

Thesis Ref. No. _____

**SERO-EPIDEMIOLOGY AND SPATIAL DISTRIBUTION OF PESTE DES
PETITS RUMINANTS VIRUS ANTIBODIES IN SOME SELECTED PASTORAL
AREAS OF SOMALI REGIONAL STATE, ETHIOPIA**

MSc Thesis



By

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Addis Ababa University, College of Veterinary Medicine and Agriculture, Department of
Veterinary Clinical studies

June, 2016

Bishoftu, Ethiopia

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RUMINANTS VIRUS ANTIBODIES IN SOME SELECTED PASTORAL AREAS OF
SOMALI REGIONAL STATE, ETHIOPIA



A Thesis submitted to College of Veterinary Medicine and Agriculture of Addis Ababa
University in partial fulfillment of the requirements for the degree of Master of Science in
Tropical Veterinary Epidemiology

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SIGNATURE

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SOMALI REGIONAL STATE, ETHIOPIA

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DEDICATION

I dedicate my MSc thesis work to those livestock herders that have lost their animals to the deadly virus called peste des petits ruminants.

STATEMENT OF THE AUTHOR

First, I declare that this thesis is my *bonafide* work and that all sources of material used for this thesis have been duly acknowledged. This thesis has been submitted in partial fulfillment of the requirements for an advanced (MSc) degree at Addis Ababa University, College of Veterinary Medicine and Agriculture and is deposited at the University/College library to be made available to borrowers under rules of the Library. I solemnly declare that this thesis is not submitted to any other institution anywhere for the award of any academic degree, diploma, or certificate.

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LIST OF ABBREVIATIONS

AAU	Addis Ababa University
AU-IBAR	African Union – Inter African Bureau for Animal Resources
CAWS	Community Animal Health Workers
C-ELISA	Competitive Enzyme-Linked Immunosorbent Assay
DIVA	Differentiation between infected and vaccinated animals
FAO	Food and Agriculture Organisation
GPS	Global Positioning System
LFD	Lateral Flow Device
LRT	Likelihood Ratio Test
MoLF	Ministry of Livestock and Fisheries
NAHDIC	National Animal Health and Diagnostic Center
OD	Optical Density
OIE	Office International des Epizooties
OR	Odd Ratio
PCR	Polymerase Chain Reaction
PA	Peasant Association
PPR	Peste des Petits Ruminants
PPRV	Peste des Petits Ruminants Virus
RNA	Ribo Nucleic Acid
RPV	Rinder Pest Virus
RT-PCR	Reverse Transcriptase Polymerase Chain Reaction
STSD	Surveillance of Trade Sensitive Diseases
TAD	Transboundary Animal Disease
VNT	Virus Neutralization Test
VS	Veterinary Sanfrontiers

ABSTRACT

A cross sectional study design with a 2 stage cluster sampling was conducted in some selected pastoral areas of Somali regional state from November to May 2015/16. The study was aimed at determining the serological status of PPR disease in sheep and goats and identifying animal and flock level risk factors in the selected Afder, and Liben zones of Somali regional state. And finally, the national status of PPR from disease outbreak reports was assessed from retrospective data. A total of 798 serum (582 goats and 216 sheep) from 19 Kebeles (peasant associations) across 8 Districts were investigated. PA level prevalence was variable (12%-64%) and the difference was significant ($X^2=53.3$ $P=0.000$). At District level, the prevalence of the disease was recorded in descending order as Dolo Bay 52% ($CI_{95\%} = 41-63$), Dolo Ado 42% ($CI_{95\%}= 36-48$), Hudet 40% ($CI_{95\%}=30-52$), Chereti 40% ($CI_{95\%}= 26-57$), Gorobeqeqsa 40% ($CI_{95\%}= 31-50$), Guradhemole 38% ($CI_{95\%}= 28-49$), Filtu 36% ($CI_{95\%}= 22-52$) and Moyale 30% ($CI_{95\%}= 20-41$). There was no significance difference in the proportion of sero-positives between Districts ($X^2= 7.46$ $p= 0.382$). The overall true prevalence was 43%. Furthermore, the prevalence in sheep 39% ($CI_{95\%}= 32-46$) was found insignificant ($X^2=0.49$ $P= 0.483$) when compared with goats 42% ($CI_{95\%}= 38-46$). The true prevalence of PPR in Sheep and goats was 41% and 44%, respectively. In addition, small ruminants aged between 36-48 months had recorded the highest prevalence 58% ($CI_{95\%}= 49-66$) out of all age groups. There was quite significant difference in sero-positivity among the age groups sampled ($X^2= 42.55$ $p= 0.000$). Female small ruminants had a statistically greater sero-prevalence rate 44% when compared with males 26% ($X^2= 16.4$ $P= 0.000$). Likewise, the likelihood of occurrence of PPR in female sheep and goats were 2.5 times more than its occurrence in males ($OR= 2.5$). The overall true flock prevalence was found to be 104% considering at least one positive in a flock. In the multivariable logistic model for both sheep and goats age group, origin and altitude were found the risk factors. However, more disease predictors were identified after adjusting the cluster effect of PAs and herds using multilevel mixed-effect logistic regression models. Therefore, in the multilevel mixed-effect logistic regression age group, origin, altitude, production system and water source were the most likely disease predictors of PPR disease in small ruminants and the variables were fit in the model using likelihood-ratio test ($P= 0.000$). Retrospectively, a

total of 1282 outbreaks were reported nationally across all regions in the 10 year period. The detection of PPR virus antibodies in all PAs and Districts suggest that the wide circulation of the virus in the study area. Hence, to curb the wide spread of the disease, strategic vaccination scheme should be followed along with training to field veterinarians and community animal health workers (CAHWS). Besides, it is recommended to conduct further studies on the characteristic of the virus circulating in the study areas plus to investigate the role of camels, cattle and wild ruminants in the epidemiology of PPR which could be a milestone in the eradication of the disease.

Key words: *Cluster, Epidemiology, Flock, Goat, PPR, Prevalence, Risk Factors, Sheep, Somali region,*

1. INTRODUCTION

Following the successful eradication of rinderpest from worldwide in 2011, a closely related disease, the peste des petits ruminants (PPR), has now become the next target for programed eradication in the regions where the disease is currently present by replicating the tools and experience used in the rinderpest eradication program (Horzinek, 2011).

As was the case with rinderpest, several technical factors favour the prospect of achieving global eradication of PPR virus. These include: the existence of the virus as a single serotype; the absence of a carrier state; the lack of any reservoir of infection outside of the small ruminant population; the availability of live attenuated vaccines which confer lifelong immunity after a single dose and are robust, safe and relatively cheap to produce; the availability of diagnostic tests for sero-monitoring of vaccination programmes and detection of virus circulation; as well as a growing political support for eradication (FAO, 2013).

PPR is a widespread, virulent and devastating disease of small ruminants. It has a significant economic impact on food security and livelihoods. PPR is therefore considered as one of the most damaging of all animal diseases in Africa, the Middle East and Asia, and it is also one of the priority diseases indicated in the FAO-OIE Global Framework for the Progressive Control of Transboundary Animal Diseases (FAO/OIE, 2015).

The disease is characterized by fever, erosive stomatitis, conjunctivitis, gastroenteritis, pneumonia and causes serious economic losses in production of small ruminants (Merck Sharp and Dohme, 2009; Elsawalhy *et al.*, 2010). The disease was first described in Cote d'Ivoire, West Africa by Gargadennec and Lalanne in 1942. PPR is also known as goat plague, pseudo rinderpest of small ruminants, pest of small ruminants, pest of sheep and goats, Kata, stomatitis pneumo-enteritis syndrome, contagious pustular stomatitis, and pneumo-enteritis complex (Braide, 1981).

The reference to the disease as a "plague" is indicative of the highly contagious nature and economic impacts that result from this disease. It was only in the late 1970s that PPR

was determined to be a distinct virus from rinderpest virus through serology, biochemical and cross-protection experiments (Hamdy *et al.*, 1976; Taylor 1979 a and b). The disease was initially thought to be confined to the countries of West Africa; however, PPR has now been confirmed present in several African, Middle East, Central and South Asia countries, as well as in China (Munir *et al.*, 2013; Libeau *et al.*, 2014). Etiological agent of PPR is a member of the genus morbillivirus. The viral genome is 15,948 nucleotides long and contains six genes encoding six major polypeptides (Bailey *et al.*, 2005).

