STUDIES ON THE EFFECTS OF THE FOWL HELMINTHS *ASCARIDIA GALLI* (NEMATODA) AND *RAILLIETINA CESTICILLUS* (CESTODA) ON GROWING CHICKENS AND OBSERVATIONS ON THE DEVELOPMENT OF THE CYSTICERCOID OF *R. CESTICILLUS*

by

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B. A., Southern Methodist University, 1941

A THESIS

submitted in partial fulfillment of the

requirements for the degree of

MASTER OF SCIENCE

Department of Zoology

KANSAS STATE COLLEGE
OF AGRICULTURE AND APPLIED SCIENCE

1943

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PART I. STUDIES ON SOME EFFECTS OF THE ROUNDWORM, *ASCARIDIA GALLI* ON GROWING CHICKENS

INTRODUCTION

Recent advances in nutritional requirements of chickens and the relation of the nutritional status of the host to parasitism have instigated new, significant investigations into the actual effects of certain helminths upon fowls. The observable effects of light parasitism on chickens provided with an adequate diet promised to be an interesting problem of practical importance. The present investigation is a study on some effects of a light infection with the roundworm *Ascaridia galli* on growing chicks receiving a diet known to be sufficient for normal growth.

MATERIALS AND METHODS

Day-old single comb White Leghorn chicks purchased from a commercial hatchery were placed in brooders in a tightly screened room. Food and water were kept before them at all times during the experiment except for short periods before weighing and taking samples for blood sugar analyses. In these cases only feed was removed from the batteries. Feed consisted of the Kansas State College Poultry Farm all-purpose mash containing the following proportions of ingredients, (in pounds):

Ground corn	200
Ground oats	200
Shorts	200
Bran	100
Soy bean meal	100
Alfalfa meal	100
Meat scraps	70
Ground limestone	20
Salt	10
Cod-liver oil	4

At the age of 23 days the chicks were weighed after a 24-hour starvation period. They were then divided into two groups of 15 chicks each matched closely according to weight, one group to be parasitized and the other to be kept as controls. Sex was not recorded. On the following day approximately 200 embryonated eggs of Ascaridia galli from cultures were fed to each chick of the parasitized group. Eggs taken from the cultures with a capillary pipette and placed on an ordinary glass slide in a small drop of water were observed and counted with the aid of a compound microscope. Only completely embryonated eggs showing no degeneration were tabulated. They were removed from the slides with small bits of filter paper which were subsequently inserted into the chick's esophagus and washed down with water.

On the 14th day after infection while the larvae were still associated with the intestinal mucosa, a 0.5 to 1.0 ml blood sample was taken from the wing veins of each chick in both groups previously starved for 12 to 24 hours. The samples were stored in small test tubes containing enough powdered sodium citrate to prevent clotting. All samples were analysed for blood sugar within three hours after they had been taken.

Hemoglobin was estimated with the New Dare Hemoglobinometer. The ceric sulfate microtitration method of Miller and Van Slyke (1936) was utilized to determine blood sugar, duplicate samples being analysed in each case. Weights were recorded on the days preceding the analyses. Four weeks after infection the chicks were subjected again to the same series of tests. At the termination of the experiment all chicks of both groups were killed and examined for worms.

REVIEW OF LITERATURE

Though considerable investigation has been made on the effects of various conditions of the host with respect to age, diet, and physical condition on the number and size of the worm, Ascaridia galli, the evidence available concerning the effects of the worm on the host is rather limited. Ackert and Titus (1924) reported a significant difference in the blood sugar of parasitized and unparasitized chicks two weeks after infection, the blood sugar of the parasitized group being lower than that of the control group. Then Ackert (1924) found the thymus glands of parasitized chicks smaller than those of control chicks. Later, Ackert and Otto (1927) in studies on the effects of parasitism with A. galli on the thyroid gland did not find any significant differences. In rather extensive studies on the growth rates of chickens parasitized with A. galli and unparasitized chicks, Ackert and Herrick (1928) showed a significant retardation of growth in the infected chicks during a period of two to four weeks after infection.

The difference corresponded to the period in which the worms were closely associated with the intestinal mucosa. However, sometimes at the end of eight weeks after parasitism, there was a greater gain in weight by the parasitized chicks. Rather heavy infections were used in this study with as many as 2000 eggs fed to one chick.

Clapham (1937) described some lesions associated with severe infections of Ascaridia larvae. She found the fowls emaciated and showing a severe enteritis. The mucous membranes were covered with petechial hemorrhages and the gut contents consisted mainly of bloody mucus. Large numbers of larvae were present.

Pratt (1940 and 1941) found that severe coccidiosis caused a marked rise in blood sugar by the sixth day after infection. The increase in blood sugar was associated with a decrease of glycogen in the liver and muscles. Withdrawal of large quantities of blood from the hearts of normal chickens produced a rise in blood sugar of approximately the same magnitude as did coccidiosis in other chickens.

Ackert (1942) reviewed the factors known to influence the natural resistance to helminth infections including dietary factors among numerous others. However, in most of the investigations reviewed, resistance was studied by observing the effects of diverse experimental conditions upon the worms.

EXPERIMENTAL DATA

Both parasitized and unparasitized groups were weighed

three times in the experiment. The data obtained from the weighings are included in Table 1. During the first two weeks after infection the difference in average weight gained by both groups was 3.7 g, only 1.63 times the standard error. Thus, this difference could have occurred readily by chance. The same applies to the average gain in weight over that of the second two weeks when the difference of 2.9 g is only 0.17 times the standard error. Considering the average gain in weight for both groups over the entire period of four weeks, one finds that the difference of 10.5 g between means could have occurred by chance since it is only 0.42 times the standard error.

Blood sugar was determined twice during the experiment: at two and four weeks after infection. The data obtained for each chick are recorded in Table 2. The difference between the means at the end of two weeks was 4.0 mg per 100 ml of blood, which is 0.44 times the standard error. At the end of four weeks the difference can be considered non-significant since it is well within the limits allowed by chance.

The only hemoglobin analyses (Table 2) were made two weeks after infection. The mean value of the parasitized group was 0.4 g/100 ml higher than the control group. Since this difference is only 0.60 times the standard error, it cannot be considered significant.

Autopsy revealed an average of 17.9 worms per chick in the parasitized group with numbers varying from one to 46. No parasites were found in any of the control chicks.

Table 1. Comparison of growth in parasitized and control chicks.

