GENERAL AND SYSTEMIC VIROLOGY
(MICRO – 303)

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BEST OF LUCK !!!

RETROVIRIDAE

The name '*retro'* is derived from the Latin word retro means "*backward*". The unique feature of the viruses of this family is the presence of *reverse transcriptase* enzyme.

Classification

The family contains three important families as listed follows;

Orthoretrovirinae

Genus	*Viruses*
Alpharetrovirus	Avian leukosis virus, avian sarcoma virus, avian myeloblastoma virus
	Rous sarcoma virus
Betaretrovirus	Murine mammary tumor virus
	Jaagsiekte sheep retrovirus
Gammaretrovirus	Murine leukemia virus, Feline leukemia virus
	Avian reticuloendotheliosis virus
Deltaretrovirus	Bovine leukemia virus
	Human T-lymphotropic viruses 1, 2
Epsilonretrovirus	Walley dermal sarcoma virus
	Fish tumor virus

Lentivirinae

Lentivirus	Human immunodeficiency viruses 1,2
	Simian, Feline, Bovine, immunodeficiency viruses
Spumavirinae	
Viruses causing vaculation of cultured cells, not associated with clinical disease	
Spumavirus	Human, Simian, Bovine, Feline Foamy viruses
	Sealion spuma virus

General Properties

- Viruses are enveloped, roughly spherical, with linear, positive sense single stranded RNA genome. The virions measure 80-130 nm in diameter.
- The helical nucleoprotein complex includes about 30 molecules of reverse transcriptase.
- The envelope of the viruses contains a lipid bilayer and glycoprotein spikes.

Difference from all other viruses

- Retroviruses differ from all other viruses in having a diploid (inverted dimmer) genome, which means that their *two RNA molecules* are identical.

Structural Characteristics

- The RNA of *non-defective* (capable to replicate) *retroviruses* contains four major genes;
  i) ***gag*** (group specific antigen) encodes for internal structural proteins.
  ii) '***pro'*** (protease) encodes for the enzyme protease.
  iii) '***pol'*** (polymerase) gene encodes for reverse transcriptase (RT), and integrase.
  iv) '***env'*** (envelope) encodes surface (su) and transmembrane(TM) glycoproteins.

- Envelope glycoproteins (su,TM) attach to specific cell receptors before virus enters cell.
- Under the influence of the RT, dsDNA copies of viral genome are formed in the cytoplasm of the cell.
- During this process repeat base sequences (several hundred basepairs called long terminal repeat LTR) are added to the ends of the DNA transcriptase.
- These transcripts (provirus) are added into the chromosomal DNA at random sites with the help of "*integrase*" causing change in cellular gene.
- The site of provirus integration determines the extent and nature of cellular changes. If the provirus of certain retrovirus is inserted close to the host genes which regulate cell division, the *proviral* LTR may increase the rate of mitosis resulting in neoplasia (insertional mutagenesis).

1. ALPHARETROVIRUS

AVIAN LEUKOSIS

Causative Agent:

- Avian leukosis virus, belongs to the genus "*Alpharetrovirus*" of the sub-family "*Orthoretrovirinae*" of family "*Retroviridae*".

Disease Properties:

- The term "*Avian leukosis complex*" is generally used to describe a number of contagious conditions of poultry which are characterized by neoplasms of the haemopoietic system. These include avian sarcomas, lymphoid leukosis, myeloblastosis, erythroblastosis and, probably osteopetrosis.
- It is not clear whether the above conditions are manifestation of a single disease or if they are caused by different but closely related species of oncornaviruses.

Distribution and Species Affected

- The avian leukosis complex is one of the most important disease problems of poultry, in all countries of the world.
- Spontaneous occurrence of the disease is almost entirely confined to domestic poultry (sometimes turkey) over 4 months old. The economic loss is very much high.

Pathogenicity:

- A number of factors influence the onset and development of avian leukosis namely:
  1) Genetic factors (some breeds are resistant while other are highly susceptible)
  2) Age at exposure (young are more susceptible than the adults)
  3) Sex (females are more susceptible than males)
  4) Strain of the virus involved (some are more pathogenic and others are less)
- Most birds in a diseased flock become infected and may remain symptomless carriers for life. Birds may die suddenly or the disease may run a protracted course with loss of bodily condition.
- Affected birds are pale and listless and may show diarrhea in the terminal stages of the illness.

- At autopsy, the disease is characterized by enlargement of the liver, spleen, kidney and gonads, with diffuse or military lesions due to massive infilteration of the tissues by lymphoid or other types of tumor cells.
- Virus is excreted in the saliva and droppings of both clinically affected and apparently healthy carrier birds. Contact spread is highly efficient among younger susceptible birds and can even occur by airborne transmission.

Ecology:

- Birds in an infected commercial flock may be divided into four broad groups:
  1) Non-infected immune birds: adult birds which do not carry the virus because they have developed neutralizing antibodies as a result of previous exposure to the virus.
  2) Infected immune birds: these are mostly viremic adult hens which have detectable amount of neutralizing antibody in their sera and are immune.
  3) Tolerant viraemic birds: because viraemic hens frequently carry an ovarian infection and pass the virus to their offspring via egg, the chickens developing from these congenitally infected embryos have large quantities of infectious virus in their blood and tissues and are immunologically tolerant. Since large amounts of virus are shed in their droppings and saliva, they remain a constant source for their horizontal spread of the virus.
  4) Non-infected non-immune birds: chicks from immune hens are virus-free upon hatching and are partially protected against contact infection for the first few weeks of their lives by passively transferred maternal (yolk antibody). At maturity, these birds are non-infected and non-immune, and a high incidence of contact infection usually results because of the presence of viraemic birds in the same flock.

Diagnosis:

- The distinct character and distribution of gross lesions in affected carcass is usually sufficient for a diagnosis of avian leukosis.
- Confirmation is based on histological examination and on the identification of the virus. The causative virus can be isolated from the blood and most soft organs by inoculation into susceptible chicks, susceptible embryonated hen's eggs and susceptible chick embryo cell cultures.
- Cell cultures tests include the following:

RIF test: inoculated embryo fibroblasts are later tested for their susceptibility to Rouse sarcoma virus. The presenc of leukosis virus is indicated by ten-fold reduction in the number of foci produced by a standard strain of Rouse sarcoma virus.

COFAL test: The COFAL reaction (*Complement fixation test avian leukosis virus*) is dependant on the formation of the group-specific protein antigen of avian leukosis virus in the infected cell cultures. Infected chick embryo fibroblasts are harvested and their fluids used as an antigen in the CFT. Samples which fix complement in the presence of a known antiserum are considered to be positive.

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