Journal of Veterinary Science & Medical Diagnosis

Research Article A SCITECHNOL JOURNAL

Comprehensive Review on Peste des Petits Ruminants in Small Ruminants in Ethiopia: with Emphasis on the Current Status and Future Prospective

Paulos Tone Dana*

Department of Veterinary Medicine, University of Gondar College of Medicine and Health Sciences, Gondar, Ethiopia

***Corresponding author:** Paulos Tone Dana, Department of Veterinary Medicine, University of Gondar College of Medicine and Health Sciences, Gondar, Ethiopia; E-mail: pau.los.@gmail.com

Received date: 23 October, 2024, Manuscript No. JVSMD-25-150829;

Editor assigned date: 25 October, 2024, PreQC No. JVSMD-25-150829 (PQ);

Reviewed date: 08 November, 2024, QC No. JVSMD-25-150829;

Revised date: 08 February, 2025, Manuscript No. JVSMD-25-150829 (R);

Published date: 15 February, 2025, DOI: 10.4172/2325-9590.1000121.

Abstract

The Food and Agriculture Organization estimates that a 62.5% of household small ruminants worldwide are susceptible to contracting the PPR virus. The billion-strong population of small ruminants in Africa, the Middle and Near East, South-West, and Central Asia is currently under risk from PPR, a significant animal virus that affects sheep and goats. 1984 following clinical observations compatible with PPR infection, it was initially suspected in Ethiopia and subsequently identified as the cause of sickness in the nation's goat population. The Peste des petits ruminant's virus, a Morbillivirus that is a member of the Paramyxoviridae family, is the cause of PPR, a widespread and economically significant viral illness of ruminants. Aerosols and direct contact between infected and vulnerable animals are the main ways that the virus is spread.

Morbidity and mortality are usually high, and PPR can create epidemics that can cause up to 100% mortality in susceptible sheep and goat populations. The diagnosis protocols range from symptomatic diagnosis to virus isolation. The estimated pooled prevalence rates in different regions of Ethiopia were ranging from 14.82% to 60.97%. PPR is a target animal disease for poverty reduction. The FAO has started a mission to over time control and eradicate PPR globally by 2030, and Ethiopia has achieved elimination or eradication through a targeted, epidemiology-driven approach. This may achieved successfully by involving all veterinary actors in the field, supporting the establishment of sustainable animal health delivery systems, continuous updating understanding of PPR epidemiology, identifying reservoirs of infection, strengthened surveillance and diagnosis, enhanced use of vaccine, all the implementations should be regularly and intensively guided by federal or regional government and the branch coordination offices, but involving all actors: Non-governmental organizations and private veterinarians in relevant areas.

Keywords: PPR virus; Current status; Future prospective; Morbidity; Ruminant

Introduction

The Peste des Petits Ruminant's Virus (PPRV) is the cause of peste des petits ruminants. The virus belongs to the Paramyxoviridae family and genus *Morbillivirus*. Goats and sheep are the main victims of this illness [1]. OIE classifies Peste des Petits Ruminant (PPR), an acute, highly contagious, and economically significant transboundary viral disease of small ruminants, as a disease that has to be reported. Severe fever, Oculo-nasal discharge, necrotizing and erosive stomatitis, enteritis, and pneumonia are the disease's clinical hallmarks [2].

Aerosol and direct contact between infected and susceptible animals are the main ways that the virus spreads [3]. The sickness takes four to six days to incubate, but it can take up to fourteen days. The infection period is usually 5–7 days, and death of the infected animal may occur within 10–12 days' post-infection due to severe dehydration and respiratory failure [4]. Morbidity and mortality are usually high, and PPR can create epidemics that can cause up to 100% mortality in susceptible sheep and goat populations [5]. Although cattle and pigs can contract PPRV, they do not add to the epidemiology because they are unable to expel the virus. Instead, PPRV mostly infects sheep and goats. Sylvatic reservoirs for PPRV have been documented, and infections and fatalities in captive wild ungulates from several species have been previously recorded [6].

Since the discovery of PPR, there have been many advances in the diagnosis of PPR in sheep and goats, and diagnosis protocols range from symptomatic diagnosis to virus isolation [7]. The gold standard for diagnosing PPR is virus isolation, although this is generally not feasible in practice [8]. As a *Morbillivirus*, PPRV is antigenically similar to the viruses that cause rinderpest in cattle, measles in humans and distemper in dogs, but can be serologically distinguished by use of commercially available Enzyme-Linked Immunoassay (ELISA) kits [9].

The Food and Agriculture Organization (FAO) estimates that 62.5% of household small ruminants worldwide are susceptible to contracting the PPR virus. PPR is a target animal illness to reduce poverty. The first report of PPR was made in the Ivory Coast, West Africa, in 1942. Today, PPR is quite common in both Africa and parts of Asia, and is emerging as a threat to other continents such as Europe. In a remote area to the east of Ethiopia, clinical PPR was detected in 1977. Clinical and serological evidence of its presence confirmed in 1991 in Addis. Ethiopia is home to lineages III and IV. In East African nations including Kenya, Sudan, Uganda, and Tanzania, Lineage III is in circulation. In Ethiopia, 0% to 52.5% prevalence of PPR was reported from different parts of the country.

Therefore, the objectives of this review manuscript are:

- To provide insight on the current epidemiology and diagnostic techniques of PPR.
- To compile existing information on the prevention and control strategies of PPR.
- To review on the socio-economic impact and opportunities for PPR eradication.

