PATHOGENICITY OF *ASCARIDIA GALLI* INFECTION IN BROILER CHICKEN WITH SPECIAL REFERENCE TO WEIGHT GAIN

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THESIS

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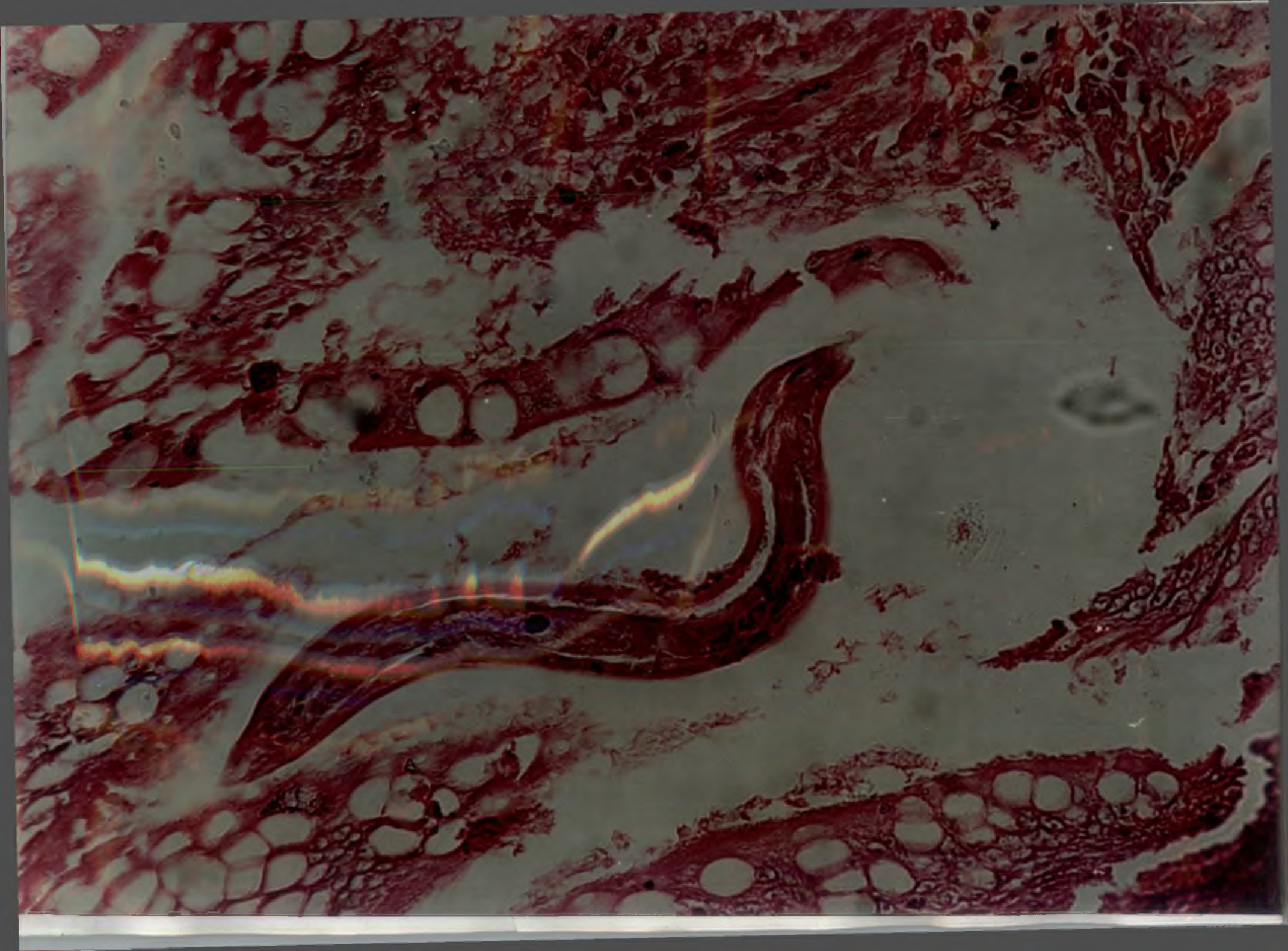

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Introduction

INTRODUCTION

In our country, of recent, broiler chicken farming is a flourishing and profit-making business in both rural and urban areas. The easy availability of good breeds of commercial broiler chicks and their feed and lesser capital investments are added advantages to those who step into this vocation. More over, members of a family themselves can manage a medium type farm without any external labour. Broiler chicken meat is comparatively cheaper and hence affordable to all income groups and there is no religious taboo against it.

The broiler industry was started as a novelty in early 'seventies' where the output was only 4 million in 1971 and 30 million in 1980. During 1990 the annual production of broiler chicken was 190 million and it is expected to be around 400 million during 1996 (Indian Poultry Industry Year Book, 1994). Although India stands as the 6th largest poultry producing country, its per capita consumption of meat is very low - about half a kilogram (566 gm/year). The consumption of meat could be increased only by making available broiler chicken meat at a lesser cost by giving much emphasis to the increased production of broiler birds.

Among others, parasitic diseases cause considerable loss in broiler chickens by affecting their general health and leading to mortality and morbidity. Ascaridiasis caused by the large round worm Ascaridia galli is the most common and pathogenic helminthic diseases of the birds (Schrank, 1788). This parasite mainly affecting the younger ones is prevalent world-wide and has been reported by various workers from India also (Dutta, 1950; Endrejat, 1964; Nadakal et al., 1972; Varghese and Peter, 1973; Gogoi, 1974; Shastri et al., 1974; Srinivasa et al., 1983; Ghosh, 1986; Raote et al., 1991 and Singh et al., 1993).

About 20 per cent of mortality in growing chicken (Gupta and Rao, 1959) and 50 per cent overall loss in poultry industry in India (Rai, 1972) is caused by A. galli. The widely practised deep litter system provides an ideal condition for the spread of the infection either alone or in conjunction with other pathogenic agencies. Even though the system of poultry rearing is gradually changing from deep litter system, A. galli infection could be invariably present in the birds which are reared in the cage system of management also. The infection may thus continue to be present in the farm for a prolonged period causing a reduction in meat production.

Present investigation

The published work on ascaridiasis in broiler chicken appears to be scanty even though the condition is found to be very common in broiler farms. Hence, a critical study on some important aspects of A. galli infection in broiler chicken such as pathogenicity, infectivity and effects on weight gain, haematological values and histopathology especially of small intestine, which would help to assess the severity of the infection and the extent of loss consequent to it was undertaken. This would, in turn, help to plan the control of infection in a broiler flock.

Review of Literature

REVIEW OF LITERATURE

Prevalence of Ascaridia galli infection

Among the nematodes it is found that Ascaridia galli is the commonest and one of the most pathogenic parasite infecting domestic fowls.

The prevalence of A. galli in broiler chicken has been reported by many from India and abroad. Reid (1958), after studying the incidence of A. galli in 1000 broilers in U.S.A., stated that 27.5 per cent of them were infected. In a large commercial broiler farm in Poland, Dzido (1973) found that, out of 1000 broilers (8-10 weeks old) 356 were positive for A. galli as evidenced by faecal examinations.

Long (1977) reported that the broilers maintained on earthern floor acquired the infection after 1st to 2nd week of transfering them into the sheds. A survey of 2 to 3 months and 4 to 5 months old broilers by Bilqees and Khan (1985) in Karachi revealed A. galli infection at the rate of 10.6 per cent and 20.4 per cent respectively.

Sharma and Kaushik (1986) after conducting autopsy of 14,564 birds in Gurgaon , Ambala and Hissar of Haryana state confirmed that 2.56 per cent of mortality was due to

A. galli infection. They also stated that 1.98 per cent of mortality took place during winter as against a mortality of 0.08 per cent in summer and the maximum mortality was in adult broilers.

Coprological study conducted by Oyeka (1989) in Nigeria found that the broilers harboured more A. galli worms (60 %) than layers (49 %). Wilson et al. (1994) stated that out of 3,452 broiler birds 41.2 per cent was positive for A. galli infection in two farms at North West Arkansas.

Pathogenicity, clinical signs and histopathology

Ackert and Tugwell (1948) observed that from 3rd to 24th day after infection of the chicks, the young worms were found within the mucous membrane of small intestine. Sadun (1950) reported that there was reduction in growth rate, atrophy of thymus, increased relative weight of the liver and spleen and high mortality rate on 14th day post infection (PI) in experimentally infected birds. He also observed anaemia, severe leucocytosis, increased total polymorphonuclear leucocytes with eosinophilic granules, marked increase in total and relative number of polymorphonuclear leucocytes with eosinophilic rods, minute haemorrhages in the duodenal region, necrosis and sloughing of mucosa. The worms were present in the lumen and crypts

and those in the crypts were sorrounded by flattened epithelium. He also found congestion during gross examination of lungs, heart and kidneys.

Tugwell and Ackert (1950) found that, between 8th to 17th day PI, majority of the young A. galli worms were burried in the mucous membrane of the small intestine with their anterior ends. Scott (1952) recorded intussusception of the bowel with prolapse and necrosis of the ends in a bird which was naturally infected with A. galli in Kenya.

Todd and Crowdus (1952) experimentally infected chicks with A. galli ova and reported that the migration of larvae into the intestinal mucosa was infrequent and most of them matured without leaving the lumen of the intestine.

Tugwell and Ackert (1952) investigated the tissue phase of A. galli and reported that it might have begun on the 1st day of parasitism, reached its intensity from 9th to 16th day and might be continued through out the 25th day. The lumen phase also began on the 1st day and reached its peak from 11th to 21st day. Tsvetaeva (1954) found that after half an hour of infection, the larvae were present in the mucosa of anterior part of duodenum, through out the small intestine after 1 to 2 hours of infection and about 19th day the young worms were seen in the intestinal lumen.

Deo and Srivastava (1955) reported that majority of third stage larvae were seen free in the lumen from 15 days PI and only a small number of them were embeded in the mucous membrane of intestine. They also corroborated that the larvae were in intimate contact with the duodenal mucosa in 10 to 12 days PI, developed rapidly in the lumen of intestine and matured in about 28 to 34 days.

Kadziolka (1956) observed vascular changes in the region of duodenum, jejunum and haemorrhagic appearance of mucous membrane after 10 to 13 days of infection.Moran and Mizelle (1956) reported that the infective eggs of A. galli hatched as early as half an hour after infection and the immature A. galli remained in the intestinal layer of mucosa in a static condition for a considerable period.The intestinal mucosal penetration by A. galli larvae was found to be rare.

Deo (1964) stated that young birds were highly susceptible to infection and marked lesions were produced when large numbers of young parasites penetrated into duodenal mucosa. The larval forms caused haemorrhage, enteritis, diarrhoea and anaemia followed by loss of appetite, abnormal thirst, unthriftiness, drowsy appearance,

ruffled feathers, drooping wings, emaciation and leg weakness. The mortality, morbidity and weight loss were highest during 3rd week PI.

Varghese (1966) stated that there was no penetration of larvae into the duodenal mucosa at any stages of their development but confirmed that the larvae at certain stages of development were seen closely adherent to the mucous membrane with their anterior extremities burried in between the crypts.

Bandyopadhyay and Jain (1967) observed haemorrhage in submucous layer extending to lamina propria, exudate in mucous coat consisting of fibrin, serum, erythrocytes and a few plasma cells, various stages of degeneration of glands of intestine and intussusception at the posterior part of ileum with adhesion of mesentric portion in a naturally infected Rhode Island Red fowl. Tongson and Mc Craw (1967) found that most of the worms were seen in the region of intestine between bile duct and meckel's diverticulum in experimentally infected chicken. Deorani (1968) noted the formation of diverticulated protuberances of 2.5 to 7 mm size on the intestinal serosa which were soft, hollow and pedunculated with an opening into the intestinal lumen. The tissue response was stated to be due to the irritation caused by A . galli and their metabolites.

Lee (1969) infected 3 and 18 month-old broiler chicken with infective eggs of A. galli and found nodule like masses on the inner wall of intestine,but could not detect larvae in the nodules. He also confirmed that the larvae stayed in the villi of small intestine for 1.5 to 2.5 months causing haemorrhage without any further development there.

Khouri and Pande (1970) confirmed that the 2nd stage juveniles were available in the lumen of small intestine, some having established contact with mucosa and the 3rd stage were recovered from the mucosa as well as the lumen. Majority of the 4th stage larvae were obtained from the lumen only. They also noted that the peak period of juvenile worms in the mucosal lining was between 8th to 15th day PI. Concurrent study by the same authors in experimentally infected chicks revealed clinical signs like droopiness, drowsiness, off feed and diarrhoea and also observed that six day old juveniles were present in the destroyed villi with their anterior ends directed towards the base of the mucosa.

Birova-Volosinovicova (1971) observed that the larvae were seen among the villi, in the lumen of Leiberkuhn glands or free in the lumen of intestine in experimentally infected chicken and concluded that histotrophic phase was absent in

the life cycle of A. galli. Dimitrov (1971) observed that the number of worms developed in the intestine decreased as the infective dose increased in experimentally infected broiler chicks.

Ikeme (1971) observed bloody diarrhoea, loss of appetite, decreased activity, ruffled plumage, drooping wings, progressive emaciation and conspicuous leg weakness in birds maintained under different levels of nutrition and infected with varying degrees of repeated doses of A. galli infective eggs. On histopathological section, extensive destruction and erosion of glandular epithelium, proliferation of the mucus secreting cells resulting in adhesion and vascular extravasation of the villi were observed. He also stated that no larvae could be seen in lungs, liver and kidneys.

Niculescu and Purcherea (1972) observed that larvae of A. galli penetrated the walls of jejunum and ileum of chicken on the 1st day of infection itself.

Herd and Naught (1975) found that histotrophic phase involved both 2nd and 3rd stage larvae rather than 3rd stage larvae alone in an experimental infection with A. galli. Pavlicek and Dykova (1975) observed a 46 to 100 per cent mortality rate, a worm burden up to 54 per cent of eggs given and a decrease in weight of 68 to 78 per cent in

cockerels of 5 to 48 days age when they received 1500 to 3000 infective A. galli eggs. They also observed marked changes in intestinal mucosa and infiltration with eosinophils in the parenchyma of liver and intestine. Sazikova (1975 a) reported partial atrophy of kidney,liver and spleen and marked reduction in weight of heart, liver, stomach and ovary and in the length of the intestine. The intestinal wall where the parasites were localized showed distension and thickening.

Vegad et al. (1979) confirmed intestinal perforation and pleuroperitonitis in A. galli infected chicken. Kaushik (1980) stated that on autopsy of a chick there was development of a diverticulum on the serosal surface of duodenum which was caused by a female adult A. galli.

Matta (1980) reported gross petechial lesions and generalized oedema in chicks 15 days after experimental infection, but could not identify any histotrophic phase. Mishra et al. (1980) on histological studies of small intestine of A. galli infected birds noted pronounced swelling of intestinal epithelia, infiltration of lamina propria by lymphocytes, macrophages and histocyte and proliferation of fibroblasts of sub mucosa. He also found the larvae of A. galli either between two villi or penetrating an individual villus.

Reddy et al. (1984) revealed that the percentage larvae associated with mucosa in the 1st two weeks of age was higher in group of chicks fed with normal feed than in those fed with deficient diets. They also noted the period of maturation of worms as 35 to 37 days after infection in birds fed with different levels of protein and could not identify the tissue phase of larvae.

Muraleedharan and Seshadri (1985) reported intussusception and volvulus associated with A. galli. infection in two white leghorn fowl of 49 weeks of age. Both birds showed ballooning and distension of the first half of intestine,one having twelve worms had intussusception and congestion of jejunum and the other with twenty worms had twisting of the bowel or volvulus.

Soulsby (1982) reported that the young birds were more susceptible and had diarrhoea, anaemia and severe enteritis. The birds were unthrifty, markedly emaciated and generally weak, perhaps with intestinal obstruction. He also stated that after natural infection, the infective eggs hatched in the intestine and the larvae were found in the intestinal mucosa from 8th to 17th day, re-entered the lumen and attained sexual maturity in 6 to 8 weeks time. Padhi et al. (1987) stated that they found adult A. galli.

worms in colon and caecum of desi fowls but did not cause any gross pathological lesions. Rai et al. (1989) diagnosed 4 cases of intussusception in white leghorn hens, where volvulus with invagination were seen mainly in distal part of duodenum, jejunum and ileum.

Mukit et al.(1991) reported two cases of intus-susception mainly of right caecum and on histopathology they observed haemorrhage, necrosis in mucosal to serosal layer and atrophy and degenerative changes of submucosal glands. Ramadan and Znada (1991) observed haemorrhage, congestion and intestinal obstruction in A. galli infected chicken. Fathithu et al. (1992) observed blood tinged diarrhoeic faeces, increased thirst, stunted growth and echymotic haemorrhages in intestine which could be visible from serosa and a generalized catarrhal enteritis. Histopathological section revealed necrosis and desquamation of intestinal villi.

Padmaja (1992) in her experimental study observed that the birds were susceptible to infection as early as 3rd day onwards and more pronounced during 10th to 20th day PI. The infected birds showed dullness, droopiness, whitish diarrhoea, weakness, emaciation, unthriftiness, pale comb and wattles and considerable reduction in body weight gain. On post mortem examination of birds up to 20days PI,

found that the intestinal contents were frothy and blood tinged and the intestinal mucosa in the region of jejunum and ileum haemorrhagic and congested. From 30 to 40 days PI, the immature or mature worms were present in the lumen of intestine. The other lesions noticed were the same as that was up to 20 days PI. However, she could not find any lesions in other organs. On histological section hyperplasia of goblet cells, infiltration of mononuclear inflammatory cells in lamina propria, submucosa, muscular layer and also in submucosal glands could be observed. The mucosa showed mild congestion and haemorrhage with mild desquamation of tip of the villi from 10th to 40th day after infection in almost all parts of the small intestine and observed migration of larvae into the duodenal and jejunal mucosa 20 days PI.

Delay in the development of feathers, dullness, weakness and blood tinged diarrhoea were noticed by Raote et al. (1992) in white leghorn male chicks infected with A. galli infective eggs followed by hydrocortisone at the rate of 10mg /kg intramuscularly. They also observed severe gastro enteritis and diarrhoea which might be due to large number of larval stages in mucosa.

Verma et al. (1993) observed diarrhoea, anorexia, dullness and intestinal obstruction in 7 weeks old

experimentally infected birds. On post-mortem they also noted thickening of the intestinal mucosa along with red bite marks. Histopathological examination revealed excessive vacuolation in epithelial cells, infiltration with mononuclear cells in the mucosal region and oedema of muscular layer. They also observed desquamation of epithelial cells, thickening of serosa and separation of serosa from muscular layer 15 days PI.

Maturation period of Ascaridia galli

Ackert (1931) reported that the young worms matured in about 50 days in one month old chicken. Feoktisov (1950) found that the worms attained maturity in 35 days in 16 to 18 days old chicken and 58 days in adult hens. Kerr (1955) recorded that prepatent period of A. galli was 30 to 35 days in chicken below 3 months age and about 50 days in older birds. Deo and Srivastava (1955) observed that the maturation period was 28 to 34 days in 4 to 8 week old birds.

Blood parameters

Sadun (1950), in heavily A. galli infected birds, ten 10 days PI observed a lower erythrocyte count (RBC) which was 2.08 and 1.55 mil/cu mm and noted a marked increase in leucocyte count (WBC). No significant difference in

leucocyte number could be found in chicken that received a mild egg dose. There was a significant increase in eosinophil count but the increase in basophils and lymphocytes was negligible.

