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Review Article

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Review on Molecular Epidimiology of Peste Des Petits Ruminants Virus in Small Ruminants in Ethiopia

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Abstract

Peste des petits ruminants is caused by a peste des petitsruminants virus belonging to thegenus *Morbillivirus*and the family *Paramyxoviridae*.It is an acute, highly contagious and fatal disease mainly affecting sheep andgoats, whereas cattle, camels and buffaloes suffer sub-clinical infections.Themorbidity and mortality rates of the disease ishigh in small ruminants. Despite the use of an effective and cheap live-attenuated vaccine, the virus has alarmingly spreading to disease-free countries.The disease is common in much of Africa, Middle East and Asia. Based on sequence analysis of the nucleoprotein and fusion gene, the virus has four lineages. In Ethiopia, the virus was detected in 1994, and subsequently, the isolate reported in 1996 wasgenetically classified to cluster in lineage III. However, recent reports indicated that lineage IV is circulating and continued to spread in Ethiopia. Molecular characterizations of circulating strains are thus important tool to understand the epidemiology of the virus and track outbreaks in the country. Such information contributes to establishing the diversity and circulation of strains in the field, trace the spatiotemporal origin of a virus, and estimate the risk of its introduction into the herd.The objective of this seminar is to review the recent advancement in the epidemiology of peste des petits ruminants in small ruminants.

Keywords: Ethiopia,epidemiology, Peste des petits ruminants, Small ruminants

1. Introduction

Peste des Petits Ruminants (PPR) is an acute, highly contagious, trans-boundary and commonly fatal disease of sheep and goats caused by peste des petits ruminant virus (PPRV), classifiedin the order*Mononegavirales*, family *Paramyxoviridae*, subfamily *Paramyxovirinae*, and genus *Morbillivirus*(1,2). The virus can also infect camels, cattle, and buffaloes,however; their role in the transmission remains ambiguous(3,4).

The virus has four genetically distinct lineages (I, II, III & IV) (5). Lineage I and IIare commonly found in Western Africa, while lineage III is circulating in Eastern Africa and the Middle East, and lineage IV is widely distributed in Asia and parts of the Middle East (6,7,8).

The genome of this virus consists of a single-strandednegative-sense RNA that contains15,948 nucleotides and encodes six structural proteins, 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 non-structural proteins, V and C where the gene order is 3′-N-P(C/V)-M-F-H-L-5′ (6,7).

Depending on the degree of predisposing factors and the virulence of the virus, the severity can be classified as peracute, acute, sub-acute, and subclinical where the acute form is the most common one (9). The virus has been regarded as the main threat and risk of infection in small ruminants in Africa, Asia and even worldwide (3). It is transmitted through close contact between infected and susceptible animals in which common signs of the infections are high fever, ocular andnasal discharges, erosive lesions on different mucous membranes, mainly in the mouth, diarrhoea, and respiratory distress (10). Symptoms are similar to those of other diseases such as rinderpest,foot-and-mouthdisease,pasteurellosis,contagious ecthyma,contagiouscaprinepleuropneumonia(CCPP),coccidiosis and bluetongue (11).

Peste des Petits Ruminants first discovered in the early 1942s in Côte d'Ivoireandprogressively expanded its geographical distribution beyond its original endemic region (12).Dramatic geographical spreadof the disease has occurred in large parts of Central Asia, South Asia, and East Asia (13).Currently, it is endemic in Asia and Africa; however, it has crossed into the territories of 70 countries in the world (14).

Diagnosis of Peste des Petits Ruminants is critical in implementing control measures, to restrain outbreaks and minimize economic losses. However, the signs of the disease are often difficult to distinguish from a number of other diseases. This situation becomes more difficult when these diseases are circulating in areas where PPR is endemic. So, clinical signs and lesions can be misleading for PPR diagnosis and it is obligatory to confirm the clinical diagnosis through laboratory testing (15).

