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Peste Des Petits Ruminants (PPR): Global and Ethiopian Aspects. A Standard Review

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Abstract: *Peste des petits ruminants* (PPR) is acute, highly contagious, virulent and devastating animal disease of domestic and wild ruminants caused by a Morbillivirus, family paramyxovirus. It is antigenically very similar to the Rinderpest virus and other members of the genus Morbillivirus including: measles virus, phocine distemper virus, canine distemper virus and dolphin morbillivirus. PPR is wide spread in many Africa and Asian countries and currently it's the the global issue causing major economic losses in tropical and sub-tropical countries. PPR is highly fatal disease of mortality up to 100% in small ruminants particularly goats but also affects camel, cattle and pigs. Wild ruminants such as antelope, buffalo, gazelles, are susceptible too and blamed as potential source of infection for domestic animals. It transmitted by the aerosol route through sneezing and coughing inducing severe lesions in organ systems rich in lymphoid and epithelial tissues, sudden dullness, high fever and in appetence, congestion of mucosa leading to serous to mucopurulent discharges, bronchopneumonia, dyspnea, diarrhea and eventually death within 5–10 days. The necropsy findings are edematous lung with pus and severe consolidation, necrotic lesions throughout the gastro intestinal tract and soiling of hindquarters with bad smelling. PPRV is routinely diagnosed on the basis of case history, geographic location, clinical examination, gross pathology and histological findings but confimatory diagnosis is conventional reverse transcription polymerase chain reaction (RT-PCR). PPR has no treatment despite antibiotics to stop secondary bacterial complications and supportive treatments. So, control and prevention is vaccination animal movement across borders combined with proper disposal carcass, decontamination of fomites and proper quarantine method. In Ethioipa PPR is endemic and among the diseases that are entailing a huge economic loss from small ruminants through limiting international trade of animals and animal products. But prevalence of the disease in different parts of country is not yet investigated; inefficiency in early detection and lack of regular on time vaccination, dynamics of the disease is not adequately known and uncontrolled animal movement across the borders are major challenges in the country. Therefore, regular mass vaccination should be carried out, true prevalence and tempo-spatial pattern of the disease should purposely have studied to implement proper intervention measures in lining with illegal animal movement control.

Key words: Morbillivirus · Small Ruminants · Paramyxovirus · Goat Plague

INTRODUCTION

Peste des petits ruminants (PPR) is a wide spread, acute, highly contagious, virulent and devastating animal disease of domestic and wild ruminants caused by a Morbillivirus [1]. Its name derived from French for "disastrous disease of small ruminants" as it is fatal disease of sheep and particularly goats it also called 'goat plague'. PRR virus belongs to order Mononegavirales, family paramyxovirus, genus Morbillivirus [2]. It is antigenically very similar to the Rinderpest virus and other members of the genus Morbillivirus including measles virus, phocine distemper virus, canine distemper virus and dolphin morbillivirus [3]. Because of the strong clinical resemblance between rinderpest, it was suggested that PPR was caused by a variant of rinderpest virus that better adapted to small ruminants that has become less pathogenic to cattle but after different serological tests and cross protection studies, it was recognized definitively as different from RPV[4].

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PPR was first described in Ivory Coast, West Africa in 1942 and later spread to many Africa and Asian countries and currently it's the the global issue causing major economic losses in tropical and sub-tropical countries of the world [5]. PPR has single serotype virus with four distinct genetic lineages (I-IV) and closely related to rinderpest virus [6]. All four lineages of PPR virus were confirmed in Africa; lineage 1 and 2 viruses have been found exclusively in West Africa, Lineage 3 has been found in east Africa whilst linages III and IV are found in Asian [7]. PPR has high mortality rate exceeding 90% in immunologically naive populations [8, 9].