A continuous increase in packed cell volume (PCV) value from 1st to 5th week PI and decreased mean value of haemoglobin during 4th week PI was observed by Ikeme (1971) in 1000 doses of A. galli infected chicken. Birova-Volosinovicova (1974) in A. galli infected birds observed that the RBC and WBC count, haemoglobin and leucogram were within the physiological limits.

An increase in total WBC count was observed by Kaushik and Sen (1978) during 1st three weeks after infection. Absolute and differential counts of heterophils, monocytes, eosinophils and basophils were found to be increased during 2nd to 8th weeks PI. They also reported a falling down of absolute and differential lymphocyte count during 4th to 8th week PI.

An absolute reduction in haemoglobin concentration and PCV were observed by Matta and Ahluwalia (1982) during first 3 to 4 weeks after experimental infection. They also observed an increase in erythrocyte sedimentation rate (ESR) during 1st three weeks PI.

Krishna Reddy and Venkataratnam (1985) studied the level of serum protein and albumin globulin ratio in A. galli infected group of birds maintained under different feed formulation and stated that the level of serum protein was always lower in infected group when compared with the uninfected control group.

Lowered thrombocyte count was observed by Sekhar and Simha (1985) in A. galli infected pullets, whereas no significant difference in thrombocyte count was observed in cockerels. In the same year they observed an increase in volume index of erythrocyte causing macrocytic anaemia in A. galli infected domestic chicken. But in pullets there was no significant change in volume index.

Sekhar and Simha (1986) observed either leucopenia due to fall in lymphocytes or leucocytosis due to rise in heterophils in both sexes of domestic fowl which had multiple helminthic infections. Sekhar et al. (1986) reported no significant reduction in mean corpuscular haemoglobin concentration (MCHC) in A. galli infected pullets, contrary to a reduction in MCHC in cockerels. Sekhar et al. (1988) noticed in infected birds a marked reduction in total erythrocyte count and blood haemoglobin level and an increase in ESR in infected birds.

An increase in lymphocytes, heterophils and eosinophils and decrease in PCV, Hb per cent, RBC, WBC, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCHb), MCHC and monocyte count were observed by Rao and Lal (1991) in A. galli infected white leghorn chicks.

Fathithu et al. (1992) reported that there was no significant reduction in PCV in A. galli infected broilers. Padmaja (1992) reported a significant decrease in RBC count after 30 days of infection and leucopenia during 10th day PI and thereafter leucocytosis. She also observed a reduction in Hb level and PCV and an increased ESR, increase in heterophils and esoinophils but without any significant increase in lymphocytes, basophils and monocytes during developmental stages of worm. No marked changes in estimation of Hb, PCV, RBC and WBC was attributed by Verma et al. (1993).

Effect of feed consumption and weight gain

Todd et al. (1949) reported that there was a progressive retardation of growth in A. galli infected New Hampshire broilers and that the weight loss was during the period of migration of immature stages.

Experimental study conducted by Todd and Hansen (1951) revealed retardation in weight gain as the number of worms

increased. Tsvetaeva (1954) in an experimental study found that the loss of weight occured during 2nd week of infection. Kadziolka (1956) in a comparative experimental study observed that the increase in weight of the infected chicks was less than control chicks.

Reid (1958) stated that combined infection of A. galli with infectious bronchitis in White Rock broilers resulted in an over all average mortality of 3.1 per cent. He also confirmed that average weight gains were less with combined infection, when compared with single infection either with worms or bronchitis. The feed consumption was lesser in combined infection than in single infection.

Reid and Carmon (1958) confirmed a significant decrease in weight gain when the dose of the infective ova was increased. Reid et al. (1958) recorded the depression in feed consumption and weight gain in combined infection of A. galli and infectious bronchitis in 3 weeks old chicks.

In a feeding trial conducted by Deo and Srivastava (1962), found a lesser weight gain in A. galli infected birds fed with diet deficient in calcium and Vitamin A. They also recorded an increased number of worm count and significant increase in length of the worms in birds fed with deficient diets. Kassai (1962) observed a loss of

weight, hypovitaminosis A and diminished resistance to other diseases in naturally A. galli infected birds.

Considerable decrease in the body weight gain and feed efficiency were recorded in an experimental study conducted by Costa (1970). An increased feed intake (4-6 %) and decreased body weight gain (6-7%) were observed in broiler chicks by Dimitrov (1971) who infected chicks with different doses of A. galli infective eggs. A significant depression in growth rate was observed by Ikeme (1971) when he repeatedly infected the birds with different doses of A. galli infective eggs. Patil et al. (1972) reported a lesser weight gain in A. galli infected broiler birds even at a lower dose rate of 150 A. galli ova.

Sazikova (1975 b) corroborated the lesser weight gain in white leghorn layers. He also reported delay in egg laying process, smaller size of eggs, reduction in fertility rate and lower vitamin A content of yolk.

Katara and Rai (1980) estimated the gain in body weight of infected birds and found to be 53, 47, 43 and 47 per cent of that by healthy controls after 10th, 20th, 30th and 40th days of infection respectively. Matta and Ahluwalia (1980) confirmed significant difference in average weight between infected and control groups of growing

chicks. Matta (1981) reported that the average production of eggs per infected hen for the period of 12 weeks was 36.5 as against 50.2 eggs in the uninfected hen. He also confirmed the loss of egg production was one egg/week/bird. Toledo and Castell (1981) in their experimental study found a highly significant difference in mean body weight after 8 weeks of infection which was 1085 g in A. galli infected birds and 1177 g in healthy controls.

Ramadan and Znada (1991) confirmed variable decrease in body weight and increase in the ratio of liver weight relative to body weight, both being proportionately related to the number of infective doses given to broiler chicken.

Padmaja (1992) reported that only 64 per cent of mean weight was gained in A. galli infected chicks when compared to the control group. Similarly subsequent reduction in weight gain was reported in A. galli infected chicken by Raote et al. (1994).

Materials and Methods

MATERIALS AND METHODS

Collection of worms

Sexually mature Ascaridia galli female worms were collected from the small intestine of fowls which were brought for post-mortem examination from the local poultry farms to the Pioneer hatcheries, Namakkal and also those slaughtered at Sakthan Thamburan market, Trichur, Kerala. Intestines were opened and the worms collected were thoroughly washed in normal saline solution to remove the debris, and brought to the laboratory in normal saline for the preparation of egg cultures.

Harvesting of eggs for setting up of cultures

The egg cultures were prepared adopting the method suggested by Riedel (1947 and 1951). The culture medium used was filtered, well aerated aquarium or well water to which one per cent commercial formalin solution (40 per cent formaldehyde) was added to prevent any fungal growth and contamination with other micro organisms. Each of the fully mature female A. galli worm was transferred on to a clean, grease free glass slide which contained a little quantity of water. The posterior end of the worm was snipped off and with the help of a fine dissecting needle, the uterus

containing the eggs were squeezed out and it was transferred with the help of a fine camel hair brush to a small sterile petri - dish which contained the culture medium. The petri dishes were covered with suitable cover dishes and kept under the room temperature for further development of eggs.

Maintainance of egg cultures

Every day morning the culture medium was changed by carefully removing the top most layer of the medium with the help of a long pointed pasteur pipette and then replaced with fresh medium.Simultaneously, the eggs were examined for the development of embryo under a binocular dissection microscope up to 8th day after setting up the culture. From the 8th day onwards the samples of eggs from the cultures were examined twice daily both in morning and evening.

In order to provide oxygen for the culture, it was aerated with the help of a pipette by slightly blowing air over the surface. Adequate care was taken to see that the depth of the medium was 1 to 2 mm as otherwise, the development of the eggs would not take place at the desired level. The above procedure was repeated up to 15 to 20 days after setting up of cultures. This method saved a large number of infective eggs from destruction due to fungus and bacterial growth. The eggs in the cultures were fully developed and became infective from 9th day onwards.

Standardization of dose for experimental infection

Cultures of 10 to 45 days old were pooled together. To obtain an uniform suspension of the eggs in cultures, the samples were homogenized for 10 minutes by using a magnetic stirrer. Afterwards a drop of the sample was taken on a slide and examined to make certain that the infective eggs were intact. 0.01 ml of pooled culture sample was transferred on to a glass slide, covered with a cover slip and all the infective eggs therein were counted under the low power of a microscope. Three consecutive counts were carried out and the average was taken.

Experimental design

Two hundred commercial day - old Vencobb broiler chicks were procured and used for the experimental trial. All the chicks were housed under identical conditions in deep litter system, providing an area of one sq. foot per bird. Wood shavings were used as the litter material. For the first three weeks of age, chicks were brooded using electric bulbs and after three weeks only night lights were provided. Upto four weeks of age chicks were fed with good quality broiler starter mash and there after with broiler finisher mash till the end of the trial. Feed and water were provided ad libitum. Standard managemental practices were followed through out the experimental period.

The chicks were wing banded, weighed and allotted randomly to different groups in such a way that weight of the chiks within a group as well as between the groups were reasonably similar.

The chicks were randomly divided into 5 groups of 40 each and housed separately. The groups were designated as A, B, C, D and E. Among the 5 groups the first 4 served as infected groups and E as the non infected control.

Chicks of A, B, C and D groups were infected orally with 500, 1000, 1500 and 2000 A. galli infective eggs respectively, when they were one day old. For the experimental infection of the chicks, the infective eggs dispersed in culture medium, were administered per os into the crop by means of a long pointed 1 ml pasteur pipette . The pipette was calibrated as 0.25, 0.5, 0.75 and 1 ml in such a way that it could hold 500, 1000, 1500 and 2000 infective eggs respectively. When the definite quantum of culture was administered to the chicks, the pipette was rinsed with tap water and administered to the same chick to ensure that all the infective eggs were received by it.

After experimental infection, two chicks from each infected group and uninfected control group were sacrificed on the 2nd, 4th, 6th, 8th, 10th, 12th and 14th day. There

after, two chicks from each group including control were sacrificed from 17th day onwards up to 56th day at an interval of four days. After sacrificing each one, the entire small intestine was cut into 4 pieces and parts of duodenum, jejunum and ileum were preserved in 10 per cent formalin for histopathological study. The other pieces were cut opened, and the contents were collected in large petri dishes in normal saline solution and left for 10 to 12 hours for salvaging the larval forms. The total number of worms collected from each individual birds were counted to assess the percentage of infection in each individual group. The external appearance and clinical signs and the gross lesions of each bird were observed prior to slaughter and during post mortem examination. The weekly body weight, weekly body weight gain, weekly feed intake and weekly feed conversion ratio was also calculated from the data collected.

Blood parameters

To study the effect of ascaridiasis on the haematalogical values of broiler birds, the blood was collected from the 9th day of experimental infection at weekly intervals up to 51st day PI i.e., at the end of the 7th week. About six samples of 2 ml blood was drawn at random from each group of birds in sterilized test tubes

containing one drop of 10 per cent solution of EDTA as an anticoagulant. Simultaneously, blood smears of each bird were also prepared for studying differential count.

Erythrocyte sedimentation rate (Wintrobe method)

ESR was determined by filling the Wintrobe tube up to zero mark with blood. Then all the tubes were kept in a vertical position for one hour and the reading taken.

Packed cell volume

PCV was determined by centrifuging the Wintrobe haematocrit tube containing the blood samples for 30 minutes at the rate of 3000 rpm and the reading taken.

Haemoglobin (Acid haematin method)

The haemoglobin percentage in the present study was determined by Acid - haematin method using 0.1 normal hydrochloric acid as the reagent.

Red blood cell and White blood cell count

Total RBC and WBC counts were recorded using a haemocytometer and Nambiyar's fluid (Nambiyar, 1961) as the diluting reagent.

Differential count (Modified copper peroxidase method)

Blood smears were stained by Modified copper peroxidase method of Sato and Sakiya (Nambiyar, 1961). Around 125 cells were counted from each blood smear and the percentage of different WBCs was calculated and the average of two counts was taken.

Gross lesion and Histopathology

Gross lesions in duodenum, jejunum and ileum were noted while sacrificing each bird during the entire period of experiment. The lesions in liver, spleen, lung and kidney were also observed if there were any. Ten per cent formalin preserved pieces of duodenum, jejunum and ileum of both infected and control group were cut and washed in running tap water over night and these were processed and sections were cut at 4 to 6 microns level, both across and longitudinal as suggested by Sheeham and Hrapchak (1980). These sections were stained with Haemotoxylin and eosin and studied for various histopathological changes.

Weekly body weight

At the end of every week, chicks were weighed individually and mean weekly body weight was calculated by

dividing total cumulative weight of the birds at the end of each week by total cumulative number of the birds at the end of each week

Weekly body weight gain

The individual body weight at day old and at the end of each week was recorded to find out the body weight gain.

Mean body weight gain = Mean body weight of bird at the end of the each week - Mean body weight of bird at the end of pervious week.

Weekly feed intake

The weekly mean feed consumption was worked out by adopting the following method. An approximate quantity of feed required in a week for each group was weighed out at the begining of every week and this feed was provided ad libitum for each group. The balance at the end of every week was weighed and the actual weekly feed intake for the particular week was worked out. This gave the weekly feed intake for each group.

The mean weekly feed intake for each chick in each group was worked out by using the following formula.

$$\text{Feed consumption/ chick/day (gm)} = \frac{\text{Total feed intake for a particular week}}{\text{Total number of birds fed in each day in a particular week}}$$

This multiplied by 7 gave the mean weekly feed intake per chick for a week.

Feed conversion ratio

The weekly and total mean feed conversion ratio was calculated based on the ratio of total feed consumption per week to weekly body weight gain and total feed consumption to total mean body weight gain respectively.

All the data collected were statistically analysed by the method described by Snedecor and Cochran (1867).

Results

RESULTS

General condition of the experimental birds

The chicks which received 500 infective eggs of Ascaridia galli (group A) appeared to be as normal as the chicks in the control group during the 1st week PI. Afterwards, all the chicks in the infected groups were dull, drooping, passing loose motion with loss of appetite and increased thirst. There was no sign of emaciation, unthriftiness and leg weakness. The comb and wattles were pale and feathers ruffled. The development of comb, wattles and feathers was delayed.

The chicks which were infected with 1000 infective eggs (group B) showed all the clinical signs as in chicks of group A, but with more severity. The chicks passed watery diarrhoeic faeces which was whitish in colour as early as 11th day PI.

In the chicks which were infected with 1500 infective eggs (group C), the clinical signs exhibited were not different from that of group A and B but were more intense. In addition, moderate leg weakness was also noticed.

The clinical signs were still more severe in the chicks which received 2000 infective eggs (group D) compared to the infected chicks in other groups. Besides, there was emaciation, unthriftiness and weakness of legs. In all the infected groups of chicks there was proportionate reduction in the growth rate and body weight gain noticed.

Gross lesions

The post-mortem examination of the infected chicks revealed varying degrees of congestion and petechial haemorrhage in mucosa of duodenum and jejunum from 4th to 14th day PI. The intestinal contents were frothy and mucus mixed.

In some segments of intestine, the contents were not only frothy but also coated with blood and mucus. From 2nd day of infection onwards,large numbers of 2nd stage larvae could be recovered from the mucosal region of duodenum and jejunum by flushing with water.

The juvenile and adult worms were found free in the lumen of duodenum and jejunum from 14th day and from 33rd day PI, respectively. Some parts of lumen of the small intestine were narrowed and the wall was thickened (group D). Majority of immature, juvenile and adult worms were

present in the duodeuum and jejunum. No gross lesions could be seen in other visceral organs viz. liver, spleen, lungs and kidneys.

Maximum number of the larvae were collected on post mortem examination on 2nd and 4th day PI by keeping the portions of small intestine in the saline. From 6th day onwards, throughout the period of experimental trial the consistency of intestinal contents were frothy and ranged from semisolid to watery in all the infected groups of chicks depending upon the dose of infection. Over and above this, pin point haemorrhages, congestion of small intestinal mucosa and more number of larvae attached to the mucosa of anterior portion of the small intestine were found upto 14th day PI. Afterwards, the juvenile worms were found free in the lumen of the intestine and some were attached to the thickened mucosa of posterior part of duodenum and the entire length of jejunum. The above observations were applicable to the chicks of all the infected groups throughout the trial.

The narrowing of intestinal lumen due to thickening of the intestinal wall and severe enteritis were noticed in chicks belonging to 'D' group sacrificed on 17th, 21st, 25th, 29th and 33rd day PI. From 41st day PI onwards, majority of the worms were detected to be seen free in the

lumen of the intestine, especially in duodenum and jejunum and they were adult ones. The other organs did not show any other lesions in any of the infected groups of birds. Before the termination of the experiment, coprological study revealed that the average prepatent period was 44 and 50 days in birds of D and C groups respectively and 59 days for A and B groups.

The larvae collected during the salvage of intestine were designated as mucosal larvae and lumen larvae. Those larvae recovered after keeping the opened small intestine in the normal saline solution for 10 hours were mucosal larvae and those found free in the lumen are lumen larvae and others, the adult worms (Table 1).

There was no mortality in chicks of both infected as well as control groups during the period of experimental trial.

