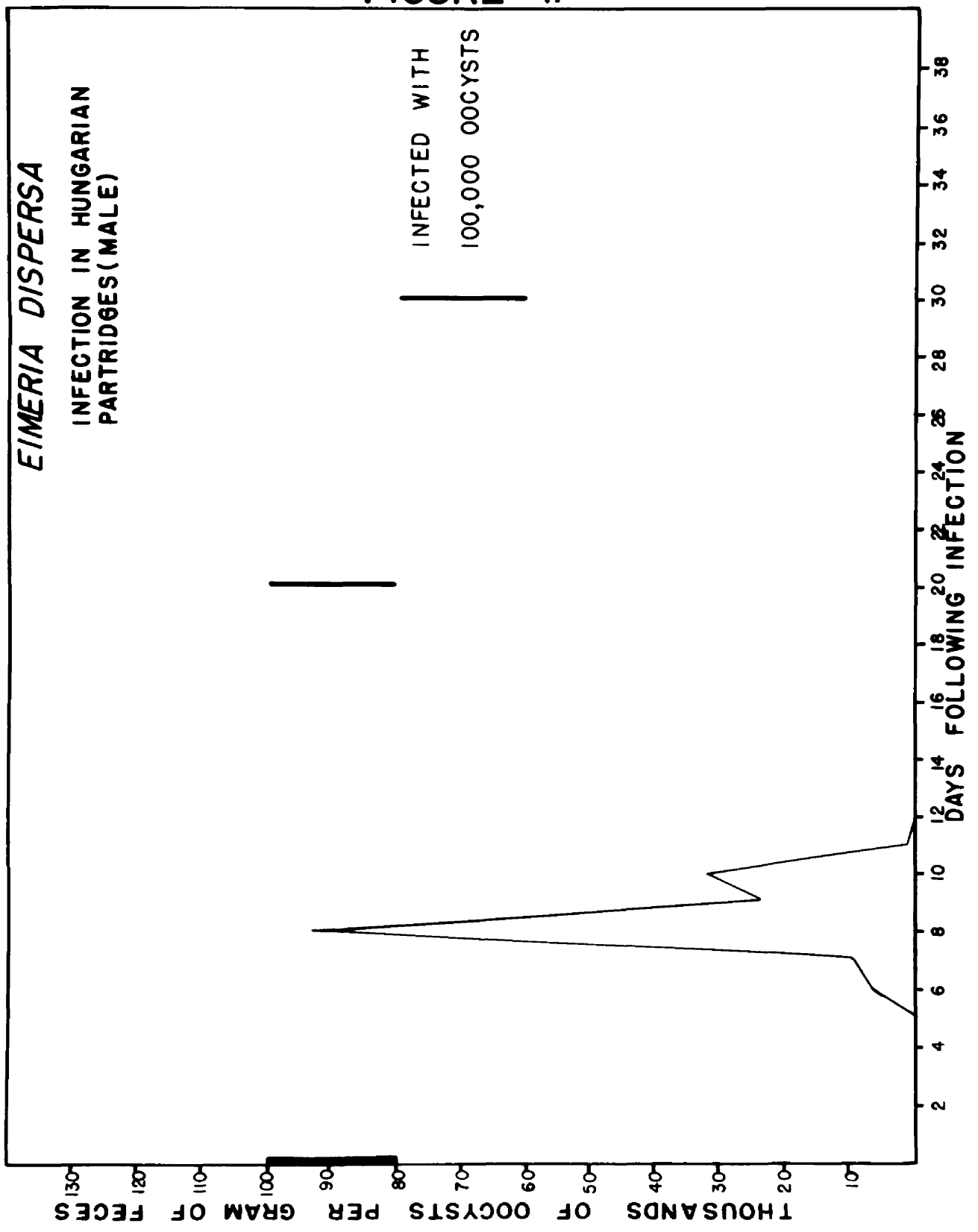

EIMERIA DISPERSA
INFECTION IN HUNGARIAN PARTRIDGES (MALE)
INFECTED WITH 100,000 OOCYSTS
THOUSANDS OF OOCYSTS PER GRAM OF FECES
DAYS FOLLOWING INFECTION

FIGURE 2.

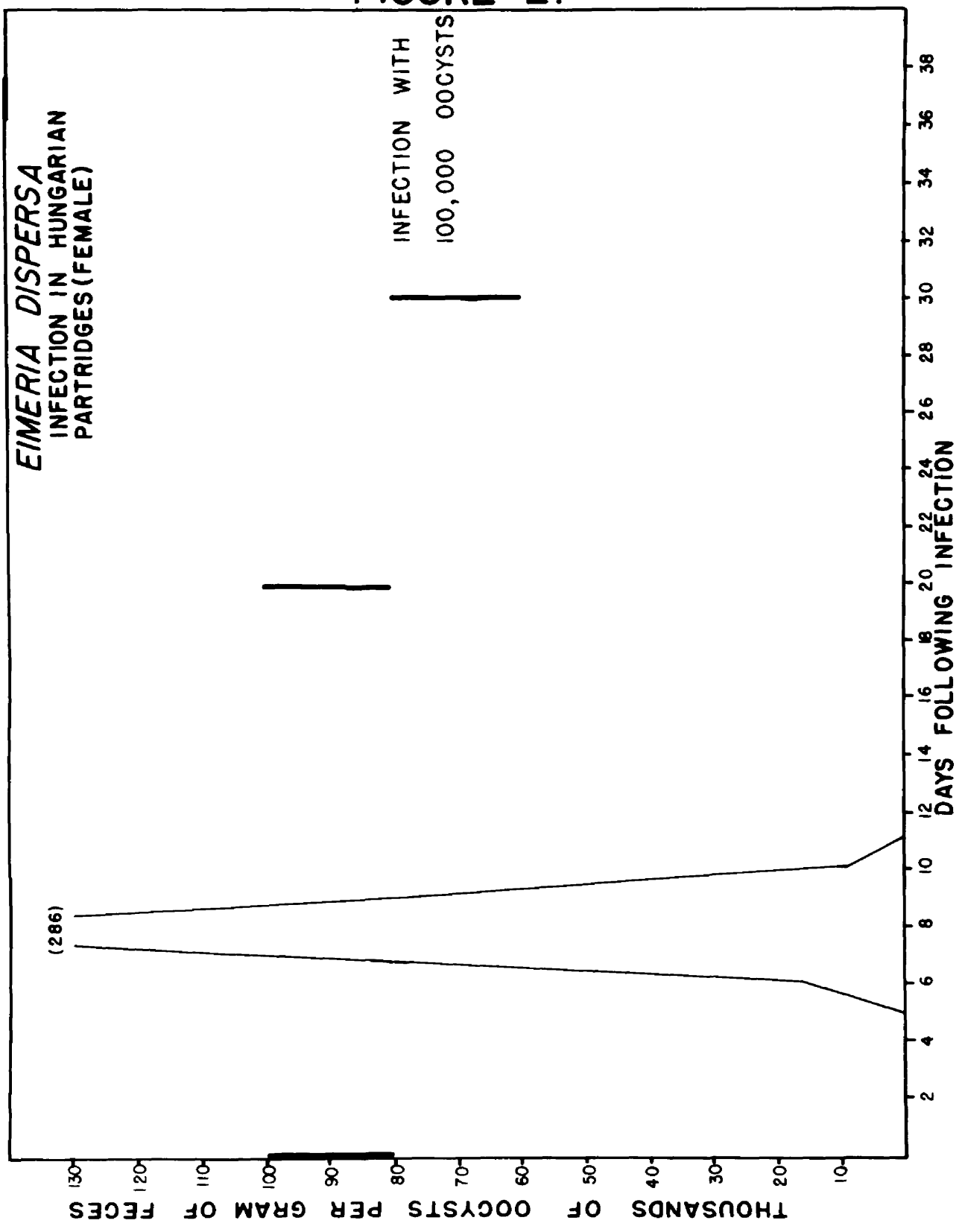

EIMERIA DISPERSA
INFECTION IN HUNGARIAN PARTRIDGES (FEMALE)
INFECTION WITH 100,000 OOCYSTS
(286)
THOUSANDS OF OOCYSTS PER GRAM OF FECES
DAYS FOLLOWING INFECTION

FIGURE 3.

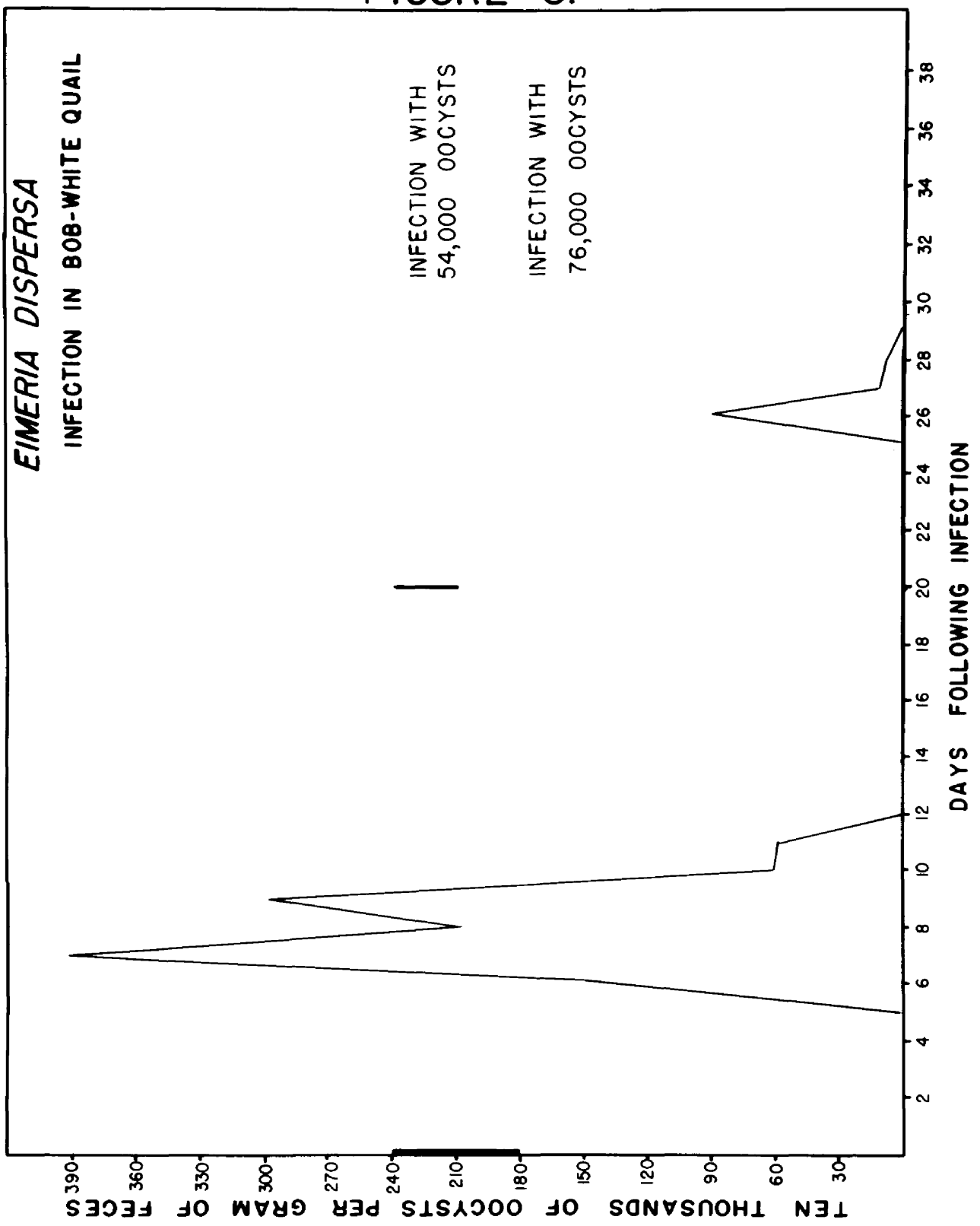

EIMERIA DISPERSA
INFECTION IN BOB-WHITE QUAIL
INFECTION WITH
54,000 OOCYSTS
INFECTION WITH
76,000 OOCYSTS
TEN THOUSANDS OF OOCYSTS PER GRAM OF FECES
30
60
90
120
150
180
210
240
270
300
330
360
390
DAYS FOLLOWING INFECTION
2
4
6
8
10
12
14
16
18
20
22
24
26
28
30
32
34
36
38

FIGURE 4.

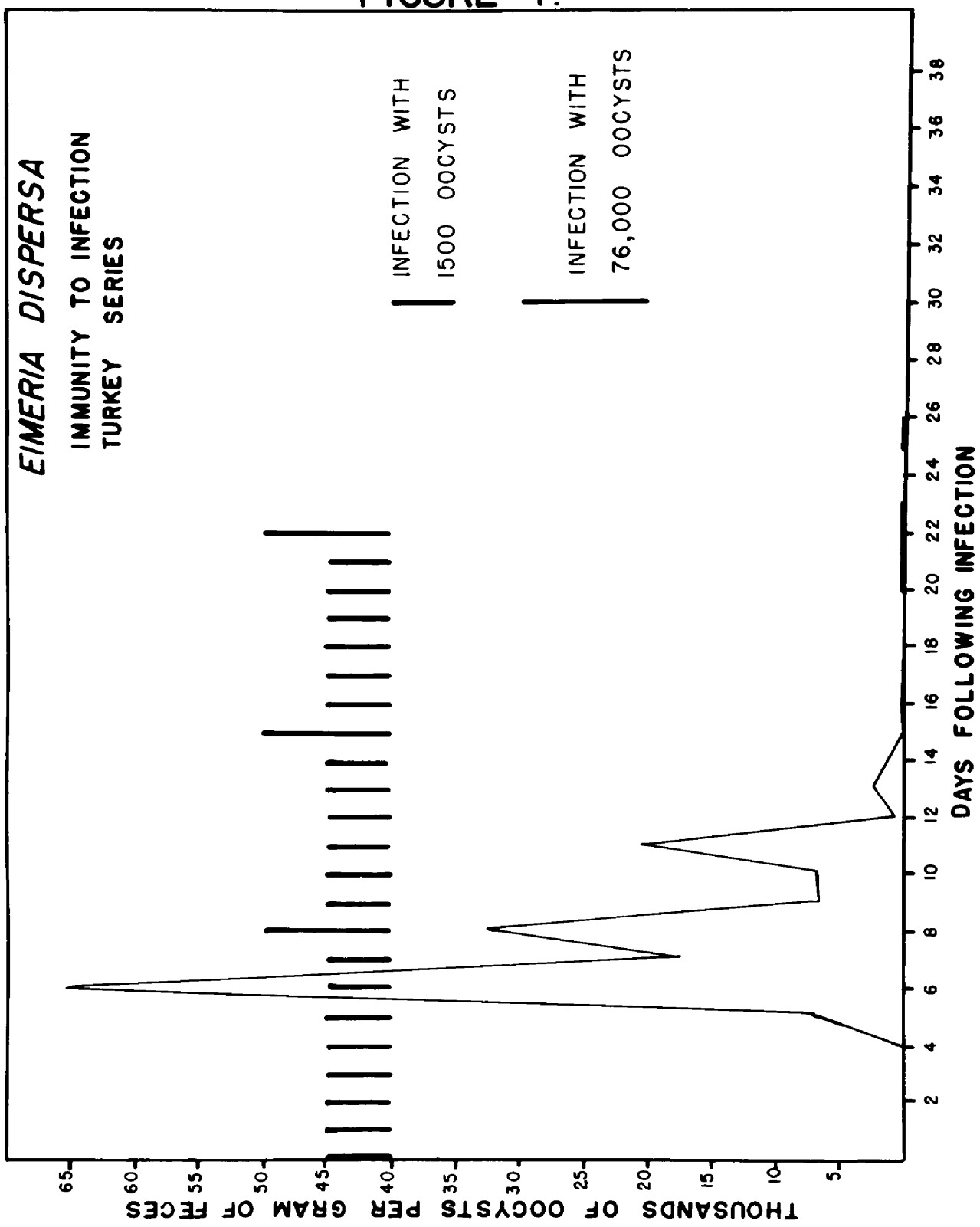
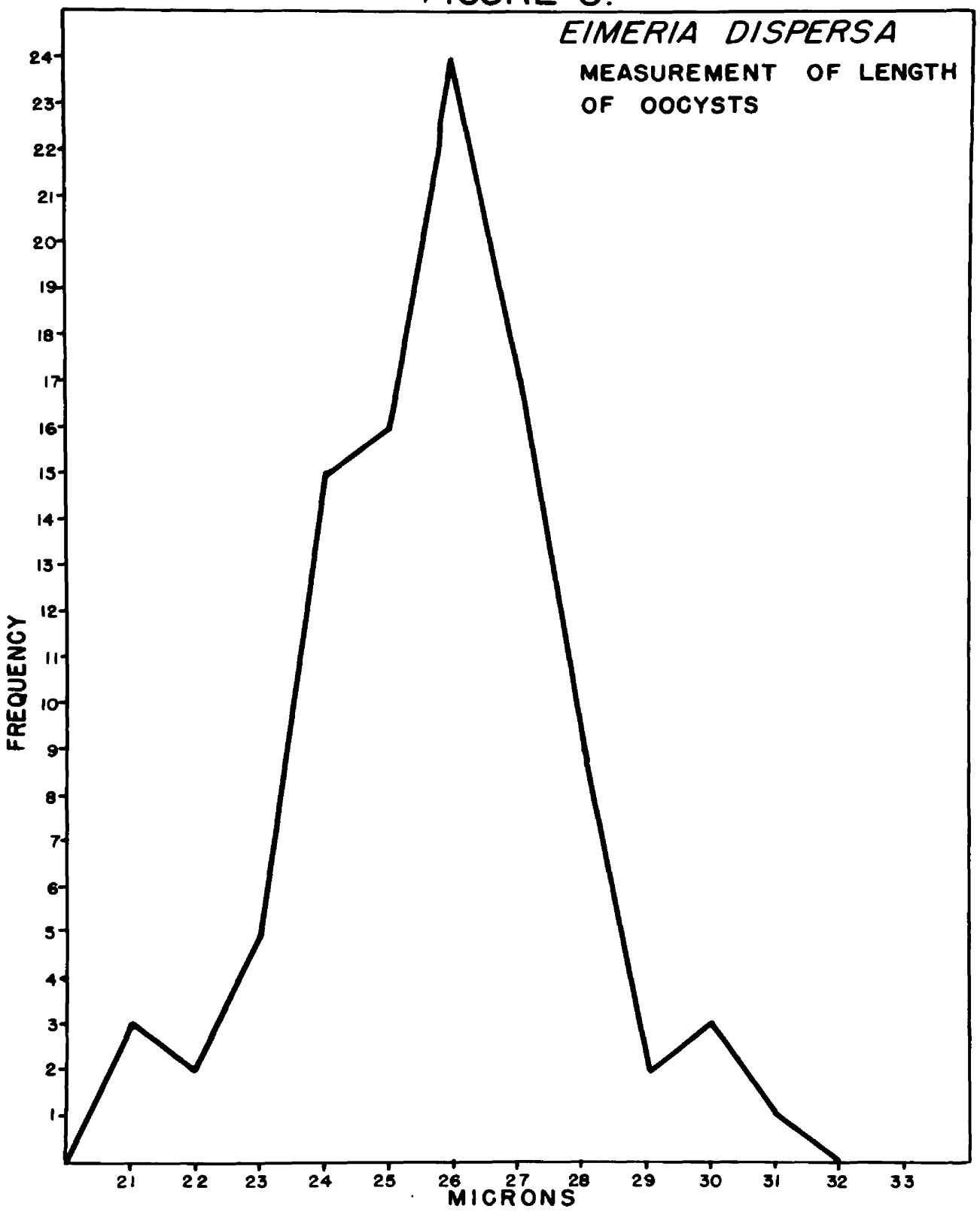

EIMERIA DISPERSA
IMMUNITY TO INFECTION
TURKEY SERIES
INFECTION WITH
1500 OOCYSTS
INFECTION WITH
76,000 OOCYSTS
THOUSANDS OF OOCYSTS PER GRAM OF FECES
DAYS FOLLOWING INFECTION

FIGURE 5.


EIMERIA DISPERSA
MEASUREMENT OF LENGTH OF OOCYSTS
FREQUENCY
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
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21
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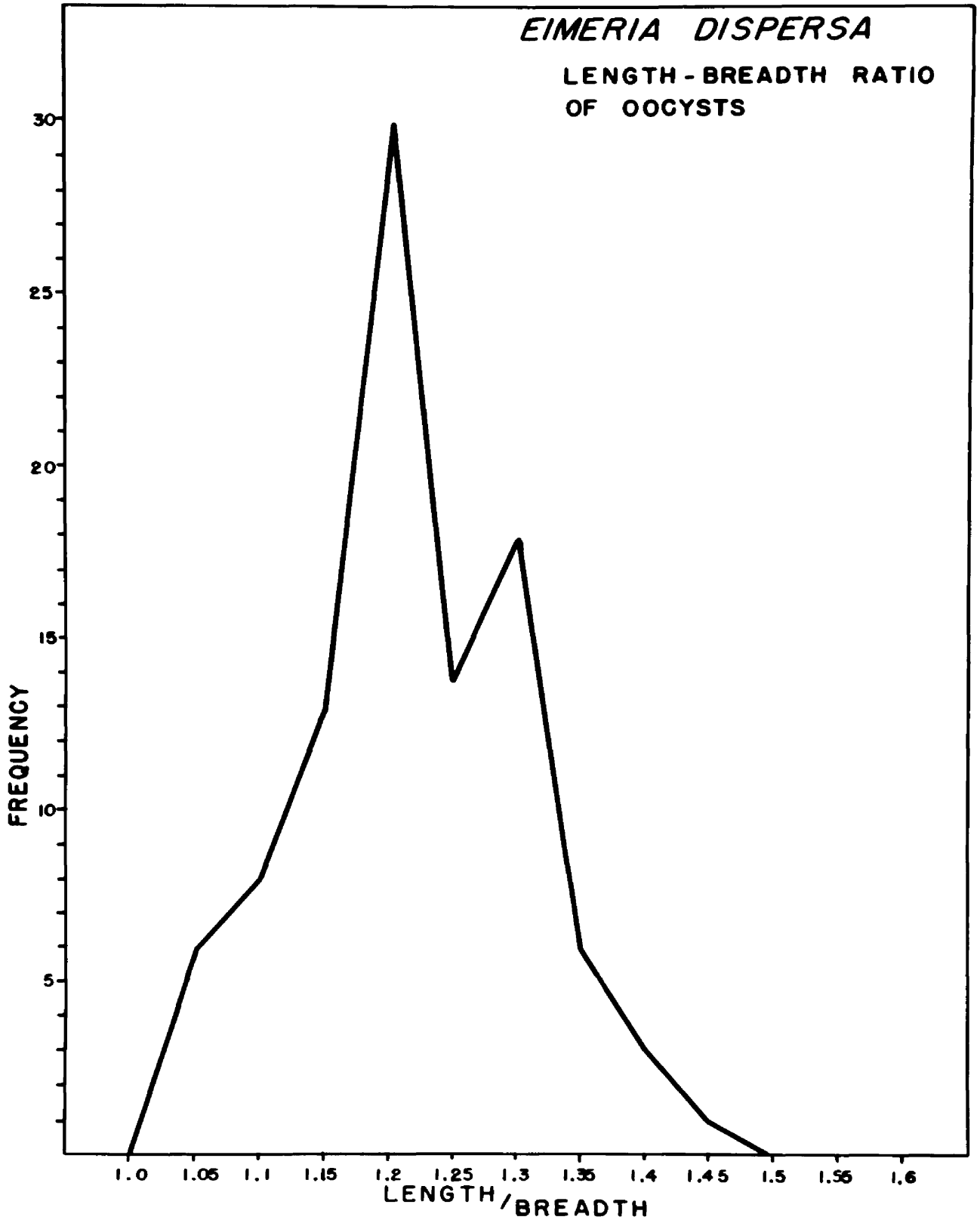
23
24
25
26
27
28
29
30
31
32
33
MICRONS

FIGURE 6.


EIMERIA DISPERSA
LENGTH - BREADTH RATIO
OF OOCYSTS
FREQUENCY
30
25
20
15
10
5
1.0
1.05
1.1
1.15
1.2
1.25
1.3
1.35
1.4
1.45
1.5
1.55
1.6
LENGTH/BREADTH

After the oocysts had been passed in the feces, approximately 48 hours were required for their sporulation. Oocysts which had been placed in 2.5 percent potassium dichromate at room temperature showed no evidence of sporulation 20 hours after their passage in the feces. At 32 hours, various stages of division were noted. At that time some oocysts had not divided, but the surface was very irregular (see Plate V-B). There were also numerous cysts in which the cytoplasmic mass had divided into four sporoblasts. In some of these the sporoblasts were round, whereas others assumed a triangular shape, and occasionally an almost square cell was observed, (see Plate V-C). Forty four hours after the oocysts were passed in the feces, sporocysts had been formed about the sporoblasts, but very few of the oocysts contained sporozoites. At fifty hours after passage in the feces, the oocysts were completely sporulated (see Figure 7-A Plate V-D). Polar granules or inclusions were never found in this species.

TISSUE STAGES

The earliest stages were observed in the epithelial cells of the suodenum 24 hours after infection. These were rounded uninucleate bodies about two microns in diameter and were located superficially to the nucleus of the epithelial cell (see Plate I-A). Division of the nucleus was first noted in these forms 44 hours after infection. Very few of these were noted at this time, but one measured six microns in diameter and possessed four nuclei. Each nucleus was

found in a vacuole, and it was assumed that the delicate lining of this was the nuclear membrane. There was apparently some lag in the development of the schizonts, since at 54 hours after infection one, two and three nucleate stages were still observed (see Plate I-B). There are apparently two types of schizonts developing in infections with this species. The relationship between these two forms is not understood, but they do not represent a multiple infection, since the original infection of the material under study was derived from a single oocyst. Tyzzer (1929) observed similar forms of schizonts and also ruled out the possibility of multiple infections. The first type of schizont was small, measured about six microns in diameter and contained at the most 15 merozoites. These merozoites were approximately four to six microns in length and about one micron wide. They had completed their development two days and 18 hours after infection, were much more numerous than the larger form and may be seen in Plate I-D and E. The early schizont of the larger type may measure 13 microns in diameter without exhibiting any apparent division of the cytoplasm (see Plate I-C). The largest schizont containing merozoites was found two days and 18 hours after infection, and measured 24 by 18 microns. These large forms contained at least 50 merozoites and probably more. An accurate count of them was not possible, and their size closely approximated that of the merozoites found in smaller schizonts (see Plate I-E).

The first asexual generation was completed by the end of the second day of infection. At this time many of the first generation merozoites had been liberated, and were to be found rounded up as trophozoites in epithelial cells in the duodenum. These trophozoites measured one to two microns in diameter. They continued to develop and increase in size without any immediate nuclear division until some of them were five to six microns in diameter. Some of these uninucleate forms were found as late as three and one half days after infection, indicating a lag either in their development, or more probably in the liberation of the first generation merozoites. The second asexual generation was completed approximately four days after infection with the formation of 18 to 23 merozoites in each schizont. The schizonts measured 11 to 13 microns in diameter, and the contained merozoites were five to six microns long and one and one half to two microns wide (see Plate II and Plate III-A).

The third generation, which was predominantly sexual, was initiated by the penetration of new epithelial cells in the duidenum by the liberated second generation merozoites. As early as four days and six hours after infection, undifferentiated gametocytes could be observed. These contained a single nucleus consisting of a karyosome within a vacuole, and a finely granular cytoplasm. This form measured up to six or seven microns in diameter. The youngest macrogametocytes measured seven to eight microns in diameter

and the cytoplasm was filled with numerous small granules, each of which appeared to be present in a small vacuole (see Plate III-B). Five days after infection, the macrogametocytes had markedly increased in size, the granules were very distinct, and the vacuoles in which they were located had also enlarged (see Plate IV-A). A form which was slightly more developed was characteristic of this species and had not been noted occurring in any of the other species. This type of macrogametocyte measured 18 to 20 microns in diameter and presented a peripheral vacuolated appearance. The vacuoles were quite large and each contained a single granule. The cyst wall had begun to form, but was as yet very thin (see Plate IV-B). A still more advanced stage possessed a thicker cyst wall and the karyosome was located centrally in a finely granular, dark, dendritic mass (see Plate IV-D). The practically mature macrogamete is pictured in Plate IV-C.

The late microgametocytes are pictured in Plate III-C, and the microgametes in Plate III-D. The latter, when observed alive under phase illumination, were actively motile and possessed two flagella, each as long as the main body.

After fertilization, which has not been observed, the oocysts are passed in the feces late on the fifth, or on the sixth day following infection.

GROSS PATHOLOGY

The earliest lesions which were found in experimentally infected birds occurred in birds killed two days and 18 hours after infection. At this time the duodenum

was slightly cream colored when observed from the serosal surface. On opening the small intestine, there seemed to be no increased exudate, although scrapings demonstrated the presence of enormous numbers of merozoites in the duodenum. Three days and five hours after infection the entire small intestine was slightly dilated, and three days and 21 hours after infection there was a slight thickening of the small intestine and the villi were very distinct and stood out discretely to the naked eye. Four and one half days after infection the duodenum was markedly cream colored when viewed from the serosal surface; it was devoid of feed and was filled with a whitish mucus. The jejunum was congested and in several birds a slight amount of hemorrhage was noted on careful inspection. The mucosa of the ileum was white and slightly thickened.

Five days after infection the entire small intestine was grossly dilated, and the duodenum and first part of the jejunum were creamy white in color, as viewed from the serosal surface. On opening the small intestine it was found to be filled, particularly in the anterior portions, with a whitish-yellow, sticky, mucoid material. Late on the fifth day of infection large numbers of oocysts could be recovered from this material. On the sixth day of infection a similar appearance was noted and occasionally on opening the duodenum four to five centimeters of the intact epithelium would slip out. By the eighth day following infection the intestinal tract was practically normal on gross

Plate I - *Eimeria dispersa*

A. Trophozoites in epithelial cells of the duodenum 24 hours after infection.

B. Trophozoites and schizonts in cross sections of epithelial cells of the duodenum two days and six hours after infection.

C. Large schizont in epithelial cell of the duodenum two days and six hours after infection.

D. Small schizonts containing merozoites, and second generation trophozoites in epithelial cells of the duodenum two days and 18 hours after infection.

E. Small and large schizonts containing merozoites in epithelial cells of the duodenum two days and 18 hours after infection.

PLATE I

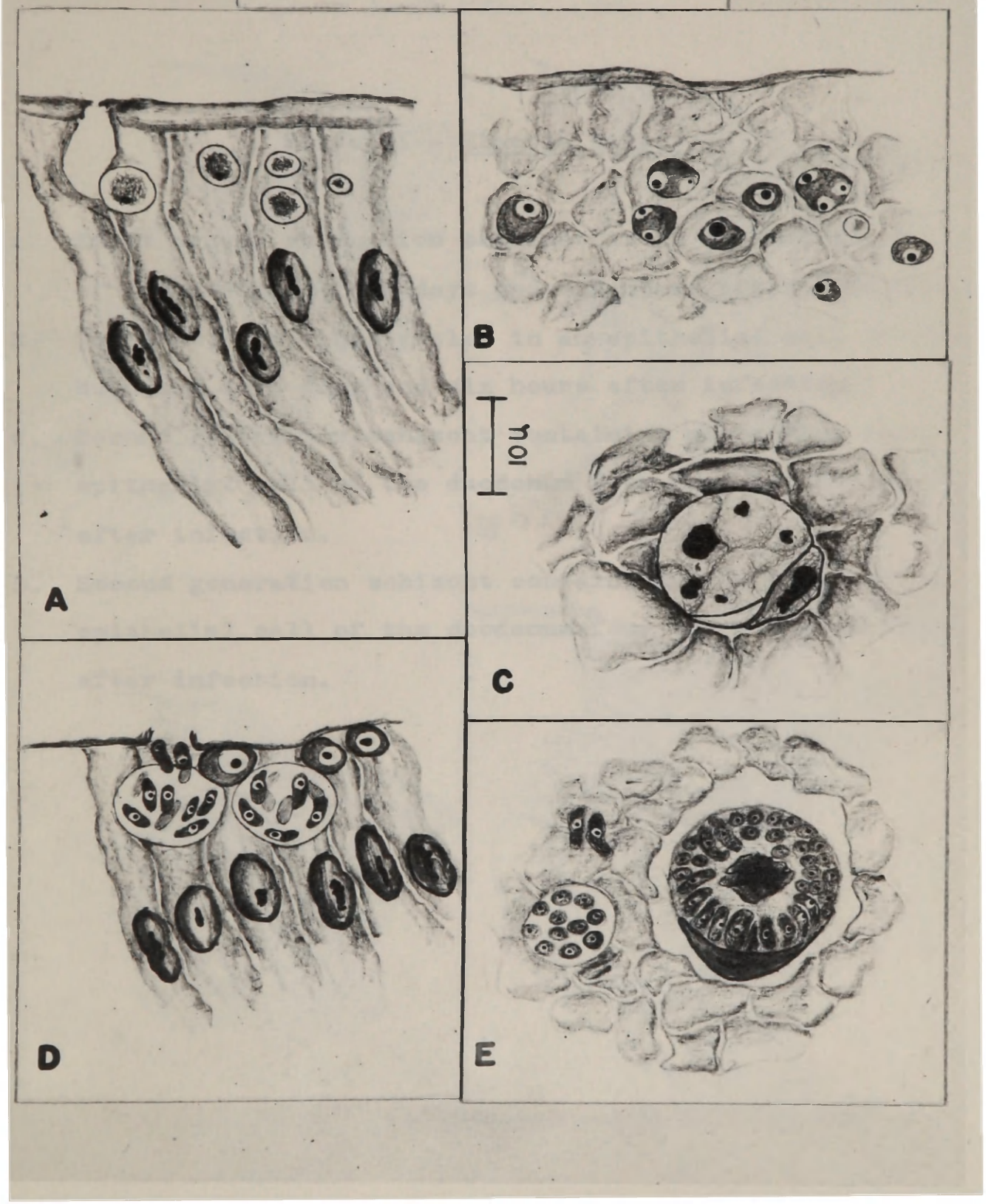


Plate II - *Eimeria dispersa*

A. Small second generation schizont in an epithelial cell of the duodenum four days and six hours after infection.

B. Schizont with eight nuclei in an epithelial cell of the duodenum four days and six hours after infection.

C. Second generation schizont containing merozoites in an epithelial cell of the duodenum four days and six hours after infection.

