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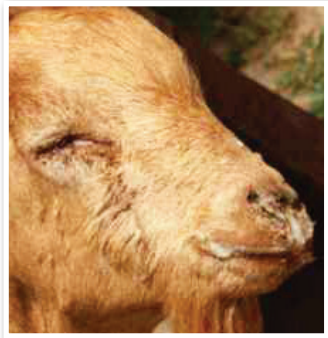
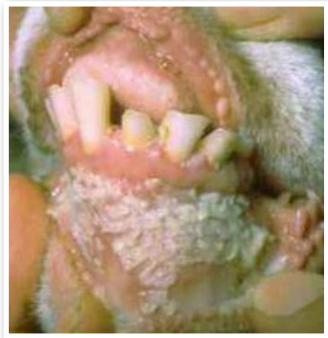
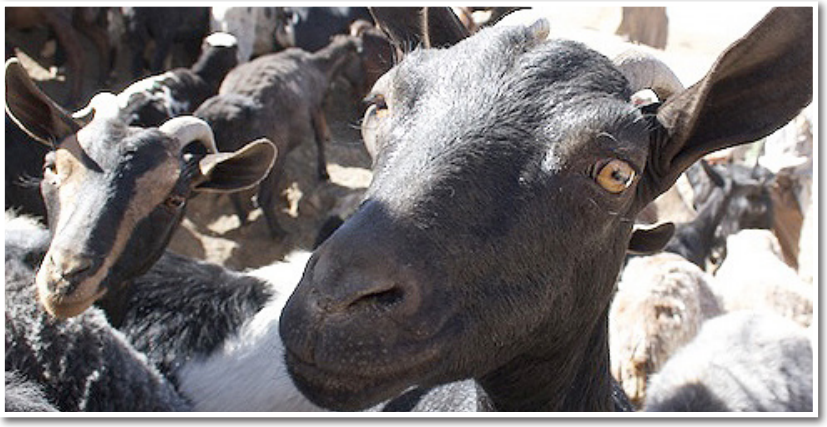
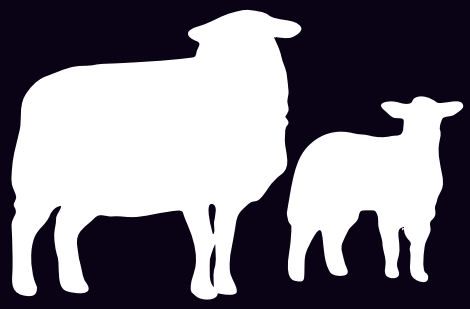
PESTE DES PETITS RUMINANTS (PPR)

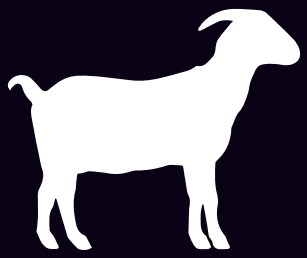
Technical Information Reporting Guide











Information cut-off, June 30, 2013

Peste des Petits Ruminants (PPR)

Technical Information Reporting Guide

Introduction to peste des petits ruminants

Peste des petits ruminants (PPR) is a highly contagious, infectious, and often fatal viral disease that affects domestic and wild small ruminants. It is also known as Ovine Rinderpest, Pest of Small Ruminants, Pest of Sheep and Goats, Stomatitis-Pneumoenteritis Complex or Syndrome, Pseudorinderpest of Small Ruminants, Kata, Goat Plague, and Contagious Pustular Stomatitis.

Animals capable of being infected by the PPR virus include: antelopes, buffalo, camels, cattle, deer, gazelles, giraffes, llamas, yaks, and wild and domestic sheep and goats. High death loss can be associated with PPR, especially in goats: 100 percent mortality in affected animals has been reported within some herds.

PPR was first described in 1942 in Ivory Coast, West Africa. The virus was thought to be restricted to West Africa but has since been recognized in other regions of Africa as well as in Asia and the Middle East. Susceptible animals in southern Africa and central Asia — near areas that harbor the PPR virus — are at risk of infection. In 2007, PPR was discovered in China. It is feasible the PPR virus is spreading, although an increased level of recognition also might explain the expanding geographic range.

A severe, fast-spreading disease, PPR is characterized by the sudden onset of fever, depression, discharge from the eyes and nose, sores in the mouth, disturbed breathing and cough, foul-smelling diarrhea, and death. The virus is not transmittable to humans. The magnitude of the disease has only become apparent in recent years and is still being clarified because PPR can be asymptomatic or misdiagnosed as Rinderpest — a closely related disease that currently is classified as eradicated by the World Organisation for Animal Health (OIE).

PPR is included as a sheep and goat disease on the OIE list of reportable diseases because of its severity, high likelihood of transmission, and threat to trade. The OIE recommends prevention and control measures including vaccination of high-risk populations along with strict controls on the movement of sheep and goats. A PPR vaccine is increasingly available. The vaccine can protect small ruminants for at least 3 years.

Occurrence and spread

Clinical disease associated with PPR virus primarily occurs in sheep and goats, however, it can also occur in wild small ruminants such as Laristan sheep, Dorcas-type gazelles, gemsbok, and Nubian ibex. Sheep and goats are not always infected equally or simultaneously. In Africa, PPR is primarily seen in goats, but sheep are more commonly infected in western and southern Asia. PPR is highly contagious when it first occurs in a new population.

Periodic PPR outbreaks also may occur in endemic regions, particularly when animals are mixed or new animals are introduced into a herd. In endemic regions, animals between 3 and 24 months 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. PPR virus, which significantly suppresses the immune system, has been identified with other concurrent infections, most notably bluetongue virus (BTV), sheep pox virus (SPV), goat pox virus (GPV), and pestivirus. Cattle, buffalo, camels, and pigs can become infected with the PPR virus but do not typically display signs of clinical disease and are unable to infect other animals.

Outbreaks of PPR can be devastating to animal conservation efforts. In 1995, an outbreak in a susceptible buffalo herd in India killed nearly all of the animals infected. In 2002, a similar outbreak occurred in captive gazelles in Saudi Arabia, resulting in the death of nearly all affected animals.