Histopathology

Group A

Mild to moderate hyperplasia of goblet cells uniformly distributed in all the segments of small intestine was observed on 2nd day PI (Fig. 1). On 4th and 6th day PI, the hyperplasia of goblet cells turned extensive. Moderate to

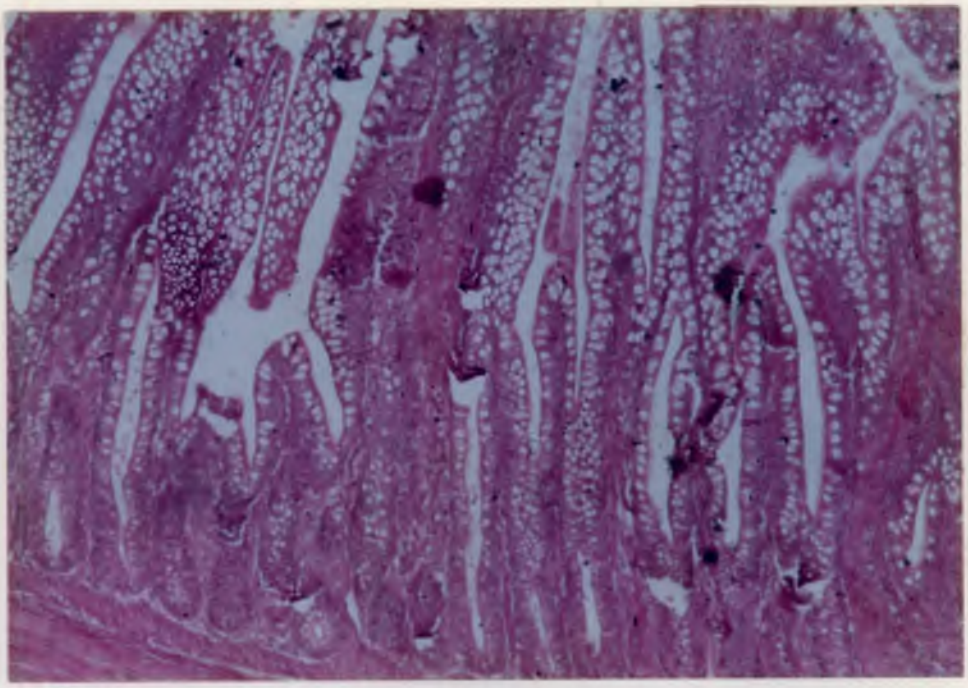


Fig. 1. Moderate goblet cell hyperplasia - duodenum (H & E) (x 160)

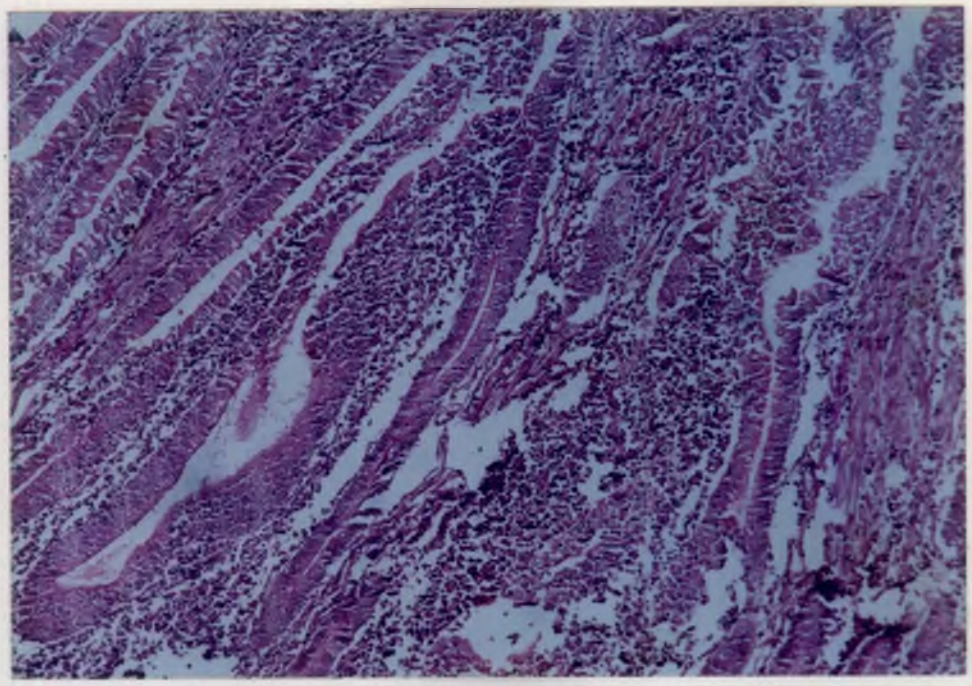


Fig. 3. Infiltration of inflammatory cells predominantly mononuclears in the lamina propria. (H & E) (x 160)

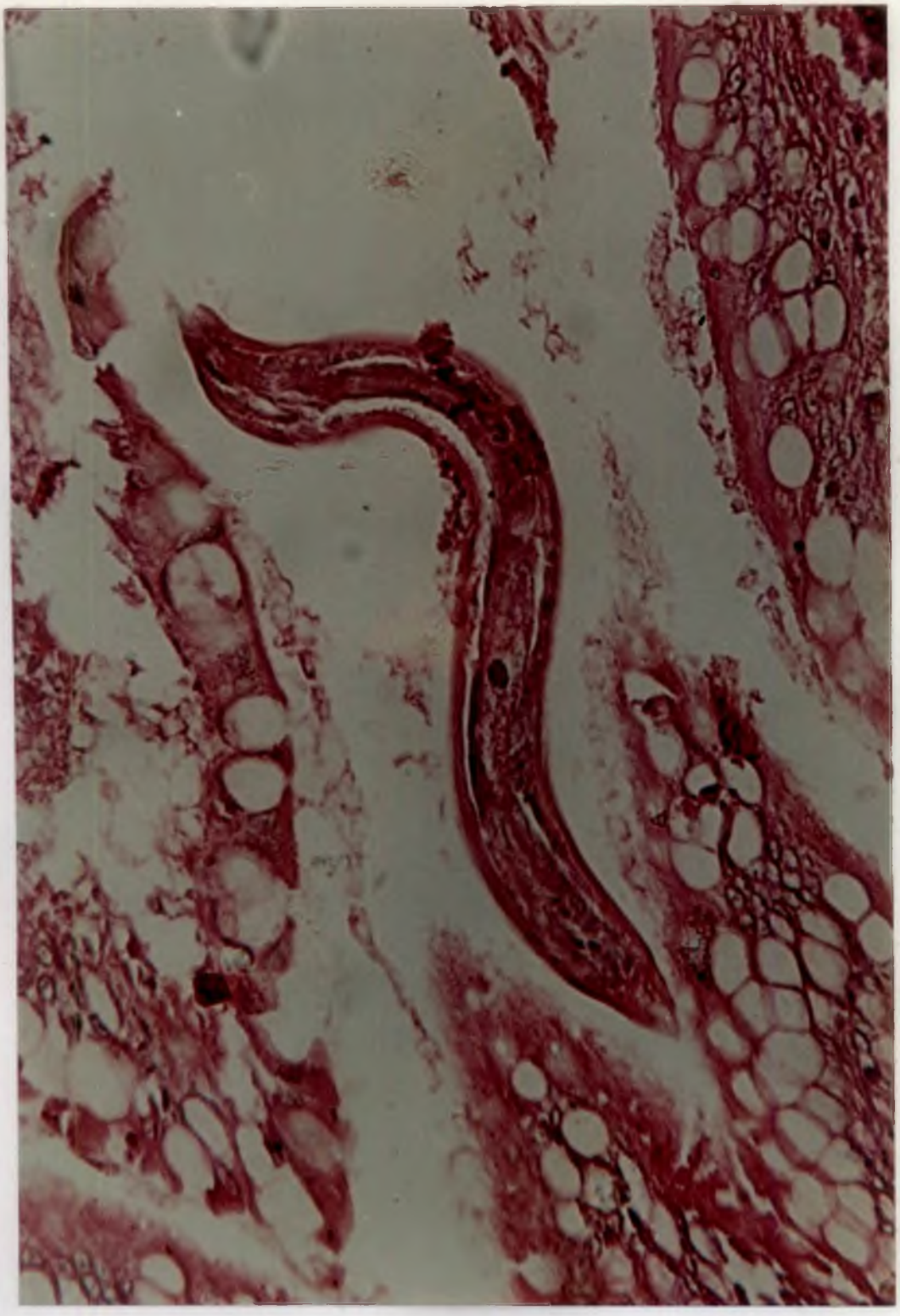


Fig. 2. Longitudinal section of A. galli in between the glands in the mucosa - No perilarval inflammatory reaction. (H & E) (x 400)

severe disruption of villi, desquamation of lining epithelium and focal necrosis were also evident. Lamina propria of a few villi and crypts revealed infiltration of inflammatory cells comprising mostly of mononuclears. Moderate desquamation of lining epithelium, focal areas of necrosis of mucosa and focal areas of inflammatory changes were observed on 8th day PI. Isolated areas of squamous metaplasia of the lining epithelium of mucosa were evident during this period.

Extensive hyperplasia of goblet cells, desquamation and focal necrosis of epithelium and focal areas of inflammation of the serosa were observed on 14th day PI. Longitudinal sections of larvae of A. galli in the glandular region of the mucosa were observed. Most of these larvae were seen between the glands without eliciting any inflammatory reaction (Fig. 2). On 17th, 21st, 25th and 29th day PI, infiltration of inflammatory cells predominantly mononuclears was observed in the lamina propria of the mucosa (Fig. 3), along with goblet cell hyperplasia and desquamation of lining epithelial cells. On 33rd and 37th day PI, desquamation of epithelium and disruption of villi were seen in association with perivascular infiltration of inflammatory cells in the serosa and muscular layers of intestine.

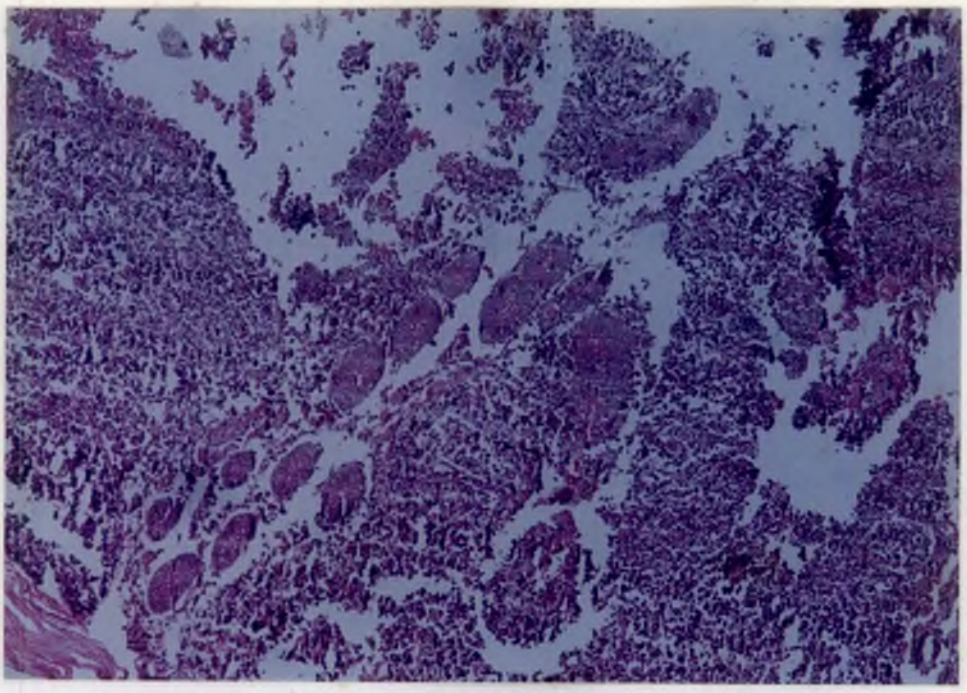


Fig. 4. Severe enteritis with disruption of villi. (H & E) (x 160)

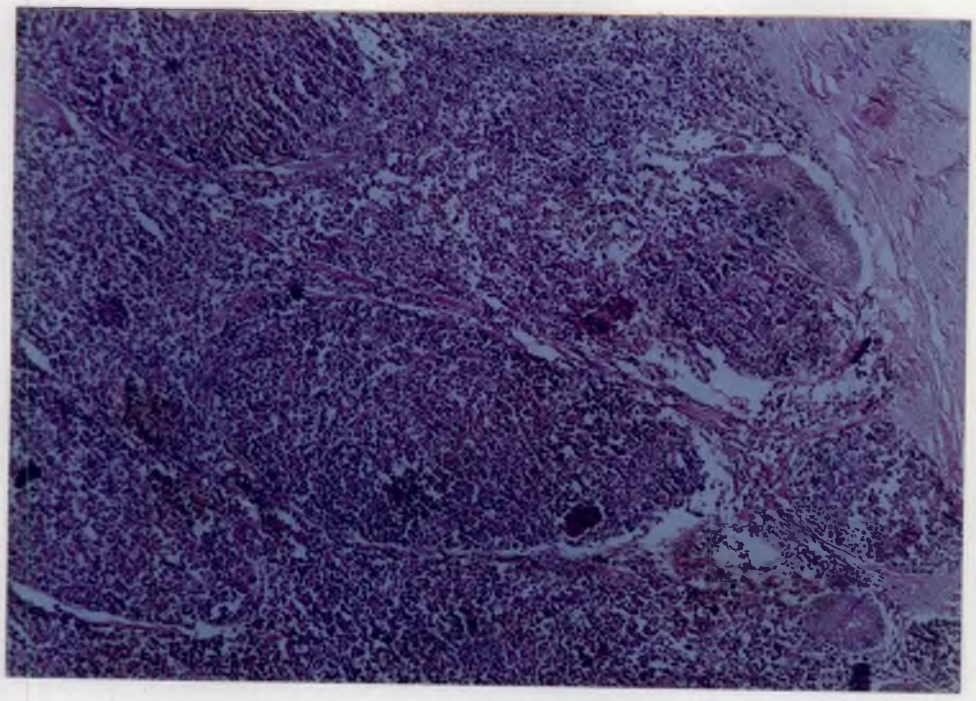


Fig. 5. Mononuclear cell aggregation in the mucosa. (H & E) (x 160)

Along with similar changes, severe enteritis was noticed on 41st day PI (Fig. 4). On 45th day PI, there was discretely distributed mononuclear cell (lymphocytic) aggregations in the mucosa noticed (Fig. 5). On 49th day PI, disruption and desquamation of epithelium of villi were observed. On 52nd and 60th day PI, mild goblet cell hyperplasia and scanty mononuclear infiltration were recorded.

Group B

On 2nd day PI, mild goblet cell hyperplasia, a few inflammatory foci and mild desquamation of epithelial cells were observed in all the segments of small intestine. Moderate desquamation of epithelial cells with extensive goblet cell hyperplasia and focal inflammatory changes were noticed on 4th day PI. Moreover, longitudinal sections of larvae were seen in between the villi causing moderate variation in the shape of the villi, but without any local inflammatory changes. Certain sections of larvae were tangential and a few were located in the crypts of Leiberkuhn (Fig. 6).

In addition to goblet cell hyperplasia, inflammatory changes and focal areas of submucosal oedema were noticed on 6th day PI. On 8th day PI, focal necrotic areas in the

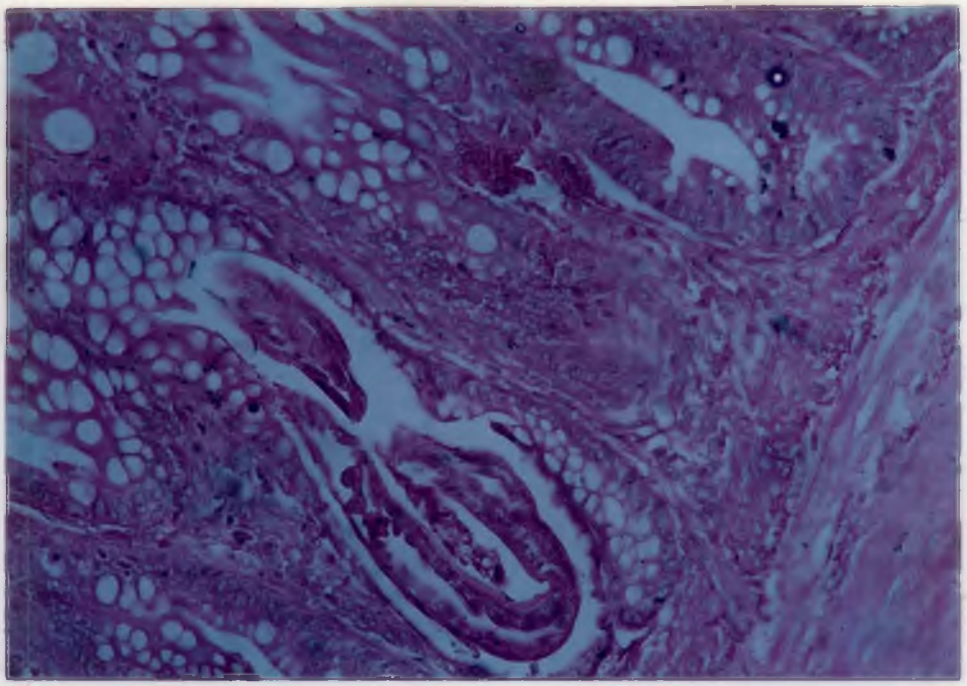


Fig. 6. Tangential section of larvae in the crypts of Leiberkuhn. (H & E) (x 400)

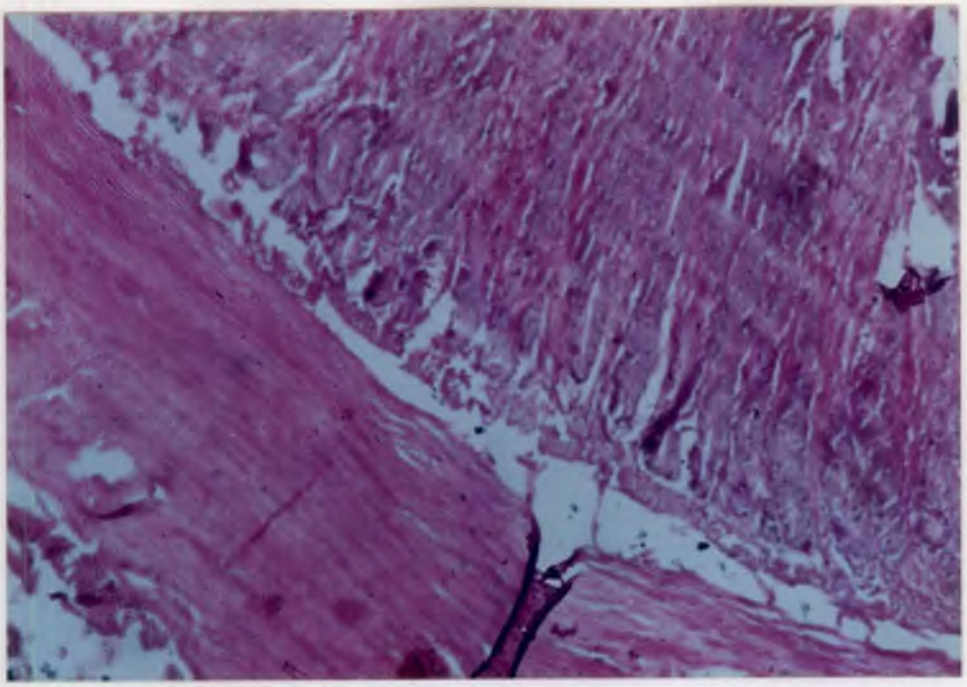


Fig. 7. Submucosal oedema with separation of submucosa from muscular layer. (H & E) (x 160)

mucosa and separation of submucosa from muscular layer were noticed (Fig. 7). From 10th to 17th day PI, mild to moderate inflammatory changes were noticed. On 21st day PI, moderate goblet cell hyperplasia, desquamation of epithelium and mononuclear cell infiltration were seen in the lamina propria of the mucosa. Evidence of mild inflammatory reaction was observed on 25th, 27th and 31st day PI.

On 33rd day PI, sections revealed presence of degenerated larvae in mucosal glands (Fig. 8) and a few glands showed mild cystic changes along with necrosis and desquamation of glandular epithelium. Development of a few lymphoid follicles in the mucosa, necrosis and desquamation of epithelial cells were observed on 41st and 45th day PI. Aggregations of lymphocytes and infiltration of inflammatory cells in the mucosa were observed on 49th day PI.

During 52nd and 60th day PI, mild inflammatory changes and goblet cell hyperplasia were recorded.