Domestic animals such as sheep and goats, camel, cattle and pigs can be affected by PPR with a various degrees of susceptibility [4]. Wild ruminants such as antelope, buffalo, hippotraginae, tragelaphinae, nigale, laristan sheep, dorcas gazelles, Nubian ibex and gemsbok are susceptible to PPR and potential source of infedtion for domestic animals [10]. PPRV is mainly transmitted by the aerosol route during close contact between animals mainly through sneezing and coughing [11]. PPRV is lymphotropic and epitheliotropic consequently; it induces the most severe lesions in organ systems rich in lymphoid and epithelial tissues [12]. The respiratory route is the likely portal to entry. Acute PPR first results in a sudden dullness of infected animals, with high fever and inappetence, after one or two days later, congestion of oral, ocular and nasal mucosae leads to serous discharges that later on become more abundant and mucopurulent, bronchopneumonia, revealed by productive cough and dyspnea and diarrhea usually appears 3 days after the oral lesions [13]. As a consequence of pneumonia and dehydration caused by diarrhea, severely affected animals may die within 5–10 days after the onset of clinical signs [8]. The necropsy findings of dead animal are edematous lung with pus and severe consolidation, necrotic lesions in the oral cavity and throughout the gastro intestinal tract and soiling of hindquarters with bad smelling [6]. PPRV is routinely diagnosed on the basis of case history, geographic location, clinical examination, gross pathology and histological findings but clinical signs and lesions can be misleading for PPR diagnosis since a number of diseases have similar out comes [14] However, conventional reverse transcription polymerase chain reaction (RT-PCR) is routinely used for virus detection [15]. PPR has no effective treatment beside use antibiotics to stop secondary bacterial complications and supportive treatments [16]. So, control and prevention of it highly rely on control of animal movement across borders combined with proper disposal carcass and the use of vaccine [17].

PPR was clinically suspected for the first time in Ethiopia in 1977 in a goat [18] and serological evidence reported in 1984 and later confirmed in 1991 with cDNA probe [19]. PPR is among the commonest of the diseases that affect small ruminants entailing a huge economic loss as it is listed trans- boundary diseases affecting the economy of the country through limiting international trade of animals and animal products [20]. Currently, PPR is endemic in Ethiopia and the National Veterinary Institute (NVI) produces live attenuated vaccine using PPR75/1 (LK6 Vero74) strain [21]. But prevalence of the disease in different parts of country is not yet investigated, 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, illegal animal movements, seasonal occurrence of the disease are great challenges to control this disease [22].Therefore, the objectives of this manuscript are to provide comprehensive document on this disease as a general and particularly in area with highest gap, Ethiopia at the same time to stimulate researchers in this potential area of research.

Historical Background of PPR: PPR was first described in Ivory Coast, West Africa in 1942 and subsequently spreaded to other regions. In the late 1970s sub-Saharan Africa, then the Middle East and Asia faced severe epidemics respectively [5, 23]. 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 [2]. 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 [23]. 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 [18, 24]. 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 [18]. 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 [19].

