PATHOLOGY OF THE REPRODUCTIVE SYSTEM IN DUCKS

By

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THESIS

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DECLARATION

I hereby declare that this thesis entitled PATHOLOGY OF THE REPRODUCTIVE SYSTEM IN DUCKS 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.

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CERTIFICATE

Certified that this thesis entitled PATHOLOGY OF THE REPRODUCTIVE SYSTEM IN DUCKS is a record of research work done independently by Sri. Jayakumar, P.M. 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.

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(Jayakumar, P.M.)

Dedicated to my beloved

parents and sisters

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Introduction

1. INTRODUCTION

Rearing of ducks is one of the important and popular occupations of farmers in the state of Kerala. The geographical location and the climatic profile are ideally suited for rearing ducks. Although, duck rearing has not developed as an industry in Kerala, there are very large number of farmers who earn their livelihood by maintaining ducks.

The ducks by nature are hardier than chicken and therefore, they are not prone to develop a large number of diseases. But the practice of rearing ducks carried out in Kerala is peculiar in that they are moved from place to place and are maintained on pasturing in paddy fields. This nomadic habit, therefore, exposes them to different micro environment and chances of getting infection are great and this perforce also contributes to the spread of infections. The duck farmer in Kerala did not consider disease problem in ducks as an important factor that hindered profitable duck farming, until the duck plague outbreak in Kerala in 1976. This was an eye opener to the farmers and scientists engaged in duck disease surveilance and control programmes. Over the years, the duck plague epidemic has been the only solitary instance in the history of duck farming in Kerala which caused considerable mortality of ducks in the state and threatened to choke the duck farming enterprise. Since this epidemic, considerable attention has been paid to the management of diseases of ducks.

Apart from the loss in flock due to infectious diseases, the loss by way of impaired growth and loss of production due to nonspecific factors are of paramount consideration in profitable farming and such losses although they may appear small in respect of individual holdings, would certainly contribute to severe national loss due to wasting of food and labour on unproductive stock. The ducks are mainly reared for production of eggs and any disorder that affects the reproductive system will have a great bearing on the production potential of the duck. In order to ensure persistent and maximum production in the flock it is imperative to investigate into the disorders affecting the reproductive system in order to understand the prevalence, nature and significance of these disorders. Eventhough, the more acute and generalised infections and epidemics which cause damage to the reproductive system can be recognised by the clinical manifestations and by bacteriological examinations, conditions which are slow and insidious in their onset and are without detectable clinical manifestations pass unnoticed for a long time thereby causing impaired production as well as permitting cryptic spread of such diseases. This group of diseases continues to be a poorly attended field of investigation and research in India and abroad.

Although, systematic investigations have been carried out on the pathology of the reproductive organs in chicken, no planned investigations have been undertaken to study the

disorders encountered in the reproductive system of ducks.

Aflatoxin is a potent hepatotoxin in large doses and in low doses it causes varying degree of degenerative and necroblotic changes in various organ systems. The adverse biological effects of this toxin have more relevance particularly in the case of ducks which have been demonstrated to be very susceptible to this toxin. Because of the favourable agroclimatic conditions in Kerala, fungal growth in feed commodities is very common and the feeds of ducks are often contaminated with aflatoxin. Besides this, investigations undertaken in the Department of Pathology have also shown that Ochratoxin, another toxic fungal metabolite is also present in many of the feed samples. The undesirable effect of these mycotoxins on the growth and production of the ducks is bound to be tremendous, particularly in low doses. Since the mycotoxins are regularly consumed by the ducks along with the feed in low doses it was decided to undertake an experimental study to assess the effects of aflatoxin and ochratoxin on the reproductive organs of ducks. The results obtained have been documented and analysed. It is presumed that the observations made on the reproductive disorders of ducks will go a long way to make it possible to advocate effective methods to combat various reproductive disorders and this would help to promote better egg production and enhance the profit of the farmers engaged in rearing of ducks.

Review of Literature

2.0. REVIEW OF LITERATURE

2.1. Pathology of the female reproductive system in chicken

2.1.1. Functional right ovary and oviduct.

Kaupp (1932) reported the presence of functional ovary and oviduct on the right side along with the presence of functional left ovary and oviduct in a hen. Morphological changes in gross appearance of the persistent right oviduct were reported. Cystic remnants of the right mullerian duct were reported by several workers (Willier, 1927; Webster, 1940; Blount, 1949; Van Trenhoven, 1957; Domm, 1964).

2.1.2. Atrophy and Hypoplasia.

Valsala and Sivadas (1971) encountered atrophy and hypoplasia of the left ovary and left oviduct in 7.5 % of the hens autopsied. Eighty-eight per cent of the cases of hypoplasia and atrophy were noticed in birds between six months and one year old. While the rest appeared in the older birds.

2.1.3. Impaction.

Impaction of the oviduct and egg bound were probably the most frequently described clinical conditions of the female reproductive system in chicken. Das and Biswal (1948), Peckham (1965), Valsala and Sivadas (1970) and Keymer (1960) described this condition in chicken. In these cases, the oviduct was occluded by masses of inspissated yolk, coagulated albumen or by the presence of fully formed egg in its lower portions. A fully formed egg lodged in the lower end of the

oviduct superimposed by inspissated material in the rest of the oviduct, was also observed. They even spilled over into the peritoneal cavity. It was more frequent in the older laying hens than in others. Cross-bred birds were reported to be more prone to develop egg bound than the other breeds. Clinically these birds were observed to adopt the egg laying posture and strained in an attempt to discharge the egg into the oviduct. At autopsy, one or more eggs in various stages of formation were seen in the oviduct.

2.1.4. Internal layers.

Internal laying was reported by Valsala and Sivadas (1970) in four hens out of 570 autopsies conducted. Thin eggs of varying shape and size were recovered from the abdominal cavity of one bird. Three of the eggs were fully formed and had hard shells, while the others were soft shelled.

2.1.5. Cystic conditions.

Valsala and Sivadas (1970) reported various cystic conditions of the right oviduct. The cyst differed in shape and size, were borne on narrow stalks, thin walled and usually distended with clear watery fluid which in some instances appeared cloudy. Multiple discontinuous cysts were also observed, appearing as blind saccular structures connected by intervening greyish white bands. Such cysts did not manifest any luminal communications with one another. They also encountered Parovarian cysts, as solitary structures which

occurred in the mesosalpinx of the left oviduct towards the region of the infundibulum. Haemorrhagic cysts of the ovary were red cyst like structures borne on twisted stalks. Associated with the stalks there were moderately distended and tortuous vessels. The fluid content of the cysts was deeply haemorrhagic and turbid, sometimes with clots of blood included. Necrotic flakes were adherent to the cyst wall. The cyst contained bluish pink staining material mixed with erythrocytes.

2.1.6. Bacterial diseases.

The reproductive system may be involved in many of the bacterial infections affecting birds. These conditions may either be acute infection causing sudden mortality without any detectable manifestations or chronic infections with localisation of infection in the reproductive organs.

In chronic cases of fowl cholera in birds, Thorp et al. (1931) observed ruptured yolk sac, ovarian abscess formation and accumulation of cheesy material with peritonitis.

Among the specific bacterial diseases of poultry, involvement of the reproductive system was reported to be common in Salmonellosis. Dexit (1962) and Singh (1967) observed that the ovary and the oviduct manifested pronounced changes in chronic pullorum disease. According to them discoloration and changes in the shape and consistency of the ova were very perceptible in the carrier hen. Besides being misshapen,

discoloured or cystic, the ova developed also long stalks instead of the normal short attachments.

Stubb (1965) reported involvement of the ovary and oviduct in Fowl Plague. These were in the nature of severe hyperaemia of the larger follicles and greyish exudation into the oviduct, the wall of which was edematous.

Valsala and Sivadas (1971) recovered Escherichia intermedia and Alkaligens faecalis from the oviduct of five birds with salpingitis. They observed severe diffuse congestion of the ovary and turbid, blood tinged exudate in the abdominal cavity. They induced salpingitis experimentally in progesterone treated birds on subsequent infection with culture of Escherichia intermedia. Sharma and Joshi (1983) studied the lesions in the reproductive organs experimentally induced by Escherichia coli. The gross lesions were swollen dark red ovaries, mis-shapen and congested ova and inflammed oviduct. Microscopic lesions were congestion, mild haemorrhages, fibrinous exudate and foci of lymphoid and plasma cells.

2.1.7. Viral infections.

Reports on viral diseases having direct bearing on the reproductive organs were very few. Among the viral specific diseases, New Castle Disease produced pathological changes in the ovary and oviduct. Abnormalities of the egg like thin and imperfect shell were reported by Clugg and Muller (1951) and attributed to the malformation of the shell producing portions of the oviduct.

In experimental studies with the New Castle Disease virus, Biswal and Morril (1954) observed marked decrease in the shell weight, and shell thickness of the egg in the birds. The lesions in the oviduct were characteristic of a mild inflammatory reaction with pronounced involvement of the albumen secreting portion. Yates et al. (1954) reported egg drop syndrome in chicken characterised by hepatitis, aplastic anaemia, haemorrhages, mild respiratory disease and a decrease in egg production.

Infectious Bronchitis, a viral respiratory disease, might affect chicken of all ages and chicken under two weeks of age. The virus was known to cause permanent damage to the oviduct which subsequently lead to conditions like soft shelled eggs or to a decrease in egg production (Hofstad, 1965). Maiti et al. (1985) reported the isolation of infectious bronchitis virus from the ovaries and oviduct of hens with a history of drop in egg production.

2.1.8. Parasitic infections.

Parasites of the oviduct and the ovary are rare when compared to the parasitic conditions of the gastro-intestinal tract in birds. The fluke Prosthogonimus belonging to the order Plagiorchidae was observed in the oviduct and bursa of Fabricius of chicken, duck and other avian species (Price, 1965). Valsala (1968) reported the incidence of Prosthogonimus infection of the oviduct in chicken. Affected birds went off production, laid thin shelled eggs or eggs without shell and

became emaciated and anaemic. Fibrinous peritonitis with adhesion and fibrino-purulent salpingitis with rupture of the organ and discharge of its contents into the abdominal cavity also were seen.

2.1.9. Protozoan infections.

The haemorrhagic disease (Bangkok Haemorrhagic Disease) of chicken characterised by diffuse cyst formation in the different organs of the body was first described by Campbell (1954) in Bangkok. Subsequently it was reported from India by Sivadas et al. (1965) and Valsala (1968). Minute whitish glistening translucent globular cysts of uniform size were observed in all the organs. The mucosa of the oviduct was practically showered with cysts many of which appeared haemorrhagic. The lesions were more pronounced in the lower portions of the oviduct. However, moderate number of cysts were seen in the ovary.

2.1.10. Chlamydial infection.

Cystic ovaries, egg peritonitis and fibrosis of the oviduct were observed by Rao (1965) in acute ornithosis.

2.1.11. Neoplastic conditions.

Jackson (1936) and Narayana et al. (1966) reported ovarian teratoma in chicken. The growths were yellowish to pearly white in appearance, rounded and varied in size from 1-3 cm in diameter.

Mayor (1968) observed that the majority of the tumour occurring in the fowl arise from the ovary. He observed firm, pink, cauliflower like growth and often there was ascites in the bird and secondary implantations were frequently observed. Sharma and Singh (1968) reported neoplasm of the ovary and oviduct in 10.5% of their material. Out of 20 neoplasms examined seven were granulosa cell tumour, 11 were leukosis complex, one, a leiomyoma and one, a fibroleiomyoma. According to Valsala (1968) the most frequent cause of death among the adult hen was neoplasms of the reproductive organs. Avian Leucosis Complex with involvement of the ovary and oviduct was recorded in the University Poultry Farm, Mannuthy, and this constituted nearly 70% of the cases. Neoplasms of the reproductive system encountered were leiomyoma, adenocarcinoma, cystadenoma and hemangioma (Valsala, 1968).

2.1.12. Salpingoperitonitis.

This condition seen in laying hens appears to be prevalent all over the world, where intensive system of breeding and rearing are in vogue. Acute and chronic manifestations of the disease were reported. Salpingoperitonitis was reported to be a complication of an initial salpingitis or oophoritis. Salpingoperitonitis has been recognised as an important if not the commonest cause for loss of production and for mortality among adult laying stock (Lindgren, 1956 and Valsala, 1968). They attributed high level of oestrogen production in the body as a factor influencing the onset of this disease.

