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## Peste des Petits Ruminants

Pest of Small Ruminants, Pest of Sheep and Goats, Stomatitis-Pneumoenteritis Complex or Syndrome, Pseudorinderpest of Small Ruminants, Kata, Goat Plague, Contagious Pustular Stomatitis

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## Overview

- Organism
- Economic Impact
- Epidemiology
- Transmission
- Clinical Signs
- Diagnosis and Treatment
- Prevention and Control
- Actions to Take

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In today's presentation we will cover information regarding the organism that causes Peste des Petits Ruminants and its epidemiology. We will also talk about the economic impact the disease has had in the past and could have in the future. Additionally, we will talk about how it is transmitted, the species it affects, clinical and necropsy signs seen, and diagnosis and treatment of the disease. Finally, we will address prevention and control measures for the disease, as well as actions to take if Peste des Petits ruminants is suspected.

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## The Organism

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## The Organism

- Family Paramyxoviridae
- Genus *Morbillivirus*
- Closely related to rinderpest virus
  - Very similar antigenically
  - Antibodies are cross-protective
  - Viruses are distinct

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Peste des petits ruminants (PPR) is a highly contagious viral disease of goats and sheep characterized by fever, erosive stomatitis, conjunctivitis, gastroenteritis, and pneumonia. The name is French for "disastrous disease of small ruminants." Goats are usually more severely affected than sheep. PPR is caused by a paramyxovirus of the genus *Morbillivirus*. It is antigenically very similar to the rinderpest virus. For many years PPR was considered a variant of rinderpest virus, specifically adapted for goats and sheep, having lost its virulence for cattle. It is now known that the two viruses are distinct, though closely related antigenically.

(Photo: The morbillivirus that causes PPR.)

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## Importance

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## History

- 1942: Cote d'Ivoire in West Africa
  - Soon spread to Nigeria, Senegal, and Ghana
- 1972: Sudan
- 1990s: Re-emerging as a result of decreases in veterinary services

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PPR was first described in Côte d'Ivoire (West Africa) in 1942. Investigators soon after confirmed existence of the disease in Nigeria, Senegal, and Ghana. In 1972 in the Sudan, a disease in goats, originally diagnosed as rinderpest, was confirmed to be PPR. The renewed spread in the 1990s of PPR, as well as other animal diseases in Africa and the Middle East, was not only related to biological factors, but to deteriorating standards in national veterinary services in these countries. Changes in government priorities have lead to decreased funding and a restructuring which has disrupted national veterinary services.

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## Economic Impact

- Presence of disease can limit:
  - Trade and export
  - Import of new breeds
  - Development of intensive livestock production
- Loss of animal protein for human consumption

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The presence of PPR can have a serious impact on the economics of a region. Economic losses are due to loss of production, death, and abortion. The presence of disease can limit trade, export, import of new breeds, and the development of intensive livestock production. PPR is a major constraint on the availability of protein for human consumption as well.

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## Epidemiology

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## Species Affected

- Principally goats and sheep
- Cattle and pigs seroconvert but do not develop or transmit disease
- Wild ungulates can be affected
  - Gazelle, deer, ibex, gemsbok
  - Limited information on species susceptibility, occurrence of disease

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Among domesticated animals, PPR is primarily a disease of goats and sheep. Cattle are usually infected asymptomatically, and are not known to transmit the disease to other animals. No clinical signs were reported in experimentally infected pigs, which also appear to be dead-end hosts. PPR can affect some wild ungulates, but there is very limited information on species susceptibility and the occurrence of disease. PPR was confirmed as the cause of two severe outbreaks, one in captive Dorcas gazelles (*Gazella dorcas*) and Thomson's gazelles (*Gazella thomsoni*) in Saudi Arabia in 2002, and the other in buffalo in India in 1995. Peste des petits ruminants is also thought to have caused another outbreak that affected both gazelles and deer in Saudi Arabia in the 1980s. White-tailed

deer (Odocoileus virginianus) can be infected experimentally. In addition, peste des petits ruminants has been reported in captive Nubian ibex, Laristan sheep and gemsbok. Whether wild ruminants are important in the epidemiology of this disease is unknown.

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Geographic Distribution

- Africa
  - South of the Sahara
  - North of the equator
- Middle East
- Parts of Asia
  - Indian subcontinent

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Presently, PPR occurs south of the Sahara desert and north of the equator in Africa, in most of the Middle East, and in parts of Asia including much of the Indian subcontinent.

