RECOGNIZING PESTE DES PETITS RUMINANTS

A field manual

EMPRES
EMERGENCY PREVENTION SYSTEM

Food and Agriculture Organization of the United Nations

Table of contents

Acknowledgements

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The colour plates appear by kind permission of the following: Front cover and Plate 5 Prof. T.U. Obi Plates 1, 4, 7, 8 and 11 Dr P.L. Roeder Plates 2, 3, 6 and 10 Dr W.P. Taylor Plate 9 Dr P.C. Lefèvre Plate 12 Commonwealth Scientific and Industrial Resarch Organization (CSIRO), Australia.

Foreword

This booklet is one of a series prepared by FAO's Emergency System for Transboundary Animal Plants and Pests (EMPRES) (Livestock) Unit as an aid to emergency preparedness for the major transboundary diseases of livestock.
Peste des petits ruminants (PPR), which is also known as goat plague, is a disease of increasing importance in Africa and Asia wherever small ruminants form an important component of agricultural food production. It can affect a broad range of species, including some antelopes, as has already been seen in zoological garden collections but, fortunately, not in the wild. The disease was once thought to be a comparatively restricted problem in West Africa, but it is now known to extend throughout most of West, Central and East Africa, reaching eastwards through western and South Asia. Undoubtedly much of this increased recognition is owing to greater awareness and the availability of new laboratory diagnostic tests. However, it is possible that the disease has actually spread, rather than just being more frequently recognized. Dramatic PPR events in the last decade in Asia and East Africa suggest that the disease may be growing in severity in newly invaded areas. Many of the other regions where small ruminant production is a significant contributor to food security are close to the PPR-affected area and there is a serious risk that the disease will spread to them, especially in southern Africa and the Central Asian republics.

Early warning is the key to early reaction for containment, control and rapid elimination.

PPR may have passed unrecognized for years in some countries because it is frequently confused with other diseases that cause respiratory problems and mortality of small ruminants. Many veterinarians, animal health workers and livestock owners in areas where PPR is absent or recently introduced are not familiar with its clinical and pathological features. The manual has been prepared to help them recognize this transboundary disease as it emerges and evolves. PPR is an important disease in its own right, but it is also very important that it be differentiated from rinderpest, particularly as the Global Rinderpest Eradication Programme (GREP) proceeds to the anticipated eradication of rinderpest by the year 2010. The aim of this manual is to make all concerned with the health of small ruminants "think PPR" and recognize it rapidly when it occurs. Comments and suggestions for improvement are welcomed for use in preparing subsequent editions.

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Introduction

Peste des petits ruminants (PPR) is a highly contagious and infectious viral disease of domestic and wild small ruminants. It was first described in Côte d'Ivoire in West Africa in 1942. Gradually it was realized that several clinically similar diseases occurring in other parts of West Africa shared the same cause - the virus now called peste des petits ruminants. Investigators soon confirmed the existence of the disease in Nigeria, Senegal and Ghana. For many years it was thought that it was restricted to that part of the African continent until a disease of goats in the Sudan, which was originally diagnosed as rinderpest in 1972, was confirmed to be PPR. The true extent of the disease has only become apparent in recent years and is still being clarified. The realization that many of the cases diagnosed as rinderpest among small ruminants in India may, instead, have involved the PPR virus, together with the emergence of the disease in other parts of western and South Asia, point to its ever-increasing importance.

PPR is an important disease in its own right, but it has also created problems because of its apparent similarity to rinderpest - the clinical signs of PPR closely resemble those of rinderpest, making differential diagnosis difficult. It should, however, be borne in mind that clinical disease caused by rinderpest in small ruminants is a relatively rare event, even in Asia.

The disease

PPR is a severe, fast-spreading disease of mainly domestic small ruminants. It is characterized by the sudden onset of depression, fever, discharges from the eyes and nose, sores in the mouth, disturbed breathing and cough, foul-smelling diarrhoea and death.

The cause

The virus which causes PPR, the peste des petits ruminants virus (PPRV), belongs to the morbillivirus group of the paramyxovirus family of viruses. It is closely related to the rinderpest virus of cattle and buffaloes, the measles virus of humans, the distemper virus of dogs and some wild carnivores, and the morbilliviruses of aquatic mammals. To date, genetic characterization of PPR virus strains has allowed them to be organized into four groups; three from Africa and one from Asia. One of the African groups of PPRV is also found in Asia. The epidemiological significance of these groupings is less clear at present than that of rinderpest virus groupings.

