PARAMYXOVIRIDAE

The paramyxoviruses were formerly grouped together with orthomyxoviruses as myxoviruses because of their affinity for the mucous membranes. Members of the family are pleomorphic viruses of about 150nm or more in diameter, they are enveloped and the genome is a single molecule of single-stranded of RNA. Generally, two types of glycoprotein spikes, which can induce the production of virus neutralizing antibodies in infected host, project from the envelope. The glycoprotein spikes are:

i. Attachment protein (F). The attachment protein mayeither be a haemagglutinin-neuraminidase protein (HN) or a protein without neuraminidase activity (G). the attachment proteins allow the virus to bind to cell surface receptors
ii. Fusion protein: This causes the virus envelope to fuse with the host cell membrane

Paramyxoviruses also possess a non-glycosylated envelope-associated membrane protein (G). they may exhibit haemagglutinating, haemolytic and neuraminidase activities. The nucleocapsid is about 13 to 18nm in diameter and possesses a helical symmetry with characteristic herring-bone appearance. Replication takes place in the cytoplasm of the host cell and mature virions are released by budding from the plasma membrane at the sites containing virus envelope proteins. Virions are very labile and sensitive to heat, desiccation, lipid solvents, non-ionic detergents and disinfectants.

Classification:

Order: Mononegavirale

Family: Paramyxoviridae

Subfamilies:

1) Subfamilies: 2)	3) Genera	4) Viruses
a. Paramyxovirinae 5)	6) i. Respirovirus	7) Bovine parainfluenza virus
	8) ii. Morbillivirus	i. Rinderpest virus ii. Peste des petits ruminants virus iii. Phocine distemper virus iv. Canine distemper virus v. Cetacean morbillivirus vi. Equine morbillivirus
	9) iii. Rubulavirus	i. Newcastle disease virus ii. Avian paramyxoviruses 2 – 9 iii. Canine parainfluenza virus 2 iv. Porcine rubulavirus
b. Pneumovirinae	10) i. Pneumovirus	11) Bovine respiratory syncytial virus 12)
	13) ii. Metapneumovirus	14) Turkey rhinotracheitis virus

Clinical infections caused by paramyxoviruses:

Generally, paramyxoviruses have narrow host range. They infect mainly mammalian and avian species. Transmission is by close contact and by aerosol. They have high affinity for the respiratory tract where replication usually takes place. Infection is generally cytolytic. Formation of syncytia and intracytoplasmic, acidophilic inclusion bodies are prominent features of paramyxovirus infections.

RINDERPEST

Rinderpest has for centuries been recognized as a major cause of morbidity and mortality in cattle and domestic buffalo. It is also called 'Cattle Plague'. The disease is endemic in parts of Africa, the Middle East and Asia. It is said to have been eradicated in Nigeria. It is caused by Rinderpest virus. Only one serotype of Rinderpest virus is recognized. Strains of the virus differ

in both host range and virulence. Susceptible animals include cattle, buffalo, giraffe, cape buffalo, eland, and warthog. Gazelle and small domestic ruminants (sheep and goats) are less susceptible.

Transmission occurs through close contact and aerosol. The virus is labile and survives for short period outside of the host. Epidemic is associated with movement of susceptible animals to endemic area or the introduction of infected animals into susceptible populations. All ages of animal are susceptible. Morbidity may reach 90% and mortality close to 100%.

Clinical signs: include fever, anorexia, depression, erosion of the oral and respiratory tract mucosae, profuse salivation, oculonasal discharge, profuse diarrhea (dark watery faeces containing mucuc, necrotic debris and blood), dehydration, wasting, collapse and death within 12 days of the onset of clinical signs.

Diagnosis:

- Clinical signs and pathological findings may be suggestive.
- Differential diagnoses include bovine viral diarrhea, infectious bovine rhinotracheitis, malignant catarrhal fever, foot and mouth disease.
- Postmortem enteric lesion: congestion of the folds of the colonic mucosa often produces a Zebra-stripe pattern
- Syncytia formation in stratified squamous epithelium of the upper alimentary tract and in crypts of the small intestine
- During an outbreak, samples for laboratory diagnosis should be collected from febrile animals which have not developed diarrhea. Suitable specimens include white cells from the buffy coat of heparinized blood samples, lymph nodes and spleen
- Rinderpest virus is detectable in tissue by immunofluorescence tests
- Agar gel immunodiffusion or a counter immunoelectrophoresis test can be used for rapid antigen detection. Ocular discharges and mesenteric lymph nodes are suitable specimens for these procedures
- Reverse transcription polymerase chain reaction method can detect rinderpest virus and differentiate it from PPR virus
- Competitive ELISA for detection serum antibodies to rinderpest virus

Prevention and Control:

- Restriction of movement
- Quarantine of imported animals
- Slaughter of infected animals
- Vaccination: modified live tissue culture rinderpest vaccine confers immunity lasting at least five years. The vaccine is easily destroyed by heat after reconstitution
- Recombinant vaccinia and capripox virus vaccines expressing either haemagglutinin protein or fusion protein of rinderpest virus have high heat stability

PESTES DES PETITS RUMINANTS

This disease is also called goat plague. It is an acute contagious disease of ruminants especially goats. It is caused by the virus pestes des petits ruminant virus; a morbillivirus. Members of the morbillivirus are closely related and induce similar clinical manifestation in affected host. The disease occurs in sub-saharan Africa, Middle East, India and Pakistan.

