EFFECT OF ASCARIDIA GALLI INFECTION ON NEWCASTLE DISEASE (RANIKHET DISEASE) VACCINATION

BY

V. VIKRAMAN

THESIS

Submitted in partial fulfilment of the requirement
for the degree

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences
Kerala Agricultural University

Department of Preventive Medicine
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
Mannuthy - Trichur

1981

DECLARATION

I hereby declare that this thesis entitled "EFFECT OF ASCARIDIA GALLI INFECTION ON NEWCASTLE DISEASE (RANIKHET DISEASE) VACCINATION" is a bonafide record of research work done by me during the course of research and that the thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title of any other University or Society.

Manmathy,
31-7-1981.

V. VIKRAMAN.

CERTIFICATE

Certified that this thesis, entitled "EFFECT OF ASCARIDIA GALLI INFECTION ON NEWCASTLE DISEASE (RANIKHET DISEASE) VACCINATION" is a record of research work done independently by Sri. V. Vikraman under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to him.

E P Paily

Dr. E.P. Paily

(Chairman, Advisory Board)
Professor
Department of Preventive Medicine.

Mannuthy,

31-7-1981.

ACKNOWLEDGEMENTS

I have great pleasure to acknowledge my indebtedness to Dr. E.P. Paily, M.V.Sc., F.R.V.C.S., Professor of Preventive Medicine under whose inspiration and guidance this work was carried out.

I am extremely grateful to Dr. P.T. Georgekutty, M.V.Sc., Associate Professor, Department of Preventive Medicine for his valuable help, suggestions and encouragement. I am also grateful to Dr. A. Ramakrishnan, Ph.D., Professor of Poultry Science, Dr. V. Sathianesan, Ph.D., Associate Professor, Department of Parasitology, members of the Advisory Committee for their valuable advice and encouragement.

My sincere thanks are due to Dr. S. Sulochana, Ph.D., Associate Professor, Department of Microbiology for her technical advice and liberal material help, and also to Dr. G. Raghunathan Nair, Assistant Professor, Department of Poultry Science for his kind help during the course of study. Help rendered by Dr. A. Rajan, Ph.D., Professor of Pathology and Miss. C.C. Annamma, M.Sc., Junior Assistant Professor, Department of Statistics is also acknowledged with thanks.

I wish to express my deep sense of gratitude to Dr. P.G. Nair, former Dean, and to Dr. M. Krishnan Nair,

Dean, College of Veterinary and Animal Sciences for affording all the facilities for the conduct of the work.

I also wish to express my thanks to Dr. K. Venugopal, M.V.Sc., and to Dr. (Mrs.) K. Baby, M.V.Sc., Assistant Professors of the Department of Preventive Medicine for their keen personal interest and the help rendered.

My thanks are due to my colleague Dr. M. Abdul Azees for his co-operation, to Dr. T.R. Raju, Veterinary Surgeon, S.A.D.U., Cannanore, to Mr. Jit Bahadur Gurung and Mr. Phuntsho Wangdi for their help. I am also thankful to Dr. C.B. Manomohan for his personal interest in the preparation of the thesis.

I extend my thanks to Mr. Velu and to Miss. Santha former attenders of the Department of Preventive Medicine and Microbiology respectively for their assistance and ready help.

My sincere thanks are also due to the Kerala Agricultural University, for granting me the fellowship for the entire period of this work.

Lastly, but not the least, I place on record my indebtedness to my loving parents for all the help and encouragement.

DEDICATED TO MY
PARENTS AND SISTERS

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INTRODUCTION

Science has transformed many areas of human endeavour. Among them, advances in agriculture have touched and transformed the lives of the largest number in all parts of the world. Revolution in poultry production is a significant phenomenon of our times. The advent of high producing birds under scientific management has made poultry production an economical proposition.

Poultry farming is being given great emphasis in Indian economy as it provides profitable employment to the people in rural areas. The development of necessary infra-structure and the appropriate technology has made available the needed inputs and services for enhanced poultry production.

The success of poultry farming depends upon maintaining a healthy flock of birds. Diseases, in general, affect the poultry industry in many ways. Among them, diseases due to bacteria, viruses, fungi and parasites play an important role in impairing health or causing death.

Among the various contagious diseases of poultry, Ranikhet disease (Newcastle disease) is of considerable

economic importance especially under intensive husbandry practices. Being the foremost among the preventable viral diseases of poultry, this continues to receive considerable attention. Vaccination, if properly and judiciously employed, will prove beneficial in controlling the disease. Although several strains of viruses have been utilised in the production of effective vaccines, the control of Ranikhet disease is yet to be streamlined, as occasional outbreaks are noticed among birds even after vaccination.

The isolated outbreaks in some protected flocks is generally attributed to breakdown of immunity or resistance. This may result from lack of production of antibodies or reduction in their production. The quality and quantity of antigens, route of administration and the physical and nutritional status of birds also play an important role in the breakdown of immunity, in addition to bad housing, poor ventilation, subclinical disease conditions and hormonal imbalance of the body.

Heavy parasitism also probably contributes for vaccination failure, as it has been observed in several cases under field conditions, that heavily parasitised birds, when vaccinated against Newcastle disease, come down with the disease in the event of an outbreak.

Ascaridia galli, the common round worm of poultry,

is almost widespread and constitutes a contributory factor for low egg production and poor quality chicken apart from lowering the vitality and rendering the bird susceptible to diseases. Heavy rainfall, high humidity, absence of extreme winter and dense vegetation are apt to create conditions congenial for the prevalence of this round worm infection in poultry in Kerala.

Hence it is thought worthwhile to investigate the role of A. galli infection on the status of immunity against Ranikhet disease.

The present investigation is designed to study

1. the post-vaccinal reactions, if any, in A. galli infected chicken vaccinated against Newcastle disease,
2. the development of immunity against Ranikhet disease in A. galli infected chicken - as assessed by haemagglutination inhibition and challenge tests - and,
3. the need for deworming poultry before vaccination against Ranikhet disease.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

NEWCASTLE DISEASE

1.1. History and Incidence

Newcastle disease was first recognised in Java (Kraneveld, 1926). This is a virus disease of considerable economic importance. The isolation of the virus was made by Doyle (1927) in Newcastle-On-Tyne. In India, it was recognised at Ranikhet by Edwards (1928) and Cooper (1931).

From the records of the Food and Agriculture Organisation (FAO) of the United Nations, Chu and Risk (1972) have quoted the occurance of Newcastle disease in 118 countries.

With the introduction of Ranikhet disease vaccine, the mortality has now been considerably brought down (Bhatia, 1960).

National Commission on Agriculture (NCA), Government of India (1976) reported that about 1,150 outbreaks of Ranikhet disease occur annually in India, and on an average, 10,724 deaths occured during 1964 to 1968 as against 7,519 during 1969 to 1973.

The elimination of the disease by slaughter was successful in South Africa, England, Australia, Sweden and Wales where the velogenic strain of the virus was

prevalent; whereas the same procedure was unsuccessful in Netherlands, West Germany and Canada where the mesogenic and lentogenic strains were prevalent (Lancaster, 1966).

Seetharaman (1960) has stressed the importance of sanitary measures in the prevention of spread of Ranikhet disease. Chu and Risk (1972) reported that vaccination was practised as a method of control in 98 out of the 118 countries where the disease was prevalent.

Under the prevailing conditions of poultry husbandry, vaccination programme on an intensive scale is the practical approach for the control of the disease. For bringing down the Ranikhet disease infection to an insignificant level, NCA (1976) emphasized the need for raising the target of vaccination to cover atleast 80 per cent of poultry population and to maintain this level of vaccination target for a period of ten years.

1.2. Vaccine Strains used

Iyer and Dobson (1940) were the first to record successful immunisation of birds against Newcastle disease with living attenuated virus vaccines. The vaccines in common use in India are prepared from the mesogenic 'Komarov' strain (Komarov and Goldsmit, 1946) and the lentogenic 'F' strain (Asplin, 1952). The

'Mukteswar' or the 'R_2B' strain has been employed as an immunising agent in many countries including India (Seetharaman, 1960). Nilakantan et al. (1960) reported the use of 'Komarov' strain as an effective immunising agent in India as revealed by Haemagglutination inhibition (HI) titre. Control by vaccination is carried out either by living or inactivated vaccines. Live vaccines are of the mild type such as 'F', 'B' or 'LaSota' or the more pathogenic strains as 'Komarov', 'H', 'Roakin' or 'Mukteswar' (Gordon, 1979).

1.3. Duration of Immunity

Komarov et al. (1948) reported that birds vaccinated with 'Komarov' strain resisted challenge after nine months. Immunity following vaccination under controlled conditions with 'R_2B' strain vaccine has been established to be atleast for four years - for the life time of the bird or for its full utility period (Seetharaman and Nilakantan, 1951).

Daubney (1952) observed that his experience with the 'Mukteswar' and 'Komarov' strains of vaccines in actual outbreaks indicated that the former produced interference against natural infection better and helped in extinguishing outbreaks more promptly. Several workers have reported that solid immunity for twelve months was conferred by the 'Komarov' strain (Ileri, 1956; Thorne

and Mac Leod, 1960; Karrar and Mustafa, 1964). The influence of maternal immunity and its effect on the production of immunity to 'Komarov' strain has been stressed by many workers (Box, 1965; Lancaster, 1966; Allan, 1969 and da Silva and Brada, 1968).

Denika (1968) reported that the post-vaccinal immunity with Newcastle disease vaccine is primarily of cellular and not of humoral nature.

Quaglio and Lombardi (1973) stressed the need for two vaccinations, the first one to neutralise the maternal antibodies and the second to induce immunity, as the maternal antibodies interfere with the vaccinal immunity of chicks. Samuel et al. (1978) reported that the birds vaccinated with RDV 'F' strain and RDV 'K' strain withstood challenge three weeks post-vaccination by 100 per cent, whereas birds vaccinated with 'CDF_{66}' strain at hatch and at six weeks age withstood challenge by 91.6 per cent only.

1.4. Assessment of Immunity

Several serological techniques have been employed for assessing the immune status of birds vaccinated against Ranikhet disease. These include Agar gel diffusion test (Ahmed et al., 1965), Serum Neutralisation test (Gillo Torrado et al., 1967; Punnose and Rai, 1971), Haemagglutination inhibition (HI) test

(Cunningham, 1966 and Lancaster, 1966), Complement fixation test (Allan and Gough, 1976), Plaque neutralisation test (Hanson, 1972). Of these, HI test is the most widely employed.

Burnet (1942) was the first to show that Newcastle disease virus (NDV) has the property of haemagglutination and that antisera will neutralise this property; and this is the basis of HI which is proved to be valuable in the diagnosis of Ranikhet disease. Fabricant (1949) compared the HI and Serum Neutralisation (SN) tests in infected chicken and found that HI test reached a positive level earlier than the SN test.

