CHAPTER: 2

REVIEW OF LITERATURE

So many workers have studied experimentally induced pathological conditions in different organs of quail. Similarly an ample of work is available on chickens. Specially the early workers mainly concentrated on chickens and it is much later the researchers have started taking interest in quails. Still literature on organ pathology and hematology due to natural infections on quail is not too much. Some notable works on quail and similar works on chicken and other avian species are reported as follows:

HEMATOLOGY:

Newell and Shaffner (1950) carried out studies on blood volume determination in chickens. They reported that the PCV (packed cell volume) value is lower at early age and it increases with the advancement of age in chickens.

Dukes (1931) reported that hematological values in birds (chickens) are influenced by age, sex, breed, climate, geographical location, seasons, day length, time of the day, nutritional status, life habit of species and such other physiological factors. He also reported that erythrocyte count at younger age is low and increases with the advancement of age.

A naturally occurring lympho-proliferative disease in Japanese quail has been reported from Mexico by Schat et.al. (1976). They detected tumor like lesions in spleen and liver.
Histology revealed a pleomorphic lymphoproliferative infiltration with dark staining large cells, lymphocytes of different sizes and reticulum like cells.

Wight et.al. (1980) reported differential and total white cell count on the peripheral blood of chickens, turkey and quails, fed from hatching in zinc deficient diet. They found severe monocytosis.

Leonard et.al. (1982) have reported standard ranges of several hematological parameters. They studied the age related hematological profiles. They reported the similar mean values as reported by other researchers although lymphocytes and monocytes count tended to be higher than the published values. They reported the values for quail as RBC 4.7 X 10^{12}/L (normal range 4.0-5.5), Hb 11.0% (normal values 11.0–15.0); PCV 42% (normal values are 30–45%) and WBC were between 12.5 to 25.0 X 10^{9}/L.

Kai and Pranklin (1984) carried out age dependent size distribution chicken blood cell analysis by utilizing a coulter counter (method). They reported that the erythrocyte number is lower in early age and gradually increased with age.

Bhattacharya (1990) studied short-term thermal stress on some hemato-biochemical constituents in Japanese quails. Respiratory stress was observed in heat stressed quails. By analyzing blood samples it was reported that the values of RBC, Hb and PCV were significantly higher in heat-exposed groups of quails.
A study on clinical signs and hematological alterations in pneumonic Aspergillosis due to *A. flavus* in Japanese quail is carried out by Pandita *et al.* (1991) They carried on study on two-week-old chick and found dullness, depression, anorexia, accelerated breathing, gasping and prostration leading to death. They reported 25% mortality in 6 weeks. Average weight was slightly lower than control chicks. No appreciable difference was observed in mean value of Hb; PCV and TEC between controlled and infected quails. In total leukocyte count there was significant increase (P less than 0.05) due to increase in percentage of heterophils and decrease in lymphocyte.

Sinovec *et al.* (1992) reported influence of high dietary salt level on blood pictures of Japanese quail. They fed Japanese quail with high concentration of salt (NaCl) at different concentration and reported significant differences (increase) in the values of mean red cell count, mean leukocyte count including WBC, heterophils, eosinophils, lymphocytes, basophils and monocytes.

Szubartowska *et al.* (1992) reported blood morphology in quails after poisoning with fenithrothion (pesticide). They found that it reduced the number of RBC, Hb level and haematocrite values but increased erythroblast and reticulocyte numbers. In males changes in blood appeared earlier than females and underwent compensation on earlier after intoxication.

Kundu *et al.* (1993) made an investigation on hematological study of different age groups of Japanese quail. Young female quails (1.5 month old), adult quails (3 months old),
males and laying females and old quails (1 year old females) showed different hematological findings. Young quails had lower Hb and RBC, adult layers had lower RBC count than adult males and old birds had low values for all hematological parameters.

Dein et al. (1994) has given avian leucocytes counting using the hemocytometer where Natt and Harrick and Unopette methods are the most common stain and diluents preparations for this method.

Devender Rao et al. (1998) carried out studies on hematobiochemical changes in sub acute malathion toxicity in Japanese quail. They reported hematobiochemical changes in quail fed with malathion at 150, 300 and 450 PPP from 4th week onward for a period of 4 weeks. They reported that feed intake; body weight gain and feed conversation ratio were not affected significantly. Leukocytosis, more specifically heterophilia and eosinophilia was significantly at higher doses during later stage of toxicity. Serum-amino-transferase activity was reduced but not significantly.

A serological survey of chickens, Japanese quail, pigeons, ducks and crow for antibodies to chicken anemia virus (LAV) is presented by Farkas et al. (1998) from Japan. These birds were tested for the presence of antibody to CAV by a micro-scale virus neutralization (VN) test. 69.2% chickens; and 61.3% quail samples were found positive for the test with titers ranging from 1:20 to 1:2560. They depicted that samples collected from quail in 1992 showed a lower rate of antibody-positive sera (7.7%) and lower antibody titers (1:20 to 1:80) than those collected in 1995 (90.2%
and 1:20 to 1:2560 respectively). None of the pigeon, duck or crow samples neutralized CAV at a 1:20 dilution. It is concluded that quail might be one of the hosts of CAV or CAV like virus.

