PATHOGENICITY OF ACUARIA HAMULOSA TO CHICKEN

BY

Y. GANESWARA RAO

ABSTRACT OF A THESIS

Submitted in partial fulfilment of the requirement for the degree

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences
Kerala Agricultural University

Department of Parasitology
COLLEGE OF VETERINARY & ANIMAL SCIENCES
MANNUTHY - TRICHUR

1980

D E C L A R A T I O N

I hereby declare that this thesis entitled "PATHOGENICITY OF ACUARIA HAMULOSA TO CHICKEN" is a bonafide 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.

Mannuthy
14-11-80.

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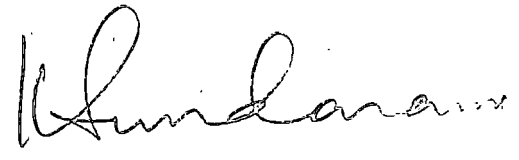
14-11-80.

C E R T I F I C A T E

Certified that this thesis entitled "PATHOGENICITY OF ACUARIA HAMULOSA TO CHICKEN" is a bonafide record of research work done independently by Shri.Y.Ganeswara Rao 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.



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Mannuthy,

IN EVER MEMORY OF
MY SISTER

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INTRODUCTION

INTRODUCTION

Rearing of chicken to supplement the family income is widely practised by many house holds in the villages and small towns all over the State of Kerala. The number of birds held by average farmers are few, but nevertheless, on an aggregate, substantial quantity of eggs are available in the State even for export to other parts of India. Almost all the farmers rear birds under backyard system. The birds have therefore ample opportunities to become infected with parasites having indirect life cycles, like spirurid worms, the incidence of which is proved to be high in Kerala.

For the reason that parasitic diseases are neither spectacular in their effect on their host, nor do they cause high mortality, a poultry grower may not be aware of their presence. Frequently the major symptom associated with parasitism is that of general unthriftness, owing to slow and protracted course which goes undetected more often, and an average farmer fails to notice the ill effects at the

early stage itself. For a successful poultry farming it is therefore imperative that a proper assessment of damage caused by parasites is understood, so that timely preventive measures could be taken. The climatic conditions and the natural fauna of intermediate hosts are congenial in the State of Kerala for a perpetual incidence of Spirurid nematodes.

While considerable information is available on most common nematodes like Ascaridia galli, Heterakis gallinae, there is paucity of information on the Spirurid group of parasites. Previous studies conducted in the State has shown that five species of Spirurid parasites are common, which are, Tetrameres mohtedai, Acuaria spiralis, Acuaria hamulosa, Gongylonema ingluvicola and Oxyspirura mansoni. The first two parasites inhabit the proventriculus, the third, the gizzard, the fourth, the wall of the crop and the fifth, the conjunctival sac. Though sporadic reports on the symptoms and histopathological lesions caused by some of these spirurid parasites in natural cases of infections have been reported, accurate assessment of their pathogenicity

has not been done, which was mainly due to the incomplete knowledge on their life cycles and range of intermediate hosts. Work conducted in the State has elucidated the life cycles of these parasites, which enables one to undertake studies on their pathogenicity through controlled experiments. Thus the pathogenicity of Acuaria spiralis and Tetrameres mohtedai has been determined accurately. It is only through such studies the relative importance of the parasite to the poultry industry of the State could be properly understood.

OBJECTIVE AND SCOPE OF PRESENT STUDY

The objective of the present study was, therefore to assess the pathogenicity of the gizzard-worm of chicken, namely Acuaria hamulosa, under controlled conditions.

The present investigation was carried out in two breeds of chicken with Acuaria hamulosa infection. The breeds chosen were, the White Leghorn (an egg strain) and the White Plymouth Rock (a broiler strain). The White Leghorn chicken were infected at two levels (mild and heavy infections) and the White Plymouth Rock were infected at one level (heavy infection) of Acuaria hamulosa.

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

REVIEW OF LITERATURE

Occurrence of Acuaria hamulosa was recorded for the first time in India by Srivastava (1939) in soft nodules in the musculature of the gizzard of a fowl received from Delhi, and later he recovered the worms from two other fowls stationed at Muktesswer. Chatterji (1939) recorded its occurrence in India and Burma in nine fowls out of fifty examined at Rangoon and Suburbs (Tamwe, Kemmendine, Kanayut, Insein). Subsequently, the occurrence of this parasite was reported by Khalerao and Rao (1944) in Utter Pradesh; Mudaliar and Alwar (1947) from Madras; Dixit (1952) from Bombay; Sundaram et al. (1962) from Kerala State; Endrejat (1964) from Assam; Shastri et al. (1974) in Marathwada region of Maharastra State, and Bali and Kalra (1975) from Punjab State. Sundaram (1971) had elucidated in detail the life cycle of Acuaria hamulosa, giving the morphology of the parasite both, within the grasshoppers (intermediate hosts) and in fowls.

According to Levine (1969), A.hamulosa occurs in the gizzards of fowls, turkeys and pheasants. Bravo (1949) has

given the description of the parasite from the proventriculus of Gallus gallus domesticus in Chinpas. Acuaria hamulosa has also been reported from the Chinese ring-necked pheasant, Phasianus colchicus torquatus (Schwart and Schwart, 1951).

Alicata (1938) has given the life history and mode of transmission of the parasite to chickens, in Hawai. Krehnert (1953) recorded a severe out break in a flock in some 60 White Leghorns which led to many deaths in 1949, in Russia. Manuel (1966) has found the presence of A.hamulosa in the gizzard of White Leghorn layers in a poultry house of the Alabang Stock Farm of the Bureau of Animal Industry, Muntinlupa, of Philippines. Hodasi (1969) reported its presence in native and introduced fowls in Ghana.

Nath and Pande (1963) and Soulsby (1965) have observed significant pathological changes in the gizzard due to infection with A.hamulosa.

The pathogenic effects of A.hamulosa have also been studied by other workers such as, Conklin (1932); Coppini (1938); Srivastava (1939); Wehr and Christensen (1942);

Wehr (1943); Casarosa (1950); Morgan and Hawkins (1953); Bochkov (1955); Gardiner (1956); Deo (1964); Soulsby (1968); Levine (1968); and Alicata (1969). Mason et al. (1978) has recorded the death of a caged-finch due to Acuaria Skrjabini in Newzealand.

The pathogenic effects of gizzard-worms of other birds were reported by different authors. Thus, Ullrich (1932), Czaplinki et al. (1956), Soulsby (1965), and Enigk et al. (1969) studied the pathogenic effect of Amidostomum anseris in geese. Mac Neill (1970) reported the pathological changes due to Amidostomum anseris in wild swans and golden eye ducks. Certain details of pathogenicity of Amidostomum Skrjabini of ducks have been studied by Dubey and Pande (1951) and Chandrasekharan (1967). Chandrasekharan (1977) assessed the pathogenicity of Amidostomum Skrjabini and Epomidostomum uncinatum under controlled experimental conditions in ducks. Ramaswamy (1977) has elucidated the detailed pathological studies on the spirurid worms of poultry, namely Tetrameres mohtedai and Acuaria spiralis under controlled conditions in fowls.

Hwang (1964) has studies the haemogram of turkey poults experimentally infected with Syngamus trachea. Kaushik and Sen (1978) have studied the leucocytic response in chicks experimentally infected with Ascaridia galli. The present investigation appears to be the first attempt to determine the pathogenicity of Acuaria hamulosa under controlled conditions.

MATERIALS AND METHODS

MATERIALS AND METHODS

COLLECTION OF WORMS

Worms for the present study were collected from infected gizzards of exotic and desi fowls, slaughtered for table purposes at the local hotels.

COLLECTION, MAINTANANCE, AND ARTIFICIAL INFECTION OF INTERMEDIATE HOSTS

Grasshoppers of the species Spathosternum prasiniferum, Oxya nitidula and Ducitia japonica caught from the paddy fields and fodder plots of the Kerala Agricultural University, Mannuthy Farm, were used as intermediate hosts for A.hamulosa.

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

COLLECTION OF INFECTIVE LARVAE

The encysted third stage infective larvae of A.hamulosa were harvested from grasshoppers on the 27th day after infection. The larvae were counted and transferred to fresh physiological saline solution. The experimental birds were infected with the larvae, within 2 hours

after dissection of the intermediate hosts.

INFECTING THE FINAL HOST

Counted numbers of infective larvae were administered directly into the crop of the chicks using a pasteur pipette.

PATHOLOGICAL STUDIES

To study the pathogenic effects of the parasite 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 - 0.4 gm per ml. of blood) as the anticoagulent. The blood samples were collected on the 14th, 29th, 36th, 43rd 50th, 60th and 70th day post infection, which corresponded to the different stages in the life cycle of the parasite.

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

The haemoglobin percentage in the present study was estimated by Cyanmethaemoglobin Method (Wintrobe, 1961) using Erma Spectrophotometer, with Drabkin's solution (Sodium Bicarbonate 1 gm, Potassium ferricyanide 200mg, Potassium cyanide 50 mg, Distilled water upto 1000 ml.) as reagent. Standard curve (Chart-1) was plotted with normal blood from healthy adult chicken using a direct reading Erma Electronic Haemo Photometer. Routine analysis for haemoglobin was carried out using the Erma Spectrophotometer.

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

Mean corpuscular volume, mean corpuscular haemoglobin, 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 and Sekiya (Nambiar, 1961) and by Wright-Leishman (Schalm, 1971). One hundred cells were counted from each smear and the average of two such counts was calculated.

The weight of the birds was 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 30 days.

All the values obtained were statistically analysed by two way classification with multiple and equal number of observations per cell.

To study the gross and histopathological changes produced by the various developmental stages of the parasite, in the host, the infected birds were autopsied on the

2nd, 6th, 10th, 14th, 18th, 22nd, 29th, 36th, 43rd, 50th, 60th and 70th day post infection. 10 per cent formalin solution was used as a fixative. Tissue were embaded in paraffin and 5 to 7 μ sections were cut. The sections were stained by Lillie Mayer's Haemotoxylin stain.

PLAN OF EXPERIMENT

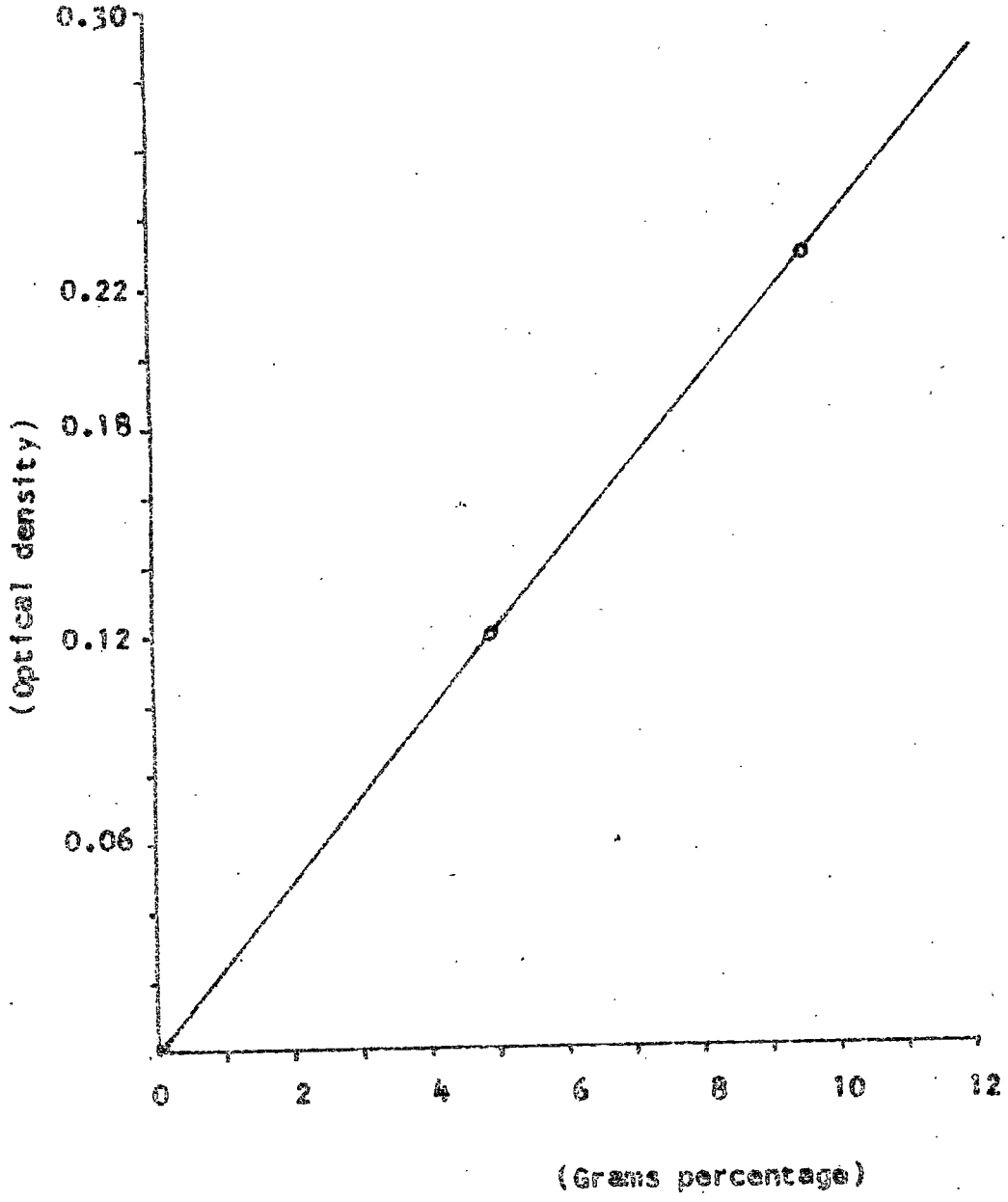
Pathogenicity was determined in White Leghorn and White Plymouth Rock breeds of chicken with A.hamulosa.

For haematological studies 12 White Leghorn chicks (9 males and 3 females) and 6 White Plymouth Rock chicks (all male), procured from University Poultry Farm, Mannuthy, were wing banded at 2 weeks of age and were seperated into 3 equal groups of 4 birds each (3 males and 1 female) in case of White Leghorn chicks and 2 groups of 3 birds each in case of White Plymouth Rock by random selection.

In case of White Leghorn chicks, the first and second grou-p, were infected with 100 and 400 larvae respectively at the age of 4 weeks; and the third group was kept as uninfected negative controls. And in the case of White Plymouth Rock chicks, the first group was infected with 400 larvae at the age of 3 weeks and the second group was kept as uninfected negative controls.

For histopathological studies 12 White Leghorn male chicks procured from the University Poultry Farm, Mannuthy, were wing banded at the age of two weeks and were infested with 200 larvae at the age of 4 weeks.

CHART-1 HAEMOGLOBIN STANDARD CURVE



RESULTS

PATHOGENICITY OF ACUARIA HAMULOSA TO WHITE LEGHORN

HAEMATOLOGICAL OBSERVATIONS

To study the haematological changes, blood samples were collected on the 14th (corresponding to the third stage), 29th, 36th and 43rd (corresponding to the fourth stage), 50th and 60th (young adult stage) and 70th day (corresponding to the mature adult) post infection.

The changes observed were as follows.

TOTAL ERYTHROCYTES

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

The observed average decrease of the erythrocyte count, in respect of Group I and Group II was 14.02% and 10.85% (not significant), respectively.

However, on further analysis the Group I (average number of worms established 47.50) showed a decrease of 18.04% ($P < 0.05$); 29.20% ($P < 0.01$); 18.39% (not significant)

43rd day (late fourth stage), which was continued till the 70th day (almost mature stage). Whereas the Group II (average number of worms established 36.25) also showed a slight reduction in the haemoglobin level on the 36th day (mid-fourth stage), which however, began to rise gradually from the 43rd day (late fourth stage) and reached the normal level by the 60th day (nearing maturity) as compared to the negative controls.

The data are presented in the table 2 and are graphically represented in the chart 3.

PACKED CELL VOLUME

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

However, the Group I (average number of worms established 47.50) showed a slight rise starting from the 29th day (early fourth stage) which was continued upto the 43rd day (late fourth stage) post infection. Thereafter, from the 50th day (young adult stage) it started

declining, which trend continued upto the 70th day (almost mature stage). Whereas the Group II (average number of worms established 36.25) showed no notable difference as compared to the negative controls.

The data are presented in the table 3 and are graphically represented in the chart 4.

MEAN CORPUSCULAR VOLUME

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

The increase in respect of the Group I (average number of worms established 47.50) was 15.64% (not significant). However, on a further analysis, the Group I showed an increase of 17.51% (P 0.05); 40.46% ($P<0.01$) and 26.68% ($P < 0.01$) on the 14th (late third stage) 29th (early fourth stage) and on 43rd day (late fourth stage) respectively. But from the 43rd day onwards it started declining and showed a slight fall on the 60th day (nearing maturation) and a slight rise

on the 70th day as compared to the negative controls.

The increase in respect of the Group II (average number of worms established 36.25) was 23.11% ($P < 0.01$) and 19.13% ($P < 0.05$) on the 29th (early fourth stage) and 70th day (almost mature stage) respectively. However, from the 36th day (mid-fourth stage) onwards it started declining and reached almost normal level on the 43rd day (late fourth stage), and from the 50th day (young adult stage) once again started rising from the 60th day which was continued upto the 70th day (almost mature stage).

The data are presented in the table 4 and are graphically represented in the chart 5.

MEAN CORPUSCULAR HAEMOGLOBIN

The average mean corpuscular haemoglobin values of the entire experimental groups showed no significant difference from those of the negative controls.

On further analysis, however, the Group I (average number of worms established 47.50) showed an increase

of 18.42% (not significant) and 48.40% ($P < 0.01$) on the 14th and 29th day respectively. But from the 29th day (early fourth stage) onwards the values commenced to decline, which by the 36th day (mid-fourth stage) reached a minimum. Thereafter, a slight rise was noticed between the period of 43rd day to the 50th day post infection. However, this slight rise in mean corpuscular haemoglobin values commenced to gradually drop upto the end of the present observation (70 days). The increase in respect of the Group II (average number of worms established 36.25) was 49.49% ($P < 0.01$) on the 29th day (early fourth stage). But thereafter, on the 36th (mid-fourth stage) and 43rd day (late fourth stage) it showed a slight reduction in the mean corpuscular haemoglobin value, which was almost normal on the 50th day as compared to the negative controls. From the 60th day it showed once again a rise which was continued upto the 70th day (almost mature stage).

The data are presented in the table 5.

MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION

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

The decrease in respect of the Group I (average number of worms established 47.50) was 12.37% ($P < 0.05$); 14.58% ($P < 0.05$) and 14.53% ($P < 0.05$) on the 36th (mid-fourth stage) 43rd (late fourth stage) and 50th day (young adult stage) respectively. The decrease in respect of the Group II (average number of worms established 36.25) was 11.96% ($P < 0.05$) on the 36th day.

The data are presented in the table 6 and are graphically represented in the chart 6.

TOTAL LEUCOCYTES

The average leucocyte count of the entire experimental groups was significantly higher than the negative controls ($P < 0.05$).

The increase in respect of Group I (average number of worms established 47.50) was 19.01% (not significant)

and in respect of Group II (average number of worms established 36.25) was 7.65% (Not significant).

On further analysis, the leucocyte count in the Group I showed an increase of 38.47% ($P < 0.05$), 64.21% ($P < 0.01$) on the 29th (early fourth stage) and 36th day (mid-fourth stage) respectively. However, on the 43rd day the leucocyte count showed a slight fall and thereafter showed an increasing trend from 50th day, which continued upto the 60th day and reached the normal level on the 70th day (almost mature stage) as compared to the negative controls.

The increase of leucocyte count in respect of the Group II was 37.74% ($P < 0.05$); and 81.88% ($P < 0.05$) on the 14th and 29th day respectively. Though the leucocyte count was at a higher level on the 36th day (mid-fourth stage) it started declining from then onwards and reached normal level on the 43rd day and continued to be at the same level upto the 50th day (young adult stage) as that of the negative controls. Later it showed a

sharp fall on the 60th day (nearing maturity) which once again rose to the normal level by the 70th day.

Thus, marked leucocytosis was noted in the period between the 14th day (late third stage) and 36th day (mid-fourth stage) post infection in both the experimental groups.