Chick No.	Wt. (g) 4-8-42	Wt. (g) 4-22-42	Gain (g)	Wt. (g) 5-6-42	Gain (g)	Total gain (g)
			Control group			
A-1727	145	227	82	407	130	262
A-1732	184	313	129	528	215	344
A-1733	189	314	125	551	237	362
A-1734	182	288	106	496	208	314
A-1736	122	204	82	395	191	273
A-1739	159	248	89	437	189	279
A-1740	180	274	94	418	144	238
A-1744	159	275	116	448	173	289
A-1746	165	266	101	455	189	290
A-1747	139	194	55	336	142	197
A-1749	160	270	110	474	204	314
A-1751	176	277	101	515	238	339
A-1752	190	308	118	525	217	335
A-1753	198	316	118	520	204	322
A-1758	204	306	102	532	226	328
Average	170.0	272.0	101.9	469.1	193.8	299.0
			Parasitized group			
A-1726	180	274	94	464	190	284
A-1728	190	277	87	270	193	280
A-1729	214	332	118	552	220	238
A-1730	115	214	99	388	174	273
A-1731	164	275	111	489	214	325
A-1735	142	262	120	478	216	336
A-1737	178	265	87	496	231	318
A-1741	159	239	80	413	174	254
A-1742	170	283	113	460	177	290
A-1743	135	232	97	439	207	304
A-1745	145	208	63	338	130	193
A-1750	204	320	116	541	221	337
A-1754	182	277	95	487	210	305
A-1755	188	300	112	517	217	329
A-1759	159	241	82	421	180	262
Average	169.0	268.0	98.2	463.0	190.9	288.5
Difference			3.7		2.9	10.5

Table 2. Comparison of the blood sugar and hemoglobin levels in parasitized and control chicks.

Chick no.	Blood sugar mg/100 ml 4-23-42	Blood sugar mg/100 ml 5-7-42	Hemoglobin g/100 ml 4-23-42	Number of worms
		Control group		
A-1726	167	137	9.6	0
A-1732	172	164	8.3	0
A-1733	224	189	8.7	0
A-1734	162	204	10.6	0
A-1736	160	132	10.0	0
A-1739	162	199	7.8	0
A-1740	156	188	12.6	0
A-1744	177	190	9.2	0
A-1746	102	158	9.6	0
A-1747	179	160	8.4	0
A-1749	198	170	8.8	0
A-1751	200	162	10.3	0
A-1752	208	160	9.6	0
A-1753	154	150	9.0	0
A-1758	176	160	8.6	0
Average	173.1	168.2	9.4	0
		Parasitized group		
A-1726	174	141	13.7	5
A-1728	131	171	14.2	1
A-1729	192	152	7.6	33
A-1730	195	175	8.8	20
A-1731	190	129	7.4	46
A-1735	133	148	12.8	11
A-1737	186	180	6.8	4
A-1741	176	184	9.6	18
A-1742	169	168	8.9	32
A-1743	166	192	7.5	2
A-1745	143	129	10.6	1
A-1750	196	186	8.2	16
A-1754	170	160	8.9	39
A-1755	150	174	9.9	9
A-1759	166	214	13.5	38
Average	169.1	166.9	9.8	17.9
Difference	4.0	1.3	-0.4	

DISCUSSION

Two weeks after infection of the chicks with embryonated eggs, the larvae of A. galli should be closely associated with the intestinal mucosa of the fowl host (Ackert, 1931). At this time any deleterious effects upon the birds should be most manifest. Using heavily parasitized chicks, Ackert and Herrick (1928) found a significant difference in weight gained during the two to four week period following infection. The light infections in the present experiment failed to cause any significant difference in the gain in weight during this period in the parasitized group when compared with that of the control group. Apparently there were too few larvae present to cause sufficient damage to the mucosa to markedly alter the digestive and absorptive processes.

Pratt (1940) showed that the loss of large quantities of blood caused the blood sugar level of the normal chickens to rise. He also reported rise in blood sugar during hemorrhage caused by cecal coccidiosis. Similar changes might be expected in the case of infections with A. galli during the period of the life cycle in which the larvae are associated with the intestinal mucosa provided the damage is severe enough to cause heavy bleeding. However, Ackert and Titus (1924) reported a lowering of blood sugar level in chicks parasitized with A. galli. No explanation was given for this finding. Damage to the intestinal walls sufficient to impair digestion and food absorption or the absorption of worm products which might alter the mechanisms regulating the blood sugar level

might be possible explanations. Clapham (1937) reported severe enteritis in heavy infections with Ascaridia larvae, supporting the former explanation. The loss of blood also reported would be expected to exert some influence toward raising the sugar level. In the present studies no significant difference between parasitized and unparasitized chicks was observed in either the glucose or hemoglobin content of the blood indicating that the light infections involved produced no severe effects on the digestive system or on the organs regulating the levels of these two components. Blood was not markedly evident in the droppings of parasitized chicks.

It must be noted that rather small groups of chickens were used in this experiment. Further studies using larger experimental groups and various degrees of infection would do much to elucidate some of the inconsistencies between this and other studies. However, if the results of this experiment can be considered significant, they would tend to show that light infections of Ascaridia galli have no marked effect on the gain in weight or in the blood sugar and hemoglobin levels of growing chicks. Such results could then be interpreted from a practical aspect to indicate that worming of chicks in flocks where the intensity of infection with this nematode is low as unnecessary. Thus expense and labor could be eliminated without significant retardation of growth in the flock and without ensuing economic loss at marketing time.

SUMMARY

1. Thirty young chickens, 15 parasitized and 15 controls,

were used to study some effects of the roundworm Ascaridia galli on growing chickens.

2. Both groups were fed a diet known to be sufficient for normal growth.

3. The parasitized group harbored from one to 46 worms with an average of 17.9 worms per chicken. The controls were free from worms.

4. Gain in weight, and blood sugar and hemoglobin levels were determined for each chicken of both groups two weeks after infection when the larvae were associated with the intestinal mucosa. Weight and blood sugar were again determined four weeks after infection when the larvae had returned to the intestinal lumen.

5. No significant differences between the means of the above mentioned characteristics in parasitized and control groups occurred.

6. The results were assumed to indicate that light infections of Ascaridia galli have no marked effects upon growing chickens supplied with an adequate diet.

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PART II. ON THE EFFECTS OF THE FOWL TAPEWORM RAILLIETINA CESTICILLUS (MOLIN) ON GROWING CHICKENS

INTRODUCTION

With the discovery by Cram (1928) that certain beetles serve as intermediate hosts for the fowl tapeworm Raillietina cesticillus (Molin), and subsequent investigations by other workers here and abroad, well-controlled, critical experiments have been made possible on the effects of this tapeworm on chickens. Lack of uniformity in the findings of different workers regarding effects of various tapeworms of chickens prompted the present study.

MATERIALS AND METHODS

The beetles Cratacanthus sp. and Amara spp. reported by Jones (1930, 1931) as successful intermediate hosts for the fowl tapeworm R. cesticillus have been found plentiful in Kansas by Case and Ackert (1939). During the summer of 1942 approximately 1500 of these beetles were collected on the campus of Kansas State College. To reduce the incidence of naturally infected individuals, they were taken at some distance from poultry yards. Some were placed in large glass dishes containing a layer of moist sand and a few dried leaves. Many survived quite well when fed meat scraps and soft-bodied insects, like small grasshopper nymphs and aphids. A considerable amount of cannibalism was noted in the case of Cratacanthus sp. To minimize cannibalism and to increase viability, and also to reduce the amount of needed care,

the beetles were stored in fruit jars with moist earth and kept at 5° C until needed for experimental purposes--a procedure described by Case and Ackert (1940) and Ackert and Case (1940).

