

## Review Article

# Review of Peste Des Petits Ruminants in Sheep

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## Abstract

*Peste des petit ruminants* [PPR] is an acute or sub-acute febrile, highly contagious and often fatal disease of sheep, goats and wild small ruminants. The disease is characterized by fever, erosive stomatitis, conjunctivitis, gastroenteritis, pneumonia and causes serious economic losses in small ruminant's production. *Peste des Petits Ruminants* is endemic in Sub-saharan Africa extending to the Arabian Peninsula, the Middle Eastern countries and India. *Peste des petit ruminants* virus is transmitted by close contact between infected and non-infected susceptible animals, which is likely to occur in common grazing and watering points. Infected animals shed PPRV in exhaled air, in secretions, and excretions. In the field, a presumptive diagnosis of PPR can be made on the basis of clinical, pathological, and epizootiological findings. However laboratory confirmation of PPR may be performed through virus isolation, detection of viral antigens, nucleic acid isolation and sequencing; and detection of specific antibody in the serum. The Food and Agricultural organization and the Office International des Epizooties have developed a global eradication strategy aimed at control and eradication of the disease by the year 2030.

## ABBREVIATIONS

PPRV: Peste Des Petits Ruminantsvirus; USD: United States Dollars, C-ELISA:Competitive Enzyme-Linked Immuno-sorbent Assay; F: Fusion Protein, RT-PCR: Reverse Transcription Polymerase Chain Reaction; PCV: Packed Cell Volume; EDTA: Ethylene Diamine Tetracetic Acid; °C: Degrees Centigrade

## INTRODUCTION

*Peste des petits ruminants* [PPR] severely affects small ruminants in almost 70 countries in Africa, the Middle East and parts of Asia. It is a highly contagious disease that causes USD 1.5 to 2 billion in losses each year in regions that are home to over 80% of the world's sheep and goats and to more than 330 million of the world's poorest people, many of whom depend on them for their livelihoods. The disease threatens food security and the livelihoods of smallholders and prevents animal husbandry sectors from achieving their economic potential.

## Historical background

*Peste des petit ruminants* [PPR] is an acute or sub-acute febrile, highly contagious and often fatal disease of sheep, goats and wild small ruminants [1]. The disease is characterized by fever, erosive stomatitis, conjunctivitis, gastroenteritis, pneumonia and causes serious economic losses in small ruminant's production [2,3]. The disease was first described in Cote d' Ivoire, West Africa [4]. *Peste des petit ruminants* is also known as goat plague, pest of sheep and goats, Kata, stomatitis-pneumoenteritis syndrome, contagious pustular stomatitis, and pneumoenteritis complex

[5]. The reference to the disease as a "plague" is indicative of the highly contagious nature and economic impacts that result from this disease. It was only in the late 1970s that PPR was determined to be a distinct virus from rinderpest virus through serology, biochemical and cross-protection experiments [6-8]. The disease was initially thought to be confined to the countries of West Africa; however PPR has now been confirmed present in several African, Middle East, Central and South Asia countries, as well as China [9,10].

## Etiology

*Peste des Petits Puminants* [PPR], 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 *Peste des petits ruminants* virus has an envelope derived from the host-cell plasma membrane, containing two transmembrane glycoproteins surrounding a nucleocapsid. The presence of the envelope renders virions sensitive to heat, lipid solvents or detergents, non-ionic detergents, formaldehyde and oxidizing agents. *Peste des petits ruminants* virus is also very sensitive to ultraviolet radiation and desiccation. Like all enveloped viruses PPRV is very sensitive to heat. The half-life of the virus at 37°C was estimated at 2 hours, and at 50°C infectivity was destroyed in 30 minutes [11]. Other studies have confirmed and clarified the thermal sensitivity of PPRV [12,13]. *Peste des petits ruminants* virus has been shown to survive in lymph nodes for 8 days at 4°C [11].

The PPR virus is also sensitive to low pH, being destroyed after death of the animal by the low pH which accompanies rigor mortis. Peste des petits ruminants virus is stable at pH between 5.8 and 9.5 but rapidly loses activity at pH below 4 or above 11 at room temperature [14]. The optimum pH for survival of PPRV is between 7 and 8 [15].

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 [16].