D. Second generation schizont containing merozoites in an epithelial cell of the duodenum four days and six hours after infection.

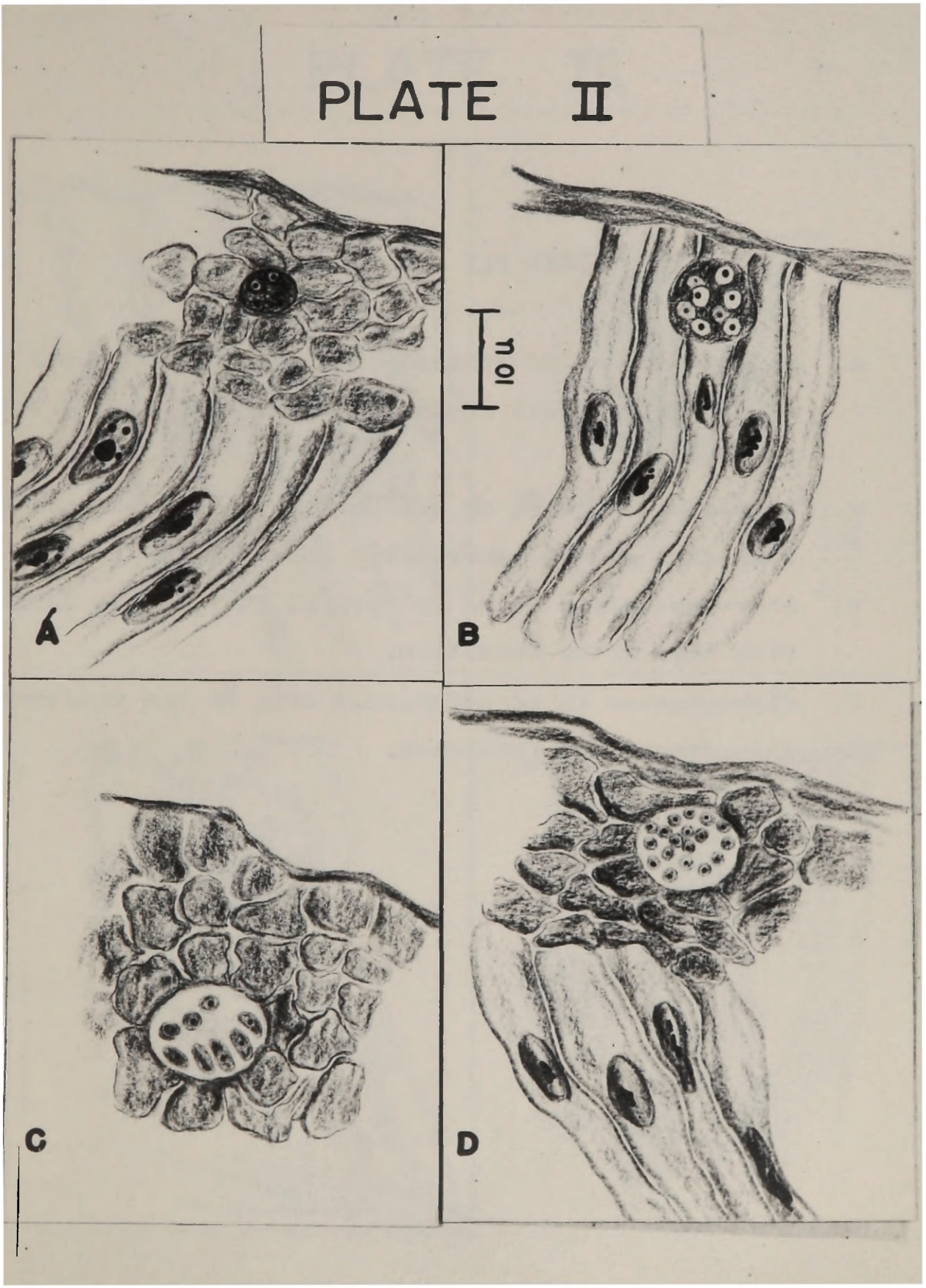


PLATE II
A
B
C
D
10u

Plate III - *Eimeria dispersa*

A. Second generation schizont containing merozoites in an epithelial cell of the duodenum five days after infection.

B. Macrogametocyte in an epithelial cell of the duodenum four days and 20 hours after infection.

C. Microgametocyte in an epithelial cell of the duodenum five days after infection.

D. Microgametes in an epithelial cell of the duodenum five days after infection.

PLATE III

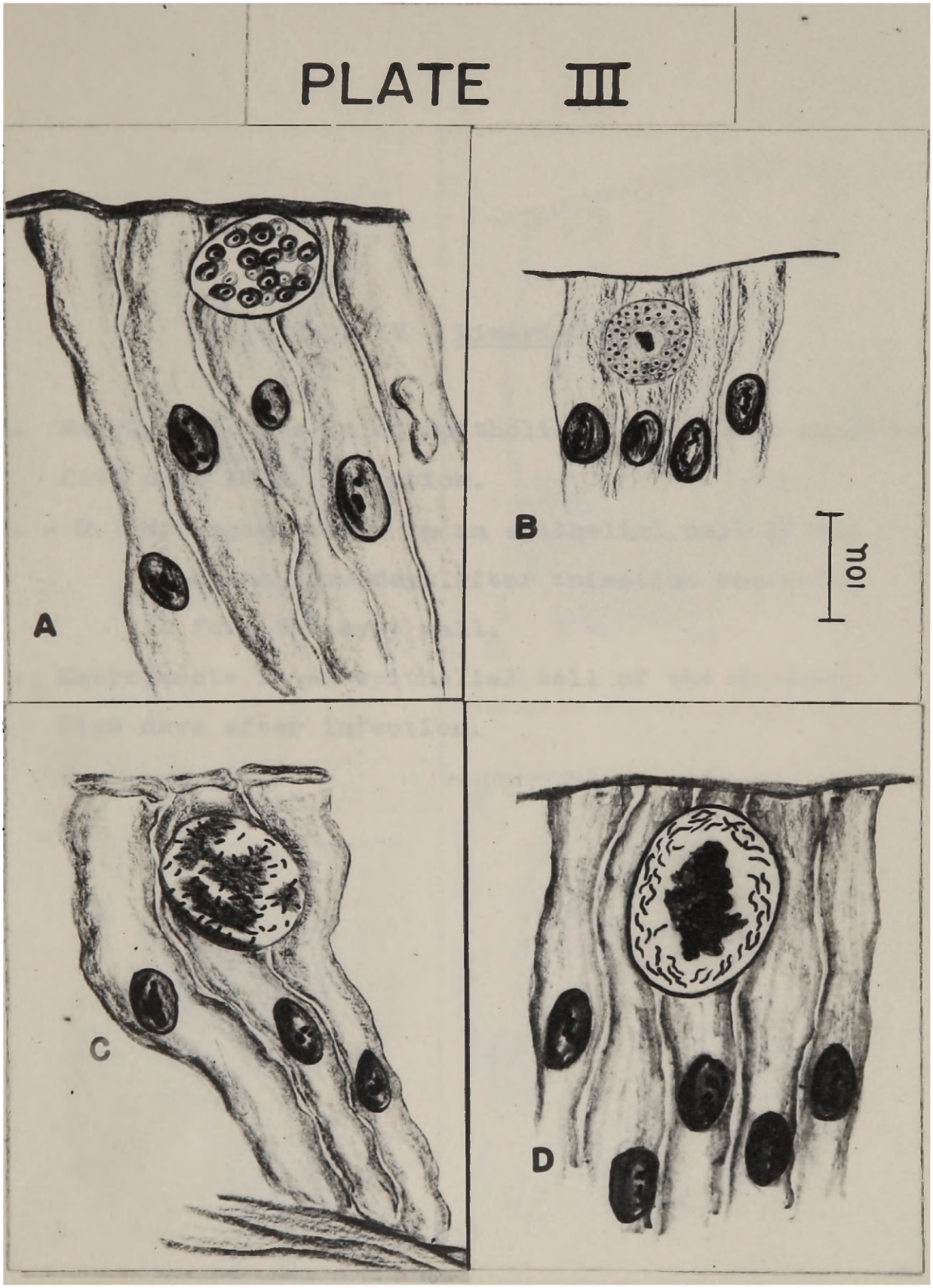


Plate IV - *Eimeria dispersa*

A. Macrogametocyte in an epithelial cell of the duodenum five days after infection.

B. - D. Macrogametocyte in an epithelial cell of the duodenum five days after infection beginning to form the cyst wall.

C. Macrogamete in an epithelial cell of the duodenum five days after infection.

PLATE IV

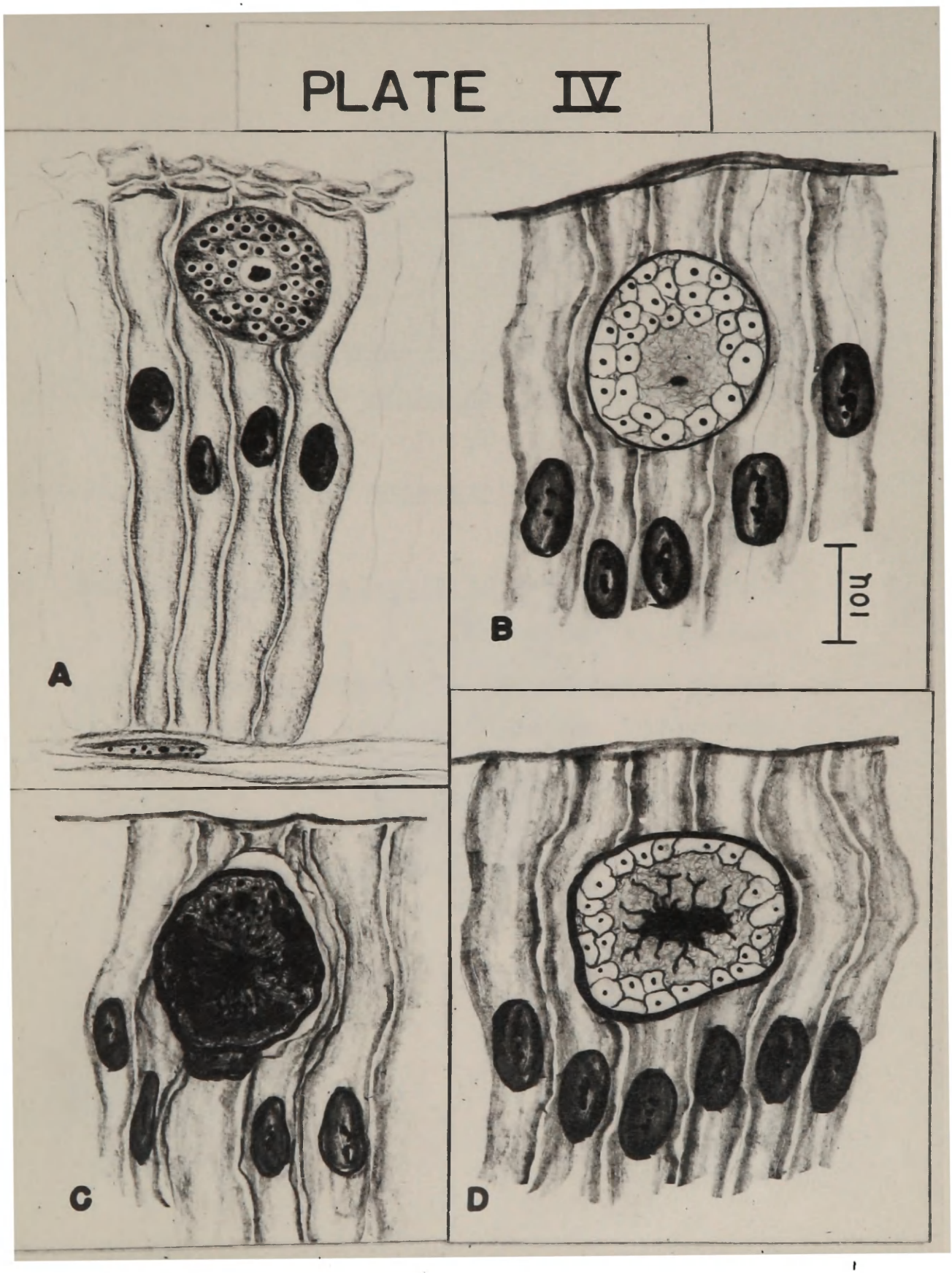


Plate V

A. Unsporulated oocyst of Eimeria dispersa.

B. Oocyst of Eimeria dispersa 32 hours after passage in feces.

C. Oocyst of Eimeria dispersa 32 hours after passage in the feces.

D. Sporulated oocyst of Eimeria dispersa 50 hours after passage in the feces.

E. Unsporulated oocyst of Eimeria meleagrimitis.

F. Sporulated oocyst of Eimeria meleagrimitis after passage in the feces.

(All drawings X1500)

PLATE V

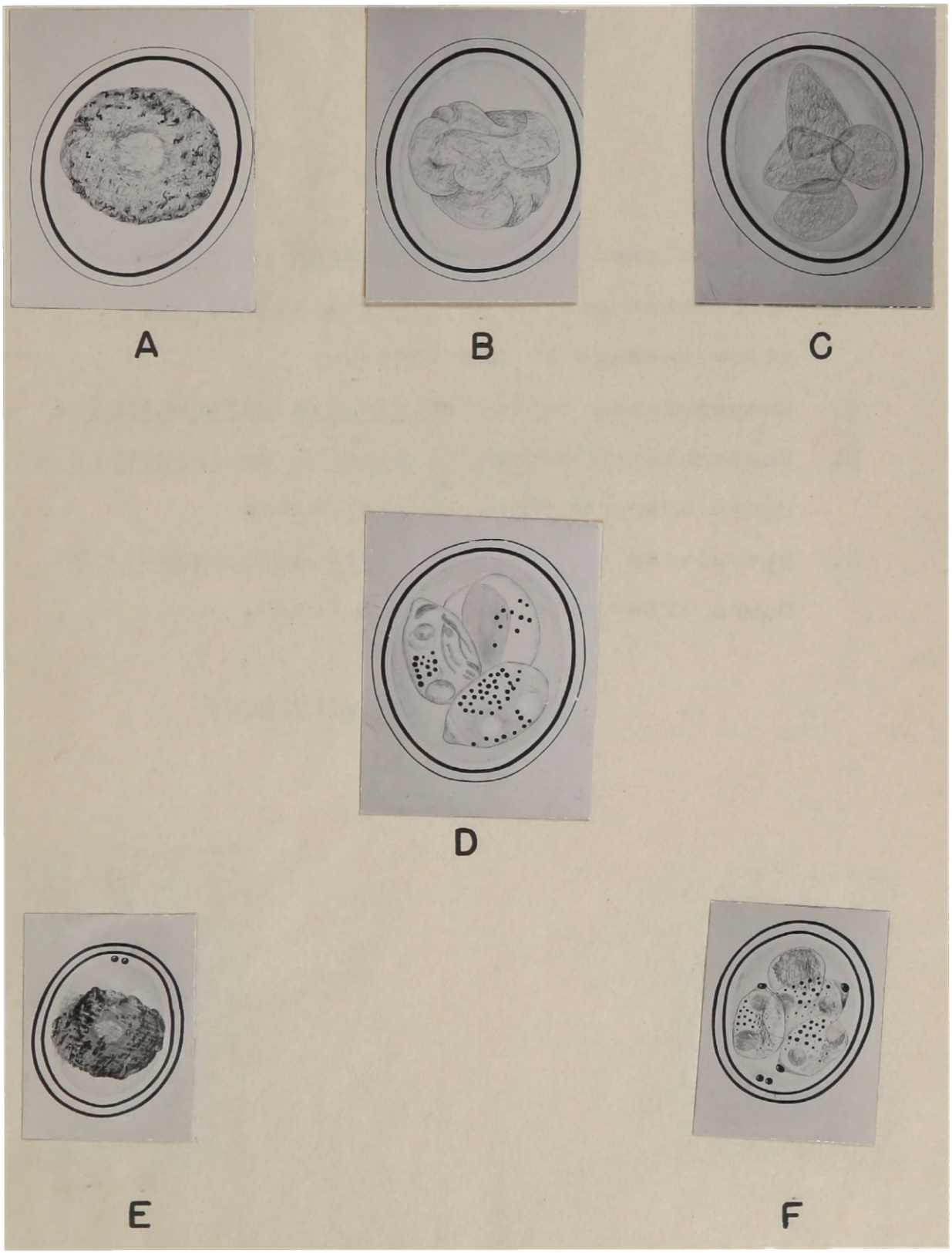


Plate VI

A. Unsporulated oocyst of Eimeria meleagridis.

B. Sporulated oocyst of Eimeria meleagridis 24 hours after passage in the feces.

C. Unsporulated oocyst of Eimeria gallopavonis.

D. Unsporulated oocyst of Eimeria gallopavonis 6 hours after passage in the feces.

E. Sporulated oocyst of Eimeria gallopavonis 24 hours after passage in the feces.

(All drawings X1500)

PLATE VI

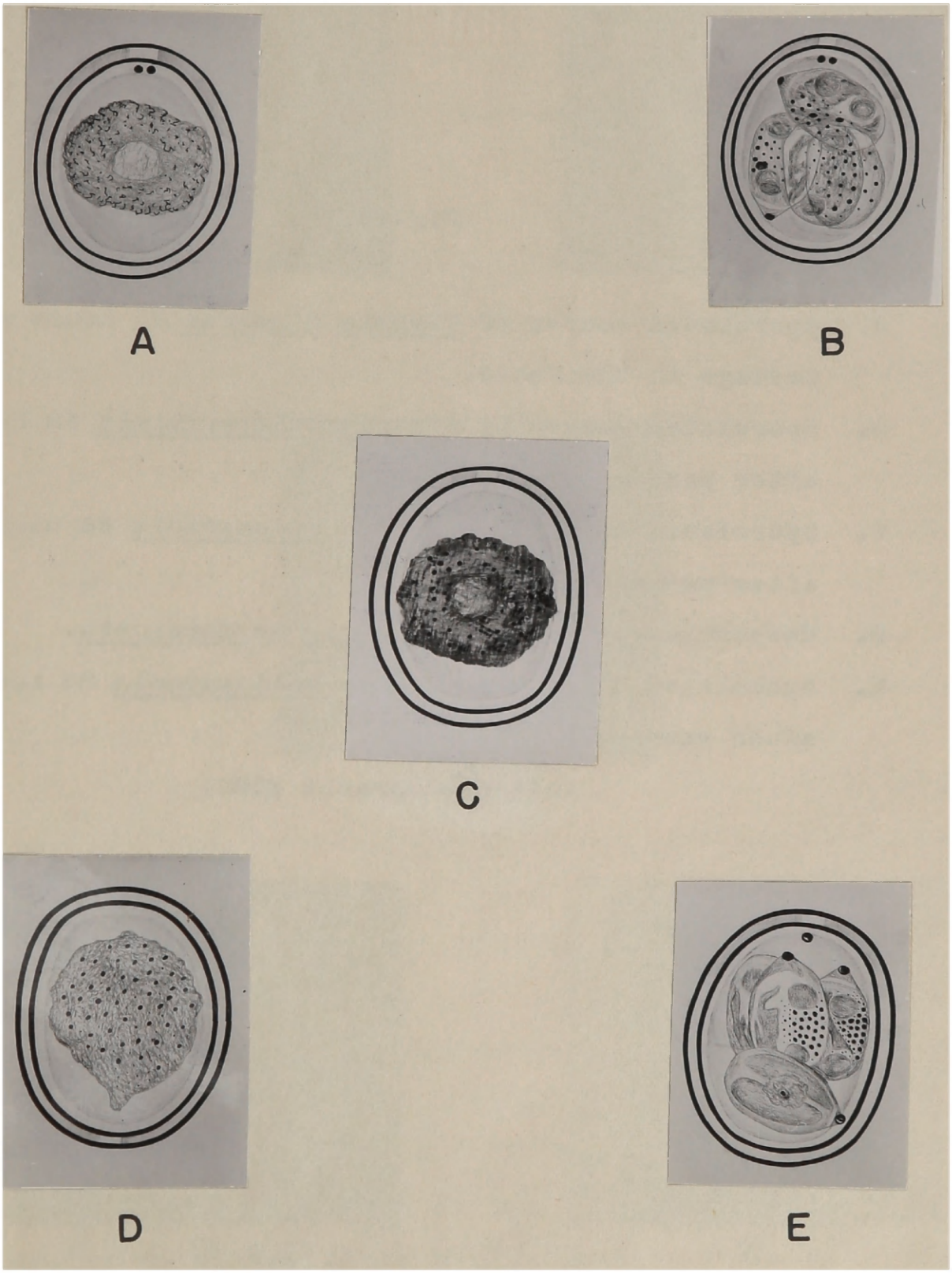


Figure 7

A. Sporulated oocyst of Eimeria dispersa 50 hours after passage in the feces.

B. Sporulated oocyst of Eimeria meleagrimitis 48 hours after passage in the feces.

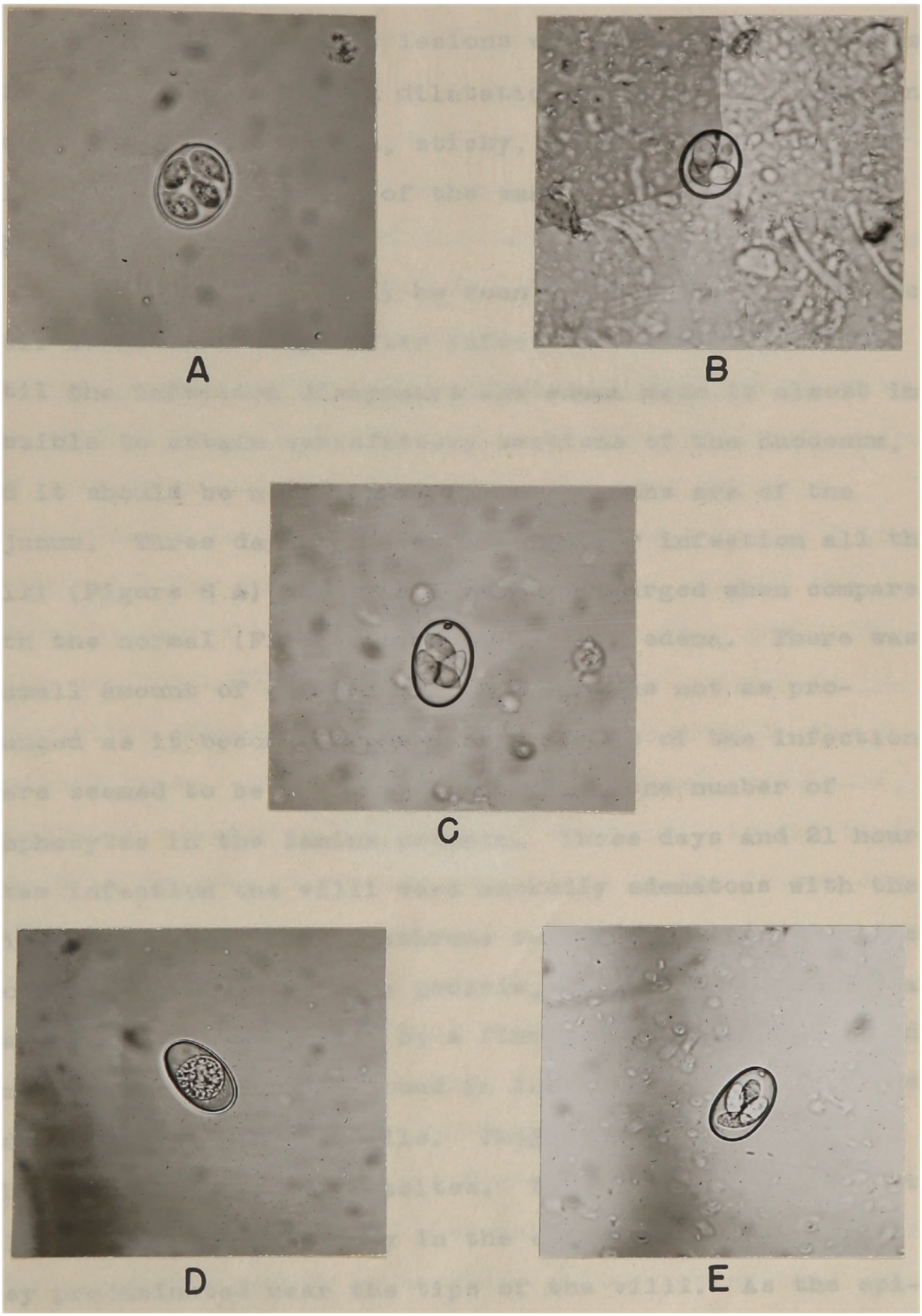
C. Sporulated oocyst of Eimeria meleagridis 24 hours after passage in the feces.

D. Unsporulated oocyst of Eimeria gallopavonis.

E. Sporulated oocyst of Eimeria gallopavonis 24 hours after passage in the feces.

(All photographs X620)

FIGURE 7



examination.

Thus, the main gross lesions which were noted in this infection were the marked dilatation of the small intestine and the creamy, yellowish, sticky, mucoid material which filled the anterior half of the small intestine.

HISTOPATHOLOGY

No marked changes may be seen in the intestinal mucosa until about three days after infection. From this time until the infection disappears the edema made it almost impossible to obtain satisfactory sections of the duodenum, and it should be noted that all photographs are of the jejunum. Three days and five hours after infection all the villi (Figure 8 A) were considerably enlarged when compared with the normal (Figure 15 A) due to the edema. There was a small amount of congestion, but this was not as pronounced as it becomes later in the course of the infection. There seemed to be a slight increase in the number of lymphocytes in the lamina propria. Three days and 21 hours after infection the villi were markedly edematous with the epithelium and basement membrane separated in many instances from the underlying lamina propria, either completely by a space or as in some areas by a fine fibrin network. At this time the parasites were found in large numbers in the lumen side of the epithelial cells. They were predominantly schizonts containing merozoites. These schizonts were not only located superficially in the epithelial cells, but they predominated near the tips of the villi. As the epi-

thelial cells of the crypts of Lieberkuhn approach the glands the number of parasites were reduced, and none have ever been observed in the glands. (Figure 8 B)

Four days and 20 hours after infection the villi were still very edematous, and there was marked congestion as shown by the dilatation of the capillaries. There was considerable necrosis in the distal quarter of the tips of the villi, which at this time contained schizonts and large numbers of gametocytes, most of which were superficial to the epithelial cell nucleus. The lymphocytic infiltration had been progressively increasing, and very few polymorphonuclear leucocytes were to be observed in the lamina propria. A few red blood cells were to be seen in the lumen, but there was no extensive hemorrhage. During the fifth day following infection the peak was reached in the destructive changes produced by these parasites in the intestinal tract. The villi were still markedly edematous, with pronounced congestion. However, the necrosis in the tips of the villi was not as pronounced as late on the fourth day of infection. There was a pronounced lymphocytic infiltration. The parasites were found in enormous numbers in the tips of the villi (Figure 9 A), but it was surprising that more of the epithelium was not sloughed off. Most of the coccidia observed at this time were gametocytes.

On the sixth day after infection the most remarkable observation was that there was a tendency for the villi to return toward a normal appearance. After noting the large

numbers of parasites present on the previous day one would have expected widespread sloughing of the epithelium, yet this did not occur. There was also a marked decrease in the amount of edema present. The lymphocytic infiltration was still pronounced.

Sections taken from the jejunum eight and nine days after infection showed a practically normal mucosa except for the persistence of a small amount of edema. However, the lymphocytic infiltration at this time was still marked (see Figure 9 B).

SYMPTOMS

This species is relatively non-pathogenic in the turkey and its pathogenicity in the Hungarian partridge and Bob-white quail was not studied. No marked symptoms were produced in experimental infections, except for a slight tendency to produce more liquid feces. There was a slight depression in weight gains (see Figure 10), but very little depression in feed (see Figure 11) and water consumption (see Figure 12).

IMMUNITY

There was a marked difference in the reaction to infection by the turkey and Hungarian partridge as compared with the Bob-white quail.