Animals affected

Clinical disease is seen in sheep and goats and has been described in zoological garden collections of wild small ruminants including Laristan sheep, Dorcas-type gazelles, gemsbok and the Nubian ibex. Cattle, buffaloes, camels and pigs can become infected but there is little or no evidence of disease associated with their infection. Probable global distribution of PPR virus infection Note: Clinical disease has not been reported by every country; for some only serological evidence exists. Source: Based on official reports to the International Office of Epizootics (OIE); published reports; other official country reports; and reports of FAO Reference and Collaborating Centres (1999).

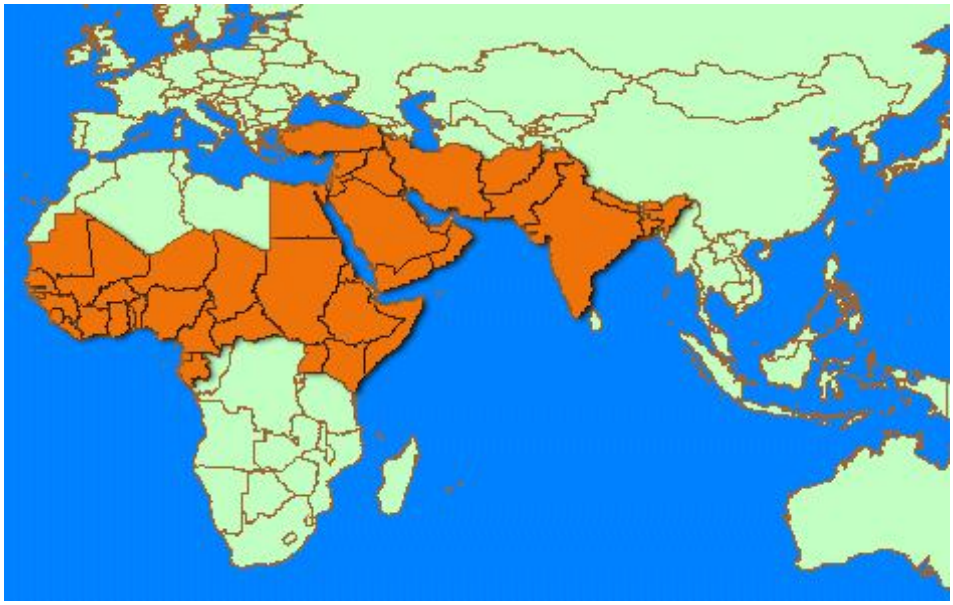
Geographical distribution

PPR infection has been recognized in many of the African countries that lie between the Atlantic Ocean and the Red Sea. The affected area extends north to Egypt and south to Kenya, in the east, and Gabon, in the west. PPR has not been recognized in most of North and southern Africa. In some of the countries where the disease has not been confirmed there are serological and/or clinical indications that the infection is, nevertheless, present. A recent (1998) serological survey in the United Republic of Tanzania did not detect any antibodies to PPR suggesting that infection has not extended that far south.

In recent years the disease has been seen in the Near East and the Arabian Peninsula, in countries including the Islamic Republic of Iran, Iraq, Israel, Jordan, Kuwait, Lebanon, Oman, Saudi Arabia, the United Arab Emirates and Yemen, and there is serological evidence from the Syrian Arab Republic and Turkey. Outbreaks of PPR are now known to be common in India, Nepal, Bangladesh, Pakistan and Afghanistan.

Countries that have imported small ruminants from these areas are advised to investigate thoroughly any disease syndrome characterized by disturbed breathing, discharges from the eyes, nose and mouth, sores in the mouth and diarrhoea in order to rule out PPR.

It is still not clear whether the apparent geographical spread of the disease in the last 50 years is real or whether it reflects increased awareness, wider availability of diagnostic tools or even a change in the nature of the virus. It seems most likely that a combination of factors is responsible for the present knowledge of its range and it is known that confusion of PPR with pneumonic pasteurellosis and other pneumonic diseases of small ruminants has delayed its recognition in some countries.



