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Reviewon Peste Des Petits Ruminants (PPR)

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Abstract: A Peste des Petits Ruminant is a contagious disease affecting goats and sheep in Sub-Saharan Africa extending to the Arabian Peninsula, Middle Eastern countries and India. It is a list a disease that is caused by the *Morbillivirus* (family *Paramyxoviridae*). Peste des Petits Ruminant Virusis related to Rinderpest virus which it shares common antigenic determinant. Goats are more susceptible to Peste des Petits Ruminant than sheep and kidsover four months and less than one year of age are also most susceptible. Morbidity and mortality can be as high as 100%and 90%, respectively. Wild animals are not believed to play a role in the spread of the virus. The disease is characterized by fever, anorexia, necrotic stomatitis, diarrhea, occulonasal purulent discharge and respiratory distress. A presumptive diagnosis is based on clinical, pathologic and epidemiological findings and may be confirmed by viral isolation and identification. There is no treatment for Peste des Petits Ruminants but animals can be treated with antibiotics to prevent secondary infections. Vaccination is the most effective way to control the disease. Methods applied for Peste des Petits Ruminants eradication includes quarantine, slaughtering, proper disposal of carcasses and contact fomites, decontamination of facilities and equipment and restriction on importation of sheep and goats from infected areas.

Key words: Peste Des Petits Ruminants · Sheep · Goat · Vaccination

INTRODUCTION

Peste desPetits Ruminants (PPR)is also known as Ovine Rinderpest, it is a contagious disease affecting goats and sheep in Africa (from Tropic of Cancer to Equator), the Middle East and the Indian Subcontinent. But, since June 2008, the disease invaded Moroccowhich indicates a crossing of the natural barrier of Sahara [1]. Peste desPetits Ruminants also known as kata, stomatitis-pneumoenteritis complex, goat plague and pseudo-rinderpest [2]. It is a list-A disease and an acute contagious disease of ruminants, particularly goats that caused by *Morbilivirus*, peste des petits ruminants virus (PPRV), which is closely related to the other members of the genus. It is in family *Paramixoviridae* [3].

PPRischaracterized by fever, anorexia, necrotic stomatitis, diarrhea, occulonasal purulent discharge and respiratory distress. It is a Rinderpest-like disease in Africa and Asia and is highly fatal in goats, less so sheep [4]. A presumptive diagnosis, based on clinical, pathologic and epidemiologic findings [5]. Definitive diagnosis requires viral isolation and identification [6].

No specific measures are available to treat the disease but antibioticsto prevent secondary infection."Stamping out" policies of slaughter and movement restrictions are appropriate where the disease appears in a previously unaffected area. Control of PPR outbreaks relies on movement control (quarantine) combined with focused ("ring") vaccination and prophylactic immunization in high risk population [7]. Therefore, the major objectives of this seminar paper are to review the impact of peste des petitsruminants on small ruminants, the current status of the disease and to point out its prevention and control measures.

Peste Des Petits Ruminants (PPR, Goat Plague Or Kata)

Historical Background: Peste des petits ruminants (PPR) was first described in 1942 by Gorgadonnec and Lalanne, who investigated the syndrome in sheep *Oviesaries* and goat *Capra hircus* in Côte d'Ivoire, West Africa. Although rinderpest virus can infect goats and sheep in Africa, most experimental infections are mild or subclincal. Therefore, it seems probable descriptions ofsevere rinderpestin small ruminants, particularly were actually

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PPR. This was demonstrated that the specific antisera and transmission studies to confirm that virus isolates originally believed to the rinderpest virus from goats in Sudan were actually the first isolates of PPR virus in eastern Africa [8].

The name peste des petitsruminants (plague of small ruminants) reflect two things about this disease. First, that it was initially described from Francophone West Africa and second, that it is a disease that kills a large number of sheep and goats [9]. Many authors prefer the name "Ovine Rinderpest". But, official agencies such as FAO and OIE use the French name "Peste des Petits Ruminants", "Peste-des-Petits-Ruminants" or "Peste-des-petits-ruminants", even in English. The French acronym, PPR, is commonly used among veterinary professionals in East Africa [1].