The data are presented in the table 7, and are graphically represented in the chart 7.

EOSINOPHILS

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

The increase in respect of Group I (average number of worms established 47.50) was 91.43% (not significant) and in respect of the group II (average number of worms established 36.25) was 85.61% (not significant).

However, on further analysis the Group I showed an increase of 152.67% ($P < 0.05$) and 824% ($P < 0.05$) on the 36th (mid-fourth stage) and 60th day(nearing maturity)

respectively. And in respect of Group II, the increase was 812.50% ($P < 0.05$) on the 14th day (late third stage). Commencing from the 14th day the differential eosinophil count of the Group II was maintained at a higher level, upto the 36th day, but from the 43rd day onwards the trend was reversed for about a week. However a second rise in eosinophilic count was noticed on the 50th day (young adult stage) which continued to be at a higher level till the 70th day (almost mature stage). The absolute eosinophil count of the experimental groups was also showed the same trend as that of the differential eosinophil count, over the negative controls.

Thus marked eosinophilia was observed for most of the experimental period.

The data are presented in the tables 8 & 13, and are graphically represented in the charts 8 & 9.

HETEROPHILS

The average heterophil count of the entire experimental groups showed no significant difference from that of the negative controls.

However, on further analysis, the differential heterophil count of the Group I (average number of worms established 47.50) showed an increase of 70.90% (P 0.01) and in the Group II (average number of worms established 36.25) showed an increase of 73.77% ($P < 0.01$), on the 30th day (young adult stage). Later a slightly higher heterophil count was maintained by the experimental birds upto the 70th day (almost mature stage) which is however statistically not significant. But the absolute heterophil count was at a higher level starting from the 29th day (early fourth stage) to the 36th day (mid-fourth stage) in the Group I which on the 43rd day came down to normal level, as compared to the negative controls. And thereafter, it rose to the peak level on the 50th day (young adult stage) which continued to be at a higher level, though showed a gradual fall from the 60th day. Whereas, the absolute heterophil count of the Group II was at a higher level starting from the 14th day (late third stage) which was at the peak

level on the 50th day (young adult stage) but on the 60th day came down to normal level, which on the 70th day rose slightly to a higher level.

Thus a relative heterophilia was noted starting from the 43rd day (late fourth stage) till to the 70th day (almost mature stage) in the experimental groups. And absolute heterophilia was observed on the 29th, 36th, 50th, 60th, and 70th day in the Group I, and starting from the 14th day to the 50th day and on the 70th day in the group II.

The data are presented in the tables 9 & 13, and are graphically represented in the charts 10 & 11.

LYMPHOCYTES

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

On further, analysis in the Group I (average number of worms established 47.30) the differential lymphocyte count showed an initial increase of 29.44% (not significant)

between the period 14th to 29th day. However, from the 29th day (early fourth stage) onwards the differential lymphocyte count started declining the reached the normal level on the 36th day as compared to the negative control. The lymphocyte count commenced to decline from the 43rd day (late fourth stage), and was below normal level even on the 70th day. Statistically, however, the decrease was 23.53% (not significant); 33.67% ($P < 0.01$) and 16.33% (not significant) on the 43rd, 50th and 60th day respectively.

The differential lymphocyte count in the Group II (average number of worms established 36.25) showed a slight rise on the 14th day (late third stage) and showed a fall on the 29th day (early fourth stage), followed by a second short rise on the 36th day (mid-fourth stage). From the 43rd day (late fourth stage) onwards, the differential lymphocyte count commenced to decline, which reached a low level on the 50th day (43.48%, significant at 1% level). The count continued

to be till the 70th day as compared to the negative controls.

The absolute lymphocyte count in the Group I showed the same trend as that of the differential lymphocyte count. But in the Group II the initial rise in the absolute lymphocyte count was maintained till the 36th day (mid-fourth stage). Commencing from the 43rd day onwards a gradual fall in the count was noticed as with the differential lymphocyte count.

Thus, lymphocytosis was noted from the 14th (late third stage) to the 36th day (mid-fourth stage) followed by slight lymphopenia in the later period (between the 43rd and 70th day post infection).

The data are presented in the tables 10 & 13 and are graphically represented in the charts 12 & 13.

BASOPHILS

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

On further analysis, Group I (average number of worms established 47.50) showed a decrease of 94.59% ($P < 0.01$) and 67.64% ($P < 0.01$) on the 29th (early fourth stage) and 36th day (mid-fourth stage) respectively. But for a slight fall on the 70th day the differential basophil count showed no notable change from the 43rd day to the 60th day. And the absolute basophil count also showed the same trend as that of the differential count.

The differential and absolute basophil counts in the Group II (average number of worms established 36.25) was generally at a low level. The decrease of differential basophil count in the Group II was 72.97% ($P < 0.01$) 52.94% ($P < 0.05$) and 53.29% ($P < 0.05$) on the 29th (early fourth stage) 36th (mid-fourth stage) and 43rd day (late fourth stage) respectively.

Thus marked basopenia was observed in the initial period of infection from 29th day to the 43rd day in both the experimental groups.

The data are presented in the tables 11 & 13 and are graphically represented in the charts 14 & 15.

MONOCYTES

The average monocyte count of the entire experimental groups showed no significant difference from that of the negative controls.

The data are presented in the table 12.

TABLE-1 Showing the total Erythrocyte count in two levels of A.hamulosa infection in White Leghorn Chicken compared with the negative controls.

DAYS	GROUP I				GROUP II				GROUP III			
	1	2	3	4	5	6	7	8	9	10	11	12
14th	2.18	1.55	1.86	1.89	2.43	1.95	2.36	1.62	2.66	2.48	2.34	1.68
29th	2.28	2.03	1.35	1.42	1.51	2.60	1.75	1.79	3.06	2.46	2.05	2.43
36th	2.17	2.11	1.87	2.17	1.62	2.04	1.86	1.62	2.20	1.99	2.02	1.60
43rd	1.70	1.80	1.76	1.81	1.66	2.46	2.08	2.27	2.62	2.06	2.08	1.72
50th	1.96	2.32	1.83	1.80	2.03	2.38	1.77	2.41	2.52	2.08	2.50	2.46
60th	1.85	2.26	1.50	1.85	1.64	1.50	2.23	1.90	1.70	1.87	2.12	2.12
70th	1.83	2.18	1.86	2.14	1.62	2.12	1.97	2.34	2.41	2.17	2.33	2.38

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

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	2	1.498	0.749	8.053**
Between days	6	0.753	0.125	1.344 n.s.
Error	75	7.042	0.093	
Total	83	9.293		

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

TABLE-2 Showing the Haemoglobin values in two levels of A.haemolose infection in White Leghorn Chicken compared with the negative controls.

DAYS	GROUP I				GROUP II				GROUP III			
	1	2	3	4	5	6	7	8	9	10	11	12
14th	9.0	6.5	6.5	7.8	8.6	6.5	7.4	5.6	8.0	7.6	8.8	6.1
29th	8.2	7.4	6.5	6.1	6.5	9.0	7.0	7.8	9.0	6.5	6.1	6.5
36th	7.8	8.6	7.0	7.4	6.5	7.4	7.0	6.5	8.2	7.1	7.8	6.5
43rd	6.5	7.4	6.5	6.1	5.2	9.0	7.0	7.8	9.5	7.4	7.8	6.1
50th	7.4	9.0	5.7	6.5	7.8	9.0	7.0	8.6	7.8	7.4	9.8	9.0
60th	8.6	7.4	6.1	5.3	7.0	7.0	9.0	8.6	7.8	7.0	7.8	8.6
70th	8.2	7.4	5.7	5.7	6.8	7.4	8.2	9.0	7.8	7.2	8.6	9.0

All values in gram percentage.

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	2	6.279	3.139	2.768 n.s.
Between days	6	4.628	0.771	0.679 n.s.
Error	75	85.061	1.134	
Total	83	95.968		

n.s. Not significant.

TABLE-3 Showing the Packed cell volume in two levels of *A.hamulosa* infection in White Leghorn Chicken compared with the negative controls.

DAYS	GROUP I				GROUP II				GROUP III			
	1	2	3	4	5	6	7	8	9	10	11	12
14th	30.0	22.0	24.0	28.0	27.0	24.0	28.0	25.0	32.0	25.0	28.0	23.0
29th	32.0	20.0	27.0	25.0	21.0	35.0	26.0	23.0	33.0	26.0	26.0	26.0
36th	24.0	29.0	24.0	27.0	21.0	25.0	23.0	23.0	25.0	23.0	24.0	18.0
43rd	24.5	24.0	24.0	24.0	19.0	28.5	23.0	24.0	27.0	22.0	24.0	19.0
50th	21.0	28.0	23.0	23.0	26.0	26.0	29.0	26.0	26.0	23.0	26.0	27.0
60th	24.0	24.0	22.0	19.0	25.0	22.0	28.0	29.0	25.0	25.0	23.0	28.0
70th	26.0	25.0	22.0	23.0	23.0	25.0	27.0	29.0	25.0	26.0	25.0	23.0

All values in percentage.

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	2	1.613	0.806	0.090 n.s.
Between days	6	140643	23.440	2.623 *
Error	75	670.054	8.934	
Total	83	812.310		

* Significant at 5% level
n.s. Not significant.

Table-4 Showing the Mean corpulscular volume in two levels of A.haemulosa infection in White Leghorn Chicken compared with the negative controls.

DAYS	GROUP I				GROUP II				GROUP III			
	1	2	3	4	5	6	7	8	9	10	11	12
14th	137.61	141.93	133.33	148.48	111.11	123.07	118.64	154.32	120.30	100.80	119.65	136.90
29th	140.35	147.73	200.00	176.05	139.07	134.61	148.37	128.49	107.84	105.69	126.82	106.99
36th	110.59	137.44	128.34	124.42	129.62	122.54	123.65	141.97	113.63	121.05	118.81	112.50
43rd	144.11	133.33	136.36	138.12	114.45	115.85	110.57	105.72	103.05	106.79	115.38	110.46
50th	107.14	120.68	125.68	127.77	128.07	109.24	112.99	116.18	103.17	110.57	104.00	109.75
60th	129.72	106.19	146.66	102.70	134.14	146.66	125.56	152.63	147.05	133.68	108.49	132.07
70th	142.07	114.67	118.27	107.47	154.32	117.92	137.05	123.93	103.73	119.81	106.38	117.64

All values in cubic microns.

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	2	4806.472	2403.236	10.907**
Between days	6	4506.950	751.158	3.409**
Error	75	16525.193	220.335	
Total	83	25838.615		

** Significant at 1% level.

TABLE-5 Showing the mean corpuscular haemoglobin in two levels of *A.haemulosa* infection in White Leghorn Chicken compared with the negative controls.

DAYS	GROUP I				GROUP II				GROUP III			
	1	2	3	4	5	6	7	8	9	10	11	12
14th	41.28	41.93	34.94	41.26	35.39	33.33	31.35	34.56	30.07	30.64	37.60	36.30
29th	35.96	36.45	48.14	42.95	43.04	38.07	40.00	43.57	27.27	26.42	29.75	26.74
36th	35.94	40.75	37.43	34.10	40.12	36.27	37.63	40.12	40.90	35.67	38.61	40.62
43rd	38.23	41.11	36.93	33.70	31.32	36.58	30.43	34.96	31.29	35.92	37.50	35.46
50th	37.75	38.79	31.14	36.11	38.42	37.81	39.54	35.68	37.69	35.57	39.20	36.58
60th	46.48	32.74	40.66	28.64	42.68	46.66	40.35	45.26	45.88	37.43	36.79	40.56
70th	44.80	33.94	30.64	26.63	41.97	34.90	41.62	38.46	32.36	33.17	35.59	37.81

All values in micromicro grams.

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	2	120.763	60.381	2.902 n.s.
Between days	6	220.815	36.802	1.769 n.s.
Error	75	1560.065	20.800	
Total	83	1901.643		

n.s. Not significant.

TABLE-6 Showing the Mean corpuscular haemoglobin concentration in two levels of *A.hemolysa* infection in White Leghorn Chicken compared with the negative controls.

DAYS	GROUP I				GROUP II				GROUP III			
	1	2	3	4	5	6	7	8	9	10	11	12
14th	30.00	29.54	27.08	27.85	31.85	27.08	26.42	22.40	25.00	30.40	31.42	26.52
29th	29.56	24.66	24.07	24.40	30.95	28.28	26.92	33.91	27.27	25.00	23.46	25.00
36th	32.50	29.65	29.16	27.40	30.95	29.60	30.43	28.26	36.00	30.86	32.50	26.11
43rd	26.53	30.83	27.08	25.41	27.36	31.57	30.43	32.50	30.37	33.63	32.50	32.10
50th	35.23	32.14	24.78	28.26	30,00	34.61	35.00	30.71	37.69	32.17	37.69	33.33
60th	35.83	30.83	27.72	27.89	31.81	31.81	32.14	29.65	31.20	28.00	33.91	30.71
70th	31.53	29.60	25.90	24.78	27.20	29.60	30.37	31.03	31.20	27.69	34.40	32.14

All values in porcentage.

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	2	95.401	47.70	6.247**
Between days	6	295.720	49.286	6.455**
Error	75	572.645	7.635	
Total	83	963.766		

** Significant at 1% level.

TABLE-7 Showing the total Leucocyte count in two levels of A.hamulosa infection in White Leghorn Chicken compared with the negative controls.

DAYS	GROUP I			GROUP II			GROUP III					
	1	2	3	4	5	6	7	8	9	10	11	12
14th	49.11	25.11	27.33	46.33	36.33	50.00	41.22	31.00	24.55	26.11	31.33	33.11
29th	24.44	21.33	20.11	35.33	35.33	21.55	23.22	20.33	12.77	15.11	13.00	14.33
36th	44.66	37.54	45.54	43.00	34.88	22.22	37.54	35.76	20.22	30.22	28.00	25.54
43rd	19.11	18.00	24.66	30.66	32.00	18.22	19.77	40.66	34.66	35.88	16.22	25.77
50th	37.33	46.00	52.22	44.00	39.11	41.11	44.00	36.00	49.11	25.33	30.22	48.66
60th	46.66	41.55	39.55	36.88	23.55	24.22	23.55	32.22	35.11	39.33	37.33	38.44
70th	44.20	44.00	34.66	39.55	37.33	44.66	32.66	39.55	39.33	36.88	41.55	44.66

All values in thousands/cu.mm of blood.

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	2	475.558	237.779	4.351*
Between days	6	3593.625	599.270	10.966**
Error	75	4098.500	54.646	
Total	83	8169.683		

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

TABLE-8 Showing the percentage of Eosinophils in two levels of *A.hamulosa* infection in White Leghorn Chicks compared with the negative controls.

DAYS	GROUP I				GROUP II				GROUP III			
	1	2	3	4	5	6	7	8	9	10	11	12
14th	2.0	7.0	3.0	2.0	9.0	3.0	9.0	4.0	2.0	4.0	1.0	1.0
29th	7.0	9.0	5.0	4.0	7.0	5.0	11.0	5.0	6.0	4.0	2.0	3.0
36th	10.0	15.0	4.0	11.0	5.0	7.0	11.0	9.0	2.0	11.0	10.0	4.0
43rd	13.0	3.0	4.5	6.0	3.0	3.5	0.5	2.5	4.0	2.0	2.5	2.0
50th	5.0	0.5	1.5	7.5	7.0	3.5	5.0	6.0	2.0	0.5	1.5	4.5
60th	9.0	7.0	0.0	2.5	6.5	0.0	3.5	5.0	0.5	1.0	0.0	0.5
70th	7.5	6.0	3.0	4.0	6.0	3.5	6.5	5.0	2.5	3.5	2.5	2.5

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	2	115.024	57.512	7.849**
Between days	6	174.833	29.138	3.976**
Error	75	549.560	7.327	
Total	83	839.417		

** significant at 1% level.

TABLE-9 Showing the percentage of Heterophils in two levels of *A.hamulosa* infection in White Leghorn Chicken compared with the negative controls.

DAYS	GROUP I				GROUP II				GROUP III			
	1	2	3	4	5	6	7	8	9	10	11	12
14th	33.0	33.0	28.0	54.0	46.0	23.0	29.0	61.0	53.0	30.0	56.0	56.0
29th	50.0	45.0	25.0	34.0	51.0	39.0	54.0	55.0	52.0	33.0	35.0	26.0
36th	49.0	51.0	46.0	43.0	37.0	55.0	47.0	36.0	61.0	42.0	54.0	50.0
43rd	57.5	48.0	47.5	48.5	59.5	57.5	54.5	44.5	46.5	33.0	37.5	51.5
50th	52.5	57.5	45.0	54.0	56.0	51.0	48.5	56.5	26.0	31.5	34.5	30.0
60th	52.5	50.5	21.0	36.5	48.5	39.5	55.5	45.5	60.5	30.0	29.5	30.5
70th	49.0	47.5	35.5	45.0	49.5	44.0	47.5	44.5	49.5	33.0	41.0	40.5

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	2	575.899	287.949	2.856 n.s.
Between days	6	602.060	100.343	0.998 n.s.
Error	75	7539.789	100.530	
Total	83	8717.748		

n.s. Not significant.

TABLE-10 Showing the percentage of Lymphocytes in two levels of *A.haemulosa* infection in White Leghorn Chicken compared with the negative controls.

DAYS	GROUP I				GROUP II				GROUP III			
	1	2	3	4	5	6	7	8	9	10	11	12
14th	63.0	60.0	67.0	43.0	41.0	56.0	57.0	33.0	43.0	60.0	35.0	42.0
29th	43.0	43.0	70.0	60.0	30.0	52.0	30.0	33.0	36.0	57.0	46.0	54.0
36th	33.0	27.0	41.0	36.0	51.0	36.0	36.0	50.0	30.0	36.0	26.0	40.0
43rd	33.5	37.5	37.5	43.0	34.5	32.5	43.5	49.5	44.5	54.0	50.5	40.5
50th	38.5	38.5	45.5	35.0	30.0	41.0	39.5	30.5	68.5	62.0	60.0	59.5
60th	36.0	37.0	66.0	50.5	42.5	56.0	35.5	44.0	37.5	61.0	66.0	62.0
70th	39.5	40.5	54.5	44.5	39.0	45.5	40.0	40.0	43.0	55.0	47.0	49.5

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	2	792.184	396.092	3.674*
Between days	6	1469.833	244.972	2.272*
Error	75	8004.900	107.798	
Total	83	10346.917		

* Significant at 5% level.

TABLE-11 Showing the percentage of Eosophils in two levels of A.hamulosa infection in White Leghorn Chicken compared with the negative controls.

DAYS	GROUP I				GROUP II				GROUP III			
	1	2	3	4	5	6	7	8	9	10	11	12
14th	1.0	0.0	0.0	0.0	2.0	3.0	1.0	1.0	2.0	5.0	5.0	0.0
29th	0.0	1.0	0.0	1.0	2.0	1.0	5.0	2.0	5.0	6.0	15.0	11.0
36th	5.0	4.0	1.0	1.0	5.0	2.0	5.0	4.0	7.0	11.0	10.0	6.0
43rd	6.0	10.5	9.5	6.0	3.0	4.5	4.5	2.5	5.0	11.0	9.0	6.0
50th	4.0	4.0	6.0	5.0	5.5	4.0	6.0	5.0	3.0	6.0	4.0	5.5
60th	2.5	2.5	7.5	10.5	2.5	3.5	5.0	4.5	1.5	7.0	4.5	6.0
70th	3.5	4.5	4.0	4.0	3.0	3.5	6.0	4.5	5.5	7.5	8.0	6.0

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	2	117.721	58.86	9.228**
Between days	6	133.364	22.227	3.484**
Error	75	478.404	6.378	
Total	83	729.489		

** Significant at 1% level.

TABLE-12 Showing the percentage of Monocytes in two levels of *A.haemulosa* infection in White Leghorn Chicken compared with the negative controls.