Probable global distribution of PPR virus infection

N.B. Clinical disease has not been reported by very country; for some only serological evidience exists. Based on official reports to the OIE; published reports, other official coutry reports and reports of FAO Reference and Collaborating Centres

Transmission and spread

The discharges from eyes, nose and mouth, as well as the loose faeces, contain large amounts of the virus. Fine infective droplets are released into the air from these secretions and excretions, particularly when affected animals cough and sneeze. Other animals inhale the droplets and are likely to become infected. Although close contact is the most important way of transmitting the disease, it is suspected that infectious materials can also contaminate water and feed troughs and bedding, turning them into additional sources of infection. These particular hazards are, however, probably fairly short-term since the PPR virus, like its close relative rinderpest, would not be expected to survive for long outside the host.

Trade in small ruminants, at markets where animals from different sources are brought into close contact with one another, affords increased opportunities for PPR transmission, as does the development of intensive fattening units. Appearance of disease in a herd or flock

When PPR occurs in an area for the first time, it is possible that acute high fever with extreme depression and death occur before any other typical signs have been seen. A more typical picture, however, is that of a fast-spreading syndrome in sheep and/or goats characterized by the sudden onset of depression, discharges from eyes, nose and mouth, abnormal breathing with coughing, diarrhoea and deaths. The outbreak will not involve cattle, whether rinderpest vaccinated or not, even if they are in contact with affected sheep and/or goats. Although both goats and sheep are susceptible to infection and may show disease, they are not always affected simultaneously. For example, in Africa PPR is seen most commonly in goats, while in western and South Asia sheep are usually the most noticeable victims. The disease can, however, strike both species with equally devastating consequences.

The appearance of clinical PPR may be associated with any of the following:

- history of recent movement or gathering together of sheep and/or goats of different ages with or without associated changes in housing and feeding;
- introduction of recently purchased animals; contact in a closed/village flock with sheep and/or goats that had been sent to market but returned unsold;
- change in weather such as the onset of the rainy season (hot and humid) or dry, cold periods (for example the harmattan season in West Africa);
  contact with trade or nomadic animals through shared grazing, water and/or housing;
- a change in husbandry (e.g. towards increased intensification) and trading practices.

In endemic areas, most of the sick and dying animals are over four months and up to 18 to 24 months of age.

Clinical signs

Clinical signs appear an average of two to six days after natural infection with the virus (the incubation period). This is followed by the sudden onset of fever with rectal temperature of at least 40° to 41oC. Affected animals are markedly depressed and appear sleepy. Their hair stands erect giving them a bloated appearance, especially the short-haired breeds. Soon after this stage, a clear watery discharge starts to issue from the eyes, nose and mouth, later becoming thick and yellow as a result of secondary bacterial infection (Figure 1). The discharges wet the chin and the hair below the eye; they tend to dry, causing matting together of the eyelids, obstruction of the nose and difficulty in breathing.

One to two days after fever has set in, the mucous membranes of the mouth and eyes become very reddened (Figure 2). Then epithelial necrosis causes small pin-point greyish areas to appear on the gums, dental pad, palate, lips, inner aspects of the cheeks and upper surface of the tongue. These areas increase in number and size and join together. The lining of the mouth is changed in appearance. It becomes pale and coated with dying cells (Figure 3) and, in some cases, the normal membrane may be completely obscured by a thick cheesy material (Figure 4). Underneath the dead surface cells there are shallow erosions. In mild cases these changes may not be severe and will require careful examination to be seen. Gentle rubbing across the gum and palate with a finger may yield a foul-smelling material containing shreds of epithelial tissue. Similar changes may also be seen in the mucous membranes of the nose, the vulva and the vagina. The lips tend to swell and crack and become covered with scabs (Figure 5).

As the disease progresses, a characteristic foul smell exudes from the mouth. Affected animals resist attempts to open their mouths because of the pain.

Diarrhoea commonly appears about two to three days after the onset of fever (Figure 6) although, in early or mild cases, it may not be obvious. The faeces are initially soft and then watery, foul-smelling and may contain blood streaks and pieces of dead gut tissue. Where diarrhoea is not an obvious presenting sign, the insertion of a cotton wool swab into the rectum may reveal evidence of soft faeces which may be stained with blood.

Affected animals breathe fast, sometimes so fast that they exhibit rocking movements with both the chest and abdominal walls moving as the animal breathes. Severely affected cases show difficult and noisy breathing marked by extension of the head and neck, dilation of the nostrils, protrusion of the tongue and soft painful coughs - they have obvious signs of pneumonia.