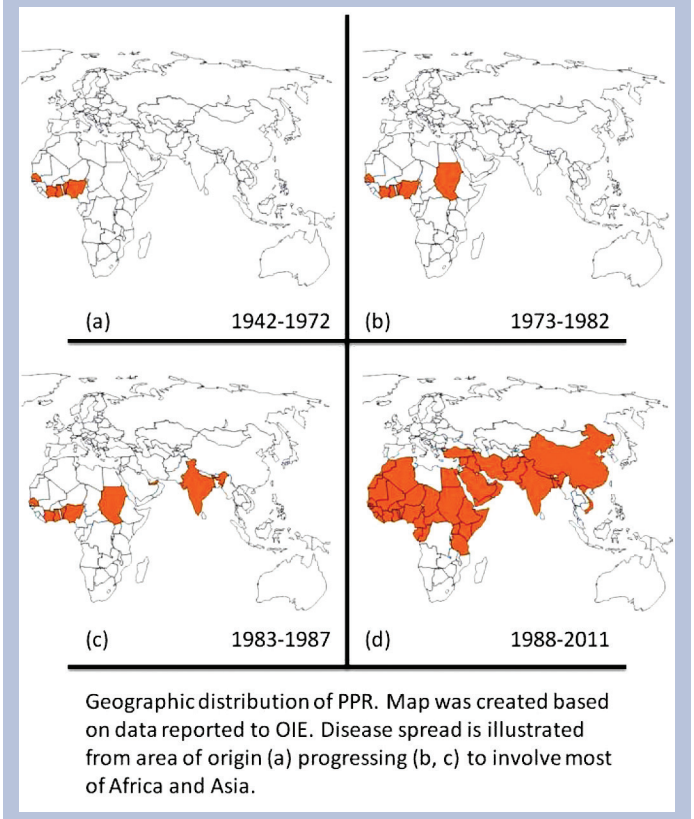
Geographic distribution

After the first report of PPR in Ivory Coast in 1942, the virus was confirmed in other West African countries: Nigeria, Senegal, Ghana, Togo, and Benin. The PPR virus is currently classified into four distinct lineages. Lineages I and II of PPR are

exclusively isolated within the West African countries where it originated. Lineage III is restricted to the Middle East and East Africa. Lineage IV is considered to be a new lineage made up of emerging viruses. It is currently most prevalent in Asian countries and is becoming the predominant lineage in Africa.

It is not clear whether the disease has spread over the last 50 years, or whether the apparent spread actually reflects increased awareness and wider availability of diagnostic tools, or even a change in the nature of the virus. Most likely, a combination of factors has contributed to the current epidemiologic disease pattern.

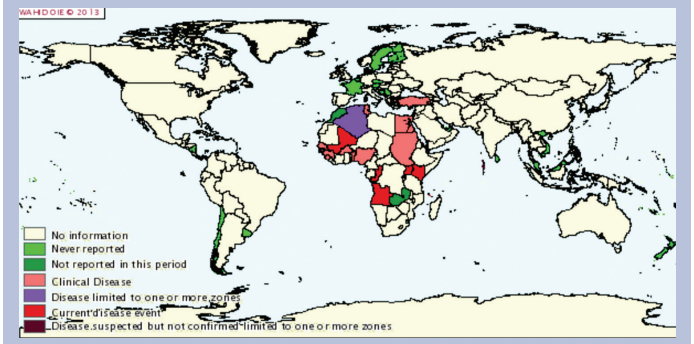
Disease distribution map of PPR



Geographic distribution of PPR. Map was created based on data reported to OIE. Disease spread is illustrated from area of origin (a) progressing (b, c) to involve most of Africa and Asia.

PPR disease map — 2013

Map from World Organisation of Animal Health (OIE) Information Disease Outbreak Maps



http://www.oie.int/wahis_2/public/wahid.php/Diseaseinformation/Diseasedistributionmap

The true extent of PPR virus spread is also unclear because it is commonly confused with similar infections. In 1972, a disease outbreak in the Sudan was originally diagnosed as Rinderpest and later confirmed as PPR. In Africa, PPR has been reported throughout the countries between the Atlantic Ocean and the Red Sea, including south to Kenya and north to Egypt. In the Middle East and Arabian Peninsula, PPR has been reported in Iran, Iraq, Israel, Jordan, Kuwait, Lebanon, Oman, Saudi Arabia, the United Arab Emirates, and Yemen. Outbreaks also commonly occur in India, Nepal, Bangladesh, Pakistan, and Afghanistan. PPR continues to spread beyond the known infected areas, including China in 2007 and Morocco in 2008.

Transmission

Peste des petits ruminants is spread through close contact between infected animals. The virus is shed in secretions (e.g., nasal and salivary) and excretions (e.g., feces) of infected animals. Inhalation of the virus (aerosol transmission) is also thought to be an important route of transmitting disease. The virus can be spread over a distance of approximately 10 meters (33 feet) after becoming aerosolized. Aerosolized transmission over longer distances is unlikely because the environment easily inactivates the virus.

Animals are considered infectious during the incubation period, which might range from 2 to 10 days. There is no carrier state; once infected, the animal either dies or recovers. Goats have been shown to shed the virus in feces for 11 to 12 weeks after recovery from PPR.

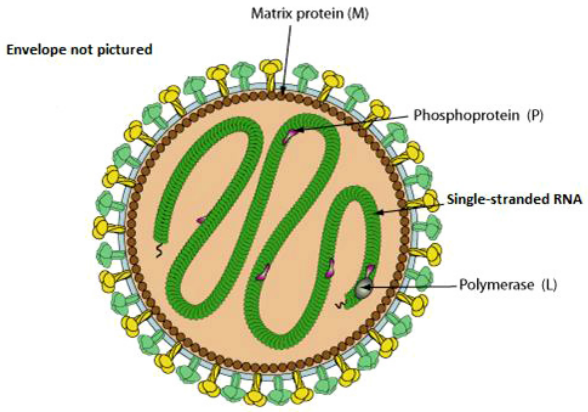
Transmission through infected meat is possible because the virus is capable of surviving for several months in frozen and salted meat. Swine may become infected through infected meat, but they are dead-end hosts. Fomites such as water, feed troughs, and bedding can transmit PPR virus for a short time but do not remain infectious for extended periods.

Spread of PPR by fomites

Virus may be mechanically carried by fomites, which include people — such as farm visitors, veterinarians, sheep and goat traders, and feed dealers. Spread can occur during auction sales, livestock shows, and similar events. Other fomite carriers include vehicles, such as feed delivery and rendering trucks; equipment; clothing or footwear; injection needles; instruments; feed, water, and bedding; and animals not susceptible to PPR.

Peste des petits ruminants virus structure

The peste des petits ruminants virus belongs to the family *Paramyxoviridae* and the genus *Morbillivirus*. Single-stranded RNA makes up its genetic component. The virus is an enveloped helical structure. Pictured is a typical member of the family *Paramyxoviridae*.



***Paramyxoviridae* virus**

Source: ViralZone: *www.expasy.org/viralzone*, Swiss Institute of Bioinformatics

Environmental stability/inactivation of the PPR virus

All members of the *Paramyxoviridae* family are very sensitive to heat.

The virus ...

- is rapidly inactivated at temperatures of 70 °C (158 °F) and above;
- remains stable in pH values between 5.8 and 10, and is inactivated at pH values of 4 or less and greater than 11;
- is capable of surviving in culture for at least 4 months at –20°C (-4°F), 8 weeks at 4°C (39°F), 1 week at 20 to 25°C (68 to 77°F), and >2.6 days at 37°C (98.6°F);
- possesses a half-life of 5 minutes in cattle blood, spleen, or lymph node at 56°C (132.8°F);
- can survive several months in frozen and salted meat; and
- is rapidly inactivated by ultraviolet light and desiccation within 4 days.

Note: pH is a scale representing acidity or alkalinity. It ranges from 0–14 (with 7 being neutral): 1 to 6 represents acidity (with 1 the highest acidity), and 8 to 14 represents alkalinity (with 14 the highest alkalinity).