Materials and Methods

Etiology of PPR virus

The Peste des Petits Ruminant's Virus (PPRV) is the cause of peste des petits ruminants. The virus belongs to the Paramyxoviridae family and genus *Morbillivirus*. The diameter of pleomorphic virions ranges from 130 to 390 nm. The virus envelope is 8-15 nm thick with glycoprotein spikes of 8.5-14.5 nm length being present throughout the membrane. Six structural proteins—the Nucleoprotein (N), Phosphoprotein (P), Matrix protein (M), Fusion protein (F), Haemagglutinin protein (H), and Large polymerase protein (L)—as well as two nonstructural proteins, V and C, are encoded by PPRV's non-segmented, single-strand RNA genome of 15,948 nucleotides. These proteins are arranged in the following order: 3′-N-P(C/V)-M-F-H-L-5′.

Based on partial sequence analysis of the F gene, the various PPR viruses (PPRV) that have been isolated thus far in each of these regions were divided into four lineages (I–IV). Viruses that were found in Africa in the 1970's are representative of lineage I. The only African lineage that did not cross the Red Sea to reach Asian nations is lineage II, which contains viruses isolated in West Africa (Ivory Coast and Guinea) in the late 1980's. Isolates from Eastern Africa (Ethiopia and Sudan) make up lineage III. Asia is the only region where PPR virus isolates belonging to lineage IV are found, including those from Israel, Iran, Nepal, Bangladesh, Turkey, and India (Figure 1).

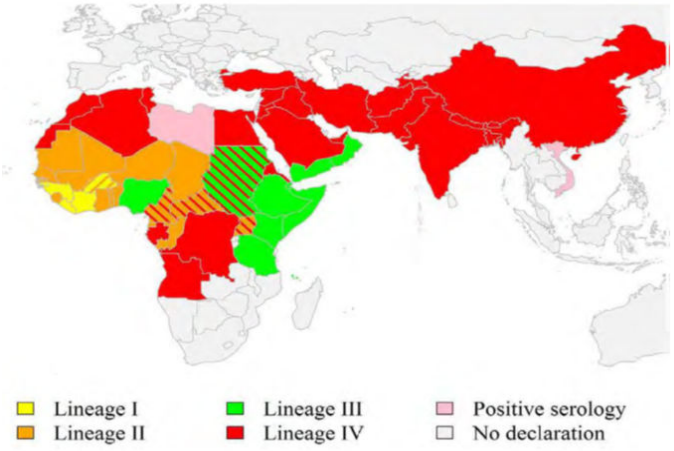

Figure 1: Map showing the worldwide distribution of PPR lineages.

Current distribution of PPR virus

PPR, sometimes known as "Goat Plague," is an acute, highly contagious, transboundary viral illness that affects sheep and goats. It causes significant morbidity and mortality and significantly reduces the productivity of small ruminants in some regions of the world. In 1942, the first reliable and scientific account of illness was published. At that time, Gargadennec and Lalanne reported an epidemic disease in West Africa's Ivory Coast (Cote d'Ivoire) that was clinically identical to rinderpest (RP) but exclusively affected small ruminants, with in-contact cattle appearing to be well. Currently, OIE has received reports of PPR disease from over 70 countries. The majority of those nations are in Africa, while some are in Asia and the Middle East.

Geographical distribution of the disease: Many nations are currently thought to be at risk for PPR illness. Based on partial sequence analysis of the F gene, the various PPR Viruses (PPRV) that have been isolated thus far in each of these regions were divided into four lineages (I–IV). Viruses that were found in Africa in the 1970's are representative of lineage I. The only African lineage that did not cross the Red Sea to reach Asian nations is lineage II, which contains viruses isolated in West Africa (Ivory Coast and Guinea) in the late 1980's. Isolates from eastern Africa (Ethiopia and Sudan) make up lineage III. The Asian isolates from Israel, Iran, Nepal, Bangladesh, Turkey, and India comprise lineage IV of PPR virus isolates, which are exclusive to Asia.

In a remote area to the east of Ethiopia, clinical PPR was detected in 1977. In 1991, Addis provided clinical and serological proof of its existence. In Ethiopia, lineage IV has recently been discovered. PPRV (Ethiopia/2010) complete genome sequence data shows a genetic clustering with isolates of lineage IV. As seen in Figure 2, the spread of PPR has steadily increased, encompassing sizable regions in Africa, the Middle East, and Asia (Figure 3).

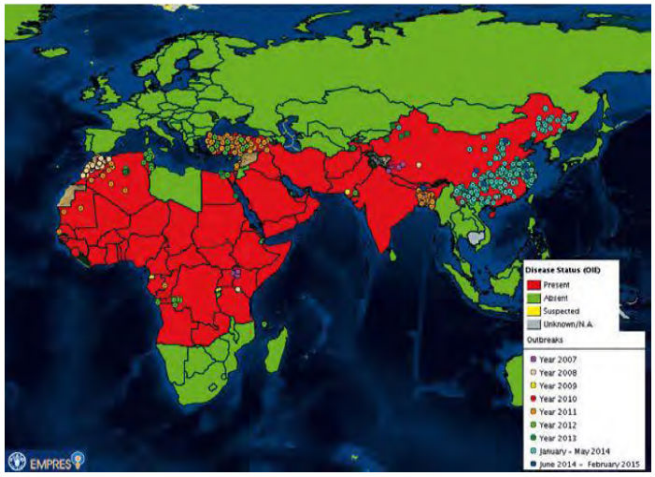

Figure 2: Current global PPR situation and occurrence of outbreaks from 2007-2014.

