

Review on the Epidemiology of Peste Des Petits Ruminants (PPR) in Ethiopia

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Abstract: Peste des petits ruminants (PPR) are an acute, highly contagious, virulent and devastating animal disease of domestic and wild ruminants caused by a Morbillivirus, family of Paramyxoviridae. It is antigenically very similar to the Rinderpest virus. PPR is widespread in many Africa and Asian countries and currently it is 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 and gazelles are also susceptible and blamed as a potential source of infection for domestic animals. PPR has single serotype virus with four distinct genetic lineages (I-IV). The aerosol route mainly transmits PPRV during close contact between animals through sneezing and coughing. The clinical signs of the virus are sudden dullness, high fever, emaciation, inappetence, muco-purulent discharges, bronchopneumonia, dyspnea, diarrhea and eventually death within 5-10 days and causing respiratory distress is the characteristic sign of PPR. 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 confirmatory diagnosis is done by Conventional Reverse Transcription Polymerase Chain Reaction (RT-PCR). PPR has no treatment despite antibiotics to stop secondary bacterial complications and supportive treatments. Control and prevention of PPR outbreaks routinely is by vaccination of animal movement across borders combined with proper disposal carcass and proper quarantine method. In Ethiopia 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 well 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, early detection and on time vaccination should be carried out and animal movement from region to region should be controlled and quarantine should be established.

Key words: Epidemiology • Ethiopia • Peste Des Petits Ruminants • Small Ruminants

INTRODUCTION

Peste des petits ruminant (PPR) is a widespread, acute, highly contagious, virulent and devastating animal disease of domestic and wild ruminants caused by a virus belonging to the genus Morbillivirus of the family Paramyxoviridae [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’. 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 [2]. 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 have

become less pathogenic to cattle but after different serological tests and cross-protection studies, it was recognized definitively as different from RPV [3].

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 [4]. PPR has single serotype virus with four distinct genetic lineages (I-IV) and closely related to rinderpest virus [5]. 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 [6]. PPR has high mortality rate exceeding 90% in immunologically naive populations [7, 8]. Domestic animals such as sheep and goats, camel, cattle and pigs can be affected by PPR with a various degrees of susceptibility [3] 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 infection for domestic animals [9]. The aerosol route commonly transmits PPRV during close contact between animals mainly through sneezing and coughing [10]. 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 mucosa 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 [11]. 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 [7]. 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 [5].

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 [12]. However, conventional reverse transcription-polymerase chain reaction (RT-PCR) is routinely used for virus detection [13]. PPR has no effective treatment beside the use of antibiotics to stop secondary bacterial complications and support the treatments [14]. Therefore, control and prevention highly rely on control of animal movement across borders combined with proper disposal carcass and the use of vaccine [15].

PPR was clinically suspected for the first time in Ethiopia in 1977 in a goat [16] and serological evidence reported in 1984 and later confirmed in 1991 with cDNA probe [17]. 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 [18]. Currently, PPR is endemic in Ethiopia and the National Veterinary Institute (NVI) produces live attenuated vaccine using PPR75/1 (LK6 Vero74) strain [19]. However, prevalence of the disease in different parts of country is not yet investigated. This is due to lack of adequate information on the dynamics of the disease in the region and inefficiency in early detection, especially 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 of this disease [20].

Therefore, the objective of this paper is to review the prevalence /epidemiology of Peste des petits ruminants (PPR) in Ethiopia.

Historical Background of PPR: PPR was first described in Ivory Coast, West Africa in 1942 and subsequently spread to other regions. In the late 1970s sub-Saharan Africa, then the Middle East and Asia faced severe epidemics respectively [4, 21]. 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 1970s by using more sensitive laboratory techniques [22]. 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 ruminants and livestock keepers [21]. 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 [16, 23].