Transmission: close contact is required for aerosol transmission. The virus is very labile.

Clinical signs: In Nigeria, Epizootic of the disease occurs during the rainy season when goats are gathered together for sale. Similar but less severe clinical infection also occurs in sheep. The disease is more severe in young animals. There is fever, dry muzzle, serous nasal discharge that become mucopurullent with secondary bacterial infection. There is erosion of the mucous membrane of the buccal cavity and marked salivation. Signs are similar to those seen in rinderpest.

Diagnosis:

Samples should be collected at the acute phase of infection. Samples to be collected include nasal and ocular swabs, unclotted blood, scrapings of buccal and rectal mucosae in live animals as well as lung, spleen and lymph nodes from animals slaughtered at the acute phase of infection. Laboratory confirmation is based on virus isolation in tissue culture and on antigen detection. Rapid antigen detection is by ELISA, counter immunoelectrophoresis and agar gel immunodiffusion. Antibody detection in serum sample is by virus neutralization and competitive ELISA. Primers are available for the detection of PPR virus nucleic acid.

BLUE EYE DISEASE IN PIGS

This disease is caused by porcine rubulavirus. It is characterized by neurological signs, corneal opacity and reproductive failure. Morbidity and mortality are highest in young pigs.

Diagnosis: Presumptive diagnosis is based on clinical signs and confirmation is by laboratory investigations. Serological testing of paired sera sample to demonstrate fourfold rise in antibody level signifying on-going infection is carried out. Antibody detection is by haemagglutination inhibition test, ELISA and virus neutralization test.

BOVINE RESPIRATORY SYNCYTIAL VIRUS

This virus causes pulmonary disease in cattle, sheep and goat. It induces characteristic syncytial in infected cells *in-vivo* and *in-vitro*. The disease is seen most commonly in animals between three and nine months old. in adults, the disease is mild and usually subclinical. Severe disease rarely occurs in adults.

Diagnosis:

- Specimens include nasal swabs, bronchoalveolar lavage fluid, lung tissue and paired serum samples.
- Since the virus is thermolabile, specimens must be transferred to the laboratory in suitable transport medium
- Commercial ELISA kits are available for the detection of viral antigen
- Immunofluorescence is a rapid and useful technique for antigen detection
- Viral antigen can be detected more rapidly in specimens from the lower respiratory tract than in nasal swabs
- Suitable serological test for demonstrating rising antibody titre include virus neutralization and ELISA. Serum samples should be taken early in the course of the disease as the antibody levels tend to rise rapidly.
- Polymerase chain reaction can be used to detected viral RNA

Control:

- Reducing stress factors within cattle herd
- Maintenance of good hygiene
- Separating calves from among older age groups
- Vaccination with modified live and inactivated vaccine: duration of protection is short therefore frequent booster dose is required

CANINE DISTEMPER

This is a highly contagious disease of dogs and other carnivores caused by Canine distemper virus, a morbillivirus in the family Paramyxoviridae. Canine distemper virus is a pantropic virus that produces generalized infection involving many organs of the body. Only one serotype of the virus is presently recognized. However, a variety of biotypes exist that vary greatly in pathogenicity and tissue tropism within the CNS. Antigenic differences have not been detected among CDV strains using such tests virus neutralization, complement fixation, antigenic precipitation and immunofluorescence. There is evidence of cross-reactivity of CDV with measles virus, rinderpest virus and Peste des petits ruminants virus.

Clinical Signs: incubation period is about one week but may be up to four weeks or more with sudden appearance of nervous signs without prior evidence of infection. Infection spreads among young dogs aged between three weeks and six months about the time of decline of maternally derived passive immunity. Affected animals may manifest biphasic fever, oculonasal discharge, pharyngitis, tonsillar enlargement, coughing, vomiting, diarrhea, skin rashes and pustules, hyperkeratosis of nose and footpad (hard pad), neurological signs such as paresis, myoclonus, epileptiform seizures and death. Manifestation of neurological signs is of grave prognosis. Recovered animals may show residual neurological abnormalities.