Animal products and by-products

Information is not available for PPR virus but it is assumed that, like Rinderpest virus, it would be rapidly inactivated by the putrefaction in the carcass of an animal dying from PPR or by a pH of 5.5 in hung meat. Rinderpest virus can be present in milk from 1 to 2 days before clinical signs develop and for as long as 45 days after recovery. Goat or sheep milk may be considered similarly infected with PPR virus.

Laboratory methods of PPR identification

Although not comprehensive, the following procedures represent key approaches used in the laboratory to diagnose PPR and confirm the presence of virus.

Biosafety Level-3 Ag Laboratory (BSL-3Ag)

These laboratories are designed to protect the environment from highly infectious animal disease-causing agents that can result in serious infection in animals only and not humans.
They contain all the features of a standard BSL-3 laboratory, including features of a BSL-4 facility* minus positive-pressure personal ventilated life-support "spacesuits" or a Class III biosafety cabinet ("glove-box").

**BSL-4 facilities provide the highest level of biosafety. They are designed to protect against exotic infectious agents that pose a high risk of human life-threatening disease for which there is no vaccine or therapy.*

Laboratory techniques

[Key information is based on World Organisation of Animal Health (OIE) Manual of Diagnostic Tests and Vaccines for Terrestrial Animals – 2013 and the Food and Agriculture Organisation (FAO) of the United Nations Animal Health Field Manual — Recognizing Peste des Petits Ruminants]

Because of the virulence of the PPR virus, laboratories that work with it must include virus-containment equipment. Only personnel working in BSL-3 containment facilities should work with the virus.

Methods of identifying the presence of PPR virus

Provisional diagnosis of PPR virus based on clinical signs or a postmortem examination must be confirmed using laboratory testing techniques to determine the presence of any of the following:

- live virus
- virus genetic material (RNA)
- virus protein components (viral antigens)
- virus-induced antibodies

In live animals, presence of the virus can be confirmed using swabs of ocular (eye) discharge, nasal (nose) and oral (mouth) mucus, or — in the very early stages of disease — whole blood collected in an anticoagulant tube. In deceased animals, organ tissue samples can be tested. Samples should be collected in the acute phase of the disease, when clinical signs are readily apparent. Ideally, samples should be collected from several animals within an outbreak.

Live virus

Growth and isolation

Laboratory growth of PPR virus in tissue (cell) culture is used to demonstrate the presence of live virus. Even when diagnosis has been carried out by more rapid techniques, the virus should always be isolated from field samples in tissue cultures for further studies.

Successful direct isolation of PPR virus can be performed through the collection of blood using heparin as an anticoagulant or by preparing a tissue suspension in 10 percent phosphate-buffered saline (PBS) or cell culture medium. The blood or tissue samples are used to inoculate mammalian cell lines. The cell cultures are incubated and then examined for cell deterioration resulting from viral activity, known as the cytopathic effect (CPE). If CPE is seen, the presence of PPR virus is confirmed. Virus can then be extracted by virus neutralization (VN) or RNA extraction and sequencing.

If no CPE is shown, the cells are exposed to a freeze-thaw procedure. The culturing procedure is repeated, and the cells are examined again for CPE. If no evidence of CPE is viewed on the second try, virus is not present and PPR cannot be confirmed.

Identification of virus genetic material

Polymerase chain reaction (PCR)

The PCR tests are sensitive "genetic fingerprinting" methods that detect genetic material, DNA. However, because the PPR virus genetic material is RNA, a modified version of the standard PCR test — the reverse transcription-polymerase chain reaction (RT-PCR) — is used. The RT-PCR test converts pieces of the PPR virus RNA to DNA copies of PPR virus genetic fragments. After conversion, an RT-PCR test can be performed on the virus.

The reverse-transcription polymerase chain reaction (RT-PCR) test is a highly sensitive technique that is capable of detecting a very low number of copies of RNA molecules. A positive test provides a high level of confidence that the PPR virus is present. These tests are useful for screening suspect cases, even before clinical signs appear. Note that PCR testing does not differentiate between live or dead virus. Results from PCR testing can be available in five hours.

Detection of viral antigens

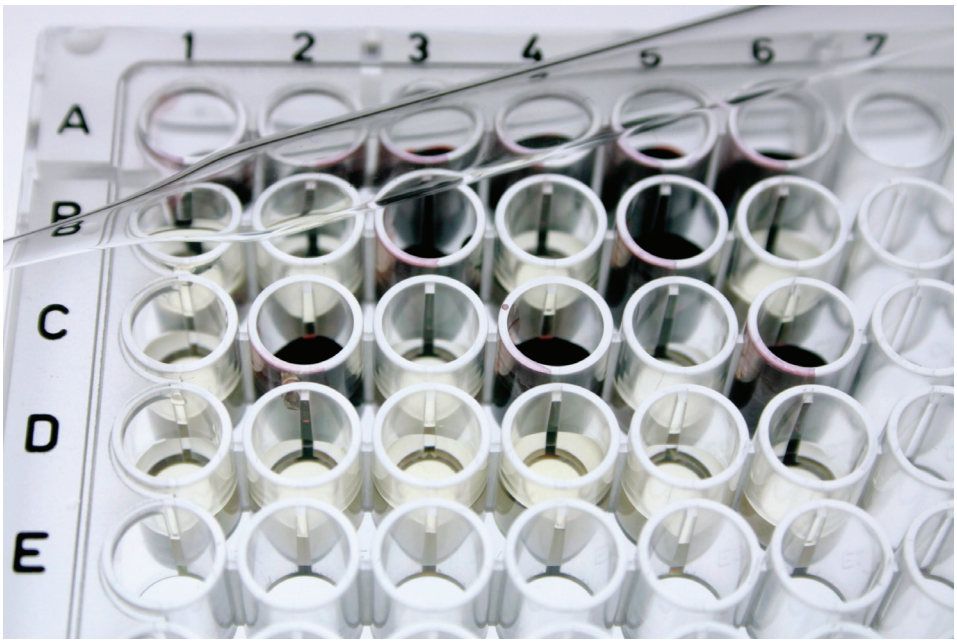
Agar gel immunodiffusion (AGID)

Agar gel immunodiffusion (AGID) is a very simple and inexpensive test that can be performed in any laboratory and even in the field. AGID gives results within one day, but it is not sensitive to mild forms of PPR and cannot differentiate between PPR and Rinderpest viruses.

Standard PPR viral antigen is prepared from infected lymph nodes, spleen, or lung material that is ground and then suspended in buffered saline. Eye or nasal swabs can be tested by inserting the sample swab into a 1-mL syringe and extracting the sample from the swab using phosphate buffered saline. The resulting eye/nasal swab-extracted sample, like the ground tissue material prepared above, may be stored at –20°C (-4°F) until used. Properly stored samples may be retained for 1 to 3 years.