In Ethiopia, PPRVwas first suspected in 1977 in a goatherd in the Afar region, in the East of the country based on clinical evidences (16). The virus was detected in 1994, and later the isolate reported in 1996 was genetically determined to cluster in lineage III (11). Lineage IV has been reported from a disease outbreak in Ethiopia in 2010 (17).

The epidemiology of the disease is poorly understood and therefore mainly based on assumptions derived from rinderpest; the epidemiological lineageand spread of the PPRV strains are not well specified. There is also continuing occurrence of PPRV in small ruminants in Ethiopia that require research in molecular characterization of the spreading virus strains and advanced phylogenetic analysis. Therefore,theobjective of this seminar paper is to review epidemiologyof PPRV in small ruminants in Ethiopia.

2. LITERATURE REVIEW

2.1 Biological Properties

Peste des Petits Ruminants (PPRV) is both lymphotropic and epitheliotropicthat results infection of epithelial cells and lymphaticswhichmakes the virus to propagate in various cell cultures like embryonic kidney cells from small ruminants, ovine embryonic dermal cells, simian kidney cells, continuous cell lines: Madin-Darby Bovine Kidney (MDBK), Multiple Sclerosis (MS), Baby Hamster Kidney 21(BHK) and Vero (18).

The life cycle of PPRV is too short (19) and the virus has two natural cellular receptors: the signalling lymphocyte activation molecule (SLAM) or CD150 protein and Nectin-4. SLAM is exclusively expressed on immune cells (lymphocyte, macrophages and dendritic cell surface not on epithelial cells), which mediates infection of immune cells and dissemination of the virus while Nectin-4 is the epithelial cell

receptor, that is not expressed in lymphocytes and dendritic cells and mediates infection of epithelial cells and is considered to play major role in virus transmission (20).

2.2 **Genome Organization**

Peste des Petits Ruminants is a linear single stranded negative-sense RNA genome that consists of 15,948 nucleotides and six genes which encode eight proteins: nucleoprotein (N), the phosphoprotein (P), the matrix protein (M), the fusion protein (F), the haemagglutininprotein (H), the large polymerase protein (L), and two non-structural proteins, V and C (fig.1) (21) and the gene order is 3′-N-P(C/V)-M-F-H-L-5′ (22).
N protein is the most plentiful viral protein and the major component of nucleocapsid core that totally encapsidates the viral RNA which is found both in the virion and infected cells (23) that directly associates with the phosphoprotein (P) and the large protein (L). The major role of N protein are: virus transcription, replication, and inhibition of β interferon (INF) production by interacting with interferon regulatory transcription factor 3 (IRF3)- to block its activation (24).

The P protein acts as a molecular intermediate that is proposed to bridge components of the ribonucleoprotein (RNP) during both the replication and transcription activities of the viral life cycle. The P gene of the morbilliviruses is responsible for coding three proteins, including phosphoprotein (P) and two nonstructural proteins (V and C), by overlapping Open reading frames (ORFs) and was found to be the most poorly conserved protein of the morbilliviruses (25).

The C protein is a small basic non-phosphorylated protein that modulateRdRp activity by interacting with the host cell protein (26) and blocking induction of type I interferons (Boxer *et al*., 2009). The V protein is phosphorylated and can bind with both N and L proteins and hence is believed to regulate RNA synthesis and contributes the virus interfering with host innate immunity by blocking INF signaling pathways (27).

Three viral proteins M, F and H are associated with the viral envelope which is derived from the host cell membrane. The M protein is located inside the envelope and forms an inner coat to PPR viral envelope which serves as a bridge between the surface glycoprotein (F and H) and the ribonucleoprotein core (28).The main role of this protein is for virus particle assembly (6).

