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Epidemiology of Peste Des Petits Ruminants in sheep and goats in Ethiopia

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Sheep and goats disease is among the main constraints of small ruminant production in the country. The peste des petits ruminants disease is widespread viral diseases that affect sheep and goats kept in a different production system. This disease is a primary effect of sheep and goats. Peste des petits ruminants are caused by peste des petits ruminants virus. The virus is a member of the genus Morbillivirus. Pest des petits ruminants disease is an extremely contagious, severe and economically important disease in not only in domestic small ruminants but also wild small ruminants. The disease is characterized in affected animals by increased respiratory rates, the extension of the head and neck, dilation of the nostrils, protrusion of the tongue, abdominal breathing, and soft painful coughs. In post mortem, necroticor hemorrhagic enteritis is usually present and linear hemorrhages or zebra stripes may be located in the colon and caecum. The infection is transmitted by direct and indirect contact. The disease is influenced by age, breeds, flock size, introduced by new or market returned animal and climatic conditions. The peste des petits ruminants disease has an enormous economic impact on production varying from direct to indirect losses. Peste des petits ruminants virus has a significant economic impact due to its highly contagious nature of the disease. With proper use of vaccine and vaccination programs together with other measures like quarantine, adequate disposal of carcasses, and movement control was suggested.

Keywords: Small ruminant, Epidemiology, Peste des petits ruminants, Ethiopia

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INTRODUCTION

Peste des petits ruminant (PPR) is an acute, extremely contagious and economically important Tran's boundary viral disease of small ruminants which is categorized by OIE as a notifiable disease. The disease is characterized clinically by severe pyrexia, oculonasal discharge, necrotizing and erosive stomatitis, enteritis and pneumonia (Balamurugan et al., 2011).

Peste des petits ruminants are caused by peste des petits ruminants virus (PPRV). The virus is a member of the genus Morbillivirus in the family Paramyxoviridae. Virions are pleomorphic, varying between 130-390 nm in diameter. The virus envelope is 8-15nm thick with glycoprotein spikes of 8.5-14.5nm length being present throughout the membrane. PPRV has a non-segmented, single-strand RNA genome of 15,948 nucleotides that encodes eight proteins including six structural proteins namely the nucleoprotein (N), the phosphoprotein (P), the matrix protein (M), the fusion protein (F), the haemagglutinin protein (H) and the large polymerase protein (L), and two nonstructural proteins V and C; in the order 3´-N-P(C/V)-M-F-H-L-5´ (Bailey et al., 2005). Four genetic lineages (I-IV) and several viral strains have been identified. The viruses Lineage IV has become particularly widespread in recent years and the virus is

similar to rinderpest virus (CFSPCH, 2015).

Peste des petits ruminants virus is more prevalent in West and Central Africa, Arabia, the Middle East, and southern Asia. This area encompasses more of the developing countries that based on small ruminants for subsistence for a source of food or as export goods. Because of the high mortality and morbidity of PPR, this disease constitutes one of the foremost hindrances of subsistence small ruminant farming (Banyard et al., 2010).

Sheep and goats are vital livestock for supporting food security because of their high reproductive capacity; faster growth rates, greater environmental adaptability, and low initial investment, and hence have a unique niche in smallholder agriculture (Tibbo, 2006). There is an immense opportunity for increased livestock production in Ethiopia with a growing human population, urbanization, economic development, domestic and export markets. However, the prevalence of different diseases is found to be a major constraint of the sector (Biruk, 2014).

The name peste des petits ruminant (plague of small ruminants) reflects two things about this disease. First, it was initially described from Francophone West Africa and second, that it is a disease that kills a large number of sheep and goats. Many authors prefer the name "Ovine Rinderpest". But, official agencies such as FAO and OIE use the French name "Peste des Petits Ruminants" (Tewodros and Melese, 2012). Peste des petits ruminant 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 (Kula, 2016).