The etiological agent, peste des petits ruminants virus (PPRV), has only one known serotype with different geographical distributions, but at least four distinct genetic lineages (I–IV) have been described based on alignments of N gene or F gene nucleotide sequences (Shaila *et al.*, 1996; Kwiatek *et al.*, 2007). Of the four known lineages of PPR virus, lineage I and II viruses have been found exclusively in West Africa. Lineage III has been found in east Africa, identified in the outbreak of 1996 in Ethiopia, also in the Arabian Peninsula and southern India (Dhar *et al.*, 2002).

The PPR disease epidemics can cause mortality rates as high as 90% in naive sheep and goat populations. In clean flocks, sheep and goats of all ages can be affected during an outbreak. However, in endemic areas the most susceptible ages are between 4 and 24 months. The disease has been associated with increased animal movement for commercial and trade purposes, transhumance and nomadic customs, climatic changes and extensive farming practices (FAO, 2008).

PPR virus has a widespread distribution spanning Africa and Asia (Nanda *et al.*, 1996; Shaila *et al.*, 1996). 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. In many areas of Asia and Africa, small ruminant production and the livelihoods of poor farmers are threatened by PPR among other trans-boundary animal diseases (TADs). With its associated high morbidity and mortality, PPR constitutes one of the major obstacles to subsistence farming (Banyard *et al.*, 2010).

PPR was first suspected in Ethiopia in 1984 following clinical observations consistent with infection with PPR (Pegram and Tereke, 1981) and was later diagnosed as the

causative agent of disease in goats in the country (Roeder et al., 1994). The virus detected in 1994, alongside a further isolate reported in 1996 was genetically determined to cluster within lineage III (Kwiatak *et al.*, 2007; Banyard *et al.*, 2010). However, in a study conducted by Muniraju *et al.*, (2014) reported the emergence of lineage IV during the 2010 outbreak in Ethiopia.

After the first confirmed cases of PPR in Ethiopia, the disease is continuously affecting small ruminant production and thus contributing to food insecurity particularly in vulnerable regions of the country. Besides, the emergence of lineage IV (Muniraju *et al.*, 2014) isolated from male goats purchased from a market, though not substantiated at field level, has pose additional threat in the control of the disease. Accurate data on the distribution of PPR infection and identification of animal and herd level risk factors for PPR are of paramount importance to control the disease with the ultimate goal of achieving eradication. However, despite its importance, the epidemiological risk factors, linkages between continuing outbreaks, and spread of PPR are not well understood with necessitates further studies.

Therefore, the objective of the studies were

General objective:

- To determine the epidemiology of PPR in Sheep and goats in the selected districts of Liben and Afdher zones of Somali regional state, Ethiopia.

Specific objective:

- To determine the PPR individual and herd level antibody prevalence in sheep and goats
- To evaluate the association with animal and herd level potential risk factors,
- To conduct the spatial distribution of PPR in the selected districts of the aforementioned zones.
- To assess the national status of the disease using a retrospective data.

1.1 Research Question and Justification of the Study

The epidemiology of PPR disease is not well understood in East Africa and more so in Ethiopia. In fact, in Ethiopia the first clinical and serological case of PPR was reported in 1984. After the first confirmed cases of PPR in Ethiopia, the disease is continuously affecting small ruminant production and thus contributing to food insecurity, particularly, in vulnerable regions of the country. The limited studies in Ethiopia are not systematically coordinated to elucidate the national status of the disease and could be suspected of misdiagnosed as the reports to MoLF from the field are based on symptoms. The Liben and Afdher zones of Somali regional states of Ethiopia are one of the most prone areas for PPR infection. They share border with Kenya and Somalia where there is frequent and uncontrolled movement of livestock along the border, which predisposes small ruminants to PPR. The disease has been reported in Somalia and Kenya since 2006. In serological survey conducted in 1999 in Ethiopia which was the largest survey on PPR ever conducted in Africa reported the highest PPR disease prevalence (52.5%) in Somali region of Liben zone, Dolo Ado district. Dolo Ado shares borders and triangulates with Somalia and Kenya. This showed the area is important to PPR distribution due to geographical location and the pastoral communities suffered massive losses due to the disease which makes PPR control relevant. However, crucial to the effective control of the disease in Somali region is the adequate understanding of the epidemiology of the disease. Thus, it becomes imperative to conduct a study on the current prevalence of the disease in the region so as to broaden the understanding of the epidemiology which will in turn help in the control of the disease and enhance all the benefits associated with small ruminants in the region.

1.2 Limitations and Challenges of the Study

Afdar livelihood zone is known for having some of the worst roads in the region. The Gorobekeksa-Guradhemole, Chereri-Gorobekesa, most of which are important trade routes for livestock and other commodities are all bad. Road access into the livelihood zone is via the Moyale-Addis road a tarmac route that links Kenya to Addis Ababa, but it does not extend into other parts of the zone. However, we accessed to the region through Bale of Oromia region in order to address the selected Somali Districts found in the border of Oromia such as Guradhemole.

Therefore, the road infrastructure from Guradhamole (Haro dibe) till to Melka Chareti and from Dolo Ado to Hay suftu district was very bad; we sunk in sand many times. Besides, the weather condition which was the hottest season in the area was one of the challenges for the team to work and move easily.

We had time limitation during the study. Because of search of pasture and water it was impossible to get and sample the animals unless we stayed overnight in villages and work early morning. As a result of this it was very difficult for us to reach all PAs within given feasible working time (that is usually morning). Even PAs within one district are much far away from each other and from district town and we forced to stay overnight in some PAs (Hager Moker, Bur amino, Kole and Melka Dida , Melka suftu, Fiquo, wadeluhabe and Kojowa). This incurs extra days in the same PA.

In some PAs we were required to treat their livestock before attempting to bled which in turn was very difficult for us since a budget was not allotted for drug and labor cost by the project. Many pastoralists nowadays are aware of the benefit of treating animals using drugs and vaccines. In our observation, in those districts we worked the pastorals request for Ivermectin and Oxytetracline when they recognized we were animal health professionals.

It was on the finale week of my study, when only three kebel remain to finish, that our vehicle was hijacked by a fully armed man together with a woman. The man forced the driver to walk up at mid night and by intimidating and using physical attack (about 12 p.m mid night) they took the car along with the driver and drove to the direction of

Yabello road and left the driver in a bush. After 60km drive he left the car and took some of our properties. The following morning the two captors were arrested at Yabello check point (kella) while using a public transport looking as if a civilians and the car was found and brought to moyale.

A team from NAHDIC (National Animal Health and Diagnostic Center) who were on different mission in Borena zone took our sample since our car was unable to move damaged during the incident. Working for around a month in those remote areas of Somali region, Ethio-Kenya and Somalia border, and when three days left to wind up our exhaustion we were a bit unlucky to encounter the above phenomenon. Lastly, our vehicle was carried by a truck to Addis Ababa and we were forced to use a public transport to get back from the study area without any injury and harm.

2. LITREATURE REVIEW

2.1. The Disease Called Peste Des Petits Ruminants

Peste des petits ruminants (PPR), also known as Kata, pseudo rinderpest, pneumo enteritis complex or stomatitis-pneumo enteritis syndrome is a severe and highly infectious viral disease of small ruminants caused by PPR virus, a *Mobilivirus* of the Family *Paramyxoviridae*. The disease is characterized by fever, erosive stomatitis, conjunctivitis, gastroenteritis, and pneumonia. The clinical disease resembles Rinderpest in cattle, which is acute, and after an incubation period of 3-6 days, the clinical symptoms become apparent, and include high rise of temperature, oral, ocular and nasal discharges, necrotic stomatitis, severe pneumonia, dyspnoea, coughing, enteritis, severe diarrhoea followed by death (Ezeokoli *et al.*, 1986; Pawaiya *et al.*, 2004).