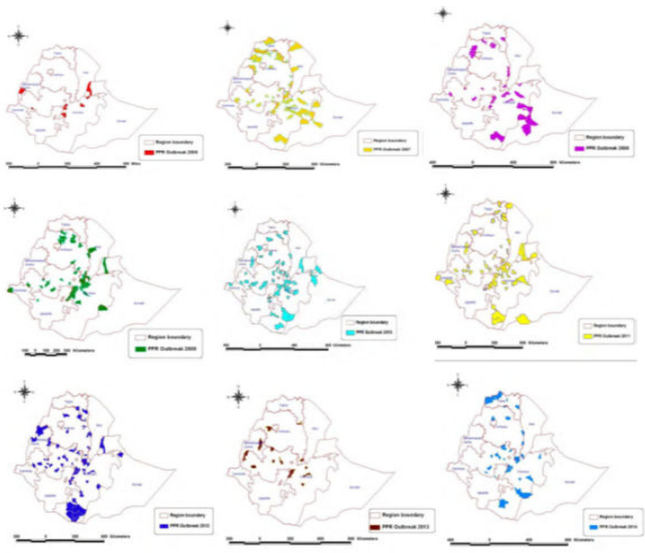

Figure 3: Maps depicting the distribution of PPR outbreak reports 2006-2014.

Molecular epidemiology of PPRV: Because antibody levels are exacerbated by ongoing vaccination programs and the epidemiological situation (endemic or epizootic), the sero-prevalence levels of PPR are typically challenging to evaluate. Sheep are generally more resilient to the illness than goats. Due to higher case mortality rates in goats, several serological surveys have found that sheep have a higher prevalence of antibodies than goats. Sero-prevalence rates in Ethiopia vary greatly between areas and even more between weredas; in 2008, they ranged from 0% to 52.5%.

An epidemic (outbreak) of PPRV in a specific region, variations in disease identification techniques, sample origin, sampling strategy, study year, study duration, and animal species could all be contributing factors to the inconsistent sero-prevalence estimates of PPR. According to Liuel et al., the estimated pooled prevalence in Afar, Amhara, Benishangul-Gumz, Gambella, Oromia, Somali, Southern nation, nationality people's region, and Tigray was 25.28%, 14.82%, 60.97%, 25.55, 23.42%, 16.29%, 16.19%, and 39.9%.

The below Figure 4 is for administrative map of Ethiopia indicating the regions and weredas boundaries. For each wereda sero-prevalence of PPR was calculated by dividing the number of positive valid samples by the number of individual sampled in the wereda. As the color gets browner higher is the sero-prevalence found in the area. The grey colored is for weredas for which no data was available.

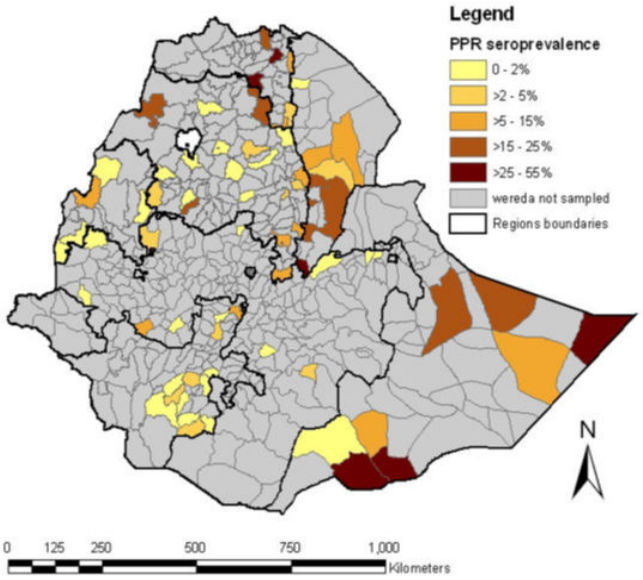

Figure 4: Sero-prevalence of PPR across wereda in Ethiopia.

Quantification of PPR by species in different regions of Ethiopia: Most recently, an overall sero-prevalence record of 30.9% from sheep and goat in pastoral and agro-pastoral area of afar and Gambella region of Ethiopia has been reported. The disease probably was introduced into Ethiopia in 1989 in the Southern Omo river valley from where it moved eastward to Borena region and then northwards along the Rift valley to Awash (Table 1).

Country/Ethiopia regions	Species	No. tested	Prevalence (%)
Afar, Borena, East Afar, Borena, East Shewa, Gambella, Jijga	Sheep	835 (cELISA)	13%
	Goat	442 (cELISA)	9%
	Cattle	910 (cELISA)	9%
	Camel	628 (cELISA)	3%
Afar (Awash Fentale)	Sheep and goat	23 (cELISA)	36.60%
North Shewa	Sheep and goat	-	29%
Gambella (Itang)	Sheep and goat	779 (cELISA)	27.30%
Afar (Adaar)	Sheep and goat	384 (cELISA)	38.30%
Afar	Sheep and goat	1653 (cELISA)	15.30%
Amhara	Sheep and goat	5992 (cELISA)	4.60%
Benishangul-Gumuz	Sheep and goat	729 (cELISA)	8%
Oromia	Sheep and goat	2290 (cELISA)	1.70%
SNNPR	Sheep and goat	1622 (cELISA)	1.80%
Somali	Sheep and goat	465 (cELISA)	21.30%
Tigray	Sheep and goat	900 (cELISA)	15.30%

Table 1: Different studies of PPR in different hosts and in different districts of Ethiopia.

Transmission of the virus: The main way that the peste des petits virus spreads is through inhalation while in close proximity to an infected animal. Saliva, urine, nasal and ocular secretions, and diarrheal feces have all been reported to contain this virus, which can be shed during

the incubation phase. Model outputs suggest that PPRV transmission was sustained in Ethiopia's lowland pastoral region through viral transmission between small ruminant village populations. Lowlands thus acted as a reservoir of infection from which PPRV spilled over into the highland sedentary region where its maintenance was unlikely. The trade of sheep and goats from Ethiopian lowlands into the neighboring countries and Gulf states makes PPRV elimination from Ethiopia's lowlands not only a national, but also a regional and even global priority.