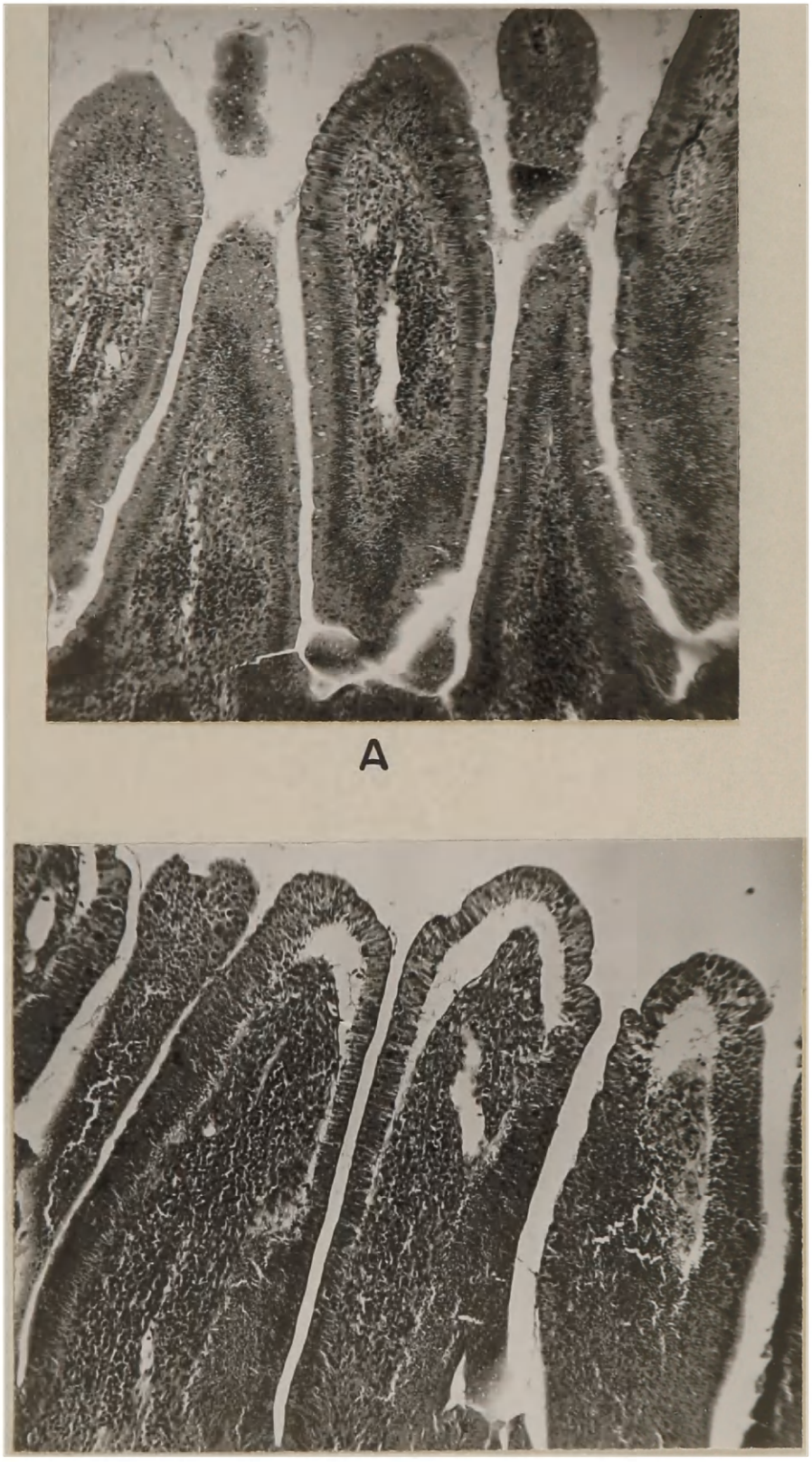
Nine three month old turkeys were infected with 1500 sporulated oocysts daily for eight days, and were then administered 76,000 sporulated oocysts. They were then given 1500 sporulated oocysts for another week, and again infected

Figure 8

A. Jejunum of two week old poult infected with Eimeria dispersa for three days and five hours (X77).

B. Jejunum of two week old poult infected with Eimeria dispersa for three days and twenty-one hours (X77).

FIGURE 8



A

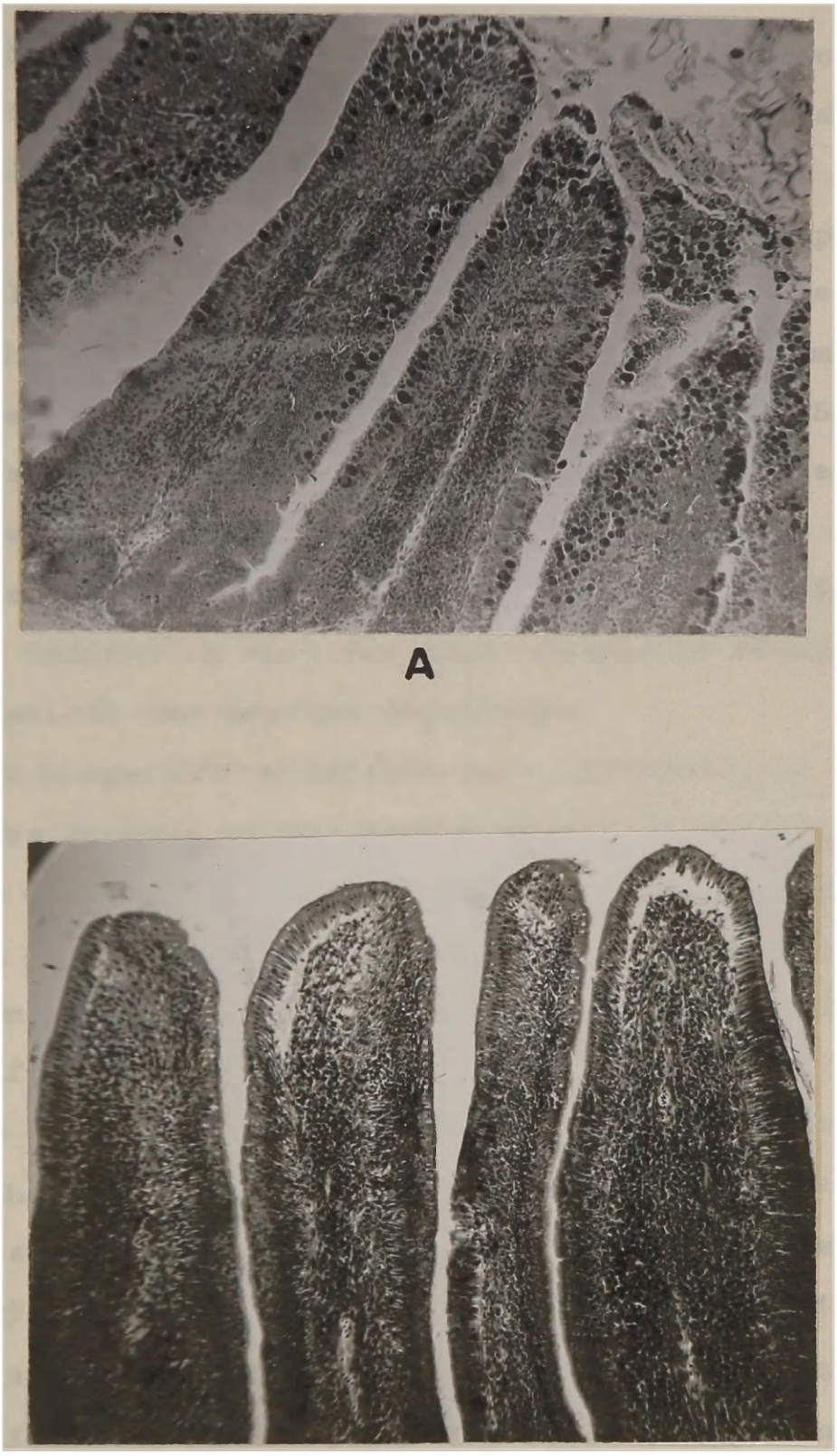
B

Figure 9

A. Jejunum of two week old poult infected with Eimeria dispersa for five days. (X77)

B. Jejunum of two week old poult infected with Eimeria dispersa for nine days. (X77)

FIGURE 9



A

B

with 76,000 sporulated oocysts. This was repeated for a third week. The results of this series of infections is presented in Figure 4. The first oocysts were recovered late on the fifth day following infection, and reached a peak of 65,300 oocysts per gram of feces six days after infection. The numbers declined rapidly from this peak with slight fluctuations and had practically disappeared by the fifteenth day following infection. Very small numbers of oocysts were recovered on the 20th, 21st, 23rd, 25th and 26th days following infection, after which fecal examinations were negative. No peaks in oocyst counts could be attributed to any of the challenge infections of 76,000 oocysts, indicating that the lower dosage of oocysts protected against the heavier infections.

Four Hungarian partridges were infected with 100,000 sporulated oocysts of the turkey strain of E. dispersa. Six days following infection small numbers of oocysts were found in the feces of the infected birds and they reached a peak on the eighth day. It is of interest to note that the two females infected reached a peak of 286,400 oocysts per gram of feces while the two males had a peak of 93,300. Due to the small number of birds used it is not felt that these results can be profitably discussed. Following the peak period of oocyst discharge, the numbers declined rapidly and the feces were negative on the twelfth day after infection. Reinfection three weeks following the original infection demonstrated that the partridges were completely resistant to further infection with this species.

The course of these two infections is shown in Figures 1 and 2.

Twelve ten week old Bob-white quail were infected with 76,000 sporulated oocysts of the turkey strain of E. dispersa. Six days following infection 1,407,000 oocysts per gram of feces were found, and this increased to a peak of 3,980,000 oocysts per gram of feces on the seventh day after infection. This dropped slightly for the next two days, and on the eleventh day the numbers of oocysts had dropped to the pre-infection level. Three weeks after the initial infection the quail were reinfected with 54,000 sporulated oocysts of the turkey strain of E. dispersa which had been passed once through the quail host. Oocysts were passed in the feces six days after infection and reached a peak of 913,000 oocysts per gram of feces on this day, and had declined to a low level ten days after infection. The course of this infection is presented in Figure 3.

FIGURE 10.

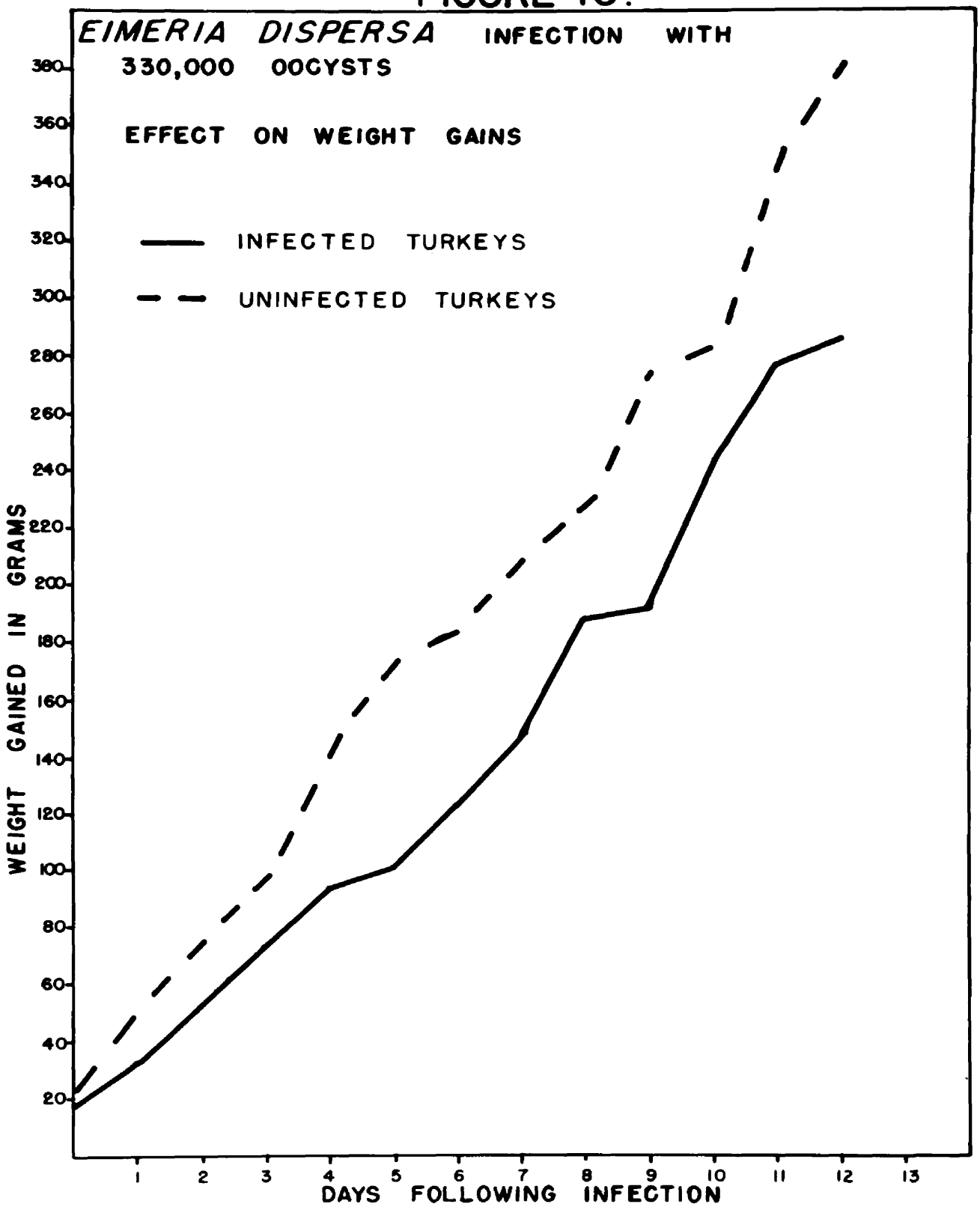
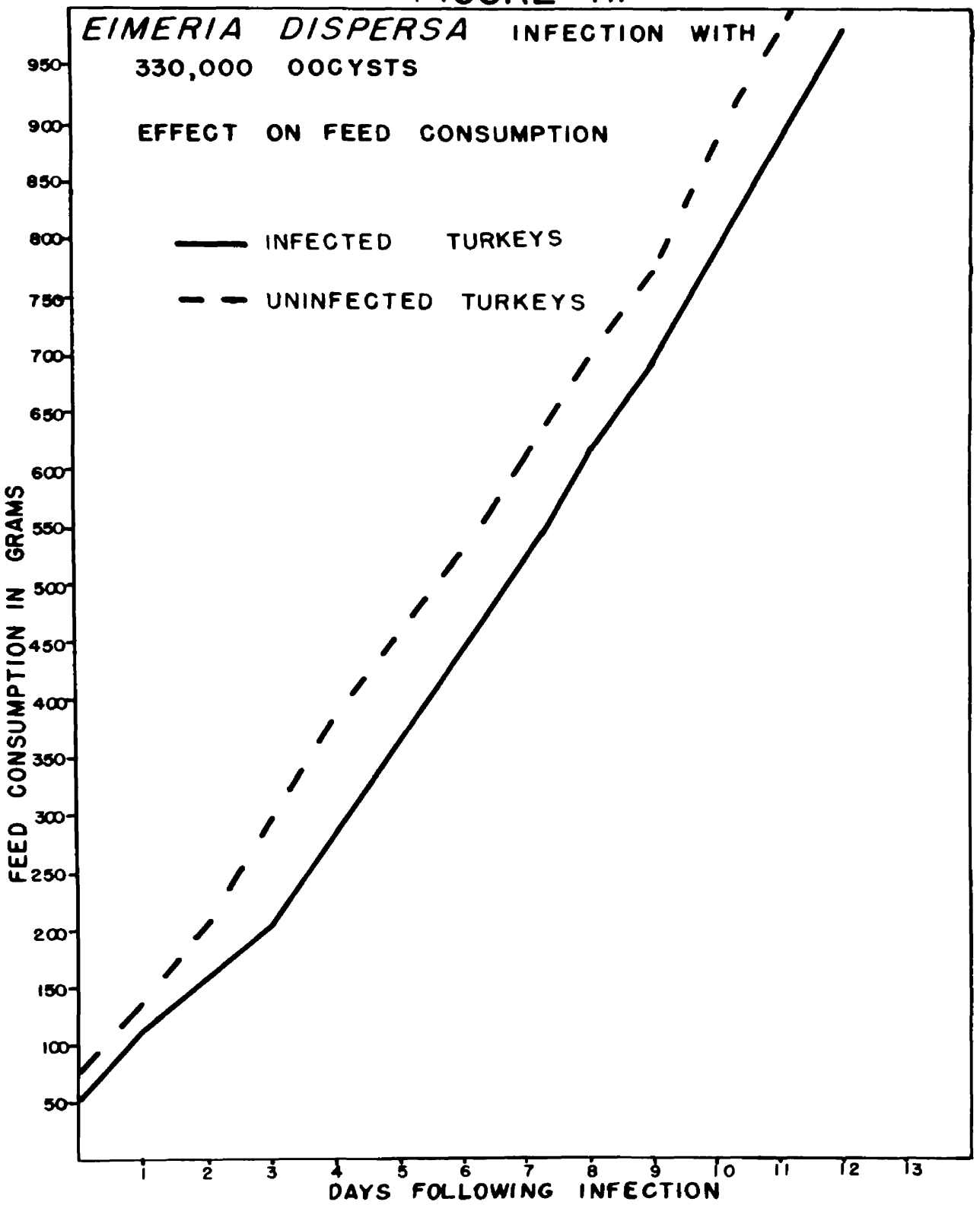

EIMERIA DISPERSA INFECTION WITH
330,000 OOCYSTS
EFFECT ON WEIGHT GAINS
INFECTED TURKEYS
UNINFECTED TURKEYS
WEIGHT GAINED IN GRAMS
380
360
340
320
300
280
260
240
220
200
180
160
140
120
100
80
60
40
20
1
2
3
4
5
6
7
8
9
10
11
12
13
DAYS FOLLOWING INFECTION

FIGURE 11.


EIMERIA DISPERSA INFECTION WITH
330,000 OOCYSTS
EFFECT ON FEED CONSUMPTION
INFECTED TURKEYS
UNINFECTED TURKEYS
FEED CONSUMPTION IN GRAMS
950
900
850
800
750
700
650
600
550
500
450
400
350
300
250
200
150
100
50
1
2
3
4
5
6
7
8
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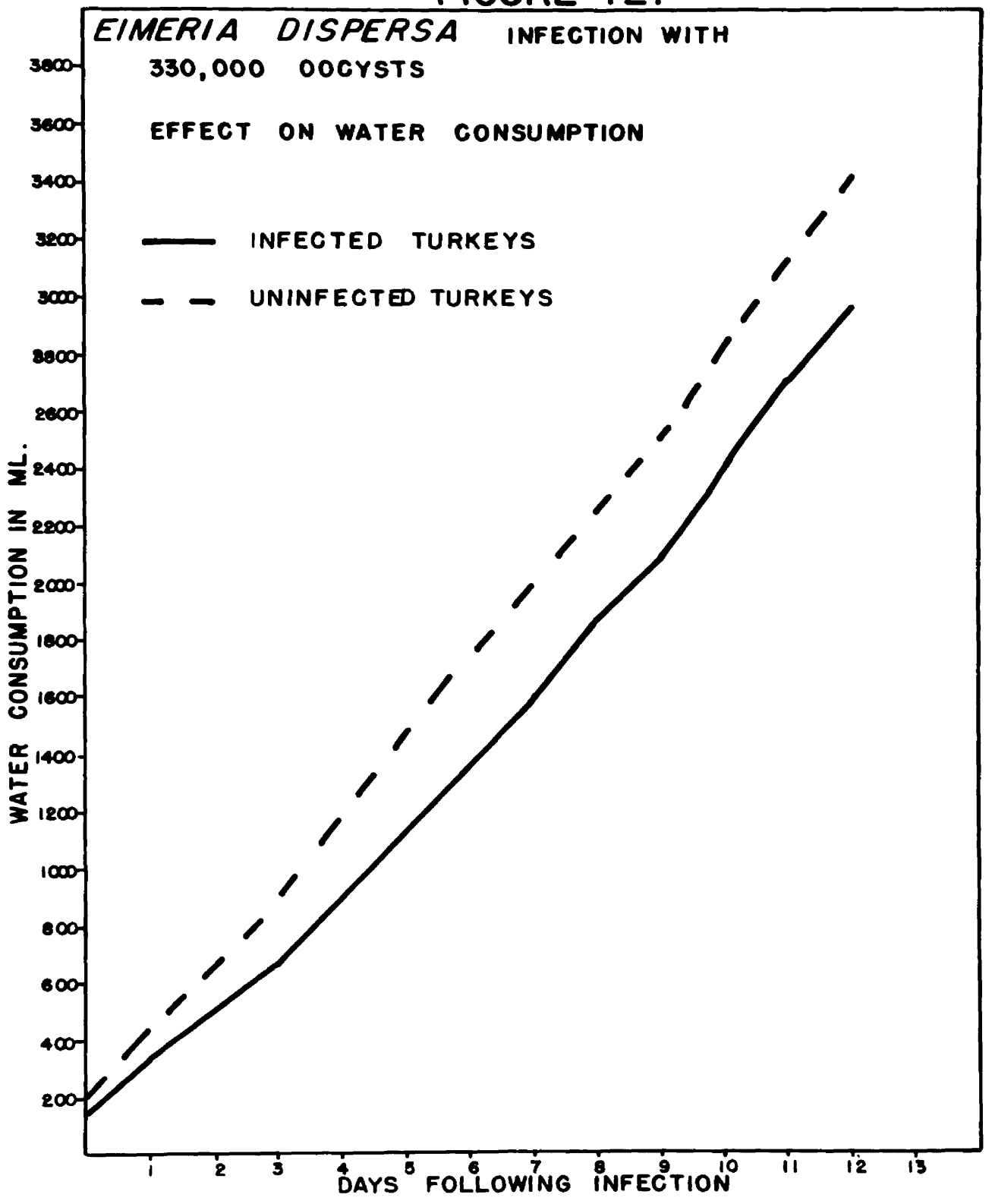
11
12
13
DAYS FOLLOWING INFECTION

FIGURE 12.


EIMERIA DISPERSA INFECTION WITH
330,000 OOCYSTS
EFFECT ON WATER CONSUMPTION
INFECTED TURKEYS
UNINFECTED TURKEYS
WATER CONSUMPTION IN ML.
3800
3600
3400
3200
3000
2800
2600
2400
2200
2000
1800
1600
1400
1200
1000
800
600
400
200
1
2
3
4
5
6
7
8
9
10
11
12
13
DAYS FOLLOWING INFECTION

Eimeria meleagrimitis Tyzzer, 1929

SOURCE

This species was first described by Tyzzer (1929) from the small intestine of the turkey. This species has been recovered from material received from Connecticut, Iowa, Kentucky, Massachusetts, Michigan, Minnesota, Missouri, Nebraska, New Jersey, Oregon, Texas, and Utah. The strain which was most thoroughly studied was obtained from Berkshire County, Massachusetts through the courtesy of Dr. K. L. Bullis.

HOST

This species has been found to develop only in the turkey.

OOCYST

The oocysts of E. meleagrimitis were passed in the feces six days after infection. They measured 15.8 to 26.9 microns in length (average 19.17 microns) (see Figure 13) and 13.1 to 21.9 microns in width (average 16.28 microns). These dimensions are based on the measurement of 100 oocysts. The oocyst is subspherical with a length breadth ratio of 1.17 (see Figure 14). The cyst wall presents a double contoured appearance in contrast with the single contoured appearance of E. dispersa (see Plate V-E). Occasionally a polar granule or inclusion body may be seen in the unsporulated oocysts, but normally they are absent in freshly passed specimens.

After the oocysts had been passed in the feces, approximately 48 hours were required for their sporulation. Many oocysts when kept in 2.5 percent potassium dichromate at room temperature had divided into four sporoblasts at the end of 24 hours. At this time the polar granules or inclusion bodies had formed, even in those cysts which had not as yet divided. The division and formation of the completely sporulated oocysts seems to be identical to that observed in E. dispersa. One to three polar granules were found in all sporulated oocysts (see Plate V-F and Figure 7 B).

TISSUE STAGES

The earliest stages found in the tissues were observed 47 hours after infection, although sections were studied from birds killed 12, 23, 29 and 40 hours after infection. These were rounded or elongated, uninucleate or binucleate forms, located superficially to the nucleus of the epithelial cell. They measured two to four microns in diameter (see Plate VII-A). They possessed a small karyosome located in a clear vacuole. Two days and 18 hours following infection the schizonts had increased markedly in size, in many the nuclei were still dividing, but in others merozoites had been formed. Forms measuring up to eight microns in diameter have been noted in which the cytoplasm had not divided. Those schizonts which contained merozoites measured eight to nine microns in diameter and contained nine to ten merozoites. The latter measured four to six

FIGURE 13.

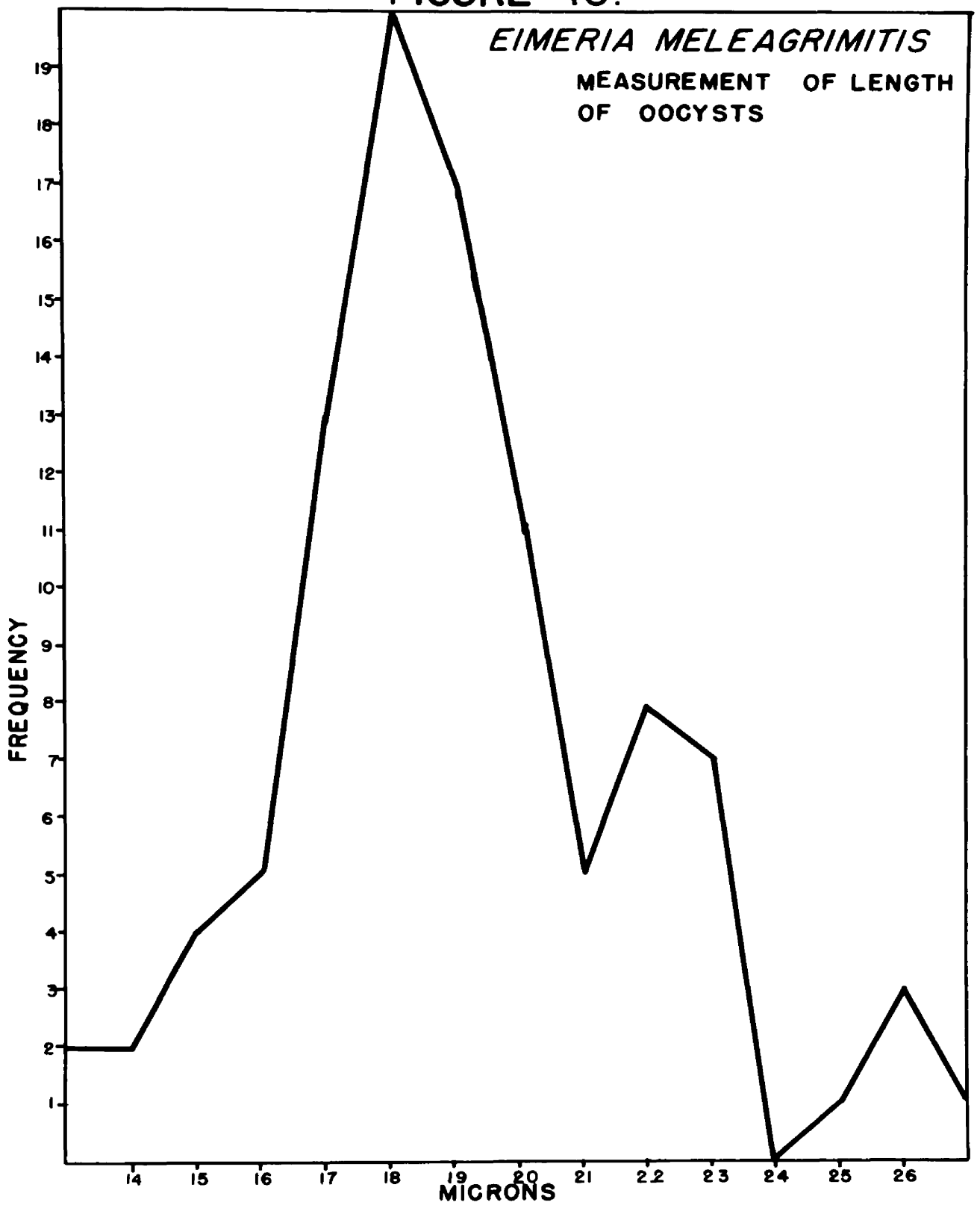
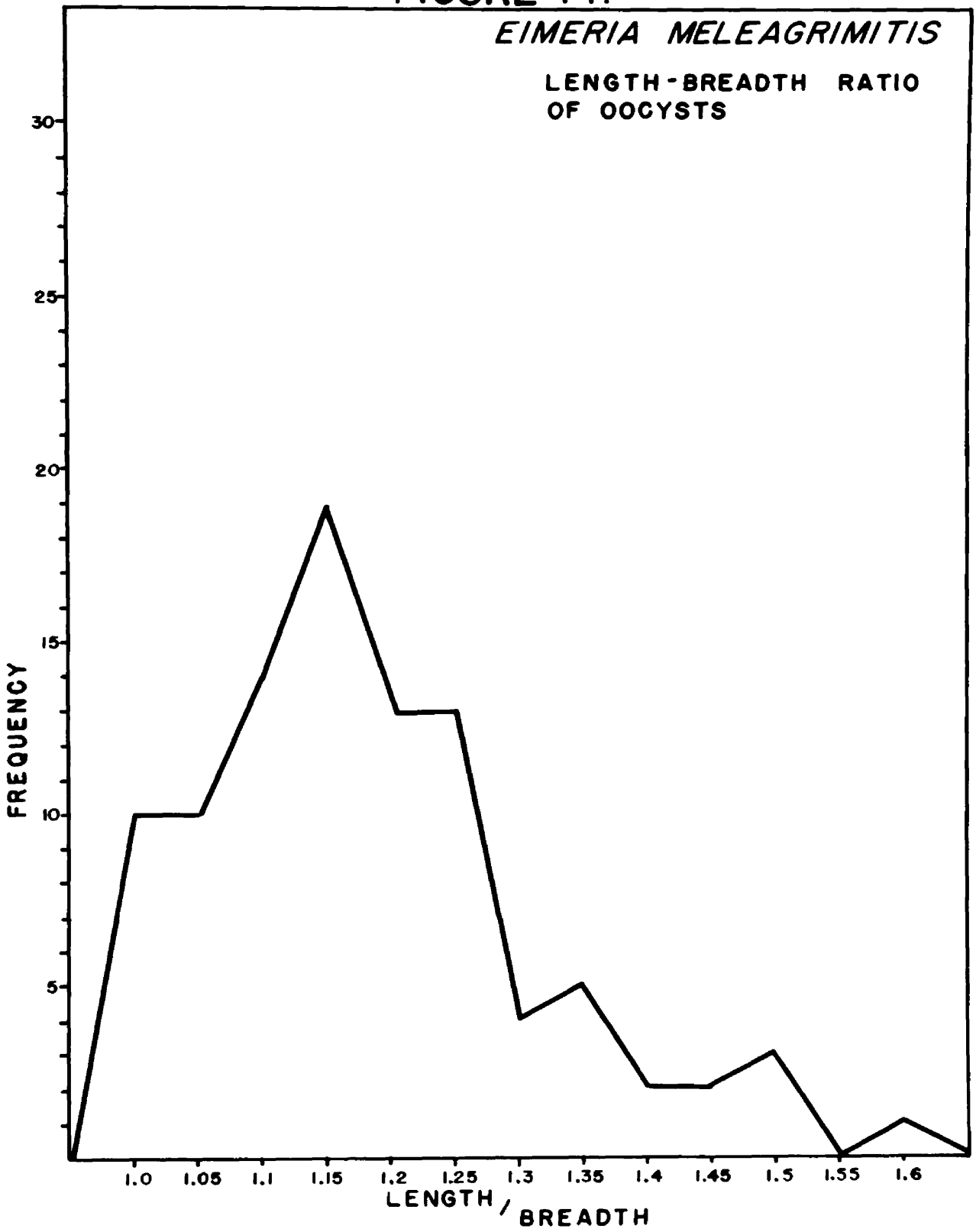

EIMERIA MELEAGRIMITIS
MEASUREMENT OF LENGTH OF OOCYSTS
FREQUENCY
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
14
15
16
17
18
19
20
21
22
23
24
25
26
MICRONS

FIGURE 14.


EIMERIA MELEAGRIMITIS
LENGTH-BREADTH RATIO
OF OOCYSTS
FREQUENCY
30
25
20
15
10
5
1.0
1.05
1.1
1.15
1.2
1.25
1.3
1.35
1.4
1.45
1.5
1.55
1.6
LENGTH / BREADTH

microns in length and about one and one half microns in width. They possessed a well defined, minute karyosome located in a small vacuole in the center of the merozoite (see Plate VII-D). Large numbers of schizonts containing merozoites were found throughout the jejunum three days after infection. None of the large schizonts such as are found in the development of E. dispersa or E. gallopavonis have been noted in infections with this species.

The first asexual generation was completed during the third day following infection. At that time many of the first generation merozoites had been liberated and were to be found as rounded up trophozoites in new epithelial cells. Many of the trophozoites, when first noted in the epithelial cells, measured about one micron in diameter, (see Plate VII-C and D). It is difficult to determine how much lag there was in the development of the first asexual generation, due to the apparent morphological similarity in many cases between the schizonts of the first and second generation. Three and one half days after infection the nucleus of many of the trophozoites had divided. Four days after infection numerous schizonts containing merozoites were observed. As noted above, many were very similar to those found in the first generation, but some were slightly larger. The larger mature schizonts measured 12 to 14 microns in diameter and contained 10 to 12 merozoites, each being five to six microns long and one to two microns wide. These forms are shown in Plate VIII.

The second asexual generation merozoites were liberated four to four and one half days after infection. In poults killed at that time enormous numbers could be found free in the lumen of the small intestine.

The third generation, which was predominantly sexual, was initiated by the penetration of new epithelial cells in the small intestine by the liberated second generation merozoites. In the case of heavy infections, much of the epithelium in the jejunum was lost, so that the sexual stages in such cases were located predominantly in the duodenum and ileum. However, in lighter infections the largest number of sexual forms would localize in the middle half of the small intestine. Four and one half days after infection, large numbers of undifferentiated gametocytes were found in the epithelial cells, and it frequently appeared that more than one parasite occurred in each epithelial cell. The parasites at this time measured from three to five microns in diameter. They possessed a single karyosome located in a lighter area either in the center or eccentrically placed in the cell. The cytoplasm was finely granular (see Plate IX-A). Five days after infection macrogametocytes could be differentiated as those forms possessing a single nucleus and hematoxylinophilic granules about the periphery of the cell (see Plates IX-B and X-B). The macrogametocytes at this time measured seven to eight microns in diameter. There were also stages present which were assumed to be macrogametocytes, of similar size, with a single nucleus, but without the hema-

toxylinophilic granules in the cytoplasm. Also, macrogametocytes were noted, possibly a slightly earlier stage than that just described, in which very minute granules were scattered throughout the cytoplasm of the parasite, each granule being in a small vacuole (see Plates IX-D and X-A). It is of interest that at this stage many of the parasites were found located below the epithelial cell nucleus, some resting on the basement membrane as in Plate X-A. Five and one half days after infection the cyst wall was observed forming about the macrogametocytes (see Plate X-C), the cytoplasm still contained many small granules, but no large granules were present at this stage in the specimens studied. A single nucleus was present and the cytoplasm filled the cell which may now be called a macrogamete. On the sixth day after infection fertilization had occurred and the cytoplasm of the zygote had contracted away from the cyst wall (see Plate X-D).

Microgametocytes were differentiated five days after infection. The nucleus had divided a number of times, and it appeared that the cytoplasm had at this time formed about some of the nuclei (see Plate IX-C). In slightly later stages the nuclei assumed the characteristic appearance noted in Plate IX. As the microgametes were formed, their chromatin staining material became elongated, and a residual body remained in the center of the large masses of microgametes (see Plates IX-D and X-C).

Asexual stages were not found in large numbers during the

fifth and sixth days of infection. They were similar to those occurring earlier except that they were usually larger and produced from 20 to 40 merozoites (see Plate X-D).