Diagnosis:

- Clinical signs may be suggestive.
- Viral antigen may be demonstrated by immunofluorescence in conjunctival or vaginal impression smears or in smears of cells from the buffy coat
- Cryostat section of lymph nodes, urinary bladder and cerebellum are also suitable for the demonstration of viral anigen
- Eosinophilic inclusions can be demonstrated in nervous and epithelial tissues
- Serological demonstration either of IgM antibodies or of a fourfold rise in antibody tire between acute and convalescence sera may be determined by virus neutralization test, ELISA, or indirect immunofluorescence. Antibody may be detected in cerebrospinal fluid
- Virus isolation may be attempted from urinary bladder, cells from the buffy coat and brain tissue. Virus isolation may prove difficult

Control: modified live vaccines are available commercially and it provides adequate protection when administered to puppies after the decline of maternally derived antibody. In endemic areas, pregnant bitches may be vaccinated to offer passive protection to their puppies for the first few weeks of life.

NEWCASTLE DISEASE

Newcastle disease occurs in poultry birds worldwide. It is caused by Newcastle disease virus designated avian paramyxovirus 1 (APMV-1). Avian paramyxovirus 1 is antigenically related to the virus of Mumps. A large number of avian paramyxovirus (APMV) isolates has been recovered from a range of domestic and wild birds worldwide. Nine species of antigenically distinct APMV are currently recognized in the genus Rubulavirus within the family Paramyxoviridae. Avian paramyxovirus 2 and 3 are associated with respiratory disease in turkeys. Newcastle disease manifests either in the respiratory or nervous form. Because of the clinical manifestations, the disease is also called Avian pneumoencephalitis.

A wide range of avian species including chicken, turkey, pigeon, pheasants, ducks and geese are susceptible to NDV. Infection is endemic in wild birds especially waterfowls. The virus is shed in all secretions and excretions of the affected host. Transmission occurs through aerosol or by ingestion of contaminated feed and water. The virus is relatively stable in the environment thereby permitting mechanical transfer of the virus through formites. Virus is present in all organs of acutely affected birds and in eggs.

Clinical signs: the incubation period is usually about five days. Respiratory, gastrointestinal and nervous signs occur in chickens. The particular clinical presentation relates to the virulence of the virus strain, its tissue tropism and the age and immune status of the host.

Pathotypes of NDV based on virulence and tissue tropism:

1. Viscerotropic velogenic isolates causing severe fatal disease characterized by haemorrhagic intestinal lesions (Doyle's form).
2. Neurotropic velogenic isolates causing acute disease characterized by nervous and respiratory signs with high mortality (Beach's form).
3. Mesogenic isolates causing mild disease with mortality confined to young birds (Beaudette's form). This presents with respiratory signs, occasionally nervous signs but low mortality.
4. Lentogenic isolates causing mild or subclinical respiratory infection (Hitchner's form).
5. Asymptomatic enteric isolates: a form that usually consists of a subclinical enteric infection.

Immunology: Newcastle disease virus induces haemagglutination inhibiting and serum neutralizing antibodies. Antibody production is rapid. It can be detected within 4 to 6 days of infection and persists for at least two years. The level of haemagglutination inhibiting antibody is a measure of immunity. Maternal antibody protects chicks for three to four weeks after hatching. Immunoglobulin G is confined to the circulation and does not protect respiratory infection but blocks viraemia. Locally produced IgA antibodies play an important role in protection in both respiratory and intestinal tracts.

Diagnosis:

- Presumptive clinical diagnosis is based on clinical signs and post mortem findings
- Suitable samples for laboratory confirmation include tracheal and cloacal swabs from live birds for virus isolation
- Postmortem samples for laboratory investigations include faeces, intestinal content and tissues from trachea, intestine, spleen, brain and lung.
- Samples may be stored at $4^{O}C$ for up to four days
- Virus isolation is carried out in embryonated egg from specific pathogen free flocks. Sample is inoculated into the allantoic cavity of the embryonated egg. After incubation, allantoic fluid is tested for haemagglutination activity
- Haemagglutination inhibition test using specific antiserum confirms the presence of NDV
- Demonstration of antibody to NDV is of diagnostic value only in unvaccinated flocks. The haemagglutination inhibition test is the most widely used assay. Commercial ELISA kits are also available
- Demonstration of viral antigen in tracheal section or impression smear using immunofluorescence test is a less sensitive technique than virus isolation

Control:

- Locating poultry farms far apart
- Preventing wild birds from having access to pens and feed-stores
- Restricted human access to farm
- Movement restriction between farms
- Thorough cleaning and disinfection of vehicles and equipment
- Some countries practice test and slaughter policy

- Vaccination: lentogenic or mesogenic strains of NDV propagated in egg or tissue culture are used in live vaccines. They are administered as sprays, in drinking water or by intranasal or intraconjunctival instillation
- The presence of maternaly derived antibodies may interfere with the efficacy of live vaccines
- Practice in Nigeria:

- Day 1 – 10: intraocular vaccination with Hitchner B-1 strain
- 3 weeks: oral vaccination in drinking water with LaSota strain
- 6 weeks: intramuscular vaccination with Komarov strain
- Quarterly intramuscular vaccination with Komarov strain in laying birds or bimonthly oral vaccination with LaSota strain