## 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. Historically, the disease was primarily associated with West Africa, but it extends in a belt across Africa immediately South of the Sahara, extending into Arabian Peninsula [17-19].

**Transmission:** Adverse climatic factors, whether seasonal or not, affect the availability of pasture and water, which leads to increased movements of small stock in search of better nutrition and shelter which then aids spread of the PPRV to susceptible groups [20]. It has been reported that in Maghreb countries of North Africa, traditional sacrifices of sheep during major Islamic festivals provide a major opportunity of seasonal clustering of small ruminants of multiple sources whose health status is often unknown, thus creating a favorable environment for the transmission and dissemination of the PPR virus [15]. The seasonal epidemiologic patterns of the PPR disease differ in different ecological systems, geographical areas and are dependent on culture and livelihood patterns of small stock owners [21]. In Pakistan, seasonal outbreaks of PPR were alluded to [22,23], suggesting that seasonal grazing patterns among nomadic livestock keepers during winter encourage diseases transmission [24]. Similar observations were made by [25] who associated PPR outbreaks in Bangladesh to winter grazing. PPR outbreaks among sheep and goats in India are described to occur any time of the year, but are most frequent during the wet [April to September / October] or cold dry [January and February] seasons [20,26].

Peste des petits ruminants virus is transmitted by close contact between infected and non-infected susceptible animals, which is likely to occur in common grazing areas. Infected animals shed PPRV in exhaled air, in secretions, and excretions approximately 10 days after the onset of fever. Sneezed or coughed out droplets by infected animals contain large amounts of virus which can spread infection. Transmission between animals in the vicinity can occur through inhalation over a distance of about 10 meters. Infected formites can act as source of infection although it is unlikely considering the rapid inactivation of the PPRV in external dry conditions. Peste des petits ruminants can be transmitted to the offspring by feeding them milk from infected dam. The virus is thought to be present in milk from 1-2 days before the signs appear and last until 45 days after complete recovery [27].

**Risk factors:** The spread of the PPR outbreaks has for a long time been associated with social, cultural and economic activities such as conflicts, disasters, livestock trade, cultural festivals, and

change of husbandry practices, nomadism and seasonal climatic and environmental changes [28,29].

In a study carried out in Turkana Kenya, the crowding of sheep and goats at watering points during the dry season was found to be a significant risk factor for PPR outbreaks in 2009 while in 2010, sick adult goats and sheep sharing of grazing and water with lambs and kids was found to be significant source of PPR outbreaks [30].

**Host range:** *Peste des petit ruminants* is a disease of sheep and goats. In general goats are more susceptible than sheep; with sheep undergoing a milder form of the disease [27]. Other domestic animals such as camels, cattle and pigs are known to undergo subclinical infection of PPR [31]. The disease has been reported in wild small ruminants in a zoo [1] and those living in the wild [32-34].

**Host determinants of PPR:** Host determinant factors of PPR spread have been reported in various studies, highlighting age, sex, breed and animal species [9]. Young animals are less likely to have developed protective antibody titers and therefore are more susceptible to PPRV [35]. This high susceptibility in the young has been reported in Ethiopia, Kenya, Pakistan, India and Turkey [20,36-38]. In Oman, the disease is reported to maintain itself in susceptible yearling population, with an increase in incidence being a reflection of increased number of susceptible young goats/sheep recruited [39].

Sex has also been reported as a risk factor for susceptibility/resistance to the disease [25,36,40,41]. Since the off-take of males, in a farm, is higher and at an early age compared to females, which end up staying in the herds for longer periods [20], females are more likely to demonstrate higher antibody titers than the males. The recruited young males, having been in the herds for a shorter period, are less likely to have been in contact with virus. Indeed, studies in Bangladesh have shown that male goats are significantly more prone to PPR than females [25]. However, studies from Pakistan have shown no significant difference between males and females, with respect to susceptibility [24].

The influences of breeds of the small ruminants on susceptibility to the disease have also been studied [24], with results showing that there are insignificant differences between goat breeds but there are significant differences between sheep breeds. Breed differences to susceptibility to PPR have been reported in other studies [27,42,43]. Goat and sheep species' differences have been highlighted as major risk factor for PPRV susceptibility [24,36,38]. Though PPR has been described in other species of animals, the camel is emerging as a key risk factor in long distance transmission of the disease [29].