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 goatherd in the Afar region, in the east of the country [16]. 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 [17]. Nowadays, Because of its major economic

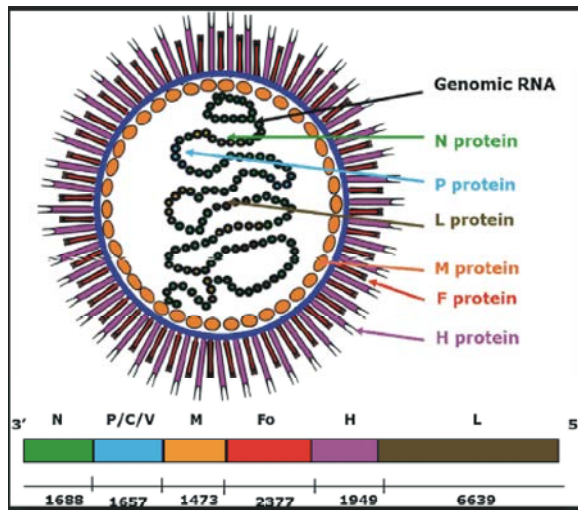


Fig. 1: Schematic representation of the PPR morbillivirus [1]

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].

Etiology: The etiological agent of Peste des petits ruminants virus (PPRV) has been classified under family Paramyxoviridae, Order Mononegavirales and Genus Morbillivirus. Similar to other morbilliviruses, PPRV is fragile and it cannot survive for long time outside the host. Like other members of the family Paramyxoviridae, *Peste des petits ruminants virus* is an enveloped, pleomorphic particle containing single stranded RNA, approximately 16 kb long with a negative polarity as a genome and it is composed of 15,948 nucleotides, the longest of all morbillivirus genomes sequenced so far [24]. The virus encodes six structural proteins; nucleoprotein (N), phosphoprotein (P), matrix Protein (M), fusion protein (F), hemagglutinin protein (H) and large polymerase protein (L) [25].

The matrix protein (M) which is located inside the envelope serves as a link between the nucleocapsid. By this position, M plays an important role in ensuring efficient incorporation of nucleocapsids into virions during the virus budding process. The haemagglutination allows the virus to bind to the cell receptor during the first step of the viral infection process. By their positions and their functions, both F and H are very important for the induction of protective host immune response against the virus [7]. However, N is the most abundant and the most

immunogenic among PPRV proteins, which does not induce protective immunity against the virus. It has been used in the development of diagnostic tests [26]. The large (L) protein is the enzymatic component of the viral transcriptase and replicase. The L proteins are multifunctional and, in addition to their polymerase activity, it has methylation, capping and polyadenylation activities [27].

Pathogenesis: PPR virus is like other morbilliviruses, it is lymphotropic and epitheliotropic consequently; it induces the most severe lesions in organ systems rich in lymphoid and epithelial tissues [16]. According to Appel and Summers, 1995 the respiratory route is the likely portal to entry. After the entry of the virus through the respiratory tract system, it localizes first by replicating in the pharyngeal and mandibular lymph 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 [28]. 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 [29]. Apoptosis of infected cells also seems to play an important role in the pathogenesis of PPRV in goats and sheep [30].

Clinical Signs: According to OIE [31] 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 of 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 [32].

The pictures shows oculonasal discharges and matting of eyelids (a) diarrhea soiling the perineum (b) submandibular edema (c) and sores and nodules on the gums and tongue (d).



Fig. 2: Clinical signs of PPR [33]



Fig. 3: Geographic distribution of PPRV lineages [40]

Epidemiological Situations

Geographical Distribution of the Disease: PPR is known to be present in a broad belt of sub-Saharan Africa, Arabia, the Middle East and Southern Asia. Major outbreaks in Turkey and India in recent years have indicated a marked rise in the global incidence of PPR [34-36]. The virus was isolated in Nigeria Taylor *et al.* [37], Sudan Taylor *et al.* [38], Saudi Arabia Abu Elzein *et al.* [39], India Nanda *et al.* [34] and Turkey Ozkul *et al.* [35]. Serological evidences were detected in Syria, Niger and Jordan, while the virus presence was confirmed with cDNA probe in Ethiopia [17].

