

# Peste des Petits Ruminants

*Ovine Rinderpest  
Pseudorinderpest,  
Goat Plague,  
Pest of Small Ruminants,  
Pest of Sheep and Goats,  
Kata,  
Stomatitis-Pneumoenteritis Syndrome,  
Pneumoenteritis Complex*

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## Importance

Peste des petits ruminants (PPR) is a highly contagious viral disease that mainly affects sheep and goats. Heavy losses can be seen, especially in goats, with morbidity and mortality rates sometimes approaching 80-100%. At one time, peste des petits ruminants was thought to be restricted to the Middle East and limited areas of Africa and Asia. Recently, its range has expanded in both Africa and Asia. In addition, infections and clinical cases have been recognized in other ungulates, particularly antelope and wild relatives of sheep and goats, but also camels and water buffalo. Some clinical cases and outbreaks in these animals have been severe, and there is a risk that PPR could threaten the conservation of certain wildlife.

## Etiology

Peste des petits ruminants virus (PPRV) is a member of the genus *Morbillivirus* in the family Paramyxoviridae. Four genetic lineages (lineages 1-4) and a number of viral strains have been identified. Lineage 4 viruses have become especially prevalent in recent years. PPRV is closely related to rinderpest virus, which has been eradicated.

## Species Affected

Among domesticated animals, peste des petits ruminants is primarily a disease of goats and sheep. PPRV has also been implicated, either alone or with other pathogens, in a few outbreaks in camels and water buffalo. Cattle can be infected but they do not seem to develop clinical signs, and are not known to transmit PPRV to other animals. No clinical signs were reported in experimentally infected pigs, which also appear to be dead-end hosts.

Many species of antelope (e.g., gazelle, impala, bushbuck, springbuck) and wild relatives of domesticated small ruminants are susceptible to PPRV. Clinical cases have been reported in gazelles (e.g., Dorcas gazelle, *Gazella dorcas*; Thomson's gazelle, *Gazella thomsoni*; Rheem gazelle, *Gazella subgutturosa marica*; Arabian gazelle, *Gazella gazella*), bushbuck (*Tragelaphus scriptus*), impala (*Aepyceros melampus*), springbuck (*Antidorcas marsupialis*), gemsbok (*Oryx gazella*), bharal (*Pseudois nayaur*), Sindh ibex (*Capra aegagrus blythi*), wild goats/ bezoar ibex (*Capra aegagrus*), Nubian ibex (*Capra nubiana*), Afghan Markhor goat (*Capra falconeri*), Barbary sheep (*Ammotragus lervia*) and Laristan sheep (*Ovis gmelini laristanica*). PPRV is thought to have been responsible for an outbreak that affected both gazelle and deer in Saudi Arabia in the 1980s, and white-tailed deer (*Odocoileus virginianus*) can be infected experimentally. Subclinical infections were reported in captive nilgai (*Tragelaphinae*). Evidence of infection (antibodies and/or virological evidence of infection) has been demonstrated in additional species, such as goitered gazelle (*Gazella subgutturosa subgutturosa*), African grey duiker (*Sylvicapra grimmia*), Bupal hartebeest (*Alcelaphus buselaphus*), waterbuck (*Kobus ellipsiprymnus*), kob (*Kobus kob*) and African buffalo (*Syncerus caffer*). 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.

PPRV (lineage IV) nucleic acids were recently detected in the tissues of an Asiatic lion (*Panthera leo persica*) that died of trypanosomiasis.

## Zoonotic potential

There is no evidence that humans are susceptible to PPRV

## Geographic Distribution

Peste des petits ruminants occurs in parts of Africa and Asia, and most of the Middle East. At present, lineages 1 through 4 have been reported in parts of Africa, and lineages 3 and 4 occur in the Middle East. In Africa, PPRV was once restricted to areas south of the Sahara desert and north of the equator, and most cases were reported in West Africa. However, viruses have now spread north, south and east of these boundaries (including into North Africa). Likewise, PPRV has historically existed on the Indian subcontinent in Asia, but recently caused outbreaks in additional countries including Nepal, Vietnam and China. Only lineage 4 is currently known to

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exist in Asia. Lineage 3 was detected in southern India in 1992, but it has not been reported since that time.

