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Review Article

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Review on Risk Factors and Socio Economic Effects of Peste des Petits Ruminants (PPR) in Small Ruminants

Berhanu Alemayehu Yebo
South West Ethiopia Regional State Bureau of Agriculture, Bonga, Ethiopia.
Corresponding Author Email: *berhanulmh@gmail.com*

Abstract

Peste des petits ruminants (PPR) are an important cause of mortality and production loss among sheep and goats in the developing world. Despite control efforts in a number of countries, it has continued to spread across the world and especially in Ethiopia, placing an increasing burden on the livelihoods of livestock keepers and on veterinary resources in affected countries. Given the similarities between PPR and Rinderpest, and the lessons learned from the successful global eradication of rinderpest, the suppression of PPR seems appealing, targeting both elimination of devastating animal disease and improving the livelihoods of the poor resource in developing countries. Risk factors associated with presence of PPR antibodies in small stock were species, age group, and vaccination status. Seasons, geographical locations and livestock management activities were also identified as risk factors to PPR disease outbreaks .Therefore, understanding social and cultural aspects of small stock management practices that could act as risk factors for PPR, and a solution to the epidemiology of PPR in pastoral and other areas. As PPR mortality rates are highly variable in different populations, conducting a sensitivity analysis of risk factors based on geographical specific mortality rate determination scenarios will indicate that investment in PPR eradication would be highly beneficial economically. Furthermore, removing one of the major constraints to small ruminant production would be of considerable benefit to many of the most vulnerable communities in Ethiopia.

Keywords: PPR, Risk factors, Sheep and goats, Socio-ecology.

1. Introduction

Ethiopia has one of the largest livestock populations in Africa providing support for the livelihoods of an estimated 80 percent of the rural poor. Animal rearing is an integral part of agricultural production and estimated livestock population approximates to 57.8 million cattle, 30 million sheep,23 million goats,10.2 million equines,1.2 million camels and 60 million poultry [1].

Peste-Des-Petits Ruminantis Virus (PPRV) is among important diseases affecting the productivity of small ruminants [2, 3]. Peste-Des-Petits Ruminantis is a highly contagious viral disease of domestic and wild small ruminants characterized by fever, anorexia, necrotic stomatitis, diarrhea, mucopurulent nasal and ocular discharges, enteritis and pneumonia [4]. Following infection via respiratory tract, which is main portal of entry, Peste-Des-Petits Ruminantis Virus replicates in oropharynx and mandibular

lymph node. The incubation period of PPRV is about 3 – 4 days prior to onset of clinical signs [5, 6]. Viremia may develop within 2-3 days and via blood it spreads to other organs and tissues like spleen, lungs, bone marrow, and mucosa of gastrointestinal tract [7].

Peste-Des-Petits Ruminantis was first reported from West African Ivory Coast in the early 1940's and later recognized as an endemic disease in West and Central Africa [8]. Afterwards the disease was reported in many other African countries including Sudan, Kenya, Uganda and Ethiopia [9, 10] (El Hag and Taylor, 1984; Wosu, 1994). In Ethiopia first PPR suspected case was encountered in 1977 following clinical observations consistent with infection with PPR (Pegram and Tereke, 1981), [11] and PPRV was later diagnosed in 1991 as the causative agent of disease in goats in the country [12].

In pastoral societies, where the livelihood survival strategies develop around the use and accumulation of animals, culture plays a particularly important role in livestock disease spread.

Some of the cultural activities, such as livestock raids and transfer of animals in marriage ceremonies, among other activities, increase probability of susceptible herds getting infection from incoming animals [13]. The world organization for animal health has identified PPR as a noticeable and economically important trans-boundary viral disease of sheep and goats, which is associated with high morbidity and mortality [14].

The PPR epidemics can cause mortality proportion of 50-80% in naive sheep and goat populations [5]. Based on assumption that goats experience an outbreak every 5 years [7]. It is an economically significant disease of small ruminants such as sheep and goats and cause an annual sum ranging from 2.47£ per goat at high loss and 0.36£ per goat at lowest loss. Effectively, the disease pushed the poorest families into destitution or near destitution and the wealthy families down one or two classes into poverty [2].

Therefore the objectives of this review paper are:-

- To review Peste Des Petit Ruminants (PPR) disease in small ruminants and its socio-economic impacts on livelihood of livestock keepers.

To review the type of small ruminant management system in livestock keepers that induces the occurrence of PPR outbreaks and spread in the community.

2. Review of Literature

21. Etiology of Peste des Petitis Ruminants (PPR)

The causative virus was first thought to be an aberrant strain of rinderpest virus that had lost its ability to infect cattle. Later molecular studies showed that it was distinct from, but closely related to, rinderpest virus. Peste-des-Petits Ruminantis is an important disease and it has also created problems because of its apparent similarity to rinderpest – the clinical signs of PPR closely resemble those of rinderpest, making differential diagnosis difficult. The history of disease backs to 1942 when first report of PPR came from Ivory Coast (West Africa). Investigators soon confirmed the existence of the disease in Nigeria, Senegal and Ghana. In 1972 in Sudan, a disease in goats that was originally diagnosed as rinderpest was confirmed to be PPR. During 1990's, PPR virus re-emerged [15].

