# EFFICACY OF CERTAIN INDIGENOUS DRUGS AGAINST ASCARIDIASIS AND CAECAL COCCIDIOSIS IN POULTRY



BY

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## CERTIFICATE

Dr.B.SREELAKSHMI has satisfactorily prosecuted the course of research and that the thesis entitled "EFFICACY OF CERTAIN INDIGENOUS DRUGS AGAINST ASCARIDIASIS AND CAECAL COCCIDIOSIS IN POULTRY" submitted is the result of original research work and is of sufficiently high standard to warrant its presentation to the examination. I also certify that the thesis or part there of has not been previously submitted by her for a degree of any University.

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## CERTIFICATE

This is to certify that the thesis entitled "EFFICACY OF CERTAIN INDIGENOUS DRUGS AGAINST ASCARIDIASIS AND CAECAL COCCIDIOSIS IN POULTRY" submitted in partial fulfilment of the requirements for the degree of MASTER OF VETERINARY SCIENCE of the Andhra Pradesh Agricultural University, Hyderabad is a record of the bonafide research work carried out by Dr.B.SREELAKSHMI under my guidance and supervision. The subject of the thesis has been approved by the Student's Advisory Committee.

No part of the thesis has been submitted for any other degree or diploma. The published part has been fully acknowledged. All assistance and help received during the course of the investigation have been duly acknowledged by the author of the thesis.

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# ABSTRACT

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## ABSTRACT

Present work was undertaken to study the efficacy of certain indigenous drugs-(Wopell, Anthelmex and Krimos) against ascaridiasis and zycox (IHP-250C) against caecal coccidiosis in the experimentally infected chicks.

To test the efficacy of three indigenous anthelmintics against ascaridiasis a total of 90 whiteleghorn chicks of one month old were randomly divided into 2 batches of 45 each. (To test the efficacy of these drugs against larval forms and adult worms of A.galli). Each batch was further divided into 5 groups.

The birds in the groups I, II, III and IV were orally inoculated with 1000 embryonated eggs of A.galli per bird and the I, II and III groups were treated with Wopell, Anthelmex and Krimos respectively. IV group kept as infected and unmedicated control. Birds in the group V were uninfected and unmedicated.

The parameters considered to evaluate the efficacy of these drugs were the average recovery of larval forms (by flushing and digestion) and adult worms of A.galli in the groups I, II and III treated with Wopell, Anthelmex and Krimos respectively.

The average recovery of larval forms were 15.33, 23.53 and 32.33 in the groups I, II and III where as the birds in these groups were treated with Wopell, Anthelmex and Krimos respectively. The efficacy of Wopell, Anthelmex and Krimos against larval forms of A.galli was 95.5, 93.1 and 90.52% respectively.

The average recovery of adult worms were 0.33, 0.83 and 1.8 in the groups I, II and III where as the birds in these groups were treated with Wopell, Anthelmex and Krimos respectively. Efficacy of Wopell, Anthelmex and Krimos against adult worms of A.galli was 98.35, 95.85 and 91%.

The parameters considered to evaluate the efficacy of zycox against E.tenella were faecal score, lesion score,

the per cent mortality during experimental period and 0-12 day P.I body weight gain were calculated. All the other selected parameters were studied on samples collected at different days P.I.

The average faecal score was 4.0 in group IV was higher than the faecal scores of 2.12, 1.25, and 1.12 in the groups I, II and III. Where as birds in these three groups treated with 4,5 and 6 ml of zycox per 10 litres of water respectively.

The average lesions score was 4 in group IV where as the average lesion scores 1.87, 1.25 and 1.12 were in groups I, II and III. Birds in these three groups were treated with 4 ml, 5 ml and 6 ml of zycox per 10 litres of water respectively.

The average oocyst counts per bird of I, II and III groups were 8.2 millions, 5.5 millions, 5.3 millions respectively and were lower than the count in group IV (36 ) which is infected and non medicated control.

Higher percentage of mortality was recorded in group IV (60%) compared to I (5%), II (1.5%) and III (1.5%) groups.

The body weight gain from 0-12 days P.I in group IV (225 g) was significantly lower than that in the groups I (310 g), II (316 g)and III (322 g). Maximum body weight gain was noticed in the group III treated with 6 ml of zycox/10 litres of water.

The histopathological changes consisting of desquamation of epithelial cells, cellular infiltration, haemorrhages, oedema and developing stages of E.tenella in the epithelial cells were of higher intensity in the group IV compared to the groups of I, II and III. Where as these groups were treated with 4 ml, 5 ml and 6 ml of zycox per 10 litres of water.

It is indicated that zycox at 6 ml/10 litres of water was efficient against caecal coccidiosis caused by E.tenella.

## LIST OF ABBEREVIATIONS

g = gram

P.I = postinfection

Pm = postmedication

OPG = Oocyst per gram faeces

# INTRODUCTION

# CHAPTER I

## 1. INTRODUCTION

Large scale commercial poultry farming has come into existence during the last three decades. Despite managemental and related problems in India, a huge loss of birds due to diseases is being faced by the poultry farmer. It is also realised that the sum total of ravages by various parasitic infections in poultry are in no way be considered less significant than other etiological agents.

Among the gastrointestinal nematode infections, ascaridiasis is the age old helminthic menace of poultry. Ascaridiasis due to Ascaridia galli is often setting back the progress of the poultry industry, incurring heavy economic loss to the nation to a tune of 180 million rupees per anum (Matta and Ahuluwalia, 1980). About 60% of domestic fowls in Andhra Pradesh were found infected with A.galli (Muralidharam and Venkataratnam, 1979-80) which affects the health of the poultry without showing any clinical symptoms. The parasites present in the host in such large numbers as to cause complete stoppage of the bowel movements (Sprehn, 1930). The presence of even a few worms may cause weakness and at times prove fatal (Mocsy, 1931).

The pathogenicity of the disease is due to the larval forms that migrate to the duodenal mucosa and adult worms usually inhabitating the lumen of small intestine. It is the larval form which causes extensive damage and affect the growth rate of birds.

Coccidiosis in poultry industry causes substantial losses which are mainly due toHigh rate of morbidity and mortality, poor weight gain and feed conversion, and decreased egg production. According to the Avian sub committee of the American Association of Veterinary Parasitologists Research Committee (1986), avian coccidiosis is one of the most important and devastating protozoan diseases affecting poultry under almost every possible climatic conditions all over the world, even under strict hygienic conditions and with the continuous usage of coccidiostats in the feed. In the face of outbreaks of coccidiosis, E.tenella is the most dangerous of the all species.

Eventhough, a number of anthelmintics and coccidiostats had been tried against ascaridiasis and coccidiosis respectively in poultry, these diseases are still prevalent throughout the globe and are not under complete control. Currently available anthelmintics are not /much effective/ against the larval forms though they may,

however, be effective against adult worms of A.galli. Prolonged use of coccidiostats have resulted in the emergence of drug-resistant strains of coccidia (Gill and Bajwa, 1979). Due to the development of resistance against a particular drug the farmers are not in a position to check the control of these diseases. Now a days a number of pharmaceutical companies are coming up with indigenous drugs against the parasitic infections.

Hence, a preliminary study has been undertaken to study the efficacy of certain indigenous drugs against ascaridiasis and coccidiosis in poultry. With the objective (a) to assess the efficacy of (1) Wopell (Kamala Powder 2 grams, Pulv.Babarang 2 grams, Pulv. Palas Papra 2 grams, Pulv. arecanuts 2 grams Pulv. Male fern 2 grams for every 10 grams of powder) from M/S Indian Herbs, (2) Anthelmex (Butae Frondosa 0.5 grams, Embelia Ribes 0.5 grams, Aristolochia Bracteata 1.5 grams, Vernonia Anthelmintica 1.0 grams, Ipomea Turpethum 0.25 grams, Pimpinella Anisum 1.0 grams, Nigella Sativa 0.75 grams, Carum Roxburghianum 1.0 grams, Cassia Aucelolata 0.75 grams, Panchalavanam 2.0 grams, Helleborus Niger 0.75 grams, Manapagu Q.S) from M/S Agasthiar Pharmaceuticals, and (3) Krimos (Dry opteris filix MAS 10 grams, Areca Catechu 10 grams,

Embella Rises 10 grams, Punica Granatum 10 grams, Mallotus Phill Ippimensis 5 grams, Nala 5 grams, Cassia Angusti Folia 15 grams, Chenopodium Album 10 grams, Artemisia Mar Itima 1 grams, Vernonia Anthelmintica 5 grams, Butea Monoperma 10 grams, Azadiracnta Indica 5 grams, Titia 1 gram, Myoscyamus Niger 3 grams) from Bhartiya Bootée Bhavan against ascaridiasis in poultry.

and (b) to study the efficacy of Zycox (IHP 250 C) from M/S Indian Herbs, against caecal coccidiosis in poultry.

# REVIEW OF LITERATURE

# CHAPTER II

## 2. REVIEW OF LITERATURE

### 2.1 ASCARIDIASIS

Much work has been done in the direction of ascaridiasis in fowl, viz, Morphology, biology of adult worms of Ascaridia galli and their larval forms, experimental infection, development of infections under varying conditions of feed, age, environment, etc., pathology, host responses and treatment. But less attention has been paid in the lines of efficacy of indigenous drugs against ascaridiasis.

Ackert (1923) found that larvae of the fowl nematode Ascaridia galli burry their anterior ends deeply between the intestinal villi and into the brunner's glands, but they seldom pass through the wall of intestine or migrate over the body of the host. Similar findings were reported by Roberts (1937) for these phases of the life cycle of A.galli.

Guberlet (1924) stated that heavily infected chicks with A.galli died on 10th or 12th day post-infection due to intestinal irritation and toxemia.

Ackert (1931) confirmed that the tissue phase of A.galli was occurring between 10th and 17th day after

infection and also stated that the young worms of A.galli matured in about 50 days in one month old chicken.

In the chicken infected with A.galli young worms were found within the mucous membrane of the intestine from 3rd to 24th day after infection (Ackert and Tugwell, 1948).

Sadun (1949) had indicated that extremely heavy single infections i.e., 203 embryonated eggs of A.galli per gram body weight caused the death of young chicken between 6 and 19 days after infection. And large number of eggs per gram body weight were required to kill older chicken.

In the chicken infected with ascaridiasis the tissue phase of A.galli might begin on the first day of parasitism by a few larvae and continue atleast to 26th day, but the majority of young larvae occurred in intestinal mucosa from 8th to 17th day after infection (Tugwell and Ackert 1950).

Feoktistov (1950) observed the maturation period of A.galli as 35 days in 16 to 18 day old chicken and 58 days in adults.

In 2 week old infected birds with A.galli, the migration of larvae into the intestinal mucosa was in

frequent and most of larvae matured without leaving the lumen of the intestine (Todd and Crowdus 1952).

Tugwell and Ackert (1952) observed that the tissue phase of A.galli might begin on the first day of infection by a few larvae and continued atleast to 26th day, but the majority of young larvae occurred in intestinal mucosa from 8th to 17th day after infection.

