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REVIEW ARTICLE

# Review on Pest Des Petits Ruminants Virus and its Socioeconomic Impact in Small Ruminants

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## Abstract

Peste Des Petits Ruminants (PPR) are a highly contagious viral disease that mainly affects sheep and goats. It belongs to the genus Morbillivirus in the family Paramyxoviridae. Today, this disease is considered a cause of mortality and morbidity in many countries of the world. The disease occurs south of the Sahara Desert and north of the equator in Africa, most of the Middle East, and parts of Asia, including the Indian subcontinent. It is transmitted by aerosols during close contact between animals, mainly through sneezing and coughing. The disease is characterized by high fever, discharge from the eyes and nose, pneumonia, necrosis and gastrointestinal tract leading to foul smelling diarrhoea. Diagnosis can be made based on clinical, pathological and epizootological findings. This has significant economic implications for food security and livelihoods. Therefore, PPR is considered one of the most damaging animal diseases in Africa, the Middle East and Asia, and is also one of the priority diseases listed in the FAO-OIE Global Framework for the Progressive Control of Trans boundary Animal Diseases. Peste des Petits ruminants are common in Ethiopia due to economic losses due to reduced production, death, abortion and cost of disease control. There is no specific treatment for PPR. However, drugs that control bacterial and parasitic complications may reduce mortality. For surveillance purposes, circular vaccination and/or vaccination of high-risk populations may also be useful. Therefore, eradication of PPR is done through a combination of quarantine, movement control, cleaning and disinfection of infected areas and vaccination.

## Abbreviations

AGID: Agar Gel Immune Diffusion; cDNA: Complementary Deoxyribonucleic Acid; ELISA: Enzyme Linked Immunosorbent Assay; FAO: Food and Agricultural Organization; HA: Haemagglutination; NVI: National Veterinary Institute; PANVAC Pan African Veterinary Vaccine Centre; PPR: Peste Des Petits Ruminants; PPRV: Peste Des Petits Ruminants Virus; RNA: Ribonucleic acid; RPV: Rinderpest Virus; RT-PCR: Reverse Transcription Polymerase Chain Reaction

## Introduction

Peste Des Petits Ruminant (PPR) is an acute viral disease of small ruminants characterized by fever, discharge from the eyes and nose, stomatitis, diarrhea and pneumonia with foul-smelling breath [1]. Because of the respiratory symptoms, PPR can be confused with Contagious Caprine Pleuropneumonia (CCPP) or pasteurellosis. In many cases,

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pasteurellosis is a secondary infection resulting from immunosuppression caused by the virus. PPR virus is mainly spread by aerosols between animals living in close contact [2]. Infected animals show clinical signs similar to those seen in the history of rinderpest, although the two diseases are caused by different virus species.

The virus that causes PPR is classified in the Morbillivirus genus of the Paramyxoviridae family due to its similarity to Rinder pest and measles viruses [3]. Viral members of this group have six structural proteins: the nucleocapsid protein (N) that surrounds the viral genomic RNA, the phosphoprotein (P) associated with the polymerase protein (L) large protein, the matrix (M) protein, the fusion protein (F), and the hemagglutinin (H) protein. The matrix protein, which is tightly attached to the inner surface of the virus envelope, forms a link between the nucleocapsid and the external glycoproteins of the virus: H and F, which are responsible for virus attachment and cell penetration, respectively. The PPRV genome also encodes two nonstructural proteins, C and V.

On the basis of its similarities to rinderpest, canine distemper and measles viruses, the virus causing PPR has been classified within the genus Morbillivirus in the family Paramyxoviridae [4].

PPR was first described in Ivory Coast (Bailey and Banyard, 2005 [3]. Since the late 1990s, it has expanded its range over large areas of Africa from North Africa to Tanzania and the Middle East, and has also spread to countries from Central Asia to South and East Asia [5].

The natural disease affects mainly goats and sheep. It is generally considered that cattle are only naturally infected sub clinically, although in the 1950s, disease and death were recorded in calves experimentally infected with PPRV- infected tissue and PPRV was isolated from an outbreak of rinderpest-like disease in buffaloes in India in 1995. Antibodies to PPRV as well as PPRV antigen and nucleic acid were detected in some samples from an epizootic disease that affected dromedaries in Ethiopia and Sudan. PPR affects a number of wild species within the order Artiodactyla some of which species are highly endangered, e.g., the Mongolian saiga antelope. The American white-tailed deer (*Odocoileus virginianus*) can be infected experimentally with PPRV. Dual infections can occur with other viruses such as pestivirus or goatpox virus.

