

# PESTE DES PETITS RUMINANTS

Aetiology Epidemiology Diagnosis Prevention and Control References

## AETIOLOGY

### **Classification of the causative agent**

Peste des petits ruminants is caused by a virus of the family *Paramyxoviridae*, genus *Morbillivirus*. Until recently, this virus was named simply *Peste des petits ruminants virus* (PPRV); the official name of this virus was changed in 2016 to *Small ruminant morbillivirus* (SRM). However, it is still commonly known as PPRV by people working in the field.

The virus exists as a single serotype but, by means of nucleic acid sequencing, it can be differentiated into four lineages (1–4). It is antigenically similar to rinderpest virus, measles virus and canine distemper virus.

### **Resistance to physical and chemical action**

Temperature:	Half-life calculation of 3 hours at 37°C or 2.2 minutes at 56°C
pH:	Stable between pH 5.0 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 as a way of making a living. 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
- Many species of antelope and wild relatives of domesticated small ruminants are susceptible to PPRV
  - Clinical cases have been reported in gazelles, bushbuck, impala, springbuck, gemsbok, bharal, Sindh ibex, wild goats/bezoar ibex, Nubian ibex, Mongolian saiga antelope, Afghan Markhor goat, Barbary sheep and Laristan sheep
  - Evidence of infection (antibodies and/or virological evidence of infection) has been demonstrated in additional species, such as goitered gazelle, African grey duiker, Bubal hartebeest, waterbuck, kob and African buffalo
  - Whether wild ruminants are important in the epidemiology of this disease is unknown
  - Currently, there is no evidence that the virus circulates in wild ruminants independently of its presence in domesticated sheep and goats
  - Experimentally, the American white-tailed deer (*Odocoileus virginianus*) is fully susceptible
- Cattle develop inapparent infections and do not transmit disease
- Pigs have been reported as being susceptible and transmitting the virus under laboratory conditions but so far not in the field
- May be associated with limited disease events in camels, but they do not appear to transmit the virus

## **Transmission**

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

## **Sources of virus**

- Tears, nasal discharge, coughed secretions, and all secretions and excretions of incubating and sick animals
- It probably occurs in milk

## **Occurrence**

The disease was first identified in the early 1940s in Ivory Coast. It has since been identified in many countries in Africa, the Middle East and Asia, partly as a result of improved detection systems and partly as a result of a significant geographical expansion of the disease that has occurred over the last 15 years, resulting in PPR now being endemic in large parts of Africa, Asia, the Near and Middle East. The disease has been reported in Europe in 2016 (Georgia) and 2018 (Bulgaria).

**PPR is one of the diseases for which the OIE has a procedure for the official recognition of disease status. For more information, visit the status portal on the OIE website [<http://www.oie.int/en/animal-health-in-the-world/official-disease-status/>]**

**For more recent, detailed information on the occurrence of this disease worldwide, see the OIE *World Animal Health Information Database (WAHID)* Interface [<https://www.oie.int/wahis/public.php?page=home>]**

## **DIAGNOSIS**

The incubation period is typically 4–6 days but may range from 3–10 days. In most cases, clinical signs appear in 3-6 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 strain, host species and breed, and the health status of host animals. Various clinical manifestations of the disease have been described in the literature. Infected animals present clinical signs similar to those of rinderpest (RP) in cattle. 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. 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.

**NB:** cattle develop anti-PPRV antibodies when exposed to the virus, but do not display clinical disease and appear not to transmit the virus. Despite the eradication of RP, the appearance of RP-like disease in cattle should not be ascribed to PPRV unless confirmed by laboratory test.

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 immuno-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
- Usually 10–15 days development with inconsistent signs; on or about 6<sup>th</sup> 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 commissures 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**

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

## Laboratory diagnosis

### Samples

#### Live animals

- 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

#### Post-mortem

- Upon necropsy aseptically collect the following tissues chilled on ice and transported under refrigeration
  - Lymph nodes (especially the mesenteric and bronchial nodes)
  - Spleen
  - Lung
  - Small intestine, especially intestinal mucosae or lymphoid areas (Peyer's patches)
- Set of tissues for histopathology should be placed in 10% neutral buffered formalin

