STUDIES ON THE

PATHOGENICITY OF TETRAMERES MOHTEDAI

AND

ACUARIA SPIRALIS OF FOWL

By

K. RAMASWAMY

THESIS

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DECLARATION

I hereby declare that this thesis entitled STUDIES ON THE PATHOGENICITY OF TETRAMERES MOHTEDAI AND ACUARIA SPIRALIS OF FOWLS is a bonafied record of research work done by me during the course of research and that the thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship, or other similar title of any other University or Society.

Place: Mannuthy

Date : 13-8-77

Ramaswamy K

(K. RAMASWAMY)

C E R T I F I C A T E

Certified that this thesis, entitled STUDIES ON THE PATHOGENICITY OF TETRAMERES MOHTEDAI AND ACUARIA SPIRALIS OF FOWLS is a record of research work done independently by Shri. K. Rameswamy under my guidance and supervision and that it has not previously formed the basis for the award of any degree, diploma, fellowship, or associateship to him.

Place: Mannuthy

Date: 13-8-1977

Name of the Guide: Dr. R.K.SUNDARAM
(Chairman Advisory Board)

Designation: Professor

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INTRODUCTION

INTRODUCTION

The Kerala State has been recognized as one of the important Poultry rearing areas within India. The rural population had taken to Poultry farming for over two decades, to augument their family income and millions of hen's and duck eggs are being exported outside the State annually. Most of the Poultry are raised on a small-scale-industry-basis, following the age old barn-yard farming methods.

Profitable Poultry rearing calls forth better managemental measures including timely attention to the control of diseases. While the losses due to mortality are immediately felt by the farmers, losses due to inciduous diseases such as those caused by parasitic helminths are not apparent to the average poultry raisers. The morbidity, loss in egg production or poor weight gain appears to account for greater losses than open infections resulting in immediate mortality.

No systematic effort has been made to determine the ill-effects due to less commonly occurring parasitic helminths such as the spirurid worms. The main reason why such studies had not been attempted before was that, the life cycles of these parasites and the range of intermediate hosts were unknown till recently. Life cycle studies conducted at Kerala Agricultural University have thrown important light on the biology of all Spirurid worms frequently met with among

poultry, so that it is now possible to critically determine the role of these parasites in bringing about losses to Poultry industry. A detailed study on the pathogenicity of the parasites to fowl would also help in the early diagnosis of the disease conditions and to institute timely measures of control.

OBJECTIVE OF THE PRESENT STUDY

The objective of the present study was, therefore, to assess the pathogenicity of two important species of Spirurid worms namely, *Tetrameres mohtedai* and *Acuaria spiralis*, under controlled conditions. The worms chosen for the study inhabit the proventriculus of fowl and quite often a mixed infection with these worms are encountered under natural conditions of poultry rearing. The present investigation was carried out on experimental monospecific *T. mohtedai* and *A. spiralis* infections and dispecific infections with both the parasites at three levels of infection. The levels chosen were; light infection, moderate infection, and heavy infection. In a series of preliminary studies, not mentioned in this work, the threshold number of infective juveniles to produce the desired level of infection was determined in each case. The parameters observed were blood changes, gross and microscopic lesions, effect on weight gain, age at first egg, effect on egg production and feed efficiency.

REVIEW OF LITERATURE

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TETRAMEROSIS

Tetrameres mohtedai was recorded for the first time in India by Bhalae Rao and Rao (1944) who collected 8 males and 10 females from the proventriculus of a fowl that died in Hyderabad (Deccan). Subsequently, its occurrence in India was reported by Anantaraman & Chandrasekharan Nair (1955) from Tamil Nadu; Sundaram *et al.* (1963) from Kerala State; Purohit *et al.* (1965) from Maharashtra State; Mukhopadhayay and Varma (1965) from Bihar and Joshi & Kamalapur (1971) from Madhya Pradesh.

Sundaram *et al.* (1963) elucidated the life cycle of the parasite for the first time using grasshoppers as the intermediate hosts. Subsequently Mukherje & Sinha (1965) found that two species of beetles *Llatongus cinctus* and *Sphaeridium quinquemaculatus* could be infected with eggs of *T. mohtedai*. Lim (1975) found that a Lepidopteran (*Setomorpha rutella*) acts as intermediate host for *T. mohtedai* in Singapore. A complete detailed description of the developmental stages of *T. mohtedai*, in both intermediate and final host was given by Sundaram (1971).

Almost all the earlier works pertaining to the pathogenicity of *T. mohtedai* were based on natural infections

in fowls and more or less confined to the histopathology of the affected proventriculus (Anantaraman & Chandrasekharan Nair (1955); Purohit et al. 1965; and Joshi & Kamalapur 1971). The present investigation, therefore, appears to be the first on the pathogenicity of Tetrameres mohtedai under controlled conditions.

The pathogenic effects of other species of Tetrameres on bird hosts were reported by many authors (Barber 1916; Cram 1928; Sugimoto & Nishiyama 1937; Srivastava 1939; Todd 1946; del Bono 1953; Popov 1953; Panebianco 1955; Raggi & Baken 1957; Nery & Dubois 1960; Sriramalu 1961; Shevtsov & Zabello 1965; Graubmann & Grafner 1967; Platt & Nelson 1969; Karstad & Sileo 1971; and Rao et al. 1972). However no controlled experiments seem to have been conducted so far; except in the case of T. fissispina by Cvetajeva (1960) and in T. anatis by Chandrasekharan (1977). Cheng (1973) stated that, when present in small numbers T. americana does not produce serious symptoms. However, when large numbers occur, the avian host portrays dullness, wasting, emaciation and even death. Death due to Tetrameres infection has also been recorded by del Bono (1953); Graubmann & Grafner (1967) and Karstad & Sileo (1971).

ACUARIASIS

The occurrence of Acuaria spiralis was reported for the first time in India by Maplestone (1932) from a bronze-winged jacana (Metopidius indicus) from the Zoological gardens Calcutta. Subsequently Bhalae Rao & Rao (1944) recorded its occurrence in the proventriculus of a fowl in Hyderabad (Deccan); Mudaliar & Alwar (1947) from Tamil Nadu; Sundaram et al. (1962) from Kerala State; Mukhopadhyay & Varma (1965) from Bihar and Joshi & Kamalapur (1971) from Madhya Pradesh. Sundaram (1971) has given a complete account of the life-cycle with details of morphology of A. spiralis both within the isopodes (intermediate hosts) and fowls.

The main seat of predilection of A. spiralis is the lumen of proventriculus. But occasionally they are also encountered in the oesophagus (Hsu 1959; Merkusheva & Kraevskia 1963; Slater 1967; and Bwangamoi 1968) and small intestine (Ahamad 1962; Hassan 1966; Diaz-Ungria & Torres Artigas 1966 and Bwangamoi 1968).

Acuaria spiralis is known to cause severe pathogenic effects in their avian hosts (Allen 1925; Cram 1928; Kocjan 1934; Bump et al. 1947; Edminster 1947; Pijuan 1949; Nescos 1954; Bendell 1955; Olano Vilchez 1958; Wehr 1959; Hwang

et al. 1961; Afnan & Mirzaians 1963; Deo 1964; Catteleer 1965; Soulsby 1965, 1968; and Karstad & Silco 1971).

The histopathological lesions in natural A. spiralis infection of fowls were studied by Joshi & Kamalapur (1971). However, no controlled studies have been undertaken on the pathogenic effects of the parasite, as the lifecycle of A. spiralis of fowl has been reported for the first time, only in 1971 by Sundaram.

HAEMATOLOGICAL CHANGES AND OTHER PATHOGENIC EFFECTS

Very little work has been done on the blood changes in helminthoses of fowl. Particularly no information is available in the literature regarding the haematological changes in T. mohtedai and A. spiralis infections of fowl. Olson & Levine (1939) have studied the blood changes in experimental Capillaria columbae infection in chicks. Wickware (1947) has recorded the blood changes in controlled infection of chicks with Heterakis gallinae. The blood picture of fowls in experimental infections with Ascaridia galli and H. gallinae before and after dehelminthisation were studied by Kenezik & Lukacova (1969). The effect of coccidia, A. galli and H. gallinae in dual infections were studied by Patil et al. (1972).

MATERIALS AND METHODS

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MATERIALS AND METHODS

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COLLECTION OF WORMS

Worms for the present study were collected from infected proventriculi of exotic and desi fowls, procured from local hotels and slaughter houses.

COLLECTION, MAINTENANCE & ARTIFICIAL INFECTION OF INTERMEDIATE HOSTS

Grasshoppers of the species Spathosternum prasiniferum, Conocephalus maculatus and Oxya nitidula caught from rice fields were used as intermediate hosts for Tetrameres mohtedai.

Isopodes of the species Porcellio laevis collected from cattle manure and cultured in the laboratory were used as intermediate hosts for Acuaria spiralis.

For the maintenance and infection of intermediate hosts, the technique described by Sundaram (1971) was adopted.

COLLECTION OF INFECTIVE LARVAE

The encysted third stage juveniles of T.mohtedai were harvested from grasshoppers on the 25th day after infection, so as to ensure infectivity of the juveniles to chicks. The larvae were counted and transferred to fresh physiological saline solution and infections were attempted within 2 hours after dissection of grasshoppers.

Third stage A. spiralis larvae were dissected out from

isopodes on the 30th day post exposure of eggs. The larvae were counted and transferred to fresh physiological saline solution and infections were attempted within 3 hours after collection.

INFECTING THE FINAL HOST

Counted number of larvae were fed directly into the crop of the birds using a pasteur pipette.

PATHOLOGICAL STUDIES

To study the pathogenic effects of the parasites on the host, observations on haematology (total erythrocytes, total leucocytes, haemoglobin, packed cell volume, mean corpuscular volume, mean corpuscular haemoglobin, mean corpuscular haemoglobin concentration and differential count), weight gain, age at first egg, intensity of laying, feed efficiency and histopathology were made.

For haematological studies blood samples were collected from the wing vein using di-sodium salt of EDTA (0.3 to 0.4 mg. per ml. of blood) as the anticoagulant. The blood samples were collected on the 6th, 13th, 24th, 38th, 52nd and the 66th day post infection in the case of pure (monospecific) Acuaria spiralis and mixed (dispecific) infections with A. spiralis and T. mohtedai, and on the 3rd, 13th, 28th, 42nd, 56th and 70th day post infection in the case of pure (monospecific) T. mohtedai infection, which corresponded to the different stages in the lifecycles of the parasites involved.

Total erythrocyte and total leucocyte counts were made by direct method using Nambiar's fluid (Nambiar, 1961) as the diluent. Duplicate counts were made and the average was taken.

Haemoglobin percentage was directly read from Erma electronic Hnemo Photo Meter. Drabkin's solution was used as the diluent. Before taking the reading the mixture was gently agitated with a glass rod to render the mixture clear by flocculating and floating off of the turbid material.

Packed cell volume was taken after centrifuging the sample in Wintrobe's tubes for 30 minutes at 3000 revolutions per minute.

Mean corpuscular volume, mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration were calculated from the values obtained above.

For differential count, duplicate smears from each sample were stained by modified copper peroxide method of Sato & Sekiya (Nambiar, 1961) which gave better differentiation of eosinophils from heterophils and by Wright-Giemsa. One hundred cells were counted from each smear and the average of the two was calculated.

The weight of the birds were recorded at weekly intervals after infection to note the weekly weight gain.

Feed efficiency was calculated by noting the amount of feed required in kilogrammes to obtain a dozen of eggs.

The female birds were kept under observation for determining the age at first egg and intensity of laying. Data regarding the latter were collected only for a period of 66 days.

All the values obtained were Statistically analysed using a randomised block design.

To study the gross and histopathological changes produced by the various developmental stages of the parasites in the host, the infected birds were autopsied on the 4th, 7th, 9th, 12th, 15th, 18th, 25th, 32nd, 39th and 55th day post infection in the case of monospecific T.mohtedai infection, 4th, 8th, 10th, 12th, 14th, 16th, 18th, 20th, 22nd, 24th, 50th and 100th day post infection in the case of monospecific A.spiralis infection and on the 2nd, 8th, 12th, 15th, 20th, 24th, 29th, 32nd, 36th, 40th, 50th and 100th day post infection in the case of dispecific infections. Bouin's Fluid and 7% formaline solution were used as routine fixatives. Tissues were embedded in paraffin and 5 to 7 μ sections were cut. Duplicate sections were stained by Lillie Mayer's Haematoxylin and by Heidenhain's Azan stain. Special stains like Picro-Mallory and Van Gieson's stain were employed wherever found necessary.

PLAN OF EXPERIMENTS

Pathogenicity was determined in monospecific A.spiralis infection, monospecific T.mohtedai infection and in dispecific infections with both the above parasites.

For haematological studies 36 White Leg Horn chicks (24 males and 12 females) procured from University Poultry Farm, Mannuthy, were wing banded at 2 weeks of age and were separated into 3 equal batches of 12 birds each (8 males and 4 females), by random selection. The 12 birds in each batch were then sub-divided into 4 equal groups of 3 birds each (2 males and 1 female) by random selection and infections were given at three levels as follows.

	Batch I monospecific *A. spiralis* infection	Batch II monospecific *T. mohtedai* infection	Batch III dispecific infections with *A. spiralis* and *T. mohtedai*.
Group I	100 juveniles each	200 juveniles each	100 + 200 juveniles each respectively
Group II	200 juveniles each	400 juveniles each	200 + 400 juveniles each respectively
Group III	400 juveniles each	600 juveniles each	400 + 600 juveniles each respectively
Group IV	Negative controls	Negative controls	Negative controls

For histopathological studies 36 White Leghorn male chicks procured from the University Poultry Farm, Mannuthy, were wing banded at two weeks of age and separated into 3 equal groups of 12 birds each. Group I birds were infected with 500 juveniles of *A. spiralis* each, Group II birds were infected with 500 juveniles of *T. mohtedai* each and Group III birds were infected with 500 juveniles each of *A. spiralis* and *T. mohtedai*.

RESULTS

PATHOGENICITY
OF
Tetrameres mohtedai

PATHOGENICITY OF TETRAMERES MOHTEDAI

HAEMATOLOGICAL OBSERVATIONS

To study the haematological alterations in three levels of monospecific Tetrameres mohtedai infection, the blood samples were collected on the 3rd (corresponding to the third stage), 13th (corresponding to the fourth stage), 28th, 42nd, 56th and 70th day (corresponding to the adult stage) post infection. The results obtained were as follows :-

TOTAL ERYTHROCYTES

The mean overall erythrocyte count of the entire experimental groups was significantly lower ($P<0.01$) than the negative controls.

The decrease in respect of Group I (average number of worms established 68) was 18.09% ($P<0.01$); in respect of Group II (average number of worms established 114.33) 22.66% ($P<0.01$) and in respect of Group III (average number of worms established 164) 23.81% ($P<0.01$).

On a further analysis Group I showed a decrease of 37.14% ($P<0.01$) on the 70th day post infection, Group II showed a decrease of 26.13% & 25.92% ($P<0.05$) on the 42nd & 56th day post infection and Group III showed a decrease of 19.86% ($P<0.05$) 25.75% & 37.14% ($P<0.01$) on the 42nd, 56th & 70th day post infection respectively.

Thus the total erythrocytes of the infected groups showed

a gradual decrease from the 42nd day post infection and was maximum by the 70th day.

The data are presented in Table 1 and are graphically represented in Chart 1.

HAEMOGLOBIN

The mean overall haemoglobin percentage of the entire experimental groups was significantly lower ($P<0.01$) than the negative controls.

The decrease in respect of Group I (average number of worms established 68) was 7.96% ($P<0.01$); in respect of Group II (average number of worms established 114.33) 11.78% ($P<0.01$) and in respect of Group III (average number of worms established 164) 17.85% ($P<0.01$)

On a further analysis, Group I showed a decrease of 16.12% ($P<0.05$) on the 70th day post infection, Group II showed a decrease of 15.25%, 15.24% ($P<0.05$) & 27.06% ($P<0.01$) on the 42nd, 56th & 70th day post infection respectively and Group III showed a decrease of 14.14% ($P<0.05$), 28.10% & 24.71% ($P<0.01$) on the 28th, 56th & 70th day post infection respectively.

Thus the haemoglobin percentage was found to be decreased on the 70th day post infection in Group I; 42 nd day post infection in Group II and from the 28th day post infection in Group III.

The data are presented in Table 2 and are graphically represented in Chart 2.

PACKED CELL VOLUME

The mean overakl packed cell volume of the entire experimental groups was significantly lower ($P<0.01$) than the negative controls.

The decrease in respect of Group I (average number of worms established 68) was not significant; in respect of Group II (average number of worms established 114.33) 8.73% (significant at 1% level) and in respect of Group III (average number of worms established 164) 8.82% (significant at 1% level).

On a further analysis, on the 56th and 70th day post infection Group I showed a decrease of 7.31% and 9.38%. ($P<0.05$) respectively; Group II showed a decrease of 20.84% and 17.72% ($P<0.01$) respectively and Group III showed a decrease of 20.84% and 16.69% ($P<0.01$) respectively.

Thus the packed cell volume of the infected birds were found to decrease from the 56th day post infection.

The data are presented in Table 3 and are graphically represented in Chart 3.

MEAN CORPUSCULAR VOLUME

The overall mean corpuscular volume of the entire experimental groups was significantly higher ($P<0.01$) than the negative controls.

The increase in respect of Group I (average number of

worms established 68) was 35.89% (P<0.01%); in respect of Group II (average number of worms established 114.33) 26.08% (P<0.01%) and in respect of Group III (average number of worms established 164) 20.87% (P<0.01%).

On a further analysis, on the 70th day post infection Group I showed an increase of 43.81% (P<0.01%); Group II 77.65% (P<0.01%) and Group III 37.11% (P<0.01%)

Thus the mean corpuscular volume was found to be almost normal upto 56th day post infection but by the 70th day a sharp increase was noticed in all the experimental groups.

The data are presented in Table 4 and are graphically represented in Chart 4.

MEAN CORPUSCULAR HAEMOGLOBIN

The overall mean corpuscular haemoglobin of the entire experimental groups was significantly higher (P<0.01) than the negative controls.

The increase in respect of Group I (average number of worms established 68) was 15.23% (P<0.01); in respect of Group II (average number of worms established 114.33) 19.59% (P< 0.01) and in respect of Group III (average number of worms established 164) 14.32% (P< 0.01).

When further analysed, Group I showed an increase of 32.97% (P< 0.01); Group II 57.33% (P< 0.01) and Group III 25.93% (P< 0.05) over the controls on the 70th day post

infection.

The mean corpuscular haemoglobin was found to be almost normal upto 56th day post infection, but by the 70th day an increase was noticed in all the experimental groups.

The data are presented in Table 5.

MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION

The mean corpuscular haemoglobin concentration of the experimental groups showed no significant difference from those of the negative controls. However by the 70th day post infection all the groups showed an average decrease of 9.30% (not significant) indicating that the anaemia is becoming hypochromic.

TOTAL LEUCOCYTES

The total leucocytes of the entire experimental groups was significantly higher ($P < 0.01$) than the negative controls.

The increase in respect of Group I (average number of worms established 68) was not significant, but in respect of Group II (average number of worms established 114.33) and Group III (average number of worms established 164) the increase were 10.01% & 27.44%(significant at 1% level) respectively over the controls.

On further analysis, Group I showed an increase of 44.02% and 119.13% ($P < 0.01$) on the 3rd and 13th day post infection respectively, Group IIIshowed an increase of 97.85% & 147.80% ($P < 0.01$) on the 3rd and 13 th day post infection respectively, and Group III showed an increase of

52.90%, 132.27% & 68.97% ($P < 0.01$) on the 3rd, 13th, & 42nd day post infection respectively. On the 56th day post infection all the experimental groups showed a significant decrease of 46.99% ($P < 0.05$) and on the 70th day post infection the decrease was 26.23%.

Thus a leucocytosis was observed upto 28th day post infection in Group I & Group II and upto 42nd day post infection in Group III. On the 56th day a leucopenia was observed in all the experimental groups which by the 70th day post infection was less marked.

The data are presented in Table 7 and are graphically represented in Chart 6.

EOSINOPHILS

The overall eosinophil percentage of the entire experimental groups was significantly higher ($P < 0.01$) than the negative controls.

The increase in respect of Group I (average number of worms established 68) was 53.64% ($P < 0.01$) in respect of Group II (average number of worms established 114.33) 42.17% ($P < 0.05$) and in respect of Group III (average number of worms established 164) it was 91.80% ($P < 0.01$) over the controls.

On further analysis, Group I showed an increase of 114.6% & 94.33% ($P < 0.05$) on the 3rd & 13th day post infection respectively, Group II showed an increase of 85.84% & 60.06%

on the 3rd & 28th day post infection respectively. Group III showed an increase of 143.35% ($P < 0.01$), 89%, 65.17%($P < 0.05$) and 127.67% ($P < 0.01$) on the 3rd, 13th, 28th & 42nd day post infection respectively.

Thus eosinophilia was observed on the 3rd and 13th day in Group I, 3rd and 28th day in Group II and the 3rd to 42nd day in Group III.

The data are presented in Table 8 and are graphically represented in Chart 5.

HETEROPHILS

The mean heterophil percentage of Group III was significantly higher ($P < 0.01$) than the negative controls.

Initially there was a decrease of 27.25% on the 3rd day post infection in all the experimental groups. But on the 42nd day post infection Groups II and III showed an increase of 50% and 50.72% ($P < 0.05$) respectively. On the 70th day post infection, Group III showed an increase of 66.47% ($P < 0.01$) over the controls.

The data are presented in Table 9 and are graphically represented in Chart 7.

BASOPHILS

The overall basophil percentage in the experimental groups showed no significant difference from the negative controls.

The data are presented in Table 10.

LYMPHOCYTES

The overall lymphocyte percentage of the entire experimental groups was significantly lower ($P < 0.05$) than the negative controls.

The decrease in respect of Group I (average number of worms established 68) and Group II (average number of worms established 114.33) were not significant, but in respect of Group III (average number of worms established 164) it was 12.94% (significant at 1% level).

On further analysis, Group III showed a decrease of 25.70% and 33.50% ($P < 0.01$) on the 42nd & 70th day post infection respectively over the controls.

Thus lymphocytopenia was observed in heavy infections from the 42nd day post infection.

The data are presented in Table 11 and are graphically represented in Chart 8.

MONOCYTES

The overall monocyte percentage of the entire experimental groups was significantly higher ($P < 0.01$) than the negative controls.

Group I (average number of worms established 68) showed no significant difference, but the increase in respect of

Group II (average number of worms established 114.33) and Group III (average number of worms established 164) were 67.43% & 77.07% ($P < 0.01$), respectively over the controls.

On further analysis, Group II showed an increase of 143.223% & 175% ($P < 0.05$) on the 56th and 70th day post infection. Group III showed an increase of 157.51% and 166.65% ($P < 0.01$) on the 56th and 70th day post infection respectively.

Thus monocytosis was observed in birds harbouring more than 114 worms from the 56th day post infection.

The data are presented in Table 12.

TABLE - 1 - Showing the total Erythrocyte count in three levels of monospecific T. mohtedai infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
3rd	2.56	2.54	2.46	2.65	3.01	2.60	2.55	2.65	2.84	2.33	3.67	3.21
13th	3.34	2.73	2.39	1.81	2.75	3.39	1.83	2.35	1.89	2.86	3.19	2.49
28th	2.03	1.81	2.00	2.33	1.47	2.02	1.99	1.85	1.88	2.33	2.53	2.25
42nd	2.09	1.33	2.05	2.17	1.51	1.52	2.16	1.92	1.56	2.51	2.27	2.26
56th	2.06	1.80	2.12	1.90	1.67	1.61	1.82	1.65	1.72	2.44	2.50	2.06
70th	1.52	1.65	1.64	0.93	1.25	1.40	1.22	1.68	1.91	2.35	2.40	2.90

All values in millions/cu.mm. of blood

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	5.039	1.679	15.270 **
Between Days	5	9.836	1.967	17.881 **
Error	63	6.936	0.110	
TOTAL	71	21.811		

** Significant at 1% level

TABLE - 2 - Showing the Haemoglobin values in three levels of monospecific T. mohtedai infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
3rd	7.6	7.4	7.4	8.2	9.0	8.8	8.0	6.8	6.6	8.0	8.2	8.4
13th	8.6	7.8	8.6	7.6	9.8	7.2	8.2	7.6	6.8	9.8	8.8	7.8
28th	8.5	8.8	8.0	8.4	5.6	7.8	6.8	7.8	6.6	8.0	8.3	8.4
42nd	7.6	7.8	8.4	7.8	6.8	6.6	7.7	8.0	6.2	7.8	8.4	8.8
56th	6.2	8.2	8.4	7.4	6.7	7.6	7.0	5.8	5.6	8.2	9.4	8.0
70th	7.8	6.6	7.0	5.6	6.4	6.6	6.2	7.0	6.0	8.0	8.9	8.6

All values in gram percentage

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	26.022	8.674	17.348 **
Between Days	5	9.522	1.905	3.810 **
Error	63	31.509	0.500	
TOTAL	71	67.053		

** Significant at 1% level.

TABLE - 3 - Showing the Packed cell volume in three levels of monospecific T. mohtedai infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
3rd	30.0	30.0	30.0	30.0	30.0	30.0	30.0	30.0	30.0	31.0	31.0	31.0
13th	31.5	29.0	31.0	31.0	34.0	27.0	28.0ø	32.0	23.5	25.5	33.0	28.5
28th	35.0	30.0	35.0	29.0	25.0	31.0	26.0	30.0	28.0	29.0	29.0	27.0
42nd	30.0	31.0	34.0	31.0	28.0	21.0	30.0	31.5	26.5	28.0	32.0	33.0
56th	31.0	29.0	29.0	26.0	25.0	25.0	27.0	24.0	25.0	31.0	35.0	30.0
70th	32.0	25.0	30.0	24.0	26.0	29.0	27.0	29.0	24.0	30.0	33.0	33.0

All values in percentage

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	228.166	76.055	13.267 **
Between Days	5	38.666	7.733	1.349 n.s.
Error	63	361.168	5.732	
TOTAL	71	628.000		

** Significant at 1% level

n.s. Not significant.

TABLE - 4 - Showing the Mean corpuscular volume in three levels of monospecific T. mohtedai infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
3rd	117.19	118.11	121.95	113.21	99.67	115.38	117.65	113.21	105.63	133.05	84.47	96.57
13th	94.31	106.23	129.71	171.27	123.64	79.65	153.01	136.17	124.34	89.16	103.45	114.46
28th	172.41	165.75	175.00	124.46	170.07	153.47	130.65	162.16	148.94	124.46	114.62	120.00
42nd	143.54	233.08	165.85	142.86	185.43	138.16	138.89	164.06	169.87	111.55	140.97	146.02
56th	150.49	161.11	136.79	136.84	149.70	155.28	148.35	145.45	145.35	127.05	140.00	145.63
70th	210.53	151.52	182.93	258.06	208.00	207.14	221.31	172.62	125.65	127.66	137.50	113.79

All values in cubic microns

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	13990.5	4663.500	8.282 **
Between Days	5	34911.8	6982.360	12.399 **
Error	63	35475.4	563.102	
TOTAL	71	84377.7		

** Significant at 1% level.

TABLE - 5 - Showing the Mean corpuscular haemoglobin in three levels of monospecific T. mohtedai infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
3rd	29.69	29.13	30.08	30.94	29.90	33.85	31.37	25.66	23.24	34.33	22.34	26.17
13th	25.75	28.57	35.98	41.99	35.64	21.24	44.81	32.34	38.98	34.27	27.59	31.33
28th	41.87	43.09	40.00	36.05	38.10	38.61	34.17	42.16	35.11	34.33	32.81	37.33
42nd	36.36	58.65	40.98	35.94	45.03	43.42	35.65	41.67	39.74	31.08	37.00	38.94
56th	30.10	45.56	39.62	38.95	40.12	47.20	38.46	35.15	32.56	33.61	37.60	38.83
70th	51.32	40.00	42.68	60.22	51.20	47.14	50.82	41.67	31.41	34.04	37.08	29.66

All values in micromicro grams.

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	641.840	213.947	7.155 **
Between Days	5	1590.920	318.183	10.642 **
Error	63	1883.720	29.900	
TOTAL	71	4116.480		

** Significant at 1% level.

TABLE - 6 - Showing the Mean corpusculer haemoglobin concentration in three levels of monospecific *T. mohtedai* infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
3rd	25.33	24.67	24.67	27.33	30.00	29.33	26.67	22.67	22.00	25.81	26.45	27.10
13th	27.30	26.90	27.74	24.52	28.82	26.67	29.29	23.75	28.94	38.43	26.67	27.37
28th	24.29	26.00	22.86	28.97	22.40	25.16	26.15	26.00	23.57	27.59	28.62	31.11
42nd	25.33	25.16	24.71	25.16	24.29	31.43	25.67	25.40	23.40	27.86	26.25	26.67
56th	20.00	28.28	28.97	28.46	26.80	30.40	25.93	32.50	22.40	26.45	26.86	26.67
70th	24.38	26.40	23.33	23.33	24.62	22.76	22.96	24.14	25.00	26.67	26.97	26.06

All values in percentage

Analysis of Variance Table

Source	df	SS	MSS	F	
Between Treatments	3	113.452	37.817	2.617	n.s.
Between Days	5	74.831	14.966	1.036	n.s.
Error	63	910.328	14.449		
TOTAL	71	1098.611			

n.s. Not significant.

TABLE - 7 - Showing the total Leucocyte count in three levels of monospecific T. mohtedai infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
3rd	40.00	41.34	38.99	45.30	52.00	68.00	48.00	34.21	45.54	30.00	28.22	25.33
15th	38.00	43.32	30.00	48.44	38.00	39.45	48.00	28.00	42.00	18.00	18.80	14.00
28th	30.00	29.50	26.00	26.00	35.10	32.00	34.88	30.00	32.88	28.50	29.10	28.00
42nd	18.40	27.10	13.50	27.80	20.80	23.10	29.80	36.70	36.40	20.70	16.90	23.30
56th	14.90	13.80	8.70	14.20	10.00	16.20	12.20	7.80	18.00	24.20	13.10	35.50
70th	14.32	24.00	10.22	17.32	14.88	15.32	16.44	18.44	16.00	21.10	21.76	23.54

All values in thousands/cu.mm. of blood.

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	2486.849	828.949	24.991 **
Between Days	5	5789.194	1157.838	34.766 **
Error	63	2098.085	33.302	
TOTAL	71	10374.128		

** Significant at 1% level.

TABLE - 8 - Showing the percentage of Eosinophils in three levels of monospecific T. mohtedai infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
3rd	5.0	5.5	4.5	5.0	5.0	3.0	3.0	6.0	8.0	1.0	3.0	3.0
13th	9.0	5.0	3.5	3.0	4.0	3.0	5.0	8.0	4.0	3.0	3.0	3.0
28th	7.0	3.0	4.5	6.0	6.0	4.0	6.0	7.0	3.5	3.0	3.0	3.0
42nd	5.0	3.0	3.0	3.0	5.0	2.0	6.0	5.0	9.5	2.0	5.0	2.0
56th	3.0	3.0	4.0	3.0	5.0	3.0	3.0	4.5	5.0	2.5	3.0	2.0
70th	2.5	3.0	3.0	2.5	3.5	3.0	4.5	3.5	1.5	2.0	2.5	1.5

Analysis of Variance Teble

Source	df	SS	MSS	F
Between Treatments	3	81.736	27.245	10.708 **
Between Days	5	33.944	6.789	2.668 *
Error	63	160.306	2.545	
TOTAL	71	275.986		

** Significant at 1% level

* Significant at 5% level.