GROSS PATHOLOGY

The earliest lesions which have been observed in experimentally infected poults occurred four days after infection. At this time there was an excessive amount of fluid in the duodenum and jejunum. Before this time the small intestine was essentially normal. Four and one half days after infection, little or no feed was present in the lumen of the small intestine, and it was filled with a clear, usually colorless fluid. The jejunum was slightly thickened and dilated, and small areas of congestion were noted. The posterior portions of the jejunum and particularly the ileum contained a greenish mucus. Five to five and one half days following infection the lumen of the small intestine was devoid of food and the mucosa of the duodenum and jejunum was white. Occasionally the mucosa of the duodenum appeared to have undergone a coagulation necrosis. Pieces of caseous, cheesy material were found scattered in the lumen of the entire small intestine. There was a large amount of fluid in the lumen which occasionally had a pinkish tinge, but large amounts of blood have never been noted, although in rare instances small strands of clotted blood might be observed. Mucus strands were numerous in the jejunum and ileum. Six to seven days after infection the small intestine was still devoid of food in the lumen. Spotty congestion was most

Plate VII - *Eimeria meleagrimitis*

A. A trophozoite and schizont in an epithelial cell of the jejunum 47 hours after infection.

B. Schizonts in epithelial cells of the jejunum two days and 18 hours after infection.

C. Second generation trophozoites in epithelial cells of the jejunum three days after infection.

D. First generation schizont containing merozoites and a second generation trophozoite in epithelial cells of the jejunum three days after infection.

PLATE VII

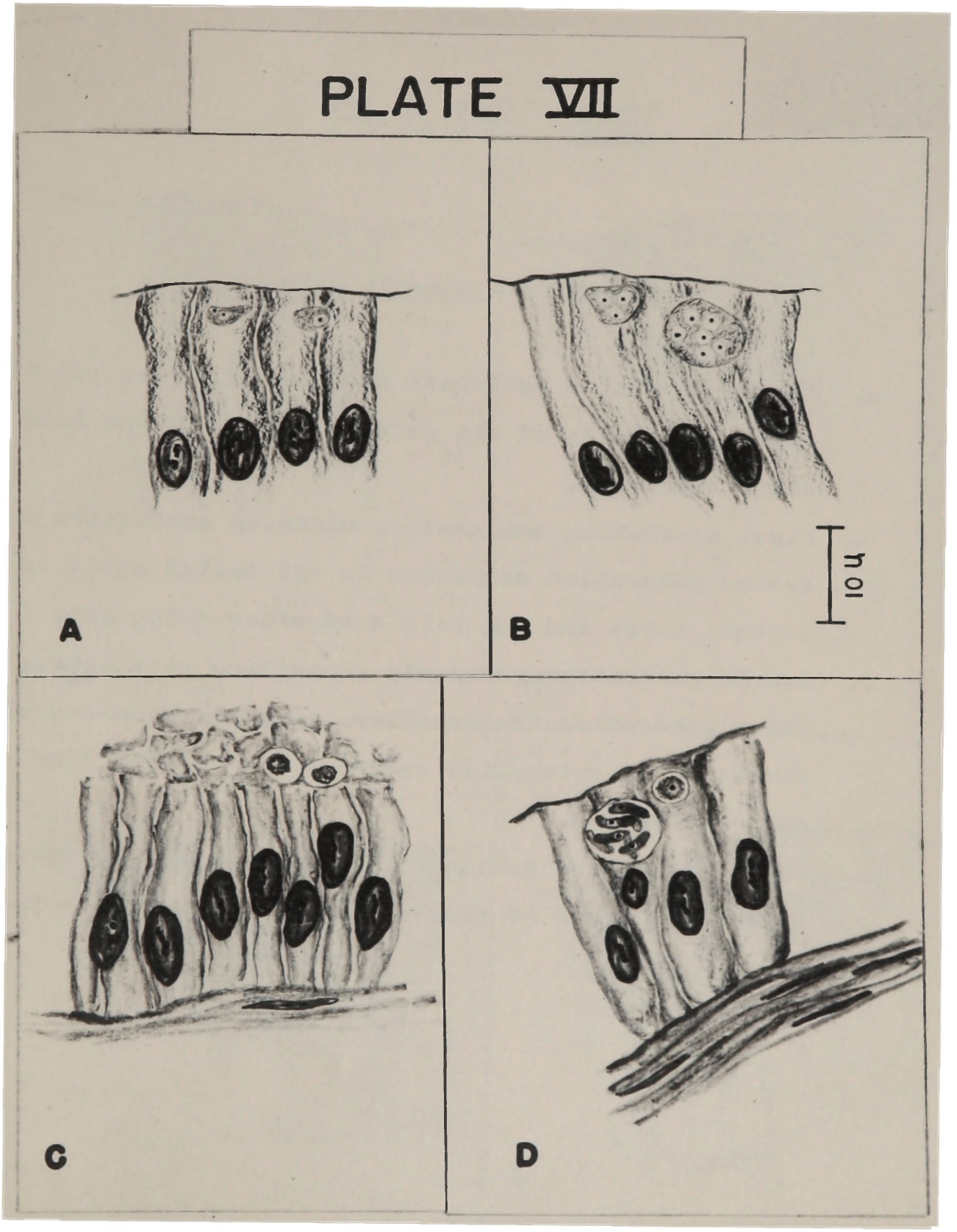


Plate VIII - *Eimeria meleagrimitis*

A. First generation schizonts containing merozoites in epithelial cells of the jejunum three and one half days after infection.

B. First generation schizonts containing merozoites and second generation schizonts in epithelial cells of the jejunum three and one half days after infection.

C. Second generation schizonts containing merozoites, and third generation trophozoites (possibly gametocytes) and schizonts in epithelial cells of the jejunum four days after infection.

D. Third generation schizont (S) and gametocyte (G) in epithelial cells of the jejunum four days after infection.

PLATE VIII

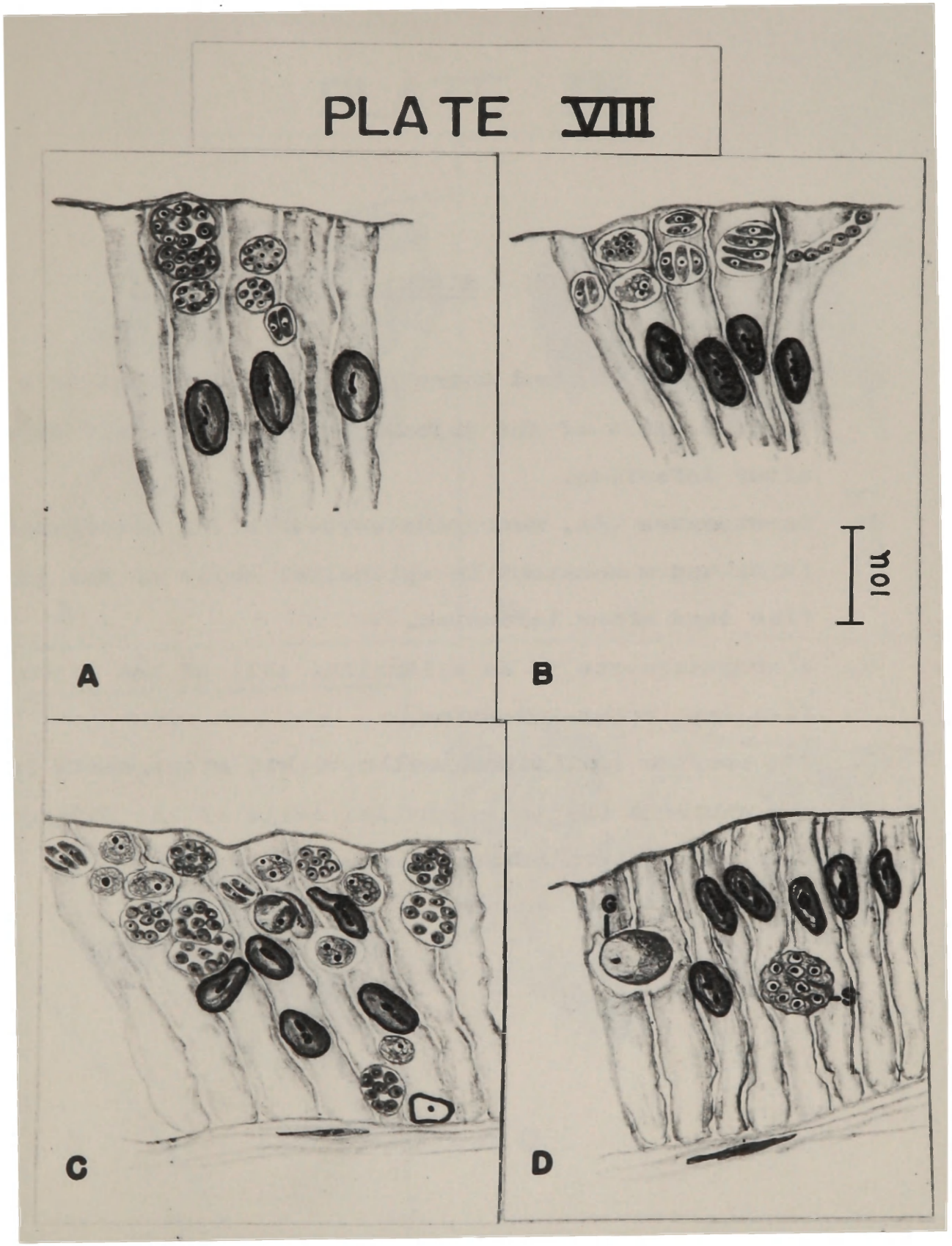


Plate IX - *Eimeria meleagrimitis*

A. Gametocytes (G) and third generation schizont in epithelial cells of the jejunum four and one half days after infection.

B. Gametocytes (G), macrogametocytes (M), a microgametocyte (MIC) and a schizont in epithelial cells of the jejunum five days after infection.

C. Microgametocyte in an epithelial cell of the jejunum five days after infection.

D. Gametocytes (G), macrogametocyte(M), microgamete (MI) and schizont (S) in epithelial cells of the jejunum five days after infection.

PLATE IX

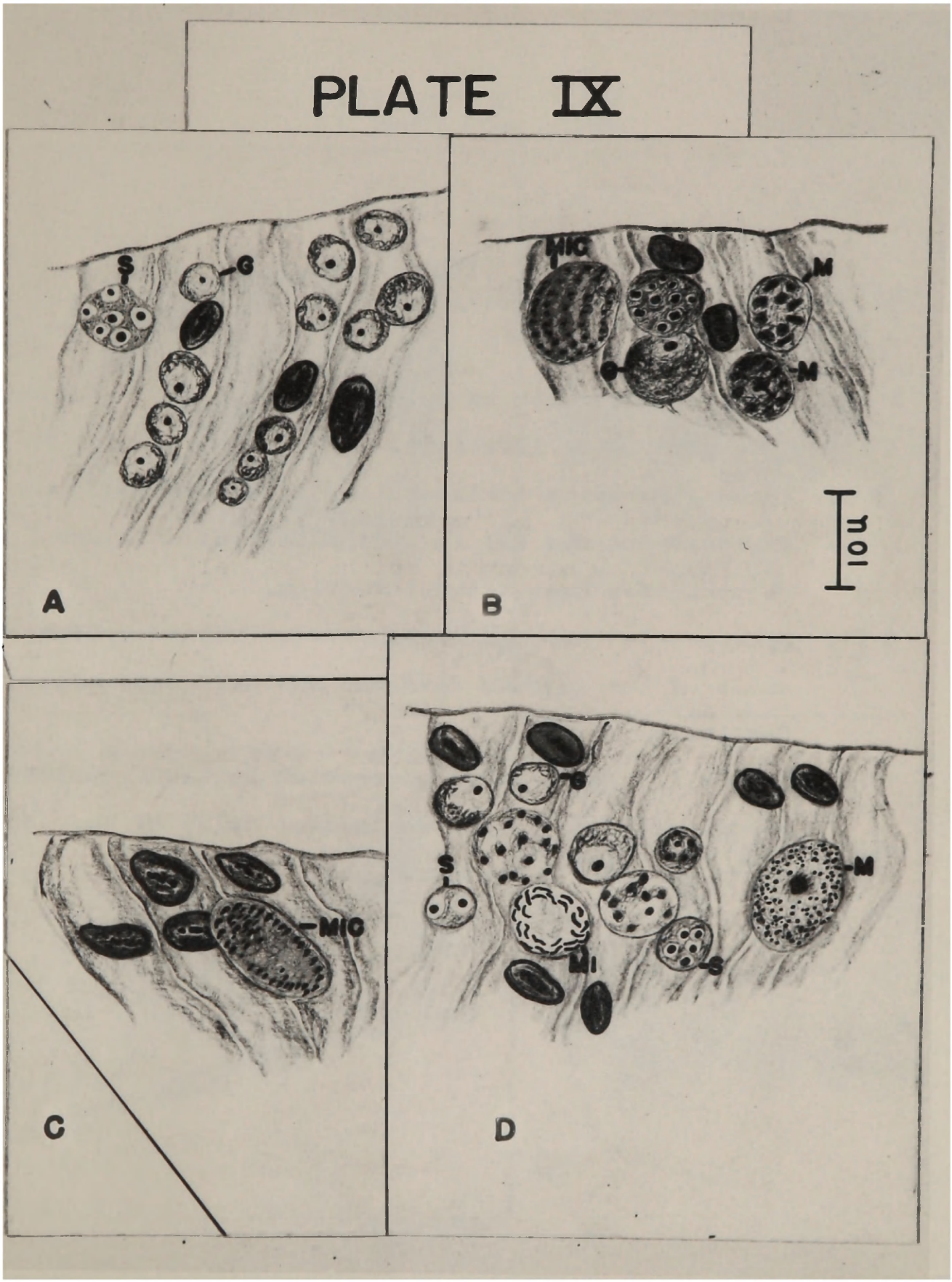


Plate X - *Eimeria meleagrimitis*

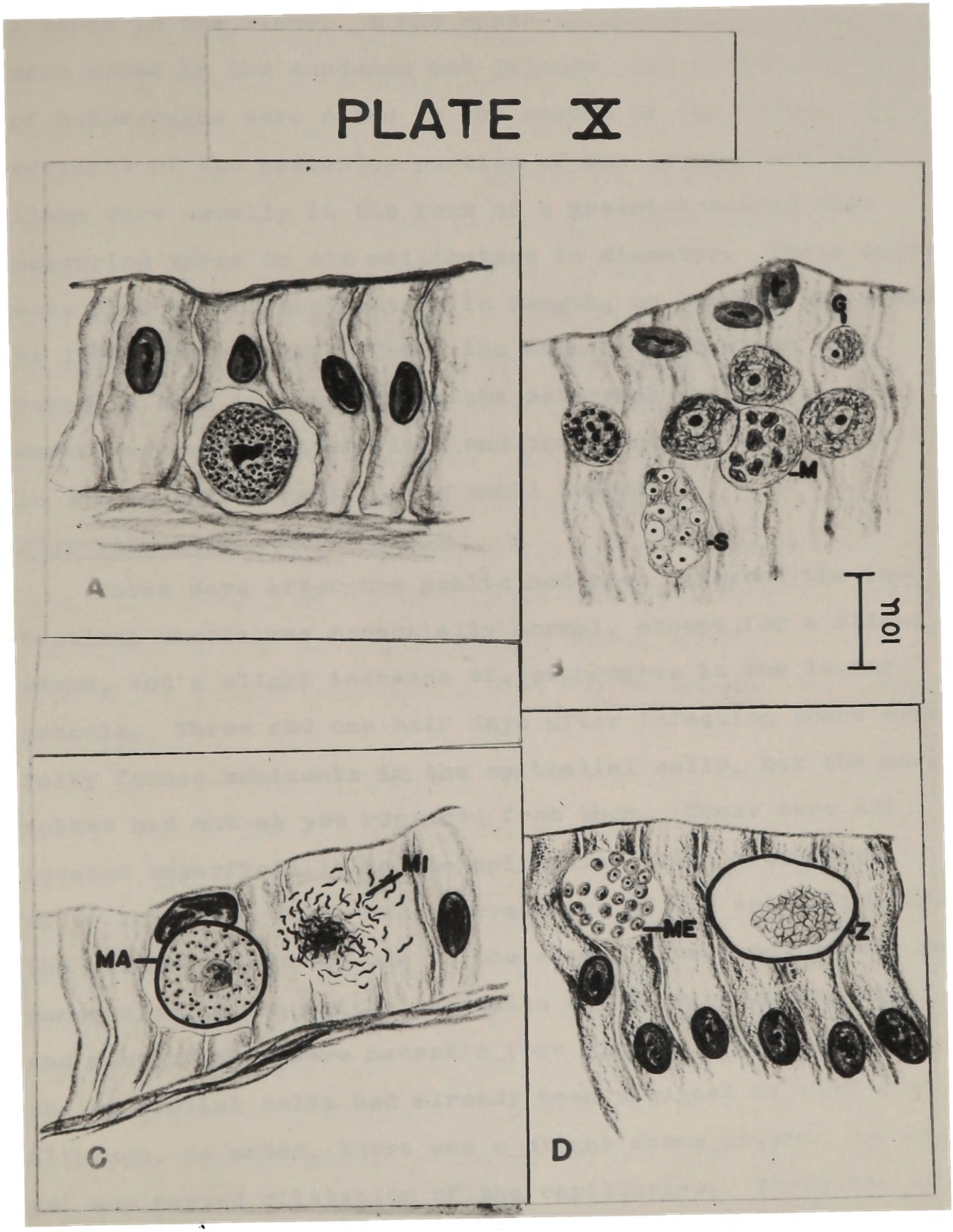
A. Macrogametocyte in an epithelial cell of the jejunum five days after infection.

B. Third generation schizont (S), gametocyte (G) and macrogametocytes (M) in epithelial cells of the jejunum five days after infection.

C. Macrogamete (MA) and microgametes (MI) in epithelial cells of the jejunum five and one half days after infection.

D. Zygote (Z) and a third generation schizont containing merozoites (ME) in the epithelial cells of the jejunum six days after infection.

PLATE X



notable in the ileum. A few spots of petechial hemorrhage were noted in the duodenum and jejunum, and minute streaks of hemorrhages were noted in the mucosa of the ileum. The contents of the posterior portion of the jejunum and the ileum were usually in the form of a greenish mucoid cast measuring three to six millimeters in diameter. These casts were five to ten centimeters in length, or longer, and could be lifted out intact. Thus, the main lesions which were noted in this infection were the catarrhal enteritis, the marked accumulation of fluid and the greenish mucoid cast in the posterior part of the small intestine.

HISTOPATHOLOGY

Three days after the poults had been infected the intestinal mucosa was essentially normal, except for a slight edema, and a slight increase of lymphocytes in the lamina propria. Three and one half days after infection there were fully formed schizonts in the epithelial cells, but the merozoites had not as yet ruptured from them. These were all located superficially in the epithelial cell. Four days after infection there was a separation of the epithelium from the basement membrane due to the edema. There was mucoid degeneration in the epithelial cells at the tips of the villi and many of them were necrotic (see Figure 15 B). Some of the epithelial cells had already been sloughed at this time. Although, as noted, there was a slight edema present but not yet any marked dilatation of the capillaries. There was an increased number of lymphocytes in the lamina propria. The

schizonts, containing merozoites, are most numerous at the tips of the villi, extending down into the crypts in decreasing numbers. None have ever been observed in the glands. In heavy infections there were hardly any unparasitized epithelial cells. Four and one half days after infection most of the epithelium on the tips of the villi had sloughed off (see Figures 16 and 17 A), due to the liberation of the second generation merozoites. The capillaries in the villi were dilated, and at the tips were only separated from the lumen by the basement membrane. It is remarkable that at this time very little hemorrhage into the lumen was noted. The tips of the villi were edematous and there was an increase in connective tissue in this location. The connective tissue nuclei were much more active near the tips of the villi than at the base. Lymphocytes were very numerous in the lamina propria.

Five days after infection the villi were grossly enlarged, due to the edema, and the capillaries were markedly dilated throughout the villus, at the base as well as near the tips (see Figure 17 B). There was a marked increase in connective tissue near the tips of the villi which extended down into each villus. The laminae propriae of the villi were packed with lymphocytes. The tips of many of the villi were still bare of epithelium, although in some areas it was noted that the epithelium had succeeded in stretching over in a thin layer and covering the villus. There were numerous sexual stages in the distal epithelium of the villi of the jejunum when epi-

thelium was present. In the case of heavy infections, when much of this epithelium had been lost, there were large numbers of gametocytes in the duodenum and ileum.

Five and one half days after infection all the villi were covered with epithelium, but they were still very edematous. However, the capillaries were not as dilated as they were 12 hours previously. There was thus a decreased amount of congestion. The edematous areas were also vacuolated in many places. There was still a greater amount of connective tissue and more lymphocytes in the lamina propria. Very few polymorphonuclear leucocytes were noted. Sections taken six days after infection presented a similar picture, but in later days there was a gradual decrease in the reaction, although the marked lymphocytic infiltration persisted through the ninth day, which was as long as the infection was followed (see Figure 18).

SYMPTOMS

This is the most serious coccidial infection which was encountered in the turkey. It seemed that there was an age resistance against this infection, although the data at the present time are not sufficient to substantiate this conclusion. However, it was possible to kill poults two to three weeks of age with an infecting dose which would not kill poults four or five months old, although feed consumption and weights were reduced in the older birds. In experimental infections 50,000 sporulated oocysts were sufficient to produce a high mortality, in some instances 100

percent, in turkey poults two to three weeks of age.

The symptoms produced by this coccidium were not characteristic. Within two to three days after infection it was noted that feed consumption began to drop, and four days after infection the feathers were ruffled, the wings drooped, eyes closed, and the birds were closely huddled. Frequently the birds held their heads between their legs and cheeped almost constantly, all this time keeping their eyes closed. The droppings were scanty and slightly fluid. At the peak of the disease, i.e. five to six days after infection, some of the fecal material appeared as cyclindrical pellets one to two centimeters in length and three to six millimeters in diameter, with the ends cut off as if with a knife. Bloody droppings were not observed in coccidiosis in the turkey, although a few flecks of blood may occasionally be noted in the feces. Death usually occurred on the fifth and sixth days of infection. If death did not terminate the disease, weight losses ceased about the seventh or eighth days after infection, and a gradual recovery ensued.

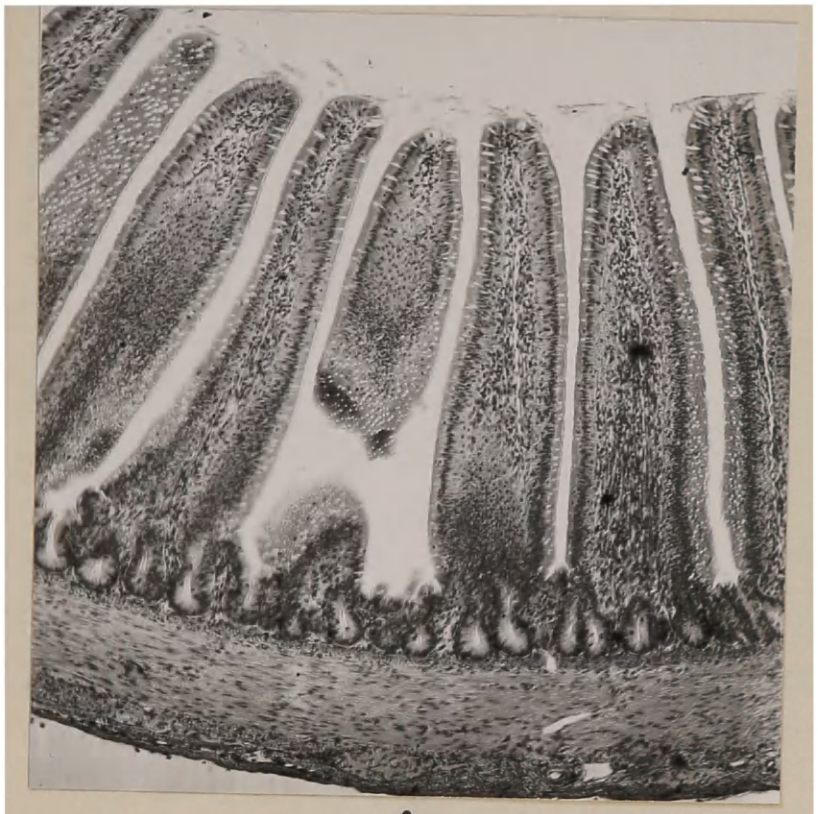
This infection has a very serious effect on the weight gains of infected poults (see Figure 19). Four days after infection there had been little depression of the weights of two week old poults infected with 330,000 sporulated oocysts. However, for the next four days the weight gains dropped, until on the seventh day following infection the poults were actually losing weight. Following the eighth day the poults began again to gain weight, but at a retarded rate. The birds have

Figure 15

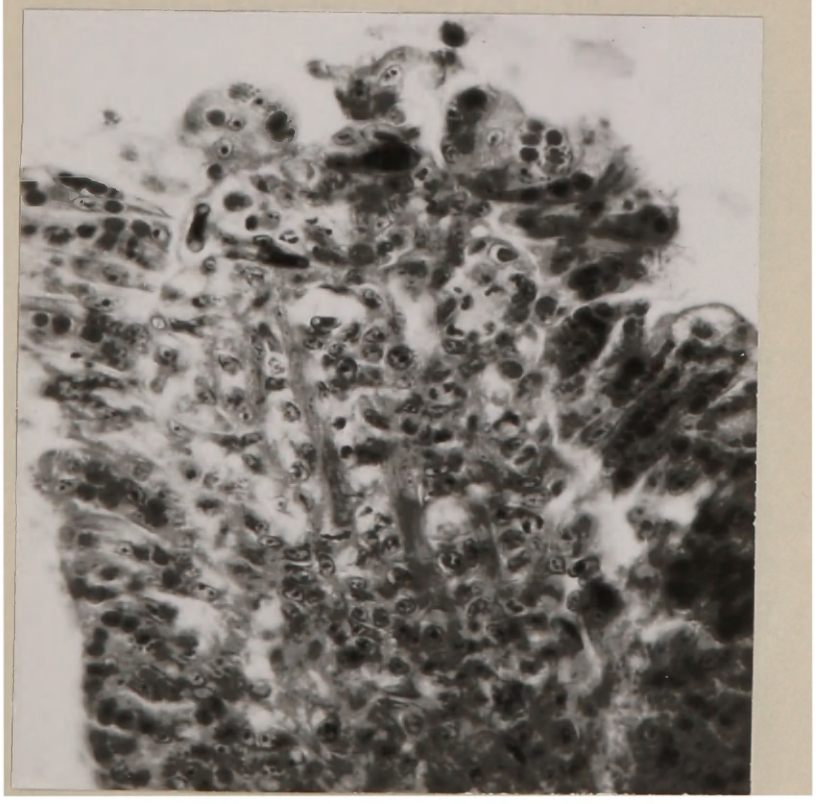
A. Jejunum of an uninfected two week old poult. (X77)

B. Jejunum of a two week old poult infected with Eimeria meleagrimitis four days. (X570)

FIGURE 15



A



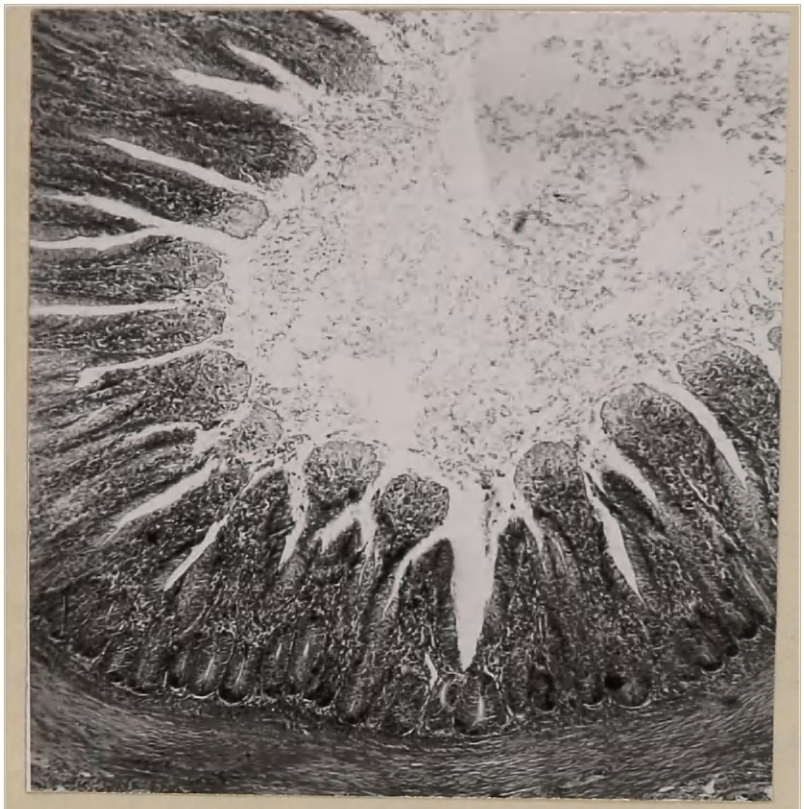
B

Figure 16

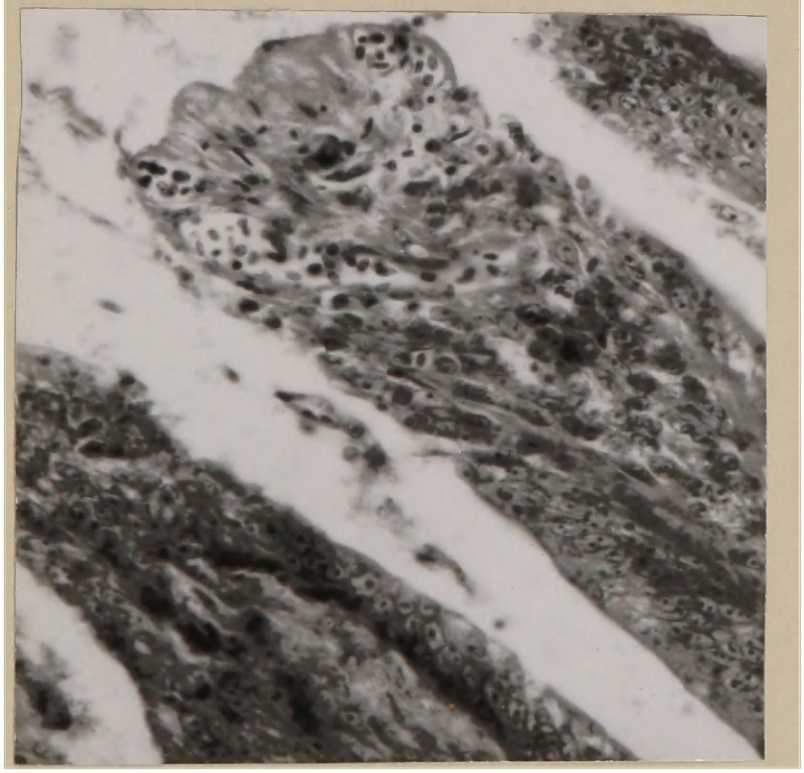
A. Jejunum of two week old poult infected with Eimeria meleagrimitis four and one half days. (X77)

B. Single villus from section shown in A. (X570)

FIGURE 16



A



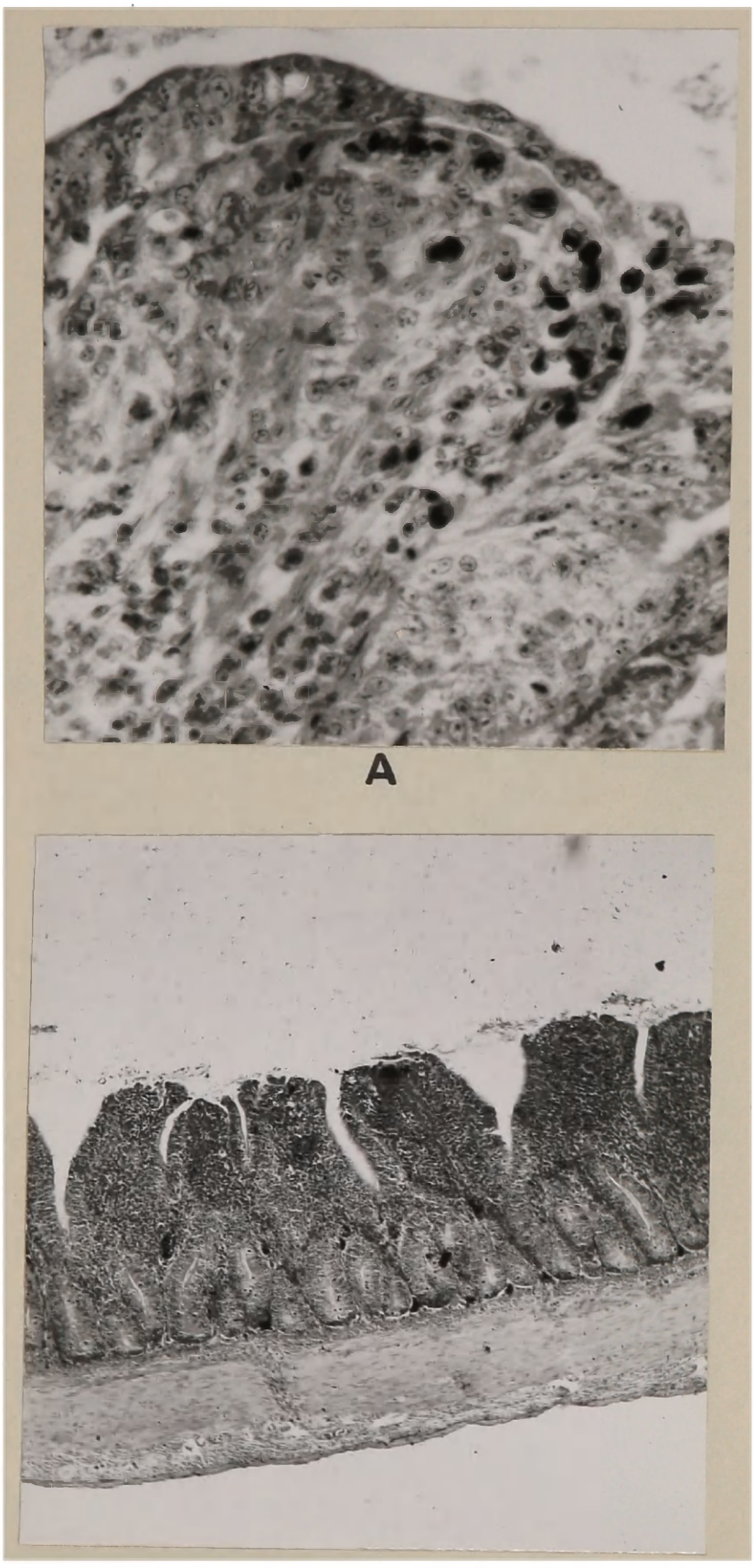
B

Figure 17

A. Ileum of two week old poult infected with Eimeria meleagrimitis for four and one half days. (X570)

B. Ileum of two week old poult infected with Eimeria meleagrimitis for five days. (X77)

FIGURE 17



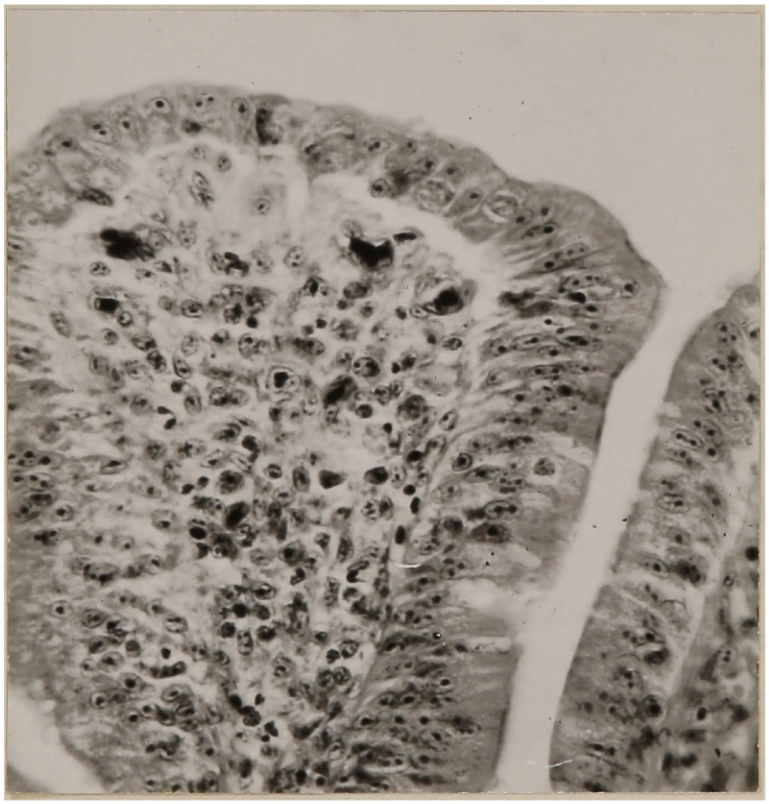
A

B

Figure 18

Jejunum of two week old poult infected with Eimeria meleagrimitis for nine days. (X570)

FIGURE 18



not been followed long enough to determine if these gains are ever made up. Feed consumption was depressed following the fourth day of infection (see Figure 20) as was water consumption (see Figure 21).