The incubation period is typically 4–6 days, but may range between 3 and 10 days. The clinical disease is acute, with pyrexia up to 41°C that can last for 3–5 days; the animals become depressed, anorexic and develop a dry muzzle. Serous oculonasal discharges become progressively mucopurulent and, if death does not ensue, persist for around 14 days. Within 4 days of the onset of fever, the gums become hyperaemic, and erosive lesions develop in the oral cavity with excessive salivation. These lesions may become necrotic. Watery blood-stained diarrhoea is common in the later stage. Pneumonia, coughing, pleural rales and abdominal breathing also occur. The morbidity rate can be up to 100% with very high case fatality in severe cases. However, morbidity and mortality may be much lower in milder outbreaks, and the disease may be overlooked. A tentative diagnosis of PPR can be made on clinical signs, but this diagnosis is considered provisional until laboratory confirmation is made for differential diagnosis with other diseases with similar signs.

At necropsy, the lesions are very similar to those observed in cattle affected with rinderpest, except that prominent crusty scabs along the outer lips and severe interstitial pneumonia frequently occur with PPR. Erosive lesions may extend from the mouth to the reticulo-rumen junction. Characteristic linear red areas of congestion or haemorrhage may occur along the longitudinal mucosal folds of the large intestine and rectum (zebra stripes), but they are not a consistent finding. Erosive or haemorrhagic enteritis is usually present and the ileo-caecal junction is commonly involved. Payer's patches may be necrotic. Lymph nodes are enlarged, and the spleen and liver may show necrotic lesions.

There are no known health risks to humans working with PPRV as no report of human infection with the virus exists. Laboratory manipulations should be carried out at an appropriate containment level determined by bio risk analysis.

- There for the Objectives of this seminar are: To review peste des petits ruminants and
- To overview the socio-economic importance of PPR

## Literature Review

### History

PPR was first described in Ivory Coast, West Africa in 1942 and subsequently spread to other

regions where it used to be named as Kata, psuedo-rinderpest, pneumoenteritis complex and stomatitis-pneumoenteritis syndrome. In the late 1970s sub-Saharan Africa, then the Middle East and Asia faced severe epidemics respectively [6]. The infection has long been considered as caused by a variant of rinderpest virus, adapted to small ruminants but recognition of PPR virus as a novel member of the Morbillivirus genus occurred only in the late 70s by using more sensitive laboratory techniques [7]. Currently, the presence of the virus has been confirmed in large areas of Asia, the Middle East and Africa; moreover, it is spreading to new countries, affecting and threatening an increasing number of small ruminant and livestock keepers [6].

The Strains of PPR virus that cause only sub-clinical diseases have been identified in several areas of the country but it was clinically suspected in Ethiopia in 1977 in a goat herd in the Afar region, in the east of the country [8]. PPR introduced to Ethiopia in 1989 in the southern Omo. River valley from where it moved east to Borana then northwards along the Rift Valley to Awash. The disease then spread northwards into the central Afar Region and eastwards into the Ogaden [8,9]. Clinical and serological evidence of its presence has been reported in 1984 and later confirmed in 1991 with cDNA probe in lymph nodes and spleen specimens collected from an outbreak in a holding near Addis Ababa. Nowadays, Because of its major economic importance, dramatic clinical incidences with high mortality rate and restrictions on animal and product movements, PPR is considered as a disease of major economic impact and has to be notified to the World Animal Health Organization (OIE).

It has received a growing attention because of its wide spread, economic impacts and the role it plays in complication of the ongoing global eradication of rinderpest and epidemiosurveillance programmes [10].

## Aetiology

Peste Des Petits Ruminants is caused by a virus that was assumed for a long time to be a variant of rinderpest adapted to small ruminants. But the studies based on virus cross neutralization and electron microscopy showed that it was a morbillivirus that had the physicochemical characteristic of a distinct virus though biologically and antigenically related to RPV. It was also shown to be an immunologically distinct virus with a separate epizootiology in areas

where both viruses were enzootic. The development of advanced diagnostic tools like specific nucleic acid probes for hybridization studies and nucleic acid sequencing have proved that PPR virus is quite distinct from rinderpest virus. The Peste Des Petits Ruminants virus is in the genus of Morbillivirus and under Paramyxoviridae family. The Morbillivirus genus also includes other six viruses: measles virus, rinderpest virus, canine distemper virus, phocine morbillivirus, porpoise distemper virus and dolphin morbillivirus [11] affecting different hosts of animals and human being.