Peste-des-Petits Ruminantis virus belongs to the genus *Morbillivirus* of the family *Paramyxoviridae* and is an enveloped, negative-sense, single-stranded RNA virus with a single serotype. The PPRV genome is 15,948 nucleotides in length, but longer variants were recently sequenced in outbreaks from China [16]. The virus exists as a single serotype but at the genetic level is divided into four distinct lineages (I-IV) based on the fusion (F) protein gene sequence [17]. It has a close antigenic relationship with other viruses of the same genus, including

Rinderpest Virus (RPV), measles virus (MV) and canine distemper viruses (CDV). These morbilliviruses have the propensity to cross species barriers, which highlights their potential towards inter-species transmission and novel host adaptation [18].

22. Transmission of The Virus

Transmission requires close contact between infected animals in the febrile stage and susceptible animals. The discharges from eyes, nose and mouth, as well as the loose faeces, contain large amounts of the virus. Fine infective droplets are released into the air from these secretions and excretions, particularly when affected animals cough and sneeze [19, 20]. Animals in close contact inhale the droplets and are likely to become infected. Although close contact is the most important way of transmitting the disease, it is suspected that infectious materials can also contaminate water and feed troughs and bedding, turning them into additional sources of infection. These particular hazards are, however, probably fairly short-term since the PPRV, like rinderpest, would not be expected to survive for long outside the host. Indirect transmission seems to be unlikely in view of the low resistance of the virus in the environment and its sensitivity to lipid solvent . There is no known carrier state for PPRV. Trade in small ruminants, at markets where animals from different sources are brought into close contact with one another, affords increased opportunities for PPR transmission, as does the development of intensive fattening units [21].

23. Host Range and Host Determinants of the Disease

Peste Des Petits Ruminantsis a disease of sheep and goats. In general goats are more susceptible than sheep; with sheep undergoing a milder form of the disease [21]. Other domestic animals such as camels, cattle and pigs are known to undergo subclinical infection of PPR [19]. The disease has been reported in wild small ruminants in a zoo and those living in the wild [22].

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 [24]. 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 [25]. Sex has also been reported as a risk factor for susceptibility/resistance to the disease . 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 [26]. 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. The influences of breeds of the small ruminants on susceptibility to the disease have also been studied by Munir *et al*, 2008, [27] 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 and Goat and sheep species differences have been highlighted as major risk factor for PPRV susceptibility [27]. 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 [28].

24. Geographic and Seasonal Distribution of PPR

It has been reported that the 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 [29]. However, the spread of the PPR outbreaks has for a long time been associated with social, cultural and economic activities such as conflicts, disasters, livestock trade, cultural festivals, and change of husbandry

practices, nomadism and seasonal climatic and environmental changes ([29, 30].