Etiology: Peste des Petits Puminants (PPR), also known as goat plague, is caused by *Paramyxovirus* of the *Morbillivirus* genus. It was first described in 1942 in Côte d'Ivoire, West Africa and is closely related to rinderpest virus, canine distemper and human measles virus. The virus has the following characteristics: It may survive at 60°C for 60 minutes, stable from pH 4.0 to 10.0, can be killed by alcohol, ether, and detergents as wellas by most disinfectants and longsurvival time with chilled and frozen tissues [10].

There are four lineages of PPR viruses have been identified; lineage 1 and 2 viruses in West Africa, lineage 3 in East Africa, Arabian and Southern India and lineage 4 in the Middle East and Asia subcontinent, reaching east as far as Nepal and Bangladesh. All four lineages have been shown to be genetically distinct from the rinderpest virus[4].

Table 1: Classification of PPR

Peste Des Petits Ruminants Virus Classification	
Group :	Group V ((-)ssRNA)
Order :	*Mononegavirales*
Family :	*Paramyxoviridae*
Genus :	*Morbillivirus*
Species:	Peste-des-petits-ruminants virus

Source:[1].

Epidemiology

Occurrence: Peste des Petits Ruminants is endemic in Sub-saharan Africa extending to the Arabian Peninsula. It is also present in Middle Eastern countries and India [11]. Historically, the diseasewas primarily associated with West Africa, but it extends in a belt across Africa immediately South of the Sahara, extending into Arabian Peninsula[7].

Morbidity And Mortality: Morbidity and mortality can be as high as 100% and 90%, respectively. When associated with other diseasessuchascapripox, mortality can be100% [10].

Transmission: Sick goats and sheep generate aerosols containing infective droplets. Successful transmission therefore requires close contact between sick and healthy animals. Fomites do not play a role in transmission of the virus [13].

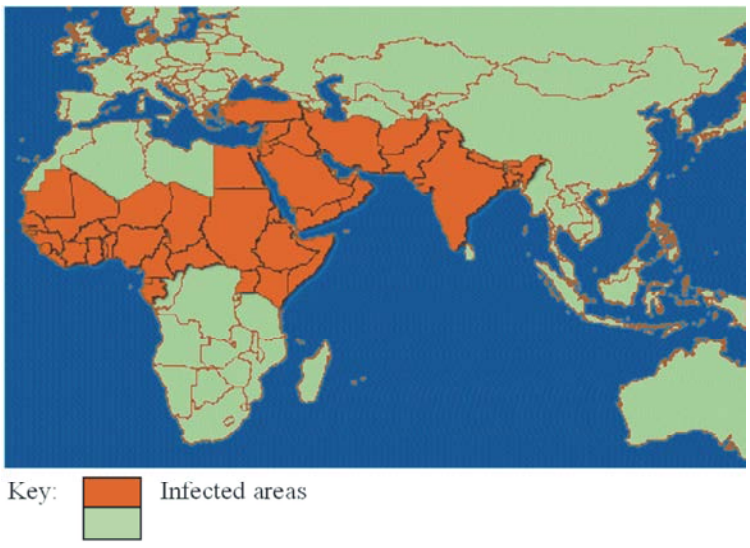

Fig. 1: Geographical distribution of PPR Key:Infected areas Source:[12]. Uninfected or no information

Risk Factors And Immune Mechanisms: Kids over four months and under one year of age are most susceptible to the disease. Sahelian breeds of sheep and goats are believed to be more resistance than the dwarf breeds in the humid and sub-humid zones of West Africa. In a particular flock, risk of an outbreak is greatly increased when a new stock is introduced or when animals are returned unsold from livestock markets. Recovered animals have lifetime immunity [4].

The disease is transmitted by direct and indirect contact [14]. Large amounts of the virus are present in all body excretions and secretions, especially in diarrheic faeces. Infection is mainly by inhalation but could also occur through conjunctiva and oral mucosa [4].