DAYS	GROUP I				GROUP II				GROUP III			
	1	2	3	4	5	6	7	8	9	10	11	12
14th	1.0	0.0	2.0	1.0	2.0	15.0	4.0	1.0	0.0	1.0	3.0	1.0
29th	0.0	2.0	0.0	2.0	10.0	3.0	0.0	5.0	1.0	0.0	2.0	5.0
36th	3.0	3.0	8.0	9.0	1.0	0.0	1.0	1.0	0.0	0.0	0.0	0.0
43rd	0.0	1.0	1.0	0.0	0.0	2.0	0.0	1.0	0.5	0.0	0.5	0.0
50th	0.5	0.0	2.0	0.5	1.5	0.5	1.0	2.0	100	0.0	0.0	0.5
60th	0.0	3.0	5.5	0.5	0.0	1.0	0.5	1.0	0.0	1.0	0.0	1.0
70th	0.5	1.5	3.0	2.0	2.5	3.5	1.0	2.0	0.5	1.0	1.5	1.5

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	2	29.899	14.949	2.707 n.s.
Between days	6	52.155	8.692	1.574 n.s.
Error	75	414.122	5.521	
Total	80	496.176		

n.s. Not significant.

TABLE-13 Showing the total, differential and absolute counts of blood leucocytes in White Leghorn Chicken infected with A.hemuloon.

	14th	29th	36th	43rd	50th	60th	70th
				GROUP-I			
Total Leucocytes	35,971	25,307	42,694	23,110	44,888	41,166	40,160
Heterophils	37/13,309	38.5/9,743	47/20,066	50/11,555	52/23,341	40/16,466	44/17,868
Lymphocytes	58/20,863	54/13,665	34.5/14,729	35/8,088	39/17,506	47/19,348	45/18,274
Monocytes	1/359	1/253	5.5/2,348	0.5/115.5	1/448	2/823	2/812
Eosinophils	3.5/1,259	6/1.518	10/4.269	6.5/1.502	3.5/1,571	5/2,058	5/2,030
Basophils	0.5/179	0.5/126	3/1.280	8/1.848	4.5/2,019	6/2,469	4/1,624
				GROUP II			
Total Leucocytes	39,638	25,110	32,610	27,666	40,055	25,888	38,554
Heterophils	40/15,855	50/12,555	43.5/14,185	53/14,662	53/21,229	47/12,167	46.5/17,927
Lymphocytes	47/18,629	36/9,039	43.5/14,185	40/11,066	35.5/14,219	44.5/11,520	41.5/15,999
Monocytes	5/1,982	4.5/1,129	1/326	0.5/138	1.5/600	0.5/129	2.5/963
Eosinophils	6/2,378	7/1,757	8/2,608	2.5/691	5/2,002	4/1,035	5/1,927
Basophils	2/792	2.5/627	4/1,304	4/1,106	5/2,002	4/1,035	4.5/1,735
				GROUP III			
Total Leucocytes	28,776	13,804	25,999	26,138	38,332	37,554	40,610
Heterophils	49/14,100	36.5/5,038	52/13,519	42/11,818	30.5/11,691	37.5/14,082	41/16,650
Lymphocytes	45/12,949	48,5/6,695	33/8,579	47.5/13,365	62.5/23,957	56.5/21,218	49/19,898
Monocytes	1/287	2/276	0.0/0.0	0.5/140	0.5/191	0.5/187	1/406
Eosinophils	2/575	4/552	6.5/1,689	2.5/703	2/766	0.5/187	2.5/1,015
Basophils	3/863	9/1,242	8.5/2,209	7.5/2,110	4.5/1,724	5/1,877	6.5/2,639

Each figure represents the average count of 4 birds.
-Nominator and denominator represents differential and absolute counts.

ACUARIA HAMULOSA INFECTION - WHITE LEGHORN

CHART-2

TOTAL ERYTHROCYTES

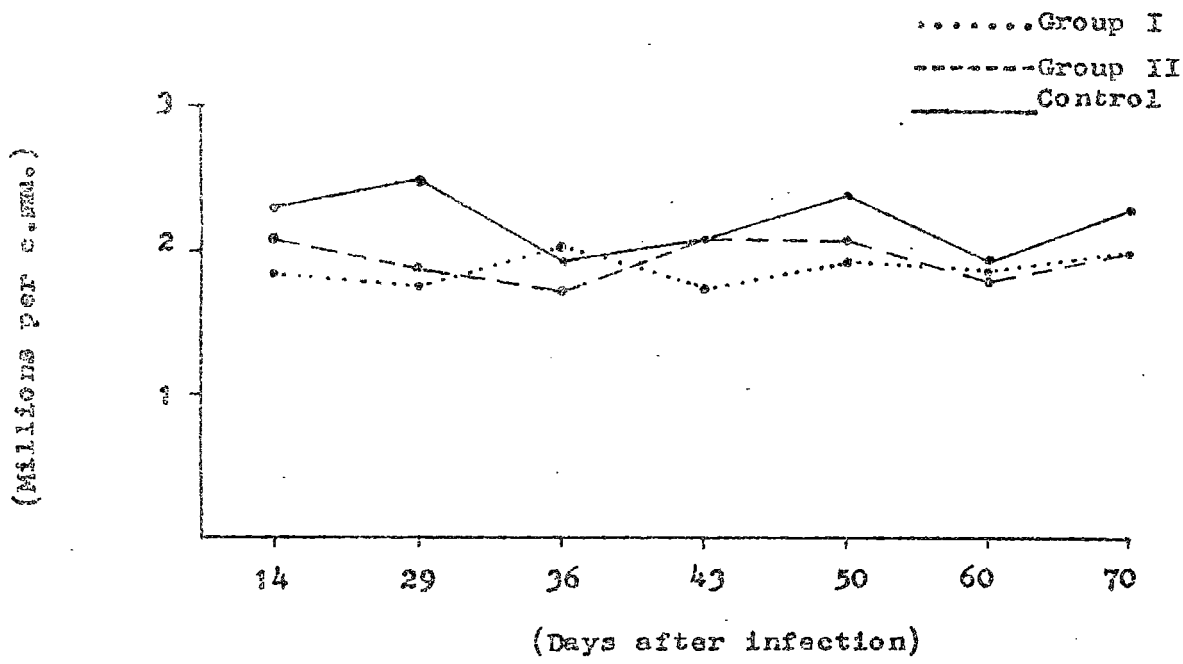
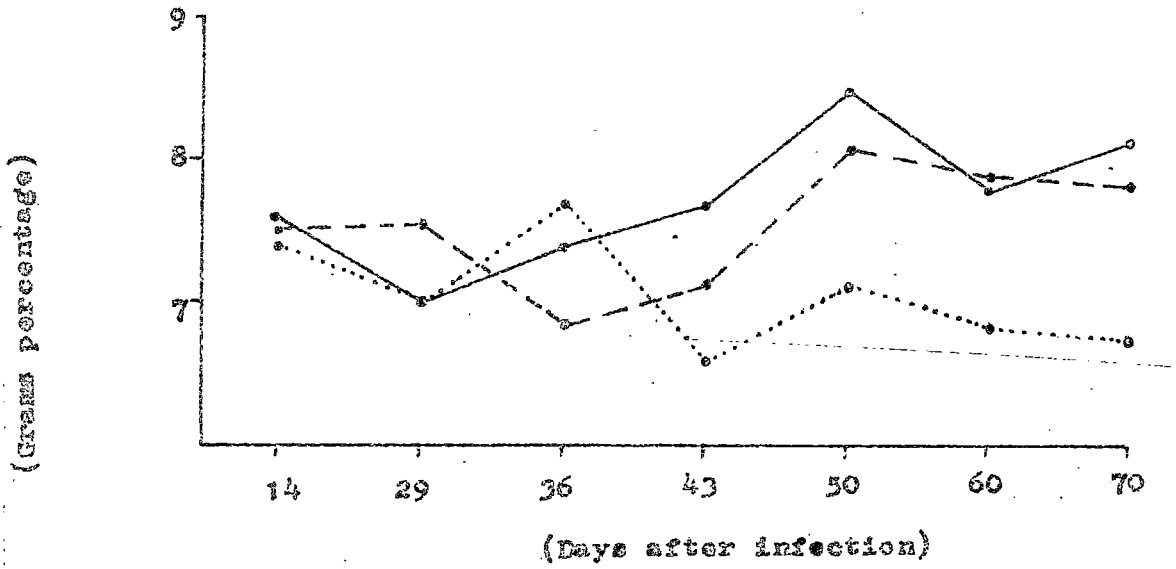


CHART-3

HAEMOGLOBIN



AGUARIA HAMULOSA INFECTION - WHITE LEGHORN

CHART-4 PACKED CELL VOLUME

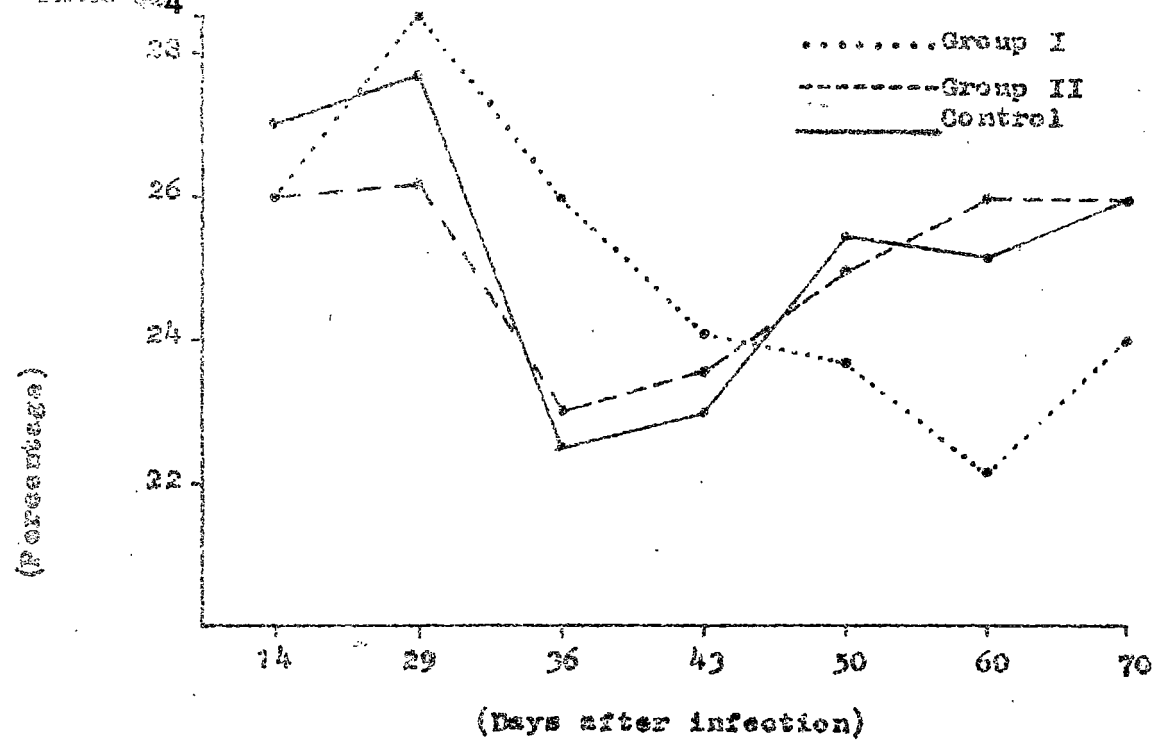
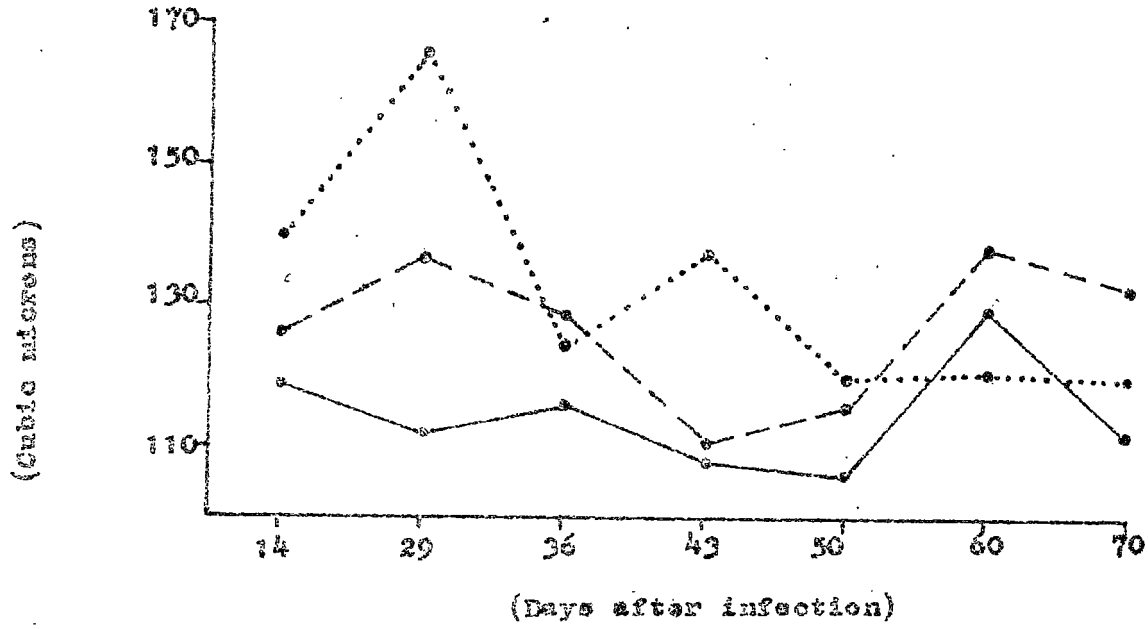


CHART-5 MEAN CORPUSCULAR VOLUME



AGUARIA HAMULOSA INFECTION - WHITE LEGHORN

CHART-6

MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION

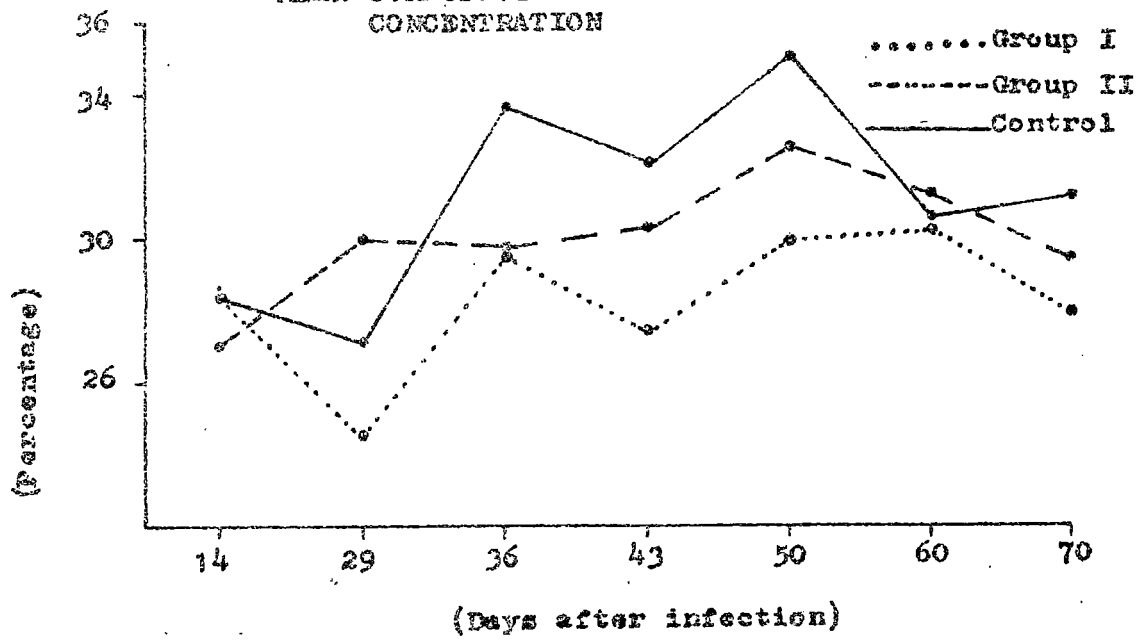
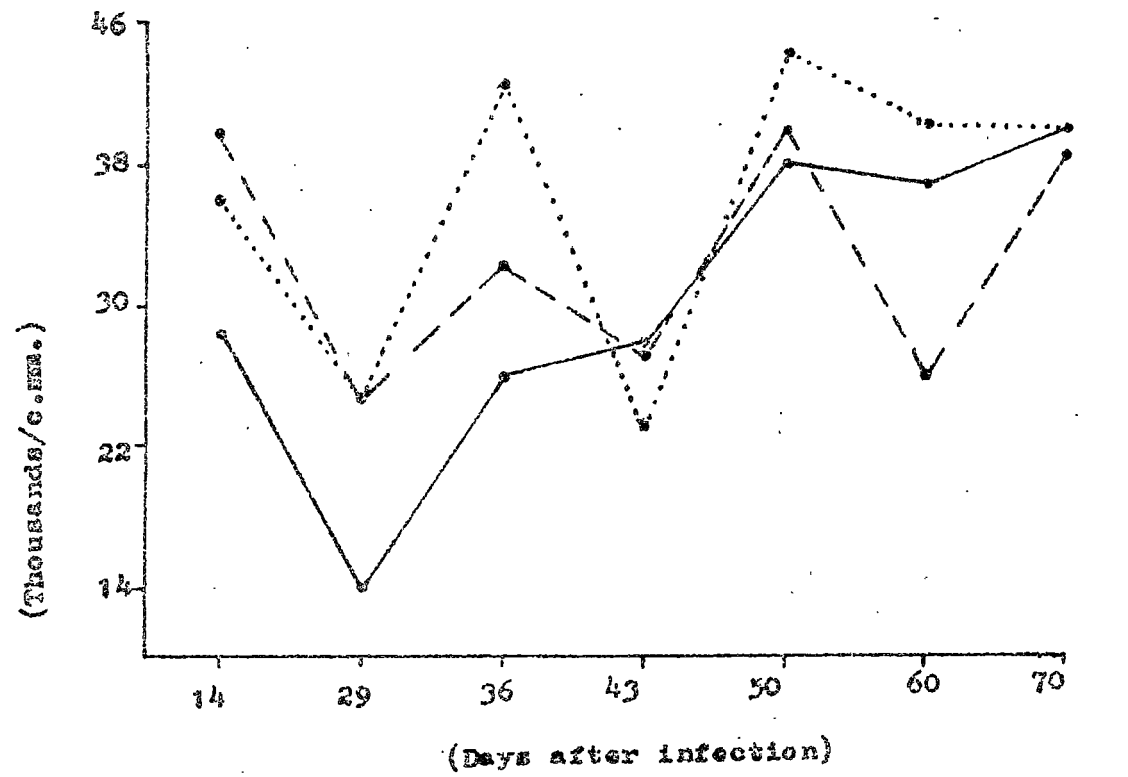