Peste des petits ruminant was first reported in Ethiopia in 1991 near Addis Ababa (Abraham, 2005). A study conducted by Kifle and Tsegaw (2008) at Metema district indicated that the prevalence of PPR was 26.3% (n=101). A serological investigation of PPR antibodies in the eastern Amhara region of Ethiopia revealed an overall prevalence of 28.1% (Biruk, 2014). Gelagay (1996) found in sheep a seroprevalence of 14.6% along roads from Debre Berhan to Addis Ababa.

Livestock keeping is the main source of livelihood for most pastoral households found in arid and semi-arid areas of East Africa. These areas are characterized by extreme climatic features of drought, flooding, low investments, fragile ecosystems and poverty levels approaching 65%. Major losses in sheep and goats in this region have been incurred from peste des petits ruminants (Gitao et al., 2010). Ethiopia has the largest sheep and goats populations in Africa with an estimated population of 28,892, 380 sheep and 29,704,958 goats (CSA, 2017). Livestock supply power for farming and transport. Livestock also supplies their owners with financial services, by providing a substitute for credit and by serving as a form of insurance. The contribution of livestock to the country's agricultural GDP is estimated to reach 47% (IGAD, 2013). The Ethiopia Livestock Master Plan (ELMP) calls for the establishment of a robust animal health information system reduced production losses by controlling prioritized animal diseases and increased export earnings by reinforcing the quarantine, inspection and certification system. Moreover, it addresses the decreased impact of zoonotic diseases on public health by controlling them and ensuring the safety of animal products, improved infrastructure, and addressing policy issues (ELMP, 2015). In Ethiopia, a risk-based (targeted) surveillance was conducted by NAHDIC during 2009 to 2011 in Afar, Oromia and Somali regions and the recorded prevalence was 60%(n=442), 59% (n=765) and 60%(n=465), respectively (FAO, 2012). An overall seroprevalence of 6.4% (n=13,651) was recorded during the 1999 national survey in seven regions of Ethiopia (Waret-Szkuta et al., 2008). This study shows PPR has been extensively circulating across Ethiopia. The introduction of PPR into a flock may be associated with several factors. Among these factors: mixing of different age groups of sheep or goats, the introduction of recently purchased animals into flocks can be mentioned. Other factors include a contact of a village flock with those that had been sent to market but returned unsold, change in weather conditions, contact with a trade or nomadic animals through shared grazing, change in husbandry practices and trading practices (Abraham, 2005). Peste des petits ruminant is the disease that causes highly economic losses in the small ruminants industry. The disease impend food security and livelihood of the people. Reducing the effect of PPR in endemic countries is thus common attention and could be considered a global public good (OIE and FAO, 2015).

Currently, the strategy of PPR vaccination is ring vaccination to control the spread of PPR infection to provide a barrier between infected and clean stock. The intervention is expected to contain the outbreak of the disease and reduce losses. The major challenge in control of PPR is the lack of adequate information on the dynamics of the disease and inefficiency in early detection. However, several diseases are widespread that hinder production and productivity. There is not enough data on the epidemiology of many diseases including PPR in Ethiopia. This disease is the most important disease of small ruminants that need epidemiological investigation in these areas. For control and prevention, the status of the disease should be known. The main objective of this paper was to review the epidemiology of peste des petits ruminants in sheep and goats in Ethiopia.

LITERATURE REVIEW

Etiology

Peste des petits ruminants (PPR) is caused by a virus (PPRV). It is classified under the order of