Deo and Srivastava (1955) observed that the larvae were in intimate contact with mucosa, 10-12 days post-infection and that the majority of the larvae were embedded in mucosa on 10th day and from 13th day onwards, almost all larvae were found free in lumen and also observed that maturation period of A.galli was 28-34 days in 4-8 weeks old birds.

Kerr (1955) observed the maturation period of A.galli was 30-32 days in chicken below 3 months and 50 days in older birds.

Lapage (1956) observed that the third stage larval forms of A.galli bury their heads in the crypts of mucosa after fifty day post-infection in chicken and also found that the young worms became adults in about 5 to 8 weeks or sometimes extend upto 14 weeks, depending on the vigour and resistance of the host.

Moran and Mizelle (1956 and 1957) found that the larvae rarely penetrated the intestinal mucosa and 99.6% of infected larvae remained in the layer of mucous adherent to the intestinal wall. The immature larvae might continue to remain a static condition in the layer of intestinal mucosa for considerable periods, the larvae failed to attain maturity even 50 days post-infection.

Edgar et al. (1957) used piperazine hexahydrate and reported 95 to 100% efficacy against adult A.galli and 75 to 100% efficacy against immature worms.

Gupta and Rao (1959) reported piperazine citrate to be effective against mature and immature worms of A.galli, 98.8% and 92.8% respectively in fowls under field conditions.

Srivastava and Malviya (1959) found piperazine citrate to be cent percent effective when given individually or in drinking water and 84.9% effective when given in mash in eliminating A.galli.

Madsen (1962) summarized the results of previous workers on the tissue phase of A.galli in chicken and concluded that in strick sense, the tissue phase could not be considered as a part of normal life cycle but rather as a reaction to factors of resistance.

Panda (1965) observed that the second stage larvae emerged from the egg were in the intestinal lumen for 9 to 10 days before invading mucosa. In about 19 to 20 days, immature adults detatched from mucosa to live in lumen and grow to became adults. Similar observations were reported by Khouri and Pande (1970).

Sharma and Sisodia (1971) tried piperazine adepate and found highly effective against adult A.galli but less effective against immature forms.

Sundar Rao (1972) reported that Wopell and Piperex were 99.67 per cent efficacious against adult forms and 95.7 and 95.3 per cent efficacious against immature forms of A.galli respectively.

Katara (1972) tried three anthelmintics piperex, Safersol and Antepar on third, fourth and fifth stage juvenils. The three drugs were ineffective against third and fourth stage larvae but cent per cent effective against fifth stage juveniles and mature worms.

Wopell given in feed at 100 mgm per chick was 70,58 and 71.06% effective against immature and mature forms of H.gallinae (Venkateswarulu ,1973).

Ovies and Tokanev (1976) compared the relative efficacy of Tetramisole and Piperazine adipate against

Narahari (1980) carried out a clinical trial to study the relative anthelmintic efficacy of piperazine hexahydrate, tetramisole hydrochloride and Wopell against natural infection with A.galli in growing pullets. It has been observed that all the three drugs were 100% effective against adult worms of A.galli and against immature forms piperazine, tetramisole and Wopell were 70.8, 94.4, and 76.4 per cent effective respectively.

Hafeez et al. (1992) studied the efficacy of anthelmex against certain gastrointestinal nematodes but in sheep and reported that the drug seems to have good eifect on trichostrongylosis in sheep.

Hafeez et al. (1993) tried the efficacy of Anthelmex against certain gsstro-intestinal round worms but in cattle and indicate that anthel-mex at a dose rate of 50 ml per calf has got good effect on Trichostrongylus sp. in cattle.

## 2.2 COCCIDIOSIS

Coccidiosis a ubiquitous disease of poultry, is caused by a protozoan parasite of Eimeria species. Of all the species, E. tenella which infects mainly the caeca is considered to be highly pathogenic. Coccidiosis in chickens continues to be a problem despite efforts to control the disease by chemical means and improved poultry house management.

Oocyst counts per gram of faeces were determined by Horton Smith and Long (1952) in E. tenella infected chickens (75,000 sporulated oocysts at the age of 2 weeks). They counted the number of oocysts per gramof faeces passed on 7th, 8th, 9th, 10th and 11th days of post-intection period as 3,24,000, 2,62,000, 1,41,000, 66,000 and 3,100 respectively.

The caecal lesions were extensive from 4th to 6th day of post-infection where edema of submucosa and muscular layer of the caecum with schizonts in the epithelial cells of the villi and cellular infiltration of more of neutrophils in lamina propria, denudation and desquamation of epithelial lining of the villi, rupture of the epithelial cells due to developing schizonts with patchy haemorrhages, open villar tips, hyperplasia of

mucosal glands were the important histopathological findings (Gill and Ray, 1957; Jagadesh et al. 1976; Padmavathi, 1984).

An experiment was carried out to develop a glycarbylamide resistant strain. A laboratory strain of E.tenella was rendered completely resistant to glycarbylamide after 9 passages in chicken receiving small doses of the drug. It was also resistant to nitrofurazone, trithiadol, zoalens, arsenosobenzene or unistat (Mc Loughlin and Gardiner, 1961).

Mukkur and Bradley (1969) observed 23 per cent mortality among birds given 50,000 sporulated oocysts of E.tenella, at the age of 4 weeks. Similar observations were reported by Kennet et al. (1974).

Kennet et al. (1974) infected the chicks with 50,000 sporulated oocysts of E.tenella and reported an oocyst count of 16 x $10^6$ per bird, and noticed an average lesion score of 2 in the infected birds. They also compared infected broiler chickens with healthy control and noticed significant depression in 0-14 day P.I. body weight gain (58g) compared to the healthy control (305g).

Giambrone et al. (1977) infected 7 day old broiler chickens with 1,50,000 sporulated oocysts of E.tenella

and reported 39% mortality among the infected birds and recorded faecal scores of 4, 4 and 4 on 5th, 6th and 7th days of post-infection respectively. They also noticed reduction of body gains of 11.9g and 128.8g on 7th and 14th day of P.I. respectively when compared to the body weight gains of 100.5g and 258.3g in uninoculated controls on respective days of post-infection.

Jasmer Singh and Hussain (1978) infected 2 week-old chickens with 50,000 sporulated oocysts of E.tenella and reported 100% mortality in the chickens and observed the maximum lesion score of 4 on5th day of post-infection in the infected birds.

Gill and Bajwa (1979) studied the drug resistance in field isolates of chicken coccidia from Punjab state. Of 158 Eimeria isolates from chicks, 113(71.5%) were resistant to sulfaquinoxaline, 93(59%) to nitrofurazone+ furazolidone, 54(34%) to amprolium, 7 to clopidol and 9 to nicarbazin, 23 were sensitive to all these drugs, none was resistant to all, 34% were resistant to two drugs,19% to three and 6% to four.

Morrison et al. (1979) observed a depression in 14-21 day P.I. body weight gain (70.1%) of infected chicken (80,000 sporulated oocysts of E.tenella) when compared to healthy control (90.4%).

Clark (1979) noticed haemorrhagic faecal droppings in chicken infected with E.tenella from 90-156 hours of post-infection and bloody faecal droppings continued to appear for a further 12-48 hours and hard core was produced 204 to 216 hours after infection.

Long et al. (1980) infected the 25th day old chickens with 50,000 sporulated oocysts of E.tenella and noticed significant reduction in body weight gain (147.9g) from 0-6 day P.I. compared to uninfected (control) chickens (254.3g).

Sasmal and Sinha (1983) reported 40.2 per cent weight gain in the E.tenella infected birds ($5 \times 10^4$ sporulated oocysts) compared to 100 per cent weight gain in uninfected birds and also recorded 70% mortality in the infected chicks.

Padmavathi (1984) infected the layer chicks with E.tenella and noticed a reduction in the body weight gains from 4th to 30th day of post-infection (88.6-157.8g) compared to the control (108.5-182.1g) and also observed maximum lesion score (4) from 4th to 6th day of post-infection, which gradually declined by 8th day of post-infection.

Venkataratnam et al. (1985) reported that caecal coccidiosis could be produced experimentally in chicks by injecting sporulated oocysts through cloaca. The pathogenesis caused was almost similar to that by oral infection.

Thyagarajan et al. (1989) infected the 28 days-old commercial broiler chicks with 50,000 sporulated oocysts of E.tenella and reported 66 per cent mortality in unmedicated infected birds.

Roy et al. (1989) infected broiler chicks with E.tenella at the age of 14 days and recorded faecal score at days 4 to 8 P.I. He reported that average faecal score was highest (4.00) in infected unmedicated group and noticed maximum lesion score (4) on 6 day P.I in infected and unmedicated group birds. He studied the efficacy of IHP-250C as coccidiostat in comparison with Bifuran against E.tenella and mixed oocysts. The results were assessed by comparison of mortality, faecal oocyst count, lesion score and weight gain. In all respects the herbal product gave better results, when added at a 0.3% concentration to feed, than a 0.2% concentation or a 0.125% concentration of Bifuran.

Guha et al. (1991) studied the prophylactic efficacy of zycox (IHP-250C) against coccidiosis in broiler chicks. IHP-250C was given in feed @ 0.2%, 0.3% and 0.4% respectively to 3 groups of broiler chicks. Among three doses, 0.3% and 0.4% drug in feed showed maximum performance index where as the dose 0.2% showed minimum performance index.

# MATERIALS AND METHODS

# CHAPTER III

# 3. MATERIALS AND METHODS

## 3.1 CHICKS AND OTHER FACILITIES

Day old white leghorn male chicks were obtained from M/S Balaji Hatcheries, Chittoor, Andhra Pradesh and they were wing banded. Out of one hundred and fifty four chicks ninety chicks were reared upto 30 days in the small animal experimental house attached to the department of Parasitology, College of Veterinary Science, Tirupati. Ninety chicks were randomly distributed into two batches of 5 groups for each as shown in tables No. 1 to 5 and 7 to 11. Each group was accommodated and maintained in individual improvised wire mesh pens in the experimental house. All the chicks were raised under similar feeding and managemental conditions.

### 3.1.2 Health condition of chicks

The chicks during the period of rearing were critically examined for any abnormality in health. They were protected against Raniket disease at the age of first week. All the chicks were apparently healthy. By faecal examination they were found negative for any parasitic disease.

### 3.1.3 Housing

Experimental house, its walls, flooring and all equipment were thoroughly scrubbed with washing soda, rinsed with 10% soln. of ammonia and washed with steeming hot water before housing the chicks. Paddy husk was used as deep litter. Routine cleaning was done every day.

### 3.1.4 *Ascaridia galli* egg cultures

#### 3.1.4.1 Collection of worms

*Ascaridia galli* worms were collected from the small intestines of naturally infected fowls which were slaughtered for sale by poultry marketing sub-centre, Tirupati and various local chicken stalls. They were put into specimen bottles, containing normal saline and carried to the laboratory. They were washed several times with distilled water to free them from debris.