H protein of the PPRV has both hemagglutinin and neuraminidase actions and named as Hemagglutinin-neuraminidase (HN) protein which is used for virus attachment to host cell and cleavage of the sialic acid residue at the carbohydrate moiety in the glycoprotein of the host. It has a hydrophobic domain at the N-terminus (amino acid position 35–38), which left over associated in mature protein (not cleaved) and acts as a signal peptide to attach the protein into the membrane (29). Whereas N-terminal 34 amino acids of the H proteins are located inside the membrane, the C-terminus is extruding outside and synthesized on ribosome of the rough endoplasmic reticulum (RER). As the protein progresses through the RER, modifications like folding and oligomerization take place to form correct antigenic epitopes. Then, protein passes through the Golgi-complex where it undergoes glycosylation (30). Degree of glycosylation contributes in determining the antigenicity and virulence of the virus (29).

The F protein makes the viral and the cell membranes to fuse, the process that allows the delivery of the viral nucleocapsid into the cell cytoplasm (31). It mediates fusion of the virus to the host cell leading to infection and neutralizing antibodies are mainly directed against H.PPRV has several conserved motifs, four of which are well known fusion peptide (FP), heptad repeat 1(HR1), heptad repeat 2 (HR 2) and a transmembrane domain (TM). During fusion of the viral and host cell membranes, FP is inserted into the host cell membrane and HR1 and HR2 interacts with each other to bring the viral and host cell membranes in a close proximity which in turn results in fusion (32,33). Large Protein is the largest viral protein and is expressed in the smallest amount in the infected cells (6). The L

protein in combination with the P protein carries out replication, transcription, capping and polyadenylation of the viral mRNAs (34).

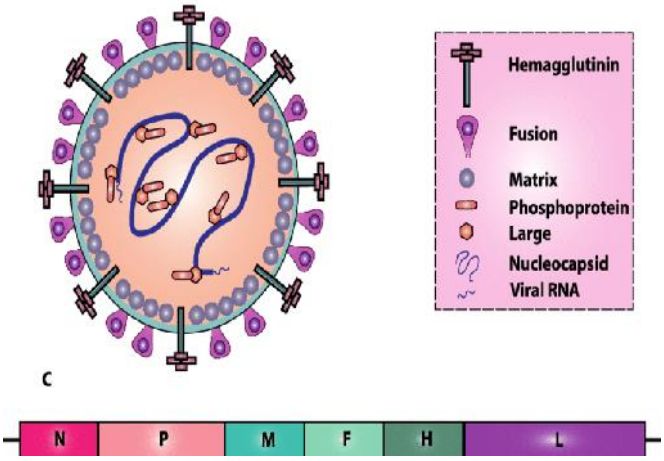

Figure 1.Schematic illustration of the PPRV genome structure and arrangement

Untranslated region (UTR) can be found at the 3' end of both genomic and antigenomic RNAs that serves as the promoter. There is a leader regionat the 3' end of the genome and a trailer region at the 5' end of the genome. The leader combines with the first non-coding sequences of the N gene to form the **genomic promoter** used by the polymerase to synthesize the messenger RNAs. The trailer and the last non-coding sequences of the L protein constitute the **antigenomic promoter** used by the polymerase to synthesize the **antigenome** (positive RNA), the intermediary of the viral genome replication (35).

3. THE EPIDEMIOLOGY OFPPRV

3.1. Determinants of Virulence

There are a number of factors contributingfor virus spreading which arise either by the host or other factors.For instance, livestock management practices, trade, movements of live animals, lack of sanitary controls among livestock producing countries and areas to consumer countries haveincreased the risk of PPRV circulation. Other factors that favor the transmission of the virus are the virus itself, animal, agro-climate conditions and the health surveillance capacity (15).