IMMUNITY

Although the immunity produced against the coccidia of the turkeys must always be considered as relative, rather than absolute, the immunity elicited by this species is not as solid as that manifested after infection with the other three species.

Nine turkeys were used in these experiments and they had already been immunized with E. dispersa as described on p. 19. One group of six birds was infected with 1280 sporulated oocysts of E. meleagrimitis daily for eight days and then administered 128,000 sporulated oocysts. This was followed by ten daily infections of 1280 sporulated oocysts and again they were given 128,000 sporulated oocysts. The course of this infection is shown in Figure 22. Oocysts were recovered in the feces of these poults six days after infection and reached a peak of 24,100 oocysts per gram of feces seven days after infection, after which the peak declined rapidly and fluctuated at a low level for the next four weeks. The oocyst counts over this period of time seemed to bear no relation to the challenge doses of larger numbers of oocysts. However, it can be seen that the initial infection with a small number of oocysts did not produce as solid an immunity as did small infections with E. dispersa, nor as do higher

FIGURE 19.

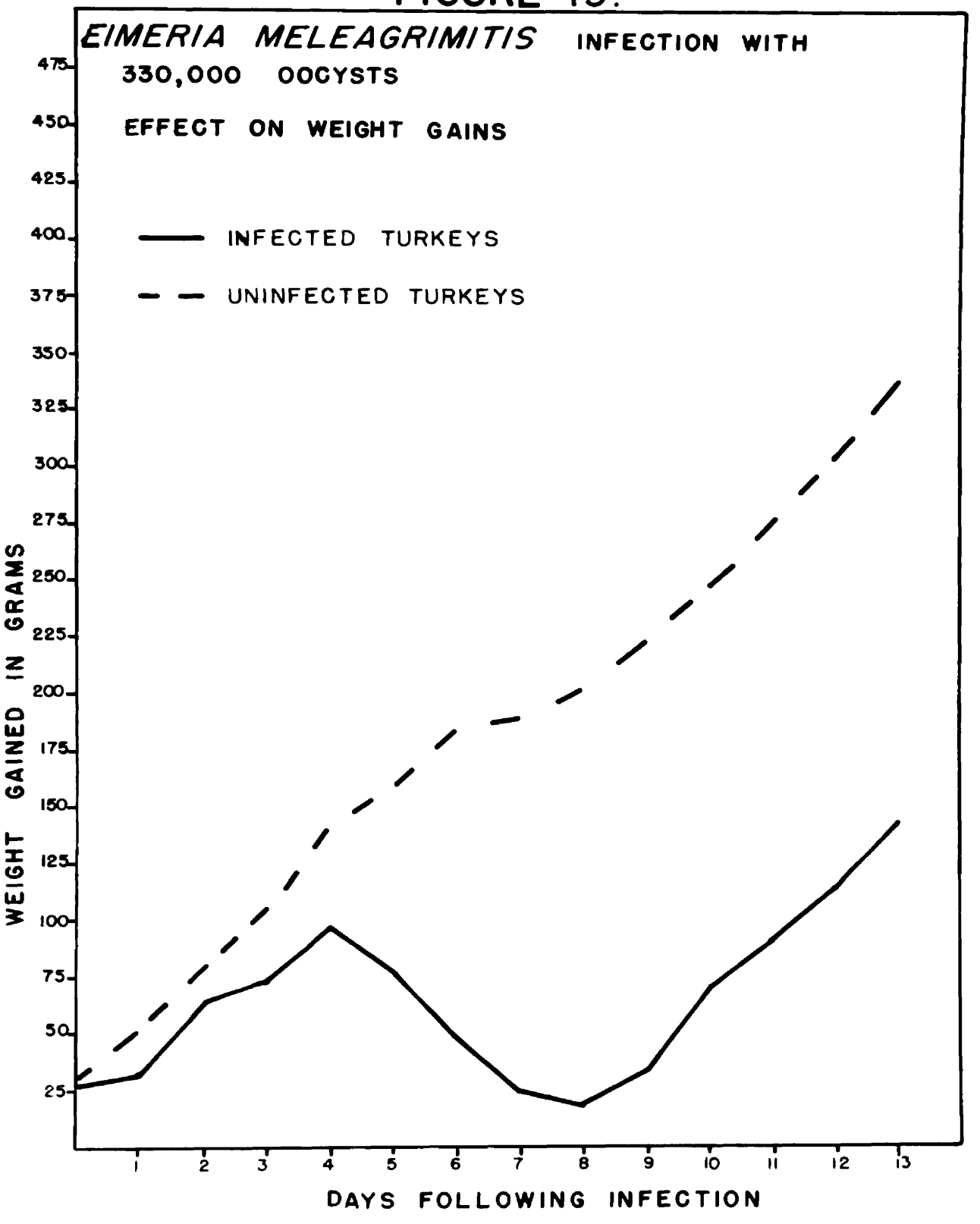

EIMERIA MELEAGRIMITIS INFECTION WITH
330,000 OOCYSTS
EFFECT ON WEIGHT GAINS
INFECTED TURKEYS
UNINFECTED TURKEYS
WEIGHT GAINED IN GRAMS
475
450
425
400
375
350
325
300
275
250
225
200
175
150
125
100
75
50
25
1
2
3
4
5
6
7
8
9
10
11
12
13
DAYS FOLLOWING INFECTION

FIGURE 20.

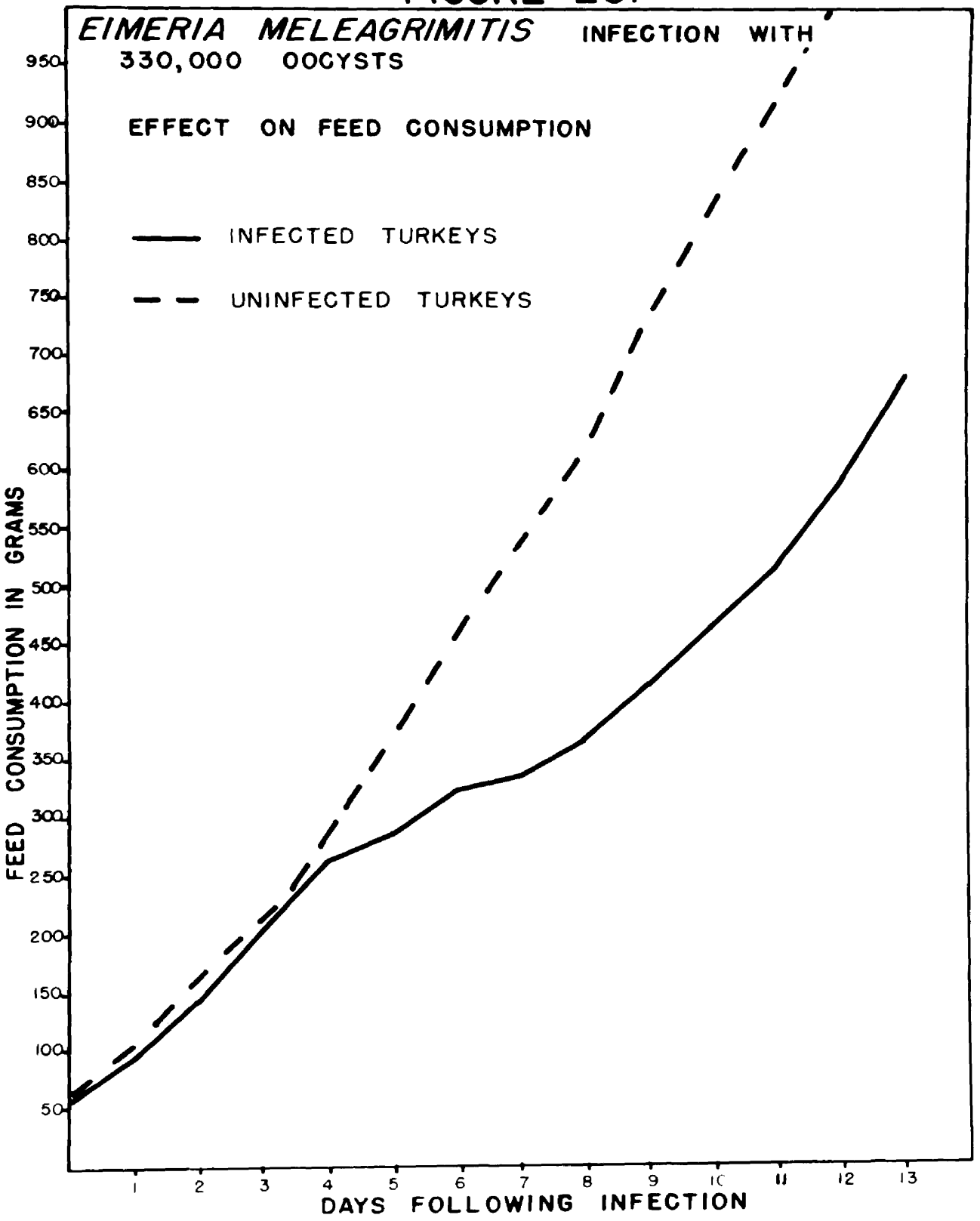
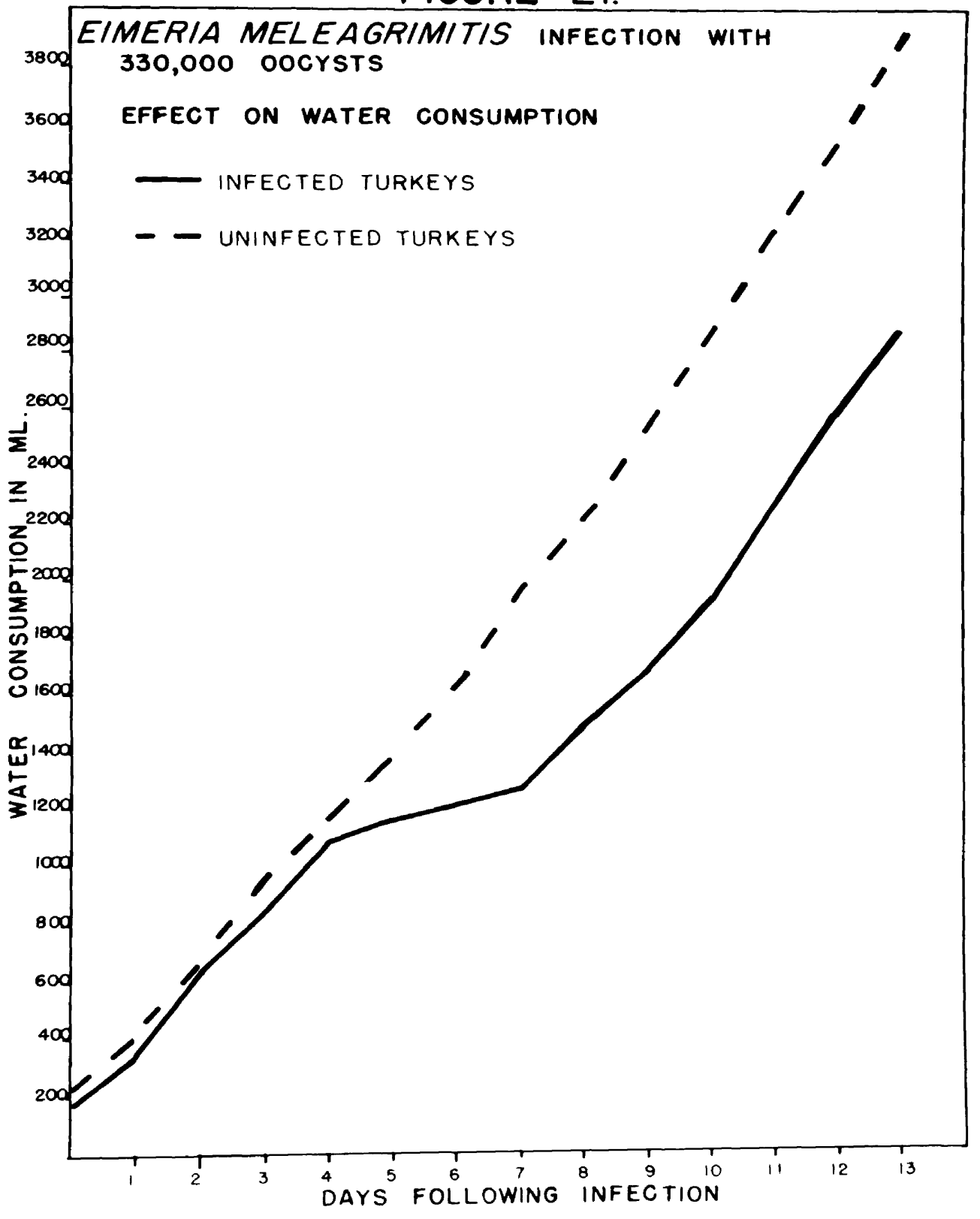

EIMERIA MELEAGRIMITIS INFECTION WITH
330,000 OOCYSTS
EFFECT ON FEED CONSUMPTION
INFECTED TURKEYS
UNINFECTED TURKEYS
FEED CONSUMPTION IN GRAMS
950
900
850
800
750
700
650
600
550
500
450
400
350
300
250
200
150
100
50
1
2
3
4
5
6
7
8
9
10
11
12
13
DAYS FOLLOWING INFECTION

FIGURE 21.


EIMERIA MELEAGRIMITIS INFECTION WITH 330,000 OOCYSTS
EFFECT ON WATER CONSUMPTION
INFECTED TURKEYS
UNINFECTED TURKEYS
WATER CONSUMPTION IN ML.
3800
3600
3400
3200
3000
2800
2600
2400
2200
2000
1800
1600
1400
1200
1000
800
600
400
200
1
2
3
4
5
6
7
8
9
10
11
12
13
DAYS FOLLOWING INFECTION

doses of E. meleagrimitis which will be described below.

Three turkeys were given an initial infection of 128,000 sporulated oocysts of E. meleagrimitis. This was followed by ten daily infections with 1280 sporulated oocysts, and then on the eleventh day 128,000 sporulated oocysts were again given. The course of this infection is presented in Figure 23. Late on the fifth day of infection a small number of oocysts were found and a peak of 399,400 oocysts per gram of feces was reached on the sixth day following infection. The numbers of oocysts declined very rapidly, and they had disappeared by the 24th day following infection, after having been present at a very low level for two weeks. Thus, this heavier infection is seen to have produced a higher degree of immunity as measured by oocyst counts than did the lower initial infection described above. From an inspection of the oocyst counts during the course of this infection, it appears that the majority of the oocysts passed resulted from the initial infection, the later infections producing practically no increase in the numbers of oocysts.

FIGURE 22

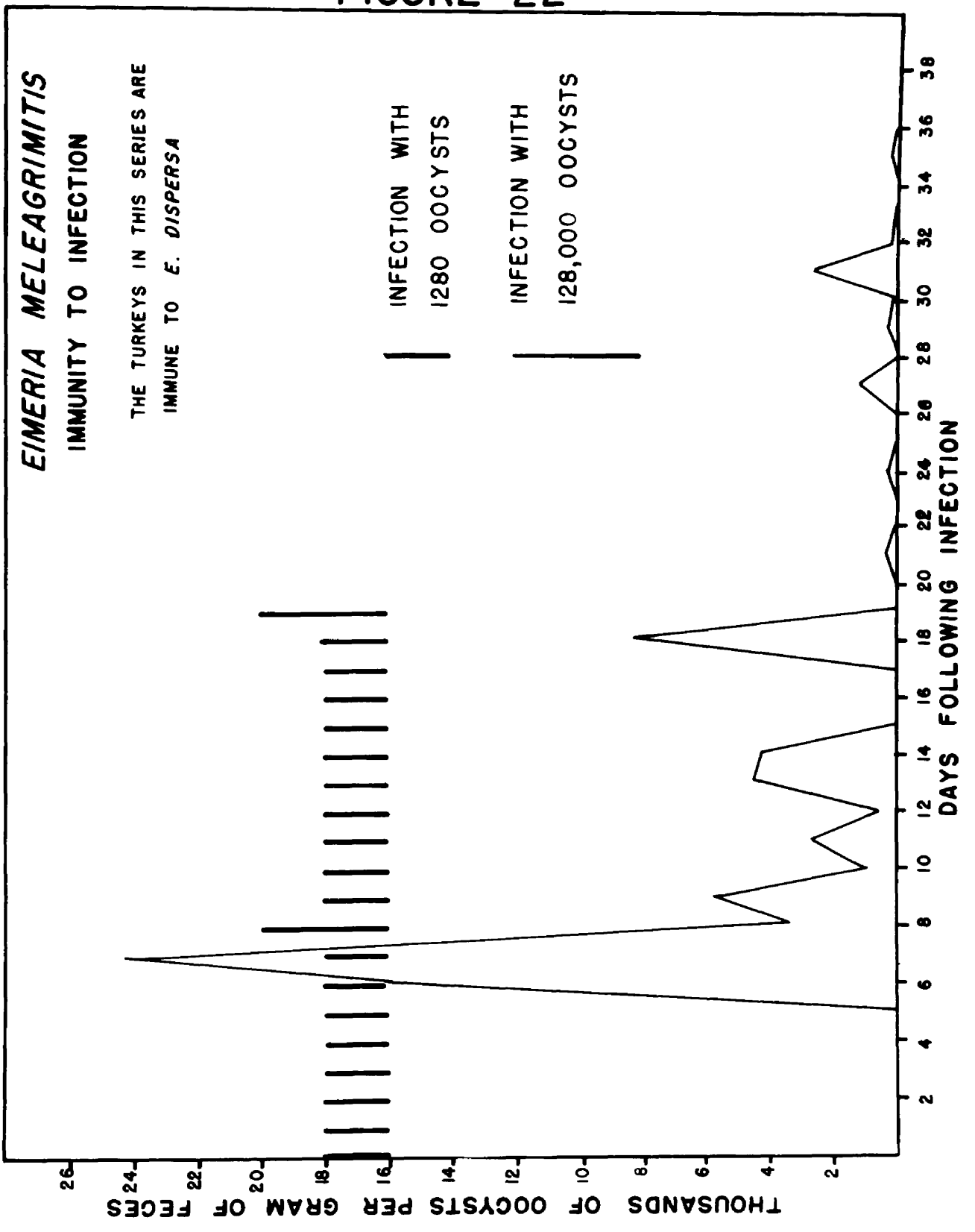
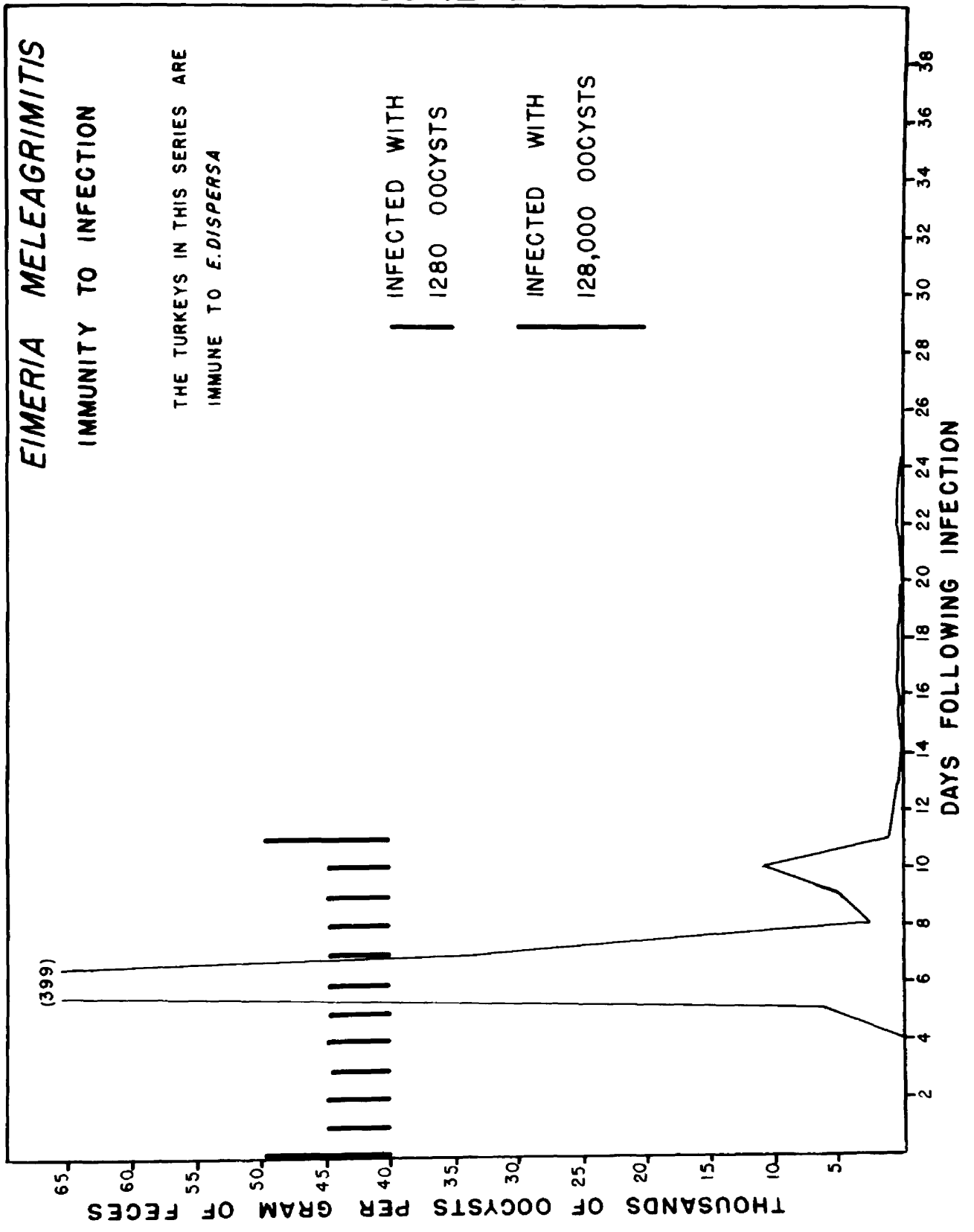

EIMERIA MELEAGRIMITIS
IMMUNITY TO INFECTION
THE TURKEYS IN THIS SERIES ARE IMMUNE TO E. DISPERSA
INFECTION WITH 1280 OOCYSTS
INFECTION WITH 128,000 OOCYSTS
THOUSANDS OF OOCYSTS PER GRAM OF FECES
DAYS FOLLOWING INFECTION

FIGURE 23.


EIMERIA MELEAGRIMITIS
IMMUNITY TO INFECTION
THE TURKEYS IN THIS SERIES ARE IMMUNE TO E.DISPERSA
INFECTED WITH 1280 OOCYSTS
INFECTED WITH 128,000 OOCYSTS
(399)
THOUSANDS OF OOCYSTS PER GRAM OF FECES
DAYS FOLLOWING INFECTION

Eimeria meleagridis Tyzzer, 1927

SOURCE

This species was originally described from the cecum of the turkey by Tyzzer (1927). It has been recovered in the course of this study from material received from Connecticut, Iowa, Maryland, Massachusetts, Michigan, Minnesota, Missouri, New Jersey, North Dakota, Oregon, Texas and Utah. The strains which were most thoroughly studied in the course of this work were obtained from Cache County, Utah through the courtesy of Dr. M. L. Miner and from Charles City, Iowa through the courtesy of Dr. N. F. Morehouse.

HOST

This species has been found to develop only in the turkey.

OOCYST

The oocysts of E. meleagridis were passed in the feces five days after infection. They measured 20.3 to 30.8 microns in length (average 24.4 microns) (see Figure 24), and 15.4 to 20.6 microns in width (average 18.12 microns). These dimensions were based on the measurement of 100 oocysts. the oocysts are ellipsoidal and have a length breadth ratio of 1.34 (see Figure 25). The cyst wall presented a double contoured appearance in constrast with the single contoured appearance of E. dispersa (see Plate VI-A). Rarely polar granules or inclusions are seen in the unsporulated oocysts, and they are not normally present in the freshly discharged oocyst.

After passage in the feces, approximately 24 hours were required for their sporulation. Polar granules were found in most of the sporulating oocysts 10 to 12 hours after their passage when they were maintained in 2.5 percent potassium dichromate at room temperature. These were present before the division of the cytoplasmic mass into sporoblasts. Some of the sporoblasts were noted to have a short, elongated handle at one end. Otherwise, the division and formation of the sporulated oocyst seemed to be similar to that described for E. dispersa (see Plate VI-B) (see Figure 7 C).

TISSUE STAGES

The earliest stages of this species were observed in the epithelial cells of the cecum 17 hours after infection. These were small, two to three microns in diameter, uninucleate, rounded up forms. These were the first generation trophozoites (Plate XI-A). Poults killed 48 hours after infection contained uninucleate trophozoites and schizonts, with some of the latter containing six to eight nuclei. The cytoplasm was observed dividing about some of the latter stages. The larger schizonts at this time measured about five microns in diameter. Both the trophozoites and the schizonts were very lightly stained, and their internal structure was difficult to study. They were also present in extremely small numbers, and yet careful examination of sections from other portions of the intestinal tract did not reveal the presence of this parasite.

The first asexual generation is terminated two and one

FIGURE 24.

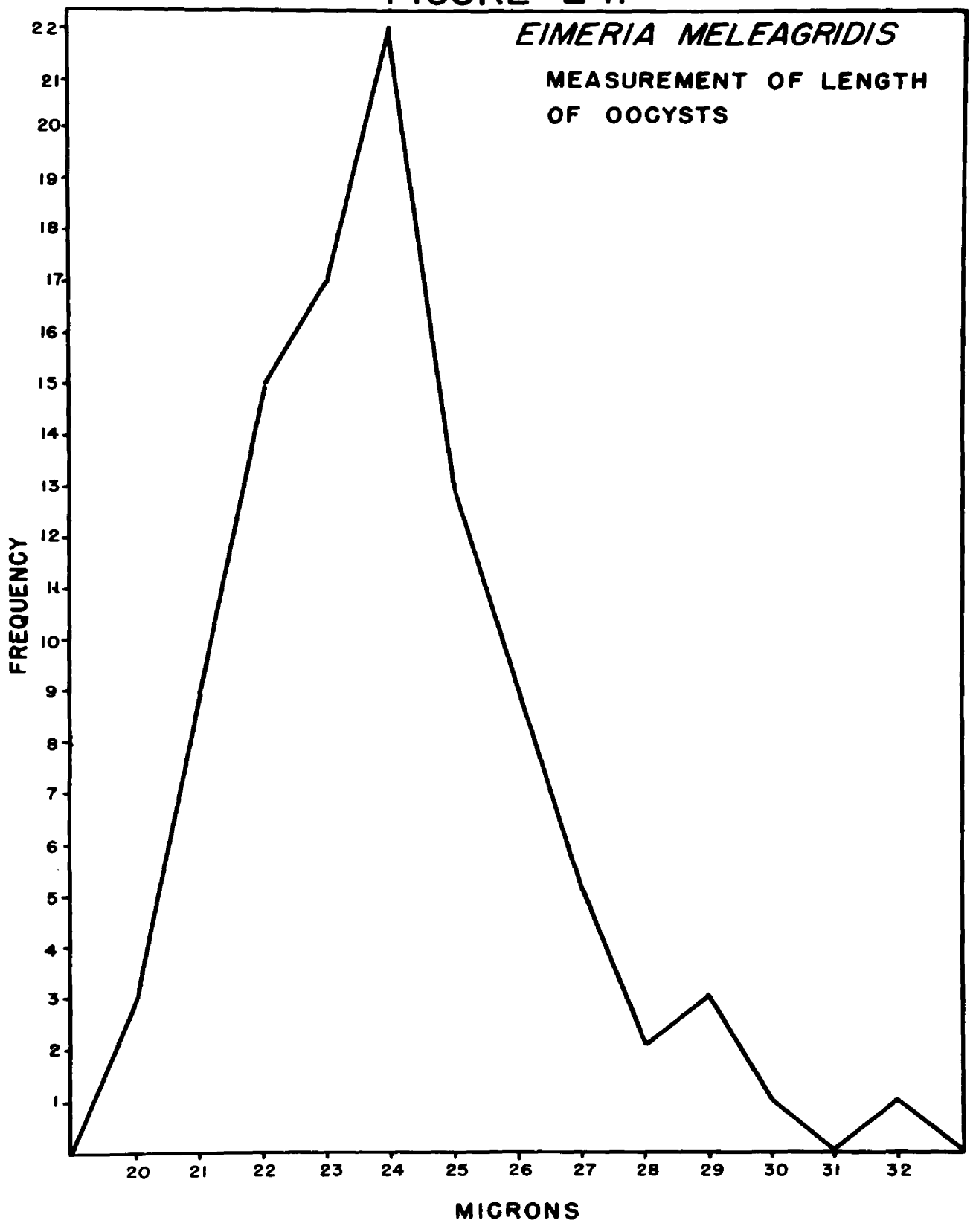
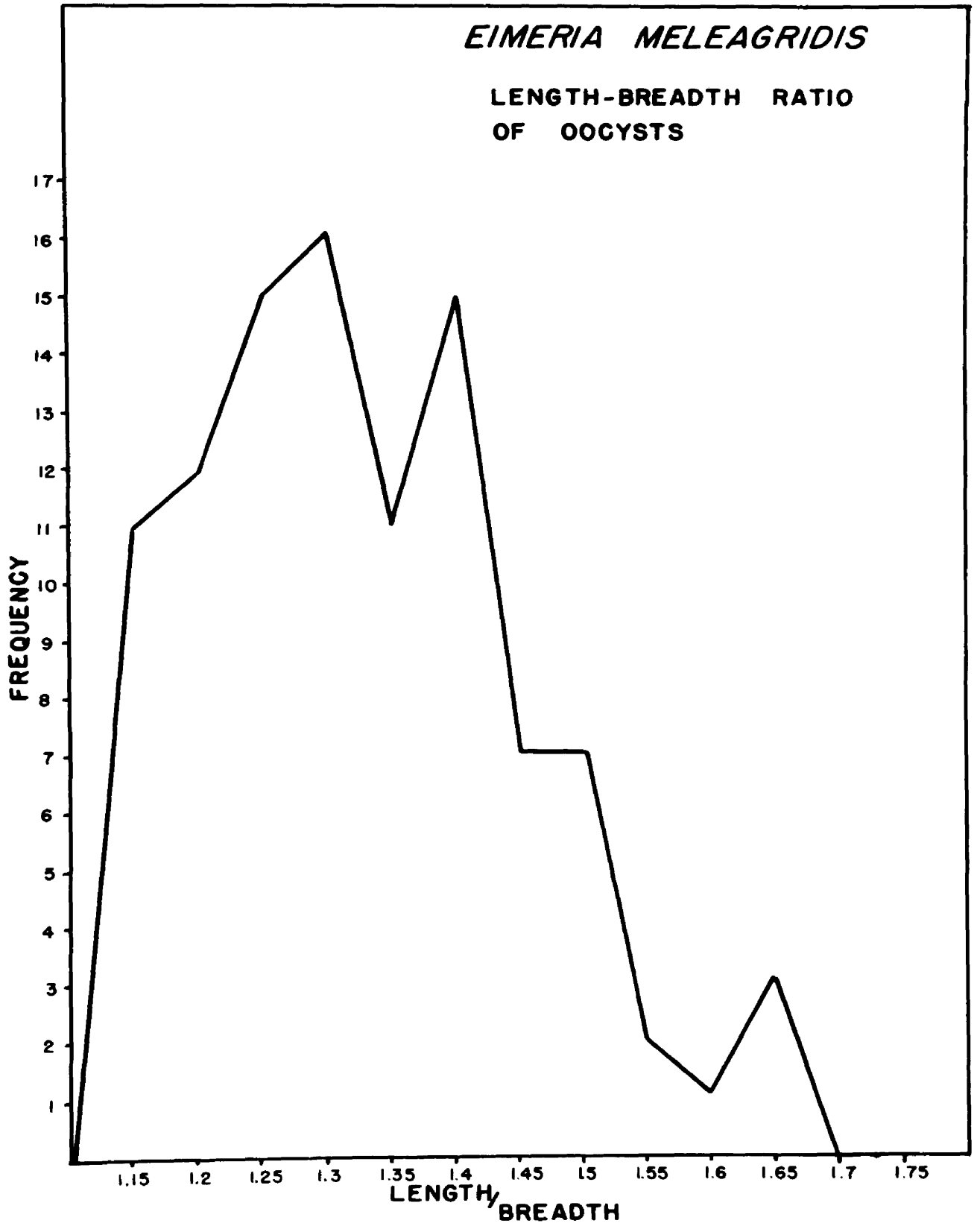

EIMERIA MELEAGRIDIS
MEASUREMENT OF LENGTH
OF OOCYSTS
FREQUENCY
22
21
20
19
18
17
16
15
14
13
12
11
10
9
8
7
6
5
4
3
2
1
20
21
22
23
24
25
26
27
28
29
30
31
32
MICRONS

FIGURE 25.