**Mihailov et.al. (1999)** carried out hematology of chickens and Japanese quail. They determined the hematological values like RBC and WBC count and Hb concentration in Japanese quail males and females (egg laying) and compared to male chickens and laying hens. They found no statistical differences between the species but some sex determined differences were identified in quails. Statistically lower thrombocytes and monocytes count were found in Japanese quail females. Also tendency towards lower Hb, lower RBC and WBC count was noted in quail females than males.

**Nazifi and Asasi (2001)** from Shizar studied the hematological and serum biochemical changes in air sac rupture of Japanese quails due to the effect of feeding of “turazolidene” and found significant decrease in WBC and lymphocyte ($P<0.05$)

**Sunil kumar et.al. (2001)** reported clinical signs, growth response and hematological changes in Japanese quails infected with *Salmonella typhimurium* and found high mortality (40%) and decrease in body weight. They also observed decrease in PCV value, Hb and Total erythrocyte count. They noticed a sharp increase in total WBC count due to increase in the number of heterophils and leukocytes.

**Ozcan et.al. (2001)** reported the effect of thyroid disorders on some hematological parameters in quail. Hb was found
high in hyperthyroid group. WBC and RBC count remained unchanged.

Reissing et al. (2002) reported hematology and serum chemistry values of lesser Rhea (*Pterocnemia pennaa*) from Argentina. Hematological and serum values were determined in chick juveniles and adults and farm lesser Rheas. Rheas have significantly lower RBC, Hb, haematocrit, red cell indices and copper than chick juveniles and the adults.

Rinesh kumar et al. (2002) carried on study on hematological changes in Japanese quail naturally infected with *Raillietina tetragona*. Hematological changes in infected young and adult Japanese quails showed no significant changes in RBC count. WBC and Hb concentration decreased significantly. Erythrocyte sedimentation rate rose significantly in adult quails. There was significant increase in percentage of heterophils and eosinophils. However lymphocytes decreased significantly.

Kaya et al. (2003) studied the effect of different dietary levels of Yucca schidigera powder on performance, blood parameters and egg yolk cholesterol of laying quails. They found that RBC, WBC and PCV values are not affected but Hb concentration increased significantly.

Yashwant singh et al. (2003) made clinico-hematological observations in Japanese quails following short-term exposure to saline drinking water. They used NaCl at different concentration (.5, 1.0 and 2.0%) in drinking water and studied certain hematological
parameters in four groups of Japanese quails. Birds on high salt water (1.0 & 2.0%) exhibited clinical signs of toxicity. Mortality was observed in birds drinking 2% salt water only. Significant Hb, PCV and Total Erythrocyte Count indicate salt induced hemoconcentration in quails. Heterophils decreased significantly which may be due to migration of the heterophils from blood to the site of injury while lymphocytes increased as a compensatory mechanism to heteropaenia. Results suggested that NaCl in concentration > 0.5% in drinking water of quails produces hemotoxicity.

Awonival et.al. (2004) reported the effect of maggot meal based diet on live stock. It led to the establishment of some indices with which the health and performance of the animals can be monitored. More important among each health condition is anemia that can be monitored using indices such as PCV, RBC, and Hb content. Their study showed that the effect of maggot meal based diets on broiler chickens erythrocyte indices was monitored as deviation from normal values may affect the physiological status of the chickens and consequently their performance. The result obtained from this study suggest that the feeding of maggot meal to broilers has no adverse effect on erythrocyte indices because of all values for PCV compared favorable well with normal values as obtained by Mitruka and Rawnsley (1970) & Ross et.al. (1978)

Islam et.al. (2004) conducted study of hematological parameters in Fayoumi, Assil and local Chickens of different ages reared in Sylhet region in Bangladesh. They determined TEC, PCV, Hb, ESR, DLC, MCV, MCH, and HCHC. They found result which
was in agreement with the work of Dukes (1955), Kai and Pranklin (1984); Sturkie (1959) and Kundu et al. (1993) i.e. the erythrocyte number, hemoglobin concentration and packed cell volume increased with the advancement of age. ESR was inversely related to age i.e. higher in early age and less in higher age. Lymphocytes and heterophils were two principle leukocytes which exerted their dominance on other leukocytes.

**Ozbey et al.** (2004) carried out study on the effects of high temperature on blood serum parameters and the egg productivity characteristics of Japanese quails. It was observed that the higher temperature increased some of the blood serum values such as glucose, Na, triglycerides (P< 0.05), cholesterol (P< 0.01) and uric acid (P< 0.01) levels but reduced the blood serum protein (P< 0.01) and albumin (P< 0.05) levels. Even if the higher temperature has led a decrease in egg productivity in all ages during the trial period, the difference that was observed in all ages except seventh week was found important (P< 0.05). The values of the egg weight in both control and experimental were found as 10.53 and 9.93 g. They have also carried out experiments on effect of high temperature on growth performance on Japanese quails having different body weights and also the effect of high temps on breeding and survival of Japanese Quails.

**Bashir et al.** (2005) reported the effect of alcohol at different doses through drinking water in quails. In Pakistan poultry farmers use ethanol for the treatment of respiratory diseases and as growth promotent but the adverse effect on health and production of
broilers are unknown. They offered 8% and 16% alcohol and observed dullness, depression and staggering gait while those given 2% and 4% ethanol showed decreased responsiveness for 2 to 3 hours. There was significant increase (P<0.05) in relative weight of pancreas. The liver weight and brain volume along with Na and K decreased significantly (P<0.05) in treated groups. RBC, PCV and Hb concentration increased significantly (P<0.05) while leukocyte count increase was not significant. Their findings suggested that ethanol at these levels has significant detorious effects on hematology, brain volume and serum electrolyte.