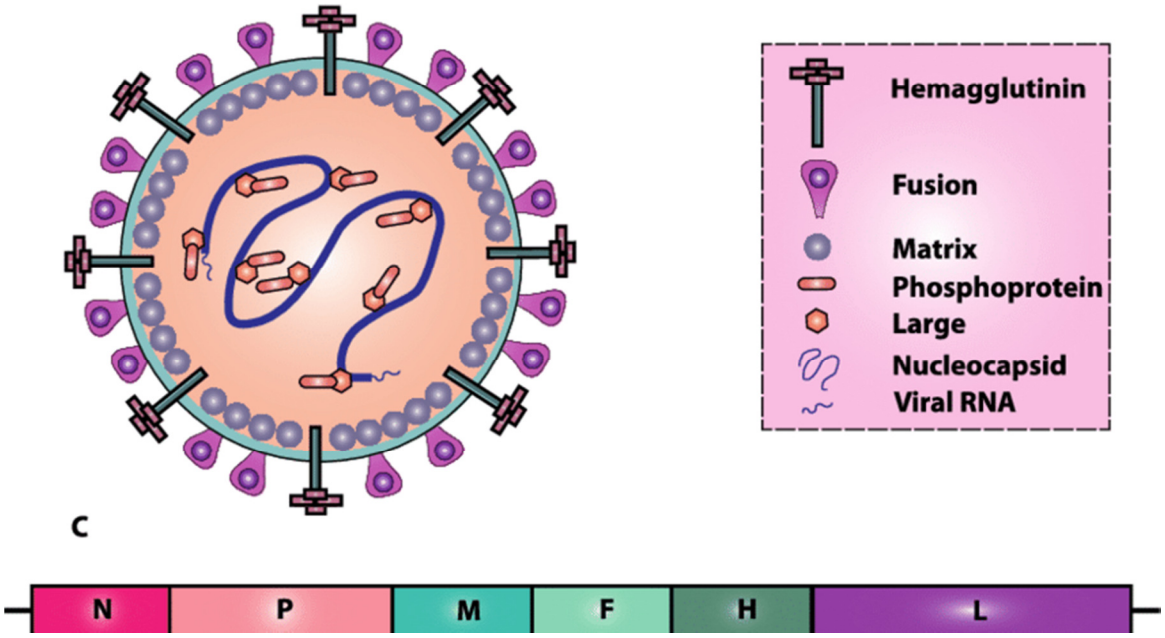

Figure 1.Schematic diagram of PPR virus, Source: Muhammad, 2015.

Mononegavirales, family Paramyxoviridae, subfamily Paramyxovirinae, and genus Morbillivirus. This virus is a genetic resemblance with Measles virus (MeV), Rinderpest virus (RPV), Canine Distemper Virus (CDV) and several other viruses that infect aquatic mammals.
The non-segmented genome encodes PPRV is nucleocapsid (N), phosphoprotein (P), matrix (M), fusion (F), haemagglutinin (H), large polymerase (L) and two non-structural proteins, C and V (Fig. 1) (Banyard et al., 2010).

PPR: The Disease

Peste des petits ruminants (PPR) is an extremely contagious, serious and economically significant disease not only in domestic small ruminants but also in wild small ruminants. The PPR is involved in the OIE list of notifiable terrestrial animal diseases due to its high morbidity and mortality (Muhammad, 2013). The disease is of emerging trans-boundary nature, which expanded from sub-Saharan Africa to the Middle East, Turkey, and the Indian subcontinent rapidly. Food and Agriculture Organization (FAO) has estimated that about 62.5% of the total small ruminant population is at risk of PPR, around the globe (Muhammad, 2015).

Epidemiological View

Geographical distribution

In the present, more than 70 countries were reported PPR disease to OIE. From those countries, most of them are African countries whereas some of them are from Asia and the Middle East. Currently, various countries are considered to be at risk for PPR disease (OIE and FAO, 2015).

The different PPR viruses (PPRV) that have been isolated so far in all these areas were classified into four lineages (I-IV) based on partial sequence analysis of the F gene. Lineage I is represented by viruses isolated in Africa in the 1970s. Lineage II 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 III is a combination of isolates from eastern Africa (Sudan and Ethiopia). Lineage IV of PPR virus isolates which includes the Asian isolates from Israel, Iran, Nepal, Bangladesh, Turkey, and India, is confined to Asia (Abraham, 2005).