TABLE - 9 - Showing the percentage of Heterophils in three levels of monospecific P. mohtedai infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
3rd	25.5	29.0	20.0	17.0	33.0	25.0	18.0	24.0	30.0	35.0	32.0	34.5
13th	32.0	36.0	37.5	20.0	35.0	31.0	23.5	40.0	32.0	42.5	23.5	28.5
28th	24.0	16.0	40.0	38.0	28.0	36.5	27.0	36.0	26.0	36.0	27.0	32.0
42nd	18.5	30.0	37.0	41.0	38.0	26.0	35.0	41.0	29.5	21.0	19.0	30.0
56th	19.0	32.0	42.0	31.0	33.0	32.0	47.0	28.5	29.0	31.5	23.5	29.0
70th	20.5	17.5	44.5	18.5	25.0	35.5	39.0	39.5	58.0	31.0	23.5	27.5

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	1134.595	378.198	7.170 **
Between Days	5	201.449	40.289	0.763 n.s.
Error	63	3322.676	52.7408	
TOTAL	71	4658.720		

** Significant at 1% level
n.s. Not significant.

TABLE - 10 - Showing the percentage of Eosophils in three levels of monospecific P. mohtedai infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
3rd	0.0	0.5	0.5	0.0	0.0	1.0	0.0	0.0	1.0	0.0	1.0	0.5
13th	0.5	0.0	0.0	0.0	0.0	0.0	0.5	0.0	0.5	0.5	0.5	0.5
28th	0.0	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
42nd	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
56th	1.0	0.5	1.0	0.5	1.0	0.0	0.5	0.5	0.5	0.5	0.5	0.5
70th	0.0	0.5	0.5	0.5	0.0	0.5	0.0	0.5	0.5	0.0	0.0	0.0

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	0.583	0.194	1.532 n.s
Between Days	5	2.292	0.458	3.612 **
Error	63	8.000	0.127	
TOTAL	71	10.875		

n.s. Not significant

** Significant at 1% level.

TABLE - 11 - Showing the percentage of Lymphocytes in three levels of monospecific T. mohtedai infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
3rd	67.0	59.0	70.0	72.0	60.0	67.0	71.0	68.0	58.0	60.0	60.0	57.0
13th	55.5	57.0	56.5	75.0	60.0	63.0	69.0	50.0	61.5	52.0	71.0	66.0
28th	68.0	80.0	55.0	52.0	64.0	58.5	66.0	55.0	68.0	60.5	69.0	63.0
42nd	73.5	63.0	58.0	52.0	54.0	67.0	55.0	48.0	56.0	75.0	72.0	67.0
56th	75.0	62.5	50.0	61.0	54.0	60.0	43.5	59.5	60.0	62.5	70.5	67.5
70th	73.5	76.0	47.5	75.5	64.5	54.5	50.0	53.5	33.5	66.0	71.5	68.5

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	1494.580	498.193	8.198 **
Between Days	5	111.030	22.206	0.365 n.s.
Error	63	3828.620	60.772	
TOTAL	71	5434.230		

** Significant at 1% level

n.s. Not significant.

TABLE - 12 - Showing the percentage of Monocytes in three levels of monospecific T. mohtedai infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
3rd	2.5	6.0	5.0	6.0	2.0	4.0	8.0	2.0	3.0	4.0	4.0	5.0
13th	3.0	2.0	2.5	2.0	1.0	3.0	2.0	2.0	2.0	2.0	2.0	2.0
28th	1.0	1.0	0.5	4.0	1.0	1.0	1.0	2.0	2.5	0.5	1.0	1.0
42nd	3.0	4.0	2.0	4.0	3.0	5.0	4.0	6.0	5.0	2.0	4.0	1.0
56th	2.0	2.0	3.0	5.0	7.0	5.0	6.0	7.0	5.0	3.0	3.0	1.0
70th	3.5	3.0	4.5	3.0	7.0	6.5	6.5	3.0	6.5	1.0	2.5	2.5

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	45.236	15.079	8.084 **
Between Days	5	89.236	17.847	9.568 **
Error	63	117.514	1.865	
TOTAL	71	251.986		

** Significant at 1% level.

IN MONOSPECIFIC T. MOHTEDAI INFECTION

CHART-1 TOTAL ERYTHROCYTES

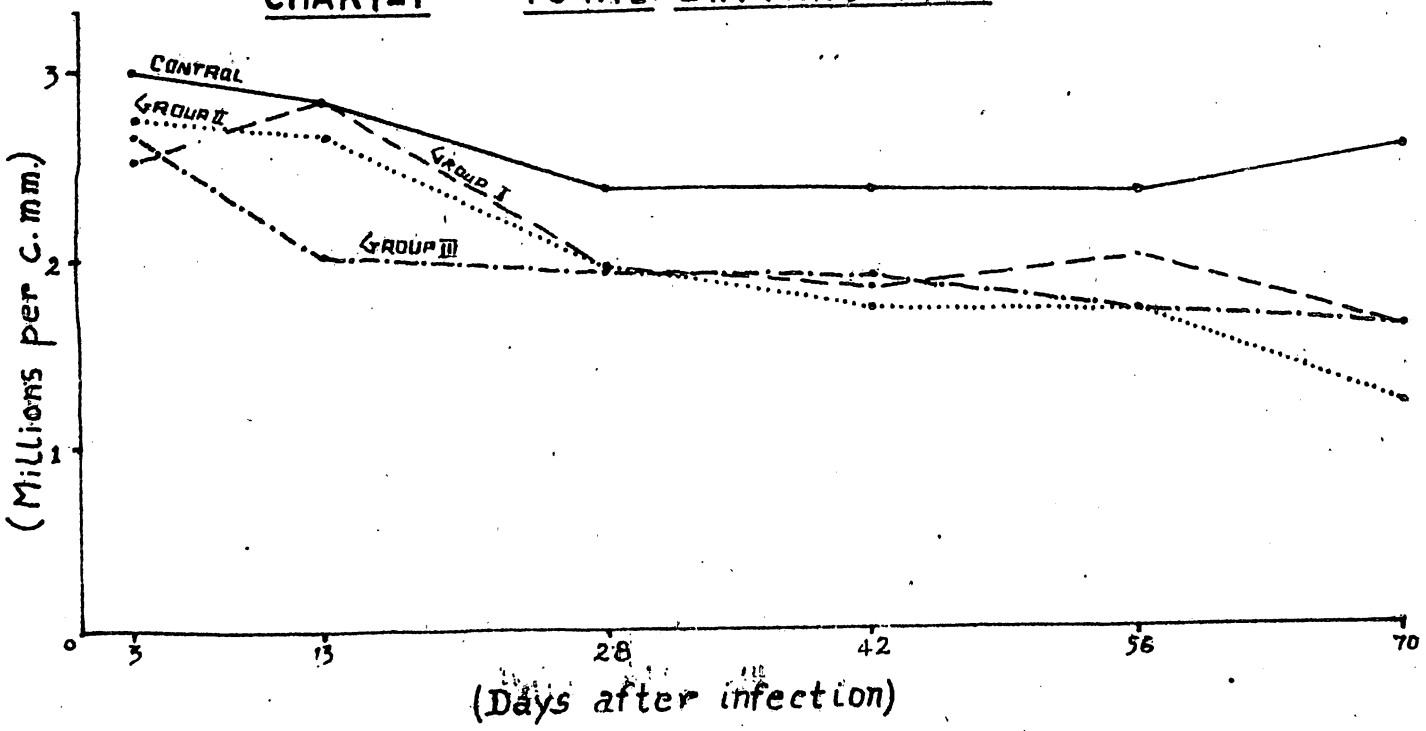
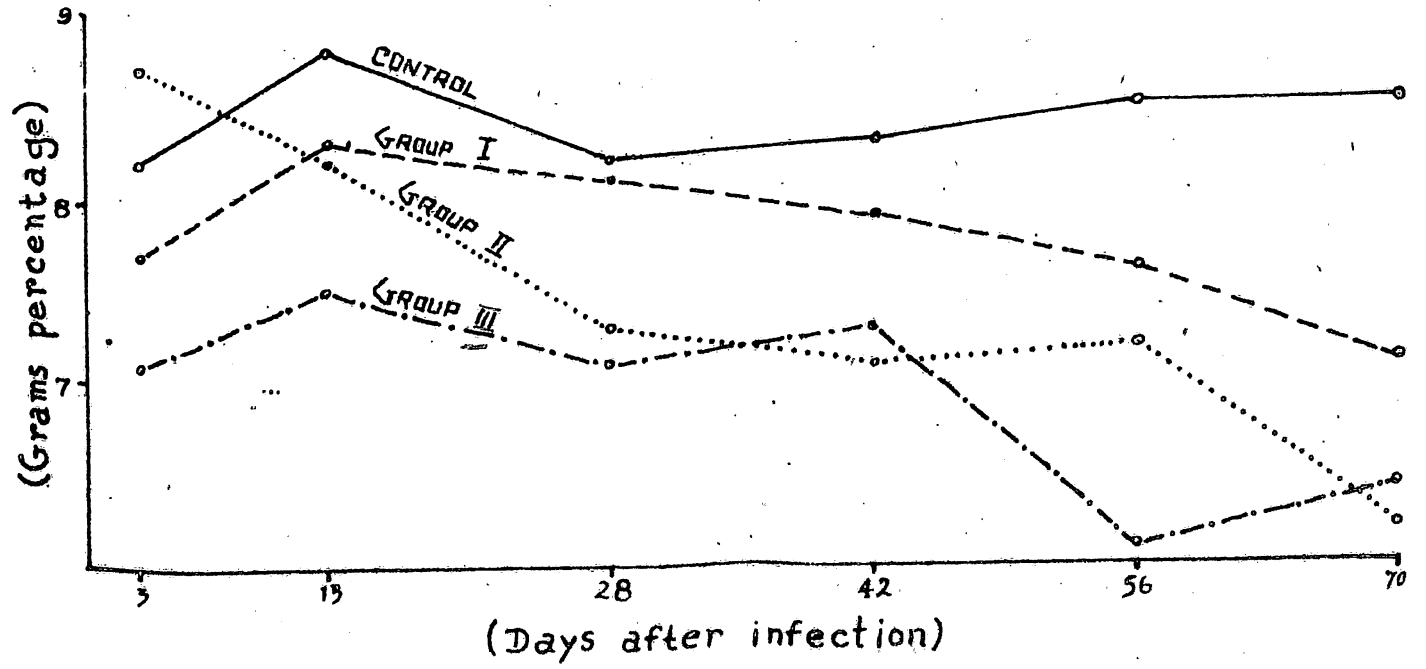


CHART-2 HAEMOGLOBIN



IN MONOSPECIFIC T. MOHTEDAI INFECTION

CHART 3 PACKED CELL VOLUME

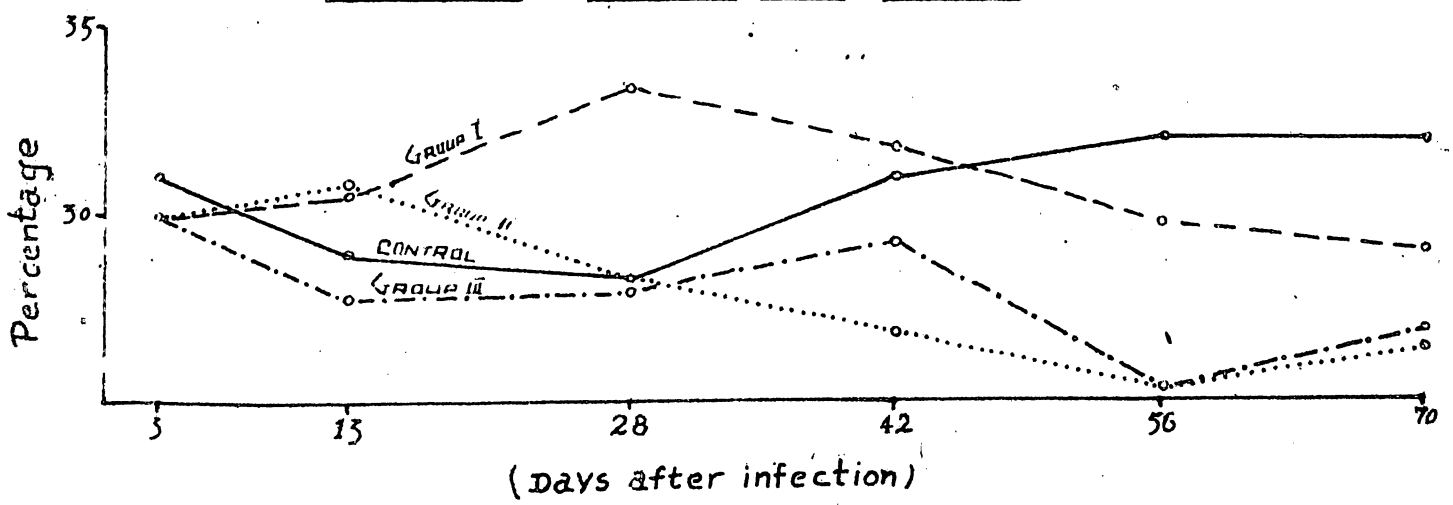
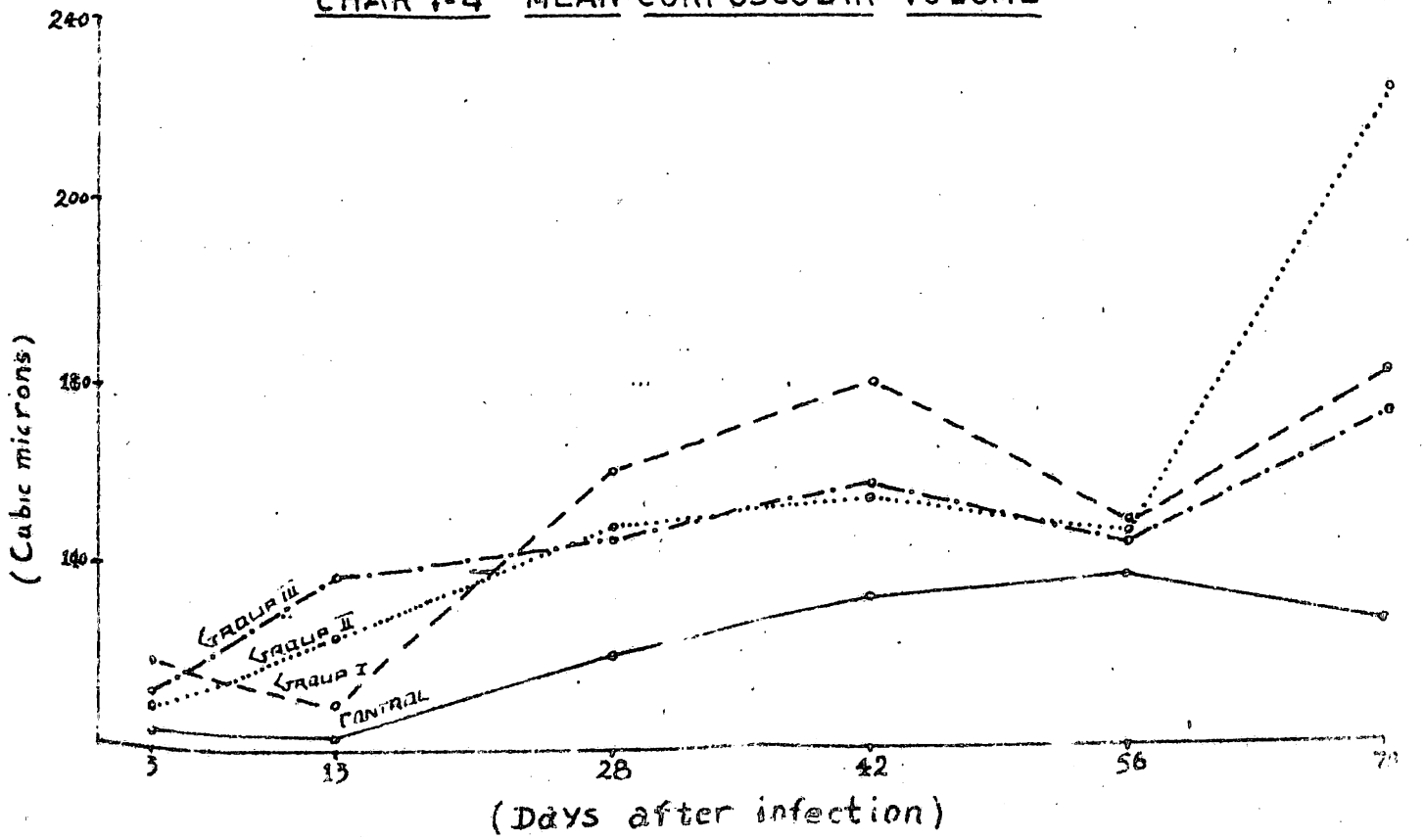


CHART-4 MEAN CORPUSCULAR VOLUME



IN MONOSPECIFIC T. MOHTEDAI INFECTION

CHART-5 TOTAL LEUCOCYTES

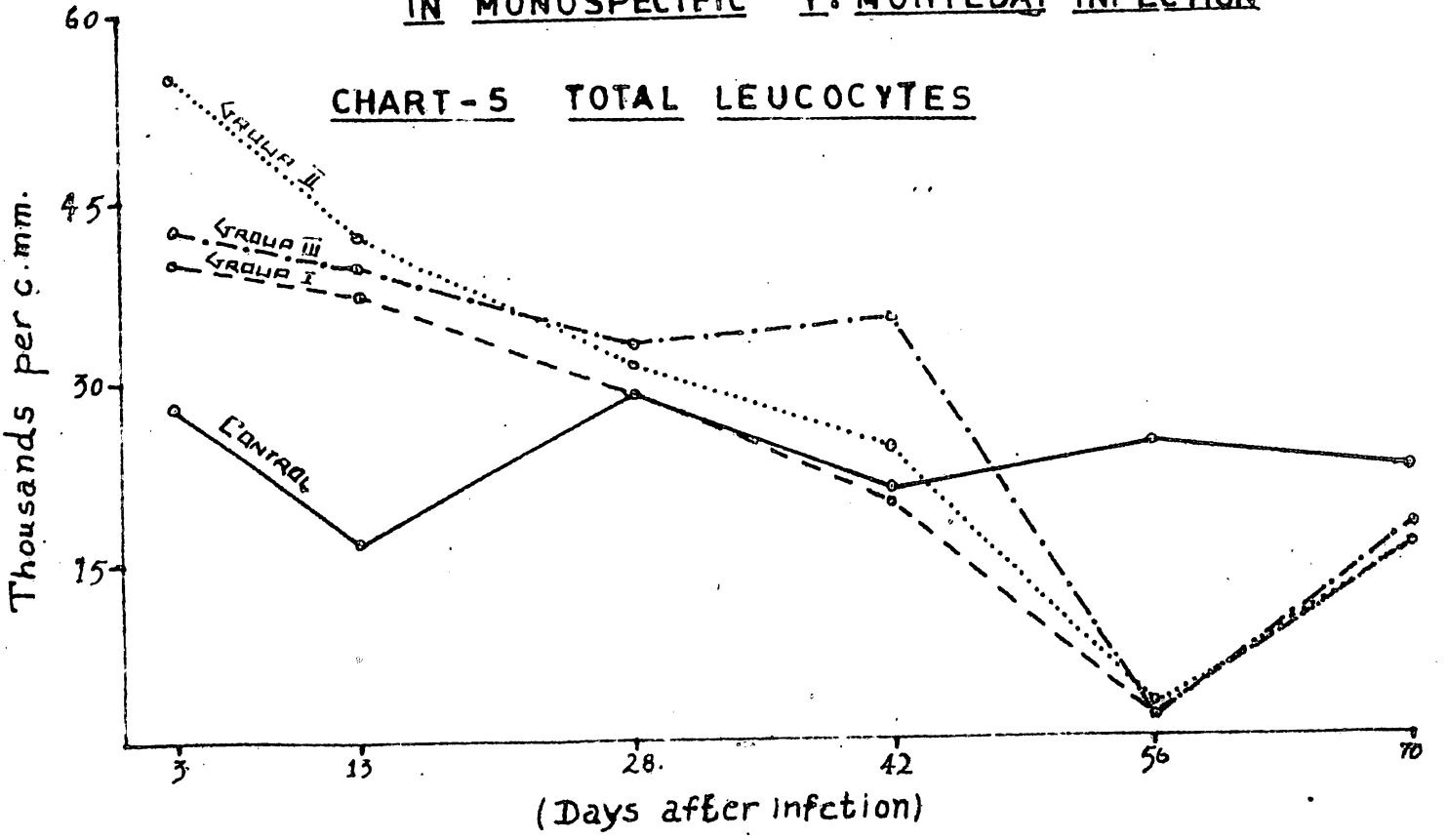
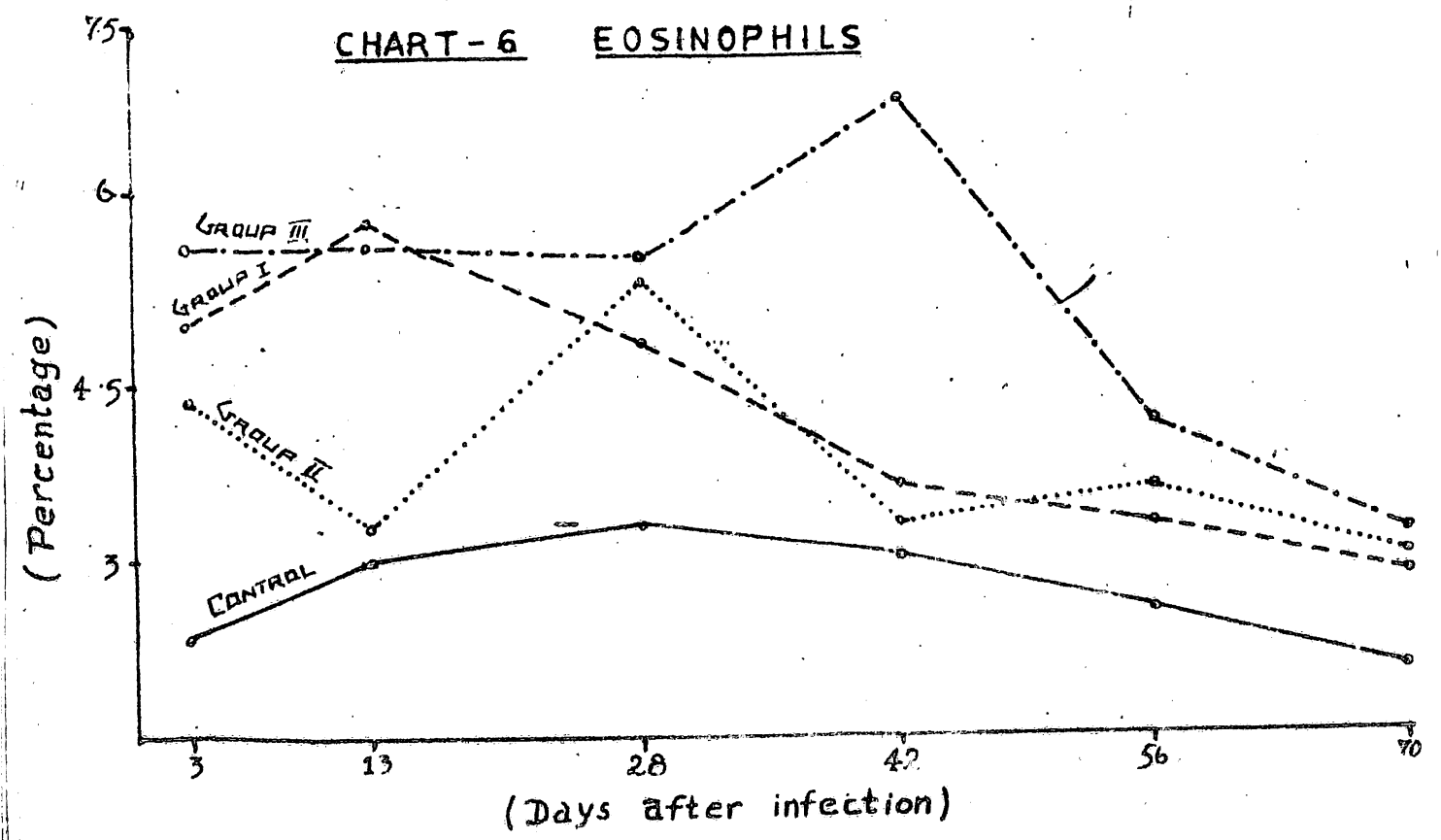


CHART-6 EOSINOPHILS



IN MONOSPECIFIC T. MOHTEDAI INFECTION

CHART-7 HETEROPHILS

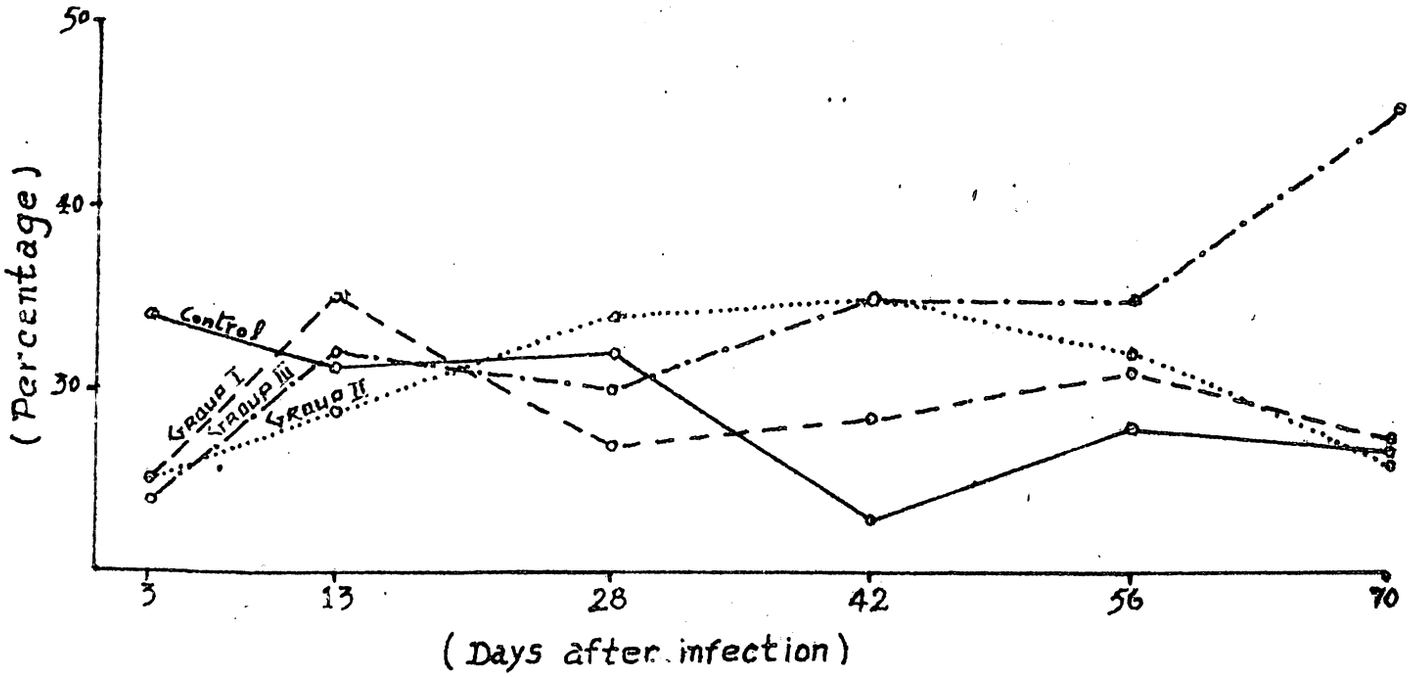
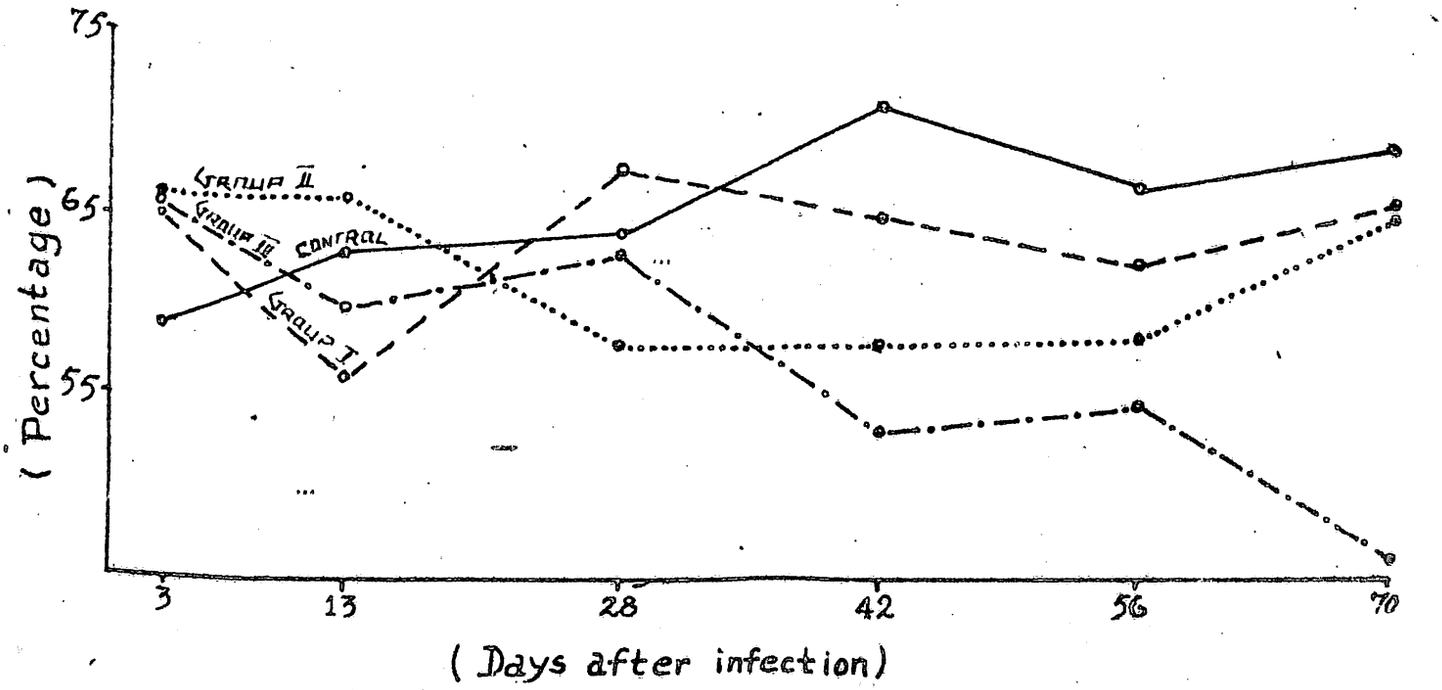


CHART-8 LYMPHOCYTES



CLINICAL SYMPTOMS

During the first two weeks of infection the chicks exhibited a marked loss of appetite and were dull and droopy. As the disease progressed the birds became weak and unthrifty due to poor feed intake. Anaemia was evident from the 6th week post infection and by the 10th week the birds had lost a considerable amount of their body weight. In layers the sexual maturity was delayed and there was decline in the rate of egg production.

WEIGHT GAIN

The mean overall weight of the experimental birds showed a significant decrease ($P < 0.01$) compared to the negative controls.

The decrease in respect of Group I (average number of worms established 68) was 19.50% ($P < 0.01$); in respect of Group II (average number of worms established 114.33) was 22.60% ($P < 0.01$) and in respect of Group III (average number of worms established 164) was 25.70% ($P < 0.01$).

On a further analysis, the decrease in respect of Group I was found to be 34.29%, 28.4% and 37.59% ($P<0.01$) on the 4th, 7th and 10th week post infection respectively; in respect of Group II was 29.73%, 30.48%, 37.04% ($P < 0.01$), 22.08% and 25.35% ($P < 0.05$) on the 5th, 6th, 7th, 8th and 9th week post infection respectively and in respect of Group III was 31.42%,

35.13%, 34.14%, 29.63%, 28.57% ($P < 0.01$) and 28.17% ($P < 0.05$) on the 4th, 5th, 6th, 7th, 8th and 9th week post infection respectively.

Thus the weight gains of the experimental groups were found to be poor during the 4th to 10th week post infection. On the 70th day post infection the average decrease in the live body weight was found to be 235 grams (19.32%) in Group I, 258 grams (21·21%) in Group II and 347 grams (28.45%) in Group III, compared to the negative controls which showed an average live body weight of 1216.66 grams.

Eventhough the strain of birds used were exclusively meant fore egg production the loss of income due to poor weight gain was calculated on the basis of the current price of live body weight (Rs.6.50/Kg.). The loss in Group I was Rs.1.50; in Group II Rs.1.70 and in Group III Rs.2.25 compared to the control birds.

The data are represented graphically in Chart 9.

SEXUAL MATURITY AND INTENSITY OF LAYING

The age at first egg was taken as the time of attainment of sexual maturity. The experimental groups showed delay in attainment of sexual maturity compared to the negative controls, which laid its first egg on the 132nd day.

The delay was 5 days in respect of Group I (number of worms established 77); 11 days in respect of Group II

(number of worms established 127) and 33 days in respect of Group III (number of worms established 184).

The rate of egg production was very poor with lack of persistancy and long pauses. During the observed period of 66 days, the total number of eggs produced by Group I was 39, by Group II 32 and by Group III only 8. Whereas the eggs produced by the negative control was 55.

The egg record of experimental and control birds are given in Table 40.

FEED EFFICIENCY

The quantity of feed in kilograms requires to produce a dozen of eggs was calculated for the female birds, since the strain of chicks used (Wychix) were exclusively meant for egg production. The feed efficiency of Group I (number of worms established 77) was 1.98; of Group II (number of worms established 127) 2.19 and of Group III (number of worms established 184) 5.25. The feed efficiency in the case of the control was found to be 1.5.

Thus the feed efficiency of the experimental birds were significantly lower when compared to the negative controls.

Calculating the loss of income on the basis of the current price of feed (Rs. 1.50/Kg.), Group I required 65 paise worth of feed, more than the controls to produce a dozen of eggs, while Group II and Group III needed Re. 1.00 and Rs. 5.60

worth of feed respectively, more than the controls.

PREPATENT PERIOD

The prepatent period of infection in the case of Group I (average number of worms established 68) was found to be in from 46 to 49 days (average 47.66 days), in the case of Group II (average number of worms established 114.33) was in from 47 to 49 days (average 48 days) and in Group III (average number of worms established 164) was in from 49 to 54 days (average 51.33 days).