EIMERIA MELEAGRIDIS
LENGTH-BREADTH RATIO
OF OOCYSTS
FREQUENCY
17
16
15
14
13
12
11
10
9
8
7
6
5
4
3
2
1
1.15
1.2
1.25
1.3
1.35
1.4
1.45
1.5
1.55
1.6
1.65
1.7
1.75
LENGTH/BREADTH

half to three days after infection. Schizonts of the second generation may be seen developing in the epithelial cells of the cecum 73 hours after infection (see Plate XI-D). The second generation merozoites were completely formed three and one half to four days following infection. They were similar to those of the first generation. Each schizont contained 10 to 14 merozoites and there was a residual mass observed in many of them (Plate XI-D). This residual mass has not been observed in the schizonts of E. dispersa or E. meleagrimitis, but do occur in those of E. gallopavonis.

The third generation was predominantly sexual and could be found in the tissues four to four and one half days after infection. The fully formed macrogametocytes measured eight to ten microns in diameter and possessed a single, large, well developed nucleus. There were numerous large, lightly staining granules in the cytoplasm (see Plate XII-C and D). The macrogametes measured up to 20 microns in diameter, possessed a partially formed cyst wall and in some the granules were still present in the cytoplasm (see Plate XII-E). Some macrogametocytes were observed as late as eight days after infection (see Plate XII-E). The latter undoubtedly belong to a second sexual generation. It was observed that the sexual stages were frequently located deep in the epithelial cells, some almost touching the basement membrane. There was a tendency for them to be below the epithelial cell nucleus, and some of these nuclei had been displaced transversely to the long axis of the epithelial cell above the

parasite. The development of the microgametocytes and microgametes seemed to be typical (see Plate XII-A and B). Small numbers of asexual stages may be found occurring in the third generation.

GROSS PATHOLOGY

The lesions presented by this infection were typical and facilitated a gross diagnosis of the condition. No lesions were noted during the first two days of infection. The first evidence of infection was noted on the third day at which time the contents of the ceca assumed a granular appearance. Three and one half days following infection the ceca were observed to be cream colored when viewed from the serosal surface, and on cross section contained a non-adherent, cream colored cheesy plug next to the mucosa. The center of this plug contained normal feces, and the entire plug could be lifted out intact. At this stage the plug might also be of a soft mucoid consistency. A few petechial hemorrhages were also observed on the mucosa. Four days after infection the ceca appeared cream colored when inspected from the serosal surface. They contained a fairly solid, caseous plug without any evidence of fecal material inside of it. The mucosa was slightly thickened, and there were rows of petechial hemorrhages in the dilated portions of the ceca. These petechial hemorrhages were also present in the constricted portions of the ceca.

Five days after infection the plugs seemed to be resolving, as they had become smaller. There were few petechial

hemorrhages present in the mucosa. Five and one half days after infection the intact cecal plugs had disappeared and were only represented by small fragments. Scattered in the mucosa at this time were small cream colored foci, one to two millimeters in diameter, which when removed were found to consist of the caseous material which made up the plugs. Six days after infection the ceca were filled with a finely granular cream colored material and a few remaining fragments of the plugs. Enormous numbers of oocysts were observed on microscopic examination of the contents. Seven days after infection the ceca were relatively normal in appearance, and very few oocysts were noted. Thus, the characteristic lesion of this infection was the cecal plug. It should be noted that although petechial hemorrhages were frequently numerous on the cecal mucosa, there was rarely any suggestion of hemorrhage in the ceca.

HISTOPATHOLOGY

There were no changes observed in the cecal mucosa until two to two and one half days after infection when there appeared to be a slight edema and an increase in the number of lymphocytes in the lamina propria. Three days after infection the edema was slightly more noticeable and the capillaries were dilated more than normal (see Figure 26 B). Four and one half days after infection (see Figure 27 A) the lymphocytic infiltration was marked, although not comparable to that which occurred in infections with E. dispersa and E.

Plate XI - *Eimeria meleagridis*

A. First generation trophozoite in an epithelial cell of the cecum 17 hours after infection.

B. First generation schizont in an epithelial cell of the cecum 73 hours after infection.

C. First generation schizont containing merozoites in epithelial cell of the cecum 73 hours after infection.

D. First generation merozoites in an epithelial cell of the cecum 73 hours after infection.

E. Second generation schizont containing merozoites in epithelial cells of the cecum four days after infection.

F. Second generation schizonts containing merozoites in epithelial cells of the cecum three and one half days after infection.

PLATE XI

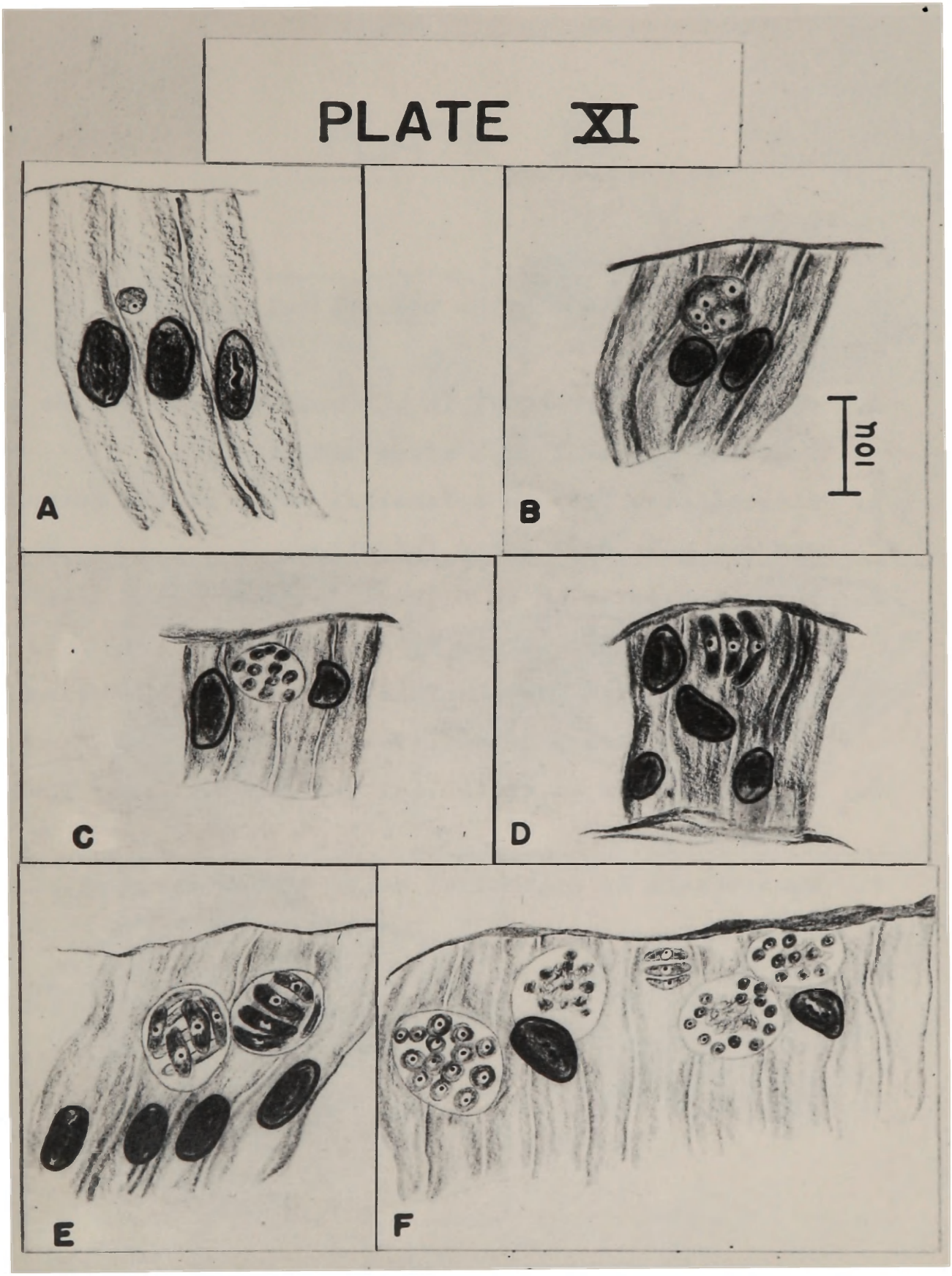


Plate XII - *Eimeria meleagridis*

A. Microgametocytes (MIC) in epithelial cells of the cecum four and one half days after infection.

B. Microgametes (MI) in epithelial cells of the cecum five and one half days after infection.

C. Macrogametocyte in an epithelial cell of the cecum four days and 17 hours after infection.

D. Macrogametocyte in an epithelial cell of the cecum four days and 17 hours after infection.

E. Macrogamete in an epithelial cell of the cecum five days after infection.

F. Gametocytes in epithelial cells of the cecum eight days after infection.

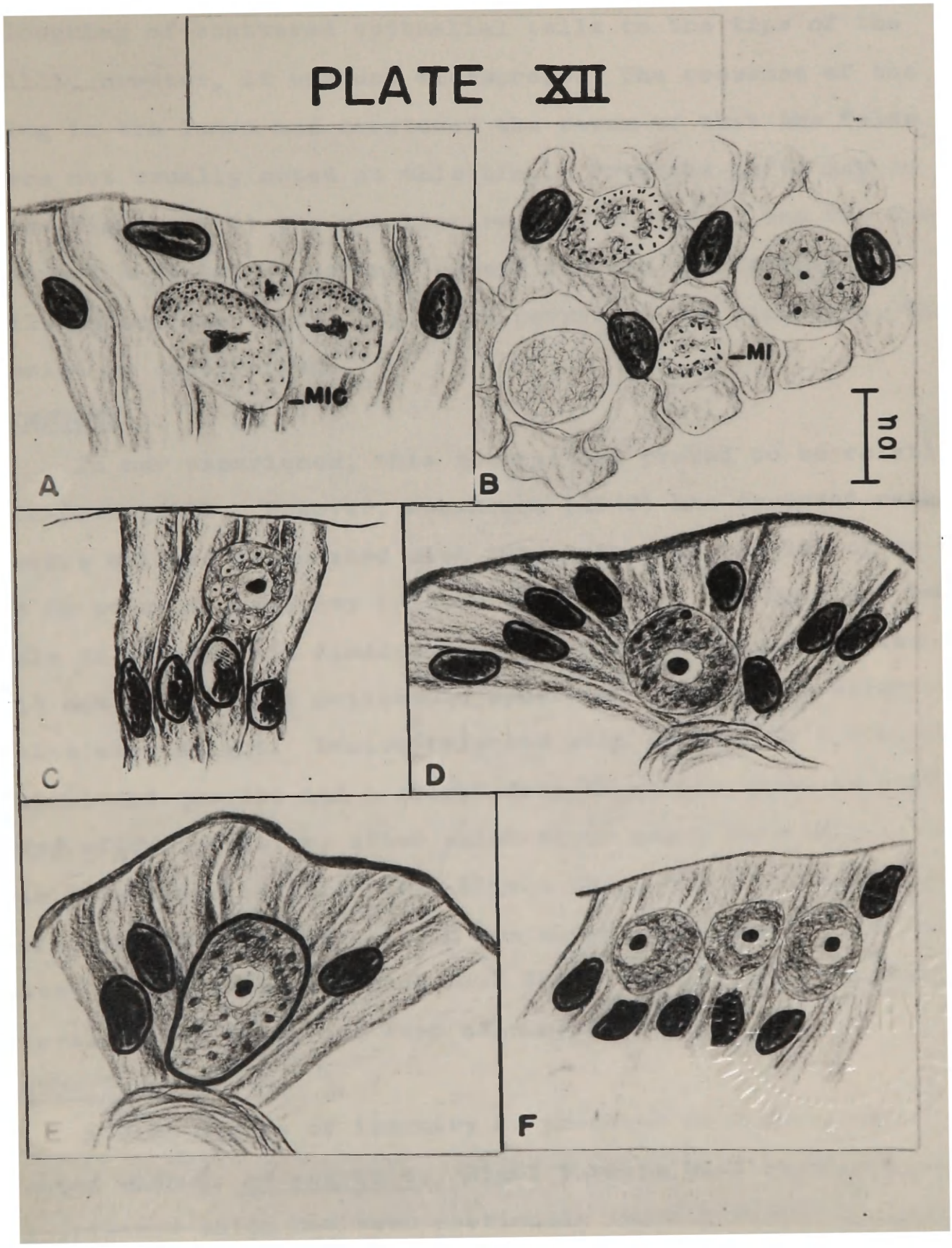


PLATE XII
MIC
MI
10u
A
B
C
D
E
F

meleagrimitis. At this time there had been some necrosis and sloughing of scattered epithelial cells on the tips of the villi, however, it was not widespread. The presence of the plug in the lumen had stretched the cecum so that the folds were not usually noted at this time. From the fifth day on (see Figure 27 B) the reaction rapidly subsided, and the ceca assumed a normal appearance except for the lymphocytic infiltration (see Figure 28) which persisted for nine days, the period of observation.

SYMPTOMS

In our experience, this species has proved to be relatively non-pathogenic. However, Morehouse (1949) has observed rather severe effects associated with this infection, including up to 50 percent mortality in some test groups. We have not been able to confirm his findings. Infections with this species did not produce any noticeable symptoms. Effects on weight gains were slight. Poults infected with 400,000 or 1,000,000 sporulated oocysts had a slight drop in weight three to four days after infection, after which their gains were very similar to uninfected birds (see Figure 29). The effects of the infection on feed consumption are shown in Figure 30, and on water consumption in Figure 31. There was observed a slight depression in water and feed consumption.

IMMUNITY

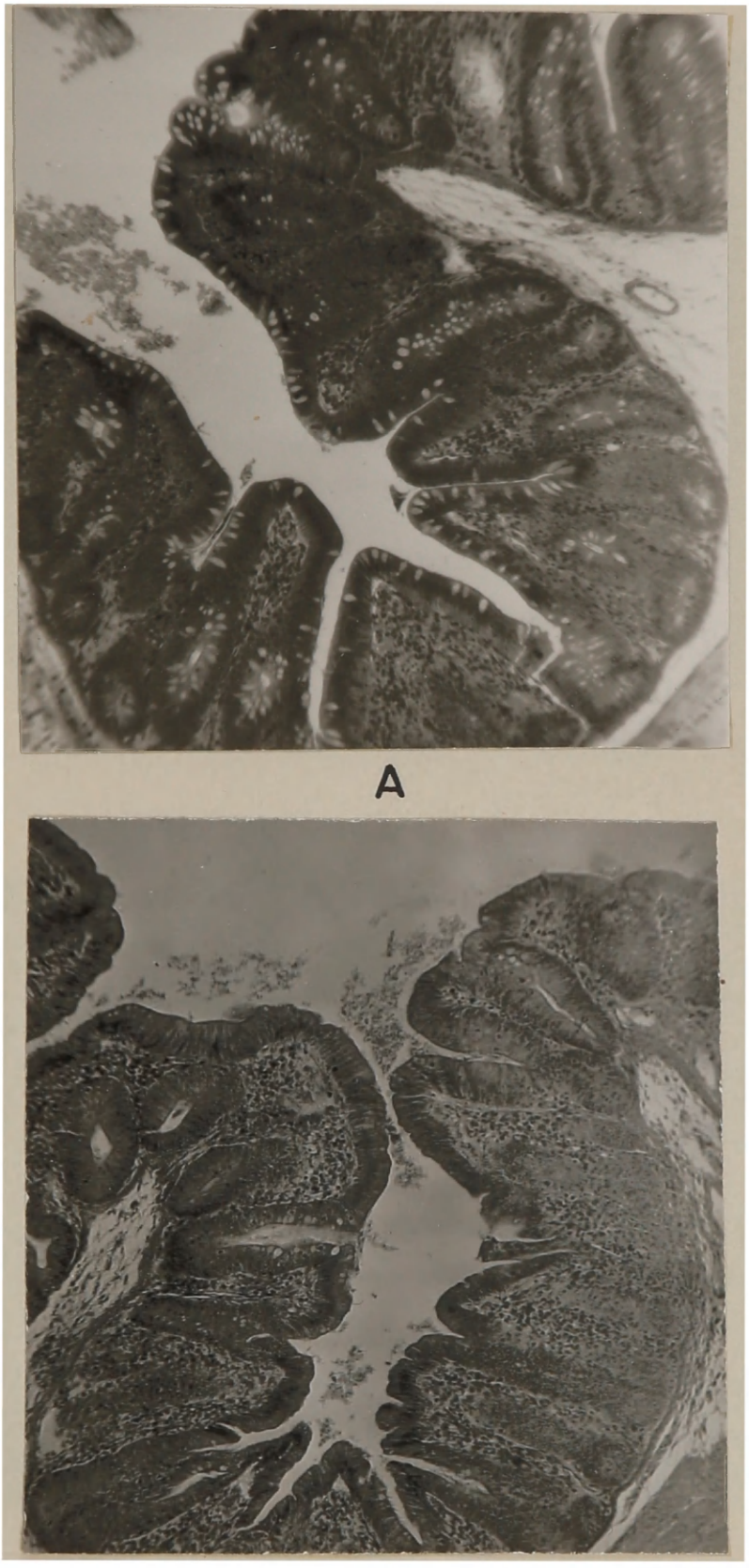
A high degree of immunity is produced in turkeys infected with E. meleagridis. Eight turkeys were used in these experiments which had been previously immunized with E. dispersa

Figure 26

A. Cecum of an uninfected two week old poult. (X77)

B. Cecum of a two week old poult infected with Eimeria meleagridis for three days. (X77)

FIGURE 26



A

B

Figure 27

A. Cecum of two week old poult infected with Eimeria meleagridis for four and one half days. (X77)

B. Cecum of two week old poult infected with Eimeria meleagridis for five days. (X77)

FIGURE 27

A

B

FIGURE 28

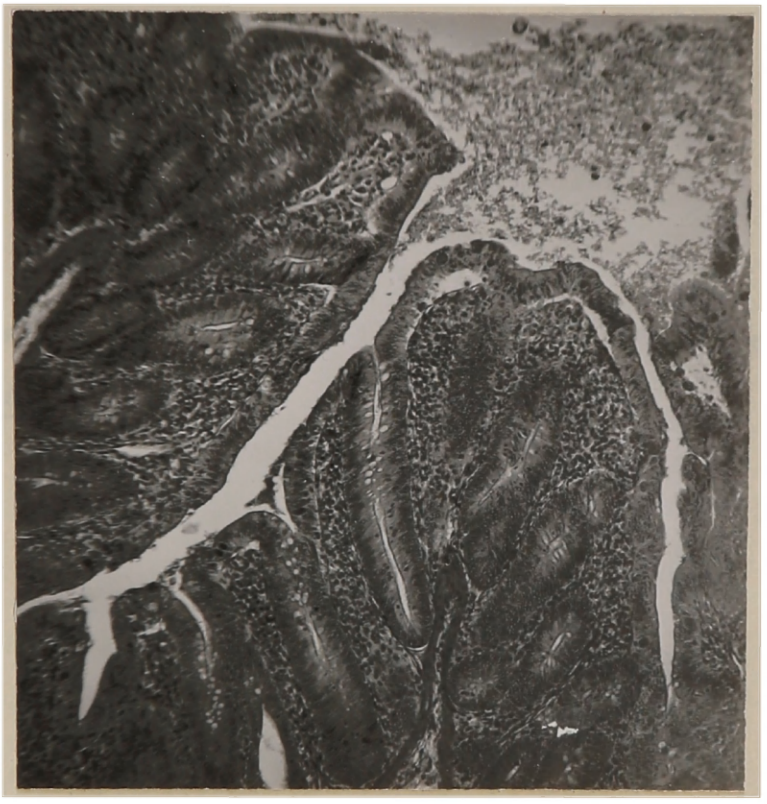


Figure 28

Cecum of two week old poult infected with *Eimeria meleagridis* for nine days. (X77)

and E. meleagrimitis as described on pp. 19 and 28. Four poults were infected with 1140 sporulated oocysts of E. meleagridis daily for 15 days and were then administered 108,000 sporulated oocysts. The course of this infection is shown in Figure 32. Oocysts were recovered in the feces five days after infection and reached a peak of 199,800 oocysts per gram of feces seven days after infection. They then declined very rapidly and had disappeared entirely by the eighteenth day following infection. The challenge infection of 108,000 sporulated oocysts failed to elicit any oocyst response, indicating that the sporozoites could not enter or failed to develop in the epithelial cells of the cecum.

Four turkey poults were given an initial infection of 108,000 sporulated oocysts of E. meleagridis, and 16 days later were again given the same number of oocysts. The course of this infection is shown in Figure 33. Five days after infection oocysts were recovered in the feces and they had reached a peak of 657,000 oocysts per gram of feces on the seventh day of infection. The numbers declined very rapidly, except for a secondary peak on the tenth day of infection, and oocysts were not recovered after the thirteenth day of infection. The challenge infection of 108,000 sporulated oocysts 16 days after the initial infection failed to elicit any oocyst response. Thus, although both the light and heavy initial infection resulted in the production of a high degree of immunity, there was a slightly more rapid disappearance of the oocysts in those birds given the heavier infection. It is also

of interest to note that the birds infected with the larger number of oocysts produced fewer oocysts per infecting oocyst than did those birds receiving the smaller number. Although only a rough comparison, the birds infected with 1140 sporulated oocysts produced at the peak of the infection, 175 oocysts per gram of feces per infecting oocyst, while those infected with 108,000 sporulated oocysts produced only six per infecting oocyst.

The larger numbers of oocysts produced by this species compared to that produced by the small intestinal species was probably more apparent than real, because counts of the oocysts of E. meleagridis were made from cecal droppings, which were less copious than those from the small intestines, hence the oocysts were concentrated.

FIGURE 29.

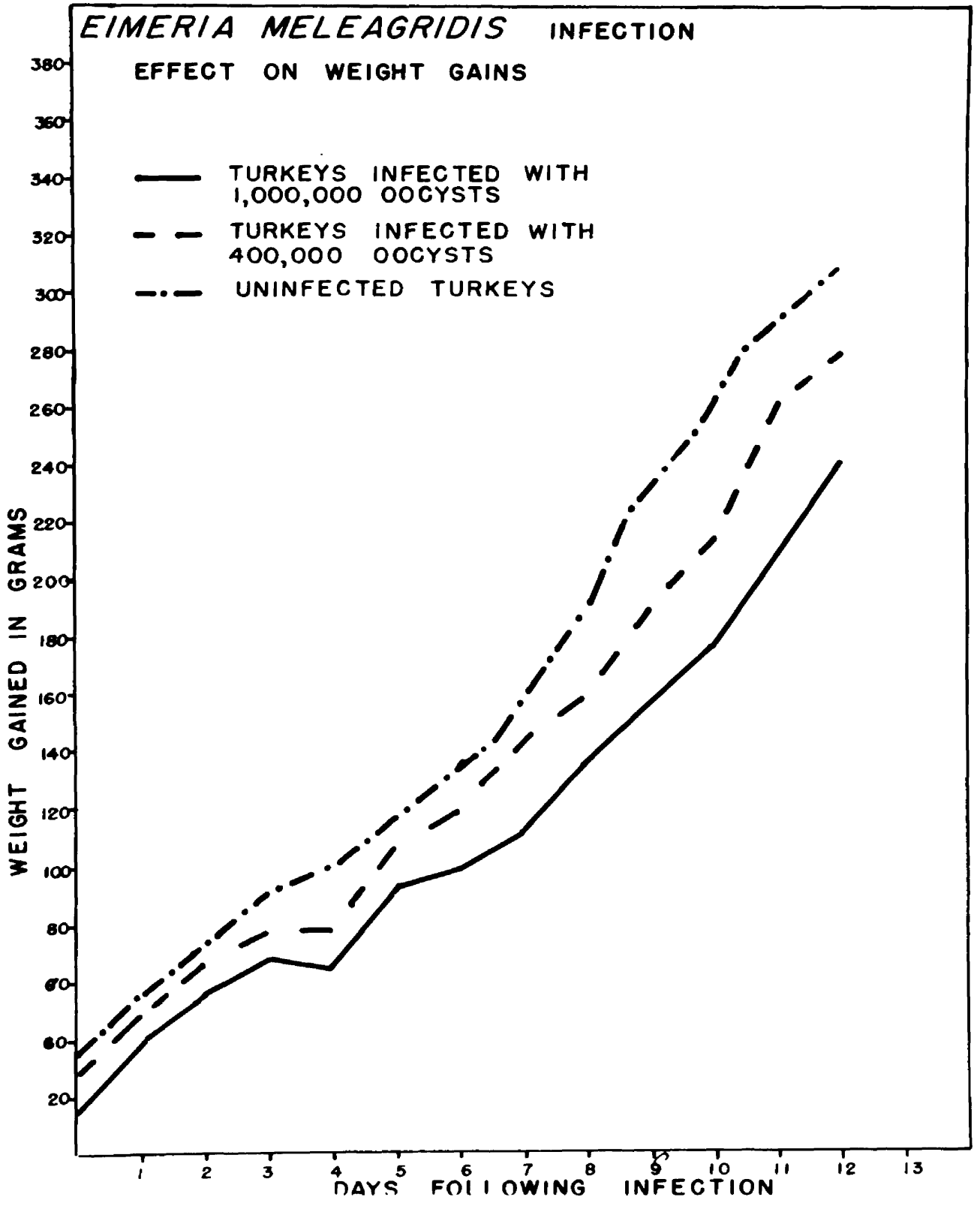

EIMERIA MELEAGRIDIS INFECTION
EFFECT ON WEIGHT GAINS
TURKEYS INFECTED WITH 1,000,000 OOCYSTS
TURKEYS INFECTED WITH 400,000 OOCYSTS
UNINFECTED TURKEYS
WEIGHT GAINED IN GRAMS
380
360
340
320
300
280
260
240
220
200
180
160
140
120
100
80
60
40
20
1
2
3
4
5
6
7
8
9
10
11
12
13
DAYS FOLLOWING INFECTION

FIGURE 30.

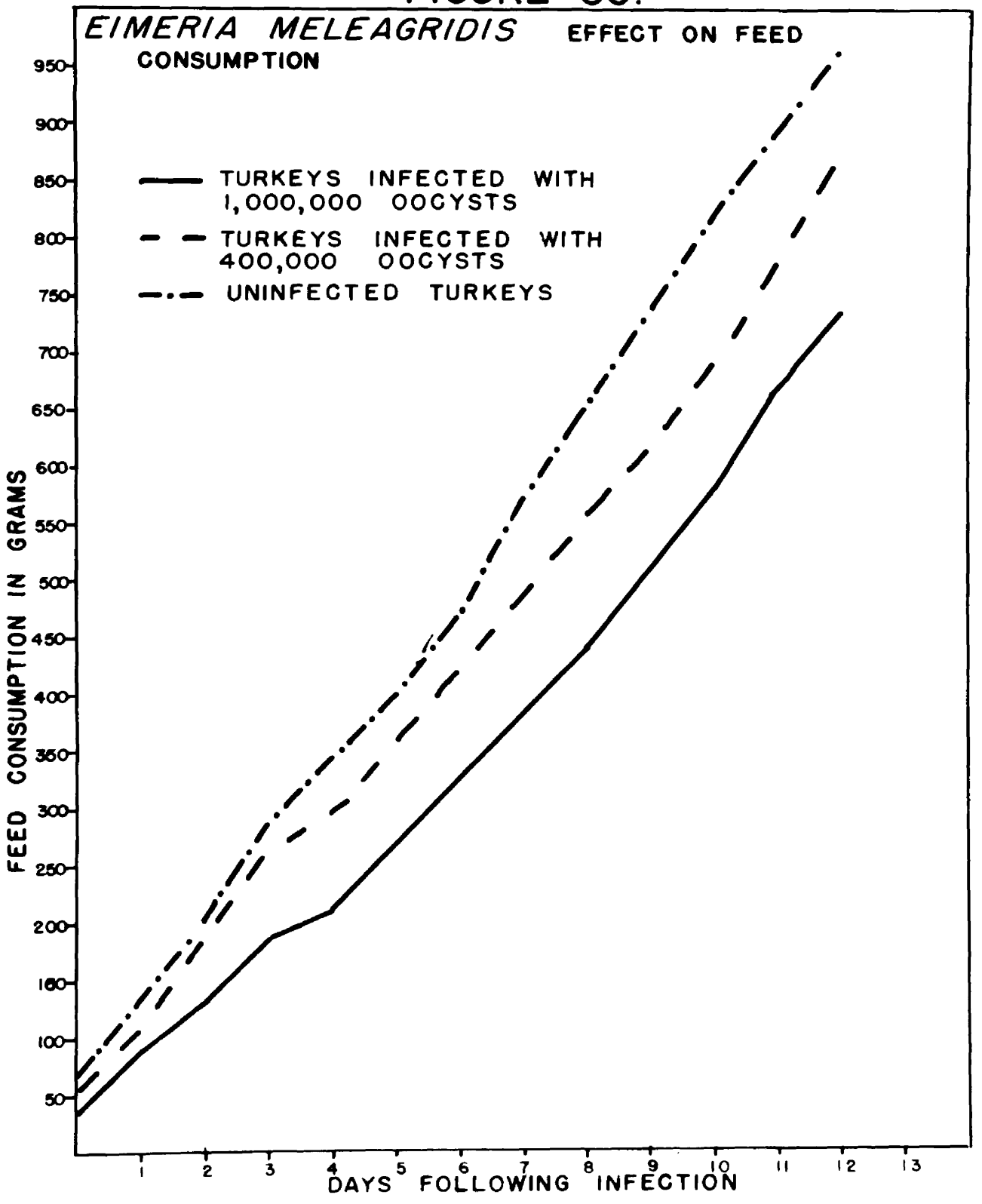

EIMERIA MELEAGRIDIS EFFECT ON FEED CONSUMPTION
TURKEYS INFECTED WITH 1,000,000 OOCYSTS
TURKEYS INFECTED WITH 400,000 OOCYSTS
UNINFECTED TURKEYS
FEED CONSUMPTION IN GRAMS
950
900
850
800
750
700
650
600
550
500
450
400
350
300
250
200
150
100
50
1
2
3
4
5
6
7
8
9
10
11
12
13
DAYS FOLLOWING INFECTION

FIGURE 31.