3.1.1 Host factors

Small ruminants are the main natural host, nevertheless other species like cattle, pigs, buffalo, and camels are newly reported (36). There is little information about susceptibility, occurrence and severity of the disease in wild small ruminants. However, current literature indicates that wild small ruminants have a critical role in the epidemiology of PPR (15). The severity of the disease might differ depending on age, sex, breed, species and seasons (2). In relation to species, goats are more susceptible than sheep in the same environmental situation. This implies that the level of PPRV antibodies is higher in sheep than goats, which deliver sheep resistant to the disease (37).So, the rate of recovery is lower in goats than in sheep. Virus infection can spread between goats without affecting nearby sheep. But mixed raisingof both sheep and goats is the major risk factor for seropositivity in sheep flocks (38).

In addition, age is the main factor for seropositivity (39) and case fatality rate is higher in young than in adults (40). Sex-based distribution of antibodies is frequently biased because the males are sold earlier and females are kept for longer. The disease rate (morbidity) increases with environmental stress such as confinement of animals during winter and rainy seasons (41). However, the effects of environment on the occurrence of PPR are solely based on the nature of animal husbandry and socio-economic status. Although there have been significant contributions in understanding the risk factors, the genetic marker of disease predisposition are not determined.

3.1.2. Non-host factors

PPRV is highly contagious and the virus is spread from infected to healthy animals via close contact (42). The virus can also be secreted in sneezing and coughing, in which transmission occur during inhalation or contact with inanimate objects. The

survival of the virus in the dam's milk has not been investigated; however, based on its resemblance with RPV, it is likely that PPRV is also secreted in milk 1–2 days before signs appear (43). Transmission is not possible through fomites, because of the short life span of the virus in dry environments (above 70°C) and in acidic (>5.6) or basic (<9.6) pH. Moreover, PPRV cannot live for a long time outside the host because of its short half-life, which is estimated to be 2.2 minutes at 56°C and 3.3 hours at 37°C (43).

3.2. Host Range

Peste des Petits Ruminants is mainly a disease of small ruminants that affects goats and sheep which exhibits different levels of virulence between them. Goats are severely affected while sheep generally undergo a mild form (44). Interestingly, cattle, bu alo, camel and pigs can develop subclinical infections, but do not excrete the virus (45).They are known to be a dead end hosts and undergo a silent or subclinical infection. Morbidity is common, mainly in susceptible goat populations and milder forms in sheep and moderately immune goat populations (3).A higher affinity of the virus for the caprine species in opposition to the ovine species and same virus being capable of inducing high mortality in goat population particularly in young goats than the susceptible sheep has been reported (46).

3.3. Transmission

Transmission requires close contact between infected animals in the febrile stage and susceptible animals because of the fragility of the virus outside the living host. The discharges from eyes, nose and mouth, as well as the loose faeces, fine infective droplets released into the air from these secretions and excretions from affected animals during coughing and sneezing contain large amounts of the virus (47). Therefore, close contact is the most important way of transmitting the disease. In accumulation, infectious materials can also contaminate water and feed troughs and bedding, turning theminto additional sources of infection. 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 (48).

3.4. Global Distribution

After first discovery in Côte d'Ivoire, the disease spread to sub-Saharan Africa, the Middle East, Turkey, India, China, Kenya, Uganda, Tanzania, Morocco and Tunisia (Figure2) (20). This indicates that the virus is highly infectious andhas emerging transboundary character**.** However, all the PPRV strains remained in the same group despite of what gene was used as basis for classification. On the other hand, it is still unclear whether differences between lineages purely reveal geographical speciation or if they are also correlated with variability in pathogenicity between isolates (49). PPRV belonging to lineages I and IIhave exclusively been isolated from the countries in West Africa, where it is once originated. Lineage III is restricted to the Middle East and East Africa. Though lineage IV was strictly considered an Asian lineage.

