

PESTE DES PETITS RUMINANTS

(Pest of Small Ruminants, stomatitis-pneumoenteritis complex or syndrome pseudorinderpest of small ruminants and kata [Pidgin English for catarrh])

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Definition [top](#)

Peste des petits ruminants (PPR) is an acute or subacute viral disease of goats and sheep characterized by fever, erosive stomatitis, conjunctivitis, gastroenteritis, and pneumonia. Goats are usually more severely affected than sheep.

Etiology (1,2,9,16) [top](#)

Peste des petits ruminants is caused by a paramyxovirus of the *Morbillivirus* genus. Other members of the genus include rinderpest virus (RPV), measles virus (MV), canine distemper virus (CDV), and phocid distemper virus (PDV) of sea mammals (seals). For many years, PPR virus was considered a variant of RPV, specifically adapted for goats and sheep, that had lost its virulence for cattle. It is now known that the two viruses are distinct though closely related antigenically.

Host Range (8) [top](#)

Peste des petits ruminants is primarily a disease of goats and sheep. However, there is one report of naturally occurring PPR in captive wild ungulates from three families: *Gazellinae* (*dorcas gazelle*), *Caprinae* (Nubian ibex and Laristan sheep), and *Hippotraginae* (gemsbok). Experimentally, the American white-tailed deer (*Odocoileus virginianus*) is fully susceptible. The role of wildlife on the epizootiology of PPR in Africa remains to be investigated. Cattle and pigs are susceptible to infection with PPRV, but they do not exhibit clinical signs. Such subclinical infections result in seroconversion, and cattle are protected from challenge with virulent RPV. Cattle and pigs do not, however, play a role in the epizootidogy of PPR because they are apparently unable to transmit the disease to other animals.

Geographic Distribution (7,12,13,21) [top](#)

Presently, PPR occurs in most African countries situated in a wide belt between the Sahara and Equator, the Middle East (Arabian Peninsula, Israel, Syria, Iraq, Jordan), and the Indian subcontinent.

Transmission [top](#)

Peste des petits ruminants is not very contagious and transmission requires close contact. Ocular, nasal, and oral secretions and feces are the sources of virus. Contact infection occurs mainly through inhalation of aerosols produced by sneezing and coughing. Fomites such as bedding may also contribute to the onset of an outbreak. As in rinderpest (RP), there is no known carrier state. Infected animals may transmit the disease during the incubation period.

Incubation Period [top](#)

Peste des petits ruminants has an incubation period of 4 to 5 days.

Clinical Signs (11,12,14,15,21) [top](#)

The disease usually appears in the acute form, with an incubation period of 4 to 5 days followed by a sudden rise in body temperature to 104-106° F (40-41° C). The temperature usually remains high for about 5 to 8 days before slowly returning to normal preceding recovery or dropping below normal before death. Affected animals appear ill and restless and have a dull coat, dry muzzle, and

depressed appetite. Accompanying these nonspecific signs are a series of changes that make up a highly characteristic syndrome. From the onset of fever, most animals have a serous nasal discharge, which progressively becomes mucopurulent. The discharge may remain slight or may progress, resulting in a profuse catarrhal exudate, which crusts over and occludes the nostrils. At this stage, animals have respiratory distress, and there is much sneezing in an attempt to clear the nose. Small areas of necrosis may be seen on the visible nasal mucous membranes. The conjunctiva usually becomes congested, and the medial canthus may have some crusting. As with the nose, there may be profuse catarrhal conjunctivitis resulting in matting of the eyelids (Fig. 88).

Necrotic stomatitis is common. It starts as small, roughened, red, superficial necrotic foci on the gum below the incisor teeth. These areas may resolve within 48 hours or progressively increase to involve the dental pad, the hard palate, cheeks and their papillae, and the dorsum of the anterior part of the tongue. Necrosis may result in shallow irregular nonhemorrhagic erosions in the affected areas of the mouth and deep fissures on the tongue. Necrotic debris may collect at the oral commissures, and scabs may form along the mucocutaneous junction of the lips. There may be excessive salivation but not to the extent of drooling.

At the height of development of oral lesions, most animals manifest severe diarrhea, often profuse but not hemorrhagic. As it progresses, there is severe dehydration, emaciation, and dyspnea followed by hypothermia, and death usually occurs after a course of 5 to 10 days. Bronchopneumonia, evidenced by coughing, is a common feature in the later stages of PPR. Pregnant animals may abort.

Secondary latent infections may be activated and complicate the clinical picture.

Gross Lesions (4,5,11,17) [top](#)

The pathology caused by PPR is dominated by inflammatory and necrotic lesions in the mouth and the gastrointestinal tract. Unlike RP, there is also a definite, albeit inconstant, respiratory system component; hence, the synonym stomatitis-pneumoenteritis complex.

Emaciation, conjunctivitis, erosive stomatitis involving the inside of the lower lip and adjacent gum, cheeks near the commissures, and the free portion of the tongue are frequent lesions. In severe cases, lesions may also be found on the hard palate, pharynx, and upper third of the esophagus (Fig. 89). The necrotic lesions do not evolve into ulcers because the basal layer of the squamous epithelium is rarely penetrated.

The rumen, reticulum, and omasum rarely have lesions. Sometimes, there may be erosions on the pillars of the rumen. The abomasum is a common site of regularly outlined erosions and often oozes blood.

Lesions in the small intestine are generally moderate, being limited to small streaks of hemorrhages and, sometimes, erosions in the first portion of the duodenum and the terminal ileum. Peyer's patches are the site of extensive necrosis, which may result in severe ulceration. The large intestine is usually more severely affected with congestion around the ileocecal valve, at the ceco-colic junction, and in the rectum. In the posterior part of the colon and the rectum, discontinuous streaks of congestion ("zebra stripes") form on the crests of the mucosal folds.