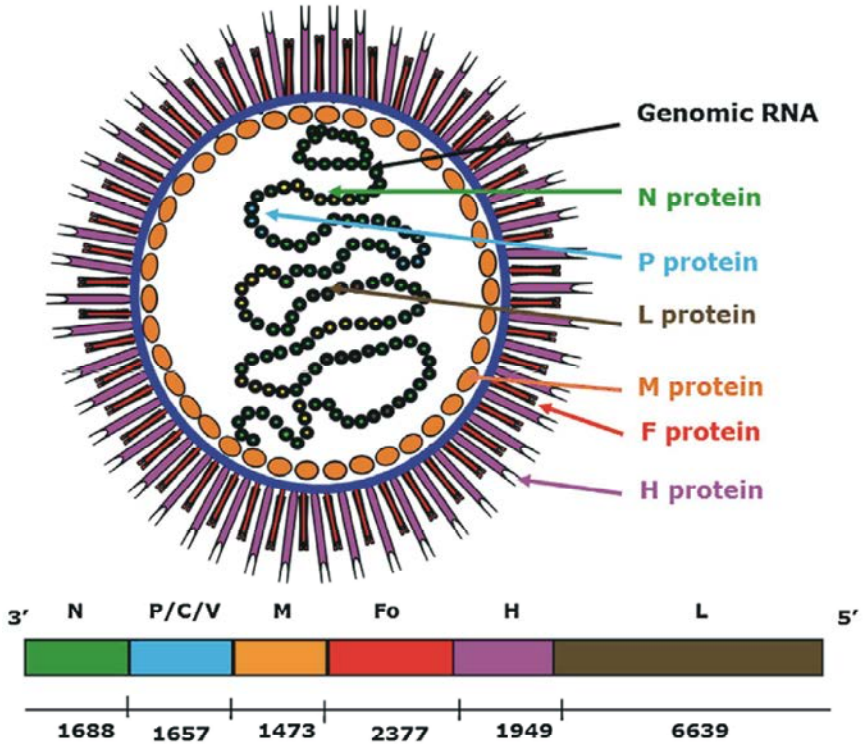


Fig. 1: Schematic representation of the PPR morbillivirus [1]

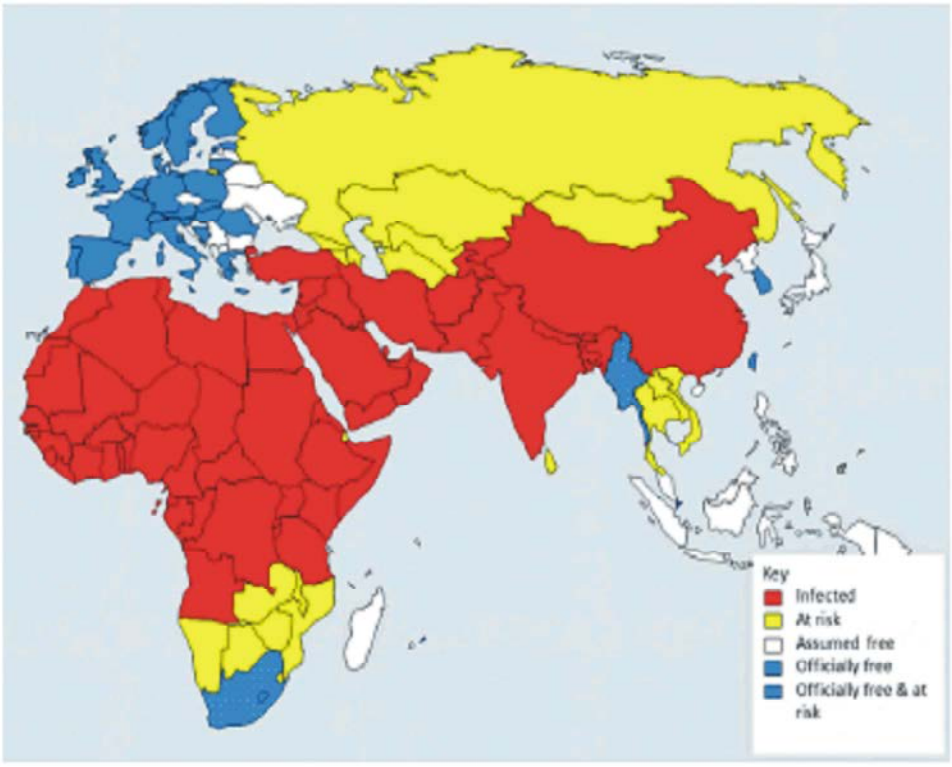
Nowadays, Because of its major economic importance, dramatic clinical incidences with high mortality rate and restrictions on animal and product movements, PPR is considered as a disease of major economic impact and has to be notified to the World Animal Health Organization (OIE) [1].