Counterimmunoelectrophoresis (CIEP)

Counterimmunoelectrophoresis (CIEP) is the most rapid test to detect PPR viral antigen. It is carried out on a horizontal surface using an electrophoresis bath consisting of two compartments connected by a bridge with a high-voltage source and filled with a buffer. An agar or agarose solution is dispensed on microscope slides in 3 mL volumes. From 6 to 9 pairs of wells are punched in the solidified agar. The pairs of wells in the agar are then filled with the sera and antigen reactants. The slide is placed on the connecting bridge and the ends are connected to the buffer in troughs by wetted porous paper. The apparatus is covered and a current of 10 to 12 milliamps per slide is applied for 30 to 60 minutes. The current is switched off, and the slides are viewed by intense light. The presence of 1 to 3 precipitation lines between pairs of wells is a positive reaction. There should be no reactions between wells with a negative result.



Detection of virus-induced antibodies

The demonstration of antibodies in PPR-infected goats and sheep can be used to support a diagnosis by antigen-detection testing. Tests that are routinely used are the virus neutralization (VN) test and the competitive ELISA tests.

Virus neutralization (VN)

This test is highly sensitive and specific, but is time-consuming because cross-neutralization with Rinderpest virus must be completed as well. **The VN test is considered to be the prescribed test for international trade for detecting PPR antibodies.** The viral serum neutralization test checks for the presence of PPR virus in blood or other fluids by adding an antibody that binds to the virus in a sample. If the correct concentration of PPR virus is present in the sample of blood or other fluid, the added antibody binds with the PPR virus to form an antigen-antibody complex, rendering it harmless. Neutralizing antibodies that are produced in response to an infection are generally responsible for the protective immunity to the PPR virus observed after an infection or vaccination.

A cross-neutralization test carried out with Rinderpest virus and a sample serum is considered to be positive for PPR when the neutralization titer is at least two-fold higher for PPR than for Rinderpest.

Immunocapture enzyme-linked immunosorbent assay (ELISA)

Indirect and competitive blocking ELISAs have proven to be good methods to detect anti-PPR virus group-reactive antibodies, especially for large-scale investigations. Both methods have been demonstrated to be adequate for PPR diagnosis. They are highly sensitive, specific, and produce results in only 2 hours.

The competitive blocking ELISA technique detects specific antibodies against PPR virus present in any ruminant species. The action of this test is to block the specific reaction between the recombinant anti-N protein absorbed on an ELISA plate and a conjugated monoclonal antibody (MAb) against PPR. PPR virus antibodies present in a suspect serum sample will block the reaction. The use of three monoclonal antibodies (MAb) anti-N proteins, allows a rapid differential identification of PPR or Rinderpest viruses.

Field diagnosis of PPR

Clinical signs

PPR is a fast-spreading disease that primarily infects domesticated sheep and goats. Outbreaks will not involve cattle. Infections are characterized by the sudden onset of depression, fever, loss of appetite, discharge from the eyes and nose, sores in the mouth, difficulty breathing, coughing, foul-smelling diarrhea, and death. Virus is present in all secretions and excretions from PPR-infected animals for approximately 10 days after the onset of fever. The incubation period can range from 2 to 10 days after infection, but most clinical signs are observed within 2 to 6 days of infection. Animals that have been infected with PPR either die or become immune. There is no apparent chronic carrier state.

In endemic regions, animals that commonly become severely infected are between the ages of 3 and 24 months. The mortality rate is highly variable, depending on the population affected, and can range from 20 percent to nearly 100 percent. Rates are higher in new populations and lower in PPR-endemic areas.

Figure 1:

PPR in a goat: purulent eye and nose discharges. Discharges from the nose and eyes in advanced PPR infection; the hair below the eyes is wet and there is matting together of the eyelids as well as partial blockage of the nostrils by dried-up purulent discharges.

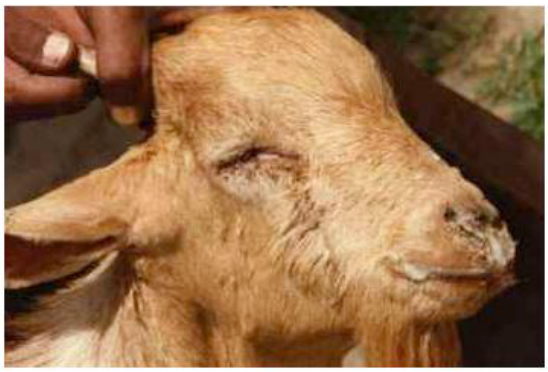


Figure 2:

PPR in a goat: inflamed (reddened) eye membranes. Reddening of the mucous membranes of the eye (the conjunctiva) in the early stages of infection. Note the purulent eye discharges.

Picture plates taken from the publication: *Recognizing Peste des Petits Ruminants – A Field Manual. Published by the Food and Agriculture Organisation of the United Nations (FAO)*

Clinical signs in other species

- **Deer:** similar to sheep and goats, with subclinical infections reported.
- **Gazelles:** anorexia, depression, fever, discharge from the eyes and nose, congested mucous membranes, salivation and diarrhea. Death rate is near 100 percent.
- **Buffalo:** depression, profuse salivation, congestion around the eyes. Not all animals exhibit fever.

Necropsy lesions

Postmortem findings on infected sheep and goats are characterized by inflammation and necrosis (tissue death) of the mucous membranes and intestinal tract. Crusting is often found around the eyes and nose, and the hindquarters are often soiled from diarrhea. Specific lesions include the following:

- Erosions of the gums, soft palate, tongue, and cheeks that may extend into the pharynx and upper esophagus.
- Swollen lips, which may or may not show erosions.
- Reddening and congestion of the nasal mucous membranes with erosions.
- Nasal matter ranging from clear to creamy yellow.
- Lungs firm to the touch with dark red or purple areas. Most lesions are found in the anterior (front) and cardiac (heart) lobes.
- Lymph nodes associated with the lungs and intestinal tract are swollen and soft.
- Intestines are congested (reddened), have hemorrhagic streaking, and may contain erosions.
- The rumen, reticulum, and omasum are not significantly changed and may contain a few erosions, but the abomasum (true stomach) generally is congested and contains erosions that ooze blood.
- Spleen may be slightly enlarged and congested.

Similar lesions are found in other species infected with PPR. In these species the abomasum is more severely affected with hemorrhages and edema (fluid accumulation in the tissues). Fluid accumulation may also be present in the liver, kidney, pancreas, and brain of those species.

Figure 3:

PPR in a goat: early mouth lesions showing areas of dead cells.
Early pale grey areas of dead cells are present on the gums.

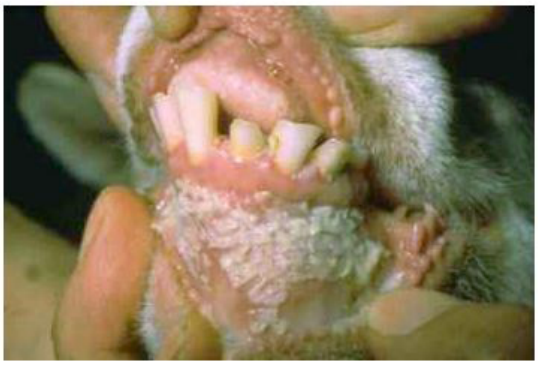


Figure 4:

PPR in a goat: later mouth lesions.
The membrane lining the mouth is completely obscured by a thick cheesy material; shallow erosions are found underneath the dead surface cells.