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Morbidity and Mortality

- Young animals most affected
  - Ages 2 months to 2 years
- Varies by species, immunity, breed
- Morbidity and mortality rates
  - Up to 100% in naïve herds
  - Lower in endemic areas
- High case fatality rate
  - Exotic ungulates

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PPR 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. 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 severity of the disease varies with the host's species, immunity and breed. The morbidity and mortality rates can reach 100%, particularly in naïve herds; however, these rates tend to be lower in endemic areas and the reported mortality rates in some individual flocks are as low as 20%. High case fatality rates have been reported when PPR virus (PPRV) infected herds of exotic ungulates.

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Transmission

Transmission of PPR mainly occurs during close contact. Inhalation is thought to be an important route of spread. PPRV is shed in nasal and ocular secretions, saliva, urine and feces. It probably occurs in milk. Although animals are not expected to become long-term carriers, one recent study reported that viral antigens were shed in the feces of clinically recovered goats for at least 11-12 weeks. Animals may also be contagious during the incubation stage. Fomites such as water, feed troughs and bedding can probably transmit PPRV for a short time, but do not remain infectious for long periods. How the virus is maintained between outbreaks is not well understood.

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Transmission

- Close contact, inhalation
- Virus shed in nasal and ocular secretions, saliva, urine, and feces
- Long-term carriers unlikely
- Role of fomites unclear
  - Do not remain infectious for long

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**Disease in Animals**

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## Clinical Signs

- Incubation period
  - 2 to 10 days
- Peracute
- Acute
  - High fever
  - Serous nasal, ocular discharge becomes mucopurulent
  - Hyperemic gums, necrotic oral lesions

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The incubation period can range from 2-10 days, with 2-6 days being typical. Peracute cases can be seen when PPR first occurs in naïve populations of sheep or goats. Most cases of PPR are acute. The characteristic signs area high fever and a serous nasal and ocular discharge that becomes mucopurulent. Matting is common around the eyes, and the nose may become obstructed. Within a few days of the onset of fever, the gums become hyperemic, and small, gray, necrotic foci, covering shallow erosions, begin to appear in the mouth.

(Photo: USDA/APHIS)

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## Clinical Signs

- Profuse diarrhea
  - Dehydration
  - Emaciation
- Rapid respiration, dyspnea
- Abortion
- Skin nodules around muzzle
- Subacute, asymptomatic disease

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Most animals develop profuse diarrhea, which may be watery, fetid and/ or blood-stained, and sometimes contain shreds of tissue. Rapid respiration is common, and dyspnea, coughing and other signs of pneumonia may be seen. Some animals 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. Severely affected animals become dehydrated and emaciated; hypothermia can precede death. Animals that do not die often have a prolonged convalescence. Subacute disease can also be seen in some animals; this form usually lasts 10-15 days. The symptoms are variable, but often include respiratory signs. Asymptomatic infections also occur.

(Photo: USDA/APHIS)

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## Post Mortem Lesions

- Inflammatory and necrotic lesions
  - Oral cavity
  - GI tract
- Emaciation
- Erosive lesions "zebra stripes"
- Bronchopneumonia
- Enlarged lymph nodes

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Post mortem lesions are similar to rinderpest, with inflammatory and necrotic lesions in the oral cavity and throughout the GI tract. The carcass is generally emaciated. The most severe lesions are seen in the large intestine, with congestion and "zebra stripes" of congestion on the mucosal folds of the posterior colon (top photo). Erosive lesions may also occur in the vulva and vaginal mucous membranes. Bronchopneumonia with consolidation and atelectasis occurs frequently. Congestion and enlargement of the spleen may be seen. The lymph nodes are generally congested, enlarged, and edematous.

(Photo: USDA/APHIS – bottom photo shows pneumonia, top photo shows zebra striping on intestine.)

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## Sampling

- Before collecting or sending any samples, the proper authorities should be contacted
- Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease

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Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease. Call before sampling as a USDA trained Foreign Animal Disease Diagnostician (FADD) will need to collect and ship the samples.