HI test has been used as an effective method to evaluate the immune response to Newcastle disease virus by many workers (Markham et al., 1954; Cherby, 1967; Chang et al., 1969; Pal et al., 1970; Marthedal et al., 1973 and Beard and Max Brugh, 1975).

Beard and Max Brugh, (1975) have reported that challange test with virulent virus along with serological findings could be of considerable significance in assessing immunity to Ranikhet disease.

Gordon (1979) stated that antibody reaction to Newcastle disease virus are most easily detected by

the H^I test which is the simplest to perform.

On the contrary, some authors have reported that there was little correlation between HI titre and results of the challange test (Winterfield and Seadle, 1957; Baggi and Lee, 1960 and Devos et al., 1976).

1.5. Vaccination Defects and Failures

Outbreaks of Ranikhet disease in vaccinated flocks have been observed under actual field conditions (Singh and Gurukripal, 1977; Manickam and Gopalakrishnan, 1978; Ajinkya, et al., 1980 and Verma, 1981). NCA (1976) has suggested that every case of breakdown in immunity among vaccinated birds should be thoroughly investigated and prompt remedial measures taken.

One of the first examples of immunologic unresponsiveness was the observation by Sulzberger (1929) that guinea pigs previously injected with chemical ^{92} did not respond to neosalphenamine.

Birds innoculated with 'Mukteswar' strain of Ranikhet disease vaccine may show some drowsiness from the third to sixth day. Later, on a proportion ranging from about one to three per cent may show inco-ordination of leg movements and about one to

two per cent may manifest symptoms of post vaccinal paralysis without any complete recovery (Seetharaman, 1960). Hanson (1972) has reported that live virus vaccines produce adverse reactions such as mild respiratory disease and drop in egg production and reaction in stock infected with Mycoplasma. Vaccination with 'Mukteswar' strain of Ranikhet disease vaccine may show inco-ordination of limbs and sometimes paralysis in one to three per cent of the vaccinated birds (Seetharaman, 1977).

Tanwani and Malik (1978) reported no vaccination reaction to the lentogenic strains including 'CDF_{66}' but four per cent of chicks vaccinated with 'R_2B' strain showed lameness and paralytic symptoms.

Biondi and Schiavo (1965) reported inhibition of antibody production in Newcastle disease after prolonged treatment with zoalene and coxistant (coccidiostat). Alinassy and Kakuk (1976) attributed subclinical infection of Infectious bursal agent (IBA) to be a probable causative factor of immunosuppression of Newcastle disease vaccine. Giambrone et al. (1976) stated that chicks reared in an environment contaminated by IBA had lower geometric mean titres in the HI test for NDV.

Brugh (1977) reported that dietary Butylated hydroxy toluene prevents the serological response of chicken to avirulent NDV.

Leutskya and Fais (1977) reported that antibody content in chicken depends on the dose of vitamin A in the diet. In chicken fed on a high dose of vitamin A in the diet, antibody content in the serum is two to five times as high as in chicken which were not given vitamin A.

Meulemans et al. (1977) reported that the immunodepressive effect of IBA on Newcastle disease vaccination can be avoided by vaccinating the chicks against Newcastle disease at day old or by using attenuated strains of IBA in all commercial farms. Nathan et al. (1977) studied the effect of starvation on antibody production of chicks and observed that starvation caused increase in plasma ascorbic acid level, decrease in leukocyte count, loss in body weight, liver, bursa of Fabricius, spleen and thymus weights. A lower antibody titre was found on the sixth day post-vaccination in the group where deprivation started on the day before or on the day of vaccination.

Toma et al. (1977) has suggested that cortisol has an inhibitory effect on humoral immunity.

Giambrone (1979) reported that experimental infection with IBD virus at hatching or at three weeks of age resulted in a depression in the antibody response of chicken to ND vaccination and increased the susceptibility of those birds to challenge with virulent NDV.

Manickam and Gopalakrishnan (1979) suggested that factors such as depletion of proteins in the diet, deficiency of vitamin A, B complex, C and E, poor ventilation and bad housing, presence of subclinical infections, alterations in the hormonal balance of the body and agents such as ionisable calcium, silica, benzene, cortisone, mustard and X-rays in large doses play an important role in breakdown of immunity.

Ajinkya et al. (1980) reported an association of IBD in all cases of outbreaks of Ranikhet disease in vaccinated flocks in and around poultry farms in Bombay. Significant reduction in HI antibody titres were observed in chicks inoculated with a mild dose of Eimeria tenella oocysts, before, at day, or ten days after vaccination (Mohammed, 1980). Verma, (1981) attributed improper storage, handling, administration of vaccines and vaccination schedule, maternal immunity status in young chicks, changing pattern of the disease virus, viral diseases such as Infectious

bronchitis, Infectious laryngiotrachéitis, Adeno-virus, Avian encephalomyelitis and IBD to be the reasnons for break in immunity with respect to New-castle disease.

ASCARIDIASIS

Of the main parasitic diseases of poultry, Ascaridiasis, especially in chicken caused by Ascaridia galli (Schrank, 1788) (Freeborn, 1923) is considered to be one of the greatest handicaps in developing the growing poultry industry. The estimated total losses due to A. galli infection in growing chicken was 3.5 million rupees for Utter Pradesh and 110 million rupees for the whole of the country (Matta and Ahluwalia, 1979).

2.1. Incidence

Dutt (1950) found that 71 per cent of birds dying at Indian Veterinary Research Institute, Izatnagar were affected with more than one species of worm and of these 56 per cent had harboured A. galli.

This large round worm of poultry has been recorded in India from fowls and other birds from almost all parts of the country (Deo, 1964). Various

authors have reported incidence of A. galli infection from different states; 30 percent among nematodes in Uttar Pradesh (Qureshi, 1950); a common parasite in Kerala (Sundaram et al., 1962); 86.6 per cent from desi fowls in Maharashtra (Sastry, 1974) and 59.7 per cent in Assam (Gogoi, 1974).

2.2. Pathogenic effects

Sprehn (1930) found the occurance of this helminth in large numbers at times even to the extent of causing complete blockage of bowel.

Baudet (1930) observed that a small number of worms might be responsible for the weakened condition of the chicken and that the number of worms obtained on post-mortem was not proportionate to the condition of the birds. Moosy (1931) reported that even the presence of four to five worms in chicken and 15 to 20 in young hens may prove fatal.

Sundaram et al. (1962) observed retarded growth, droopiness, emaciation and diarrhoea in A. galli infected chicken. Dee (1964) reported droopy wings and leg weakness in heavy infection with A. galli in chicken. Seneviratna (1966) reported partial paresis and diarrhoea in birds infected with A. galli.

Droopiness, drowsiness, off feed and diarrhoea have been attributed to be the symptoms of ascaridiasis in chicken (Khouri and Pande, 1970).

Pavlicek and Dykova (1975) in their experimental study on five to six day old chicks have noted an establishment of upto 54 per cent of the eggs given and a decrease in weight of 68 to 78 per cent compared to the control birds. Amer et al. (1976) reported unthriftiness, general weakness and weight loss in A. galli infected chicken. Soulsby (1976) observed haemorrhagic enteritis, diarrhoea and anaemia in A. galli infection in chicken. Walker and Farrell (1976) observed a significant reduction in the metabolisability of the dietary energy and nitrogen retention in chicken infected with A. galli.

2.3. Blood changes

Georgekutty and D'souza (1967) reported reduction in haemoglobin, lymphocyte and monocyte count and an increase in heterophils and eosinophils in artificially infected chicken. Ikemi (1971) reported that A. galli infection had no significant effect on haematocrit, haemoglobin or plasma protein values.

Dubinsky et al. (1976) stated a reduction in levels of protein, lipids, glucose, albumin, globulin

coefficient and aspartate aminotransferase activity in blood samples of A. galli infected chicken maintained on a cereal ration deficient in aminoacids. Kaushik and Sen (1978) reported leukocytosis with corresponding absolute heterophilia and eosinophilia in A. galli infected chicken. Peter (1978) observed that there is reduction in haemoglobin, total erythrocytes count and an increase in leukocytes in ascaridiasis in chicken.

2.4. Effect on Ranikhet disease vaccine

Reports on the influence of A. galli infection on immunity to Ranikhet disease is scanty.

Food and Agricultural organisation (1960) reported that Newcastle disease vaccination may sometimes enhance the effects of a parasitic infestation. Seneviratna (1966) suggested that birds with heavy worm infection or those in poor condition should not be vaccinated against Newcastle disease as this would cause appreciable mortality. Tizard (1977) attributed disturbances in protein metabolism due to heavy worm infection to be a possible factor of vaccination failure. Vaccination reactions such as inco-ordination of limbs and sometimes paralysis with 'Mukteswar' strain of Ranikhet disease vaccine may become more acute if the birds are affected with

round worms, coccidiosis or weakness on account of malnutrition (Seetharaman, 1977).

MATERIALS AND METHODS

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Preliminary Procedures

a. Preparation of egg cultures

The worms for the preparation of egg cultures were collected from the intestines of naturally infected chicken of exotic and desi fowls slaughtered for table purposes at the local hotels and also from fresh carcasses brought for autopsy to Department of Pathology, College of Veterinary and Animal Sciences, Mannuthy.

The method adopted by Varghese (1966) was followed for collection and culturing of eggs.

Mature female worms were thoroughly washed in water to remove mucus and dirt. The cleaned live female worm was spread on a glass slide and the posterior end at the level of the anus was snipped off. The uteri together with the rest of the female genital organs were then milked out with the help of two dissection needles. Part of the intestine which came out along with the uteri and ovaries was discarded. For making cultures, only part of the uterus containing mature eggs was used. The dissected out ova from the uterus of several worms were pooled

in petridish and clean well water was added in sufficient quantities to give a depth of approximately three millimeters. Thus, a series of 23 such cultures were made for inducing artificial infection. The cultures were daily observed for the development of embryo, pooled and kept for inducing infection.

b. Standardisation of the culture

After uniform mixing, 0.1 ml of the culture was drawn with a graduated pipette and spread over a slide. The total number of ova were counted, and 100 such counts were made, and on an average it was found that 0.1 ml of the culture contained about 200 ova.

c. Mode of infection

The experimental infection was induced orally by administering embryonated eggs of A. galli with the help of a pipette.

d. Assessment of infective dose of A. galli.

To assess the infective dose of A. galli and to study the deleterious effects of worm burden, ten, day-old white leghorn chicks were procured from the University Poultry Farm, Mannuthy and reared under

parasite free conditions in electric cage brooders. They were confirmed to be parasite free by faecal sample examination at an interval of three days. When they attained four weeks of age, they were divided into two groups of five birds each. These two groups were artificially infected with embryonated eggs of A. galli at a dose rate of 1,000 and 1,500 respectively. They were maintained for another six weeks to recognise the development of deleterious effects, if any. Birds from each group were randomly slaughtered to record the worm burden.

e. Ranikhet disease vaccine

Freeze dried 'Komarov' strain of Ranikhet disease vaccine (RDV'K') supplied by Veterinary Biological Institute, Palode, Trivandrum, was used for the study. One ampoule of the vaccine was reconstituted with 100 ml of chilled normal saline and 0.5 ml was given subcutaneously to each bird immedeately after reconstitution.

f. Challenge virus

Velogenic NDV supplied from the Department of Microbiology, College of Veterinary and Animal Sciences, Mannuthy, was used for challenge.