#### 3.1.4.2 Collection and culturing of eggs

The collection and culturing of eggs of *Ascaridia galli* was same as described by Chatterjee and Singh (1968). The gravid female worms were placed on clean glass slides individually in a few drops of water and

their anterior end was excised. Uteri were separated and dissected for isolation of eggs. The tissue debris mixed with eggs was removed by treating with 1 per cent sodium hydroxide for three minutes and then centrifuged at 100 rpm. This was followed by three washings with distilled water to remove the chemical. Then the eggs were cultured in the medium consisting of 100 ml distilled water to which 10 drops of 2% formalin were added. A number of cultures in petridishes were made at the same time and kept in incubator at 32°C. The cultures were examined twice daily to observe the embryonation. Larval movement inside the egg was taken as the criteria for full embryonation. The embryonation was completed by about 7th day. Such embryonated eggs were stored at 4°C until further use.

3.1.5 Experimental infections

Egg cultures of the seventh day were pooled for infecting the experimental chicks of one month old to keep the infectivity at a constantly high level. Embryonated egg counts were made on the same day and the fully embryonated eggs were calculated in 1 ml of the culture. Then required quantity of distilled water was added to contain 1000 infective eggs in 1 ml of suspension.

Before giving the infection, the control group V of batch I and II birds were separated from the birds which were to be infected, kept separately in individual improvised wire mesh pens in the experimental house. A uniform dosage of 1000 infective eggs of A.galli in 1 ml of distilled water was given orally by means of graduated pipette to individual chicks of groups I to IV of batches I and II.

3.1.6 Postmortem examination

The birds were killed at different intervals of post-infection as tabulated under results (Table No.1 to 5). A.galli larvae were collected from both lumen and mucosa as described in technical Bulletin No.18(1971) of Manual of Veterinary Parasitological Laboratory Techniques.

Flushing:

The intestine from the gizzard to the yolksac diverticulum was removed, stripped of mesentrics and divided into lengths of approximately 30 cm. The intestinal contents were squeezed out gently with fingers and collected in a separate flask containing distilled water. Then the intestine was flushed with warm distilled water quickly and free worms were flushed into jars. The worms

were allowed to remain in the intestinal debris for approximately 12 hours to straighten out and die. They were then counted and preserved in vials containing 10 per cent formalin.

Digestion:

The freshly flushed intestines were opened and cut into small pieces. They were digested in jars containing 250 ml of digestion fluid to free larvae from the intestinal mucosa (mucosal larvae) the digestion fluid was prepared by adding 8g pepsin, 20 ml concentrated hydrochloric acid and 23 ml saturated salt solution to 940 ml water. The resulting litre of fluid was sufficient to digest about 500 g tissue. The contents of jar were agitated at one hour intervals for 12 hours and kept at room temperature. At the end of 12 hours, the supernatent fluid was drawn out and then the jar was filled with tap water and allowed to stand. Once again the supernatant fluid was removed. Small volumes of the residue was transferred to petridishes and examination with steriomicroscope facilitated the isolation of larvae from mucosa which were counted and preserved in 10 per cent formalin.

### 3.1.7 Treatment trial

For treatment trials the chicks were allotted at random into 2 batches (five groups for each batch). The first three groups of each batch were infected and treated. The fourth group of I and II batches were infected and untreated and so kept as infected controls and the fifth groups were kept as uninfected controls. These were kept in separate pens in the experimental house, managed and fed on the same lines as per the previous experiments. Before initiation of fresh experiment, the experimental house, its walls and flooring and other equipment were thoroughly disinfected on the lines given previously.

The drugs Wopell (M/S Indian Herbs), Anthelmex (Agasthiar Pharmaceuticals) and Krimos (Bhartiya Bootee Bawan) were used at prescribed dose rates as shown in tables.

The birds were sacrificed at different intervals of post infection as tabulated (Table No. 1 to 5) for collection of immature forms from the lumen of intestine if any, as described before, to note the efficacy of the drug on the immature fo rms of A.galli.

So also the above drugs were used on the same experimental basis, as given above, to note the efficacy of the drugs on the adult worms of A.galli. Pretreatment EPG were counted and recovery of adult worms if any on postmortem were recorded.

### 3.2 CARE AND MANAGEMENT OF CHICKS

Out of one hundred and fifty four chicks, sixty four were reared in brooders upto 3 weeks age, and were divided into five groups as shown in table 1. These chicks were fed with coccidiostatic drug free chick mash and water ad lib. The housing and management of these chicks was same as described previously.

#### 3.2.1 Isolation and sporulation of E.tenella oocysts

Caeca were obtained from the fowls naturally infected with E.tenella the contents of the caeca were centrifuged with normal saline (0.85% Nacl) the sediment was subjected to repeated washings and centrifugations with normal saline till the supernatant obtained was clear. Then the sediment was resuspended in sheather's sugar solution (granulated sucrose-454 g, phenol-6.7 ml and tap water-355 ml) and allowed for 2 hours for the oocysts to float. The top most layer containing the oocysts was

collected with a pipette and the fluid thus collected was centrifuged after adding water, to settle the oocysts in the sediment. The oocysts were allowed to sporulate in 2.5 per cent potassium dichromate solution in shallow petri-dishes at room temperature ($27^{o}$ to $29^{o}$C). The petridish was closed partially with it's lid to minimise the evaporation of dichromate solution. Sufficient quantity of 2.5 per cent potassium dichromate solution was added occasionally to make up the loss due to evaporation. The petridishes containing the oocysts were agitated gently now and then and with the help of pipette, air was bubbled into the solution to facilitate oxygenation. Majority of the oocysts sporulated within 24 to 48 hours.

The sporulated oocysts with the fluid medium were centrifuged and their exogenous characters were studied. The oocysts assigned to E.tenella were picked up in single numbers on to a felatin flake under low magnification with a fine pasture pipette. The flakes containing single sporulated oocysts were fed to a number of one day old white leghorn chicks. Infection was observed to have been established in about 25 percent of chicks. The prepatent period was observed to be 7 days. The morphological characters of the oocysts and the type of lesions confirmed the infection to be solely due to E.tenella. The oocysts

raised in the chicks from single oocyst infections were again sporulated at room temperature and were propagated in chicks for harvest of a crop of oocysts to be used in experiments. The isolated strain was maintained by serial passage through chicks in the laboratory. Oocysts harvested as a crop were sporulated in 2.5 per cent dichromate, washed thoroughly with clean water to get rid of dichromate. The sporulated oocysts thus washed were adjusted to the required dose of inoculum in 1 ml of water.

3.2.2 Experimental design

In this experiment sixty four white leghorn chicks of 3 weeks age were utilised. They were wing banded individually and were randomly assorted to 5 groups of 16, 16, 16, 8 and 8. Each group of chicks was housed in a separate pen. All possible sanitary and hygienic measures were adopted during the experimental period.

3.2.3 Dose of infection

At the age of 21 days the birds belonging to the groups I, II, III, IV were infected with 70,000 sporulated oocysts of E.tenella per os, per bird by means of sterile glass syringe. These oocysts were suspended in 1 ml of distilled water. Group V served as uninfected and unmedicated control. Group IV served as infected and unmedicated control.

3.3 The following parameters were observed.

3.3.1 Mortality

Mortality was recorded on the whole and expressed as per cent mortality.

3.3.2 Faecal scoring

Faecal droppings were observed thrice a day on 4th,6th,8th and 10th day of post infection and were scored, according to the method of Morehouse and Baron (1970).

3.3.3 Lesion scoring

Four birds selected at random from each group were sacrificed on 5th, 7th, 9th and 12th days of post infection and were autopsied. The caecal mucosa was examined at autopsy for gross pathological changes (lesions) and lesions were scored qualitatively and graded as an arbitary scale of 0 to 4 in the manner described by Johnson and Reid (1970). An average of the lesion scores of all the sacrificed birds on different post infection days i.e., 5th, 7th, 9th and 12th were calculated and taken as a group lesion score for that day of infection.

### 3.3.4 Oocyst count

Oocyst output was counted daily over 24 hours for birds under different treatments on 5th, 7th, 9th and 11th days of post infection. The dilution method was adopted to count the oocysts.

The oocysts were counted in each of the samples using a Mcmaster Counting Chamber. The mean of the number of oocysts counted from three samples from each bird was corrected by multiplication with the dilution factor for expressing as average number of oocysts per bird.

### 3.3.5 Body weight gain

The birds were weighed on 0 and 12th day of post infection and the body weight gain of 0-12 days post infection was calculated individually, for each group of chicks.

### 3.3.6 Histopathological studies

Small pieces of caecum were collected and fixed in 10 per cent buffered formalin. After fixation they were processed by paraffin embedding method. Section of 5 u thick were cut and stained by Haematoxylin and Eosin stain.

Fig.No.1: Photomicrograph showing unsporulated oocyst of coccidia. x 100

Fig.No.2: Photomicrograph showing sporulated oocyst of coccidia. x 100

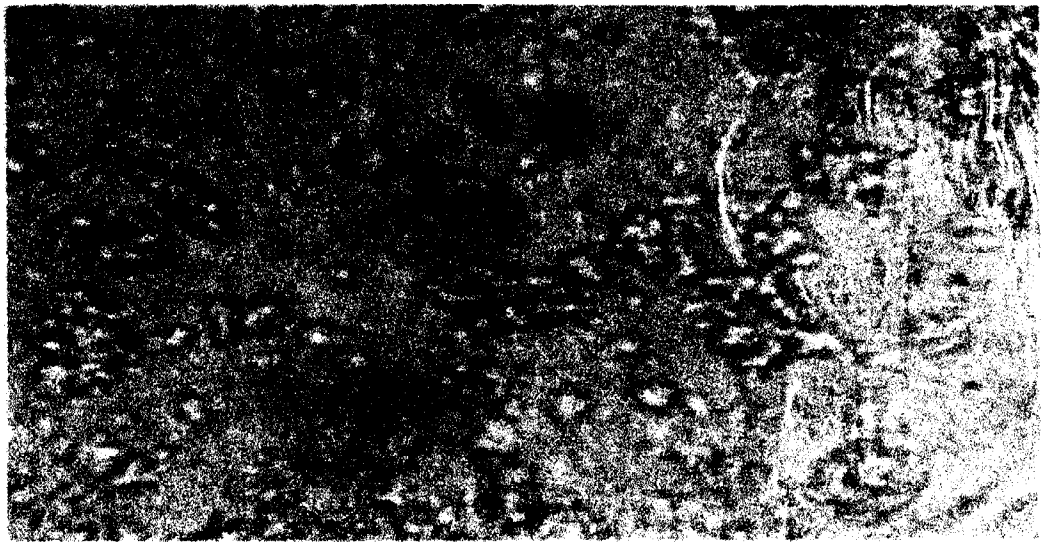


Fig.No.3: Photomicrograph showing the egg of Ascaridia galli x 450

Fig.No.4: Photomicrograph showing embryonated egg of A.galli x 150

# RESULTS

# CHAPTER IV

# 4. RESULTS

## 4.1 DRUG TRIALS AGAINST LARVAL FORMS OF A.GALLI

The results of trial groups (I, II and III) and control groups (IV, V), giving the details of dose of iniection, number of days post infection and number of days post-medication on which the bird was sacrificed and the number of larvae recorded both in lumen (by flushing) and mucosa (by digestion) were recorded in table No. 1 to 5.