FECAL SAMPLE EXAMINATION (Coccidiosis):

Zaprianov (1979) carried out Eimeria tenella studies in quails and Eimeria kofoidi studies in chickens. Studies on two types of coccidiosis, E. tenella isolated from diseased chickens and E. kofoidi isolated from diseased young quails were carried out. Ten 14 days old quails of the species Alektore in graeca cypriatis and thirty 10, 20 and 30 days old chickens of white plymouth breed were used. By cross invasion of sporulated oocyst of both coccidia types, the shizogonal development in non-specific host birds was observed. It was established that E. tenella finds suitable conditions for and endogenous development, no matter how slight, in the ceacum of the non specific host — the young quails. E. kofoidi, however does not find similar suitable conditions for its development in the alimentary canal of chickens and passes transit. Under certain definite conditions quail can be potential reservoir for E. tenella in nature.
Dimerdash et al. (1984) reported coccidiosis in quails from U.S.A.

Ruff et al. (1988) studied effect of coccidiosis on reproduction in male Japanese quail. They exposed the male quails before sexual maturity to oocyst of Eimeria uzura at 16 or 30 days of age and sampled at 37 days. Quails with high doses of Eimeria were having reduced testes and lower circulatory androgen concentration compared to control males. They observed no difference in average weight of testes by 51 days.

Rai et al. (1989) studied mortality pattern and incidence of poultry disease in Andaman and reported high mortality (55.55%) due to coccidiosis in layers.

Miranda et al. (1992) reported clamydeosis in red tailed hawk (Buteo jamaicensis) with clinical signs like respiratory distress and diarrhea. Necroscopy revealed emaciated carcass, splenomegaly, fibrinous pericarditis, airsacculitis and perihepatitis. Microscopically they reported fibrinous pericarditis, airscculitis, myocardial necrosis, necrotizing hepatitis, splenic necrosis, reticuloendothelial cell hyperplasia, interstitial pneumonia, focal pancreatic necrosis and fibrinous exudates covering lung, liver, spleen, heart and pancreas.

Panda and Dwivedi (1995) studied chemoprophylactic use of some non-coccidiostal agents in experimentally induced
coccidiosis in Japanese quails from Hyderabad. They found clopidol having maximum chemoprophylactic activity.

**Rao et al. (1995)** reported influence of dietary aflatoxin or *E. uzura* infection in Japanese quails. They found significantly less weight than normal chicks either receiving aflatoxin or coccidial alone. They also observed increased morbidity, mortality and decreased efficiency of feed utilization. Combination of *E. uzura* and aflatoxin resulted in reduction in PCV and Hb i.e. it showed additive effect.

**Awadalla (1998)** from Egypt has reported the influence of dietary aflatoxin on the severity of coccidial infection in quail. The infection with the combination of aflatoxin and coccidia produced higher mortality rate, higher fecal scores with increased oocyst output. Reduction in weight gain, decreased feed and utilization in also reported. The lower RBC, Hb values and total WBC count in infected quail was observed with coccidia and aflatoxins.

**Badawy et al. (1999)** carried out study on some protozoan parasites in migrant-quail (*Coturnix coturnix*, coturnix). Out of 87 migrating common gray quails collected at Alexandra, Egypt, during autumn 1997. 79 (90.8%) were infected with protozoan parasites. The 79 isolates included *Plasmodium* species (24, 30.4%), *Tritichomonas* species (6, 7.4%) and *Eimeria* Species (66, 83.5%). Three species of *Plasmodium* (*P. durae*, *P. falax* and *P. circumferum*), one of *Tritichomonas* species (*T. eberthi*) and 4 of *Eimeria* (*E. uzura*, *e. bateri*, *E. garnhami* and *E. tsunodai*) were identified. Transmission experiments in domestic (Japanese) quails
using intestinal and cecal isolates of *Eimeria* oocysts obtained from the common gray quails were successful. The prepatent and R patent periods of infection were measured and the histo-pathological changes in the intestine of the Japanese quails due to coccidial infections are described.

*Looszova et al.* (2001) reported increased total leukocyte count due to *Eimeria* infection (*E. colchici* and *E. tenella*).

*Kadam et al.* (2002) studied the hemato-biochemical changes in due to experimentally induced coccidiosis in chicks. They reported the decreased values of Hb, PCV and Total Erythrocyte Count and increase in Total WBC Count.

*Alan Kocan et al.* (2003) reported 28% morbidity due to *Eimeria* species in quails from Okahama.