Thus the prepatent period of infection was found to be not influenced by the number of worms established.

The data are presented in Table 13.

PERCENTAGE OF ESTABLISHMENT

All the infection experiments, during the current studies were conducted with infective juveniles (encysted third stage) aged 25 days, dissected out from grasshoppers as outlined under materials and methods. It was found in preliminary experiments that the number of larvae harboured by the intermediate host had apparently no influence on the subsequent establishment of the parasites, as the larvae from heavily infected grasshoppers established to the same extent as larvae from lightly infected grasshoppers. In these experiments it was also noted that more than one larva could

be found in a single cyst as mentioned by Sundaram (1971). However in exceptionally heavy infections as many as over 20 larvae could be seen in a single cyst (Plate II Fig. 1) and a total of over 4000 juveniles could be harvested from a single grasshopper. This constitute an additional observation on the development of the worm within the intermediate host.

The percentage of establishment was calculated from the number of worms found in each bird at autopsy and the number of infective larvae administered to them. For better correlation of results the percentage of establishment was considered rather than the number of juveniles administered, in all the experiments.

In Group I (infected with 200 juveniles each) the average number of worms recovered was 68, (22.55% males & 77.45% females) giving an establishment percentage of 34%.

In Group II (infected with 400 juveniles each) the average number of worms recovered was 114.33,(30.61% males & 69.39% females) giving an establishment percentage of 28.58%.

In Group III (infected with 600 juveniles each) the average number of worms recovered was 164,(34.76% males & 65.24% females) giving an establishment percentage of 27.33%.

No worms were present in Group IV as it constituted the negative controls.

Thus the percentage of establishment decreased when the

infective dose was raised. Since it was not the aim of the present investigation to find out the quantum of infective dose to give a maximum percentage of establishment, no attempt in this direction was made.

The data are presented in Table 13.

TABLE - 13 - Showing the details of prepatent period and percentage of establishment in monospecific *T. mohtedai* infection at three levels and negative controls.

	Chick number	Number of infective juveniles given	Prepatent period in days	Number of worms developed			Percentage of infn estc-blishment
				Males	Females	Total	
	1	200	46	14	63	77	38.50%
A	2	200	48	19	47	66	33.00%
	3	200	49	13	38	61	30.50%
	TOTAL	600				204	34.00%
	4	400	48	41	86	127	31.75%
B	5	400	49	29	73	102	25.50%
	6	400	47	35	79	114	28.50%
	TOTAL	1200				343	28.58%
	7	600	54	58	126	184	30.67%
C	8	600	51	54	112	166	27.67%
	9	600	49	59	83	142	23.67%
	TOTAL	1800				492	27.33%
	10						
D	11	NIL	All the birds remained negative				
	12						

A - GROUP I B - GROUP II
C - GROUP III D - GROUP IV

EFFECT ON WEIGHT GAIN IN MONOSPECIFIC I. MOHTEDAI INFECTION

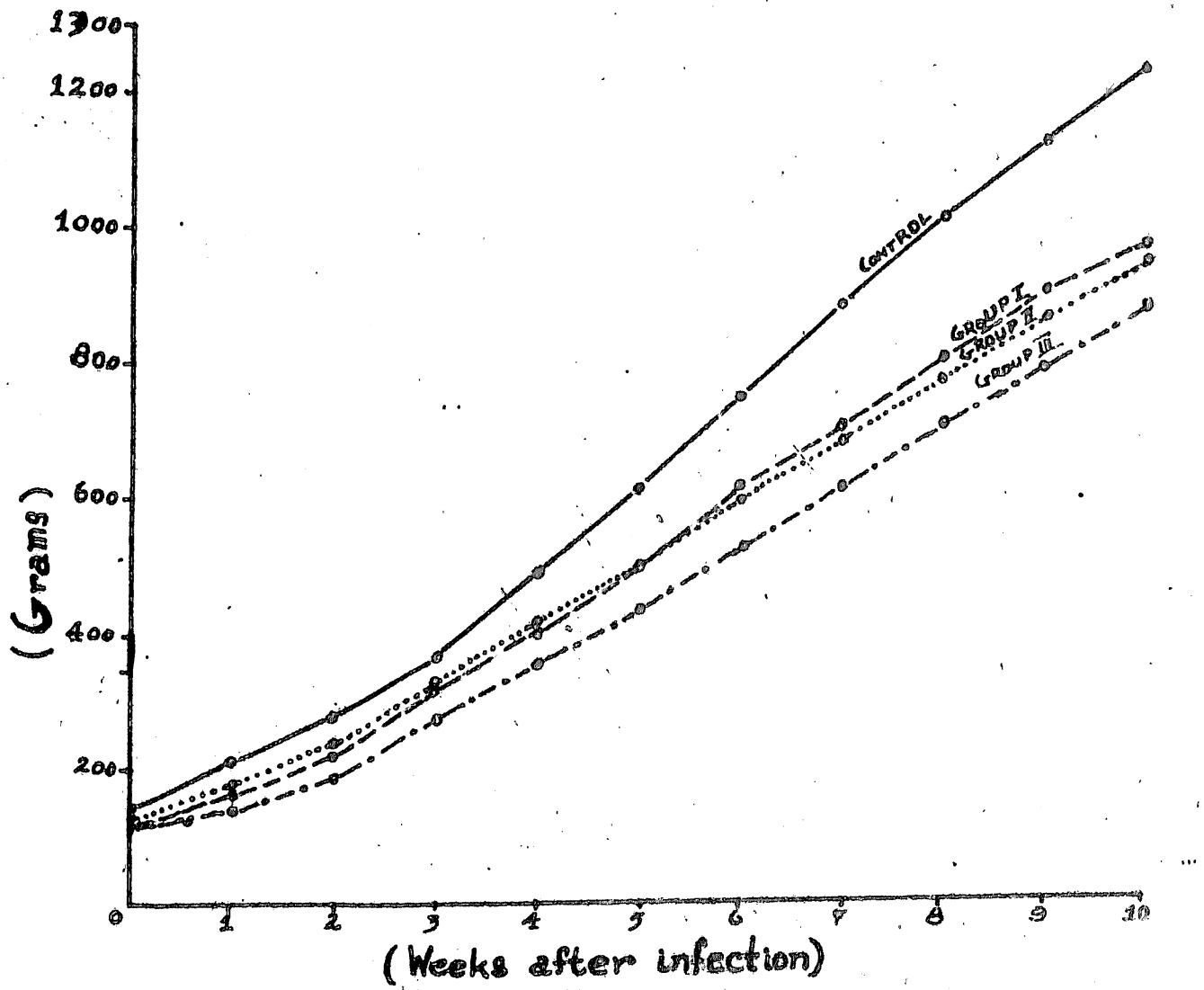


CHART - 9

HISTOPATHOLOGICAL OBSERVATIONS

To study the histopathological changes produced by the various developmental stages of *Tetrameres mohtedai* in the proventriculus of the host, chicks were infected with 500 juveniles and were slaughtered on the 4th (corrosponding to the third stage), 7th, 9th, 12th & 15th (corrosponding to the fourth stage), 32nd, 39th & 55th day (corrosponding to the adult stage) post infection. The changes obsarved were as follows : -

GROSS LESIONS

Unopened proventriculus of the chick slaughtered on the fourth day post infection appeared normal to the naked eye. When the organ was cut open the lumen was found to be filled with copious mucous secreation. Numerous petechial haemorrhages were seen on the mucous membrane especially around the glandular openings. Large number of juveniles were found embedded in the mucus in these areas.

On the twelth day post infection the proventriculus was one and a half times the normal size with a comparatively hard texture. When the organ was opened proventricular wall was found to be very much thickened and the lumen contained straw coloured exudate. The mucous membrane was intensely congested with focal areas of erosion of the epithelial lining.

On the twenty fifth day post infection pin head sized

black spots were observed on the serosal surface representing the glands infected by the female worms. When the organ was cut opened a mucopurulent exudate was seen blocking the glandular openings and the infected glands were well raised above the surface of the mucosa.

On the fifty fifth day post infection the serous surface of the proventriculus appeared mottled and the size of the organ was increased than the normal (Plate.. I Fig. 1). When cut opened the lumen of the organ was found to be very much reduced due to thickened wall. The glandular openings were very prominant in the mucosa. Cheesy semisolid exudate along with engorged female worms were observed within the compound glands.

MICROSCOPIC LESIONS

On the 4th day post infection

The proventriculus of the chick slaughtered on the fourth day post infection showed within the lamina propria cut sections of the juveniles surrounded by a severe granulocytic reaction (Plate II Fig. 2). The inflammatory reaction was seen to extend even upto the submucosa in some places. The lumen of the proventriculus was packed with desquamated epithelial cells and necrotic cell debris. Focal hyperplastic changes in the lining cells and goblet cells were evident.

On the 7th day post infection

On the 7th day post infection tubular glands showed focal

areas of degeneration and necrosis. The lamina propria was thickened due to infiltrating cells chiefly comprising of granulocytes. Submucosal oedema was present especially in the inter-glandular region. Compound glands showed hyperplasia of the lining epithelium and lymphoid aggregation in the glandular tissue (Plate II Fig 3). Cut sections of juveniles were present in the lumen of the compound glands along with abundant necrotic cell debris.

On the 9th and 12th day post infection

On the 9th day post infection complete destruction of the tubular glands was noticed near the opening of the infected glands with mild fibroplasia, histiocytic reaction and focal lymphoid hyperplasia in the lamina propria. Many of the compound glands also showed histiocytic reaction and their lumina were packed with inflammatory cellular exudate and juveniles. Similar changes were observed on the twelth day post infection, but were more pronounced (Plate II Fig. 4)

On the 15th day post infection

On the fifteenth day post infection pronounced histiocytic reaction predominated by granulocytes and plasma cells were found surrounding the juveniles within the compound glands (Plate III Fig. 1). Hypertrophy of the muscular coat of the organ, fibroplasia and engorgement of the blood vessels of the lamina propria were some of the other changes noticed.

On the 18th day post infection

On the 18th day post infection all most all the compound glands showed pronounced cellular reaction with marked inter - glandular oedema. The lumen of the glands contained juveniles along with necrotic cellular materials.

On the 25th day post infection

On the 25th day post infection the fibroplastic changes in the lamina propria became more pronounced and the cellular reaction was less, indicating a tendency towards chronicity. Fibroplastic changes were observed in some of the compound glands also along with degenerative changes. Some of the glands possessed immature female worms in their lumina.(Plate III Fig. 2)

On the 32nd and 39th day post infection

Histopatological changes observed on the 32nd and 39th day post infection were those of a chronic proventriculitis involving the compound glands. The affected glands showed pressure atrophy and flattening of the glandular acini to accomodate the worms. Their lumina were found to be lined by a stratified layer of epithelium strengthened by a layer of fibrous connective tissue below (Plate IV Fig. 1). Fibrous tissue invasion was also observed in the muscular coat at some places. Copious cellular material and erythrocytes were found within the intestine of the female worms indicating that they have been feeding on the host

tissue and blood (Plate III Fig. 3).

On the 55th day post infection

Changes observed in the lamina propria on the fiftyfifth day post infection were those of a reparative and regenerative process. Few of the compound glands showed complete loss of acini with mature female worms in their lumina (Plate IV Fig. 2).

PLATE I - Proventriculus - monospecific T.mohtedai infection - Note the light grey spots (indicated by arrow) on the serosa of the organ showing the affected compound glands by the female T.mohtedai worms.

PLATE I

PLATE II

Fig. 1. A single cyst carrying the third stage juveniles of T.mohtedai within - collected from a heavily infected grasshopper (25th day). This cyst contained 27 juveniles.

Fig. 2. Proventriculus - T.mohtedai infection 4th day - Note the granulocytic reaction surrounding the juveniles.

Fig. 3. Proventriculus - T.mohtedai infection 7th day - Note the lymphoid aggregation in the glandular tissue (indicated by arrow), hyperplastic lining epithelium and necrosis of the mucous membrane.

Fig. 4. Proventriculus - T.mohtedai infection 12th day- Note the marked inter-glandular oedema, fibroplasia and hyperplastic lining cells of the compound glands.

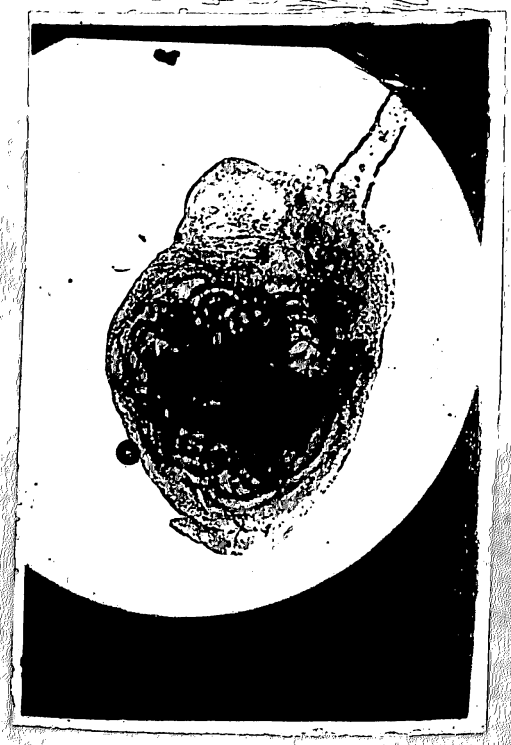


Fig. 1

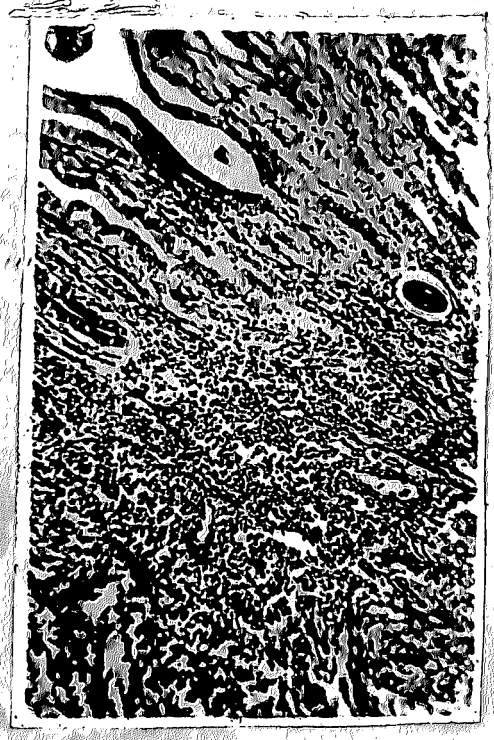


Fig. 2

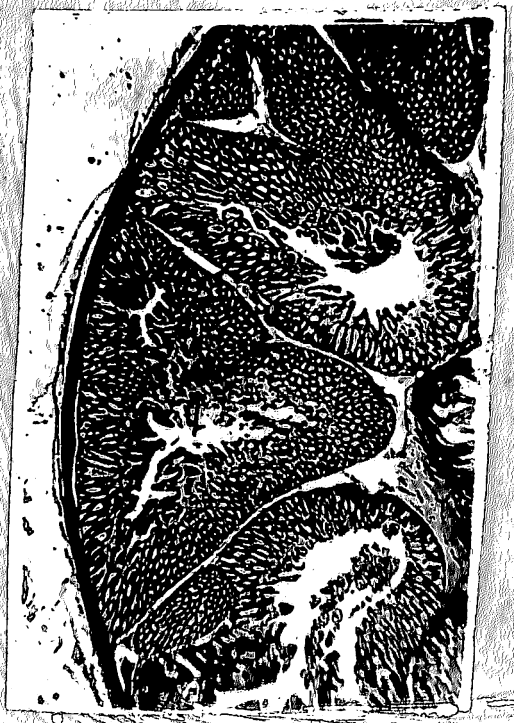


Fig. 3

Fig. 4

PLATE III

Fig. 1. Proventriculus - T.mohtedai infection 15th day- Pronoumced histiocytic and granulocytic infiltration surrounding the juvenile.

Fig. 2. Proventriculus - T.mohtedai infection 25th day- Note that the fibroplastic changes have extended even into some of the compound glands. An immature female worm (indicated by arrow) is seen within the lumen of a compound gland.

Fig. 3. Proventriculus - T.mohtedai infection 39th day- 1.Portion of the glandular tissue of the organ.
2. Portion of female T.mohtedai worm Arrow points to the erythrocytes within the gut of the worm.

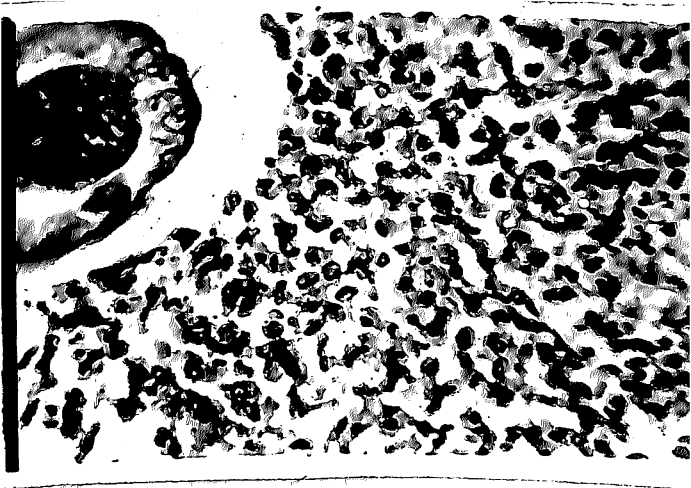


Fig. 1

Fig. 2

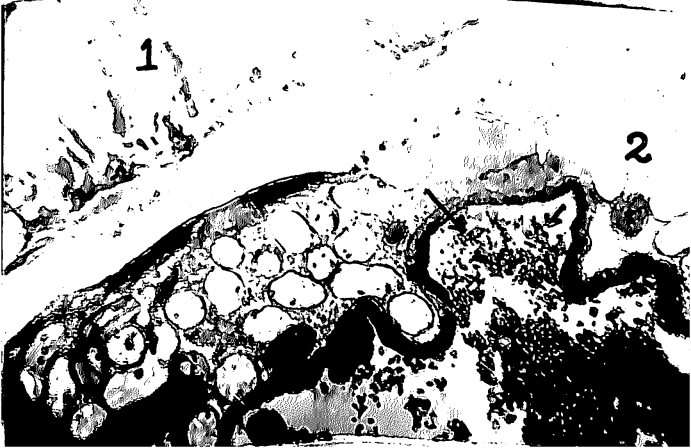


PLATE III

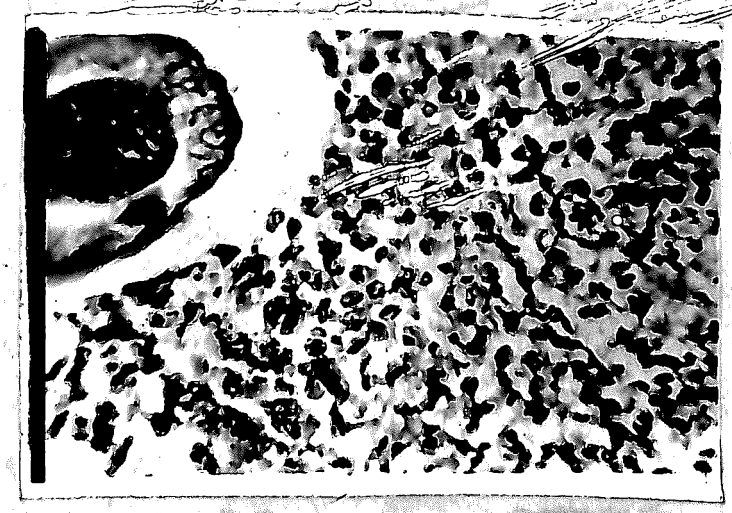


Fig. 1

Fig. 2

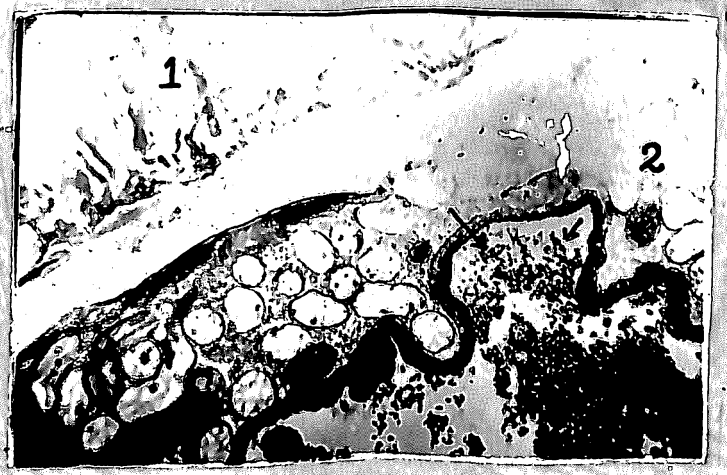


Fig. 3

PLATE IV

Fig. 1. Proventriculus - T.mohtedai infection 32nd day - A female worm has been cut transversely. Note the damaged and atrophied glandular tissue. The epithelial layer lining the gland is highly flattened.

Fig. 2. Proventriculus - T.mohtedai infection 55th day - Note the marked pressure atrophy, cystic dialatation and focal areas of necrosis of the compound glands.

PLATE IV

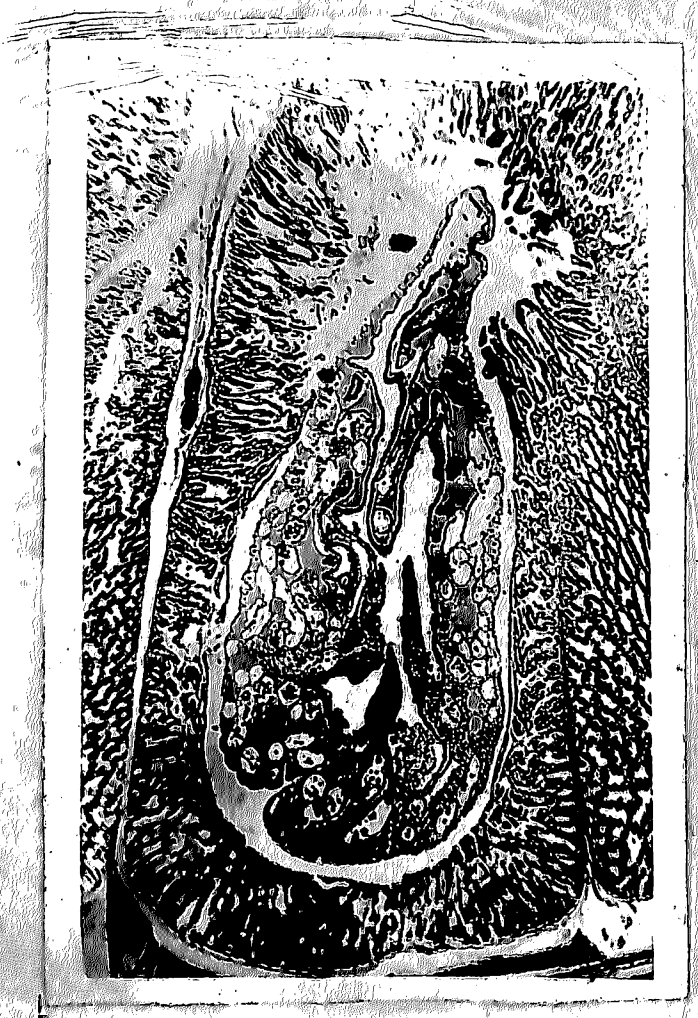


Fig. 1

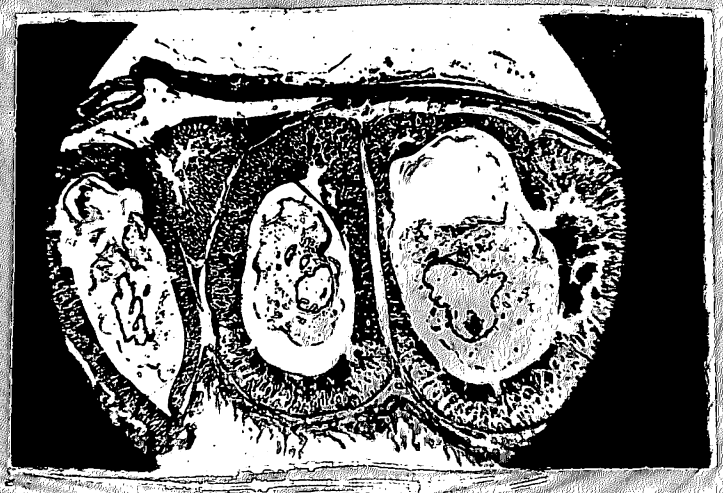


Fig. 2

DISCUSSION

DISCUSSION

The pathogenic studies conducted during the present investigation appears to be the first, under controlled conditions for monospecific Tetrameres mohtedai infection.

Among the haematological observations made, progressive anaemia was observed as an important clinical manifestation of T.mohtedai infection. Anaemia was reported as a clinical symptom in natural T.fissispina infection by Sugimoto and Nishiyama (1937) and in natural T.mohtedai infections by Anantaraman & Chandrasekharan Nair (1955). Chandrasekharan (1977) observed a microcytic hypochromic anaemia in T.anatis infection in ducks. In the present investigation the type of anaemia observed was normocytic normochromic initially, indicating an acute blood loss probably due to the blood sucking habits of the female worms; which is supported by the fact that erythrocytes were found within the gut of the female worms (Plate III Fig.3). Later as the decease progressed a macrocytic anaemia slightly becoming hypochromic was evident towards the 70th day post infection indicating the development of an iron deficiency anaemia.

Villela & Ribeiro (1955) making observations on the haemoglobins from the perientric fluid of female T. confusa found that it was similar to that of the host (fowl), but differed in

its much greater ability to hold oxygen. Later Railey (1973) working on T.fissispina found that most of the blood ingested by the female worms were digested to haematin. Based on the assumption that females only are active blood suckers, it could be calculated from the data obtained in this work that a single female worm causes an average decrease of 0.039 gram percentage of haemoglobin and 0.021 millions of erythrocytes per cu.mm. of blood during a period of 30 days after the worms have matured (40th to 70th day post infection).

Olson & Levine (1939) has recorded eosinophilia and heterophilia in chickens experimentally infected with Capillaria columbae. Similar changes were observed by Wickware (1949) in a controlled infection of chickens with Heterakis gallinae, but the increase obtained was not related to the parasite present. In the present study eosinophilia was evident during the early phase of infection especially during the invasive period. However in heavy infections (as in Group III) eosinophilia was continued to be noted upto 42nd day post infection.

Initially there was a severe leucocytosis but as the disease progressed leucopenia was observed. The initial leuco - cytosis may be due to the host's response to the invading juveniles. The leucopenia observed during the later stage, that is, when the worms have matured, indicate an immuno - supression which is also supported by the fact that cellular reaction was minimum in histopathological studies of the organ during this

period. Compared to 56th day after infection, leucopenia was less marked on the 70th day post infection, indicating that there is a trend towards the reversal of pathogenic effect and that a host - parasite balance is well underway.

The observations made by Anantaraman & Chandrasekharan Nair (1955) on natural T.mohtadai infection and by Cram (1928); Sugimoto & Nishiyama (1937); Srivastava (62 9); Soulsby (1965) and Chandrasekharan (1977) on other species of Tetrameres infection, that the infected birds show lack of appetite, droopiness, marked loss of weight and emaciation were seen in the present study also during the course of infection.

The maximum worm load assessed by Anantaraman & Chandra - sekharan Nair (1955) in respect of natural T.mohtedai infection was 19 females, in a bird and by Sundaram (1971) was 137 males and 124 females. During the present studies the maximum worm load obtained was 27 males and 173 females (number of juveniles administered being 600). The prepatent period of infection was found to be similar to those observed by Sundaram (1971). The percentage of establishment showed an inverse proportion to the number of juveniles administered .

Anantaraman & Chandrasekharan Nair (1955) observed chronic catarrhal changes with occassional desquamation of superficial epithelium and lymphocytic infiltration in the tunica propria of proventriculi in chicks naturally infected with T.mohtedai.

Other changes observed by them were pressure atrophy and total disappearence of the tissue in some of the compound glands. Similar changes were observed in the present study on the 55th day post infection. Cvetajeva (1960) working on T.fissispina observed dialatation of the glandular lumen with desquamated cells on the 5th day post infection and degenerative and attrophic changes from 10th day post infection. During the present studies on the 4th day post infection severe granulocytic reaction was observed in the lamina propria surrounding the worms with necrosis and desquamation of epithelial cells lining the organ. By the 7th day post infection when the worms had migrated into the proventricular glands changes in the form of acute inflammation and periglandular oedema were evident in the compound glands. In 9 to 12 days post infection the initial granulocytic reaction in the lamina propria was replaced by lymphoid hyperplasia suggesting the immune reaction towards the invading juveniles. By the 15th day post infection plasma cells were seen among the infiltrating cells within the compound glands. Cvetajeva (1960) observed pressure atrophy and cystic dialatation of the affected compound glands on the 18th day in T.fissispina infection, Whereas in the present study T.mohtedai worms were found to be only in the young adult stage and were not large enough to cause pressure atrophy. However pressure atrophy and cystic dialatation were noticed from the 25 th day post infection (Plate III Fig. 2). The cellular

reaction were minimum with initiation of fibroplastic changes on the 25th day post infection, showing a tendency towards chronicity. By 39th day post infection the reaction were more or less confined to the affected glands indicating that a host - parasite balance has been reached to a certain degree. By 55th day post infection the host - parasite relationship was more or less balanced as indicated by the reparative and regenerative process going on in the organ. This is further proved by the fact that even in very heavy artificial infections (average number of adult worms established 164) where almost all the glands were parasitised, mortality of the experimental chicks did not result. Therefore the present study agrees with the observations made by Cram (1931) and Soulsby (1965) on T. americana infection that the greater damage is done when the young worms invade the wall of the proventriculus.

Joshi & Kamalapur (1971) while describing the histopathological changes caused by T. mohtedai in naturally infected proventriculi, have mentioned, proliferation of connective tissue cells in the lobular septum around the worm, chronic catarrhal changes with necrosis of the superficial epithelium, metaplasia of epithelial cells into stratified squamous variety and pressure atrophy of deep glands in some of the sections examined by them. In the present study fibroplasia and atrophic changes were seen during 25th to 55th day post infection, however, the metaplastic changes mentioned by them were not observed.

The proliferative adenamatous reaction of the glandular crypts described by Alves et al. (1954); Zajicek (1959); Tsvetaeva (1960); Soulsby (1965) and Graubmann & Grafner (1967) was not observed in the present study in T.mohtedai infection, however, such change was seen in Aquaria spiralis infection.

To understand to what extent the pathogenic effects of T.mohtedai causes economic loss to the Poultry industry, the egg production and feed efficiency of the infected female birds were observed. It had been calculated that the heavily infected bird required Rs. 5.60 worth of feed more than the control to produce a dozen of eggs. The live body weight was also found to be considerably low, thereby causing a further loss (the calculated loss being Rs. 2.25 per bird). These two factors unmistakably point out that T.mohtedai is economically important.

PATHOGENICITY
OF
Acuaria spiralis

PATHOGENICITY OF ACUARIA SPIRALIS

HAEMATOLOGICAL OBSERVATIONS

To study the haematological alterations in three levels of monospecific Acuaria spiralis infection, the blood samples were collected on the 6th (representing the 3rd stage), 13th (representing the 4th stage), 24th, 38th, 52nd and 66th day (representing the adult stage) post infection. The results obtained were as follows :

TOTAL ERYTHROCYTES

The mean overall erythrocyte count of the entire experimental groups was significantly lower ($P < 0.01$) than the negative controls.

The decrease in respect of Group I (average number of worms established 70) was 11.75% ($P < 0.01$), in respect of Group II (average number of worms established 144.33) 22.27% ($P < 0.01$) and in respect of Group III (average number of worms established 246.33) 22.39% ($P < 0.01$).

On a further analysis Group I showed a decrease of 16.45% ($P < 0.05$) on the 66th day post infection, Group II showed a decrease of 39.76% ($P < 0.01$) & 31.73% ($P < 0.05$) on the 24th & 66th day post infection respectively and Group III showed a decrease of 29.97%, 32.71% & 37.21% ($P < 0.01$) on the 38th, 52nd & 66th day post infection respectively.

Thus the total erythrocyte of the infected groups dropped significantly on the 38th day and was maximum by the 66th day post infection.

The data are represented in Table 14 and are graphically presented in Chart 10.

HAEMOGLOBIN

The mean haemoglobin percentage of the entire experimental groups was significantly lower ($P < 0.05$) than the negative controls.

The decrease in respect of Group I (average number of worms established 70) was 0.91% (not significant); in respect of Group II (average number of worms established 144.33) 5.18% (significant at 5% level) and in respect of Group III (average number of worms established 246.33) 8.94% (significant at 1% level) over the controls.

On a further analysis Group I showed a decrease of 10.17% ($P < 0.05$) on the 66th day post infection; Group II showed a decrease of 13.36% ($P < 0.05$) on the 66th day post infection and Group III showed a decrease of 12.53% ($P < 0.05$) and 16.48% ($P < 0.01$) on the 52nd and 66th day post infection respectively.