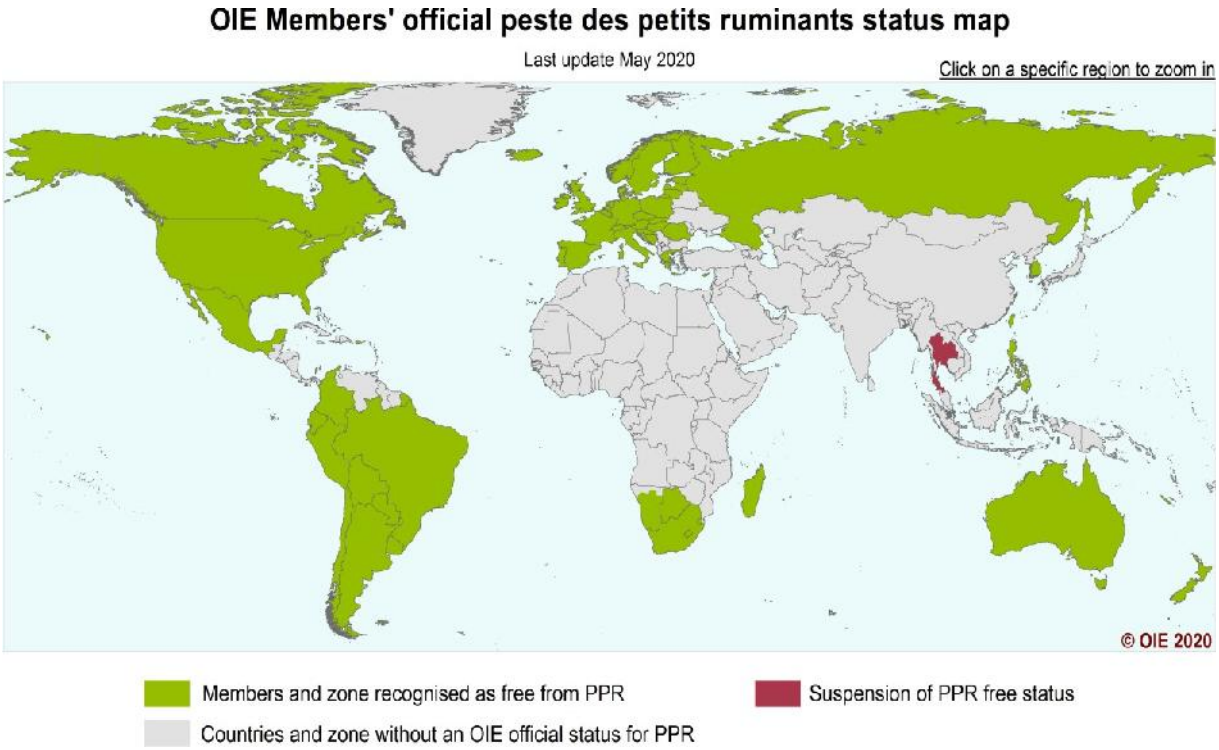

Figure 1. Official Status of PPR in OIE member countries. **(**Source: OIE, 2020) [31].

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 [12, 32]. The geographic distribution of the disease has increased rapidly in recent years, as it has in much of Eastern Africa, with the emergency of lineage IV during the 2010 outbreak in Ethiopia [23].

Small ruminants in Ethiopia mainly thrive on free-range pasture land, shrubs and forest cover. Due to the shrinkage in pasture land and forest area, these animals move to long distance in search of fodder and water during dry season. This phenomenon is common due to different summer and winter grazing grounds depending upon the altitude. PPR is transmitted through direct contact between infected animal and susceptible population [33].

During nomadism, animals come in contact with local sheep and goat population from where they pick up the infection or spread disease if nomadic flock is pre-exposed. Therefore, migratory flocks play an important role in transmission epidemiology of PPR. Movement of animals and introduction of newly purchased animals from the market also play an important role in transmission and maintenance of the virus. This could be one of the possible reasons for higher frequency of PPR outbreaks between March to June (Figure 4), which was also corresponded to lean period of kidding [34].

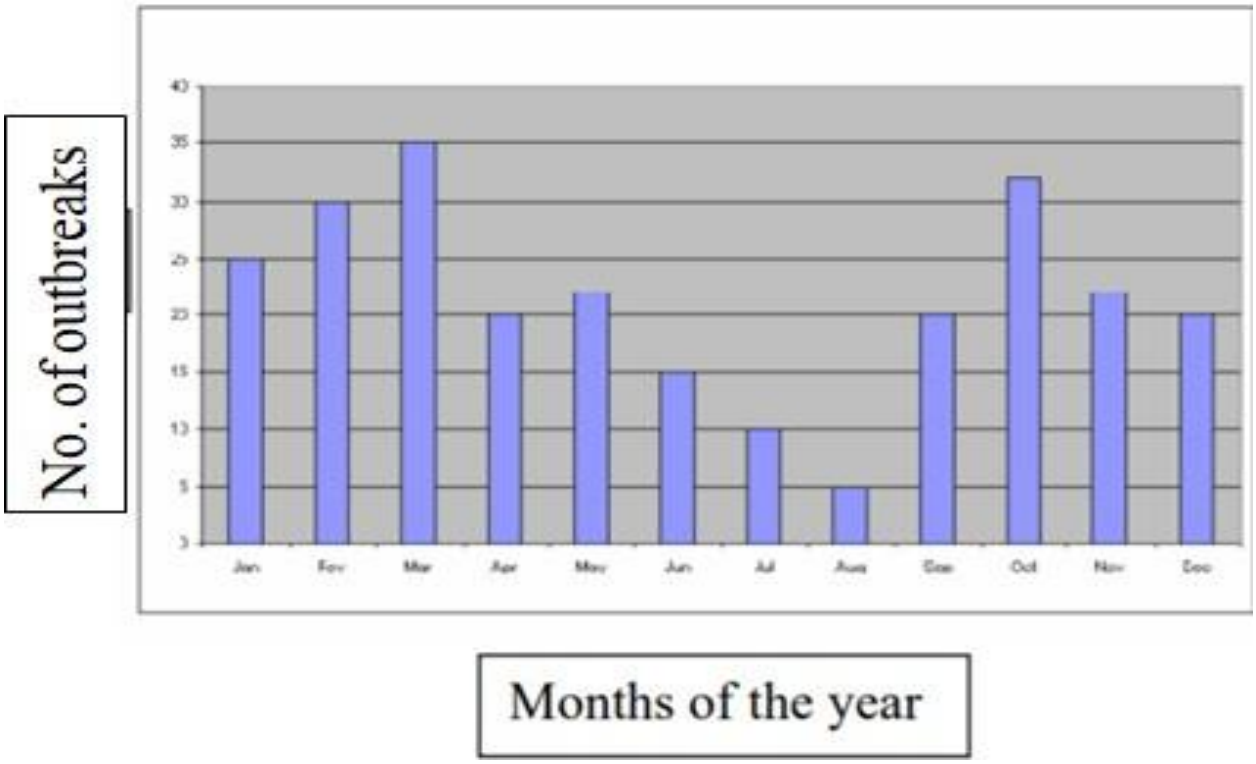

Figure 2. PPR seasonal disease pattern (Source: Abraham, 2005) [34].