## Transmission

Transmission of PPRV mainly occurs during close contact. Inhalation is thought to be an important route of spread. This virus can be shed during the incubation period, and has been found in nasal and ocular secretions, saliva, urine and feces. It probably occurs in milk. While long-term carriage is not thought to occur, some recent studies have detected viral antigens and/or nucleic acids in the feces of clinically recovered goats for as long as 1 to 3 months. Whether live virus is also present, and if so, for how long, has not been determined. Little is known about virus shedding and transmission in other species, such as camels and antelope. PPRV is relatively fragile in the environment, and long distance aerosol transmission is unlikely; in cool temperatures and in the dark, this virus has been shown to spread for approximately 10 meters.

Fomites such as water, feed troughs and bedding can probably transmit PPRV for a short time, but do not remain infectious for long periods. There is very little information on the survival of PPRV in the environment; however, this virus is very similar to rinderpest virus, which is inactivated by ultraviolet light and desiccation within 3-4 days or less (depending on the specific environment), and normally survives for very short periods in carcasses. Temperatures above 70°C, as well as pH less than 5.6 or greater than 9.6, are also expected to inactivate PPRV. Rinderpest virus has been reported to survive for a time in refrigerated meat, and for several months in salted or frozen meat, and the survival of PPRV in meat might be similar.

## Disinfection

PPRV can be inactivated by many disinfectants including alkalis (sodium carbonate, sodium hydroxide), halogens (sodium hypochlorite), phenolic compounds, citric acid, alcohols and iodophores.

## Incubation Period

The incubation period can range from 2 to 10 days; in most cases, clinical signs appear in 3-6 days.

## Clinical Signs

The severity of the clinical signs can vary with the animal's species, breed and immunity to PPRV. Immunosuppression caused by this virus can exacerbate concurrent infections, contributing to the clinical signs.

### Sheep and goats

Peracute cases can be seen when PPRV first infects naïve populations of sheep or goats. In this form, the clinical signs are generally limited to high fever, severe depression and death. More often, the illness is subacute or acute. In acute cases, the initial signs include a sudden high fever, inappetence, marked depression and somnolence.

Serous nasal and ocular discharges appear soon after the onset of clinical signs; these discharges generally become mucopurulent from secondary bacterial infections. Matting is common around the eyes, and the nose may become obstructed. Within a few days, the gums become hyperemic, and small, gray, necrotic foci, covering shallow erosions, begin to appear in the mouth. (If these lesions are difficult to find, rubbing a finger across the gums and palate may recover foul-smelling exudates and shreds of tissue.) The oral lesions are painful, and animals may resist opening their mouths. Salivation is usually increased. In some cases, the mouth lesions resolve rapidly. In others, they enlarge, spread and coalesce. While lesions are most common on the lips and gums, they can also be found on the dental pad, palate, cheeks and their papillae, and tongue. In severe cases, the mouth may be completely covered in thick, cheesy material. The lips are often swollen, cracked and crusted, and the breath of animals with severe stomatitis is fetid. Necrotic lesions may also be detected on other mucous membranes, including those of the nasal cavity, vulva and vagina.

Many animals also develop profuse diarrhea, which may be watery, fetid and/ or blood-stained, and sometimes contains shreds of tissue. Severely affected animals can become dehydrated and emaciated. Rapid respiration is also common, and dyspnea, coughing and other signs of pneumonia may be seen. Pasteurellosis is a frequent complication. In addition, some animals may abort. In the late stages of the disease, small nodules resembling contagious ecthyma or sheep/goat pox can appear in the skin around the muzzle. The cause of these lesions is unknown. Deaths are usually the result of dehydration and/or pneumonia. Animals that do not die often have a prolonged convalescence.