2.1.13. Oophoritis and Salpingitis.

Singh et al. (1977) examined 1246 laying hens for the evidence of reproductive disorders. They found that 27.52% of the birds had pathological changes in the reproductive system and this included egg peritonitis, salpingitis and oophoritis.

Batra and Singh (1978) examined 2180 adult hens. They found oophoritis, salpingitis, impaction of oviduct, cystic oviduct and egg bound conditions in 375 birds. Major reproductive problems in chicken and turkey were low egg production, poor egg shell quality, low fertility and low hatchability (Opel, 1979). Keymer (1980) recorded oophoritis, salpingitis and ruptured oviduct in poultry. The disorders were frequent in domestic fowl than in non-domesticated species.

2.2. Pathology of female reproductive system in ducks

Published data on the incidence and pathology of the reproductive system of ducks are only few.

2.2.1. Parasitic infections.

Macy (1934) described cessation, decreased egg production, formation of soft-shelled egg and manifestation of egg peritonitis as a pathogenic effect of Prosthogonimus infection. Disturbances in egg production, discharge of calcareous substance, and prolapse of uterus were reported in Prosthogonimus infection in the oviduct of ducks (Sreekumaran, 1968).

2.2.2. Neoplastic conditions.

Rao et al. (1980) reported the incidence of papillary adenocarcinoma and cystadenoma of the ovary in ducks.

2.2.3. Miscellaneous disorders.

Bhowmik (1983) indicated that the main reason for mortality in adult ducks was egg peritonitis (11.53%) followed by non-specific enteritis (9.88%), post-vaccinal paralysis (7.54%) and impaction of oviduct (2.54 %).

2.3. Pathology of the male reproductive system in chicken

Batra and Singh(1978) recorded hypoplastic and neoplastic testes in four out of 64 male birds which were subjected for post-mortem examination. Teratoma arising from the left testicle in a Kadaknath breed of poultry was reported by Raote (1986).

No reports are available describing the pathological features encountered in the testis of ducks.

2.4. Effect of aflatoxin on the reproductive system

2.4.1. Chicken.

Sims et al. (1970) stated that dietary aflatoxin caused decline of egg production in laying hens. Garlich et al. (1973) hypothesised that the reduced quantities of yolk precursors were preferentially channelled to ova already comitted to maturation. Therefore, the decline in egg production was delayed because, aflatoxin inhibited the commitment of ova to maturation rather than affecting the maturation

process itself. In broiler breeders aflatoxin depressed fatty acid synthesis (Donaldson et al., 1972) and impaired lipid transport from the liver to the blood (Tung et al., 1973). By feeding dietary aflatoxin at the rate of 20 µg/g diet for four weeks, it was observed that although the birds exhibited signs of aflatoxicosis like decrease in body weight, serum total protein and serum total lipid, increased liver weight and liver fat, there was no significant effect on semen volume, sperm count, DNA, RNA and protein content of sperm, testicular histology or weight of testis (Wyatt et al., 1973). Jacobson and Wiseman (1974) showed that aflatoxin fed to broiler breeders was transferred to the egg yolk and white.

Briggs et al. (1974) concluded that aflatoxin did not affect the reproductive system of the mature male by the generally accepted mechanism of inhibition of DNA dependent RNA polymerase since the DNA, RNA and protein content of the sperm were unaffected. Culvenor (1974) described that the maximum permissible dose of aflatoxin in chicken was 0.20 ppm (200 ppb). The pathological changes caused by aflatoxin were characterised by deterioration in condition, decrease in growth, fall in egg production, subcutaneous haemorrhage and death (Butler, 1974).

Aflatoxin was shown to be detrimental to hatchability in broiler breeders (Howarth and Wyatt, 1976) and they concluded that the rapid effect of aflatoxin on hatchability was due to a sudden transfer of aflatoxin or a potent breakdown product

into the egg. Experimentally aflatoxin B (8.1 ppm) and G (1.0 ppm) were incorporated into the feed of five laying hens and in mature cocks for three weeks (Hafez et al., 1978). Egg laying ceased during the whole period. Histopathology showed follicular atresia of ovary but testicles were unaffected. Sharlin et al. (1980) observed that aflatoxin added to the feed at the rate of 20 µg/g diet for five weeks in mature White Leghorn males, resulted in decreased semen volume and testis weight and a disruption of germinal epithelium. But it had no effect on per cent fertile eggs or per cent hatch of fertile eggs. The decline in semen volume was preceeded by decrease in body weight and decrease in feed consumption. White Leghorn males appeared to be more susceptible to aflatoxin than broiler males. Feeding of 200 µg of aflatoxin to male chicks from the day old upto 35 days resulted in increased serum alkaline phosphatase, enlarged congested liver and atrophied testis. The seminiferous tubules were less well developed and smaller. There was more connective tissue between them and there was no histopathological evidence of spermatogenesis (Mohiddin, 1982). Mahipal and Kaushik (1983) described the pathological conditions including reduced feed consumption, reduction in growth, decreased production and carcinogenicity in the liver of birds. They observed that if the feed contained aflatoxin more than 20 ppb, the health and productivity of poultry flocks were severely affected. Chicken fed diet containing groundnut meal was more affected by addition of

aflatoxin B_1 at the dose rate of 50-400 µg/kg feed than those fed fish meal (Ostrowski Meissner, 1984). Jegadeesh et al. (1986) reported that aflatoxin fed at 20 ppm of the diet adversely affected semen characteristics in White Leghorn cocks when fed for eight weeks. Such adverse effects were not seen in the cocks fed ten ppm of aflatoxin in the diet. Wolnak et al. (1986) encountered pale liver, enlarged and haemorrhagic ovaries which were significantly smaller than those from the control hens. The ovary contained only small ova in hen fed a diet which contained 3310 µg/kg aflatoxin B_1 and 1680 µg/kg aflatoxin B_2.

2.4.2. Japanese quail.

Dietary aflatoxin caused a delayed onset in the decline of egg production in Japanese quail (Sawhney et al., 1973). Doerr and Ottinger (1980) fed aflatoxin (10 µg/g diet) to Japanese quail from 1-3 and 2-4 weeks of age. After two weeks of feeding toxin, body weight and testes weights were approximately 30% less than the controls. Serum testosterone level in both treatment groups was non-significantly different from controls at five weeks of age. However, in males fed aflatoxin from 2-4 weeks of age, the testosterone level was depressed by 50% when compared to values at six weeks of age. Among the females fed aflatoxin, follicular development was greatly reduced in both treatment groups even at six weeks of age. It was concluded that sexual maturity in birds fed aflatoxin from 2-4 weeks of age was delayed longer than birds fed

aflatoxin from 1-3 weeks of age, and females were more sensitive to the residual effects of aflatoxin than males.

In a relative study, Ottinger and Deorr (1980) observed that an expected increase in testosterone level and testicular weight was delayed longer among male Japanese quails fed aflatoxin (10 µg/g diet) from 2-4 weeks of age than among quails fed aflatoxin from 1-3 weeks of age. Compared to controls, the delay in the onset of sexual behaviour was the same for both groups. Blankford et al. (1981) reported retarded growth, lowered body weight and significant depression of gonadal body weight in mixed sex juvenile Japanese quail, when fed 10 ppm of aflatoxin for 14-28 days.

2.4.3. Ducks.

The maximum permissible dose of aflatoxin in the duckling was reported to be 0.03 ppm (Culvenor, 1974). Mukit and Kwatra (1978) recorded a natural case of aflatoxicosis in Khaki Campbel ducks in Assam, characterised by a moderate reduction in haemoglobin concentration, total erythrocyte count and a moderate leukocytosis due to an increase in the circulating heterophils. Abdullahs and Lee (1981) fed starter pellet containing aflatoxin at the rate of 80-100 µg/kg of feed and this resulted in nervous symptoms, drop in egg production and death in a flock of 2000 ducks. Jin et al. (1983) reported heavy loss among Beijing ducks in a duck farm in China, where the cause was feeding of mouldy maize which contained more than 100 µg of aflatoxin per kg. The symptoms

seen in ducklings were nervous signs, diarrhoea and retarded growth and in older ducks, diarrhoea, emaciation, drop in egg production, occasional deaths in breeders. In ducklings, the prominent histological features were toxic hepatitis with biliary hyperplasia, and in breeders cirrhosis and carcinoma of the liver. Replacement of deteriorated maize with fresh stock stopped the loss. In ducks addition of aflatoxin B_1 (40-400 µg/kg feed) in diets containing groundnut meal gave more toxic effects like reduction, in growth and protein utilisation and liver damage, than in diets with fish meal (Ostrowski-Meissner, 1904).

2.5. Mechanism of action of aflatoxin

Aflatoxin's primary action disrupted carbohydrate and lipid metabolism and inhibited protein synthesis (Hsieh, 1979). These mechanisms cannot explain all the effects of aflatoxin in the mature broiler breeder or White Leghorn male nor account for the differences in response between these two types of birds. A review of literature revealed three other explanations for the effects of aflatoxin on the mature male fowl.

2.5.1. Aflatoxicosis, a nutritional deficiency.

Mature broiler breeders were more resistant than White Leghorns since they were 2.5 times heavier and had 2.5 times more crude body fat than mature White Leghorn males (Mitchell et al., 1926; 1931) and therefore were tolerant to decreased food consumption. Parker and McSpadden (1943) reported

decreased semen volumes and fertility in Rhode Island Reds, after an 18% loss in body weight due to restricted feed consumption. In a study using paired feeding to examine the effects on essential fatty acid deficiency on mature White Leghorn males showed that reduced comb size, testis size and pituitary gonadotropin levels were caused by a decrease in appetite and energy intake rather than the deficiency per se (Engster et al., 1978). A reduction in pituitary gonadotropins would explain the reduced testis size and semen volume observed in aflatoxin treated White Leghorns. Furthermore, a decrease in body weight was linked to lowered plasma testosterone levels by Wilson et al. (1979). They observed a 53% decrease in circulating testosterone after an 18% loss in body weight in mature White Leghorns fed a 2% protein diet. The primary evidence to explain the effect of aflatoxin on nutrition was the significant decrease in feed consumption and concomitant loss in body weight observed in aflatoxin treated White Leghorns (Sharlin et al., 1980).

2.5.2. Aflatoxin - an anti androgen.

Structural similarity between aflatoxin and steroid hormones was first noted by Williams and Rabin (1969). In a later study, Rabin et al. (1970) concluded that aflatoxin can compete with sex steroid hormones for binding sites on the endoplasmic reticulum of rat liver cells. More recently, Patterson and Roberts (1972) showed that androstenedione, a testosterone precursor, competitively inhibited the in vitro

reduction of aflatoxin B_1 to aflatoxicol in the avian liver. According to them certain enzymatic systems could not distinguish sex steroids from aflatoxin. Therefore, a structural similarity existed. This they opined, raised the possibility that aflatoxin could act as an anti-androgen. Anti-androgens affected the action of testosterone by interfering with the uptake of testosterone by target cells, intracellular metabolism, or binding of metabolites to the cytoplasmic steroid receptor complex (Mainwaring, 1977). The ability of aflatoxin, or its metabolites, to cross the blood - testis barrier has not been investigated.

2.5.3. Inhibition of steroidogenesis - A secondary effect of aflatoxin.

Leutinizing Hormone was shown to stimulate the conversion of cholesterol to pregnenolone (Sulimovici and Boyd, 1969). Eik-Nes (1970) stated that the male gonad was capable of synthesising cholesterol therefore, a decrease in cholesterol precursors, namely acetate occurred before steroid synthesis was impaired. Since androgen levels influenced metabolic machinery in the testes, the presence of androgen was more important than the availability of steroid precursors. Precursor levels and the rate of steroidogenesis were controlled by androgens. Wyatt et al. (1973) noted a 3-fold increase in liver lipid and a decrease in serum total lipid in broiler breeder males fed aflatoxin and suggested that lipid transport from the liver was impaired unexpectedly. No significant differences in the serum cholesterol between treated and

control groups was observed. Since cholesterol was a precursor to steroid hormones, any interference with cholesterol metabolism affected steroid levels. Accepting the hypothesis that the primary effect of aflatoxin was a nutritional deficiency, any changes in levels of androgen precursors were attributed to a decrease in *LH* and a corresponding drop in androgen caused by an inadequate diet (Sharlin et al., 1980).