Morbilliviruses are structurally linear, non-segmented, single stranded, negative sense RNA viruses with genomes 15–16 kb in length and 200 nm in diameter. The major site of this virus propagation is lymphoid tissue and acute diseases are usually accompanied by profound lymphopenia and immunosuppression, leading to the host susceptible for secondary and opportunistic infections [12]. The PPRV genome is 15,948 nucleotides nearly 16 kb in length, although a variant virus with an additional 6 nucleotides has been detected in the recent Chinese epizootic. The genome contains six transcription units encoding in sequential order, the nucleocapsid (N) protein, the phospho (P) protein, the matrix (M) protein, the fusion (F) protein, the hemagglutinin (H) protein and the large (L) protein which together with the P protein forms the viral RNA-dependent RNA polymerase [13].

There is only one serotype of PPRV, but phylogenetic analysis based on partial N or F gene sequences groups PPRV strains into lineages I, II, III and IV. Historically, PPRV strains found in Africa belonged to lineage I and lineage II and were mainly prevalent in West Africa. Lineage III is mostly found in Arabia and recently it circulating in East Africa, but has also been isolated in southern India in study. Lineage IV is usually found in Asia and so termed the Asian lineage. A recent review of currently available molecular epidemiological data was carried out in Africa and showed that since 2008 lineage IV has also been continually identified in Africa [14].

## Epidemiology

**Host range:** Clinically PPR is seen in both sheep and goats however, goats are more susceptible than sheep [15]. Camels are considered susceptible to PPR but this is still to be clarified by experimental infections. It has been shown that camels can seroconvert to the PPRV [16].

Recent observations in Sudan suggest that camels could be affected by PPR, as they can show clinical expression of the disease and positive results were detected by serological tests, including reverse transcription polymerase chain reaction (RT-PCR), and PPRV was isolated in cell culture. In one study, antibodies against PPR were detected in Ethiopia in 3% of the 628 tested camels [8,17,18].

Peste des petits ruminants can affect some wild ungulates, but there is very limited information on species susceptibility and the occurrence of disease. Peste des petits was confirmed as the cause of two severe outbreaks, one in captive Dorcas gazelles (*Gazelladorcas*) and Thomson's gazelles (*Gazellathomsoni*) in Saudi Arabia in 2002 and the other in buffalo in India in 1995. It is also thought to have caused another outbreak that affected both gazelles and deer in Saudi Arabia in the 1980s. White-tailed deer (*Odocoileusvirginianus*) can be infected experimentally. In addition, peste des petits ruminants have been reported in captive Nubian ibex, Laristan sheep and gemsbok. Whether wild ruminants are important in the epidemiology of this disease is unknown [19].

**Risk factors:** Kids over 4 months and under 1 year of age are most susceptible to the disease. Sahelian breeds of sheep and goats are believed to be more resistant than the dwarf breeds in the humid and sub-humid zones of West Africa. In a particular flock, the 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 [20].

Confinement and restricted movement of the animals, due to rainy seasons in tropical countries, may affect the nutritional status of the animals and hence predispose them to PPRV infection. Sero positive PPR cases were reported during the months of December, January and February followed by the months of September and October. December to February appeared to be the period of high risk for small ruminants to PPR infection. The least seroprevalence was observed from March to August.

The migration of animals during the coolest months may be one of the reasons for the higher frequency of PPR outbreaks during the months of December, January and February. However, limited fodder also makes animals nutritionally deficient, resulting in an increased susceptibility to further infections. Climatic factors favorable for the survival

and spread of the virus may also contribute to the seasonal distribution of PPR outbreaks. With the start of the rainy season between (June/July and August/September), the migratory activity of animals is reduced due to the increased availability of local fodder. The nutritional status of the animals is also improved; resulting in an increased resistance to infection. These factors may play a key role in limiting the transmission of disease. PPR prevail throughout the year in the country [21,22].

Climatic condition is also a major factor and outbreaks are most frequent during the rainy season or the cold dry season. In subtropical areas, the occurrence of the disease is more common during winter and rainy seasons [18,21,22]. The disease has been associated with increased animal movement for commercial and trade purposes, transhumance and nomadic customs, climatic changes and extensive farming practices.