In the respiratory system, small erosions and petechiae may be visible on the nasal mucosa, turbinates, larynx, and trachea. Bronchopneumonia may be present, usually confined to the anteroventral areas and is characterized by consolidation and atelectasis. There may be pleuritis, which may become exudative and results in hydrothorax.

The spleen may be slightly enlarged and congested. Most lymph nodes throughout the body are enlarged, congested, and edematous. Erosive vulvovaginitis similar to the lesions in the oral mucocutaneous junction may be present.

Morbidity and Mortality (13,21) [top](#)

The incidence of PPR in an enzootic area may be similar to that of rinderpest (RP) in that a low rate of infection exists continuously. When the susceptible population builds up, periodic epizootics (outbreaks) occur, that receive more attention than usual. Such epizootics may be characterized by almost 100 percent mortality among affected goat and sheep populations.

The prognosis of acute PPR is usually poor. The severity of the disease and outcome in the individual is correlated with the extent of mouth lesions. Prognosis is good in cases where the lesions resolve within 2 to 3 days. It is poor when extensive necrosis and secondary bacterial infections result in an unpleasant, fetid odor from the animal's breath. Respiratory involvement is also a poor prognostic sign. A morbidity rate of 80-90 percent and a case-fatality rate of 50-80 percent are not uncommon — particularly in goats.

Young animals (4 to 8 months) have more severe disease, and morbidity and mortality are higher. Both field and laboratory observations indicate that PPR is less severe in sheep than in goats. Nevertheless, field outbreaks have been

reported in the humid zones of west Africa in which no distinction could be made between the mortality rates in sheep and in goats. Poor nutritional status, stress of movement, and concurrent parasitic and bacterial infections enhance the severity of clinical signs.

Diagnosis (7,9,13,19-22) [top](#)

Field Diagnosis [top](#)

In the field, a presumptive diagnosis can be made on the basis of clinical, pathological, and epizootiological findings.

Laboratory confirmation is an absolute requirement — particularly in areas or countries where PPR has not previously been reported.

Specimens for the Laboratory [top](#)

Specimens to submit include blood in EDTA anticoagulant, clotted blood or serum (if possible, paired sera), mesenteric lymph nodes, spleen, lung, tonsils, and sections of the ileum and large intestine.

Swabs of serous nasal and lachrymal discharges may also be useful. All samples should be shipped fresh (not frozen) on ice within 12 hours after collection.

Laboratory Diagnosis [top](#)

A wide range of laboratory procedures have been described for detecting virus or viral antigen, viral nucleic acid, and antibody.

Differential Diagnosis (1) [top](#)

Rinderpest. Clinical RP is rare in goats and sheep in Africa. In India, these species are quite often involved in RP outbreaks. Clinically, RP and PPR are similar, but the former should be the prime suspect if the disease involves both cattle and small ruminants.

Confirmation requires virus isolation and cross-neutralization.

Pasteurellosis. Enzootic pneumonia or the septicemic form of pasteurellosis is characterized by obvious respiratory signs, infrequent diarrhea, and a fatality rate rarely exceeding 10 percent.

Contagious caprine pleuropneumonia. There is no digestive system involvement, and the clinical signs and lesions are confined to the respiratory system and pericardium.

Bluetongue. Swelling of the lips, muzzle, and oral mucosa, together with edema of the head region, should serve to differentiate bluetongue from PPR. Coronitis, common in bluetongue, is not a feature of PPR. Also, sheep are more affected than goats.

Heartwater. There is often central nervous system involvement, including convulsions. There is no diarrhea.

Contagious ecthyma (contagious pustular dermatitis, orf). The orf virus causes proliferative, not necrotic lesions, that involve the lips rather than the whole oral cavity. The absence of nasal discharges and diarrhea also distinguish orf from PPR.

Foot-and-mouth disease. This condition is comparatively mild, and the most characteristic clinical sign, lameness, is not a feature of PPR.

Nairobi sheep disease. Sheep are more severely affected than goats. It is limited geographically to parts of east and central Africa (Kenya, Uganda, Tanzania, Ethiopia, Somalia and Congo [formerly Zaire]). Diagnosis requires isolation and serologic identification of the virus.

Coccidiosis. There is no upper digestive tract and respiratory system involvement.

Plant or mineral poisoning. Several plants and minerals may cause severe intestinal lesions. Case history and absence of fever should distinguish poisoning from PPR.

Treatment [top](#)

There is no specific treatment for PPR. However, drugs that control bacterial and parasitic complications may decrease mortality.

Vaccination [top](#)

The tissue culture rinderpest vaccine at a dose of $10^{2.5}$ TCID₅₀ protects goats for at least 12 months against PPR. The vaccine is currently used in many African countries for vaccination against PPR. The efficacy notwithstanding, its wide use is

disadvantageous for the ongoing Pan-African rinderpest campaign (PARC) because it is impossible to determine if seropositive small ruminants have been vaccinated or naturally infected with RPV. A homologous attenuated PPR vaccine is being tested and may soon be commercially available.

Control and Eradication (3,6,13,21) [top](#)

Eradication is recommended when PPR appears in new areas. Methods that have been successfully applied for RP eradication in many areas would be appropriate for PPR. These should include quarantine, slaughter, and proper disposal of carcasses and contact fomites, decontamination, and restrictions on importation of sheep and goats from affected areas.

Public Health [top](#)

Peste des petits ruminants is not infectious for humans.

GUIDE TO THE LITERATURE [top](#)

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