Host range: Although cattle and pigs are also susceptible to infection, they do not add to the epidemiology because they are unable to expel the virus. The Peste des petits ruminant's virus mostly affects sheep and goats. Both sheep and goats are clinically sensitive to PPR, but goats are more vulnerable than sheep. Goat breeds have a significant impact on susceptibility; for example, Guinean dwarf goat breeds from West Africa, such as Djallonke, Kirdi, and Lagoon, are far more vulnerable than the main Sahelian breeds. Abraham et al. reported that camels in Ethiopia have a 3% antibody sero-prevalence. Both tiny ruminants in the wild and those in zoos have been reported to have the illness.

Social ecology and seasonality of the PPR disease: According to reports, the recent PPR disease outbreaks have been linked to the small ruminants' complete exposure to PPRV due to the discontinuation of rinderpest vaccination and the loss of antibody cross protection between the PPR and rinderpest. However, social, cultural, and economic activities like wars, natural catastrophes, cattle trading, cultural festivals, changes in husbandry techniques, nomadism, and seasonal climatic and environmental changes have long been linked to the propagation of PPR outbreaks.

The model suggested frequent PPRV incursions into highlands from lowlands. In the search for grazing and watering points, pastoral flocks may move toward highlands, where they then mix with sedentary flocks. Moreover, goats and sheep traded from lowlands into highlands are moved through several markets, over long distances. Such marketing systems are likely to promote viral amplification, as observed with other species. While most animals traded from lowlands would end up in abattoirs, they could infect highland animals brought to markets. Unsold highland animals returning to their village of origin could then spread the infection. If these interfaces between pastoral and sedentary populations were characterized spatiotemporally, they could be targeted by vaccination to reduce viral spillover.

According to Abraham and Waret-Szkuta et al., the seasonality of PPR in Ethiopia has been linked to the seasonal movement of small stock in search of water and pasture resources during dry seasons, social animal exchange, and livestock marketing, which show seasonal patterns with pick outbreaks occurring in March–June and October–November.

Potential risk factors and wild life susceptibility to the disease: Children under one-year-old and older than four months are particularly vulnerable to the illness. It is thought that Sahelian sheep and goat breeds are more resilient than the dwarf varieties found in West Africa's humid and sub-humid regions. Introducing a new stock or returning unsold animals from livestock markets significantly increases the probability of an outbreak in a given flock. Animals that have recovered are immune for life. The wet or cold dry seasons are when outbreaks are most common, and climate conditions are another risk factor. Risk factors for PPR disease include large flock sizes, animals that go to animal markets, and insufficient veterinarian care.

A complicating element is the susceptibility of wildlife to PPR; infection and clinical disease have been documented in gemsbok, ibex, Thomson's gazelle, and Dorcas gazelle (in captive populations). No research has been done on the function of wild species as a reservoir. However, more research is required about possible PPR spread through wild species given the involvement of wildlife in the epidemiology of rinderpest. Recently, an outbreak of PPR in really free-ranging Sindh ibex was verified by immune-capture ELISA and PCR in Pakistan in 2010, resulting in 36 deaths. This outbreak may have been linked to sharing pasture and water with a herd of goats that were thought to be infected.

Diagnostic techniques and control approaches

Clinical observations, distinctive symptoms, epidemiology, post-mortem lesions, and laboratory confirmation using a variety of serological and molecular approaches are used to provisionally diagnose PPR.

Pathological lesions and clinical manifestations: The incubation period for Peste des petits ruminants is two to ten days, after which there is a sudden onset of pyrexia (40-42°C) that may last for three to five days, severe depression, anorexia, and clear nasal and ocular discharges that turn muco-purulent due to secondary microorganism infection. In addition to matting around the eyelids, crusts may form on the nose, interfering with the nostrils and causing respiratory difficulty. The oral and ocular mucosae membranes get congested one to two days after the symptom appears. The multifocal pinpoint necrosis of the gum, dental pad, palate, lips, inner parts of the cheeks, and side of the tongue is the goal of this. According to Sunelle, these death zones should even merge.

At first, most PPR outbreaks were identified using common clinical symptoms. However, it can be challenging to differentiate the symptoms of PPR from those of several other illnesses, including bluetongue and foot-and-mouth disease. Clinical and pathological abnormalities can be used to make a provisional diagnosis of PPR. With the exception of the PPRV's exceptional affinity for lung tissues, the clinical manifestation of PPR is essentially comparable to that of RP. High fever (pyrexia), oculo-nasal discharges, necrotizing and erosive stomatitis, gastroenteritis, diarrhea, and bronchopneumonia are the disease's clinical manifestations. The animal may either die or recover from the illness.

The disease has several stages:

- Incubation period.
- Prodromal phase (febrile).
- Mucosal phase (ocular and nasal discharges, hyperaemia of conjunctiva and anterior nares mucosa, and erosions on the tongue, palate, lips, and other parts of the oral mucosa) (Figure 5).
- Diarrheal stage.
- In non-fatal cases, "recovery stage," during which sheep and goats that recover from PPR develop an active, lifelong immunity.

Depending on the severity of the illness, PPR might present as acute, mild, or per acute.

Retrogressive and necrotic alterations in lymphoid tissues and gastrointestinal and respiratory system epithelial cells define and predominate the pathophysiology of PPR. Consolidation, color changes in the lungs, and occasionally frothy mucus seen in squeezed

lung pieces are the most noticeable lesions in PPR-infected animals. The antero-ventral regions of the right lung are often affected; parts of the lungs turn dark red or purple and become firm to the touch, primarily in the anterior and cardiac lobes (Figure 6).