CHART-7

TOTAL LEUCOCYTES



ACUARIA HAMULOSA INFECTION - WHITE LEGHORN

CHART-8

EOSINOPHILS

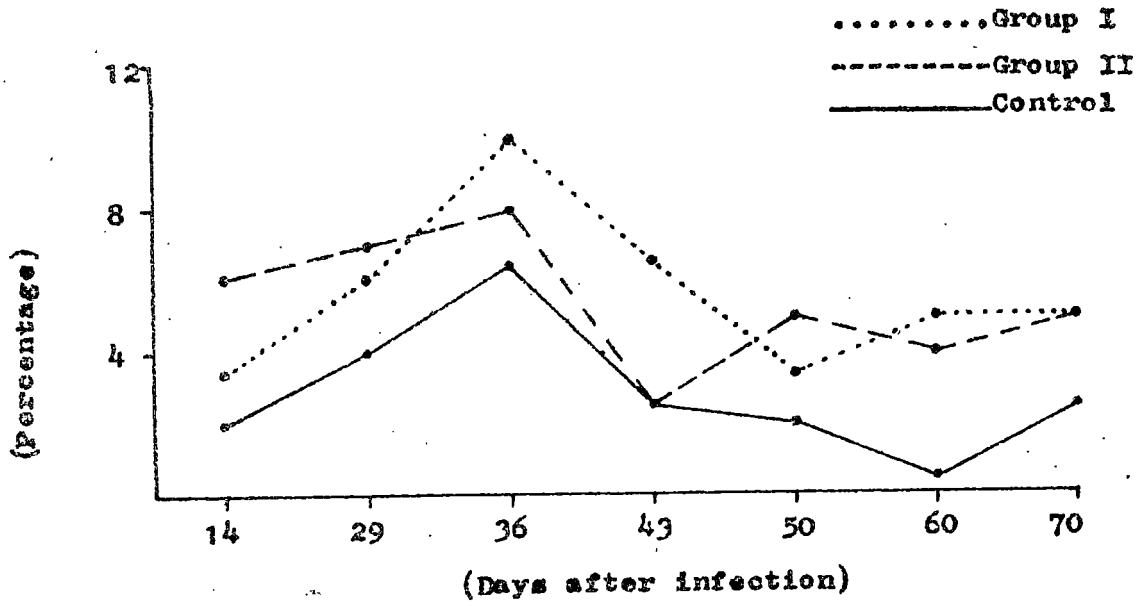
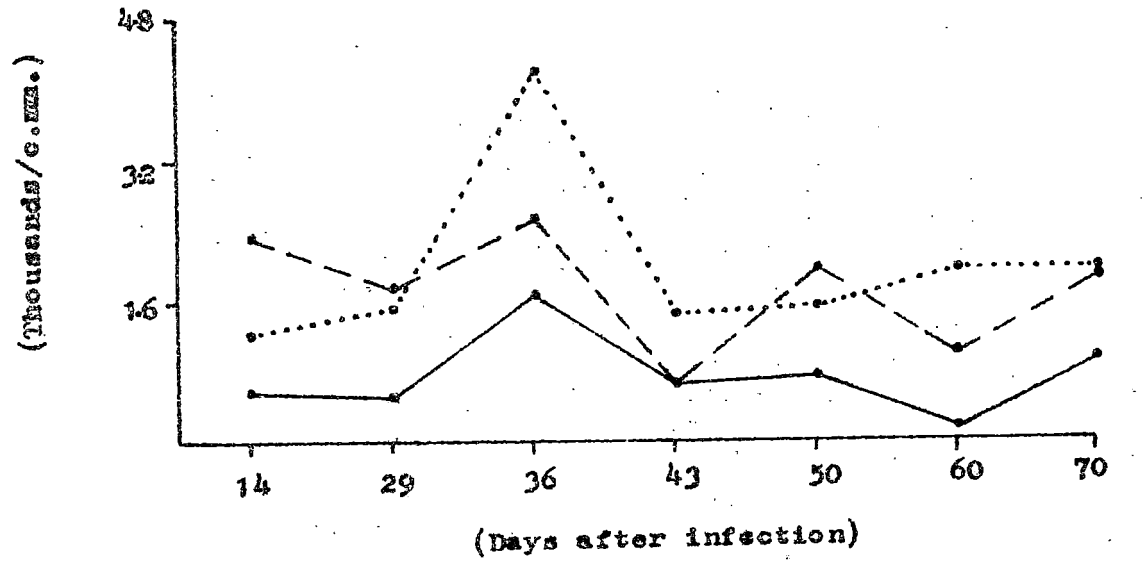


CHART-9

EOSINOPHILS ABSOLUTE



ACUARIA HAMULOSA INFECTION - WHITE LEGHORN

CHART-10

HETEROPHILS

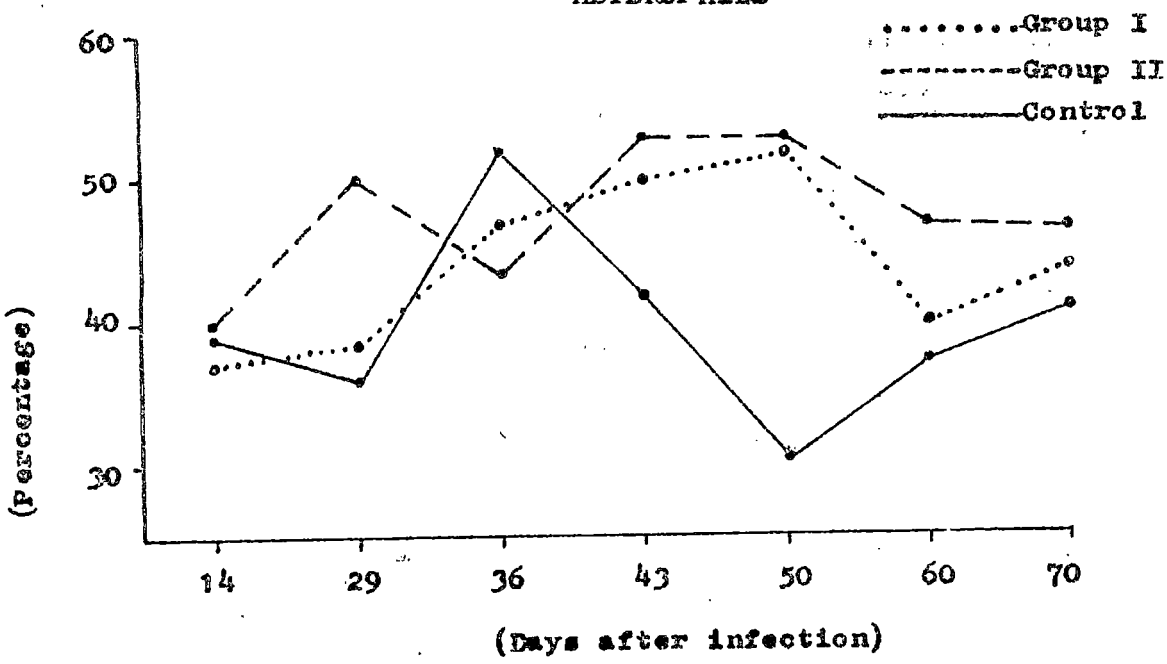


CHART-11

HETEROPHILS ABSOLUTE

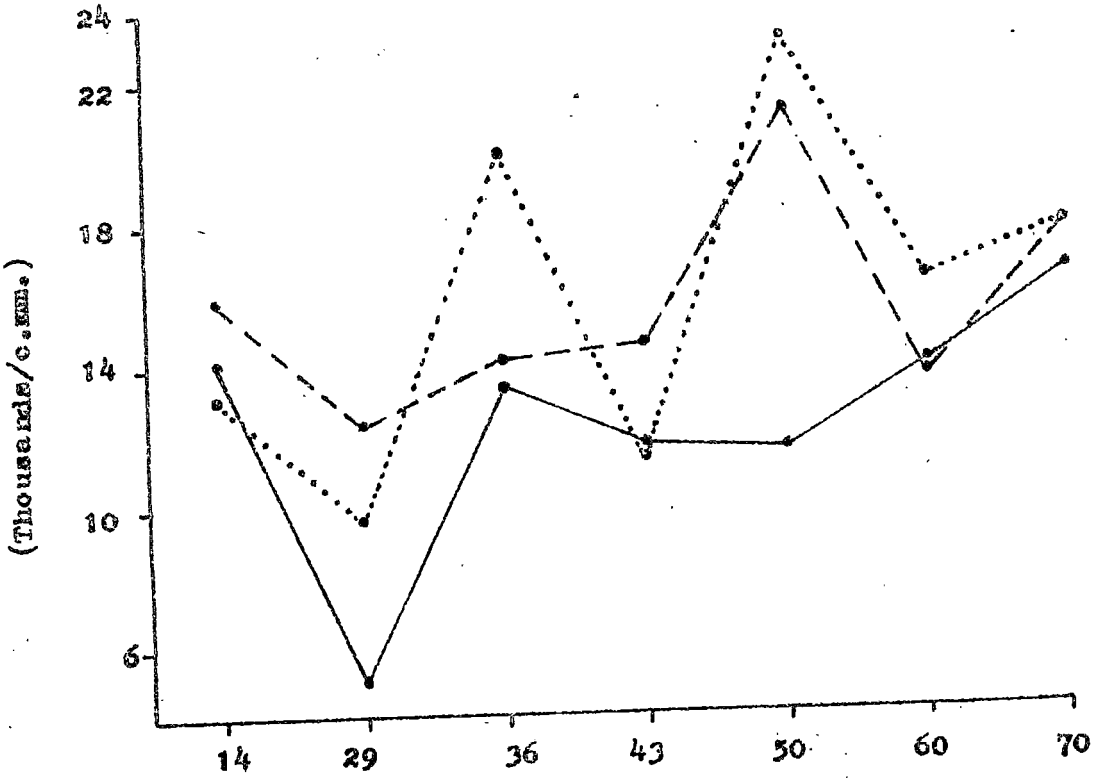
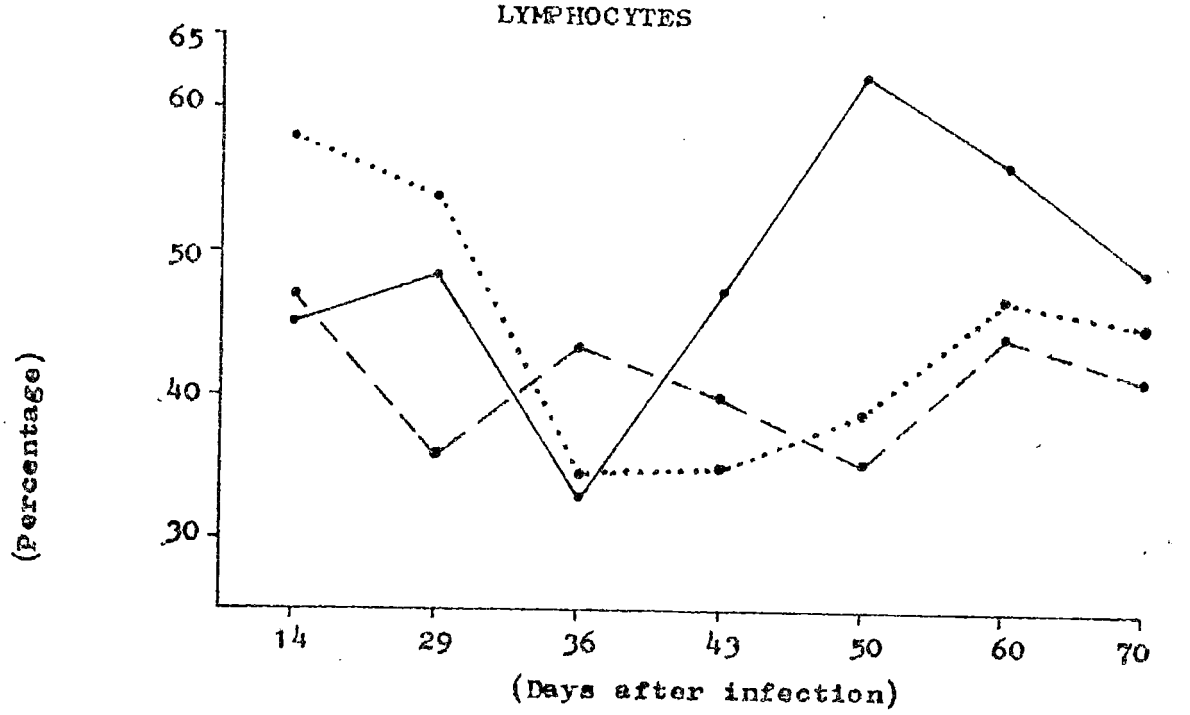


CHART-12

ACUARIA HAMULOSA INFECTION - WHITE LEGHORN

LYMPHOCYTES



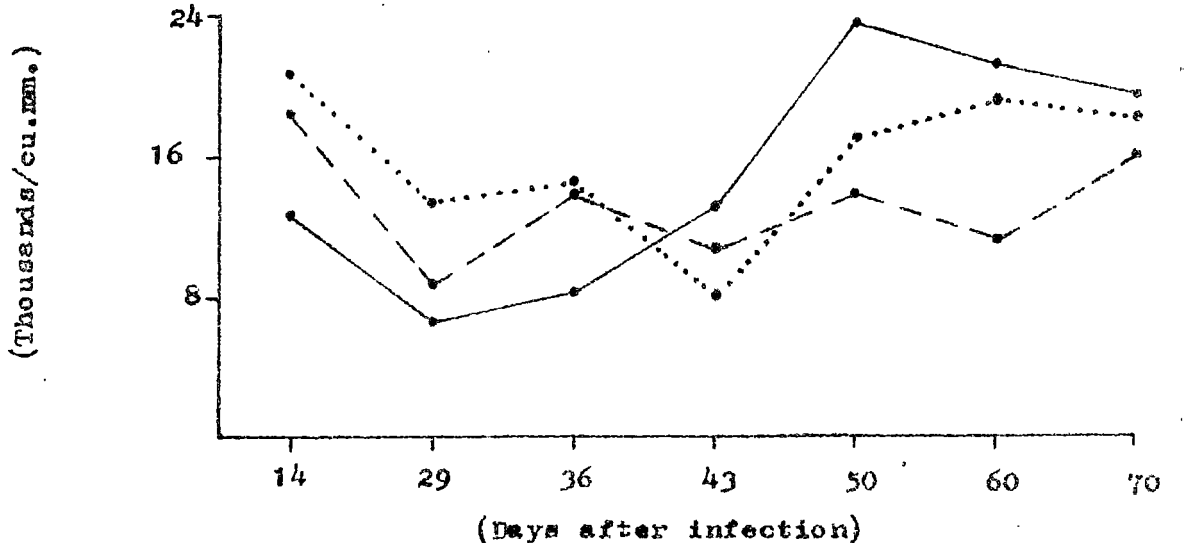
.....Group I

--------Group II

————Control

CHART-13

LYMPHOCYTES ABSOLUTE



ACUARIA HAMULOSA INFECTION-WHITE LEGHORN

CHART-14

BASOPHILS

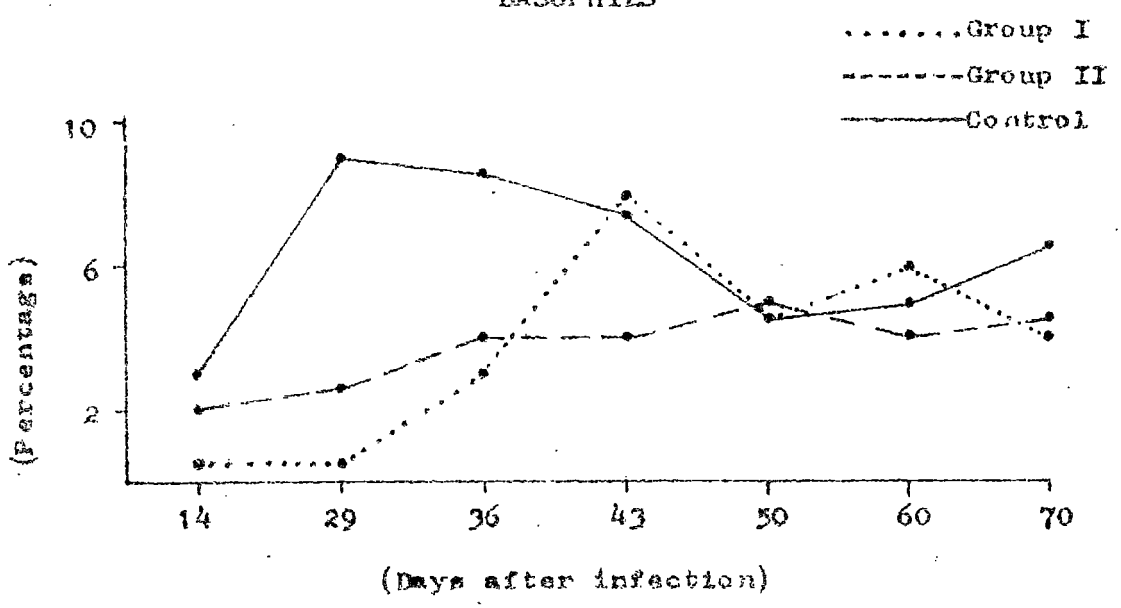
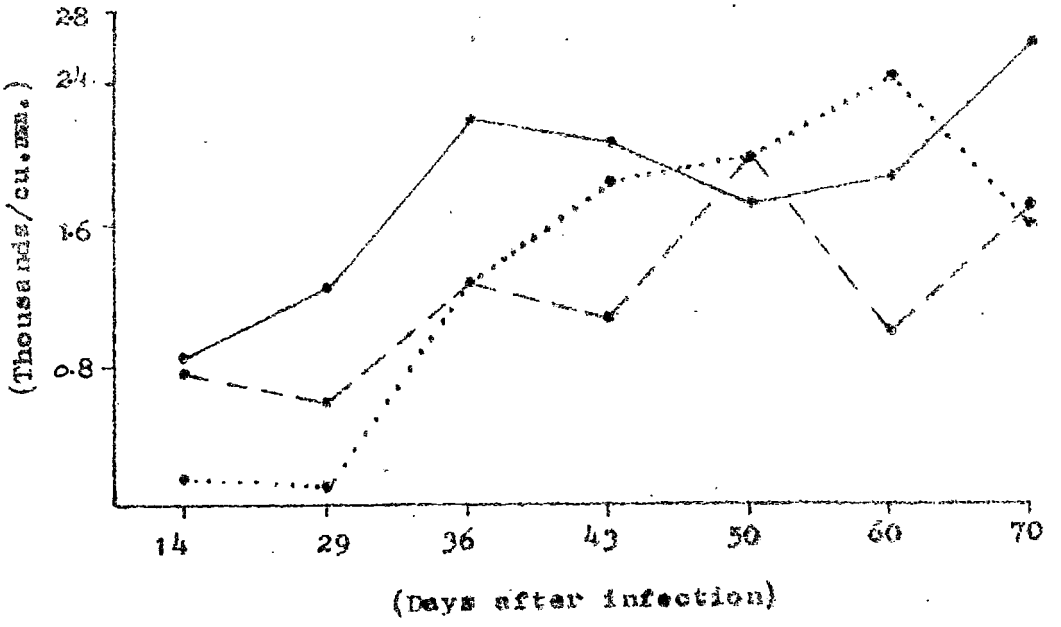


CHART-15

BASOPHILS ABSOLUTE



PATHOGENICITY OF ACUARIA HAMULOSA TO WHITE PLYMOUTH ROCK

HAEMATOLOGICAL OBSERVATIONS

To study the haematological changes, blood samples were collected on the 14th (corresponding to the third stage), 29th, 36th and 43rd (corresponding to the fourth stage), 50th, 60th (corresponding to the young adult stage) and 70th day (corresponding to the mature adult stage) post infection.

TOTAL ERYTHROCYTES

The average erythrocyte counts of the experimental group showed no significant difference from those of the negative controls. However, on further analysis the Group I (average number of worms established 15.66) showed a decrease of 15.24% ($P < 0.05$), and 10.50% (not significant), on the 50th day (young adult stage) and 70th day (almost mature stage) post infection respectively.

The data are presented in the table 14.

HAEMOGLOBIN

The average haemoglobin per cent of the experimental group was found to be not significant from that of the negative controls.

A decrease of 9.30% and 11.63% (not significant) on the 50th (young adult stage) and 70th day (almost mature stage) post infection respectively was noticed.

The data are presented in the table 15 and are graphically represented in the chart 16.

PACKED CELL VOLUME

No significant difference was observed in the average packed cell volume of the experimental group, compared to the negative controls.

However, on the 70th day (almost mature stage) a decrease of 16.66% ($P < 0.05$) was observed.

The data are presented in the table 16.

MEAN CORPUSCULAR VOLUME

The average mean corpuscular volume of the experimental group showed no significant difference from that

of the negative controls.

The data are presented in table 18.

MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION

The average mean corpuscular haemoglobin concentration of the experimental group showed no significant difference from the negative controls.

The data are presented in the table 19.

TOTAL LEUCOCYTES

The average leucocyte count of the experimental group showed no significant difference from that of the negative controls.

However, on further analysis Group I (average number of worms established 15.66) showed an increase of 16.92% (not significant) and 30.48% ($P < 0.05$) on the 29th day (early fourth stage) and 36th day (mid-fourth stage) respectively. Commencing from the 43rd day (late fourth stage) the total leucocyte count began to decrease, and by the 50th day, it was almost normal. A second rise in leucocyte count was noticed from the 60th day and

continued to remain slightly higher than normal even on the 70th day (almost mature stage). The increase (14.22%) however was not statiscally, significant.

The data are presented in the table 20 and are graphically represented in the chart 17.

HETEROPHILS

The average heterophil count of the experimental group was found significantly higher than the negative controls ($P < 0.01$).