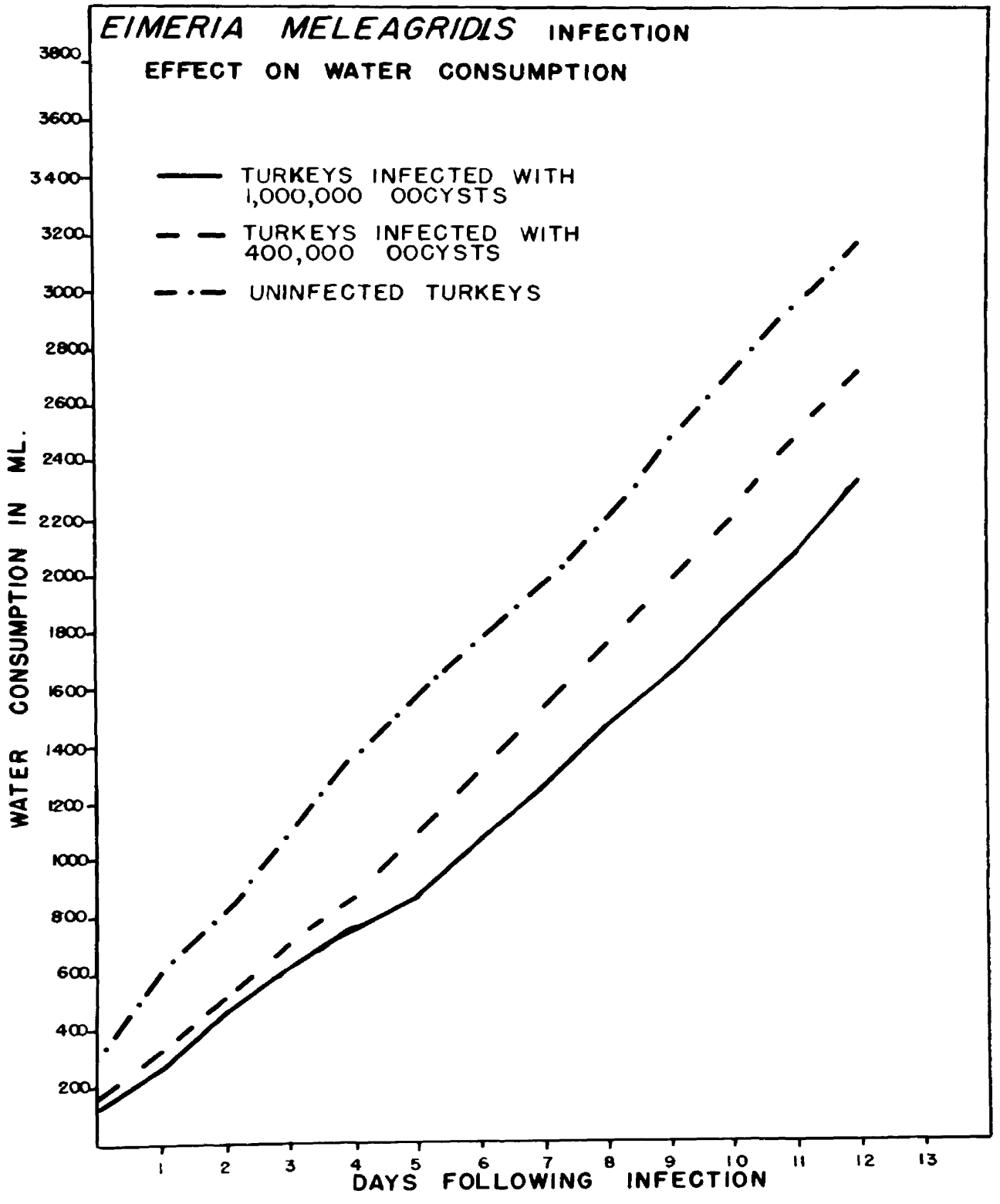

EIMERIA MELEAGRIDIS INFECTION
EFFECT ON WATER CONSUMPTION
TURKEYS INFECTED WITH 1,000,000 OOCYSTS
TURKEYS INFECTED WITH 400,000 OOCYSTS
UNINFECTED TURKEYS
WATER CONSUMPTION IN ML.
3800
3600
3400
3200
3000
2800
2600
2400
2200
2000
1800
1600
1400
1200
1000
800
600
400
200
1
2
3
4
5
6
7
8
9
10
11
12
13
DAYS FOLLOWING INFECTION

FIGURE 32.

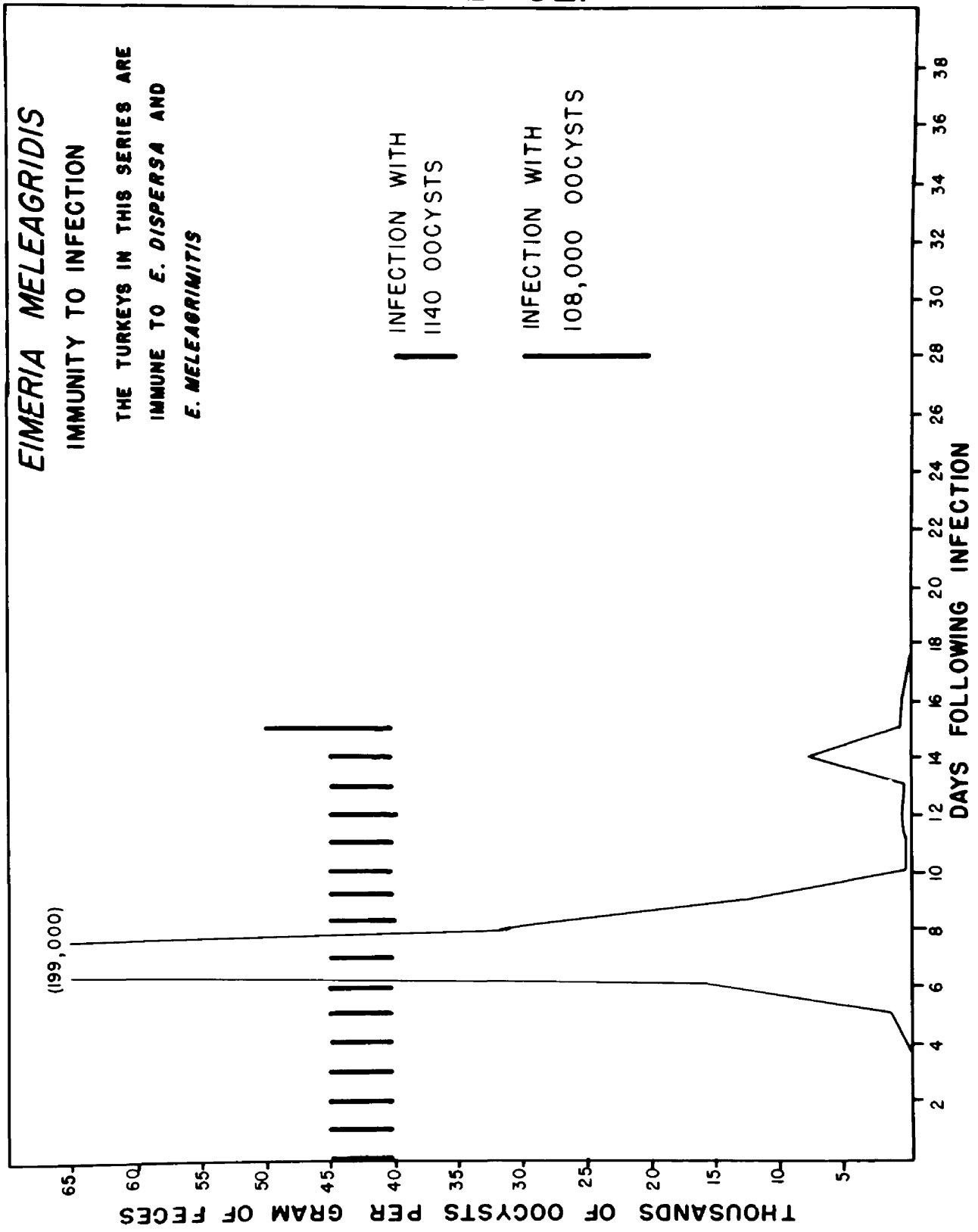
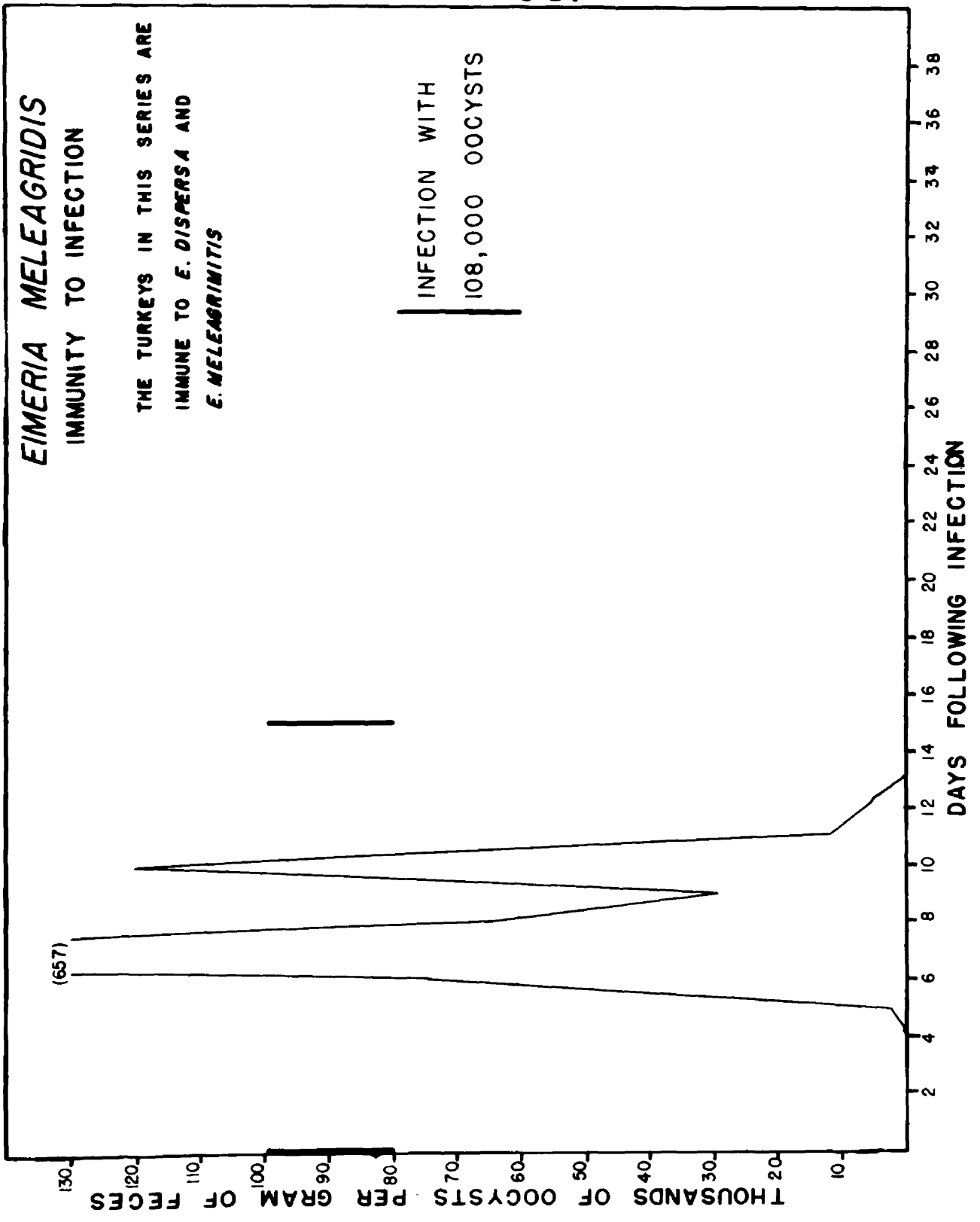

EIMERIA MELEAGRIDIS
IMMUNITY TO INFECTION
THE TURKEYS IN THIS SERIES ARE IMMUNE TO E. DISPERSA AND E. MELEAGRIMITIS
INFECTION WITH 1140 OOCYSTS
INFECTION WITH 108,000 OOCYSTS
(199,000)
THOUSANDS OF OOCYSTS PER GRAM OF FECES
DAYS FOLLOWING INFECTION

FIGURE 33.


EIMERIA MELEAGRIDIS
IMMUNITY TO INFECTION
THE TURKEYS IN THIS SERIES ARE IMMUNE TO E. DISPERSA AND E. MELEAGRIMITIS
INFECTION WITH 108,000 OOCYSTS
(657)
THOUSANDS OF OOCYSTS PER GRAM OF FECES
DAYS FOLLOWING INFECTION

Eimeria gallopavonis new species

SOURCE

This species was isolated only once, and then from material received from Worchester County, Massachusetts through the courtesy of Dr. G. Snoeyenbos. This species was isolated in a pure culture by the administration of oocysts to turkey poults which were immune to E. dispersa, E. meleagrimitis and E. meleagridis. An infection was produced by this method which proved to be different from the other three occurring in the turkey.

HOSTS

Attempts were made to establish this species in the pheasant, Hungarian partridge and Bob-white quail. No infection occurred in three mature pheasants to which 41,000 sporulated oocysts of this species were administered. It was not possible to obtain coccidia free pheasants, so that this attempt at infection cannot be regarded as conclusive evidence that they were not susceptible to this species. Four Hungarian partridges were infected with 41,000 sporulated oocysts and a mild infection developed. Oocyst counts of this species in the partridge are given in Table 1. In the case of the infection of the Hungarian partridge, as in the pheasant, coccidia free birds were not obtainable, although they had not been passing oocysts in their feces for several weeks prior to this infection. Twelve Bob-white quail were given 41,000 sporulated oocysts of this species, but they did not become infected.

Table 1.

Infection of the Hungarian partridge with

E. gallopavonis.Oocyst counts.

Date		Oocysts per gram of feces
December	22	0
	23	0
	24*	0
	25	0
	26	0
	27	0
	28	0
	29	0
	30	4,500
	31	10,000
January	1	11,900
	2	11,000
	3	0
	4	0
	5	0
	6	0
	7	0

* Date of infection.

OOCYST

The oocysts of this species are passed in the feces six days after infection. They measured 22.2 to 32.7 microns in length (average 27.1 microns) (see Figure 34), and 15.2 to 19.4 microns in width (average 17.23 microns). These dimensions were based on the measurement of 100 oocysts. The oocysts are long ellipsoidal and have a length breadth ratio of 1.52 (see Figure 7 - D) which is the largest of any of the coccidia which so far have been encountered in the turkey. There is no perceptible micropyle, although the area over one end is slightly paler than the rest of the cyst wall. The cyst walls appeared as double refractile lines giving a characteristic double contoured appearance, (see Figure 7 - D and Plate VI C).

The oocysts from the turkey which most closely resemble those of this new species are those of E. meleagridis. The oocysts of these two species could not be differentiated with any certainty. These oocysts are also very similar to those of E. phasiani occurring in the pheasant, and although at the time of the study no oocysts of this species were available it is doubtful if they could be defferentiated. However, it has not been possible to establish this species in the pheasant, although the limited number of pheasants used makes this attempt inconclusive. The prepatent period for E. phasiani is five days in the pheasant, whereas the prepatent period of E. gallopavonis in the

FIGURE 34.

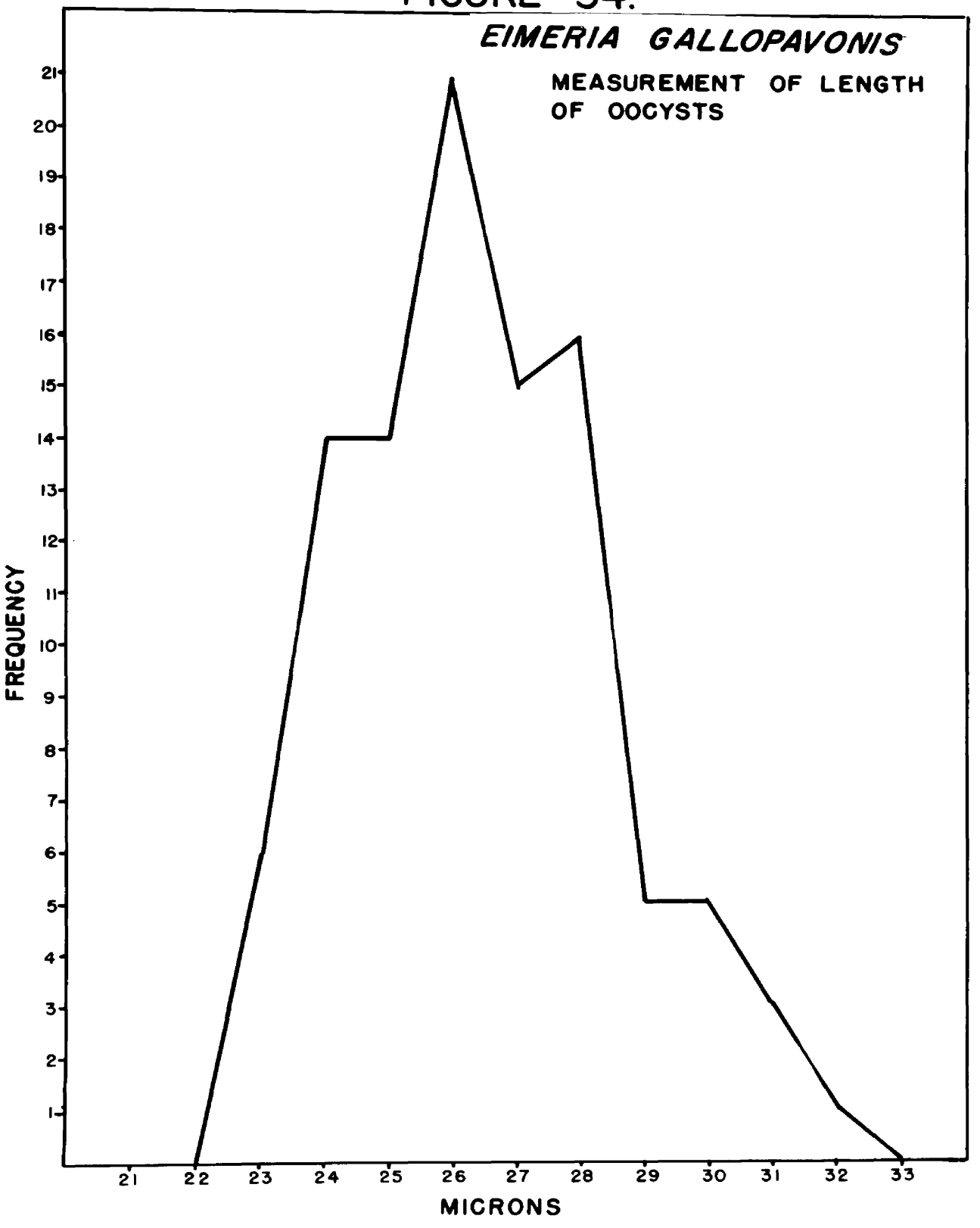
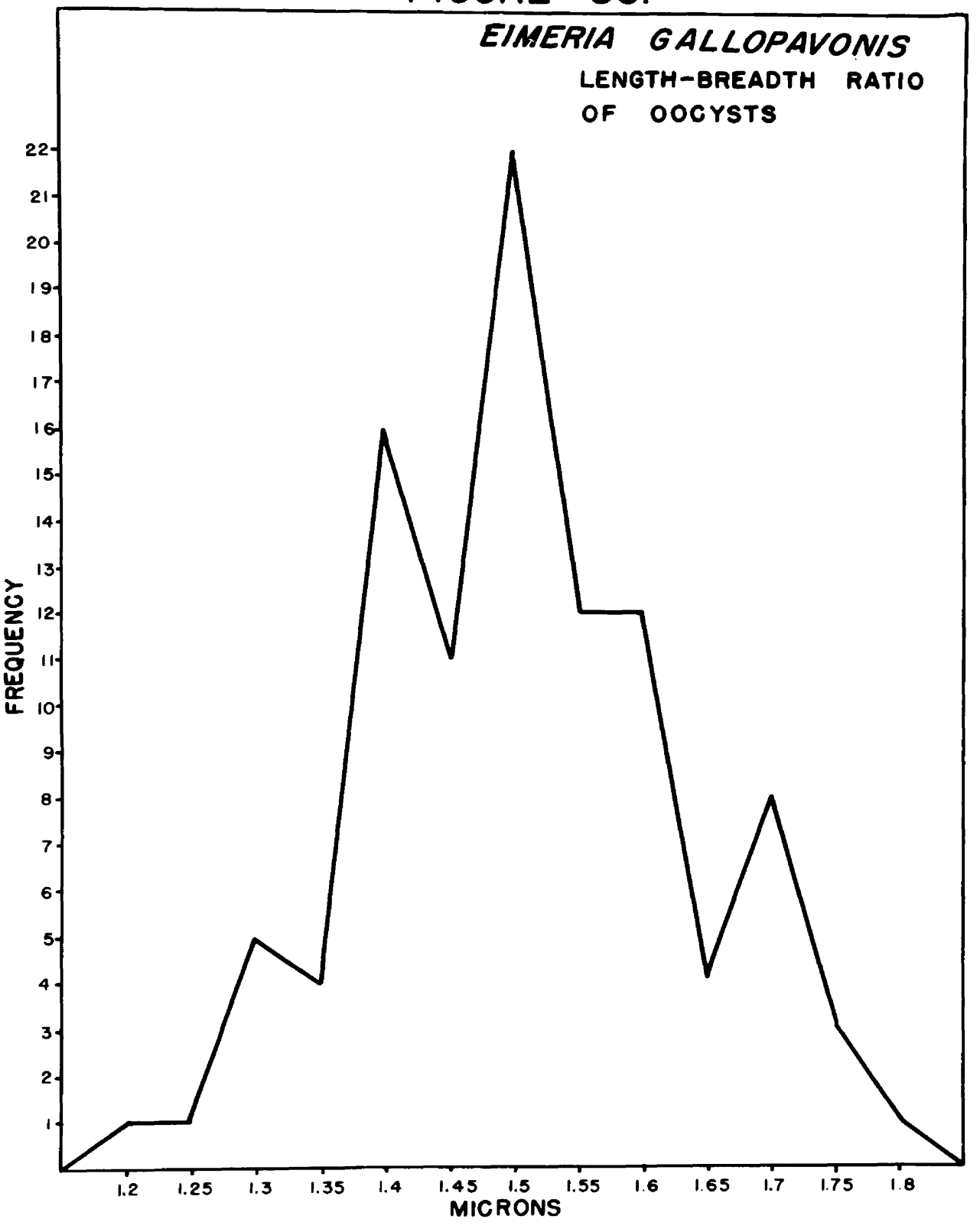

EIMERIA GALLOPAVONIS
MEASUREMENT OF LENGTH OF OOCYSTS
FREQUENCY
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
21
22
23
24
25
26
27
28
29
30
31
32
33
MICRONS

FIGURE 35.


EIMERIA GALLOPAVONIS
LENGTH-BREADTH RATIO
OF OOCYSTS
FREQUENCY
22
21
20
19
18
17
16
15
14
13
12
11
10
9
8
7
6
5
4
3
2
1
1.2
1.25
1.3
1.35
1.4
1.45
1.5
1.55
1.6
1.65
1.7
1.75
1.8
MICRONS

turkey is six days. Furthermore, Tyzzer (1929) was not able to infect the turkey with E. phasiani.

The sporulation time for this species was 24 hours, or slightly more, when it was kept in 2.5 percent potassium dichromate at room temperature. Several hours after passing in the droppings of infected poults, the cytoplasmic mass was observed to possess small polar protrusions at one end, and occasionally on both (see Plate VI). Whether the polar granules were formed in this manner was not observed, but at this time no polar granules were present in the oocysts. Fully sporulated oocysts were observed in about 24 hours.

TISSUE STAGES

No turkeys infected with this species were available for study prior to three days after infection. At this time a few schizonts containing merozoites were present in the ileum and rectum. These schizonts resembled those produced in the cecum by E. meleagridis in that they were found to possess a residual mass. Approximately eight merozoites were found in each schizont (see Plate XIII A B). It was assumed that these stages represented the end of the first asexual generation. Four days after infection, stages of the second asexual generation were found in the ileum, cecum and rectum. The stages which were found in the ileum and cecum were small unimucleate trophozoites and were rare. They measured three to five microns in diameter. In the rectum the parasites were most numerous

at this stage of the infection, and there were apparently two different types of development. There were numerous uninucleate forms (see Plate XIII D) and also a number which contained three to five nuclei (see Plate XIII C D). There was no evidence in any of these of division of the cytoplasm about the nuclei, most of which were found located in lighter staining vacuoles. In addition to these small forms, there were also observed very large schizonts containing large numbers of merozoites. The former were approximately 20 microns in diameter, and contained a large undetermined number of small merozoites (see Plate XIV A). These large schizonts resembled those observed in the duodenum in infections with E. dispersa two to three days after infection. The main differences were that these were slightly larger, occurred four days after infection and were located in the rectum. They have been observed in no other location. The precursors of this large schizont could not be determined from the material available for study, but it is fairly certain that the small forms described above four days after infection do not develop into the large schizonts. No other schizonts containing merozoites were found in the tissues of the intestinal tract four days after infection.

Five days after infection numerous small schizonts containing 10 to 12 merozoites were found in both the ileum and rectum, and a small number had localized in the ceca. These were very similar to the asexual stages de-

scribed in the first generation (see Plate XIII A B), with most of the schizonts containing a residual mass. No large schizonts were found at this time. In addition to the asexual stages which were most numerous, gametocytes were also present (see Plate XIV C). Small numbers of these sexual stages were localized in the ileum, ceca and rectum.

Six days after infection the rectum was most heavily parasitized with only occasional forms being located in the ileum and ceca and for the first time rare forms were also observed in the posterior portions of the jejunum. A few asexual stages were present, but the predominent forms were sexual, and it was assumed that these represented the third generation. A few schizonts containing three to six nuclei were observed, but most were larger and contained 10 to 12 nuclei (see Plate XIV D). Gametocytes (see Plates XIV D and XV A) were very numerous at this stage of the infection. They contained a single nucleus located in a lightly staining vacuole and a finely granular cytoplasm. They measured from five to ten microns in diameter. At the same time macrogametocytes were also noted (see Plate XV A).

Seven days after infection the epithelial cells of the rectum were very heavily parasitized and contained mostly macrogametocytes and macrogametes. Some large schizonts, as observed six days after infection, were also present. In the material available for study very few microgametocytes and microgametes were noted, although those that were found seemed to be similar to those present in the other

species of the turkey.

PATHOLOGY AND SYMPTOMS

Conclusions concerning the lesions produced by this species could not be drawn from the small amount of material available. However, from a study of sections it was noted that very marked edema, sloughing and lymphocytic infiltration had occurred. In the original report received concerning this material, Dr. Snoeyenbos of the University of Massachusetts states, "Birds showed marked ulceration of the mucosa of the lower small intestine, with a yellow exudate over the ulcers". He obtained this material from a flock of 500 six week old turkey poults which had suffered a mortality of two percent at the time a diagnosis was made, the loss apparently produced by this coccidium. Further study is required to determine the pathogenicity of this species.

IMMUNITY

Six turkey poults which were immune to E. dispersa, E. meleagrimitis and E. meleagridis were infected with 41,000 sporulated oocysts of E. gallopavonis. Six days after infection oocysts were recovered in the feces, and on the seventh day after infection a peak of oocyst production of 84,400 oocysts per gram of feces was observed. The oocysts in the droppings declined rapidly until they had disappeared by the fourteenth day following infection. Fifteen days following the original infection the same poults were

Plate XIII - *Eimeria gallopavonis*

A. B. First generation schizonts containing merozoites in epithelial cells of the rectum three days after infection.

C. D. Second generation schizonts in epithelial cells of the rectum four days after infection.

PLATE XIII

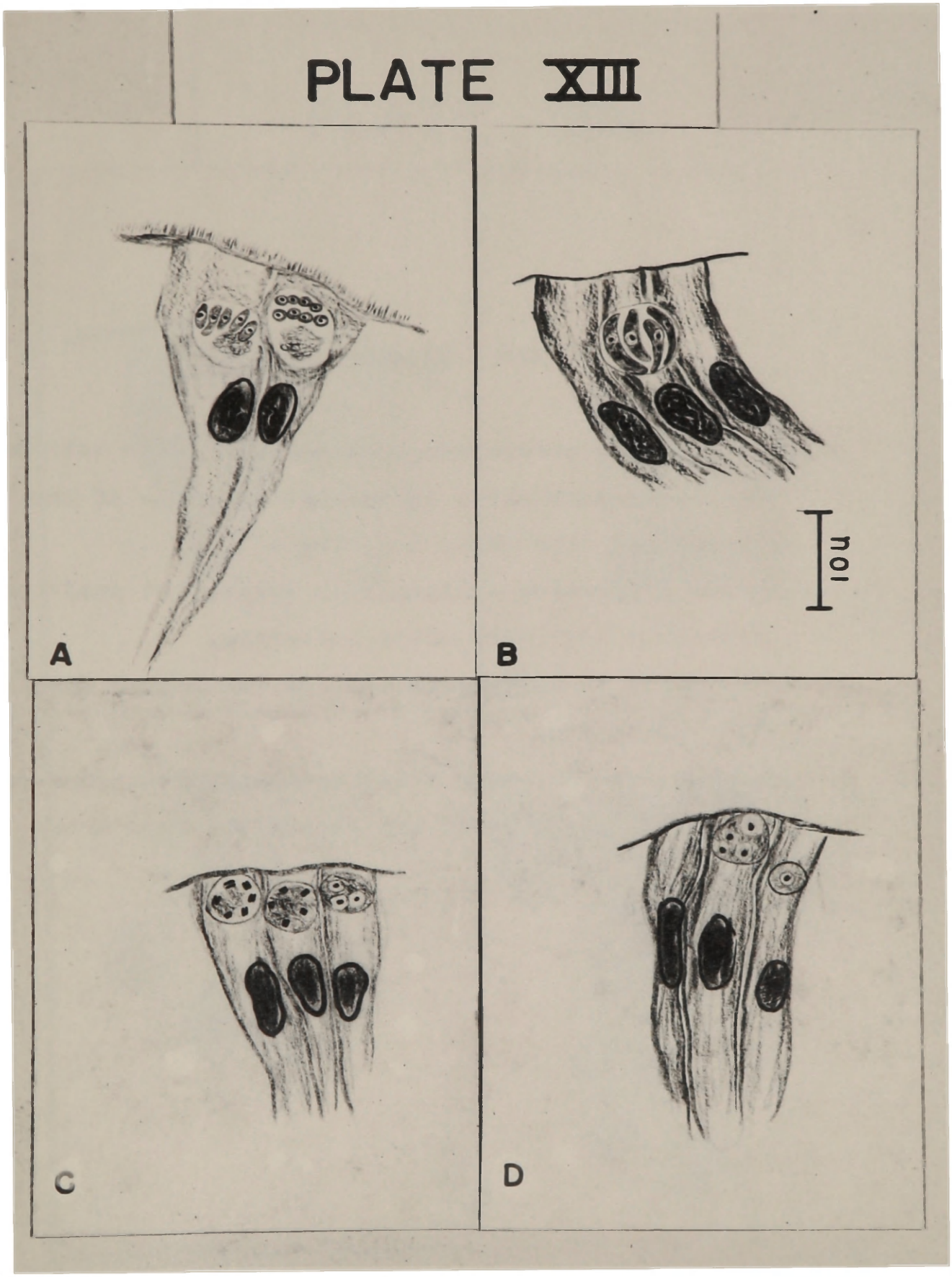


Plate XIV - *Eimeria gallopavonis*

A. Small second generation schizont and large schizont containing merozoites in epithelial cells of the rectum four days after infection.