*Teixeira et al.* (2004) reported coccidiosis in Japanese quails (*Coturnix coturnix japonica*) as characterization of a naturally occurring infection in commercially rearing farm. They carried out the study in order to identify species of genus *Eimeria* and characterize a naturally occurring infection in a commercial rearing farm. For this purpose fecal examination, oocyst counting and morphological study were performed besides necropsy and histo-pathology to confirm diagnosis. Three species of the genus *Emieria* were found and identified as *E. tsunodai* *E. uzura* and *E. bateria*. The natural infection was characterized as sub-clinical because of mild and nonspecific clinical signs. Nevertheless, coccidiosis was considered as an important disease because endogenous stages of parasite and a high number of oocyst in the faces were associated with
intestinal lesions. They reported villous erosion, hyperplasia of crypts of liberkuhn, and infiltration of inflammatory cells associated with edema. The results suggest that such infections might represent a limiting factor to this branch of modern poultry industry.

**Zaher Ahmad Radi (2004)** carried out study on epizootic of combined *Clostridium perfringers*, *Eimieria species* and *Capillaria species* enteritis and *Histomonas species* hepatitis with *Escherischia coli* septicemia in Bobwhite quails. They found anorexia, weakness, diarrhea, dehydration and loss of weight, death, multifocal subserosal intestinal ulceration with subcutaneous perforation and peritonitis and multifocal necrotizing hepatitis. Microscopically they reported severe necro-ulcerative and heterophilic enterocolitis and typhitis (bacteria *Capillaria species*) multifocal nacro-granulomatous hepatitis (due to protozoa *Histomanas species*). *E. Coli* were isolated from spleen, intestine and liver.

**HISTOPATHOLOGY:**

A fungal infection in Japanese quail have been reported by **Matsumoto et.al. (1978)** from Japan. *Aspergillon fumigatus* was involved in an out break of aspergillosis in experimental quails. Typical granulomatous inflammatory changes were seen in lung nodules at post-mortem examination.

**Dhillon et.al. (1980)** have studied out break of histomoniasis in bob white quails. They reported 95% mortality in quail over 3 weeks of age. In their article they have reported 1-2 mm
in diameter, occasionally subscapular multifocal splenic necrosis and also necrotic foci on liver. Histology revealed that focal necrosis is associated with variable number of *Histomonas*.

An epidemiological and pathological study in outbreak of *Salmonella bareilly* infection in chickens and quails have been carried out by Kapoor *et.al.* (1980). The out break of this infection resulted in a mortality of 92.5% (in 900 quail chicks) It seems to be first report of *S. bareilly* in quail. Pathological results were similar in chicks and quails but was more severe in quails particularly pericarditis and enteritis.

Pradhan *et.al.* (1980) reported bronchitis in Japanese quail and studied the pathological lesions.

Chang and Hamilton (1982) carried out a study on experimentally induced aflatoxicosis in young Japanese quail. Japanese quail was fed with different levels of diatary aflatoxins. From hatching to 4 weeks of age, they were comparatively resistant. At higher dose (5 µg/g or more) growth inhibition occurred. 50% mortality was seen at a dose of 20 µg /g. The acute oral dose was 19.2 ± 4.8 mg/kg. These values are up to 10 times those reported in the literature of quail. Unlike chickens, the spleen size of quail was little affected at lower doses (1.2 µg /g).

Panigrahi *et.al.* (1982) reported acute out break of fowl cholera in quail. They reported high mortality in *Coturnix coturnix japonica* and *Colinus virginianus*. Causative organism reported was *Pasteurella multocida* type 3.

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Thon, Kneiesberg and Dixon (1982) have reported epithelial deciliation and hyperplasia and infiltration of inflammatory cells in trachea, bronchi and nasal cavity in quail due to cryptosporidiosis (*Cryptosporidium* infection). The gross lesion due to the infection was pathological lesions and excess mucus secretion in trachea and nasal cavity.

Derieuze (1983) observed reactions of Bobwhite quails, Japanese quails, guinea fowl and Mallard to avirulent and virulent *Pasteurella multica*. He found the species was moderately virulent to Japanese quail by oral rout and highly virulent to Bobwhite quail by stickwing. It was avirulent to guinea fowl and Mallard.

Sah, Mall and Mohanti (1983) have described Septicemic proteus infection in *Coturnix coturnix japonica* chicks. They reported that proteus infection was incriminated as the cause of severe depression, coma and high mortality in quail chicks. The pathological lesions were comprised of congestion of lung and mucus exudation in trachea. They isolated *P. mirabilis* from the heart blood and lungs of affected chicks. The pathogenesity of the isolates was also tested in young albino mice and week old quail chicks, died from the infection within 48 hrs of inoculation. Association of *P. mirabilis* with septisemic disease in Japanese quail has apparently been demonstrated for the first time.

Didier et.al. (1984) reported Magnesium deficiency in Japanese quail. Mg-deficiency was characterized by a depressed growth, high mortality rate decreased hematocnt and Mg and Ca

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plasma concentration. Mg deficiency reduced the Mg concentration in heart by 44%.

Pradhan, Mohanti and Mukit (1985) reported Marek's disease in Japanese quail. They found visceral tumors. The routine postmortem showed gross lesions mostly in the spleen and liver. Microscopic lesions were common in spleen, proventriculus, liver, and duodenum.

Da Silva et.al. (1989) reported occurrence of coligranulomatosis in Japanese quail in Vberlandia, Minas, Gerais and Brazil. 85% quails were affected by coligranulomatosis. Drop in egg production and 15% mortality observed. Histo-pathological lesions were located in mesenteries, intestine, gizzard, heart, liver, oviducts and ovaries.