2.6. Effect of ochratoxin on the reproductive system

2.6.1. Chicken.

Huff et al. (1980) observed that the simultaneous presence of ochratoxin and aflatoxin in poultry feeds at a level of 2 ppm was found to exert synergic toxic effects. Although, the growth inhibition and increase in liver weights were the same, the tan yellow colour of the liver, characteristic of aflatoxicosis was masked or lost in the interaction group, besides affecting the carcase quality due to the intestinal ruptures causing economic loss to the industry (Warren and Hamilton, 1980). Kubena et al. (1983) reported significant reduction in body weight and feed efficiency, when day old male broiler chicks were fed with diet containing 3 ppm of ochratoxin A. Feeding of ochratoxin at 0, 0.25, 0.5, 1 and 2 mg/kg feed to hens for 12 weeks resulted in lowering of percentage of hen day egg production. Between 8-12 weeks, there was significant reduction of egg weight. Percentage of blood and meat spots in the egg at eight weeks were markedly increased at 0.5 to 2 mg/kg level. Tohala (1983) and Huff et al. (1984) observed

that the effect of ochratoxin A persisted longer than aflatoxin. Dwiveda and Burns (1985) indicated a reduction in the size of the thymus and bursa and this was associated with reduction in the population of lymphoid cells, and increase in size of the liver, proventriculus and kidney. Maximum changes were seen in the proximal convoluted tubules and most of these showed the presence of hyaline bodies in the lumen.

2.6.2. Ducks.

Bodnarchuk and Kaspruk (1984) observed 42% mortality in ducks after an illness for 2 to 6 days due to the presence of ochratoxin A in the feed. Haemorrhage in various tissues, catarrhal gastritis and enteritis were the lesions seen.

Materials and Methods

3.0. MATERIALS AND METHODS

3.1. Survey studies

retrospective survey on the various disease conditions prevalent in ducks in Kerala State was undertaken based on the records maintained at the Government Duck Farm, Niranam. The data recorded during a 11 year period from 1975-1985 at the duck farm, Niranam were collected and analysed. The diseases encountered were classified and the percentage of mortality due to various diseases was estimated. Based on the analysis of the data documented an assessment of the disease situation prevalent was made. Statistical analysis was carried out to reveal the causes of mortality, yearly incidence of diseases and nature of diseases prevalent (Steel and Torrie, 1960).

3.2. Studies on spontaneous disorders of the reproductive system

3.2.1. Source of material.

One hundred and sixty-five females and fifteen male culled Khaki Campbel ducks were brought from the duck farm, Niranam for the study.

Ducks brought for post-mortem examination at the Department of Pathology, College of Veterinary and Animal Sciences, Mannuthy, from various parts of the State and from the University Poultry Farm, Mannuthy were also included for the study.

3.2.2. Method of examination.

The ducks were sacrificed by decapitation and exsanguination. Autopsy was performed as per the protocol prescribed (SIDA, 1984). The gross lesions encountered were recorded. The reproductive organs were dissected out for further investigation.

3.2.3. Collection of materials.

The ovary was collected separately and the salpinx and uterus as one piece. They were weighed. The organs were examined in detail exposing the salpinx and uterus and gross lesions if any were recorded. Representative samples of tissues from the ovary and different parts of the oviduct (isthmus, infundibulum, magnum and uterus) were preserved in 10% formalin for histopathological examination.

3.2.4. Histopathological studies.

Tissues collected as mentioned above were processed by routine paraffin embedding technique (Armed Forces of Institute of Pathology, 1968). Paraffin sections cut at 4 μ thickness were stained routinely with Haematoxylin and Eosin method of Harris (Bancroft and Cook, 1984). Sections were also stained with P.A.S., Van Gieson's and Acid Fast, whereever required (Bancroft and Cook, 1984).

3.3. Experimental study

3.3.1. Pathology of the reproductive system in experimental aflatoxicosis.

3.3.1.1. Experimental ducks.

Twelve, healthy male, 3 to 4 month old cross-bred ducks were purchased from a local farmer. They were managed according to the standard recommendations. The feed was tested and found free of aflatoxin and ochratoxin. The ducks were randomly divided into two groups, each group contained six ducks.

3.3.1.2. Source of the toxin.

Crystalline aflatoxin B_1 was obtained from Makor Chemicals Ltd., Israel.

3.3.1.3. Mode of administration and dose schedule.

Group I contained six ducks and aflatoxin B_1 was administered at the rate of 25 µg per bird daily for a period of three months. The toxin was dissolved in 2.5 ml of propylene glycol and injected into the crop.

Group II contained six ducks and they served as controls. They were given 2.5 ml of propylene glycol as injection into the crop daily for a period of three months.

3.3.1.4. Observations made.

Daily feed intake was recorded. The body weight was recorded once in every fortnight. Clinical symptoms, if any were recorded. Detailed post mortem examination was conducted on the ducks died during the course of the experiment.

3.3.1.5. Mode of collection of tissues.

The ducks were sacrificed at the end of the third month, by decapitation and exsanguination. Autopsy was performed as per the protocol described (SIDA, 1984). The gross lesions were recorded and the reproductive organs were weighed.

Representative samples of tissues from the testis were preserved in 10% formalin. They were processed by routine paraffin embedding technique (Armed Forces of Institute of Pathology, 1968). Paraffin sections cut at 4 μ thickness were stained routinely with Haematoxylin and Eosin method of Harris (Bancroft and Cook, 1984).

3.3.1.6. Analysis of data.

Numerical data were analysed according to the procedures described by Steel and Torrie (1960).

3.3.2. Pathology of the reproductive system in experimental ochratoxicosis

3.3.2.1. Experimental ducks.

Twelve, healthy male cross-bred ducks aged 3-4 months were purchased from a local farmer. They were maintained according to the standard recommendations. The feed was tested and found free of aflatoxin and ochratoxin. The ducks were randomly divided into two groups of six each.

3.3.2.2. Source of the toxin.

Ochratoxin A was obtained from Makor Chemicals Ltd., Israel.

3.3.2.3. Mode of administration and dose schedule.

Group I, contained six ducks.Ochratoxin was administered daily at the rate of 25 µg per bird for a period of three months. The toxin was injected, in 2.5 ml of 4.24% sodium-bicarbonate solution into the crop.

3.3.2.4. Observations made.

Daily feed intake was recorded. A record of body weight was noted once in every fortnight. Clinical symptoms if any manifested were also observed.

3.3.2.5. Method of collection of tissues.

The ducks were sacrificed at the end of the third month, by decapitation and exsanguination. Autopsy was performed as per the protocol described (SIDR., 1984). The gross lesions were recorded and the testes were weighed.

Representative samples of tissues from the testis were preserved in 10% formalin. They were processed by routine paraffin embedding technique (Armed Forces of Institute of Pathology, 1968). Paraffin sections cut at 4 µ thickness were stained routinely with Haematoxylin and Eosin method of Harris (Bancroft and Cook, 1984).

3.3.2.6. Analysis of data.

Numerical data were analysed according to the procedures described by Steel and Torrie (1960).

RESULTS

4.1. Survey studies

A detailed survey on the various disease conditions prevalent in ducks was conducted based on the records maintained at the Government Duck Farm, Miranam. During the period from 1975 to 1985, 8746 ducks died in the farm due to various diseases. The disease conditions encountered were classified as hepatosis, hepatitis, enteritis, tuberculosis and miscellaneous disorders. The latter category included pulmonary edema, omphalitis and transport stress. The overall profile of the disorders encountered has been graphically represented in Fig.1.

A high incidence of hepatosis was seen in the year 1975, 1976, 1977, 1978, 1979, 1984 and 1985. The highest incidence was recorded (90.19%) in the year 1985. During the year 1986, 194 female ducks were brought from the Miranam farm and sacrificed. All these had lesions of hepatosis and this was confirmed by histological examination. However, in the26 male ducks, the liver lesions were minimal or absent. The prevalence of enteritis was very high (36.86%) in 1980, when compared to all other years. Tuberculosis was first reported in the year 1975 (2.08%). The highest incidence was recorded in the year 1980 (12.39%). The miscellaneous disorders were found to be high (41.5%) in 1981. The incidence of hepatitis (66.18%) was high in 1982.

HEPATOSIS
ENTERITIS
HEPATITIS
TUBERCULOSIS
MISCELLANEOUS
PERCENTAGE
1975
1976
1977
1978
1979
1980
1981
1982
1983
1984
1985
86.64
5.08
0.39
2.09
5.80
50.71
12.77
2.13
8.51
25.89
49.09
11.36
1.40
2.81
35.34
64.05
9.86
1.16
6.67
18.26
57.51
23.72
1.19
0.59
16.99
2.72
36.86
19.34
2.39
28.70
0.71
17.45
31.37
8.96
41.51
0.60
3.38
66.18
3.86
9.28
85.13
4.81
0.34
0.45
9.28
87.36
1.00
0.72
1.20
10.59
90.19
1.00
7.40
0.52
1.81

The prevalence of reproductive disorders encountered in males and females is shown in table 1.

Table 1. Prevalence of reproductive disorders

Female:

Total number of ducks examined = 194

Total number in which the reproductive disorders were recorded = 55

Disorders encountered	Number of cases	Prevalence rate	Percentage of the reproductive disorders
Hypoplasia of the left ovary and oviduct	11	5.67	20
Impaction of the oviduct	3	1.546	5.45
Haemorrhagic cyst in the ovary	4	2.06	7.27
Salpingoperitonitis	6	3.09	10.9
Oophoritis	27	13.92	49.09
Cystic right oviduct	1	0.5	1.82
Mycotic salpingitis	1	0.5	1.82
Tuberculous salpingitis	2	1.03	3.63

Male:

Total number of ducks examined = 26

Total number in which reproductive disorders were recorded = 3

Atrophic testis	2	7.69	66.6
Seminoma	1	3.84	33.3

4.2. Disorders of the female reproductive system

4.2.1. Hypoplasia of the left ovary and oviduct.

Gross pathology.

Out of 194 female ducks examined, in 10 instances left ovary and oviduct were poorly developed. In all these instances the birds were fully grown adult ducks. But the reproductive organs were very small in size (Fig.2). Mature follicles were not seen in any of these cases. Scattered small greyish white granules constituted the only evidence of the ovarian bunch. Although, these ducks were over 10 months in age, the ovary grossly appeared like that of 2-3 month old duck. The left oviduct was poorly developed and appeared as a thin cord in all. The different regions were poorly defined, the lumen was narrow. There was absence of any activity in any region of the oviduct and the muscular portion of the wall of the oviduct appeared thin in all instances.

Histopathology.

Histologically the ovary in these birds was characterised by the presence of a few follicles lined by a single layer of more or less flattened cells which were occasionally discontinuous. The cells were low cuboidal and had shrunken nucleus. The follicles contained pale thin colloid. There were focal areas of interstitial fibrosis as well as scattered accumulation of mononuclear cells.

In the oviduct, the mucosa was thrown into minute folds.

There was no glandular activity in the secretory tubular glands which were by themselves very few and scattered in distribution. Another characteristic feature was focal fibrosis of the submucosa and atrophy of the muscular layer.

4.2.2. Impaction of the oviduct.

Gross pathology.

Impaction of the oviduct was observed in three out of 194 adult female ducks examined. The location and the degree of impaction varied with different cases. The impaction of the oviduct was in the magnum and uterine portion in two instances and in the infundibulum in one case and was characterised by the presence of inspissated caseous, cheesy or moderately hard onion peal shaped yellowish white material in the lumen (Fig.3). The wall of the oviduct was thin and the vessels of the oviduct were moderately and diffusely engorged. The ovary was active and contained three to four mature follicles and numerous developing follicles.

Histopathology.

Histopathological examination of the affected portion showed focal areas of degeneration, necrosis and desquamation of epithelial lining. There was severe infiltration of the submucosa with lymphocytes and heterophils. There was glandular hyperplasia in focal areas. The inflammatory reaction was seen extending into the muscular coat causing partial destruction of the muscular layers.

4.2.3. Haemorrhagic cyst in the ovary.

Gross pathology.

This condition was characterised by cysts containing masses of blood clot or by the presence of large blood clots in the ovary. It was observed in four Khaki Campbell ducks. In one duck, a brownish grey stalk of about 1 cm in length was seen to arise from the middle of the ovary, and its distal end ended in a large dark brown cyst which contained moderate quantity of dark brown turbid fluid, brownish yellow necrotic masses and dark red blood clots. The necrotic material close to the cyst was adherent to it. The oviduct was moderately developed and was very pale.

In other cases the ovary was found to contain four to five dark brown glistening cysts of 1-2 cm in diameter with short, narrow stalk. The cyst wall was smooth and brownish and contained dark brown free masses of lysed blood and caseous material. Besides the cysts, the ovary contained mature follicles and numerous developing follicles. The oviduct was well developed and normal in size.