Morphology of PPRV: Peste des petits ruminants (PRR) virus belongs to order Mononegavirales, family paramyxovirus, genus Morbillivirus [25]. It is antigenically very similar to the Rinderpest virus and other members of the genus Morbillivirus including measles virus, phocine distemper virus, canine distemper virus and dolphin morbillivirus [3]. PPR virus is single stranded RNA, lymphotropic, non-segemented and enveloped pleomorphic, negative in polarity, with diameter range from 150 to 700 nm, with a mean of 500 nm [26]. Due to the presence of helicoidal nucleocapsid surrounded by lipoproteic envelope PPRV can be easily destroyed by means of lipid solvents and is very delicate, especially outside the host. The genome of nucleocapsid is surrounded by three viral proteins: nucleoprotein (N), phosphoprotein (P) and the large protein (L). Most importantly nucleoprotein (N protein) is the major viral protein wich plays an important role in inducing antiviral immunity. Currently, the great interest in this protein is the use of its cDNA as a potential specific diagnostic probe [1]. The viral envelop which derives from the host cell membrane are associated three viral proteins: the matrix protein (M) which is located inside the envelope and serves as a link between the nucleocapsid and the two external viral proteins, the fusion protein (F) and the hemagglutinin (H) which allows the virus to bind to the cell receptor during infection (Fig. 1). F and H are very important for the induction of protective host immune response against the virus however, N is the most abundant immunogenic among PPRV proteins, does not induce protective immunity against the virus rather it has been used in diagnostic tests [15].

Susceptible Animals and Risk Factors: Domestic animals such as sheep and goats, camel, cattle and pigs are susceptible to PPR in a variety of degrees [27]. Clinically PPR is seen in both sheep and goats however, goats are more susceptible than sheep. Breed of goats play important role in susceptibility as Guinean breeds specially, West African dwarf goats such as: Lagoon, Kirdi and Djallonké breeds are considerably more susceptible than the major Sahelian breeds [28]. Age is also important, with animals aged 3 to 18 months being more severely affected than adults or unweaned young. Furthermore, Climatic condition is also a major factor and

outbreaks are most frequent during the rainy season or the cold dry season. Wild ruminants such as antelope, buffalo, hippotraginae, tragelaphinae, nigale, laristan sheep, dorcas gazelles, Nubian ibex and gemsbok are susceptible to PPR and potential source of infedtion for domestic animals [29]. The existence of sylvatic reservoirs for PPRV has been reported with infections and deaths in captive wild ungulates from several species such as antelopes, emsbok, bharals, ibex impala and gazelles [30]. Its experimentally proved that white-tailed deer *(Odocoileus virginianus)* is susceptible to PPR [31]. Cattles are susceptible to infection but they are asymptomatic. PPR is now recognized as an emerging disease in camelids causing respiratory syndrome [32, 33]. In subtropical areas, the occurrence of the disease is more common during winter and rainy seasons [15, 26, 34]. In tropical countries confinement and restricted animals movement due to rainy seasons, may affect the nutritional status of the animals and hence predispose them to PPRV infection [24]. On another hand, PPR can also occur mostly during the cool, dry season in most endemic areas of Africa [35, 36].

Epidemiology: PPR is widely spreaded in the intertropical regions of Africa, Arabian Peninsula and Middle East and Asia [37]. 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 [36]. However, this does not necessarily mean that PPR originated in West Africa [38] 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 [39]. 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 [14]. 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 [40]. 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 [11]. Therefore, based on molecular epedimeology currently all four linages are found in Africa while linage III and IV found in Asian continent.



Map 1: Spatial distribution of peste des petits ruminants [41]

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 [42]. In Asia Lineage IV is more prominent along with localized Lineage III, PPR was first discovered in southern India in 1987 subsequently spread across the Arabian Peninsula, the Middle and Far East and several Asian countries like China, Iran and Pakistan Kazakhstan, Jordan and Saudi Arabia [43, 44].