Thus the percentage of haemoglobin was found to be decreased from 52nd day post infection in Group III and on the 66th day post infection in groups I & II.

The data are presented in Table 15 and are graphically represented in Chart 11.

PACKED CELL VOLUME

The mean overall packed cell volume of the experimental groups showed no significant difference from that of the negative controls.

The data are presented in Table 16.

MEAN CORPUSCULAR VOLUME

The overall mean corpuscular volume of the entire experimental groups was significantly higher ($P < 0.01$) than the negative controls.

The increase in respect of Group I (average number of worms established 70) was 19.48% ($P < 0.01$); in respect of Group II (average number of worms established 144.33) 25.83% ($P < 0.01$) and in respect of Group III (average number of worms established 246.33) 33.25% ($P < 0.01$) over the controls.

On a further analysis, Group I showed an increase of 35.05%, 36.57% & 12.67% ($P < 0.05$) on the 38th, 52nd & 66th day post infection respectively; Group II showed an increase of 39.56% & 35.50% ($P < 0.05$) on the 6th & 66th day post infection respectively and Group III showed an increase of 45.17% ($P < 0.01$), 38.67% ($P < 0.05$) & 56.59% ($P < 0.01$) on the 38th, 52nd & 66th day post infection respectively.

Thus the mean corpuscular volume was nearly normal upto 24th day post infection but thereafter an increase was noticed in all the experimental groups.

The data are presented in Table 17 and are graphically represented in Chart 13.

MEAN CORPUSCULAR HAEMOGLOBIN

The overall mean corpuscular haemoglobin of the entire experimental groups was significantly higher ($P < 0.05$) than the negative controls.

The increase in respect of Group I (average number of worms established 70) was 15.71% ($P < 0.05$); in respect of Group II (average number of worms established 144.33) 19.91% ($P < 0.01$) and in respect of Group III (average number of worms established 246.33) 22.58% ($P < 0.01$) over the controls.

On a further analysis, Group I showed an increase of 34.69% and 8.50% ($P < 0.05$) on the 38th and 66th day post infection respectively, Group II showed an increase of 37.13% and 27.96% ($P < 0.05$) on the 6th and 66th day post infection respectively and Group III showed an increase of 39.62%, 29.53% & 26.61% ($P < 0.05$) on the 38th, 52nd & 66th day post infection respectively.

The mean corpuscular haemoglobin showed an increase which was maximum on the 38th day post infection in Group I and Group III. Thereafter there was a fall in the value but had not reached normal upto 66th day post infection.

The data are presented in Table 18.

MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION

The overall mean corpuscular haemoglobin concentration of the entire experimental groups was significantly lower ($P < 0.05$) than the negative controls.

The decrease in respect of Group I (average number of worms established 70) was 3.09% (not significant) in respect of Group II (average number of worms established 144.33) 4.49% (not significant) and in respect of Group III (average number of worms established 246.33) 7.83% (significant at 1% level) over the controls.

On a further analysis, Group II showed a decrease of 5.65% ($P < 0.05$) and Group III showed a decrease of 18.43% ($P < 0.05$) on the 66th day post infection.

Thus birds harbouring more than 114 worms showed a decrease in the mean corpuscular haemoglobin concentration on the 66th day post infection.

The data are presented in Table 19 and are graphically represented in Chart.

TOTAL LEUCOCYTES

The mean total leucocytes of all the experimental groups showed a significant increase ($P < 0.01$) on the 13th day post infection compared to the negative controls. But thereafter a decrease was noticed.

The increase in respect of Group I (average number of worms

established 70) was 77.33% ($P < 0.01$) in respect of Group II (average number of worms established 144.33) 67.25% ($P < 0.01$) and in respect of Group III (average number of worms established 246.33) was 83.21% ($P < 0.01$) over the negative controls. Later groups I & II showed a decrease of 51.31% and 62.28% ($P < 0.05$) respectively on the 66th day post infection. Group III showed a decrease of 59.37%, 36.20%, 52.22% & 55.34% ($P < 0.05$) on the 24th, 38th, 52nd & 66th day post infection respectively.

Thus leucocytosis was observed on the 13th day post infection in all the experimental groups. But from the 24th day post infection leucopenia was evident in Group III and on the 66th day post infection in groups I & II.

The data are presented in Table 20 and are graphically represented in Chart 14.

EOSINOPHILS

The mean overall eosinophil percentage of the entire experimental groups was significantly higher ($P < 0.01$) than the negative controls.

The increase in respect of Group I (average number of worms established 70) was 100.00% ($P < 0.05$); in respect of Group II (average number of worms established 144.33) 158.50% ($P < 0.01$) and in respect of Group III (average number of worms established 246.33) 219.31% ($P < 0.01$) over the controls.

On a further analysis, on the 13th day post infection

Group I showed an increase of 204.76% ($P<0.01$) and Group II showed an increase of 228.57% ($P<0.01$). GroupIII showed an increase of 414.29% ($P<0.01$) and 342.91% ($P<0.05$) on the 13th and 38th day post infection respectively.

Thus eosinophilia was observed on the 13th day post infection in all the experimental groups.

The data are presented in Table 21 and are graphically represented in Chart 15.

HETEROPHILS

The mean overall heterophil percentage of the entire experimental groups was significantly higher ($P<0.05$) than the negative controls.

The increase in respect of Group I (average number of worms established 70) was 11.95% (not significant), in respect of Group II (average number of worms established 144.33) 19.38% (significant at 5% level) and in respect of Group III (average number of worms established 246.33) 28.92% (significant at 1% level) over the controls.

On a further analysis, all the groups showed an initial decrease on the 13th day post infection. Later Group II showed an increase of 84.62% ($P<0.01$) and 64.71% ($P<0.05$) on the 24th and 66th day post infection respectively. Group III showed an increase of 110.25% ($P<0.01$) and 28.48% ($P<0.05$) on the 24th and 38th day post infection respectively.

Thus heterophilia was evident in Group II & Group III from the 24th day post infection, but in Group I it was not significant.

The data are presented in Table 22 and are graphically represented in Chart 16.

BASOPHILS

The mean overall basophil percentage of the experimental groups showed no significant difference from that of the negative controls.

The data are presented in Table 23.

LYMPHOCYTES

The mean overall lymphocyte percentage of the entire experimental groups was significantly lower ($P < 0.01$) than the negative controls.

The decrease in respect of Group I (average number of worms established 70) was 11.36% ($P < 0.01$); in respect of Group II (average number of worms established 144.33) 18.32% ($P < 0.01$) and in respect of Group III (average number of worms established 246.33) 25.95% ($P < 0.01$).

On a further analysis, Group I showed a decrease of 36.39% ($P < 0.01$) on the 24th day post infection, Group II showed a decrease of 56.33% ($P < 0.01$) & 24.37% ($P < 0.05$) on the 24th and 66th day post infection respectively and Group III

showed a decrease of 25.34% ($P < 0.05$), 71.16% ($P < 0.01$) and 21.85% ($P < 0.05$) on the 13th, 24th & 38th day post infection respectively.

Thus when the worm burden was between 70 and 144 lymphocytopenia was evident from the 24th day post infection. But when the worm burden was 246 it was seen from the 13th day post infection itself.

The data are presented in Table 24 and are graphically represented in Chart 17.

MONOCYTES

The mean overall monocyte percentage of the entire experimental groups was significantly higher ($P < 0.05$) than the negative controls.

The increase in respect of Group I (average number of worms established 70) was 21.03% (not significant), in respect of Group II (average number of worms established 144.33) 42.02% (significant at 5% level) and in respect of Group III (average number of worms established 246.33) 44.04% (significant at 5% level) over the controls.

On a further analysis, on the 66th day post infection Group II showed an increase of 123.61% ($P < 0.05$) and Group III showed an increase of 139.21% ($P < 0.05$).

Thus monocytosis was observed when the worm burden was above 70 on the 66th day post infection.

The data are presented in Table 25.

TABLE-14 - Showing the total Erythrocyte count in three levels of monospecific *A. spiralis* infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	2.40	2.70	2.10	2.20	2.10	1.07	1.44	3.60	2.01	2.50	3.10	2.20
13th	1.86	2.68	2.95	2.30	2.75	2.82	2.35	2.17	2.22	2.51	2.79	2.67
24th	1.61	3.48	3.31	2.40	2.54	2.32	2.32	2.46	2.44	2.79	2.87	2.31
38th	2.25	2.10	2.08	2.05	2.34	2.09	2.26	2.00	1.40.	2.94	2.23	2.91
52nd	1.94	1.92	2.19	2.03	1.93	2.25	1.72	2.04	1.67	2.88	2.28	2.91
66th	2.01	1.87	2.52	1.65	1.90	1.68	1.61	1.61	1.59	2.57	2.53	2.56

All values in millions/cu.mm. of blood.

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	6.192	2.064	9.8**
Between Days	5	2.363	0.473	2.246*
Error	63	13.268	0.195	
TOTAL.	71	21.823		

** Significant at 1% level.
* Significant at 5% level.

TABLE-15 - Showing the Haemoglobin values in three levels of monospecific A.spiralis infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	6.88	7.2	7.8	7.4	7.0	5.4	7.0	5.2	8.0	8.4	7.4	6.2
13th	7.2	7.4	9.0	7.6	8.6	8.0	8.2	7.4	7.0	7.6	7.6	8.2
24th	8.5	7.7	8.9	8.9	7.8	6.8	7.6	8.6	7.4	8.0	8.4	8.0
38th	8.0	8.6	8.0	8.0	6.0	8.8	7.4	7.0	7.2	7.8	7.6	7.2
52nd	7.8	7.8	8.4	8.8	7.8	7.4	7.4	7.0	7.8	8.4	8.6	8.4
66th	6.4	8.0	8.4	8.2	6.6	7.2	7.0	7.0	8.0	8.4	8.6	8.4

All values in gram percentage.

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	12.195	4.065	10.531**
Between Days	5	8.712	1.742	4.513**
Error	63	24.288	0.386	
TOTAL	71	45.195		

** Significant at 1% level.

TABLE-16 - Showing the Packed cell volume at three levels of monospecific A.spiralis infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	29.0	28.0	28.0	25.0	25.0	25.0	23.0	24.0	24.0	28.0	28.0	29.0
13th	30.0	28.0	30.5	28.5	36.0	32.0	29.0	30.0	29.0	26.0	26.0	26.0
24th	27.0	28.0	28.0	30.0	29.0	25.0	30.5	34.0	32.0	28.0	30.5	29.0
38th	34.0	35.0	35.0	34.0	30.0	31.0	33.0	31.0	31.0	33.0	33.0	29.0
52nd	29.0	29.5	31.0	31.0	27.0	27.00	24.0	27.0	30.0	28.0	27.0	32.0
66th	24.0	29.0	32.0	26.0	29.0	28.0	35.0	27.5	27.0	31.0	29.0	31.0

All values in percentage

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	44.923	14.974	2.622 n.s
Btween Days	5	233.750	46.750	8.187**
Error	63	359.702	5.710	
TOTAL	71	638.375		

n.s. Not significant.

** Significant at 1% level.

TABLE-17 - Showing the Mean corpuscular volume in three levels of monospecific A.spiralis infection compared with the negative controls.

DAYS	GROUP I			GROUP II				GROUP III			GROUP IV	
	1.	2	3	4	5	6	7	8	9	10	11	12
6th	120.83	103.70	133.33	113.64	119.05	233.65	159.72	633.89	119.40	112.00	90.32	131.82
13th	161.29	104.48	103.74	123.91	130.91	113.48	123.40	138.25	130.63	103.59	98.19	97.38
24th	167.70	80.46	84.59	125.00	114.17	107.76	131.47	138.21	131.15	100.36	106.27	125.54
38th	151.11	166.67	168.27	165.85	128.21	148.33	146.02	155.00	221.43	112.25	147.98	99.66
52nd	149.49	153.65	141.55	152.71	139.90	120.00	139.54	132.35	179.64	97.22	118.48	109.97
66th	119.40	155.08	126.98	157.58	152.63	172.62	217.39	170.81	169.81	120.62	114.62	121.09

All values in cubic microns.

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	18205.60	6068.533	10.123**
Between Days	5	13393.50	2678.700	4.468**
Error	63	37767.00	599.476	
TOTAL	71	69366.10		

** Significant at 1% level.

TABLE-18 - Showing the Mean corpuscular haemoglobin in three levels of monospecific A.spiralis infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	28.33	26.67	37.14	33.64	33.33	50.47	48.61	14.44	39.80	33.60	23.87	28.18
13th	38.71	27.61	30.61	33.04	31.27	28.37	34.89	34.10	31.53	30.28	27.24	30.71
24th	52.80	22.13	26.89	37.08	30.71	29.21	32.76	34.96	30.33	28.67	28.27	34.63
38th	35.56	40.95	38.46	41.38	25.64	42.11	32.74	35.00	51.43	26.53	34.08	24.74
52nd	40.21	40.63	38.36	41.38	40.42	32.89	43.02	34.31	46.71	29.17	37.72	28.87
66th	31.84	42.78	33.33	49.70	34.74	42.86	43.48	43.48	38.99	32.69	33.99	32.89

All values in micro micro grams

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	900.577	300.192	8.094**
Between Days	5	495.180	97.036	2.67*
Error	63	2336.429	37.086	
TOTAL	71	3732.186		

** Significant at 1% level.
* Significant at 5% level.

TABLE - 19 - Showing the Mean corpuscular haemoglobin concentration in three levels of monospecific A.spirclis infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	23.45	25.71	27.86	29.60	28.00	21.60	30.44	22.61	33.33	30.00	26.43	21.38
13th	24.00	26.43	29.51	26.50	23.89	25.00	28.28	24.67	24.14	29.23	29.23	31.54
24th	31.48	27.50	31.79	29.67	26.90	27.20	24.92	25.29	23.13	28.57	29.23	31.54
38th	23.53	24.57	22.86	23.53	20.00	28.39	22.42	22.58	23.23	23.64	23.03	24.83
52nd	26.90	26.44	27.10	27.10	28.89	27.41	30.83	25.93	26.00	30.00	31.85	26.25
66th	26.67	27.59	26.25	31.54	22.76	24.83	20.00	25.45	22.96	27.10	29.66	27.10

All values in percentage

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	98.074	32.691	5.096 **
Between Days	5	147.916	29.583	4.612 **
Error	63	404.151	6.415	
TOTAL	71	650.141		

** Significant at 1% level.

TABLE - 20 - Showing the total Leucocytes in three levels of monospecific A. spiralis infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	12.00	16.00	8.80	12.00	30.00	23.10	12.00	10.00	11.00	12.00	8.67	8.00
13th	38.00	61.40	38.88	64.00	25.54	40.88	63.55	51.10	28.22	48.66	50.88	38.44
24th	45.32	42.00	22.00	34.00	15.54	14.00	11.54	13.54	9.32	46.44	34.00	44.22
38th	22.00	15.10	20.00	28.00	18.00	22.80	13.50	26.60	18.40	51.00	28.00	42.70
52nd	25.10	17.80	27.80	20.90	43.50	28.00	14.00	50.00	11.30	50.40	51.30	53.10
66th	23.20	14.22	9.32	10.00	18.00	8.22	13.32	18.67	10.89	26.00	23.11	26.89

All values in thousands/cu.mm. of blood.

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	2942.712	980.904	9.642 **
Between Days	5	8058.720	1611.744	15.843 **
Error	63	6409.314	101. 735	
TOTAL	71	17410.746		

** Significant at 1% level.

TABLE - 21 - Showing the percentage of Eosinophils in three levels of monospecific A. spiralis infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	7.0	9.0	3.0	9.0	7.0	7.0	12.0	9.0	7.0	6.0	4.0	3.0
13th	22.0	19.0	23.0	16.0	26.0	27.0	39.0	34.0	35.0	8.0	6.0	7.0
24th	4.0	3.0	5.0	4.0	2.0	6.0	4.5	5.0	7.0	1.5	1.0	2.0
38th	4.5	2.0	2.0	8.0	4.0	10.0	12.0	6.0	13.0	4.0	2.0	1.0
52nd	4.0	4.0	2.5	13.0	6.5	9.5	2.5	5.5	3.5	3.0	4.0	2.0
66th	5.0	4.5	1.5	2.0	1.5	3.0	1.5	3.0	1.0	1.5	1.5	5.0

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	606.121	202.040	10.565 **
Between Days	5	3048.351	609.670	31.881 **
Error	63	1204.775	19.123	
TOTAL	71	4859.247		

** Significant at 1% level.

TABLE - 22 - Showing the percentage of Heterophils in three levels of A. spiralis infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	30.0	30.0	25.0	26.0	20.0	29.0	27.0	40.0	32.0	26.0	35.0	30.0
13th	20.0	21.0	25.0	12.0	19.0	18.0	14.0	15.5	13.0	29.0	30.0	27.0
24th	51.0	41.0	52.0	57.0	59.0	64.0	63.0	72.0	70.0	49.5	32.0	25.0
38th	22.0	20.0	24.0	24.0	31.0	28.0	37.0	34.0	30.5	15.0	16.0	24.0
52nd	23.5	28.0	40.0	30.0	29.5	22.0	27.5	40.5	31.5	26.0	21.0	21.0
66th	42.0	23.0	40.0	52.0	28.0	46.0	30.5	36.0	38.5	36.5	17.5	28.5

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	1183.625	394.542	6.161 **
Between Days	5	7838.791	1567.758	24.481 **
Error	63	4034.459	64.039	
TOTAL	71	13056.875		

** Significant at 1% level.

TABLE - 23 - Showing the percentage of Eosophils in three levels of A. spiralis infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	0.0	0.0	1.0	1.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
13th	0.5	0.0	1.0	0.0	0.0	0.5	0.0	0.5	0.0	0.0	1.0	0.0
24th	1.0	3.0	4.0	2.0	1.0	2.0	4.0	4.0	3.0	2.0	1.0	0.0
38th	0.0	0.0	0.0	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0
52nd	0.0	0.0	0.5	0.0	0.0	0.5	0.5	0.0	0.5	0.5	0.5	0.5
66th	0.5	0.5	0.0	0.0	0.5	0.5	0.0	0.5	1.0	0.0	0.5	1.5

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	4.083	1.361	3.770 *
Between Days	5	41.167	8.233	22.806 **
Error	63	22.750	0.361	
TOTAL	71	68.000		

* Significant at 5% level

** Significant at 1% level.

TABLE - 24 - Showing the percentage of Lymphocytes in three levels of monospecific A.spiralis infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	58.0	55.0	67.0	60.0	69.0	63.0	59.0	46.0	58.0	63.0	57.0	64.0
13th	55.0	58.0	49.0	66.0	54.0	50.0	46.0	43.0	49.0	61.0	60.5	64.0
24th	38.0	48.0	32.0	29.0	32.0	20.0	22.5	16.0	15.0	43.5	63.0	70.0
38th	72.5	75.0	73.0	66.0	59.0	57.0	49.0	57.0	55.0	80.0	76.0	74.0
52nd	71.0	66.5	56.0	56.0	62.5	66.5	69.0	52.0	63.0	69.0	74.0	74.0
66th	48.0	68.5	54.5	41.5	64.5	46.0	63.5	55.5	54.0	59.0	76.5	59.5

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	3480.22	1160.073	19.348 **
Between Days	5	7327.85	1465.570	24.443 **
Error	63	3777.42	59.959	
TOTAL	71	14585.49		

** Significant at 1% level.

TABLE-25 - Showing the percentage of Monocytes in three levels of monospecific A.spiralis infection compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	5.0	6.0	4.0	4.0	4.0	1.0	13.0	5.0	3.0	5.0	4.0	3.0
13th	2.5	2.0	2.0	5.5	1.0	4.0	1.0	7.0	2.5	2.0	2.5	2.0
24th	6.0	5.0	7.0	8.0	6.0	8.0	6.0	4.0	5.0	3.5	3.0	4.0
38th	1.0	3.0	1.0	2.0	4.0	5.0	2.0	3.0	1.0	1.0	6.0	1.0
52nd	1.5	1.5	1.0	1.0	1.5	0.5	0.5	2.0	1.5	1.5	2.5	2.5
66th	4.5	3.5	4.0	4.5	5.5	4.5	5.0	5.0	5.5	3.0	2.0	1.5

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	29.760	9.920	3.145*
Between Days	5	131.615	26.323	8.996**
Error	63	184.344	2.926	
TOTAL	71	345.719		

* Significant at 5% level.
** Significant at 1% level.

IN MONOSPECIFIC A. SPIRALIS INFECTION

CHART-10 TOTAL ERYTHROCYTES

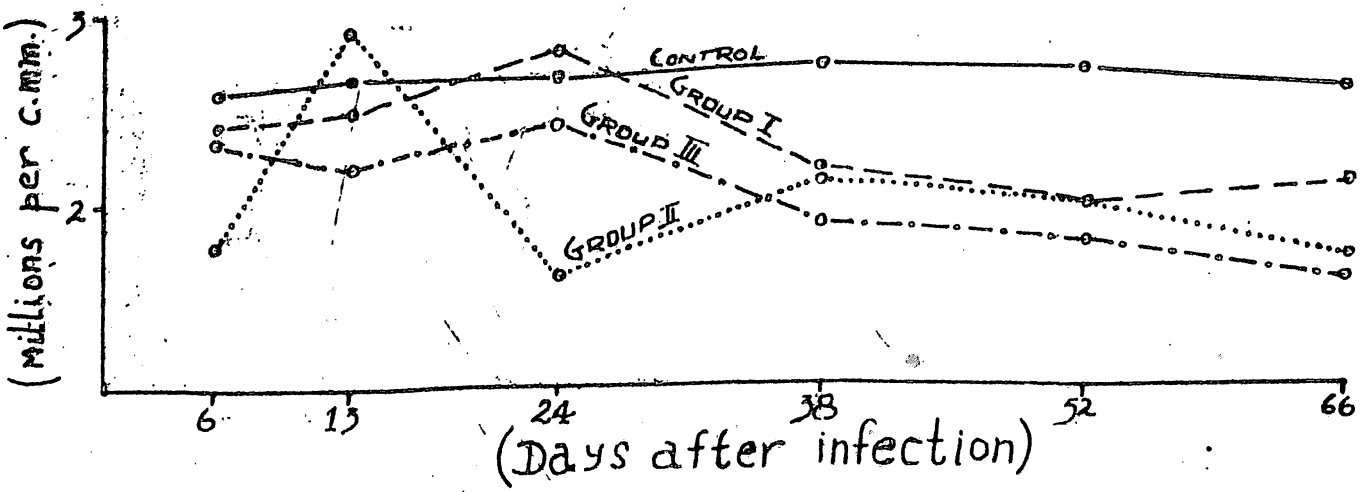
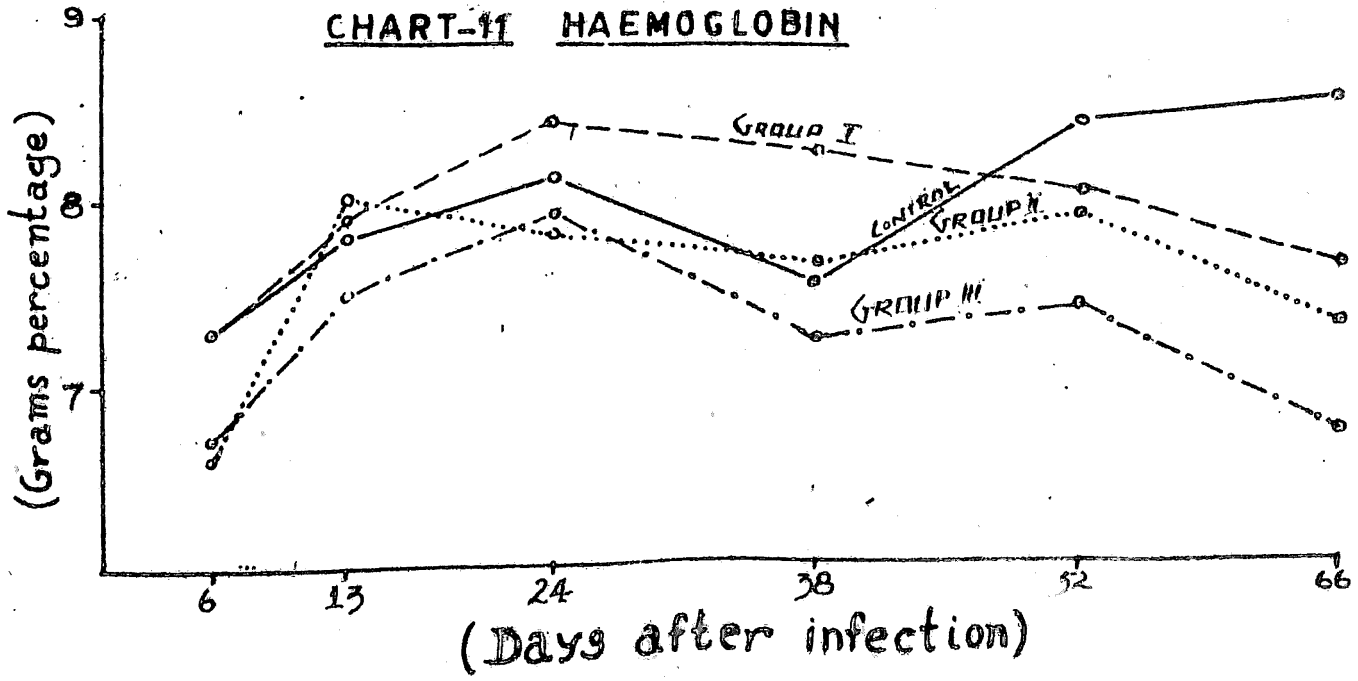


CHART-11 HAEMOGLOBIN



IN MONOSPECIFIC A. SPIRALIS INFECTION

CHART 12 MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION

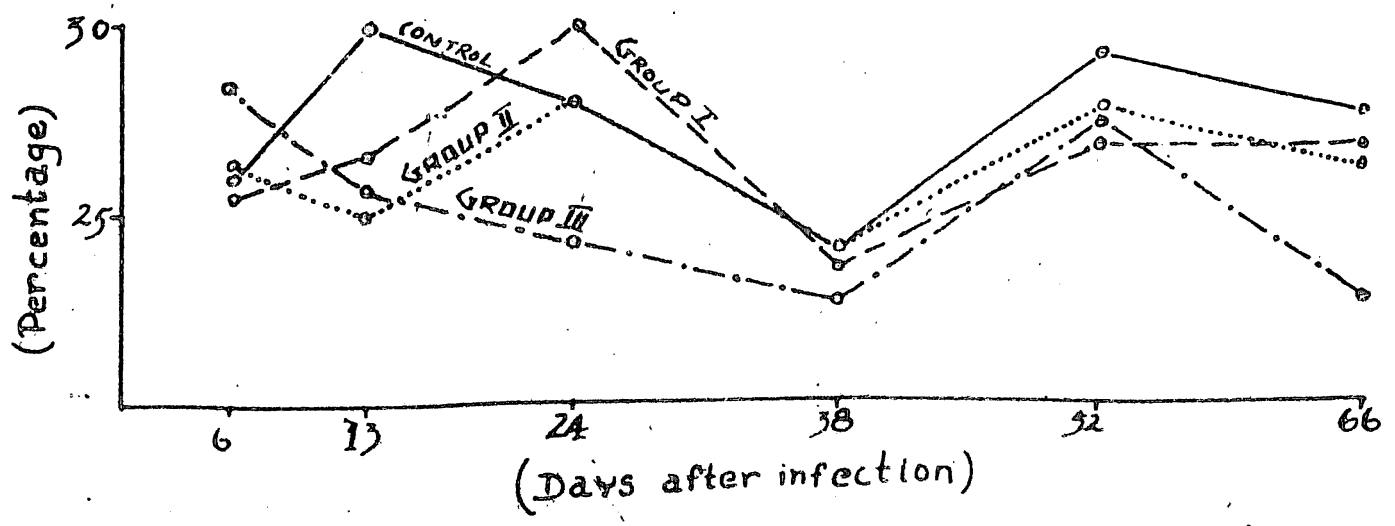
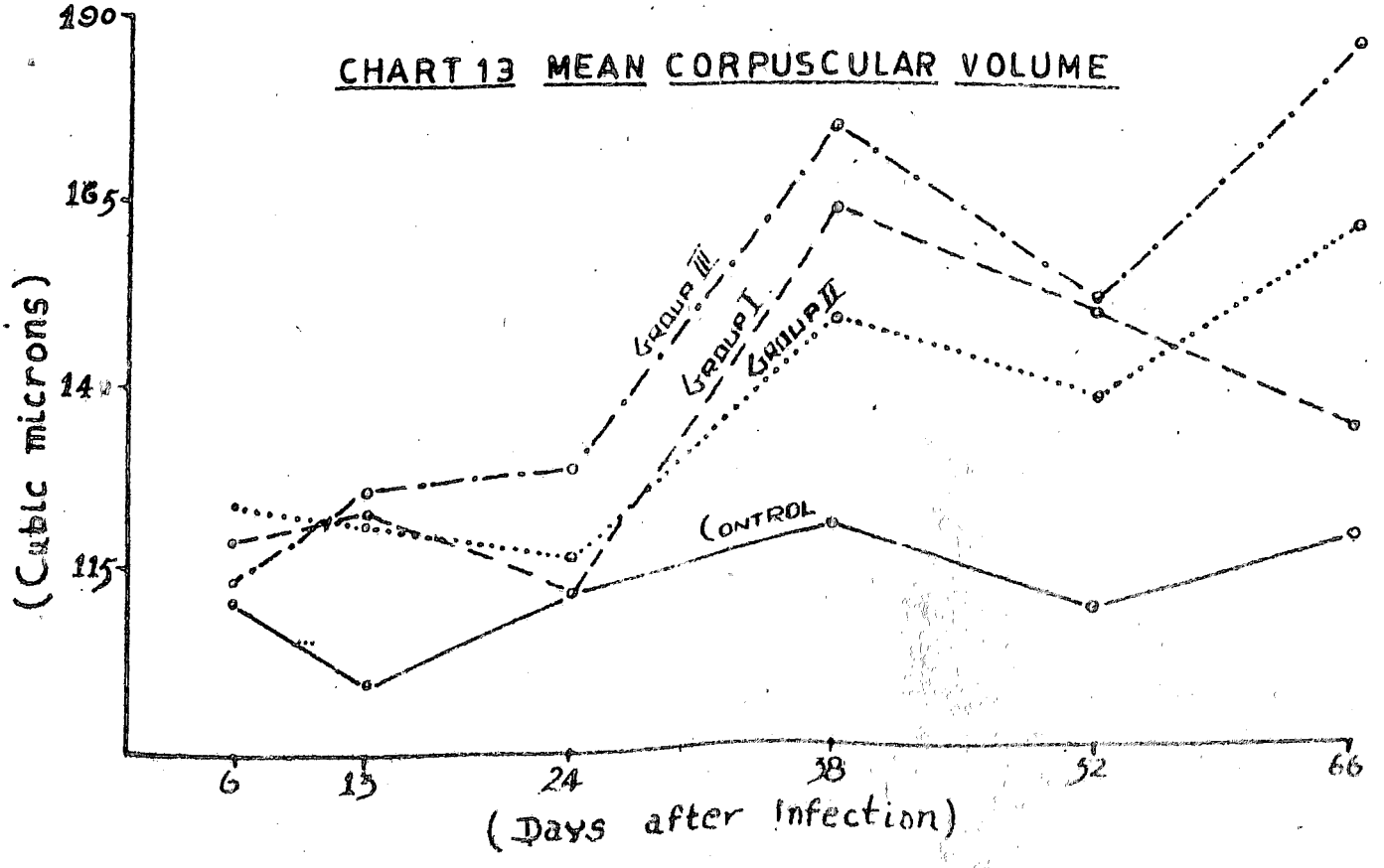