Some animals have subacute or mild cases of PPR, which can last up to 2 weeks. The clinical signs are variable, but often include respiratory signs. Asymptomatic infections are also seen.

### Camels

Respiratory disease was the predominant syndrome in PPRV-infected camels during one outbreak in Ethiopia. This outbreak may have been complicated by *Streptococcus equi*. Concurrent infections with PPRV and other respiratory pathogens were found in the lungs of apparently healthy camels sampled from abattoirs in Sudan.

An outbreak among camels in Sudan was characterized by sudden death in some animals, and a more prolonged course in others. The most prominent clinical signs in the latter cases were yellowish diarrhea, which later became bloody, and abortions. Other reported signs included subcutaneous edema, submandibular swelling, "chest pain," infrequent coughing, decreased milk production, weight loss and increased water consumption. Although all ages were affected, fatal cases were most common in animals that were pregnant or had recently given birth.

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## **Water buffalo, cattle and pigs**

A highly fatal outbreak in water buffalo was characterized by depression, profuse salivation and conjunctival congestion; however, the animals were not reported to be febrile. Experimentally infected 3-5-month-old water buffalo calves developed a fever but no other clinical signs, and died in 30- 35 days. Gastrointestinal lesions were found in these calves at necropsy.

Cattle are usually asymptomatic; however, clinical signs have been reported in experimentally infected calves, and it is possible that some cattle in poor condition might become symptomatic. If they did, the syndrome would probably resemble rinderpest.

Experimentally infected pigs remained asymptomatic.

## **Wild ungulates**

Clinical signs have been described in a few exotic species. Deer can have signs similar to sheep and goats, but subclinical infections have also been reported. Captive gazelles became severely ill during one outbreak. The initial signs were anorexia and depression, followed by fever, lacrimation, congested mucous membranes, nasal discharges, small erosions on the tongue, salivation and diarrhea. All affected animals died. Similar signs were reported in Sindh ibex. Elevated respiratory rates, lacrimation, congested mucous membranes, ocular and nasal discharges, sneezing and ocular lesions (ulcerative keratitis and conjunctivitis) were documented in wild goats. Clinical descriptions of live animals were not available for one outbreak in the United Arab Emirates; however, necropsy findings indicated involvement of the lower gastrointestinal tract (e.g., catarrhal to hemorrhagic colitis) and lungs (congestion, subacute bronchiointerstitial pneumonia with occasional suppurative or fibrinopurulent pneumonia). However, no lesions were found in the upper digestive and respiratory tracts, including the oral mucous membranes, during this outbreak. Lameness has been reported in some wild ruminants, but not definitively linked to PPR.

## **Post Mortem Lesions**

The postmortem lesions are characterized by inflammatory and necrotic lesions in the oral cavity and throughout the gastrointestinal tract. The respiratory tract is also affected in many cases.

The carcass is often emaciated and/or dehydrated, and there may be evidence of diarrhea, serous or mucopurulent oculonasal discharges, crusted scabs on the lips, and necrotic stomatitis. Erosions, which are shallow and sharply demarcated from normal epithelium, may be found in the mouth, and sometimes in the pharynx and upper esophagus. Similar lesions may be detected on the vulva and vaginal mucous membranes of some animals. Erosions are common in the abomasum, but the rumen, reticulum and omasum are not significantly involved (although erosions are occasionally found on the pillars of the rumen).

Hemorrhagic streaks and erosions may also occur in the duodenum and the terminal ileum, but other segments of the small intestine are generally spared. The Peyer's patches often have extensive necrosis, which can lead to ulceration. The most severe lesions are seen in the large intestine, particularly around the ileocecal valve, at the cecocolic junction and in the rectum. "Zebra stripes" or "tiger stripes" of congestion, hemorrhage or darkened tissue are sometimes found in the posterior part of the colon on the mucosal folds. (These stripes can also be seen in animals with diarrhea and tenesmus from other causes). Respiratory lesions are also common, and may include congestion of the lungs; small erosions and petechiae in the nasal mucosa, turbinates, larynx and trachea; and bronchopneumonia. Blood-tinged, frothy exudates have been reported in the tracheas of some experimentally infected goats. The lymph nodes, particularly those associated with the respiratory and gastrointestinal tracts, are generally congested, enlarged and edematous, and the liver and spleen may have necrotic lesions. In peracute cases, the lesions may be limited to congestion of the ileocecal valve and bronchopneumonia.