Until 2000, lineage IV was limited to Asia countries and the Middle East. On the other hand, this lineage had recently been recognized in African countries (Sudan, Morocco, Tunisia, and Algeria). This condition, together with the first discovery of the disease in Uganda, Kenya, and Tanzania during 2006-2007, indicated a shift in disease dynamics on the continent (OIE, 2011). Recently, lineage IV has been identified in Ethiopia. The full genome sequence data of PPRV (Ethiopia/2010) clusters genetically with lineage IV isolates (Muniraju et al., 2014). The distribution of PPR has progressively expanded, covering large areas in Africa, the Middle East, and Asia as shown in the maps below (Fig. 2) (Sunelle, 2012).

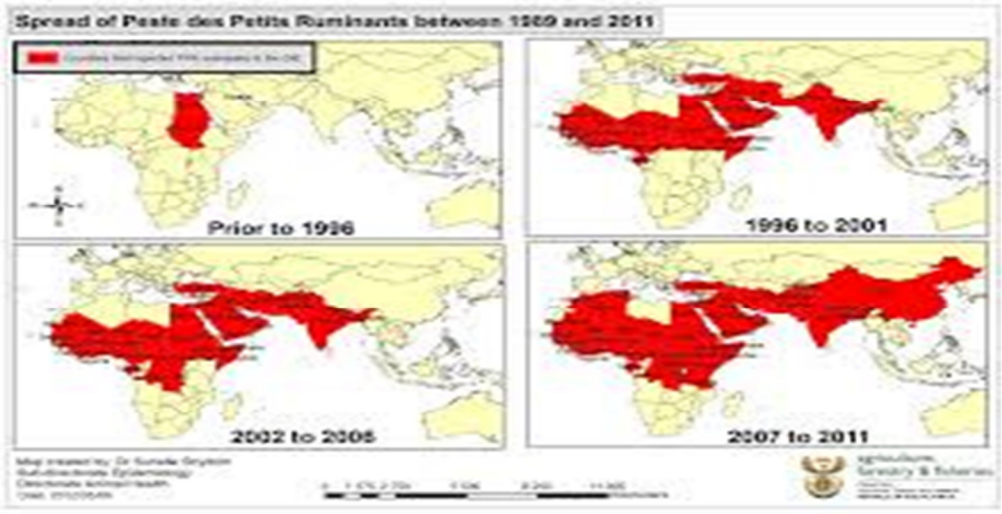

Figure 2. Distribution of PPR at different times

Host range and reservoirs

Peste des petits ruminants virus primarily infects sheep and goats, although both cattle and pigs are susceptible to infection, but do not contribute to the epidemiology as they are unable to excrete the virus (Abu-Elzein et al., 2004; Kinne et al., 2010). 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 of West African dwarf goats such as Lagoon, Kirdi, and Djallonke breeds are considerably more susceptible than the major Sahelian breeds (Abu-Elzein et al., 2004). Peste des petits ruminants are now recognized as an emerging disease in camelids causing respiratory syndrome in Sudan (Khalafalla et al., 2010). According to Abraham et al. (2005), a 3% antibody seroprevalence was recorded in camels in Ethiopia. Serological evidence of camel exposure to PPRV was confirmed in Tanzania with an overall seroprevalence of 2.6% (Swai et al., 2011). Pest des petits virus circulates in domestic ruminants and acts as a source of virus for wildlife (Hamed et al., 2016). Peste des petits ruminant was detected from different wild ruminants (Table 1).