Plan of the Experiment

Sixty, day-old white leghorn chicks of the same hatch vaccinated with RD'F' vaccine at day-old stage were procured from the University Poultry Farm, Mannuthy. All these birds were wing banded for identification. They were then reared under parasite free conditions in electric cage brooders. These chicks were fed with ad libitum quantities of chick mash supplied from the University Poultry Farm and clean drinking water. Eight birds died subsequently and the remaining 52 birds were randomly divided into three groups as A, B and C when they attained three weeks of age. The details pertaining to the treatments in each of these groups were as follows:

Group A

This group consisted of 20 birds. They were artificially infected with 1,500 embryonated eggs of A. galli (0.75 ml of the culture), at four weeks of age. When they attained eight weeks of age they were vaccinated with Ranikhet disease vaccine.

Group B

This group consisted of 20 birds. They were artificially infected with 1,000 embryonated eggs of A. galli at four weeks of age. When they attained

six weeks of age, they were vaccinated with Ranikhet disease vaccine.

Group C

This group consisted of twelve birds and served as control. They were vaccinated with Ranikhet disease vaccine at six weeks of age and they were not infected with eggs of A. galli.

Haematological Studies

Haematological studies were conducted one day prior to vaccination, and also at 30, 60 and 90 days post-vaccination. Randomly selected five birds from each group were used for the study.

Haematological study included estimation of erythrocytes, leukocytes, differential count, haemoglobin and serum protein content. For the study, two ml.of blood was collected from the wing vein using disodium salt of EDTA (0.3 gram per ml. of blood) as the anticoagulant. Two ml. of blood was collected for separating serum which was used for the estimation of serum protein.

Total erythrocytes and leukocytes were counted by the technique adopted by Nambiar (1961) as described below.

The following fluid was used for counting erythrocytes and leukocytes.

Stock solutions

Solution A - Sodium citrate two per cent.

Solution B - 0.1 per cent Gention violet in Ringer's solution.

Solution C - 0.1 per cent Brilliant Cresyl blue in Ringer's solution.

Solution D - Neutral Formalin.

The above four solutions were made and working solution was prepared daily before making the counts.

Solution A - 1 ml.

Solution B - 2 ml.

Solution C - 1 ml.

Solution D - 3 drops.

After mixing it was filtered. Duplicate counts of erythrocytes and leukocytes were made and the average was taken.

The haemoglobin percentage was estimated by Sahli's acid hematin method.

For differential count, duplicate smears from each sample were stained by modified copper peroxidase method described by Sato and Sekiya (Valsala, 1968).

Total serum protein content was estimated by the Biuret assay method of Inchiosa (1964). A standard curve with Bovine Serum Albumin was plotted as per details set out below:

The Volume of Albumin solution, the protein concentration in each tube and the corresponding colorimetric readings

Sl. No.	Quantity of Albumin solution (ml.)	Water added (ml.)	Concentration of protein (mg.)	Colorimetric reading
1.	0.25	2.25	1.25	34
2.	0.50	2.00	2.50	70
3.	0.75	1.75	3.75	105
4.	1.00	1.50	5.00	137
5.	1.25	1.25	6.25	174
6.	1.50	1.00	7.50	207
7.	2.00	0.50	10.00	280
8.	2.50	nil	12.50	315

Serological studies

Two ml. of blood was collected by venipuncture using sterilized syringes and needles. The blood was transferred to clean dry test-tubes and stored in the refrigerator until used. The serum was separated by centrifugation of the tubes.

The antibody titre of birds were estimated by

using Haemagglutination inhibition (HI) technique at 7, 14, 21, 28, 35, 42, 49, 56, 63, 70, 77, 84, 91, 98, and 105 days post vaccination. Beta method of HI technique using eight HA units of the virus and 0.5 per cent of chicken red blood cells was used for determining the antibody titre (Poultry Biologics, 1963).

Histopathological studies

To study the histopathological changes produced, the bursa of Fabricius, brain and sciatic nerve from paralysed birds died during the course of the experiment were collected and fixed in ten per cent formalin solution. The tissues were embedded in paraffin and four microns sections were taken and stained by Haematoxylin Eosin stain (Luna, 1968).

Challenge test

Velogenic NDV having an HA titre of 2,560 supplied from the Department of Microbiology, College of Veterinary and Animal Sciences, Mannuthy, was diluted to a final concentration of 1:100 and 0.1 ml. of this 1:100 diluted virus was injected subcutaneously into two birds having lowest HI titres from each group. Challenge test was conducted on all the birds when they attained 24 weeks of age. They were then

kept under observation for a period of 15 days.

Statistical analysis

Standard techniques of Analysis of Varience and related tests as given in Snedecor and Cochran (1967) were followed.

RESULTS

RESULTS

Egg Cultures.

During the present investigation 23 egg cultures of A. galli were made. Daily observation of the cultures revealed that the vermiform or the tadpole stage was reached by the seventh day. The infective stage (egg containing the second stage larva) was reached on the ninth day in thirteen cultures and on the tenth day in rest of the cultures. No change in the size of the eggs was noticed during its development to the infective stage.

Assessment of Infective Dose.

Details regarding the number of worms recovered in both groups are furnished in Table 1. The lowest number of worms recorded was nine while the highest was 63 with a mean of 37.4 in case of birds given 1,000 infective ova while it was 21 and 187 respectively with a mean of 82.6 in the case of birds given 1,500 infective ova. Forty and sixty per cent of the birds given 1,000 and 1,500 A. galli eggs respectively showed loss of apetite, diarrhoea and enteritis. However, in all cases birds exhibited stunted growth. Mortality was observed in a single case which was fed with 1,500 ova. The number of worms recovered

from this bird was 187.

Haematological Studies.

Erythrocyte Count.

The average erythrocyte count of groups A and B were significantly lower than the group C ($P<0.05$). The mean erythrocyte count in millions per cubic millimetre (10^6/c. mm) were 1.81, 1.79 and 2.24 in groups A, B and C respectively. The results are presented in the Table 2.

Leukocyte Count.

The average leukocyte count of groups A and B were significantly higher than the group C ($P<0.05$). The mean leukocyte count in thousands per cubic millimetre were 36.54, 40.87 and 25.08 in groups A, B and C respectively. The results are presented in the Table 3.

Haemoglobin.

The mean haemoglobin percentage of groups A and B were significantly lower than the group C ($P<0.05$). The mean haemoglobin value in grams percentage were 7.69, 7.64, and 10.27 in groups A, B and C respectively. The results are presented in the Table 4.

Serum Protein.

The average serum protein content of group A and B were significantly lower than group C($P<0.05$). The mean percentage serum protein of groups A, B and C were 2.75, 3.31 and 4.63 respectively. The results are presented in the Table 5.

Differential Count.

The values of the differential count are set out in the Tables 6 to 8.

Heterophils.

The average heterophil count of group A was significantly lower than the group C ($P<0.01$). However, no significant difference was observed between groups B and C. The mean heterophil count in percentage of groups A, B and C were 38.75, 44.75, and 46.15 respectively.

Eosinophils.

The average eosionophil count of groups A and B was significantly higher than that of the group C ($P<0.05$). The mean eosinophil count in percentage of groups A, B and C were 5.65, 6.45 and 2.40 respectively.

Monocyte.

The mean monocyte count of group A and B showed no significant difference from that of group C. The mean monocyte count in percentage of groups A, B and C were 1.15, 1.60 and 1.35 respectively.

Lymphocyte.

The average lymphocyte count of group A showed significant increase from that of group C ($P<0.05$). The mean lymphocyte count in percentage of groups A, B and C were 53.95, 46.50 and 48.30 respectively.

Basophil.

The average basophil count of groups A and B was significantly lower than group C ($P<0.01$). The mean basophil count in percentage of groups A, B and C were 0.50, 0.75 and 1.75 respectively.

HI Titre.

The mean HI titre of groups A and B were significantly lower than group C ($P<0.05$). Analysis of HI titre during different periods showed that the decrease in HI titre of group A and B were observed from 28th day post-vaccination onwards. The mean HI titres of groups A and B were 226.33 and 466.33 respectively, and in group C it was 808.12. The data pertaining to HI titres of different groups are

presented in Tables 9 to 19 and Figure 2.

Post-vaccinal Reactions.

Post-vaccinal reactions were observed in six birds of group A (30 per cent) and five birds of group B (25 per cent). The most prominent symptom observed was paralysis of the legs (Plate 1). Four birds showed paralysis of both legs while seven showed only on one leg. Of the birds that showed unilateral paralysis, five were affected on the left side and two on the right side. Paralysis of the left wing was noted in two birds of group A. All the affected birds showed droopiness. One bird in group B had weakness of the neck. All the birds which showed reactions died. These reactions were observed from the fifth to the 11th day post-vaccination in case of group A and from the fifth to the 13th day post-vaccination in case of group B. However, one bird in group B which showed symptoms of paralysis from the sixth to the tenth day after showing apparent recovery died on the 52nd day post-vaccination.

Post-mortem examination of the dead birds showed empty bowel, haemorrhagic enteritis and fluidity of the intestine. The highest number of worms recovered was 223 from a bird of group A and the lowest number

recovered was 39 from a bird of group B.

Post-vaccination reactions were not seen in any of the birds of group C. One bird in this group died due to non-specific causes.

Histopathological Studies.

Bursa of Fabricius showed abundant well formed plicae. Numerous well organised follicles and moderately thick stroma were observed. Both central and peripheral lymphoid cells within the follicles were hypertrophic and hyperplastic (Plate 2).

Brain showed small foci of degeneration in the cerebrum (Plate 3).

Sciatic nerve showed slight oedema and axonal degeneration characterised by swelling (Plate 4).

Challenge Test.

Out of the six birds used for challenge, four birds from the experimental group (groups A and B) showed similar symptoms such as droopiness, loss of apetite and generalised weakness. Greyish white diarrhoea and droopy wings were observed in three and two cases respectively. These birds recovered from the illness by the 12th day. Both the birds that were subjected to challenge from the control group (group C) resisted the challenge without showing

any symptoms. However, no mortality was observed after challenge in any of the groups.

TABLES

Table 1. Number of worms recovered after experimental infection with *A. galli* eggs.

Serial Number	Number of ova administered	
	1,000	1,500
1	9	21
2	17	42
3	39	66
4	59	97
5	63	187*
Mean	37.4	82.6

* Died on the 29th day post-infection.

Table 2. Erythrocyte count (millions/c.mm) in birds of groups A, B and C.

