PESTE DES PETITS RUMINANTS

AETIOLOGY

Classification of the causative agent

Peste des petits ruminant virus (PPRV) classified in the family Paramyxoviridae, genus *Morbillivirus.* By means of nucleic acid sequencing, PPRV can be differentiated into four lineages (1–4). It is antigenically similar to rinderpest virus.

Resistance to physical and chemical action

Temperature:	Half-life calculation of 2 hours/37°C; virus destroyed at 50°C/60 minutes
pH:	Stable between pH 5.8 and 10.0; thus inactivation at pH<4.0 or >11.0
Disinfectants/chemicals:	Effective agents include alcohol, ether and common detergents; susceptible to most disinfectants, e.g. phenol, sodium hydroxide 2%/24 hours
Survival:	Survives for long periods in chilled and frozen tissues

EPIDEMIOLOGY

Peste des petits ruminants (PPR) represents one of the most economically important animal diseases in areas that rely on small ruminants. Outbreaks tend to be associated with contact of immuno-naïve animals with animals from endemic areas. In addition to occurring in extensive-migratory populations, PPR can occur in village and urban settings though the number of animals is usually too small to maintain the virus in these situations.

- Morbidity rate in susceptible populations can reach 90–100%
- Mortality rates vary among susceptible animals but can reach 50–100% in more severe instances
- Both morbidity and mortality rates are lower in endemic areas and in adult animals when compared to young

Hosts

- Goats (predominantly) and sheep
    - Breed-linked predisposition in goats
- Wildlife host range not fully understood
    - documented disease in captive wild ungulates: Dorcas gazelle (*Gazelle dorcas*), Thomson's gazelles (*Gazella thomsoni*), Nubian ibex (*Capra ibex nubiana*), Laristan sheep (*Ovis gmelini laristanica*) and gemsbok (*Oryx gazella*)
- Experimentally the American white-tailed deer (*Odocoileus virginianus*) is fully susceptible
- Cattle and pigs develop inapparent infections and do not transmit disease
- May be associated with limited disease events in camels

Transmission

- Mainly by aerosols or direct contact between animals living in close quarters
- Fomites may be means of spreading infection; bedding, feed and water troughs
- No carrier state
- Seasonal variations: more frequent outbreaks during the rainy season or the dry cold season
    - Also associated with seasonal periods of increased local trade in goats

Sources of virus

- Tears, nasal discharge, coughed secretions, and all secretions and excretions of incubating and sick animals

Occurrence

PPR was first described in Côte d'Ivoire, but it occurs in most African countries from North Africa to Tanzania, and in nearly all Middle Eastern countries up to Turkey. PPR is also wide-spread in countries from central Asia to south and south-east Asia. Recent incursions into China (Tibet) and Morocco have caused serious disease outbreaks and disease has been reported to be moving southwards in East Africa.

For more recent, detailed information on the occurrence of this disease worldwide, see the OIE *World Animal Health Information Database* (WAHID) Interface [http://www.oie.int/wahis/public.php?page=home] or refer to the latest issues of the *World Animal Health* and the OIE *Bulletin*.

DIAGNOSIS

The incubation period is typically 4–6 days, but may range from 3–10 days. For the purposes of the OIE *Terrestrial Animal Health Code*, the incubation period for the PPR is 21 days.

Clinical diagnosis

Disease severity depends on various factors: PPRV lineage, species, breed, immune status of animals. Various clinical manifestations of the disease have been described in the literature. Infected animals present clinical signs similar to those of rinderpest in cattle but with the eradication of this disease worldwide, its differentiation is of little or no importance.

A tentative diagnosis of PPR can be made based on clinical signs, but this diagnosis is considered provisional until laboratory confirmation is made for differential diagnosis with other diseases with similar signs. Two signs often seen in PPR and not in RP are crusting scabs along the lips and development of pneumonia in later stages of disease. Sheep and goats that recover from PPR develop an active immunity and antibodies have been demonstrated 4 years after infection; immunity is probably life-long.