Group C

On 2nd day PI, mild to moderate goblet cell hyperplasia, desquamation of superficial epithelial cells and swelling of endothelial cells of blood vessels of serosa were noticed (Fig. 9). A few eosinophils and neutrophils

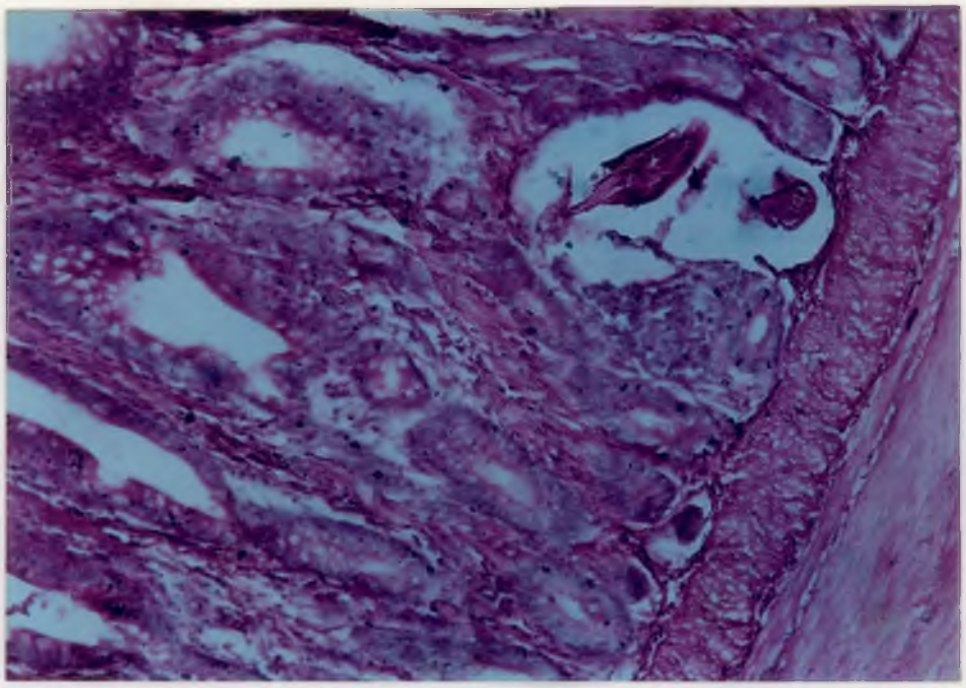


Fig. 8. Degenerated larvae in the mucosal glands. (H & E) (x 250)

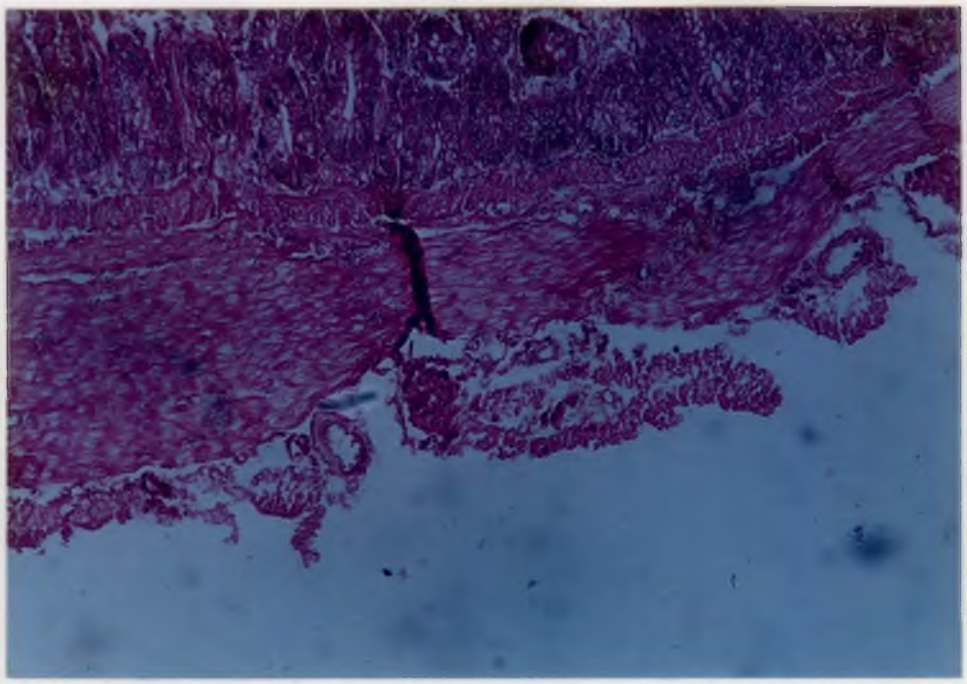


Fig. 9. Swelling of endothelial cells of serosal blood vessel. (H & E) (x 250)

were also observed in the lamina propria. Squamous metaplasia of the lininig epithelial cells were evident in a few villi of small intestine. On 4th and 6th day PI also similar changes were observed.

In addition to the changes observed on 2nd, 4th and 6th day PI, moderate congestion,endothelial swelling, cystic dialatation of a few mucosal glands and mild fibrosis of mucosa were observed on 8th day PI. On 10th day PI, goblet cell hyperplasia was conspicuous. Sections of larvae at the tip of the villi (Fig. 10) and embeded in the glandular region of mucosa were noticed (Fig. 11).

Severe goblet cell hyperplasia, appearance of cystic gland and severe desquamation of epithelium were evident on 12th day PI. On 14th day PI in addition to these changes larval sections were demonstrated at the tip of the villi. Severe desquamation of epithelium, goblet cell hyperplasia and haemorrhages were evident on 17th day PI.

On 21st,25th and 29th day PI, moderate goblet cell hyperplasia, desquamation of epithelium, focal inflammatory changes, intestinal congestion and necrosis of glandular epithelium were observed. The cystic changes and desquamation of epithelium of mucosal glands were evident

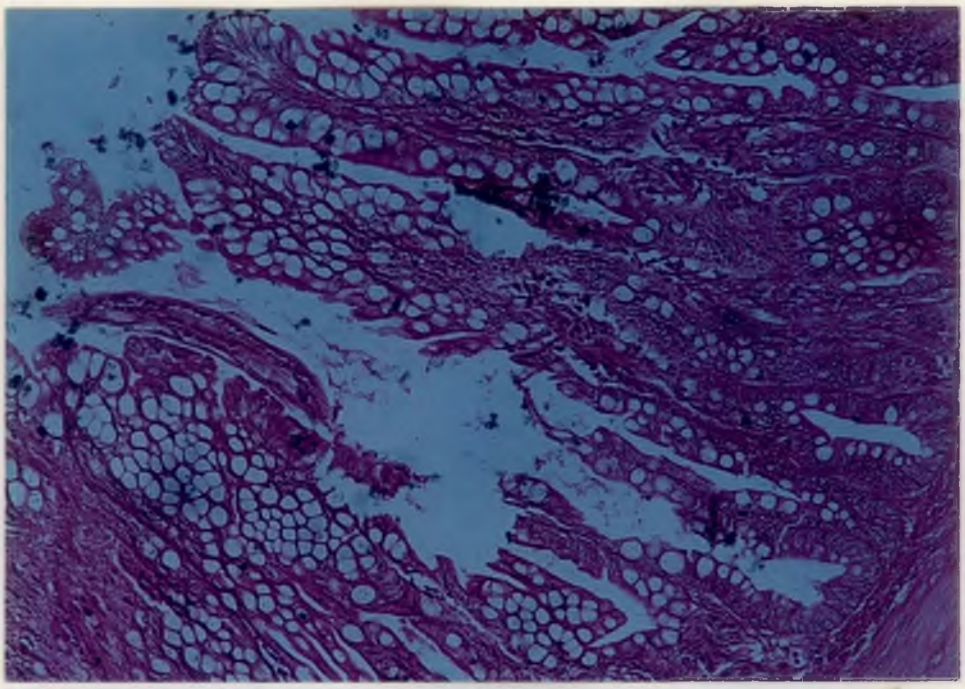


Fig. 10. Larval section at the tip of the villi. (H & E) (x 250)

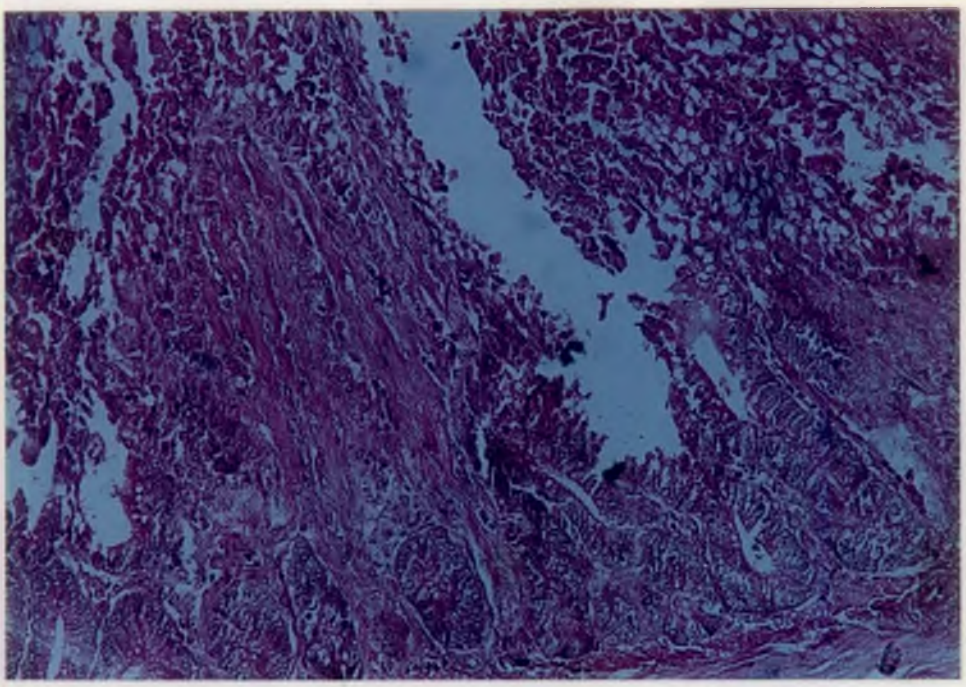


Fig. 12.Focal interglandular fibrous tissue proliferation. (H & E) (x 250)

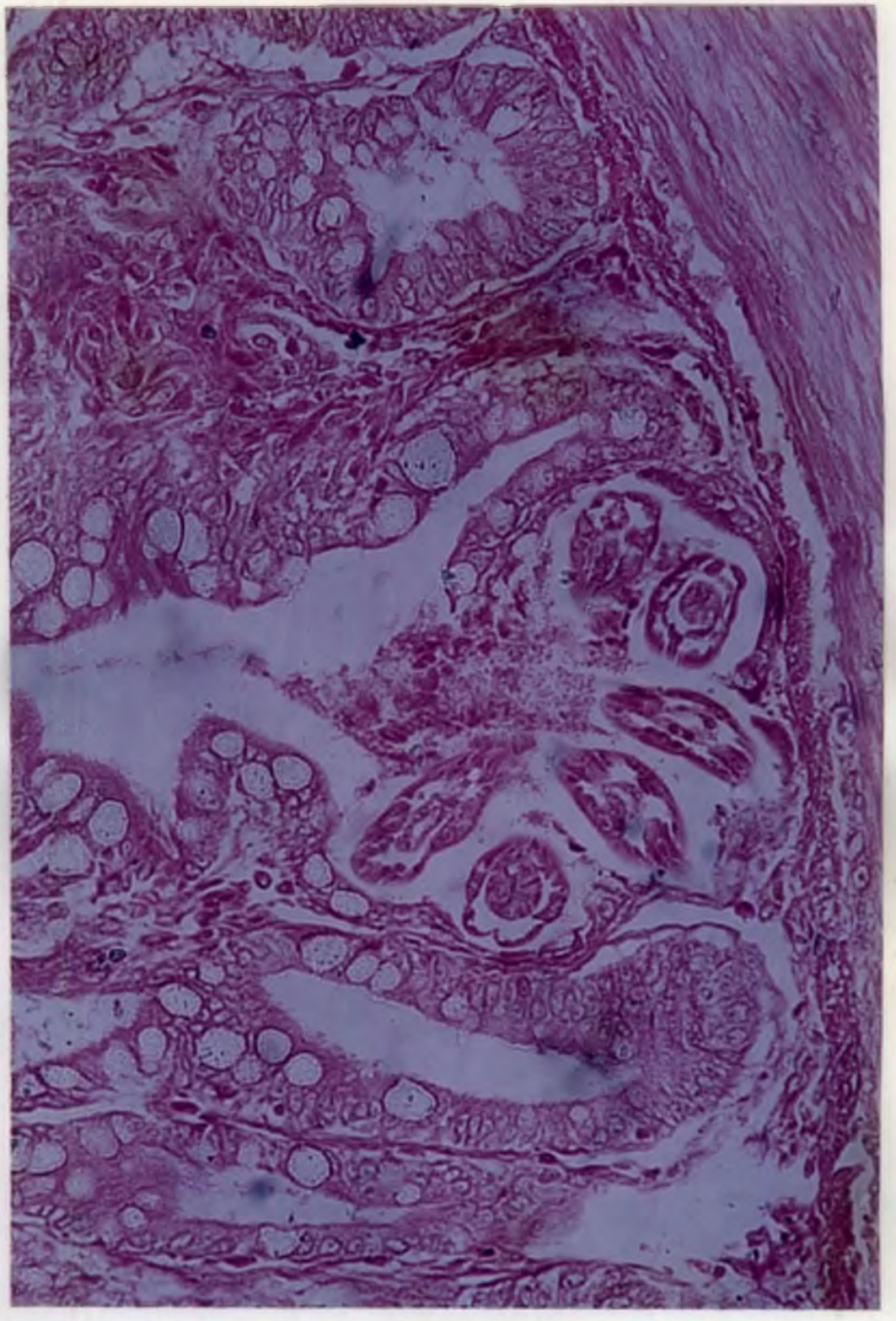


Fig. 11.Larval sections embeded in the glandular region of mucosa (H & E) (x 250)

on 33rd day PI. On 37th and 41st day PI, mild inflammatory changes, goblet cell hyperplasia and desquamation of epithelium were observed.

Severe goblet cell hyperplasia with severe focal infiltration of inflammatory cells was noticed on 45th day PI. On 49th day PI, isolated areas of aggregation of lymphoid cells were noticed in the mucosa. On 53rd and 56th day PI, mild inflammatory reaction and severe enteritis were noticed respectively.

Group D

On 2nd day PI, extensive desquamation of epithelial cells, disruption of villi, severe goblet cell hyperplasia, focal infiltration of inflammatory cells and moderate swelling of endothelial cells of the serosal blood vessels were noticed. Further, isolated areas of focal necrosis of the mucosal glands and focal areas of interglandular fibroblastic proliferation were also observed (Fig. 12).

Extensive desquamation of epithelial cells, swelling of endothelium of serosal blood vessels and moderate haemorrhages into the glandular region were evident on 4th day PI. In the duodenum, focal inflammatory changes in the

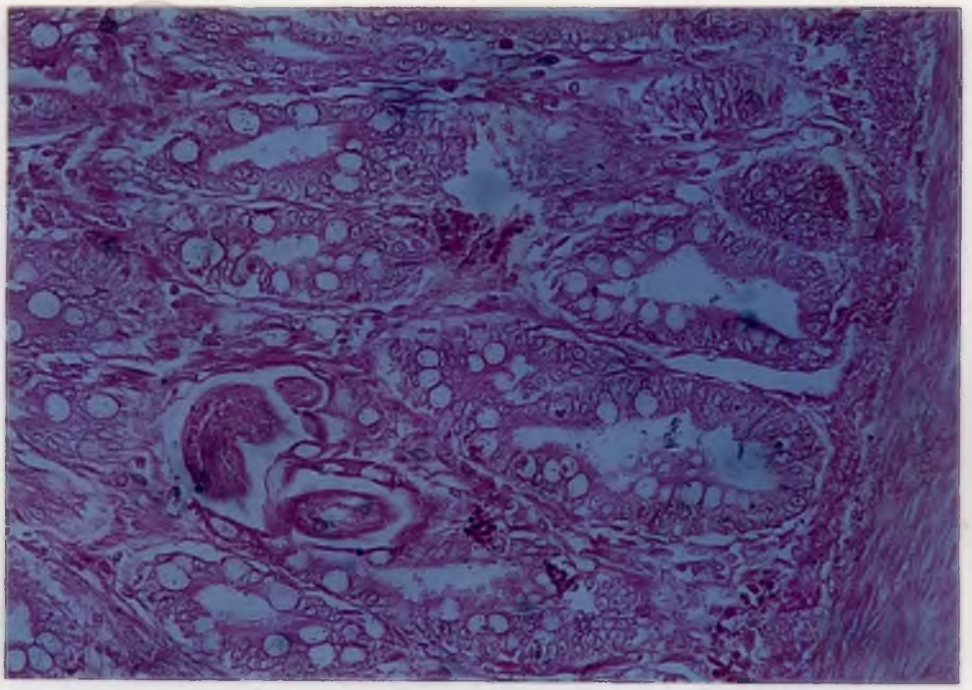


Fig. 13.Section of larvae at the tip of the villi. (H & E) (x 400)

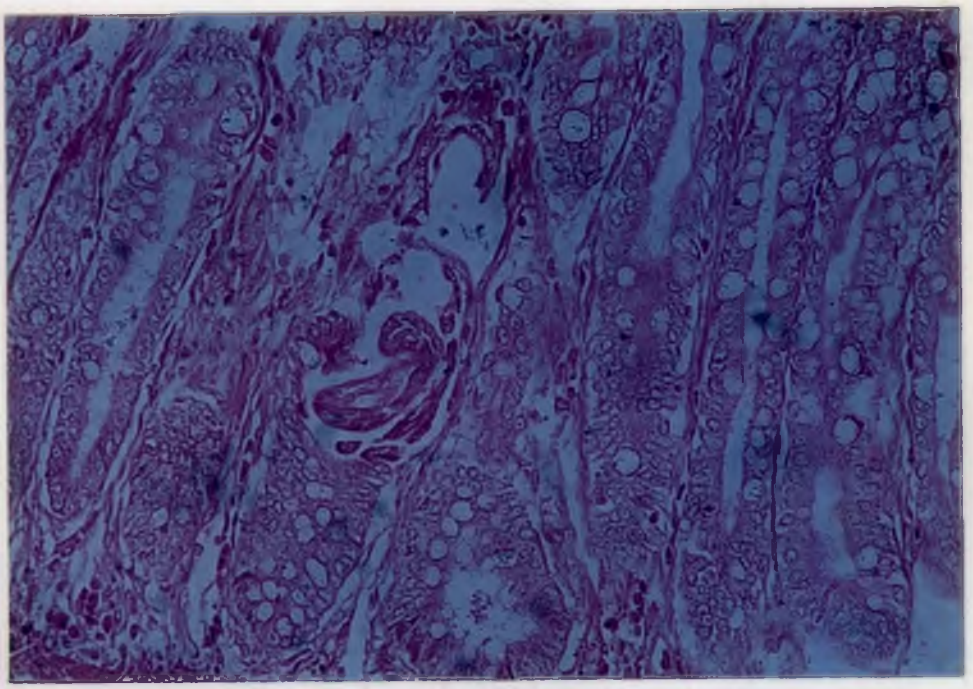


Fig. 14.Section of larvae in the deep mucosa inside the lumen of the gland (H & E) (x 400)

villi along with isolated cystic dilatation of glands were seen. A few cystic glands were conspicuous on 8th day PI.

Moderate goblet cell hyperplasia, cystic dilatation of mucosal glands and focal inflammatory reaction were observed on 10th, 12th and 14th day PI. Severe desquamation of epithelium, goblet cell hyperplasia and haemorrhages were evident on 17th day PI. In addition, sections of larvae were observed at the tip of the villi and in the deep mucosa and inside the lumen of mucosal glands (Fig. 13 and 14). On 17th day PI, some sections of larvae were seen at the tip of villi and also embeded in the glandular region of mucosa. Inflammatory reaction consisting of mononuclear cell infiltration was noticed in the lamina propria along with moderate goblet cell hyperplasia and desquamation of lining epithelium on 21st day PI.

On 37th day PI, desquamation of lining epithelium, disruption of villi and severe inflammatory reactions were noticed. Severe enteritis with disruption of mucosa were observed on 41st day PI. Lymphoid aggregations of varying sizes were observed on 49th and 52nd day PI. On 56th day PI, severe enteritis was noticed along with disruption of mucosal epithelium.

Blood parameters

The haematological values of infected as well as uninfected control chicks were observed at weekly intervals from 9th to 51st day PI. The results were statistically analysed and presented in Tables 2 to 11.