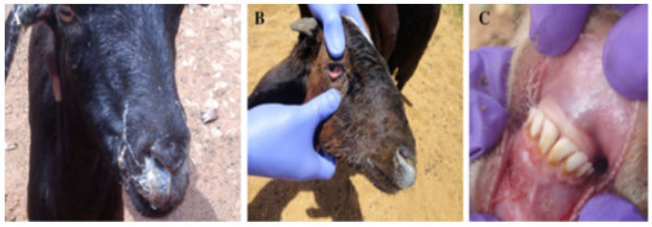

Figure 5: PPR virus infected animals showing nasal discharge (a) congestion of conjunctiva (b) and Bran-like deposits-discrete tiny necrotic ulceration or foci in the mucous membrane (c).

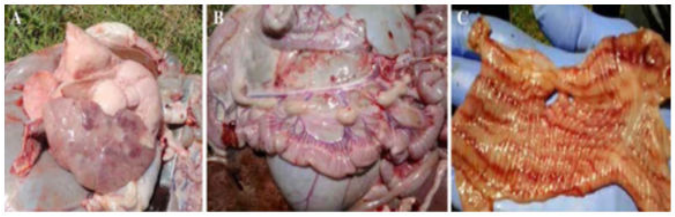

Figure 6: Post-mortem lesion showing congestion and consolidation of lobes of lung (a) enlarged oedematous and congested intestinal mesenteric lymph nodes (b) and colon showing discontinuous streaks of congestion and haemorrhages (Zebra markings) on the mucosal folds (c).

Conventional tests/assays: PPRV-specific antibodies have been used for the detection of virus antigen in tissue, swabs, conjunctival smears and formalin-fixed tissues by various researchers in different assays/tests. Earlier, AGID/AGPT was a frequently used method for the detection of RPV and PPRV antigens in the clinical and post-mortem samples but remains relatively insensitive. CIE was comparatively more sensitive and rapid method than that of AGPT, but failed to differentiate between PPRV and RPV infection. Viral Neutralization Test (VNT): Is applied to a serum sample; this technique needs also cell culture facilities.

Among all the methods, isolation of the virus remains the ‘‘gold standard’’ for diagnosis of PPR. The PPRV can be isolated and grown *in vitro* in primary bovine and sheep cells as well as established cell lines such as Vero (African green monkey kidney) cells and Marmoset B-lymphoblastoid-B95a cells. The virus manifests specific Cytopathic Effect (CPE) after 3–5 days of infection, which include initial rounding of the infected cells in grape-bunch-like clusters, followed by vacuolation, granulation of the cell cytoplasm, fusion of the monolayer cells and formation of syncytia, which are characteristics of PPRV.

Enzyme-Linked-Immunosorbent Assay (ELISA): Immunocapture and sandwich ELISAs are available to efficiently detect antigens in the tissues and secretions of PPRV infected animals. Both these assays utilize Monoclonal Antibodies (MAbs) directed against the N protein of PPRV. Both assays are rapid, sensitive and specific with a detection limit of 100.6 TCID50/well. Since the MAbs used in these assays are raised against the non-overlapping domains of the N protein of PPR and RP viruses, this assay can be used to differentiate PPRV from RPV infected animals. The Lateral Flow Device (LFD)-based test for PPR using monoclonal antibody C77 recognizing the H protein of PPRV.

Molecular diagnostic techniques: Real-Time Polymerase Chain Reaction (RT-PCR) is an accurate, rapid and reliable method that can be used for the detection and also for the quantization of specific DNA molecules. The conventional RT-PCR has been developed for the specific amplification of the NP gene or the amplification of the fusion (F) gene and is established in various laboratories. The real-time PCR assay specific for PPRV and the Loop-Mediated Isothermal Amplification Technique (LAMP-RT-PCR) is also available for the genome detection of PPRV.

Pen side tests: For the detection of PPRV antigen in tissue homogenate/swab samples of sheep and goat origin, a straightforward dot-ELISA has also been developed utilizing either anti-M protein MAb or anti-N protein Mab. This test may be used as a pen side test for PPR diagnosis and has been found to be helpful for screening a large number of clinical samples. It is also appropriate for animal disease investigation laboratories in the field. When compared to s-ELISA for PPR diagnosis, this assay demonstrated relative diagnostic sensitivity (82.5%) and specificity (91%). Although it was developed, the lateral flow test for the detection of PPRV antigen and antibody was not suitable for widespread use. However, this test has the advantages of quick, easy to perform and does not involve technical skill or expertise and hence user friendly, useful as pen side diagnostic test.

Control and prevention strategies

There is no treatment for PPR but it helps to give broad-spectrum antibiotics to stop secondary bacterial complications. The controls of PPR require an effective vaccine and for this purpose, several vaccines including both homologous and recombinant vaccines have been developed. Nowadays, efficient live attenuated PPR vaccines are available that can induce lifelong protective immunity in vaccinated animals.

The challenges in control activities arise it is not possible to distinguish vaccinated animals from those that have recovered from natural infection. A Differentiation of Infected from Vaccinated Animals (DIVA) vaccine/test would improve epidemiological data by allowing tracking of infection in areas where there has been partial vaccination. Animals that have been infected are detected by the presence of antibodies to the N protein, while vaccination coverage can be assessed by the presence of antibodies to the H protein in the absence of antibodies to the N protein. Thermo-stabilizing PPR vaccine is well-suited for transport without icebox cold. Currently, the Pan African Vaccine Center (PANVAC) which is found in Debre Zeit, Ethiopia is producing and distributing effective PPR vaccines for Ethiopia and some African countries.