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## Clinical Diagnosis

- PPR should be considered in:
  - Sheep, goats, or gazelle
  - Acutely febrile, highly contagious disease
  - Oral or GI signs

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PPR should be considered in sheep or goats with any acutely febrile, highly contagious disease with oral erosions and/or gastrointestinal signs. The photo shows dried exudate on the muzzle and around the eye resulting from rhinitis and conjunctivitis

(Photo: Gray Book at http://www.vet.uga.edu/vpp/gray_book/photos/pages/088.htm)

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## Differential Diagnosis

- Rinderpest
- Bluetongue
- Contagious ecthyma
- Foot and mouth disease
- Heartwater
- Coccidiosis
- Mineral poisoning
- Contagious caprine pleuropneumonia
- Pasteurellosis

The differential diagnoses include rinderpest (although many reports of 'rinderpest' among small ruminants may have been PPR), bluetongue, contagious ecthyma, foot and mouth disease, heartwater, coccidiosis and mineral poisoning. The respiratory signs can resemble contagious caprine pleuropneumonia (CCPP) or pasteurellosis; pasteurellosis can also be a secondary complication of peste des petits ruminants.

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## Laboratory Diagnosis

- Virus isolation
- Antigen detection
- RT-PCR
- Serology
- Samples
  - Discharges, oral lesions, whole blood

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PPR can be confirmed by virus isolation, but recovery of the virus is not always successful. Cultures are examined for cytopathic effect (CPE); 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). CEIP and ICE can distinguish PPRV from rinderpest virus, but the AGID test cannot differentiate these two viruses. Immunofluorescence and immunochemistry can be used on conjunctival smears and tissue samples collected at necropsy. Viral nucleic acids can be detected with reverse transcription PCR (RT-PCR). Serological tests include virus neutralization and competitive ELISA assays. Both tests can distinguish PPR from rinderpest. In live animals, swabs of ocular and nasal discharges, and debris from oral lesions should be collected. Whole, unclotted blood (in heparin or EDTA) should be taken for virus isolation and PCR.

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Treatment

- No specific treatment
- Drugs to control bacterial and parasitic complications
  - May decrease mortality
- Supportive care

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

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Disease in Humans

Humans are not affected

PPRV does not infect humans.

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Prevention and Control

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Recommended Actions

- IMMEDIATELY notify authorities
- Federal
  - Area Veterinarian in Charge (AVIC)
    http://www.aphis.usda.gov/animal_health/area_offices/
- State
  - State veterinarian
    www.usaha.org/stateanimalhealthofficials.aspx
- Quarantine

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If you suspect a case of PPR, state or federal authorities should be notified immediately. Animals suspected with PPR should be isolated, and the farm should be quarantined until definitive diagnosis is determined.

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Prevention and Control

- Quarantine
- Movement controls
- Euthanasia of infected and exposed animals
- Cleaning and disinfection of infected premises

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PPR can be eradicated with a combination of quarantines, movement controls, euthanasia of infected and exposed animals, and cleaning and disinfection of infected premises. Methods that have been successfully applied for rinderpest eradication would be appropriate for PPR.

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## Vaccination

- Outbreaks
  - Ring vaccination, high-risk populations
- Endemic areas
  - Used to control disease
- Vaccine types
  - Attenuated rinderpest vaccine
  - Homologous, attenuated PPR vaccine
  - Recombinant vaccine



In an outbreak, ring vaccination and/or vaccination of high-risk populations can be helpful. PPR is controlled in endemic areas by vaccination. An attenuated tissue culture rinderpest vaccine was used previously. However, a homologous PPR vaccine is now available and endorsed for use in countries that have followed the 'OIE pathway' for epidemiological surveillance for rinderpest; this vaccine gives strong immunity and can eliminate confusion during serological surveillance for rinderpest. A recombinant capripox-based PPR vaccine, able to protect against both capripox and PPR, is also in development.

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## Disinfection

- PPR virus killed by most common disinfectants
  - Alkalis (sodium carbonate, hydroxide)
  - Halogens (sodium hypochlorite)
    - 2% for 24 hours
  - Phenolic compounds
  - Citric Acid
  - Alcohols
  - Iodophores



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

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## Additional Resources

- World Organization for Animal Health (OIE)
  - www.oie.int
- U.S. Department of Agriculture (USDA)
  - www.aphis.usda.gov
- Center for Food Security and Public Health
  - www.cfsph.iastate.edu
- USAHA Foreign Animal Diseases ("The Gray Book")
  - http://www.aphis.usda.gov/emergency_response/downloads/nahems/fad.pdf



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## Acknowledgments

*Development of this presentation was funded by grants from the Centers for Disease Control and Prevention, the Iowa Homeland Security and Emergency Management Division, and the Iowa Department of Agriculture and Land Stewardship to the Center for Food Security and Public Health at Iowa State University.*

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