Although seasonal occurrence of PPR virus outbreaks is disputed, disease transmission is certainly affected by animal movement for which socioeconomic factors and variations in agro climatic conditions are responsible. [35]. With the start of rains, the movement of animals is restricted due to the easy availability of local fodder. Nutritional status of the animals also gets improved during the rains. This may reduce disease transmission after the start of rains and during the period of easy availability of fodder. Similar observations were also recorded by Taylor, 1984 [19] in tropical humid zone of Southern Nigeria during a period of 5 years of observations [34].

25. Clinical Sign of the Disease

The virus is present in the secretions of infected animals. Close contact between animals enhance inhalation of droplets that are released into the water, feed, and bedding from the affected animals are the main sources of infection. However, the virus does not survive for a long time outside the body of a host animal [36]. Depending on any predisposing factors and the virulence of the infecting virus, clinical manifestation for PPR can be seen in per-acute, acute, subacute and sub- clinical forms. However, PPR in sheep and goats is generally observed as an acute disease. The per-acute form of disease is often seen in kids infected at the age of 4 months and older during the time frame whereupon any pre-existing maternal antibody levels wane. This per-acute form of disease has a short incubation period (2 days) with a rapid development of pyrexia with body temperature rising to 40-42°C. Depression, congestion of mucous membranes, oculo-nasal discharge, dyspnoea and profuse watery diarrhea lead to the death of infected animals within 4-5 days [23].