Host Range: PPR is primarily a disease of sheep and goats. There have been several reports of PPR occurring in other species, particularly caprine wild ungulate from three families: Gazellinae (*Dorcas gazelle*), Caprinae (*Nubian ibex* and Laristan sheep) and Hppotraginae (Gemsbok). Cattle, buffaloes, camels and pigs are susceptible to infection but do not inhibit clinical signs and unable to transmit the disease to other animals [10]. Goats and sheep are the natural host of PPR. Goats appear to be more susceptible and suffer a more severe clinical disease than sheep [11].

Serological evidence suggests that the virus can spread from sheep and goats to domestic cattle and buffalo. Although experiments suggest that the infection in cattle is subclinical, there may be circumstances when this is not the case. In the original study, when PPRV was inoculated into 15-month old calf, it induced pyrexia of 42°C for 48 hours [9].

Economic Importance: PPR is regarded the most important disease of goats and sheep in West Africa and possibly in all countries where the disease occurs. In many of those countries, these animals are the major sources of animal protein [4]. The study found that PPR infection resulted in better of households slipping into poverty, while the poor and very poor became destitute. The estimated livestock asset losses due to PPR range from 65 to 100% in four wealth categories that caused among other things, shifts in food consumption, food and income sources. The percentage of livestock-derived income loss due to PPR varies between 21 and 90% [15].

Juan Lubroth, FAO's Chief Veterinary Officer, noted that, "sheep and goats are critical to food and income security for pastoral communities. The presence of this disease directly affects a family's wealth, hence the veterinary service of the countries in the region must review their preparedness plans, strengthen border control and improve surveillance '' [15].

Public Health Issues: Peste des petits does not cause infection in human; therefore, there are no public health issues to be considered [1].

Biosecurity Concerns: Like rinderpest, Peste des Petits Ruminants requires close contact with an infected animal for transmission to occur. Nevertheless, goats and sheep and traded and may be carried over long distance, the disease can be easily introduced to a new herd or even a new country unknowingly from animals incubating PPR or showing only mild lesions [4].

Pathogenesis: It is assumed that the pathogenesis of PPR is similar to that of rinderpest [8]. Route of infection is respiratory and is spread by airborne droplets. All secretions and excretions of infected animals are contagious throughout the course of the disease, but no carrier state exists (Pugh, 2002). During acute phase of the disease, virus is shade in all secretions and excretions[3].

PPR viruspenetrates the retropharyngeal, mcosa, sets up a viremia and specifically damages the alimentary, respiratory and lymphoid systems. Infected cells undergo necrosis and the respiratory system, also undergoes proliferation. Death may occur from severe diarrhea and dehydration, before respiratory lesions become severe, or is hastened by concurrent diseases such as pneumonic pasteurollosis, coccidiosis or coliform enteritis. Lymphoid necrosis is not marked as in rinderpest and the possibility immunosuppression has not been investigated. Most sheep and some adult goats recover. [4].

The pathogenesis in sheep and goats in general similar to those of rinderpest, except that the disease is more acute in onset, especially in goats and follows a more rapid course. Another difference is the marked involvement of the respiratory tract; affected animals have dyspnea, hyperpnea and coughing. There is also a marked serous to mucopurulentnasal and ocular discharge and erosion or ulceration of the pharyngeal epithelium may be diffuse[2].

Symptoms And Clinical Signs: Peste des Petits Ruminants is an important disease in it is own right, but it has also created problems because it is apparently similarity to rinderpest. The clinical signs of PPR closely

resemble those ofrinderpest,this makes a differential diagnosis difficult. It should, however, be borne in mind that clinical disease used by rinderpest in small ruminants is relatively rare event [7].

The clinical signs range from subacute in sheep to fulminating fatal illness in goats although in apparent infections occur in both species especially in nomadic animals that are endemically exposed [13].

Acute Form: The acute form is seen mainly in goats and similar to rinderpest in cattle except severe respiratory distressis a common feature of PPR. Signs generally appear 3 to 6 days after being in contact with an infected animal [4]. Susceptibility to infection rises with age; however, the disease is highly fatal in the young animals[10].