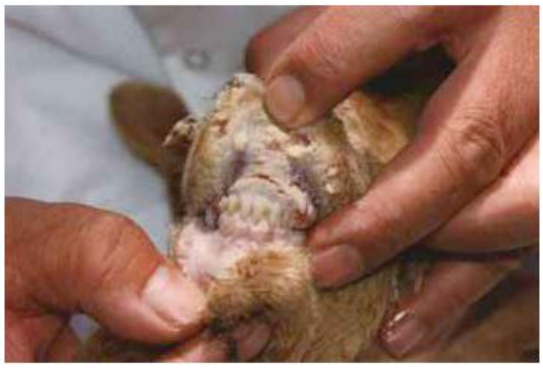


Figure 5:

PPR in a goat: swollen, eroded lips.
The lips are swollen, edematous, and show areas of erosion.

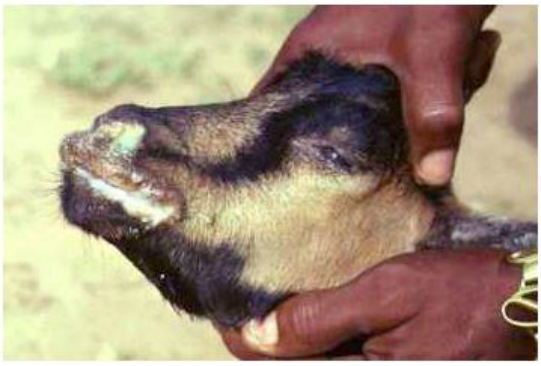


Figure 6:

PPR in a goat: signs of diarrhea.
The hindquarters are soiled with liquid feces.

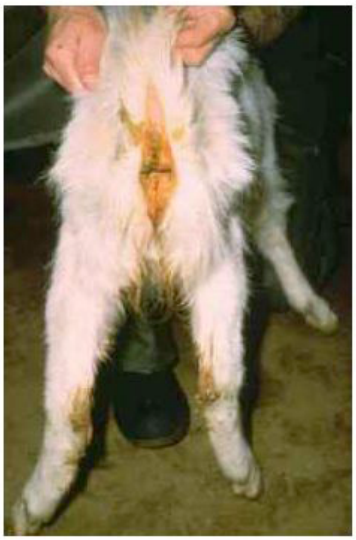


Figure 7:

PPR in a goat: nodular lesions around the mouth. Such nodules are a common finding in the later stages of PPR infection.

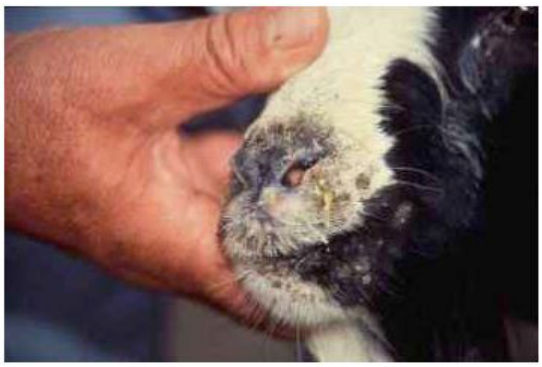


Figure 8:

PPR in a goat: the early lesions of pneumonia. Note the small, red, solid areas of lung tissue caused directly by PPR virus infection.

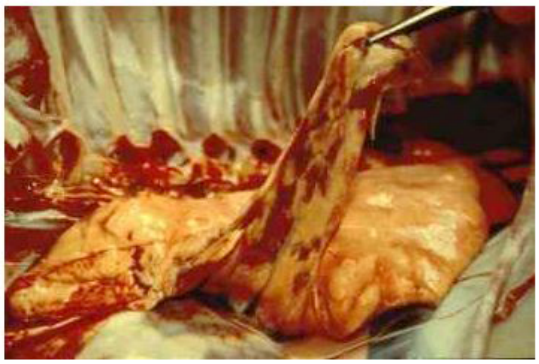


Figure 9:

PPR in a sheep: advanced pneumonia. Note the extensive dark red/purple areas, firm to the touch, in the anterior and cardiac lobes of the lungs. Although such pneumonia is commonly seen in PPR, it is caused by secondary bacterial infection, most commonly *Pasteurella haemolytica.* These lesions are typical of pneumonic pasteurellosis.

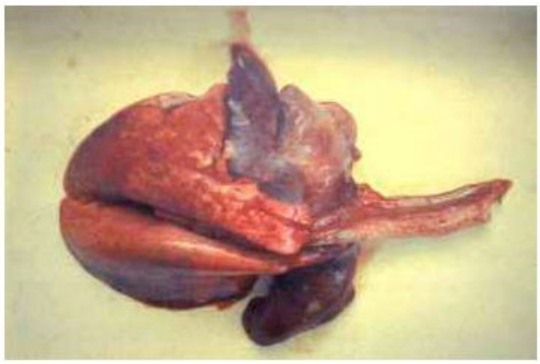
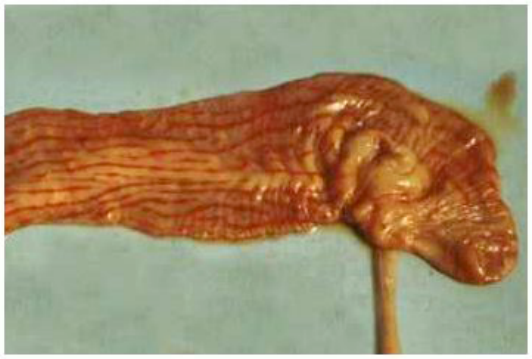


Figure 10:

PPR in a goat: "zebra striping" in the large intestine. Note the lines of hemorrhage along the tips of the folds of the lining of the caecum and colon. Later, individual hemorrhages join up and, after death, turn black.



Diseases with signs clinically indistinguishable from PPR

PPR is very similar to other diseases occurring in sheep and goats, making its diagnosis difficult and often confusing. The following diseases appear clinically similar to PPR:

Rinderpest is a viral disease caused by a morbillivirus of the family *Paramyxoviridae*. It most commonly affects sheep and goats when they have had close contact with infected cattle. It is primarily seen in Asia. The clinical signs of Rinderpest and PPR are very similar, and disease confirmation requires a specialized laboratory. Due to the progress the Global Rinderpest Eradication Program has made in eradicating this disease, it is extremely important to correctly differentiate it from PPR.

Contagious caprine pleuropneumonia is a bacterial disease caused by *Mycoplasma.* It only affects goats and is characterized by fever, difficulty breathing, and coughing. Diarrhea and oral lesions are not commonly associated with this infection.

Pneumonic pasteurellosis is a bacterial disease caused by *Mannheimia haemolytica.* It affects both sheep and goats and causes only respiratory signs associated with pneumonia.