Genetic relationship between PPR viruses isolated from different geographical regions was studied by sequence comparison of the F-protein gene. Four lineages

were revealed [36, 40]. (Fig. 3). Lineage 1 is represented by viruses isolated in Africa in 1970s (Nigeria/1975/1, Nigeria/1975/2, Nigeria/1975/3, Nigeria/1976/1 and Senegalese strain). Lineage 2 which includes viruses isolated in the late 1980s in West Africa (Ivory Coast and Guinea) is the only African lineage that did not cross the Red Sea to the Asian countries. Lineage 3 is a combination of isolates from Sudan (Meilig /1972) [41], Ethiopia [17]. Lineage 4 of PPR virus isolates, which includes the Asian isolates from Israel/1994, Iran/1994, Nepal/1995, Bangaldesch/1993 and India [36], is confined to Asia. Recently, it was reported in Turkey [35]. The presence of the two African lineages in Asia beside a distinct Asian lineage may be taken as indication of the trade route of spread of the disease.

Current Status of PPR by Species in Different Regions of Ethiopia: In Ethiopia, Clinical PPR was suspected in 1977 in afar region, east of the country [17, 42]. Clinical and serological evidence of its presence confirmed in 1991 in Addis Ababa [43, 44] has reported that 14.6% of sheep sampled along four roads from Debre Berhan to Addis Ababa were seropositive for PPR. [45] has also reported an overall seroprevalence of 1.7% in Oromia, 21.3% in Somalia, Amhara region of Ethiopia, [6] has also reported an overall seroprevalence of 30.9% from sheep and goat in pastoral and agro-pastoral area of afar and Gambella region of Ethiopia. More recently

(from 2012-2018), an overall seroprevalence record of 40.2% from sheep and goat in the pastoral community from Afar region of Ethiopia has been reported by Tolosa, Ragassa and Belihy [46]; 2.1% from sheep and goats in Four Districtsof Bench Maji and Kafa Zones of South West Ethiopia has been reported byMussie, Tesfu and Yilkal [47]; 41% from sheep and goats in some selected pastoralareas of Somali regional state of Ethiopia has been reported by Wondimagegn [48]. 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 [17].

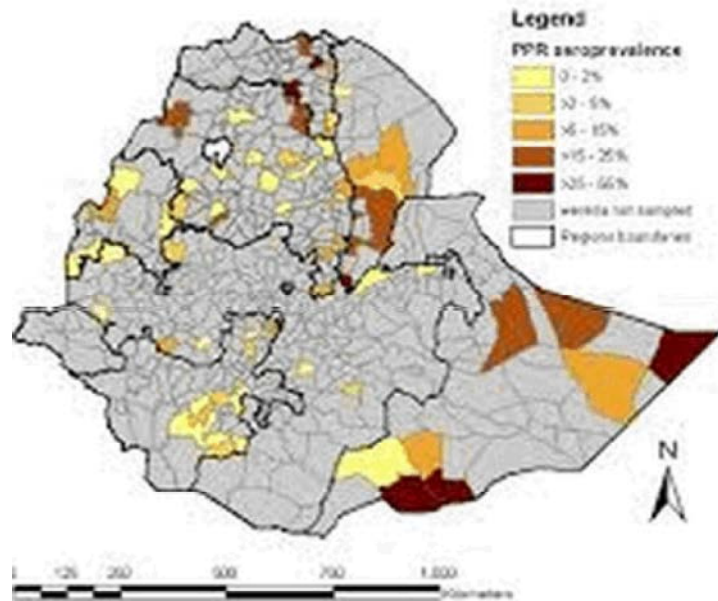


Fig. 4: Seroprevalence of PPR in Ethiopia [29]