Table 1. Detection of PPRV in Wildlife Species

Specie	Latin name
Laristan sheep	*Ovis gmelinilaristanica*
Gemsbok	*Oryx gazella*
Dorcas gazelles	*Gazella dorca*
Thompson's gazelle	*Eudorcas thomsonii*
Nubian Ibex	*Capra nubiana*
Indian buffalo	*Bubalus bubalus*
African Grey dukier	*Sylvicapra grimma*
Arabian oryx	*Oryx leukoryx*
Bubal hartebeests	*Alcelaphus buselaphus*
Buffaloes	*Syncerus caffer*
Defassa waterbuck	*Kobus defassa*
Kobs	*Kobus kob*
Arabian mountain gazelles	*Gazella gazellecora*
Springbuck	*Antidorcas marsupialis*
Arabian gazelles	*Gazella gazella*
Barbary sheep	*Ammotragus lervia*
Bushbucks	*Tragelaphus scriptus*
Impala	*Aepyceros melampus*
Rheem gazelles	*Gazella subguttoros amarica*
Afghan Markhor goat	*Capra falconeri*

Source: Banyard et al., 2010.

Transmission

Pest des petits virus is primarily transmitted through inhalation during close contact with infected animals. This virus can be shed during the incubation period and has been found in nasal and ocular secretions, saliva, urine and diarrheic feces (Radostits et al., 2007). The PPRV most likely transmitted through fomites like water, feed troughs, and bedding for a short time, however, infectious was not persisted for a long period. There is limited information on the persistence of the virus in the environment. However, the virus is inactivated by ultraviolet light and dryness within 3-4 days or less and usually persists for short periods in the carcasses. The PPRV may be inactivated with temperatures above 70°C and PH <5.6 or >9.6 (CFSCH, 2015).

Risk factors

Age is an important risk factor, with animals aged 3 to 18 months being more severely affected than adults or unweaned young (Kinne et al., 2010). Kids over 4 months and under 1 year of age are most susceptible to the disease. Sahelian breeds of sheep and goats are believed to be more resistant than the dwarf breeds in the humid and sub-humid zones of West Africa. In a particular flock, the risk of an outbreak is greatly increased when new stock is introduced or when animals are returned unsold from livestock markets. Recovered animals have lifetime immunity (Radostits et al., 2007). Climatic condition is also a risk factor and outbreaks are most frequent during the rainy season or the cold dry season (Kinne et al., 2010).

Pathogenesis and clinical signs

Pathogenesis

Peste des petits ruminants virus is transmitted mainly by aerosols between animals living in close contact (Abraham, 2005). Then, the virus enters the retropharyngeal mucosa, develops viremia and particularly damages the alimentary, respiratory and lymphoid systems. Infected cells undergo necrosis, and in the respiratory system, also proliferation. Death may occur from severe diarrhea and dehydration, before respiratory lesions become severe, or is hastened by concurrent diseases such as pneumonic pasteurellosis, coccidiosis or coliform enteritis (Radostits et al., 2007).

Clinical findings

Peste des petits ruminants have an incubation period of 2-10 days, followed by the sudden onset of pyrexia (40-42^{0}C) that could last for 3-5 days, severe depression, anorexia, and clear nasal-and ocular discharges that become mucopurulent because of secondary microorganism infection. Crusts could type on the nose, leading to the interference of the nostrils and respiratory distress, whereas matting along the eyelids may additionally result. One to 2 days following the onset of the symptom, the oral and ocular mucose membranes become congestion. This then is aiming to multifocal pin-point necrosis of the epithelial tissue of the gum, dental pad, palate, lips, inner aspects of the cheeks, and also the side of the tongue. These death areas extend and should even coalesce (Sunelle, 2012).

Diarrhea normally seems concerning 2 to 3 days once the onset of fever though, in early or gentle cases, it is going to not be obvious. The feces are at first soft then watery, malodourous and should contain blood streaks and items of dead gut tissue. Wherever diarrhea is not presenting sign, the insertion of a cotton swab into the rectum could reveal proof of soft feces which will be stained with blood (FAO Animal Health Manual No.5).

Affected animals have obvious signs of pneumonia, characterized by raised in respiratory rates, the extension of the head and neck, dilatation of the nostrils, protrusion of the tongue, rales, and soft painful coughs. A standard feature within the later stages of the infection is the formation of tiny nodular skin lesions on the skin of the lips and around the muzzle. Abortions could occur in pregnant animals. Death typically follows among 7-10 days from the onset of the clinical signs due to severe dehydration, emaciation, symptom, and hyperthermia. Some infected animals could recover when a drawn-out recovery (Sunelle, 2012).