Such victims may eventually become dehydrated with sunken eyeballs, and death often follows within seven to ten days from onset of the clinical reaction. Other animals will recover after a protracted convalescence.

A common feature in later stages of the disease is the formation of small nodular lesions in the skin on the outside of the lips around the muzzle (Figure 7). The exact cause of these is not known (possibly Dermatophilus infection or reactivation of a latent contagious ecthyma infection - orf or "sore mouth") but they cause confusion because of their similarity to the symptoms of primary contagious ecthyma or even sheep/goat pox.

Up to 100 percent of the animals in a flock may be affected in a PPR outbreak with between 20 and 90 percent dying. These proportions are usually lower in endemic areas where older animals have survived earlier infection. Pregnant animals may abort.

In summary, suspect PPR if you see any combination of:

- the sudden onset of a febrile illness affecting sheep and/or goats; eye, nose and mouth discharges with sores in the mouth, with or without scabs or nodules around the mouth;
- pneumonia;
- a significant death rate.

Any appearance of one or more of these signs in combination must be considered suspicious.

Post mortem findings

The carcass of an affected animal is usually emaciated, the hindquarters soiled with soft/watery faeces and the eyeballs sunken. The eyes and nose contain dried-up discharges. The following changes may be seen:

Mouth

Dirty-white, false membranes; erosions on the gums, soft and hard palates, tongue and cheeks and into the oesophagus.

Lips

Swollen; erosions and possibly scabs or nodules in late cases.

Nasal cavity

Congested (reddened) lining; clear or creamy yellow exudates; erosions.

Lungs

Dark red or purple areas; firm to the touch, mainly in the anterior and cardiac lobes (evidence of pneumonia) (Figures 8 and 9).

Lymph nodes (associated with the lungs and the intestines)

Soft and swollen. Abomasum Congested (reddened) lining; haemorrhages.

Small intestines

Congested (reddened) lining; haemorrhages; some erosions.

Large intestines (caecum, colon and rectum)

Small red haemorrhages along the folds of the lining, joining together as time passes and becoming darker, even green/black in stale carcasses (Figure 10).

Differential diagnosis

PPR is frequently confused with other diseases that present fever and grossly similar clinical signs, especially when it is newly introduced. When carrying out an investigation, examination of the way the disease behaves in the herd or flock is as important as the findings on a single goat or sheep. The most frequent sources of confusion are:

Mouth lesions

Could be a symptom of: rinderpest, foot-and-mouth disease, bluetongue or contagious ecthyma (orf or "sore mouth").

Difficult breathing

Could be a symptom of: pneumonic pasteurellosis or contagious caprine pleuropneumonia (CCPP).

Diarrhoea

Could be a symptom of: coccidiosis or gastro-intestinal helminth infestations. Pneumonia is usually a very obvious presenting sign in PPR so, without doubt, pneumonic pasteurellosis and CCPP have caused the most difficulty in differential diagnosis.

Pneumonic pasteurellosis

is a purely respiratory disease of sheep and goats caused by the bacterium Pasteurella haemolytica. Dark red/purple areas, firm to the touch, are evident mainly in the anterior and cardiac lobes of the lung (Figure 9). There are no oral lesions or diarrhoea. The numbers of affected and dead animals are usually lower than for PPR except under exceptional conditions of stress and crowding such as can occur when large numbers of sheep are assembled for trade. The main problem of differentiation arises when oral lesions and diarrhoea are either absent or not very obvious in PPR, as is sometimes the case. Using appropriate culture media, Pasteurella haemolytica bacteria are easily isolated in pure and profuse culture from pneumonic lungs of sheep, even from the lungs of PPR-affected animals. Isolation of Pasteurella haemolytica bacteria from the lungs of sheep, therefore, neither confirms a diagnosis of primary pneumonic pasteurellosis nor

rules out the presence of PPR. Diagnostic tests for detecting PPRV should be carried out in all suspected cases of pneumonic pasteurellosis where there is a risk of PPR.