**Economic significance:** *Peste de Petit Ruminants* virus has a widespread distribution spanning Africa and Asia [44,45]. These areas encompass much of the developing world that relies heavily on subsistence farming to supply food or goods for trade, and small ruminants provide an excellent supply of both. Unfortunately, in many areas of Asia and Africa, small ruminant production and therefore the livelihoods of poor farmers is threatened by PPR among other trans-boundary animal diseases [TADs]. With its associated high morbidity and mortality, PPRV constitutes one of the major obstacles to subsistence farming

[46]. Small stock and mainly sheep and goats are the main farm animals owned by the poor in most developing countries. Goats, and sheep “considered as mobile banks”, are reared as sources of not only milk and meat for family consumption, but also of income that can easily be mobilized for paying household expenditures, particularly in lean times. In addition to this economic role, sheep and goats have significant socio cultural roles. They are used as gifts or emblems for traditional rituals and religious purposes [28].

**Pathogenesis:** The route of infection is respiratory and is spread by airborne droplets. All secretions and excretions of infected sheep are contagious throughout the course of the disease, but no carrier state exists [47]. During acute phase of the disease, virus is shed in all secretions and excretions.

*Peste des petits ruminants* virus exhibits lympho-epitheliotropism [48]. Lymphotropic nature, common to all Morbilliviruses, causes a severe leukopenia in infected animals, which promotes the development of secondary infections by bacterial agents or parasitic opportunists who take advantage of the induced immunosuppression and severe clinical pictures [9]. Infection occurs primarily through naso-pharyngeal route via inhalation of virus particles [15]. Then, the virus multiplies in lymphoid organs before regional spread through blood to the epithelial cells of respiratory and gastro-intestinal tract [21].

The *Peste des petits ruminants* virus causes cytopathic effect that is distinguished from that of other *Morbilliviruses*, by its characteristic appearance of multinucleated cells capable of forming round mini syncytia and intracytoplasmic inclusion bodies and eosinophilic intra-nuclear inclusion bodies [49]. The fusion between an infected cell and neighboring cells [syncytia] aided by viral fusion protein [F] which is expressed on the surface of infected cells is one way of spreading the virus. This process of spreading the infection from cell to cell through fusion allows the virus to continue the infectious process free of neutralizing antibodies as nucleocapsids migrate from cell to cell without passing through the external environment.

## SYMPTOMS AND CLINICAL SIGNS

### Acute form

The acute form of PPR is rare in sheep. Signs generally appear 3 to 6 days after being in contact with an infected animal [50]. The disease is highly fatal in the young animals accompanied by a sudden rise in body temperature to 40-41.3°C [50]. It is 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 becomes mucopurulent 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 and Tanzania [51]. Death usually occurs within one week of the onset of the illness.

### Sub-acute form

The sub-acute form is more common in sheep but they also occur in goats. This form has a longer incubation period of about 6 days. Sub-acutely affected animals are not severely affected and lack characteristic clinical signs and therefore mortality is usually very low. Lesions such as oral crusts due to mucosal discharges may appear making the disease to be confused with contagious ecthyma. Infected animals develop low grade pyrexia [39-40°C] and recover in 10-14 days and remain immune protected [52].

### Clinical Pathology

Hemorrhages in the digestive system and the liver reduce number of erythrocytes and hematocrit values significantly in animals naturally infected with PPRV. The virus has affinity for lymphoid organs contributing to marked immune-suppression as indicated by leucopenia, monocytes depletion and lymphopenia (Table 1).

### Competitive Enzyme-Linked Immunosorbent Assay [C-ELISA]

The C-ELISA is considered suitable for large scale testing due to its simplicity and availability of the recombinant antigen [55]. C-ELISA sensitivity is 99.4 % and specificity 94.5%. A competitive ELISA based on PPRV monoclonal antibodies specific for haemagglutinin [H] protein [56] or nucleoprotein [N] [55] was developed for detection of antibodies to PPRV in serum samples of sheep and goats.

### Experimental infection

In contrast to natural PPR infections, experimental infection of susceptible animals with PPRV results in a clinical disease with high morbidity rate and low mortality rate [57,58]. Sheep experimentally infected with PPRV exhibited characteristic lesions at the lip commissure (Figure 1), severe diarrhea (Figure 2), gooseberry-like mesenteric lymph nodes (Figure 3) and intestinal congestion (Figure 4). A severe experimental form of disease has been reproduced in sheep and goats [59].