Days before/ post-vaccination	Group A					Group B					Group C				
	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
Before vaccination	1.56	1.72	1.68	1.50	1.66	1.76	1.58	1.56	1.64	1.69	2.18	2.17	2.09	2.01	2.08
30th	1.62	1.78	1.74	2.04	1.68	1.89	1.73	1.68	1.61	1.72	2.26	2.11	2.35	2.27	2.41
60th	1.94	2.06	1.79	1.89	1.89	1.84	1.78	1.86	1.76	1.84	2.19	2.15	2.42	2.36	2.26
90th	1.87	1.98	1.92	2.01	1.95	2.08	2.01	1.86	1.93	1.99	2.28	2.18	2.28	2.39	2.28
Mean	1.81					1.79					2.24				

Analysis of Variance Table

Source	df	SS	MSS	F
Between groups	2	0.630	0.315	31.50*
Period	3	2.514	0.838	
Error	54	0.543	0.010	
Total	59	3.687		

Critical difference (CD) = 0.064
Difference between means of groups A & C = 0.446*
Difference between means of groups B & C = 0.422*
*Denotes significant difference.

Table 3. Leukocyte count (thousands/c.mm) in birds of groups A, B and C.

Days before/ post-vaccination	Group A					Group B					Group C				
	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
Before vaccination	43.72	44.45	38.39	34.68	38.64	48.72	42.18	50.18	46.74	44.18	25.60	18.88	27.94	30.60	22.34
30th	38.67	42.38	33.58	33.71	37.82	36.96	40.67	39.68	40.10	40.62	22.35	21.34	28.96	24.78	27.96
60th	37.48	41.68	36.21	28.64	28.16	41.38	40.11	37.67	41.70	40.91	21.68	20.66	26.70	27.60	25.60
90th	38.23	41.72	34.86	29.73	27.64	34.18	38.68	32.18	38.00	41.00	26.00	21.32	28.20	28.40	24.80
Mean			36.54					40.87					25.08		

Analysis of Variance Table

Source	df	SS	MSS	F
Between groups	2	2640.487	1320.243	82.80*
Period	3	205.608	68.536	
Error	54	860.991	15.944	
Total	59	3707.086		

CD = 2.53
Difference between means of groups A & C = 11.461*
Difference between means of groups B & C = 15.707*
* Denotes significant difference.

Table 4. Haemoglobin (grams percentage) in birds of groups A, B and C.

Days before/ post-vaccination	Group A					Group B					Group C				
	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
Before vaccination	5.8	6.6	7.2	5.8	5.4	6.6	7.2	4.4	5.6	6.0	9.6	10.2	10.8	7.2	9.2
30th	6.0	7.2	7.8	6.8	7.8	7.2	8.4	6.2	9.2	7.8	10.8	10.4	12.6	8.6	9.6
60th	8.2	9.4	10.2	8.2	7.8	8.8	9.2	6.6	9.0	7.8	10.8	11.0	12.8	8.4	9.6
90th	7.8	9.0	10.6	8.0	8.2	10.6	9.4	7.8	10.8	8.2	11.2	11.0	13.0	8.4	10.2
Mean			7.69					7.84					10.27		

Analysis of Variance Table

Source	df	SS	MSS	F
Between groups	2	83.89	41.93	27.60*
Period	3	51.55	17.18	
Error	54	82.32	1.52	
Total	59	217.76		

CD = 1.284
Difference between means of groups A & C = 2.58**
Difference between means of groups B & C = 2.43**
*Denotes significant difference at five per cent level.
**Denotes significant difference at one per cent level.

Table 5. Total serum protein (grams/100 ml.) in birds of groups A, B and C.

Days before/ post-vaccination	Group A					Group B					Group C				
	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
Before vaccination	2.20	2.10	2.70	2.60	2.40	3.00	2.60	2.20	2.20	2.70	4.00	3.80	3.60	4.20	4.00
30th	2.40	2.20	3.20	2.90	2.70	3.20	2.60	2.70	2.40	2.70	4.40	4.70	4.40	3.90	3.90
60th	2.80	2.40	3.30	3.10	2.80	3.80	2.20	2.90	3.20	3.60	4.90	5.20	4.60	5.10	5.40
90th	2.80	2.60	3.30	3.10	3.40	3.80	3.10	3.00	3.00	3.40	5.40	5.70	5.40	5.20	4.90
Mean			2.75					3.31					4.63		

Analysis of Variance Table

Source	df	SS	MS	F
Between groups	2	8.005	4.003	29.219*
Period	3	43.592	14.530	
Error	54	7.416	0.137	
Total	59	59.013		

CD = 0.235
Difference between means of groups A & C = 1.885*
Difference between means of groups B & C = 1.720*

*Denotes significant difference.

Table 6. Differential count of leukocytes (%) in birds of groups A, B and C.

	No.	Before vaccination					Post-vaccinal interval: 30th					60th					90th				
		H	L	M	E	B	H	L	M	E	B	H	L	M	E	B	H	L	M	E	B
Group A	1	37	58	1	3	1	33	62	0	5	0	35	56	1	8	0	37	57	1	4	1
	2	38	54	2	6	0	42	49	1	7	1	41	53	1	5	0	39	52	0	8	1
	3	47	38	5	10	0	28	59	2	9	2	39	56	1	4	0	32	57	1	9	1
	4	35	59	1	5	0	37	60	0	3	0	44	54	0	2	0	41	49	3	5	2
	5	42	48	1	8	1	41	55	1	3	0	47	50	0	3	0	40	53	1	6	0
Group B	1	46	45	1	8	0	39	49	2	9	1	41	49	1	8	1	43	49	1	6	1
	2	40	47	5	6	2	50	36	4	8	2	44	44	2	9	1	53	41	1	5	0
	3	39	52	2	6	1	40	52	1	6	0	41	50	1	8	0	50	42	1	6	1
	4	40	51	1	7	1	43	48	2	6	1	48	41	2	9	0	54	41	1	4	0
	5	43	52	1	4	0	44	50	1	4	1	49	46	0	4	1	48	44	1	6	1
Group C	1	49	45	1	2	3	38	56	2	3	1	52	44	1	2	1	41	49	1	2	6
	2	47	46	1	3	3	49	48	1	1	1	49	44	2	4	1	47	50	0	1	2
	3	51	44	1	3	1	48	48	1	2	1	44	51	1	2	2	44	50	1	2	3
	4	42	52	2	3	1	44	49	2	3	2	46	50	1	2	1	47	49	1	3	0
	5	47	47	2	2	2	49	44	1	4	2	40	53	2	3	2	49	47	3	1	0

H - Heterophil; L - Lymphocyte; M - Monocyte;

E - Eosinophil; B - Basophil.

Table 7. Mean values of differential count in birds of groups A, B and C.

	H	L	M	E	B
Group A	38.75	53.95	1.15	5.65	0.50
Group B	44.75	46.50	1.60	6.45	0.75
Group C	46.15	48.30	1.35	2.40	1.75

Differential count - Overall comparison of groups - Chi-square value with one df.

Groups	H	L	M	E	B
A v/s C	12.90**	6.244*	0.30	26.24**	13.82**
B v/s C	0.43	0.683	0.42	37.068**	8.00**

* Denotes significant difference at five per cent level

** Denotes significant difference at one per cent level.

H - Heterophil

L - Lymphocyte

M - Monocyte

E - Eosinophil

B - Basophil

Table 8. Group wise comparison of differential count - Chi-square value with one df.

Groups	H	L	M	E	B	Periods
A v/s C	3.147	1.007	0.520	8.020**	5.333*	Before vaccination
A v/s C	5.400*	3.019	0.818	4.900**	1.600	30th day
A v/s C	1.430	1.427	1.600	2.314	7.000*	60th day
A v/s C	3.647	1.031	0.000	12.902**	2.250	90th day
B v/s C	1.766	0.350	0.520	7.360**	2.571	Before vaccination
B v/s C	0.324	0.168	0.889	8.696**	0.333	30th day
B v/s C	0.149	0.305	0.769	12.255**	1.600	60th day
B v/s C	0.840	1.697	0.091	9.000**	4.570	90th day

Critical value of Chi-square with one df = 3.84

* Denotes significant difference at 5% level.

**Denotes significant difference at 1% level.

Table 9. HI titre at varying intervals post-vaccination among experimental birds of group A.

No	Post-vaccination interval (Days)														
	7	14	21	28	35	42	49	56	63	70	77	84	91	98	105
1	40	80	80	160	160	320	320	320	320	320	320	160	160	160	160
2	80	80	80	80	160	160	160	160	160	320	160	160	160	80	80
3	40	40	40	80	160	320	320	320	640	640	640	640	320	320	320
4	160	160	160	640	640	640	320	320	160	160	160	160	160	160	160
5*	20	-	-	-	-	-	-	-	-	-	-	-	-	-	-
6	20	40	40	320	640	1280	1280	1280	640	640	640	640	320	80	80
7	20	20	80	80	640	640	640	640	640	160	160	160	160	80	80
8*	40	-	-	-	-	-	-	-	-	-	-	-	-	-	-
9*	20	-	-	-	-	-	-	-	-	-	-	-	-	-	-
10	160	320	320	1280	1280	640	1280	640	640	640	320	160	160	160	80
11	20	160	160	160	160	160	160	80	80	80	80	80	80	40	40
12	40	160	160	160	80	80	80	80	80	80	80	40	40	20	20
13	20	20	80	80	160	160	160	160	80	20	40	40	40	40	40
14*	20	-	-	-	-	-	-	-	-	-	-	-	-	-	-
15	40	80	80	320	160	160	80	160	80	80	80	40	40	40	40
16	40	40	160	320	320	160	160	160	160	80	80	80	40	40	40
17*	40	-	-	-	-	-	-	-	-	-	-	-	-	-	-
18	20	80	320	320	160	320	320	320	320	80	80	160	80	80	40
19*	20	-	-	-	-	-	-	-	-	-	-	-	-	-	-
20	40	320	320	640	640	320	160	160	160	160	80	80	80	20	20

* Died.

Table 10. HI titre at varying intervals post-vaccination among experimental birds fo group B.