Histopathology.

The wall of the haemorrhagic cyst was composed of thick wavy bands of collagenous tissue; and was lined by a single layer of columnar epithelium which showed degeneration in focal areas. Externally a serosal lining was evident. Distributed profusely in the collagenous layer were numerous

capillaries of varying dimensions, all of which were engorged with blood. The cavity of the cyst contained free erythrocytes, partially organised blood and purple staining mass of yolk material (Fig.4).

4.2.4. Salpingoperitonitis.

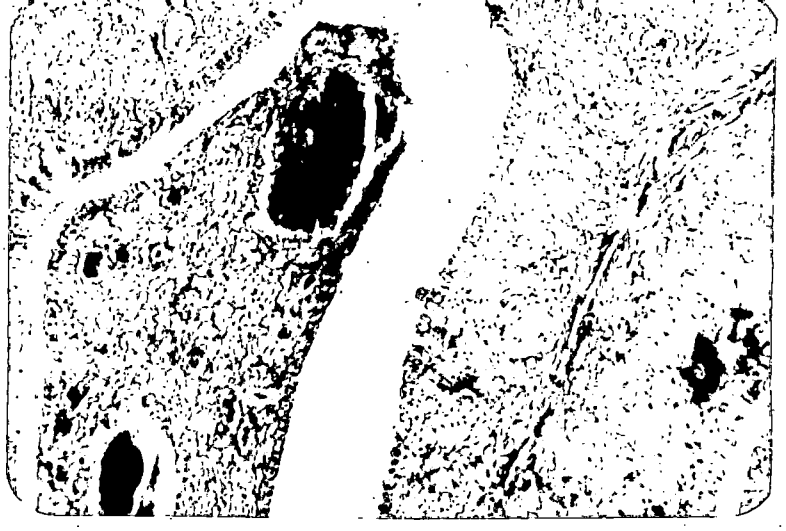
Grosa pathology.

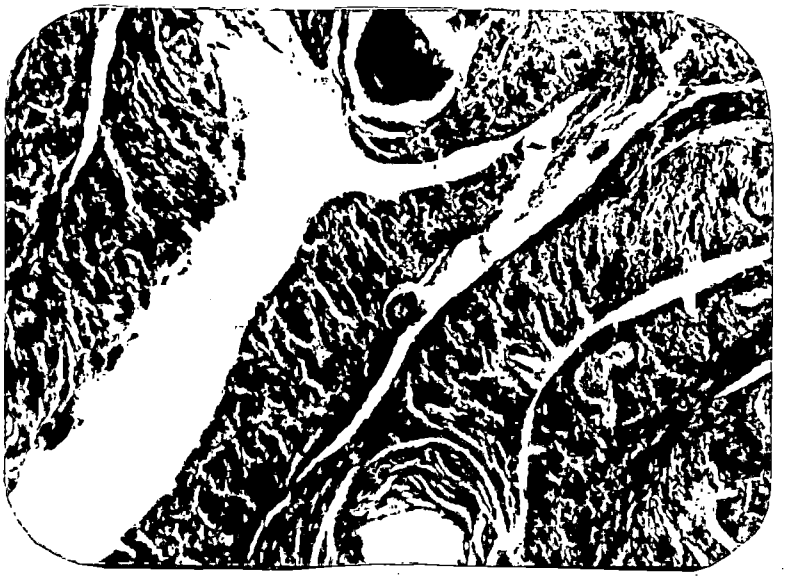
Out of 194 ducks examined six ducks showed salpingitis. In all the cases salpingitis was associated with peritonitis as a complex group of inflammatory reaction. The constant finding in these ducks was a pronounced deposit of peritoneal fat which appeared abnormal in amount. The intestinal serosa and mesentric folds in between the intestinal loops were covered by moderately thick deposits of fibrinous material which also extended over the peritoneal wall. Moderate diffuse hyperaemia of the peritoneal and mesenteric vessels was observed.

The serosa of the oviduct was hyperaemic and the vessels in the mesosalpinx were engorged. Moderate quantity of fibrinous plastic exudate often covered the outer surface of the oviduct. The mucosa of the oviduct invariably showed severe diffuse hyperaemia.

Histopathology.

In the oviduct, pronounced changes were observed in the infundibulum and magnum. Deciliation, degeneration and desquamation of the epithelial lining were marked. The lumen





contained homogenous pink staining exudate in which there were numerous heterophils and mononuclear cells. There was severe edema of the submucosa and fibrinous exudate caused distension of the submucosal layer and this resulted in the separation of muscular layer of the oviduct. Focal or diffuse areas of necrosis of the secreting glands were a constant feature. Infiltration of the submucosa with heterophils and mononuclear cells was diffuse and severe (Fig.5). and in some instances cystic dilatation of the glands in the submucosa was observed (Fig.6). The cellular reaction was very meagre and uniformly spread over the submucous layer. The capillary engorgement in the submucosal layer was a constant finding and in some instances fibrin thrombi appeared to occlude the lumen of the capillaries in the oviduct. There was proliferation of smooth muscle fibres.

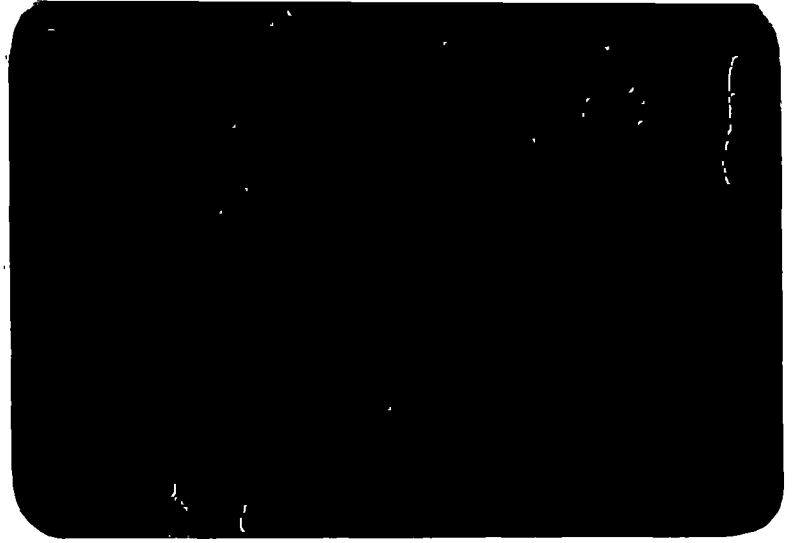
4.2.5. Mycotic salpingitis.

Gross pathology.

Out of 194 ducks examined, one duck had mycotic salpingitis and the organism was identified as Aspergillus fumigatus on cultural examination. Numerous greyish white nodules of 2-3 mm in diameter were seen scattered throughout the serosa of the oviduct (Fig.7). Similar nodules were seen in the lungs also. The oviduct was poorly developed and different regions were poorly defined and the lumen was narrow.

Histopathology.

Histopathological examination of the oviduct revealed the



presence of multifocal granulomas involving the serosa and mucosa. The central area of caseation necrosis was surrounded by infiltration of few mononuclear cells and a mantle of foreign body giant cells. The whole structure was covered by fibroblasts (Fig.8). PAS staining revealed the presence of septate hyphae of the fungus in the central caseous material in the submucosa (Fig.9 and Fig.10).

4.2.6. Tuberculous salpingitis.

Gross pathology.

Among the 18 cases of tuberculosis recorded while examining 194 ducks in two instances the oviduct was seen involved. There were many millet sized, moderately hard nodules scattered in the mesosalpinx in the region of the magnum of the oviduct. Similar nodules were distributed in the lungs and peritoneum also. Cut sections of these nodules revealed yellowish caseous necrotic mass at the centre.

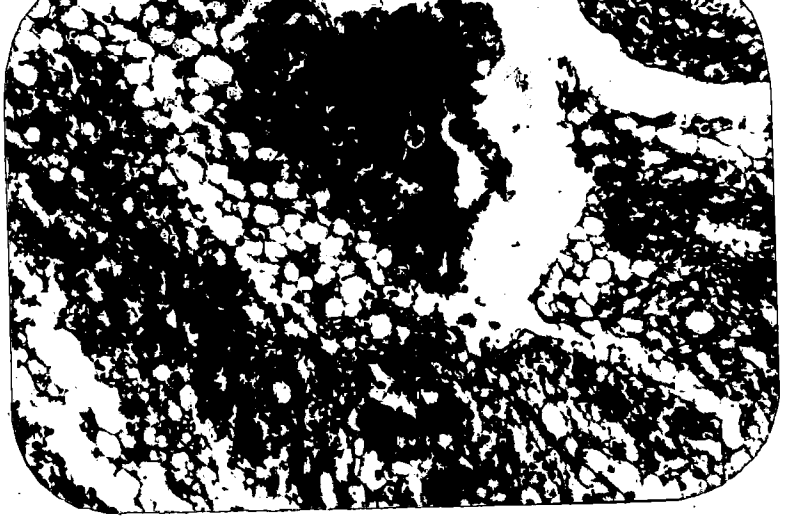
Histopathology.

Histologically the tubercle showed typical structure of a granuloma. A central caseous mass was surrounded by a zone of lymphocytes and mononuclear cells. The whole structure was encapsulated by connective tissue of varying thickness. Acid fast staining revealed numerous acid fast bacilli in the lesion.

4.2.7. Oophoritis.

Gross pathology.

Oophoritis was recorded in 27 cases out of 194 ducks



examined. The follicles were covered with thin greyish white sheets of necrotic material, which in certain instances caused the ovarian follicles to be matted together. The surface of the follicle showed diffuse congestion, with the vessels standing out prominently. Besides the necrotic tissue covering the follicles there was often a thick film of pale yellow cheesy inspissated mass of yolk enveloping the ovary as well as layered over the adjacent peritoneal surface. A few ova were misshapen, greenish or grey in colour and appeared to be fused. Some follicles were haemorrhagic. The discoloured misshapen follicles contained small amounts of viscid brownish yellow opaque fluid.

Histopathology.

In the ovary there was diffuse accumulation of a purple staining homogenous exudate. This infiltrated into the ovarian parenchyma. Severe engorgement of the capillaries was often seen, together with focal areas of haemorrhage (Fig.11). There was focal degeneration and desquamation of the ovarian germinal epithelium which in certain places in the subcortical region had proceeded to necrosis. In the surrounding area there was mononuclear and heterophilic infiltration. The primary follicles in some instances underwent necrosis. The cellular reaction in the ovary was chiefly one of plasma cells and heterophils. Moderate fibrosis and lymphoid infiltration, sclerosis of the blood vessels and proliferation of smooth muscles were occasionally observed in few cases (Fig.12 and Fig.13).

4.2.8. Cystic right oviduct.

Gross pathology.

Only one instance of cystic right oviduct was observed out of 194 ducks examined. The cyst was 6 cm in diameter and contained a clear serous fluid. The left ovary and oviduct were normal in appearance and size. The cloacal portion of the cyst served as a tubular stalk which had no communication with the cloaca (Fig.14). The left ovary and oviduct were normal in appearance and size.

Histopathology.

The cyst wall was composed of bands of smooth muscle fibres which had a tendency for longitudinal arrangement. Lining the cavity of the cyst was a single layer of ciliated columnar epithelium. In between the columnar epithelial cells there were a few scattered goblet cells which contained small amount of mucinous material. The cavity of the cyst contained homogenous eosinophilic material. The cyst wall was covered by serous coat.

4.3. Disorders of the male reproductive system

Out of 26 male ducks examined, the conditions encountered were atrophic testis and seminoma.

4.3.1. Atrophic testis.

Gross pathology.

Testes were very small greyish white and firm (Fig.15). The weight of the testes is shown in table 2.