In European PPRV is first reported inTurkey in 1996 and the potential for PPRV to spread across the rest of Europe became the concern of interest, moreover out break of PPR in Morocco increased the threat of movement of infected animals into southern Europe specially Spain because of exchanges exist between Morocco and Spain where both ovine and caprine populations are important [45].

Morbidity and Mortality: The morbidity and mortality rates of PPR can be up to 100% in severe outbreaks but in milder outbreaks, mortality rate may be reduced to 50% while morbidity rate still remains high in both cases [46]. Mortality rate is high in the susceptible young animals (4-8 months), animal with poor nutritional status, Stress and concurrent parasitic and bacterial infections also enhance the severity of the disease [47].

Transmission: PPRV is mainly transmitted by the aerosol route during close contact between animals mainly through sneezing and coughing [42]. The affected animals are important source of transmission during incubation periods, subclinical cases or before the onset of clinical signs [48]. Animals affected by PPR shed the virus in exhaled air, in secretions and excretions from natural orificies approximately 10 days after the onset of fever [25]. 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 [24]. 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 facilitate the transmission of this highly contagious disease [44].

Pathogenesis: PPRV is lymphotropic and epitheliotropic consequently; it induces the most severe lesions in organ systems rich in lymphoid and epithelial tissues [18]. According to Appel and summers, [49] the respiratory route is the likely portal to entry. After the entry of the virus through the respiratory tract system, it localizes first replicating in the pharyngeal and mandibular lymph nodes as well as tonsil. Viremia may develop 2-3 days after infection and 1-2 days before the first clinical sign appears. Subsequently viremia results in dissemination of the virus to spleen, bone marrow and mucosa of the gastro-intestinal tract and the respiratory system. Acute disease is usually accompanied by lymphopenia and immuno-suppression, leading to secondary opportunistic infections [50]. The virus can be isolated from nasal discharges from the day ninth of virus infection. PPRV then starts multiplying in the gastrointestinal tract, which leads to stomatitis and diarrhea [44]. Apoptosis of infected cells also seems to play an important role in the pathogenesis of PPRV in goats and sheep [51].

Clinical Signs: According to OIE [52], in acute cases of PPR sudden fever may observe that stay will for 5-8 days before the animal either dies or begins to recover. A clear nasal discharge that eventually becomes grey and sticky exudate with severe inflammation of the mucous membrane of the nose, causing respiratory distress is the characteristic sign of PPR. It also causes erosion of nasal and oral mucous membranes, severe oculonasal discharge and congestion of conjunctiva with matted eyelids, profuse non-hemorrhagic diarrhea, severe dehydration, progressive emaciation, difficult breathing and death within 5-10 days in affected animal. Bronchopneumonia with productive cough and dyspnea is common late in the disease while abortion may be seen in pregnant animals [53]There is a severe leucopenia which facilitates secondary bacterial infection especially complication with pulmonary infections caused by Pasteurella species are common in the later stages of the disease. The severity of the disease and prognosis in the individual is correlated with the extent of the mouth lesions as its good in cases where the lesions resolve within 2 to 3 days and poor when extensive necrosis and secondary bacterial infections result in a fetid odor from the animal's mouth. The prognosis of acute cases and cases with respiratory sign involvement are usually poor. Histopathologically PPR virus causes epithelial necrosis of the mucosa of the alimentary and respiratory tracts marked by the presence of eosinophilic intracytoplasmic and intranuclear inclusion bodies where as multinucleated giant cells (syncytia) can be observed in all affected epithelia as well as in the lymph nodes [54].

Post Mortem Lesions: The necropsy findings of dead animal are edematous lung with pus and severe consolidation, necrotic lesions in the oral cavity and

throughout the gastro intestinal tract and congested intestinal mucosa, eyes and nose will have a dirty white/grey discharge and soiling of hindquarters with watery feces usually possess bad smelling on dead animal body. The most severe lesions are seen in the large intestine, with congestion and "zebra stripes" of congestion on the mucosal folds of the posterior colon. Erosive lesions may also occur in the vulva and vaginal mucous membranes. There is congestion and enlargement of the spleen and lymph nodes [52].