Glisson et.al. (1989) has reported Interstitial pneumonia in three flocks of Japanese quail of approximately 75000 birds each. They observed high mortality rate beginning at 24 to 28 days. Gross lesions were either absent or if present composed of either multifocal pale area on liver, spleen or lungs. Lesions were slightly darker than the normal color of the organ. Histo-pathology revealed focal splenic and hepatic necrosis and interstitial pneumonia. They isolated Pasteurella multocida serotype 3 and 4 from affected organs. They treated these quails successfully with chlortetracycline.

Jack and Reed (1990) studied experimentally induced quail bronchitis in Bobwhite quails (Colinus virginianus). The quail were inoculated with 10(6) man tissue culture infective dose of quail
bronchitis virus at 1, 3, 6 and 9 weeks of age by intra-tracheal, intra-peritoneal and subcutaneous routes. Quail showed necrotizing tracheitis, proliferative and necrotizing bronchitis and pneumonia. Multifocal necrotizing hepatitis, necrotizing splenitis with or without hyperplasia of splenic mononuclear phagocyte, bursal lymphoid necrosis and bursal atrophy is also reported. Lesions reported were more severe in quails. Large intra-nuclear inclusion characteristics of adenovirus infections were identified in trachea, lung, liver and bursa. This is the first report of histo-pathology of experimentally induced quail bronchitis.

Buchholz and Fairbrother (1992) reported pathogenisity of Salmonella in Northern Bobwhite quails and Mallard ducks. 10 days old bob white quails and Mallard ducks were administered orally and intravenously with Salmonella pullorum at selected concentration. Mortality in bobwhite quail was 65—100% where as in none of the Mallard ducks were died due to infection. Bacteria showed colonies in all organs of the body like lung liver, heart & kidney. Slight to moderate diffuse or multifocal necrotizing inflammation were present in all organs.

Miranda et al. (1992) reported gross and microscopic lesions in red tailed hawk (Buteo jamaicensis). They observed respiratory distress and diarrhea. Nacroscopy revealed emaciated carcass, spleenomegaly, fibrinous pericarditis, airsacculitis and perihepatitis. Microscopically they observed fibrinous pericarditis, airsacculitis, myocardial necrosis, narcotizing hepatitis, splenic necrosis, reticuloendothelial cell hyperplasia, interstitial pneumonia and focal pancreatic necrosis. They also
noticed intracytoplasmic chlamydial inclusion bodies in macrophages in fibrinous exudates covering air sacs, pericardium, spleen, liver, heart, lung and pancreas.

Pattnaik et al. (1993) reported brooders pneumonia in Japanese quails. It is a common fungal infection affecting lungs and air sacs of young poultry birds. In their paper they reported six cases of brooders pneumonia in Japanese quails. Affected quails were having open mouth breathing and poor health status. A number of small yellowish nodules were seen in the lung, which was mildly congested. The causative organism detected as *Aspergillus fumigatus*. Histo-pathologically the lesions showed typical granulomatous reaction. Alveolar macrophages were the major cells involved. Besides this the lesions were characterized by well-demarcated foci of necrosis with infiltration of macrophages, few giant cells and heterophils.

Dubey et al. (1994) have reported experimental toxoplasmosis in Japanese quail. 24, five month old battery hatched Japanese quails were inoculated with different doses of *Toxoplasma gondii* oocyst. Out of 24, 11 quails died. Quails that survived showed protozoal pneumonia, myocarditis or meningoencephalitis. They also showed hypertrophic spleen. *Toxoplasma gondii* antibodies were found in the sera of all the quails.

Jack et al. (1994) studied pathology of quail bronchitis. They inoculated 1-week-old quails (intra-tracheal) by quail bronchitis virus and sacrificed them 2, 4, 8, 16 and 24 hours after inoculation. They carried on pathology of tracheal epithelium and found

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deciliated tracheal epithelium which formed irregular laminar border. Tracheal epithelium was partly desquamated (or 4th & 5th day after inoculation) but minimal leukocyte infiltration. As compared to tracheal epithelium, bronchial epithelium showed more leukocyte infiltration and lymphocytes and heterophils on 3rd day after inoculation. Hyperplasia of splenic macrophages is also noticed.

Singh et al. (1995) studied pathogenesis of inclusion body hepatitis virus (avian adenovirus) in Japanese quail and broiler chicken by experimentally inoculating IBH virus derived from quails. Virus was inoculated intra-peritoneally at 3, 4, 5, 6 or 7 week of age. Lesions were noted in liver, kidney and lungs. They noted pale swollen, mottled liver, swollen nephritic kidney congested and pneumonic lungs. Result indicate that their isolate is pathogenic for quail above 3 weeks of age.

Senthivelan et al. (1996) reported pathogenicity of quail isolate of Newcastle disease virus (NDV) in chicks. Histopathological examination showed severe depletion and necrosis of lymphocytes in the lymphoid organs and mononuclear cell infiltration in liver.