Foot-and-mouth disease is a viral disease caused by an *Apthovirus* of the family *Picornaviridae.* It is highly contagious and affects all cloven-hooved animals. Clinical signs associated with this infection include lesions in the oral cavity that are often small and do not exhibit foul smells. Respiratory signs and diarrhea are not associated with this disease.

Bluetongue viral infections are caused by an *Orbivirus.* Sheep are most commonly affected, and other domestic ruminants rarely show clinical signs. The disease can manifest as primarily a reproductive illness resulting in abortion, stillbirth, and weak lambs. Bluetongue can also progress as a vascular disease resulting in edema (fluid accumulation) of various tissues including the face, lips, muzzle, ears, and lungs.

Contagious ecthyma, which is also known as Orf, is caused by a *poxvirus*. The virus infects sheep and especially lambs. It is characterized by vesicle and pustule formation on the lips, nostrils, face, and eyelids as well as feet and udders.

Clinical features of PPR in sheep and goats PPR has 3 forms of disease, each with varying levels of clinical signs and associated death rates.	
Peracute	
Susceptible Population	New populations
Signs of Disease	• Acute high fever, 40 to 41.3°C (104 to 106°F) • Extreme depression • Death
Death Rate	Close to 100 percent
Acute	
Susceptible Population	New populations and populations in endemic regions
Signs of Disease	• High fever, 40 to 41.3°C (104 to 105°F) • Discharge from eyes and nose • Reddening of the mucus membranes • Pinpoint spots of tissue death (appear grey) on the mucous membranes that coalesce over time and result in tissue slough, erosions, and a foul smell • Diarrhea that is profuse, foul smelling, and may contain blood and sloughed tissue from the gastrointestinal tract • Difficulty breathing and flared nostrils • Increased respiratory rate • Coughing • Dehydration • Refusal to eat and emaciation • Abortion • Hypothermia • Death
Death Rate	Ranging from 20 to 90 percent
Subacute	
Susceptible Population	Populations in endemic regions
Signs of Disease	• Difficulty breathing and increased respiratory rate • Coughing • May be asymptomatic
Death Rate	Ranging from 20 to 90 percent

Economic importance and impact of PPR

PPR is considered a disease of major economic impact, particularly in the inter-tropical regions of Africa, in the Arabian Peninsula, the Middle East, and Asia. Small ruminant farming is critical to alleviate poverty in areas such as these, where sheep and goats play an important role in food security and income generation for many families. Aside from animal death, the disease also affects animal weight and milk production.

PPR is extremely virulent, with mortality rates as high as 80 to 100 percent in acute outbreaks, making it effectively capable of wiping out large portions of a flock. Although data are incomplete on PPR's economic impact, in Kenya, for example, direct and indirect losses from PPR were estimated at nearly $12 million (U.S.) per year. The financial damage includes lost income from milk and meat, the cost of disease-control measures, and lost trade. For the world's 1 billion poor farmers — most in sub-Saharan Africa and Asia — PPR is a devastating and direct cause of persistent poverty. Already present across a wide swath of Asia, the Near East, and Africa, the virus continues to spread to new countries and threatens an increasing number of livestock keepers and small ruminant populations.

Medical treatment for PPR

There is no treatment for PPR. However, mortality rates may be decreased by the use of drugs that control the bacterial and parasitic complications associated with viral infection.

PPR disease in humans

Peste des petits ruminants does not cause disease in humans.

General prevention and control of PPR

Useful actions to prevent or limit PPR viral outbreaks

Effective import policies and animal inspections

Reporting:

Registration of all commercial and noncommercial livestock holdings with veterinary and biosecurity authorities.

Quarantine:

Strict quarantine and control of the movements of animals, animal products, and people to prevent the spread of disease from infected to uninfected premises.

Physical barriers:

The rapid inactivation of PPR virus in the environment aids eradication efforts; this virus is thought to remain viable for less than 4 days outside the animal.

Prevent infections in susceptible wildlife and captive wild animals such as gazelles by eliminating their contact with sheep and goats.

Vaccination:

Peste des petits ruminants can be controlled in endemic areas by vaccination. Animals that recover develop a lasting immunity that persists at least 3 years and possibly throughout the animal's life.

PPR outbreak control

The control of PPR outbreaks requires quarantine, vaccination, movement control, disease monitoring, sanitary slaughter, and cleaning and disinfection. Specific control measures are outlined below:

- Whenever PPR is suspected, local veterinarians should be informed immediately. PPR is listed in the OIE Terrestrial Animal Health Code, which means countries are obligated to report disease outbreaks according to OIE criteria.
- No treatment exists for PPR infections, but medications may be helpful in reducing mortality rates by controlling secondary bacterial infections that often accompany the primary viral disease.

- Restrict movement into and out of protected areas.
  - Declaration of infected premises, dangerous contact premises, and suspect premises, along with the establishment of a restricted and control area. Declaration of these areas will ensure that the diseased and disease-free areas are well defined for domestic and international recognition and the continuation of trade.
  - Restricted areas must include any feral goat herds that may have had contact with infected or dangerous contact animals.
- Destruction (stamping-out) of all ruminants with confirmed PPR.
  - As soon as possible after the diagnosis of PPR, all sheep, goats, and camels on an infected premise should be destroyed and disposed of, preferably on the premises, using deep burial or burning.
  - If a contact premise contains relatively few susceptible animals in addition to the dangerous contact animals, all will be destroyed.
  - If the number of stock is large, with clear separation of groups, then only the dangerous contact animals need to be destroyed; the susceptible in-contact animals will be quarantined and observed for signs of disease. This approach is possible because the virus survives only a few days outside the host.
- Effective cleaning and disinfection of contaminated areas of all premises.
  - PPR virus is sensitive to a wide range of disinfectants due to its large size, lipid-containing virus envelope, and sensitivity to both acid and alkaline conditions. In general, the alkalis (sodium carbonate, sodium hydroxide) and the halogens (chloride) are suitable for disinfecting buildings, wooden structures, concrete surfaces, equipment, and vehicles. For personal disinfection, citric acid, alcohols, and iodophors are suitable.
  - The PPR virus only survives 4 days outside the host. During that time the virus can be spread through contaminated water and feed troughs as well as bedding. To prevent this type of spread, the virus can be inactivated using common disinfectants including sodium carbonate, sodium hydroxide, sodium hypochlorite, phenolic compounds, citric acid, alcohols, and iodophores.
- Infected carcasses should be disposed of properly by deep burial or burning.

- Use ring vaccination.
  - If a disease outbreak outstrips the resources available for its control by stamping out, ring vaccination may be used to provide a buffer zone of immune animals around the disease area until the outbreak can be brought under control.
  - If the disease becomes more widespread than anticipated, it may be necessary to use vaccine more extensively to supplement the continuing stamping-out strategy.

Vaccines

In general, vaccine production involves using cell cultures to produce viruses, which are then processed to become live viruses incapable of causing disease (attenuated vaccines) or nonliving, inactivated viruses (inactivated vaccines). To strengthen the immune response elicited by a vaccine, carriers called adjuvants are added before the vaccine is prepared for distribution. Ideally, the vaccine produced will be a DIVA vaccine (Differentiates between Infected and Vaccinated Animals) that allows serological testing to differentiate between animals that are vaccinated for disease prevention and those that have been naturally infected with the disease-causing virus. At this time, not all vaccines developed are considered DIVA vaccines.