Contagious caprine pleuropneumonia (CCPP)

is a disease of goats (sheep are not affected) caused by a Mycoplasma sp. Like PPR, it is characterized by fever, difficult/abnormal breathing and coughing, but there mouth lesions or diarrhoea are not present in CCPP. At post mortem examination, the lung lesions in CCPP are more diffuse and a fibrinous fluid is found in the chest cavity. Fibrin deposits cover the lungs and are frequently connected to the chest wall by fibrinous strands (Figure 11). In PPR high-risk areas it is advisable to rule out PPR by laboratory testing of, at least, serum samples from convalescent flocks, even if CCPP is suspected.

Rinderpest disease

in small ruminants has been described primarily in Asia. Generally, this disease occurs in small ruminants only when they are in contact with affected cattle or buffaloes, so it is important during investigations to examine all species. Confirmation requires the resources of a specialist laboratory (see Sources of assistance). The samples required for laboratory confirmation of both rinderpest and PPR are identical. As the Global Rinderpest Eradication Programme (GREP) progresses, it becomes increasingly important that PPR and rinderpest be differentiated because, at this stage of the programme, any outbreak of rinderpest anywhere represents an international emergency.

Foot-and-mouth disease (FMD)

is more commonly seen in sheep than goats. The most important distinguishing features of FMD, other than the appearance of the lesions, are the absence of breathing problems and diarrhoea, and the presence of lameness (often marked). Sudden death of very young lambs without other signs often occurs. The oral lesions when present are often very small and difficult to see; the mouth does not exude such a foul odour as in PPR. Bluetongue, like PPR, is characterized by fever, discharges and oral lesions (Figure 12). However, it differs from PPR in: the presence of oedema of the head region; bluish discoloration of the oral cavity, the coronary band of the hooves and the less hairy parts of the body; and lameness.

Bluetongue

virus infection is endemic throughout the regions of the world affected by PPR. Clinical disease is, however, not generally experienced in indigenous breeds in these countries, being mainly restricted to exotic introduced animals. The presence of antibody to bluetongue viruses in single samples does not confirm a provisional diagnosis of bluetongue.

Contagious ecthyma (orf, "sore mouth", contagious pustular dermatitis)

is often confused with PPR because of the nodules and thick scabs sometimes seen on the lips in the late stages of PPR. Confusion is especially likely to arise in severe cases of orf where lesions extend into the mouth and nose. In uncomplicated orf, there is usually no oral necrosis, diarrhoea or pneumonia.

Diagnosis of PPR

The International Office of Epizootics (OIE) Manual of Standards for Diagnostic Tests and Vaccines contains guidelines on the collection of samples and the diagnostic techniques for diagnosis of PPRV infection. A provisional diagnosis of PPR can be made from epidemiological and clinical features. A disease characterized by discharges, diarrhoea, and deaths with breathing problems in sheep and/or goats, but not in-contact cattle, with mainly adolescents being affected and dying must arouse a suspicion of PPR. The observation of characteristic post mortem changes would further strengthen the provisional diagnosis.

Laboratory confirmation

Because of the necessity to detect PPR amid a number of other acute diseases with grossly similar presenting signs, and to differentiate it from rinderpest, some laboratory tests need to be carried out. These tests may detect the virus itself, evidence of the presence of the virus (virus antigen or genetic material) or antibodies against the virus found in blood serum.

Detection of virus antigens by the agar gel immunodiffusion test (AGIDT) is a relatively simple, fast

and cheap process. It is extremely useful as an initial test, but it does not discriminate between PPR and rinderpest viruses and further tests are needed to do this. Histopathology combined with immunohistochemical staining (e.g. immunoperoxidase) is a useful procedure because it is performed on formalin-fixed material and can discriminate between PPR and rinderpest when performed with specific monoclonal antibodies. Virus antigens can also be detected by immunocapture ELISA (ICE) which is rapid and sensitive, and differentiates between PPR and rinderpest. Standardized reagent kits are commercially available for AGIDT and ICE.

Detection of virus genetic material is performed by the reverse transcriptase polymerase chain reaction (RT PCR) which requires specialist facilities and expertise. Despite its high cost, it is now one of the tests used most frequently in reference centres, together with enzyme linked immunosorbent assay(ELISA), because it is rapid, accurate, highly sensitive and can discriminate between PPR and rinderpest. Combining this test with nucleotide sequencing provides virus characterization information that is useful in epidemiological studies. Detection of the virus is done by isolation of the PPR virus in cultured cells. This method of diagnosis can be very valuable as it provides live virus for biological characterization studies. If facilities are available, it should always be attempted and isolated viruses stored for later studies.

