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REVIEW ARTICLE

# Poultry diseases of viral origin causing neoplasia and production losses: an overview

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**Abstract:** The economically important neoplastic diseases of poultry are caused by viruses. These diseases are responsible for economic loss due to both mortality and depressed performance. Marek's disease virus (MDV), avian sarcoma virus (ASV) and avian leukosis virus (ALV) are prevalent throughout the world and demands control strategies for implementation.

**Key words:** Economic losses; Neoplasia; Poultry diseases

## Introduction

Marek's disease virus speads through horizontal transmission and hygienc practices should be implemented to avoid the spread of the disease among hatching eggs and day-old chicks. Transmission of ALV and REV occurs both horizontally and vertically (through the egg), and measures to prevent international spread are more demanding. Marek's disease is controlled by vaccination. Avian leukosis can be controlled by virus eradication programmes, mainly at the primary breeding level. However, still there are insufficient or no strong research reports to comment that these tumor diseases in poultry poses to be a public health hazard.[1]

### Marek's disease

Marek's disease is caused by DNA virus belonging to the subfamily *Alphaherpesvirinae* under the family *Herpesviridae*. It occurs in four clinical forms known as neurolymphomatosis, acute Marek's disease, ocular lymphomatosis and cutaneous Marek's disease.[2]

Peripheral nerves are enlarged and infiltrated with immature mononuclear cells. The virus matures in feather follicle cells, sheds into environment and transmits the disease to healthy birds through respiratory secretions. Many apparently healthy birds are lifelong carriers and shedders of the virus. There is no transmission through eggs. Tumor material containing the virus can also transmit the disease. As the virus is cell associated, cell free filtrate does not transmit the disease. In cutaneous form, there are nodular lesions particularly at the feather follicles. There are lymphoid tumors in gonads, lings, liver and kidneys. Affected organs become enlarged and pale due to infiltration of lymphocytes. In the clinical forms of neurolymphomatosis, there is progressive paralysis of one or more limbs, incoordination, drooping of limbs and head. Blindness occurs in ocular lymphomatosis. Chickens infected with MDV produce humoral and cell-mediated immunity. The disease commonly occurs in 2-4 months old chicken. Only a few percentages of infected chickens develop clinical form of the disease. There is depression in acute cases.[2,3]

In lymphoid cells, there is transformation of the virus resulting in proliferation of cells without cell death. No virus is produced in these cells. The infected cells contain Marek's associated tumor specific surface antigen (MATSA). In transformed cells, MATSA can be detected by immunodiffusion and immunoflourescent techniques using serum prepared with transplantable tumor cells. In non-lymphoid cells, either there is productive infection resulting in the production of infective virion as in case of feather follicle cells or production of non-infectious virion.

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The non-infectious virion produces intranuclear inclusions and causes cell death.[2,7]

Viruses belonging to the family *Retroviridae* cause avian leukosis and avian sarcoma disease in chickens which is usually characterized by lymphoma, leukemia, anemia and osteopetrosis.
For avian leukosis and sarcoma, three classes of viruses are involved[2]:

**Endogenous viruses:**
These viruses occur in the genome of chicken as DNA provirus. Generally, they are not expressed and when expressed they are non-pathogenic.

**Exogenous viruses which are competent for replication:**
They possess *gag*, *pol* and *env* oncogenes in viral genome. Generally, these viruses are non-pathogenic. Some of these exogenous viruses acquire *c-onc* (cellular oncogene) and then rapidly induce malignant tumors. A small percentage of birds infected with this virus may develop lymphoma or leukaemia.

**Exogenous viruses which are defective for replication:**
Some of the rapidly transforming viruses acquire the oncogene (*gag, pol, env*) by loosing part of their genome and become replication defective. They require a helper virus for their replication.

Avian leukosis and avian sarcoma viruses induce variety of transmissible neoplasms. ALV predominantly affects haemopoietic cells and ASV produces pathogenic response in fibroblastic cells. Both the viruses have common internal antigens. Transmission of disease mainly occurs vertically, but horizontally also in birds in close contact. Cellular response in avian leucosis is predominantly of B-cell type. Removal of bursa prevents lymphoid leucosis. Major internal antigen is present in supernatant and in the cells of infected cultures which can be detected by Resistance inducing factor (RIF) and complement fixing avian leucosis (COFAL) tests. Marek's disease is RIF negative and lymphoid leucosis is RIF positive. Eggs for human vaccine production must be free from RIF negative hens. In both viruses, tumor specific cell surface antigen (TSSA) in present. In sarcoma virus transformed cells, tumor specific transplantable antigen (TSTA) has been demonstrated. There occurs some degree of immunosuppression due to both these viral diseases. These viruses can be isolated in cell cultures from plasma, serum, tumor masses or embryos of infected chicken. Rous sarcoma virus induces pocks on CAM of infected eggs and produces tumors in embryos by intravenous or yolk sac inoculation. Leucosis virus replicates in chicken embryos by intravenous route and kills chicks in 1-2 weeks after hatching.[2,4]

Exogenous non-defective avian retroviruses produce tumors, only in birds that are congenitally infected and have a persistent viraemia. There is high probability of lymphoid cell infection and transformation by leukosis virus. These viruses produce the following types of disease[2,5-7]:

**Lymphois leukosis (Visceral lymphomatosis):**
It is the common form of avian leukosis and occurs mostly in 4-8 months of birds. The comb may become pale and shriveled. Tumors may be present in liver, spleen bursa and other organs. Removal of bursa in early age arrests the development of lymphoid leukosis. The development of lymphoid leukosis is slow. There is lack of feed intake, weakness, emaciation and enlarged abdomen. Leukemia is not very common. Lymphoblastoid cells are rarely found in blood circulation. The disease is popularly called big liver disease among farmers. In affected birds, the lymphoblasts may secrete large amount of IgM.

**Osteopetrosis (thick leg):**
In this form of disease, bones are affected by uniform of irregular thickening. Generally, long bones are affected. In advanced cases, there is osteoma and osteogenic sarcoma. The birds may have anemia and lesions of lymphoid leukosis.

**Renal tumors:**
When renal cells are affected, nephroblastoma may develop which can be diagnosed during necropsy of the birds. The defective avian leukosis viruses replicate with the help of non-defective leukaemia viruses (generally exogenous type) and produce a variety of neoplasms. These viruses produce three types of diseases:

**Erythroblastosis:**
It occurs on two forms: Proliferative and anemic forms. In the former, the virus propagates rapidly and large numbers of erythroblasts are found in the blood circulation. In anemic form, there is anaemia and only a few circulating erythrocytes.

**Myeloblastosis:**
In this syndrome, large numbers of myeloblasts are present in the blood. The symptoms are similar to erythroblastosis. In this disease, leukaemia is a major feature. The incubation period is about 10 days.

**Myelocytomatosis:**
Rate of the development of disease is rapid. Various mesenchymal cells are affected and lesions produced are those of sarcoma. The target cells are non-granulated myeloblasts. The symptoms are similar to erythroblastosis. Tumor growth may extend up to bones.

Avian leukosis can be diagnosed by the history of the disease in the flock, clinical symptoms, gross

and histopathological findings. Differential diagnosis should be made with Marek's disease.[2,7]

## Conclusion

The disease can be controlled by applying strict hygienic measures, elimination of infected flocks and starting with a new genetically resistant strain. Immunization using inactivated or attenuated live virus vaccines provides limited success.

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