Vaccine types

Attenuated (live) vaccines

Laboratory manipulation transforms the live, disease-causing virus into a live, non-disease-causing version. Vaccines of this type create a strong cellular and antibody response to the virus, resulting in lifelong

immunity. Live vaccines result in viremia (viral presence within the blood) and have a small risk for virus reassortment* that could result in a disease outbreak.

Inactivated (killed) vaccines

Laboratory methods are used to kill the virus. These vaccines are easier to store and transport and do not have the risk of viral reassortment.* The immune response initiated by these vaccines is usually considered much weaker and short lived, as compared to that of live vaccines. Typically, multiple doses and boosters are needed to achieve and maintain immunity.

Monovalent and polyvalent vaccine

A monovalent vaccine is prepared using only one serotype of the virus. A vaccine prepared using two or more serotypes of the virus is a polyvalent vaccine.

Attenuated virus vaccine for PPR

Vaccination is considered the major method of preventing PPR disease spread and occurrence. Vaccines are only beneficial when given to prevent disease and are not effective as a treatment. Although PPR virus can be classified into four lineages, only a single serotype of PPR virus is known. This allows a single vaccine to be used for mass immunization of affected animals without the need to determine which lineage of PPR is present. Identifying the PPR virus lineage may become a more essential prerequisite in the future for diagnosis, epidemiology, and control. Researchers have suggested that continuous use of vaccine not containing the specific lineage present for an area may lead to the generation of new lineages, or allow the existing population to eventually evade protection.

The current vaccine used for PPR protection is designated as a DIVA vaccine. An attenuated tissue culture vaccine based on one of the first isolates of PPR virus is widely used for vaccination of small ruminants in almost all endemic areas. Vaccinated animals will not transmit PPR to nearby healthy flocks. The vaccine appears to be safe for pregnant animals and in field conditions induces protective immunity in at least 98 percent of the vaccinated animals.

Vaccines against PPR are used in sheep and goats older than 4 months of age. Newly purchased animals should be quarantined for 21 days before vaccination to ensure they are not incubating a PPR infection. If quarantine is not an option, vaccination is recommended on the day of purchase. It is recommended that the vaccine be repeated annually.

Although it has been done in the past, the use of Rinderpest vaccine to protect against PPR is now contraindicated. Its use produces antibodies to Rinderpest in the animal, thus compromising accurate serosurveillance for the disease by creating false-positive serological results for Rinderpest and putting at risk the current status of Rinderpest as a worldwide eradicated disease.

The main limitation to the attenuated PPR vaccine is thermostability, especially in the scenario when PPR is only endemic in tropical countries. PPR virus, as with other morbilliviruses, is heat labile, and therefore heat sensitivity poses a serious problem in the live attenuated vaccines used under hot climate conditions. In addition, since the disease is prevalent in most developing countries, due to poor infrastructure it is difficult to maintain the cold temperature necessary to ensure vaccine potency. All of these factors inevitably result in the loss of vaccine potency at the end time of its use. To alleviate this drawback, development of a heat-tolerant product is needed.

Viral reassortment: Because the virus used in the vaccine is alive, it may have the ability to transform back into its disease-causing (virulent) form. Can sometimes occur when live vaccines are used.

Peste des petits ruminants disinfectants

The virus is:

- Of intermediate to large size and contains lipid, which makes it susceptible to detergents.
- Susceptible to dehydration and often does not persist long outside of the host, except in cool, moist environments.

Disinfectants generally used to inactivate the PPR virus include:

- **Soaps and detergents**
  Solid or liquid — strength as appropriate; contact time — 10 minutes
- **Oxidizing agents**
  - *Virkon®* powder — final strength, 2 to 3%; contact time — 10 minutes
  - *Sodium hypochlorite* (liquid bleach) — final strength, 2 to 3% solution in water; contact time — 10 to 30 minutes
  - *Calcium hypochlorite (*solid); contact time — 10 to 30 minutes
- **Alkalis**
  - *Sodium hydroxide (*caustic soda pellets) — final strength, 2% weight-to-volume; contact time — 10 minutes
  - *Sodium carbonate*
  - *Anhydrous* (powder) — (final strength, 4% weight-to-volume; contact time — 10 minutes
  - *Washing soda (*crystals) — final strength, 10% weight-to-volume; contact time — 30 minutes
- **Acids**
  - *Citric acid* (powder) — final strength, 0.2% weight-to-volume; contact time — 30 minutes
- **Aldehyde**
  - *Glutaraldehyde* (solution) — final strength, 2% weight-to-volume; contact time — 10 minutes

Notes:

Peste des petits ruminants

Peste des petits ruminants (PPR) is a highly contagious, infectious, often fatal viral disease affecting domestic and wild small ruminants. High death loss can be seen, especially in goats: 100 percent mortality has been reported within some herds.

Disease names: PPR can also be known as Ovine Rinderpest, Pest of Small Ruminants, Pest of Sheep and Goats, Stomatitis-Pneumoenteritis Complex or Syndrome, Pseudorinderpest of Small Ruminants, Kata, Goat Plague, and Contagious Pustular Stomatitis.

Animals affected: Antelope, buffalo, camels, cattle, deer, gazelles, giraffes, llamas, yaks, and wild and domestic sheep and goats. It can also occur in wild small ruminants (Laristan sheep, Dorcas-type gazelles, gemsbok, and Nubian ibex). Cattle, buffalo, camels and pigs can become infected with PPR virus but do not typically display signs of clinical disease and do not spread the disease to other animals.

Pathogen scientific name/type: The PPR virus belongs to the family *Paromyxoviridae* and the genus *Morbillivirus*.

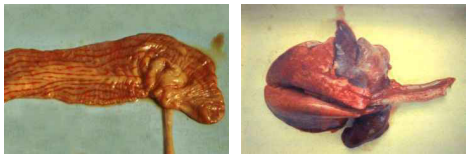
Geographic distribution: PPR was first reported in West Africa in Cote d'Ivoire in 1942. The virus has been reported throughout countries that lie between the Atlantic Ocean and the Red Sea in Africa, including south to Kenya and north to Egypt. PPR has been reported in the Islamic Republic of Iran, Iraq, Israel, Jordan, Kuwait, Lebanon, Oman, Saudi Arabia, the United Arab Emirates, and Yemen. Outbreaks also commonly occur in India, Nepal, Bangladesh, Pakistan, and Afghanistan. PPR has been seen in China and has also been reported in Morocco in North Africa.

Human disease threat: None.