In the I, II and III groups treated with Wopell, Anthelmex and Krimos the average recovery of larval forms were 15.33, 23.33 and 32.33 respectively. In IV group (infected and unmedicated) average recovery of larval forms were 341. The comparative efficacy of Wopell, Anthelmex and Krimos against larval forms of A.galli was given in table No.6. The tabulated results showed that Wopell, Anthelmex and Krimos were 95.5, 93.1 and 90.5% efficacious against larval forms of A.galli. The uninfected and unmedicated control did not develop any infection.

## 4.2 DRUG TRIALS AGAINST ADULT WORMS OF A.GALLI

The details of the efficacy of Wopell, Anthelmex and Krimos against adult worms are given in tables No. 7 to 12.

In the I, II, III groups of birds infected with 1000 embryonated eggs of A.galli and treated with Wopell, Anthelmex and Krimos. The average recovery of adult worms was 0.33, 0.83 and 1.8 respectively. In the IV group (infected and unmedicated). The average recovery of adult worms were 20. It is indicated that Wopell, Anthelmex and Krimos were 98.35, 95.85 and 91.0% efficient against adult worms of A.galli.

The efficiency in all experiments on drug trials was determined by using the formula $\frac{t_1-t}{t_1} \times 100$ where '$t_1$' is the average number of larval/adults in infected controls. 't' is the average number of larval/adults survived in the medicated group.

4.3 The efficacy of zycox at different concentrations has been clearly shown by different parameters in Tables No. 13and 14. Efficacy of zycox on different parameters.

In the group of I, II, III and IV each bird infected with 70,000 sporulated oocysts of E.tenella. Birds in groups I, II and III were treated with 4 ml, 5 ml and 6 ml of zycox per 10 litres of water respectively for a period of 7 days. Treatment was given 2 days post infection. Group IV was infected but not medicated. Group V was uninfected and unmedicated control.

4.3.1 Faecal score

In the group I the average faecal score of four observations was 3.5, 2.5, 1.5 and 1.0 on 4, 6, 8 and

10 days post infection with an average faecal score of group was 2.12 and the faecal score protection was 47%.

In the group II the average faecal score of every four observations was 3.0, 1.0, 1.0 and '0' on 4, 6, 8 and 10 days post infection with an average faecal score of group was 1.25 and the faecal score protection was 68.95%.

In the group III the average faecal score of four observations was 2.5, 1.5, 1.5 and '0' on 4, 6, 8 and 10 days post infection with an average faecal score of group was 1.12 and the faecal score protection was 72%.

Birds in group IV infected but left as unmedicated. The average faecal score of every four observations on 4, 6, 8 and 10 days of post infection was 4.0 with average of group faecal score 4.00 and the faecal score protection was 0.00.

Group V kept as uninfected and unmedicated control and faecal score for coccidiosis was zero on 4,6, 8 and 10 days of post infection.

Among the infected and medicated groups the average faecal score of group I was comparatively higher (2.12) than that of groups II (1.25) and III (1.12). Statistically there was no significant difference between three

medicated groups (I, II and III). But each of the medicated groups was differing significantly with group IV.

4.3.2 Lesion score

The datas regarding lesion score was present in table No.13.

Lesion scores were taken on 5, 7, 9 and 12 days of post infection for 5 groups.

In group I the average lesion score of 4 observations on 5, 7, 9 and 12 days post infection was 3, 2, 1.5 and 1.0 with an average lesion score of group was 1.87 and the lesion score protection per cent was 53.25.

In group II the average lesions score of 4 observations on 5, 7, 9 and 12 days of post infection was 2.5, 1.5, 1.0 and '0' with an group average 1.25 and the lesion score protection per cent was 68.75.

In group III the average lesionscore of 4 observations on 5, 7, 9 and 12 days post infection was 2, 1.5, 1.0 and '0' and the group average was 0.12 and the lesion score protection per cent was 72.

In group IV the average lesion score of 4 observations on 5, 7, 9 and 12 days post infection was 4.00 and group average was 4.00

The average lesion score was highest (4.00) and lesionscore protection per cent was lowest in IUC (IV) group. No lesions could be seen for coccidiosis in UUC (V) group. By day 12 post infection the lesion score was reduced to '0' in group II and III; 1.0 in group I. Group III has recorded lowest mean number of lesion score. Statistical comparison between the groups I, II and III exhibited no significant difference in the lesion score at 5 per cent level.

4.3.3 Oocyst count

The efficacy of zycox at different concentrations was shown in table No.14.

The oocyst production per bird was counted on 5, 7, 9 and 11 days of post infection.

The faecal droppings collected from group V (UUC) was free from oocysts.

In the group I the average oocyst count for four observations on 5, 7, 9 and 11 days post infection was 13, 9, 7 and 4 (millions) and the group average was 8.2.

In the group II the average oocyst count for four observations on 5, 7, 9 and 11 days post infection was 8.3, 6.7, 4.5 and 3.5 (millions) and group average was 5.5.

In the group III the average oocyst count for four observations on 5, 7, 9 and 11 days post infection was 9, 6.2, 3.7 and 2.3 (millions) and group average was $2.3 \times 10^3$

In the group IV (IUC) the average oocyst count for four observations on 5, 7, 9 and 11 days of post infection was 46, 50, 30 and 18 (millions) and group average was 36. Perusal of the oocysts counts given in table 2 revealed a significantly higher rate of oocyst production in group IV compared with groups (I, II and III).

4.3.4 Mortality

The group wise per cent mortality in response to different treatments has been shown in the table 14.

The mortality recorded in the groups I, II, III and IV was 5, 1.5, 1.5 and 60 per cent respectively. There was no mortality in the healthy control (group V).

4.3.5 Body weight

The group wise per cent weight gain in response to different treatments has been shown in the table 14.

The body weight gains from 0-12 days post infection of groups I, II, III, IV and V were 310, 316, 322, 225 and 320 g respectively and the corresponding percentage body weight gains were 96.87, 98.75, 100.6, 70.30 and 100% respectively.

The birds of all medicated groups showed significantly higher body weight gain in comparison to IUC group (IV).

4.3.6 Histopathological studies

Autopsy of birds were done on 5, 7, 9 and 12 days of post infection.

On day 5 P.I.

Group I

Gross lesions: The caeca was greatly distended few haemorrhagic patches seen on caecal wall.

Microscopic lesions:

: The denudation and desquamation of the epithelium, the developed schizonts were seen in the cytoplasm of the glandular epithelial cells. Edema of submucosa and muscular layer of the caecum, and cellular infiltration of lymphocytes in the mucous and submucous layers, distorsion of villi were noticed.

Group II

Gross lesions: The caeca was moderately distended.

Microscopic lesions:

: Edema of submucosa and muscular layers and thickening of mucosa was seen. Distorsion of the villi. Glands showing hyperplastic changes and also hyperplasia

of lymphoid follicles with proliferation of lymphocytes. Schizonts were seen in most of the epithelial cells. Desquamation and denudation of epithelial cells.

Group III

Gross lesions: Caeca was moderately extended.

Microscopic lesions:

Oedema and congestion of submucosa and muscular layers, the desquamated and degenerated epithelium forming part of the caecal core was lying free in the lumen. Infiltration of lymphocytes around the glandular epithelial cells. The developed schizonts were seen in glandular epithelial cells.

Group IV

Gross lesions: Numerous haemorrhagic patches on caecal wall were noticed. Caeca was distended with blood clots.

Microscopic lesions:

Cellular infiltration in lamina propria denudation and desquamation of epithelial lining of the villi were observed. Edema of submucosa and muscular layers of caecum. More number of schizonts in the epithelial cells of the villi and also rupture of epithelial cells due to developing schizonts. Congestion and haemorrhages were marked in the mucosa, submucosa and muscular layers.

On day 7 P.I

Group I

Gross lesions: Caecum was still in distended conditions.
Microscopic lesions:

Edema of muscular layer, distortion of villi, thickening of submucosa and lamina propria, more number of goblet cells, denudation and desquamation of the epithelial cells with schizonts. Infiltration of lymphocytes and fibroblasts were noticed. The schizonts, gametocytes and forming oocysts were seen in most of the epithelial cells.

Group II

Gross lesions: Caeca was slighly distended.
Microscopic lesions:

Infiltration of lymphocytes and fibroblasts, hyperplasia of mucosal glands, few of the epithelial cells of the villi contained oocysts.

Group III

Gross lesions: Caeca was slightly distended.
Microscopic lesions:

More number of goblet cells were observed. Only few of the epithelial cells contained oocysts. Infiltration of lymphocytes and fibroblasts.

Group IV

Gross lesions: Caeca was greatly distended.
Microscopic lesions:

Most of the epithelial cells of mucosa contained oocysts. There was destruction and desquamation of epithelial cells. Infiltration of lymphocytes were seen in mucosal, submucosal and muscular layers. Glandular hyperplasia.

On 9 day P.I.

Group I

Gross lesions: Distension of caeca were slight.
Microscopic lesions:

Hyperplasia of mucosal glands, fibrous tissue proliferation was seen. Lymphoid hyperplasia and infiltration of more of lymphocytes were seen, less number of oocysts in the glandular epithelial cells could be detected.

Group II

Gross lesions: Caecal distension disappeared.
Microscopic lesions:

Very few epithelial cell contained oocysts. Diffuse cellular infiltration in the mucosa was observed. Hyperplasia of lymphoid follicles was seen. Fibrous tissue proliferation was noticed.

Group III

Gross lesions: Caecal distension disappeared.
Microscopic lesions:

Number of oocysts in the epithelials were less cellular infiltration of lymphocytes were seen.

Group IV

Gross lesions: Distension of caeca was still noticed.
Microscopic lesions:

Diffuse cellular infiltration in the mucosa. Most of the epithelial cells contained oocysts. Hyperplasia of lymphoid follicles. Connective tissue proliferation in submucosa and muscular layers.

On day 12 P.I

Group I

Gross lesions: Nothing abnormal could be seen.
Microscopic lesions:

Regeneration of caecal mucosa hyperplasia of lymphoid follicles. No oocysts could be detected in the epithelial cells.

Group II

Gross lesions: Nothing abnormal could be noticed.
Microscopic lesions:

Regeneration of caecal mucosa. No oocysts could be detected in the epithelial cells.

Group III

Gross lesions: Nothing abnormal could be found.
Microscopic lesions:

No oocysts could be detected in the epithelial cells. Caecal mucosa was regenerated.

Group IV:

Gross lesions: Distension of caeca was still observed.
Microscopic lesions:

Connective tissue proliferation in sub-mucosa and muscular layer and cellular infiltration were seen. Hyperplasia of lymphoid follicles. Few of the epithelial cells contained oocysts.

Group V

Birds in group V did not show any pathological lesions throughout the experiment.