The acute form of PPR is accompanied by a sudden rise in body temperature to 104-106 °F (40-41.3°C) [16]. And it also accompanied by dullness sneezing serous discharge from the eyes and nostrils. A day or two day later, discrete lesions develop in the mouth and extend over the entire oral mucosa, forming diaphtheric plaques. There is a profound halitosis and the animal is unable to eat because of a sore mouth and swollen lips. Nasal and ocular discharge becomesmucopurulent and the exudate dries up, matting the eyelids and partially occluding the external narres. Diarrhea develops 3-4 days after the onset of fever. It is a profuse and faeces may be mucoid and blood tinged. Dyspnea and coughing occur later and the respiratory signs are aggravated when there is secondary bacterial pneumonia. Erosion has been described in the vulva and prepuce. Abortion has been reported during outbreak in India. Death usually occurs within one week of the onset of the illness [4].

Subacute Form: The subacute form is more common in sheep but they also occur in goats. The signs and lesions are less marked and few animals may die within two weeks, but most recover [4]. The subacute form lasts for about two weeks [17].

Clinical Pathology: A leucopenia occurs but is not as marked as in rinderpest. As diarrhea develops there is a progressive hemo-concentration and low serum sodium and potassium.More recently, competitive or blocking enzyme-linked immune-sorbent assay (c-ELISA) has been developed based on monoclonal antibodies. Specific antibodies specific for neucleocapsid (N) or hemoagglutinin (H) protein of PPR and rinderpest viruses and which enable differential diagnosis of the two viruses. The efficacy of c-ELISA compares very well with virus isolation test for detection and titration of antibodies to PPRV in goats and sheep. Viral antigen also can be detected in buffy coat, body secretions, feces, lymph node and tonsils by immune histochemical dot-ELISA methods as well as by AGID and CIEP. Furthermore, the RT-PCR has been reported to be more rapid and far more sensitive than conventional titration technique on vero cells. Unlike rinderpest, PPR viral antigen is still high in tissues of animals dying from the disease [4,17].

Necropsy Finding: In addition to the signs mentioned abovecharacteristic necropsy lesions often occur. These lesions are usually seen in the digestive and respiratory system, but can be seen in other systems as well [10]. The carcass of an affected animal is usually emaciated; the hindquarter soiled with soft/watery feces and eyeball 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 into the esophagus. Lips: Swollen lips; erosions and possibly scab or nodules in late cases, Nasal cavity: Congested (reddened) lining, clear or creamy exudates, erosions. Lungs: Dark or purple areas firm to touch, mainly in the anterior and cardiac lobes (evidence of pneumonia). Lymph nodes (associated with the lungs and the intestine): Soft and swollen. Abomasum congested (reddened) lining, hemorrhages. Small intestine: Congested (reddened) lining hemorrhages, some erosions. Large intestine (caecum, colon and rectum): Small red hemorrhage along the folds of lining, joining together as time passes and becoming darker, even green-black in stale carcasses (zebra stripes) as reported by FAO [12]. The skin is usually covered with bad smelling [18]. At necropsy, the following specimens should be collected for virology and histopathology: Lungs, Small and large intestines, oral mucosa, tonsil and mesenteric lymph nodes [4, 13].

Diagnosis: A presumptive diagnosis is based on clinical, pathologic and epidemiologic findings may be confirmed by viral isolation and identification [16]. There are several important points to observe when using the service of the laboratory; these areproviding epidemiological and clinical details with the samples, always sampling several animals in an outbreak, keeping samples cool during transfer to the laboratory (preferable on melting ice) and reducing the time in transit to the minimum, mark sample bottles carefully with an indelible pen and record details of each sample's origin for submission to the laboratory [12].

Differential Diagnosis: Other diseases cause diarrhea or pneumonia in sheep and goats may pose diagnostic challenge but a history of recent introduction of new stock and the clinical and postmortem findings of stomatitis, typical for PPR. Laboratory tests arerequiringruling out rinderpest [4,7].