Weekly body weight

The body weight of each bird in all the infected groups as well as control group was taken at weekly interval from the end of the 1st week PI upto end of last week of experimental trial. From the data collected, the mean body weight of each bird in infected groups as well as in control group were found out and the result of their statistical analysis are presented in Table 12.

Weekly body weight gain

The gain in body weight for both infected and control group of birds were calculated and its statistical analysis are presented in Table 13 and Fig. 15.

Weekly feed intake

The weekly feed intake for each bird in each group was calculated for both infected as well as control group and these are presented in Table 14 and Fig. 16, the statistical analysis is presented in Table 15.

Feed conversion ratio (FCR)

The weekly and total FCR was calculated from the data and are presented in Table 16 and Fig. 17, the statistical analysis is presented in Table 17.

Table 1. Numbers of mucosal larvae, lumen larvae and adults collected during sacrifice of experimental chicks.

Days of sacrifice	Infected groups															
	A				B				C				D			
	ML	LL	A	POI	ML	LL	A	POI	ML	LL	A	POI	ML	LL	A	POI
2nd day	497	-	-	49.7	758	-	-	37.9	1118	-	-	37.3	1952	-	-	48.48
4th day	510	-	-	51.0	690	-	-	34.5	1242	-	-	41.4	1818	-	-	45.45
6th day	267	-	-	26.7	477	-	-	23.85	723	-	-	24.1	811	-	-	20.28
8th day	227	-	-	22.7	593	-	-	29.65	871	-	-	29.03	932	-	-	23.30
10th day	149	-	-	14.9	483	-	-	24.15	876	-	-	29.2	787	-	-	19.60
12th day	281	-	-	28.1	488	-	-	24.1	521	-	-	17.37	503	-	-	12.58
14th day	143	20	-	16.3	353	17	-	18.5	321	25	-	11.5	393	47	-	11.00
17th day	120	15	-	13.5	280	21	-	15.05	307	28	-	11.17	282	32	-	7.85
21st day	135	20	-	15.5	208	15	-	11.15	311	10	-	10.7	270	31	-	7.53
25th day	27	164	-	19.1	15	290	-	15.75	24	370	-	13.13	30	478	-	12.70
29th day	-	148	-	14.8	-	152	-	7.6	-	199	-	9.97	54	454	-	12.7
33rd day	-	144	20	13.4	-	170	2	8.6	-	119	10	4.3	-	62	28	2.25
37th day	-	38	-	3.8	-	82	-	4.1	-	190	35	7.5	-	117	32	3.73
41st day	-	58	6	64	-	177	12	9.5	-	347	34	11.57	-	189	50	5.98
45th day	-	25	12	3.7	-	36	14	2.5	-	32	37	2.3	-	67	35	2.55
49th day	-	25	22	4.7	-	37	24	3.05	-	72	35	3.57	-	78	48	3.15
53rd day	-	40	22	6.2	-	61	27	4.4	-	107	37	4.57	-	110	72	4.55
56th day	-	-	-	-	-	-	-	-	-	103	79	6.07	-	115	87	5.05
60th day	-	40	31	7.1	-	140	69	4.73	-	-	-	-	-	-	-	-

Note: 1. No. of experimental chicks sacrificed on each day was 2.

2. Chicks of the control group sacrificed along with the experimental group did not reveal the presence of larvae or adults.

3. ML: Mucosal larvae; LL : Lumen larvae; A: Adults.

4. POI: Percentage of infection.

Table 2. Erythrocyte sedimentation rate (in mm/h) in experimental chicks.

Days of collection (Post infection)	Infected groups				Control group	'F' value	Remarks
	A	B	C	D	E		
9th day	2.17±0.31	2.33±0.21	2.33±0.49	2.42±0.49	1.83±0.31	0.38	NS
16th day	2.33±0.21	2.50±0.34	2.67±0.42	2.83±0.31	1.83±0.31	1.40	NS
23rd day	2.50±0.23	2.83±0.21	2.33±0.21	2.50±0.34	2.33±0.42	0.43	NS
30th day	2.33±0.42	2.17±0.31	2.83±0.31	2.83±0.40	2.03±0.37	1.06	NS
37th day	2.00 ± 0.26^{ac}	2.17 ± 0.40^{abc}	2.50 ± 0.23^{bc}	3.00 ± 0.36^{b}	1.50 ± 0.23^{a}	3.74	*
44th day	2.17±0.17	2.00±0.26	2.50±0.23	2.50±0.23	2.00±0.26	1.22	NS
51st day	1.67±0.50	1.67±0.21	2.33±0.42	2.50±0.23	2.00±0.36	1.11	NS

N S Non Significant
* significant at 5% level

Means bearing common letter in the superscript in row wise do not differ significantly

Table 3. Packed cell volume (in percentage) in experimental chicks.

Days of collection (Post infection)	Infected groups A	B	C	D	Control group E	'F' value	Remarks
9th day	30.43±0.69	30.15±0.84	30.26±1.85	29.26±1.33	30.90±1.45	0.18	NS
16th day	31.25±1.03	29.97±0.61	29.09±0.43	29.62±0.67	30.92±0.54	1.42	NS
23rd day	31.50±0.88	31.52±1.55	27.98±0.84	27.83±0.80	31.16±1.19	2.63	NS
30th day	29.40±0.73	29.93±0.39	28.50±1.41	28.62±0.61	32.10±0.69	2.51	NS
37th day	32.12 ± 1.07^{bde}	30.86 ± 0.54^{ad}	31.81 ± 0.63^{ae}	30.04 ± 0.53^{a}	34.79 ± 0.89^{c}	4.54	**
44th day	33.53 ± 2.27^{bd}	31.56 ± 0.56^{ad}	29.08 ± 1.01^{a}	30.44 ± 0.49^{a}	36.36 ± 1.33^{c}	4.00	*
51st day	34.16±1.73	32.69±1.56	31.20±1.03	32.56±2.12	34.37±1.34	0.54	NS

N S Non Significant
* Significant at 5% level
** Significant at 1% level

Means bearing common letter in the superscript in row wise do not differ significantly

Table 4. Haemoglobin (in gms percentage) in experimental chicks.

Days of collection (Post infection)	Infected groups A	B	C	D	Control group E	'F' value	Remarks
9th day	10.27±0.63	9.32±0.48	9.42±0.15	9.03±0.55	9.75±0.70	0.63	NS
16th day	9.65 ± 0.12^{a}	9.79 ± 0.30^{a}	9.64 ± 0.08^{a}	9.39 ± 0.07^{a}	10.85 ± 0.31^{b}	6.51	**
23rd day	9.93 ± 0.13^{bc}	9.76 ± 0.20^{ac}	9.68 ± 0.12^{ac}	9.53 ± 0.16^{a}	10.71 ± 1.33^{b}	4.39	**
30th day	10.16 ± 0.24^{bc}	9.17 ± 0.05^{ac}	9.21 ± 0.11^{ac}	8.66 ± 0.44^{a}	10.66 ± 0.24^{b}	8.67	**
37th day	10.25 ± 0.25^{b}	9.42 ± 0.40^{ac}	9.87 ± 0.23^{bc}	8.93 ± 0.17^{a}	9.98 ± 0.32^{bc}	3.18	*
44th day	9.19±0.34	9.26±0.37	9.51±0.60	9.09±0.37	9.79±0.12	0.44	NS
51st day	9.54±0.43	9.26±0.51	8.96±0.15	8.75±0.14	9.78±0.30	1.17	NS

N S Non Significant
* Significant at 5% level
** Significant at 1% level

Means bearing common letter in the superscript in row wise do not differ significantly

Table 5. Red blood cell count (in millions/cu mm) in experimental chicks.

Days of collection (Post infection)	Infected groups				Control group E	'F' value	Remarks
	A	B	C	D			
9th day	2.90±0.17	2.88±0.09	2.72±0.12	3.01±0.20	2.93±0.09	0.59	NS
16th day	3.14 ± 0.14^{bc}	2.79 ± 0.13^{ac}	2.80 ± 0.12^{ac}	2.67 ± 0.16^{a}	3.34 ± 0.18^{b}	3.69	*
23rd day	3.05 ± 0.06^{bcd}	2.87 ± 0.09^{ad}	2.67 ± 0.11^{ac}	2.63 ± 0.16^{a}	3.14 ± 0.09^{bd}	4.52	**
30th day	2.98±0.12	2.78±0.17	2.83±0.12	2.61±0.14	3.14±0.10	2.29	NS
37th day	2.96±0.09	2.79±0.15	2.64±0.20	2.46±0.21	3.06±0.11	2.34	NS
44th day	2.83±0.17	2.72±0.14	2.71±0.13	2.65±0.18	3.00±0.10	0.91	NS
51st day	2.77±0.12	2.66±0.12	2.71±0.11	2.59±0.14	2.88±0.09	0.77	NS

NS Non Significant
* Significant at 5% level
** Significant at 1% level

Means bearing common letter in the superscript in row wise do not differ significantly

Table 6. Total lecuocyte count (in thousands/cu mm) in experimental chicks.

Days of collection (Post infection)	Infected groups				Control group E	'F' value	Remarks
	A	B	C	D			
9th day	28.03±0.97	25.22±1.83	26.96±1.20	30.58±1.02	27.10±1.09	2.42	NS
16th day	33.00±1.84^{b}	36.40±0.84^{bd}	36.11±0.94^{bd}	37.89±1.20^{cd}	22.87±1.03^{a}	24.84	**
23rd day	34.19±1.43^{b}	39.51±0.70^{cd}	36.22±1.43^{bd}	38.34±1.27^{bd}	25.48±0.69^{a}	23.27	**
30th day	30.29±1.12^{b}	30.07±0.60^{b}	30.51±1.38^{b}	35.55±0.96^{c}	25.02±1.49^{a}	10.43	**
37th day	25.54±1.00^{a}	29.88±1.27^{b}	32.42±0.66^{b}	31.06±0.62^{b}	24.31±1.35^{a}	11.90	**
44th day	26.27±0.94^{ac}	25.99±1.29^{ac}	30.84±0.76^{b}	27.29±1.32^{bc}	22.68±1.34^{a}	6.47	**
51st day	28.10±1.24	28.64±1.26	28.48±1.03	27.88±1.12	28.81±0.98	2.59	NS

NS Non Significant
* Significant at 5% level
** Significant at 1% level

Means bearing common letter in the superscript in row wise do not differ significantly

Table 7. Heterophil count (in percentage) in experimental chicks.

Days of collection (Post infection)	Infected groups				Control group E	'F' value	Remarks
	A	B	C	D			
9th day	32.01 ± 0.62^{ae}	33.35 ± 1.16^{be}	38.28 ± 0.58^{cf}	40.23 ± 1.19^{df}	29.62 ± 0.50^{a}	15.75	**
16th day	36.39 ± 1.16^{b}	36.70 ± 1.10^{b}	37.09 ± 0.56^{b}	38.00 ± 1.15^{b}	28.29 ± 0.26^{a}	16.14	**
23rd day	35.52 ± 0.72^{b}	36.62 ± 0.60^{b}	35.48 ± 0.82^{b}	36.35 ± 0.68^{b}	29.79 ± 0.29^{a}	16.24	**
30th day	37.39 ± 0.70^{b}	37.17 ± 0.75^{b}	36.57 ± 0.85^{b}	37.86 ± 0.52^{b}	29.36 ± 0.35^{a}	25.18	**
37th day	34.07 ± 1.08^{b}	36.42 ± 0.99^{bd}	36.71 ± 0.90^{cd}	35.71 ± 1.19^{bd}	29.39 ± 0.36^{a}	8.65	**
44th day	34.32 ± 0.60^{c}	32.52 ± 0.50^{bd}	31.41 ± 0.49^{ad}	32.73 ± 0.69^{bd}	30.48 ± 0.45^{a}	5.64	**
51st day	30.84 ± 0.60^{a}	32.16 ± 1.17^{b}	33.19 ± 0.68^{b}	33.38 ± 0.73^{b}	29.98 ± 0.45^{a}	3.13	*

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

Means bearing common letter in the superscript in row wise do not differ significantly

Table 8. Eosinophil count (in percentage) in experimental chicks.

Days of collection (Post infection)	Infected groups A	B	C	D	Control group E	'F' value	Remarks
9th day	2.48 ± 0.37^{b}	3.47 ± 0.44^{b}	3.94 ± 0.54^{b}	3.37 ± 0.37^{b}	1.84 ± 0.29^{a}	3.82	*
16th day	3.16 ± 0.22^{a}	4.44 ± 0.77^{ac}	4.80 ± 0.43^{ac}	6.14 ± 0.55^{bc}	1.96 ± 0.24^{a}	10.58	**
23rd day	3.47 ± 0.27^{ac}	4.23 ± 0.57^{ac}	5.11 ± 0.44^{bc}	5.63 ± 0.41^{bc}	1.71 ± 0.32^{a}	12.73	**
30th day	2.76 ± 0.17^{a}	3.49 ± 0.30^{ac}	5.24 ± 0.46^{bc}	5.56 ± 0.53^{bc}	1.68 ± 1.43^{a}	21.82	**
37th day	3.81 ± 0.34^{acd}	4.25 ± 0.19^{ace}	5.28 ± 0.56^{bc}	6.36 ± 0.75^{bde}	1.78 ± 0.27^{a}	14.00	**
44th day	3.92 ± 0.31^{ac}	4.09 ± 0.60^{ac}	5.83 ± 0.31^{bc}	6.28 ± 0.55^{b}	1.74 ± 0.14^{a}	20.95	**
51st day	2.66 ± 0.08^{ab}	3.09 ± 0.22^{ab}	3.08 ± 0.21^{ab}	4.16 ± 0.38^{b}	2.06 ± 0.20^{a}	8.82	**

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

Means bearing common letter in the superscript in row wise do not differ significantly

Table 9. Basophil count (in percentage) in experimental chicks.

Days of collection (Post infection)	Infected groups A	B	C	D	Control group E	'F' value	Remarks
9th day	3.11 ± 0.37^{b}	2.97 ± 0.29^{b}	4.17 ± 0.49^{b}	4.19 ± 0.52^{b}	2.23 ± 0.31^{a}	3.94	*
16th day	2.81 ± 0.28^{b}	3.54 ± 0.52^{b}	3.50 ± 0.33^{b}	4.19 ± 0.45^{b}	1.99 ± 0.09^{a}	4.38	**
23rd day	3.30 ± 0.55^{b}	3.35 ± 0.44^{b}	4.36 ± 0.47^{b}	4.57 ± 0.94^{b}	1.06 ± 0.25^{a}	7.45	**
30th day	2.03 ± 0.35^{b}	2.80 ± 0.32^{b}	3.18 ± 0.18^{b}	3.29 ± 0.21^{b}	1.83 ± 1.15^{a}	6.24	**
37th day	2.29 ± 0.20^{b}	2.39 ± 0.55^{b}	2.97 ± 0.27^{b}	4.22 ± 0.68^{b}	1.58 ± 0.15^{a}	5.47	**
44th day	2.95 ± 0.20^{a}	4.62 ± 0.37^{b}	5.28 ± 0.55^{b}	4.45 ± 0.66^{b}	2.51 ± 0.22^{a}	8.36	**
51st day	2.33 ± 0.20	2.49 ± 0.19	2.27 ± 0.46	2.39 ± 0.42	1.55 ± 0.13	1.34	NS

NS Non Significant
* Significant at 5% level
** Significant at 1% level

Means bearing common letter in the superscript in row wise do not differ significantly

Table 10. Lymphocyte count (in percentage) in experimental chicks.

Days of collection (Post infection)	Infected groups				Control group E	'F' value	Remarks
	A	B	C	D			
9th day	53.63 ± 0.94^{c}	50.51 ± 1.53^{b}	45.05 ± 1.20^{a}	43.03 ± 1.23^{a}	57.75 ± 0.94^{d}	21.96	**
16th day	48.42 ± 1.19^{b}	46.24 ± 0.84^{b}	46.51 ± 1.05^{b}	43.63 ± 1.04^{a}	60.92 ± 0.99^{c}	36.33	**
23rd day	50.08 ± 0.59^{c}	48.30 ± 0.50^{b}	46.93 ± 0.68^{b}	44.49 ± 1.07^{a}	59.84 ± 0.39^{d}	61.00	**
30th day	49.85 ± 0.46^{c}	48.45 ± 0.76^{c}	46.09 ± 0.86^{b}	43.81 ± 0.83^{a}	59.96 ± 0.20^{d}	70.52	**
37th day	53.24 ± 1.13^{c}	49.89 ± 0.57^{c}	46.14 ± 0.87^{b}	45.52 ± 1.30^{a}	60.68 ± 0.47^{d}	37.78	**
44th day	52.68 ± 0.57^{b}	52.24 ± 0.70^{b}	50.20 ± 0.54^{a}	49.05 ± 1.12^{a}	59.29 ± 0.44^{c}	24.14	**
51st day	56.64 ± 0.67^{b}	55.59 ± 1.21^{b}	52.56 ± 0.79^{a}	52.16 ± 0.34^{a}	60.22 ± 0.55^{c}	13.46	**

** significant at 1% level

Means bearing common letter in the superscript in row wise do not differ significantly

Table 11. Monocyte count (in percentage) in experimental chicks.

Days of collection (Post infection)	Infected groups A	B	C	D	Control group E	'F' value	Remarks
9th day	8.73±0.42	8.51±1.54	8.53±0.80	8.83±0.57	8.44±0.38	0.08	NS
16th day	8.21±0.60	8.54±0.37	9.14±0.18	8.32±0.83	7.17±0.73	1.10	NS
23rd day	8.07±0.58	7.86±0.53	8.46±0.57	8.52±0.35	7.73±0.30	0.50	NS
30th day	7.98 ± 0.15^{ac}	8.12 ± 0.28^{ac}	9.01 ± 0.36^{bc}	9.51 ± 0.23^{b}	7.22 ± 0.09^{a}	10.16	**
37th day	7.24 ± 0.44^{ab}	7.11 ± 0.41^{ab}	8.79 ± 0.46^{b}	8.38 ± 0.33^{ab}	6.70 ± 0.21^{a}	4.47	**
44th day	6.78±0.39	7.03±0.25	7.46±0.43	7.49±0.40	6.33±0.22	1.75	NS
51st day	6.57±0.63	7.04±0.18	7.12±0.18	7.43±0.23	6.23±0.31	1.43	NS

NS Non Significant
** significant at 1% level

Means bearing common letter in the superscript in row wise do not differ significantly

Table 12. Mean weekly body weight (in gms) of A. galli. infected chicks.

Age in weeks	Infected groups A	B	C	D	Control group E	F value	Remarks
0	45.50±0.32	45.50±0.32	44.70±0.26	43.50±0.19	46.00±0.21	--	--
I	146.27±4.43^{a}	125.16±4.55^{c}	109.14±4.31^{b}	107.42±4.43^{b}	147.86±4.31^{a}	20.29	**
II	281.11±8.51^{a}	229.58±9.02^{b}	215.56±8.51^{b}	148.57±8.36^{c}	310.00±8.07^{d}	56.90	**
III	548.57±17.48^{d}	448.16±18.38^{a}	382.38±17.48^{b}	245.46±17.08^{c}	528.26±16.71^{d}	51.42	**
IV	898.24±23.48^{c}	676.00±24.99^{a}	611.77±23.48^{a}	461.67±22.81^{b}	934.44±22.81^{c}	74.62	**
V	1214.00±35.00^{a}	976.92±37.60^{b}	907.33±48.11^{b}	752.44±33.89^{c}	1328.75±33.89^{d}	63.82	**
VI	1549.09±48.11^{a}	1385.00±50.46^{b}	1332.73±48.11^{b}	1092.33±46.00^{c}	1897.50±46.00^{d}	53.07	**
VII	1803.33±52.14^{a}	1611.25±55.30^{b}	1587.78±52.14^{b}	1326.56±52.14^{c}	2195.46±47.16^{d}	53.31	**
VIII	1962.50±89.01^{a}	1835.00±89.01^{a}	1785.00±89.01^{ab}	1555.00±72.67^{b}	2435.71±67.28^{c}	21.69	**

** Significant at 1 per cent level

Means bearing common letter in the superscript in row wise do not differ significantly.