CHART 13 MEAN CORPUSCULAR VOLUME



IN MONOSPECIFIC A. SPIRALIS INFECTION

CHART-14 TOTAL LEUCOCYTES

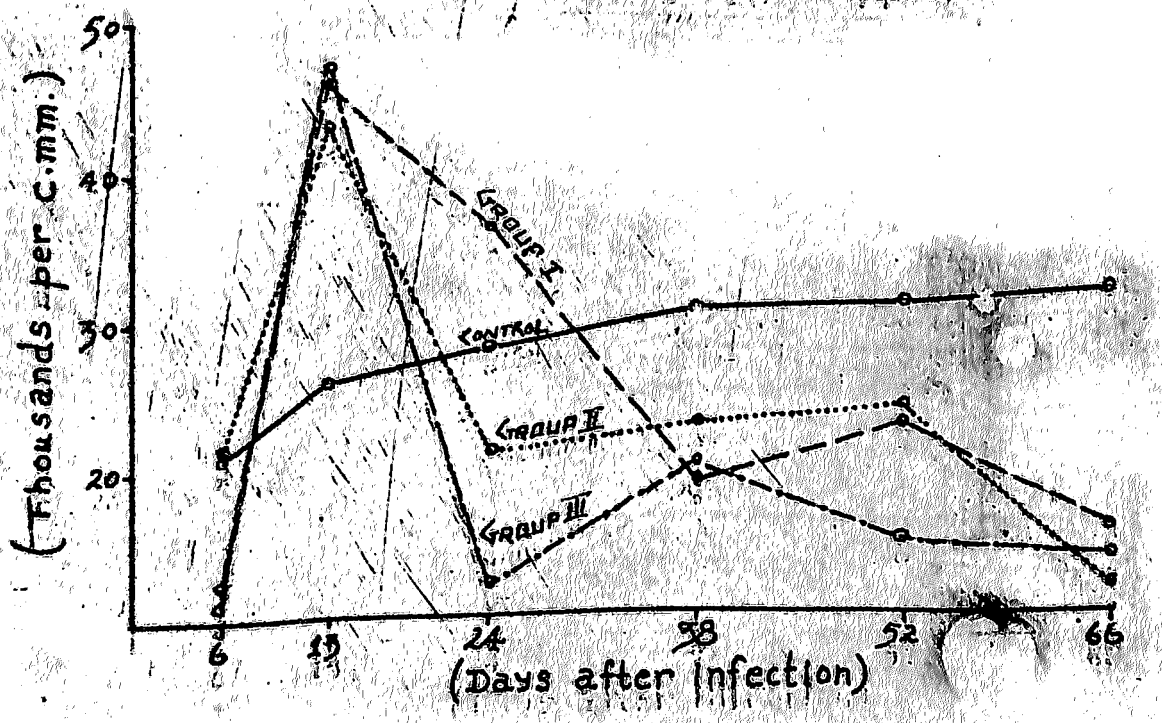
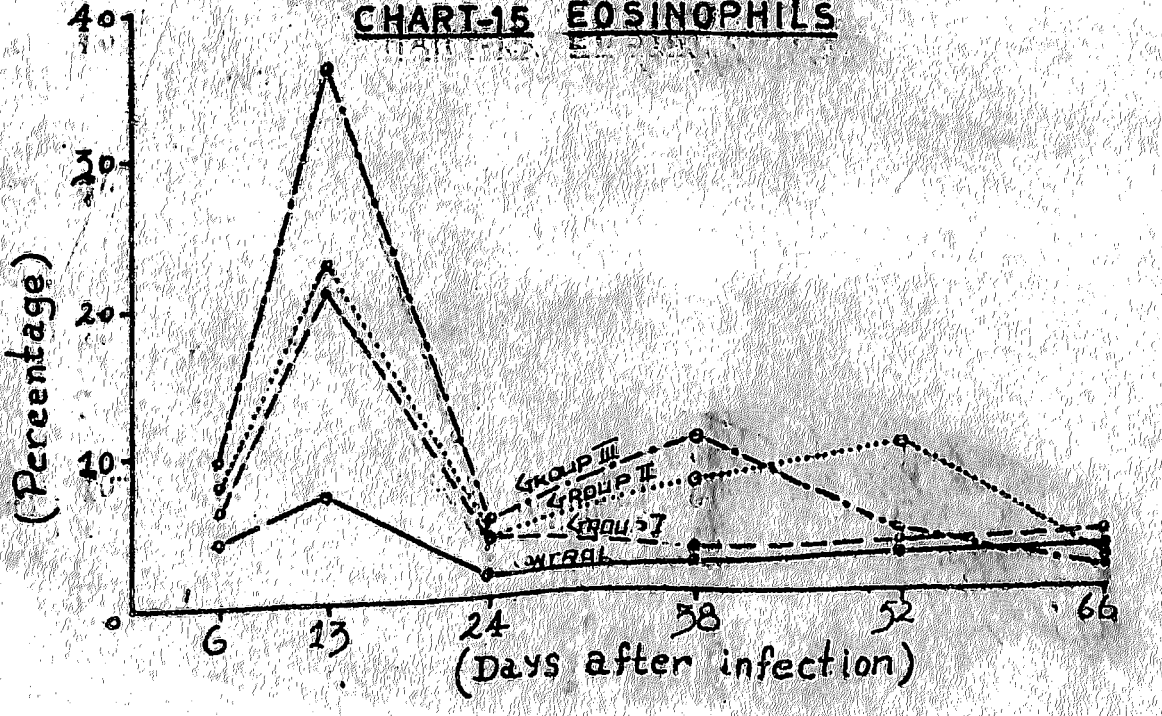


CHART-15 EOSINOPHILS



IN MONOSPECIFIC A. SPIRALIS INFECTION

CHART-16 HETEROPHILS

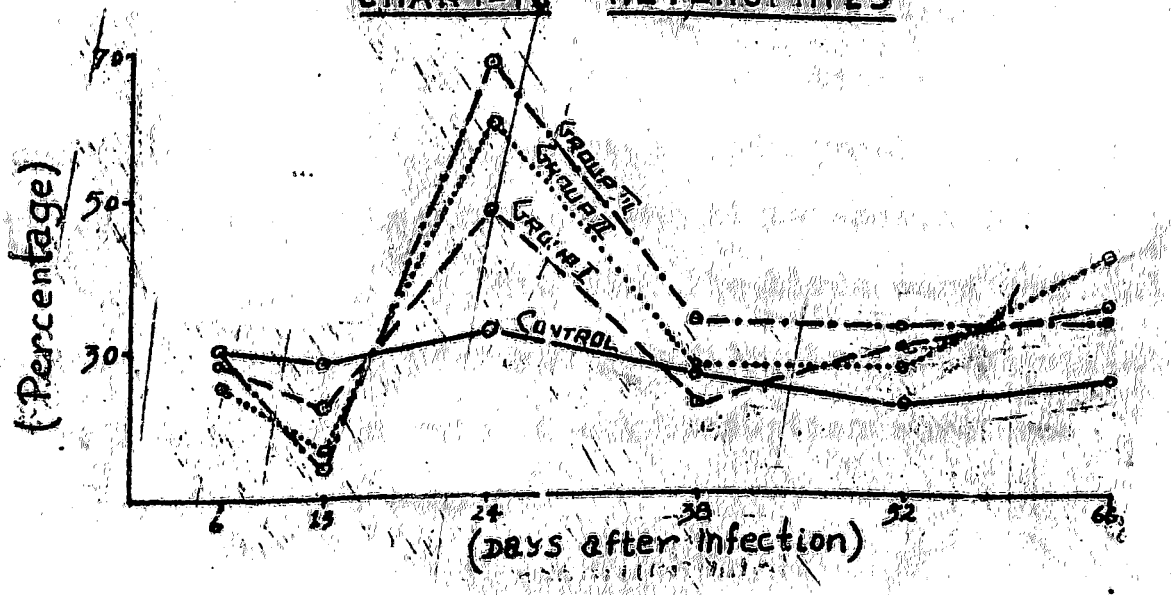
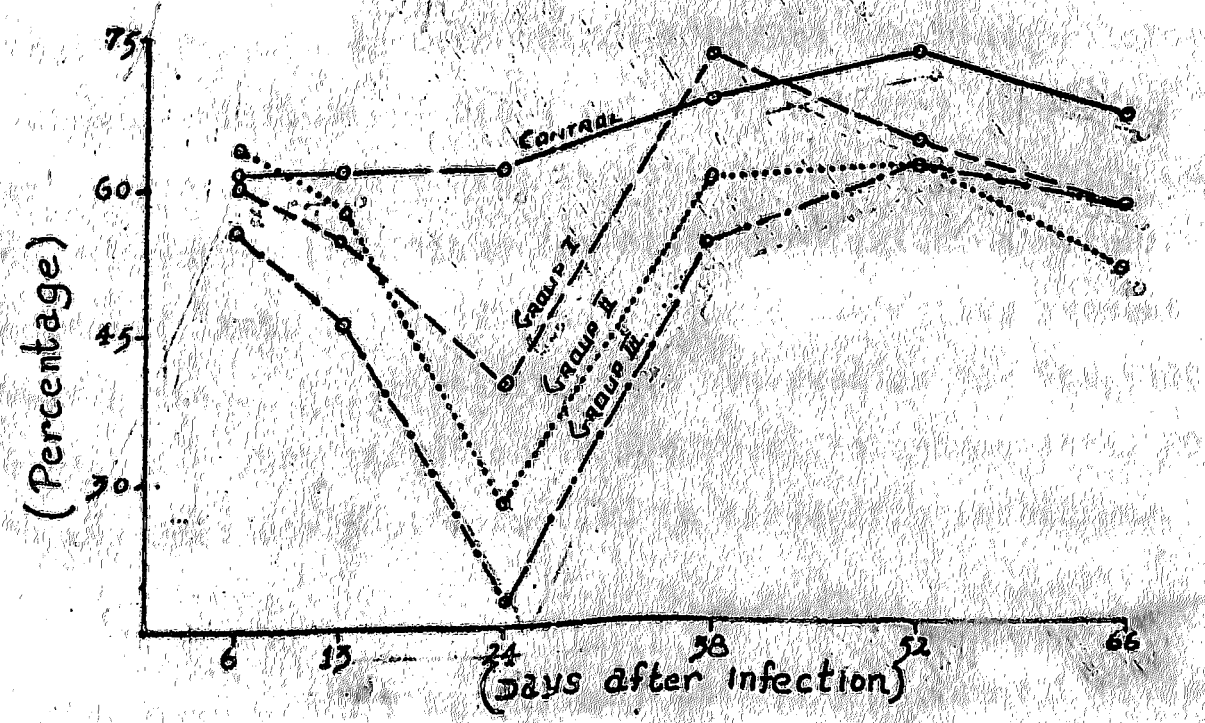


CHART-17 LYMPHOCYTES



CLINICAL SYMPTOMS

The infected birds were very dull and droopy, often seen huddled towards the corner of the cages. During the first two weeks post infection,,a rise in the body temperature was noticed and the birds showed lack of appetite and unthriftiness. The droppings became diarrhoeic from the 4th week post infection onwards and the birds showed considerable loss in body weight. By 10th week the birds were weak and emaciated. Mortality, delayed maturity, lack of persistancy in laying and reduced rate of egg production wefe also noticed as described below :

MORTALITY

In monospecific A. spiralis infection experiments 3 birds out of 12 (25%) died when they received 500 juveniles as the infective dose (data from chicks infected for hidtopathological studies). Similarly 4 birds out of 12 (33.33%) also died when they received the same infective dose of A. spiralis (data from dispecific infections for histopathological studies). Thus a mortality rate of 29.16% was obtained during the present investigation. The mortality were observed on the 8th, 10th, and 20th day post infection in monospecific and on 15th, 20th, 24th and 29th day post infection in dispecific infections.

WEIGHT GAIN

The mean overall weight gain of the experimental birds

showed a significant decrease ($P<0.01$) compared to the negative controls.

The decrease in respect of Group I (average number of worms established 70) was 26.13% ($P<0.01$) in respect of Group II (average number of worms established 144.33) 34.16% ($P<0.01$) and in respect of Group III (average number of worms established 246.33) 37.24% ($P<0.01$).

On a further analysis, the decrease in all the experimental groups was found to be significant ($P<0.01$) during the 7th, 9th & 10th week post infection. During these periods, Group I showed a decrease of 39.54%, 78.05% & 46.99% respectively, Group II showed a decrease of 44.19%, 68.29% & 73.49% respectively and Group III showed a decrease of 60.46%, 58.54% & 74.70% respectively.

Thus the weight gains of the experimental groups were found to be poor during the 7th to 10th week post infection. On the 70th day post infection the average decrease in the live body weight was found to be 303 grams (24.04%) in Group I, 385 grams (30.52%) in Group II and 425 grams (33.69%) in Group III compared to the negative controls which showed an average live body weight of 1261.66 grams.

The data are presented graphically in Chart 18.

SEXUAL MATURITY AND INTENSITY OF LAYING

The age at first egg was taken as the time of attainment of sexual maturity. It was found that in experimental groups

the age at first egg was delayed compared to the negative control which laid its first egg on the 129th day.

The delay was 7 days in respect of Group I, 23 days in respect of Group II and 46 days in respect of Group III.

The rate of production greatly varied in the experimental groups with lack of persistancy and /or exceptionally long pauses. During an observed period of 66 days the total number of eggs laid by Group I (number of worms established 71) was 43, by Group II (number of worms established 154) 10 and by Group III (number of worms established 251) 3 only, whereas the eggs produced by Group IV (negative control) was 59.

The egg record of experimental and control groups is presented in Table 40.

FEED EFFICIENCY

The quantity of feed in kilograms required to produce a dozen of eggs was calculated for the female birds, since the strain of chicks used in the experiments ("Wychix") were exclusively meant for egg production. The feed efficiency of the female bird of Group I (number of worms established 71) was 1.84, of Group II (number of worms established 154) 5.52 & of Group III (number of worms established 251) 8.799. Whereas in respect of Group IV (negative control) the feed efficiency of the female bird was 1.399.

The feed efficiency of the experimental groups was thus significantly lower than the negative control. Calculating

the loss of income on the basis of the current price of the feed (Rs. 1.50/Kg.), Group I required 65 paise worth offeed more than the controls to produce a dozen eggs. While groups II & III needed Rs. 6.15 and Rs. 11.10 worth of feed respectively more than the negative control.

PREPATENT PERIOD

The prepatent period of infection in the case of Group I (average number of worms established 70) was found to be in from 24 to 26 days (average 25 days), in the case of Group II (average number of worms established 144.33) in from 26 to 29 days (average 26.66 days) and in the case of Group III (average number of worms established 246.33) in from 26 to 29 days (average 27 days). Hence the number of worms established had little influence on the prepatent period of infection and that even in heavy infections some of the female worms developed normally.

The data are presented in Table 26.

PERCENTAGE OF ESTABLISHMENT

The percentage of establishment was calculated from the number of worms found in each bird at autopsy and the number of infective juveniles administered to them.

In Group I (infected with 100 juveniles each) the average number of worms recovered was 70 (36.19% males and 63.81% females) giving an establishment percentage of 70.

In Group II (infected with 200 juveniles each) the

average number of worms recovered was 144.33 (35.10% males and 64.90% females) giving an establishment percentage of 72.16.

In Group III (infected with with 400 juveniles each) the average number of worms established was 246.33 (38.57% males and 61.43% females) giving an establishment percentage of 61.58.

In Group IV no worms were present as it constituted the negative control group.

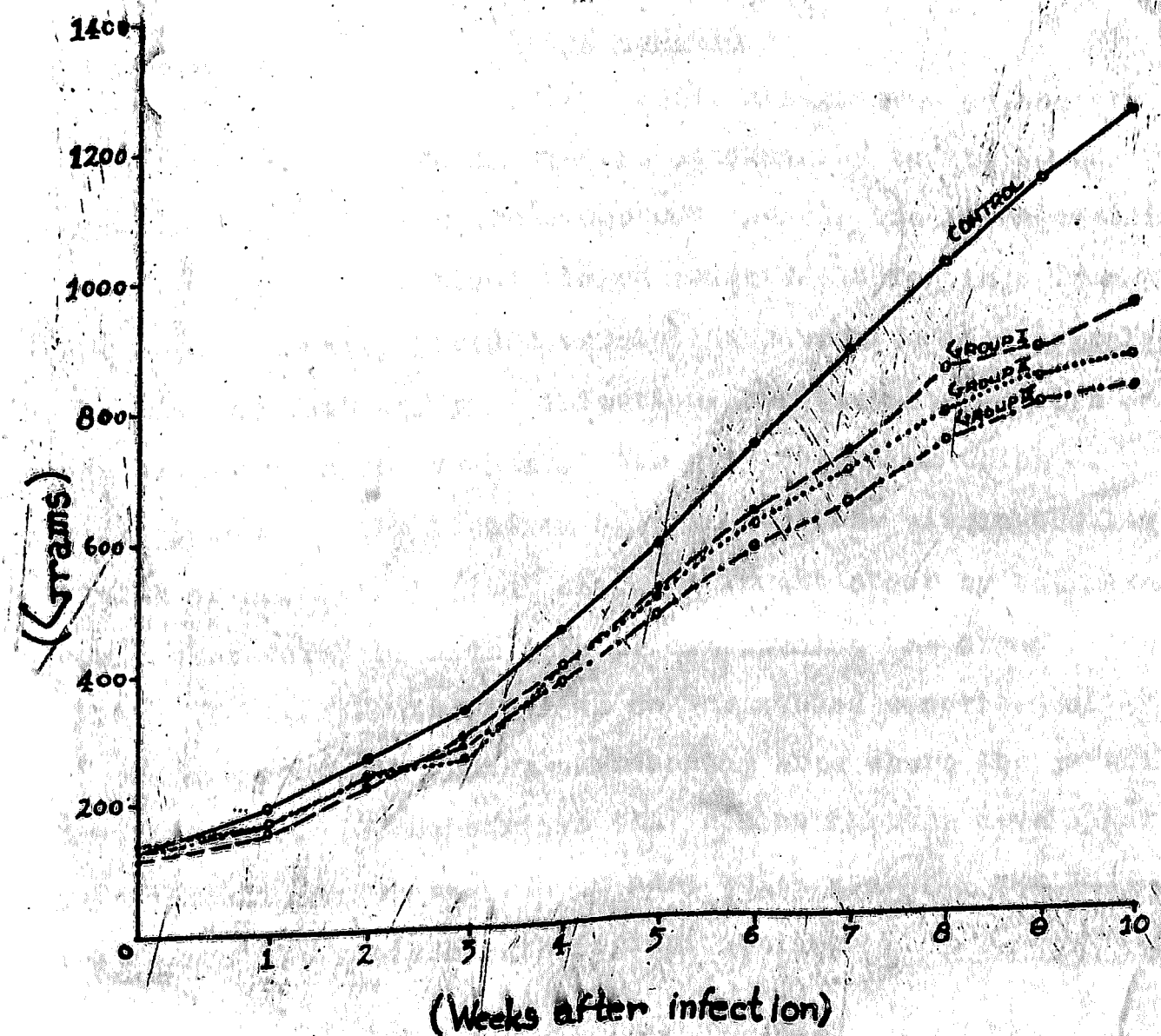
The data are presented in Table 26.

TABLE - 26 - Showing the details of prepatent period and percentage of establishment in monospecific *A. spiralis* infection at three levels and negative controls.

	Chick number	Number of infective juveniles given	Prepatent period in days	Number of worms developed			Percentage of establishment
				Male	Female	Total	
A	1	100	25	18	53	71	71.00%
	2	100	26	28	37	65	65.00%
	3	100	24	30	44	74	74.00%
	TOTAL	300				210	70.00%
B	4	200	26	55	99	154	77.00%
	5	200	25	37	95	132	66.00%
	6	200	29	60	87	147	73.50%
	TOTAL	600				433	72.16%
C	7	400	29	97	154	251	62.75%
	8	400	26	62	124	186	46.50%
	9	400	26	126	176	302	75.50%
	TOTAL	1200				739	61.58%
D	10	NIL	All the birds remained negative				
	11						
	12						

A - GROUP I
B - GROUP II
C - GROUP III
D - GROUP IV

CHART-18 EFFECT ON WEIGHT GAIN IN MONOSPECIFIC A. SPIRALIS INFECTION



HISTOPATHOLOGICAL OBSERVATIONS

Gross and microscopic changes in the proventriculus of chicks infected with 500 juveniles each of *Acuaria spiralis* were studied on the 4th (representing the 3rd stage) 8th, 10th, 12th & 14th (representing the 4th stage), 16th, 18th, 20th, & 22nd (representing the young adult stage), 24th, 50th and 100th day (representing the adult stage) post infection. The changes observed were as follows :

GROSS LESIONS

The proventriculus of the chick slaughtered on the 4th day post infection showed no visible abnormality to the naked eye, however when the organ was cut opened, the lumen contained a large quantity of blood tinged exudate. Scrappings from the mucosa readily yielded developing juveniles of *A. spiralis*.

On the 10th day post infection, an irregular whitish patch was observed on the serosa of the proventriculus which corrosponded to the location of the juveniles within the lumen of the organ. Pathological changes brought about by the parasite could therefore, be made even before opening the organ (Plate VI Fig. 1). The lesions on the mucosa comprised of ulcers and necrosis of the surrounding area where the juveniles have attached. It was curious that almost all the developing juveniles clustered towards an area which probably was the reason for the visible pathological lesion.

On the 16th day post infection the proventricular wall was thick and the lumen contained a considerable amount of debris along with copious mucus secretion. The mucous membrane appeared congested and showed numerous small ulcers. Some of the glandular openings were very prominant and on section juveniles were found within the lumen of these glands.

On the 22nd day post infection, corresponding to the pale areas in the serosa, the mucous membrane showed elevated areas covered by mucopurulent exudate. Mature worms were found buried with their heads deep in these areas.

On the 100th day post infection the proventriculus was almost two and a half times the normal size. The organ had a hard texture and was round in shape. On opening the lumen was found to be greatly reduced due to the nodular lesions on the mucosa. Almost the entire portion of the mucous membrane was involved and the worms were found deeply buried in these growths (Plate V).

MICROSCOPIC LESIONS

4th day post infection:

On the fourth day post infection, focal areas of desquamation and necrosis of glandular epithelium was seen over the affected area (Plate VI Fig. 2). The goblet cells were hyperplastic and the lumen of the organ contained abundant necrotic cellular materials. Cut sections of the juveniles were seen partially embedded in the lamina propria. The

submucosal connective tissue was hypertrophied and the blood vessels were found engorged. Mild hypertrophy of the circular muscle fibres were noticed beneath the affected areas.

8th day post infection

By eighth day post infection severe non-keratinizing squamous cell metaplasia of the lining cells in the lamina propria was seen. Some portions of the tubular gland had undergone compensatory hyperplastic changes. Moderate diffuse thickening due to cellular infiltration, chiefly consisting of granulocytes, were observed in the lamina propria. Some of the compound glands contained juveniles in their lumen, encircled by necrotic cell debris and desquamated epithelial cells (Plate VI Fig.3 & Plate VII Fig.1).

10th day post infection

On the tenth day post infection complete denudation of the mucous membrane was noticed in focal areas with adjacent areas showing compensatory hyperplastic changes. Juveniles were equally found in both these areas. Most of the compound glands showed accumulation of inflammatory exudate in their lumina but no juveniles were discernable (Plate VI Fig.4).

12th day post infection

On the twelth day post infection pronounced and circumscribed multiple, lymphoid nodule formations (Plate VIII Fig.1) were noticed in the lamina propria where the worms were

found embedded within the mucous membrane (Plate VII Fig. 2).

14th day post infection

On the fourteenth day post infection deep ulcers, necrotic foci, lymphoid hyperplasia and severe cellular infiltration with granulocytes were observed in the lamina propria. Numerous juveniles were found buried deep in the lining epithelium. The mucous membrane was completly destroyed and the compound glands in the vicinity of the affected portions showed marked inflammatory changes (Plate VIII Fig. 2).

16th day post infection

Proventriculus examined on the sixteenth day post infection showed juveniles within the lumina of some of the compound glands indicating that the worms may occasionally invade the compound glands (Plate VIII Fig. 3). An increase in the smooth muscle fibres were observed in the lamina propria.

18th day post infection

On the eighteenth day post infection mature female worms were found embedded inthe lamina propria surrounded by necrotic cellular materials derived from the damaged glands (Plate VIII Fig. 4). Lymphoid hyperplasia, mild fibroplasia and a mononuclear histiocytic reaction were some of the other changes noticed. Many of the compound glands showed loss of

lining cells and degenerative changes. Interglandular oedema was very prominant.

20th to 24th day post infection

On the twentieth to twenty second day post infection fibroplastic changes were noticed in the lamina propria along with the histiocytic reaction (Plate IX Fig. 1 & 2). By twenty fourth day post infection the submucosa, periglandular region and muscular coat were invaded by fibrous connective tissue. Worms present within the lumen of the proventriculus caused severe necrosis of the lamina propria (Plate IX Fig. 3).

50th and 100th day post infection

On the fiftieth day post infection fibro-adenamatoid growths were seen in the lamina propria which became more pronounced and pedunculated by hundredth day post infection. The cellular reaction was minimum and the lamina propria in focal areas were completely sloughed off. (Plate IX Fig. 4). Cut sections of the worms were found embedded within the fibro-adenamatoid growths.

The overall histopathological changes, therefore, clearly indicate that the worms are highly pathogenic to their host resulting in extensive pathological changes involving almost all the layers of the organ. The mortality noted in Acuaria spiralis infection was, in all probability, due to these changes.

PLATE V - Proventriculus - monospecific A. spiralis infection - Note the pronounced nodular lesions occupying almost the whole of the mucous membrane of the organ.

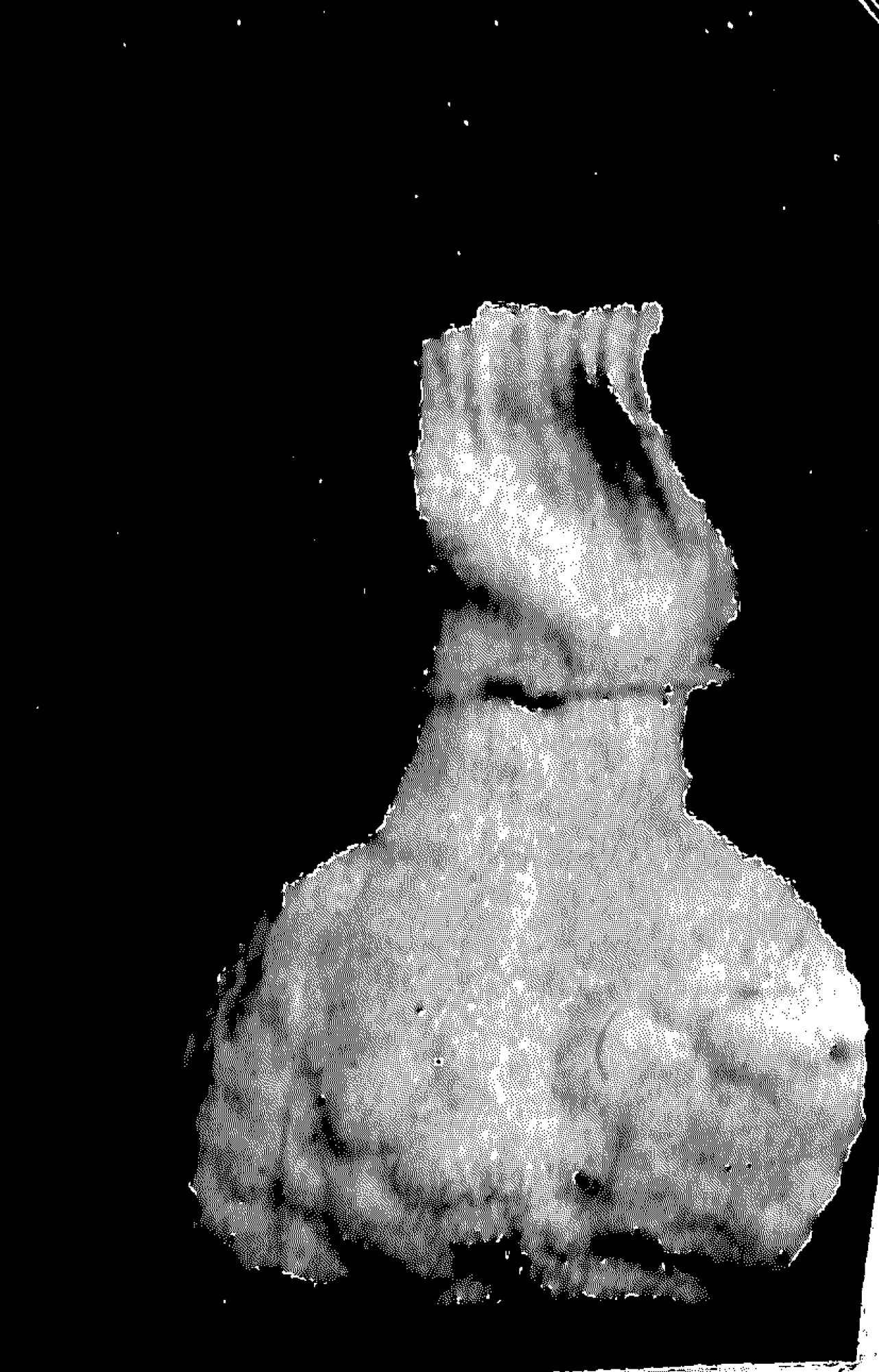


PLATE VI

Fig. 1. Proventriculus - A.spiralis infection. Note the irregular whitish patch on the serosa which corrosponded to the location of juveniles within the lumen of the organ. ×2·5

Fig. 2. Proventriculus - A.spiralis infection 4th day- Note the focal areas of desquamation and necrosis of the mucous membrane. H&E ×50

Fig. 3. Proventriculus - A.spiralis infection 8th day- Severe non-keratinizing squamous cell metaplasia of the lining epithelium in the lamina propria (indicated by arrow). Juveniles are seen within the compound gland. H&E ×50

Fig. 4. Proventriculus - A.spiralis infection 10th day- Focal area in the mucous membrane compleatly denuded and there is accumulation of inflammatory exudate within the lumen of a compound gland. Juveniles (indicated by arrow) are seen within the lumen of the proventriculus. H&E ×50

PLATE VI

Fig. 1

Fig. 2

Fig. 3

Fig. 4

PLATE VII

Fig. 1. Proventriculus - A.spiralis infection 8th day - Note the metaplastic changes in the mucous membrane and the juvenile within the compound gland. H&E ×70

Fig. 2. Proventriculus - A.spiralis infection 12th day- Note the juveniles embedded within the hyperplastic tubular glands and the pronounced circumscribed, multiple lymphoid nodules in the lamina propria.
H·Agan × 70

PLATE VII

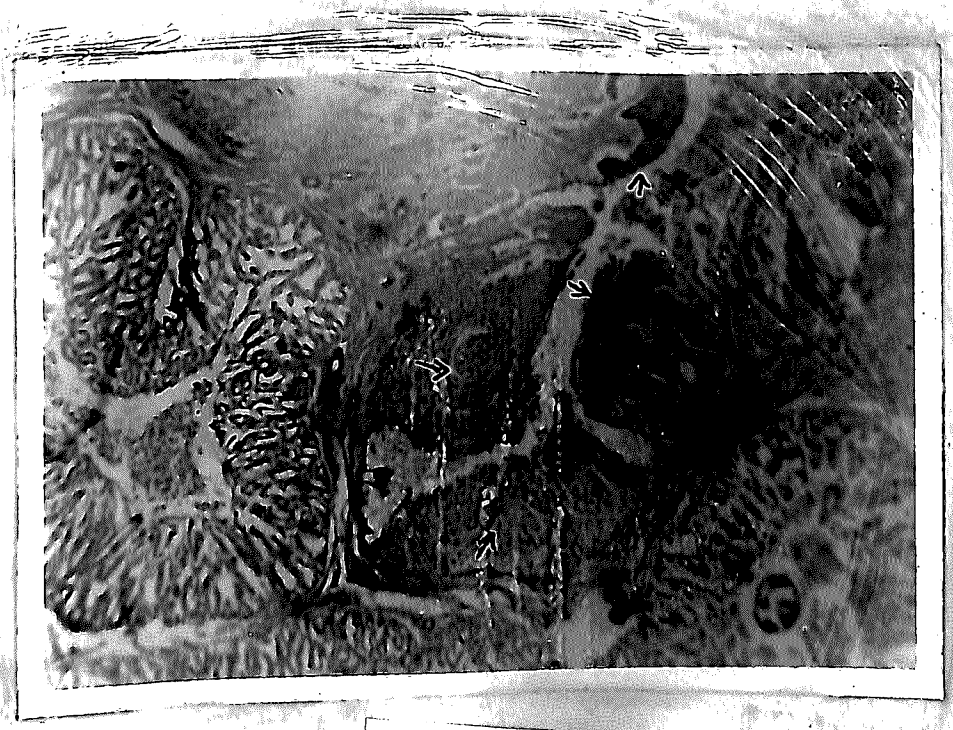


Fig. 1

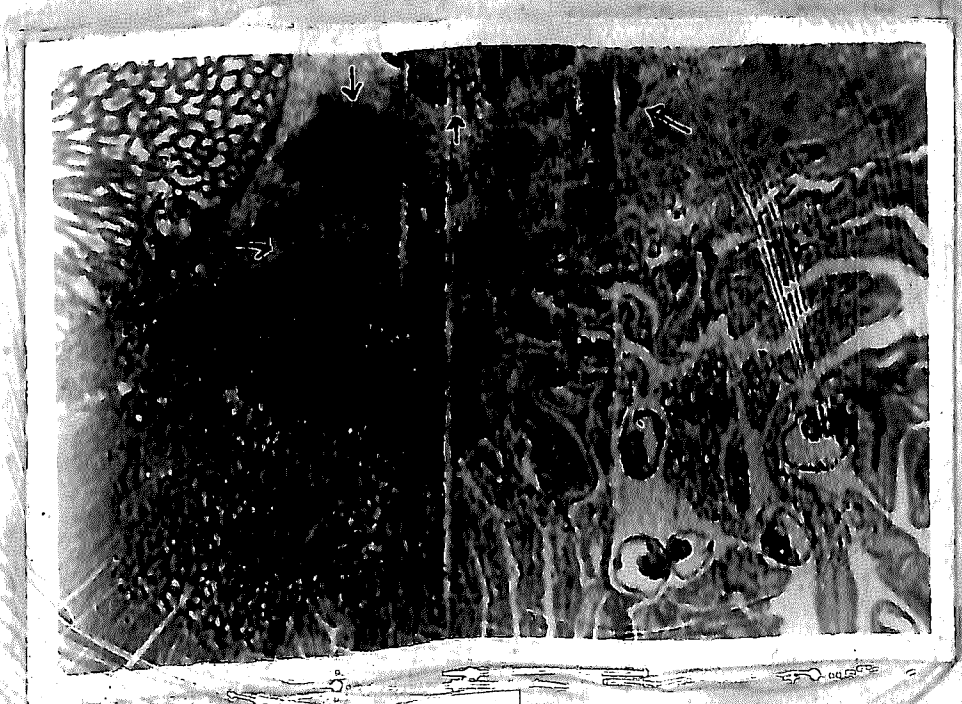


Fig. 2

PLATE VIII

Fig. 1. Proventriculus - A.spiralis infection 12th day- Lymphoid hyperplasia in the lamina propria. x100

Fig. 2. Proventriculus - A.spiralis infection 14th day- Note the severely damaged mucous membrane where the juveniles have been attached. Portion of the compound gland also shows marked inflammatory reaction
H&E x 50

Fig. 3. Proventriculus - A.spiralis infection 16th day- Note the juveniles within the compound glands.
H & E x 50

Fig. 4. Proventriculus - A.spiralis infection 18th day- Female worm is seen embedded in the mucous membrane surrounded by necrotic cellular debris. Note the mild fibroplasia and submucosal oedema.
H·Azan x 50

Fig. 1.