Diagnosis: PPRV is routinely diagnosed on the basis of case history, geographic location, clinical examination, gross pathology and histological findings but clinical signs and lesions can be misleading for PPR diagnosis since a number of diseases including rinderpest, contagious caprine pleuropneumonia, bluetongue, Pasteurellosis, contagious ecthyma, foot and mouth disease, heartwater, coccidiosis, Nairobi sheep disease and mineral poisonings have similar out comes [52]. However, several rapid, specific and sensitive laboratory methods are available for confirmation. Conventional reverse transcription polymerase chain reaction (RT-PCR) is routinely used for virus detection due to its very high specificity and sensitivity. More recently, one-step real-time RT-PCRs have been developed and shown to be the most sensitive techniques for PPRV genome detection [55, 56]. ELISA technique is an accurate screening test for diagnosis of PPR [57]. Immunocapture ELISA (ICE) also can be used since it is rapid, specific and rather sensitive for PPRV antigen detection in sick animals [58]. Since the virus is circulating and excreted for approximately 10 days after the onset of fever, samples including blood, body fluids (Lachrymal and nasal discharges) and damaged organs and tissues, must be collected during the acute phase of the disease for PPR virus isolation. But PPR virus isolation is time consuming and the preservation of samples collected under field conditions is not always adequate for successful laboratory results. African green monkey kidney cells (Vero) have been for a long time the cells of choice for the isolation and propagation of PPRV, however, some isolates may not grow well in these cells and nowadays, transformed monkey cells expressing sheep/goat signaling lymphocytic activation molecules (SLAM or CD150), the virus cellular receptors, have been shown to possess increased sensitivity [59].

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 [16].

Prevention and Control: Control of PPR outbreaks routinely based on movement control combined with proper disposal carcass and the use of vaccine. Restriction on importation of sheep and goats from affected areas or newly introduced animal should be quarantined for three weeks. Additionally, 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 [52].

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. Sheep and goats vaccinated with an attenuated strain of PPR or that recover from PPR develop an active life-long immunity against the disease [60]. Several homologous PPR vaccines are available, being cell culture-attenuated strains of natural PPRV [61]. 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. There have also been three published reports on the preliminary results from recombinant capripox-based PPR vaccines that are able to protect against both capripox and PPR [62, 63, 64].