### Necropsy finding

The carcass of a PPR affected animal is usually emaciated, the hindquarters soiled with soft/watery faeces and the eyeballs sunken. The eyes and nose contain dried-up discharges. Lips may be swollen and possibly scabs or nodules in late cases. The nasal cavity is congested [reddened] lining with clear or creamy yellow exudates and erosions. The pathology caused by PPR is dominated by necrotizing and ulcerative lesions in the mouth and the gastro-intestinal tract. Erosion in the oral cavity is a constant feature affecting the gums, soft and hard palates, tongue and cheeks and into the oesophagus. The abomasum is congested with multiple haemorrhages. The rumen reticulum and omasum rarely exhibit lesions. Occasionally, there may be erosions on the pillars of the rumen. The omasum is a common site of regularly outlined erosions often with oozing blood. Lesions in the small intestine are generally moderate, being limited to small streaks of hemorrhages and, occasionally, erosions in the first portions of the duodenum and the terminal ileum. The large intestine is usually more severely affected, with congestion around the ileo-

**Table 1:** Mean hematological parameters and their standard error for experimentally infected sheep before and after infection. Source: [Maina et al., 2015] [58].

Parameters	Before infection (n=5)	After infection (n=5)	Mean of the Difference	P-value	t
	X±Se	X±Se	d±Se		
<b>WBC (X 10<sup>3</sup>/ML)</b>	19.57±7.70	61.68±25.42	42.11±28.04	0.0305	3.3581*
<b>HGB (mg/dl)</b>	9.46±2.15	10.34±1.57	0.88±1.12	0.1535	1.7569
<b>RBC (X10<sup>6</sup>/ML)</b>	8.69±2.29	12.56±1.54	3.88±1.97	0.0117	4.404*
<b>PCV (%)</b>	21.72±7.23	26.42±3.64	4.68±4.83	0.0949	2.17
<b>MCV (fl)</b>	16.42±1.57	21.6±2.49	5.18±2.77	0.0139	4.18*
<b>MCH (fl)</b>	10.94±0.58	8.14±0.55	2.8±1.08	0.0044	5.7972*
<b>MCHC (g/dl)</b>	45.04±6.56	38.64±2.04	6.4±5.97	0.0746	2.40
<b>Platelets (10<sup>3</sup>/ml)</b>	838±542.18	209.6±32.62	628.4±515.94	0.0528	2.7235
<b>Neutrophils (%)</b>	46.2±15.51	58.4±15.53	12.2±3.90	0.0022	6.9949*
<b>Lymphocytes (%)</b>	53.2±16.39	36.4±11.55	16.8±5.81	0.0029	6.47*
<b>N/L</b>	1.098±0.951	1.942±1.378	0.8436±0.461	0.0012	4.09*

Subscript \* in the same row indicate there was a significant difference (P<0.05)

Key:

X - Mean

Se - Standard error of mean

d - Mean of the difference

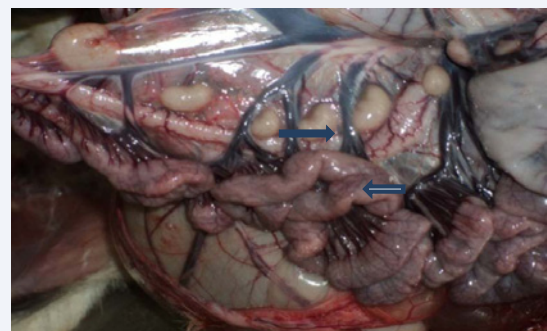
**Abbreviations:** WBC: White Blood Cells; HGB: Hemoglobin Concentration; RBC: Red Blood Cells

PCV: Packed Cell Volume; MCV: Mean Corpuscular Volume; MCH: Mean Corpuscular Hemoglobin; MCHC: Mean Corpuscular Hemoglobin Concentration

These observations are predominant particularly during acute phase of disease [53]. The number of eosinophils may remain unaltered because these cells are primarily associated with parasitic infections [54]. On the other hand; the vaccine virus induces only a transient lymphopenia without significantly affecting the immune response to nonspecific antigen or to itself. As diarrhea develops there is a progressive hemo-concentration and low serum sodium and potassium.