Fig. 2

Fig. 3

Fig. 4

PLATE IX

Fig. 1. Proventriculus - A. spiralis infection 20th day - Lamina propria grately thickened due t infiltrating cells and fibreplasia.
Van Gieson X 50

Fig. 2. Proventriculus - A. spiralis infection 22nd day - Worms embedded within the mucous membrane cause great tissue damage
H.Azan X 50

Fig. 3. Proventriculus - A. spiralis infection 24th day - Note ;-
Necrosis and Disruption of the mucous membrane
Fibrosed lamina propria
Dissolution of the glandular tissue of the compound gland and
Periglandular oedema.
H & E X 50

Fig. 4. Proventriculus - A. spiralis infection 100th day- Mucous membrane is compleately denuded and there is marked fibrosis. Arrow indicates portion of the fibro-adenomatoid growth in the lamina propria.
H & E A 20

Fig. 1

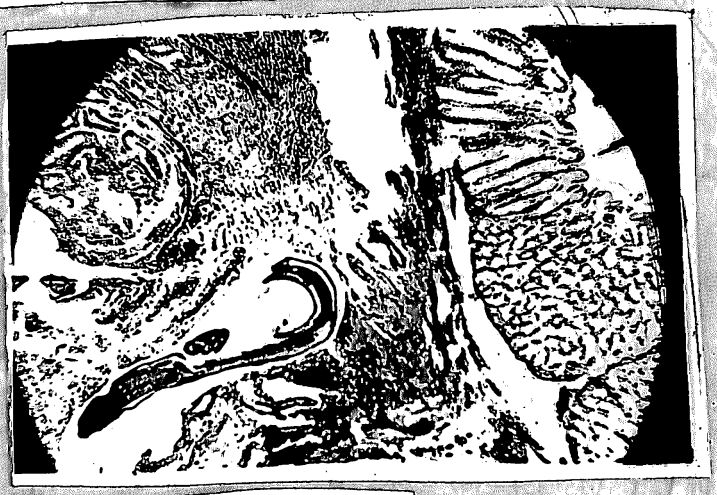


Fig. 2

Fig. 3

Fig. 4

DISCUSSION

DISCUSSION

The present investigation seems to be the first controlled study on the various pathogenic effects caused by the different developmental stages of *Acuaria spiralis* in fowls.

Deo (1964) has stated that in spite of the voracious appetite young chickens become anaemic and lost weight. The haematological studies conducted during the present investigation indicate that in *A. spiralis* infection a macrocytic hypochromic anaemia occurs during the 9th week post infection. This anaemia may be due to a deficiency of some of the essential nutrients as a result of the infection.

Eosinophilia and heterophilia were observed by Olson & Levine (1939) in controlled *Capillaria columbae* infection and Wickware (1947) in *Heterakis gallinae* infection of fowls. In present study, in *A. spiralis* infection, eosinophilia was observed during the 2nd week post infection and heterophilia and lymphocytopenia during the 3rd and 4th week post infection. From the 24th day post infection leucopenia was evident.

The maximum worm load noticed in natural *A. spiralis* infection by Sundaram (1971) was 174 males and 282 females. In the present study the maximum worm load obtained was 129 males and 184 females (number of juveniles administered being 400).

During the present study the *A. spiralis* worms were found only within the proventriculus, though it has been recorded

from oesophagus by Hsu (1959); Merkusheva & Kraevskia (1963); Slater (1967) and Bwangamoi (1968) and from small intestine by Ahmad (1962); Hassan (1966); Diaz-Ungria & Torres Artigas (1966) and Bwangamoi (1968).

According to Bump et al. (1947) A. spiralis is the only parasite pathogenic to ruffed grouse "though the bird may be host to 14 species of round worms". Allen (1925) ascribed A. spiralis to be the chief causative agent of "Grouse disease" in North-eastern United States and Kocjan (1934) reported that A. spiralis causes an enzootic disease in Pigeon. Cram (1928) observed that heavy infestation of this parasite resulted in death of many carrier pigeons in Texas. Mortality due to A. spiralis has also been reported by Edminister (1947) in ruffed grouse and Hwang et al. (1961) in pigeons. Bendell (1955) opined that parasitism due to D. nasuta (A. spiralis) and Plagiorynchus formosus was an important mortality factor in chicks and a major cause of population stability. Soulsby (1965) observed that death occurs in A. spiralis infection during 2 to 3 weeks after the onset of clinical signs. Karstad & Silco (1971) believed that A. spiralis is responsible for the death of many birds during autumn and winter. During the present investigation mortality was observed on the 8th, 10th and 20th day post infection. Three out of the twelve chicks infected with 500 juveniles succumbed to the infection in one experiment (in monospecific infection) and four out of twelve chicks

infected with the same dose died in another experiment (in dispecific infections). Thus making a mortality percentage of 29.16%. This indicates that the LD_{50} of Acuaria spiralis infection may be slightly above 500. However, in natural infections desi birds are found to harbour nearly 500 worms with apparently no clinical symptoms indicating that desi birds are more resistant to the infection than the exotic birds.

The observation made on the weight gain of the birds indicate that as a result of A. spiralis infection the birds lose considerable amount of their body weight. In infected layers the egg production was greatly supressed and their feed efficiency was also very low. The prepatent period of infection observed were found to be similar to those made by Sundaram (1971).

Deo (1964) and Soulsby (1968) have stated that the pathogenic effect of the parasite depends on the severity of infection. Similar observations were made in the present study, where the maximum pathogenic effects were seen in heavily infected (Group III) birds.

According to Soulsby (1965) the proventriculus may be as large as gizzard in heavy infections, due to marked hypertrophy of the proventricular wall and the mucous membrane may be completely destroyed, the various layers of tissue being indistinguishable from one another. During the present study in heavy infections the whole of the mucous membrane

was found to be covered by nodular growths. The characteristic appearance of the A. spiralis lesion on the serosa observed during the present investigation (Plate VI Fig. 1) has not been reported earlier.

Normally the parasites do not invade the compound glands, but in the present study juveniles were found within the lumen of the compound glands on the 8th and 16th day post infection (Plate VI Fig. 3) and (Plate VIII Fig. 3) indicating that in heavy infections the juveniles may enter the compound glands. The days in which the juveniles were found within the compound glands correspond to the 3rd and 4th larval moult observed by Sundaram (1971).

Intense cellular reaction especially eosinophilic infiltration into the mucosa were described by Soulsby (1965). In the present study the juveniles (especially the 4th stage) were found to evoke a severe granulocytic reaction. Pronounced lymphoid hyperplasia observed during this period indicate the immunity reactions.

Hwang et al. (1961) observed a chronic catarrhal type of inflammation characterised by epithelial desquamation, papillary proliferation, hypersecretion of mucus, congestion and secondary bacterial invasion of the superficial mucosa. Joshi & Kamalapur (1971) studying the histopathological lesions produced by natural A. spiralis infection in the proventriculi of fowls observed proliferation of fibrous tissue, desquamation and necrosis of epithelial cells,

was found to be covered by nodular growths. The characteristic appearance of the A. spiralis lesion on the serosa observed during the present investigation (Plate VI Fig. 1) has not been reported earlier.

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ulceration of the mucosa, interglandular oedema and infiltrating cells. Soulsby (1965) has mentioned an adenomatous proliferation of the glandular epithelium with marked desquamation and ulceration. In the present study severe non-keratinizing squamous cell metaplasia were observed on the 8th day post infection and by 10th day post infection there was complete denudation of the mucous membrane with pronounced granulocytic reaction. A marked lymphoid hyperplasia was observed from the 12th day post infection marking the development of local immunity. By the 18th day post infection mononuclear histiocytic reaction were evident in the lamina propria with degenerative changes of the compound glands. From the 20th to 24th day post infection fibroplasia were evident, indicating that the pathological process tends to become chronic after this period. Pedunculated fibroadenomatoid growths were formed in the lamina propria from the 50th to 100th day post infection which were quite apparent to the naked eye. These changes indicate that the worm(A. spiralis) has also some ability to cause tumour formation just as some of the other spirurid worms (Spirocerca lupi, Gnathostoma spinigerum or Habronema megastoma) in other hosts. The foci of rod shaped bacteria observed by Hwang et al. (1961) was not seen in the present study.

Thus the results obtained in the present study indicate that A. spiralis infection may cause a great economic loss to the Poultry industry.

PATHOGENICITY
OF
Tetrameres mohtedai & Acuaria spiralis
IN
DISPECIFIC INFECTIONS

PATHOGENICITY OF TETRAMERES MOHTEDAI & ACUARIA SPIRALIS IN DISPECIFIC INFECTIONS

HAEMATOLOGICAL OBSERVATIONS

To study the blood alterations in 3 levels of dispecific infections with Tetrameres mohtedai and Acuaria spiralis, blood samples were collected on the 6th, 13th, 24th, 38th, 52nd and 66th day post infection representing the various developmental stages of the parasites involved.

TOTAL ERYTHROCYTES

The mean overall erythrocyte count of the entire experimental groups was significantly lower ($P < 0.01$) than the negative controls.

The decrease in respect of Group I (average number of worms established 64.66 of T. mohtedai and 71.66 of A. spiralis) was 3.55% (not significant), in respect of Group II (average number of worms established 82 of T. mohtedai and 113.33 of A. spiralis) 7.76% (significant at 5% level) and in respect of Group III (average number of worms established 124 of T. mohtedai and 244.66 of A. spiralis) 16.56% (significant at 1% level).

On a further analysis Group I showed a decrease of 12.18% ($P < 0.05$) on the 52nd day post infection, Group II showed a decrease of 10.35% ($P < 0.05$) on the 24th day post infection and Group III showed a decrease of 8.85% ($P < 0.05$), 25.21%,

37.96% & 37.27% ($P < 0.01$) on the 24th, 38th, 52nd & 66th day post infection respectively.

Thus the total erythrocytes of the experimental groups was found to be decreased from the 24th day post infection upto the observed period of 66th day post infection

The data are presented in Table 27 and are graphically represented in Chart 19.

HAEMOGLOBIN

The mean overall haemoglobin percentage of the entire experimental groups was significantly lower ($P < 0.01$) than the negative controls.

The decrease in respect of Group I (average number of worms established 64.66 of *T. mohtedni* and 71.66 of *A. spiralis*) was 9.92% ($P < 0.05$), in respect of Group II (average number of worms established 82 of *T. mohtedni* and 113.33 of *A. spiralis*) 14.51% ($P < 0.01$) and in respect of Group III (average number of worms established 124 of *T. mohtedai* and 244.66 of *A. spiralis*) 18.40% ($P < 0.01$).

On a further analysis Group I showed a decrease of 11.86% ($P < 0.05$) on the 66th day post infection, Group II showed a decrease of 14.81%, 16.98% ($P < 0.05$) & 18.52% ($P < 0.01$) on the 38th, 52nd & 66th day post infection respectively and Group III showed a decrease of 15.19% ($P < 0.05$), 24.91% & 25.93% ($P < 0.01$) on the 38th, 52nd & 66th day post infection respectively.

Thus the haemoglobin percentage of the experimental groups was found to decrease from 38th day post infection and was maximum on the 66th day.

The data are presented in Table 28 and are graphically represented in Chart 20.

PACKED CELL VOLUME

The mean overall packed cell volume of the entire experimental groups was significantly lower ($P<0.01$) than the negative controls.

The decrease in respect of Group I (average number of worms established 64.66 of T.mohtedai and 71.66 of A.spiralis) was 1.86% (not significant), in respect of Group II (average number of worms established 82 of T.mohtedai and 113.33 of A.spiralis) 2.60% (significant at 5% level) and in respect of Group III (average number of worms established 124 of T.mohtedai and 244.66 of A.spiralis) 6.69% (significant at 1% level).

On a further analysis Group II showed a decrease of 6.67% ($P<0.05$) on the 66th day post infection. Group III showed a decrease of 5.76% ($P<0.05$), 7.88% & 17.78% ($P<0.01$) on the 24th, 52nd & 66th day post infection respectively.

Thus the packed cell volume was found to be decreased from the 52nd day post infection in groups II & III, while in group I it was not significant.

The data are presented in Table 29 and are graphically represented in Chart 21.

MEAN CORPUSCULAR VOLUME

The overall mean corpuscular volume of the entire experimental groups was significantly higher ($P < 0.05$) than the negative controls.

The increase in respect of Group I (average number of worms established 64.66 of *T. mohtedni* and 71.66 of *A. spiralis*) was 2.43% (not significant), in respect of Group II (average number of worms established 82 of *T. mohtedni* and 113.33 of *A. spiralis*) 6.77% (not significant) and in respect of Group III (average number of worms established 124 of *T. mohtedni* and 244.66 of *A. spiralis*) 17.36% (significant at 1% level).

On a further analysis, Group III showed an increase of 29.06%, 51.90% & 30.92% ($P < 0.01$) on the 38th, 52nd & 66th day post infection respectively.

Thus the increase in the mean corpuscular volume was found to be maximum on the 52th day post infection in Group III. Whereas in groups I & II it was not significant.

The data are presented in Table 30 and are graphically represented in Chart 22.

MEAN CORPUSCULAR HAEMOGLOBIN

The overall mean corpuscular haemoglobin of Group III (average number of worms established 124 of *T. mohtedni* and 244.66 of *A. spiralis*) was significantly higher ($P < 0.01$) than the negative controls. An increase of 10.55% was obtained. Group I and Group II showed no significant difference.

Further analysis revealed that the increase in Group III was maximum on the 52nd day post infection.

The data are presented in Table 31.

MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION

The overall mean corpuscular haemoglobin concentration of the entire experimental groups was significantly lower ($P < 0.05$) than the negative controls.

The decrease in respect of Group I (average number of worms established 64.66 of *T. mohtedai* and 71.66 of *A. spiralis*) was 2.18% (not significant), in respect of Group II (average number of worms established 82 of *T. mohtedai* and 113.33 of *A. spiralis*) 6.62% (significant at 1% level) and in respect of Group III (average number of worms established 124 of *T. mohtedai* and 244.66 of *A. spiralis*) 6.72% (significant at 1% level).

On a further analysis, Group I showed a decrease of 9.90% ($P < 0.05$) on the 66th day post infection; Group II showed a decrease of 14.13% & 12.68% ($P < 0.01$) on the 52nd and 66th day post infection respectively. Group III showed a decrease of 11.63% ($P < 0.05$) on the 38th day post infection, 17.54% ($P < 0.01$) and 10.02% ($P < 0.05$) on the 52nd and 66th day post infection, respectively.

The data are presented in Table 32.

TOTAL LEUCOCYTES

The total leucocytes showed a significant increase ($P < 0.01$) of 18.36% in Group I (average number of worms established 64.66 of *T. mohtedai* and 71.66 of *A. spiralis*); 41.11% in Group II (average number of worms established 82 of *T. mohtedai* and 113.33 of *A. spiralis*) and 33.65% in Group III (average number of worms established 124 of *T. mohtedai* and 244.66 of *A. spiralis*) on the 6th day post infection. A slight increase was also observed on the 13th day in all the groups, but was not significant. However, by 24th day post infection a steep decrease of 48.79% and 43.46% ($P < 0.01$) respectively were observed in Group II and Group III. In Group I the decrease was evident only from the 38th day post infection. The decrease obtained was maximum on the 52nd day post infection in all the groups.

Thus leucocytosis was observed on the 6th & 13th day post infection in all the experimental groups followed by a leucopenia on the 24th, 38th, 52nd & 66th day post infection.

The data are presented in Table 33 and are graphically represented in Chart 23.

EOSINOPHILS

The mean overall eosinophil percentage of the entire experimental groups was significantly higher ($P < 0.01$) than the negative controls.

The increase in respect of Group I (average number of

worms established 64.66 of T.mohtedai and 71.66 of A.spiralis) was 214.69% ($P < 0.01$), in respect of Group II (average number of worms established 82 of T.mohtedai and 113.33 of A.spiralis) was 174.83% ($P < 0.01$) and in respect of Group III (average number of worms established 124 of T.mohtedai and 244.66 of A.spiralis was 245.45% ($P < 0.01$).

On a further analysis Group I showed an increase of 611% ($P < 0.01$) and 286,27% ($P < 0.05$) on the 13th & 24th day post infection respectively. Group II showed an increase of 383.3% ($P < 0.01$) and 225.02% ($P < 0.05$) on the 13th & 38th day post infection respectively and Group III showed an increase of 477.77%,($P < 0.01$) 279.10%, 180.02% & 230.03% ($P < 0.05$) on the 13th, 24th, 38th & 66th day post infection respectively.

Thus marked eosinophilia was evident in all the experimental groups on the 13th day post infection.

The data are presented in Table 34 and are graphically represented in Chart 24.

HETEROPHILS

The mean overall heterophil percentage of the entire experimental groups was significantly higher ($P < 0.05$) than the negative controls.

The increase in respect of Group I (average number of worms established 64.66 of T.mohtedai and 71.66 of A.spiralis) was 3.43% (not significant), in respect of Group II (average number of worms established 82 of T.mohtedai and 113.33 of

DISCUSSION

DISCUSSION

In natural infections quite often chicks were found to harbour both T.mohtedai and A.spiralis simultaneously. Similar observations were made by Sundaram (1971) who reported that 8.8% of 2427 proventriculi examined showed mixed infection. Hence the present study was undertaken to know to what extent both these parasites could cause pathological alterations in fowls. Since the location of both the parasites is proventriculus the studies have to be designed for monospecific and dispecific infections to note the individual and combined effect of parasitism on the host. No controlled experiments have been attempted earlier to study the combined effects of T.mohtedai and A.spiralis in fowls, since the life cycle of both these parasites were elucidated only recently by Sundaram (1963, 1971).

The haematological changes observed were macrocytic hypochromic anaemia, leucopenia, eosinophilia, heterophilia lymphocytopenia and monocytosis.

While conducting the experiment for histopathological studies, mortality of 4 chicks out of 12 (33.33%) infected with 500 juveniles each of A.spiralis and T.mohtedai, occurred. Since T.mohtedai was found to be not lethal to chicks (even at a dose rate of 600 juveniles) it is apparent that the reason for death is mainly due to A.spiralis. Moreover similar results were obtained in monospecific A.spiralis infection also where

A. spiralis) 21.52% (significant at 5% level) and in Group III (average number of worms established 124 of T. mohtedai and 244.66 of A. spiralis) 20.69% (significant at 5% level).

On a further analysis, Group II showed an increase of 69.92%, 32.12% and 41.56% ($P < 0.05$) on the 24th, 52nd and 66th day post infection respectively and Group III showed an increase of 52.03%, 25.46% and 28.57% ($P < 0.05$) on the 24th, 52nd and 66th day post infection respectively.

Thus heterophilia was observed on the 24th, 52nd and 66th day post infection in Group II & Group III. In Group I it was not significant.

Thed data are presented in Table 35 and are graphically represented in Chart 25.

BASOPHILS

The mean overall basophil percentage of the experimental groups showed no significant difference from that of the negative controls.

The data are presented in Table 36.

LYMPHOCYTES

The mean overall lymphocyte percentage of the entire experimental groups was significantly lower ($P < 0.01$) than the negative controls.

The decrease in respect of Group I (average number of worms established 64.66 of T. mohtedai and 71.66 of A. spiralis) was 4.31% (not significant), in respect of Group II (average

number of worms established 82 of T.mohtedai and 113.33 of A.spiralis) 14.12% (significant at 1% level) and in respect of Group III (average number of worms established 124 of T.mohtedai and 244.66 of A.spiralis) 16.21% (significant at 1% level).

On a further analysis, Group I showed a decrease of 29.76% ($P < 0.01$) on the 13th day post infection, Group II showed a decrease of 27.38% & 22.44% ($P < 0.01$) on the 13th & 66th day post infection respectively and Group III showed a decrease of 32.74% & 25.85% ($P < 0.01$) on 13th & 66th day post infection respectively.

Thus lymphocytopenia was observed on the 13th & 66th day post infection in all the experimental groups.

The data are presented in Table 37 and are graphically represented in Chart 26.

MONOCYTES

The mean overall monocyte percentage of the entire experimental groups was significantly higher ($P < 0.05$) than the negative controls.

The increase in respect of Group I (average number of worms established 64.66 of T.mohtedai and 71.66 of A.spiralis) was 27.85% (not significant), in respect of Group II (average number of worms established 82 of T.mohtedai and 113.33 of A.spiralis) 46.83% (significant at 5% level) and in respect of Group III (average number of worms established 124 of

T.mohtedai and 244.66 of *A.spiralis*) was 50.62% (significant at 5% level).

Further analysis showed an increase of 100.30% ($P < 0.05$) in Group II and 178.97% ($P < 0.05$) in Group III on the 66th day post infection.

Thus monocytosis was observed on the 66th day post infection in groups II & III.

The data are presented in Table 38.

TABLE - 27 - Showing the total Erythrocyte count in three levels of dispecific infections with T. mohtedai and A. spiralis compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	2.56	2.13	2.31	2.67	2.31	2.13	2.39	2.12	2.07	2.44	2.15	2.36
13th	2.38	2.23	2.58	2.14	2.15	2.18	2.80	2.51	2.38	2.37	2.12	2.22
24th	1.91	1.91	1.96	1.61	2.12	1.65	2.06	1.80	1.61	2.13	1.98	1.89
38th	1.74	2.37	2.08	2.19	1.61	1.86	1.73	1.70	1.58	2.13	2.14	2.43
52nd	1.75	1.93	2.01	1.97	1.84	2.14	1.73	1.32	1.67	2.29	2.02	2.17
66th	2.16	1.79	2.24	1.71	2.13	1.97	1.31	1.38	1.45	2.34	2.08	2.18

All values in millions/cu.mm. of blood.

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	1.552	0.517	9.223 **
Between Days	5	2.964	0.593	10.572 **
Error	63	3.532	0.056	
TOTAL	71	8.048		

** Significant at 1% level.

TABLE - 28 - Showing the Haemoglobin values in three levels of dispecific infections with T. mohtedai and A. spiralis compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	7.0	7.0	6.8	6.8	6.8	6.8	6.2	7.0	7.4	8.4	6.6	6.4
13th	7.6	7.2	8.2	7.8	7.6	7.6	7.6	7.4	7.6	7.4	6.2	7.8
24th	8.0	8.0	6.5	7.4	7.4	6.8	7.0	6.2	6.8	7.8	7.0	7.3
38th	7.6	7.8	9.0	7.6	7.4	8.0	7.6	8.0	7.3	8.4	9.0	9.6
52nd	8.6	7.6	8.4	7.4	7.2	7.4	6.0	6.7	7.2	9.3	8.4	8.8
66th	7.6	8.0	8.2	7.8	7.2	7.0	6.2	6.6	7.2	9.2	8.8	9.0

All values in gram percentage

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	14.641	4.880	4.715 **
Between Days	5	10.814	2.163	2.089 n.s.
Error	63	65.207	1.035	
TOTAL	71	90.662		

** Significant at 1% level

n.s. Not significant

TABLE - 30 - Showing the Mean corpuscular volume in three levels of dispecific infections with T. mohtedai and A. spiralis compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	117.19	140.85	129.87	112.36	129.87	140.85	125.52	141.51	144.93	127.05	144.19	131.36
13th	121.85	130.04	116.28	130.84	139.53	142.20	100.00	115.54	126.05	126.58	141.51	130.63
24th	151.83	146.60	153.06	180.12	141.51	175.76	140.78	150.00	161.50	140.85	146.46	148.15
38th	160.92	126.58	139.42	136.99	173.91	155.91	167.63	176.47	177.22	145.54	130.84	127.57
52nd	160.00	150.26	149.25	147.21	146.74	140.19	242.72	212.12	173.65	126.64	153.47	133.64
66th	134.26	162.01	133.93	163.74	138.15	137.06	183.21	173.91	179.31	132.48	144.23	133.03

All values in cubic microns

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	6521.8	2173.933	7.111 **
Between Days	5	10855.6	2171.120	7.101 **
Error	63	19261.1	305.732	
TOTAL	71	36638.5		

** Significant at 1% level.

TABLE - 31 - Showing the Mean corpuscular haemoglobin in three levels of dispecific infections with T. mohtedai and A. spiralis compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	27.34	32.86	29.44	25.47	29.44	31.92	25.94	33.02	35.75	34.43	30.70	27.12
13th	31.93	32.29	31.78	36.45	35.35	34.86	27.14	29.48	31.93	31.22	29.25	35.14
24th	41.88	41.88	33.16	45.96	34.91	41.21	33.98	34.44	42.24	36.62	35.35	38.62
38th	43.68	32.91	43.27	34.70	45.96	43.01	43.93	47.06	46.20	39.44	42.06	39.51
52nd	49.14	39.38	41.79	37.56	39.13	34.58	58.25	50.76	43.11	40.61	41.58	40.55
66th	35.19	44.69	36.61	44.07	33.80	35.53	47.33	47.83	49.66	39.32	42.31	41.28

All values in micro micro grams

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	190.78	63.593	3.269 *
Between Days	5	1736.15	347.233	17.849 **
Error	63	1225.55	19.453	
TOTAL	71	3152.48		

* Significant at 5% level
** Significant at 1% level

TABLE - 32 - Showing the Mean corpuscular haemoglobin concentration in three levels of dispecific infections with T. mohtedai and A. spiralis compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	23.33	23.33	22.67	22.67	22.67	22.67	20.67	23.33	24.67	27.10	21.29	20.65
13th	26.21	24.83	27.33	27.86	25.33	24.52	27.14	25.52	25.33	24.67	20.67	26.90
24th	27.59	28.57	21.67	25.52	24.67	23.45	24.14	22.96	26.15	26.00	24.14	26.07
38th	27.14	26.00	31.03	25.33	26.43	27.59	26.21	26.67	26.07	27.10	32.14	30.10
52nd	30.71	26.21	28.00	25.52	26.67	24.67	25.05	23.93	24.83	32.07	27.10	30.34
66th	26.21	27.59	27.33	27.86	24.83	25.93	25.83	27.50	27.69	29.68	29.33	31.03

All values in percentage

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	66.226	22.075	6.448 **
Between Days	5	203.116	40.623	11.866 **
Error	63	215.674	3.423	
TOTAL	71	485.016		

** Significant at 1% level.

TABLE - 33 - Showing the total Leucocyte count in three levels of dispecific infections with T. mohtedai and A. spiralis compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	38.00	39.60	42.00	32.60	33.22	35.20	52.60	34.00	56.00	19.55	28.00	22.00
13th	42.22	35.32	44.00	36.00	26.88	56.00	29.10	66.00	24.44	42.66	27.32	44.66
24th	40.33	39.32	42.66	24.00	14.00	21.10	23.10	20.88	21.32	35.20	38.10	42.10
38th	15.80	15.80	16.00	14.20	20.40	13.80	19.10	15.10	10.80	24.00	18.00	43.30
52nd	18.70	22.70	15.30	19.10	23.80	13.80	15.10	26.40	15.30	23.30	25.50	42.20
66th	13.54	22.88	13.54	18.00	21.32	11.54	14.00	22.00	21.32	28.66	14.00	27.76

All values in thousands/cu.mm. of blood.