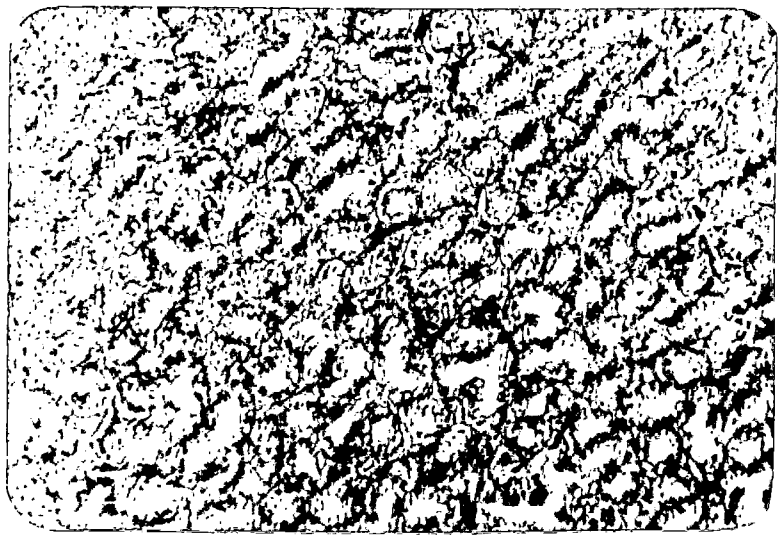


Table 2. Reproductive disorders in males

Live weight (g)	Weight of testis (g)	Percentage of body weight	Gross lesion
1170	3	0.256	Atrophic testis
1430	4	0.279	Atrophic testis
2240	280	12.5	Seminoma

Histopathology.

The tubules were narrow and were lined by a single layer of flattened atrophied cells which did not show any mitotic activity, denoting cessation of spermatogenesis. There was absence of spermatozoa within the lumen. There was increased peritubular connective tissue (Fig.16).

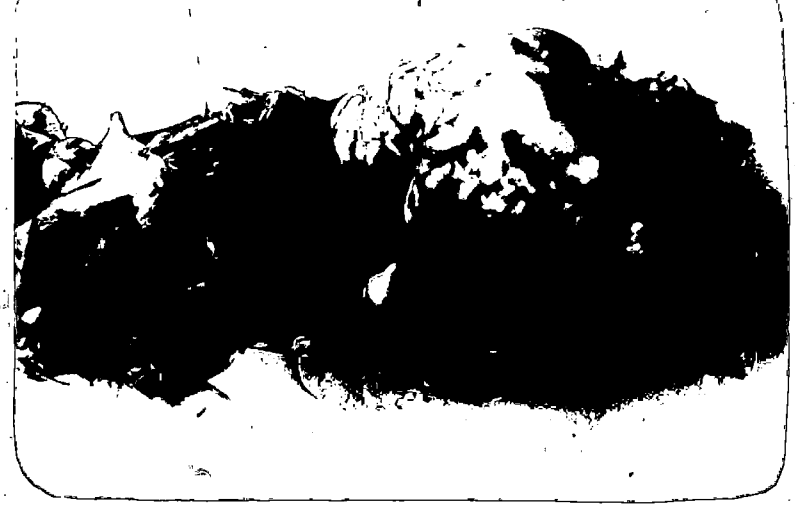
6.3.2. Seminoma.

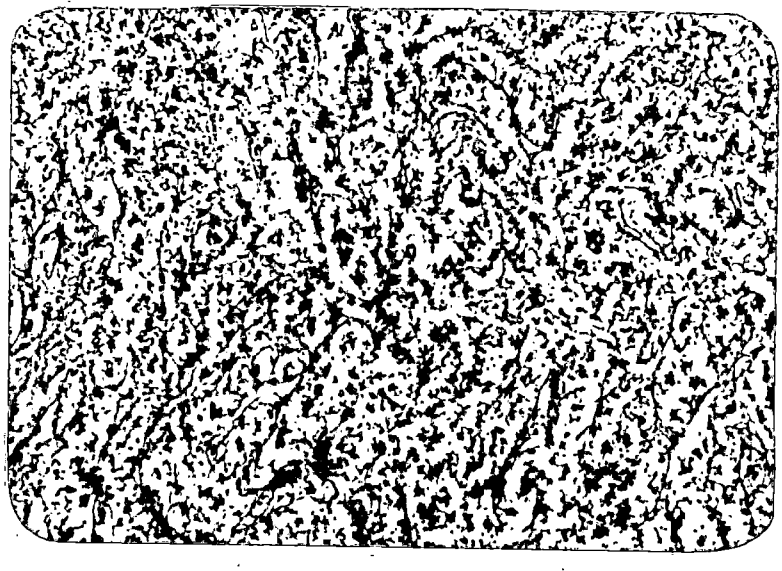
Gross pathology.

This was recorded in one duck. The tumour involved both the testis. Both the testis together weighed 280 g. There was a large greyish white mass involving the left testis and this almost filled up the peritoneal cavity. The surface was coarsely granular, and was moderately firm to cut. The growth in the right testis was bigger than the left one (Fig.17) and weighed 210 g.

Histopathology.

There were many seminiferous tubules of varying sizes with large, closely packed cells. The cells were round and





polygonal in shape and had a central roughly spherical hyperchromatic nucleus and a variable but abundant amount of eosinophilic cytoplasm. In certain areas the cells were not arranged in any defined architectural pattern, and did not produce an obvious intracellular matrix (Fig.18). Fine trabeculae divided the mass of cells into compartments. Scattered foci of lymphocytes were present in the midst of the neoplastic cells. Tubules did not show any sperms but some of them contained degenerated, desquamated epithelial cells.

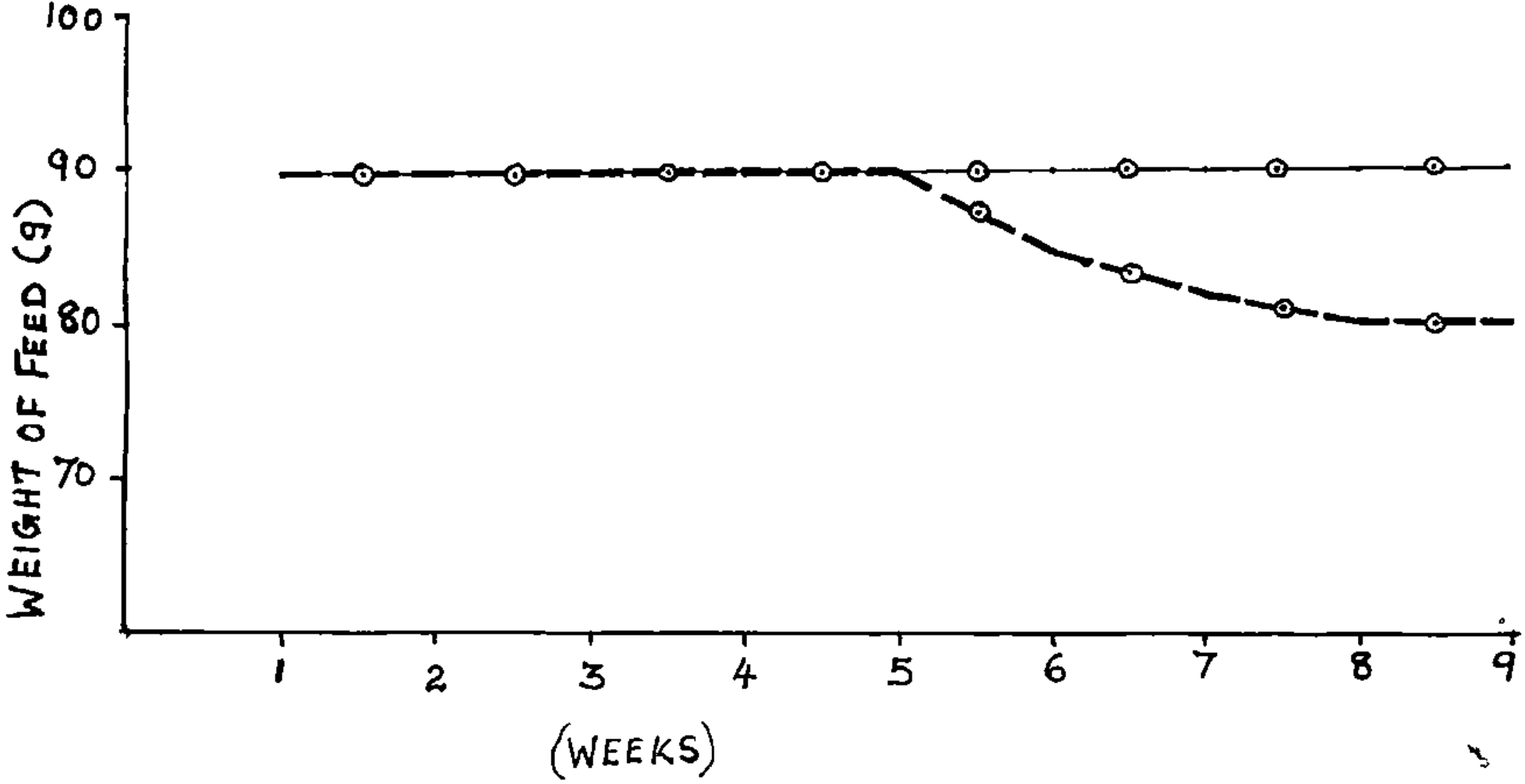
4.4. Experimental aflatoxicosis

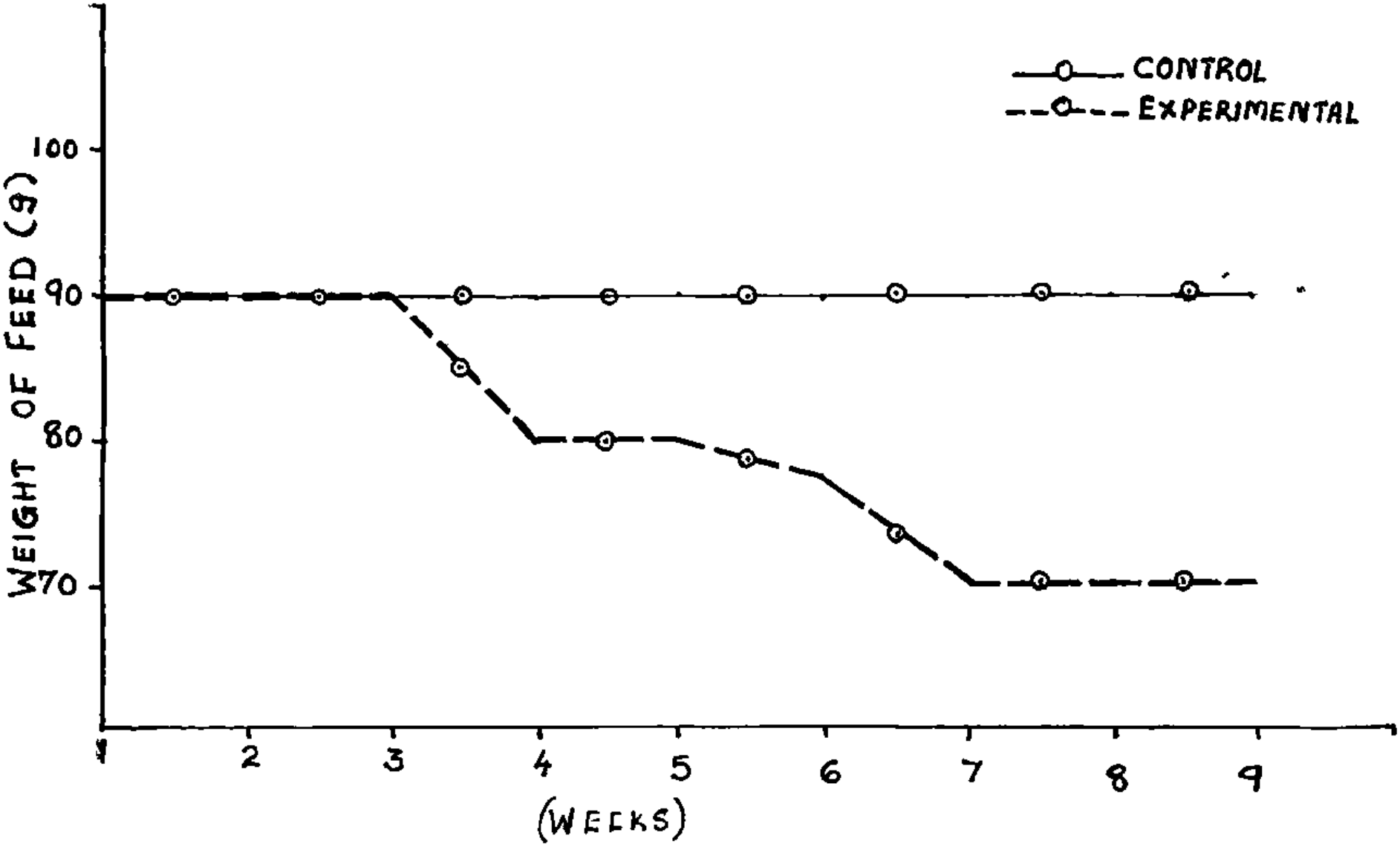
Group I, contained six ducks. Aflatoxin B_1 was administered at the rate of 25 µg per bird daily for a period of three months. The toxin was injected into the crop in 2.5 ml of propylene glycol.