Examination of droppings of chickens in a local packing company, for gravid proglottids of R. cesticillus, showed three infected birds which were subsequently purchased. These chickens were placed in batteries provided with coarse screen floors and metal dropping pans. Moistened papers placed in the pans shortly after noon each day prevented excessive drying of the proglottids. Since most of the proglottids were passed during the afternoon as reported by Wetzel (1934) and Reid, Ackert and Case (1938), the feeding of the beetles was done in the early evening when the still active gravid proglottids could be picked easily from the recent droppings. Fresh proglottids were placed in Petri dishes with beetles starved for at least 24 hours. Beetles observed to ingest one or two proglottids were removed from the Petri dish and placed, usually in pairs, in a four-drachm vial prepared as illustrated in Figure 1. Both Blotting paper and paper wad were moistened with water. In subsequent feedings, a small

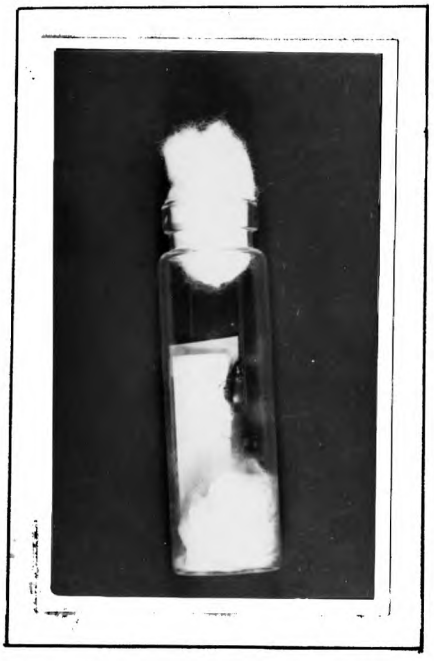

Fig. 1. Storage vial for beetles.

bit of meat was placed on the blotting paper, easily accessible to the insects. These beetles were stored in an incubator at temperatures ranging from 28° C to 31° C. Daily additions of moisture were necessary. During incubation, while cysticercoids were developing within them, the beetles were fed small bits of meat on alternate days. It was necessary at frequent intervals to remove dead beetles and old particles of meat not eaten since conditions were very favorable for mold growth. At times it was necessary to transfer beetles to fresh vials because of accumulation of waste materials, debris and fungi. The entire collection of beetles was kept in the incubator until a period of 21 days had elapsed after the last beetles had been fed.

Day-old single-comb White Leghorn chicks from a commercial hatchery were placed in clean brooders in a tightly screened room. Later, they were transferred to larger metal batteries with sufficient room for normal growth. The chicks were fed a mixed feed obtained from the Kansas State College Poultry Farm which contained the following ingredients (in pounds):

All-purpose mash[1]	200
Ground wheat	100
Ground corn	100
Ground limestone	16
Dried buttermilk	20
Cod-liver oil	0.6

This feed was available to the chicks at all times during the experiment except for short periods before weighing and before the determination of blood sugar. Clean water was present at all times.

At the age of 39 days the chicks previously starved for

[1] The ingredients for the all-purpose mash have been given in Part I.

24 hours were weighed on a torsion spring balance and divided into two groups matched according to weight. Twenty-two chicks were placed in each group. On the following day one group of chicks designated as the Parasitized Group was fed cysticercoids of the tapeworm R. cesticillus obtained from the beetles previously discussed.

A method for the removal of cysticercoids faster than that described by Reid, Ackert and Case (1938) was devised. Since none of the beetles were to be kept for identification, no precautions were taken to preserve any structures. To facilitate manipulation, the beetles were first drowned, a procedure followed by Reid, Ackert, and Case (1938) and Ackert and Case (1938). Then, while holding either the legs or the head and thorax between the thumb and fore-finger of one hand, the elytra and membranous wings were easily removed with pointed forceps held in the other hand. A dissecting needle ground to form a knife-edge at the tip was used to loosen the membranous dorsal covering of the beetle's abdomen. While grasping the head and thorax with a pair of forceps and submerging the entire insect in a Syracuse watch glass partially filled with water, the cysticercoids were freed easily by teasing the viscera with a dissecting needle and flushing with a stream of water from a capillary pipette. When no more cysticercoids could be removed from the body cavity and viscera, the beetle was removed from the dish. In this manner a considerable number of beetles could be examined in a relatively short period of time. With the aid of a low power binocular microscope the cysticercoids could be counted

as they were drawn into a capillary pipette provided with a Hoffman screw compressor attached to the rubber bulb, an arrangement described by Reid, Ackert and Case (1938). One hundred mature cysticercoids were placed directly into the esophagus of each chicken of the Parasitized Group by means of the pipette and were washed down with water. Later in the same day each of these chickens was fed two infected beetles. This group was parasitized at the age of 40 days.

At various intervals following the infection of one group, both groups of chickens were subjected to certain tests. Weights were recorded nine days after infection and at weekly intervals thereafter. In each instance the feed troughs were removed from the batteries at approximately 10:00 o'clock in the evening and weighings were made between 9:00 and 10:00 o'clock the next morning. A starvation period of this short duration did not result in the loss of the tapeworm strobila as did longer periods reported by Reid (1940, 1942a, 1942b).

Blood sugar was determined by the ceric sulfate micro-titration of Miller and van Slyke (1936). Blood for these analyses collected from a wing vein by means of a syringe was transferred immediately to small test tubes containing enough powdered sodium citrate to prevent clotting until the analyses were made. In each case of blood sugar determination, the feed troughs were removed from the batteries the evening before the blood samples were taken in order that a fasting blood sugar level would be obtained. Only as many samples were taken at one time as could be analyzed in the next three hours. Duplicate analyses were made on each sample. Hemoglobin was

determined by the new Dare Hemoglobinometer. The first series of analyses were made on blood taken from the wing veins. All subsequent hemoglobin determinations were made on samples obtained by clipping a small portion of the comb, the blood being collected directly into the hemoglobinometer pipette. Samples of blood for differential white cell counts were obtained also from the comb. Thin smears were stained with Giemsa's stain using a phosphate buffer to control the pH. At the termination of the experiment all of the chickens were sacrificed and examined for worms.

REVIEW OF LITERATURE

Before the discovery by Cram (1928) that certain ground beetles serve as intermediate hosts for the tapeworm Raillietina cesticillus (Molin), studies on the effects of this tapeworm on chickens were made of necessity on naturally infected birds. Many such animals studied by earlier workers possessed mixed infections not only of various species of tapeworms but also of parasites distantly removed from this class. The studies of Gutberlet (1916) were made on naturally infected fowls. Among other symptoms reported by him from observation and examination of infected fowls were great hunger and thirst, nervousness, emaciated appearance, drooping wings, catarrh of the intestine and, in heavy infections, anemia. These symptoms were attributed to tapeworms in general.