Environmental stability: PPR virus is rapidly inactivated at temperatures of 70 °C (158 °F) and above; stable in pH values between 5.8 and 10; inactivated at pH values of 4 or less and greater than 11; capable of surviving in culture for at least 4 months at –20°C (-4°F), 8 weeks at 4°C (39°F), 1 week at 20–25°C (68–77°F) and >2.6 days at 37°C (98.6°F); possesses a half-life of 5 minutes in cattle blood, spleen, or lymph node at 56°C (132.8°F); capable of surviving several months in frozen and salted meat; and rapidly inactivated by ultraviolet light and desiccation within 4 days. Information is not available for PPR virus but it is assumed that, like Rinderpest virus, it would be rapidly inactivated by the putrefaction in the carcass of an animal dying from PPR or by a pH of 5.5 in hung meat. Also extrapolated from Rinderpest characteristics, virus can be present in milk for as long as 45 days after recovery.

Spread: PPR virus is spread by close contact between infected animals. The virus is shed in secretions (e.g., nasal and salivary) and excretions (e.g., feces) of infected animals. Aerosol transmission of PPR virus is also an important route of transmission. The virus can be spread over a distance of approximately 10 meters (33 feet) after becoming aerosolized. Animals are considered infectious during the incubation period, which might range from 2 to 10 days. Goats have been shown to shed the virus in feces for 11 to 12 weeks after recovery from PPR. Transmission can be through infected meat, as the virus is capable of surviving for several months in frozen and salted meat. Fomites such as water, feed troughs, and bedding can transmit PPR virus for a short time.

Pictures depicting clinical signs of ocular and nasal discharge as well as erosive lesions of the gums. Taken from: *http://www.fao.org/docrep/003/x1703e/x1703e00.htm*



Necropsy lesions of the lungs and intestines. Pictures taken from: *http://www.fao.org/docrep/003/x1703e/x1703e00.htm*

Signs: Three forms of PPR: **1. Peracute:** Acute high fever 104–106°F (40–41.3°C); extreme depression; death. Mortality approaching 100 percent. **2. Acute:** High fever 104–105°F (40–41.3°C); discharge from eyes and nose; reddening of mucous membranes; pinpoint spots of tissue death on mucous membranes which coalesce over time and result in tissue slough, erosions, and foul smell; profuse, foul-smelling diarrhea possibly containing

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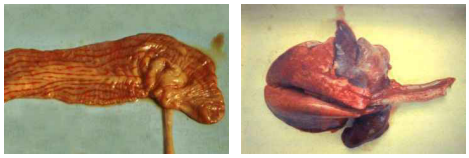

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sloughed tissue from the gastrointestinal tract; difficult breathing, with flared nostrils; increased respiratory rate; coughing; dehydration; refusal to eat; emaciation; abortion; hypothermia. Mortality ranging from 20 to 90 percent. **3. Subacute:** Seen mostly in areas where PPR is endemic. Difficulty breathing and increased respiratory rate; coughing; may be asymptomatic. Mortality ranging from 20 to 90 percent.

PPR can be confused with other diseases such as Rinderpest, Contagious Caprine Pleuropneumonia, Pneumonic Pasteurellosis, Foot and Mouth Disease, Bluetongue, and Contagious Ecthyma.

Laboratory tests: *Growth and isolation* of PPR virus confirms the presence of live virus. Virus should always be isolated from field samples even when diagnosis is made by more rapid techniques. *Polymerase chain reaction (PCR)* — Highly sensitive technique that detects presence of genetic material from PPR virus; PCR test useful for screening suspect cases; results in 5 hours; does not differentiate between presence of live or dead virus. *Agar gel immunodiffusion (AGID)* — Simple and inexpensive test capable of being performed in laboratory or field settings; not sensitive to mild forms of PPR and cannot differentiate between PPR and Rinderpest; results in 1 day. *Counterimmunoelectrophoresis (CIEP)* — Most rapid test for PPR virus antigen detection. *Virus neutralization (VN)* — Test is highly sensitive and specific but time-consuming; cross-neutralization with Rinderpest virus must be completed along with it; VN test is the prescribed test for international trade. *Immunocapture enzyme-linked immunosorbant assay (ELISA)* — Used to detect anti-PPR virus group-reactive antibodies; good for large-scale investigations; high sensitivity and specificity; differentiates between PPR and Rinderpest viruses; results available in 2 hours.

Treatment/vaccine: No current treatment is available for PPR. Supportive care and antibiotics may assist by treating associated secondary infections. PPR can be controlled in endemic areas by vaccination. Vaccines against PPR are commercially available in the live (attenuated) form for use in sheep and goats 4 months of age or older.

Prevention and control:

To prevent or limit a PPR outbreak

- Effective import policies and animal inspections.
- Registration of all commercial and noncommercial livestock holdings.
- Strict quarantine and control of the movements of animals, animal products, and people.
- Infections in susceptible wildlife and captive wild animals such as gazelles can be prevented by eliminating contact with sheep and goats.
- Peste des petits ruminants can be controlled in endemic areas by vaccination.

To control PPR during an outbreak

- Whenever PPR is suspected, local veterinarians should be informed immediately.
- Restrict movement into and out of protected areas.
  - Institute declaration of infected premises, dangerous contact premises, suspect premises, and establish restricted and control areas.
  - Restricted areas must include any feral herds that may have had contact with infected or dangerous animals.
- Destruction (stamping-out) of all ruminants with confirmed PPR.
- Carcasses resulting from stamping-out efforts on an infected premise should be destroyed and disposed of, preferably on the premises, using deep burial or burning.
- Effective cleaning and disinfection of contaminated areas of all premises.
- Use ring vaccination.
  - If a disease outbreak outstrips the resources available for its control by stamping out, ring vaccination may be used to provide a buffer zone of immune animals around the disease area until the outbreak can be brought under control.

Epidemiological investigations

- Strict quarantines and animal movement bans around infected premises.
- Epidemiological investigations tracing sources of infection/spread; isolating sick ruminants until cause of illness is determined; standard biosecurity, including cleaning and disinfecting clothing, equipment, and vehicles entering and leaving infected premises to reduce the risk of introducing or spreading disease via fomites; and employing lab services to test ruminants suspected or known to be infected or exposed to the PPR virus.
- Disinfection using: soaps/detergents; alkalis (2% sodium hydroxide); oxidizing agents (2–3% sodium hypochlorite/calcium hypochlorite); acids (0.2% citric acid); aldehydes (2% glutaraldehyde).

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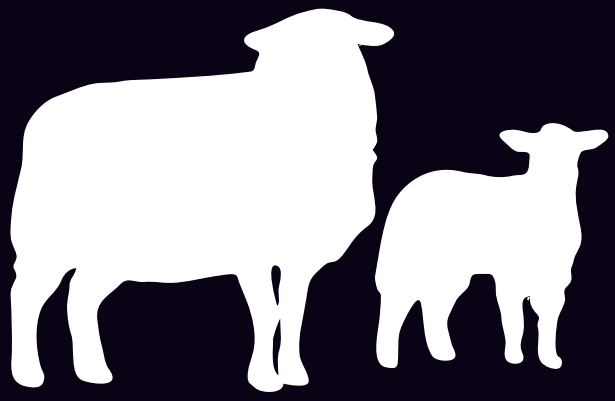

PESTE DES PETITS RUMINANTS (PPR)