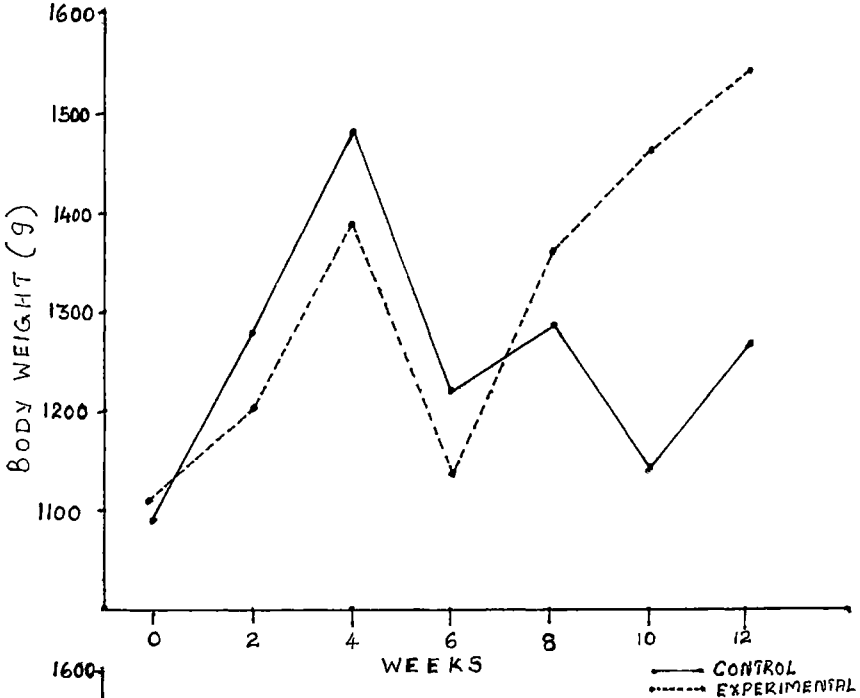
Group II, contained six ducks, and they served as control and were given 2.5 ml of propylene glycol as injection into the crop daily for a period of three months.

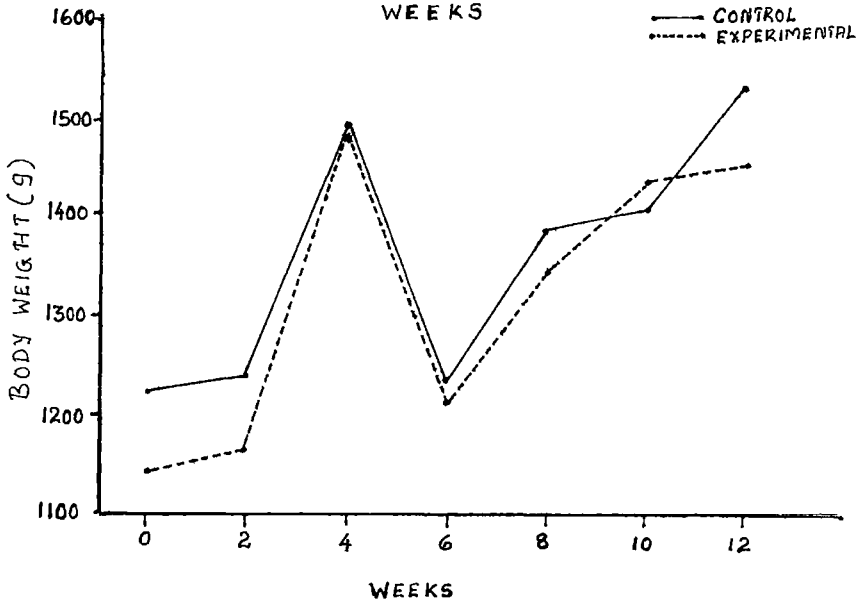
4.4.1. Feed intake.

The data on the feed intake of ducks during the experimental period are given in Fig.19. From the third week onwards considerable reduction in feed intake was observed in the experimental group, whereas in the control ducks, the feed intake was stationary.


100
90
80
70
WEIGHT OF FEED (g)
1
2
3
4
5
6
7
8
9
(WEEKS)


CONTROL
EXPERIMENTAL
WEIGHT OF FEED (g)
100
90
80
70
2
3
4
5
6
7
8
9
(WEEKS)


1600
1500
1400
1300
1200
1100
BODY WEIGHT (g)
0
2
4
6
8
10
12
WEEKS
CONTROL
EXPERIMENTAL


1600
1500
1400
1300
1200
1100
BODY WEIGHT (g)
0
2
4
6
8
10
12
WEEKS

4.4.2. Weight gain.

The data on the body weight of ducks during the experimental period are given in Fig.20. All the birds gained body weight till the fourth week. But the rate of weight gain was more in the control birds. Subsequently there was a steady reduction in the body weight in both the groups towards the sixth week. From the sixth week onwards, there was appreciable increase in body weight in the control group.

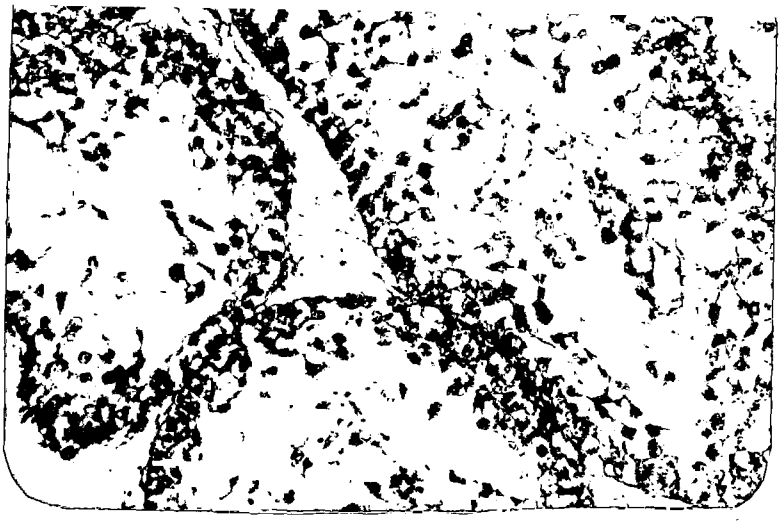
Statistical analysis employing 't' test to find out the effect of toxin on the body weight showed that the effect was not significant ($P<0.01$).

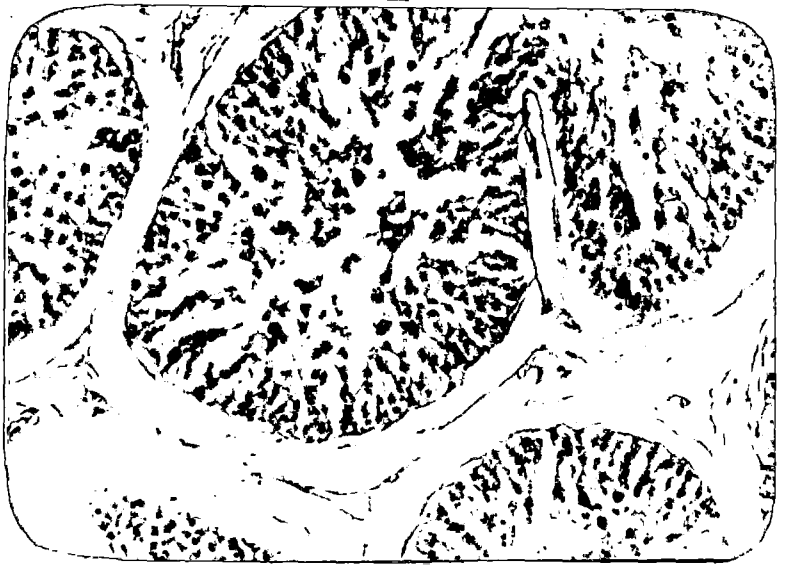
4.4.3. Clinical symptoms.

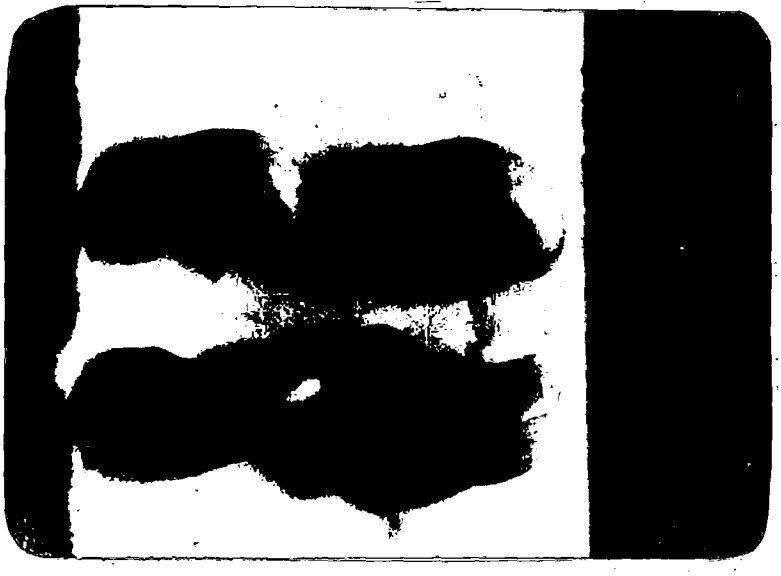
The ducks were apparently normal and healthy during the first six weeks. Subsequently there was progressive listlessness, unthriftiness and weakness and they were unable to walk (Fig.21). These symptoms progressively increased in intensity and they appeared very weak when they were sacrificed on the third month.

4.4.4. Autopsy findings.

The ducks were sacrificed on the 90th day. Necropsy examination revealed reduction in the size of the testis (Fig.22). Most of the ducks in the experimental group had significantly low testicular weight but higher liver weight than the control group (Fig.23).







Statistical analysis revealed that the effect of toxin on the weight of testis was highly significant ($P < 0.01$).

4.4.5. Histopathology.

There was pronounced histological changes in the testis of the experimental group of ducks when compared to the control birds. The testis from the aflatoxin fed ducks showed no changes in the size of the seminiferous tubules, but there was marked disruption in the organisation of the germinal epithelium. Absence of sperm bundles, a reduced germinal epithelium and debris filled lumen were seen. Vacuolation of the cytoplasm of the cells of intermediate zone and accumulation of edmatous fluid in the interstitial tissue were also observed (Fig.24). The basement membrane appeared degenerated and disrupted in some of the tubules (Fig.25).

4.5. Experimental ochratoxicosis

Group I, contained six ducks. Ochratoxin was administered at the rate of 25 µg per bird daily for a period of three months. The toxin was injected into the crop in 2.5 ml of 4.24% sodium bicarbonate solution as injection into the crop daily for a period of three months.

4.5.1. Feed intake.

The data on the feed intake of ducks during the experimental period are given in Fig.26. From the fifth week onwards a gradual reduction in the feed intake was noticed in the experimental group.

TESTES } EXPERIMENTAL
LIVER
TESTES } CONTROL
WEIGHT OF LIVER AND TESTES (%)
3·26
0·78
3·44
0·95
1·81
0·73
2·24
0·90
3·63
0·07
3·46
0·04
3·27
1·45
2·55
1·19
2·65
0·08
2·63
1·00
2·60
1·10
2·80
1·30
EXPERIMENTAL DUCKS
CONTROL DUCKS

LIVER
TESTES } EXPERIMENTAL
LIVER
TESTES } CONTROL
WEIGHT OF LIVER AND TESTES (%)
3·28
0·98
2·72
1·23
2·50
0·94
2·92
0·73
3·10
1·24
2·62
0·75
2·87
1·31
2·80
1·20
2·89
1·28
2·70
1·20
2·63
1·18
2·75
1·25
EXPERIMENTAL DUCKS
CONTROL DUCKS

4.5.2. Weight gain.

The data on the body weight of ducks during the experimental period are given in Fig.27. The control group of birds recorded an appreciable increase in the body weight.

Statistical analysis employing 't' test to find out the effect of toxin on the body weight showed that the effect was not significant ($P<0.01$).

4.5.3. Clinical symptoms.

The ducks were apparently normal and healthy during the first ten weeks. Subsequently there was slight unthriftiness and by the 12th week moderate weakness was observed.