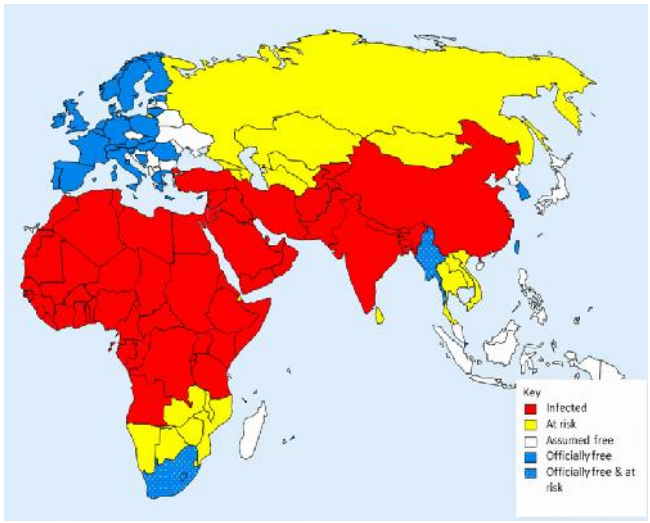

Figure 2. Spatial Distribution of PPRV
Source :(50)

3.6. Distribution in Africa

PPRV is currently present in some countries in the North Africa region where the situation has

evolved in recent years. The disease occurred for the first time in Morocco in 2008, with a virus belonging to lineage IV and the same lineage IV is also present in Tunisia and Algeria which is widespread in Egypt (51).All countries in Eastern Africa are infected where a regional strategy has been developed. (52).

Southern Africa is currently free from PPR which is officially recognised by the OIE. Following the introduction of PPR in a few countries, the Southern African Development Community (SADC) developed a regional PPR control strategy in 2010 in order to control PPR virus circulation in these countries, to prevent the disease from spreading to adjoining countries and ultimately to achieve the eradication of PPR from the SADC region (53).

Central and West African countries are infected and they are facing multiple constraints in controlling and eradicating PPR. At a regional level, the relevant Economic Community of West African States (ECOWAS), Central African Economic and Monetary Community (CEMAC), Western African Economic and Monetary Union (WAEMU), and other regional organisations need to enhance their political commitment as well as their financial and technical support together with their development partners (51).

3.7. Distribution in Ethiopia

Among viral diseases of sheep and goats, PPR is the main top constraint to small ruminant production in Ethiopia. It is widely distributed in the country and high sero-prevalence of the disease has been reported from different parts of the country in small ruminants (2)cattle and camels (3).There are differentfactors that contributes the spreadof the virus.The important risk facors which significantly identifiedare:Species,Age, Market, Sex,Flock size,Markets and Agro-ecology (54).

3.7.1. Species

In relate to species, goats are more susceptible than sheep in the same environmental situation. This implies that the level of PPRV antibodies is higher in sheep than goats, which deliver sheep resistant to the disease (37). So, the rate of recovery is lower in goats than in sheep. Virus infection can spread between goats without affecting nearby sheep. But mixed rising of both sheep and goats is the major risk factor for seropositivity in sheep flocks (38).

3.7.2. Age

Age is the main factor for seropositivity (39) and case fatality rate is higher in young than in adults due to unmature immune status and the decline of maternal antibodies and nutritional related factors (40).

3.7.3. Sex

With respect to sex, PPRseroprevalence found infemales was slightly higher than males. This is due to Females are kept longer in a flock for the breedingpurposes while males are either sold out or slaughteredfor meat purposes.In addition, females become pregnant and lambing/kidding, theimmunity status becomes lowered which results theirability to resist the challenge of the infection will be low(39).

3.7.4. Flock size

Large herds with a high density of animals often associated with high contact between different levels of viral susceptibility animals which affoards the spread of virus easily (47).

3.7.5. Markets

Legal and illegal cross-border movements of animals like Imports and exports lacking health inspections increasing the spread of virus between livestock rearing areas towards meat consuming areas (15).

3.7.6. Grazing

The prevalence of PPR higher in communal grazing systems than private land grazing. This is due tovulnerability of small ruminant flocks for infection sinceopencommunal grazing systems infect pastures and watering points (54).