**Geographic distribution:** PPR is widely spread in the intertropical regions of Africa, Arabian Peninsula and Middle East and Asia [23]. Previously it was considered that PPR confined to West Africa but later on it has expanded to cover large regions of Africa, the Middle East and Asia by chronological spread from West Africa to Eastward [24]. However, this does not necessarily mean that PPR originated in West Africa [25] rather, the global spread of PPR is probably related to the progressive control and eradication of rinderpest as cessation of rinderpest vaccination campaigns and loss of antibody cross-protection between the two diseases consequently, small ruminants are fully exposed to PPR [26].

Based on the sequence analysis of F and most divergent N genes (most appropriate for molecular characterization) the strains of PPRV can be grouped into four lineages (I–IV), which are genetically distinct [18]. The three first lineages were historically settled in Africa, Lineage III is also common to south part of Middle East countries like Yemen, Qatar and Oman and unexpectedly once southern India [27]. The fourth lineage was until recently confined to Asia, including Turkey and the Arabic peninsula but within a remarkably short time, it spread to a large part of the African continent [5]. Therefore, based on molecular epidemiology currently all four lineages are found in Africa while lineage III and IV found in Asian continent [4]. PPR virus exists as a single serotype but at the genetic level is divided into four distinct lineages (I– IV) based on the fusion (F) protein gene



sequence [5]. Lineage number I includes the group of virus strains found in West Africa where the disease was first identified (Côte d'Ivoire) and also where the first virus isolation was made (Senegal). Lineage II consists of a group of viruses that were initially found in Nigeria. Lineage III, which was first identified in East Africa, is shared between Africa and the Middle-east on both sides of the Red Sea. Lineage IV, a unique lineage in Asia, covers a large area from Turkey to Southern Asia through the Arabian Peninsula [28].

Currently, the disease is widespread in western, central, eastern and northern Africa and the four genetic lineages are all present in different regions of the African continent. Therefore, based on molecular epidemiology currently all four lineages are found in Africa while lineage III and IV found in Asian continent [5].

### Transmission

PPRV is mainly transmitted by the aerosol route during close contact between animals mainly through sneezing and coughing [5]. The virus spread through ingestion and conjunctival penetration; by licking of bedding, feed and water troughs are also common. Furthermore, Infection may spread to offspring through the milk of an infected dam [9]. Moreover, mixed populations sheep and goats, the introduction of new animals into a herd/flock, congregation of susceptible animals at grazing land and watering points and intensive type farming system alimentary facilitate the transmission of this highly contagious disease [29].

The affected animals are important source of transmission during incubation periods, subclinical cases or before the onset of clinical signs [30]. PPRV is secreted in tears, nasal discharges, and secretion from coughing and in feces of infected animal. The virus is shed from the intestine and is found in feces at the end stage of the disease approximately 10 days after the onset of fever [31].

### Life cycle of PPR virus

Peste des petits ruminant's virus life cycle starts with the attachment of haemagglutinin to the cell-surface receptors, then fusion of the virion envelope with cellular membranes occur and then the nucleocapsid virus is released into the cytosol of infected cell. The virus polymerase binds to the single promoter located at the 3' end of the genome. It partially uncoats the nucleocapsid and transcribes

the genes into positive stranded mRNAs which are then translated into structural and non-structural proteins. Transcription either terminates after a gene or continues to the next gene downstream, which means that genes close to the 3' end of the genome are transcribed in the greatest abundance, whereas those toward the 5' end are least likely to be transcribed, a phenomenon known as transcriptional gradient. Because the N gene is located at the 3' end of the genome, it is the most expressed gene [18]. The N concentration in the cell then determines when the L switches from gene transcription to genome replication. Replication results in full length, positive stranded antigenomes that are in turn transcribed into negative-stranded virus progeny genome copies. Newly synthesized structural proteins and genomes self-assemble and accumulate on the cell membrane, bud off from the cell and in the process gaining their envelopes from the cellular membrane they bud from.

### Pathogenicity and immunity

Peste des petits ruminants' virus has significant affinity to lymphoid and epithelial tissue of respiratory and gastrointestinal tracts. After the entry of the virus through respiratory system, it replicates itself in lymph nodes (pharyngeal and mandibular lymph nodes) and tonsil [32]. Subsequently, the virus enters the retropharyngeal mucosa and spreads to local lymphatic tissue for further replication. Consequently, it produces primary viremia within 2 to 3 days. The viremia facilitates spread of the virus to other lymphoid tissues and organs such as spleen, bone marrow and mucosae of gastrointestinal and respiratory tract where it cause severe damage replicating in endothelial, epithelial and monocyte cells [33,34].