The natural disease affects mainly goats and sheep, but is usually more severe in goats where it causes severe morbidity and mortality (Raghavendra *et al.*, 2000). Infection rates in sheep and goats increase with age, and the disease, which varies in severity, is rapidly fatal in young animals (Wosu, 1994). Generally, cattle are considered to be sub-clinically infected with the disease. However, in poor conditions it might be possible that cattle develop lesions following PPRV infection, clinical signs of which would be ascribed to rinderpest, because of the similarity of the two diseases clinically. Moreover, PPRV was isolated from an outbreak of rinderpest-like disease in buffaloes in India in 1995 (Govindarajan *et al.*, 1997). It was also suspected to be involved in the epizootic disease that affected one-humped camels in Ethiopia in 1995–1996 (Roger *et al.*, 2001). Indeed, PPRV antigen and PPRV nucleic acid were detected in some pathological samples collected during that outbreak (Roger *et al.*, 2001), but no live virus was isolated. Cases of clinical disease have been reported in wildlife resulting in deaths of gazelles in captivity (Elzein *et al.*, 2004).

Before the recognition of PPR as a disease entity in small ruminants, there were historical records of outbreaks of Rinderpest like diseases in sheep and goats that did not cause disease in cattle. Furthermore, diagnostic tests that are capable of differentiating between the two viruses only became available in the past 20 years. Therefore, it was likely that,

in the past, many cases of PPR were ascribed to RPV (Baron, 2011). The idea that PPR has been in existence as a distinct disease for a long time is supported by the phylogenetic tree of the morbilliviruses, which shows that both RPV and PPRV were a similar distance from their most recent common ancestor. Assuming both viruses mutate according to the same evolutionary „clock“, PPRV must have been in circulation for as long as RPV.

In all regions where PPR is endemic, it constitutes a serious threat to small ruminant production. The disease is said to be the fastest growing disease of small ruminants in developing countries (Baron, 2011). Therefore, it influences the livelihood of poor farmers, the main owners of sheep and goats. Hence its control is a major priority for programs aimed at poverty alleviation.

2.2. Etiology

The etiological agent, *Peste des petits ruminants virus* (PPRV) has been classified under family Paramyxoviridae, Order Mononegavirales and Genus Morbillivirus (Tober *et al.*, 1998). Similar to other morbilliviruses, PPRV is fragile and it cannot survive for long time outside the host. Its half-life has been estimated to be 2.2 minutes at 56 °C and 3.3 hours at 37 °C (Rossiter and Taylor, 1994).

Like other members of the family Paramyxoviridae, PPR virus is an enveloped pleomorphic particle. The genome of PPRV is single stranded RNA, approximately 16kb long with negative polarity (Haas *et al.*, 1995). PPR virions, as other morbilliviruses, are enveloped, pleomorphic particles containing single strand RNA as the genome. It is composed of 15, 948 nucleotides, the longest of all morbillivirus genomes sequenced so far. This genomic RNA is wrapped by the nucleoprotein (N) to form the nucleocapsid into which are associated two other viral proteins: the phosphoprotein (P) and the large protein (L) (Diallo, 2007).

The phosphoprotein is the cofactor of L, the viral RNA dependent RNA polymerase (RdRp). To the viral envelop which derives from the host cell membrane are associated three viral proteins: the matrix protein (M) which is located inside the envelope and serves as a link between the nucleocapsid and the two external viral proteins, the fusion

protein (F) and the haemagglutinin (H). 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. However N the most abundant and also the most immunogenic among PPRV proteins does not induce protective immunity against the virus. It has been used in the development of diagnostic tests (Diallo, 2007).

2.2.1. *Biology of the Virus*

Structure of PPR virus

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 (Barrett *et al.*, 2005). The genome of PPR virus is so far the longest of all the morbilliviruses, consisting of about 15,948 nucleotides. Intact virion has a diameter of about 130-390nm with the thickness of the ribonucleoprotein measuring approximately 14-23nm (Durojaiye *et al.*, 1985). It is wrapped by a nuclear protein which is associated with two other proteins: the phosphoprotein (P) and the viral RNA dependant RNA polymerase (L). On the viral envelope are found two other viral proteins, Haemagglutinin (H) and Fusion (F) proteins, which are very important for the induction of protective host immune response against the virus (Chauhan *et al.*, 2009).

Structural proteins of the virus

The virus encodes six structural proteins; nucleoprotein (N), phosphoprotein (P), matrix Protein (M), fusion protein (F), hemagglutinin protein (H), large polymerase protein (L), and non-structural proteins V and C (Bailey *et al.*, 2007). Among the structural proteins, N protein is antigenically the most conservative among the morbilliviruses and is highly immunogenic in spite of its internal location (Libeau *et al.*, 1995). The F and H proteins are associated with the viral envelope where they are believed to play important roles in induction of protective immunity (Chauhan *et al.*, 2009). 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, have methylation, capping and polyadenylation activities (Lamb and Kolakofsky, 2001). The Matrix (M) proteins are basic membrane associated molecules that interact with surface glycoproteins in the lipid envelope as well as the virion ribonucleoprotein.

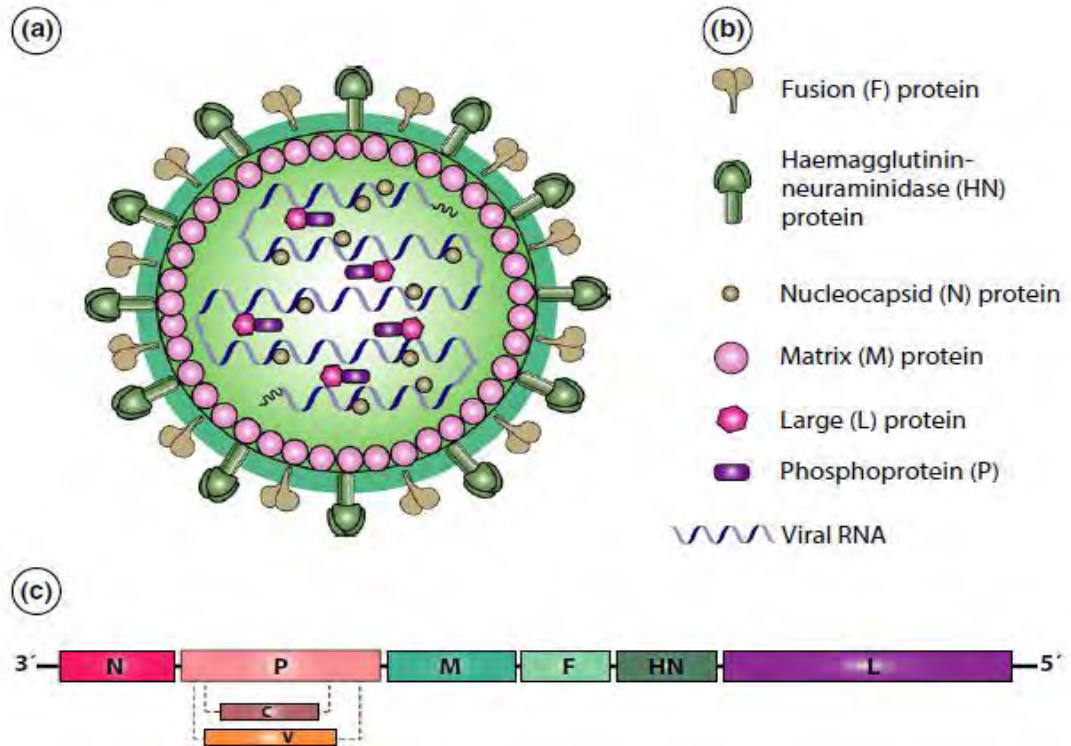


Figure 1: (a) Schematic structure of a typical morbillivirus (PPRV). (b) The structural components of PPR. (c) The genome organization of all known genes of PPRV.

2.2.2. Physiochemical properties of the virus

The molecular weight of the genome is 5.8×10^6 while the diameter of the virion measures about 150-300nm. The virion is very sensitive to heat, lipid solvents, non-ionic detergents, formaldehyde and oxidising agents (Kingsbury, 1990). The virus is usually destroyed at 50°C for 60 minutes or 37°C for 2 hours. However, it survives for long periods in chilled and frozen tissues (OIE, 2009).

2.3. Pathogenesis and Clinical Signs

There is variation in the inherent resistance of different breeds of sheep and goats to PPRV (Couacy-Hymann *et al.*, 2007). There is anecdotal evidence that younger animals show higher mortality rates, but this has not been confirmed by experiments. Sheep and goats infected with PPRV show a similar, if slightly less severe, clinical picture to that seen in cattle infected with RPV. There is a rare per-acute form of the disease causing death four to six days after the onset of fever. The more frequent acute form is characterized by a sudden rise in body temperature, peaking at 2–2.5 °C above normal. The mucous membranes of the eyes and nose become congested and there is noticeable discharge from the eyes and nose (Lefevre and Diallo, 1990).