In 2013, the OIE and FAO together decided to develop a PPR global control and eradication strategy for a 5-year action plan (2013-2017). The task of eradicating PPR can benefit from a series of favorable elements. These include the experience gained from eradicating rinderpest (RP), several favorable technical aspects in terms of diagnosis and surveillance, effective and inexpensive vaccines that covers all known strains/lineages of the virus, no long-term virus carriers and no significant role of wildlife, a growing social and political commitment from various decision-makers at national, regional and global levels. This plan aims to control and eradication of PPR with other major diseases by reinforced veterinary service and global animal health systems, the improvement of animal health will reduce the impact of these diseases. This, in turn, strengthens the contribution made by the small ruminant sector to global food security

and economic growth while at the same time improving the livelihoods of smallholders and poor farmers. There are various tools to implement a global PPR control strategy. These include WAHIS (World Animal Health Information System), PPR Monitoring and Assessment Tool (PMAT), Post-Vaccination Evaluation (PVE), vaccines, surveillance, diagnosis, regional and international laboratory networks, OIE standards and the Performance of Veterinary Services (PVS) pathway and others.

Ethiopia has launched a national progressive PPR control strategy. The strategy will adopt geographic approaches. The initial area of operation will include at least the epidemiologically interconnected pastoral areas of the country, where a progressive control (ultimately leading to eradication) program will be implemented in strategically defined epidemiologically important (sub)-ecosystems. These areas include Somali and Afar regional states, pastoral districts of Oromia and South Omo zone of SNNPR. The strategy of the highland lowland interface was similar to that of the pastoral areas. It contains a district border with pastoral areas of the country through seasonal grazing and marketing.

The approach towards controlling PPR can be divided into three inter-dependent phases based on the epidemiology of the disease and prioritizing available resources. The first phase will establish a better understanding of the disease situation and implement disease control strategies that progressively controlled the PPR until the stage that there is evidence that the sub-ecosystem is clinically PPR disease free. The second phase was started when the veterinary services stop vaccination in the sub-ecosystem and intensify clinical disease search/surveillance to verify the absence of clinical disease for about two years and simultaneously prevent reintroduction of PPR. The third phase will serologically verify the absence of circulating antibodies and by this the absence of possible mild disease circulating in all susceptible species.

The four steps (Figure 7) that make up the PPR GCES's step-by-step eradication strategy also serve as a summary of the program's operations. These phases, which involve a multi-stage, multi-country process involving evaluation, control, eradication, and maintenance of PPRV-free status, correspond to a combination of declining levels of epidemiological risk and rising levels of prevention and control. This goes from stage 1, when the epidemiological situation is being evaluated, to stage 4, where the nation is prepared to apply for OIE-official PPR-free status after demonstrating that there is no virus circulation at the zonal or national level. Stage 2 involves the implementation of control measures, such as immunization, and stage 3 directly relates to the eradication of PPR (Figure 8).

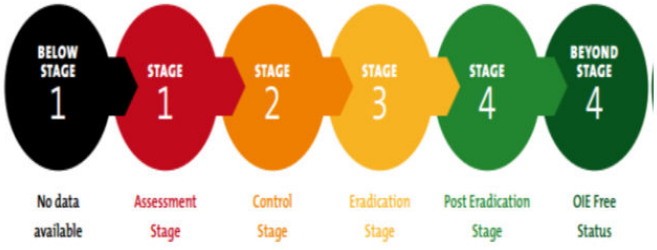

Figure 7: The four stages of the PPR GCES (FAO/OIE, 2015).

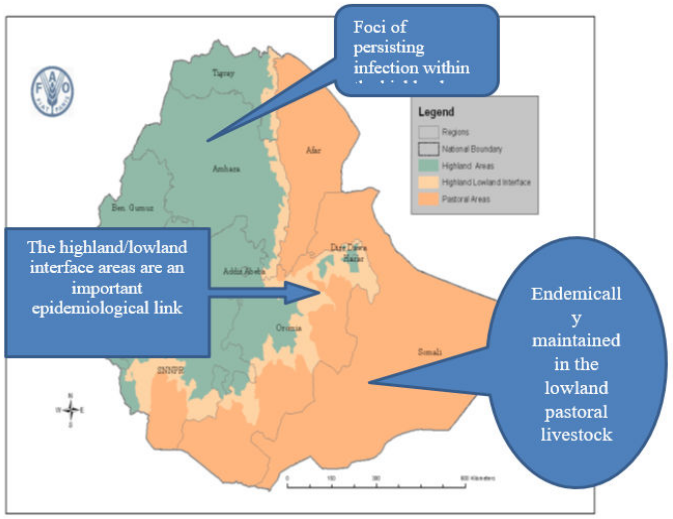

Figure 8: Current PPR situation and understanding.

Moreover, Ethiopia has successfully vaccinated over 24.5 million sheep and goats during the January 2016 to April 2019 period and the technical capacities of 2,361 veterinary officers have also been enhanced on vaccine handling and management techniques in six operational regions of the project. This has been possible through the support of the EU-SHARE PPR project (European Union's Supporting Horn of Africa Resilience), which is generously funded by the European Union (EU), FAO (Figure 9).

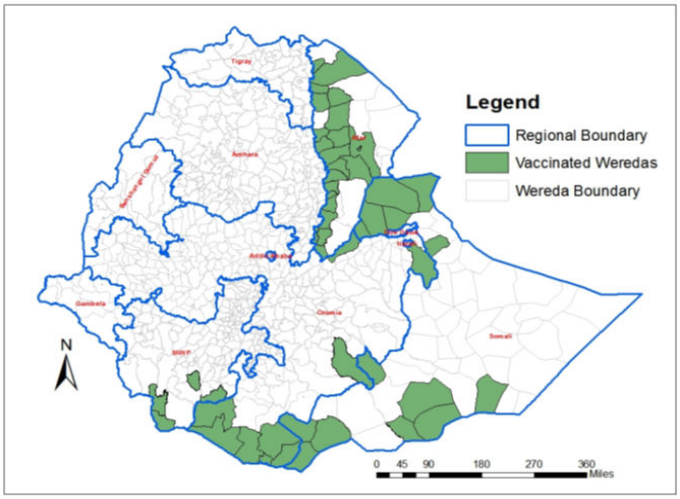

Figure 9: A total of sheep and goats vaccinated against PPR, June 2016 to January 2018.