Chandra and Kumar (1998) presented a review on quail bronchitis. This review covered the epidemiology, etiology, pathology, diagnosis and control of the disease. The causative agent was adenovirus that was similar to chicken embryo lethal orphan (CELO) virus.
Kalpana and Sriram (1998) studied pathology of mercuric chloride toxicity in Japanese quail. They enumerated the gross lesions observed in Japanese quail experimentally poisoned with HgCl$_2$ for 8 weeks. The development of lesions throughout the duration of the study as well as the lesions observed after withdrawal of HgCl$_2$ treatment is presented. Chronic dosing of HgCl$_2$ in drinking water at 250 ppm/ml resulted in growth depression and lesions in the liver, kidney, gastrointestinal tract and lymphoid organ including spleen. Upon withdrawal of mercury treatment, scars were still apparent in the liver, kidney, heart and intestine.

Kaprczynski et.al. (1998) studied quail bronchitis and found that the bronchitis viruses first infects lungs and then go to bursa. They made detection of in ovo-inoculated infectious bronchitis virus by immuno-histochemistry and in situ hybridization with riboprobe in epithelial cells of the lung and cloacal bursa.

Nakamura et.al. (1998) studied systemic, amyloidosis in laying Japanese quail. It showed decreased egg production, anorexia, white diarrhea, and subcutaneous abscess of the head. Histologically the amyloid deposited predominantly in the spleen and liver. A lesser degree of amyloidosis in heart and lung was also observed.

Anjali et.al. (1999) presented a study on effect of experimentally induced thiamine deficiency in Japanese quails. They reported mean concentration of RBC and TK activity is lower.
Out break of Septicemic colibacillosis in Japanese quail is reported by Arenas et.al. (1999). They carried out an etiological and pathological study on out break of septicemia caused by the 0165 sero group of *E. coli* in Japanese quail. The found high mortality (90%) and on treatment with amoxicilnine in drinking water (200 mg/l) mortality rate dropped markedly.

Asrani et.al. (1999) have reported infections in quail chicks by oral administration with $14.5 \times 10^8$ *Candida albicans* cells. Mortality up to 18% was noted in infected group given tetracycline added water. The lesions in the gizzard, liver, lungs and heart were noticed in the infected group kept on tetracycline. The lung parenchyma was replaced by diffused granulomatous mass comprising large number of multinucleated giant cells, mononuclear cells and a few heterophils. The adjoining parenchyma was severely congested with slight serofibrinous exudates in the tertiary bronchioles. In heart a small necrotic area infiltrated with macrophages and heterophils was noticed on one quail chick that died at fourth post inoculation day Simultaneous occurrence of granulomatous lesions in the lungs, liver and heart suggested development of systemic infection possibly due to hematogenous spread.

Batta et.al. (1999) carried out experimental studies on clamydiosis in quails. They infected quails with pneumonia viruses (*Lanydia psittaci*) isolated from calf pneumonia, swine pneumonia, goat abortion, sheep abortion, kid enteritis and calf conjunctivitis and found lesions in spleen and lungs.
Corton et al. (1999) reported main lesions in Japanese quail. The pathology of lung samples showed inflammation. Birds were severely emaciated with low body weight, severe atrophy of skeletal musculature and epicardial fat, small dark tanned liver and distended bladder. They also reported presence of coccidia from the same birds.

Premkumar et al. (1999) studied patho-morphological changes in liver, heart, spleen, pancreas, kidney and bursa of fabricia of 36 week old chicks fed diet containing Mm from 1 to 8 week of age.

Jayaramu et al. (1999) reported effect of formaline vapors and formaldehyde fumigation in trachea and lungs of chicks. Histopathologically they found deciliation, degeneration, desquamation and necrosis of pseudo stratified ciliated columnar epithelial cells of trachea. They also observed extensive infiltration of lymphoid cells and heterophils and deposition of fibrin like material in the tracheal lumen and para bronchi of lungs. Lungs showed excessive exudation in the primary and secondary bronchi with segmental to total deciliation and hyperplasia of gabled cells as the concentration of formaldehyde and duration of exposure increased. Degeneration and vacuolation of para bronchi, presence of fibrinous exudates in primary and secondary bronchi was also observed.

Craig et al. (2000) reported pathological lesions in Peking duck (Anas platyrhynchos). They studied the effect of different doses of Riemella anatipeifer (a gram negative bacteria) infection and observed pathological lesions in bursa of fabricius and spleen.
showing varying degree of lymphoidal depletion and necrosis within cortical and medullary region.

**Devendra Rao and Yadgirkar (2000)** described pathology for malathion toxicity in Japanese quail. Malathion is an organo-phosphorus group of insecticide. It is the safest and least toxic insecticide. It is applied to birds against ectoparasites or in feed. They observed mild congestion in pericardium. Heart revealed focal areas of degenerative changes of cross-striations and sarcoysis in cardiac muscle fibers and hemorrhage. The fed level was @ 300 and 450 ppm. Pathological changes in liver, intestine, duodenum and bursa of fabricia are also reported.