No	Post-vaccination interval (Days)														
	7	14	21	28	35	42	49	56	63	70	77	84	91	98	105
1	20	20	80	80	160	320	320	320	320	320	160	160	160	160	160
2	20	80	160	160	320	320	320	320	320	160	160	160	160	80	80
3	40	160	640	640	640	320	320	320	160	80	80	80	80	80	80
4*	80	160	160	160	160	-	-	-	-	-	-	-	-	-	-
5	160	160	640	1280	640	640	320	640	640	320	320	320	160	160	160
6	320	320	1280	1280	1280	1280	640	640	640	320	320	320	160	160	160
7	40	160	1280	1280	640	640	1280	640	640	640	640	320	160	160	160
8	80	320	2560	2560	640	640	640	640	160	640	160	160	160	160	160
9	80	80	80	80	320	320	160	160	160	160	160	160	80	80	80
10	40	40	40	160	160	320	320	160	160	160	160	80	160	160	80
11*	80	320	1280	1280	640	640	640	-	-	-	-	-	-	-	-
12	80	320	1280	1280	1280	320	640	640	640	640	640	160	160	80	80
13	40	320	640	1280	1280	640	640	640	640	640	640	640	1280	640	640
14*	40	-	-	-	-	-	-	-	-	-	-	-	-	-	-
15	160	1280	5120	5120	5120	1280	1280	1280	640	640	640	640	640	640	640
16	20	320	320	160	160	160	160	160	160	160	160	40	40	80	80
17	40	320	1280	1280	1280	640	640	640	640	640	320	320	40	40	40
18*	80	80	-	-	-	-	-	-	-	-	-	-	-	-	-
19	40	640	640	320	80	160	160	160	160	160	80	80	80	80	40
20*	40	-	-	-	-	-	-	-	-	-	-	-	-	-	-

* Died

Table 11. HI titre at varying intervals post-vaccination among experimental birds of group C.

No	Post-vaccination interval (Days)														
	7	14	21	28	35	42	49	56	63	70	77	84	91	98	105
1	80	320	320	1280	1280	1280	1280	1280	640	640	640	640	640	640	640
2	40	160	640	1280	2560	2560	1280	1280	640	1280	1280	1280	1280	640	640
3	20	80	80	320	320	320	320	160	160	80	80	80	80	80	40
4	80	160	320	1280	1280	2560	1280	1280	1280	1280	320	320	320	320	320
5*	80	320	320	320	-	-	-	-	-	-	-	-	-	-	-
6	80	160	1280	1280	1280	640	640	640	640	320	320	320	320	160	160
7	40	640	2560	5120	5120	5120	1280	1280	1280	640	640	320	640	640	640
8	40	160	160	640	320	320	320	320	320	320	320	320	320	320	320
9	40	160	320	1280	1280	1280	1280	1280	1280	320	640	640	640	640	640
10	80	320	5120	5120	640	640	640	640	640	640	640	640	640	640	640
11	160	160	320	320	640	2560	2560	2560	640	640	640	640	320	320	320
12	80	320	640	640	640	640	320	640	640	640	640	320	640	640	640

* Died

Table 12. Mean HI titres of experimental birds at varying post-vaccination periods.

Days post-vaccination	Group A	Group B	Group C
7th	54.29	78.67	67.27
14th	114.29	302.67	240.00
21st	148.57	1069.33	967.27
28th	331.43	1130.67	1687.27
35th	377.14	933.33	1512.73
42nd	382.86	533.33	1687.27
49th	388.57	522.67	1018.18
56th	342.86	490.67	1032.73
63rd	297.14	405.33	741.82
70th	247.14	378.67	647.27
77th	208.57	309.33	560.00
84th	185.72	242.67	501.82
91st	131.43	234.67	530.91
98th	99.29	184.00	472.73
105th	85.71	176.00	454.55
Mean	226.33	466.33	808.12

Table 13. Analysis of variance - Group wise comparison of HI titres.

Source	df	SS	MSS	F
Between groups	2	2567423.983	1283711.996	10.278*
Error	42	5245947.787	124903.519	
Total	44	7813371.770		

Critical difference (CD) = 260.422

Difference between means of groups A & C = 582.121*
Difference between means of groups B & C = 341.998*

* Denotes significant difference.

Table 14. Analysis of variance - Period wise comparison of HI titres (Seven days post-vaccination).

Source	df	SS	MSS	F
Between groups	2	9641.026	4820.513	1.666ns
Error	49	141766.667	2893.197	
Total	51	151407.693		

ns. Not significant.

Table 15. Analysis of variance - Period wise comparison of HI titres (28 days post-vaccination)

Source	df	SS	MSS	F
Between groups	2	10290530.441	5145265.221	3.523*
Error	40	58423143.978	1460578.599	
Total	42	68713674.419		

CD for groups A & C = 475.439

CD for groups B & C = 455.666

Difference between means of groups A & C = 1355.84*
Difference between means of groups B & C = 556.60*

*Denotes significant difference.

Table 16. Analysis of variance - Period wise comparison of HI titres (49 days post-vaccination)

Source	df	SS	MSS	F
Between groups	2	12193512.829	6096756.414	9.475*
Error	38	24450389.610	643431.306	
Total	40	36643902.439		

CD for groups A & C = 654.141
CD for groups B & C = 635.900

Difference between means of groups A & C = 1298.701*
Difference between means of groups B & C = 1157.273*

*Denotes significant difference.

Table 17. Analysis of variance - Period wise comparison (70 days post-vaccination) of birds of groups A, B and C.

Source	df	SS	MSS	F
Between groups	2	1004832.771	502416.385	6.704*
Error	37	2773077.229	74948.032	
Total	39	3777910.000		

CD for groups A & C = 223.475
CD for groups B & C = 220.173

Difference between means of groups A & C = 400.130*
Difference between means of groups B & C = 268.606*

* Denotes significant difference.

Table 18. Analysis of variance - Period wise comparison of HI titres (91 days post-vaccination)

Source	df	SS	MSS	F
Between groups	2	1032364.329	516182.165	7.438*
Error	37	2567635.671	69395.559	
Total	39	3600000.000		

CD for groups A & C = 215.038
CD for groups B & C = 211.860

Difference between means of groups A & C = 399.481*
Difference between means of groups B & C = 296.242*

* Denotes significant difference.

Table 19. Analysis of variance - Period wise comparison of HI titres (105 days post-vaccination)

Source	df	SS	MSS	F
Between groups	2	886584.416	443292.208	14.533*
Error	37	1128575.584	30502.043	
Total	39	2015160.000		

CD for groups A & C = 142.565
CD for groups B & C = 140.459

Difference between means of groups A & C = 368.831*
Difference between means of groups B & C = 278.545*

* Denotes significant difference.

ILLUSTRATIONS

Fig. 1. Calibration curve for protein determination.

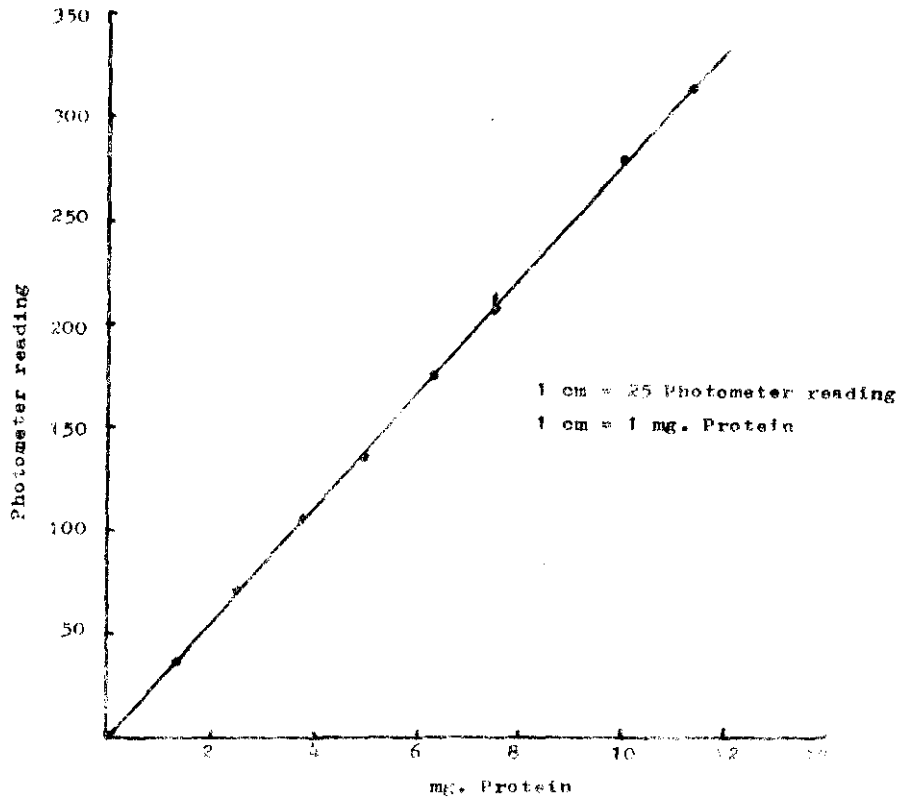
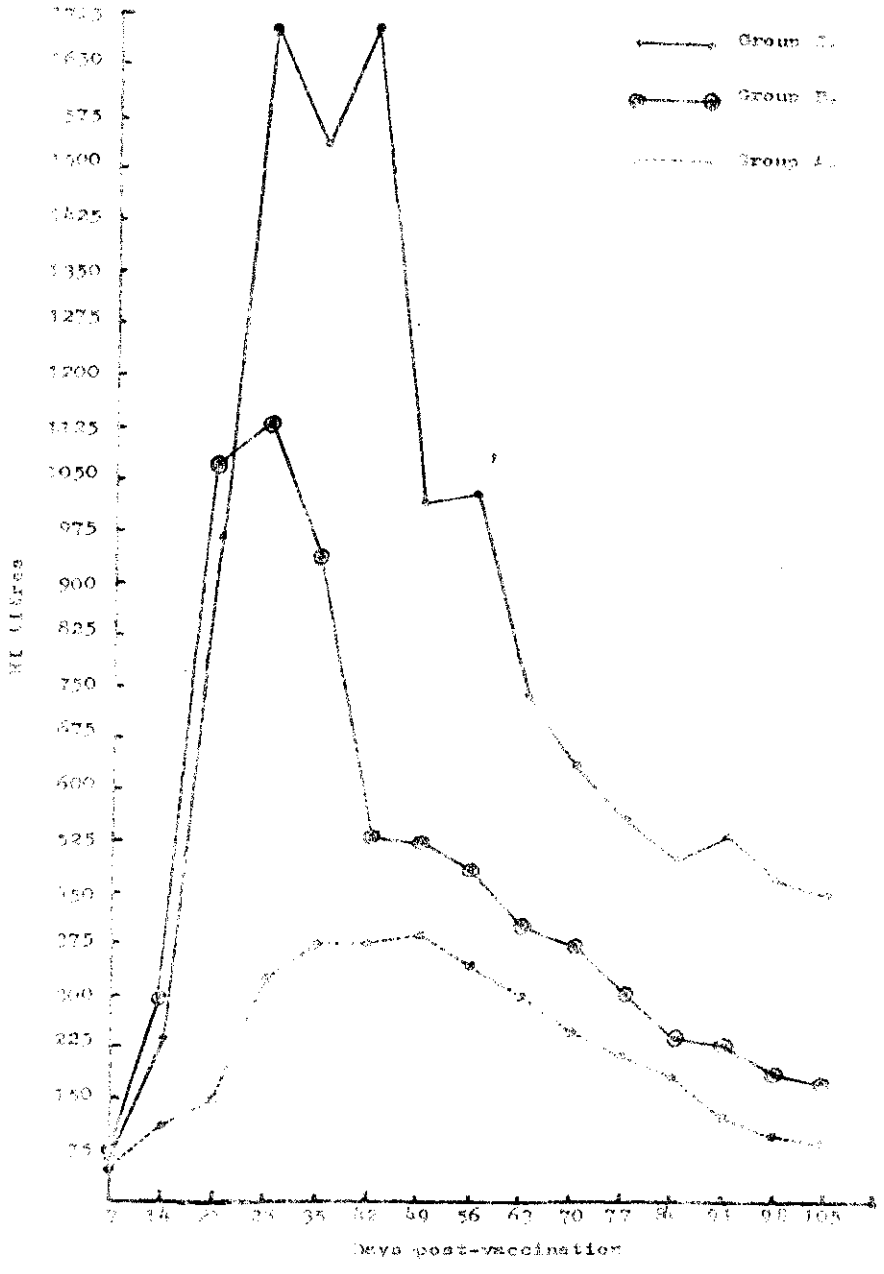


Fig. 2. Mean HI titres of groups A, B and C from seven to 105 days post-vaccination.