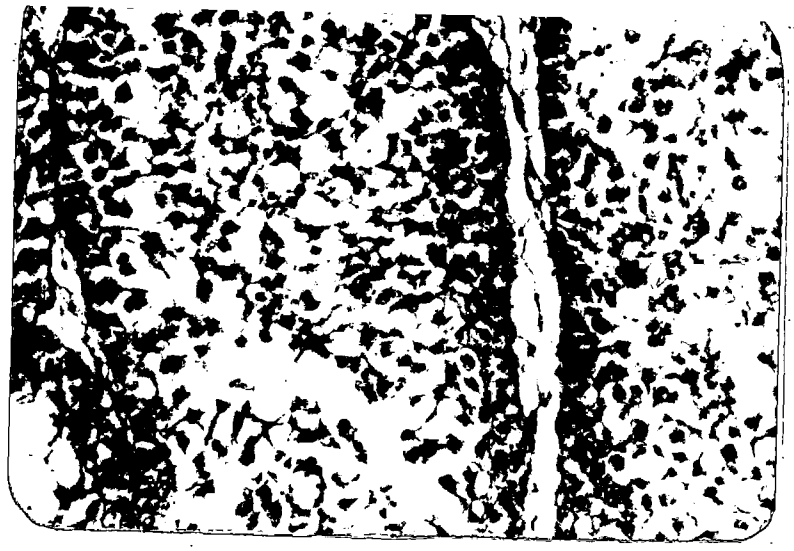
4.5.4. Autopsy findings.

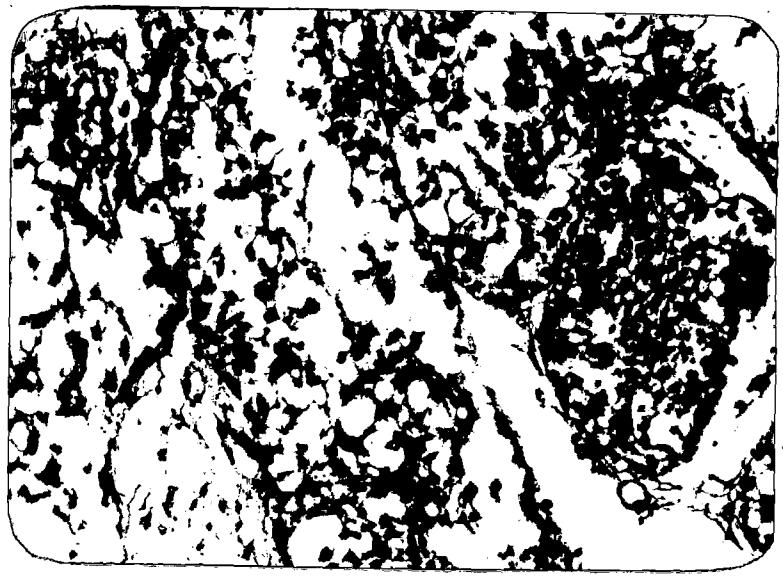
The ducks were sacrificed on the 90th day. Necropsy examination revealed reduction in the size of the testis. Most of the ducks in the experimental group had significantly smaller testis weight but higher liver weight than the ducks in the control group (Fig.28).

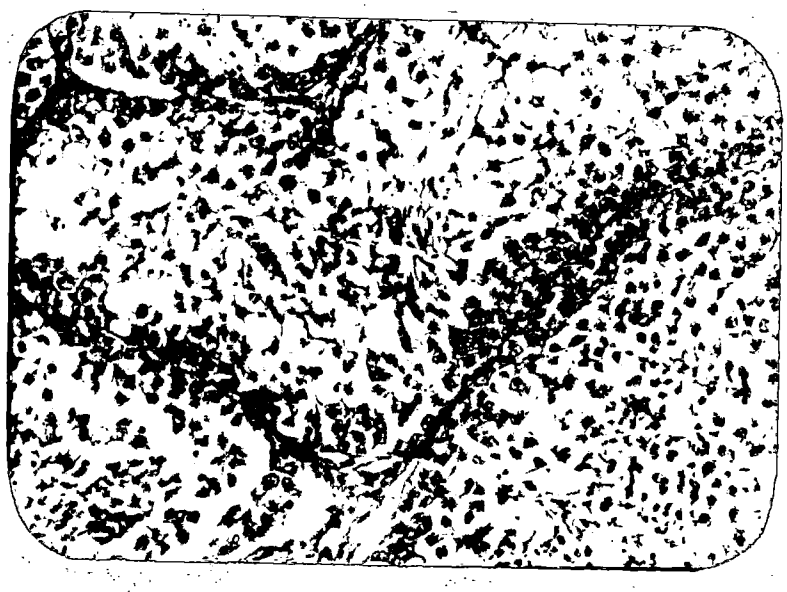
Statistical analysis employing 't' test, revealed that the effect of the toxin on the testis was highly significant ($P<0.01$).

4.5.5. Histopathology.

The microscopical lesions in the testis were prominent. The testis of the ducks fed ochratoxin showed no changes in the size of the seminiferous tubules. But there was marked







depletion of spermatozoa within the lumen of the seminiferous tubules (Fig.29). Degeneration of cells in the superficial and intermediate zones and disappearance of intermediate and superficial zones were seen (Fig.30). The basement membrane of the seminiferous tubules was found to be unaffected (Fig.31).

Discussion

DISCUSSION

Detailed studies have been carried out on the pathology of the reproductive organs in chicken. However, no planned investigations have been undertaken to study the disorders encountered in the reproductive system of ducks. The present study, therefore is a pioneering investigation undertaken to elucidate the various reproductive disorders encountered in ducks.

A retrospective survey was conducted in order to gauge the prevalence of various disease conditions encountered at the Duck Farm, Niranam. Analysis of the data, on the various disease conditions prevalent in ducks, at the Government Duck Farm, Niranam showed that during the period 1975 to 1985, hepatosis was the most important disease encountered. The incidence of the condition showed progressive increase particularly during the period 1983 to 1985 and this condition has caused great economic loss. Besides the economic loss caused by mortality, the presence of ducks with damaged liver in the farm will perforce seriously hamper productivity in ducks. The survey studies undertaken, therefore, indicated that hepatosis besides causing mortality is also an important factor which will threaten profitable duck farming.

Sriraman *et al.* (1978) from their studies also concluded that hepatosis is an important disease condition in ducks. It is pertinent to point out that dietary aflatoxin is one of the

important aetiological factors associated with hepatosis in ducks. Though groundnut cake was excluded as a feed ingredient in the ration of ducks reared in the farm, the presence of aflatoxin in other feed ingredients as reported by Maryamma et al. (1982) may be responsible for causing hepatosis. The examination of the liver of the ducks brought from the Mirnam farm for studying the reproductive disorders had also revealed hepatosis and the histological changes were suggestive of aflatoxicosis. This observation is important and it points out the need for regular screening of the feeds for the presence of mycotoxins. Tuberculosis appears to be endemic in the farm. The incidence was more during the year 1976, 1978, 1980 and 1981. So far there has not been any record of tuberculosis in ducks in Kerala. However, Singh et al. (1968) and Sriraman et al. (1978) recorded the prevalence of tuberculosis in ducks in India. Maryamma et al. (1971) recorded tuberculosis in a crow from Kerala. In this context it may be pointed out that crow can act as an agent responsible for spreading the infection, as they are in the habit of visiting duck pens. This is a most undesirable situation in a farm since in a confined flock of ducks, the disease will spread like wild fire. The situation prevalent in the farm, makes it mandatory to screen all the ducks for tuberculosis and rigorous culling has to be practised. It is not desirable to keep even a single infected duck, because this will contaminate the whole area and this would result in perpetuation of infection

and heavy loss in the flock. It would also be worth while to deflock the farm and restock it after some time.

Enteritis was prevalent in the farm all the years under investigation and it may again be considered as part of the picture of mycotoxicosis. However, whether these are primary bacterial or viral infections, cannot be ruled out since no efforts were made to identify the cause of this condition.

Out of 194 ducks systematically examined eleven ducks showed hypoplasia of the left ovary and oviduct. The ovary and oviduct were very small and there was no indication of earlier functional activity on gross or histological examination. This observation leads to the conclusion that these are hypoplastic cases and not cases of atrophy. Valsala (1968) reported this condition in chicken, and she explained that this condition might be due to failure of stimulation of growth and development of the reproductive organs due to deficiency of follicle stimulation from the pituitary. This surmise, however, needs further elucidation both on biochemical and histological basis.

Impaction of the oviduct was observed in 1.55% of the ducks autopsied. This compares with the percentage of incidence in chicken reported by Das and Biswal (1948), Peckham (1965), Valsala and Sivadas (1971) and Keymer (1980). In searching for the cause of impaction, the possibility that pre-existing inflammatory conditions of the oviduct might be

responsible, would suggest itself, especially because in many of these instances of impaction, inflammatory changes were also observed. However, not all cases of salpingitis manifested evidence of impaction, which should be the case if salpingitis alone was capable of causing impaction. Obstruction by external pressure, either by the enlarged bursa of Fabricius or neoplasms on the mesosalpinx might obviously precipitate impaction by interfering with the normal passage of egg through the oviduct. A lack of tone of the muscular wall of the oviduct is more likely to be the inciting factor for these cases. Such lack of tone might result from pressure, exhaustion, nutritional deficiency, hormonal imbalance, dehydration and other factors. Inertia of the oviduct associated with age and senility would probably appear to be capable of inducing impaction.

The appearance of haemorrhagic cystic structures in the ovary deserves special mention, because of the difference they manifest from other types of ovarian cysts. In all the four ducks in which this condition was met with, involvement of the vascular structures of the ovary was common. This observation is similar to that reported in chicken by Valsala and Sivadas (1970). Based on the gross and histologic character, these cysts appear to have formed as a result of haemorrhage into the follicles from engorged varicose veins. Hence varicosity of the ovarian vessels would appear to be the primary defect from which the haemorrhagic follicle results.

Salpingoperitonitis was observed in six ducks. This observation reconciles with the reported finding of Lindgren (1956) and Valsala (1968) in chicken. She indicated that the high production induced exhaustion of the oviduct and ovary and the nonspecific stress that accompanied such exhaustion predisposed to infection. She also observed high incidence of salpingoperitonitis in good layers in which the oestrogen activity was naturally very high. Lindgren (1956) reported that the uterine defence mechanism in mammalian species has been shown to be adversely affected by progesterone administration, which inhibits phagocytic activity. It is possible that a similar tendency for suppression of the reticulo-endothelial reaction in the oviduct under progesterone influence prevails in ducks also. In ducks, the bursa regresses before sexual maturity, and the natural antibody formation might be incomplete and such ducks would therefore be more susceptible to infection which might flare up as the progesterone level become high with consequent inhibition of uterine defence mechanism. No attempt was made in the present study to find out whether the ducks manifested any variation in the development and regression of the bursa that would account for the increased susceptibility to infection. However, it is postulated that in some groups, bursa development may not reach complete proportion before regression thereby exposing these birds to the risk of infection in the production period.

Oophoritis was observed in 27 ducks autopsied. This was

invariably associated with peritonitis. This could have resulted from the high production stress leading to the exhaustion of the ovary. The non-specific stress associated with production may predispose to Escherichia coli infection. Similar condition was documented in chicken by Valsala (1968). She reported that Escherichia intermedia is of aetiologic significance in salpingitis and oophoritis. Sharma and Joshi (1983) reported that swollen dark red ovaries, mis-shapen and congested ova and inflammed oviduct were the lesions in experimentally induced salpingoperitonitis in chicken by Escherichia coli. The lesions observed in this study were similar to those reported by them and Escherichia coli might be the causative factor. However in this investigation no efforts were made to identify the organisms involved by cultural examination.

Abnormalities relating to the persistence of the right oviduct and its transformation into cysts were observed. The nature and purpose of regression of one of the paired gonadal structures in many of the avian species has not been correctly understood. Assuming that it is an adaptation to help the birds in flight, the persistence of such a structure must be considered as a retrograde manifestation. The right ovary and oviduct are known to be potentially present in the developing embryo and they regress after having initially shown some development. Hence, remnants of these structures may naturally be expected to remain as minute bodies and such instances

would not strictly constitute abnormalities in development. It is only when these structures are grossly too large, they attract attention. One instance of cystic right oviduct was observed in a duck. Similar condition was reported in chicken by many workers (Knapp, 1922; Willier, 1927; Blount, 1949; Valsala, 1968). They observed that long inbreeding might result in retrograde manifestations in oviductal development in some flocks.