The increase was 32.29% ($P < 0.05$) over the controls. On further, analysis the differential heterophil count in the Group I (average number of worms established 15.66) showed an increase of 69.52% ($P < 0.01$); 20.91% ($P < 0.05$); 54.81% ($P < 0.01$); 37.33% ($P < 0.01$); and 25.44% ($P < 0.05$), on the 29th (early fourth stage) 36th (mid-fourth stage) 43rd (late fourth stage) 50th (young adult stage) and 70th day (almost mature stage) respectively. And, the absolute heterophil count was also, showed the same trend starting from the 29th (early fourth stage) and remained

to be high till the 70th day (almost mature stage).

Thus, marked heterophilia was observed starting from the 29th day (early fourth stage) till the end of the experiment.

The data are presented in the tables 21 & 26, and are graphically represented in the charts 18 & 19.

LYMPHOCYTES

The average lymphocyte count of the experimental group was found significantly lower than the negative controls ($P < 0.01$).

The decrease in the Group I (average number of worms established 15.66) was 26.93% ($P < 0.01$). On further analysis the differential lymphocyte count of the group I showed a decrease of 39.49%, 43.85%, 39.02% ($P < 0.01$); 24.01% ($P < 0.05$), and 25.15% ($P < 0.01$), on the 29th (early fourth stage) 36th (mid-fourth stage 43rd (late fourth stage) 50th (young adult stage) 60th (nearing maturity) and 70th day (almost mature stage) respectively. The absolute lymphocyte count was also

found to be low, starting from the 29th day till the end of the experiment.

Thus a marked lymphopenia was observed starting from the 29th day (early fourth stage) which was constant till the end of the experiment.

The data are presented in the tables 23 & 26, and are graphically represented in the charts 20 & 21.

MONOCYTES

The average monocyte count of the experimental group was significantly higher than the negative controls ($P < 0.01$).

On further analysis the differential monocyte count of the Group I (average number of worms established 15.66) showed an increase of 1,150% and 303.03% ($P < 0.05$) and the 43rd day (late fourth stage) and 70th day (almost mature stage) respectively. The absolute monocyte count was also found to be higher on the 29th day (early fourth stage) and it showed a slight decrease on the 36th day (mid-fourth stage). Thereafter, from

the 43rd day (late fourth stage) it started once again to rise to reach a peak level on the 60th day. The monocyte count continued to be high till the 70th day.

Thus, monocytosis was noted starting from the 29th day (early fourth stage) and was maintained upto the end of the experiment. The data are presented in the tables 24 & 26 and are graphically represented in the charts 22 & 23.

EOSINOPHILS

The average eosinophil count of the experimental group was significantly higher than the negative controls ($P < 0.01$).

The increase in the Group I (average number of worms established 15.66) was 179.48% ($P < 0.01$) over the negative controls. On further analysis, the differential eosinophil count of the Group I showed an increase of 383.92%, 300.75%, 287.96%, 255.19%, 150% and 177.33% ($P < 0.01$), on the 29th (early fourth stage) 36th (mid-fourth stage) 43rd (late fourth stage) 50th(young adult stage) 60th (nearing maturity) and 70th day

respectively. The absolute eosinophil count was also higher commencing from the 29th day (early fourth stage) and was maintained throughout the experimental period.

Thus, marked eosinophilia was also noted starting from the 29th day (early fourth stage) till the 70th day (almost mature stage).

The data are presented in the tables 22 & 26 and are graphically represented in the charts 24 & 25.

BASOPHILS

The average basophil count of the experimental group showed no significant difference from the negative control.

Though not significant the differential basophil count of the Group I (average number of worms established 15.66) showed, a decrease of 22.50% than the negative controls.

On further analysis the differential basophil count showed a decrease of 65.52% ($P < 0.01$) and 43.40% (not significant) on the 36th and 43rd day respectively.

Thereafter, it was remained almost normal upto the 70th day.

The absolute basophil count also showed the same trend throughout the experiment as that of the differential basophil count.

Thus a transient basopenia was observed during the period between 29th (early fourth stage) and 43rd day (late fourth stage) post infection.

The data are presented in the tables 25 & 26 and are graphically represented in the charts 26 & 27.

TABLE-14 Showing the total Erythrocyte count in *A.hamulosa* infection in White Plymouth Rock compared with the negative controls.

DAYS	GROUP I			GROUP II		
	1	2	3	4	5	6
14th	2.36	1.82	1.77	2.16	1.46	1.33
29th	2.50	1.80	2.22	1.77	2.46	1.99
36th	2.13	1.95	1.83	2.48	1.69	2.13
43rd	1.80	1.58	1.83	1.95	1.73	2.16
50th	2.32	1.89	1.53	2.20	2.70	1.80
60th	2.18	1.71	1.94	2.17	1.85	1.73
70th	2.08	1.93	1.89	2.38	1.96	2.24

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

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	1	0.043	0.043	0.472 n.s.
Between days	6	0.532	0.088	0.967 n.s.
Error	34	3.109	0.091	
Total	41	3.684		

n.s. not significant.

TABLE-15 Showing the Haemoglobin values in *A.haemulosa* infection in White Plymouth Rock compared with the negative controls.

DAYS	GROUP I			GROUP II		
	1	2	3	4	5	6
14th	8.6	8.6	6.5	8.2	5.6	6.1
29th	8.2	6.5	7.8	6.5	7.4	6.5
36th	8.7	7.8	6.1	8.2	6.5	5.7
43rd	7.8	5.7	7.4	8.2	7.0	8.2
50th	9.0	7.4	5.7	8.2	10.2	6.1
60th	8.6	9.0	6.8	9.0	9.0	8.2
70th	8.2	9.0	7.2	10.4	9.0	8.2

All values in gram percentage.

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	1	0.046	0.046	0.028 n.s.
Between days	6	13.535	2.255	1.406 n.s.
Error	34	54.514	1.603	
Total	41	68.095		

n.s. Not significant.

TABLE-16 Showing the Packed cell volume in *A.hamuloss* infection in White Plymouth Rock compared with the negative controls.

DAYS	GROUP I			GROUP II		
	1	2	3	4	5	6
14th	30.0	27.0	22.0	30.0	25.0	21.0
29th	28.0	18.0	23.0	25.0	25.0	22.0
36th	27.0	25.0	21.0	26.0	22.0	21.0
43rd	26.0	22.0	19.0	26.0	22.0	23.0
50th	24.0	22.0	18.0	26.0	25.0	21.0
60th	25.0	22.0	19.0	23.0	23.0	19.0
70th	19.0	21.0	20.0	24.0	26.0	22.0

All values in percentage.

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	1	8.595	8.595	0.970 n.s.
-Between days	6	65.238	10.873	1.227 n.s.
Error	34	301.239	8.859	
Total	41	375.072		

n.s. Not significant.

TABLE-17 Showing the Mean corpuscular volume in A.hamulosa infection in White Plymouth Rock compared with the negative controls.

DAYS	GROUP I			GROUP II		
	1	2	3	4	5	6
14th	127.11	148.35	124.29	138.88	171.23	157.89
28th	112.00	100.00	103.60	141.83	101.62	110.55
36th	128.57	128.20	114.75	104.83	130.17	98.59
43rd	144.00	139.24	103.82	133.33	127.16	106.48
50th	103.00	120.02	117.64	118.18	92.59	116.66
60th	114.67	128.65	97.93	107.98	124.32	109.82
70th	91.34	108.80	105.82	100.84	132.65	98.21

All values in cubic microns.

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	1	91.553	91.553	0.421 n.s.
Between days	6	6023.940	1003.990	4.620 **
Error	34	7387.144	217.268	
Total	41	13502.637		

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

TABLE-18 Showing the Mean corpuscular haemoglobin in A.hamulosa infection in White Plymouth Rock compared with the negative controls.

DAYS	GROUP I			GROUP II		
	1	2	3	4	5	6
14th	36.44	47.25	36.72	37.96	38.35	45.86
29th	32.80	36.11	35.13	36.72	30.08	32.66
36th	40.84	40.00	33.33	33.06	38.46	26.76
43rd	43.33	36.07	40.43	42.05	40.46	37.96
50th	38.79	40.43	37.25	37.27	37.77	33.88
60th	39.44	52.63	35.05	41.47	48.64	45.08
70th	39.42	46.63	38.09	43.69	45.91	36.60

All values in micromicro grams.

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	1	5.713	5.713	0.033 n.s.
Between days	6	441.726	73.621	0.428.n.s.
Error	34	5846.005	171.941	
Total	41	6293.444		

n.s. Not significant.

TABLE-19 Showing the Mean corpuscular haemoglobin concentration in A.haemulosa infection in White Plymouth Rock compared with the negative controls.

DAYS	GROUP I			GROUP II		
	1	2	3	4	5	6
14th	28.66	31.85	29.54	27.33	22.40	29.04
29th	29.28	36.11	33.91	26.00	29.60	29.54
36th	32.22	31.20	29.04	31.53	29.54	27.14
43rd	30.00	25.90	38.94	31.53	31.81	35.65
50th	37.50	33.63	31.66	31.53	40.80	29.04
60th	34.40	40.90	35.78	33.13	39.13	41.05
70th	43.15	42.85	36.00	43.33	34.61	37.27

All values in percentage.

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	1	23.655	23.655	1.799 n.s.
Between days	6	602.797	100.466	7.644 **
Error	34	446.868	13.143	
Total	41	1073.320		

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

TABLE-20 Showing the total Leucocyte count in *A.hamulosa* infection in White Plymouth Rock compared with the negative controls.

DAYS	GROUP I			GROUP II		
	1	2	3	4	5	6
14th	17.55	34.55	12.00	25.11	9.11	25.88
29th	26.00	41.77	37.32	34.22	29.00	26.66
36th	42.00	29.55	26.44	26.22	24.44	24.44
43rd	22.66	31.55	35.33	25.55	34.88	38.00
50th	43.33	45.33	42.22	44.66	45.33	42.44
60th	42.00	36.66	39.33	29.55	46.22	35.33
70th	34.00	38.00	42.22	36.88	35.11	28.00

All values in thousands/cumm. of blood.

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	1	66.327	66.327	1.768 n.s.
Between days	6	1945.230	324.205	8.642 **
Error	34	1275.434	37.512	
Total	41	3286.991		

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

TABLE-21 Showing the percentage of Heterophils in *A.hamulosa* infection in White Plymouth Rock compared with the negative controls.

DAYS	GROUP I			GROUP II		
	1	2	3	4	5	6
14th	29.0	28.0	21.0	28.0	35.0	26.0
29th	38.0	52.0	49.0	26.0	32.0	24.0
36th	44.0	35.0	71.0	44.0	35.0	42.0
43rd	53.5	61.0	62.0	32.5	53.5	28.0
50th	48.0	53.0	53.5	48.0	26.0	38.5
60th	44.5	43.5	48.5	38.5	39.5	36.5
70th	42.5	52.5	45.5	43.0	32.0	37.0

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	1	1377.149	1377.149	21.107 **
Between days	6	1414.452	235.742	3.613 **
Error	34	2218.310	65.244	
Total	41	5009.911		

** Significant at 1% level.

TABLE-22 Showing the percentage of Eosinophils in *A.hannaloa* infection in White Plymouth Rock compared with the negative controls.

DAYS	GROUP I			GROUP II		
	1	2	3	4	5	6
14th	2.0	5.0	5.0	4.0	3.0	5.0
29th	9.0	8.0	7.0	1.0	1.0	3.0
36th	4.0	4.0	8.0	1.0	0.0	3.0
43rd	7.0	6.0	2.5	1.0	2.0	1.0
50th	9.5	4.5	5.5	2.0	2.5	1.0
60th	5.5	5.5	4.0	1.5	3.5	1.0
70th	4.5	5.0	3.0	2.0	0.5	2.0

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	1	128.625	128.625	47.288 **
Between days	6	16.321	2.720	0.943 n.s.
Error	34	98.084	2.884	
Total	41	243.030	2.884	

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

TABLE-23 Showing the percentage of Lymphocytes in A.humuloss infection in White Plymouth Rock compared with the negative controls.

DAYS	GROUP I			GROUP II		
	1	2	3	4	5	6
14th	64.0	63.0	59.0	62.0	61.0	64.0
29th	44.0	33.0	41.0	68.0	62.0	65.0
36th	43.0	56.0	18.0	43.0	54.0	45.0
43rd	35.0	26.0	32.5	57.5	42.5	66.5
50th	30.5	33.0	36.5	42.5	64.5	57.0
60th	42.0	41.5	41.5	57.5	53.5	53.5
70th	46.0	33.0	46.0	47.5	63.5	56.0

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	1	2430.482	2430.482	32.245 **
Between days	6	1627.821	271.303	3.599 **
Error	34	2562.727	75.374	
Total	41	6621.030		

** Significant at 1% level.

TABLE-24 Showing the percentage of Monocytes in *A.hannuloza* infection in White Plymouth Rock compared with the negative controls.

DAYS	GROUP I			GROUP II		
	1	2	3	4	5	6
14th	3.0	2.0	3.0	3.0	0.0	3.0
29th	3.0	1.0	1.0	0.0	0.0	0.0
36th	1.0	3.0	1.0	3.0	1.0	0.0
43rd	0.0	5.0	1.0	0.0	0.0	0.5
50th	1.5	4.5	0.0	1.0	1.0	0.0
60th	2.0	4.5	1.0	0.0	0.0	0.0
70th	3.0	3.5	1.5	0.5	1.0	0.5

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	1	22.881	22.881	12.884 **
Between days	6	8.369	1.394	0.845 n.s.
Error	34	56.036	1.648	
Total	41	87.286		

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

TABLE-25 Showing the percentage of Basophils in *A.hamulosa* infection in White Plymouth Rock compared with the negative controls.

DAYS	GROUP I			GROUP II		
	1	2	3	4	5	6
14th	2.0	2.0	3.0	3.0	1.0	2.0
29th	6.0	6.0	2.0	5.0	5.0	8.0
36th	6.0	2.0	2.0	9.0	10.0	10.0
43rd	4.5	2.0	2.0	9.0	2.0	4.0
50th	10.5	5.0	4.5	6.5	6.0	3.5
60th	6.0	5.0	5.0	2.5	3.5	9.5
70th	5.0	6.0	4.0	7.0	3.0	4.5

Analysis of variance Table

Source	df	SS	MSS	F
Between groups	1	15.482	15.482	2.766 n.s.
Between days	6	81.738	13.623	2.433 *
Error	34	190.310	5.597	
Total	41	187.530		

* Significant at 5% level
n.s. Not significant.

TABLE-26 Showing the total, differential and absolute counts of blood leucocytes in White Plymouth Rock infected with *A.hemolosa*.

	14th	29th	36th	43rd	50th	60th	70th
			GROUP-I				
Total Leucocytes	21,370	35,036	32,666	29,851	43,628	39,332	38,073
Heterophils	29/6,197	46/16,116	50/16,333	59/17,612	51.5/22,468	45.5/17,896	47/17,894
Lymphocytes	62/13,249	39/13,664	39.5/12,903	31/9,253	33.5/14,615	42/16,519	41.5/15,800
Monocytes	2/427	2/700	2/653	2/597	2/872	2.5/983	2.5/951
Eosinophils	4/854	8/2,802	5/1,633	5/1,492	6.5/2,835	5/1,966	4/1,522
Basophils	3/641	5/1,752	3.5/1,143	3/895	6.5/2,835	5/1,966	5/1,903
			GROUP-II				
Total Leucocytes	20,036	29,962	25,036	32,814	44,147	37,036	33,332
Heterophils	29/5,810	27/8,089	40/10,014	38/12,469	37.5/16,555	38/14,073	37.5/12,499
Lymphocytes	62/12,422	65/19,475	47.5/26,124	55.5/18,211	54.5/24,060	55/20,369	55.5/18,499
Monocytes	3/601	0/0	1.5/824	0.5/164	0.5/220.5	0/0	0.5/166
Eosinophils	4/801	2/599	1.5/824	1.0/328	2/882	2/740	1.5/500
Basophils	2/400	6/1,797	9.5/5,224	5/1,640	5.5/2,428	5/1,851	5/1,666

Each figure represents the average count of 3 birds
Nominator and denominator represents differential and absolute counts.

ACUARIA HAMULOSA INFECTION - WHITE PLYMOUTH ROCK

CHART-16

HAEMOGLOBIN

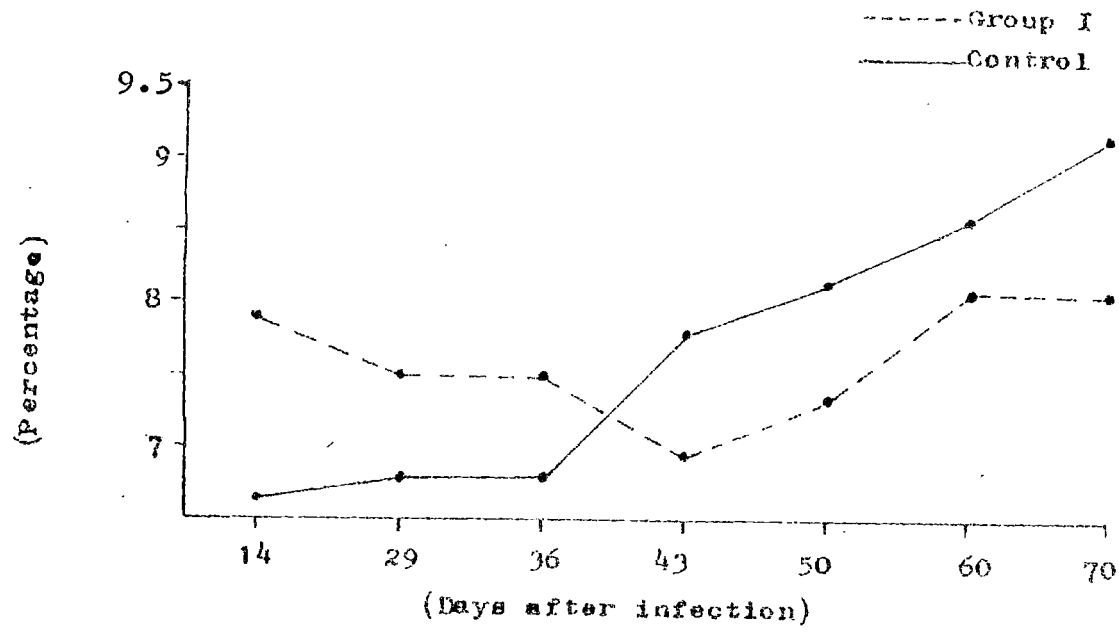
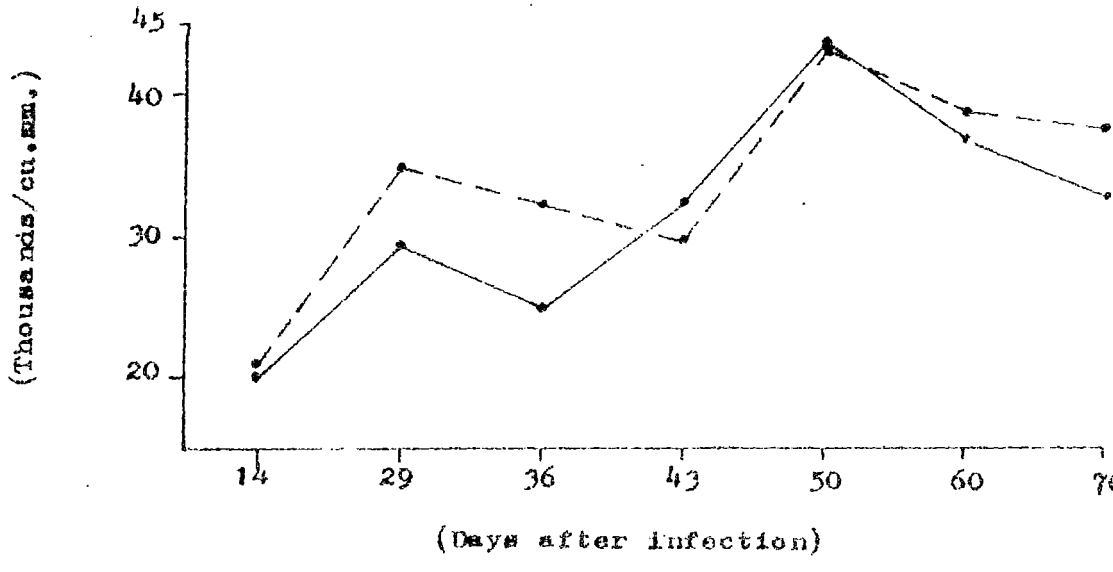