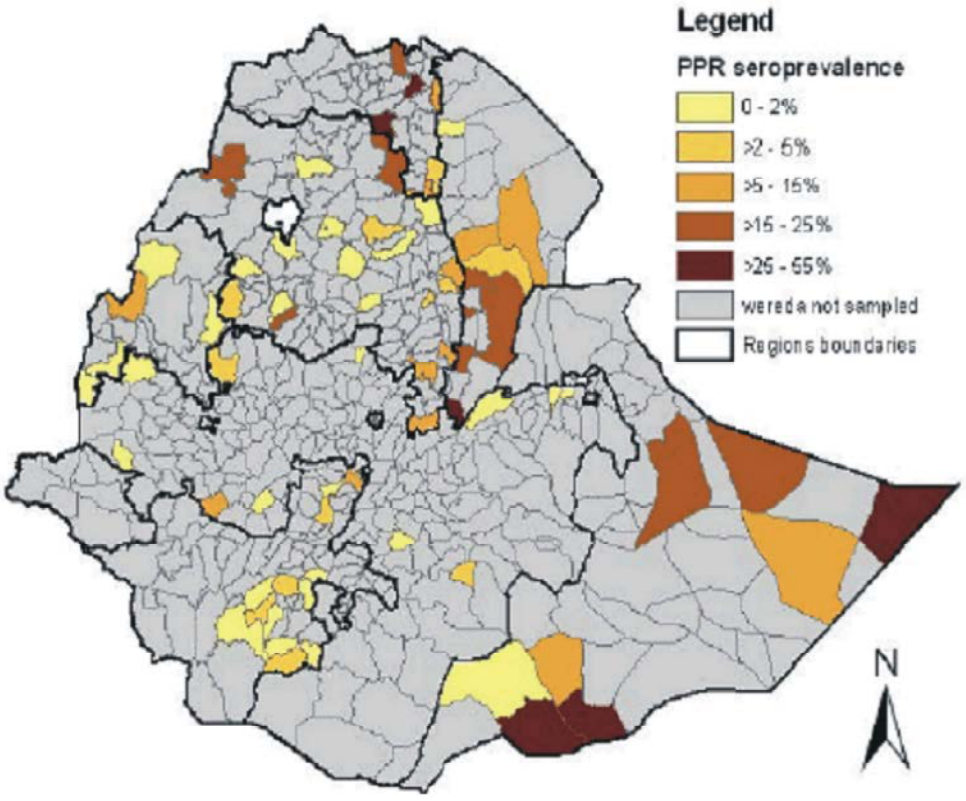
Apportunities and Challenges to Eradicate PPR: PPRV has been proposed as the next candidate after eradication of Rinder pest. Recovering animals always develop a strong life-long immunity clearing the virus [65]. Homologous live attenuated vaccine provides a life-long immunity after a single administration however; it has a low thermal stability with half-life of 2–6 h at 37°C after reconstitution [4]. According to Jones *et al*. [41] PPR vaccines currently in use are able to induce protective immunity against all known serotypes; immunity is lifelong, whether due to natural infection or vaccination; infection is transmitted primarily by direct contact and the virus does not persist in the environment; infected

animals are infectious for a short period of time and there is no carrier state; while a number of different wildlife ungulate species can be infected, there is no evidence to indicate that wildlife populations play an important role in virus maintenance; an effective, robust, safe and affordable vaccine is available; a thermo-stable vaccine has been developed; and sensitive and specific diagnostic tests are available.

According to Mariner *et al*. [66] considering the wide distribution of PPR and its multiple target host species which have an intense mobility, it will be a long process that cannot exclusively rely on mass vaccination. Goats and sheep are more numerous and reproduce rapidly than cattle, which creates greater challenge for the vaccination strategy. When compared to cattle value of sheep and goat per head is lower with an associated lower investment per head on health care, in spite of playing an important role in food security and livelihoods, utilising marginal grazing unsuitable for cattle or for crop production. Hence, transmission is mainly by direct contact movement control is effective but is difficult to implement in many of the infected countries where extensive and mobile production systems are common. As experienced from RP theoretically, mass annual vaccination is an effective control measure, but in practice is difficult to achieve and is costly.

Therefore, a more effective time-bound strategy is therefore required which will achieve eradication and avoid the need for long-term costly control programmes to repeat the achievement deserved on RP [41]. PPR specific epidemiological features and socio-economic cons iderations will also have to be taken into account and sustained international, coordinated and funded strategy based on a regional approach of PPR control will be the guarantee toward success.

Current Status of PPR in Ethiopia: It was in 1977 that PPR clinically suspected for the first time in Ethiopia in a goat herd in the Afar region, east of the country [67] and later confirmed in 1991 with cDNA probe in lymph nodes and spleen specimens collected from an outbreak in a holding near Addis Ababa [67]. In 1994 Roger and Bereket [67] (CIRAD-EMVT report n°96006, Montpellier, 1996) reported seroprevalences of up to 33% and 67% in sheep and goats respectevly. In 1996 Gelagay found that 14.6% of sheep sampled from Debre Berhan were seropositive [68].