Differential Diagnosis

Frequently, PPR is confused with other diseases that have grossly similar clinical signs. These diseases include rinderpest, foot and mouth disease (FMD), bluetongue, contagious ecthyma (Orf), pneumonic pasteurellosis, contagious caprine pleuropneumonia (CCPP), and gastro-intestinal helminths infestations. The most frequent sources of confusion are the mouth lesions, which could be due to rinderpest, FMD, bluetongue or orf; difficult breathing, which could be due to pneumonic pasteurellosis or CCPP or diarrhea which could be due to coccidiosis or gastro-intestinal helminths infestations (Dilli et al., 2011).

Diagnostic Methods

Clinical diagnosis

Clinical diagnosis of PPR in the field is based on symptoms such as pyrexia, lachrymation, nasal discharges, oral erosion, pneumonia, diarrhea, and death. Historic epidemiologic data of PPR within the region or farms will facilitate field personnel to report a

suspicious case. A differential clinical diagnosis ought to be created with different syndromic diseases. However, it is recommended to sample sick animals for a confirmatory diagnosis (Couacy-Hymann, 2013).

Post mortem diagnosis

Widespread erosive lesions occurred, that extending from the oral cavity to the reticulo-rumen junction. Besides, apical pneumonia, enlarge, edematous, congested lymph nodes, pleuritis and hydrothorax may be also present. The spleen is congested and enlarged, and necrotic lesions may be present. Necroticor hemorrhagic enteritis is usually present and linear hemorrhages or zebra stripes may be located in the colon and caecum (Sunelle, 2012).

Laboratory diagnosis

Conventional techniques

a) Virus isolation

This technique needs cell culture facilities which are not common in many laboratories in the developing countries. Where this is possible, primary cell culture from lamb or kid kidney and lung were used for the virus isolation along with different cell lines such as Vero cells, MDBK (Madin-Darby Bovine Kidney), marmoset-derived cell line (B95a). Recently, it has been developed a new and very sensitive cell line using the monkey cell expressing sheep-goat SLAM (Signaling Lymphocyte Activation Molecule) receptor. Usually, cultures are examined for the cytopathic effect in the days following infection of a monolayer with suspect material. The identity of the virus can be confirmed by virus neutralization or molecular techniques. Alternatively, specific antigens and antibodies can be detected (CouaCy-Hymann, 2013).

b) Antigen detection

Counter-Immuno-Electrophoresis (CIEP) and Agar Gel Immuno-Diffusion (AGID) tests using hyper-immune serum:

- ✓ Agar Gel Immuno-Diffusiontest is simple and can be performed in a basic laboratory but remains relatively insensitive. Moreover, it cannot distinguish PPRV from RPV.

Counter-Immuno-Electrophoresisis sensitive and specific and able to differentiate PPRV from the RPV sample (CouaCy-Hymann, 2013).Immunohistochemistry (IHC) on tissue samples:

- ✓ It allows the localization of specific PPRV antigens in a pathological tissue sample (CouaCy-Hymann, 2013).

c) Antibodies detection

Viral neutralization test (VNT): is applied to a serum sample; this technique needs also cell culture facilities (CouaCy-Hymann, 2013).