3.7.7. agro-ecology

The seroprevalance of PPR in lowlandarea is high (39). This is due to different production systems with exchanges and movements in areas of lowland being more common and involving larger numbers of animals. In Ethiopia, small ruminants mainly flourish on free range pasture lands, shrubs and forest grounds. These influence the availability of these resources and the movement of animals becomes necessary in order to ensure the provision of feed and water. This is mostly important during the dry season and in low altitude areas where resources are scarce(54).

In Ethiopia, the presence of the disease was first suspected in 1977 in the eastern part of the country, Afar based on clinical evidences (16). The virus was detected in 1994, and subsequently the isolate reported in 1996 and genetically classified to cluster in lineage III(55).

Recent reports indicated that lineage IV is circulating and continued to spread in Ethiopia (56). This might indicate the lineage distribution of PPR is currently changing. So far, the epidemiological lineagesand spread of the PPR strains are not well understood. Molecular characterizations of circulating strains are thus important tool to understand the epidemiology of PPR virus and track outbreaks in the country. Such information contributes to establishing the diversity and circulation of strains in the field, trace the spatiotemporal origin of a virus, and estimate the risk of its introduction into the herd(39).

PPRV was characterized and phylogenetically analysed based on the fusion gene (F), which classified all the strains of PPRV into four distinct lineages (8). Afterwards, it appeared that phylogenetic analysis based on the nucleoprotein gene (N) presented a better molecular epidemiological pattern (57). It is currently preferred over F gene-based phylogenetic analysis and the use of the haemagglutinin- neuraminidase (HN) gene, in addition to the F and N genes, could give better declaration and allow tracing of virus transmission within outbreaks (15).

Moreover, it is crucial to note that countries once exclusively carrying a single lineage are now simultaneously reporting the presence of several lineages i.e. Ethiopia, Sudan and Uganda (58). In the majority of cases, the newly introduced lineage is lineage IV (59).Advanced sequencing technologies have enabled molecular epidemiologic studies of viruses in which whole gene and complete genome data are used to enhance and explain the evolutionary dynamics of virus (60). This analysis will allow a more precise evolutionary and phylogenetic assessment of the relationships between lineages (57).

Serological studies conducted in different parts of the country indicate, PPRV is widespread in small ruminants.Thus, based on the easily reached literature the prevalence of PPRV in small ruminants recorded by different workers (Table-1).

Table -2: Species-wise seroprevalence of PPR in different parts of Ethiopia

	Sheep		Goat		
Area	No.tested	Prevalence (%)	No.tested	Prevalence (%)	Reference
Gurage	190	24.2	200	34	Gizachew (61)
Tigray	-	-	240	47.5	Berihun*et al*. (62)
Oromia	293	50.85	407	46.68	Getachew*et al*. (63)
Afar	94	41.5	135	39.3	Fikru*et al*. (64)
Somali	216	41	58	24	Wondimagegne(65)
Afar	251	29.5	912	31.3	Megersa *et al*.(2)
Amhara	329	14.89	343	21.57	Tsegaw *et al*. (66)

The seroprevalence in different parts of the country were not unifrm.Thesecan beexplained by sample size difference, geographical andseasonal effects, host population density, vaccination and the social environments.

4. Conclusion

PPR is an acute, highly contagious and fatal viral disease mainly affecting sheep and goats with high morbidity and mortality rates. Despite the use of an effective and cheap live-attenuated vaccine, the virus has alarmingly spreading to disease-free countries. The disease is common in much of Africa, the Nearand Middle East and Asia. Based on sequence analysis of the nucleoprotein and fusion gene, the virus has four lineages.PPR is widely distributed in different parts of Ethiopia and two lineages of the virus (III and IV) are recorded from the country. Therefore, molecular characterizations of circulating strains are important tool to understand the epidemiology of the virus and track outbreaks in the country.

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