Stafseth (1935), reporting on the pathology of the host's intestine as found in autopsy of birds naturally infected with R. cesticillus, described a condition of capillary congestion

with an infiltration of lymphocytes and polynuclear cells in the villi on either side of the crypt occupied by the worm. He found that hemorrhages were not common; however, there was some enteritis in heavily infected birds.

Controlled studies by Ackert and Case (1938) on fowls experimentally infected with the tapeworm R. cesticillus showed less gain in weight and a lower blood sugar in the parasitized chickens than in the non-parasitized group. Infections of long standing were reported to cause a significant lowering of the hemoglobin.

Taylor (1933) working with 12 week old chickens artificially infected with Davainea proglottina was unable to find any noticeable disturbance of health in birds harboring as many as 3000 to 4000 worms; however, Levine (1938) in infections of approximately the same magnitude found a difference of two to six ounces between the parasitized and non-parasitized groups of chicks from the 35th day to the 136th day of the experiment. In experiments with the tapeworm Hymenolepis carioca, Luttermoser (1940) found that two week old chicks infected with 1000 cysticercoids gained as much over a six week period as did the non-infected group.

Variations in the results obtained by different workers as to the extent of deleterious effects of tapeworms on domestic fowls led to an inquiry into the reasons for those variations. Ackert and Reid (1937) introduced the factor of age resistance when they reported young chickens more susceptible to infections of Raillietina cesticillus than older chickens. Recently, the

role of the nutritional status of the host has been introduced as a major factor in the manifestation of deleterious effects from parasite infections in general. Harwood and Luttermoser (1938) showed that chickens infected with R. cesticillus gained only two-thirds as much weight in a two week period as their uninfected controls when both groups were fed a manganese deficient diet; however, uninfected chicks gained only slightly more than the parasitized when both groups were fed a more adequate diet. Later, Luttermoser and Allen (1942) reported that there were significant differences in weight gained by chickens parasitized with R. cesticillus and unparasitized chickens when both groups were placed on a low protein diet. Again, when a parasitized and unparasitized group were fed a more adequate diet high in protein, there was no marked difference in weight.

EXPERIMENTAL DATA

The data obtained in the present experiment were compared by the usual statistical methods. No attempt has been made to allow for sex variations.

The data obtained from periodic weighings through the course of the experiment are recorded in Tables 1 and 2. Comparison of the total gain in weight by both groups over the entire period of 65 days of the experiment showed that the Control Group gained 53.0 g more than did the Parasitized Group. However, since this difference was only 1.41 times the standard error it could not be considered significant.

Table 1. Blood sugar and weight data for Parasitized Group

Chick No.	Weight (g) 10-9-42	Weight (g) 12-13-42	Gain (g)	Blood Sugar mg/ 100 ml 10-25-42	Blood Sugar mg/ 100 ml 12-10-42	Number of Worms
A-1762	172	993	821	158	147	25
A-1764	154	1022	868	180	164	42
A-1765	153	783	630	144	145	76
A-1768	110	636	526	165	144	25
A-1772	188	857	669	162	173	150
A-1773	168	839	671	180	169	58
A-1776	168	1020	852	181	153	133
A-1778	194	1153	959	164	142	33
A-1779	177	800	623	132	164	133
A-1781	148	816	668	145	151	16
A-1782	233	1142	909	151	172	34
A-1785	185	877	692	163	187	39
A-1786	141	849	709	168	149	56
A-1788	241	1051	810	146	154	40
A-1792	152	851	699	141	164	31
A-1793	215	1088	873	144	152	161
A-1797	177	1105	928	157	137	16
A-1799	160	689	529	157	144	90
A-1839	213	1239	1026	167	129	172
A-1840	225	1206	981	171	157	2
Average	176.6	950.8	772.1	158.8	154.8	61.5

Table 2. Blood sugar and weight for Control Group

Chick No.	Weight (g) 10-9-42	Weight (g) 12-13-42	Gain (g)	Blood sugar mg/ 100 ml 10-25-42	Blood sugar mg/ 100 ml 12-10-42
A-1767	161	1141	980	137	117
A-1769	201	1034	837	161	170
A-1770	150	833	683	185	134
A-1771	147	938	791	154	132
A-1777	121	978	857	194	140
A-1780	179	1293	1114	169	147
A-1783	154	913	759	178	200
A-1787	183	975	792	160	143
A-1789	197	1213	1016	158	172
A-1790	215	998	783	176	143
A-1791	244	1308	1064	158	131
A-1794	230	1306	1076	201	140
A-1795	201	1169	958	153	155
A-1796	223	1171	948	161	148
A-1798	150	628	478	155	149
A-1800	154	882	728	146	159
A-1835	174	821	647	132	152
A-1836	185	944	759	166	163
A-1837	171	677	506	167	137
A-1838	187	1005	818	152	136
A-1841	112	778	666		149
A-1842	164	1056	892	187	195
Average	177.4	1002.5	825.1	164.2	150.5

Table 3. Hemoglobin and differential leukocyte count data for Parasitized Group

Chick No.	Hemoglobin g/ 100 ml 10-25-42	11-20-42	12-2-42	Differential White Cell Count (%) 12-2-42 Lymph.	Mono.	Poly.rods	Poly.gran.	Baso.
A-1762	8.5	9.8	10.4	76	10	9	1	4
A-1764	7.4	9.9	13.8	74	2	16	1	7
A-1765	9.5	8.5	11.9	71	7	13	3	6
A-1768	8.2	12.2	14.2	75	7	17	1	0
A-1772	10.6	9.2	14.9	50	6	42	0	2
A-1773	8.1	9.4	13.8	71	4	16	2	7
A-1776	8.2	14.4	13.6	78	4	16	1	1
A-1778	7.9	9.8	10.8	88	3	6	1	2
A-1779	9.6	14.2	11.4	79	3	15	1	2
A-1781	8.4	9.1	10.2	80	3	16	0	1
A-1782	9.2	10.8	11.9	79	4	15	1	1
A-1785	8.9	9.2	10.4	65	5	26	2	2
A-1786	9.0	8.9	12.2	90	2	6	0	2
A-1788	8.6	9.9	10.5	84	5	10	1	0
A-1792	7.8	9.6	14.4	94	2	2	0	2
A-1793	7.5	9.7	10.7	87	3	9	1	0
A-1797	8.0	8.2	12.5	77	5	16	0	2
A-1799	9.5	9.8	10.5	66	4	24	3	3
A-1839	8.7	11.4	13.3	62	5	27	2	4
A-1840	9.3	13.2	14.3	74	3	18	1	4
Average	8.6	10.4	12.3	76.0	4.4	15.9	1.1	2.6