Detection of antibodies for diagnosis requires the collection of two blood samples, three weeks apart, from the same animals, which is not always feasible in the field. Exceptionally, in a country that can be certain that it was free from PPR, testing single samples taken late in the course of the disease (at least a week after the appearance of clinical signs) can be diagnostic. Surveys for antibodies are very useful to determine the presence or absence of infection and its extent in a population. Competitive ELISA has now largely replaced the virus neutralization test.

Samples required for laboratory testing

The chances of a successful laboratory confirmation of the clinical diagnosis increase as the numbers of samples examined and animals sampled increase. There are several important points to observe when using the services of a laboratory:

1. Provide epidemiological and clinical details with the samples.
2. Always sample several animals in an outbreak.
3. Keep samples cool during transfer to the laboratory (preferably on melting ice) and reduce the time in transit to the minimum.
4. Mark sample bottles carefully with an indelible pen and record details of each sample's origin for submission to the laboratory.

The samples required are:

Tears

Cotton buds or swabs of absorbent cotton wool are inserted into the conjunctival sac and swirled around to collect tears. The bud/swab is broken off into a container and about 150 microlitres of sterile phosphate-buffered saline (PBS pH 7.2 to 7.6) are added (if available).

Gum debris

This material can be collected by a spatula or finger rubbed across the gum and inside the upper and lower lips. The material collected is then scraped into a container and 150 microlitres of PBS are added (if available).

Tissues

It is recommended that the following tissues be collected during post mortem examination: lymph nodes found around the lungs (mediastinal) and alimentary tract (mesenteric); portions of the spleen and the lungs.

Two sets of each tissue are required; one set is chilled but not frozen, and the other is put in 10 percent formalin solution to preserve the samples. Where cold storage is a problem, as is often the case, formalin can be used to preserve the samples when they are sent to the laboratory.

Unclotted blood

This is needed for virus isolation and should be collected in bottles containing anticoagulants (heparin or ethylenediamine tetracetic acid [EDTA]).

Clotted blood or serum

These are needed for antibody detection.

National laboratories will provide guidance about exactly which samples are required, but it is advisable to collect as many of the samples listed above as possible when dealing with an outbreak.

Control of PPR

Control of PPR outbreaks relies on movement control (quarantine) combined with the use of focused ("ring") vaccination and prophylactic immunization in high-risk populations. Until recently, the most practical vaccination against PPR made use of tissue culture rinderpest vaccine. Recently, a homologous PPR vaccine has been developed and the vaccine seed is available through the Pan African Veterinary Vaccine Centre (PANVAC) at Debre Zeit, Ethiopia, for Africa, or CIRAD-EMVT at Montpellier, France, for other areas. This vaccine of choice is becoming increasingly available. The vaccines can protect small ruminants against PPR for at least three years.

The use of rinderpest vaccine to protect small ruminants against PPR is now contraindicated because its use produces antibodies to rinderpest which compromise serosurveillance for rinderpest, and thereby the Global Rinderpest Eradication Programme.

Sources of assistance

Differentiating between rinderpest and PPR to obtain a definitive identification of PPR can be difficult, especially when the disease is encountered for the first time and national laboratories lack adequate facilities. Samples for diagnostic confirmation can be submitted to either the FAO World Reference Laboratory for Rinderpest at the Institute for Animal Health, Pirbight Laboratory, United Kingdom or the FAO Collaborating Centre at the International Cooperation Centre on Agrarian Research and Development, Department of Breeding and Tropical Veterinary Medicine (CIRAD-EMVT) Laboratory, Montpellier, France, which can assist with the diagnosis of PPR. Addresses are given below.

It should be noted that submission of samples to any laboratory outside the country of origin is always subject to prior agreement with the recipient and transportation in containers meeting International Air Transport Association (IATA) regulation standards. Detailed instructions for the collection and dispatch of rinderpest samples (which are also applicable to PPR samples) are contained in the publication ***Collection and submission of diagnostic specimens to the FAO World Reference Laboratory for Rinderpest,*** which can be obtained from FAO EMPRES; it can also be supplied electronically as an attachment to e-mail or by fax on request.

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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.

FIGURE 3:
PPR in a goat: early mouth lesions showing areas of dead cells
Early pale, grey areas of dead cells on the gums.