Table 13. Mean weekly body weight gain (in gms) in A. galli. infected chicks.

Age in weeks	Infected groups A	B	C	D	Control group E	'F' value	Remarks
0	45.50± 0.32	45.50± 0.32	44.70± 0.26	43.50± 0.19	46.00± 0.02	—	--
I	100.50± 4.71^{c}	79.00± 4.66^{b}	64.00± 4.38^{a}	63.00± 4.51^{a}	102.00± 4.38^{c}	18.15	**
II	135.00± 7.98^{c}	105.00± 8.47^{b}	107.00± 7.98^{b}	42.00± 7.84^{a}	163.00± 7.57^{d}	33.16	**
III	267.00± 15.54^{d}	218.00±16.34^{c}	166.00±15.54^{b}	96.00±15.18^{a}	218.00±14.85^{c}	17.92	**
IV	350.00± 18.65^{b}	228.00±19.76^{a}	229.00±18.56^{a}	217.00±18.04^{a}	406.00±18.04^{c}	22.19	**
V	316.00± 24.90^{b}	301.00±26.75^{b}	296.00±24.90^{b}	291.00±24.11^{a}	395.00±24.11^{c}	11.10	**
VI	335.00± 43.19^{a}	408.00±45.30^{ab}	426.50±43.19^{b}	340.00±41.86^{ab}	569.00±41.36^{c}	5.17	**
VII	160.00± 34.54^{b}	224.00±34.54^{a}	197.00±34.54^{a}	234.00±28.20^{a}	241.00±26.11^{a}	5.83	**
VIII	254.00± 26.41	226.00±28.42	255.00±26.45	228.50±27.98	297.00±23.12	1.29	NS

** Significant at 1 per cent level

NS Non Significant

Means bearing common letter in the superscript in row wise do not differ significantly.

Table 14. Mean weekly feed consumption (g) per bird in infected chicks.

Age in weeks	Infected groups A	B	C	D	Control group E
I	120	109	135	105	145
II	280	235	182	142	251
III	573	448	411	363	581
IV	649	609	599	496	689
V	689	633	608	502	718
VI	886	866	845	691	1057
VII	875	980	957	887	1035
VIII	860	869	845	843	1026
Total	4932	4749	4582	4029	5502

Table 15. Comparison of cumulative feed intake (g) in A. galli. infected chicks and with control.

Sl. No.	Groups	Cumulative feed intake (Mean ± SE)		't' value	Remarks
1.	A Vs B	616.50±100.60	593.60±110.70	0.1530	NS
2.	A Vs C	616.50±100.60	572.80±109.20	0.2946	NS
3.	A Vs D	616.50±100.60	503.60±104.10	0.7798	NS
4.	A Vs E	616.50±100.60	687.80±124.60	-0.4450	NS
5.	B Vs C	593.60±110.70	572.80±109.20	0.1342	NS
6.	B Vs D	593.60±110.70	503.60±104.10	0.5924	NS
7.	B Vs E	593.60±110.70	687.80±124.60	-0.5649	NS
8.	C Vs D	572.80±109.20	503.60±104.10	0.4581	NS
9.	C Vs E	572.80±109.20	687.80±124.60	-0.6941	NS
10.	D Vs E	503.60±104.10	687.80±124.60	-1.1342	NS

NS Non Significant

Table 16. Mean weekly feed conversion ratio of A. galli infected chicks.

Age in weeks	Infected groups				Control group
	A	B	C	D	E
I	1.1940	1.3797	2.1093	1.6666	1.4215
II	2.0740	2.2380	1.7009	3.3809	1.5493
III	2.1461	2.0550	2.4759	3.7812	2.6651
IV	1.8542	2.6770	2.6157	2.2857	1.6970
V	2.1803	2.1029	2.0540	1.7250	1.8177
VI	2.6447	2.1225	1.9835	2.0323	1.8576
VII	3.3850	3.8451	3.3137	3.7906	3.4545
VIII	5.4687	4.3750	4.8578	3.6812	4.2946
Total	2.62±0.46	2.60±0.33	2.64±0.34	2.79±0.27	2.35±0.34

Table 17. Comparison of mean feed conversion ratio of A. galli infected chicks and with control.

Sl. No.	Groups	Cumulative feed conversion ratio (Mean ± SE)			't' value	Remarks
1.	A Vs B	2.6183±0.469	Vs	2.5987±0.356	0.1283	NS
2.	A Vs C	2.6183±0.468	Vs	2.6389±0.362	0.0587	NS
3.	A Vs D	2.6183±0.468	Vs	2.7857±0.279	-0.2093	NS
4.	A Vs E	2.6483±0.462	Vs	2.3447±0.368	0.5587	NS
5.	B Vs C	2.5987±0.356	Vs	2.6389±0.362	-0.0792	NS
6.	B Vs D	2.5987±0.356	Vs	2.7857±0.279	-0.4813	NS
7.	B Vs E	2.5987±0.356	Vs	2.3447±0.368	0.4965	NS
8.	C Vs D	2.6389±0.362	Vs	2.7857±0.279	-0.3214	NS
9.	C Vs E	2.6389±0.362	Vs	2.3447±0.368	0.5704	NS
10.	D Vs E	2.7857±0.279	Vs	2.3447±0.368	0.9565	NS

NS Non Significant

Fig. 15. Graph showing mean weekly body weight gain (in gms) in experimental chicks

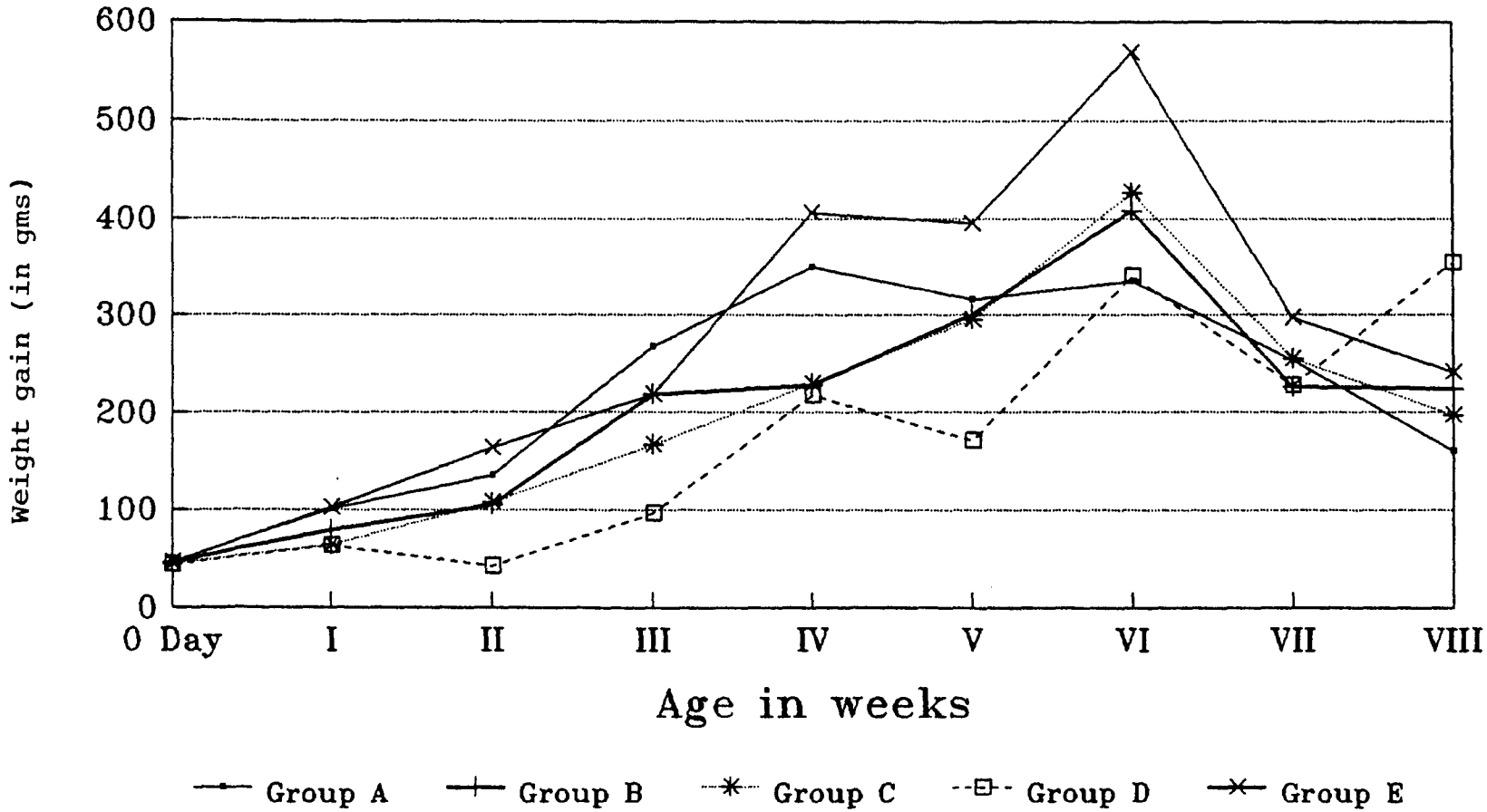


Fig.16. Graph showing mean weekly feed intake (in gms) in experimental chicks

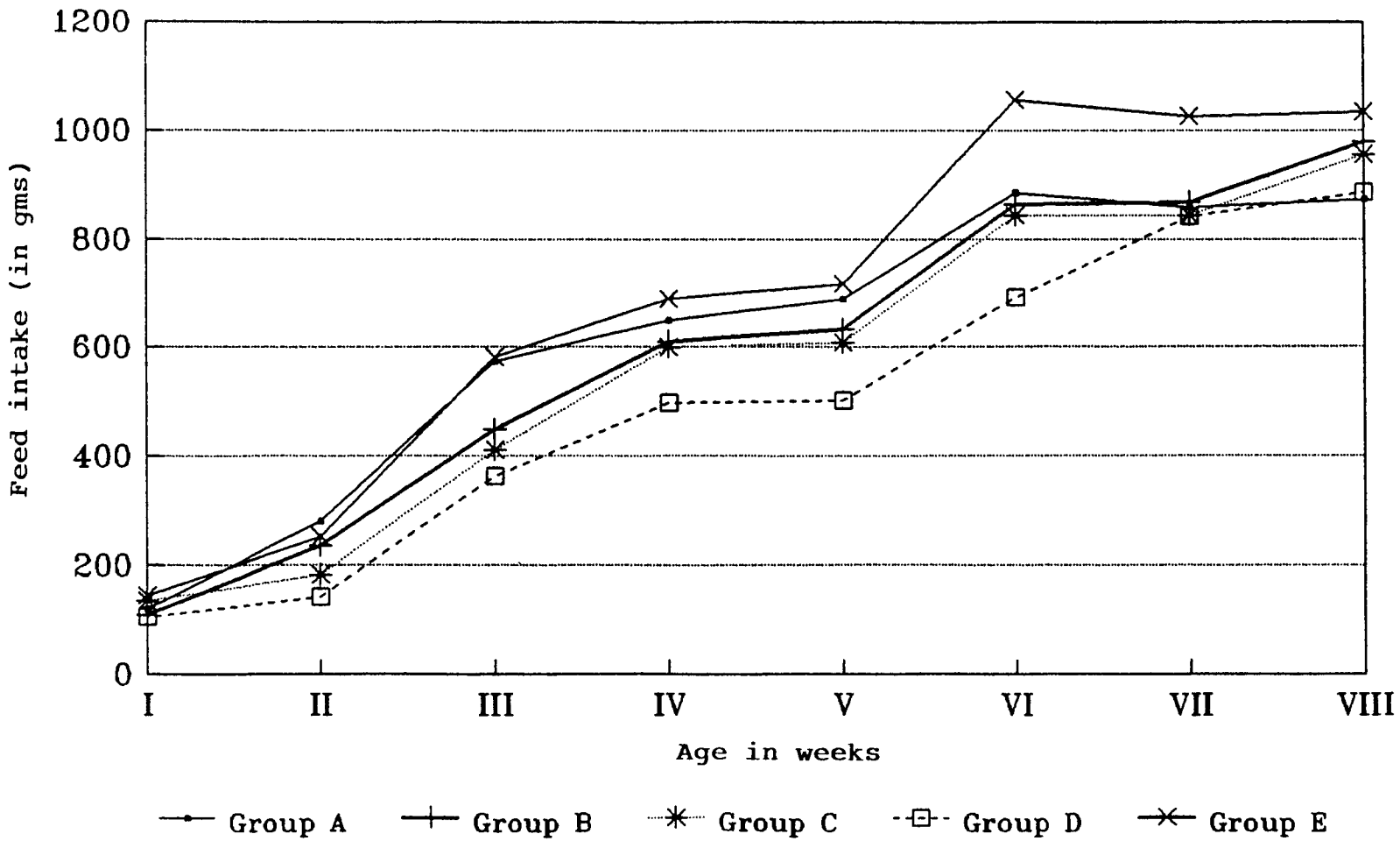
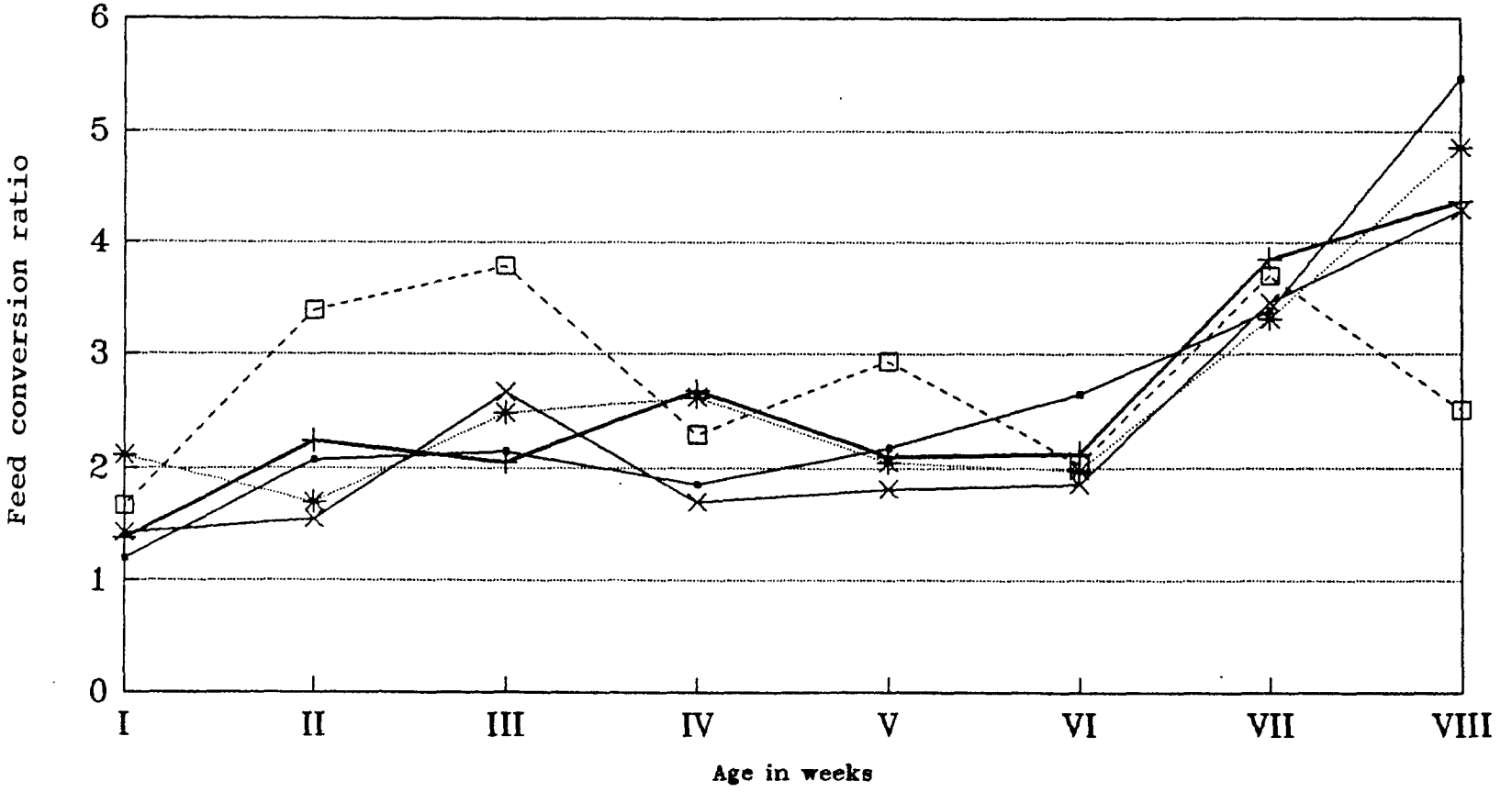


Fig. 17. Graph showing mean weekly feed conversion ratio in experimental chicks



Discussion

DISCUSSION

General condition

In the present experimental trial, in general, the infected birds were found to be dull, drooping, passing loose motion with loss of appetite, reduction in growth rate and increased thirst. When the birds of group C and D which received a higher dose of infection showed from 10th day PI, emaciation, unthriftiness, ruffled feathers, leg weakness and anaemia besides the late development of comb, wattles and feathers.

Tugwell and Ackert (1952) reported that the birds had signs of general weakness from 9th day PI. Deo (1964), Soulsby (1982) and Verma et al. (1993) observed that the young birds were more susceptible than the older ones. The above observations concur with the findings in the present investigation.

In this study, clinical signs of droopiness, off feed, drowsiness and diarrhoea noted in the infected birds are in agreement with the observations made by Khouri and Pande (1970), Fathithu et al. (1992), Padmaja (1992) and Verma et al. (1993).

In the present trial even though the birds were maintained with good quality commercial bbroiler mash, the

clinical signs of dullness, emaciation and weakness of legs were predominant in birds that had received 1500 and 2000 infective eggs of A. galli. These observations are in agreement with that of Deo (1964), Soulsby (1982) and Padmaja (1992). The delay in the development of feathers as reported by Raote et al. (1992) and the paleness and delay in the development of comb and wattles as observed by Padmaja (1992) in addition to the above,were noted in this trial also.

Gross lesions

In the present experimental trial, varying degrees of congestion and petechial haemorrhage in the mucosa of duodenum and jejunum which at certain places coated with blood and the frothy and mucoid intestinal contents were observed in all the groups of infected birds from 4th to 14th day PI. These findings are contrary to those of Sadun (1950) and Kadziolka (1956), who had reported vascular changes in the region of duodenum and jejunum and haemorrhagic appearance of mucosa from 10 to 13 days of infection and Padmaja (1992) who observed gross lesions from 10th to 20th day PI. The narrowness of some parts of lumen thickening and distension of the wall of small intestine noted in the present study (group D) are in pact with the reports of Sazikova (1975) who noted marked distension and

thickening of intestinal wall and who observed distension of first half of intestine. Muraleedharan and Seshadri (1985) and Verma et al. (1993) also observed thickening of the intestinal mucosa along with red bite marks. Obstruction of intestine was noticed in the present trial with immature and adult worms in birds which had received the highest dose of infection. This observation is in agreement with the reports of Soulsby (1982), Ramadan and Znada (1991), Padmaja (1992) and Verma et al. (1993).