Acute form

- Sudden rise in body temperature (40–41°C) with effects on the general state: animals become depressed or restless, anorexic and develop a dry muzzle and dull coat
  - pyrexia can last for 3–5 days
- Serous nasal discharge becoming mucopurulent and resulting, at times, in a profuse catarrhal exudate which crusts over and occludes the nostrils; signs of respiratory distress
  - in surviving animals, mucopurulent discharge may persist for up to 14 days
- Within 4 days of onset of fever, gums become hyperaemic, and erosive lesions develop in the oral cavity with excessive salivation
  - necrotic stomatitis with halitosis is common
  - erosions may resolve or coalesce
- Small areas of necrosis on the visible mucous membranes
- Congestion of conjunctiva, crusting on the medial canthus and sometimes profuse catarrhal conjunctivitis
- Severe, watery, blood-stained diarrhoea is common in later stages
- Bronchopneumonia evidenced by coughing is a common feature; rales and abdominal breathing
- Abortions may occur
- Dehydration, emaciation, dyspnoea, hypothermia and death may occur within 5–10 days
- Survivors undergo long convalescence

Peracute form

- Frequent in goats; especially situations of inmuno-naïve introductions into instances of circulating PPRV
- High fever, depression and death
- Higher mortality

Subacute form

- Frequent in some areas because of local breed susceptibility; form commonly seen in experimentally infected animals

- Usually 10–15 days development with inconsistent signs; on or about 6th day post-infection, fever and serous nasal discharge is observed
- Fever falls with onset of diarrhoea and, if this is severe, may result in dehydration and prostration

Lesions

Lesions associated with PPR are very similar to those observed in cattle affected with rinderpest, except prominent crusty scabs along the outer lips and severe interstitial pneumonia frequently occur with PPR

- Emaciation, conjunctivitis, erosive stomatitis involving the inside of the lower lips and adjacent gum near the commisures and the free portion of the tongue
- Lesions on the hard palate, pharynx and upper third of the oesophagus in severe cases
- Rumen, reticulum and omasum rarely have lesions
- Small streaks of haemorrhages and sometimes erosions: in the first portion of the duodenum and the terminal ileum
- Necrotic or haemorrhagic enteritis with extensive necrosis and sometimes severe ulceration of Peyer's patches
- Congestion around the ileo-caecal valve, at the caeco-colic junction and in the rectum
    - 'Zebra stripes' of congestion in the posterior part of the colon
- Small erosions and petechiae on the nasal mucosa, turbinates, larynx and trachea
- Bronchopneumonia is a constant lesion
- Possibility of pleuritis and hydrothorax
- Congestion and enlargement of spleen and liver
- Congestion, enlargement and oedema of most of the lymph nodes
- Erosive vulvovaginitis may exist

Differential diagnosis

- Rinderpest
- Contagious caprine pleuropneumonia
- Bluetongue
- Pasteurellosis (also may occur as secondary infection to PPR)
- Contagious ecthyma
- Foot and mouth disease
- Heartwater
- Coccidiosis
- Mineral poisoning

Laboratory diagnosis

Samples

- Swabs of the conjunctival discharges and from the nasal and buccal mucosae
- For virus isolation, polymerase chain reaction (PCR) and haematology:
    - whole blood collected in EDTA; preferably collected in early stages of disease
    - blood and anticoagulant should be mixed gently
- For serologic needs, clotted blood can be collected at the end of an outbreak
- Upon necropsy aseptically collect the following tissues chilled on ice and transported under refrigeration
    - Lymph nodes (especially the mesenteric and bronchial nodes)
    - Spleen
    - Lung (especially intestinal mucosae)
- Set of tissues for histopathology should be placed in 10% neutral buffered formalin

Procedures

It should be noted that no live rinderpest virus can be permitted in any test system.

Identification of the agent

- *Agar gel immunodiffusion*
    - simple and inexpensive test that can be performed in any laboratory and even in the field

    - standard PPR viral antigen is prepared from mesenteric or bronchial lymph nodes, spleen or lung material
    - results are obtained in one day, but the test is not sensitive enough to detect mild forms of PPR due to the low quantity of viral antigen that is excreted.
- *Counter immunoelectrophoresis*
    - most rapid test for viral antigen detection
    - carried out on a horizontal surface using a suitable electrophoresis bath
    - presence of 1–3 precipitation lines between pairs of wells is a positive reaction
    - there should be no reactions between wells containing the negative controls
- *Imunocapture enzyme-linked immunosorbent assay*
    - using two monoclonal antibodies (MAb) raised to the N protein, allows a rapid identification of PPRV
        - positive cut-off value is calculated from the blank control as three times the mean absorbance values of the control wells
    - sandwich ELISA is widely used in India