**Thakur et.al. (2000)** from Patna (Bihar) carried out study about the pathology of lung in Japanese quail due to natural infections. They reported different types of pneumonic lesions in quail. Suppurative pneumonia was observed in 83(37.05%) cases. Microscopically, scattered alveoli were seen packed with edematous fluid and focal infiltration of large number of heterophils along with mononuclear cells. Lungs of 72 birds (32.14%) showed the tissue changes of hemorrhagic pneumonia. Ruptured blood vessels and air alveoli packed with erythrocytes were noticed. Fibrinous pneumonia was observed in 37 birds(16.52%). Microscopically, there were fibrinous exudates in the intra-alveolar spaces. Lungs of 11 naturally dead Japanese quail (4.92%) showed changes in conformity with chronic interstitial pneumonia in which marked proliferation of fibrous connective tissue in the intra-alveolar spaces was detected. Emphysematous changes were also observed in 10 quails (4.46%). Microscopically the alveolar wall were found to be thin and air...
vesicles appeared severely dilated Lung specimens of 6 quails (2.68%) were having changes similar to chronic non-suppurative pneumonia. There was infiltration of mononuclear cells in the para and meso-bronchial spaces. There was marked proliferation of connective tissue in the widened inter-alveolar spaces. Granulomatous changes were detected in 5 quails (2.23%). Histopathologically, section revealed central necrotized area in the lung parenchyma surrounded by infiltration of mononuclear cells. According to them reasons for occurrence of such high percentage of pneumonia was probably the unhygienic management of farm and conducive climate of Bihar.

Yasunori et al. (2000) carried out microscopical, immunochemical and morphological study on pulmonary neuro-epithelial endocrine system in quails and reported cell apoptosis and emphysema (loss of alveolar structure) due to inhibition of VEGF receptors in quails.

Genchev et al. (2001) carried out study on effect of availability of Datura Stramoni (thorn-apple) seeds on the general condition and performance of Japanese quail. They reported the effect on blood characters and the development of internal organs. They examined 140 quails, (4 groups of 35 quails in each) fed with Datura stramonium seed in their daily ration at different % from 7 to 35 days of age (0, 0.16%, 0.25% AND 0.33% Dautra seeds in feed). They analyzed the total erythrocyte count total blood protein, total blood albumin and globulin. They also reported some pathological lesions. The alkaloid concentration in the intestine and liver in the birds from third and fourth group (i.e. at higher doses) resulted in a degree of liver dystrophy, cardiac hypertrophy, follicular activity in
the ovaries and hypertrophy of seminal ducts accompanied by absence of spermatogenesis.

Jakhar and Sadana (2001) reported the effect of selenium supplementation on the growth response in experimental aflatoxicosis in Japanese quails and found a significant decrease in weight ($P < 0.05$), dullness, depression and anorexia and reduced feed intake. They also reported fatty changes in liver, hemorrhage in liver and in myocardium.

Perkins and Swayne (2001) studied pathology of pheasants, chicks, pigeons, ducks and quails due to A chicken Hong Kong 220/97 (H5N1) avian influenza virus. Quails showed mild mortality (14%). The gross lesions observed by them in quail were splenomegaly with parenchymal molting (in about 42% quails), pulmonary consolidation with edema and congestion to hemorrhage (in 92% quails). Larynx, trachea and air sacs do not showed any significant lesions. However the laryngeal epithelium and bronchioles were associated with lymphoid tissue containing minimal to mild apoptotic lymphoid depletion, heterophilic inflammation and mild degenerative changes of overlying epithelium. Cardiovascular system showed acute inflammation in all species including quails. Endothelial cells were hypertrophied or swelling accompanied by heterophilic parementing to vascular exocytosis, identified in capillaries of lung and spleen. Endocardial hemorrhage, rapid multifocal to confluent myocardial degeneration and necrosis was observed.
Mu
tinelli et.al. (2002) presented clinical, gross and microscopic findings in different avian species, including quails, naturally infected by H7N1 low and high pathogenicity avian influenza epidemics in Italy during 1999 and 2000. In general the clinical signs in younger birds evolved into a more severe respiratory alteration which in some cases resulted in and subcutaneous emphysema and was associated with mortality rates ranging from 40% to 97%. The most striking post mortem lesions were the presence of a fibrinous clot in the sinuses and trachea and lungs were congested and in some cases hemorrhagic. Petechial hemorrhages were also observed in some cases on the epicardium and on the cecal tonsils. In quails and layers the disease moved much slowly within the flock. They also reported fibrinoid necrotic foci in spleen. Spleen appeared reduced in size. Heart appeared congested and enlarged.

Murakami et.al. (2002) reported their findings on occurrence of conjunctivitis sinusitis and upper region tracheitis in Japanese quail. Mortality rate was 5.7% per day. They reported caseous exudates and air sacculitis. Microscopically non-purulent or purulent inflammation accompanied by lymphoid hyperplastic tissue. With germinal centers was observed by them in the occlusofacial respiratory mucosa. They investigated pathogen as *Mycoplasma gallisepticum* and *Pasteurella multocida* serotype D.

Sunilkumar et.al. (2002) described the sequential pathological changes in Japanese quail experimentally infected with *salmonella typhimurium*. 100 quail chicks were randomly divided into two groups A and B. Birds in group A were infected with 1 ml NSS
containing 2 X 105.5 organisms of 18h broth culture of *S. typhimurium* where as birds in group B were given sterile NSS and served as control. These birds were scarified at different intervals of days after inoculation to study sequential gross and microscopic changes in different organs. Salient gross changes included fibrinous pericarditis, splenomegaly, hepatomegaly, and pale granular appearance of liver. Histo-pathologically, there was fibrinous pericarditis, fatty changes with necrotic foci and granuloma formation in liver, patchy pneumonia and lymphoid nodules in lungs, degeneration with increased inter-follicular connective tissue in bursa, hyalinization of lymphoid follicles and RE cell hyperplasia in spleen and catarrhal enteritis.