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

Regions	Species	No. of tested	Prevalence (%)	Reference
Afar, Borena, East Shewa, Gambella, Jijja	Sheep	835(cELISA)		
	13	[16]		
	Goat	442(cELISA)	9	[16]
	Cattle	910(cELISA)	9	[16]
Afar (Awash Fentale)	Camel	628 (cELISA)	3	[16]
	Sheep and Goat	23 (cELISA)	36.6	[49]
Gambella (Itang)	Sheep and Goat	779 (cELISA)	27.3	[6]
Afar (Adaar)	Sheep and Goat	384 (cELISA)	38.3	[6]
Afar (Ab Ala)	Sheep and Goat	1653(cELISA)	15.3	[45]
Benishangul-Gumuz (Guba)	Sheep and Goat	729(cELISA)	8	[45]
Somali (Dolo Odo)	Sheep and Goat	465 (cELISA)	21.3	[45]
Oromia (E. Shewa & Arsi)	Sheep and Goat	700 (cELISA)	48.43	[50]
Tigray (Kukfto, Adigudem, Chercher and Maychew)	Goat	240 (cELISA)	47.5	[51]
Oromia (Adama)	Sheep and Goat	384 (cELISA)	30.2	[52]
Afar (Adar and Mille)	Sheep and Goat	229 (cELISA)	40.2	[53]
Somali (Afdher and Liben)	Sheep and Goat	798 (cELISA)	41	[48]
SNNPR (Siltie and Gurage)	Sheep and Goat	390 (cELISA)	29.2	[54]
SNNPR (BenchiMaji& Kafa)	Sheep and Goat	968(cELISA)	2.1	[47]

Table 2: Different studies of PPR in different hosts and in different regions of Ethiopia without specific zones or woreda

<i>Regions</i>	<i>Species</i>	<i>No. of tested</i>	<i>Prevalence (%)</i>	<i>Reference</i>
Amhara	Sheep and Goat	5992(cELISA)	4.6	[45]
Oromia	Sheep and Goat	2290 (cELISA)	1.7	[45]
SNNPR	Sheep and Goat	1622 (cELISA)	1.8	[45]
Tigray	Sheep and Goat	900 (cELISA)	15.3	[45]

Transmission: PPRV is mainly transmitted by the aerosol route during close contact between animals through sneezing and coughing [10]. The affected animals are important source of transmission during incubation periods, subclinical cases or before the onset of clinical signs [55]. Animals affected by PPR shed the virus in exhaled air, in secretions and excretions from natural orifices approximately 10 days after the onset of fever [56]. Spread through ingestion and conjunctival penetration, by licking of bedding, feed and water troughs are common. Furthermore, Infection may spread to offspring through the milk of an infected dam [23]. 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 [29].

Host Range: Peste des petits ruminants is a disease of sheep and goats. In general, goats are more susceptible than sheep; with sheep undergoing a milder form of the disease [57]. Other domestic animals such as camels, cattle and pigs are known to undergo subclinical infection of PPR [38]. The disease has been reported in wild small ruminants in a zoo and those living in the wild [58, 59, 60].

Host Determinants of the Disease: Host determinant factors of PPR spread have been reported in various studies, highlighting age, sex, breed and animal species [23]. Young animals are less likely to have developed protective antibody titers and therefore are more susceptible to PPRV [61]. This high susceptibility in the young has been reported in Ethiopia, Kenya, Pakistan, India and Turkey; thus, age of small ruminants is a key risk factor for susceptibility/resistance to the disease [35, 45, 62, 63]. In Oman, the disease is reported to maintain itself in susceptible yearling population, with an increase in incidence being a reflection of increased number of susceptible young goats/sheep recruited [64].

Sex has also been reported as a risk factor for susceptibility/resistance to the disease [45, 65, 66, 67]. The off-take of male small stock for social economic activities is higher and at an early age compared to

females which end up staying in the herds for longer periods for productive purposes females [63]. Therefore, females are more likely to demonstrate antibody titers than the males. The recruited young males, having been in the herds for a shorter period, are less likely to have been in contact with virus. Indeed, studies in Bangladesh have shown that male goats are significantly more prone to PPR than females [66]. However, studies from Pakistan have shown no significant difference between males and females, with respect to susceptibility [68].