Destruction of the lymphoid tissues causes lymphopenia and significant immuno-suppression on the host leading to secondary opportunistic infection by increasing susceptibility of the animals to other microorganisms [35,36].

### Clinical signs

Sheep and goats are the primary hosts for PPRV. However, goats are highly susceptible to the disease than sheep. Incubation period of the virus can range from 3 and 10 days even though the typical period is 4 to 6 days. At acute stage of PPR disease, the animals usually exhibit clinical signs such as fever (up to 41°C) lasting for 3 to 5 days, depression, anorexia and muzzle dryness [36]. They can also show excessive

salivation, watery nasal and lachrymal discharges that gradually changes to mucopurulent. Erosive lesions are also formed in oral cavity and may become necrotic as the disease stage progress. Subsequently, the animals develop diarrhea and cough with labored abdominal breathing in the later stage of infection. The disease condition may last for 14 days before recovery from infection or leads to death during the acute stage of infection. In general, the clinical signs considerably depending on the virulence of virus [37].

### Immunity to PPRV infection

Sheep and goats that recover from PPR develop an active immunity against the disease and resist infection with PPR. Antibodies have been demonstrated for four years after infection, however, immunity is lifelong. Young animals from dams with previous history of PPR are protected up to 3 - 4 months of age by maternal antibodies. Clostral immunity protects kids and lambs until they are weaned. Therefore, the age of three months should be considered a suitable and optimum time for effective immunization of small ruminants against PPR. The presence of high level of maternal antibodies has an immunosuppressive effect on the immune system of neonates and would interfere with the degree of immunologic response to active immunization. The duration of passive immunity is 120 days as estimated by the VNT compared to 90 days by C-ELISA. There were no differences in the length of maternal immunity in dams vaccinated with TCRP vaccine between 0 and 2 months and those vaccinated at 5 months [38]. Sheep and goats vaccinated with the attenuated RBOK strain of RP virus did not develop clinical disease when infected with PPR virus. The Schwarz vaccine strain of measles virus did not protect sheep and goats against PPR virus while canine distemper virus did have some cross- protection [7].

### Diagnosis of PPRV infection

**Clinical diagnosis:** Although a tentative diagnosis of PPR can be preferred based on clinical signs, laboratory confirmation is required for differential diagnosis from other diseases with similar signs. Clinically, the disease is characterized by proliferative and self- resolving lesions around the muzzle (which becomes dry) and lips of involved animals, serous nasal and ocular discharge which become mucopurulent [14]. PPR is characterized by fever, pneumonia, profuse diarrhoea and lameness, inflammation of the mucous membrane of the respiratory and digestive tracts. Scabs or nodules can be seen on the lips, the

small intestine is congested and haemorrhages may be present, lymph nodes associated with lungs and intestinal tract are soft, swollen and focally or diffusely congested. The symptoms of PPR are very similar to those of rinderpest, only the formation of small nodular skin lesions on the outside of the lips around the muzzle and the development of pneumonia during the later stages of the disease are seen in PPR but not in rinderpest. On Post mortem, the carcass is usually emaciated and soiled with soft/watery faces.

Disease severity and the clinical signs depend on various factors: PPRV lineage, species, breed and immune status of animals. So a definitive diagnosis of PPRV infection cannot be based on clinical impressions alone, but must rely on laboratory confirmation.

**Laboratory diagnosis:** The measuring mechanism for diagnosis paste des petits ruminants infected small ruminants routinely diagnosed based on several combinations such as clinical examination, gross pathology, histological findings and laboratory confirmation to implement control measure. The test made is rapid, specific and sensitive methods for diagnosis [39]. For paste des petits ruminant's diagnosis, the sample can be taken from swabs of the mucous membrane of the eye, nose and blood. Sample also can be isolate from these organs such as large intestine, lungs and spleen. After isolation of these samples the isolate should be transported within the cold chain and refrigeration. To detect PPR can be used for serological and molecular diagnostic tests [40]. The laboratory techniques used for the detection of the virus includes virus isolation, detection of viral antigens, nucleic acid sequencing and detection of specific antibody in serum [41]. PPR virus is cross-reacts with rinderpest virus. Virus isolation is a definitive test but is labor intensive, incontinent and takes a long time to complete. Currently, antigen capture ELISA and reverse transcription-PCR are the preferred laboratory tests for confirmation of the virus.