There are many observations made by several workers on gross lesions caused by A. galli in domestic fowls. The formation of pedunculated and diverticulated protuberances on the intestinal serosa was reported by Deorani (1968) and Kaushik (1980). Lee (1969) noticed the formation of nodule like masses on the inner wall of intestine. Intestinal perforation and pleuroperitonitis were observed by Vegad et al. (1979) in chicken which were naturally infected with A. galli. Matta (1980) noticed the generalized oedema in chicks after 15 days PI. The ballooning, twisting and intussusception of the small intestine were reported by Scott (1952), Muraleedharan and Seshadri (1985), Rai et al. (1989) and Mukit et al. (1991). Atrophy of thymus, increased relative weight of liver and spleen had been reported by Sadun (1950). Sazikova (1975) reported a marked reduction in the weight of heart and liver besides their atrophy. Though

the adult worms were localized in the region of caecum and colon they did not cause any pathological lesion (Padhi et al., 1987). Congestion of lungs, heart and kidneys and focal diffused areas of haemorrhage in the liver was reported by Sadun (1950) and Padmaja (1992) respectively. None of these observations could not be found in the present trial.

Collection of worms from intestine

In the present experimental trial, the larvae of A. galli could be recovered from the mucosa of the small intestine from 2nd to 17th day PI. Ackert and Tugwell (1948) reported that from 3rd to 24th day after experimental infection, the young worms were located within the mucous membrane of small intestine. But, in this trial, from 14th day PI on wards majority of the young worms were found free in the lumen and the few were within the mucosa of small intestine.

Varghese (1966) opined that the larvae at certain stages of development were seen closely adherent to the mucous membrane with their anterior extrimities burried in between the crypts. This observation favourably compares with the findings of the present study. But Birova - Volosinovicova (1971) had reported that all the larvae were seen free in the lumen of the intestine without migration into the intestinal mucosa. Padhi et al. (1987) reported

that adult A. galli were seen in caecum and colon but no such observation were made in the present study. Padmaja (1992) observed that during post mortem examination of the infected chicks, majority of the larvae were found attached to the intestinal mucosa, from 10th to 20th day PI whereas in the present trial this was noticed from 2nd to 17th day PI. She had also reported that young worms could be found free in the lumen only after 30 days PI, but in the present work, young worms were found free in the lumen of intestine as early as 14 days PI.

The present observation on the presence of larvae in the mucosa anddin the lumen of the small intestine are in agreement with many workers with minor variations.

Tugwell and Ackert (1950) found that majority of young A. galli worms were seen in the mucous membrane of small intestine between 8th to 17th day PI. Tsvetaeva (1954) noticed the presence of young worms only in the lumen of the small intestine. Deo and Srivastava (1955a) reported that 3rd stage larvae were found free in the lumen after 15 days PI. Khouri and Pande (1970) confirmed the availability of 2nd and 3rd stage larvae in the mucosa as well as in the lumen followed by 4th stage larvae and juvenile worms in the lumen of intestine between 8th to 15th day PI.

On the contrary, Todd and Crowdus (1952) reported that all the worms matured without leaving the lumen of the intestine, stressing the absence of tissue-phase. In the present study, the larvae were seen attached to mucosa as early as 2nd day PI and juvenile worms were seen free in the lumen from 14th day PI onwards.

From the Table 2 it is evident that the number of larvae hatched out in the intestine and developed to subsequent stages were high in those groups of chicks which received low dose of infection. During 2nd to 6th day PI, the number of larvae hatched out were high in all the experimental chicks and the number of larvae developed into adult worms were decreased from 8th to 14th day PI and much less from 14th day PI onwards in all the groups. This may be due to the age resistance and is also evidenced from the gain in body weight which was higher during 5th and 6th weeks PI. This observation is in correlatin with the findings of Dimitrov (1971) who stated that the number of worms developed in the intestine decreased as the infective dose increased in experimental infection in broilers. Reddy et al. (1984) in their comparative study stated that the highest percentage of larvae was recovered from intestinal mucosa of chick fed with normal diet during the first 2 week PI. Pavlicek and Dykova (1975) reported a highest worm burden of upto 54 per cent of eggs given, when the birds

were infected with 1500 to 3000 infective A. galli eggs, while in the present study the highest worm burden of 51 per cent was obtained in birds infected with 500 infective eggs of A. galli.

Mortality and Morbidity rate

In the present study there was no mortality in any of the infected group of chicks including the controls. But the morbidity rate was high, as almost all the infected chicks showed symptoms of ascaridiasis with varying degrees of severity, proportional to the dose of infection. The literature on mortality rate in chicks due to A. galli infection appears to be scanty. However, Sadun (1950) observed heavy mortality in chicks which had received 14000 infective eggs of A. galli on 14 days PI. But at the same time he reported that the birds tolerated a milder dose of 500 embryonated eggs of A. galli.

Deo (1964) stated heavy mortality and loss of weight during the 3rd week after infection while Pavlicek and Dykova (1975) reported a 46 to 100 per cent mortality rate in A. galli infected birds, both these are in contrast with the present findings.

Maturation period of Ascaridia galli

The prepatent period of A. galli which received different doses of infective eggs (500, 1000, 1500 and 2000 numbers) was 59, 59, 50 and 44 days respectively.

The available literature on the prepatent period of A. galli in chicken appears to be limited. According to Ackert (1931) it was about 50 days. Feoktisov (1950) reported that the worms attained maturity in 35 days in younger chicken and in 58 days in adult hens. According to Kerr (1955), Deo and Srivastava (1955 a) it was between 30 to 35 days and 28 to 34 days in younger birds respectively. The variations in the maturation period of A. galli may be due to a variation in the dose of infection. Further Reddy et al. (1984) noted that the maturation period was 35 to 37 days PI. The present observation is favouring the findings of Ackert (1931) and Feokitsov (1950) and contrary to the findings of Kerr (1955), Deo and Srivastava (1955a) and Reddy et al. (1984).

Blood Parameters

Erythrocyte Sedimentation Rate (ESR)

The mean ESR values of all experimental groups of birds are presented in Table 2. The ESR was found increased in all the infected groups than the control group on every week

after 9th day PI up to 37th day PI. On 37th day PI, a statistically significant increase ($P < 0.05$) than the control group was noticed in groups C and D. Among the infected groups significant increase was noticed in group D than group A .Later, 44th day PI onwards the trend was reversed and there was gradual decrease in ESR.

Matta and Ahluwalia (1982), Sekhar et al. (1988) and Padmaja (1992) also reported comparable increase in ESR during first 3 weeks PI.

Packed Cell Volume (PCV)

The data on PCV with its statistical analysis are furnished in Table 3. Substantial decrease in PCV was noticed in all infected groups than control group. But the difference was statistically significant in all the infected groups at 1 per cent level on 37th day ($P < 0.01$) and 5 per cent level on 44th day PI ($P < 0.05$) only.

On 37th day PI, among the infected groups, significant decrease in ($P < 0.01$) PCV was noticed in group D over the group A. On 44th day PI, PCV was decreased ($P < 0.05$) in groups C and D than group A. Later, PCV of infected birds reached comparable levels with control group.

These observations are in agreement with those of Matta and Ahluwalia (1982), Rao and Lal (1991) and Padmaja (1992).

On the contrary, an increase in PCV after infection was reported by Ikeme (1971). Differing with both these findings Fathithu et al. (1992) and Verma et al. (1993) reported lack of marked variations in PCV in A. galli infected chicken.

Haemoglobin

Data on haemoglobin concentration and its statistical analysis are given in Table 4. There was reduction in the haemoglobin concentraion in all the infected groups than the controls. But the reduction was statistically significant at 1 per cent level ($P<0.01$) on 16th, 23rd, 30th day PI and 5 per cent level ($P< 0.05$) on 37th day PI.

On 16th day PI, significant reduction was noticed in all the infected groups than control. Among the infected groups, significant difference did not exist. On 23rd and 30th day PI, groups B,C and D recorded statistically significant reduction ($P< 0.01$) over control. Among the infected groups, group D was significantly differed from group A. On 37th day PPI,a significant decrease ($P< 0.05$) in haemoglobin was noticed in group D than control and over groups A and C among the infected groups.

Ikeme (1971), Matta and Ahluwalia (1982), Sekhar et al. (1985), Rao and Lal (1991) and Padmaja (1992) reported a

significant decreasein haemoglobin content in Ascaridia infection. The decrease in the content may be due to penetrating activities of larval stages of A. galli resulting in destruction of mucosa and rupture of small blood vessels. But the histopathologgicalsections revealed a less number of haemorrhagic cases, the reduction could be due to starvation anameia.

But Birova - Volosinovicova (1974) and Verma et al. (1993) reported that there were no variation in haemoglobin level in Ascaridia infection. The observation of the present investigation lent support to the findings of the former workers.

Red blood cell count

The data on RBC and its statistical analysis are presented in the Table 5. Although reduction in RBC was noticed in all the infected groups, the variation was statistically significant only on 16th and 23rd day PI ($P < 0.05$) and ($P < 0.01$) respectively. Significant reduction was noticed in groups B, C and D ($P < 0.05$) over the control on 16th day PI. Among the infected groups RBC count of group D was significantly differed ($P < 0.05$) from that of group A. On 23rd day PI, group C and D significantly

differed from control ($P < 0.01$) and among the infected groups, group D was significantly differed from group A,B and C.

Sadun (1950), Sekhar and Simha (1985), Sekhar et al. (1988), Rao and Lal (1991) and Padmaja (1992) observed severe anaemia and significant decrease in RBC count in birds infected with A. galli. The findings of present study are in agreement with their observations, but contrary to those of Birova - Volosinovicova (1974) and Verma et al. (1993) who stated that the RBC counts were found within the physiological limits.

The increase in ESR and decrease in PCV and total RBC count recorded in this study established evidence of anaemia in all the infected birds, although very specific correlation with the stage and dose of infection could not be drawn from the data analysed, but it is worthwhile to point out that whereever anaemia was found statistically significant, birds of group D was the most affected. This indicated a positive correlation between the dose rate and the intensity of anaemia in ascaridiasis.

White blood Cell Count

The statistically analysed data on WBC count are presented in Table 6. There was a significant increase in

all the infected groups than controls from 16th day PI till 44th day PI ($P < 0.01$). On 16th day PI, the difference was significant at 1 per cent level ($P < 0.01$) in all the infected groups than control. Among the infected groups, group D significantly differed ($P< 0.01$) from group A. On 23rd day PI, the difference was significant ($P <0.01$) in all the infected groups than control. Among the infected groups, significant increase ($P < 0.01$) was noticed in group B than group A. On 30th day PI, significant increase ($P<0.01$) was noticed in all infected groups than control. Among the infected groups, group D significantly differed ($P < 0.01$) from groups A, B and C. On 37th day PI, significant increase was noticed ($P <0.01$) in infected groups B,C and D than control and group A. On 44th day PI, significant increase was noticed ($P < 0.01$) in groups C and D than control. Among the infected groups, group C significantly differed from group A and B ($P < 0.01$).

Later, on 51st day PI, the total WBC counts were turned to normal in the infected groups. The findings of Sadun (1950), Kaushik and Sen (1978) and Padmaja (1992) also recorded comparable increase in total WBC count in ascaridiasis. There are many controversial reports on the WBC count in ascaridiasis. Rao and Lal (1991) observed a significant decrease in WBC count, whereas Birova -

Volosinovicova (1974) and Verma et al. (1993) recorded no change. Alternatively, either a significant decrease or increase was reported by Sekhar and Simha (1986).

An increase in the total WBC indicates the existence of an inflammatory reaction and in the present study evidence of enteritic lesions was observed consistantly both grossly as well as histopathologically. Clinical signs also confirmed existence of enteritis.

Heterophil

Data on heterophil count with statistical analysis are presented in Table 7. A significant increase in heterophil count was observed from 9th day PI till the end of the trial. On 9th day PI, significant increase ($P < 0.01$) was noticed in groups B, C and D than group A. Among the infected groups, groups C and D significantly differed from groups A and B. On 16th, 23rd and 30th day PI, a statistically significant ($P < 0.01$) increase in hetrophil count was noticed in all the infected groups than the control, but significant difference did not exist among the infected groups. On 37th day PI also the infected groups showed significant increase ($P < 0.01$) in heterophil count than control. Among the infected groups, group C showed significant difference from group A ($P < 0.01$). On 44th day PI, significant increase in the count was noticed

in groups A, B and D than control ($P < 0.01$). Among the infected groups, group A significantly differed ($P < 0.01$) from other groups. On 51st day PI, statistically significant increase ($P < 0.05$) in heterophil count was noticed in groups B, C and D than group A and control.

Although Birova-Volosinovicova (1974) stated that the leucogram values were within the physiological limits. Kaushik and Sen (1980), Rao and Lal (1991) and Padmaja (1992) reported an increase in the heterophil count in experimental studies. In the present study, an increase in the total WBC count was observed in the infected birds and this might partially be due to the heterophilia. It is also to be noted that lesions of enteritis were seen throughout the intestinal segments of experimental birds without any marked relationship with the sections of larvae. The parasitic load might have affected the disease resistance of the bird and in turn resulted in flaring up of bacterial enteritis. This might explain the heterophilia and leucocytosis observed in the present study.

Eosinophil

The data on eosinophil count and its statistical analysis are presented in Table 8. A significant increase ($P < 0.05$ and $P < 0.01$) in eosinophil count in infected groups throughout the experimental study was observed.

On 9th day PI, all the infected groups significantly differed ($P < 0.05$) from control group and were homogeneous. On 16th day PI, group D statistically differed ($P < 0.01$) from control group. Among the infected groups, group D significantly differed ($P< 0.01$) from group A. On 23rd day PI, group C and D significantly differed ($P < 0.01$) from control. On this day all the infected groups were homogenous and did not differ significantly.

On 30th day PI, significant increase ($P<0.01$) was noticed in groups C and D than conttrol,but significantly differed from group A. On 37th day PI also groups and D showed an significant increase ($P<0.01$)than control, but all infected groups were homogenous.

On 44th day PI groups C and D significantly differed ($P < 0.01$) from control. Among the infected groups, group D significantly differed from groups A and B. On 51st day PI, statistically significant increase in count ($P < 0.01$) was noticed in group D than control. Infected groups did not differ significantly among themselves.

Sadun (1950), Kaushik and Sen (1978), Rao and Lal (1991) and Padmaja (1992) also reported an increased eosinophil count in A. galli infected chicken. Eosinophilia in parasitic infection is a characteristic clinical feature and the present study also substantiates this concept.

Basophil

The summarised data on mean basophil count and its statistical analysis are set out in Table 9. A significant increase in basophil count from 9th to 44th day PI ($P < 0.05$ and $P < 0.01$) had been observed. Later, on 51st day PI, though the count was higher in the infected groups it did not differ significantly.

On 9th day PI, all the infected groups significantly differed ($P < 0.05$) from control group. On 16th, 23rd, 30th and 37th day PI also the infected groups differed significantly ($P < 0.01$) from control group. On 44th day PI, groups B, C and D significantly differed ($P < 0.01$) from control and infected group A.

These findings are in concurrence with the observations of Kaushik and Sen (1978) and Padmaja (1992) but contrary to the observation of Sadun (1950) who observed neither increase nor decrease in basophil count in birds which received a milder dose of infection.

Lymphocyte

The summarised data on mean lymphocyte count and its statistical analysis are presented in Table 10. There was significant decrease in lymphocyte in all the infected groups of chicken from 9th day PI till the end of trial

($P < 0.01$). In later part of experiment, the lymphocyte count gradually increased but it was still lesser than the control group.

On 9th day PI, all the infected groups significantly differed ($P < 0.01$) fromcontrol. Among the infected groups, a significant decrease ($P < 0.01$) in lymphocyte was noticed in groups C and D than group A and B and also the group B was significantly differed from group A.

On 16th and 23rd PI, among infected groups, group D significantly differed from control and other 3 infected groups. From 30th day PI till the end of the experiment significant reduction in lymphocyte was noticed ($P < 0.01$) in groups C and D over the control and other infected groups.

A significant decrease in lymphocyte in A. galli infected chicken was reported by Kaushik and Sen (1978). On the contrary, Sadun (1950),Rao and Lal (1991) and Padmaja (1992) stated that there was a significant increase in lymphocyte in A. galli infected chicken.

Although, the lymphocyte count was consistently lower than the control values in this study during the initial period, towards the end of experiment this trend was

reversed and an increase in the count was characteristically observed. This probably reflects an immunological response to the long standing infection.

Monocyte

The data on monocyte count and its statistical analysis are given in Table 11. There was an increase in the monocyte count in all the infected groups of chicks when compared to control, but the difference was statistically significant on 30th and 37th day PI only ($P < 0.01$).

On 30th day PI, statistically significant increase in monocyte count ($P < 0.01$) was noticed in infected groups C and D than control. Among the infected groups, group D showed a significant increase ($P < 0.01$) in the count than groups A and B. On 37th day PI, group C ($P < 0.01$) significantly differed from control group,but the infected groups were homogenous.

Similar findings were reported by Kaushik and Sen (1978) and Padmaja (1992), whereas Rao and Lal (1991) observed a decreased monocyte count in A. galli infected chickens.

The variations in the basophil and monocyte counts observed in this study needs to be analysed further in

detail to elucidate its significance in the pathogenicity of A. galli infection in broiler chicken.

Histopathology

The histopathological changes of various segments of small intestine of the experimental chicken were studied from 2nd to 14th day PI at 2 days interval in all groups and 14th to 60th day PI at 4 days interval in A and B groups. In groups C and D, experiment was carried out till 56th day PI.

In all the infected groups of birds mild to moderate goblet cell hyperplasia of intestinal mucosa was uniformly observed along with mild desquamation of epithelial cells of the villi. A few inflammatory foci characterized with infiltration of mononuclear cells and a few heterophils and eosinophils were also observed. Further, evidence of focal necrosis of intestinal mucosa in isolated areas was also recorded.

Sadun (1950), Kadziolka (1956), Bandhyopadhyay and Jain (1967), Lee (1969), Ikeme (1971), Pavlicek and Dykova (1975), Mishra et al. (1980), Reddy et al. (1984). Mukit et al. (1991), Fathithu et al. (1992), Padmaja (1992) and Verma et al. (1993) also observed similar changes in various experimental studies.

Different dose levels of A. galli infective eggs were tried in different groups of experimental birds in order to assess the probable dose dependant variations in the histopathological lesions. The variations observed were only minor and hence could not be absolutely correlated with the infective doses.

Other than the general changes like goblet cell hyperplasia and desquamation of epithelium, squamous metaplasia of lining epithelial cells in isolated areas of mucosa was evident on 2nd and 8th day PI in groups A and C of experimental chicken. Similar occasional occurrence of metaplastic changes leading to even cornification were reported by Ikeme (1971) in experimental A. galli infection in chicken.

Focal areas of submucosal oedema and separation of submucosa from muscular layer were observed on 8th day PI in group B chicks. Although Verma et al. (1993) reported oedema, the lesion was observed in the muscular layer and separation of muscular layer from serosal layer was recorded on 15 days PI due to necrosis of fatty tissues.