CHART-17

TOTAL LEUCOCYTES



ACUARIA HAMULOSA INFECTION - WHITE PLYMOUTH ROCK

CHART-18

HETEROPHILS

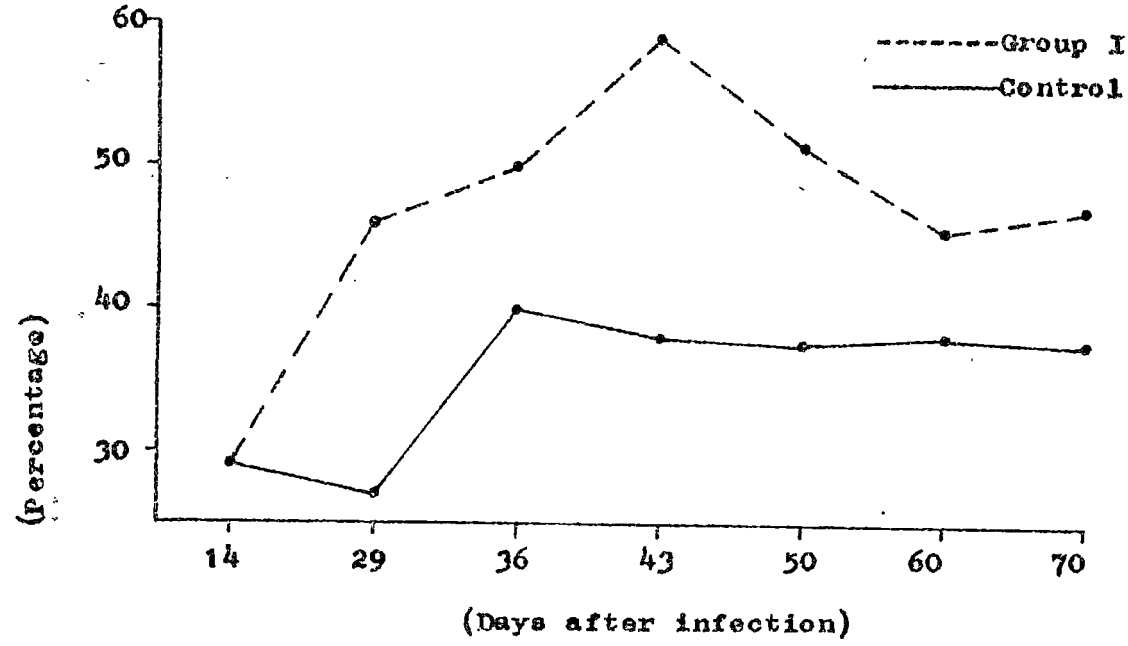
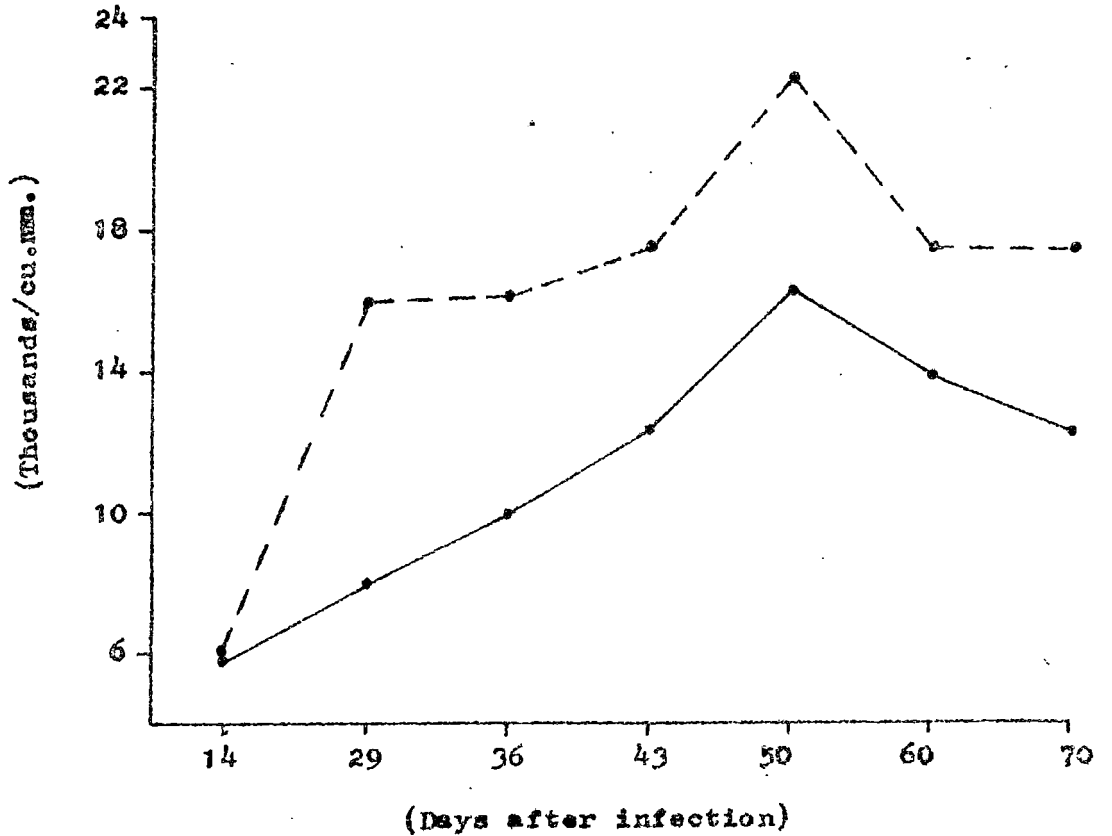


CHART-19

HETEROPHILS ABSOLUTE



ACUARIA HAMULOSA INFECTION - WHITE PLYMOUTH ROCK

CHART-20

LYMPHOCYTES

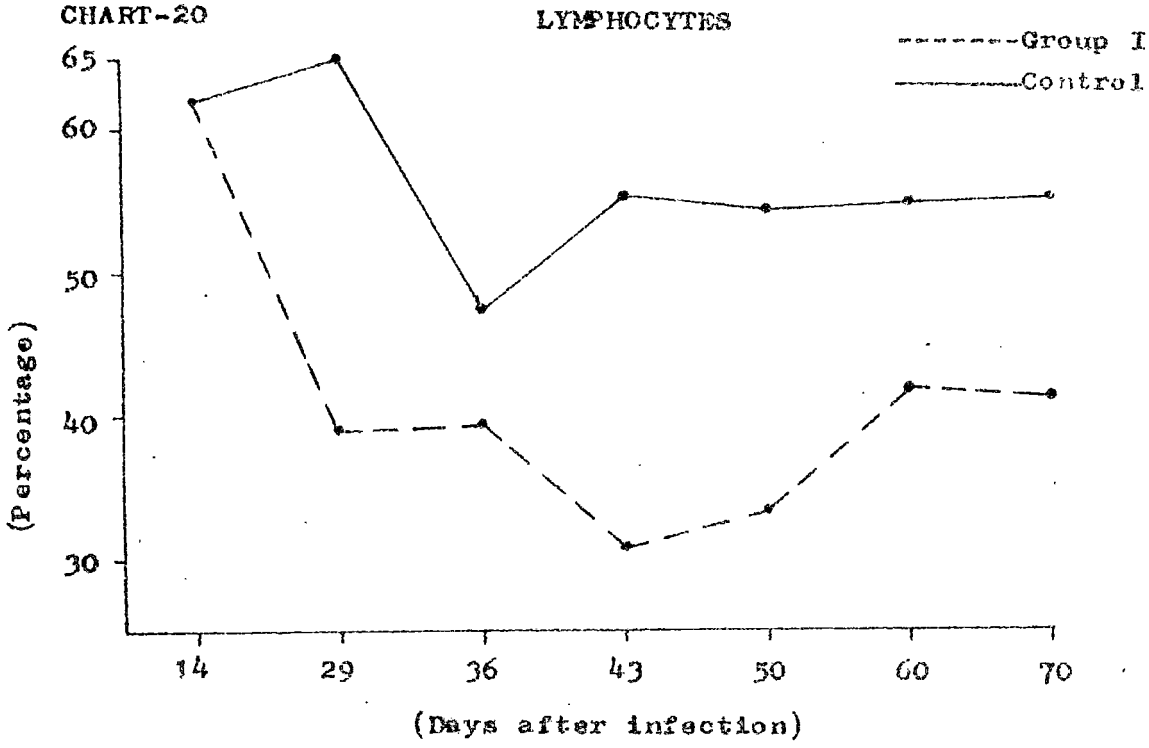
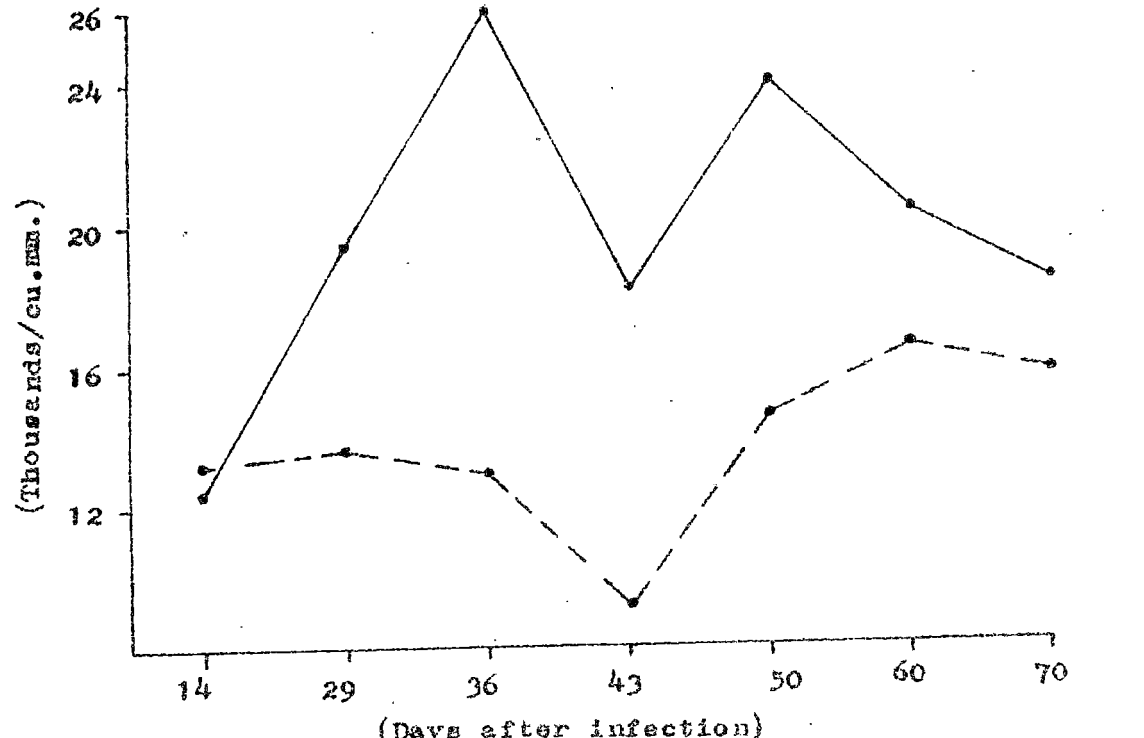


CHART-21

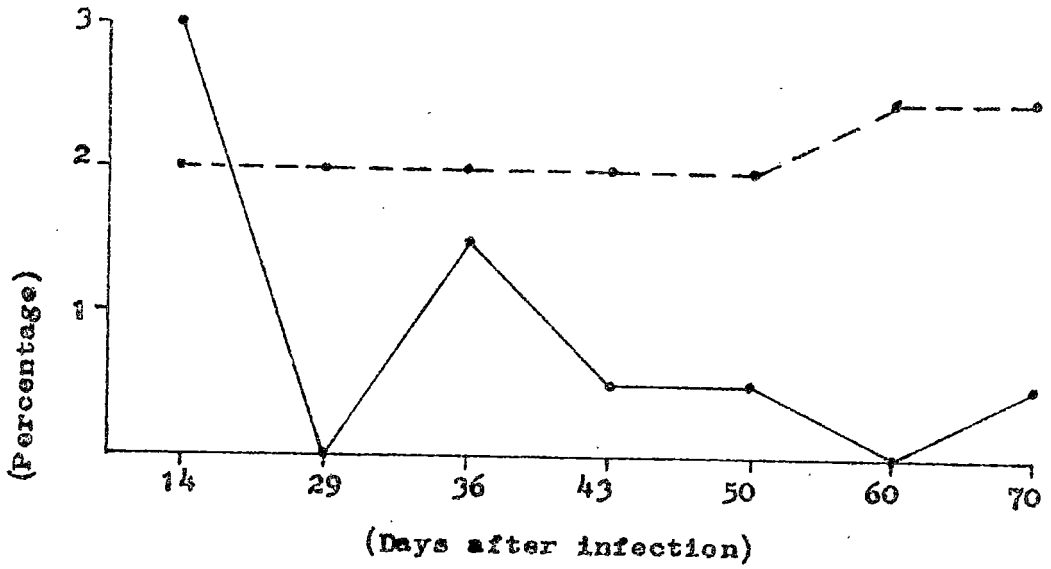
LYMPHOCYTES ABSOLUTE



ACUARIA HAMULOSA INFECTION - WHITE PLYMOUTH ROCK

CHART-22

MONOCYTES

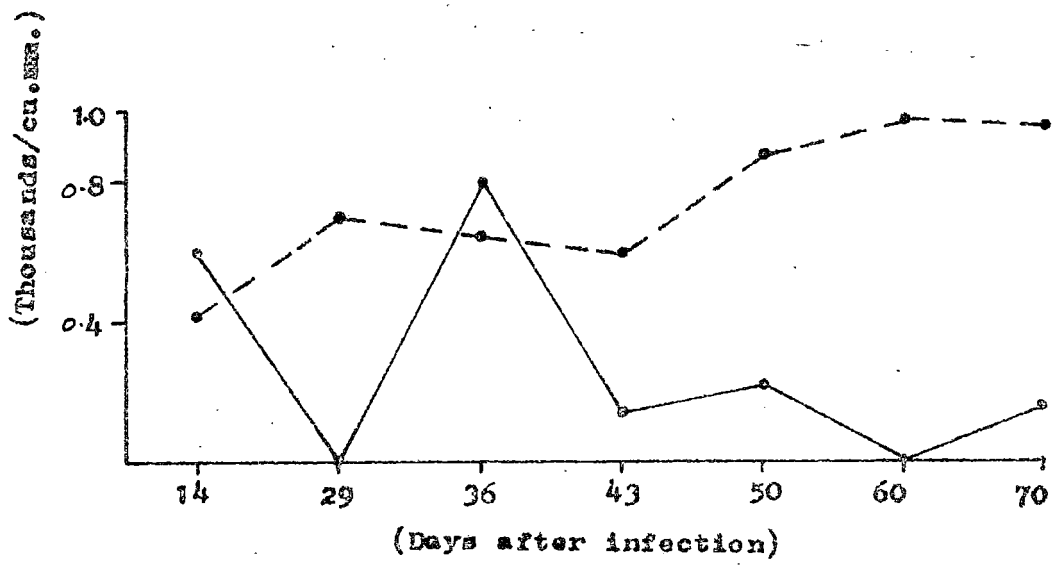


-------Group I

———Control

CHART-23

MONOCYTES ABSOLUTE



ACUARIA HAMULOSA INFECTION - WHITE PLYMOUTH ROCK

CHART-24

EOSINOPHILS

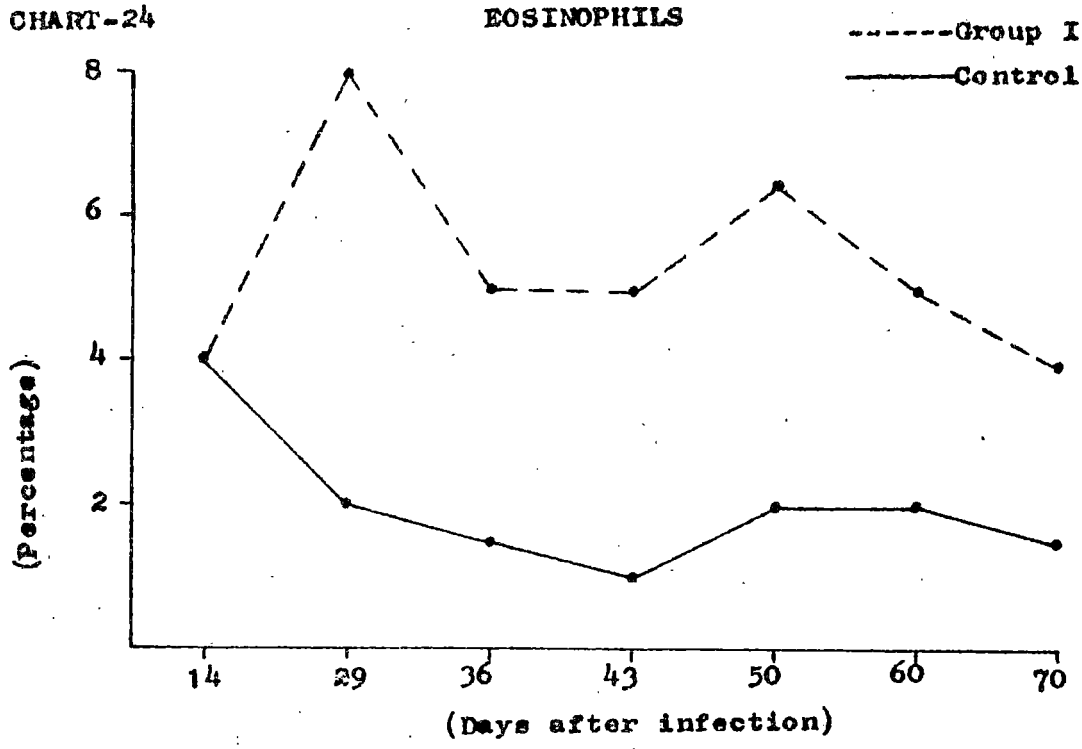
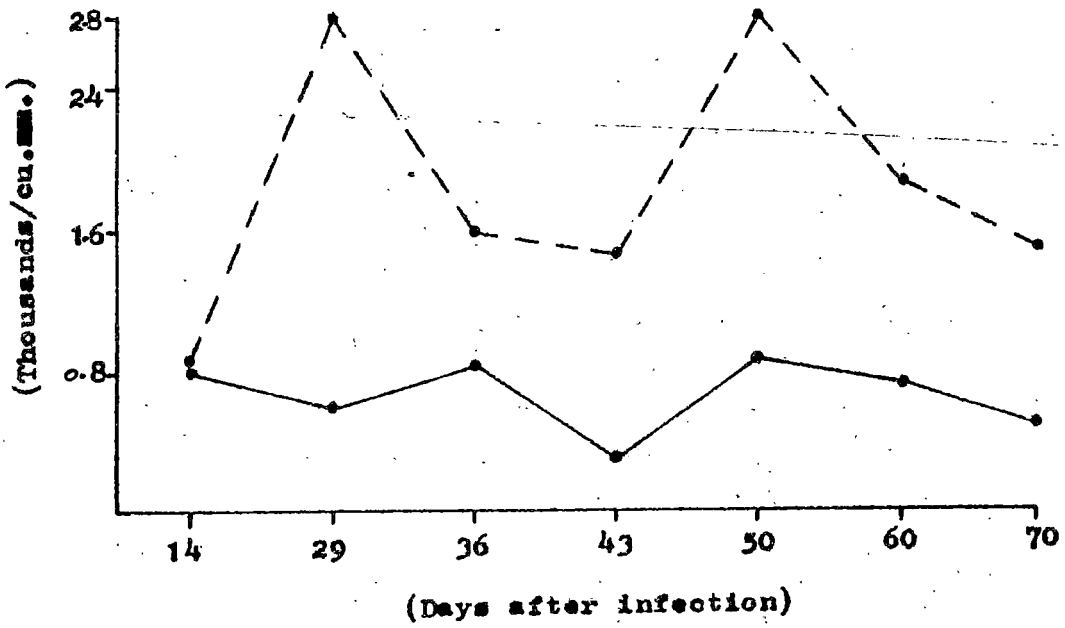