There is a marked and rapid loss of circulating white blood cells (leucopenia) at this time, starting from two to three days post infection (dpi). The white blood cell count will remain low (about 20% of normal) and will return to normal only during the convalescent phase. As the disease progresses, congestion can be seen in the gums, and necrotic lesions appear in the epithelial tissue lining the mouth, first in the gums and the inside of the lower lip, and in severe cases can be seen on the top and sides of the tongue and in other parts of the buccal mucosa. The necrotic areas throughout the mouth and gums readily erode. As the disease progresses further, diarrhea develops, which is occasionally bloody (Lefevre and Diallo, 1990).

Many animals with PPR show abnormally rapid or labored breathing, and a productive cough. By this stage, the animal is apathetic, with labored breathing and an unwillingness to move. Convalescence, if it occurs, takes several weeks. Any animals that are pregnant at the time of infection will abort. The white blood cell count slowly returns to normal and the oral lesions heal over a period of two to three weeks. This transient loss of white cells, and the generalized immunosuppression that can go on for even longer, means that the animal is susceptible to activation of latent or chronic infections (e.g. with parasites) or to secondary infection by other pathogens. The virus infection, on the other hand, completely resolves in recovered animals, and there is no persistent infection or carrier state (Lefevre and Diallo, 1990).

In post-mortem examination, PPRV infection reveals significant lung pathology, with patches of congestion in the lung tissue and signs of pneumonia. Animals show extensive damage to mucous membranes of the digestive tract and to lymphoid organs. Immuno-histological examination shows that the virus is primarily lymphotropic, with epithelial tissue involvement in only later stages of infection (Pope *et al.*, 2013). Further details on the pathology of PPR disease can be found in Wohlsein and Saliki (2006).

The morbidity and mortality rate varies enormously (up to 100 %) depending on the species infected, the age of the animals, the prevalence of secondary infectious agents and the PPRV lineage involved (Zahur *et al.*, 2009; Kivaria *et al.*, 2013; OIE, 2013; Chowdhury *et al.*, 2014).

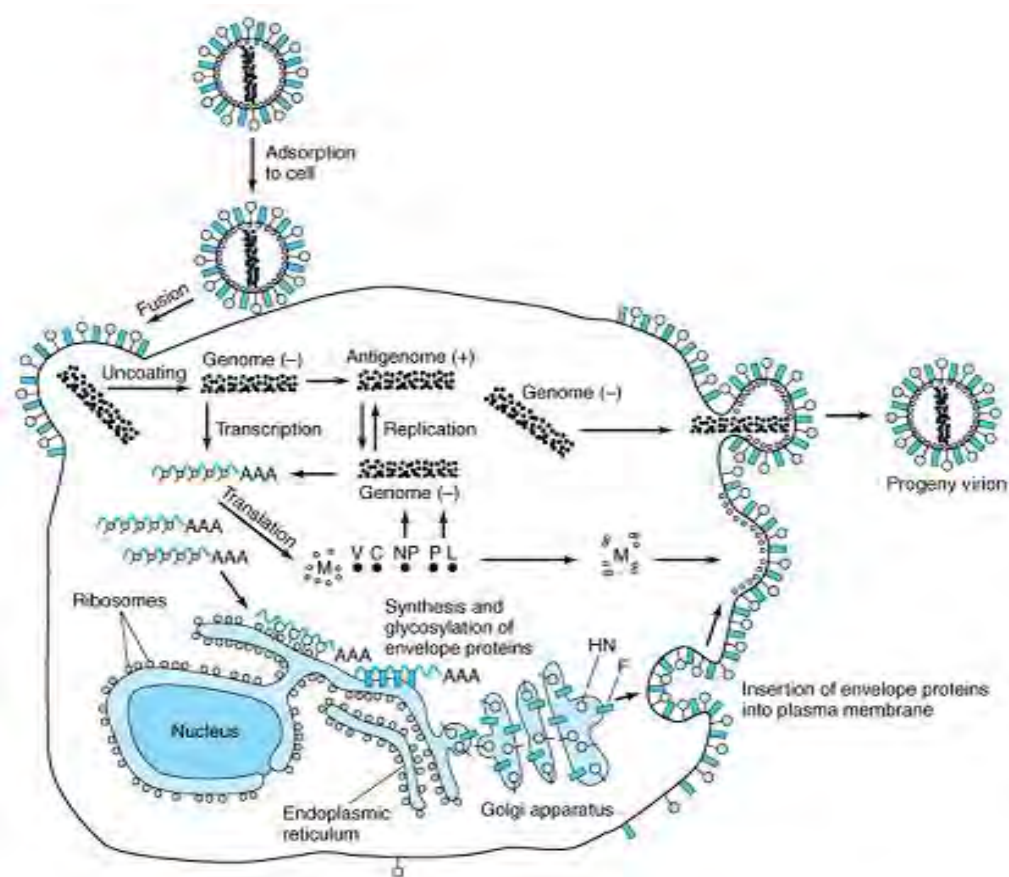


Figure 2: The process of Morbillivirus virus replication

Source: (Brooks *et al.*, 2008)

2.4. Differential Diagnosis

Other diseases cause diarrhea or pneumonia in sheep and goats may pose diagnostic challenge but a history of recent introduction of new stock and the clinical and postmortem findings of stomatitis, typical for PPR. Laboratory tests are requiring ruling out rinderpest (*Radostitis et al*, 2007 &LPP, 2006). 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 (OIE, 2002).

2.5. Epidemiological Situations

2.5.1. Geographical distribution of the disease

Since it was first identified in the early 1940s in Côte d'Ivoire, the disease has spread throughout Africa, South Asia and China (Figure. 3). In the last 15 years, it has expanded into previously non-infected regions. As a result, PPR is now endemic in large parts of the Middle East, Central Asia, South Asia and East Asia and is expected to spread into Southern Africa and Southeast Asia. Populations of the northern Mediterranean region are also at high risk. If left uncontrolled, and with the increasing global flow of livestock products to meet consumer demands, PPR will likely make inroads in Mongolia as well as to other countries in the Caucasus and Europe that have historically been free of the disease (OIE and FAO, 2015).

There are many gaps in current understanding about the epidemiology of PPR. There are many reports with different scenarios of animal species involved in the outbreaks: goats alone, sheep alone, or sheep and goats together. While large ruminants are believed to be relatively resistant, there have been reports indicating the involvement of PPRV in respiratory disease in camels (Roger *et al.*, 2000) in Africa or rinderpest-like disease in buffaloes in India (Govindarajan *et al.*, 1997).

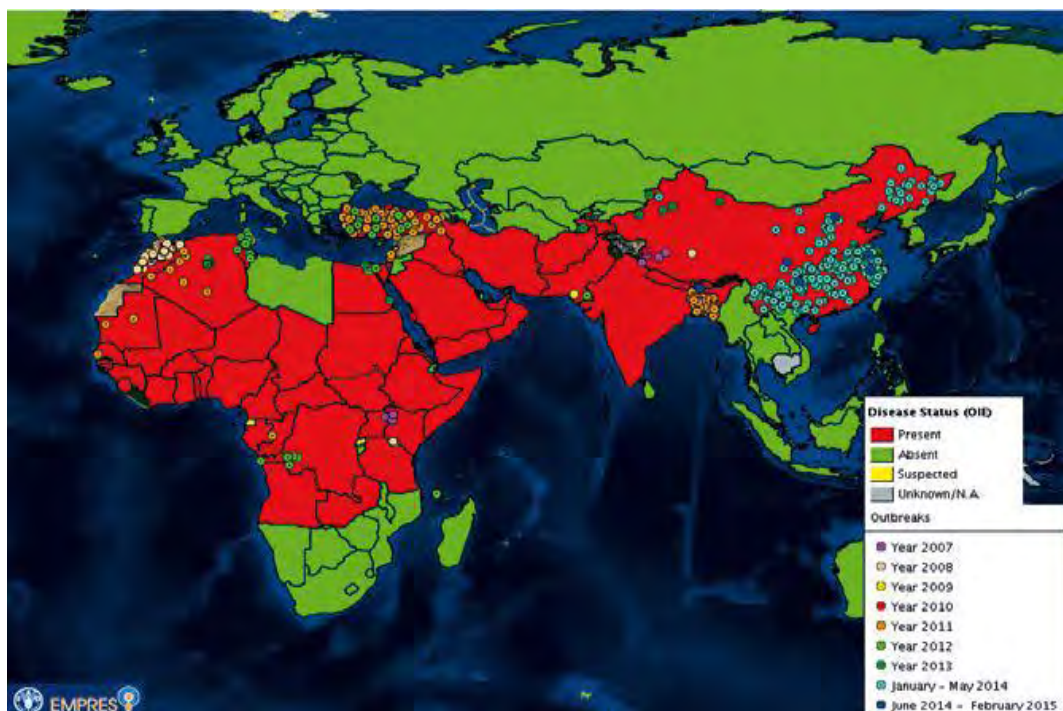


Figure 3: Current global PPR situation and occurrence of outbreaks from 2007-2014.
Source: (OIE WAHIS and FAO EMPRES-I, 2015)