*For antibody detection competitive ELISA and virus neutralization are the OIE-recommended tests [42]. Some of these testes are described below.*

**Gene detection:** The PCR is the most sensitive assay for confirming PPR diagnosis. A rapid and specific TaqMan-based, real-time quantitative reverse transcription PCR (qRT-PCR) has been described for the detection of Peste Des Petits Ruminants Virus (PPRV). The Primers and probe were designed based on the nucleocapsid protein gene sequence. The

real-time qRT-PCR assay allows the rapid, specific and sensitive laboratory detection of PPRV in tissue samples from field cases [43].

**Virus sequencing:** Nucleotide sequencing of the PCR product offers the opportunity to differentiate specific PPR virus lineages and more effectively trace the source of outbreaks and enhance our understanding of the epidemiology of PPR virus. Such like technology is available in PPR OIE reference laboratories. The use of this assay becomes vital in understanding virus circulation, the distribution of different virus strains and the differing roles this technology might play in the epidemiology of the disease in the field [41].

**Competitive ELISA:** A mab -based competitive ELISA is available to detect PPR-specific antibodies in blood for monitoring the response of national herds (which may be multispecies) to PPR vaccination. This can be implemented as a standard assay for global use. Implementation should include a system of internal controls and performance monitoring to ensure standardization of results and international confidence in the data generated [41].

**Serum neutralization test:** This was one of the earliest serological assays used for determination of protective immunity to PPR virus infection. It is the prescribed test for international trade in the OIE Terrestrial Manual. The principle of this test is for titrating serum antibodies by evaluation of their neutralization effect on virus infectivity on cells. To this end, serum dilutions are incubated with the viral suspension and distributed over a cell culture in tubes or plates. After one to two weeks incubation, neutralizing antibodies will inhibit visible Cytopathic Effects (CPE) comparatively to the virus alone. The serum titre is the last dilution of the test serum for which no CPE is observed. This reaction requires culture stocks of sensitive cells and vaccine virus [44].

PPR disease was clinically suspected for the first time in Ethiopia in 1977 in a goat herd from Afar region eastern part of the country. Clinical and serological evidence of its presence has been reported in 1984 and later confirmed in 1991 with cDNA probe in lymph nodes and spleen specimens collected from an outbreak in a holding near Addis Ababa. There are reports of the overall sero-prevalence of 9% in goats and 13% in sheep in different parts of Ethiopia. It was also reported that 14.6% of sheep sampled along four roads from Debre Berhan to Addis Ababa were seropositive. In 1999 national sero-surveillance

of PPR conducted in Ethiopia, the overall sero-prevalence of 6.4% in both goats and sheep ranging from 0% to 52.5% was estimated. At species level sero-prevalence of sheep 46.68% was approximately the same with that of goats 50.85% which may resulted from equal exposure of sheep and goat because they are herded together and communal grazing [45].

**Treatment:** There is no treatment for PPR but it helps to give broad spectrum antibiotics to stop secondary bacterial complications and supportive treatment like dextrose normal saline for restoration of body ionic fluid balance [4]. Affected goat with stomatitis, enteritis and pneumonia were treated with penicillin and streptomycin reinforced with broad-spectrum chloramphenicol. However, mortality rates can be reduced by the use of drugs that control the bacterial and parasitic complications. Specifically, oxytetracycline and chlortetracycline is recommended to prevent secondary pulmonary infections.

**Prevention and control:** Control of PPR in non-infected countries may be achieved using classical measures such as restriction of importation of sheep and goats from affected areas or newly introduced animal should be quarantined for three weeks, sanitary slaughter and proper disposal of carcasses and contact fomites and decontamination of affected premises in case of introduction. Control of PPR outbreaks can also rely on movement control (quarantine) combined with the use of focused ("ring") vaccination and prophylactic immunization in high-risk populations [4,8]. Carcass and contact fomites should be buried or burned, barns, tools and other items that have been in contact with the sick animals must be disinfected with common disinfectants such as phenol, sodium hydroxide 2%, virkon as well as alcohol, ether and detergents. Vaccination should be carried before the start of the rainy season and annually in endemic areas [28].

