Retroviridae

Retroviruses are enveloped, single stranded RNA viruses that replicate through a DNA intermediate (provirus) using an RNA-dependent DNA polymerase (reverse transcriptase (RT)). Within a cell the retroviral RNA genome is reverse transcribed into a DNA copy, and it is the proviral DNA form that serves as the intracellular retroviral genome. The proviral DNA is covalently integrated in the DNA of the infected host cell. Highly oncogenic retroviruses often have an oncogene. The integration of retroviruses is not at a specific site within the cellular DNA, rather integration can occur at many sites. The integrated DNA provirus act as a eukaryotic gene. It may be transcribed into mRNA and genomic RNA using host cell enzymes to produce more virus or it may remain latent for long periods of time and replicate when the cellular DNA is replicated by the cell.

Oncogenesis by Retroviruses

In oncogenic retroviruses, all or part of an oncogene exists in the viral genome, usually in place of viral genes. This retroviral oncogene is responsible for the ability of oncogenic retrovirus to cause oncogenic transformation of a cell. Each of these has a corresponding gene that can be found in the genome of normal cells. The normal gene that corresponds to a viral oncogene is termed a c-oncogene and the viral version is called a v-oncogene.

Classification

Depend on the viral transmission there are two types:

1- **Exogenous retroviruses** spread horizontally from animal to animal, similar to the mechanism of transmission of other kinds of viruses.
2- **Endogenous retroviruses** are transmitted vertically from fathers to offspring. The endogenous retroviruses persist as integrated DNA proviruses that are passed from generation to generation through the DNA in the gametes of the host animal species. Thus, the endogenous proviral genome occurs in each cell of the animal.

Depend on the host there are three types:

1- **Ecotropic** strains replicate only in cells from animal species of origin.
2- **Xenotropic** strains replicate only in cells of other species. Most of the endogenous retroviruses are also xenotropic.
3- **Amphotropic** strains replicate in both.

Avian Leukosis (lymphoid leukosis)

lymphoid leucosis, the most common and economically important disease caused by avian leukosis virus (ALV), the comb may be pale, shriveled, and occasionally cyanotic. Inappetence, emaciation, and weakness occur frequently. Enlargement of the liver, bursa of Fabricius, kidneys, and the nodular nature of the tumors can sometimes be detected on palpation.

Etiologic Agent

ALVs are classified into five subgroups, A–E, on the basis of differences in their viral envelope glycoprotein antigens

Distribution, Reservoir, and Transmission

ALVs occur naturally in chickens and most flocks of chickens worldwide harbor various strains of ALV. Even in infected flocks, the frequency of lymphoid tumors is typically low and mortality is usually 2% or less. The reservoir host for ALV is the infected chicken. Transmission can be either vertical (from hen through egg) or horizontal. Vertically infected chicks are immunologically tolerant to the virus and fail to produce neutralizing antibodies, and remain viremia for life. Horizontal infection is through infected saliva and feces and is characterized by transitory viremia followed by the development of antibodies. Tumors are more frequent in vertical than horizontal infections.

Pathogenesis and Pathology.

Under natural conditions, the most common disease caused by ALV is lymphoid leukosis. Transformation of lymphocytes occurs in the bursa of Fabricius, usually at a few months after infection. These early ALV-induced lesions sometimes regress, while others enlarge and eventually spread to other visceral organs. Grossly visible neoplasms are of variable size and organ distribution, almost always involve the liver (a synonym for lymphoid leucosis is big liver disease), spleen, and bursa of Fabricius.

Host Response to Infection

Chickens exposed to ALV virus fall into four classes:

(1) No viremia with no antibodies: includes birds that are genetically resistant.

(2) No viremia with antibody: most exposed chickens are included in this category, and antibody persists throughout the life of these birds and is passed via the yolk to progeny chicks. The passive immunity provided by

such antibodies generally lasts for 3–4 weeks. Although virus-neutralizing antibodies restrict the amount of virus, they have little direct effect on growth of the virus induced neoplasms.

(3) Viremia with antibody: Few chickens occur in this category, which may represent chickens that are in the process of clearing an acute infection with ALV.

(4) Viremia with no antibody: Most chickens in this category acquire ALV vertically when in the egg and are immunologically tolerant to the virus. Hens transmit virus to a high proportion of their progeny through the egg.

Laboratory Diagnosis

ALV can usually be isolated from plasma, serum, tumor tissue, and albumin, or from the embryo of infected eggs. Since ALV is generally not cytopathogenic, complement fixation, fluorescent antibody, or radioimmunoassay tests must be used to detect and identify the viruses in cell culture. An ELISA test is used for direct detection of virus in egg albumen or vaginal swabs.

Treatment and Control

Attempts to produce effective vaccines have been largely unsuccessful. It is possible to eradicate ALV from chickens by establishing breeder flocks that are free of exogenous ALVs.