Table 4. Hemoglobin and differential leukocyte count data for Control Group

Chick No.	Hemoglobin g/ 100 m. 10-25-42	11-20-42	12-2-42	Differential White Cell Count (%) 12-2-42 Lymph.	Mono.	Poly.rods	Poly.gran.	Baso.
A-1767	9.0	12.9	14.7	71	7	17	2	3
A-1769	9.1	16.2	15.6	69	7	21	1	2
A-1770	7.3		10.9	74	9	14	1	2
A-1771	8.9	10.2	11.1	79	13	6	0	2
A-1777	8.4	9.1	10.5	76	4	15	2	3
A-1780	8.9	9.6	10.7	65	8	20	4	3
A-1783	8.4	9.5	11.4	85	2	9	1	3
A-1787	7.3	9.6	10.9	92	5	3	0	0
A-1789	10.0	10.9	12.5	80	3	13	1	3
A-1790	9.2	9.3	13.4	74	3	17	2	4
A-1791	8.2	10.2	13.7	84	3	9	1	3
A-1794	9.1	9.4	14.8	75	4	18	1	2
A-1795	6.9	9.3	11.3	85	3	9	1	2
A-1796	9.9	10.5	14.5	68	7	21	4	0
A-1798	8.4	10.5	14.3	57	9	28	3	3
A-1800	9.3	9.7	12.6	87	3	5	0	5
A-1835	8.9	10.5	11.2	88	2	6	2	2
A-1836	8.0	9.6	13.2	73	6	15	1	5
A-1837	7.0		13.9	63	7	26	2	2
A-1838	7.7	9.8	11.2	86	4	7	0	3
A-1841		12.0	14.6	89	3	6	0	2
A-1842	8.3	10.0	14.6	73	4	15	2	6
Average	8.5	10.4	12.8	76.9	5.3	13.6	1.4	2.7

Blood sugar was determined twice during the experiment. The first determination was made 15 days after infection and the second over the period of 58 to 61 days after infection. Tables 1 and 2 include the values obtained for these analyses. Statistical analysis showed the differences between means of 5.4 m. per 100 ml in the first and 4.3 mg per 100 ml in the second series of analyses were 1.0 and 0.38 times the standard error, respectively. Neither of these differences could be considered significant.

Hemoglobin was determined at 15, 41, and 53 days after infection. The results are recorded in Tables 3 and 4. Fifteen days after infection the Parasitized Group averaged 0.1 g hemoglobin per 100 ml blood higher than the Control Group, a difference which was only 0.31 times the standard error. There was no difference between the means 41 days after infection, and 53 days after infection the Control Group averaged 0.5 g hemoglobin per 100 ml blood higher than the Parasitized Group. The latter value was 1.02 times the standard error and could not be considered a significant difference.

Smears for the differential white cell counts were made 29 days after infection. Statistical comparison of the results are recorded in Table 5. Examination of the last column of this table will reveal that none of the differences of the means between the Parasitized and Control Groups approached significance.

Table 5. Statistical analysis of the white cell differential counts 29 days after infection

Type of cell	Mean		Diff. of MEAN	Standard Dev.		Standard error	Diff./ Std. error
	Control	Parasit.		Control	Parasit.		
Lymphocytes	76.9	76.0	0.9	9.6	10.6	3.03	0.39
Monocytes	5.3	4.4	0.9	2.3	2.1	0.67	1.37
Polyneuclear rods	13.6	15.9	2.3	9.6	8.8	2.80	0.82
Polyneuclear granules	1.4	1.1	0.3	1.17	0.88	0.31	0.97
Basophiles	2.7	2.6	0.1	1.38	2.01	0.29	0.34

At the termination of the experiment the Parasitized Group was found to harbor an average of 61.5 worms per chicken ranging from two to 172. The Control Group had none.

DISCUSSION

Attempts to experimentally evaluate the effects of tapeworms on chickens has led only to confusion. Workers have disagreed on results not only with tapeworms of different species but also in cases where the same species has been used. These discrepancies could be attributed to differences in one or more of the conditions other than species of host or parasite.

Of significance are the findings of Harwood and Luttermoser (1938) and Luttermoser and Allen (1942) who showed tnat parasitized chickens receiving an adequate diet did not differ significantly in tne weignt gained over the experimental period from unparasitized chickens receiving the same diet. However, when the diet was deficient in an essential substance, differences began to appear, the parasitized chickens gaining less weight than the controls on the same deficient diet.

Advances in the knowledge of the nutritional requirements of chickens are constantly being made. It is not improbable that some of the earlier workers failed to include certain essential food materials in the feed given to their experimental chickens. With such an assumption such differences as have been apparent could be explained without difficulty.

Both parasitized and control chickens of this experiment

were fed on a diet which had been shown to be sufficient for growing chicks. No significant differences in weight, hemoglobin, blood sugar, or differential leukocyte count was observed between these two groups. The number of tapeworms in the parasitized group ranged from two to 172, averaging 61.5. These data indicate that growing chickens receiving an adequate diet are not markedly affected by moderate infections of the fowl cestode Raillietina cesticillus. The recent findings of Luttermoser et al substantiate these findings.

SUMMARY

1. Forty-four young chickens divided into two similar groups of 22 chickens each were used in a study on some effects of the fowl tapeworm Raillietina cesticillus (Molin) on growing chickens.

2. One group was fed mature cysticercoids of R. cesticillus and harbored an average of 61.5 worms per chicken at the termination of the experiment.

3. Both groups were fed a diet known to be sufficient for normal growth.

4. Various tests including blood sugar, hemoglobin, weight and differential leukocyte counts were applied to the individuals in both groups of fowls at various intervals after infection.

5. No significant differences were observed between the means of the values obtained in each of the tests mentioned above.

6. The results were interpreted to indicate that chickens fed an adequate diet are not markedly affected by moderate infec-

tions of the tapeworm Raillietina cesticillus.

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PART III. STUDIES ON THE DEVELOPMENT OF THE CYSTICERCOID OF THE FOWL TAPEWORM, RAILLIETINA CESTICILLUS (MOLIN)

INTRODUCTION

Although other papers have appeared describing the development of the cysticercoid of Raillietina cesticillus (Molin), none has presented detailed figures showing many of the various stages through which the organism passes in development. With the material and equipment available, it was possible to made a detailed study of the gross morphology of the diverse stages. It is the purpose here to present a series of photomicrographs to supplement previous studies by other workers.

MATERIALS AND METHODS

During the course of the experiment to determine the effect of Raillietina cesticillus (Molin) on chickens as described in Part II of this thesis, it was observed that some of the beetles previously fed on gravid proglottids of the tapeworm died from time to time. Examination of such beetles revealed many immature stages of the cysticercoids in the body cavity. As the storage vials were examined at daily intervals, no beetles removed from them had been dead more that twenty-four hours. High humidity within the vials prevented excessive drying of the beetles. Many forms still living were removed from the dead insects. Proof that many stages were still alive was seen in the motion of the early

forms observed microscopically and the successful infection of chickens with mature cysticercoids from the dead beetles (Plate I).