2.5.2. Molecular epidemiology of PPRV

Peste des petits ruminants virus has only one serotype with four distinct lineages (1, 2, 3 and 4) on the basis of partial sequence analysis of fusion protein (F) and (N) genes. These gene sequence analyses of PPR viral isolates have demonstrated the involvement of each of the four PPRV lineages with specific geographical niches. The gene sequence analysis of nucleoprotein (N) has been found to be more precise map marker because of its conserved nature therefore allowing a more precise geographical distribution of different lineages concordant with the historic areas of trade or transhumance of small ruminants in some affected areas (Kwiatek *et al.*, 2007).

Lineage 1 and 2 are found exclusively in West Africa countries, Lineage 3 is found in Eastern Africa and Middle East while Lineage 4 is found in South Asian countries, Middle East and China (Dhar *et al.*, 2002). Sudan has lineage 3 and 4 circulating in the country (Saeed *et al.*, 2010) while recently lineage 4 has been found circulating in Morocco and other North African countries (De Nardi *et al.*, 2011). Lineage 1, 2 and 4 were confirmed circulating in Uganda while Lineage 3 is responsible for PPR outbreaks

in Tanzania (Luka *et al.*, 2012; Kivaria *et al.*, 2013). However, the lineage of the PPRV circulating in Kenya has not been established (Banyard *et al.*, 2010).

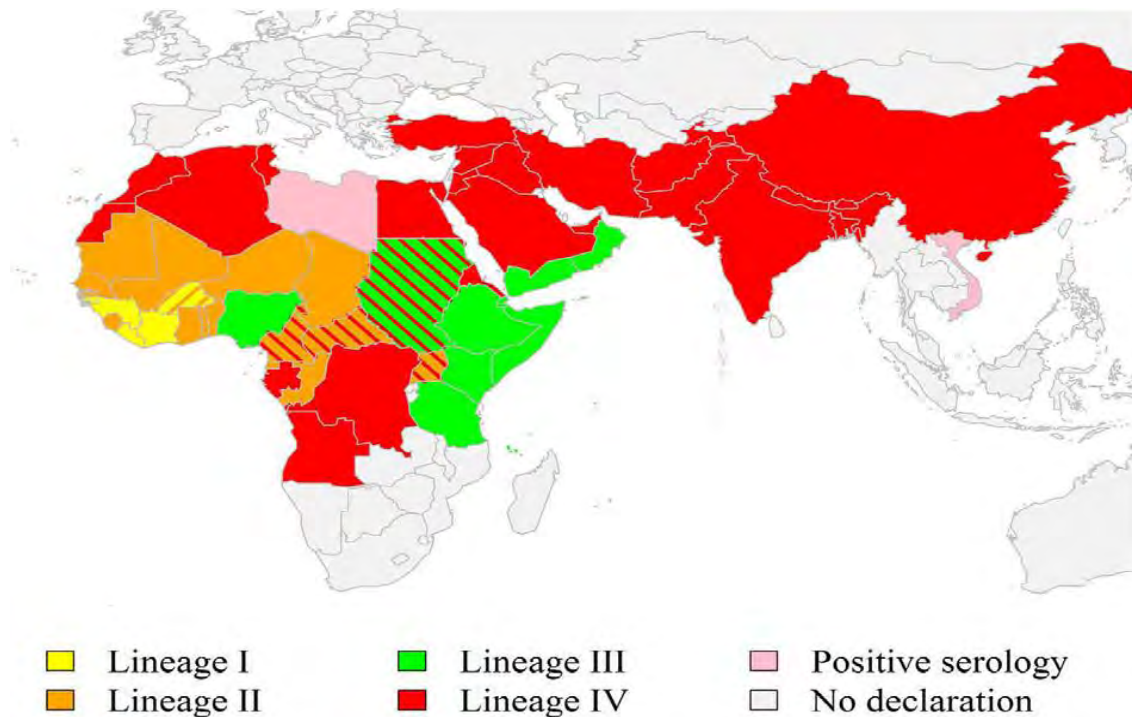


Figure 4: Map showing the worldwide distribution of PPR lineages

Source: (Libeau *et al.*, 2014)

2.5.3. Quantification of PPR by species in different regions of Ethiopia

In Ethiopia, Clinical PPR was suspected in 1977 in afar region, east of the country (Pegram and Tereke, 1981. Roeder *et al* 1994). Clinical and serological evidence of its presence confirmed in 1991 in Addis Abraham *et al.* (1994). Gelagay (1996) has reported that 14.6% of sheep sampled along 4 roads from Debre Berhan to Addis Ababa were seropositive for PPR. Waret-Szkuta *et al.* (2008) has also reported an overall seroprevalence of 1.7% in Oromia, 21.3% in Somalia, Amhara region of Ethiopia. Most recently, an overall seroprevalence record of 30.9% from sheep and goat in pastoral and agro-pastoral area of afar and Gambella region of Ethiopia has been reported Megersa *et al.* (2011).

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 (Gopilo *et al.*, 1991, Roeder *et al.*, 1994).

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

Country	Species	No. tested(*)	prevalence	Reference
Ethiopia(Region)				
Afar, Borena, East	Sheep	835 (cELISA)	13	Abraham <i>et al.</i> , 2005
Shewa, Gambella, Jijga	Goat	442 (cELISA)	9	Abraham <i>et al.</i> , 2005
	Cattle	910 (cELISA)	9	Abraham <i>et al.</i> , 2005
	Camel	628 (cELISA)	3	Abraham <i>et al.</i> , 2005
Afar (Awash Fentale)	Sheep and Goat	23 (cELISA)	36.6	Delil <i>et al.</i> , 2012
North Shewa	Sheep and Goat	-	29	Gelagay, 1996
Gambella(Itang)	Sheep and Goat	779 (cELISA)	27.3	Megersa <i>et al.</i> , 2011
Afar (Adaar)	Sheep and Goat	384 (cELISA)	38.3	Megersa <i>et al.</i> , 2011
Afar	Sheep and Goat	1653 (cELISA)	15.3	Waret-Szkuta <i>et al.</i> , 2008
Amhara	Sheep and Goat	5992 (cELISA)	4.6	Waret-Szkuta <i>et al.</i> , 2008
Benishangul-Gumuz	Sheep and Goat	729 (cELISA)	8	Waret-Szkuta <i>et al.</i> , 2008
Oromia	Sheep and Goat	2290 (cELISA)	1.7	Waret-Szkuta <i>et al.</i> , 2008
SNNPR	Sheep and Goat	1622 (cELISA)	1.8	Waret-Szkuta <i>et al.</i> , 2008
Somali	Sheep and Goat	465 (cELISA)	21.3	Waret-Szkuta <i>et al.</i> , 2008
Tigray	Sheep and Goat	900 (cELISA)	15.3	Waret-Szkuta <i>et al.</i> , 2008

2.5.4. Transmission of the virus

Transmission requires close contact between infected animals in the febrile stage and susceptible animals (Braide, 1981) because of the lability of the virus outside the living host. 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 (Bundza *et al.*, 1988; Taylor, 1984).

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 (Lefèvre and Diallo, 1990). 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.

2.5.5. 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 (Lefevre and Diallo, 1990). Other domestic animals such as camels, cattle and pigs are known to undergo subclinical infection of PPR (Taylor, 1984). The disease has been reported in wild small ruminants in a zoo (Furley *et al.*, 1987) and those living in the wild (Ogunsanmi *et al.*, 2003; Sharawi *et al.*, 2010; Kinne *et al.*, 2010).