Munir *et al.* [68] have also studied the influences of breeds of the small ruminants on susceptibility to the disease, with results showing that there are insignificant differences between goat breeds but there are significant differences between sheep breeds. Breed differences to susceptibility to PPR have been reported in other studies [57, 69]. Goat and sheep species differences have been highlighted as major risk factor for PPRV susceptibility [45, 67, 68]. Though PPR has been described in other species of animals, the camel is emerging as a key risk factor in long distance transmission of the disease particularly those used in trade caravans [70].

Social Ecology and Seasonality of the PPR Disease: It has been reported that the recent PPR disease outbreaks have been attributed to the cessation of rinderpest vaccination and loss of antibody cross protection between the PPR and rinderpest, leaving the small ruminants fully exposed to PPRV [70]. However, the spread of the PPR outbreaks has been for a long time associated with social, cultural and economic activities such as conflicts, disasters, livestock trade, cultural festivals and change of husbandry practices, nomadism and seasonal climatic and environmental changes [70, 71]. It has been reported that in Maghreb countries of North Africa, traditional sacrifices of sheep during major Islamic festivals provide a major opportunity for seasonal clustering of small ruminants of multiple sources whose health status is often unknown, thus creating a favorable environment for the transmission and dissemination of the PPR virus [72]. In the Sahel region, sero-prevalence of 75% is observed in pastoralist small ruminants and in most cases the disease is muted or subclinical [73].

Clinical PPR is more prevalent in the humid and sub humid regions of West Africa with morbidity of 80 to 90% resulting in mortality of about 50 to 80% [57]. These epidemics in West Africa, which coincide with wet rainy seasons, have been associated with seasonal animal husbandry patterns, livelihood activities among the settled and pastoralist communities [74]. However, Opasina and Putt [75] have reported PPR disease outbreaks in South west Nigeria during dry season, in different ecological zones.

In Sudan, PPR outbreaks in camels coincided with the seasonal movement of animals towards autumn green pasture [76], while other studies by Abdalla *etal.*[65] revealed significant association between prevalence of PPR and winter season. Seasonality of PPR in Ethiopia has been attributed to seasonal movement of small stock in search for water and pasture resources during dry seasons, social exchange of animals and livestock marketing which exhibit seasonal patterns with pick outbreaks being experienced in March-June and October-November [16, 45].

Potential Risk Factors of PPR: Kids over four months and under one year of age are most susceptible to the disease. Sahelian breeds of sheep and goats are believed to be more resistance than the dwarf breeds in the humid and sub-humid zones of West Africa. In a particular flock, risk of an outbreak is greatly increased when a new stock is introduced or when animals are returned unsold from livestock markets. Recovered animals have lifetime immunity [77].

Pattern of the Disease: 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 remains high in both cases [15]. Mortality rate is high in the susceptible young animals (4-8 months), animal with poor nutritional status, Stress and concurrent parasitic and bacterial infections enhance the severity of the disease [78]. There are considerable differences in the epidemiological pattern of the disease in the different ecological systems and geographical areas. In the humid Guinean zone where PPR occurs in an epizootic form, it may have dramatic consequences with morbidity of 80%-90% accompanied with mortality between 50 and 80% [57]. While in arid and semi-arid regions, PPR is seldomly fatal but usually occurs as a subclinical or inapparent infection opening the door for other infections such as Pasteurellosis [57]. Though outbreaks in West Africa coincide with the wet rainy

season, Opasina and Putt [75] observed outbreaks during the dry season in two different ecological zones. A high morbidity of 90% accompanied with 70% case fatality was reported from Saudi Arabia [39]. Serological data from Nigeria revealed that antibodies occur in all age groups from 4-24 months indicating a constant circulation of the virus [37].

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 [31].

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 ethyma, foot and mouth disease, heartwater, coccidiosis, Nairobi sheep disease and mineral poisonings have similar outcomes [31]. 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 [76, 79]. ELISA technique is an accurate screening test for diagnosis of PPR [80]. Immunocapture ELISA (ICE) also can be used since it is rapid, specific and rather sensitive for PPRV antigen detection in sick animals [81].

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 [82].