The most prominent lesions in an outbreak among camels in Sudan included congestion and consolidation of the lungs (primarily the apical lobes), and inflammation and hemorrhages of the small intestine and stomach, together with enlarged lymph nodes and a pale, fragile liver. Oral lesions (swelling of the lips and hemorrhagic ulcers on the tongue) were reported in one animal.

The gross lesions in wild small ruminants and water buffalo are generally reported to be similar to those in sheep and goats. However, hemorrhagic and edematous gastroenteritis was found to involve the abomasum and all segments of the intestines in infected water buffalo. The presence of oral lesions might also be inconsistent in some wild species. Small erosions were found on the tongue of gazelles in one outbreak, and the esophagus contained thick mucoïd deposits along the walls. However, oral lesions and erosive mucosal lesions were absent from the upper intestinal and respiratory tracts of affected ungulates during another outbreak. In addition, congestion has been reported in visceral organs such as the liver, kidney, pancreas, spleen and brain of wild ruminants.

## **Diagnostic Tests**

PPRV, its nucleic acids or antigens can be detected in whole blood, swabs of ocular and nasal discharges and/or swabs of buccal and rectal mucosa. In one study, nucleic acids were detected in the buffy coat, but serum was much less likely to be diagnostic. Samples for virus isolation should be collected during the acute stage of the disease, preferably from animals with a high fever that have not yet developed diarrhea. At necropsy, samples can be collected from lymph nodes (particularly the mesenteric and bronchial nodes), lungs, spleen, tonsils and affected sections of the intestinal tract (e.g. ileum and large intestine).

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In endemic regions, PPR is often diagnosed by detecting viral nucleic acids with RT-PCR assays. This disease can also be confirmed by virus isolation, but recovery of the virus is not always successful. At present, PPRV is usually isolated in African green monkey kidney (Vero) cells, although other cell lines have also been employed. The identity of the virus can be confirmed by virus neutralization or other methods.

PPRV antigens can be detected by immunocapture ELISA (ICE), counter immunoelectrophoresis (CIEP) or agar gel immunodiffusion (AGID). CIEP and ICE can distinguish PPRV from rinderpest virus, but the AGID test cannot differentiate these two viruses. AGID is also relatively insensitive, and may not be able to detect small quantities of viral antigens in milder forms of PPR. Immunofluorescence and immunochemistry can be used on conjunctival smears and tissue samples collected at necropsy.

Serological tests include virus neutralization and competitive ELISA assays. Both tests can distinguish peste des petits ruminants from rinderpest; this is not always possible with older serological tests such as complement fixation. Whenever possible, paired sera should be taken rather than single samples. However, in countries that are PPRV-free, a single serum sample (taken at least a week after the onset of clinical signs) may be diagnostic.

## Treatment

There is no specific treatment for PPR; however, supportive care and treatment of bacterial and parasitic coinfections may decrease mortality.

## Control

### Disease reporting

A quick response is vital for containing outbreaks in PPRV-free regions. Veterinarians who encounter or suspect this disease should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

### Prevention

PPRV is a short-lived virus in the environment, and it is usually spread by direct contact, and introduced by infected animals. Import controls, movement restrictions, testing and quarantine are used to exclude the virus from nonendemic areas.

In regions where peste des petits ruminants is not endemic, it can be eradicated with a combination of quarantines, movement controls, euthanasia of infected and exposed animals, and cleaning and disinfection of infected premises. Ring vaccination and/or vaccination of high-risk populations may also be helpful. The rapid inactivation of PPRV in the environment aids eradication; this virus is thought to remain viable for less than four days outside the animal. Carcasses are generally buried or burned. Care

should be taken to prevent the virus from spreading to susceptible or potentially susceptible wild populations such as deer, gazelles, wild sheep or feral goats.