Another consistent histological feature observed in this study was cystic changes of a few mucosal glands along with necrosis and desquamation of glandular epithelium. These changes were observed on 33rd day PI in group B, on

6th, 12th and 33rd day PI in group C and 6th, 8th, 10th and 12th day PI in group D. Although some of the earlier workers (Khouri and Pande, 1970; Pavlicek and Dykova, 1975; Mishra et al., 1980, Reddy et al., 1984 and Mukit et al., 1991) failed to observe similar lesions, Padmaja (1992) reported cystic changes of submucosal glands in experimental ascaridiasis.

Inflammatory changes of the intestine stretching from mild to very severe enteritis were noticed in different periods of observation. Severe enteritis was characteristically observed in 41st day PI in group A, 45th day PI in group B,53rd and 56th day PI in group C and on 41st day PI in group D. Similar inflammatory changes were observed by earlier workers also (Sadun, 1950; Khouri and Pande, 1970; Mishra et al., 1980; Reddy et al., 1984, Fathithu et al., 1992; Padmaja, 1992 and Raote et al., 1992).

In addition to the inflammatory changes, mild fibrosis of mucosa was noticed on 4th and 6th day PI in group C chicken. These findings are in agreement with observations of Sadun (1950) and Verma et al. (1993). Focal areas of interglandular fibroblastic proliferation was also observed in mucosa of heavily infected chicken on 2nd day PI. Similar findings were reported by Mishra et al. (1980) and Verma et al. (1993) also.

In all the infected groups of chicks, lymphocytic aggregations in the mucosa were observed especially towards the end of the experiment i.e. on 41st, 45th, 49th and 52nd day PI. Similar diffuse lymphoid infiltration as well as a few lymphofollicular aggregations had been reported in cases of ascaridiasis of both natural (Deorani, 1968) and experimental (Kaushik, 1980) infection.

Mild to moderate congestion and haemorrhage of mucosa observed on 4th, 6th and 17th day PI in heavily infected chicken lent support to the observations made by earlier workers (Sadun, 1950; Bandhyopadhyay and Jain, 1967; Lee, 1969, Kohuri and Pande, 1970; Mukit et al., 1991 and Padmaja, 1992).

Considerable difference of opinion had been recorded in the literature in the existence of a tissue migratory phase of the different larval stages during infection of A. galli. While research workers like Moran and Mizelle (1956), Varghese (1966), Birova- Volosinovicova (1971), Matta (1980) and Reddy et al. (1984) recorded their failure to detect any stage of larvae in the tissue, a number of other workers like Sadun (1950), Tugwell and Aekert (1952), Tsvetaeva (1954), Deo and Srivastava (1955 a), Lee (1969), Bandhyopadhyay and Jain (1967), Khouri and Pande (1970), Ikeme (1971), Niculescu and Purcherea (1972), Herd and Mc

Naught (1955), Mishra et al. (1980), Soulsby (1982), Padmaja (1992) and Verma et al. (1993) reported the presence of sections of larvae in different layers of intestinal wall after experimental infection.

In the present study, sections of larvae were detected in the villi as early as 4th day PI and persisted upto 17th day PI.

On the duration of the tissue phase also various concepts had been recorded. Tugwell and Ackert (1952) reported that tissue - phase of A. galli might have begun on the 1st day of parasitism and reached its intensity from 9th to 16th day and might be continued through out the 20th day. Tsvetaeva (1954) observed that half an hour after the infection larvae were seen in the mucosa of anterior part of duodenum and were present throughout the intestine 1 to 2 hours of infection. Further, Niculescu and Purcherea (1972) observed the larvae of A. galli penetrating the walls of jejunum and ileum on the 1st day of infection itself. Deo and Srivastava (1955) reported that larvae were in intimate contact with mucosa 10 to 12 days PI. Lee (1969) confirmed that larvae stayed in the villi of small intestine for 1.5 to 2.5 months without any development there, whereas Soulsby (1982) reported that infective larvae were hatched out after natural infection

and found in the intestinal mucosa from 8th to 17th day after infection. The observations of the present study are in agreement with that of Soulsby (1982), that the larvae were noticed in the section up to 17 days PI.

On the 4th day PI, in the birds of group B longitudinal sections of larvae were observed in the villi causing moderate alteration to their shape. Certain sections of larvae were tangential and a few were located in the crypts of Leiberkuhn. In those birds which belonged to group A longitudinal sections of larvae were revealed on 14th day PI in the glandular region of mucosa, most of them being located between the glands without any inflammatory reaction.

Sections of larvae were identified on 10th day PI in the birds belonging to group C. These were either seen at the tip of villi or embeded in the glandular region of mucosa without any inflammatory reaction. Larvae persisted upto 17th day PI and sections were observed at the tip of the villi. In heavily infected group of chicken (group D) sections of larvae were seen in the deep mucosa and some times even inside the lumen of the mucosal glands.

The extent of penetration of the intestine by the larvae had been studied by a number of workers. Sadun

(1950) identified A. galli larvae in the crypts and observed flattened intestinal epithelium around it. Fibrous tissue encapsulation and degenerative changes of the larvae were also recorded. Although such encapsulated larvae could not be detected in the present study, degenerated larvae in the mucosal region were observed in the section of intestine of the birds of group B on 33rd day PI. Padmaja (1992) also observed on 30 and 40 days PI, cut sections of degenerated larvae at the submucosal region and opined that host tissue reaction might have lead to the death of organism. Khouri and Pande (1970) in an experimental study reported that 6 day old juveniles occurred among the destroyed villi with their anterior ends directed towards the base of mucosa and in another section juveniles cut in various planes with their anterior ends abbuting against the muscularis mucosa. Even though extensive destruction of villi and desquamation of epithelium were observed in the present study, sections of larvae could be seen only in the mucosa and not in muscularis mucosa or further deeper tissues. This observation lent support to the findings of Ikeme (1971) where in the larvae were identified mainly in the mucous membrane between or occasionally within the villi. Further, Mishra et al. (1980) opined that cut section of the larvae of A. galli were seen either between two villi or penetrating an individual villus.

Bandhopadhyay and Jain (1967) noticed cross sections of nematode sorrounded by massive haemorrhage and cellular debris. Padmaja (1992) observed cut section of larvae in the intestinal mucosa along with other inflammatory changes. However, in the present study, the sections of larvae did not reveal any inflammatory changes or haemorrhagic lesions around them.

Weekly body weight

The data on weekly body weight presented in the Table 12 revealed that the uninfected birds had the higher body weight than the infected. The mean body weight of the birds were the highest in the uninfected control group i.e. group E and followed by birds in group A, B, C and D in the descending order.

The mean initial body wieght (in gms) were 45.5±0.32, 45.5±0.32, 44.7±0.26,43.5±0.19 and 46.0±0.21 for birds in groups A,B,C,D and E respectively. The mean total body weight (in gms) at the end of experimental trial was 1962.5±89.01, 1835.0±89.01, 1785.0±89.01, 1555.0±72.67 and 2435.71±67.28 for groups A, B, C, D, and E respectively. From the Table 12 and Fig. 1 it is logical that uninfected control group birds had the highest total body weight whereas the birds which received highest infective dose had the lowest body weight i.e. group D and followed by C, B and

A in ascending order. From this it is evident that the reduction in the body weight is directly proportional to the dose of infection. The data presented in the Table 12 revealed that there was a statistically significant difference in body weight ($P < 0.01$) in each week in infected groups when compared to the uninfected control group.

The effect of A. galli on mean body weight in chicken had been reported by many workers. Kassai (1962) reported a loss of weight and hypovitaminosis A in naturally infected birds. Matta and Ahluwalia (1980) confirmed that there was a significant difference in average weight between infected and control group of birds and this was directly proportional to the increasing dose of infection. A highly significant decrease in mean body weight in A. galli infected birds was reported by Toledo and Castell (1981). Padmaja (1992) in an experimental study with chicks infected with 3500 embryonated ova of A. galli found that the mean body weight of infected group was 225.2 gms and that for the control group was 358.5 gms. The observations made by these workers were supportive of the present findings. It is thus concluded that A. galli infection adversely influence the body weight of chicken and it is direclty proportional to the intensity of infection.

Body weight gain

On perusal of data on weekly body weight gain presented in the Table 13 and Fig. 15 it is apparent that there was a statistically significant difference in body weight gain ($P<0.01$) between groups in each week. The average gain in body weight from 0 to 8 weeks of age for group E was 2390 gms, where as for groups A, B, C and D it was 1917, 1789, 1740 and 1511 gms respectively in descending order. A ststistically ($P<0.01$) detrimental effect on body weight gain between control and infected groups was evident not only on overall body weight gain for 0 to 8 weeks, but also in respect of gains made during each weekly interval.

The observation in the present study revealed lower body weight gain in the infected birds as compared to control chicks, and this supports the observations of earlier workers.

A loss of weight in the Ascaridia infected chicks was observed by Tsvetaeva (1954). Kadziolka (1956) reported that the weight gain of the infected chicks was less than control chicks. A marked decrease in the body weight gain was recorded by Costa (1970). Katara and Rai (1980) reported that the gain in body weight of infected birds was 53, 47,

43 and 47 per cent after 10th, 20th, 30th and 40th days of infection respectively. A subsequent reduction in body weight gain had been reported by Raote et al. (1994).

The observation that heavier the dose of infection, lesser the gain in body weight of growing chicks is confirmed by earlier workers. Todd et al. (1949) reported that there was a reduction in gain in body weight when the number of infective eggs increased and weight loss was observed during the period of migration of immature stages. Sadun (1950) recorded a lesser gain in body weight in chicks infected with 14000 embryonated eggs of A. galli (160.5g) as against (230.1g) in chicks received 500 embryonated egggsof A. galli, and 241g in control bird on 32nd day PI. Todd and Hansen (1951) revealed that there was a retardation in gain in body weight in birds when the infective dose of A. galli was increased.Similar findings were observed by Reid and Carmon (1958) and they also stated that there was a decrease in gain in body weight in birds three weeks PI. Dimitrov (1971) who infected with different doses of A. galli infective eggs in broiler chicks, recorded lesser body weight gain (6-7%). Ikeme (1971) reported a lesser weight gain and significant depression in growth rate in growing birds when repeatedly infected with varying doses of A. galli infective eggs. Even at an

infective dose of 150 A. galli eggs, a lesser gain in body weight was noticed by Patil et al. (1972). Corroborated results on lesser weight gain in white leghorn layers was reported by Sazikova (1975). Highly significant decrease in mean body weight was observed in A. galli infected birds by Toledo and Castell (1981). Ramadan and Znada (1991) also reported a variable decrease in body weight which was directly proportional to the doses given to broiler chicken.

In the present experimental trial, though the birds were fed with good quality broiler mash, irrespective of dose of infection, lesser gain in body weight was noticed in infected group of birds when compared to control group. The difference in gain in bodyweight was statistically significant.

Feed intake

On perusal of data on weekly feed intake presented in the Table 14 and Fig. 16 it is evident that irrespective of dose of infection the weekly feed intake was increasing from the 1st week PI till the end of the experimental trial in all the infected groups. The mean feed intake for the different groups were 5502, 4932, 4749, 4582 and 4029 gms for groups E, A, B, C and D in descending order.

The results of statistical analysis of cumulative feed intake data in Table 15 revealed, that there was no statistically significant difference in feed intake between groups.

The literature available on the effect of different doses of A. galli in infected chicken on weekly feed intake appears to be scanty. In the present trial there was no significant difference in feed intake between groups though the intake was gradually decreased numerically with the increasing dose of infection.

Costa (1970) in an experimental study reported a lesser feed intake. Dimitrov (1971) reported an increased feed intake (4-6%) and decreased body weight gain in broilers infected with different doses of A. galli eggs.

Feed conversion ratio

The data presented on mean feed conversion ratio in Table 16 and Fig. 17 showed that the feed conversion ratio of the birds in infected and control groups A,B,C,D and E were 2.62 ± 0.46, 2.60 ± 0.33, 2.64 ± 0.34, 2.79 ± 0.27 and 2.35 ± 0.34 respectively. The feed conversion ratio was best for the group E, followed by groups B, A, C and D in descending order.

On persual of statistical analysis set out in Table 17 showed that feed conversion ratio did not significantly differ between groups eventhough the feed conversion ratio numerically appeared better for the control group than infected groups. The literature on feed efficiency in A. galli infected chicken appears meagre. Costa (1970) reported a lesser feed efficiency in A. galli infected chicken. The observations made in the present trial is in agreement with this.

Summary

SUMMARY

An investigation was carried out with an objective to assess the pathogenicity of Ascaridia galli infection in broiler chicken. Two hundred numbers of day - old broiler chicks constituted the experimental flock. The chicks were divided randomly into 5 groups (A,B,C,D,and E), each consisting of 40 chicks. Groups A,B,C and D were experimentally infected with 500,1000,1500and 2000 embryonated eggs of A. galli and retaining E as the control group.

The study involved general clinical signs, gross lesions, morbidity and mortality rate, blood parameters at weekly intervals and histopathology of small intestine at specific intervals. In addition, a correlation was also made on weekly body weight, weekly body weight gain and weekly feed intake and the data obtained was used to work out the weekly feed conversion ratio.

From the results the following conclusions were drawn:

1. The intensity of various clinical signs observed in all the infected groups of chicks were directly proportional to the dose of experimetnal infection. In general in all the infected groups, the chicks were

dull, drooping, passing loose motion with loss of appetite and showed increased water intake. The comb and wattles were pale, feathers ruffled and their devlopment delayed. There was no sign of emaciation, unthriftiness and leg weakness in group A chicks, but such symptoms were invariably observed in chicks of groups C and D.

2. Gross lesions consisted of varying degrees of congestion, frothy and mucus-mixed intestinal contents, some times coated with blood. Thickening of the wall and narrowing of the lumen of intestine leading to obstruction and severe enteritis were seen associated with large numbers of worms.

   During post mortem examination, large numbers of larvae were recovered from 2nd day PI. From 14th day PI, majority of young worms could be seen free in the lumen of small intestine. Subsequently from 33rd day PI, the adult worms were found in the lumen.

There was no mortality in both infected and control groups of birds, but the morbidity rate was high in all the infected groups.

3. Haematological studies revealed consistent increase in erythrocyte sedimentation rate in infected groups

from 9th day PI till the end of the experiment, but the increase was statistically significant only on 37th day PI ($P < 0.05$).

Packed cell volume was decreased in all the infected groups than the control from 9th day PI. But the decrease was statistically significant at 1 per cent level ($P < 0.01$) on 37th day PI and 5 per cent ($P < 0.05$) level on 44th day PI.

There was reduction in haemoglobin concentration in all the infected groups than control. It was statistically significant at 1 per cent level on 16th , 23rd and 30th day PI ($P < 0.01$) and at 5 per cent level on 37th day PI ($P < 0.05$).

Total erythrocyte count was decreased in all the infected groups than control ones. But the decrease was statistically significant only on 16th and 23rd day PI at 5 per cent ($P < 0.05$) and 1 per cent ($P < 0.01$) level respectively.

Total leucocyte count was significantly increased ($P < 0.01$) in all the infected groups of birds from 16th day PI onwards upto 44th day PI. But there was no significant difference noticed on 9th and 51st day PI.

From 9th day PI, statistically significant increase in heterophil count ($P < 0.01$) was noticed in all the infected groups of birds.But the difference was only at 5 per cent ($P < 0.05$) level on 51st day PI.

The eosinophil count was significantly increased ($P < 0.01$) from 16th day PI till the end of the trial. But the increase was only at 5 per cent level ($P < 0.05$) on 9th day PI.

A significant decrease ($P< 0.01$) in the lymphocyte count was observed in all the infected groups throughout the experimental period.

4. Histopathological observations revealed mild to moderate goblet cell hyperplasia uniformly distributed in all the segments of intestine, moderate to severe disruption of villi, desquamation of epithelium, focal necrosis of mucosa and infiltration of mononuclear cells in the lamina propria of few villi and crypts. Focal inflammatory changes in serosa and severe enteritis were also recorded.

Other lesions were submucosal oedema, separation of submucosa from muscular layer, cystic changes in mucosal

gland with necrosis, squamous metaplasia of lining epithelial cells, congestion and mild fibrosis of mucosa. Degenerated larvae in the mucosa were observed in a few cases.

The tissue-phase of larval migration was observed from 4th to 17th day PI. This was evidenced by the presence of sections of larvae at various locations of intestinal mucosa like the villi, crypts of Leiberkuhn and mucosal glands. Peri larval inflammatory reaction was not detected in any of the sections. On 33rd day PI, degenerated larvae were observed in the submucosal region.

5. The mean body weight of birds was highest for control group (2435.71±67.28 g) followed by groups A, B, C and D i.e. 1962.5±89.01, 1835.0±89.01, 1785.0±89.01 and 1555.0±72.67 g respectively.

6. A continuous increase in gain in body weight was noticed from 1st week PI up to 6th week PI. But the increase in weight gain was lesser than the control group. There was statistically significant decrease ($P < 0.01$) in the weight gain in infected groups in each week PI. The mean body weight gain for groups A, B, C, D and E was 1917, 1789, 1740, 1511 and 2390 gms respectively.

7. The mean feed intake was highest for the group E followed by groups A, B, C and D. i.e. 5502, 4932, 4749, 4582 and 4029 gms respectively. The mean feed conversion ratio was highest for the group E followed by groups B, A, C and D i.e. 2.35±0.34, 2.60±0.33, 2.62±0.46, 2.64±0.34 and 2.79±0.27 respectively. Though a lesser feed intake and feed conversion ratio was noticed in all the infected groups the difference was not statistically significant.

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PATHOGENICITY OF *ASCARIDIA GALLI* INFECTION IN BROILER CHICKEN WITH SPECIAL REFERENCE TO WEIGHT GAIN

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ABSTRACT OF A THESIS

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ABSTRACT

A study carried out to assess the pathogenic effect of the developmental stages of Ascaridia galli in broiler chicks for a period of eight weeks, revealed clinical signs like general weakness, unthriftiness, ruffled feathers and loss of appetite, besides the delayed development of comb, wattles and feathers. On post-mortem examination of the experimentally infected chicks, large numbers of larvae were recovered from 2nd day PI onwards and the lumen larvae were detected from 14th day PI. The intestinal contents were frothy, mucus mixed and had many immature worms. In the small intestine the worms embeded in the mucosa with their anterior extremities.

Histopathological studies revealed disruption of villi, desquamation of epithelium, infiltration with mononuclear cells, focal necrosis, congestion and haemorrhage in the mucosa and cystic changes of mucosal glands.

Sections of larvae in the mucosa at different histological locations were detected on 4th , 10th, 14th and 17th days PI. In addition, cut sections of degenerated larvae were also observed.

Haematological studies indicated a statistically significant increase in erythrocyte sedimentation rate, total leucocyte count, total heterophil and eosinophil counts. Packed cell volume, haemoglobin content and total erythrocyte count were significantly decreased. Lymphocyte count was significantly decreased ($P < 0.01$) in all the infected groups upto 37th day PI; later the count was gradually increased in all the infected groups.

The mean body weight and mean body weight gain in the infected group of chicks were significantly lesser than the control group chicks ($P < 0.01$). The mean weekly feed intake and mean weekly feed conversion ratio were lesser in the infected groups of chicks than control ones, although the difference was not statistically significant.

The morbidity rate was high in all the infected chicks, but mortality was not recorded during the experimental period. Coprological study confirmed the maturation period for A. galli in groups A, B, C and D as 59, 59, 50 and 44 days respectively.