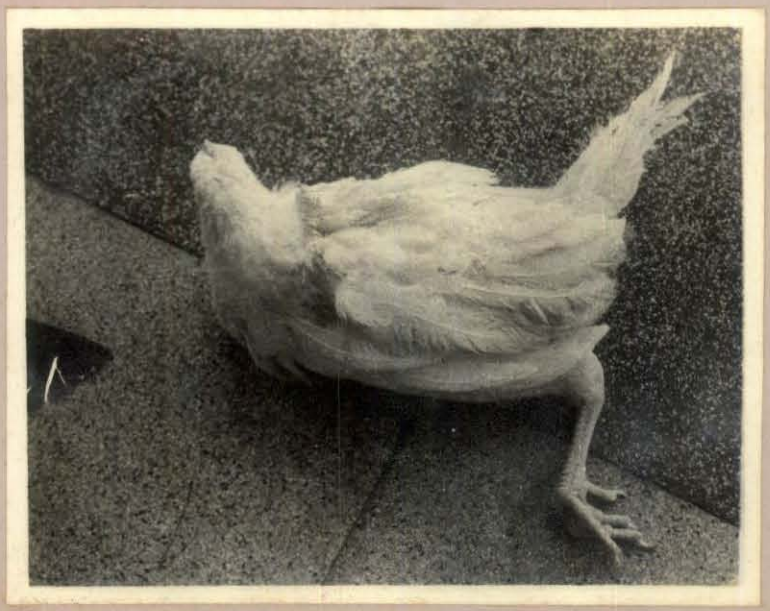


Plate 1. Showing the post-vaccinal reactions in a paralysed bird of group A.

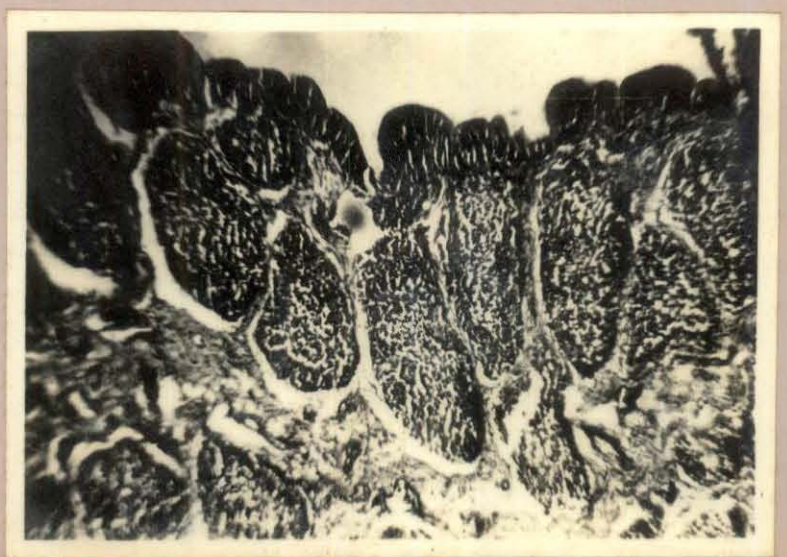


Plate 2. Showing the histopathological changes in the Bursa of Fabricius of a paralysed bird of group A.

Plate 3. Showing the histopathological changes in the Brain of a paralysed bird of group A.

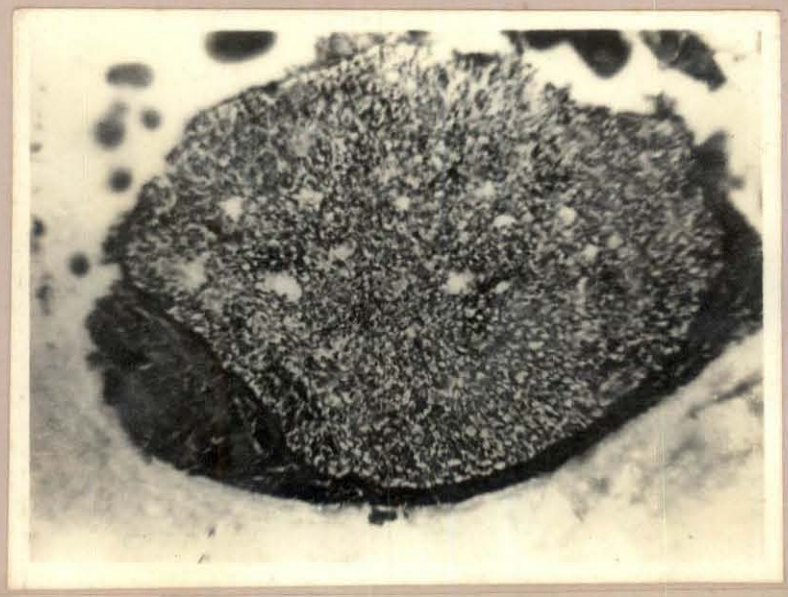


Plate 4. Showing the histopathological changes in the Sciatic nerve of a paralysed bird of group A.

DISCUSSION

DISCUSSION

The present investigation was intended to study the effect of A. galli infection on the immune response and post-vaccinal reactions in birds vaccinated with Ranikhet disease vaccine. From the perusal of the literature it appears that very little work seems to have been done on the effect of A. galli on Newcastle disease vaccination. Such a study will be more relevant in developing countries like India where the poultry are maintained under poor dietary and unhygienic conditions which will favour high prevalence of parasitism.

In this study, artificial infection of chicks with 1,000 and 1,500 ova of A. galli resulted in the development of, on an average, 37.4 and 82.6 worms respectively (Table 1). Baudet (1930) reported that the number of worms was not proportional to the condition of birds and that even a small number of worms might be responsible for weakened condition of chicken. Mocsy (1931) has opined that even the presence of four to five worms in chicken and 15 to 20 in young hens may prove fatal. However, during the preliminary assessment of the infective dose, only a single case of mortality could be observed

which had a heavy infection with 187 worms.

The symptoms observed in birds during the preliminary study were loss of apetite, droopiness, off feed, stunted growth, unthriftiness and diarrhoea. Similar symptoms have also been described by Sundaram et al. (1962), Seneviratna (1966), Khouri and Pande (1970), Amer et al. (1976) and Soulsby (1976). However, the symptoms such as droopy wings, leg weakness and partial paresis described by Deo (1964) and Seneviratna (1966) were not observed.

Haematological studies revealed a significant reduction in haemoglobin and erythrocyte count while the leukocyte count showed a significant increase as compared with the control (Tables 2 to 4). Eosinophilia was marked in both groups when compared with the control (Tables 6 to 8). This is in agreement with the findings of Georgekutty and D'souza (1967), Kaushik and Sen (1978) and Peter (1978).

The average serum protein content of A. galli infected groups were significantly lower than that of the control group. Dubinsky et al. (1976) and Tizard (1977) have also observed similar reduction in the level of serum protein in A. galli infected chicken. These reduction in the serum protein levels may probably be due to reduced intake of feed as the birds

were showing inanition and unthriftness. Moreover, it is also possible that the dietary protein was not fully available to the birds as they were harbouring a heavy worm infection.

The mean HI titres of groups A, B and C were 226.33, 466.33 and 808.12 respectively and these differences were statistically significant. The group A which was infected with 1,500 ova had the lowest mean HI titre and in group B where the infective dose was only 1,000 eggs, the mean HI titre was higher when compared to group A. Maximum mean HI titre was observed in group C which was not infected with A. galli. This indicated that ascaridiasis has defenite influence in reducing the immune response to Newcastle disease vaccination and that the HI titres differ at different levels of infection as it was observed that heavier the infection, lower the HI titre.

The mean peak HI titre of groups A, B and C were 388.57, 1130.67 and 1687.27 and these were observed during 49th, 28th and 28th day post-vaccination respectively. In the case of group A, the worms would have fully developed at the time of vaccination and since a period of 28 to 29 days are normally

required for the full development of the worm as observed in the preliminary studies and substantiated by the findings of Deo and Srivastava (1955), and Varghese (1966). Moreover, the number of worms developed in this group will be more as the birds in this group received 1,500 A. galli ova. These factors may be the probable causes for a low HI titre and for delay in reaching the mean peak HI titre. In group B the worms were likely to be immature, since they had reached only 14 days of age at the time of vaccination, and the inoculum was also low when compared to group A. In the light of these factors a higher mean HI titre could be attained earlier (28th day) when compared to group A. The mean peak HI titre was reached earlier in group B when compared to group A. This could be probably because the immature worms perhaps may not have much influence on the antibody production. In group C, the mean HI titre was the highest and also the peak was reached on the 28th day as it did not have the influence of either the immature or the adult worms.

This lowered mean HI titre, mean peak HI titre and the delay in reaching the peak observed between the groups A and B in comparison to C may probably

be due to the poor utilisation of protein by the birds, as it is indirectly reflected on the serum protein level. This impaired protein utilisation may be influenced more by the presence of adult worms when compared to immature as evinced by the difference in HI titre of groups A and B. Tizard (1977) has attributed imbalance of protein metabolism to be a cause of vaccination failure.

In group C the mean HI titre reached the peak on 28th day post-vaccination and remained almost stationary till 42nd day, and the HI titre declined gradually to 454.55 by 105th day post-vaccination. In group B although the HI titre reached the peak in the 28th day post-vaccination, it started declining rapidly till it reached a titre of 176.00 by 105th day post-vaccination. In group A the highest peak was reached by 49th day post-vaccination and the HI titre declined sharply when compared to group B and reached 85.71 by 105th days post-vaccination (Fig. 2). These differences in the rate of decline of HI titre in groups A, B and C may be due to the presence of worms. It was observed during the preliminary study that feeding of 1,500 and 1,000 ova resulted in the development on an average 82.6 and 37.4 worms respectively. Thus it

can be inferred that group A have almost double the worm burden when compared to group B which may be the probable reason for showing the sharp decline of HI titre and reaching almost less than half of the HI titre when compared to group B by the time it reached 105 days post-vaccination.