Peste des petits ruminants is controlled in endemic areas by vaccination. Animals that recover develop good immunity, which persists for at least four years and possibly lifelong. Some vaccines are also reported to be highly effective and provide long-term immunity. Animals may shed some vaccine strains.

To help protect susceptible wildlife and captive wild animals in endemic regions, they should not be allowed to have contact with sheep and goats. It might also be possible to vaccinate exotic species, but there is currently little information on the safety and efficacy of sheep/goat PPR vaccines in other animals. One outbreak among Sindh ibex at a wild animal park seemed to be controlled by vaccinating domesticated small ruminants and disinfecting common water sources in nearby villages.

## Morbidity and Mortality

Exposure to PPRV can be common among small ruminants in endemic regions, with seroprevalence rates ranging from < 2% to greater than 50%. These rates are often higher in sheep than goats. Several studies reported that up to 20% of camels and cattle also had antibodies to PPRV, and one study from Pakistan found that 42% of cattle and 67% of water buffalo (most born after rinderpest had been eradicated) were seropositive.

Peste des petits ruminants is highly contagious when it first occurs in a naïve population. Periodic outbreaks may also be seen in endemic regions, particularly when animals are mixed or new animals are introduced into a herd. Some epizootics are associated with changes in weather, such as the beginning of the rainy season or a cold, dry period. How this virus is maintained between outbreaks is uncertain. Some sources suggest that it might circulate subclinically in small ruminant populations, emerging when immunity wanes or naïve animals are introduced.

The severity of the disease varies with the host's species, immunity, breed, age and concurrent illnesses or infections. Some isolates can cause serious illness in one breed of goats, but mild disease in another. At least two comparative experimental studies, as well as some outbreaks in the field, have suggested that clinical cases are more severe in goats than sheep; however, there have also been reports of outbreaks among sheep where signs in goats were mild. In endemic regions, animals between three months and two years of age are most severely affected; young animals that are still nursing and older animals tend to be spared.

The morbidity and case fatality rates in small ruminants can reach levels of 80-90% or greater, particularly in naïve herds and young animals; however, these rates tend to be lower in endemic areas, and morbidity and mortality rates in some individual flocks are reported to

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be as low as 10-20%. Few cases have been reported in other domesticated ruminants. In an outbreak among water buffalo in India, the case fatality rate was 96%. Fifty of 385 water buffalos were affected; most (38) of these cases occurred in animals that had been recently introduced into the herd and were not yet vaccinated against rinderpest. During a countrywide outbreak among camels in Ethiopia, the morbidity rate was greater than 90%, and the mortality rate ranged from 5% to 70%. In another outbreak affecting camels in Sudan, mortality rates were 0% to 50%, and fatalities were most common in animals that were pregnant or had recently given birth.

High case fatality rates have been reported in some exotic ungulates. During one outbreak in captive gazelles, the morbidity rate was 51% and the case fatality rate was 100%. High case fatality (100%) was also reported among wild ungulates, including various antelope species. In an outbreak in the United Arab Emirates. An outbreak among wild Sindh ibex in Pakistan was likewise severe.

## Internet Resources

Food and Agriculture Organization (FAO) of the United Nations. Recognizing peste des petits ruminants. A field manual

<http://www.fao.org/docrep/003/x1703e/x1703e00.htm>

The Merck Veterinary Manual

<http://www.merckvetmanual.com/>

United States Animal Health Association. Foreign Animal Diseases

[http://www.aphis.usda.gov/emergency\\_response/downloads/nahems/fad.pdf](http://www.aphis.usda.gov/emergency_response/downloads/nahems/fad.pdf)

World Organization for Animal Health (OIE)

<http://www.oie.int>

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

<http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/>

OIE Terrestrial Animal Health Code

<http://www.oie.int/international-standard-setting/terrestrial-code/access-online/>

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