**Vaccination:** Live attenuated vaccines are effective against PPR virus and now widely available. Since the global eradication of Rinderpest, heterologous vaccines should not be used to protect against PPR [4]. It has been withheld from being used because of its interference with the Pan-African Rinderpest Campaign (PARC), since it is impossible to determine if seropositive small ruminants have been vaccinated or naturally infected with RPV [8]. Sheep and goats vaccinated with an attenuated strain of PPR or that recover from PPR develop an active life-long immunity



against the disease [4]. Several homologous PPR vaccines are available, being cell culture-attenuated strains of natural PPRV [46].

In 1998, the OIE World Assembly (formally OIE International Committee) endorsed the use of such a vaccine in countries that have decided to follow the 'OIE pathway' for epidemiological surveillance for rinderpest in order to avoid confusion when serological surveys are performed. Homologous PPR vaccine attenuated after 63 passages in vero cell was used and produced a solid immunity for 3 years. The PPRV homologous vaccine was found to be safe under field conditions even for pregnant animals and it induced immunity in 98% of the vaccinated animals. The PPRV vaccine has been tried for protection of cattle against RP and it was found very effective. PPR vaccine seed is available through the Pan African Veterinary Vaccine Centre (PANVAC) at Bishoftu, Ethiopia, for Africa [8].

There have also been two published reports on the preliminary results from recombinant Capripox-based PPR vaccines that are able to protect against both capripox and PPR [47,48]. Rinderpest and PPRV both belong to morbillivirus with cross reactivity and relatively similar immunological response and clinical feature. Rinderpest is a fatal and acute disease in cattle while in sheep and goat characterized as a sub-acute and mild disease [10]. It is assumed that PPRV is the consequence of rinderpest natural passage in sheep and goat. Seroprevalence surveys showed seropositive case causes a humoral response against PPRV in cattle and buffalo [8]. PPR virus in cattle is now a threat, whether the cattle should be vaccinated to control PPR or not. However, the vaccination of cattle with goats and sheep is not cost beneficial [31].

**Socioeconomic impact of PPR:** PPRV is currently considered one of the main animal trans-boundary pathogens that constitute a significant threat to livestock production in developing countries. In those areas affected by the disease, PPR is considered a major limiting factor in the development of the small ruminant industry [49]. This is especially evident in many countries in Africa and Asia where sheep and goats play an integral role in sustainable agriculture and employment [27]. The potential and real economic impacts of PPR outbreaks are extremely high and the impact of the disease on the poorer sections of society is disproportionate, reflecting an intrinsic dependence on sheep and goat farming [27].

According to the study in Afar Regional state,

North Eastern Ethiopia Was reported about 63.3% of the total population of sheep and goats were lost each year due to PPR. The financial loss due to mortality in the affected animal farm was on an average 2,146,875.00 birr/92,140.56\$ both in sheep farm and in goat farm [50].

According to the study conducted in Ada'ar and Mile districts of Afar Regional state by Gizaw, et al. [50] the prevalence of PPR was 92 (40.2%) out of 229 analyzed serum samples. Another study conducted in Selected districts of Silte and Guraghe Zones of South Regional state, The Overall prevalence was found to be 29.2% (114/390) [51]. Out of 700 blood samples obtained from goats and sheep, research conducted in the East Shewa and Arsi zones found that 48.43% were overall PPR seropositive [45].

A cross-sectional study reported by [52], indicates that PPR was widely prevalent in small ruminants in the study areas. All villages, except one in Gambella, had seropositive cases. Such a high prevalence in most of the villages (more than 30%) suggests a remarkable contagious nature of the disease, covering wide geographic areas and infecting perhaps most of the susceptible animals in affected villages. The overall seroprevalence (30.5%) of transmit the virus to susceptible small ruminant population and, therefore, the movement of animals plays an important role in the transmission and maintenance of PPRV in nature.

Because of the negative economic impact on countries affected by PPR, the disease is one of the priorities among international and regional livestock disease research and control programs [53]. The disease has also been ranked by pastoral communities as one of the top ten diseases of small ruminants [54].

**PPR control measures and challenges in Ethiopia:** Currently, the strategy of PPR vaccination is ring vaccination to control the spread of PPR infection to provide a vaccinated barrier between infected animals and clean stock. The intervention is expected to contain the outbreak of the disease and reduce losses. The vaccine is provided by the federal government in coordination with, FAO and several NGO's. Mass annual vaccination programs have also been practiced since 2005 with annual vaccination coverage reaching nearly 6 million heads (20%) of small ruminant are vaccinated.