Molecular techniques

a) Antigen detection

Immuno-capture Enzyme-Linked Immuno-Sorbent Assay (IC-ELISA): It is a sensitive and specific method to detect the presence of PPRV antigens. It is easy to run and is well established in many laboratories in developing countries (CouaCy-Hymann, 2013).

b) Antibodies detection

Detection of antibodies against PPRV is carried out by using ELISA techniques. Currently, the use of competitive PPRV-specific anti-H (H-cELISA) or anti-N (N-cELISA) monoclonal based ELISA is routinely effective in laboratories where the disease exists. Both competitive ELISA tests can be used equally for the detection of PPRV antibodies (CouaCy-Hymann, 2013).

c) Genome detection

Real-time Polymerase Chain Reaction (RT-PCR) is an accurate, rapid and reliable method that can be used for the detection and also for the quantization of specific DNA molecules (Vinayagamurthy et al., 2012). The conventional RT-PCR has been developed for the specific amplification of the NP gene or the amplification of the fusion (F) gene and is established in various laboratories. The real-time PCR assay specific for PPRV and the loop-mediated isothermal amplification technique (LAMP-RT-PCR) is also available for the genome detection of PPRV (CouaCy-Hymann, 2013).

Treatment, Control, and Prevention

There is no treatment for PPR but it helps to give broad-spectrum antibiotics to stop secondary bacterial complications (Bharath et al., 2016). The controls of PPR require an effective vaccine and for this purpose, several vaccines including both homologous and recombinant vaccines have been developed (Abubakar et al., 2011). Nowadays, efficient live attenuated PPR vaccines are available that can induce lifelong protective immunity in vaccinated animals (OIE and FAO, 2015; Rebecca et al., 2015). The challenges in control activities arise it is not

possible to distinguish vaccinated animals from those that have recovered from natural infection. A differentiation of infected from vaccinated animals (DIVA) vaccine/test would improve epidemiological data by allowing tracking of infection in areas where there has been partial vaccination. Animals that have been infected are detected by the presence of antibodies to the N protein, while vaccination coverage can be assessed by the presence of antibodies to the H protein in the absence of antibodies to the N protein (Rebecca et al., 2015). Thermo-stabilizing PPR vaccine is well-suited for transport without icebox cold. Currently, the Pan African Vaccine Center (PANVAC) which is found in Debre Zeit, Ethiopia is producing and distributing effective PPR vaccines for Ethiopia and some African countries.

In general, the control of the disease is more effective by applying measures such as the slaughter of the infected herd, correct disinfection and adequate disposal of carcasses, movement control, emergency vaccination and quarantine (CouaCy-Hymann, 2013). Other preventive actions include public awareness creation, quick report, surveillance, and treatment of products and by-products (AUSVETPLAN, 1996).

Current Status of PPR in Ethiopia

In Ethiopia, several outbreaks have been reported for the federal ministry of agriculture at different times. Lineage III and IV are found in Ethiopia. Lineage III is circulating in East Africa countries such as Kenya, Sudan, Uganda and Tanzania (Banyard et al., 2010). In Ethiopia, 0% to 52.5% prevalence of PPR was reported from different parts of the country (Waret-Szkuta et al., 2008). Reasons for this may be related to different production systems with exchanges and movements in areas of lowlands being more frequent and involving larger numbers of animals.

Table 2. Prevalence of PPR in the Seven Surveyed Regions of Ethiopia

Region	Numbers of sample collection	Prevalence (%) (95%CI)
Afar	1653	15.3(13.6-17.0)
Amhara	5992	4.6(4.0-5.1)
Benishangul Gumuz	729	8.0(6.0-9.9)
Oromia	2290	1.7(1.2-2.2)
SNNPR	1622	1.8(1.1-2.4)
Somali	465	21.3(17.6-25.0)
Tigray	900	15.3(13.6-15.9)
Total	13,651	6.4(6.0-6.8)

Source: Waret-Szkuta et al., 2008.

According to Biruk (2014), the overall seroprevalence of PPR in sheep and goats in the Eastern Amhara region in the unvaccinated flock was 26.9% (n=133) and 28.6%(n=196), respectively. A cross-sectional seroprevalence study conducted in the southern region of Tigray revealed 47.5% prevalence in goats (Berihun et al., 2014). According to Abraham et al. (2005), PPR antibody seroprevalence in unvaccinated herd/flock was 3% (n=628) in camels, 9% (n=910) in cattle, 9% (n=442) in goats and 13% (n=835) in sheep in Ethiopia.