2.5.6. Host determinants of the disease

Host determinant factors of PPR spread have been reported in various studies, highlighting age, sex, breed and animal species (Munir *et al.*, 2013). Young animals are less likely to have developed protective antibody titers and therefore are more susceptible to PPRV (Luka *et al.*, 2011). 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 (Waret-Szkuta *et al.*, 2008, Abubakar *et al.* 2009, Singh *et al.*, 2004b; Ozkul *et al.* 2002). 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 (Taylor *et al.*, 1990).

Sex has also been reported as a risk factor for susceptibility/resistance to the disease (Abdalla *et al.*, 2012; Sarker and Islam, 2011; Swai *et al.*, 2009; Waret-Szkuta *et al.*, 2008). 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 (Singh *et al.*, 2004b). 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 (Sarker *et al.*, 2011). However, studies from Pakistan have shown no significant difference between males and females, with respect to susceptibility (Munir *et al.*, 2008).

The influences of breeds of the small ruminants on susceptibility to the disease have also been studied by Munir *et al.* (2008), 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 (Lefevre and Diallo, 1990; El Hag and Taylor 1984; Diop *et al.*, 2005). Goat and sheep species differences have been highlighted as major risk factor for PPRV susceptibility (Swai *et al.*, 2009, Munir *et al.*, 2008, Waret-Szkuta *et al.*, 2008). 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 (Libeau *et al.*, 2011).

2.5.7. 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 (Libeau *et al.*, 2011). 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 (FAO. 2009b, Libeau *et al.*, 2011).

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 (Dufour, 2010). In the Sahel region, sero-prevalence of 75% is observed in pastoralist small ruminants and in most cases the disease is muted or subclinical (Grenfell and Dobson, 1995).

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% (Lefevre and Diallo, 1990). These epidemics in West Africa, which coincide with wet rainy seasons, have been associated with seasonal animal husbandry patterns and livelihood activities among the settled and pastoralist communities (Mai *et al.*, 2004; William and Barker, 2001). However, Opasina and Putt (1985) 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 (Khalafalla *et al.*, 2010), while other studies by Abdalla *et al.* (2012) 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 (Abraham G, 2005, Waret-Szkuta *et al.*, 2008).

2.5.8. *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 (Radostitis *et al.*, 2007).

The disease is transmitted by direct and indirect contact (Carter *et al.*, 1993). Large amounts of the virus are present in all body excretions and secretions, especially in diarrheic faeces. Infection is mainly by inhalation but could also occur through conjunctiva and oral mucosa (Radostitis *et al.*, 2007).

2.5.9. Wild life Susceptibility to the disease

Wildlife susceptibility to PPR is a complicating factor, with infection and clinical disease reported in Dorcas gazelle (in captive groups), Thomson's gazelle, gemsbok and ibex (Wohlsein and Saliki, 2006; Gur and Albayrak, 2010), as well as in wild sheep such as bharals in Tibet and in wild goats in Kurdistan (Bao *et al.*, 2011; Hoffmann *et al.*, 2012). Recently, an outbreak of PPR in truly free-ranging Sindh ibex was confirmed by immunocapture ELISA and PCR in Pakistan in 2010 with 36 deaths, possibly associated with the sharing of water pasture with a presumed infected goat herd (Abubakar *et al.*, 2011).

The role of wild species as a reservoir has not been studied. However, considering the role of wildlife in the epidemiology of rinderpest (Shanthikumare *et al.*, 1985; Anderson *et al.*, 1990; Couacy-Hymann *et al.*, 2005; Kock *et al.*, 2006; Rossiter *et al.*, 2006), further research is needed about potential PPR spread through wild species. This may have serious repercussions in Ethiopia, where several wild ruminant species have the opportunity to contact during grazing and watering.

Table 2: Detection of PPRV in wild life species.

Species	Latin name	Reference
Laristan sheep	<i>Ovis gmelini laristanica</i>	Furley <i>et al.</i> (1987)
Gemsbok	<i>Oryx gazella</i>	Furley <i>et al.</i> (1987)
Dorcas gazelles	<i>Gazella dorcas</i>	Furley <i>et al.</i> (1987)
Thompson's gazelle	<i>Eudorcas thomsonii</i>	Abu-Elzein <i>et al.</i> (2004)
Nubian Ibex	<i>Capra nubiana</i>	Furley <i>et al.</i> (1987)
Indian buffalo	<i>Bubalus bubalus</i>	Govindarajan <i>et al.</i> (1997)
African Grey dukier	<i>Sylvicapra gramma</i>	Ogunsanmi <i>et al.</i> (2003)
Arabian oryx	<i>Oryx leukoryx</i>	Frolich <i>et al.</i> (2005)
Bubal hartebeests	<i>Alcelaphus buselaphus</i>	Couacy-Hymann <i>et al.</i> (2005)
Buffaloes	<i>Syncerus caffer</i>	Couacy-Hymann <i>et al.</i> (2005)
Defassa waterbuck	<i>Kobus defassa</i>	Couacy-Hymann <i>et al.</i> (2005)
Kobs	<i>Kobus kob</i>	Couacy-Hymann <i>et al.</i> (2005)
Arabian mountain gazelles	<i>Gazella gazella cora</i>	Kinne <i>et al.</i> (2010)
Springbuck	<i>Antidorcas marsupialis</i>	Kinne <i>et al.</i> (2010)
Arabian gazelles	<i>Gazella gazelle</i>	Kinne <i>et al.</i> (2010)
Barbary sheep	<i>Ammotragus lervia</i>	Kinne <i>et al.</i> (2010)
Bushbucks	<i>Tragelaphus scriptus</i>	Kinne <i>et al.</i> (2010)
Impala	<i>Aepyceros melampus</i>	Kinne <i>et al.</i> (2010)
Rheem gazelles	<i>Gazella subgutturosa mar</i>	Kinne <i>et al.</i> (2010)
Afghan Markhor goat	<i>Capra falconeri</i>	Kinne <i>et al.</i> (2010)

Source: Ashley *et al.*, 2010

2.5.10. Pattern of the disease

In general, morbidity is common, particularly in fully susceptible goat populations. Mild forms of the disease may occur in sheep and partially immune goat populations. 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% (Lefèvre and Diallo, 1990). While in arid and semi-arid regions, PPR is seldom fatal but usually occurs as a subclinical or in apparent infection opening the door for other infections such as Pasteurellosis (Lefèvre and Diallo, 1990). Though outbreaks in West Africa coincide with the wet rainy season, Opasina and Putt (1985) 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 (Abu Elzein *et al.*, 1990).

Serological data from Nigeria revealed that antibodies occur in all age groups from 4-24 months indicating a constant circulation of the virus (Taylor, 1979b). In Oman the disease persisted on a year round basis maintaining itself in the susceptible yearling population (Taylor *et al.*, 1990). Therefore, an increase in incidence reflects an increase in number of susceptible young goats recruited into the flocks rather than seasonal upsurge in the virus activity, since its upsurge pend on the peak of kidding seasons (Taylor *et al.*, 1990). Moreover, the susceptibility of young animals aged 3 to 18 months was proved to be very high, being more severely affected than adults or unweaned animals (Taylor *et al.*, 1990).

2.6. Current Diagnostic Techniques

Earliest possible diagnosis of PPR is crucial in implementing control measures, to contain outbreaks and minimize economic losses. Initially, the majority of PPR outbreaks were diagnosed based on typical clinical signs. However, the signs of PPR are often difficult to distinguish from those caused by a number of other diseases, such as foot-and-mouth disease and bluetongue disease (Munir *et al.*, 2013). This situation becomes even more complicated when these diseases are circulating in areas where PPR is endemic. Thus, it is necessary to confirm the clinical diagnosis through laboratory testing (Munir *et al.*, 2013). Currently, the diagnosis of PPRV is made based on demonstration of antibodies, which is a good indication because an animal infected with PPRV carries antibodies for life, with the development of a sustained antibody response.

2.6.1. Serological detection

Most of the available diagnostic assays have been developed based on the N protein. Owing to the presence at the 3' end of the genome of PPRV, the N protein produced in quantities higher than any other structural proteins because attenuation occurs at each intergenic region between two genes (Lefevre *et al.*, 1991; Yunus and Shaila, 2012). The antibodies produced against the N protein don't protect the animals from the disease. Due to abundance of the N protein it remains the most acceptable target for the design of PPRV diagnostic tools (Diallo *et al.*, 1994).