When carrying out an investigation, examination of the way the disease behaves in the herd or flock is important as the finding on a single goat or sheep. The most frequent sources of confusion are: Mouth lesions-could be a symptom of rinderpest, FMD, Blue tongue or Contagious ecthyma (Orfor"Sore mouth"), Difficult breathing-could be a symptom of pneumonic pasteurollosis or CCPP and Diarhea-could be a symptom of coccidiosis or gastrointestinal helminth infestations. Pneumonia is usually a very obvious presenting sign in PPR. Therefore, without doubt, pneumonic pasteurollosis and CCPP have caused the most difficult in differential diagnosis [19].

Pneumonic pasteurollosisis purely respiratory disease of sheep and goats caused by the bacterium *Pasteurellahaemolytica*. Dark red-purple areas, firm to the touch, are evident mainly in the anterior and cardiac lobes of the lung. There are no oral lesions or diarrhea. The numbers of affected and dead animals are usually lower than for PPR except under exceptional condition of stress and crowding such as when large numbers of sheep assembled for trade. The main problem of differentiation arises when oral lesions and diarrhea are absent or not very obvious in PPR, as is sometimes the case. Using appropriate culture media, *Pasteurellahaemolytica*can be easily isolated in pure and profuse culture from pneumonic lungs of sheep; even the lungs of PPR affected animals. Isolation of *Pasteurellahaemolytica* from the lungs of sheep, therefore, neither confirms a diagnosis of primary pneumonic pasteurollosis nor rules out the presence of PPR. Diagnostic test for detecting PPR virusshould be carried out in all suspected cases of pneumonicpasteurollosis whereis a risk PPR [12].

CCPP is a disease of goats (sheep are not affected) caused by *Mycoplasma* species. Like PPR, it is characterized by fever, difficult or abnormalbreathing and coughing, but their mouth lesions or diarrhea is not present in CCPP. At postmortem 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. 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[20].

Rinderpest, 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 investigation to examine all species. Confirmation requires the resources of a specialized laboratory. The samples required for laboratory confirmation of both rinderpest and PPR are identical. As the Global Rinderpest Eradication Programme (GREP) progress, it becomes increasingly important that PPR and rinderpest be differentiated because, any outbreak of rinderpest anywhere represents international emergency [19].

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 diarrhea 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[12].

Bluetongue like PPR is characterized byfever, discharges and oral lesions. 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. Bluetonguevirus infection is endemic throughout the region 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 virus in single samples does not confirm a provisional diagnosis of bluetongue [20].

Contagious ecthyma (Orf, "Sore mouth", contagious purulent 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 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, diarrhea or pneumonia [12, 17].

Specimens Required For Diagnosis: Whole blood collected with heparin (blood and anticoagulant should be mixed gently) should be submitted for serological diagnostic. Serological tests include Virus seroneutralization (recommended by OIE),

Competition ELISA, Counter immnoelectrophoresis, Agar gel immunodiffusion and Immunodiffusion inhibition test.Samples can also be submitted for identification of the agent. Such techniques include detection of the antigen by immunological method (counterimmunoelectrophoresis and ELISA), virus identification and virus RNA detection using PPR-specific DNA probes or amplification by PCR [11].

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 of 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 postmortem examination: Lymph nodes found around the lungs (mediastinal) and alimentary tract (mesenteric), portionsof the spleen and the lungs. Two sets of each tissues are required; one set of chilled but not frozen and the other is put in 10% formalin solution to preserve the samples. Where cold storage is a problem, as is often the case, formalin can be used to preserve the sample when they are sent to the laboratory [19]. Uncloatted blood- this is needed for virus isolation and should be collected in bottles containing anticoagulant (Heparin or Ethylenediaminetetracetic acid [EDTA]), Clotted blooded or serum-these are needed for antibody detection. National laboratory will provide guidance about exactly which samples are required, but it is advisable to collect as many of the sample listed above as possible when dealing with an outbreak [12].