Examination of the dead beetles for cysticercoids was made in a manner similar to that described for the surviving ones in Part II. All forms were fixed in Bouin's fluid. The whole mounts were stained with menatoxylin or picrocarmine and mounted in Canada balsam or clarite. Paraffin sections of the mature cysticercoids and of the beetle gut were stained by the azo-carmine modification of the Mallory triple staining technique.

The photomicrographs were made with a Zeiss microscope equipped with apochromatic lenses. A ground glass screen and cut film holder provided by the manufacturers converted a Solar enlarger into a satisfactory camera.

REVIEW OF LITERATURE

Cram (1928) succeeded in producing the cysticercoids of Raillietina cesticillus (Molin) in the beetle Anisotarsus agilis by feeding gravid proglottids. This report appears to have been the first actual demonstration of the cysticercoid of this tapeworm. Though Ackert (1918) succeeded in infecting some chickens with this tapeworm by allowing them to feed on large numbers of house flies, he did not demonstrate the cysticercoids by dissection and isolation from the flies.

Wetzel (1933) accompanied his report of successful development of the cysticercoids in certain beetles in Germany with measurements and a drawing of a longitudinal section of the cyst.

Later (1934) he described the development from onchosphere to mature cysticercoid including several good figures of longitudinal sections of diverse stages. Ackert and Reid (1936) made further studies on mature cysticercoids, with measurements and sketches of the invaginated and evaginated forms. Joyeux and Baer (1937 and 1938) redescribed the development; however, they added nothing new to Wetzel's account (except differences in the length of time for development attributed to differences in temperatures prevailing in the two localities and a few additional drawings). Reid, Ackert, and Case (1938) made further observations on the period of development and included a detailed drawing of an onchosphere with its surrounding membranes.

DISCUSSION OF OBSERVATIONS

Eggs of Raillietina cesticillus expressed from gravid proglottids consisted of an onchosphere surrounded by several membranes which have been described in detail by Reid, Ackert and Case (1938). Figure 1 of Plate II presents a photomicrograph of an unstained egg freshly pressed from a proglottid in nearly isotonic saline. All of the membranes described by Reid et al (1938) are visible in the illustration.

Sections made through the mid-gut of appropriate beetles two to three hours after ingestion of a gravid proglottid of R. cesticillus revealed numerous onchospheres free of their membrances distributed throughout the lumen, many closely associated with the wall. No detailed study was made of the

factors which caused the onchospheres to break from their membranes within the beetles; however, direct microscopic examination of gravid proglottids teased apart in warm physiological saline (0.85 percent NaCl) containing a low concentration of sodium carbonate showed considerable activity of the onchospheres with many penetrating through the entire system of membranes by means of their hooks. They continued to be quite active for some time after freeing themselves showing characteristic onchosphere movement in the alkaline saline solution.

Since digestion in many of the carabids simulates tryptic digestion in higher animals, it was considered feasible that the slightly alkaline reaction would serve as a stimulus to the onchospheres in addition to salt concentration (Reid, Ackert and Case, 1938) and temperature. The mechanical macerating effect of the beetles' mandibles during the ingestion of the proglottid might have torn the membranes from some of the onchospheres, thus aiding in their excape. However, sections through the mid-gut of beetles made shortly after the ingestion of a proglottid showed many onchospheres still contained within their membranes.

Only dead beetles were examined for the stages occurring within the body cavity. Since they had been fed gravid proglottids from one to three times during their captivity before death, most of the beetles presented an array of diverse stages whose period of development could not be determined accurately. However, their sequence in the cycle was followed with relative ease.

Examination of beetles found dead the morning after feeding upon a proglottid revealed some still active onchospheres in the body cavity which differed very little from those observed to have escaped from ova. In addition to these, other early stages were found to be present. Some showed an increase in size resulting from cell division and formed nearly spherical hyaline masses. Undulating movements in the periphery of these forms were observed. Upon close examination of the globules, the embryonic hooks were found distributed without definite arrangement near one side--the hook-bearing pole. Increase in size through rapid cell division resulted in larger, solid spherical masses devoid of motility (Plate II, Fig. 2).

Further increase in size was accompanied by the development of a comparatively large cavity, or primitivhöhle, causing this stage to resemble a blastula. Examination of some of these stages showed a cellular proliferation at the pole opposite the embryonic hooks (Plate II, Fig. 3). Progressive growth of the hook-free pole was accompanied by the formation of a small projection at the hook-bearing pole which developed into a weakly attached spherical mass of relatively small size also containing a cavity (Plate II, Figs. 4 and 5). The embryonic hooks usually were contained in this structure. Wetzel (1934) compared this stage to the procercoid of Diphyllobothrium. As later stages failed to show the small spherical hook-bearing structure, it was assumed to have been lost. This observation is in agreement with that of Wetzel (1934).

Continuation of cellular proliferation at the hook-free

pole resulted in the elongation of the developing cysticercoid. Figure 6 (Plate II) shows such a stage with two bands of nuclear condensation near the anterior end destined to become the two rows of rostellar hooks. Rapid growth of the cells of the walls filled the cavity, or primitivhöhle, with loose parenchymatous tissue. A marked constriction (O_1) just posterior to the two rows of developing hooks established the identity of the rostellum (Plate II, Fig. 7). Another constriction (O_{11}) somewhat less marked at this stage and posterior to the first constriction, defined the boundary between the scolex and the body of the cysticercoid. Condensation of nuclear material into four definite masses between the two areas of construction (three such masses are visible in Fig. 7, Plate II) forecast the formation of the four weak suckers in the fully developed scolex. Figure 9 (Plate III) shows proliferation of these areas forming lateral projections. A third constriction (O_{111}) present in Figures 9 and 10 (Plate II) divided the scolex and future inner cyst wall from the body of the cysticercoid.

It was observed that the rostellum in stages of development more advanced than shown in Figure 7 (Plate III) could be either retracted or relaxed. Figures 8 and 11 (Plate II) show a retracted rostellum, while Figures 9, 10 and 12 show the rostellum relaxed.

The rostellar hooks appearred to be completely developed in stages as early as those shown in Figure 10 (Plate III). Sections not contained in this series of figures showed that the suckers were well-developed at this stage. Other than

the enlargement of the basal portion forming the typical scolex of R. cesticillus, no major changes were observed in the scolex from this stage to the mature cysticercoid.

The portion of the developing cysticercoid posterior to the third constriction underwent considerable development and differentiation. The original cavity or primitivhöhle was filled in with loose parenchymatous tissue. Coinciding with and extending beyond the differentiation of the scolex, various tissue layers were formed in this portion. Its increase in size can be noted in Figures 9 to 12 (Plate II). Since a detailed description of the structure of these layers has been given by Wetzel (1934) and since this part of the thesis has been limited mainly to the changes in gross morphology, such histological details will be omitted. A cysticercoid in the process of invagination is shown in Fig. 11 (Plate II).