Table 1: Showing the details of efficacy of Wopell against larval forms of *Ascaridia galli*

Batch I

Group I

| Wing band No. of chick | Postmortem conducted | | No. of larval forms recovered | | |
|---|---|---|---|---|---|
| | Days of P.I | Days of postmedication | Flushing | Digestion | Total |
| 4 | 7 | 1 | 24 | 12 | 36 |
| 2 | 7 | 1 | 13 | 18 | 41 |
| 1 | 12 | 6 | 9 | 20 | 29 |
| 4 | 12 | 6 | 16 | 10 | 26 |
| 7 | 17 | 11 | 4 | 9 | 13 |
| 8 | 17 | 11 | 6 | 4 | 10 |
| 10 | 20 | 14 | 3 | 2 | 5 |
| 6 | 20 | 14 | 8 | 1 | 9 |
| 9 | 22 | 16 | 2 | 0 | 2 |
| 11 | 22 | 16 | 2 | 1 | 3 |
| 3 | 24 | 18 | 1 | 2 | 3 |
| 12 | 24 | 18 | 3 | 2 | 5 |

Total: 184

Average: 15.33

Note:
Dose of infection: 1000 embryonated eggs of *A.galli* per chick.
Wopell was given once only
Dose: @ 100 mgm per chick
Mode of administration: in the feed
Wopell was given 6 days of post infection

Table 2 : Showing the details of efficacy of Anthelmex against larval forms of A.galli.

Group II

| Wing band No. of chick | Postmortem conducted | | No. of larval forms recovered | | |
|---|---|---|---|---|---|
| | Days of P.I | Days of PM | Flushing | Digestion | Total |
| 16 | 7 | 1 | 11 | 37 | 48 |
| 14 | 7 | 1 | 16 | 6 | 22 |
| 18 | 12 | 6 | 9 | 4 | 26 |
| 20 | 12 | 6 | 26 | 8 | 34 |
| 15 | 17 | 11 | 4 | 14 | 18 |
| 19 | 17 | 11 | 10 | 15 | 25 |
| 21 | 20 | 14 | 20 | 9 | 29 |
| 23 | 20 | 14 | 12 | 21 | 33 |
| 17 | 22 | 16 | 4 | 7 | 11 |
| 22 | 22 | 16 | 2 | 13 | 15 |
| 24 | 24 | 18 | 7 | 5 | 12 |
| 13 | 24 | 18 | 2 | 7 | 9 |
| | | | | Total: | 282 |
| | | | | Average: | 23.53 |

Note:

1. Dose of infection: 1000 embryonated egg of A.galli per chick
2. Anthelmex was given once only
3. Dose of: @ 0.5 ml/chick.
4. Mode of administration: Orally
5. Drug was given 6 days of P.I.

Table 3 : Showing the details of efficacy of Krimos against larval forms of A.galli in chicken.

Group III

| Wing band No. of chick | Postmortem conducted | | No. of larval forms recovered | | |
|---|---|---|---|---|---|
| | Days P.I | Days PM | Flushing | Digestion | Total |
| 25 | 7 | 1 | 56 | 2 | 56 |
| 27 | 7 | 1 | 44 | 4 | 48 |
| 30 | 12 | 6 | 32 | 6 | 42 |
| 31 | 12 | 6 | 18 | 28 | 46 |
| 29 | 17 | 11 | 31 | 7 | 38 |
| 32 | 17 | 11 | 16 | 20 | 36 |
| 34 | 20 | 14 | 8 | 17 | 25 |
| 35 | 20 | 14 | 11 | 9 | 20 |
| 26 | 22 | 16 | 2 | 16 | 18 |
| 28 | 22 | 16 | 7 | 17 | 24 |
| 36 | 24 | 18 | 12 | 3 | 15 |
| 33 | 24 | 18 | 6 | 14 | 20 |

Total: 388

Average: 32.33

Note:

1. Dose of infection: 1000 embryonated eggs of A.galli per chick
2. Krimos was given twice. Repeated after a week.
3. Dose of: @ 0.5 gm/chick.
4. Mode of administration: given with feed.
5. Drug was given 6 days of post infection

Table 4 : Showing the larval forms recovered in infected and unmedicated control

Group IV

| Wing band No. of chick | Postmortem conducted Days P I | No. of larval forms recovered Flushing | Digestion | Total |
|---|---|---|---|---|
| 40 | 7 | 179 | 183 | 362 |
| 38 | 12 | 193 | 201 | 494 |
| 37 | 17 | 172 | 188 | 360 |
| 41 | 20 | 97 | 89 | 186 |
| 42 | 22 | 124 | 116 | 240 |
| 39 | 24 | 75 | 68 | 143 |

Total: 2046

Average: 341

Table 5 : Showing the number of larval forms recovered in uninfected and unmedicated control.

| Wing band No. of chick | Postmortem conducted | No. of larval forms recovered Flushing | Digestion | Total |
|---|---|---|---|---|
| 44 | 7 | - | - | - |
| 43 | 12 | - | - | - |
| 45 | 22 | - | - | - |

Table 6: Showing the comparative efficacy of certain indigenous drugs (Wopell, Anthelmex and Krimos) against larval forms of A.galli

| Name of the drug | Average larval forms surviving in infected control | Average larval forms surviving in infected and medicated group | % of larval survival | % of effica |
|---|---|---|---|---|
| Wopell | 341 | 15.33 | 4.5 | 95.5 |
| Anthelmex | 341 | 23.53 | 6.9 | 93.1 |
| Krimos | 341 | 32.33 | 9.48 | 90.52 |

Table 7 : Showing the details of efficacy of Wopell against adult worms of*A.galii*.

Batch II

Group I

| Wing band No. of chick | EPG before treatment | Postmortem conducted i.e. Days of post medication | No. of adult worms found on postmortem |
|---|---|---|---|
| 50 | 2000 | 1 | 1 |
| 53 | 1600 | 1 | - |
| 52 | 1900 | 1 | - |
| 47 | 2300 | 1 | - |
| 49 | 3800 | 2 | 2 |
| 46 | 1600 | 2 | - |
| 55 | 2200 | 2 | - |
| 48 | 2800 | 2 | - |
| 51 | 3000 | 3 | 1 |
| 56 | 1400 | 3 | - |
| 57 | 1200 | 3 | - |
| 54 | 2000 | 3 | - |
| | | Total: | 4 |
| | | Average: | 0.33 |

Note:

1. Dose of infection: 1000 embryonated eggs of *A.galli* per chick.
2. Wopell was given 44 days of post infection
3. Eggs were first detected in the droppings of experimental chicks on42 day post infection.
4. Eggs counts were done as per stoll's method. EPG was calculated.

Table 8 : Showing the details of efficacy of Anthelmex against adult worms of A.galli.

Group II

| Wing band No. of chick | EPG before treatment | Postmortem conducted i.e. Days of post medication | No. of adult worms found on postmortem |
|---|---|---|---|
| 60 | 2500 | 1 | - |
| 59 | 2700 | 1 | 1 |
| 68 | 3000 | 1 | - |
| 67 | 2200 | 1 | 1 |
| 65 | 1600 | 2 | - |
| 63 | 2300 | 2 | 2 |
| 66 | 3800 | 2 | 3 |
| 62 | 3300 | 2 | - |
| 58 | 2100 | 3 | 1 |
| 61 | 1500 | 3 | 1 |
| 64 | 1900 | 3 | - |
| 69 | 2800 | 3 | 1 |

Total: 10

Average: 0.83

Note:

1. Dose of infection: 1000 embryonated eggs of A.galli/chick.
2. Anthelmex was given 44 days of post infection.
3. Eggs were first detected in the droppings of experimental chicks on 42 day post infection
4. Egg counts were done as per Stoll's method. EPG was calculated.

Table 9: Showing the details of efficacy of Krimos against adult worms of A.galli.

Group III

| Wing band No. of chick | EPG before treatment | Postmortem conducted i.e. Days of post medication | No. of adult worms found on postmortem |
|---|---|---|---|
| 78 | 4800 | 1 | 5 |
| 81 | 2000 | 1 | - |
| 80 | 2200 | 1 | 2 |
| 72 | 1800 | 1 | 3 |
| 74 | 1600 | 2 | 1 |
| 76 | 2300 | 2 | 4 |
| 77 | 2800 | 2 | 2 |
| 73 | 3200 | 2 | - |
| 70 | 2600 | 3 | 3 |
| 75 | 2400 | 3 | - |
| 71 | 1900 | 3 | 2 |
| 79 | 2100 | 3 | - |

Total: 22

Average: 1.8

Note:

1. Dose of infection: 1000 embryonated eggs of A.galli per chick.
2. Krimos was given 44 days of post infection.
3. Eggs were first detected in the droppings of experimental chicks on 42 day post infection.
4. Egg counts were done as per Stoll's method. EPG was calculated.

Table 10: Showing the number of adult worms of A.galli recovered in infected and unmedicated control

Group IV

| Wing band No. of chicks | Dose of infection (embryonated eggs of A.galli) | Postmortem conducted Days of postinfection | No. of adult worms recovered |
|---|---|---|---|
| 86 | 1000 | 45 | 30 |
| 84 | ,, | ,, | 15 |
| 82 | ,, | 46 | 20 |
| 83 | ,, | ,, | 28 |
| 85 | ,, | 47 | 16 |
| 87 | ,, | ,, | 21 |
| | | Total: | 120 |
| | | Average: | 20 |

Table 11: Showing the number of adult worms of A.galli recovered in uninfected and unmedicated control

Group V

| Wing band No. of chick | Dose of infection (embryonated eggs of A.galli) | Postmortem conducted Days of post infection | No. of adult worms recovered |
|---|---|---|---|
| 89 | - | 45 | - |
| 88 | - | 46 | - |
| 90 | - | 47 | - |

Table 12 : Showing the comparative efficacy of certain indigenous drugs (Wopell, Anthelmex and Krimos) against adult worms of A.galli.

| Name of the drug | Average number of adults surviving in infected control (group IV) | Average number of adults surviving in infected and medicated control | % of adults survivals | % of efficacy |
|---|---|---|---|---|
| Wopell | 20 | 0.33 | 1.65 | 98.35 |
| Anthelmex | 20 | 0.83 | 4.15 | 95.85 |
| Krimos | 20 | 1.80 | 9.0 | 91.00 |