Post-vaccination reactions were observed in six birds (30 per cent) of group A and five birds (25 per cent) of group B. The reaction observed were mostly of the paralytic nature. The important symptoms observed were droopiness, paralysis of legs, wings and followed by mortality in all the birds. In the control (group C) none of the birds showed any reaction. Post-vaccinal reactions have been reported with 'Mukteswar' (R_2B) and 'Komarov' strains of Ranikhet disease vaccine by several workers (Seetharaman, 1960; Thorne and MacLeod, 1960; Hanson, 1972 and Tanwani and Malik, 1978). Seetharaman (1977) have noted that similar reactions occur in one to two per cent of the birds after vaccination without complete recovery. However, in the present study none of the birds in group C had any post-vaccinal reactions. The higher rate of post-vaccinal reactions (25 to 30 per cent) in group A and B can be attributed to the effect

of ascaridiasis. In support of this, FAO (1960), Seneviratne (1966) and Seetharaman (1977) have reported that if birds have a very heavy worm burden, the vaccination reactions may become more acute. Under field conditions where the incidence of worm infection may be comparatively high the chances of vaccination reaction may be more.

For assessing the resistance to Newcastle disease, two birds each from groups A and B (40 HI titres each) and two birds from group C showing lowest HI titres (160 and 320) were challenged. All the birds withstood the challenge. However, the birds of groups A and B showed mild form of the disease while none of the birds in the control group showed any symptoms. Danchev et al. (1978) and Peterson (1978) reported that the birds with HI titre of 1:40 or higher can withstand the challenge while those with 1:20 or less may be susceptible. The mild symptoms shown by the birds of groups A and B after challenge, although they had a titre of 1:40, may be due to the worm burden.

The results of the present study clearly indicated that the immune response of birds due to Newcastle disease vaccination is reduced by A. galli infection,

the magnitude of reduction being related to the severity of worm burden. Furthermore, it also revealed that post-vaccinal reactions are more in birds infected with A. galli. Ackert (1927), Pande, and Krishnamurthy (1959) and Georgekutty and D'souza (1967) have observed that vitamin A status of birds play an important role in the resistance to A. galli infection. During the present study the birds were given feed containing adequate quantity of vitamin A. In this context it is relevant to point out that, under conditions of poultry rearing practiced by the farmers where the vitamin A status of birds is likely to be low, there is increased possibility of higher worm burden than observed in this experiment and consequently the impact of poorer immune response as well as severe vaccination reactions are likely to be more.

Therefore, deworming of birds periodically should be a routine practice that could be suggested to farmers to improve the efficacy of Newcastle disease vaccination control programme.

SUMMARY

SUMMARY

The effect of A. galli infection on Newcastle disease vaccination was investigated under controlled conditions. White Leghorn chicken were utilised for the study. A total of 52 chicks of four weeks of age were grouped into A, B and C, with 20, 20 and 12 birds respectively. Groups A and B were infected with 1,500 and 1,000 ova of A. galli respectively and group C was left as uninfected control. The birds of group A were vaccinated at eight weeks of age while group B and C were vaccinated at six weeks of age with RDV'K' strain.

The parameters studied were, haematological changes estimated at one day prior to vaccination, 30, 60 and 90 days post-vaccination, post-vaccinal reactions, HI titres from seventh day to 105 days at weekly intervals and resistance to challenge with Newcastle disease virus.

The haematological changes observed were reduction in erythrocytes, haemoglobin, serum protein, heterophils and basophils and increase in leukocytes and eosinophils in the A. galli infected groups.

The post-vaccinal reactions were observed in 30 and 25 per cent of birds in group A and B respectively. These birds showed droopiness, unthriftiness, paralysis of legs and wings. All the birds which had these reactions died. On autopsy, the birds showed empty bowel, haemorrhagic enteritis, fluidity of intestine and worms. None of the birds in group C had any reaction.

The mean HI titres of groups A and B were found significantly lower than the control group C and this decrease in HI titres was noticed from 28th day post-vaccination. The mean peak HI titres of groups A, B and C were reached on 49th, 28th and 28th day post-vaccination respectively. The rate of decline of the HI titre after reaching the peak was comparatively rapid in groups A and B when compared to C. The lowered mean HI titre, the delay in reaching the mean peak HI titre and the rapid decline of the HI titre in groups A and B may be due to the presence of worms.

After challenge with virulent Newcastle disease virus, all the birds withstood the challenge. However, the birds in groups A and B developed a mild form of the disease and recovered.

The results of the study indicated the necessity of deworming of birds as a routine procedure in the control programme of Ranikhet disease under field conditions.

REFERENCES

REFERENCES

Ackert, J.E. (1927) Some intestinal worms of chickens and their control. Cited by Deo (1964).

*Ahmed, A.A.S., Sokkar, I.M.H. and Elsisi, M.A. (1965) Studies on Newcastle disease virus with the aid of agar gel diffusion technique. J.vet.Sci. 2 : 135 - 146.

Ajinkya, S.M., Surrashe, B.D. and Sardesh Pande, P.D. (1980) Breakdown in immunity to Ranikhet disease (Newcastle disease) with Infectious Bursal disease (Gumboro disease) in broiler chicks. Indian vet.J. 57 (4) : 265 - 269.

Alinassy, K. and Kakuk, T. (1976) Immunosuppressive effect of a naturally acquired subclinical bursal agent infection on Vaccination against Newcastle disease. Vet. Rec. 99 (22) : 435 - 437.

Allan, W.H. (1969) Response to tissue culture derived Newcastle disease vaccine in presence of maternal antibodies. Res. Vet. Sci. 10 (2) : 222 - 224.

Allan, W.H. and Gough, R.E. (1976) Comparison between Haemagglutination Inhibition and Complement Fixation tests for Newcastle disease. Res. vet. Sci. 20 (1) : 101 - 103.

*Amer, A.A., El-Amrousi, S. and El-Hammardy, H.Y. (1976) Trials with concurat and low doses of Mansonil in the treatment of poultry naturally and experimentally infected with Ascaridia galli. Veterinary Medical Review. (1) : 18-31.

Asplin, F.D. (1952) Immunisation against Newcastle disease with a virus of low virulence (strain F) and observations on subclinical infection in partially resistant fowls. Vet. Rec. 64: 245 - 249.

Baudet, E.A.R.F. (1930) Cited by Deo (1964).

Beard, C.W. and Max Brugh. (1975) Immunity to Newcastle disease. Am. J. vet. Res. 36 (4): 509 - 512.

Bhatia, H.M. (1960) Ranikhet disease. Animal Diseases and Pests. 1 : 45.

*Biondi, E. and Schiavo, A. (1965) Effect of some coccidiostats on production of Newcastle disease Haemagglutination inhibition antibodies. Acta med. vet. (11): 159 - 168.

Box, P.G. (1965) The influence of maternal antibody on vaccination against Newcastle disease. Vet. Rec. 77 (19): 246 - 250.

Brugh, M.Jr. (1977) Butylated hydroxy toluene protects chicken exposed to Newcastle disease virus. Science (Wash., D.C.) 197 (4310):1291 - 1292.

Burnet. (1942) Cited by Hanson, R.P. (1972).

Chang, P.W., Yates, V.J., Pendleton, J.A. and Flanagan, T.D. (1969) Immune response of chicken to six strains of Newcastle disease as measured by Haemagglutination inhibition test. Avian Dis. 13 (1): 46 - 52.

*Cherby, J. (1967) Application of Haemagglutination innibition test in diagnosis of Newcastle disease. Bull. Soc. Sci. vet. Lyon 69:309-336.

Chu, H.P. and Risk, J. (1972) Newcastle disease. A world poultry problem. World Animal Review. 2: 33 - 43.

Cooper, H. (1931) Cited by Seetharaman (1960).

Cunningham, C.H. (1966) A Laboratory guide in Virology. Burgess publishing Company, Minneapolis. 6th ed. pp. 47 - 60.

*Danchev, P., Semov, P. and Danchev, Y. (1978) Serological reactions for evaluating the immunity of Fowls to Newcastle disease virus. Veterinarnomeditisinski. Nauski. 15(4):80-92.

*da Silva, M.M. and Brada, W. (1968) Congenital immunity to Newcastle disease in chicks. Pesquisa. agropec. bras. 3 : 303 - 305.

Daubney, R. (1952) Cited by Seetharaman (1960).

Deo, P.G. (1964) Round worms of Poultry. I.C.A.R. New Delhi. pp. 16 - 48.

Deo, P.G. and Srivastava, H.D. (1955) Study on the biology and Life history of Ascaridia galli. Cited by Deo (1964).

*Devos, A.H., Viaene, N.J. and Spanoghe, L. (1976) Newcastle disease. Effect of vaccines and method of vaccination on circulating antibodies and on clinical resistance to infection. Vlaams. Diergeneesk. Kundig. Tijdschrft. 45 (12): 405 - 425.

*Donika, G.G. (1968) Role of immune fowl in the epidemiology of Newcastle disease. Dokl. Akad. Selkhoz. Nauk. (3): 38 - 40.

Doyle, T.M. (1927) Cited by Seetharaman (1960).

*Dubinsky, P., Leston, P. and Rybos, M. (1976) Effect of Ascaridia galli infection upon components of blood serum of the host fed with cereal nutrition. Biologia. Czechoslavakia. 31 (11): 829 - 839.

Dutt, S.C. (1950) Cited by Deo (1964).

Edwards, J.T. (1928) A new fowl disease. Cited by Lancaster (1966).

Fabricant, (1949) Cited by Bruner, D.W. and Gillespie, J.H. In Hagan's Infectious Diseases of Domestic Animals. Cornell University Press, London. 6th ed. pp. 1069.

FAO (1960) Report of the IV joint meeting on Animal Health held at Manila, Philippines. pp. 42.

Freeborn. (1923) Cited by Deo (1964).

Georgekutty, P.T. and D'souza, B.A. (1967) Effect of Methyridine, Piperazine citrate and a worm pill in Ascaridia galli infection. Indian vet. J. 44 (9): 751 - 759.

Giambrone, J.J. (1979) Effect of early Infectious Bursal disease virus infection on immunity to Newcastle disease in adult chickens. Poult. Sci. 58 (4): 794 - 798.

Giambrone, J.J., Edison, C.S., Page, R.K., Fletcher, O.J., Berger, B.O. and Kleven, S.H. (1976) Effect of Infectious Bursal agent on the response of chicken to Newcastle disease and Marek's disease vaccination. Avian. Dis. 20 (3): 534 - 544.

Gogoi, A.R. (1974) Occurance of Nematodes and Trematodes in the local Fowls in Assam. Kerala J. vet. Sci. 5 (2): 131 - 134.

Gordon, R.F. (1979) Poultry Diseases. English Language Book Society, London. pp. 85 - 90.