Treatment: There is no treatment for goat plague but skilled professionals can use special medicines to help animals recover [18]. Affected goat with stomatitis, enteritis and pneumonia were treated with penicillin and streptomycin reinforced with broad-spectrum chloramphenicol [9]. However, mortality rates can be reduced by the use of drugs that control the bacterial and parasitic complications. Specifically oxytetracyclineand chlortetracycline are recommended to prevent secondary pulmonary infections [10].

Valuable sick animals in the early stages of the disease should be isolated and given hyperimmuneserum, which may be obtained from cattle hyperimmunized against rinderpest. Lesions around the eyes, nostrilsand mouth should be cleaned and good nursing provided the disease may precipitate other infections and the animals harbor without clinical signs, such as blood and internal parasites. Thus, it may be necessary to administer antiprozoal and anthelmintics as well [4, 17].

PREVENTION AND CONTROL

The disease can be prevented by not introducing new stock from unknown sources, especially animals bought at livestock markets. In addition animals returned unsold from markets should be segregated unless the entire herd or flock has been vaccinated [4].

Vaccination is the most effective way to gain control epidemic PPR. Taking advantage of the cross relationship between the viruses and its ready availability, Rinderpest tissue culture vaccine was initially used to protect small ruminants [9]. Vaccinate all sheep and goats that have been in contact with sick animals. Look at the vaccinated animals very closely every day. If one shows signs of disease put it with the sick ones that have been isolated. It is important to vaccinate animals in areas where the disease does not often happen if there is a risk of sick animals from elsewhere being brought through the area[18].

Recently, a homologous PPR vaccine has been developed and the vaccine seed is available through the PANVAC at Debre Zeit, Ethiopia, Africa, or CIRAD-EMVT Montpellier, France, for other areas. This vaccine of choice is becoming increasingly available. The vaccine can protect small ruminants against PPR for at least for 3 years [20]. The newly developed recombinant vaccine capripox viruses expressing the fusion (F) and hemagglutinin (H) protein genes of the rinderpest virus are also effective against PPR [4]. Butthe use rinderpest vaccine to protect small ruminants against PPR is now contraindicated because its uses produce antibodies to rinderpest which compromise sero-surveillance for rinderpestand thereby the Global Rinderpest Eradication Programme [12].

Methods applied for rinderpest eradication may be appropriate for PPR. These include the following: quarantine, slaughter, Proper disposal of carcasses and contact fomites, decontamination of facilities and equipment, restriction on importation of sheep and goats from infected areas [10].

CONCLUSION AND RECOMMENDATION

PPR is primarily a problem of sheep and goats in Africa (from Tropic of Cancer to Equator), the Middle East and the Indian subcontinent. Historically, the disease was primarily with West Africa but it extends in a belt across Africa immediately south of Sahara and Arabian Peninsula. It is widely distributed in goat rearing areas of the countries. The studies on PPR infection resulted in better-off households slipping into poverty, while the poor and very poor became destitute. In a particular flock, the risk of outbreak is greatly increased when a new stock is introduced or when animals are returned unsold from livestock market. Other disease that cause diarrhea, or pneumonia may pose a diagnostic challenge but a history of recent introduction of new stock and the clinical and postmortem findings of stomatitis, enteritis and pneumonia are typical for PPR. There is no specific treatment for PPR. Hence, an attenuated cell culture adapted rinderpest virus vaccine provides protection against PPR for at least four years. But this vaccine will affect any subsequent surveys for evidence of rinderpest, thereby the Global Rinderpest Eradication Programme.

Based on the above conclusion the following recommendations are forwarded:

- Restriction on importation of the animal from infected areas.
- Quarantine any animals which are unknown health status before mixing them with the rest of the herd.
- Any animals suspected of having PPR should be reported to veterinarian in charge immediately.
- Use of focused ("ring") vaccination and prophylactic immunization in high risk population.
- Attenuated PPR vaccine should be used not to compromise surveillance for rinderpest.
- The veterinary services in the country must review their preparedness plans, strengthen border control and improve surveillance.
- Generally, early reporting is the key to early reaction for containment, control and rapid elimination.

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