Moreover, because the HN protein is the most diverse among all the members of morbilliviruses, RPV and PPRV share only 50% similarity in their HN proteins. The HN protein determines cell tropism; most of the protective host immune response is raised against HN protein. Therefore, serological assays have also been developed targeting HN protein (Munir *et al.*, 2012a, 2013). Commercial ELISAs are available based either on the HN (Saliki *et al.*, 1993; Anderson and McKay, 1994; Singh *et al.*, 2004) or N proteins (Libeau *et al.*, 1995) for specific detection of antibodies against PPRV, in any susceptible host. The sensitivity and specificity of these assays can be as high as 90% and 99%, respectively.

2.6.2. Antigen detection

Immunocapture (Libeau *et al.*, 1994) and sandwich ELISAs (Saliki *et al.*, 1994) are available to efficiently detect antigens in the tissues and secretions of PPRV-infected animals. Both these assays utilize monoclonal antibodies (MAbs) directed against the N protein of PPRV. Both assays are rapid, sensitive and specific with a detection limit of 100.6 TCID₅₀/well. Since the MAbs used in these assays are raised against the non-overlapping domains of the N protein of PPR and RP viruses, this assay can be used to differentiate PPRV- from RPV-infected animals (Libeau *et al.*, 1994). The lateral flow device (LFD)-based test for PPR using monoclonal antibody C77 recognizing the H protein of PPRV (Anderson *et al.*, 1990; Anderson and McKay, 1994).

2.6.3. Genome detection

To overcome several shortcomings of the serological and antigen detections, such as the requirement of sera in well-preserved format, several PCRs have been developed for PPRV with wide range of sensitivities, specificities and detection limits (Munir *et al.*, 2013). Despite the high sensitivity and specificity of these assays, and their validity to detect both vaccine and field viruses, none of the assays is a formally approved OIE method. For this they need further extensive validation. None of the assays is field applicable since they require thermocycler and electrophoresis apparatus for RT-PCR, and real-time PCR for probe or SYBR Green-based assays. However, with the development of LAMP assay, on-site detection can be proposed. It is highly plausible to

combine the simple procedures for RNA extraction using Whatman FTA card (Munir *et al.*, 2012b, 2012c) and using the RT-LAMP assay for isothermal amplification. This could possibly be applied for field diagnosis of PPRV. Recently, a novel and non-amplification strategy was proposed in which two probes complementary to the target sequences (one conjugated to magnetic microparticles, the second to gold nanoparticles labelled with horseradish peroxidase) were used (Tao *et al.*, 2012). On specific binding to the target, the system allows magnetic separation and substrate detection. It was proposed to be quick (45 minutes), cheap and sensitive (17.6 ng/μl) for PPRV detection. This method holds great potential, especially when it is multiplexed for the detection of several pathogens in the same clinical sample.

2.7. 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 of time 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 (Kumar *et al.*, 2013).

2.8. Socio-Economic Impact of PPR

Peste des Petits Ruminants virus has a widespread distribution spanning Africa and Asia (Nanda *et al.*, 1996; Shaila *et al.*, 1996). 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 threatened 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 (Barnyard *et al.*, 2010).

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. *Peste des Petits Ruminants* disease is a constraint to international trade, although this impact is mitigated in local and regional markets due to wide geographic distribution of the disease at present (Elsawalhy *et al.*, 2010). 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 (Bailey *et al.*, 1999).

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 (FAO 2012b; Baron, 2012; Soumare, 2013; Domenech, 2013). An international study conducted by Perry *et al.*, (2002) 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 (Diallo, 2006).

It is estimated that one billion small ruminants or about 62.5% of global domestic small ruminant population is at risk of infection with PPR (FAO, 2009a). 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 (Diallo 2006; Munir *et al.*, 2013).

3. MATERIALS AND METHODS

3.1. Study Areas

The study area was one of the regions selected by STSD (Surveillance of Trade Sensitive Diseases) project of AU-IBAR. By taking Somali regional state as a cluster the study was conducted in Afdher and Liben pastoral livelihood zones. From the aforementioned zones, eight Districts were selected, namely Guradhemole, Goro baqaqsa, Chereti, Dolo Bay, Dolo Ado, Filtu, Hudet and Moyale Districts of Afdher and Liben zones, respectively.

The Somali Region is geographically located in south-eastern part of Ethiopia, between 4° and 11° N latitude and 40° and 48° E longitude. The altitude of the region ranges between 400-1600 meters above sea level (masl), with most areas lying below 900 masl, it is the second largest region in Ethiopia. It is bounded by Kenya and Somalia to the south, the Republic of Djibouti and the Somali region to the north, Somalia to the east and southeast and Oromiya region to the west.

The region covers a total area of 350,000km² consisting of 9 administrative zones, 52 districts (Woredas) and 703 PAs. The 9 zones are Jigjiga, Shinile, Liban, Afdher, Godey, Korahay, Warder, Dagahbrur and Fik. The major perennial rivers in the region are Wabi Shebelle, Genale, Dawa and Weyib.

Afdher administrative zone

Afdher Zone is one of the nine zones in the Somali National Region State of Ethiopia and it is located in the south west of the region. The zone borders Somalia to the east, Gode zone to the north-east, Liben zone in the west and Fiq zone to the north. Afdher consists of eight districts: Harghelle (capital), El-Karre, Goro Baqaqsa, Guradamole, Dollo Bay, Charati, Barey and West Imey.

There are two distinct agro-ecological areas in Afdher zone: the “highland” area (Gora-damole, Goro-baqaqsa and parts of El Karre) and semi-arid lowlands (Barey, Harghelle, Dolo-Bay, Charati, and West Imey). The typical vegetation coverage of the

zone is classified as open shrub and grassland areas (lowland semi-arid areas) and thick vegetative (thorny) bush (highland areas). The zone has three rivers running through: Ganale and Shabelle are permanent; the Wayb is seasonal.

Rainfall is low and erratic, averaging only around 250-300 mm per year, although annual precipitation is relatively higher and more stable in the highland areas. As in much of Somali Region, the first rainy season is the *gu*; it is relatively more important for production and occurs from April to June. The second season is called the *deyr*; it takes place from October to December. The *karan* rains in neighboring areas of Oromia (Bale zone) sometimes extend into parts of the higher altitude areas. The dry periods of the year are the *hagaa*, from July to September, and the *jilaal*, from January to March.

The main sources of water for animals and people include the three rivers mentioned above; boreholes, hand-dug wells and seasonal ponds, which dry up during the dry season. Despite the rivers, water scarcity is a major problem in parts of this livelihood zone in most years. The highland areas between Hargelle and Adadle Districts and parts of Gorobaqqa are especially insecure in terms of water. A salt-mine (*God-cusbo*) is found in the area; it provides an income sources for some better off and middle households, but its benefits do not extend widely throughout the livelihood zone (SC-UK, 2001).

Guradhemole district

Guradhemole is one of the Districts in the Somali region of Ethiopia. Part of the *afder* zone, guradhemole is bounded on the south by the Genale dorya river which separates it from the *liben* zone, on the west by *kersa dula*, on the north by the Oromia region, and on the east by *goror bekeksa*. Towns in this district include *harodube* and *kundi*.

The altitude of this District ranges from 200 to 1500 meters above sea level. The other perennial river in the guradhemole is Genale or Ganaane Dorya Mena River, Dumal and Webielan. Guradhemole has a very green highland, as of 2008, this district has neither all-weather gravel road nor any community roads; about 12.3% of the total population has access to drinking water (Kene, 2008).

Gorobaqasha district

Part of the Afder Zone, Goro Bekeksa is bounded on the south by the Ganale Dorya River which separates it from the Liben Zone, on the west by Guradamole, on the north by the Oromia Region, on the northeast by Elekere, and on the southeast by Cherti.

Chereti (Weyib) district

Cherti is bordered on the southwest by the Ganale Dorya River which separates it from the Liben Zone, on the west by Goro Bekeksa, on the north by Elekere, on the east by Afder, and on the southeast by Dolobay. The altitude of this district ranges from 750 to 1700 meters above sea level. Other rivers in Cherti include the Mena and the Weyib.