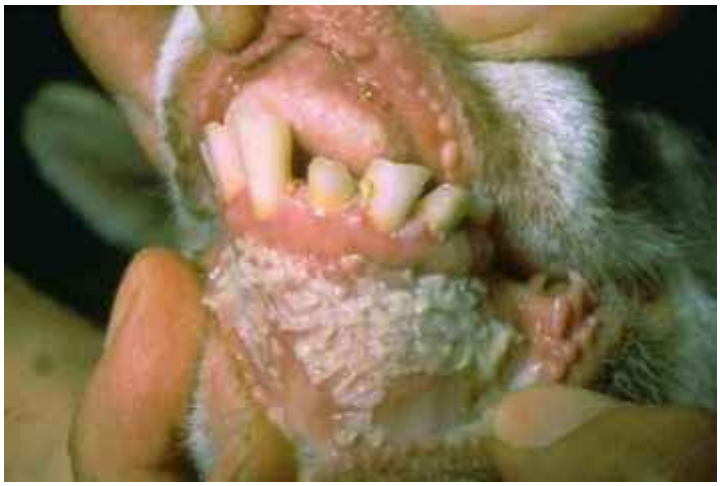

FIGURE 4:
PPR in a goat: later mouth lesions
The membrane lining the mouth is completely obscured by a thick cheesy material; shallow erosions are found underneath the dead surface cells.

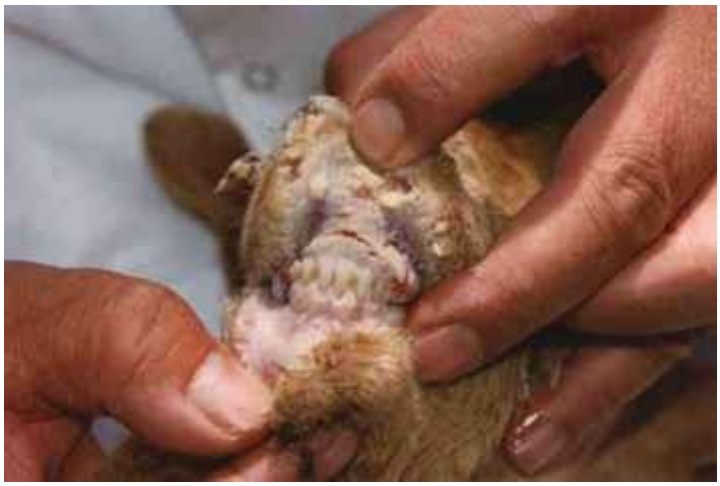

FIGURE 5:
PPR in a goat: swollen, eroded lips
The lips are swollen, oedematous and show areas of erosion.

FIGURE 6:
PPR in a goat: signs of diarrhoea
The hindquarters are soiled with liquid faeces.

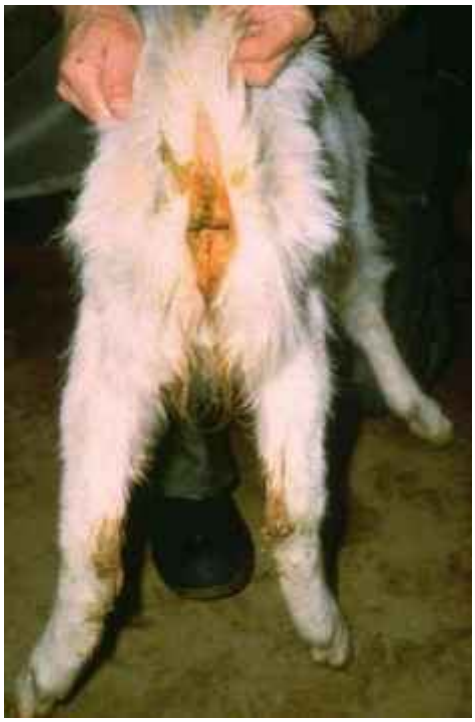

FIGURE 7:
PPR in a goat: nodular lesions around the mouth
Such nodules are a common finding in the later stages of PPR infection.

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.