CHART-25

EOSINOPHILS ABSOLUTE



ACUARIA HAMULOSA INFECTION - WHITE PLYMOUTH ROCK

CHART-26

BASOPHILS

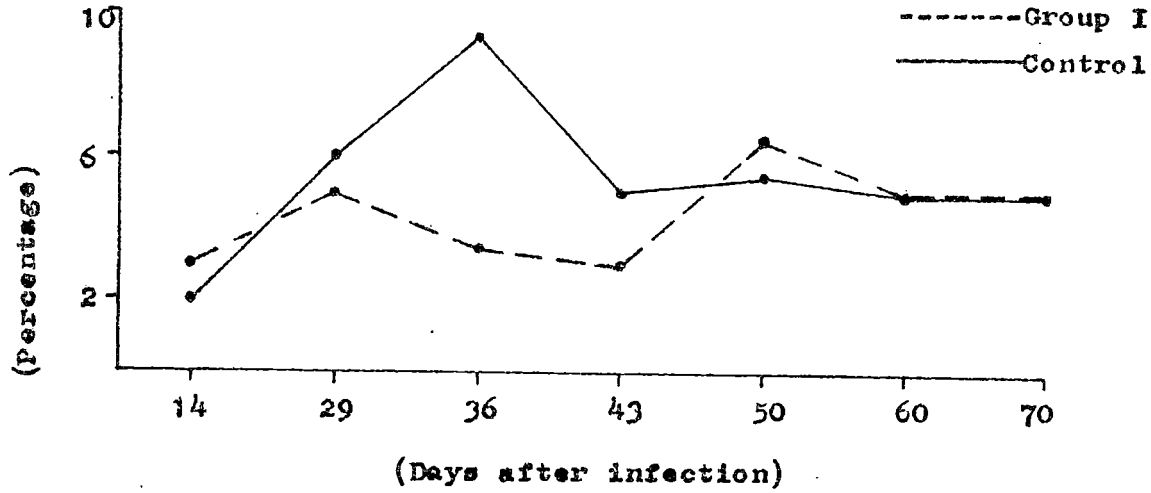
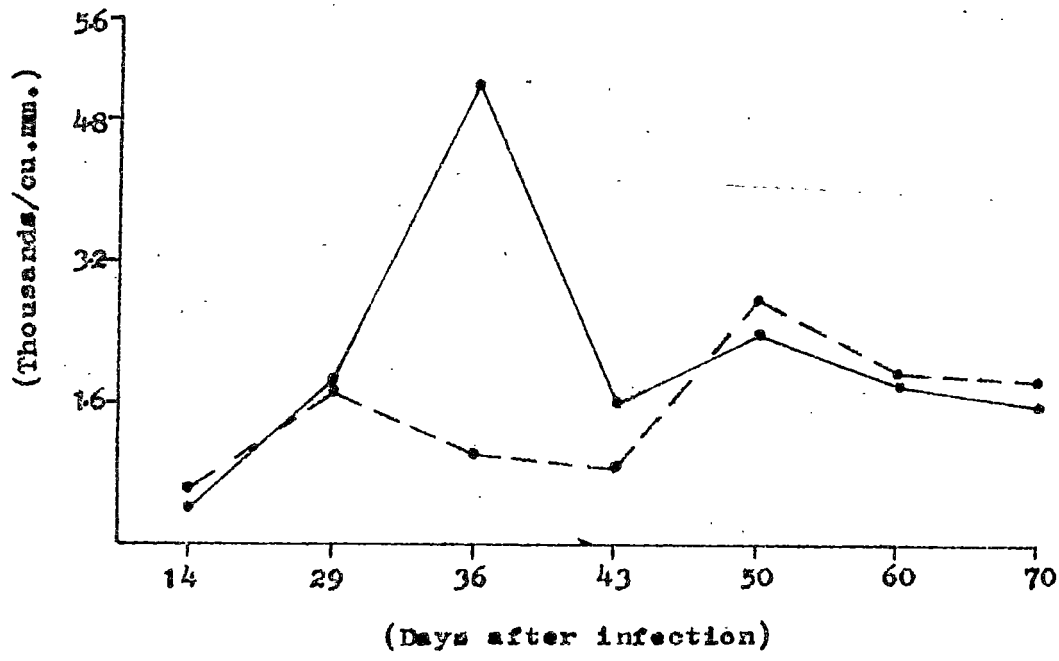


CHART-27

BASOPHILS ABSOLUTE



CLINICAL SYMPTOMS

During the first three weeks of infection the chicks exhibited no noticeable symptoms of the disease. However as the infection progressed, from the 5th week onwards, the birds became unthrifty. Anaemia was evident from the 7th week post infection in all the infected birds. The experimental birds had lost considerable amount of their body weight by 7th week in case of White Plymouth Rock and by 10th week in the case of White Leghorn respectively. In layers the sexual maturity was delayed and there was decline in the rate of egg production.

WEIGHT GAIN

The average weight of the experimental White Plymouth Rock birds showed a significant decrease ($P < 0.01$) compared to the negative controls.

On a further analysis, the Group I (average number of worms established 15.66) showed a decrease of 28.30% ($P < 0.05$) and 28.50% ($P < 0.01$) on the 7th and 8th week

post infection respectively.

Thus the weight gains of the experimental group was found to be poor during the 7th and 8th week of infection. On the 70th day post infection the average decrease in live body weight was found to be 276.67 grams (27.57%) compared to the negative controls, which showed an average live body weight of 1003.33 grams.

Since the White Plymouth Rock strain of birds were used mainly for meat purpose, the loss of income due to poor weight gain was calculated on the basis of the current price of live body weight (Rs.10/Kg). The loss was Rs.2.76 compared to the negative controls.

Whereas, the average weight of the experimental White Leghorn birds showed no significant difference compared to the negative controls.

However, on a further analysis, the Group I (average number of worms established 47.50) showed a decrease of 11.85% (not significant) and 22.28% ($P < 0.05$) on the 9th and 10th week of post infection respectively.

In respect of Group II (average number of worms established 36.25) the decrease was 11.44% (not significant) on the 10th week post infection.

Thus the weight gains of the experimental groups were found to be poor during the 9th and 10th week post infection. On the 70th day post infection the average decrease in the live body weight was found to be 185 grams (22.28%) in the Group I and 95 grams (11.44%), in Group II compared to the negative controls, which showed an average live body weight of 830 grams.

The data are represented graphically in charts 28 & 29.

SEXUAL MATURITY AND INTENSITY OF LAYING

The experimental groups showed a delay in the attainment of sexual maturity compared to the negative controls, which laid its first egg on the 182nd day.

The delay was 13 days in respect of Group I (average number of worms established 16) and 21 days in respect of Group II (average number of worms established 33).

The rate of egg production was poor with a longer pause in both the experimental groups. During the observed period of 30 days, the total number of eggs produced by Group I was 12 and by Group II was only 6. Whereas the eggs produced by the negative control was 26.

The egg record of experimental and control birds are given in Table-29.

FEED EFFICIENCY

The quantity of feed in Kilograms required to produce a dozen of eggs was calculated for the female birds, since the White Leghorn strain was used mainly for egg production. The feed efficiency of Group I (number of worms established 16) was 2.49, and of Group II (number of worms established 53) was 3.72. The feed efficiency in the case of the control was found to be 1.7.

Thus the feed efficiency of the experimental birds was low when compared to the negative control.

Calculating the loss of income on the basis of the

current price of feed (Rs.1.65/Kg), Group I required Rs.1.30 worth of feed, while the Group II needed Rs.1.68 worth of feed, more than control to produce a dozen of eggs.

PREPATENT PERIOD

The prepatent period of infection in case of White Leghorn birds Group I (average number of worms established 47.50) was found to be in from 73 to 87 days (average 79.5 days) and in the case of Group II (average number of worms established 36.25) was in from 78 to 82 days (average 81.25 days). And in the case of White Plymouth Rock, the prepatent period of infection was found to be 90 days.

Thus the prepatent period of infection was found to be not influenced by the number of worms established. The data are presented in the Tables 27 & 28.

PERCENTAGE OF ESTABLISHMENT

During the present study, all the experimental birds were infected with infective larvae (encysted third stage)

aged 27 days, dissected out from grasshoppers as outlined under, materials and methods. Heavy infection of grasshoppers could be obtained by the above method (Fig.1). A heavily infected grasshopper often yielded over 4000 larvae.

It was observed during the present studies that there was little correlation between the number of larvae administered and the final percentage of establishment. Particularly in the case of White Rocks the percentage of worms established widely varied. In some preliminary attempts only 30% of the birds showed eggs at the end of prepatent period. In the rest of the birds minute calcarious nodules in the muscle were seen and no live larvae could be deducted. For final experimentation therefore, a batch of White Rocks were infected and data pertaining to various parameters were collected. For statistical analysis data from only those birds which showed eggs at the end of prepatent period or developing live larval stages were used.

The worms had a tendency to accumulate in masses towards the soft musculature separating the two halves of the gizzard, where they produce eventually nodules. In White Leghorn birds, when a large number of worms developed in the gizzard musculature, a few parasites could be found even under serosa.(Fig.2).

In White Leghorn, the Group I (infected with 100 larvae each) the average number of worms recovered was 47.50 (21.75% males, and 25.75% females) giving an establishment percentage of 47.50%. Whereas in Group II (infected with 400 larvae each) the average number of worms recovered was 36.25 (6.91% males and 5.16% females) giving an establishment percentage of 12.08%.

In White Plymouth Rock the Group I (infected with 400 larvae each) the average number of worms recovered was 15.66(2.0% males and 1.91% females) giving an establishment percentage of 3.91%.

The data are presented in Tables 27 & 28.

TABLE-27 Showing the details of prepatent period and percentages of establishment of *A.hamuloea* in White Leghorn.

Chick number	Number of infective larvae given	Prepatent period in days	Number of worms developed			Percentage of establishment
			Male	Female	Total	
1. (Male)	100	80	25	28	53	53%
2 (Female)	100	78	13	3	16	16%
3 (Male)	100	73	33	38	71	71%
4 (Male)	100	87	16	34	50	50%
Total	400		87	103	190	47.50%
5 (Male)	400	78	--	10	10	2.5%
6 (Female)	400	80	41	12	53	13.25%
7 (Male)	400	81	23	20	43	10.75%
8 (Male)	400	82	19	20	39	9.75%
Total	1200		83	62	145	12.08%
9 (Female)	Nil	All birds remained negative.				
10 (Male)	Nil					
11 (Male)	Nil					
12 (Male)	Nil					

TABLE-28 Showing the details of prepatent period and percentage of establishment of A.hammulosa in White Plymouth Rock

Chick number	Number of infective larvae given	Prepatent period in days	Number of worms developed.			Percentage of establishment.
			Male	Female	Total	
1	400	--	1	1	2	0.50%
2	400	--	1	-	1	0.25%
3	400	90	22	22	44	11.00%
Total	1200		24	23	47	3.91%
4	Nil	All birds remained negative				
5	Nil					
6	Nil					

ACUARIA HAMULOSA INFECTION EFFECT ON WEIGHT GAIN

CHART-28

WHITE LEGHORN

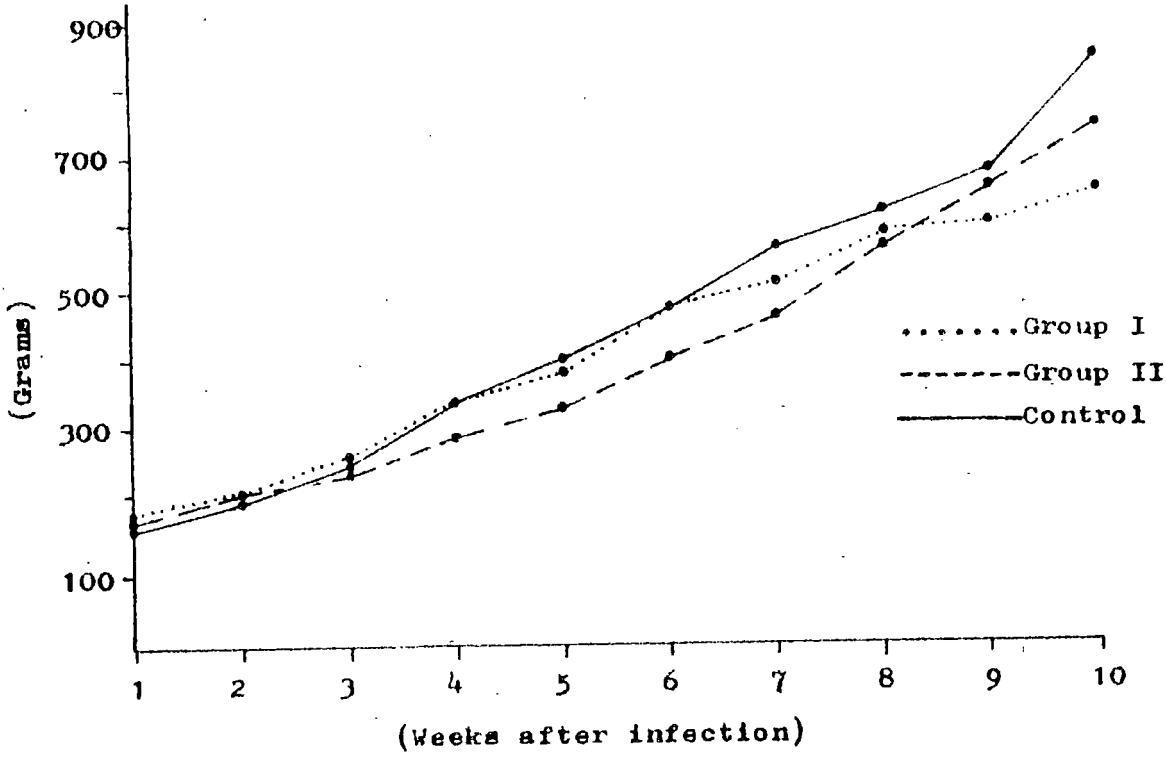


CHART-29

WHITE PLYMOUTH ROCK

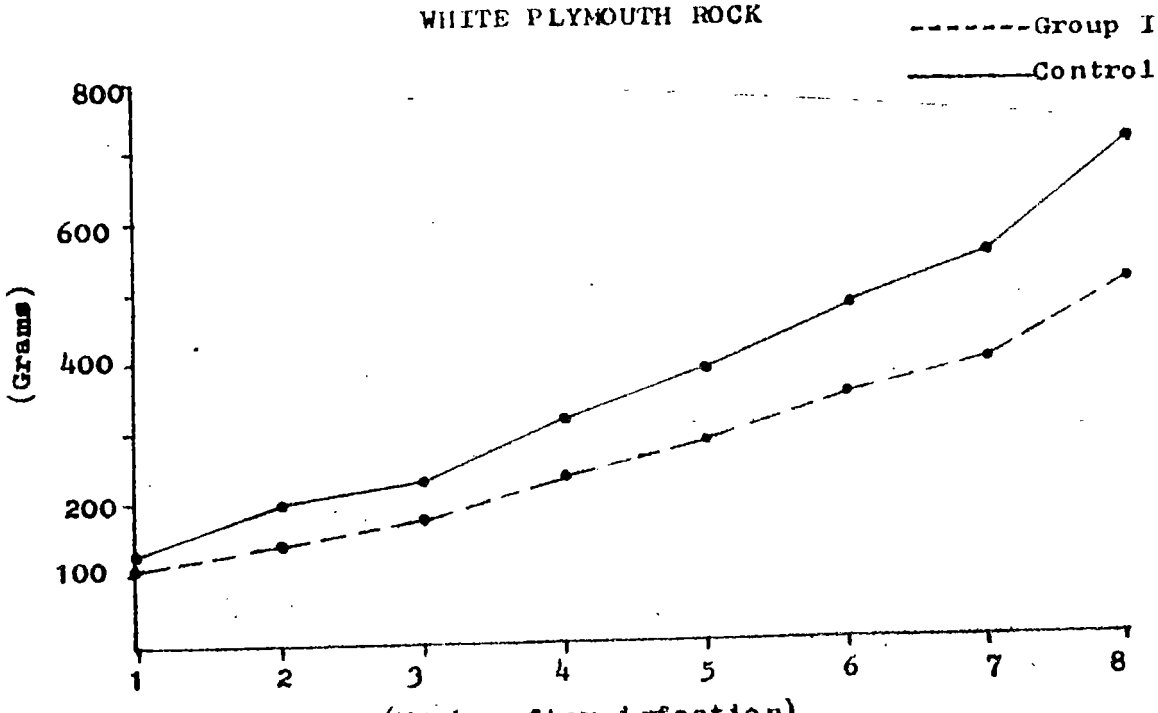


TABLE - 29 EGG RECORD OF EXPERIMENTAL AND CONTROL BIRDS

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	TOTAL
GROUP I														X	X					X	X	X	X		X	X	X	X	X	X	12
GROUP II																						X				X	X	X	X	X	6
GROUPIII	X		X	X		X	X		X	X	X	X	X	X	X	X	X	X	X	X		X	X	X	X	X	X	X	X	X	26

HISTOPATHOLOGICAL STUDIES

To study the histopathological changes produced by the various developmental stages of Acuaria hamulosa in the gizzard of the host, chicks were infected with 200 larvae and were slaughtered on the 2nd, 6th, 10th 14th (corresponding to the third stage) 18th, 22nd, 29th, 36th, 43rd (corresponding to the fourth stage) 50th, 60th and 70th day (corresponding to the adult stage) post infection. The changes observed were as follows.

GROSS LESIONS

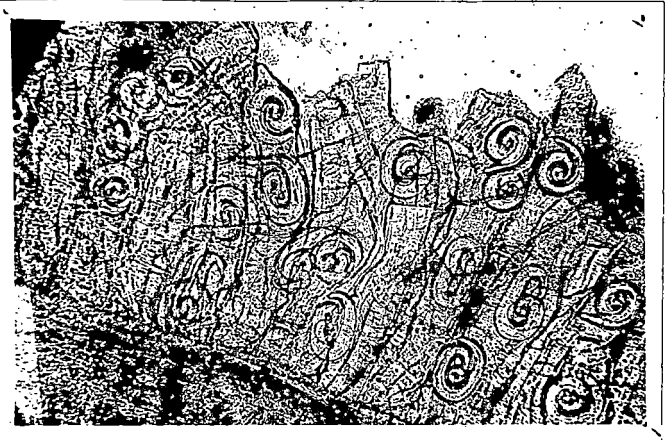
No macroscopic lesions were detectable upto the 18th day post infection.

On the 22nd, 29th and 36th day post infection, erosion of the keratin layer in the form of ulcers were noted, at the anterior and posterior sacs of the gizzards. Petechial haemorrhages were seen on the mucosa of the gizzard, underneath the keratin layer.

On the 43rd day post infection paddy grain sized soft nodules were found in the musculature of the anterior and posterior sacs of the gizzard. The keratin coat was eroded due to the migratory juveniles. The infected area presented a dirty brownish discolouration and could easily be identified from normal healthy surface of the keratin layer.

On the 50th, 60th and 70th day post infection a large number of soft nodules were found in the musculature of the anterior and posterior sacs. Owing to haemorrhage and accumulation of inflammatory exudate in the worm tracts, the infected areas (nodules) presented a purplish colour, which could be discerned even from the serous surface. The keratin layer was greatly thickened with areas of sloughing and necrosis, and could be easily peeled off. On the 70th day post infection the nodules attained their maximum size, which showed out as hemispherically bulging areas with surface discolouration.