Table 13: Showing the efficacy of Zycox (IHP-250C) at different concentrations against caecal coccidiosis

| Groups No. | Number of birds | Dose of drug/ 10 litres of water | Faecal score days of P.I 4 | 6 | 8 | 10 | Average | Faecal score protection % | Lesion score days of P.I 5 | 7 | 9 | 12 | Average | Lesion score protection % |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| I | 16 | 4 ml | 3.5 | 2.5 | 1.5 | 1.0 | $2.12^{b} \pm 0.48$ | 47.0 | 3 | 2 | 1.5 | 1.0 | $1.87^{b} \pm 0.39$ | 53.25 |
| II | 16 | 5 ml | 3.0 | 1.0 | 1.0 | 0.0 | $1.25^{bc} \pm 0.54$ | 68.95 | 2.5 | 1.5 | 1.0 | 0.0 | $1.25^{bc} \pm 0.45$ | 68.75 |
| III | 16 | 6 ml | 2.5 | 1.5 | 0.5 | 0.0 | $1.12^{bc} \pm 0.48$ | 72.00 | 2.0 | 1.5 | 1.0 | 0.0 | $1.12^{bc} \pm 0.37$ | 72.00 |
| IV (IUC) | 8 | - | 4.0 | 4.0 | 4.0 | 4.0 | $4.0^{a}$ | 00.00 | 4.0 | 4.0 | 4.0 | 4.0 | $4.0^{a}$ | 00.00 |
| V (UUC) | 8 | - | - | - | - | - | $0.00^{c}$ | 100.0 | - | - | - | - | $0.00^{c}$ | 100.0 |

Note: 1. Dose of infection: 70,000 sporulated oocysts of E.tenella
2. Drug was given 2 days post infection for 7 days
3. Each value is the mean value of four birds
4. Mean values subscribed with similar alphabets within the same column are non significant (P 0.05)
5. $\bar{X} \pm$ = Mean standard error

Table 14: Showing the effect of IHP-250C on oocyst output, mortality and weight gain of whiteleghorn chicks experimentally infected with caecal coccidiosis

| Group No. | Dose of drug per 10 litres of water | Oocyst count millions/bird days of P.I | | | | Average | % Mortality | Body weights Days of P.I(gms) | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 5 | 7 | 9 | 11 | | | 0 | 12 | Average gms | % |
| I | 4 ml | 13.0 | 9.0 | 7.0 | 4.0 | $8.2^{b}$ | 5.0 | 470 | 780 | 310 | 96.87 |
| II | 5 ml | 8.3 | 6.7 | 4.5 | 3.5 | $5.5^{bc}$ | 1.5 | 484 | 800 | 316 | 98.75 |
| III | 6 ml | 9.0 | 6.2 | 3.7 | 2.3 | $5.3^{bc}$ | 1.5 | 472 | 794 | 322 | 100.60 |
| IV | - | 46.00 | 50.00 | 30.00 | 18.00 | $36.00^{a}$ | 60 | 475 | 700 | 225 | 70.30 |
| V | - | - | - | - | - | $0.00^{c}$ | - | 480 | 800 | 320 | 100.00 |

Note: 1. Dose of infection: 70,000 sporulated oocysts of E.tenella
2. Drug was given 2 days post infection
3. Each value is the mean value of four birds
4. Mean value subscribed within the same column are non-significant (P 0.05).

Fig No. 5 : Photo graph showing caeca of I,IV and III groups on 5 day P.I. Caecum (IV) with blood clots. Caeca of I,II groups with haemorrhages x

Fig No. 6 : Photo graph showing caeca of III group on 5 day P.I. showing congestion x

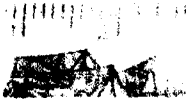
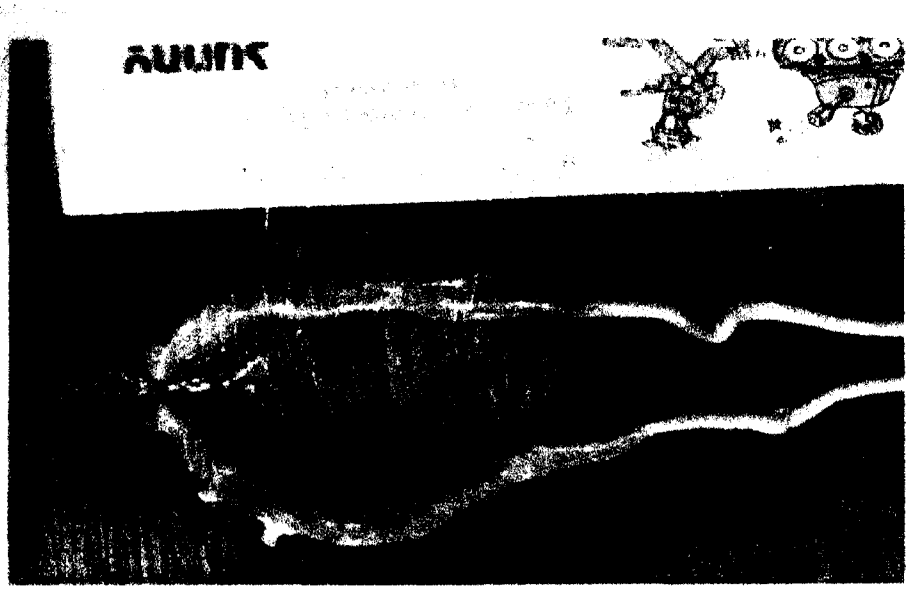


Fig No. 7 : Photo[illegible]graph showing caeca of II and III groups on 7 day P.I. caeca with haemorrahges x

Fig No. 8 : Photo[illegible]graph showing caeca of I group on 7 day P.I. caecal contents with blood.



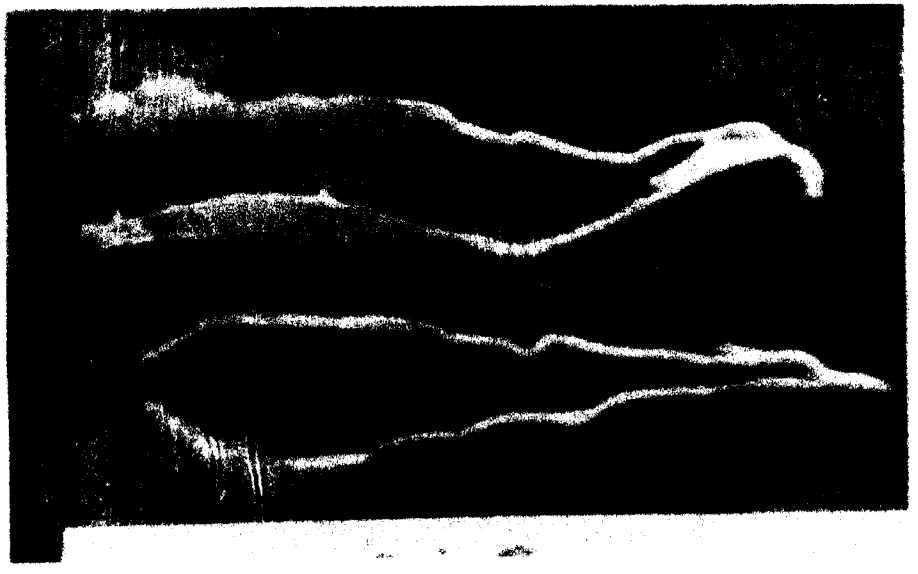


Fig No. 9 : Photo[illegible]graph showing caeca of I and II groups on 7 day P.I. caeca with distension and dilatation x

Fig No. 10 : Photo[illegible]graph showing caeca of III group on 7 day P.I. distension of caeca x

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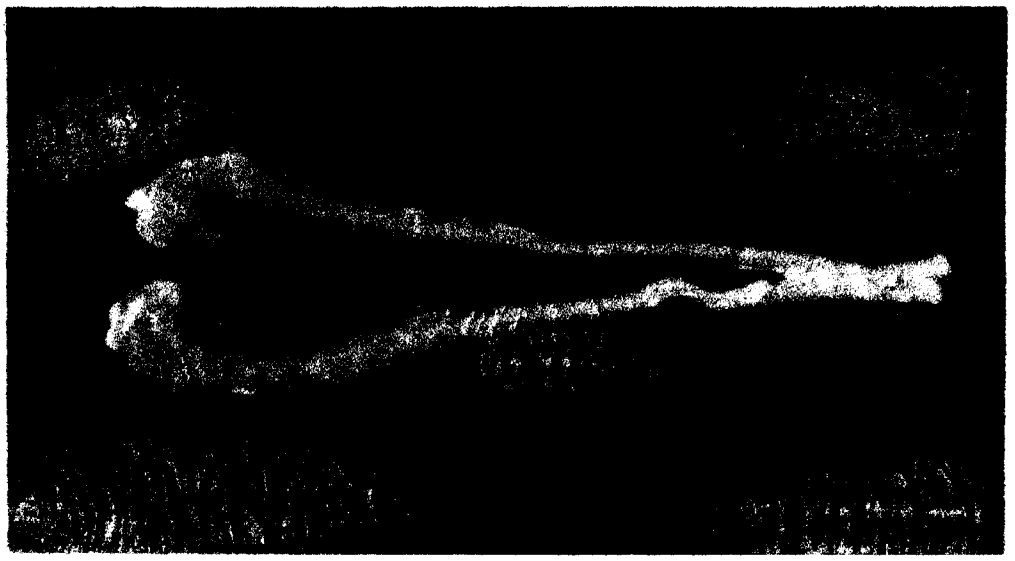
Fig No. 11 Photograph showing caeca of I group on 12 day P.I. x
Any abnormal could be detected.

Fig No. 12 : Photograph showing caeca of II group on 12 day P.I. x
Any abnormal could be detected.



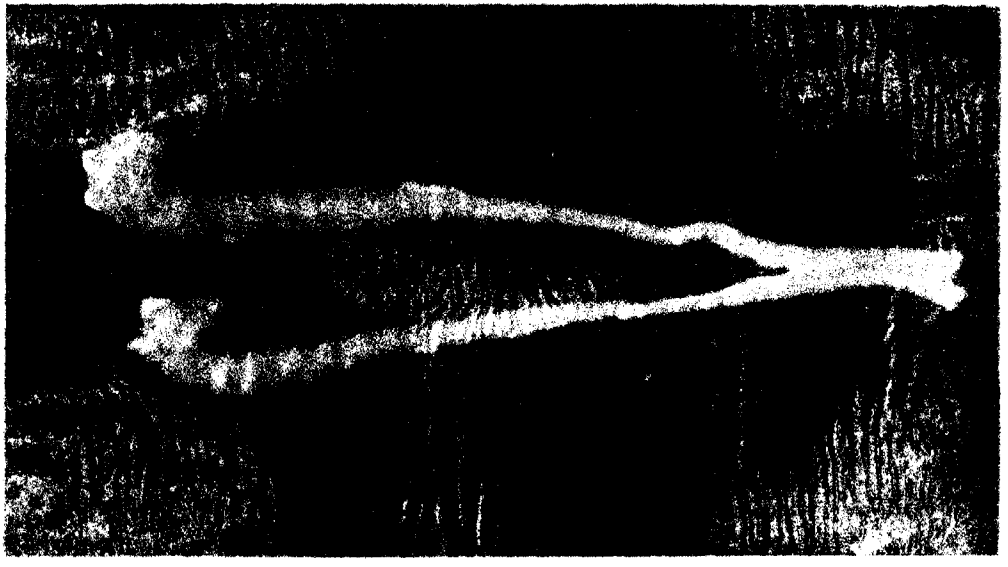
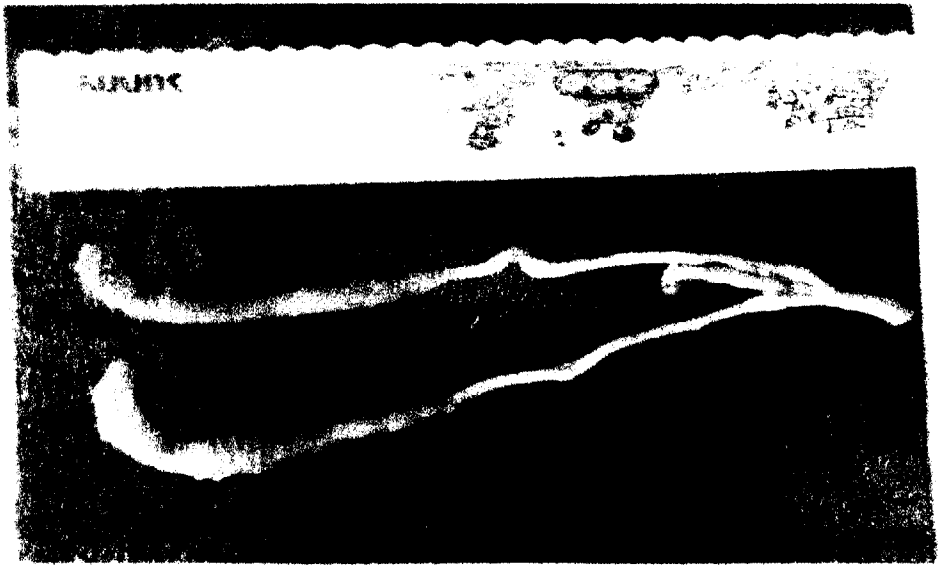