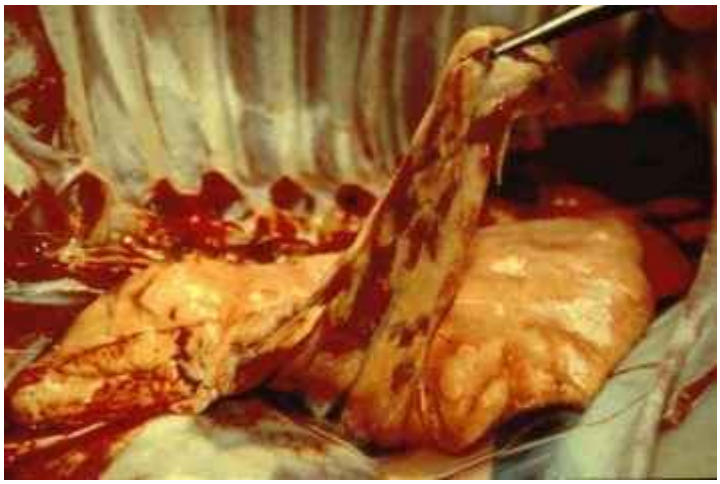

FIGURE 9:
PPR in a sheep: advanced pneumonia
Note the extensive, dark red/purple areas, firm to the touch, in the anterior and cardiac lobes of the lungs. Although such pneumonia is commonly seen in PPR, it is caused by secondary bacterial infection, most commonly Pasteurella haemolytica. These lesions are typical of pneumonic pasteurellosis.

FIGURE 10:
PPR in a goat: "zebra striping" in the large intestine
Note the lines of haemorrhage along the tips of the folds of the lining of the caecum and colon. Later, the individual haemorrhages join up and, after death, turn black.

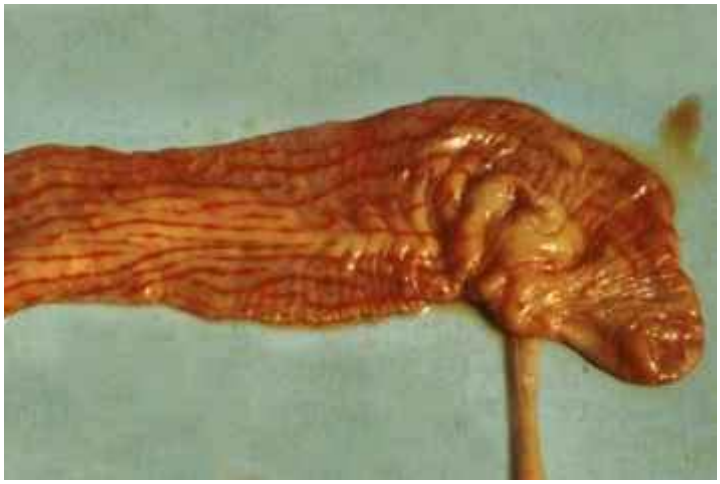

FIGURE 11:
Typical lesions of contagious caprine pleuropneumonia (CCPP) in a goat
Note the yellowish, fibrinous deposit on the surface of the lungs and adhesions to the inside of the rib cage.

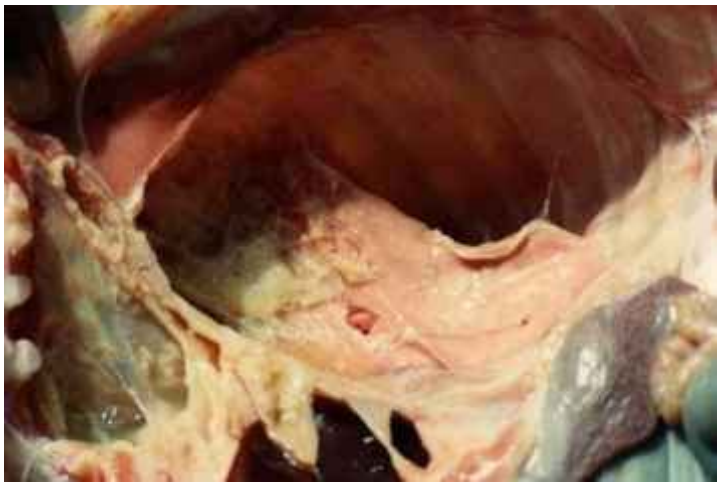

FIGURE 12:
Bluetongue disease in a sheep
Note the bluish discoloration of the coronary bands of the hoof. The lips will usually be found to be swollen and discoloured blue at the same time.