*Grillo Torrado, J.M., DE Muslera, B.P. and Perez Arrieta, A. (1967) Methods of controlling using inactivated vaccines against Newcastle disease by titration of neutralising antibodies. Revta. Investness. Agropec. Ser. 4(6): 65 - 80.

Hanson, R.P. (1972) Newcastle disease. In Diseases of Poultry. (Hofstad, M.S., Calnek, B.W., Hemboldt, C.F., Reid, W.M. and Yoder, H.W. -Ed). The Iowa state University Press, Ames, 6th ed. pp. 634 - 636.

Ikemi, M.M. (1971) Observations on the pathogenicity and pathology of Ascaridia galli. Parasitology. 63 : 169 - 179.

*Ileri, S.Z. (1956) The Roskin and Komarov Newcastle disease vaccines. Bull.OIE. 46 : 476 - 483.

Inchiosa, M.A. (1964) Direct biuret determination of total protein in tissue homogenate. J. Lab. Clin. Med. 61 : 319.

Iyer, S.G. and Dobson, N. (1940) Cited by Seetharaman (1960).

*Karrar, G. and Mustafa, E. (1964) Newcastle disease in Sudan. Bull OIE. 62 (1): 891 - 896.

Kaushik, R.K. and Sen, A.B. (1978) Studies on leukocyte response of chicks experimentally infected with Ascaridia galli. Indian J. Anim. Sci. 48 (6): 444 - 449.

Khouri, S.R. and Pande, B.P. (1970) Notes on the Histopathology in Ascaridia galli - An experimental study. Indian vet.J. 47(6): 472 - 474.

*Komarov, A. and Goldsmit, L. (1946) Preliminary observation on the modification of a strain of Newcastle disease virus by intracerebral passage through ducklings. Vet. J. 102 (7): 212 - 218.

*Komarov, A., Goldsmit, L. and Khane, Y. (1948) Duration of immunity following vaccination with 'Haifa' strain Newcastle disease vaccine. Refuah. vet. Palestine. (5): 50 - 75.

*Kraneveld, F.C. (1926) A poultry disease in Dutch East Indies. Ned. Indisch. Bl. Diergeneesk. 38 : 448 - 450.

Lancaster, J.E. (1966) Newcastle disease. A review of some of the literature published between 1926 and 1964. Canada Department. Agri. Monograph. (3): 1 - 134.

*Leutskya, Z.K. and Fais, D. (1977) Antibody synthesis stimulation by vitamin A in chicks. Biochin. Biophys. Acta. 475 (2): 216 - 217.

Luna, G.L. (1968) Manual of histologic staining methods of the Armed Forces Institute of Pathology. Mc Graw Hill Book Company, London. 3rd ed. pp. 1 - 37.

Manickam, R. and Gopalakrishnan, C.A. (1979) Breakdown of immunity in poultry. Poultry International. 17 (10): 194 - 195.

Markham, F.S., Cox, H.R. and Dottorff, C.A. (1954) Newcastle disease. A serologic study in vaccination and revaccination. Cornell vet. (44): 324 - 345.

*Marthedal, R.E., Velling, G. and Badstue, P.B. (1973) The application of Haemagglutination Inhibition test in the control of Newcastle disease. Bulletin de l' office International des Epizooties 79 (1/2): 51 - 58.

Matta, S.C. and Ahluwalia, S.S. (1979) Studies on the effect of Ascaridia galli infection on growth rates of chicks. Indian J. Poult. Sci. 14 (1): 37 - 38.

Methods for examination of Poultry Biologics. (1963) Publication No. 1038. National Academy of Science. National Research Council, Washington, D.S. 35 - 39.

Meulemans, G., Halen, P., Vindevogel, H. and Gauffaux, M. (1977) The immuno depressive effect of Infectious Bursal agent on vaccination against Newcastle disease. Res. vet. Sci. 22 (2): 222 - 224.

Mocsy, (1931) Cited by Deo (1964).

*Mohammed, M.A. (1980) Effect of subclinical infection of Eimeria tenella on the response of chickens to vaccination against Newcastle disease. Archiv fur Geflugelkunde. 44 (2): 47 - 49.

*Nambiar, K.T.K. (1961) Study on the Haematology of Domestic Fowl. M.Sc. Thesis submitted to the University of Madras.

Nathan, D.B., Heller, E.D. and Perek, M. (1977) The effect of starvation on Antibody production of chicks. Poult. Sci. 56 (5): 1468 - 1471.

National Commission on Agriculture. (1976) Report. Ministry of Agriculture and Irrigation. Government of India. Part VII: 472 - 473.

Nilakantan, P.R., Sakkubai, P.R. and Dhanda, M.R. (1960) Observation on the effects of immunisation of fowls with different vaccine strains of Ranikhet (Newcastle) disease virus. Indian vet. J. 37(10): 503 - 508.

*Pal, O., Zamora, N. and Polanco, R. (1970) Immunological experiments with Newcastle disease using Haemagglutination inhibition test. Revta Cubana cienc. vet. (1): 31 - 40.

Pande, P.C. and Krishnamurthy, D. (1959) Inter-relationship between hypovitaminosis A and Ascaridia galli infestation in poultry. Poult. Sci. 38: 13 - 25.

*Pavlicek, J. and Dykova, J. (1975) Experimental infection with Ascaridia galli in young chickens of different age. Acta veterinaria Brno. 44 (3): 223 - 233.

Peter, C.T. (1978) Studies on the use of Cashew nut shell liquid as an anthelmintic against ascaridiasis in domestic fowls. Annual Progress Report. Emeritus Scientist's Schome of the I.C.A.R: 3 - 14.

Peterson, C.H. (1973) Newcastle disease. In Servicemans Poultry Health Hand Book. pp. 83 - 86.

Punnose, K.T. and Rai, B.N. (1971) Serum Neutralisation tests in Primary Calf kidney Monolayers with Newcastle disease virus. Kerala J. vet. Sci. 2 (2): 147 - 155.

*Quaglio, G. and Lombardi, D. (1973) Interaction between vaccine virus and specific parental antibodies against Newcastle disease in chicks. Folia vet. Latina. 3 (3): 387 - 399.

Qureshi, S.H. (1950) Cited by Gogoi (1974).

Raggi, L.G. and Lee, G.G. (1960) Response of birds to intranasal vaccination with B_1 strain of Newcastle disease virus. Avian Dis. 4(2): 187 - 194.

Samuel, R., Padmanaban, V.D., Jayaraman, M.S., Masilomony, P.R. and Nachimuthu, K. (1978) Immunogenicity of CDF_{66} strain of Newcastle disease virus in comparison to 'F' and 'K' strain. Indian vet. J. 55 (11): 843 - 847.

Sastry, U.V., Kamath, G.R. and Chafoot, M.A. (1974) Cited by Gogoi (1974).

Seetharaman. C. (1960) The Ranikhet disease of Poultry. I.C.A.R. New Delhi. pp. 3 - 14.

Seetharaman, C. (1977) Ranikhet disease. In Hand Book of Animal Husbandry. I.C.A.R. New Delhi. pp. 332.

Seetharaman, C. and Nilakantan, P.R. (1951) Cited by Seetharaman (1960).

Seneviratna, P. (1966) Diseases of Poultry. Ed Seneviratna. pp. 36 - 37.

Sehrank, F. (1788) Cited by Deo (1964).

Singh, R. and Gurukripal. (1977) Outbreaks of Ranikhet disease in vaccinated poultry flock in Punjab. Virus isolation and serological studies. Indian J. Anim. Hlth. 16(2): 179 - 181.

Snedecor, G.W. and Cochran, G.W. (1967) Statistical methods. Oxford and IBH Publishing Company. 6th ed.

Soulsby, E.J.L. (1976) Helminths, Arthropods and Protozoa of Domesticated Animals. Bailliere Tindall and Cassel Ltd. London. pp. 167 - 168.

Sprehn, C. (1930) Cited by Deo (1964).

Sulzberger. (1929) Cited by Carpenter, P.L. (1965) Immunology and Serology. W.B. Saunders Company, Philadelphia. 2nd ed. pp. 121.

Sundaram R.K., Radhakrishnan, C.V. and Padmanabha Iyer, R. (1962) A note on the common parasitic helminths of fowls in Kerala. Kerala vet. J. 1 (1): 17 - 21.

Tanwani, S.K. and Malik, B.S. (1978) A comparative study of immune and antibody response of lentogenic strain CDF_{66} and other vaccine strains of Ranikhet disease virus. Indian vet. J. 55 (4): 267 - 268.

Thorne, A.L.C. and Mac Leod, A.J. (1960) The production and properties of Newcastle disease vaccine ('Komarov' strain) in Nigeria. Brit. vet. J. 116 (1) : 427 - 435.

Tizard, I.R. (1977) An Introduction to Veterinary Immunology. W.B. Saunders Company, Philadelphia. pp. 179 - 181.

*Toma, V., Giurgea, R. and Borsa, M. (1977) Thymobursal reactions under the immunosuppressive action of Cyclophosphamide. Zentralblatt fur veterinarmedizin. 24(B) (8): 656 - 661.

Valsala, K.V. (1968) Reproductive Pathology in the Hen. M.Sc. Thesis submitted to the University of Kerala.

Varghese, C.G. (1966) Studies on the common Nematodes in Poultry. M.Sc. Thesis. submitted to the University of Kerala.

Verma, R. (1981) Latest trends in Ranikhet disease. Poultry guide. 18 (6): 47 - 49.

Walker, T.R. and Farrell, D.J. (1976) Energy and Nitrogen Metabolism of Diseased chickens. Interaction of Ascaridia galli infestation and vitamin A status. Br. Poult. Sci. 17 (1): 63 - 77.

Winterfield, R.W. and Seadle, E.H. (1957) Newcastle disease immunisation studies II. The immune response of chicks vaccinated with Newcastle disease vaccine administred through drinking water. Poult. Sci. 36(1): 54 -64.

* Original not consulted.

EFFECT OF ASCARIDIA GALLI INFECTION ON NEWCASTLE DISEASE (RANIKHET DISEASE) VACCINATION

BY

V. VIKRAMAN

ABSTRACT OF A THESIS

Submitted in partial fulfilment of the requirement
for the degree

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences
Kerala Agricultural University

Department of Preventive Medicine
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
Mannuthy - Trichur

1981

ABSTRACT

Under controlled conditions, the effect of Ascaridiasis on Newcastle disease vaccination was investigated. Haematological studies revealed reduction in erythrocytes, haemoglobin, serum protein, heterophils and basophils and an increase in leukocytes and eosinophils in A. galli infected birds. Post-vaccinal reactions, mostly of a paralytic nature, were observed in 25 - 30 per cent of the infected birds. The immune response of birds to Newcastle disease as revealed by the HI titres, is reduced by A. galli infection, the magnitude of reduction being related to the severity of worm burden. Although, all the birds resisted challenge, A. galli infected birds developed a mild form of disease. The need for routine deworming of birds before vaccination is stressed.