Fig No. 13 : Photograph showing caeca of III group on 12 day P.I. x
Any abnormal could be detected.

Fig. No. 14 Photograph showing caeca of IV group on 12 day P.I. x
Distension and dilation of caeca.



# DISCUSSION

# CHAPTER V

## 5. DISCUSSION

Even though, a number of anthelmintics and coccidiostats had been tried against ascaridiasis and coccidiosis respectively, in poultry, these diseases are still prevalent throughout the globe and are not under complete control.

Due to the development of resistance against a particular drug, the farmers are not in a position to check the control of these diseases. Hence, now-a-days a number of pharmaceutical companies are coming up with indigenous drugs against the parasitic infections. Preliminary study undertaken to test the efficacy of certain indigenous drugs, Wopell from M/S Indian Herbs, Anthelmex from M/S Agasthiar Pharmaceuticals and Krimos from M/S Bhartiya Bootee Bawan against ascaridiasis in poultry and zycox from M/S Indian Herbs against caecal coccidiosis.

The chicks in the batch I (group I, II and III) were sacrificed at definite intervals so as to have seventh day to twenty fourth day, post infections to note the larval forms both in lumen, as well as in mucosal tissue. This period was chosen since it takes generally seven to twenty two days post infection, for

the second stage larvae liberated from the eggs in the small intestine, to become immature adults (Ackert 1931, Lapage 1956, Deo and Srivastava 1955, Khouri and Pande 1970a). So also largely the so called tissue phase of the larvae had been recorded during this period with consequential damage to the mucosa of small intestine (Ackert 1931, Lapage 1956). Though the tissue phase was said to begin much earlier i.e. even on first day post infection (Ackert and Tugwell 1948, Tugwell and Ackert 1952).

The chicks in batch II were sacrificed 45 days post infection. This period was chosen since young worms matured in about 28-50 days in one month old chicken (Ackert 1931 and Feoktisov 1950). All the chicks in the infected and medicated groups were sacrificed on 1, 2, and 3 days after the drug was given to note the adult survivals.

In the present study it has been observed that the per cent survival of larval forms and adult worms from the chicks infected with 1000 embryonated eggs of A.galli and treated with Wopell at the dose rate of 100 mg per chick was 4.5 and 1.65. It is indicated that Wopell was 95.5 and 98.35% efficacious against larval forms and adult worms of A.galli.

In the past the drug trial with Wopell against larval forms and adult worms of A.galli has been conducted by Sundar Rao (1971) and observed that Wopell given in the feed at dose rate of 100 mg per chick was 95.67% efficacy against larval forms and adult forms of A.galli. Narahari (1980) observed that Wopell was 76.4% efficacy against immature forms in the natural infection of chicks with A.galli but 100% effective against adult worms of A.galli. in naturally infected chicks. Thus indicating that compound acted upon adult worms effectively, but was not very effective against immature worms. The present observations could be favourably compared with the findings of Sundara Rao (1971) and Narahari (1980).

In the present study the per cent survival of larval forms and adult forms from the chicks infected with 1000 embryonated eggs of A.galli and treated with Anthelmex at the dose rate of 0.5 ml/chick were 6.9 and 4.15. It indicated that Anthelmex was 93.1 and 95.85% efficacious against larval forms and adult worms of A.galli in chicks.

The present observations on the efficacy of Anthelmex against larval forms and adult forms of A.galli could not be compared with any other findings due to lack of literature on its efficacy against A.galli in chicks. But Hafeez et al. (1992 and 1993) observed that Anthelmex was effective against Trichostrongylosis in sheep and cattle.

Anthelmex which is less efficient than Wopell against larval forms and adult worms of A.galli in poultry could also be proved as a good drug of choice for poultry as the same is available in liquid forms which is more economical to farmers.

In the present study the per cent survival of larval forms and adult forms from the chicks infected with 1000 embryonated eggs of A.galli per chick and treated with Krimos at the dose rate of 0.5 g per chick were 9.48 and 9.0. It gave an indication that the Krimos was 90.52 and 91% efficacious against the larval forms and adult forms of A.galli in chicks.

The present observations on the efficacy of Krimos against larval forms and adult forms of A.galli could not be compared with any other findings due to lack of literature except that of Iqbal Hussain (1980) who reported Krimos as a good anthelmintic.

Therefore it is noted that Wopell, Anthelmex and Krimos are 95.5, 93,1and90.52%efficacious against larval forms of A.galli.and 98.35, 95.85 and 91.00 % efficacious against adult worms of A.galli, under the experimental conditions followed in the present study. Among the

anthelmintics used even though Wopell and Anthelmex are equally good drugs against larval form and adult forms of A.galli . It is tempted to report that Anthelmex is available in liquid form which will be preferred by the farmer for easy and safety administration. More over it is always advisable to change the anthelmintics regularly to avoid the drug resistance, the above three indigenous drugs would be drug of choice for the farmer.

In the present study to test the efficacy of zycox against caecal coccidiasis in birds, faecal scoring was done on 4, 6, 8 and 10 days of P.I and the average faecal score in the group IV (IUC) was 4.0 by observing faecal droppings with blood and continued to remain high. The present faecal score of 4 observed in the group IV was in accordance with the observations of Clark (1979), and Roy et al. (1989) who also noted that faecal score of 4 on 5 to 7 days.

The average faecal score in group I (treated with 4 ml), group II (treated with 5 ml),group III (treated with 6 ml of zycox) was 2.12, 1.25 and 1.12 respectively and the faecal score protection was 47.0, 68.95 and 72.00 per cent. It is indicated that zycox at the rate of 6 ml/ 10 litres of water gave the highest faecal score protection (72%) compared to other medicated groups of I, II.

The present observations are in agreement with the findings of Roy et al.(1990) and Guha et al. (1991) as Roy et al. (1990) observed that faecal score as 1.0 in the groups treated with zycox (powder form) at the dose rate of 2 gm/kg feed and 3 gm/kg feed in the experimentally infected chicks of 2 weeks age with $5 \times 10^5$ sporulated oocysts of E.tenella by sprinkling on the litter, and Guha et al. (1991) observed the average faecal score as 1.6, 1.2 and 1.2 in the groups treated with zycox at the rate of 2 gm/kg feed (0.2%), 3 gm/kg feed (0.3%) and 4 gm/kg feed (0.4%).

Lesion score:

In the present study the lesion score given on the basis of necropsy findings in the caeca revealed maximal intensity of lesions in group IV (4). The present observations of lesion score in group IV (IUC) was in agreement with the findings of Schanzel (1967), Kennet et al. (1967), Jasmer Singh and Hussain (1978), Sasmal and Sinha (1983), Padmavathi (1984), Roy et al. (1990) and Guha et al. (1991), as they also observed lesions core of 4 from 4 to 6 day of post infection.

The average lesion score was 1.87, 1.25, 1.12 and 4 in the groups of I, II, III and IV respectively. By considering the lesion score among infected groups it was found that it was highest in IUC (IV) and lowest in group III

The lesion score protection per cent was 53.25, 68.75 and 72.00 in the I, II and III group respectively where as it was 0.00 in IUC group (IV). The present observations are in accordance with the findings of Roy et al. (1990) who also observed lesion score of 1.6 and 1.2 and also lesion score protection per cent of 60 and 70 in the groups treated with 0.2% and 0.3% zycox (IHP-250C) against caecal coccidiosis.

Oocyst count:

In the present trial the oocyst output daily over 24 hours per bird was undertaken on 5th, 7th, 9th and 11th days of P.I. The faecal droppings collected from group V (UUC) were negative for the coccidial infections. The average oocyst out put was highest ($36 \times 10^3$) in group IV (IUC) while it was comparatively much lower in infected, medicated groups (I, II and III). Where as the average oocyst out put was 8.2, 5.5 and 5.3 in the I, II and III groups respectively and there was no such distence between II and III groups. Roy et al. (1990) Guha et al. (1991) reported the similar types of results on oocyst out put by using IHP-250C in the experimental case of coccidiosis in chicks.

Mortality:

In the present study there was no mortality in group V. The per cent mortality in group IV (IUC) was 60%. These findings were in accordance with those of Sasmal and Sinha (1983), Thyagarajan et al. (1987), who observed 70% and 66 per cent mortality in unmedicated infected birds respectively.

In the present study it has been observed that the per cent mortality in group I, II and III was 5, 1.5 and 1.5% respectively. The survival of birds in IMC groups (I, II and III) was highest compared to IUC group (IV). Roy et al. (1990) observed 93.34 and 100% survivality of birds in the groups treated with 0.2% and 0.3% zycox (IHP-250) respectively. Guha et al. (1991) observed 10, 3 and 2% mortality in the groups treated with 0.2%, 0.3% and 0.4% IHP-250 respectively. The present observations are in agreement with the findings of Roy et al. (1990), and Guha et al. (1991).

Body weight gain:

In the present experiment the average weight gain of groups I, II, III, IV and V (for 0-12 days P.I) was 310, 316, 322, 225 and 320 gms respectively. The per cent weight gain was 96.87, 98.75, 100.6, 70.3 and 100 respectively.

It has been observed that weight gains of infected, medicated groups (I, II, III) were higher than infected, unmedicated control group IV. This support the findings of Kennet et al. (1974), Giambrove et al. (1977), Morrison et al.(1979) who also observed the depression in body weight gains in the chicks infected with E.tenella and unmedicated.