Among the possible factors for the persistence and cystic transformation of the right oviduct, Morghan and Crob (1959) have mentioned hormonal imbalance or unidentified suppressors of tissue regression. The role of oestrogen in causing persistence of right oviduct in day old chicken has been experimentally demonstrated by Williamson (1964). However, this does not explain the persistence of these structures in the adult birds. But here, it is possible that in this instance, there has been an initial and early phase of hyper-oestrogenism which caused primary manifestation of partial development of the right oviduct.

Mycotic salpingitis due to Aspergillus fumigatus was observed in one duck. There was infection in the air sac and the lesion might have spread from the air sac to the salpinx. There was extensive nodular lesions in the salpinx and the histological examination showed that the lesions were originating in the serosa and extended into the muscular coat. This observation is a proof to conclude that the involvement

of the salpinx was the result of extension of infection from the airsac. So far there has not been any report of mycotic salpingitis in ducks.

Tuberculosis involving the oviduct was observed in two ducks. On perusal of the available literature, no reports were seen describing tuberculous salpingitis in ducks. In these two instances the liver, spleen and peritoneum were involved and the involvement of the salpinx was only a manifestation of generalisation from the peritoneum.

The distribution of lesions in tuberculosis observed in this investigation was similar to that reported by Singh et al. (1968) who reported involvement of the liver, lung and kidney in tuberculosis in ducks. Sriraman et al. (1978) also reported tuberculosis of liver, lung, spleen and kidney in ducks. However, those workers did not record the involvement of the oviduct.

The pathological changes encountered in the testis were only few. Atrophy of the testis was seen in two instances and seminoma in one duck. The seminoma was bilateral and the growths were relatively big and histologically the tubular pattern was maintained. There has not been any record of seminoma of the testis in ducks and this would appear to be the first report.

In experimental aflatoxicosis administration of aflatoxin B_1 at the rate of 25 µg per bird resulted in decreased feed

intake and reduced weight gain. Ottinger and Beerr (1980) reported decreased body weight in immature Japanese quails, which were fed aflatoxin. Sharlin et al. (1980) indicated that aflatoxin B_1 when fed at high levels (20 µg/g diet) to immature White Leghorn male chicken depressed appetite or made the feed unpalatable and this they attributed as the reason for decreased feed intake and reduced weight gain. Later they observed that chicks and adults could overcome the appetite depressing effects of dietary aflatoxin after four weeks of ingesting contaminated feed. But how aflatoxin causes a temporary decrease in feed consumption remains speculative. Polin and Wolford (1973) hypothesised that appetite is controlled by receptors in the crop interacting with the hypothalamus and the whole system can be modified by metabolic alterations arising elsewhere in the body. Aflatoxin, could therefore affect appetite as a result of its influence on the hepatic metabolism. Here it may be pointed out that in both the experimental and control groups there was a steady reduction in body weight from the fourth week onwards, upto the sixth week. There is no other explanation for this except for the fact that this reduction may be due to a change in the brand of feed that was effected during the experimental period.

In this experiment, there was apparent increase in the weight of the liver and a reduction in the body weight. However, statistically they were not significant ($P<0.01$). Wyatt et al. (1973) observed enlargement of the liver after

four weeks of dietary aflatoxin in broiler breeders. They noted a three-fold increase in liver lipid and a decrease in serum total lipid in broiler breeder males fed aflatoxin and suggested that lipid transport from the liver was impaired. This they attributed as the reason for the liver enlargement.

Histologically, there was significant hepatic degeneration, necrosis and biliary hyperplasia in the ducks doced with aflatoxin and these are characteristic changes in aflatoxicosis in ducks and has been recorded by earlier workers (Asplin and Carnaghan, 1961). This observation supports the conclusion that the high incidence of hepatosis observed in the duck farm, Niranam might be due to dietary aflatoxin. Significant reduction in the weight of the testis was observed in all the experimental ducks. This observation is consistent with that of Doerr and Ottinger (1980) in immature Japanese quails and Sharlin et al. (1980) in mature White Leghorn males. They pointed out that the decreased feed consumption resulting from the ingestion of aflatoxin and the accompanying loss of body weight were responsible for the reduction in the testicular weight.

Histologically there was significant degenerative changes in the testis characterised by degeneration of the tubular epitholium and aspermatogenesis. This also may account for the reduction of the weight of testis. The study undertaken has therefore, clearly demonstrated the deleterious biological effects of aflatoxin on the testis. This has great practical

relevance since ducks are regularly consuming aflatoxin in the feed and this will result in variable degree of testicular pathology leading to lowered fertility and hatchability of eggs. This would be an important problem for farmers and may partly explain the poor hatchability of duck eggs experienced by them. In chicken, Howarth and Wyatt (1976) clearly demonstrated that aflatoxin is detrimental to hatchability. Since the ducks were much more sensitive to aflatoxin than chicken, the magnitude of the problem is much greater in ducks. This observation again stresses the need for screening the feed of ducks for aflatoxin to prevent subfertility and infertility of eggs.

Administration of ochratoxin A resulted in decreased feed intake and reduced weight gain. This is a finding similar to that observed in aflatoxicosis, and gives proof to the conclusion that both aflatoxin and ochratoxin have almost similar effect on the growth. Similar observations were reported in broiler chicks by Kubena et al. (1983). They reported significant reduction in body weight and feed efficiency, when day old male broiler chicks were fed a diet containing 3 ppm of ochratoxin A. This they attributed to the appetite depressing effect of ochratoxin. Prior et al. (1980) reported that chicks and adults can overcome the appetite depressing effects of dietary ochratoxin after four or five weeks of ingesting contaminated feed.

The increase in the weight of the liver and the reduction in body weight seen in experimental ducks were not found to be statistically significant ($P<0.01$). In both the experimental and control groups, there was a steady reduction in body weight from the fourth week onwards, upto the sixth week. This reduction in body weight seen both in the control and experimental groups may be due to a change in the feed that was made during the experimental period and similar observations were made in the ducks dosed with aflatoxin during the same period. But it may be pointed out that the reduction in the body weight was much more pronounced in the experimental group when compared to the control group.

In the ducks dosed with ochratoxin there was significant reduction in the weight of the testis. Similar observations were reported by Dwivedi and Burns (1985) in chicken dosed with ochratoxin. The reduction in the weight of the testis seen in ducks dosed with aflatoxin and ochratoxin in these experiments was comparable. Pronounced degenerative changes were seen in the testis and this can no doubt lead to infertility, subfertility and poor hatchability of eggs. Dwivedi and Burns (1985) documented similar pathobiological changes in the testis of chicken dosed with ochratoxin. However, histologically no changes were seen in the basement membrane of the seminiferous tubules in ochratoxin group. Whereas in aflatoxin group, degeneration of the basement membrane of the seminiferous tubules, edema and absence of sperm bundles were the

lesions encountered. This is an observation which would lead to the conclusion that aflatoxin has more severe biological effects on the testicular tissue than ochratoxin and the former is to be considered as a more potent toxin which would affect more significantly the productivity of ducks.

The feed that is given to ducks in the field situation is likely to contain both aflatoxin and ochratoxin and the synergestic effect of these two has to be borne in mind. Eventhough, the feed sample may contain an amount below the permissible level of these mycotoxins when both of these are present, the synergestic effect is bound to cause extensive damage to the liver and reproductive organs and this can be a major cause for poor hatchability in the farm. The experimental studies carried out, therefore focusses attention to the important problem of the damaging effects of mycotoxins on the reproductive organs. This implies that there is need to have regular screening of feed for these two mycotoxins and since the ducks are very sensitive they should be given a feed which is almost free of these toxins.

Summary

SUMMARY

A study on the pathology of the reproductive system in ducks was carried out for a period of two years from 1983 to 1985. A detailed survey on the various disease conditions prevalent at the Duck Farm, Niranam was made based on the data recorded during a 11 year period from 1975 to 1985. The studies revealed the incidence of the following diseases namely, hepatosis (64.58%), hepatitis (9.07%), enteritis (8.58%), tuberculosis (2.61%) and miscellaneous disorders (15.13%). The latter category included pulmonary edema, omphalitis and transport stress.

During the study, 194 female and 26 male ducks were subjected to detailed post mortem examination. The reproductive disorders encountered in the females were hypoplasia of the left ovary and oviduct (20%), impaction of the oviduct (5.45%), haemorrhagic cyst in the ovary (7.27%), salpingoperitonitis (10.9%), mycotic salpingitis (1.82%), tuberculous salpingitis (3.63%), oophoritis (49.09%) and cystic right oviduct (1.82%). In the males atrophic testis (7.69%) and seminoma (3.84%) were the disorders encountered. The gross and histologic features of these conditions were described in detail.

Aflatoxin B_1 was administered to six ducks at the rate of 25 µg per duck daily for a period of three months. Clinically all the experimental ducks showed unthriftiness and weakness. Considerable reduction in feed intake was observed

in the experimental group from the third week onwards. The experimental ducks showed reduced weight gain compared to the control ducks.

There was reduction in the weight of the testis. Histologically, degeneration of the basement membrane, marked disruption in the organisation of the germinal epithelium, absence of sperm bundles, debris filled lumen, vacuolation of the cytoplasm of the cells of the intermediate zone and accumulation of edematous fluid in the interstitial tissue were the lesions observed.

Ochratoxin A was administered at the rate of 25 µg per duck daily for a period of three months. Clinically, all the experimental ducks showed moderate weakness and showed gradual reduction in feed intake from fifth week onwards. An appreciable increase in body weight was observed in the control group.

When the ducks were sacrificed at the end of the third month, ducks in the experimental group showed significantly low testicular weight than the control groups.

Histologically, marked depletion of spermatozoa within the lumen of the seminiferous tubules, degeneration of cells in the superficial and intermediate zone and disappearance of the intermediate and superficial zones were observed. The basement membrane of the seminiferous tubules was unaffected.

The experimental studies undertaken with aflatoxin and ochratoxin showed significant pathoanatomical changes in the testicular tissue and it was surmised that these mycotoxins can cause subfertility and infertility in ducks.

The observations made indicated that aflatoxin has more severe biological effects on the testicular tissue than ochratoxin. The investigations undertaken pointed out the need for giving a feed which is free of mycotoxins to ducks in order to ensure maximum productivity.

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PATHOLOGY OF THE REPRODUCTIVE SYSTEM IN DUCKS

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ABSTRACT OF A THESIS

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ABSTRACT

A study was conducted on the pathology of the reproductive system in ducks. A survey conducted on the prevalence of diseases based on the data for a period of 11 years at the Government Duck Farm, Miranem revealed the incidence of hepatosis (64.58%), hepatitis (9.07%), enteritis (8.58%), tuberculosis (2.61%) and miscellaneous disorders (15.13%). The latter category included pulmonary edema, omphalitis and transport stress.

In a separate study, the reproductive organs of 194 female ducks and 26 male ducks were examined systematically and gross and histopathological lesions encountered were studied. The diseases encountered in females were hypoplasia of left ovary and oviduct (20%), impaction of oviduct (5.45%), haemorrhagic cyst in the ovary (7.27%), salpingoperitonitis (10.9%), oophoritis (49.09%), cystic right oviduct (1.82%), mycotic salpingitis (1.82%) and tuberculous salpingitis (3.63%). In males atrophic testis (66.3%) and seminoma (33.3%) were the diseases recorded.

Experimentally, pure aflatoxin B_1 and ochratoxin A were administered to six ducks each, at the rate of 25 µg/bird/duck daily for a period of three months. Clinically all the experimental ducks showed unthriftiness. But it was more pronounced in aflatoxin group. In both aflatoxin and ochratoxin group, the birds showed reduced weight gain and decreased feed intake.

There was reduction in the weight of the testis in both aflatoxin and ochratoxin groups. Histologically, in the aflatoxin group, marked disruption in the organisation of germinal epithelium, absence of sperm bundles and edema of the interstitial tissue were the lesions observed. In the ochratoxin group, the basement membrane was found to be unaffected. Both aflatoxin and ochratoxin were found to cause degenerative changes in the testicular tissue. However, aflatoxin was found to cause more pronounced changes than ochratoxin.

From the studies made, it was concluded that both aflatoxin and ochratoxin can induce degenerative changes in the testis and it was surmised that this would lead to subfertility and infertility. The need for feeding a diet free of mycotoxins to ensure profitable duck farming was stressed.