A recently matured evaginated cysticercoid is shown in Fig. 12 (Plate II). Tissue differentiation within the cyst is visible in this photograph. Plate III is a photomicrograph of the longitudinal section through a mature invaginated cysticercoid showing the typical position of the scolex with its retracted rostellum. The mode of attachment of the scolex to the inner cyst wall is also revealed. An irregular cavity at the posterior end opening to the exterior (as shown in Plate III) has been described by Wetzel (1934) as the excretory bladder of the cysticercoid.

SUMMARY

1. Diverse immature stages of the cysticercoid of Raillietina cesticillus (Molin) were obtained from experimentally infected beetles dying during the course of another experiment.

2. A study supplemented with photomicrographs was made on the gross morphology of successive stages in the development from onchosphere to mature cysticercoid.

3. These observations agree with and supplement studies by other workers.

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EXPLANATION OF PLATES

Plate I

Small intestine of chicken ten days after being fed large numbers of cysticercoids of Raillietina cesticillus taken from dead beetles. (The gut is split longitudinally.)

Plate II

Fig. 1. Unstained egg from gravid proglottid of R. cesticillus showing hooks and membranes. (Greatly enlarged)

Fig. 2. Early stage of cysticercoid consisting of a solid spherical mass of cells. (X142)

Fig. 3. Early stage of cysticercoid showing primitive cavity. (X142)

Fig. 4. Early stage showing beginning of cellular proliferation at hook free pole and developing projection at hook bearing pole. (X142)

Fig. 5. Later stage showing increase in size and fully developed hook bearing sphere. (X142)

Fig. 6. Stage showing loss of hook bearing sphere and the developing rostellar hooks. (X142)

Fig. 7. Obliteration of primitive cavity, beginning of sucker formation, and development of rostellum. (X142)

Fig. 8. Later stage showing retracted rostellum. (X150)

Fig. 9. Stage of approximately same degree of development as Fig. 8 but with relaxed rostellum. (X142)

Fig. 10. Increase in size of scolex and cyst proper. (X142)

Fig. 11. Cysticercoid in the process of invagination. Scolex fully developed. (X142)

Fig. 12. Fully developed evaginated cysticercoid with relaxed rostellum. Fibrillar tissue of cyst clearly visible. (X142)

Plate I.

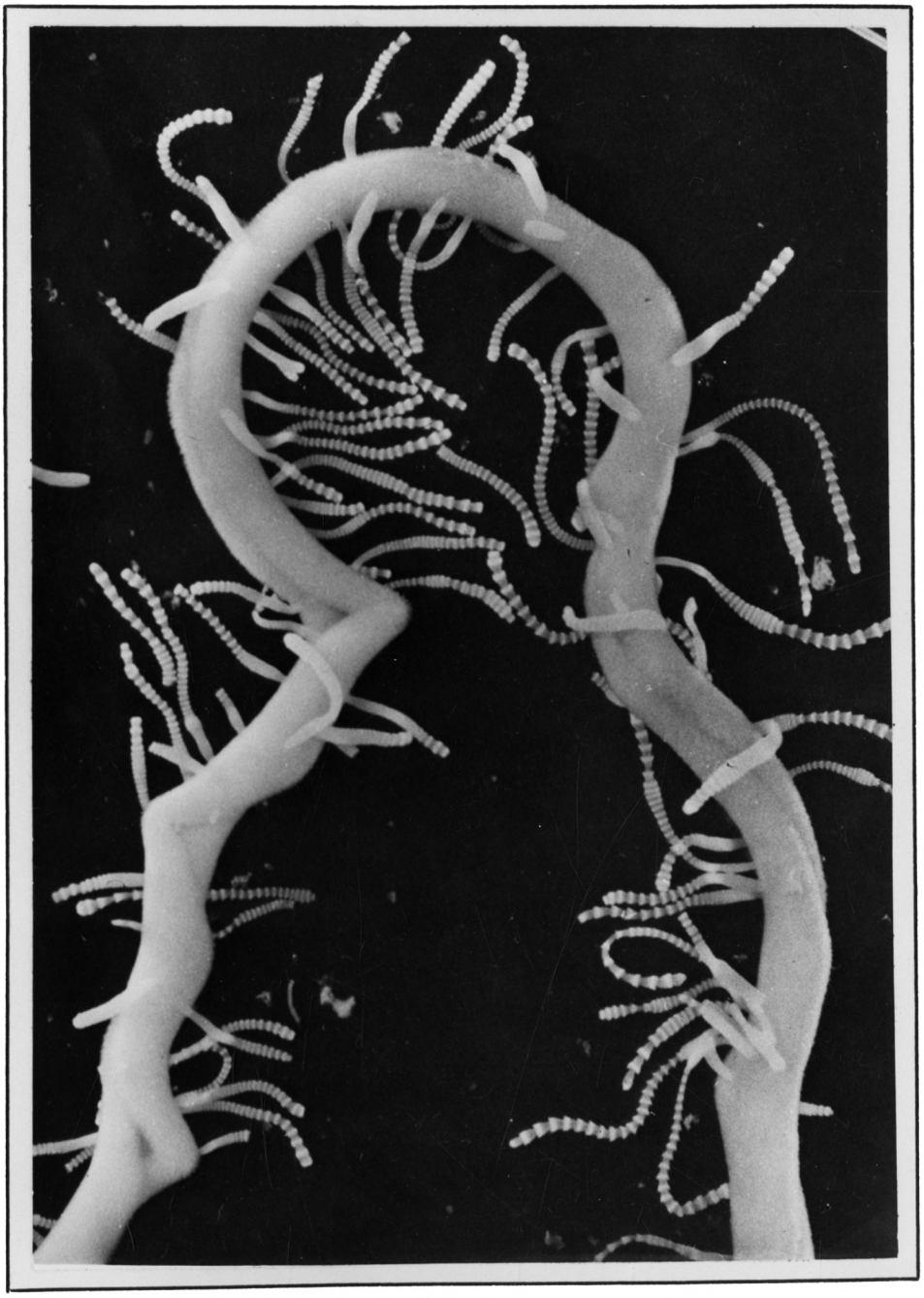

Plate II.