A study of mycobacteriosis is presented by Tell *et.al.* (2003). They studied histo-pathology in Japanese quail intravenously inoculated with *Mycobacterium avium*. Abnormal clinical findings were decreased activity, feather erection and sudden death. Mean body weight and packed cell volume declined. Mean total white blood cells (heterophils bands and monocytes) increased from 28 days post inoculation onward. Pathological findings were severe hepatomegaly, lesions in liver, spleen intestine, lungs and gonads. Less affected tissues were gizzard, heart, pancreas and brain. *Mycobacterium avium* was isolated from liver, spleen & intestine

Bell *et.al.* (2003) studied ulcerative enteritis on Bobwhite quails, chickens and Turkey. They reported that quails showed acute susceptibility to ulcerative enteritis showing 100% mortality. Primary lesions (punctuate of ulceration), and hemorrhage were seen in
lower third of small intestine, ceca and liver (necrotic foci). Besides these the lesions were also seen in spleen which may be enlarged and either hemorrhagic or necrotic. They isolated *Clostridium colium* however in differential diagnosis histomoniasis and coccidiosis caused greatest problem.

**Kwon et.al. (2003)** studied gross and microscopic lesions in Baikal Teals (*Anas Formosa*). They reported out break of fowl cholera from Korea. Gross lesions were multifocal necrotic foci in liver with enlargement, petechial or ecchymotic hemorrhage on heart and mucoid exudates in duodenal mucosa. Microscopically hepatic necrosis, hemorrhage and necrosis in myocardium, hemorrhagic enteritis and fibrinous exudates in para bronchial lumen were observed.

**Shrilatha et.al. (2003)** from Tirupati reported pathological studies of pasturellosis. The causative organism reported is *Sellmonella pasturella* yielded from liver and heart of Japanese quail. Mortality rate observed was 33.7% in the month of October. They reported hemorrhage in heart, spleen and congested lungs along with liver necrosis and hemorrhages in intestinal mucosa in Japanese quails.

**Fethy Yilmaz et.al. (2004)** worked on pathology of Japanese quails experimentally infected with avian influenza A virus H7N1 Subtype. They reported that the virus occur in many avian species such as turkey, geese and pheasants, but develops more rapidly in Japanese quails as compared to other avian species and wild quails might have played role in spread of the disease (Perer et.al 2003). They found decreased food and water consumption.
calmness, reluctance and diarrhea in some birds. Macroscopic findings reported by them were mild in severity and were observed mostly in respiratory system, liver, heart and intestine. The trachea and upper respiratory tissue were congested and had accumulation of mucus or exudates. It was seen that bilateral ventral part of the lungs were covered with slightly dark focus and had edematous appearance. There were sub-epicardial and in some cases sub-endocardial hemorrhages in the heart, and approximately 1 ml liquid was observed in the pericardium. Microscopically, the tracheal mucosa was inflamed as characterized by congestion, edema and deciliation, necrosis and detachment of epithelial cells. Severe infiltration of lymphocyte, macrophages and infrequently heterophils were observed in the lamina propria, which increased the thickness of tracheal mucosa. Mild to moderate interstitial pneumonia was a common lesion in quails. Infiltration of macrophages lymphocytes and few heterophils were seen in addition to hyperemia, edema and hemorrhages in lungs. In the epithelia of the primary bronchi, degeneration, desquamation and focal hyperplasia as well as peribronchial edema and congestion were notable. The virus produced focal lymphohistocytic myocarditis with myocytic degeneration and necrosis. Myocardial hyperemia and hemorrhages were found in the sub-epicardial region. No substantial macroscopic and microscopic findings were determined in the spleen and air sacs.

Jakhar and Sadana (2004) from Hissar, Haryana, studied sequential pathology of experimental aflatoxicosis in quail and the effect of selenium supplementation in modifying the diseases process. Feeding of aflatoxin B1@ 1 PPM to 2 week old Japanese quail for a period of 8 weeks produced gross and microscopic
changes in the liver, skeletal muscles, heart and bursa fabricia. These included fatty changes, bile duct hyperplasia and lymphoid aggregation in liver, hemorrhages in thigh, breast muscles and myocardium, mild depletion of lymphocytes, cystic degeneration and fibrous tissue proliferation in bursa of fabricius. More or less similar lesions were seen in quail chicks fed on aflatoxin with sodium selenite @ 5 PPM but these were of lesser intensity and appeared at later stages of the experiment, thereby indicating that supplementation of selenium had some protective action against the toxic effect of aflatoxin B1 in Japanese quail.

Ozbey and Ozcelik (2004) carried out experiment on the effect of high temperature on growth performance of Japanese quails with different body weights. In this study they determined the effect of high environmental temperature on live weight, feed consumption, feed efficiency, survivality and some carcass features of Japanese quails with different body weights. During this work, total 550 quails chicks were used in addition, two different temperature groups as being the control (18-24°C) and experiment (35°C) groups and two different body weight groups consisting of heavy (live weight > 27 g) and light (live weight < 27 g) groups were identified. As the result of research, the live weight, feed consumption, feed efficiency and survivality were found less at 35°C compared with 18-24°C. in both heavy and light weight groups. Such decrease was noted as more efficiently in heavy groups.