Chicks infected with E.tenella and medicated group (III) i.e. treated with 6 ml zycox/10 litres of water showed higher weight gain than UUC group (V). Kirticar and Basu (1975) proved that H.andysentrica, Acorus calamus and E.ribes have carminative and appetising effect in man which might have acted as a growth stimulating factor under this study. The observations of the present study are similar to the findings of Guha et al. (1991) who observed mean weight gain per cent of 98.91, 100.09 and 100.53 in the groups treated with zycox @ 2 gm, 3 gm and 4 gm per kg feed respectively.

Histopathology:

In the present study caeca from the chicken belonging to the group V (UUC) were apparently normal. The absence of pathological changes were due to the fact that the birds were run on normal diets and were maintained under disease free atmosphere.

The histopathological changes consisting of desquamation of epithelial cells, cellular infiltration, haemorrhages, oedema, and developing stages of E.tenella at various depths of caecal wall were of higher intensity in the group IV compared to the infected and medicated groups I, II and III which might be due to the composite effects of the ingredients of IHP-250C. The same has been reported by Kirtikar and Basu, 1975.

Among the different groups treated with zycox at different dose rates (4 ml, 5 ml, 6 ml/10 litres of water), the group of birds treated with 6 ml of zycox showed negligible lesions on 12 days post infection.

# SUMMARY

## CHAPTER - VI

## 6. SUMMARY

An experimental study was carried out to note the relative anthelmintic efficacy of three indigenous drugs Wopell, Anthelmex and Krimos against larval forms and adult worms of Ascaridia galli in experimentally infected white leghorn chicks and also to study the efficacy of zycox (IHP-250C) a herbal product at different concentrations against Eimeria tenella in white leghorn chicks. It has observed that Wopell, Anthelmex and Krimos were 95.5, 93.1 and 90.52 per cent effective against the larval forms of A.galli. Against adult worms of A.galli Wopell, Anthelmex and Krimos were 98.35, 95.85 and 91.00 per cent effective respectively.

It has observed that zycox (IHP-250C) at different dose rates of 4,5 and 6 ml/10 litres of water showed the faecal score protection of 47%, 68.95 and 72% respectively when compared to 0.00% to the control group (IV).

Similarly the lesion score protection per cent was recorded as 53.25, 68.75 and 72.00 in birds treated with 4, 5, 6 ml of zycox/10 litres of water when compared to 0.00% in the control group and the average oocyst count was 8.2, 5.5, 5.3 millions/bird in chicks treated with 4, 5, 6 ml of zycox/10 litres of weight when compared to 36 millions/bird in the control group of birds.

The per cent mortality in birds treated with 4 ml, 5 ml and 6 ml of zycox per 10 litres of water was 5, 1.5 and 1.5 when compared to 60%, in birds infected and unmedicated group (IV).

The percentage weight gain in birds treated with 4, 5 and 6 ml of zycox/10 litres of water was 96.87, 98.75, and 100.06% respectively when compared to 70.30% in birds infected but not treated group (V).

The comparative efficacy of zycox at different dose levels indicated that 6 ml of zycox/10 litres of weight seems to be more efficacious than the dose rates of 4 and 5 ml of zycox/10 litres of water against caecal coccidiosis (E.tenella in white leghorn chicks). The efficiency of the dose rate of 6 ml/10 litres of water was also supported by the findings observed in the faecal score and lesions score protection, oocyst count, % mortality body weight gains and gross and histopathological lesions.

# LITERATURE CITED

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Ackert J E 1923 On the life history of fowl nematode Ascaridia perspicillum (Rud). Anatomy Record 26: 336.

Ackert J E 1931 The morphology and life-history of fowl nematode Ascaridia lineata (Schneidar). Parasitology 23: 360-379.

Ackert J E and Tugwell R L 1948 Tissue phase of Ascaridia galli life-cycle elucidated by artificial digestion apparatus. Journal of Parasitology 34: 32.

Clark P L 1979 Coccidial infection with E.tenella and caecal defecation in chickens. British Poultry Science 20: 317-322.

Deo P G and Srivastava H D 1955 Studies on the biology and life history of Ascaridia galli (Scharank, 1788). Proceedings of Indian Science Congress 3: 282-283.

Edgar S A, Davis D C and Frazier J A 1957 The efficacy of some piperazine compounds in the examination of helminths from experimentally and naturally infected poultry. Poultry Science 36: 495-510.

Feoktistov P I 1950 Quoted from Deo 1964.

Giambrone J J, Anderson W I, Reid W M and Eidson C S 1977 Effect of Infectious Bursal Disease on the severity of Eimeria tenella infections in broiler chicks. Poultry Science 56: 243-246.

Gill B S and Ray H N 1957 Life cycle and cytology of E.tenella Railliet and Lucet, 1891 with notes on symptomology and pathology of the infection. Proceedings of zoological society of calcutta-Mookerji. Memo pp.357-383.

Gill B S and Bajwa R S 1979 Drug resistance in field isolates of chicken coccidia from Punjab State. Indian Journal of Parasitology 3: 131-134.

Guberlet J E 1924 Notes on the life-history of Ascaridia perspicillum (Rud). Trans. Amer. Micro. Soc. 43: 152-156.

Guha C, Majumdar P and Pramanik A K 1991 Field trials of zycox (IHP-250C) against coccidiosis in broiler chicks. Indian Journal of Indigenous Medicines 29-31.

Gupta B R and Rao S V B 1959 A preliminary note on the Anthelmintic action of piperazine citrate on the large round worm of poultry Ascaridia galli under field conditions. Indian Veterinary Journal 36: 475-478.

Hafeez Md, Rasool S, Changalva Rayulu V, Rajasekhar B, Narendra Babu G and Venkateswarulu U 1992 Anthelmex against certain gastrointestinal nematodes of sheep. Livestock Adviser Vol. XVII. 34-35.

Hafeez Md, Venkateswarulu U, Rasool Syed and Narendra Babu G 1993 Anthelmex. An Anthelmintic against certain gastro-intestinal round worms of cattle. Livestock Advisor Vol.XVIII 29-31.

Horton-Smith C and Long P L 1952 Nitrofurazone in the treatment of caecal coccidiosis in chickens. British Veterinary Journal 108: 17-55.

Iqbal Hossain Md 1980 Report. Product Index, Bhartiya Bootee Bhavan pp.23.

Jagadesh Babu K S, Seshadri S J and Syed Mohiuddin 1976 Studies on the pathology of field cases of coccidiosis in poultry. Indian Veterinary Journal 53(1): 47-54.

Jasmer Singh and Hussain O H 1978 Therapeutic activity of Amprolium and codrinal against Eimeria tenella infection and effect of medication on the development of immunity. Indian Veterinary Journal 55: 56-60.

Johnson J K and Reid W M 1970 Anticoccidial drugs. Lesion scoring techniques in battery and floor for experiments with chicken. Experimental Parasitology 28: 30-36.

Katare R P 1972 Effect of anthelmintics on the developing stage of Ascaridia galli in experimentally infected chicken. Proceedings of 59th Indian Science Congress Part III. 554 (Abstr.).

Kennett R L, Mantor S and Gallo A 1974 Efficacy studies with Robenidine, a new type of anticoccidial, in the diet. Poultry Science 53: 978-986.

Kerr K B 1955 Age of chicken and rate of maturisation of Ascaridia galli. Journal of Parasitology 41: 233-235.

Lapage G 1956 Veterinary Parasitology pp.171: Oliver and Boyd Edinburg Tweeddale Court. London 39 A. Welbeck Street, W.I.

Long P L, Johnson J andWyatt R D 1980 Eimeria tenella: Clinical effects in partially immune andsusceptible chickens. Poultry Science 59: 2221-2224.

Madsen H 1962 The so called tissue phase in nematodes Journal of Helminthology 36: 143-144.

McLoughlin D K and Gardiner J L 1961 Drug resistance in Eimeria tenella. 1. The experimental development of a glycarbylamide, resistant strain. Journal of Parasitology (4) 1001-1006.

Moran J F Jr and Mizelle J D 1956 Notes on the habitat and tissue phase of Ascaridia galli (Schrank, 1788). Journal of Parasitology 42: 18.

Morehouse, Neal F and Baron, Robert R 1969 Coccidiosis: Evaluation of coccidiostats by mortality, weight gains, and fecal score . Experimental Parasitology 28: 25-29.

Morrison W D, Ferguson A E and Leeson S 1979 Efficacy of salinomycin and stenorol against various species of Eimeria and effect on chick performance. Poultry Science 58: 1160-1166.

Mukkur T K S and Bradley R E 1969 E.tenella: Packed blood cell volume, haemoglobin and serum protein of chicken correlated with the immune state. Experimental Parasitology 26: 1-16.

Narahari D 1980 Studies on the relative efficacy of certain anthelmintics against Ascaridia galli in chickens. Poultry Review 23-25.

Padmavathi P 1984 Studies on certain pathophysiological and immunological aspects in Eimeria tenella Railliet and Lucet, 1891 infection in chicks. Ph.D. thesis, Andhra Pradesh Agricultural University, Hyderabad.

Panda NC 1965 Routine versus diagnostic treatment in poultry against Ascaridiasis. Indian Veterinary Journal. 42: 615-621.

Roy S, Sasmal N K and Misra S K 1989 Efficacy of IHP-250C As coccidiostat in comparison with Bifuran against Eimeria tenella and mixed oocysts infection in broiler chicks. Indian Veterinary Journal 402-406.

Sadun E H 1949 The effect of single infections of variable size on the resistance of chicken to the nematode Ascaridia galli. American Journal of Hygeine 49 : 101-116.

Sharma ND and Sisodia C S (1971) Efficacy of piperazine adipate against Ascaridia galli worms in poultry. Gujarat Veterinary Journal 5 : 35-38.

Sasmal N K and Sinha P K 1983 Efficacy of manensin and bsalocid against amprolium-resistant strain of Eimeria tenella in poultry. Indian Journal of Animal Sciences 53 (7) : 794-749.

Srivastava H D and Malviya Hc 1959 Control of large intestine round worm of poultry Ascaridia galli Proceedings of 46th Indian Science Congress 4 : (Abstr.) 32.

Sundar Rao 1972. NVSc thesis. A.P.Agricultural University.

Technical Bulletin No. 18. 1971, Mannual of Veterinary parasitological laboratory Techniques. Ministry of Agriculture, Fisheries and Food, Her majesty's stationary Office, London. pp. 36-37.

Todd A C and Crowdus D H 1952 on the life history of Ascaridia galli. Trans. Amer. Micro. Soc. 71 : 282-287.

Thyagarajan D, Harahari D, Kothandaraman P and Ebenezer Raja E 1987 The effect of four different Anti-coccidial agents in experimental caecal coccidiosis in chicken. Kerala Journal of Veterinary Science 18 (1) : 31-36.

Tugwell R L and Ackert J E 1950 Further studies on the tissue phase of the life cycle of Ascaridia galli Journal of Parasitology 36 : 16 (Suppl.).

Venkataratnam A, Reddy K R, Reddy P R and Hafeez Md 1985 Caecal coccidiosis - Experimental transmission through cloaca and pathogenesis. Cheiron 14 : 2.

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