Map 2: PPR seroprevalence of PPR in Ethiopia, [44]

Survey conducted to explore the spatial distribution and to investigate risk factors of PPR in Ethiopia by collecting 13 651 serum samples from 7 out of the 11 regions were analyzed by competitive enzyme-linked immunosorbent assay (cELISA) and reported overall of 6.4% Seroprevalence with very heterogeneous seroprevalence across regions and even more across *wereda*, with prevalence estimates ranging from 0% to 52.5%. But most of the samples were collected from the northern part of the country and particularly in the Amhara region (43.9% of total samples collected) which may contribute for variability between prevalence among region. This study concluded that PPRV circulation has been very heterogeneous; the values for the prevalence may reflect the endemic or epidemic presence of the virus or the various degrees of mixing of animals in the different areas and production systems. Age, sex and species appear as a risk factor for seropositive status, the linear effect seeming to confirm in the field that PPRV is highly immunogenic [69].

A Cross-Sectional Epidemiological Study Conducted from September 2006 to June: 2007 in Awash Fentale District, Afar, Ethiopia to investigate seroprevalence and post-vaccination sero-conversion rate using c-ELISA reported 1.70% prevalence that area. At the same time small ruminants in the area were vaccinated using the attenuated homologous PPR virus (Nigeria 75/1) strain vaccine, produced at National Veterinary Institute (NVI) in Debre-Zeit, Ethiopia resampled after fourteen days of vaccination. The post vaccination seroconversion rate in the population was found to be 61.13%, indicating relatively weak herd immunity. The main reason for the low sero-conversion could be the thermolabile nature of the vaccine, since no statistically significant difference was observed between small ruminants vaccinated by Veterinary Professionals and Community Animal Health Workers (CAHWs), using Chi-squared test at 95% CI ($P > 0.05$). This signifies the need for thermostable vaccine that could potentially increase the herd immunity in addition to that being administered by CAHWs independently. The current finding indicated that CAHWs could participate in vaccination campaigns in such areas as Afar, where there are few veterinarian's despite of the huge livestock populations, as means of pastoralists' livelihood [13].

It is also reported that PPR outbraek occurred in the pastoralist areas of Yabello *woreda* in September 2008 and later in Dire and Moyale *woredas* in March 2009. The disease has also been found to exist in Afar and Keyeryou pastoral areas in Ethiopia. However, these reports were not confirmed through laboratory tests. As a result, further diagnostic tests are being conducted through the Yabello regional and Sebeta federal veterinary diagnostic laboratories. As the Borana zone government is undertaking its annual vaccination of livestock against CBPP (Contagious Bovine Pleuro Pneumonia) with logistical support from CARE Ethiopia, PPR vaccination will also be included in *woredas* where outbreaks have been reported [70].

Megersa *et al*. [7] doing cross-sectional study reported 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 this study is much higher than the findings in previous studies carried out in the country; 6.8% by Abraham et al. [18] and 6.4% by Waret- Szkuta *et al*. [69]. This could be attributed to the nature of production systems, large flock size, uncontrolled animal movement and frequent contact between flocks. Mobile pastoral herds may often come into contact with local sheep and goats and facilitate contact transmission of PPRV from infected to susceptible animals. Likewise, infected migratory animals may.

PPR Control Measures and Challenges in Ethiopia: Yami and Merkel [71] claimed that, 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 [44]. 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 [47]. 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.

CONCLUSION

PPR is an important animal disease which now threatens the billion strong ruminant's population in Africa, the Middle and Near East, South West and Central Asia. PPR is a disease of animals, sheep and goats, which contribute significantly to the livelihoods of rural poor farmers, its control should therefore be considered in programs that aim at alleviating poverty in developing countries. In addition, as disease of public concern and thus its control should benefit from all international concerning organizations. In Ethiopia beside seasonal occurance of the disease illegal animal movement within and across the borders is a great hindrance for prevention and control of the disease; therefore, regular mass vaccination should be carried out, tempo-spatial pattern of the disease should purposely have studied to implement proper intervention measures in lining illegal animal movement control.

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