B. Second generation schizonts in epithelial cells of the rectum four days after infection.

C. Gametocyte in epithelial cell of the rectum four days after infection.

D. Gametocytes (G) and a third generation schizont (S) in epithelial cells of the rectum six days after infection.

PLATE XIV

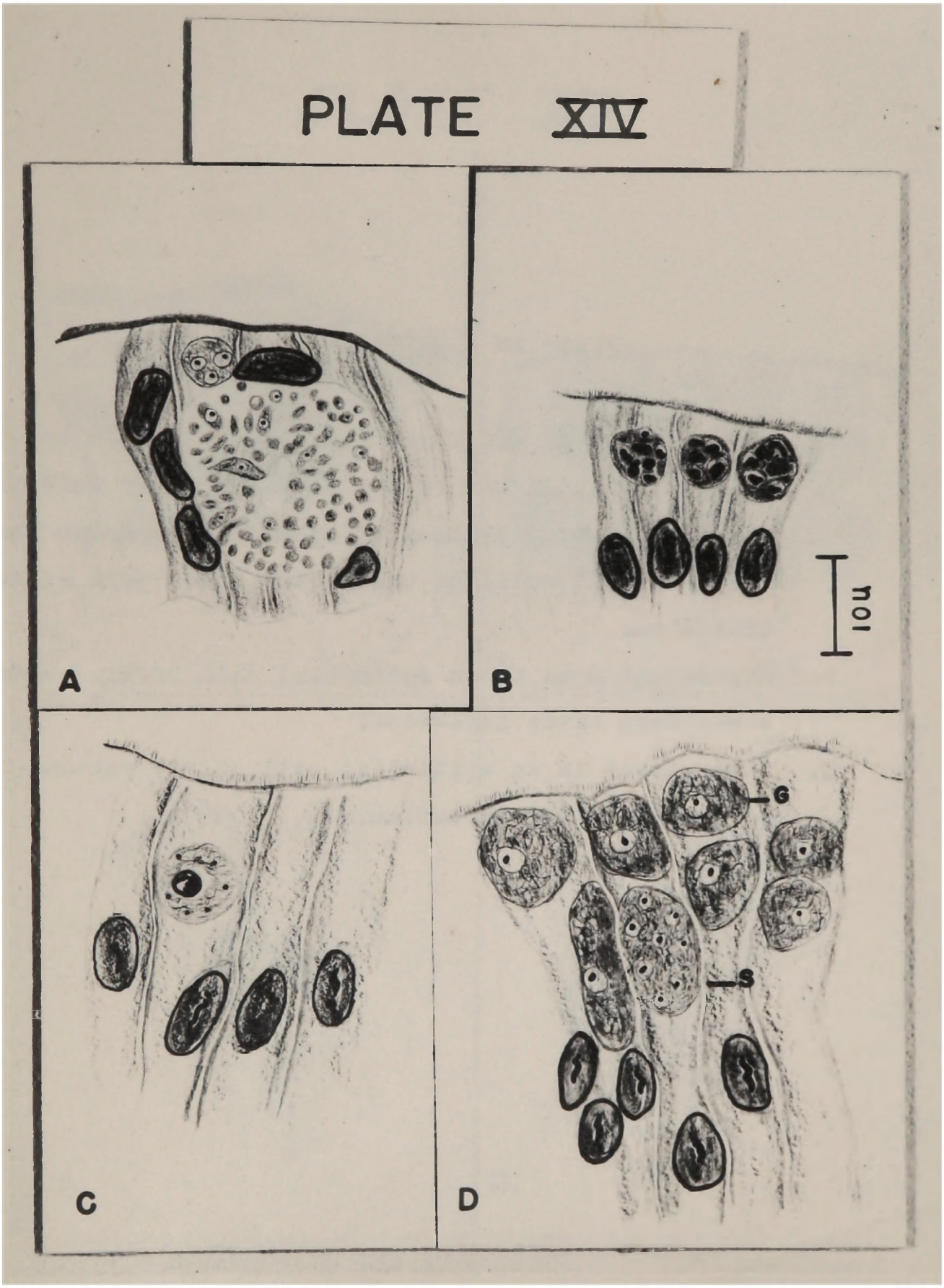


Plate XV - *Eimeria gallopavonis*

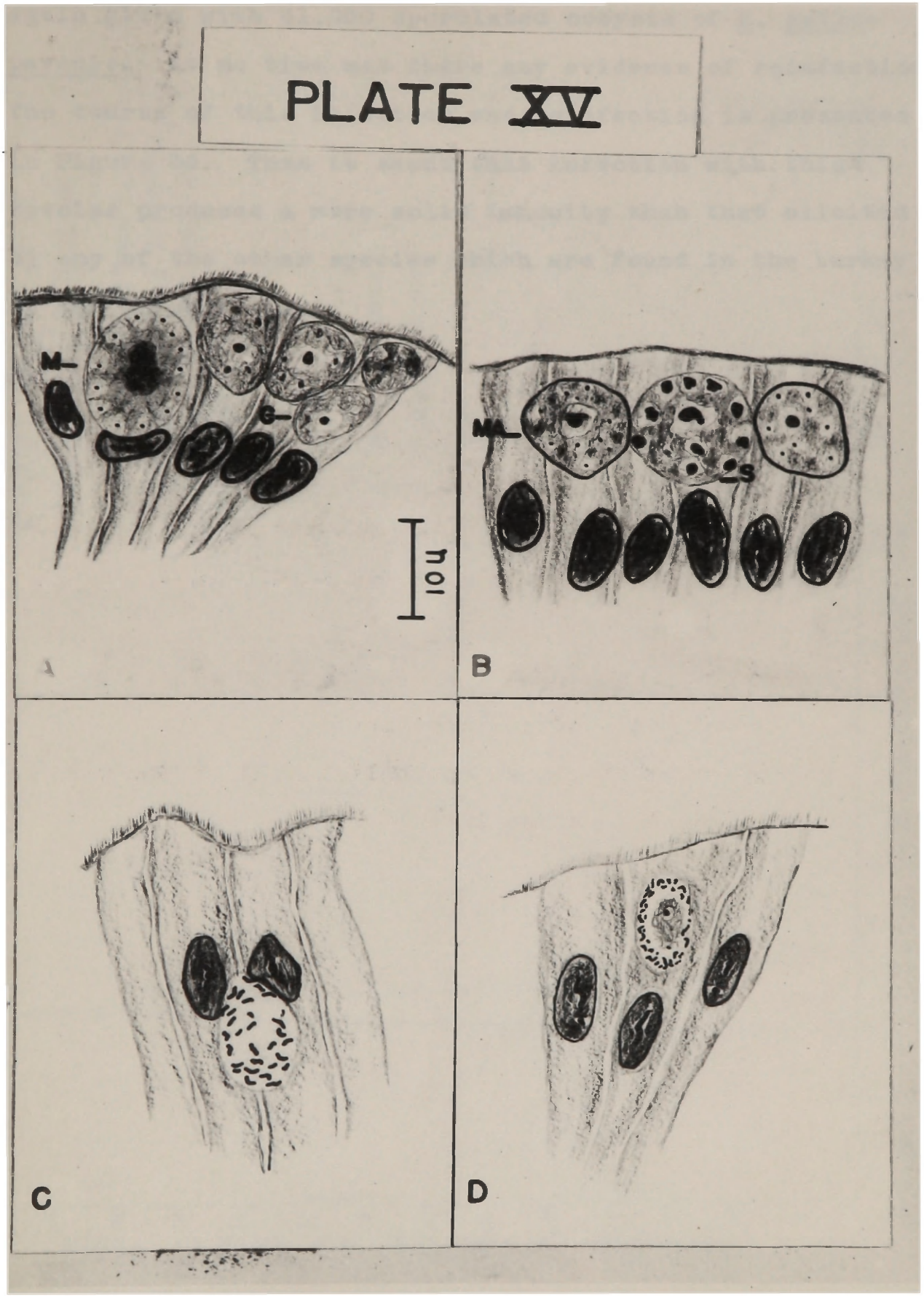
A. Gametocytes (G) and a macrogametocyte (M) in epithelial cells of the rectum six days after infection.

B. Third generation schizonts (S) and macrogamete (MA) in epithelial cells of the rectum seven days after infection.

C. Microgametocyte in an epithelial cell of the rectum seven days after infection.

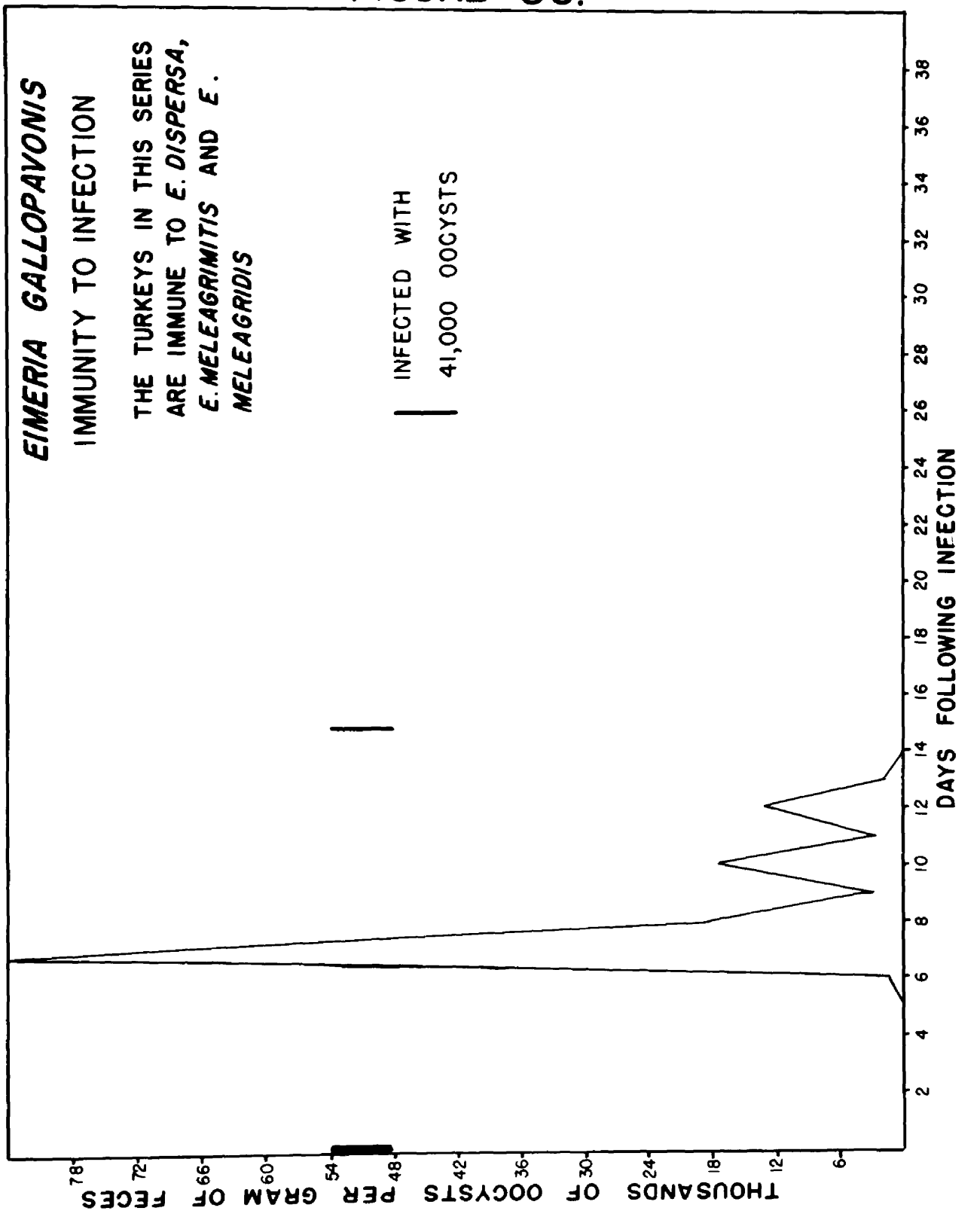
D. Microgamete in an epithelial cell of the rectum seven days after infection.

PLATE XV



again given with 41,000 sporulated oocysts of E. gallopavonis. At no time was there any evidence of reinfection. The course of this infection and reinfection is presented in Figure 36. Thus it seems that infection with this species produces a more solid immunity than that elicited by any of the other species which are found in the turkey at the present time.

FIGURE 36.


EIMERIA GALLOPAVONIS
IMMUNITY TO INFECTION
THE TURKEYS IN THIS SERIES ARE IMMUNE TO E. DISPERSA, E. MELEAGRIMITIS AND E. MELEAGRIDIS
INFECTED WITH 41,000 OOCYSTS
THOUSANDS OF OOCYSTS PER GRAM OF FECES
6
12
18
24
30
36
42
48
54
60
66
72
78
DAYS FOLLOWING INFECTION
2
4
6
8
10
12
14
16
18
20
22
24
26
28
30
32
34
36
38

DISCUSSION

The results presented in the previous sections clearly indicate that there are at least four species of coccidia in the turkey, and it is possible from material observed by the writer that more may be present. This is, of course, not inconceivable when the multiplicity of species of the genus Eimeria in other animals is considered. It is felt that the species which are described all clearly meet the requirements of specific designation. A summary of their characteristics is presented in Table 2. The localization of them in the host is rather sharply delineated. E. dispersa predominated in the duodenum and decreased rapidly in numbers progressing posteriorly in the small intestine, with no tissue stages having been observed in the ceca or rectum. E. meleagrimitis occurred throughout the small intestine. However, in the first and second generations it predominated in the middle third, but after the extensive epithelial destruction which occurred, large numbers of the third generation asexual stages were found in the duodenum and jejunum. A few tissue stages of this parasite were also observed in the ceca and rectum, but these were rare. E. meleagridis was localized in the ceca with occasional forms being found in the jejunum and rectum. E. gallopavonis was found in the jejunum and rectum, with very few localized in the ileum or ceca. In this species almost all sexual stages occurred in the rectum, very few locating in any other area. It is felt that statements

in the literature describing E. meleagridis in the rectum and jejunum in large numbers, were based on a mixed infection, in which the forms in these locations were those of E. gallopavonis.

Host limitations of these species are not striking except in the case of E. dispersa. As Tyzzer (1929) noted, the lack of apparent host specificity by this form is an exception in the genus Eimeria. In this work it has been possible to produce infections with this species in the turkey, Bob-white quail and Hungarian partridge. It was not possible to establish the infection in the pheasant or chicken, although Tyzzer described the infection in these birds. As pointed out in the description of the experimental results, it is possible that the pheasants which were available might have been immune to infection with this species. However, there may be a strain difference in these species in the various hosts. Thus Tyzzer was able to infect the turkey with E. dispersa from the quail, but could not infect pheasants with E. dispersa from the quail.

E. meleagridis and E. meleagrimitis would only develop in the turkey, but there was a possibility that a transitory infection with E. gallopavonis was established in the Hungarian partridge.

It is of interest to note, in connection with a discussion of host specificity, that E. dispersa of quail origin has a prepatent period of four days in the quail,

Table 2. Summary of characteristics of the coccidia occurring in the turkey.

Species	Host	Location	Prepatent Period Days	Sporulation hours	Polar granule	Oocyst wall*	Average Length	Average width
E. dispersa Tyzzer, 1929	Turkey Bob-white quail Hungarian partridge Pheasant Chicken ?	Duodenum	6	48	Absent	Single	26.07u	21.04u
E. meleagrimitis Tyzzer, 1929	Turkey	Jejunum	6	48	Present	Double	19.1u	16.2u
E. meleagridis Tyzzer, 1927	Turkey	Cecum	5	24	Present	Double	24.4u	18.1u
E. gallopavonis new species	Turkey Hungarian partridge	Ileum Rectum	6	24	Present	Double	27.1u	17.2u

*Oocyst wall double or single contoured

but when Tyzzer transferred these species to the turkey the prepatent period was lengthened.

The determination of the natural host for E. dispersa has certain practical implications concerned with the transmission of the organisms from wild birds to domesticated turkeys. While this controversial subject cannot be answered with any degree of certainty, it would appear that the Bob-white quail is the normal host for this coccidium, and that it has become adapted to the turkey, Hungarian partridge and pheasant. From an evolutionary point of view it is frequently thought that the parasite which has most recently become associated with a host is the most pathogenic, and hence might be considered to be incompletely adapted to that host. On the other hand, those forms which are well adapted, and have been associated with the host for the longest period of time, are the least pathogenic. Unfortunately, there is little evidence concerning the pathogenicity of this species. No marked pathogenicity has been observed in any of the host birds in which this infection has been established during the course of this work. Tyzzer quotes a personal communication in which marked pathogenicity is attributed to this coccidium occurring in the quail. Thus, if it is to be assumed that this species is the most pathogenic in the quail, and that pathogenicity is evidence of lack of adaptation to the host, it could be said that the turkey or the Hungarian partridge is the normal and the quail the abnormal host.

It was pointed out that the prepatent period of E. dispersa in the quail was originally determined by Tyzzer to be four days, and that when this species was introduced into the turkey the prepatent period was lengthened to five or six days. The turkey strains of E. dispersa which have been studied in the course of this work had a prepatent period of six days. There is considerable evidence available which would indicate that the presence of a parasite in an abnormal host results in a lengthening of the prepatent period. This has been well shown in several of the nematodes. Hegner and West (1941a) noted that the asexual cycle of Plasmodium cathemerium in the canary was three to five days whereas in the duck it was three to eight days. They ascribed the greater length of the prepatent period in the duck to the greater quantity of blood in this host as compared to the canary. In the case of coccidiosis there seems to be no apparent reason why a greater length of the intestinal tract, such as is found in the turkey and Hungarian partridge as compared with the Bob-white quail, should influence the length of the prepatent period.

A parasite should be able to be readily transferred among normal hosts, in order that the species may easily perpetuate itself. From the results presented in this paper it seems probable that this would occur in infections of any of the hosts under consideration. It might seem that E. dispersa in the Bob-white quail had better chances of perpetuation than in the turkey or Hungarian partridge,

because of the enormous numbers of oocysts which are passed in the feces. However, when it is considered that the quail ranges over much greater areas than the turkey, there does not appear to be any great difference. In fact, when considered in this light, it may be seen that the infection in the Hungarian partridge has the least opportunity for perpetuation.

Considering that the intestinal tract of the Bob-white quail is much shorter than that of the other birds the number of oocysts passed was proportionately greater. Even if the numbers of oocysts passed were calculated as to the number of oocysts passed per gram of feces originating from one infecting oocyst it may be seen that the greatest number of oocysts were passed by the quail. Thus, it is concluded that the Bob-white quail is the normal, or best adapted host for the development and perpetuation of E. dispersa.

The oocysts of coccidia are frequently used as a method for specifically differentiating them. While this method of determination has certain value for morphologically distinct forms, e. g. E. intricata in sheep, oocysts do not lend themselves to specific determination without the aid of other criteria. The oocysts of the species of coccidia occurring in the turkey may be divided into three groups, which may be easily separated. These are: 1. E. dispersa, 2. E. meleagrimitis and 3. E. meleagridis and E. gallopavonis. E. dispersa may be rather easily separated from the other species occurring in the turkey by its large,

broad ovoidal shape and the fact that the cyst wall presents a single rather than a double contoured appearance. The oocysts of the other species described in the turkey are double contoured. E. dispersa is further differentiated from all other known species of the genus Eimeria in gallinaceous birds by the absence of polar granules, or inclusion bodies, in the sporulated oocyst. The significance of these granules is not known at the present time. In the other species these granules are present in the sporulated oocyst, although they were frequently lacking in the unsporulated oocyst. It is also of interest to note that this species, which lacks the polar granules, was the one with the greatest lack of host specificity.

The oocysts of E. meleagrimitis may be differentiated by their small size and subspherical appearance. The oocysts of E. gallopavonis and E. meleagridis cannot be separated at the present time. The former is a longer and narrower oocyst, but the measurements overlap to such an extent that differentiation of the oocysts is not possible. For this reason it is not difficult to understand why E. meleagridis had been described as occurring in the jejunum and rectum as well as in the ceca.

The prepatent period of the four species occurring in the turkey was very similar, the oocysts being passed sometime during the fifth day after infection, except in infections with E. gallopavonis where no oocysts were passed until the sixth day of infection. In this species small

numbers of oocysts were found in the feces six days after infection, with the peak of oocyst production occurring on the seventh to ninth days. In the case of E. meleagrimitis infections it was found that in poults which were initially infected with 1280 sporulated oocysts the prepatent period was six days, whereas in another group infected with 128,000 sporulated oocysts the prepatent period was slightly over five days. However, in these birds with a prepatent period of five days the oocysts passed on the fifth day expressed as a percentage of the oocyst peak was 1.3 percent, compared with 67 percent for those with the prepatent period ending on the sixth day. This would indicate that the prepatent period of this species is normally six days. The prepatent period of E. dispersa in the turkey was slightly under six days, but the turkey strain of this species in the Hungarian partridge and Bob-white quail had a prepatent period of six days. The first oocysts in infections with E. meleagridis are passed on the fifth day of infection, but in small numbers, accounting for only two percent of the peak number of oocysts passed. The peak of oocyst production in all four species occurred on the second or third day of patentcy.

From the studies presented in the experimental section, no definite conclusions can be drawn as to the length of the patent period, except for that of E. gallopavonis which was nine days. In infections with the other three species the poults were receiving multiple infections, which would

undoubtedly increase the length of the patent period. However, in these cases of multiple infections the length of the patent period was 22 days in infections with E. dispersa, 19 days in initial infections with 128,000 and 31 days in initial infections with 1280 sporulated oocysts of E. meleagrimitis, and 12 days in initial infections with 1140 and 9 days in initial infections with 91,000 sporulated oocysts of E. meleagridis.

Infections with E. dispersa and E. meleagridis could be easily differentiated by the gross lesions which they produced. Poults which were infected with E. dispersa exhibited a marked dilatation of the small intestine and a creamy, yellowish, sticky mucoid material which filled the anterior portion of the small intestine. The presence of the broad,ovoidal oocysts should serve to distinguish this condition from any other. E. meleagrimitis, which is also present in the small intestine, did not produce such clear cut lesions, except in severe infections. In the latter instance there was a marked accumulation of fluid in the small intestine, a catarrhal enteritis, and a greenish mucoid cast in the posterior half of the small intestine. In the small intestine of poults infected with E. meleagrimitis there was never observed in the course of this work the yellowish mucoid material found in E. dispersa infections. Thus, although the latter infection, while more difficult to diagnose by the lesions, should not be confused with E. dispersa infections. Furthermore, the much

smaller, subspherical oocysts would facilitate a diagnosis.

Turkeys infected with E. meleagridis usually had a non-adherent cream colored cheesy or mucoid plug in the ceca before and at about the time oocysts were being produced. Numerous scattered petechial hemorrhages were also frequently observed in infections with this form, both in the dilated and constricted portions of the ceca. However, hemorrhage into the ceca was not noted.

There was not sufficient material available to study the lesions produced by infections with E. gallopavonis. However, when large numbers of elongate oocysts are found in the rectum in the absence of oocysts and there is evidence of infection in the cecum, it must be concluded that the infection is produced by E. gallopavonis rather than E. meleagridis.

Coccidiosis in the turkey is not characterized by hemorrhage, which is so frequently present in certain forms of this disease in other species of birds and mammals. In fact, except for a slight pinkish tinge to fluids in the small intestine, and petechial hemorrhages in the mucosa of the ceca, there was practically no gross or microscopic evidence of loss of blood into the lumen of the alimentary tract. Some species of coccidia, at certain stages of their development, may be seen through the serosal surface of the intestinal tract as white opacities. This type of lesion has not as yet been observed in any of the species of coccidia involving the turkey.

The pathological response of turkeys to the various species of coccidia is strikingly different. E. meleagrimitis and E. dispersa, both located in the small intestine and found in a similar position in the epithelial cells, gave rise to very different reactions. Both these forms were usually located superficially to the nucleus of the epithelial cells, and were concentrated in greatest numbers on the tips of the villi, decreasing in numbers as they progressed down the epithelial cells of the crypts, and were entirely absent from the glands. However, the reaction to E. meleagrimitis was essentially one of necrosis and sloughing of the epithelium, while the reaction to infection with E. dispersa was primarily congestion and edema. It is not possible to explain this difference, except to postulate the presence of some substance or substances which possess a much greater damaging effect to the intestinal epithelium in one species than in the other. Whether such a substance is produced at all, or in greater quantities by one species than the other is not known.

A lymphocytic infiltration was present in all infections with the four species of coccidia of the turkey. An infiltration with polymorphonuclear leucocytes is usually anticipated in an acute inflammatory process such as would be expected to be produced by E. meleagrimitis. However, this infection was marked by the absence of cells of this type. This also occurred in infections with E. dispersa, E. meleagridis and E. gallopavonis. The reasons for this

marked lymphocytic infiltration are not known. However, it would explain why there is a well developed immunity against these species, since the lymphocytes have been demonstrated to play an important role in this phenomenon.

It is not possible at the present time to estimate accurately the overall loss from coccidiosis in turkeys. It was possible to obtain figures from 43 flocks throughout the United States, totaling 78,430 turkeys, in which there was an estimated mortality of 3.9 percent from coccidiosis. It must be borne in mind that these birds were brought to poultry pathologists for a diagnosis due to obvious disease in the flock. For this reason it is probable that the losses in all flocks may be much lower. Furthermore, it was assumed that coccidia were the primary cause of death, which is an unwarranted assumption. The losses in these flocks, which varied in size from 100 to 6,000 turkeys ranged from 0 to 50 percent. The poults in most of the flocks, which suffered mortality from coccidiosis, were under eight weeks of age, with the majority being three to five weeks old. It was not possible, with any degree of certainty, to determine which coccidium was responsible for the heaviest losses, due to the presence of mixed infections. However, from the figures available most of the mortality was apparently produced by the species inhabiting the small intestine, rather than the cecal form.

In the experimental work it was observed that E. meleagrimitis was the only form which produced serious effects

in the turkey. In the case of turkey poults, two to three weeks of age, it was possible to produce a 100 percent mortality. It was not possible to produce any mortality in four month old turkeys. There was also marked depression in the weight gains and feed consumption. The weight losses began the fourth day after infection, at the time of the maturing and rupture of the second generation schizonts, and continued to fall until the eighth day after infection at which time weight increases were again noted. The weights of infected birds have not been followed longer than two weeks after infection, although it might be presumed from the results obtained in chickens by Mayhew (1932) that this weight impairment might persist for several months. The effects of the infection on feed consumption are also noted on the fourth day following infection, and there was a marked interference with it for the next four days. Water consumption by poults following infection with this species pursued a similar course.

The results obtained on weight gains in turkeys infected with E. dispersa did not give clear cut results, however there did appear to be a slight impairment of the ability of the birds to utilize their feed and convert it into gains. Feed and water consumption gave similar results. Although there was little marked effect of this infection on poults it cannot be doubted that, over a period of time, there would be a greater consumption of feed per pound of gain in weight and hence a greater economic loss,

than in birds which had never been infected.

Very similar results were obtained in infections with E. meleagridis. In this case it was possible to use two dosages with which to infect the turkeys, one being comparable to that of the infections of the two species described above, and another of 1,000,000 sporulated oocysts. Turkeys which were infected with the smaller number of oocysts did not differ markedly from uninfected birds, however the poults receiving the larger initial infection did lose a little more in weight. This was also reflected in their feed and water consumption. Thus, a species such as E. meleagridis, and probably also E. dispersa, might, if administered in large numbers, produce loss, although possibly not mortality.

To date no experiments have been carried out to determine the effects of E. gallopavonis on the turkey.

The development of immunity to the coccidia of the turkey has given very interesting results. Each species, with which the turkey was infected, was capable of establishing itself in this host and running through its course of development independently of the other three. Poults have been infected daily with varying numbers of sporulated oocysts of E. dispersa, as shown in Figure 4, and it has been found that no oocysts could be recovered from the feces 26 days or later after the first infection. At the peak of this infection there were 43 oocysts produced per gram of feces for every oocyst administered. These

poults were then infected with E. meleagrimitis and were found to be susceptible to infection. They were infected with varying numbers of sporulated oocysts, and oocysts were present in the feces until 36 days after infection, at which time they could no longer be demonstrated. In infections with this species two infecting dosages were used. When poults were initially infected with 1280 sporulated oocysts it was found that at the peak of the infection there were 18 oocysts produced per gram of feces for every sporulated oocyst administered. In contrast, when they were given an initial infection of 128,000 sporulated oocysts there were only 3.1 oocysts per gram of feces for every sporulated oocyst administered. Thus it is apparent that with heavier infections fewer oocysts were produced in relation to the number administered. There might be several explanations for this. It is possible that with heavier infections there might be a crowding factor which would result in the production of fewer oocysts. However, the most probable reason is that in heavier infections there is a much greater destruction of epithelial cells by the asexual stages, and thus fewer cells in which the sexual forms, and hence the oocysts, could develop. Therefore, the heavier infections would produce greater losses in the birds, but the lighter infections, while they would not produce as many oocysts, would perpetuate the infection more effectively.

The course of infection of E. meleagridis in poults immune to both E. dispersa and E. meleagrimitis was studied.

Infections were produced with both small and large infective doses of sporulated oocysts. There was no apparent inhibition of this infection due to immunity to the other two species. Poults which were given an initial infection of 1140 sporulated oocysts and subsequent daily infections of varying numbers, as shown in Figure 32, passed oocysts in the feces for 17 days after infection, and then oocysts could no longer be recovered. Poults which were initially infected with 108,000 sporulated oocysts (see Figure 33) passed oocysts in the feces for a slightly shorter time, 13 days. Again it was noted that the numbers of oocysts which were passed per infecting oocyst were much smaller in the heavier infection. Thus, those infected with 1140 sporulated oocysts passed 174 oocysts per gram of feces at the peak of the infection for each infecting oocyst in contrast to six oocysts per gram of feces in infections with 108,000 sporulated oocysts. This could again be accounted for by lack of suitable epithelial cells in which the sexual forms could develop, due to their destruction by the asexual stages.

Finally, the poults which were immune to the above three species were infected with E. gallopavonis. These birds continued to pass oocysts in the feces for 13 days after infection, and then they could no longer be recovered. This species apparently does not produce as many oocysts as the other three species, although comparable infections were not available. However, in the poults to which 41,000

sporulated oocysts were administered it was found that at the peak of the infection two oocysts per gram of feces were produced for every oocyst administered.

Unfortunately the technique employed in producing immunity in the poults with the four different species of coccidia makes comparison of the infections and the resulting immunity difficult. Although there can be no doubt as to the lack of production of an absolute immunity to coccidiosis, there do seem to be some antigenic differences in the four species. Infections of turkeys with 41,000 sporulated oocysts of E. gallopavonis resulted in an infection in which oocysts were passed for 14 days. A challenge infection of 41,000 sporulated oocysts administered 14 days after the first did not result in infection. Thus, this species produced what might be considered an absolute immunity against the challenge infection. It is not known how long this resistance might persist. Infection with 108,000 sporulated oocysts of E. meleagridis resulted in an infection in which oocysts were discharged for 13 days. A challenge infection of 108,000 sporulated oocysts on the 14th day following the first infection did not result in infection. However, when 1140 sporulated oocysts were administered daily for 15 days, oocysts were passed in the feces for 17 days. In this case a challenge administered on the fifteenth day following the first infection failed to produce infection. Thus, it may be seen that with this species there is a slightly more rapid

acquisition of immunity when larger infections are used for immunization.

Turkeys were administered 1500 sporulated oocysts of E. dispersa daily for eight days and then 76,000 sporulated oocysts of the same species. Further infections were administered as shown in Figure 4. The main course of this infection was passed in 15 days, although small numbers of oocysts continued to be passed in the feces until 26 days after the original infection. Infection with this species in the Hungarian partridge produced a period of patency which terminated on the twelfth day after the initial infection, and no infection occurred from a challenge infection administered on the twentieth day. Bob-white quail infected with this species had a period of patency which terminated on the twelfth day following infection, however a challenge infection 20 days following the original resulted in a period of patency which persisted for four days after which the oocysts could not be demonstrated in the feces. Thus, the immunity produced against this species was most highly developed in the turkey and Hungarian partridge, and although there was apparently an immunity developed in the Bob-white quail to infections with this species, it was not as highly developed as in the other hosts. Furthermore, the resistance produced in the turkey cannot be considered to be produced as promptly as in infections with E. gallopavonis and E. meleagridis.

Turkeys were infected with 1280 sporulated oocysts of E. meleagrimitis and then further infected as shown in Figure 22. In this case the period of patency persisted for 36 days. However, if 128,000 sporulated oocysts were given as the initial infection the major course of the infection had passed by the fourteenth day following infection, although oocysts passed in the feces until 24 days following the first infection. Thus it may be seen that, although this species produced an immunity against reinfection, it was not as effective as the other species. Furthermore, it can be seen that larger numbers of infecting oocysts produce a higher degree of resistance than do smaller numbers. Tyzzer (1929) has shown that severe infections with E. tenella in the chicken will excite a prompt and well marked immunity, but that single light infections with small numbers of oocysts were not sufficient to protect.

CONCLUSIONS

Coccidiosis in turkeys has been demonstrated to be caused by at least four species, Eimeria meleagridis, E. meleagrimitis, E. dispersa and E. gallopavonis. Of these four, E. gallopavonis is described as a new species and E. dispersa originally described from the Bob-white quail has been shown to occur commonly in the turkey. The development forms in the life histories of these species have been studied and described.

The gross and histopathology of these infections is discussed. The symptoms produced by the various species were noted and the effects on weight gains, feed and water consumption are discussed.

The immunity produced by the different species was followed, and it was noted that the resistance developed in the turkey against each species, was specific.

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