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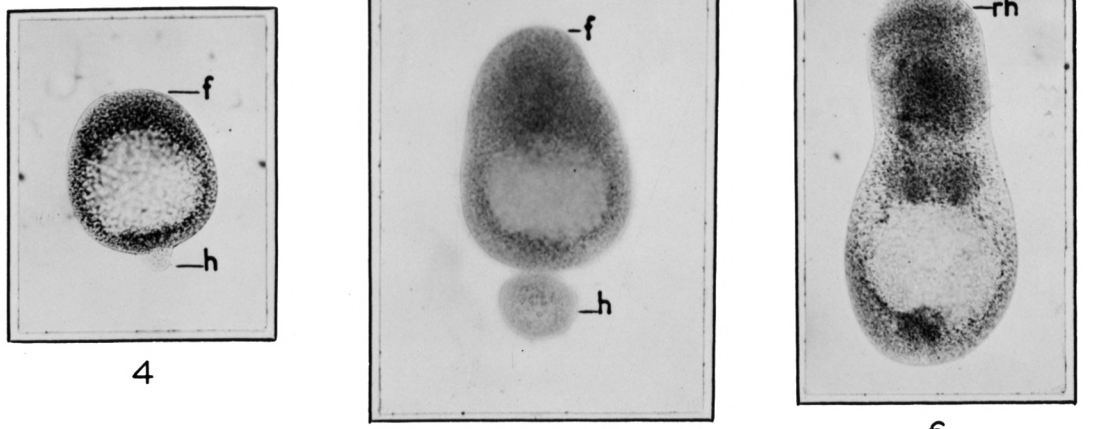
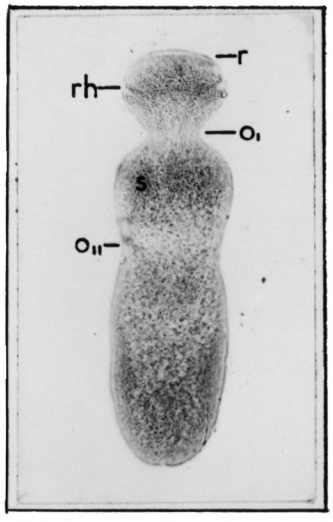


4 5 6

Plate II (continued).



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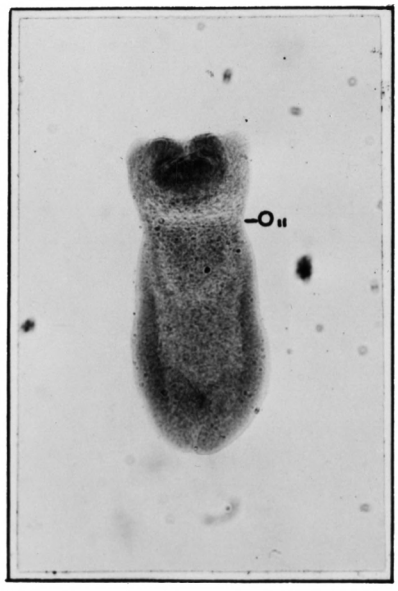

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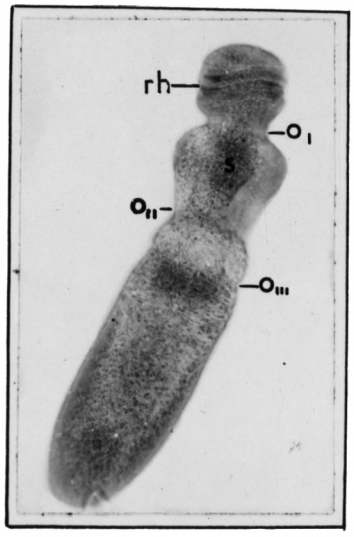

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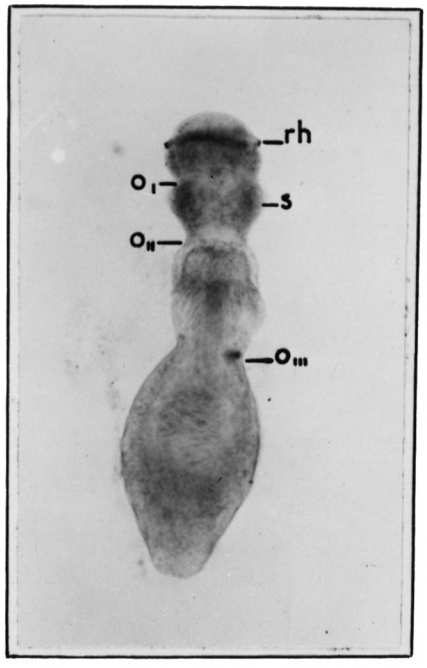

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Plate III.

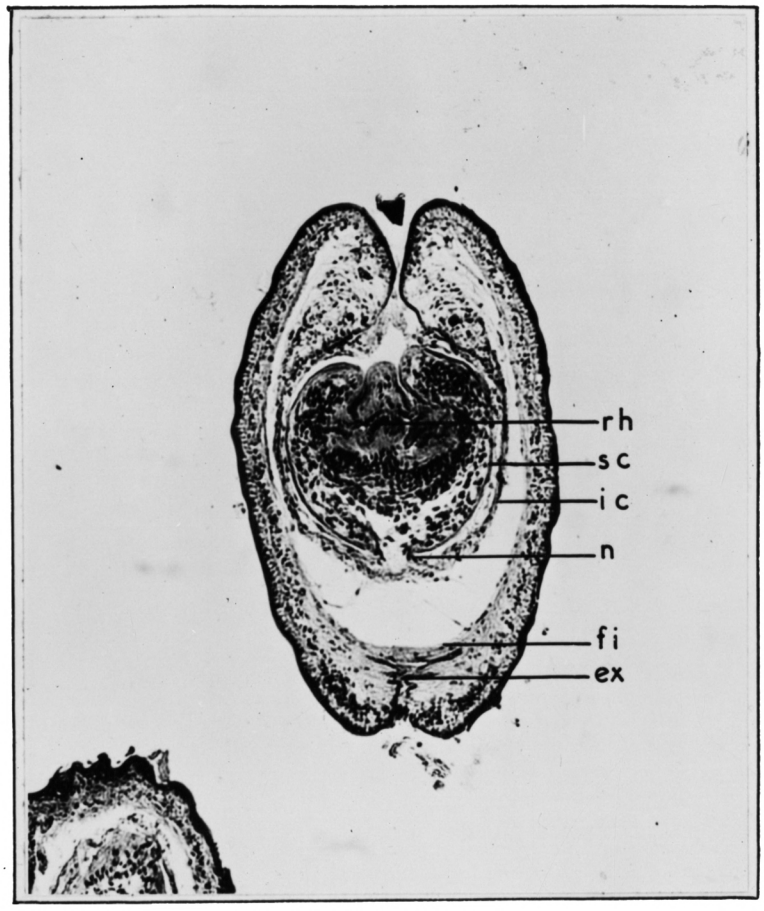

Plate III

Longitudinal section of mature cysticercoid of *Raillietina cesticillus* showing various tissue layers and mode of attachment of scolex to inner cyst wall. Azocarmine stain. (X300)

Key to abbreviations used in the plates

c	primitive cavity	O_1	first constriction
ex	excretory bladder	O_{11}	second constriction
f	hook free pole	O_{111}	third constriction
fi	fibrillar tissue	r	rostellum
h	hook bearing pole	rh	rostellar hooks
ic	inner cyst wall	s	sucker masses
n	neck	sc	scolex