Socio-economic impact of PPR

According to the Food and Agriculture Organization (FAO), 62.5% of global domestic small ruminant's population is at risk of being infected by PPR virus. PPR is a target animal disease for poverty alleviation.

To better understand the impact of PPR in all settings, a well-planned cost-benefit analysis of PPR is required, comparing policies and responses that take into account both the direct and indirect costs associated with PPR. However, the annual global impact of PPR has been estimated to be between USD 1.4 billion and USD 2.1 billion.

Cost-benefit analyses have also been conducted in various nations, identifying losses that amply support the pursuit of national and international PPR eradication programs.

Disease can restrict commerce and export, import new breeds, and promote intensive livestock production, all of which reduce human consumption of animal protein. Vaccination must be administered more regularly and extensively since the turnover rate of small ruminant populations is significantly higher than that of bigger cattle. However, losses could be reduced and certain regions should be able to be released from PPR if this can be accomplished under a scheme of progressive control. The most crucial factor in starting any disease management program is cost-benefit analysis. PPR epidemics can have a significant impact on livestock productivity, and controlling or eradicating the illness can be quite expensive. Epidemics affect not only individual farmers but also the agricultural industry and as a consequence, the national economy. PPR is present in countries, which are either developing or under-developed thereby adding to the economic woes.

Opportunities for PPR eradication

The epidemiology and biology of the PPRV are very much similar to those of the RPV. Therefore, there are enough reasons to control and eradicate PPR very much in a similar way like rinderpest. Like RPV, there are several aspects that may favor eradication of PPR:

- PPRV has a single serotype, and strains from various lineages are thought to exhibit perfect cross-protection.
- Vaccine is considered to provide life-long immunity.
- There is no carrier state.
- For the disease to spread effectively, the animals must come into intimate touch with one another.
- The virus requires a constant supply of vulnerable animals to survive because it is easily damaged by heat and sunshine and cannot endure for very long outside the host.
- The right diagnostic instruments are at hand. However, the vaccine may be a waste of public money and, at worst, contribute to the virus's spread if it is not utilized extensively, widely, and enough to prevent the virus's spread in endemic areas.

The eradication from a geographically defined area may require a sustained effort and regional cooperation. To reduce the problem of thermo-stability, vaccinations during the cold season is advocated. During the subsequent years, mass vaccination of target population (above 5 months), creation of immune belts at the borders, targeted sero-surveillance will be the required strategies to be employed before the final push for eradication.

The main lesson from GREP is that understanding disease epidemiology is important factor for its eradication. Therefore, control programs, field data would be generated by its rigorous surveillance and its epidemiological understanding of the paste des petits ruminants, supported, and analysed risk and geographical distribution of the disease in small ruminants and large ruminant, also using mapping in geo-referenced mapping systems using GIS.

Production of a thermo-stable vaccine in the RP campaign was another important step. Economic and political is a current issue impetus to drive the eradication campaign to complete eradication of the paste des petits ruminants by 2030.

Results and Discussion

The continued spread of PPR has become an essential animal health concern in endemic countries despite the existence of an effective vaccine. Re-emergence of PPR through different lineages has been dis-closed in recent years with high losses of livestock, and the threat is now heightened for PPR-free countries at the border of the current endemic areas. Currently, the rapid spread of the disease in small ruminant populations is attributed to the highly contagious nature of the disease and a combination of factors, including exponential population growth, globalization of travel and exchanges, and lack of proper control. Such a dramatic extension of the disease has resulted in increased recognition by the international community and definition of needs and commitments for organizing global control and foreseeing, as for rinderpest, a possible eradication.

The possibility of eradicating PPR worldwide depends on a number of technical aspects. Coordination of the public and private players in animal health management is necessary, as is the execution of cooperative regional campaigns with ongoing international funding and commitment. Corollary activities such as evaluation of the effectiveness of vaccination and development of new vaccines are to be encouraged and funded to help sustain the gains achieved.

In addition, the key elements to better and efficiently control PPR should be based on a thorough understanding of the dynamics of the target disease. Thus, to meet the threat of PPR, a better understanding is needed of the underlying drivers of spread listed above and other epidemiological factors which influence its dynamics. Thus, it is essential to elucidate the role of evolutionary factors critical on the emergence, virulence, and adaptation to novel hosts. Success of control should rely on all innovations made in the field of virus identification and epidemiology investigation and modeling.

The integration of molecular epidemiology and quantitative knowledge is a key element to understand the bio-ecological processes and enable dynamic mapping of health risks. To this end, a greater resolution of sampling of host populations and of genome sequencing must be foreseen throughout the geographic range of the virus. This work will allow grasping the genetic diversity of PPRV and evolutionary forces acting on genomic diversity. This information as well as relevant epidemiological data related to sampled animals (e.g., relevant species and animal mobility) is needed to establish the diversity of strains in the field, trace the spatiotemporal origin of a virus, and estimate the basis of its introduction into the herd. The plasticity of the virus in samples of diseased hosts (goats, sheep, and camels) will also lead to better understanding of the epidemiology of PPRV circuits. This information in particular will assist in our understanding of the mechanism of replacement or extinction or of the coexistence of viruses in some areas and not in others to produce predictive models and how they may be influenced by factors such as vaccination, migration, and transhumance. The results of these studies will be interesting aids to support decision making in an optimized approach of PPR control.