Differential Diagnosis: Other diseases cause diarrhea or pneumonia in sheep and goats may pose diagnostic challenge but a history of the recent introduction of new stock and the clinical and postmortem findings of stomatitis, typical for PPR. Laboratory tests are requiring ruling out rinderpest [77]. In addition to rinderpest, other conditions that should be considered in differential diagnosis include contagious caprine pleuropneumonia, bluetongue, pasteurellosis, contagious ecthyma, foot and mouth disease, heart water, coccidiosis and mineral poisoning [83].

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 [33].

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 [31].

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 [84]. Several homologous PPR vaccines are available, being cell culture-attenuated strains of natural PPRV [85]. 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 [86, 87].

Opportunities Presented Regarding 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: (i) there is only one serotype of PPRV and it is believed that perfect cross protection appears to exist within strains from different lineages. (ii) Vaccine is considered to provide life-long immunity. (iii) There is no carrier state. (iv) a close contact between the animals is required for effective transmission of the disease. (v) Virus does not survive for a long period outside the host as it is readily destroyed by heat and sunlight and hence needs continuous source of susceptible animals for survival. (vi) Appropriate diagnostic tools are available. However, unless the vaccine is used sufficiently, widely and thoroughly to stop transmission of the virus in the endemic areas, it may simply be wasting the public funds and at worst helping the virus to perpetuate [88].

Socio-economic Impact of PPR: *Peste des Petits Ruminants* virus has a widespread distribution spanning Africa and Asia [34, 36]. These areas encompass much of the developing world that relies heavily on subsistence farming to supply food or goods for trade and small ruminants provide an excellent supply of both. Unfortunately, in many areas of Asia and Africa, small ruminant production and therefore the livelihoods of poor farmers is threatening by PPR among other trans-boundary animal diseases (TADs). With its associated high morbidity and mortality, PPRV constitutes one of the major obstacles to subsistence farming [10]. The socio-economic significance of PPR is a result of heavy losses at production level and market effects along the value chain.

The socio-economic losses associated with PPR mainly result from the high mortality rate that is characteristic of the disease. This negatively affects income from production and value addition in small ruminants marketing chains. However, the direct economic losses caused by the disease are aggravated by the sanitary measures imposed by authorities to control animal movement and by trade restrictions on animal by-products [25]. 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 [20, 89].

An international study conducted by Perry *et al.*, (2002) [90] ranked PPR in the top ten diseases affecting small ruminants. The disease has also been ranked by pastoral communities as one of the top ten diseases of small ruminants [3]. It is estimated that 10% of the total impact of the disease is on trade and public expenditure and 90% on herd productivity for example one billion small ruminants or about 62.5% of global domestic small ruminant population is at risk of infection with PPR [71]. In Ethiopia, FAO estimated that losses associated with PPR reached an average of US\$ 375 per flock, with an average of 143 small ruminants per flock (an average loss of more than US\$ 2 per animal) [91]. However, there are very few economic studies related to the economic impact of the PPR and the data available on losses due to the disease is scanty [3, 23].

CONCLUSION AND RECOMMENDATIONS

Peste des petits ruminants (PPR) is an acute, highly contagious, virulent and devastating animal disease of domestic and wild ruminants caused by a Morbillivirus, family of Paramyxoviridae. It is antigenically very similar to the Rinderpest virus. The virus is highly fatal disease of mortality up to 100% in small ruminants particularly goats but also affects camel, cattle and pigs. PPR is an economically important animal disease, which now threatens the billion-strong ruminant population worldwide including Ethiopia; and it is one of the animal diseases whose control is considered important for poverty alleviation in countries like Ethiopia. In addition, as disease of public concern and thus its control should benefit from all international concerning organizations. In Ethiopia beside seasonal occurrence 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 with PPR homologous vaccine that is recommended by the OIE should be implemented in high-risk areas of the country.
- ▶ Tempo-spatial pattern of the disease should purposely studied to implement proper intervention measures in lining illegal animal movement control.
- ▶ Control programmes of PPR should be supported by field data generated by rigorous epidemiological surveillance, risk analysis and geo-referenced mapping systems using GIS.

- ▶ Animal movement from region to region should be controlled and quarantine should also be established.

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