### Procedures

#### Identification of the agent

- *Nucleic acid detection and identification*
  - The preferred methods for virus detection, for reasons of sensitivity and specificity; can detect virus from all four lineages of the virus
  - Reverse-transcription PCR (RT-PCR) techniques based on the amplification of parts of the N or F protein genes have been developed for the specific diagnosis of PPR
    - 1000 times more sensitive than classical virus isolation on Vero cells and results are obtained in 5 hours, including the RNA extraction
    - another format of the N gene-based RT-PCR has also been described, in which amplicon is detected by ELISA through the use of a labelled probe. This RT-PCR-ELISA, is ten times more sensitive than the standard RT-PCR
  - several reverse-transcription real-time PCR (RT-qPCR) assays have been developed for the specific detection of PPRV nucleic acid. These are the most sensitive test available. Users are advised to contact the OIE Reference Laboratories for PPR (see <https://www.oie.int/en/scientific-expertise/collaborating-centres/list-of-centres/>) for advice on the current best assay
- *Immunocapture enzyme-linked immunosorbent assay*
  - available as a commercial kit; uses two monoclonal antibodies (MAb) raised to the N protein, allows rapid identification of PPRV
  - a sandwich ELISA is widely used in India but has not been validated outside that country
- *Culture and isolation methods*
  - even when diagnosis has been carried out by rapid techniques, the virus should always be isolated from field samples in cell cultures for further studies
  - PPRV isolation is best carried out using a cell line expressing the virus receptor (SLAM). Cell lines expressing goat or canine SLAM are very susceptible to the virus and are available from the OIE Reference Laboratories
  - PPRV may also 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
- *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*
  - a more rapid version of AGID
  - 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 with wells containing the negative controls

#### *Serological tests*

- *Virus neutralisation*
  - 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 3.7.9 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**

The OIE and the FAO, in their joint strategy for control and eradication of PPR, have set the goal of eradicating the disease by 2030. This strategy has three components, (i) the technical step-wise approach (Stage 1 to Stage 4) to control and eradicate the disease (ii) the strengthening of Veterinary Services in order to be able to carry out the technical component and (iii) the control of other priority small ruminant diseases together with PPR in view of increasing the impact of the control efforts.

### ***Sanitary prophylaxis***

- Exposed or infected animals should be slaughtered and the carcasses should be burned with deep burial
- 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 the virus 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 zoological species may be considered

### ***Medical prophylaxis***

- No specific treatment. However, supportive care and treatment of bacterial and parasitic coinfections may decrease mortality
- Antibiotics may prevent secondary pulmonary infections (oxytetracycline, chlortetracycline)
- 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 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
- These attenuated PPRV vaccines are commercially available

**For more detailed information regarding vaccines, please refer to Chapter 3.7.9 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*.**

## **REFERENCES AND OTHER INFORMATION**

- Adams M.J., Lefkowitz E.J., King A.M.Q., Harrach B., Harrison R.L., Knowles N.J., Kropinski A.M., Krupovic M., Kuhn J.H., Mushegian A.R., Nibert M., Sabanadzovic S., Sanfacon H., Siddell, S.G., Simmonds P., Varsani A., Zerbini F.M., Gorbalenya A.E. & Davison A.J. (2017). – Changes to taxonomy and the International Code of Virus Classification and Nomenclature ratified by the International Committee on Taxonomy of Viruses (2017). *Arch. Virol.*, **162**, 2505–2538.
- Brown C. & Torres A., Eds. (2008). - USAHA Foreign Animal Diseases, Seventh Edition. Committee of Foreign and Emerging Diseases of the US Animal Health Association. Boca Publications Group, Inc.
- Coetzer J.A.W. & Tustin R.C. Eds. (2004). - Infectious Diseases of Livestock, 2nd Edition. Oxford University Press.
- FAO and OIE (2015) – Global Strategy for the Control and Eradication of PPR
- Kahn C.M., Ed. (2005). - Merck Veterinary Manual. Merck & Co. Inc. and Merial Ltd.
- Munir M., Zohari S. & Berg M. (2013). Molecular Biology and Pathogenesis of Peste des Petits Ruminants Virus, SpringerBriefs in Animal Sciences, DOI: 10.1007/978-3-642-31451-3\_2
- Spickler A.R. & Roth J.A. Iowa State University, College of Veterinary Medicine – last updated in August 2015 <http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.htm>
- Schulz C., Fast C., Schlottau K., Hoffmann B. & Beer M. (2018). – Neglected Hosts of Small Ruminant Morbillivirus. *Emerg. Infect. Dis.*, **24**, 2334–2337
- Schulz C., Fast C., Wernery U., Kinne J., Joseph S., Schlottau K., Jenckel M., Hoper D., Patteril N.A.G., Syriac G., Hoffmann B. & Beer M. (2019). – Camelids and Cattle Are Dead-End Hosts for Peste-des-Petits-Ruminants Virus. *Viruses*, **11** (12). pii: E1133. doi: 10.3390/v11121133
- Taylor W.P. & Barrett T. (2007) Rinderpest and peste des petits ruminants. *In: Diseases of sheep*, fourth edition. I.D. Aitked, editor. Blackwell Publishing.
- World Organisation for Animal Health (2019). - Terrestrial Animal Health Code. OIE, Paris.
- World Organisation for Animal Health (2019). - Manual of Diagnostic Tests and Vaccines for Terrestrial Animals. OIE, Paris.

\*

\* \*

<p>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 (<a href="mailto:scientific.dept@oie.int">scientific.dept@oie.int</a>). Last updated January 2020</p>
------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------