**Figure 1** An experimentally infected sheep having ulcerated oral lesions at the commissure of the mouth on day 12 post infection. [Courtesy: Maina et al., 2015] [58].



**Figure 3** Swollen and enlarged mesenteric lymph nodes (arrows) from an experimentally infected sheep having a goose berry-like appearance. (The sheep died on day 17 post infection) [Courtesy: Maina et al., 2015] [58].



**Figure 2** An experimentally infected sheep having severe watery diarrhea with soiling of the hind limbs on day 13 post infection [Courtesy: Maina et al., 2015] [58].



**Figure 4** Intestinal mucosa from an experimentally infected sheep appearing severely congested. Courtesy: [Maina et al., 2015] [58].

cecal valve, at the ceco-colic junction and in the rectum. In the posterior part of the colon and the rectum, discontinuous streaks of congestion “zebra stripes” form on the crests of the mucosal folds. In the respiratory system, small erosion and petechiae may be visible on the nasal mucosa, turbinates, larynx and trachea. Bronchopneumonia may be present, usually confined to the antero-ventral areas, and is characterized by consolidation and atelectasis. The lung is dark red or purple with areas firm to the touch, mainly in the anterior and cardiac lobes show evidence of pneumonia. Lymph nodes associated with the lungs and the intestines are soft and swollen [60].

### Histopathology of the disease

*Peste des petits ruminants* virus causes epithelial necrosis of the mucosa of the alimentary and respiratory tracts marked by the presence of eosinophilic intracytoplasmic and intranuclear inclusion bodies [61]. Multinucleated giant cells [syncytia] can be observed in all affected epithelia as well as in the lymph nodes where there is severe depletion of lymphocytes (Figure 5), [49]. In the lungs multifocal degeneration, ulceration and necrosis, followed by alveolar type II pneumocytes hyperplasia which mostly ends up with syncytial cell formation is a prominent feature [9]. Infiltration of the lymphocytes, plasma cells and histiocytes into the alveolar septae leads to its hypertrophy and desquamation with alveolar casts [9]. Intestinal lesions are characterized by blunted villi, degeneration of surface and crypt epithelial cells; expansion of lamina propria by a primarily mononuclear infiltration with scattered syncytial cells [49].

### Diagnosis of the disease

In the field, a presumptive diagnosis of PPR can be made on the basis of clinical, pathological, and epizootiological findings. However laboratory confirmation is an absolute requirement. Diagnosis of PPR may be performed through virus isolation, detection of viral antigens, nucleic acid isolation and sequencing; and detection of specific antibody in the serum [21].

### Virus isolation

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 and the isolated viruses are stored for later studies [50]. Samples for virus isolation include heparinized blood, eye and nasal swabs [from

live animals], tonsil, mesenteric lymph nodes, spleen, section of colon and lung from necropsied cases. For successful isolation, samples must be collected during the hyperthermic phase [62] and submitted to the testing laboratory in cold ice. The most widely used cell culture systems are primary lamb kidney and ovine skin [63,64] and Vero cells [65].

## MOLECULAR TECHNIQUES

### Nucleic acid recognition methods

Reverse transcription polymerase chain reaction [RT-PCR] techniques based on the amplification of parts of the N and F protein genes has been developed for the specific diagnosis of PPR [66,67]. This technique is 1000 times more sensitive than classical virus titration on Vero cells [66] with the advantage that results are obtained in 5 hours, including the RNA extraction, instead of 10–12 days for virus isolation.