1



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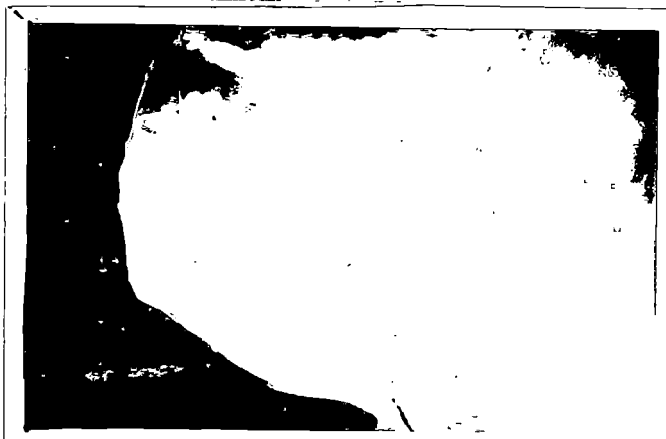

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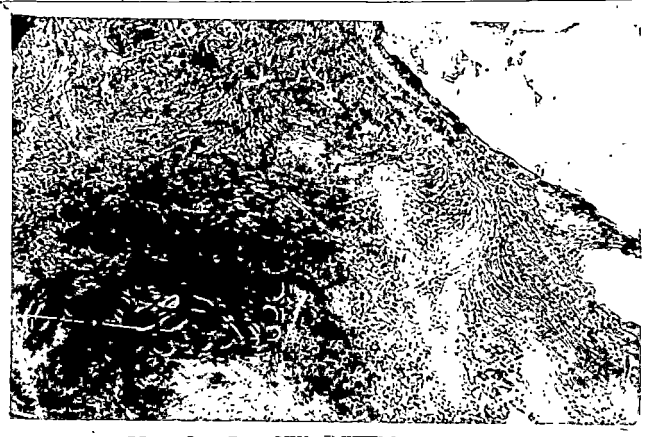

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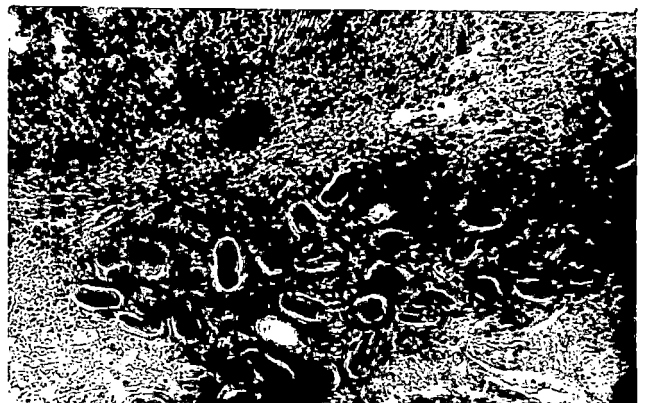

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10th day post infection

There was no evidence of necrosis, in the gizzard of the chick slaughtered on the 10th day post infection. But the lamina propria showed severe defuse infiltration with heterophils, which extended even into the muscular coat. Moderate intermuscular oedema was observed in the muscular coat, which was first seen on the 6th day post infection.

14th day post infection

On the 14th day post infection, there was desquamation of the keratin layer with the formation of ulcers. Severe defuse infiltration with heterophils were also evident. Focal non-suppurative myositis was evident in the inner muscular coat. Larval stages were seen under the keratin layer in the superficial mucosa (Fig.6).

18th day post infection

On the 18th day post infection, the keratin layer showed small focal erosions and necrosis. Diffuse

infiltration of lymphocytes were seen in the mucosa. Larval stages were seen in the lamina propria (Fig.7&8).

22nd day post infection

On the 22nd day post infection, the keratin layer showed focal ulceration and necrosis. Defuse infiltration of heterophils and lymphocytes were evident in the lamina propria. The muscular coat was oedematous and showed focal infiltration with heterophils and lymphocytes.

29th and 36th day post infection.

On the 29th day post infection there was defuse infiltration of heterophils and lymphocytes within the mucosa. Below the muscularis mucosa, the muscular tissue showed dense infiltration with heterophils and lymphocytes and focal areas of necrosis could be seen. Larval stages were seen embeded in the interstitial tissue of muscular coat (Fig.9&10). Invasion into the muscular tissue therefore, commences from about the

29th day post infection. Migrating tracks were descernible even upto the outer longitudinal layer of the musculature in some cases. These tracks were filled with necrotic material, which comprised of tissue debris, disintegrating erythrocytes, lymphocytes and a few heterophils. Immediately surrounding the necrotic masses were seen several foreign body giant cells (Fig.11). Mononuclear infiltration and a mild degree of fibrosis was also noticed around the tracks. On the 36th day post infection, the histological changes were same as seen on the 29th day post infection.

On the 43rd day post infection necrotic and infiltrative changes were more pronounced with extensive myositis of the gizzard musculature. The developing juveniles were found in the deeper layers of musculature. The tracks produced by them were well limited by fibrous encapsulation.(Fig.12) obviously as an attempt on the part of the host to contain the parasite.

9

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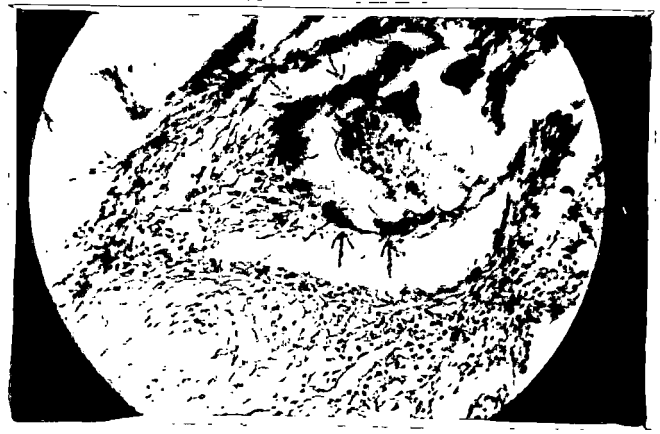

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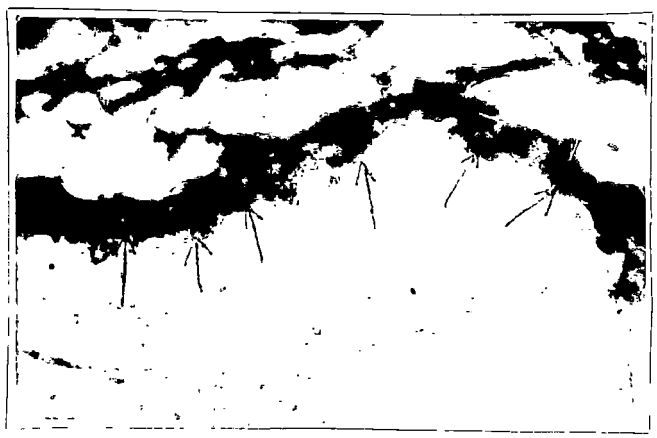

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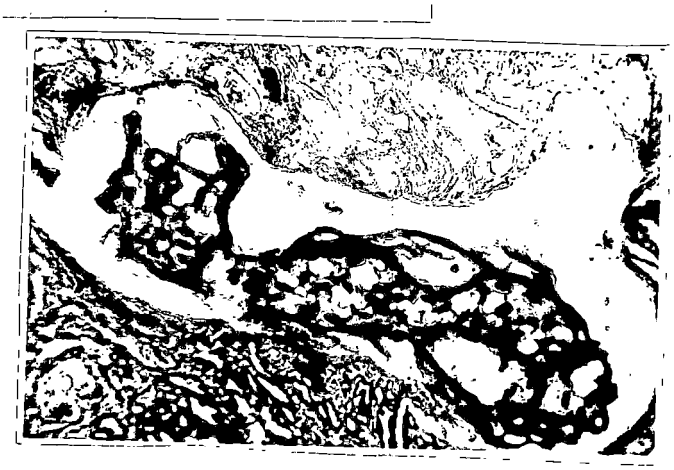


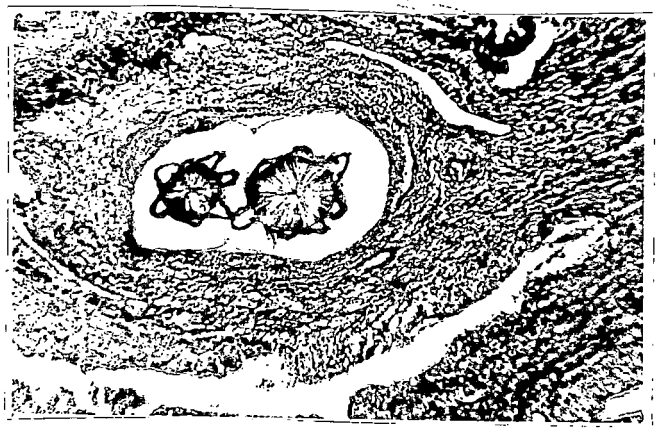
50th day post infection

On the 50th day post infection the muscular coat showed the parasite, encircled by lymphocytes plasma cells and fibroblasts (Fig.13 & 14). The essential features of the migratory track of the parasites within the muscle fibres were same as those seen on the 29th day. The fibrosis around the track were more pronounced with a more or less continuous chain of giant cells immediately following the necrotic mass (Fig.15). In occasional areas the parasites were also seen. Extensive oedema and sub-acute to chronic focal myositis were also invariably association with the worm tracks. At other adjacent areas in the muscular coat, heavy infiltration with mononuclears and capillary haemorrhage were noticed (Fig.16).

60th day post infection

On the 60th day post infection, parasites were seen embeded in the muscular coat (Fig.17). The fibrous tissue reaction was far advanced. Infiltration with lymphocytes

17



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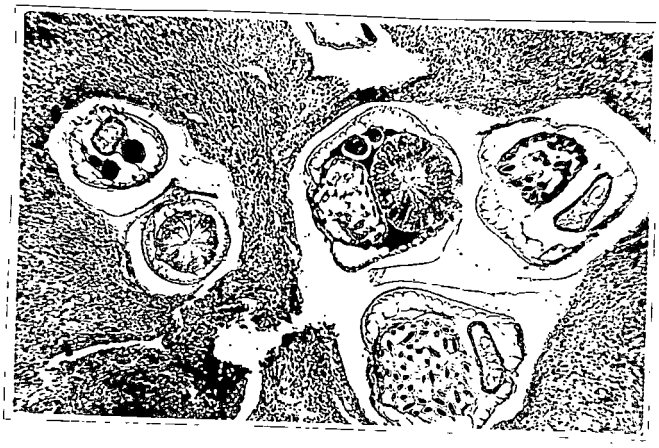

was as heavy as observed on the 50th day post infection. Parts of the parasite bodies were also seen projecting beyond the mucosa into the lumen of the organ. Developing eggs could also be seen in the uteri of the worms, indicating that the worms have established communicating channels with the lumen of the gizzard evidently for the discharge of eggs.

70th day post infection

On the 70th day post infection the histopathological lesions were essentially the same as observed on the 60th day post infection. Eggs with advanced stages of development could be seen in the uteri of worms. The overall picture was that of a chronic granulomatous change surrounding the worm tracks (Fig.18 & 19).

DISCUSSION

DISCUSSION

From the available literature it appears that no controlled studies seems to have been made previously on the pathogenicity of Acuaria hamulosa to chicken. The present investigation was therefore, a preliminary attempt to assess the effect of the parasite on its host under controlled experimental conditions.

Unlike, Tetrameres mohtedai and Acuaria spiralis, predictable establishment of worms in the experimental hosts was not observed in the case of A.hamulosa. This was perhaps due to encapsulation of the larvae, especially the third stage juveniles. Within the mucosa as a part of host response (Fig.3). Many of the larvae perhaps, perish in the process of encapsulation. There was, therefore, no satisfactory correlation between the infective dose and the final percentage of worms established. To cite specific instances, the average number of worms developed to adult stage in White Leghorn chicks that received 100 infective larvae was 47.50; whereas only 12.03 worms established when the infective dose was raised to 400 larvae. In White Rock

breed only 3.91 worms established with an infective dose of 400 larvae. It is probable that the response of the host and consequent rejection of infection during the early stages is directly proportional to the dose of infective larvae. Ackert et al.(1931) also observed a similar phenomina with Ascaridia lineata of chicken. Michel (1963) explains that "the proportion of large doses of infective larvae or eggs which become established is often smaller than that of small doses and in some cases the evidence is clear that, it is initial establishment that is affected and not the rate of subsequent loss".

Ramaswamy (1977) had observed a normocytic normochromic anaemia in the initial period, followed by a hypochromic anaemia in later stage of controlled T. mohtedai infections in fowls. Chandrasekharan (1977) had observed microcytic hypochromic anaemia and leucocytosis in controlled infection of Amidostomum skrjabini and Epomidiostomum uncinatum in ducks. He noticed, anaemia, weakness, in-appetance, and listlessness as clinical symptoms of Amidostomiasis and Epomidiostomiasis in ducks.

Deo (1964), Gardiner (1964), Morgan & Hawkins (1953) Wehr (1943), Levine (1969) and Soulsby (1968) have stated that, when present in small numbers Acuaria hamulosa do not cause any noticeable effect on the health of the birds, but in heavy infestations the worms produce emaciation, droopiness, weakness and anaemia. Lindsey (1968) has reported symptoms of wasting, anaemia and general weakness in poultry infected with A.hamulosa. In the present investigation, the chicks exhibited no noticeable symptoms during the first three weeks of infection; but as the infection progressed the birds became unthrifty. Anaemia was evident from the 43rd day in White Plymouth Rock, and from 50th day post infection in White Leghorn birds. These results are in agreement with those of the above authors.

Kaushik and Son (1978) have reported leucocytosis with corresponding absolute heterophilia during the initial period of controlled Ascaridia galli infections in chicks. They have observed absolute eosinophilia from second to fourth weeks of infection. Hwang (1964) has reported a marked relative heterophilia

and monocytosis with a corresponding lymphocytopenia in turkey poults heavily infected with Syngamus trachea larvae. Olson and Levine (1969) have reported eosinophilia and heterophilia in controlled Capillaria columbae infection in chickens. Ramaswamy (1977) has observed an initial eosinophilia and leucocytosis in the invasive period and a hetrophilia in the later period of T.mohtedai infection in fowls. He also observed leucocytosis and eosinophilia in the initial period and a leucopenia heterophilia and lymphocytopenia in the later stages of monospecific A.spiralis and dispecific A.spiralis and T. mohtedai infections. Chandrasekharan (1977) has observed leucocytosis in Amidostomiasis and Epomidiostomiasis in ducks.

In the present investigation, during the invasive period an initial leucocytosis from 14th day to 36th day post infection was observed in both the breeds of chickens, which was an agreement with the findings of Kaushik and Sen (1978), Ramaswamy (1977). A mild degree of persistent eosinophilia was observed in both the breeds of chicks, throughout the experimental period of 70days; whereas, it is observed from the earlier reports, that generally eosinophilia characterises the initial response of the host to the

Parasite. During the later stages eosinophilia is reported to be less evident. A sustained heterophilia was also observed, during the present investigation in the case of both breeds of chicks, and no appreciable leucopenia was observed during any stage of infection.

The maximum worm load noticed in natural A.hamulosa infection by Chatterji (1939) was 24 adult worms and by Manuel (1966) 22 adult worms. In the present study a maximum of 71 worms (33 males & 38 females) could be recovered, which points out that the birds may acquire very heavy infection under experimental conditions. The prepatent period of infection was found to be similar to those observed by Sundaram (1971).

The limited observations made on the weight gain of the White Rock birds indicate that as a result of A.hamulosa infection the birds lose considerable amount of their body weight. The economic loss due to decreased body weight was calculated to be Rs.2.76 per bird compared to the control birds. In White Leghorn layers the sexual maturity was delayed by 13 days in the Group I and 31 days in Group II compared to the control bird. The rate of egg productio

and the related feed efficiency of the infected layers was low when compared to the negative control. It was calculated that the birds of Group I and Group II required Rs. 1.30 and Rs.1.68 worth of more feed respectively compared with the controls to produce a dozen of eggs.

Soulsby (1965) and Levine (1968) have observed marked destruction of parasitized glands, hypertrophy of the adjacent glands, marked infiltration with lymphocytes and eosinophils, and fibrous tissue elements. Soulsby (1965) had reported haemorrhagic inflammation of the tunica propria with necrosis and destruction of the horny lining. In the present investigation also, during the initial phase of infection, the same changes were observed. Botsenko (1952) reported haemorrhagic inflammation of the mucosa between 35 to 55 days after infection with A.humulosa, and nodule formation on the 90th day. Whereas in the present investigations, erosion of the keratin layer and haemorrhagic inflammation was observed between the periods of 6th day and 22nd day post infection and nodule formation was evident on the 43rd day post infection. A zone of foreign body giant cells were regularly seen around the necrotic masses and within the worm tracks. These cells have

probably a scavenging function. Similar zone of giant cells has also been reported by Nair (1973) in artificially induced inflammatory reaction around the larvae of A.suum and T.canis in the subcutaneous tissue of chicken.

SUMMARY

SUMMARY

The pathogenic effect of A.homalosa, a spirurid parasite occuring in the musculature of gizzard of fowl was determined under controlled conditions. Two breeds of chicken namely the White Leghorn (an egg strain) and the White Plymouth Rock (a broiler strain) were used to study the effect on the host. The parameters observed were haematological changes, weight gain, sexual maturity, intensity of laying, feed efficiency and histo-pathological changes.

1. The haematological changes consisted of an initial leucocytosis from 14th day to 36th day post infection, and a mild eosinophilia throughout the observed period of 70 days in both breeds of chickens. The White Leghorn birds showed heterophilia and lymphocytophenia from 43rd to 70th day post infection; whereas the White Plymouth Rock birds showed a heterophilia and lymphocytopenia from 29th day to the 70th day post infection.

2. The weight gain of the infected White Rock birds were found to be poor on the 7th and 8th weeks after infection. The economic loss due to decreased body weight was calculated to be Rs.2.76 per

bird compared to the control bird during the observed period of 70 days.

3. In White Leghorn layers, the sexual maturity was delayed by 10 days in low worm burden and by 21 days in heavy infection compared to the control bird. The rate of egg production was also less in the infected birds. During the observed period of 30 days the total number of eggs laid were 12 and 6 respectively in low and heavey infection, whereas the control bird laid 26 eggs during this period.

4. The feed efficiency of the infected layers was low when compared to the negative controls. In low and heavy infection, Rs.1.30 and Rs. 1.68 worth of more feed respectively were required to produce one dozen eggs compared to the controls.

5. The prepatent period of infection was found to be in from 73 to 90 days (average 81 days) and was not influenced by the number of worms established.

6. Third stage juveniles became encapsulated as a result of host interaction, within 2 days after the administration of infective larvae. Many appeared to have perished since there was no correlation between the size of the infective dose and the total number of worms established.

7. The gross pathological changes of the affected gizzards included petechial haemorrhages on the mucosa and erosion of the keratin layer on the 32nd day post infection; nodule formation in the muscular coat with erosion, and thickening of the keratin layer on the 43rd day post infection; enlargement of gizzard with brownish surface discolouration, and accumulation of inflammatory exudate in the worm tracks, on the 70th day post infection.

8. The microscopic lesions observed were, during the initial phase of infection (2nd to 18th day) severe cellular reaction, haemorrhage and necrosis of the superficial mucosa; desquamation of the keratin layer, infiltration with lymphocytes and heterophils in the lamina propria; focal infiltration with heterophils and lymphocytes in the muscular coat; invasion into the muscular tissue by the parasites with migratory tracks filled with necrotic mass, surrounded by giant cells and mild degree fibrosis on the 29th day; fibrous encapsulation of parasites, pronounced necrosis and extensive myositis on the 43rd day; pronounced

giant cell aggregation and fibrosis around the necrosed worm tracks, extensive muscular oedema and sub-acute to chronic focal myositis on the 50th day; advanced fibrous tissue reactions on the 60th day; and chronic fibrous tissue reactions on the 70th day post infection.

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PATHOGENICITY OF ACUARIA HAMULOSA TO CHICKEN

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THESIS

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ABSTRACT

Controlled experiments were conducted with commonly occuring spirurid worm of chicken, Aquaria hamulosa, in two breeds of chicken, namely the White Leghorn (an egg strain) and the White Plymouth Rock (a broiler strain).

The blood changes conducted revealed an initial leucocytosis from 14th day to 36th day, followed by heterophilia from 43rd day to 70th day post infection, and a mild eosinophilia throughout the experimental period. The infected White Plymouth Rock birds lost considerable amount of their body weight. In White Leghorn layers, there was great delay in sexual maturity and poor feed efficiency as indicated by reduced egg production. The percentage of establishment in the experiment was found to be not correlated to the infective dose. The pathological changes in the affected gizzards indicated an acute inflammation during the invesive phase of juveniles, followed by a chronic reaction, which by 60th day post infection was well established indicating the development of a host-parasite balance, towards the later stages of infection.