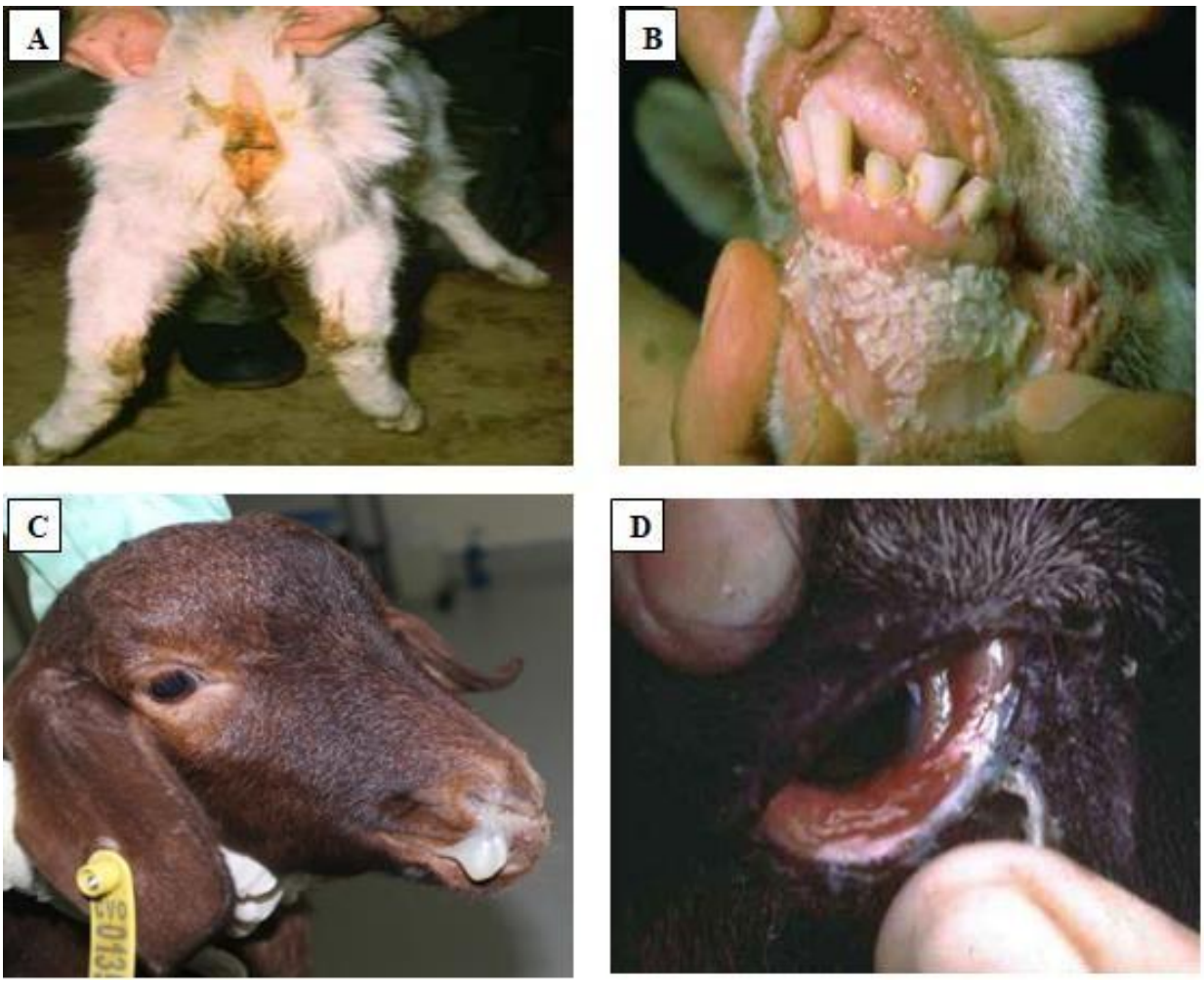

Figure 3. Clinical signs f PPR disease in sheep and goats.

A) PPR in a goat: signs of diarrhea (the hindquarters are soiled with liquid faeces). B) PPR in a goat: early mouth lesions showing areas of dead cells. C) Nasal discharge in sheep. D) Eye lesion. (Source: Bamouh *et al*., 2019; FAO and OIE, 2016) [37, 38].

In the acute form of disease, a 3 to 4 days incubation period precedes the development of pyrexia and the onset of other clinical disease signs, including watery oculo-nasal discharge, congestion of the mucous membranes of the buccal cavity, conjunctiva of the eye and the vulva. A diarrheic phase follows, often resulting in the generation of bloody faecal matter leading to dehydration and ultimately death of the animal. As the disease progress, the watery oculo-nasal discharge may become mucopurulent and can occlude the nostrils [26].

In the subacute form of disease, the animals do not develop severe clinical disease and low mortality rates are seen. With this form of infection, the animals may develop temperatures ranging from 39 to 40°C, but do not develop the characteristic clinical signs normally associated with PPRV infection. Animals usually recover from the disease within 10-14 days. A subclinical form of disease is also seen in large ruminants (buffalo and cattle), where the infected animals are able to clear virus in the complete absence of clinical disease, but seroconvert to PPRV, often generating strong neutralizing antibody responses [39].

26. Gross Pathology of PPR Disease

Many pathological characteristics are common at post-mortem examination following PPRV infection. Ulcerative to necrotic lesions are clearly evident throughout the buccal cavity in PPRV-infected sheep and goats at post-mortem [40]. The buccal papillae, dental pad, gum, dorsal surface of tongue, palatine tonsil and hard palate are mainly affected. Congestion of the digestive tract, particularly the duodenum, abomasum, ileum, caecum and colon, is often seen. Extensive congestion along the longitudinal folds of the caecum, colon and rectum may be evident as zebra striping. The ileo-cecal valve can also demonstrate extensive mucosal hemorrhage. In severe cases, hyperaemic, oedematous and ulcerative mucosa are also seen throughout the intestines [23].

The enlargement of lymph nodes accompanied with necrosis and hemorrhage, particularly the mesenteric lymph nodes and atrophied congested spleen are also sometimes seen in PPRV- infected goats in the field [41]. Similar to the buccal mucosa, the nasal mucosa becomes hyperaemic. The caudal part of the trachea and bronchus may contain froth. Pulmonary congestion and oedema with varying degree of red and grey consolidations can be seen with severe infections. Congestion of lungs and bronchopneumonia was also seen in PPR-infected West African dwarf goats and was associated with bacterial infection [42].