### Specimens required for diagnosis

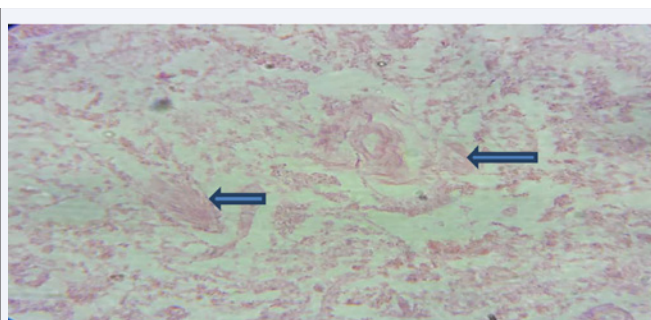
Tears- cotton buds or swabs of absorbent cotton wool are inserted into the conjunctival sac and swirled around to collect tears. The bud of swab is broken off and placed in Phosphate buffered saline. Gum debris- this material is scraped with a spatula from the gums and placed in Phosphate buffered saline. The tissues to sample include: Lymph nodes found around the lungs [mediastinal] and alimentary tract [mesenteric], portions of 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. It was found that even after three years of storage in formalin, this sample can still be used to recover RNA for PPR confirmation [68]. Unclothed blood is needed for virus isolation and should be collected in bottles containing anticoagulant [Heparin or Ethylenediaminetetraacetic acid [EDTA], serum is needed for antibody detection.

## PREVENTION AND CONTROL

There is no specific treatment against PPR. Control of the disease in previously non-infected countries can be effected through strict quarantine, movement controls, restriction of importation of sheep and goats from affected areas, rapid identification, humane slaughter, disposal of affected animals and burning or burying carcasses and effective cleaning and disinfection of contaminated areas and clothing with lipid solvent solutions of high or low pH. Effective disinfectant agents include alcohol, ether, phenol, sodium hydroxide and common detergents. In areas where PPR is endemic, the commonly employed control mechanism is vaccination [69].

Vaccination is the most effective way to gain control epidemic PPR. In a situation where goats are reared together with sheep, the mixed herd model established that sheep were the main drivers of PPR transmission. *Peste des petits ruminants* disease in sheep herds was seen to persist longer than was the case in the goats and thus may serve as the reservoir for virus in between outbreaks. A simulation of the model showed that vaccination coverage of 50% of combined sheep and goats herds was enough to curtail the spread of the PPR disease within 254 days in Turkana, Kenya [70].

Although PPRV is classified into four lineages based on F



**Figure 5** Lymph node: Note severe lymphocytic depletion within the cortex with arrows indicating network of trabeculae that are remaining (H&E x400) Courtesy:[Maina et al., 2015] [58].

and N genes, they all belong to a single serotype. Therefore vaccination using vaccine prepared from any of the lineage will provide protection against all the lineages. A live attenuated culture vaccine based on Nigeria75/1 strain is widely used for vaccination and immunization in almost all the PPRV endemic areas of the world. This vaccine is safe for pregnant dams and induces immunity in at least 98% of the vaccinated animals in the field [71]. This vaccine protects immunized small ruminants for a period of up to 3 years. The major drawback in using this vaccine is thermo stability especially since PPRV is a disease of tropical countries. Recently, a freeze dried form of this vaccine has been prepared in an excipient containing trehalose to make it thermo stable. This fortified vaccine is resistant to temperature as high as 45 degrees Celsius for 14 days with negligible loss in efficacy. The use of this vaccine to protect small ruminants will lead to effective control of PPR in developing countries [69].

### FAO/OIE ERADICATION PROGRAMME

It is expected that the control and eradication of PPR will improve incomes from small ruminant husbandry systems and lead to their improved profitability and productivity. In 2013, the OIE and FAO jointly decided to embark upon the control of PPR on a global scale and develop a 'PPR Global Control and Eradication Strategy', [named 'Global Strategy'] with a strong willingness to address the animal health problems in a systematic way through approaching horizontal as well as more disease-specific [vertical] issues [72]. The strategy recognizes that situations and contexts can be very different between and even within countries. Consequently, the proposal is to begin by controlling the disease in areas where it is highly endemic and then to consolidate these control efforts by concentrating on areas where a low endemic level has been reached and where eradication is a feasible objective or is already underway. For countries already free of PPR, the Global Strategy is designed to maintain this status. The duration of each stage is variable and will depend on the context [72].

### DISCUSSION AND CONCLUSION

PPR is primarily a problem of sheep and goats in Africa the Middle East and the Indian subcontinent. Historically, the disease was primarily reported in West Africa but has now extended in a belt across Africa and the Arabian Peninsula. It is widely distributed in sheep and goat rearing areas of all the countries. The impact of peste des petits ruminants on livestock rearing communities is very huge and pushes many of them into poverty. 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. If all countries can embark on comprehensive vaccination targeting over 50 % of their small ruminant population, then the disease can be wiped out following the recommendations of OIE/FAO by the year 2030.

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