PPR is an important animal viral disease of sheep and goats, which now threatens the billion-strong small ruminant population in Africa, the Middle and Near East, South-West and Central Asia. PPR is the one of the priority animal diseases whose control is considered important for poverty alleviation. But still PPR is a poorly recognized disease, particularly with regard to epidemiological features such as transmission dynamics under different production systems that is important to support control policy decisions. A great deal of more

research into this aspect of the disease is need of an hour. The fact that PPRV can infect cattle, buffaloes and camels gives PPR an even higher priority, particularly in the current situation where RP has been eradicated. Though, natural transmission of the virus occurs in other species of animals like cattle, buffaloes and camels, generally not affected with clinical form of disease but seroconvert.

The availability of an effective marker vaccine along with its companion serological tests will greatly assist in designing effective control and eradication programmes. Now the disease has been brought under control in goats and sheep by available effective and safe live attenuated cell culture PPR vaccines. The vaccines are effective in the face of natural outbreaks or experimental challenge and significantly reduce the mortality. The present scenario of PPR in Ethiopia the studies to be undertaken with the objective to know the exact epidemiology unit in every corner of the country to perform effective vaccination coverage for delineated area to control the circulation of virus within and between districts and potential risk factors to formulate modules for forecasting and forewarning. The epidemiology of PPR is likely to change due to vaccination as the disease occurs more severely in the naïve population only. More research is needed on the host-virus interaction through cellular receptor, immunological events including protective mechanisms, development of marker vaccine to differentiate between virulent and vaccine virus antibodies and also on development of thermo-stable vaccine. Analytical study about incidence of disease would be extremely useful and elicit widespread interest by providing sufficient additional information, especially in the epidemiology of the disease, which is important to support control policy decisions.

Conclusion

Although there is a project launched by the OIE and FAO developed jointly as the global strategy for the progressive control and eradication of PPR Globally by 2030, yet there is need to have comprehensive and timely coordination between national and regional (IGAD) level programs to combat this threat. Because PPR virus is highly contagious and the control and eradication strategy must equally implement within and between not only all IGAD member countries but also globally. This could only be achieved by the combined efforts of local and national authorities as well as political will; along with continuous support and strengthening by international agencies.

Currently Ethiopia using a targeted, epidemiology-driven approach achieved elimination strategy. But currently re-introduction of the infection of PPR virus to already vaccinated area is becoming a common challenge. This may mainly associated with failure in effective mapping of epidemiologic unit to be vaccinated after confirmation of the virus, failure in early reporting of disease outbreak to laboratory for early confirmation and intervention, time gap between vaccination/intervention and diagnosis of PPR virus, failure in 100% coverage of vaccination in area delineated, uncontrolled animal movement associated with search of feed and water, social interaction and culture, local animal marketing or animals easily moving within or between neighboring districts, uncontrolled animal movement along national or international bordering, failure in intensive and regular monitoring and evaluation system especially activities at ground, commitment among veterinarian experts(field and office staffs).

Focused approach (similar to RP) or Clearing ecosystem by ecosystem and preventing reintroduction may achieved successfully by involving all veterinary actors in the field, supporting the establishment of sustainable animal health delivery systems especially developing strong, fast and inclusive reporting systems and effective using of existing once like DOVAR and ADNIS, continuous updating understanding of PPR epidemiology (clearly mapping of areas in to endemic, high-risk and unknown), identifying reservoirs of infection, strengthened surveillance and diagnosis, enhanced use of thermo-stable vaccine for targeted vaccination areas, all the implementations should be regularly and intensively guided by Federal or Regional Government and the Branch Coordination Offices, but involving all actors: NGOs, CAHWs and private veterinarians in relevant areas. The commitment of public and private sector resources for implementation of the strategy will enhance its success. In particular, the allocation of public resources for key actions identified in the strategy will catalyze the implementation of the national strategies.

References

1. Abraham G (2005) Epidemiology of peste des petitd ruminant virus in Ethiopia and molecular studies on virulence. Trop Anim Health Prod 18: 1-176.
2. Abraham PL, Kenfe G, Barrett T (1994) PPR in Ethiopian goats. Trop Anim Health Prod 26: 69-73.
3. Abubakar M, Rajput ZI, Arshed MJ, Sarwar G, Ali Q (2011) Evidence of peste des petits ruminant's virus (PPRV) infection in Sindh Ibex (Capra aegagrus blythi) in Pakistan as confirmed by detection of antigen and antibody. Trop Anim Health Prod 43: 745-747.
4. Abu-Elzein EME, Housawi FMT, Bashareek Y, Gameel AA, Al Afaleq AI, et al. (2004) Severe PPR infection in gazelles kept under semi-free range conditions. J Vet Med B Infect Dis Vet Public Health 51: 68–71.
5. Anderson EC, Jago M, Mlengeya T, Timms C, Payne A, et al. (1990) A serological survey of rinderpest antibody in wildlife and sheep and goats in northern Tanzania. Epidemiol Infect 105: 203-214.
6. Anderson J, McKay J (1994) The detection of antibodies against Peste des petits ruminants virus in cattle, sheep and goats and the possible implications to rinderpest control programmes. Epidemiol Infect 112: 225–231.
7. Bailey D, Banyard AC, Dash P, Ozkul A, Barrett T (2005) Full genome sequence of PPR virus, a member of the *Morbillivirus* genus. Virus Res 110: 119-124.
8. Balamurugan V, Saravanan P, Sen A, Rajak KK, Bhanuprakash V, et al. (2011) Sero-epidemiological study of PPR in sheep and goats in India between 2003 and 2009. Rev Sci Tech 30: 889-896.
9. Banyard AC, Parida S, Batten C, Oura C, Kwiatek O, et al. (2010) Global distribution of Peste des petits ruminant's virus and prospects for improved diagnosis and control. J Gen Virol 91: 2885–2897.