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	959.668	319.889	31.778 **
Between Days	5	4856.165	971.233	96.484 **
Error	63	634.175	10.066	
TOTAL	71	6450.008		

** Significant at 1% level.

TABLE - 34 - Showing the percentage of Eosinophils in three levels of dispecific infections with T. mohtedai and A. spiralis compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	9.0	7.0	5.5	6.5	2.0	5.5	8.0	2.0	12.0	4.0	3.0	3.0
13th	37.0	16.0	11.0	15.5	16.0	12.0	11.0	24.0	17.0	3.0	4.0	2.0
24th	16.0	4.0	7.0	3.0	6.5	13.0	10.0	10.5	6.0	1.0	2.0	4.0
38th	9.0	5.0	4.0	15.0	10.5	7.0	15.0	8.0	5.0	4.0	4.0	2.0
52nd	1.0	6.0	1.5	6.0	2.0	2.0	7.5	3.5	5.5	2.0	1.5	2.0
66th	9.0	8.0	6.0	8.0	9.0	2.0	13.0	6.0	14.0	3.0	3.0	4.0

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	747.153	249.051	14.977 **
Between Days	5	765.569	153.113	9.208 **
Error	63	1047.597	16.629	
TOTAL	71	2560.319		

** Significant at 1% level

TABLE - 36 - Showing the percentage of Eosophils in three levels of dispecific infections with T. mohtedci and A. spiralis compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	0.5	0.0	0.5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.0	0.0
13th	0.0	0.5	0.5	1.5	0.0	0.0	0.5	0.5	0.0	1.0	0.0	0.5
24th	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
38th	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
52nd	0.5	0.0	0.0	0.0	0.5	0.5	0.0	0.5	2.0	0.5	1.0	0.0
66th	0.0	1.0	0.0	0.0	0.0	1.0	0.5	0.5	0.0	0.0	0.0	1.0

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	0.344	0.115	0.790 n.s.
Between Days	5	2.489	0.498	3.434 **
Error	63	9.135	0.145	
TOTAL	71	11.968		

n.s. Not significant

** Significant at 1% level.

TABLE - 37 - Showing the percentage of Lymphocytes in three levels of dispecific infections with T. mohtedai and A. spiralis compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	71.0	59.0	70.5	72.5	70.0	65.5	64.0	69.0	66.0	72.0	72.0	76.0
13th	46.0	44.0	28.0	56.0	29.0	37.0	42.0	28.0	43.0	50.0	61.0	57.0
24th	68.0	76.0	54.0	53.0	45.5	63.0	61.5	52.5	60.0	31.0	72.5	72.5
38th	72.0	76.5	76.0	60.5	68.0	53.0	44.5	56.0	73.0	65.0	74.5	60.0
52nd	71.0	62.0	63.5	64.0	53.5	57.0	49.0	57.5	64.0	65.5	73.0	65.0
66th	64.0	62.0	58.0	50.0	52.0	57.0	47.5	52.5	52.0	72.0	72.0	61.0

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	2299.01	766.337	13.430 **
Between Days	5	4627.74	925.548	16.220 **
Error	63	3594.70	57.059	
TOTAL	71	10521.45		

** Significant at 1% level.

TABLE - 38 - Showing the percentage of Monocyte in three levels of dispecific infections with T. mohtedai and A. spirelis compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III			GROUP IV		
	1	2	3	4	5	6	7	8	9	10	11	12
6th	1.5	6.0	5.5	1.0	5.0	1.0	6.0	1.0	9.0	3.0	1.0	4.0
13th	1.0	3.0	3.5	3.0	3.0,	5.0	3.5	1.0	3.0	2.0	1.0	3.5
24th	1.0	4.0	1.0	4.0	2.5	5.5	2.0	1.5	2.0	3.0	1.5	1.0
38th	2.0	1.0	4.0	1.5	5.0	1.0	0.5	2.0	4.0	1.0	2.0	2.5
52nd	3.0	2.5	1.5	1.0	3.0	2.5	2.5	2.0	2.0	2.5	1.5	3.0
66th	2.0	3.0	5.0	5.0	6.0	3.0	4.0	5.0	10.5	3.0	2.0	2.0

Analysis of Variance Table

Source	df	SS	MSS	F
Between Treatments	3	65.622	21.874	8.514 **
Between Days	5	41.642	8.328	3.672 **
Error	63	142.899	2.268	
TOTAL	71	250.163		

** Significant at 1% level.

IN DISPECIFIC INFECTIONS WITH
T. MOHTEDAI & A. SPIRALIS

CHART 19 TOTAL ERYTHROCYTES

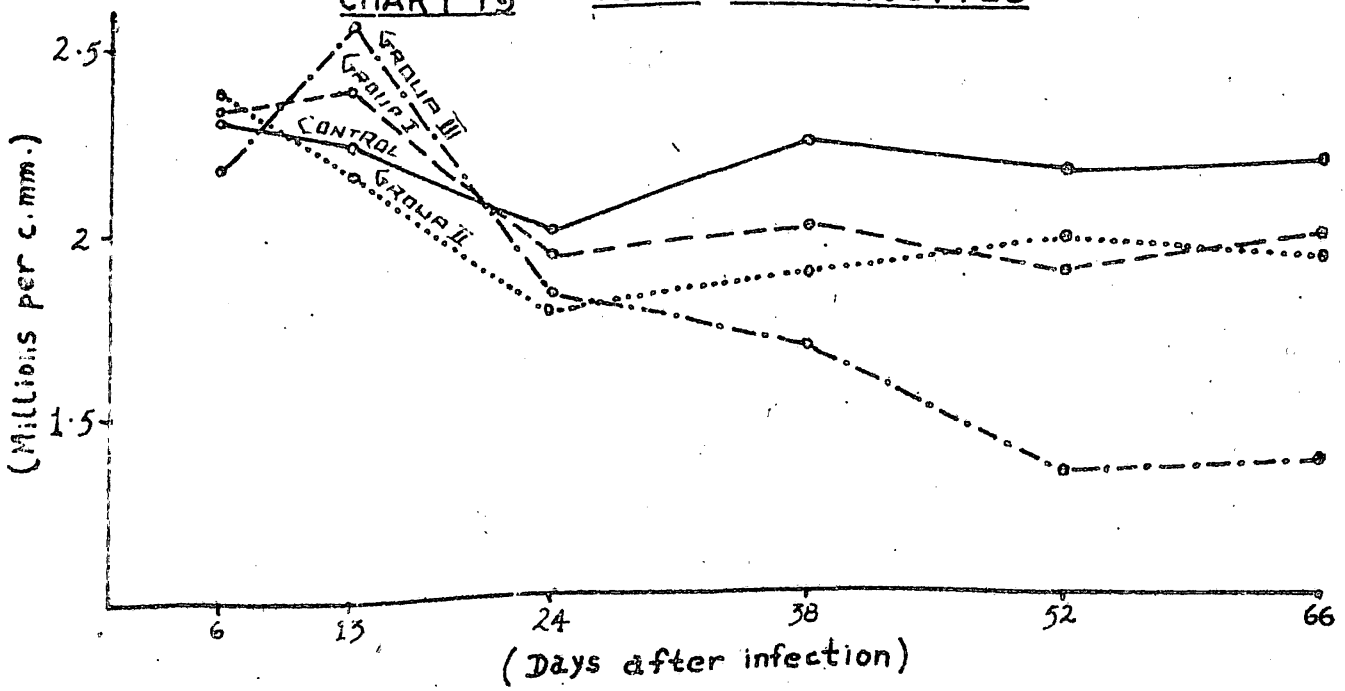
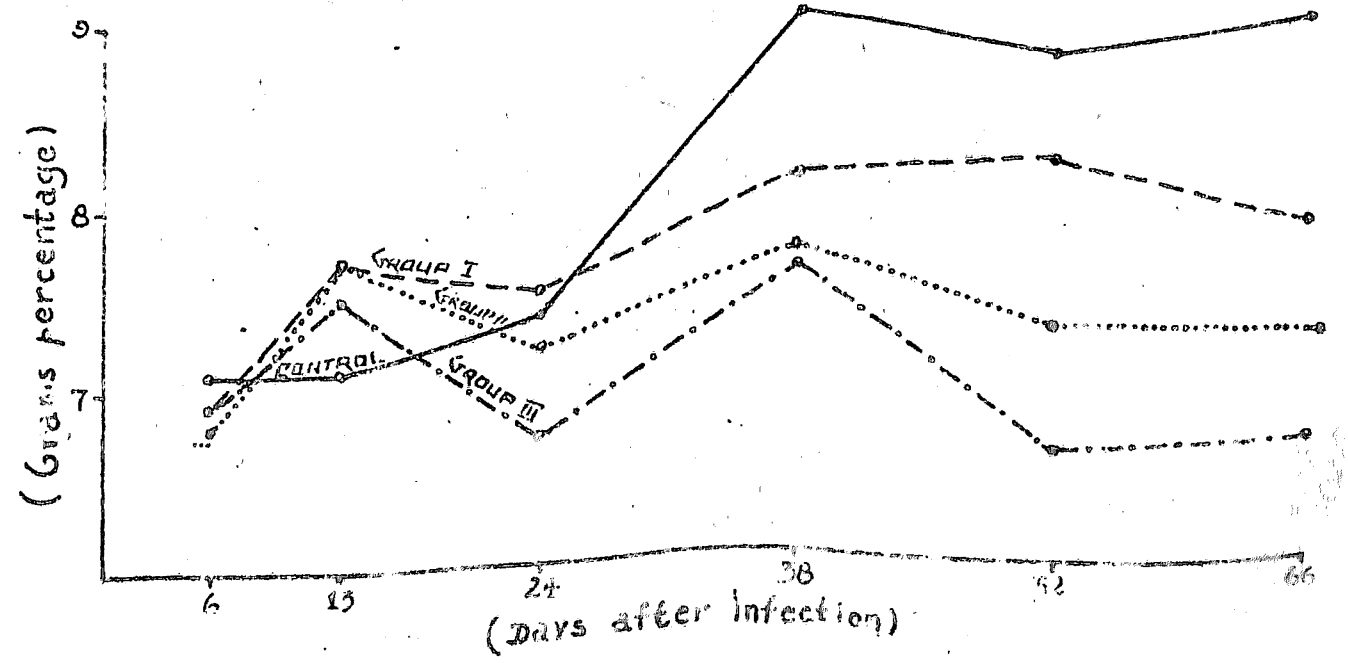


CHART 20 HAEMOGLOBIN



IN DISPECIFIC INFECTIONS WITH
T. MOHTEDAI & A. SPIRALIS

CHART-21 PACKED CELL VOLUME

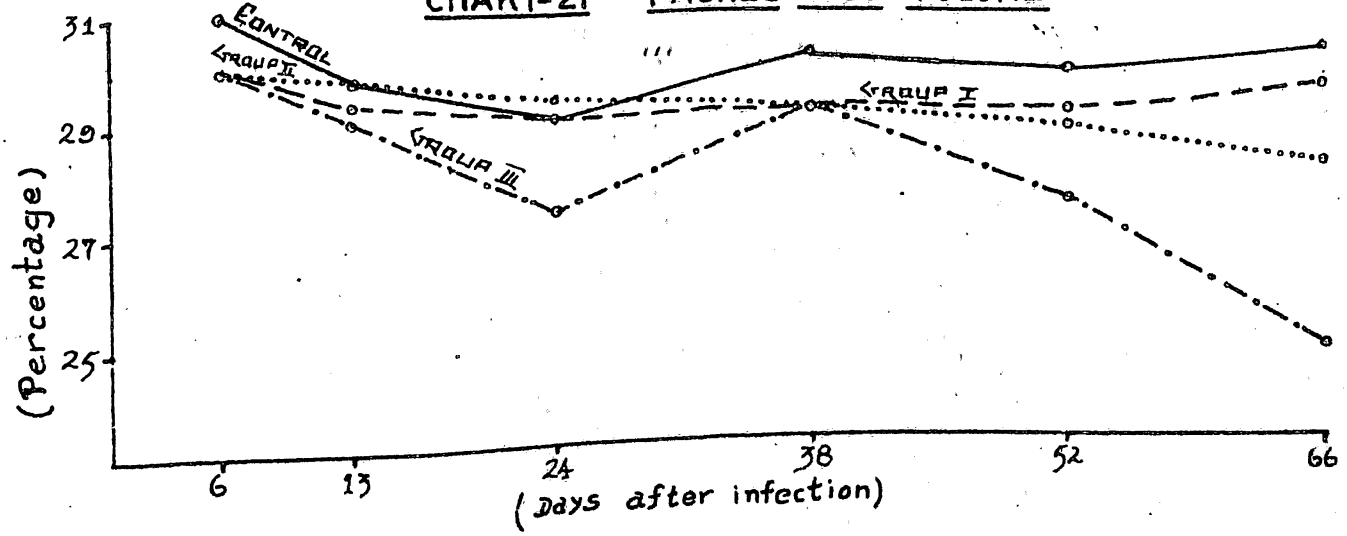
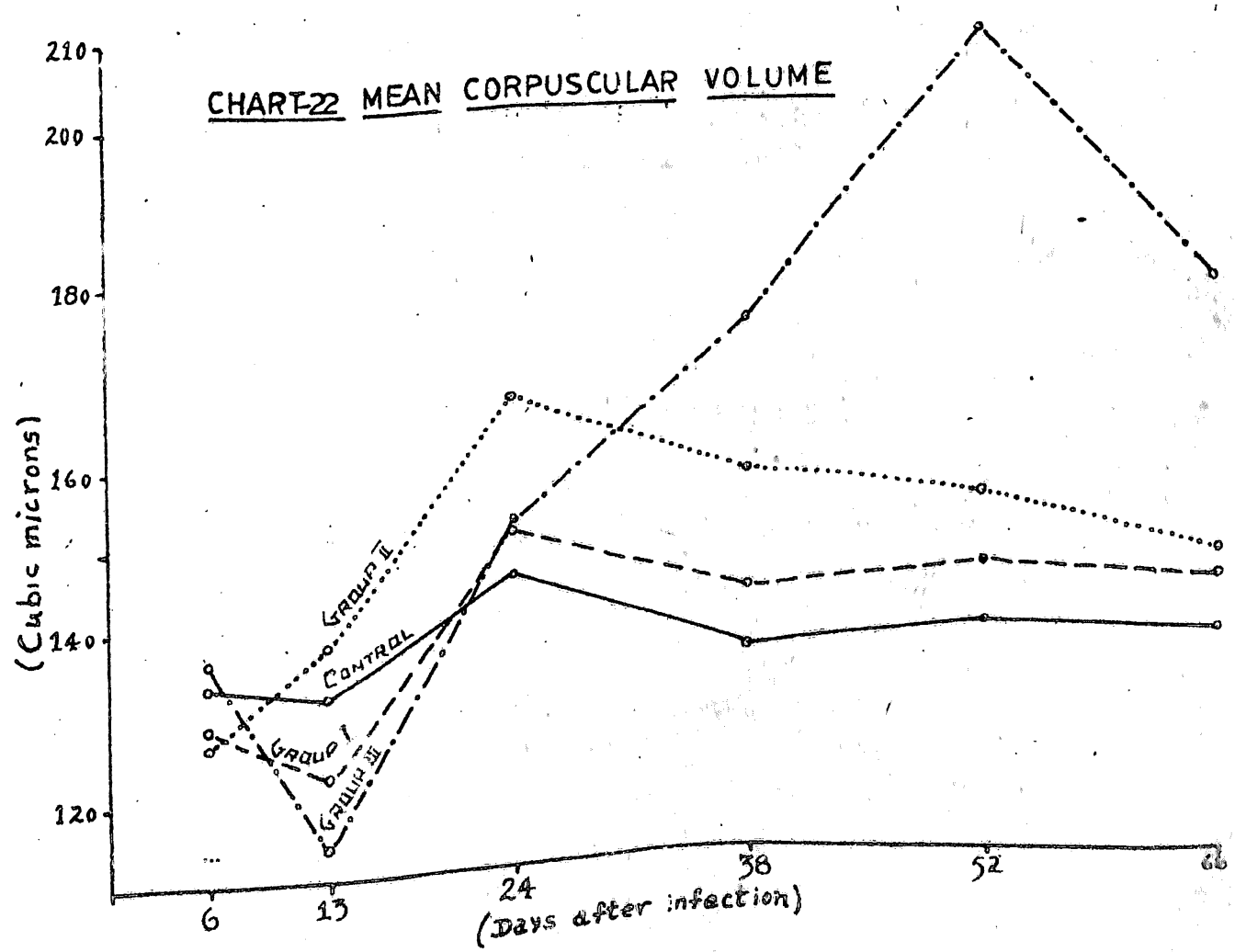


CHART-22 MEAN CORPUSCULAR VOLUME



IN DISPECIFIC INEECTIONS WITH
T. MOHTEDAI & A. SPIRALIS

CHART 23 TOTAL LEUCOCYTES

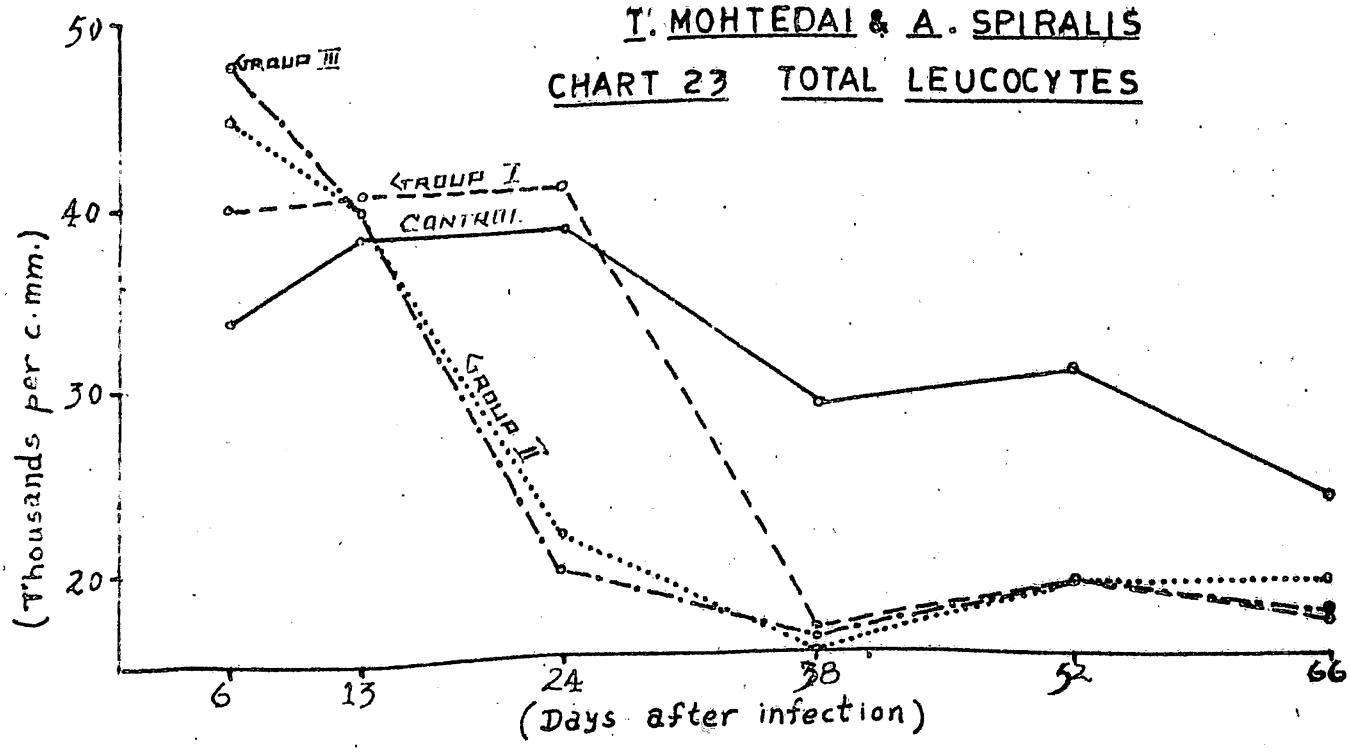
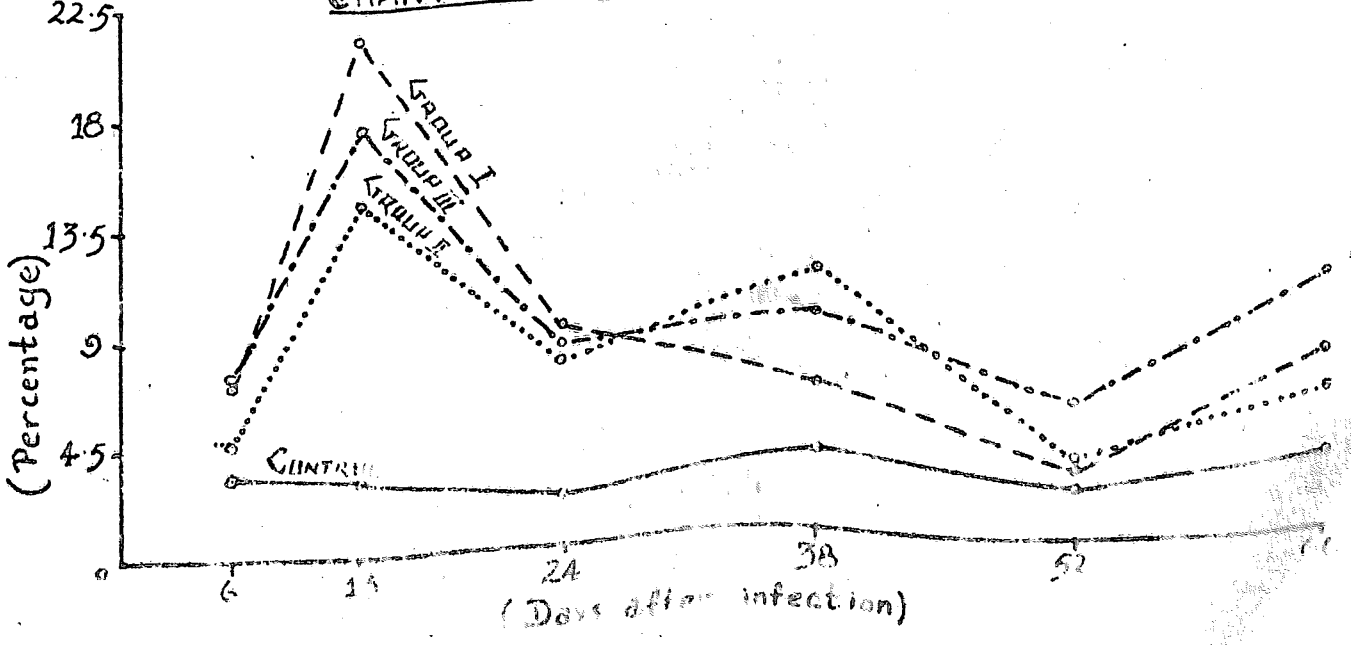


CHART-24 EOSINOPHILS



IN DISPECIFIC INFECTIONS WITH
T. MOHTEDAI & A. SPIRALIS

CHART 25 HETEROPHILS

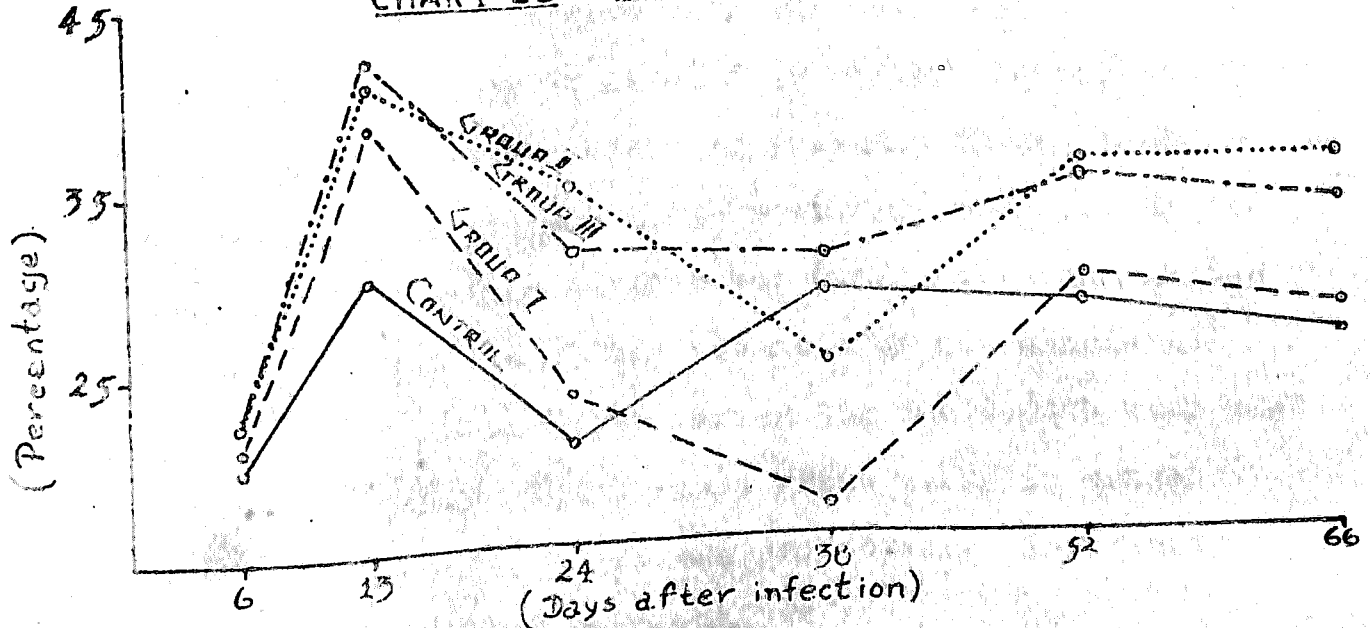
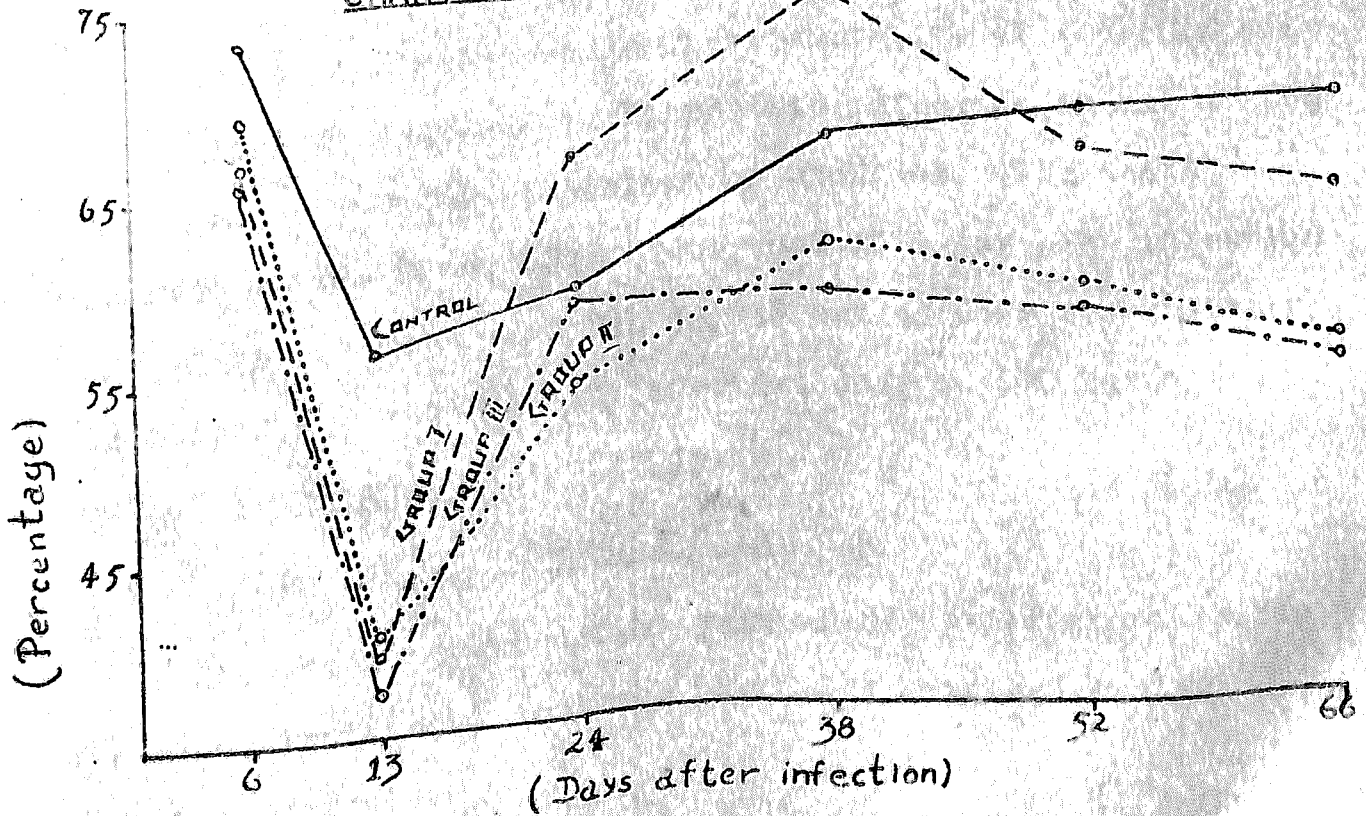


CHART 26 LYMPHOCYTES



CLINICAL SYMPTOMS

The affected birds were very dull with ruffled feathers and there was evidence of loss of appetite. During the first two weeks post infection a slight increase in the body temperature was noticed and the birds were very droopy. By the 5th week post infection anaemia was discernable and as the disease progressed, the birds became weak and emaciated. Mortality of chicks occurred during the 2nd to 4th week post infection. In Mayers there was a great delay in sexual maturity and reduced rate of egg production. Important details of infection were as follows :

MORTALITY

As has been mentioned earlier mortality of 4 chicks out of 12 (33.33%) occurred in dispecific infection in one of the experiments (for histopathological studies). The death occurred on the 15th, 20th, 24th, and 29th day post infection (age of the chicks being 29, 34, 38 and 43 days respectively). The chicks had received an infective dose of 500 juveniles each of *T. mohtedai* & *A. spiralis*.

WEIGHT GAIN

The mean overall weight gain of the experimental birds showed a significant decrease ($P < 0.01$) compared to the negative controls.

The decrease in respect of Group I (average number of worms established 64.66 of T.mohtedai and 71.66 of A.spiralis) was 36.39% ($P < 0.01$), in respect of Group II (average number of worms established 82 of T.mohtedai and 113.33 of A.spiralis) 37.52% ($P < 0.01$) and in respect of Group III (average number of worms established 124 of T.mohtedai and 244.66 of A.spiralis) 48.15% (P 0.01).

On a further analysis, Group I showed a decrease of 67.19%, 37.50%, 44.00% & 60.00% ($P < 0.01$) by the 5th, 6th, 7th & 10th week post infection respectively. Group II showed a decrease of 54.69%, 38.75%, 44.00%, 50.00% & 73.75% ($P < 0.01$) by the 5th, 6th, 7th, 8th & 10th week post infection respectively. Group III showed a decrease of 50.00% ($P < 0.05$), 37.66%, 76.56%, 42.50%, 53.34% ($P < 0.01$), 40.00% ($P < 0.05$) and 66.25% ($P < 0.01$) by the 2nd, 4th, 5th, 6th, 7th, 8th & 10th week post infection respectively.

Thus the weight gain of the experimental groups were found to be poor during the 5th to 10th week post infection. On the 10th week post infection the average live body weight was found to be 793 grams in Group I, 777 grams in Group II and 682 grams in Group III compared to the negative controls which showed an average live body weight of 1175 grams.

The data are represented in Chart 27.

The loss of income due to decreased body weight was calculated on the basis of the current price (Rs.6.50/Kg. of live body weight). Thus the loss was found to be Rs.2.50 per bird in Group I, Rs.2.60 per bird and Rs.3.20 per bird in groups II & III respectively when compared to the negative controls.

SEXUAL MATURITY AND INTENSITY OF LAYING

The age at first egg was taken as the attainment of sexual maturity. The control bird laid its first egg on the 131st day, while the bird in Group I gave the first egg on the 137th day (a delay of 6 days), in Group II on the 163rd day (a delay of 32 days) and in Group III on the 182nd day (a delay of 51 days).

In all the infected birds the egg production was very low due to lack of persistancy and/or due to exceptionally long pauses. During the observed period of 66 days, the total number of eggs laid by Group I (number of worms established, 70 T.mohtedai and 83 A.spiralis) was 42, by Group II (number of worms established, 106 T.mohtedai and 112 A.spiralis) was 11 and by Group III (number of worms established, 113 T.mohtedai and 191 A.spiralis) was only 2. Whereas the eggs produced by Group IV (negative control) was 53.

The egg records of experimental and control groups are presented in Table 40.

FEED EFFICIENCY

The quantity of feed in kilograms required to produce a dozen eggs was calculated for the female birds, since the strain of chicks used in the experiments (Mychix) were exclusively meant for egg production. The feed efficiency in Group I (average number of worms established, 70 T.mohtedai and 83 A.spiralis) was 1.99, in Group II (number of worms established, 106 T.mohtedai and 112 A.spiralis) 4.53 and in Group III (number of worms established, 113 T.mohtedai and 191 A.spiralis) 11.4. Whereas in respect of Group IV (negative controls) the feed efficiency was 1.51.

The feed efficiencies of the experimental groups were thus found to be significantly lower than the negative controls.

Calculating the loss of income on the basis of the current price of the feed (Rs.1.50/Kg.), Group I required 70 paise worth of feed more than the controls to produce a dozen of eggs while groups II & III needed Rs.4.50 and Rs.14.80 worth of feed respectively more than the negative controls.

PREPATENT PERIOD

The prepatent period of infection was calculated by noting the day in which the first egg of the parasites appeared in the droppings. The eggs of T.mohtedai were easily distinguished from those of the A.spiralis by the presence of

the small knob at one pole of the egg shell, by the shape & size of the egg and by the transverse rows of body spines at the head end of the larva as described by Sundaram (1971).

The prepatent period of T.mohtedai in Group I was found to be in from 47 to 49 days (average 47.66 days), in Group II, in from 47 to 48 days (average 47.66 days) and in Group III, in from 49 to 50 days (average 49.33 days).

The prepatent period of A.spiralis in Group I was in from 26 to 27 days (average 26.33 days); in Group II, in from 24 to 25 days (average 24.66 days) and in Group III, in from 24 to 25 days (average 24.66 days).

Thus the development of the parasites in dispecific infections was normal and without any mutual inhibitory effects.

The data are presented in Table 39.

PERCENTAGE OF ESTABLISHMENT

The number of worms developed in each bird was found out, to note the percentage of establishment in each group so as to correlate their population with the various parameters chosen to determine their pathogenicity.

In Group I (infected with 200 juveniles of T.mohtedai and 100 juveniles of A.spiralis), the average number of worms recovered was 64.66 (17.01% males and 82.99% females) of T.mohtedai and 71.66 (24.65% males and 75.35% females) of A.spiralis, thus giving an establishment percentage of 32.33

for T.mohtedai and 71.66 for A.spiralis.

In Group II (infected with 400 juveniles of T.mohtedai & 200 juveniles of A.spiralis), the average number of worms recovered was 82 (23.98% males and 76.02% females) of T.mohtedai and 113.33 (24.12% males and 75.88% females) of A.spiralis, thus giving a percentage of establishment of 20.50 and 56.66 respectively.

In Group III (infected with 600 juveniles of T.mohtedai and 400 juveniles of A.spiralis), the average number of worms recovered was 124 (16.40% males and 83.60% females) of T.mohtedai and 244.66 (36.78% males and 63.22% females) of A.spiralis, thus giving an establishment percentage of 20.66 and 60.16 respectively.

No worms were present in Group IV as it constituted the negative controls.

The data are presented in Table 39.

TABLE - 39 - Showing the details of prepatent period and percentage of establishment in dispecific infection with T. mohtedai and A. spiralis at three levels and the negative controls.