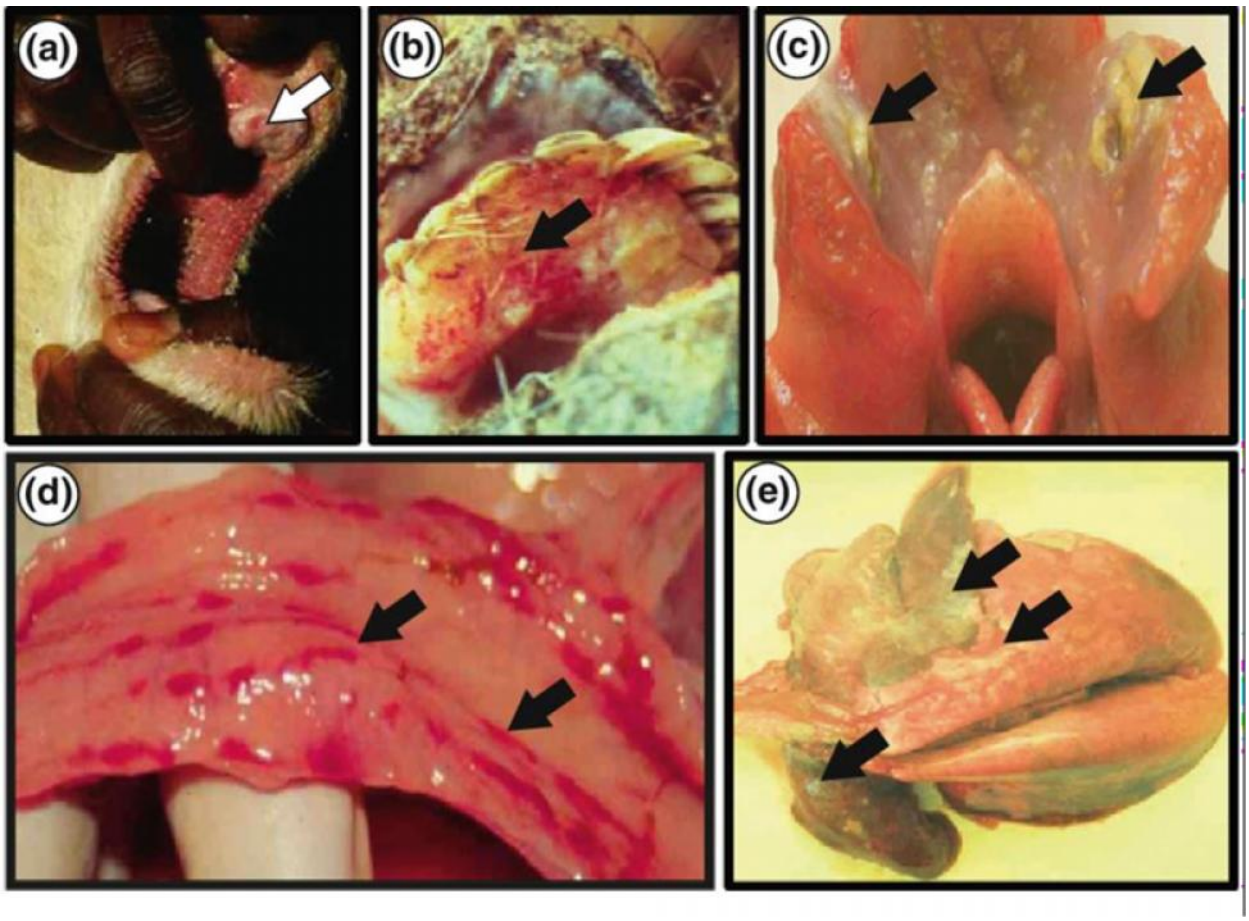

Figure 4. Gross pathological lesions at post-mortem in goats infected with PPR virus.
a) Necrotic lesions on dental pad; b) lesions on gum and oral cavity; c) oro-pharyngeal sections showing necrotic lesions on palatine tonsils and small fibrin deposits on the base of tongue; d) zebra striping in large intestine and e) consolidated lungs leading to pneumonia (Source: Parida *et al*., 2015) [43].

27. Diagnosis

27.1. Tentative diagnosis

Presumptive diagnosis of PPR is based on clinical signs presented in sick animals and postmortem lesions. Such diagnosis in PPRV endemic zones could play an important role in early warning in disease-symptomatic surveillance when coupled to digital diagnostic technology [44, 45, 46].

However, definitive laboratory diagnosis of PPR is the key to achieving accurate result because PPRV infections manifest similar clinical picture with other diseases such as bluetongue, contagious caprine pleuropneumonia, capripox and foot-and-mouth disease [47].

27.2. Competitive ELISA

Competitive ELISA (cELISA) is one of the most extensively used tests for serological screening and diagnosis of PPRV infected animals. The Principle of competitive ELISA assays are:-. (A) Coating the antigen on a polystyrene plate. (B) Addition of serum sample and monoclonal antibodies raised against the N protein of PPRV. (C) Addition of secondary antibodies labeled with enzyme. (D) Addition of substrate and reading the plate [29].

27.3. Virus isolation

Viral isolation using primary cells (bovine, ovine and caprine kidney and lung epithelial cells) requires multiple, sequential blind passages and takes up to weeks in culture before the development of any cytopathic effect [40]. Virus isolation is expensive and time consuming, thus, it cannot be deployed for routine diagnostic, but it can only be used as gold standard for further disease confirmation and in research purpose. Thus, establishment of cell lines with high infection efficiency to PPRV will be of help in confirming viable PPRV in the last phase of global PPRV eradication [48].

28. Prevention and Control of the Disease

In 2015, FAO and OIE launched a Global Control and Eradication Strategy for PPR which utilizes a progressive stepwise approach with four stages: 1) Assessment of epidemiological situation; 2) Implementation of control activities; 3) Eradication; 4) Post-eradication [38]. Control of PPR outbreaks relies on movement control (quarantine) combined with the use of focused ("ring") vaccination and prophylactic immunization in high-risk populations. Following a PPR outbreak, the disease is generally controlled through the subcutaneous administration of a live attenuated PPRV vaccine. Although the vaccine provides long-term immunity, generation of virus neutralizing antibodies does not occur rapidly enough to prevent rapid spread of the disease among in-contact animals [49].