Even though, the National veterinary Institute (NVI) is producing sufficient doses of live attenuated

tissue culture homologous PPR vaccine (PPR 75/1 Vero 76, attenuated, freeze dried) to satisfy the vaccination programs, a progressive control campaign based on repeated vaccination of all susceptible small ruminants is difficult and unaffordable. The major challenge in control of PPR in the region is lack of adequate information on the dynamics of the disease in the region and inefficiency in early detection, especially because communities and even most of the animal health workers on ground are not familiar with the disease symptoms and may dismiss it as simple pneumonia, CCPP and Orf [29].

Furthermore, several agro-ecological conditions accompanied with seasonal occurrence of the disease, movement of infective small ruminants within the country and cross-border particularly, the pastoral areas of Afar, Somali and Oromia are well known for significant movement of small ruminants and other livestock species within these regions, to towards central high lands where important livestock markets and export abattoirs are located. Moreover, a cross-border seasonal movement in search of pasture and water in pastoral areas of Kenyan border is also a great challenge to control this widely spreading disease [55]. Therefore, strict animal movement control within the country and cross-border should be effective and use of epidemiological intelligence to initially target endemic populations and high-risk areas will be essential (Figures 1-4).

## Conclusion and Recommendations

Peste-des-Petits Ruminants is one of the most important economical diseases in Ethiopia, since it had been confirmed in goats in 1991. The epidemiology of the disease is much more complex than previously thought with added differences in pathogenicity and virulence. The pastoral community has developed a very comprehensive description of PPR disease overtime thus is a repository of livestock disease information for their locality.

Seasons, geographical locations and livestock management activities were also identified as risk factors to PPR disease outbreaks. Risk factors associated with presence of PPR antibodies in small stock were species, age group, geographical administrative areas and vaccination status.

Understanding this spatial-temporal heterogeneity of risk factors will greatly improve design of disease control measures against PPR. However, it cannot be generalized that risk factors in

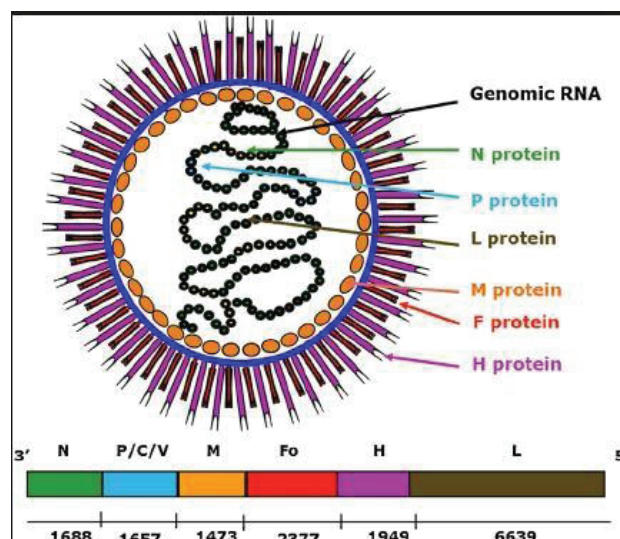


Figure 1 Schematic representation of the PPR morbillivirus.



Figure 2 Official Status of PPR in OIE member countries.



Figure 3 Common clinical signs of acute PPRV infection in small ruminants: Early oral lesions and necrosis of the gum [27].

one year are similar in other similar seasons of other years considering livestock are in constant move in search of pasture and water. The disease has the potential of destroying livelihoods and reducing most herders to destitution consigning them into the ever growing internally displaced camps of economically challenged people.



**Figure 4** Clinical signs in goats with peste des petits ruminants.

Based on the fact and information mentioned in the review the following recommendations are forwarded.

- Quarantine and restrictions on movement of sheep and goats from affected areas. The affected area should be quarantine by avoiding introduction of healthy animals.
- Proper disposal of carcasses of shoats dying of the disease (burned or buried) and disinfecting contact fomites. Most common disinfectants (phenol, sodium hydroxide, alcohol, ether, and detergents) can be used.
- Focused “ring vaccination” in surrounding areas where outbreaks have been detected.
- Advice farmers/pastoralists to keep newly purchased sheep and goats separate from other animals.
- Advise farmers/pastoralists to isolate animals with signs of PPR immediately and to move their healthy animals to other clean area.
- Further study should be conducted on the epidemiology, economic importance, and prevention of PPR

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