	Chick number	Number of infective juveniles given		Prepatent period in days		Number of worms developed						Percentage of establishment	
						Male		Female		Total			
		T.m.	A.s.	T.m.	A.s.	T.m.	A.s.	T.m.	A.s.	T.m.	A.s.	T.m.	A.s.
A	1	200	100	47	27	12	22	58	61	70	83	35.00%	83.00%
	2	200	100	47	26	14	14	38	48	52	62	26.00%	62.00%
	3	200	100	49	26	7	17	65	53	72	70	36.00%	70.00%
	TOTAL	600	300							194	215	32.33%	71.66%
B	4	400	200	47	24	24	25	82	87	106	112	26.50%	56.00%
	5	400	200	48	25	21	39	73	99	94	138	23.50%	69.00%
	6	400	200	48	25	14	18	32	72	46	90	11.50%	45.00%
	TOTAL	1200	600							246	340	20.50%	56.66%
C	7	600	400	49	25	19	65	94	126	113	191	18.80%	47.75%
	8	600	400	50	25	17	129	42	184	59	313	9.83%	78.25%
	9	600	400	49	24	27	76	173	154	2200	230	33.33%	57.50%
	TOTAL	1800	1200							372	734	20.66%	60.16%
D	10	NIL		All the birds remained negative									
	11												
	12												

A - GROUP I
B - GROUP II
C - GROUP III
D - GROUP IV

T.m. - Tetrameres mohtedai
A.s. - Acuaria spiralis

CHART-27 EFFECT ON WEIGHT GAIN IN DISPECIFIC INFECTIONS WITH T. MOHTEDAI & A. SPIRALIS

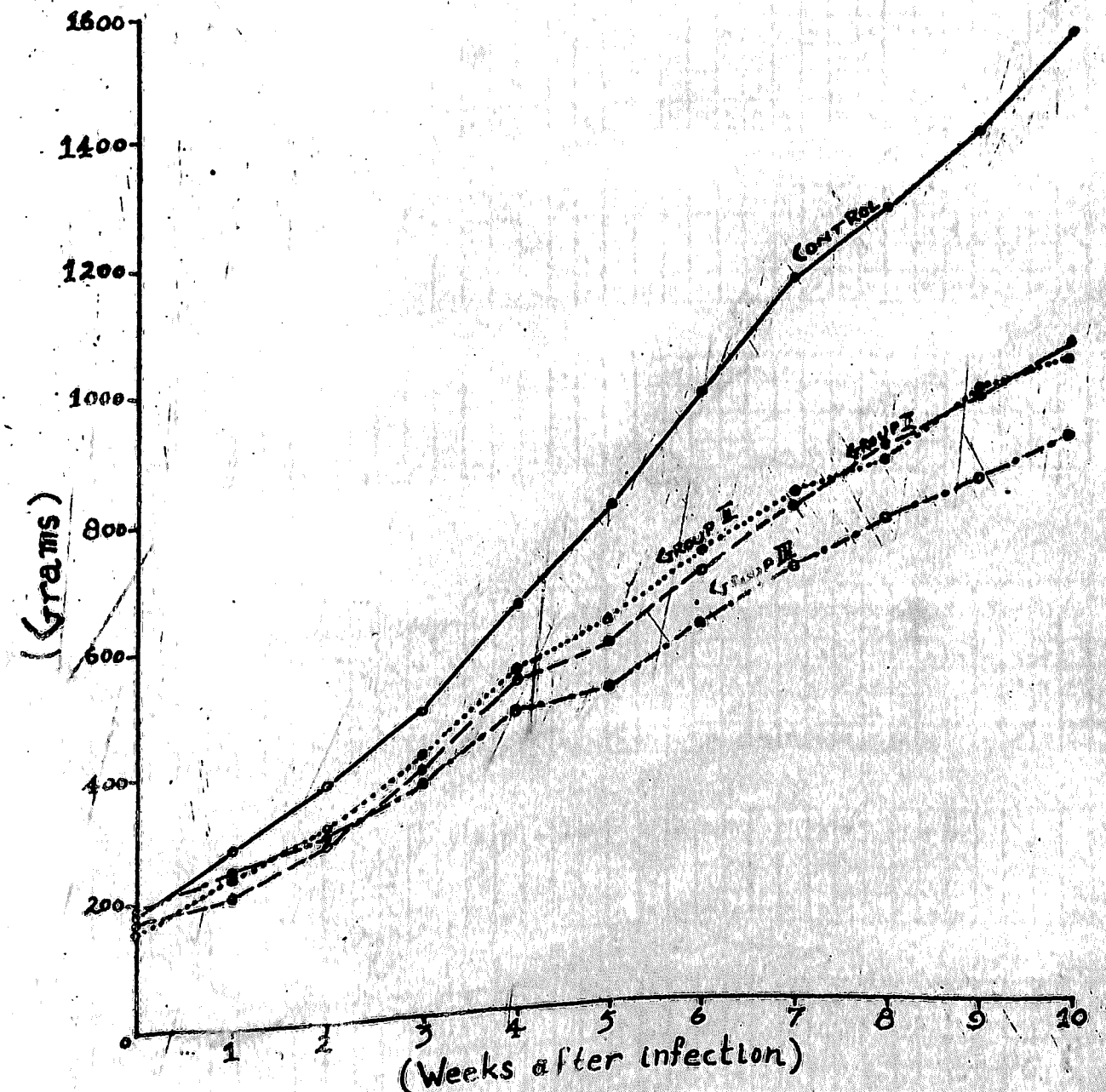
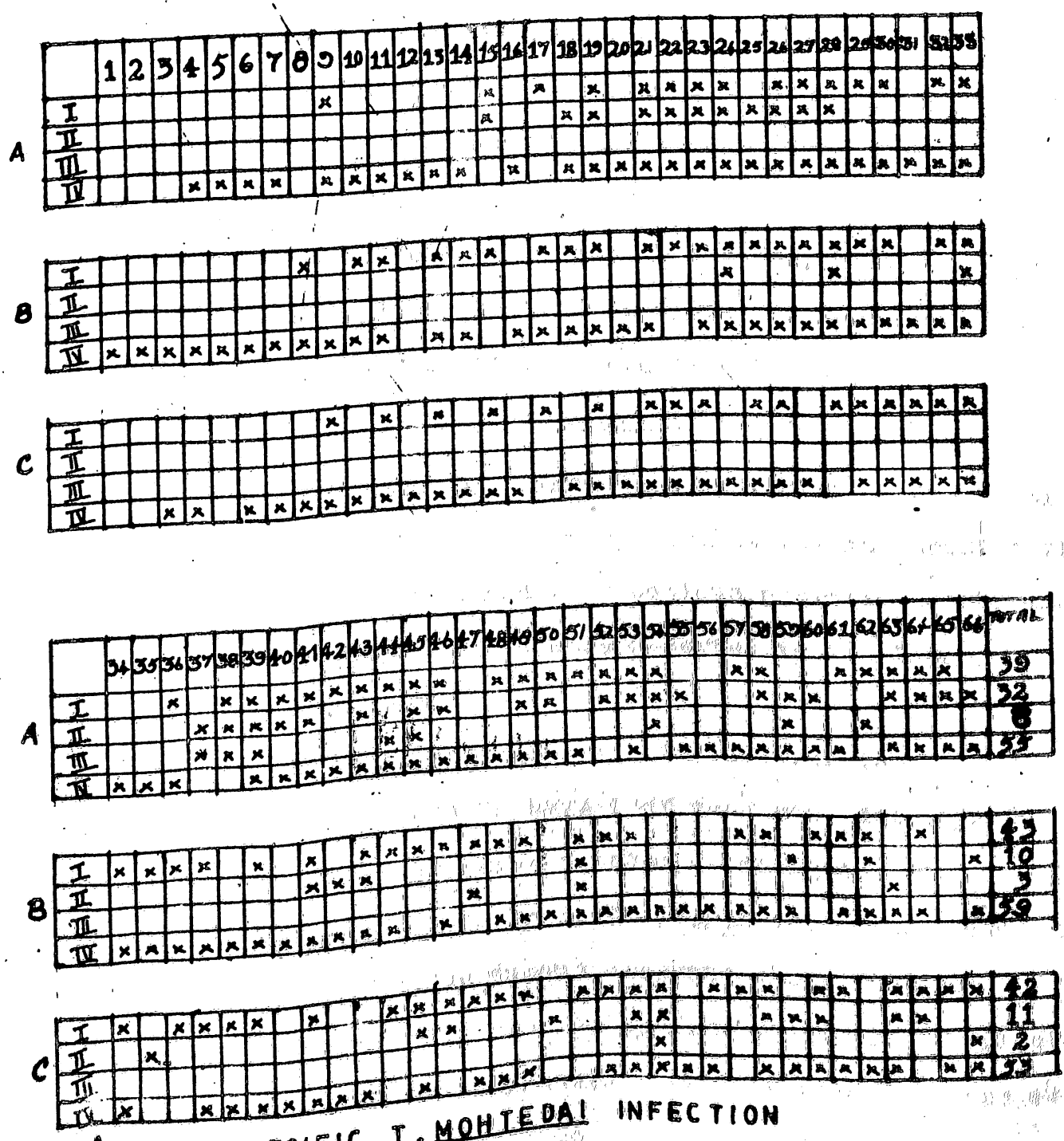


TABLE-40. EGG RECORD OF EXPERIMENTAL AND CONTROL BIRDS



A – MONOSPECIFIC I. MOHTEDAI INFECTION

B – MONOSPECIFIC A. SPIRALIS INFECTION

C – DISPECIFIC INFECTIONS WITH I. MOHTEDAI & A. SPIRALIS

PATHOLOGICAL CHANGES IN DISPECIFIC TETRAMERES MOHTEDAI & ACUARIA SPIRALIS INFECTIONS

Gross and microscopic lesions produced in the proventriculus by the various developmental stages of Tetrameres mohtedai and Acuaria spiralis in dispecific infections were studied after infecting a batch of chicks with 500 juveniles each of the parasites. Observations were made on the 2nd, 8th, 12th, 15th, 20th, 24th, 29th, 32nd, 36th, 40th, 50th and 100th day post infection. The changes noticed were as follows :

GROSS LESIONS

On the second day post infection no visible abnormality was noticed in the unopened proventriculus but on incision the lumen was found to be packed with copious necrotic debris and mucus. Numerous small ulcers were observed in the highly congested mucous membrane.

By the 12th day post infection the size of the proventriculus was found to have increased considerably. The serosa showed irregular white patches denoting the predilection sites of A. spiralis worms. On cutting open the organ, elevated areas were visible on the mucous membrane where numerous A. spiralis juveniles were seen to be firmly attached in clusters.

On the 20th day post infection the proventriculus was one and a half times the normal size and the white patches were more clearly seen on the serosa than on the 12th day . On

opening the organ the wall appeared thick and the lumen was found to be filled with straw coloured inflammatory exudate. The glandular openings were very prominant and were well raised above the surface. On section some of the glands showed juveniles of both the parasites within their lumina.

On the hundredth day post infection the proventriculus was three times the normal size and was almost spherical in shape with a hard texture. The oesophageal junction was sharply demarkated due to the spherical shape of the enlarged organ. On the serosa numerous black spots and pale areas were visible corresponding to the location of T.mohtedai and A.spiralis worms (Plate X Fig.1.). When cut opened the lumen of the proventriculus was found to have been nearly obliterated by the marked nodular growths from the mucous membrane due to Acuariasis. Most of the gland contained mature female T.mohtedai worms. Some of the glands had undergone liquifactive changes and appeared vacuolated.

MICROSCOPIC LESIONS

2nd day post infection

The proventriculus of the chick slaughtered on the second day post infection showed necrosis and damage to the lining epithelium of the tubular glands. Lamina propria in focal areas were completely destroyed with frank haemorrhagic spots. Numerous juveniles were seen embedded among the cellular exudate and some of the juveniles were found to have invaded the compound glands (Plate X Fig. 2).

8th day post infection

Moderate diffuse thickening of the lamina propria due to granulocytic infiltrating cells were noticed on the eigth day post infection. Most of the compound glands were packed with necrotic cellular inflammatory exudate and juveniles. Submucosa showed oedema and engorgement of blood vessels (Plate X Fig.3).

12th day post infection

On the 12th day post infection the lamina propria was very much thickened in focal areas due to lymphoid hyperplasia. The infiltrated granulocytes were mostly found packed at the periglandular region and submucosa. An increase in the connective tissue with necrotic foci were evident in the submucosa (Plate XI Fig.1). Juveniles of T.mohtedai within the compound glands caused pronounced histiocytic reaction. Juveniles of A.spiralis were seen embedded within the hyperplastic tubular glands causing marked mucosal changes. Thus lesions charecteristic of both Acuariasis and Tetramerosis were evident.

15th day post infection

On the 15th day post infection the lamina propria showed mild fibroplasia and an increase in the smooth muscle fibres. Histiocytic reaction was evident beneath the surface epithelium (Plate XI Fig.2). Focal areas in the lamina propria showed non-keratinizing squamous cell metaplasia.

20th to 24th day post infection

Changes noticed in the proventriculus on the 20th to 24th day post infection had the characteristics of a chronic inflammation as evidenced by the fibroplastic changes in the lamina propria and interglandular region. Cut sections of the juveniles of both T.mohtedai and A.spiralis were seen in the lumen of some of the compound glands on the 20th day post infection indicting that some of the latter have also entered the glands (Plate XI Fig.3). However, on the 24th day post infection, mature A.spiralis worms were found embedded in the mucous membrane of the proventriculus. Hypertrophy of the circular muscle fibres were observed in the muscular coat of the organ. Some of the compound glands contained immature female T.mohtedai worms in their lumina (Plate XI Fig.4).

29th to 32nd day post infection

On the 29th day post infection the compound glands were also found to be invaded by fibrous connective tissue and their lumina contained immature female and male T.mohtedai worms (Plate XII Fig.1). The A.spiralis worms attached to the mucous membrane of the organ induced marked necrotic changes. Pressure atrophy and degeneration of glandular acini of the compound glands were apparent on the 32nd day post infection (Plate XII Fig.2)

36th day post infection

On the 36th day post infection the lamina propria showed

fibro-adenamatoid growths characteristic of A. spiralis infection along with pressure atrophy of the compound glands due to female T. mohtedai worms.

40th to 50th day post infection

By 40th day post infection the lamina propria had completely sloughed off in some areas and the mucous membrane showed squamous cell metaplasia at other places. Most of the compound glands were filled with mature female T. mohtedai worms. Usually a single female worm was found in a gland but occasionally more than one worm was present in some of the glands. The glandular tissue had undergone marked atrophy with simultaneous reduction in the thickness of the wall of the glands. In some of the glands the cystic dialatation was very pronounced so that they appeared as mere pockets holding the parasites (Plate XII Fig.3).

100th day post infection

On the 100th day post infection severe chronic proventriculitis was observed with completely fibrosed lamina propria, submucosa and few of the compound glands. Pedunculated fibro-adenamatoid growths were evident in the lamina propria, wherever A. spiralis worms have burrowed deep into the mucous membrane (Plate XII Fig.4). Mature female T.mohtedai worms were found lodged within some of the compound glands. Vacuolated spaces representing the degenerated compound glands were also seen in some places.

PLATE X

Fig. 1. Proventriculus - Dispecific infections with T.mohtedai and A.spiralis .-
The organ is almost spherical having a mottled appearence. Black spots represent the glands affected by female T.mohtedai worms.

Fig. 2. Proventriculus - Dispecific infections with T.mohtedai and A.spiralis - 2nd day.
Lamina propria shows pronounced granulocytic reaction and disruption. Note the juvenile within the lumen of the compound gland (indicated by arrow). H & E X 20

Fig. 3. Proventriculus - Dispecific infections with T.mohtedai and A.spiralis 8th day - Note the greately thickened lamina propria due to the infiltrating cells. Juveniles are seen partially embedded in the mucous membrane.
H & E X 20

PLATE X

Fig. 1

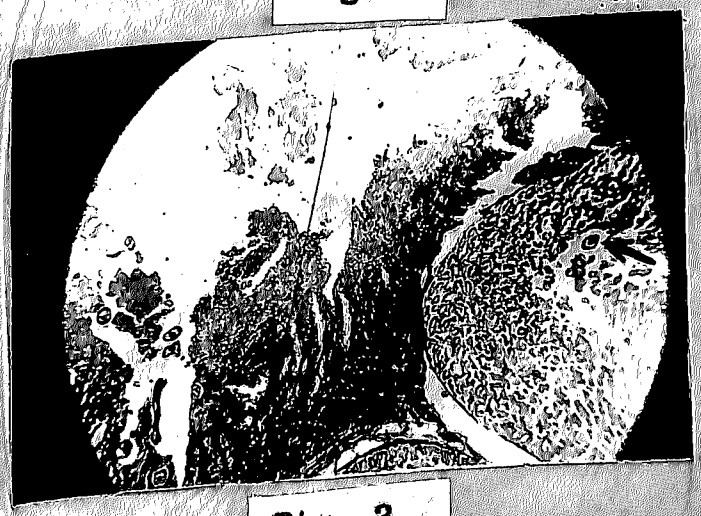


Fig. 2

Fig. 3

PLATE XI

Fig. 1. Proventriculus - Dispecific infections with T.mohtedai and A.spiralis 12th day - Note the diffuse lymphoid hyperplasia, granulocytic infiltration in the peri-glandular region and submucosa, necrotic areas in the lamina propria and the hyperplastic tubular glands.in which is seen embedded an A.spiralis worm (indicated by arrow) H & E X 100

Fig. 2. Proventriculus - Dispecific infections with T.mohtedai and A.spiralis 15th day - Note the mild fibroplasia, engorged blood vessel and increase in the smmeth muscle fibres. Inflammatory cells are seen beneath the surface epithelium. H & E X 100

Fig. 3. Proventriculus - Dispecific infections with T.mohtedai and A.spiralis 20th day - Note the A.spiralis juveniles within the compound gland. H & E X 100

Fig. 4. Proventriculus - Dispecific infections with T.mohtedai and A.spiralis 24th day - Lymphoid hyperplasia and fibroplasia are seen in the lamina propria. Mucous membrane severely damaged due to A.spiralis worms. One of the compound glands show pressure atrophy due to the female T.mohtedai worm. H & E X 100

Fig. 1

Fig. 2

Fig. 3

Fig. 4

PLATE XII

Fig. 1. Proventriculus - Dispecific infections with T.mohtedai and A.spiralis 29th day - Note the severe fibroplastic changes in the lamina propria and submucosa. Fibrous connective tissue invasion is seen in the compound glands also which carry the T.mohtedai worms. one gland contains both male (indicated by arrow) and female T,mohtedai worm inits lumen. An A.spiralis worm is seen embedded within the lamina propria. H.Azan X 100

Fig. 2. Proventriculus - Dispecific infections with T.mohtedai and A.spiralis 32nd day - Lumen of the proventriculus, showing severe damage to mucous membrane and the presence of A.spiralis. Note the pressure atrophy and flattened lining epithelium in the compound gland carrying the female T.mohtedai worm H & E X 100

Fig. 3. Proventriculus - Dispecific infections with T.mohtedai and A.spiralis 50th day - Note the very pronounced cystic dialatation of the compound gland containing a female T.mohtedai worm. Only a small portion of the glandular tissue remains. 1.- Gut of the female T.mohtedai worm. A.spiralis worms are seen embedded within the compleately damaged mucous membrane. H & E X 100

Fig. 4. Proventriculus - Dispecific infections with T.mohtedai and A.spiralis 100th day - Note the compleately denuded mucous membrane, severe fibrosis and the fibro-adenamatied growths in the lamina propria. H & E X 100

Fig. 1

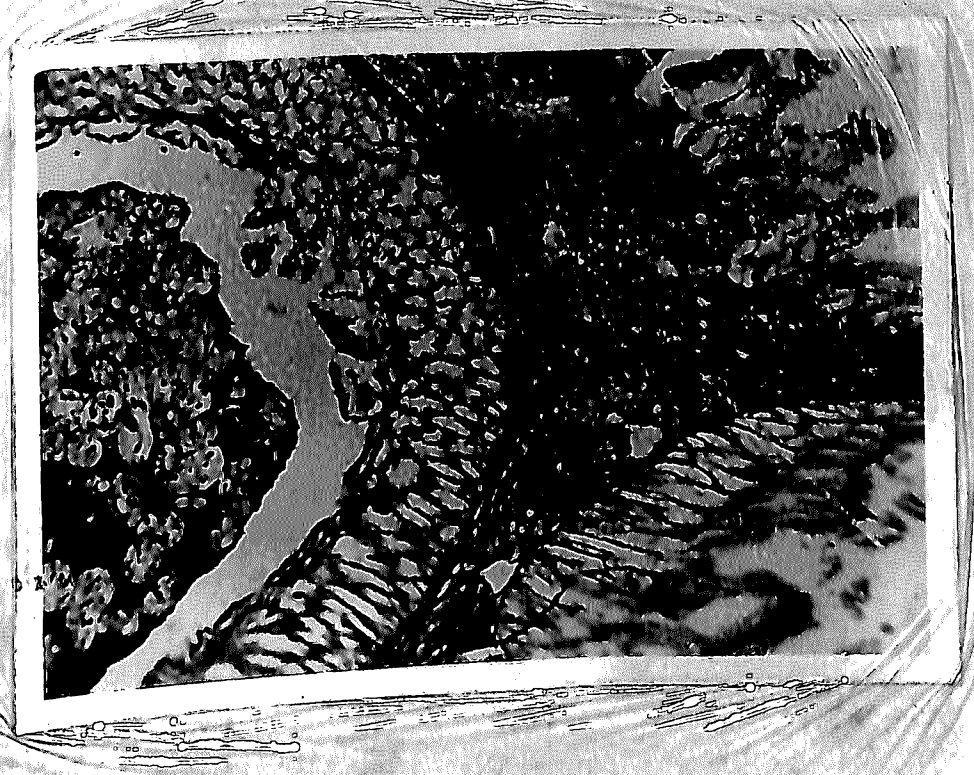
Fig. 2

Fig. 3

Fig. 4

PLATE XIII - Proventriculus - dispecific infections with T.mohtedai and A.spiralis 40th day - Note the highly flattened glandular tissue of the compound carrying the female T. mohtedai worm with cystic dialatation. The mucous membrane shows necrotic areas caused by A.spiralis worms. H & E × 70

PLATE XIII



three out of twelve chicks (25%) infected with 500 juveniles succumbed to the infection. Thus this forms a further evidence to the fact that A.spiralis may cause death of chicks in heavy infections. In the present study therefore the total mortality due to acuariasis was 7 out of 24 chicks (29.16%).

Birds with dispecific infections lost considerable amount of their body weight. The economic loss due to this has been calculated as Rs.3.20 per bird in heavy infections. In layers the sexual maturity was delayed for nearly 2 months and the rate of egg production was greatly lowered. It has been calculated that a heavily infected bird required Rs.14.80 worth of feed more than the control to produce a dozen eggs.

The histopathological changes in mixed infections presented the combined characteristics of Tetramerosis and Acuariasis as described earlier under monospecific infections of each. However, juveniles of T.mohtedai were found to invade the compound glands even by the 2nd day post infection (Plate X Fig.2). This early migration may be due to the increased population (1000 larvae) and small amount of surface area available in the mucosa of the proventriculus for attachment (being young chicks). Juveniles of A.spiralis were found within some of the compound glands on the 20th day post infection (Plate XI Fig.3), indicating that they too invade the glands if their population is considerable. The white patches on the serosa denoting the predilection sites of the

A. spiralis worms were evident in dispecific infections also from the 12th day post infection and this could be taken as a visible indication of Acuariasis even without cutting open the organ.

Anataraman & Chandrasekharan Nair (1955) while describing the histopathological lesions in natural T. mohtedai infections found that in some of the compound glands there is total disappearence of the glandular tissue leaving the lobules as empty spaces lined occasionally with a thin layer of epithelial cells and that there was absence of entire normal glandular secreation in other lobules. Similar changes were observed in the present study also in the effected glands on the 100th day post infection that is after the full establishment of the parasite. The fibro-adenamatoid growths caused by A. spiralis were seen as early as 36th day post infection.

Great economic loss due to a dual sublethal parasitic infection with coccidia, Ascaridia galli and Heterakis gallinae were reported by Patil et al. (1972). The present study also very clearly points out that a mixed infection with Tetrameres mohtedai and Acuaria spiralis can cause a considerable amount of economic loss to the Poultry industry.

SUMMARY

SUMMARY

Pathogenic effects of two commonly occurring spirurid worms of fowl viz. Tetrameres mohtedai and Acuaria spiralis were determined under controlled conditions. The studies were conducted in respect of monospecific and dispecific infections with the above parasites, at three levels of infection viz. low, medium and heavy. The parameters observed were haematological changes, weight gain, sexual maturity, intensity of laying, feed efficiency and histopathological changes.

In monospecific Tetrameres mohtedai infection :

1. The haematological changes consisted of a normocytic normochromic anaemia from 28th to 56th day post infection, followed by a macrocytic anaemia upto the observed period of 70 days post infection at all the three levels of infections. An eosinophilia and leucocytosis were seen from the 3rd to 28th day post infection in low and medium infections and from the 3rd to 42nd day post infection in heavy infection. A heterophilia and lymphocytopenia were observed from the 42nd day upto an observed period of 70 days post infection in the case of heavy infection. Leucopenia and monocytosis were evident on the 56th and 70th day post infection at all the three levels of infection.

2. The weight gain of the infected birds were found to be poor from the 5th week post infection and continued to be

at low level until the completion of the experiment (10th week post infection). The economic loss due to the decreased body weight was calculated to be Rs. 1.50 per bird in low infection, Rs. 1.70 per bird in medium infection and Rs. 2.25 per bird in heavy infection compared to the control bird during the observed period of 70 days.

3. In layers the sexual maturity was delayed by 5 days in low infection, 11 days in medium infection and 33 days in heavy infection compared to the control bird. The rate of egg production was also less in the infected birds. During an observed period of 66 days the total number of eggs laid were 43, 22, and 8 respectively in low, medium and heavy infections, whereas the control bird laid 55 eggs during thid period.

4. The feed efficiency of the infected birds were very low when compared to the negative controls. It was calculated that in low infection the female required Re.0.70 worth more feed to produce one dozen eggs and medium & heavy infections the birds required Re. 1.00 and Rs. 5.60 worth of feed more than the controls.

5. The prepatent period of infection was found to be in from 46 to 54 days (average 49 days) and was not influenced by the number of worms established.

6. The percentage of establishment of worms were found to be inversely proportionnal to the number of juveniles administered.

7. Gross pathological changes of the affected proventriculi included, petechial haemorrages in the mucous membrane on the 4th

day post infection; intense congestion with focal areas of erosion of the mucosa on the 12th day; dialated compound glands with immature female worms on the 25th day and greatly reduced lumen of the organ on the 55th day post infection.

8. The microscopic changes observed were severe granulocytic reaction on the 4th to 7th day post infection, lymphoid hyperplasia from the 9th to 12th day; plasma cell reaction on the 15th day; interglandular oedema and fibroplastic changes on the 18th to 25th day and pressure atrophy and cystic dialatation on the 32nd to 39th day post infection. The reparative process found on the 55th day post infection indicates, the development of a host-parasite balance.

9. The decrease in the live body weight and reduced rate of egg production seen in monospecific T. mohtedai infection therefore clearly indicate that the parasite is economically important.

In monospecific Acuaria spiralis infection :

10. The haematological changes consisted of, leucocytosis and eosinophilia on the 13th day post infection; leucopenia, heterophilia and lymphocytopenia from the 24th day post infection upto an observed period of 66 days post infection; a macrocytic hypochromic anaemia on the 52nd and 66th day and monocytosis on the 66th day post infection.

11. The weight gain of the infected birds was found to be poor from 7th week and continued to be at low level until

the completion of the experiment (10 weeks post infection). The economic loss due to the decreased body weight was calculated to be Rs. 1.97 per bird in low infection, Rs. 2.50 in medium infection and Rs. 2.75 per bird in heavy infection.

12. In layers the sexual maturity was delayed by 7 days in low infection, 23 days in medium infection and 46 days in heavy infection compared to the negative control. Consequently the rate of egg production was less in infected birds. During an observed period of 66 days the total number of eggs laid by the female bird with low infection was 43, with medium infection was 10 and with heavy infection was only 3, as compared to the 59 eggs laid by the negative control bird.

13. The feed efficiency of the infected birds were therefore very low. It was calculated that in low infection the female required Rs.0.65 worth of feed more than the control. Similarly Rs.6.15 and Rs.11.10 worth of more feed was required in cases of medium and heavy infections respectively.

14. The prepatent period of infection was found to be in from 24 to 29 days (average 26.22 days) and showed little variation at the three levels of infection.

15. A 25% mortality was observed in experimental chicks infected with 500 juveniles.

16. Gross pathological changes of the affected proventriculi included the appearence of an irregular whitish patch on the serosa by the 10th day post infection, corresponding to the location of the juveniles within the lumen of the organ.

congestion, ulceration and necrosis of mucous membrane were observed on the 16th day post infection. Small elevated areas were seen in the mucosa by the 22nd day in places where the juveniles were found attached in clusters. By 100th day post infection the whole of the mucous membrane was found to be studded with nodular growths.

17. The microscopic changes observed in the mucous membrane were of an acute inflammation with a granulocytic reaction and non-keratinizing squamous cell metaplastic changes by the 8th day post infection. Juveniles of A. spiralis were found within the compound glands on the 8th and 16th day post infection. Lymphoid hyperplasia was evident on the 12th and 14th day. The lesions became chronic from the 18th day and by 100th day pedunculated fibro-adenamatoid growths were found wherever the worms were attached.

In dispecific infections with Tetrameres mohtedai and Acuaria spiralis :

18. The haematological changes observed at all the three levels of infections were leucocytosis and eosinophilia on the 13th day post infection; leucopenia and heterophilia from the 24th day upto the observed period of 66 days after infection. A macrocytic hypochromic anaemia from the 52nd day until the completion of the experiment (66th day post infection) and a lymphocytopenia and monocytosis towards the 66th day post infection were also evident.

19. The weight gain of all the infected birds at all the three levels were found to be very poor from the 5th week to the observed period of 10 weeks post infection. The economic loss due to the decreased body weight was calculated to be Rs.2.50 per bird in low infection, Rs.2.60 per bird in medium infection and Rs.3.20 per bird in heavy infection compared to the negative controls.

20. In layers the sexual maturity was delayed by 6 days in low infection, 32 days in medium infection and 51 days in heavy infection compared to the negative control. The rate of egg production was also low. During an observed period of 66 days the total number of eggs laid by the female bird with low infection was 42. In medium and heavy infection the eggs laid were 11 and 2 respectively, as compared to 53 eggs laid by the control during the same period.

21. The feed efficiency of the infected birds were therefore very low at all the three levels. It was calculated that the female birds in low, medium and heavy infections required Rs.0.70, Rs.4.50 and Rs.14.80 worth of feed, more than the controls to produce a dozen eggs.

22. The prepatent period of infection was found to be in from 47 to 50 days (average 48.22 days) in the case of T.mohtedai and from 24 to 27 days (average 25.22 days) in the case of A.spiralis.

23. A 33.33% mortality was observed in experimental chicks infected with 500 juveniles each of A.spiralis and T.mohtedai.

24. Gross pathological changes in the affected proventriculi included petechiel haemorrhage in the highly congested mucous membrane on the 2nd day post infection and irregular white patches on the serosa charecteristic of the A. spiralis infection, on the 12th day post infection. T. mohtedai worms were found in most of the compound glands on the 100th day post infection and the mucosa showed nodular growths charecteristic of A. spiralis infection. Some of the glands showed liquifactive changes.

25. The microscopic changes observed were an acute inflammation of the mucous membrane with severe granulocytic reaction during the early stages followed by a chronic change involving almost the entire organ from the 24th day post infection. Juveniles of A. spiralis were also found within the compound glands on the 20th day. Fibro-adenamatoid growths charecteristic of A. spiralis infection were evident on the 36th day and pressure atrophy with cystic dialatation of the compound glands were evident from 40th day post infection due to the female T. mohtedai worms. The pathological picture was therefore a combination of the changes seen in the monospecific infections.

26. The results obtained in the present study indicate that both Tetrameres mohtedai and Acuaria spiralis are very pathogenic to their host viz. the fowl and are therefore economically important. The results also show that the loss due to morbidity of infection was more than those due to mortality.

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STUDIES ON THE

PATHOGENICITY OF TETRAMERES MOHTEDAI AND ACUARIA SPIRALIS OF FOWLS

By

RAMASWAMY K.

ABSTRACT OF A THESIS

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ABSTRACT

Controlled experiments were conducted at three levels of monospecific and dispecific infections with two commonly occurring spirurid worms of fowls viz. *Tetrameres mohtedai* and *Acuaria spiralis*.

The blood studies conducted in monospecific *T. mohtedai* infection revealed a normocytic normochromic anaemia and eosinophilia during the initial phase and later a macrocytic anaemia, heterophilia and slight monocytosis by 66th day post infection. The infected birds lost considerable amount of their body weight. In layers there was a great delay in sexual maturity and poor feed efficiency as indicated by greatly reduced egg production. The percentage of establishment of worms in these experiments were found to be inversely proportional to the number of juveniles administered. The pathological changes in the affected proventriculi indicated an acute inflammation during the invasive phase of juveniles, followed by a chronic reaction which by 55th day post infection was well established indicating the development of a host - parasite balance towards the later stages of infection.

Among the blood changes in monospecific *A. spiralis* infection, eosinophilia was evident initially followed by a marked leucopenia and development of a macrocytic hypochromic anaemia. Other effects due to parasitism were, delayed maturity, reduced rate of egg production, poor feed efficiency,

loss of weight, emaciation and a mortality rate of 25% in chicks. The gross pathological changes in Acuariasis included the appearance of an irregular whitish patch on the serosa and formation of nodular growths in the mucosa which in heavy infections occupy the whole of the mucous membrane. Juveniles of A. spiralis were found to invade the compound glands in very heavy infections. The microscopic changes indicate a severe acute inflammation during the initial stages. Later as the disease became chronic pedunculated fibro-adenamatoid growths were evident on the mucous membrane.

In dispecific infections with T. mohtedai and A. spiralis the haematological changes observed were eosinophilia during the early stages followed by a leucopenia and heterophilia. As the disease progressed a macrocytic hypochromic anaemia developed. There was a great decrease in the live body weight of the infected birds. A mortality of 33.33% were observed in chicks with heavy infection. In layers the sexual maturity was delayed and egg production was greatly suppressed with consequent poor feed utilization. The gross and microscopic changes found in the affected proventriculi indicated a combination of lesions observed under monospecific infections of both the parasites.

The results obtained show that the loss due to morbidity (mainly as a result of decreased body weight and reduced rate of egg production) was considerable. These facts indicate that both T. mohtedai and A. spiralis are economically important.