Since 1972 OIE recommended the use of TCRPV for PPR prophylaxis in West Africa, which was continued for longtime. The vaccine was successfully used to control PPR in West African and other African countries. Considering the close antigenic relationship between RPV and PPRV, the live attenuated RP vaccine was tested in goats for vaccination against PPR and provided a protection for a period of 1 year. After the launch of rinderpest eradication programme, which stimulated the development of homologous PPR vaccine(s) by the world community. Hence the practice of heterologous PPR control was abolished in most countries. The first homologous PPR vaccine was developed using live attenuated Nigerian strain PPRV Nig 75/1 after 63 passages in Vero cells produced a solid immunity for 3 years [50]. During 1975, this virus was isolated from a dead PPRV infected goat in Nigeria [48]. Several vaccine trials had been conducted during 1989-1996 which demonstrated the efficacy of this vaccine in 98,000 sheep and goats in the field. The vaccine was safe under field conditions even for pregnant animals and induced immunity in 98 % of the vaccinated animals [50]. The vaccinated animals did not develop

any disease following challenge with virulent PPRV strains and thereby this vaccine was used worldwide (Africa, Middle East and Sothern Asia) for effective control of PPR. In a cross protection study, PPR vaccine was found to protect cattle effectively against RP [51].

Until recently, the most practical vaccination against PPR was made use of tissue culture rinderpest vaccine. Recently, a homologous PPR vaccine has been developed and the vaccine seed is available through the Pan African Veterinary Vaccine Centre (PANVAC) at Debre Zeit, Ethiopia, for Africa, or CIRAD-EMVT at Montpellier, France, for other areas. This vaccine of choice is becoming increasingly available. The vaccines can protect small ruminants against PPR for at least three years [52].

29. PPR Risk Factors Along the Small Ruminants Value Chain

The epidemiology of PPR in Eastern Africa is less clearly understood. The link between the disease pattern and factors that could influence the disease dynamics, including socio-cultural and economic factors such as nomadism, transhumance, livestock trade or livestock rustling, has yet to be fully established [53].
Peste Des Petits Ruminantsis disease outbreaks are associated with social, cultural and economic activities that promote host contacts such as livestock trade, cultural festivals, husbandry practices Ohta, 1982, [54] as well as nomadism, environmental and climatic factors [30, 55]. These factors are known to have seasonal variability. Generally it is thus becoming increasingly evident that human factors, more so the cultural activities, play a great role in the emergence and reemergence of the infectious animal diseases [53]. In pastoral societies where the survival strategies develop around the use and accumulation of animals, cultural activities play a particularly important role in livelihood sustainability. Some of the cultural activities such livestock raids and exchange of animals in marriage ceremonies among others activities, increases the probability of susceptible herds contacting infection from incoming infected animals [13]. Therefore, a better understanding of social and cultural aspects of small stock management practices thought to elevate risk of introduction and spread of PPR, become in large, a part of designing solutions to the social ecological challenges of PPR occurrence in pastoralists [6]. This would entail carrying out a PPR disease risk analysis that focuses on risk identification and risk assessment [56].

According to the 2015/16 CSA survey result, about 12.29 million sheep and 11.74 million goats were born during the reference period in the rural sedentary areas of Ethiopia while the estimated numbers of sold, slaughtered or, deaths of small ruminants were about 6.85 million sheep, and about 5.35 million goats [57].

A significant number of livestock are exported from Ethiopia through informal channels. Ministry of Agriculture data from 2002 to 2008 show that about 1.7 million live animals are informally exported each year, most of them being small ruminants [58] (Legesse and Fdiga, 2014). These animals are sourced from different regional states and cross borders informally to different neighboring countries. Most of these animals are informally exported to Somalia and smaller numbers to Djibouti, the Sudan and Kenya. The animals exported to Djibouti and Somalia are re- exported to different Middle East countries and thus a risk to small ruminant population because of the disease spread [59].

The general risk factors for the occurrence of PPR can be categorized as :-

i. Livestock rearing practices

Pastoralism: this can be expressed as animal movement through nomadism or seasonal transhumance, Cross-border pastoralism routes, Modification of usual herding routes to avoid areas afflicted by drought, insecurity or conflict, the sharing of pasture land and water points facilitates, frequent and repeated contact between

animals of unknown health status, Mixing of vulnerable (young) and high risk (sick adult) animals [60].

ii. Herd management

Large herds with a high density of animals which is often associated with intensive livestock farming, herds combining animals with different levels of viral susceptibility (such as goats mixed with sheep or small ruminants mixed with dromedarie, introduction of animals of unknown origin without health guarantees into a herd, unsold animals from market and trade reintroduced into a herd without observing any quarantine measures, Regrouping individuals of varying ages and origins destined for sale, Mixing of local sedentary herds with transhumant herds, Animals of different ages forced to live closely together, Accommodating animals in transit, Replacement rate of animals in a herd are the main herd health management shortfalls [61].