Nucleic acid recognition methods

- reverse transcription PCR (RT-PCR) techniques based on the amplification of parts of the N and F protein genes has been developed for the specific diagnosis of PPR
    - 1000 times more sentsitive than classical virus titration on Vero cells and results are obtained in 5 hours; including the RNA extraction
- multiplex RT-PCR, based on the amplification of fragments of N and M protein genes, has been reported
- another format of the N gene-based RT-PCR has also been described
    - analyses of amplicon is detected by ELISA through the use of a labelled probe
    - this new format, RT-PCR-ELISA, is ten times more sensitive than the classical RT-PCR
- a real time RT-PCR assay has been developed for the specific detection of PPRV nucleic acid. It can detect virus from all four lineages of the virus

- *Culture and isolation methods*
    - even when diagnosis has been carried out by rapid techniques, the virus should always be isolated from field samples in tissue cultures for further studies
    - PPRV may be isolated in primary lamb kidney/ lung cells and some cell lines (Vero, B95a)
    - Monolayer cultures are inoculated with suspect material (swab material, buffy coat or 10% tissue suspensions) and examined daily for evidence of cytopathic effect

Serological tests

- Virus neutralisation (the prescribed test for international trade)
    - test is sensitive and specific but time-consuming
    - standard neutralisation test is now usually carried out in 96-well microtitre plates although roller-tube cultures may be used. Vero cells are preferred, but primary lamb kidney cells may also be used
- Competitive enzyme-linked immunosorbent assay
    - based on use of MAbs that recognise virus proteins: those where the MAb recognises the N protein and use recombinant N protein produced in baculovirus as the antigen; and those with a viral attachment protein (H) specific MAb and antigen consisting of purified or part purified PPRV (vaccine strain)advice on the use and applicability of ELISA methods is available from the OIE Reference Laboratories for PPR

For more detailed information regarding laboratory diagnostic methodologies, please refer to Chapter 2.7.11 Peste des petits ruminants in the latest edition of the OIE *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* under the heading "Diagnostic Techniques".

PREVENTION AND CONTROL

- No specific treatment
- Antibiotics may prevent secondary pulmonary infections (oxytetracycline, chlortetracycline)

Sanitary prophylaxis

- Epidemic outbreak situations: when the disease appears in previously PPR-free zones or countries
    - rapid identification, humane slaughter and disposal of affected animals and their contacts; carcasses burned or buried
    - strict quarantine and control of animal movements
    - effective cleaning and disinfection of contaminated areas of all premises with lipid solvent solutions of high or low pH and disinfectants as described above; includes physical perimeters, equipment and clothing
    - careful consideration to use of vaccine; strategic ring vaccination and/or vaccination of high-risk populations
    - monitoring of wild and captive animals
- Endemic outbreak situations: when is continually circulating
    - most commonly employed control mechanism is vaccination
        - sheep and goats vaccinated with an attenuated strain of PPR or that recover from PPR develop an active life-long immunity against the disease
    - monitoring of wild and captive animals; especially avoiding contact with sheep and goats
        - protective vaccination of zoologic species may be considered
- Exposed or infected animals should be slaughtered and the carcases should be burned with deep burial.

Medical prophylaxis

- Since the global eradication of rinderpest, heterologous vaccines should not be used to protect against PPR
- Several homologous PPR vaccines are available, being cell culture-attenuated strains of natural PPRV. In 1998, the OIE World Assembly (former OIE International Committee) endorsed the use of such a vaccine in countries that have decided to follow the 'OIE pathway' for epidemiological surveillance for rinderpest in order to avoid confusion when serological surveys are performed
    - vaccine gives strong immunity
- Attenuated PPRV vaccines are commercially available
- There have also been two published reports on the preliminary results of the development of recombinant capripox-based PPR vaccines that are able to protect against both capripox and PPR

For more detailed information regarding vaccines, please refer to Chapter 2.7.11 Peste des petits ruminants in the latest edition of the *OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* under the heading "Requirements for Vaccines".

For more detailed information regarding safe international trade in terrestrial animals and their products, please refer to the latest edition of the OIE *Terrestrial Animal Health Code*.

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The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Scientific and Technical Department (scientific.dept@oie.int). Last updated April 2013.