Ethiopia has launched a progressive PPR control strategy. The strategy will adopt geographic approaches. The initial area of operation will include at least the epidemiologically interconnected pastoral areas of the country, where a progressive control (ultimately leading to eradication) program will be implemented in strategically defined epidemiologically important (sub)-ecosystems. These areas include Somali and Afar regional states, pastoral districts of Oromia and South Omo zone of SNNPR. The strategy of the highland lowland interface was similar to that of the pastoral areas. It contains a district border with pastoral areas of the country through seasonal grazing and marketing (FAO, 2012).

The approach towards controlling PPR can be divided into three inter-dependent phases based on the epidemiology of the disease and prioritizing available resources. The first phase will establish a better understanding of the disease situation and implement disease control strategies that progressively controlled the PPR until the stage that there is evidence that the sub-ecosystem is clinically PPR disease-free. The second phase was started when the veterinary services stop vaccination in the sub-ecosystem and intensify clinical disease search/surveillance to verify the absence of clinical disease for about two years and simultaneously prevent reintroduction of PPR. The third phase will serologically verify the absence of circulating antibodies and by this the absence of possible mild disease circulating in all susceptible species (FAO, 2012).

Global PPR Control Strategy

In 2013, the OIE and FAO together decided to develop

a PPR global control and eradication strategy for a 5-year action plan (2013-2017). The task of eradicating PPR can benefit from a series of favorable elements. These include the experience gained from eradicating rinderpest (RP), several favorable technical aspects in terms of diagnosis and surveillance, effective and inexpensive vaccines that covers all known strains/lineages of the virus, no long-term virus carriers and no significant role of wildlife, a growing social and political commitment from various decision-makers at national, regional and global levels. This plan aims to control and eradication of PPR with other major diseases by reinforced veterinary service and global animal health systems, the improvement of animal health will reduce the impact of these diseases. This, in turn, strengthens the contribution made by the small ruminant sector to global food security and economic growth while at the same time improving the livelihoods of smallholders and poor farmers (OIE and FAO, 2015).

There are various tools to implement a global PPR control strategy. These include WAHIS (World Animal Health Information System), PPR monitoring and assessment tool (PMAT), post-vaccination evaluation (PVE), vaccines, surveillance, diagnosis, regional and international laboratory networks, OIE standards and the performance of veterinary services (PVS) pathway and others(OIE and FAO, 2015).

Economic Significance

Peste des petits ruminants are generally considered a major constraint for small ruminant production (Rossiter and Taylor, 1994; Nanda et al., 1996). This disease causes serious economic losses in small ruminants' production due to its extremely contagious nature of the disease. Likewise, PPR is common in areas that highly depend on subsistence farming of small ruminants for food supply and export (Muhammad, 2013). Collectively, Muhammad (2013) reviewed that PPR causes a loss of US$1.5 million annually in Nigeria, US$39 million in India and at least US$1.5 million in Iran. It was estimated that about US$15 million loss in Kenya (Thombare and Sinha, 2009).

CONCLUSION

Conclusively peste des petits ruminant is a serious viral disease that has a great economic impact throughout small ruminant production areas. This is recognized as an essential disease of sheep and goats worldwide. In Ethiopia peste des petits ruminant is the main constraint to sheep and goats production system. This disease is widely distributed in almost all parts of the country imposes great losses on the economic development of the country. Peste des petits ruminant disease is transmitted through direct and indirect contact. Peste des petits ruminants virus has a significant economic impact due to its highly contagious nature of the disease. This virus primarily infects sheep and goats. The disease is influenced by age, breeds, flock size, introduced by new or market returned animal and climatic conditions. An effective peste des petits ruminants disease prevention and control method should involve an effective vaccination program, adequate disposal of carcasses, movement control, emergency vaccination, and quarantine is vital a part of the peste des petits ruminant disease management.

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