iii. Markets and trade

Gathering animals in live animal markets, Legal and illegal cross-border movements of animals, Imports and exports without health inspections, Increasing commercial trade between livestock rearing areas towards meat consuming areas to meet growing demand for animal protein and Trade routes are highly important risk factors [62].

iv. Social and cultural practices

Religious festivals giving rise to intense trade movements and the setting up of slaughter centers, Trading, loaning and giving animals that could be infected, Theft of animals, Risky livestock farmer behavior by moving animals in PPR areas to disease-free areas, Migration of rural populations in infected areas towards disease-free urban areas, Fleeing areas of socio-political or climate insecurity [63].

v. Environment

Some of the environmental risk factors affecting the occurrence of PPR includes variability of climate factors according to the season (temperature, humidity, wind), Agro-ecological zones (mountains, plains), Agro-pastoral zones with a high density of small ruminants, Agro-pastoral border zones [64].

vi. Health surveillance

Insufficient knowledge about the disease in disease-free areas and of some people keeping animals with insufficient health monitoring systems, difficult access to veterinary services, medicines, and vaccines, lack of trained health officers and veterinarians are among the factors which hamper the health surveillance and can constantly to the epidemiology of the disease [65].

2.10. Socio Economic Impact of PPR

The presence of disease can limit trade and export, import of new breeds and development of intensive livestock production, which in turn diminishes the consumption of animal protein in human. The turnover rate of small ruminant populations is much faster than that of larger livestock, so vaccination has to be used more intensively and more frequently [14]. But, if this can be achieved within a programme of progressive control, losses could be minimized and certain areas should be able to be freed from PPR. Cost benefit analysis is the most important criteria for launching any disease control programme. Epidemics of PPR have enormous consequences in terms of the dramatic effects this disease can bring about on livestock productivity and the high costs of control or eradication. Epidemics affect not only individual farmers but also the agricultural industry and as a consequence, the national economy [44].

The prevalence of such infectious and economically important animal diseases in Ethiopia excludes the country from profitable international markets thereby greatly reducing the country's foreign exchange earnings [66]. According to [67] FAO (2013), PPR can result in huge losses due to mortality in susceptible flocks from 10 to 100 percent and morbidity from 50 to 100 percent with significant economic, food security and livelihood impacts.

The 2015/6 CSA survey shows that the economic loss from small ruminant mortality due to diseases was estimated to be 4,989,677 Sheep and 5,582,924 goats in number. In spite of the fact that PPR and other disease outbreaks are under reported, due to the poor reporting system in Ethiopia, this figure says a lot about the impact of the diseases on the livelihood of the people in the country. PPR could be one of the causes for such huge losses [57].

Sheep and goats contribute a quarter of the domestic meat consumption, half of the domestic wool requirements, 40 percent of fresh skins and 92 percent of the value of semi-processed skin and hide export trade in Ethiopia [68].
It is estimated that 1,078,000 sheep and 1,128,000 goats are used in Ethiopia for domestic consumption annually. Moreover, according to the data obtained from the Ministry of Livestock and Fisheries during the last 3 years (2015-17) on average about 20,000 tons of red meat have been exported almost 99% to the gulf countries especially UAE and Saudi Arabia. Among the exported animal commodities 95% was goat meat, 4.5% sheep mutton and 0.5% [68].

According to the Ethiopian Leather Industry Development Institute (2011) [69] export earnings of the Ethiopian leather industry for the fiscal year 2010-11 rose above US$ 104.1 million. Many households depend on sheep and goat production to feed and educate their families, pay their immediate expenses etc. Women often have access and control over small ruminants making it an important resource of income for them.

However, when they lose their small ruminants, they fall out of livestock production leading to and turn to selling of firewood, grass and charcoal [70].

Conclusion and Recommendations

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

Seasons, geographical locations and livestock management activities were also identified as risk factors to PPR disease outbreaks. Risk factors associated with presence of PPR antibodies in small stock were species, age group, geographical administrative areas and vaccination status. Understanding this spatial-temporal heterogeneity of risk factors will greatly improve design of disease control measures against PPR. However, it cannot be generalized that risk factors in one year are similar in other similar seasons of other years considering livestock are in constant move in search of pasture and water. The disease has the potential of destroying livelihoods and reducing most herders to destitution consigning them into the ever growing internally displaced camps of economically challenged people.

Based on the above conclusion the following recommendations are forwarded:

- Appropriate data regarding the status of PPR are very essential if the eradication of this devastating disease is to be successful
- The key risk factors of PPR in small ruminant has to be known to provide a good basis for crafting a comprehensive PPR control strategy.
- Vaccination against PPR should be carried out regularly preferably on annual basis

to improve the herd immunity to levels that can prevent the spread of the PPR disease.

- Veterinary offices have to advice the local community on requisite measures to take to safeguard themselves against the negative impact of PPR.

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