Dolo Bay district

Dolobay is one of the districts in the Somali Region of Ethiopia. Part of the Afder Zone, Dolobay is bordered on the south by the Provisional Administrative Line with Somalia, on the west by the Ganale Dorya River which separates it from the Liben Zone, on the northwest by Chereti, on the north by Afder, and on the east by Bare.

Liban Administrative Zone

Liban Zone is one of the nine administrative Zones of Somali National Regional State (SNRS). The Zone is located in the extreme south-western corner of the Region and has borders with Kenya on the south, Afder Zone (SNRS) on the east and Oromia Region on the north and west. The Zone has got three districts, namely Filtu, the capital, Dolow Ado and Moyale.

The altitude ranges from 300 to 1500 meters above sea level. This is a semi-arid zone, with temperatures reaching as high as 38° C in the hottest periods of the year (March to May) and dipping to 19° C during cooler times (July to September). Rainfall is bi-modal, with the *gu* season occurring from April to June and the *deyr* from October to December; annual rainfall ranges between 300–550 mm. The rest of the year is dry. The

jilaal season, from January to March, is a time of particular hardships and resource scarcity; the *hagaa* season, from July to September, is also dry, but slightly less difficult.

The ground cover consists of swaths of open grass, broken by shrubs, bushes and forests. In Filtu and Dekasuftu districts, the topography is flat in the central areas of the livelihood zone gradually descending to the east towards the Ganale River and to the west towards the Dawa River. Hills and mountains increasingly become prominent features in the areas around the two rivers. Most of this livelihood zone's population within Elkare is located in elevated mountainous areas. Soil types include black clay which is fertile mainly around the river basins and at Hayadimtu; scattered red soils are found in Seru and parts of Ayinle; and black reddish mixed soils are mostly found in Elkare (SC-UK, 2001).

Filtu districts

Filtu is one of the districts of Liben zone, located in the northern part, which is in the Somali Regional State at 120 km to the east of Negelle town, Oromia region. This is at a frontier line between the traditional territories of the Somali and Oromo groups occupying the south western part of Ethiopia. Two perennial rivers, Ganale and Dawa, traverse it, and define the eastern and western boundaries of both Dollo Ado and Filtu districts.

It is bounded by other Liben zone districts; Dollo Ado and Moyale in the south, by Afdher zone of Somali Regional State in the east, and by Guji Zone of Oromia Regional State in the west and north. The topography is generally characterized by flat to gentle slopes with altitude of 1264 m a.s.l. The area is also described by rock outcrops, Savanna-Acacia-Commiphora woodland in the lower landscapes and scrub vegetation on the escarpments.

Filtu district is located at south eastern part of the country under Liban zone of Ethiopian regional state and 730km away from Addis Ababa with total population of 140,978 according to 2007G.c population and housing conducted in Ethiopia. 85 percent of population of district is living in rural areas with pastoral and agro pastoral livelihood.

The socioeconomic status of people highly depends on livestock. The district is bordered by Afder zone in Northeast, Dolo Ado district in the east, Dekasuftu district in west and Mubarek and Moyale district to south east. The district has 28 PAs. Geographically the district covers 17,000 km². In Filtu district there are two rivers Genale and Dawa which follow parallel on North and Southern border of district. Filtu district receives average rainfall 250-500 mm. The district temperature ranges from 27-35 degree celsius (SC-UK, 2001).

Dolo Ado districts

Dolo-Ado is located in the angle formed by the confluence of the Ganale River with the Dawa River, and bordered to the west by Mubarak (one of the six districts Liben Zone comprises), to the northwest by Filtu (one of the six districts Liben Zone comprises), to the southeast by Somali, on the north and east by Afder Zone, and on the south by Kenya. Its topography is more of plane. The altitude of the district ranges from 200 to 1000 meters above sea level and the annual temperature ranges between 38°C and 42°C.

Dolo-Ado district is found in two livelihood zones Dawa-Ganale Riverine livelihood and Filtu-Dollo pastoral. Dawa-Ganale Riverine the main livelihood activities are irrigated farming along the Dawa and Ganale rivers and livestock keeping mainly of shoats and cattle.

The Dawa-Ganale Riverine is the main livelihood group in the district with about 70% of the population falling in this livelihood group. Milk and milk products forms the backbone of the daily household survival among this group. Milk provides an important nutritional component for children and when made into ghee combines a high energy value with long storage. Mandera is the main regional livestock serving both neighboring Somalia and Ethiopia regions. Livestock sold in this market is then traded in Kenya livestock market.

Like Filtu, Dolo Ado has a bimodal rainfall seasons; the main rainfall season from March to May which in Somali language called “gu’u” and the short rainfall season from

October to November, “dayer”. The average rainfall that the area gets is less than 400 mm (SC-UK, 2001).

Hudet districts

Hudet is among the 6 districts of Liban zone. The district is administratively divided into 13 major and 13 minor PAs. It is situated in southern Ethiopia or south west of Somali regional state. It is bounded by the north Liban district of Guj zone (Oromia), to the west Arero district of Borena zone (Oromia), to the south Dhas district of Borena zone (Oromia), to the east Mubarak district, south east Moyale district of liban zone, and to the north east Filtu district of liban zone.

It occupies an area of 14400 km². It falls between 39-41° longitude and between 4° and 45" latitude. The topography of the district is mostly characterized by low land and hilly especially around river Dawa with elevation of between 852-1400 above mean sea level with average annual rainfall of 700mm-1100mm.

Moyale district

Moyale district of Liben Zone is one of the three districts found in Liben Zone of Somali National Regional State, Ethiopia. Mandera and Moyale districts of Kenya border the district to the South and Southeast. The Dawa River forms the eastern border. The North and Northwest sides of the district border Borena Zone of Oromia Region. The district is very large with about 271,971 km² of land.

The altitude ranges between 900-1100m above sea level: thus the land is classified as Kolla (lowland). The majority of the population dependent on livestock: they are pastoralists. Camel keeping is dominant followed by cattle and shoats. The population mostly lives in scattered and temporary settlements. Some 90,657 km² of the district, or about 33%, is suitable for agriculture use. However, the area is prone to frequent drought and hence the amount of land cultivated is usually low because of lack of rainfall (SC-UK, 2001).

3.2. Study Animals

The study population used were black head ogaden sheep and Somali goat breeds aged 6 months of age or older for sampling to avoid those with maternal immunity (Ata *et al.*, 1989, Awa *et al.*, 2002, Balmurgan, 2012). The date of the last vaccination in the kebele (PAs) was determined from District archives, by interviewing field veterinarians and community animal health workers (CAWS). Animals born since the last vaccination were sampled using dentition.

3.3. Study Design, Sampling Method and Sample Size Determination

A cross sectional study design was used with a 2 stage clustered sampling scheme during November to May 2015–2016. This study was part of the national survey supported by AU-IBAR through STSD (Surveillance of trade sensitive Disease) project and coordinated by the Ministry of Livestock and Fisheries (MoLF). Therefore, out of the designated regions at national level, the study was undertaken in part of Somali regional state taking two zones (Afdar and Liben) as a cluster.

Sampling frame were list of PAs in the study Woredas (districts). Multi-stage sampling with a 3 stage sampling technique were implemented (PA>village>Herd); the PAs were preselected randomly using point coordinates (CSA-OUCHA, 2014) from the list of PAs available, and the districts where the PAs dropped were included in the study. Three villages were selected purposively based on accessibility and willingness of herd owners for each selected PA, from the herds in each village one flock was selected conveniently based on herd owner's willingness to bled their animals. A village was considered to be a "herd", which is made up of a number of household herds. Within the selected herd 14 sheep and goats (cumulative sum of both species) were selected randomly.

Hence, the number of herds required to estimate the prevalence of an event was calculated using *promesa software* (<http://www.promesa.co.nz/>). The sample size calculation used an absolute error of 5%, confidence interval of 95%, with expected prevalence of infected flocks of 30% (Appendix 4) and assuming an intra-cluster correlation coefficient (measure of variation with clusters) of 0.2. To account for

clustering with PA and herd, a design effect of 2 was used (Dohoo *et al.*, 2003). In the study 30% and 20% used for animal and herd level prevalence respectively were estimates obtained after reviewing different published literatures (Appendix 4).

The number of sheep and goats to be sampled within each herd was calculated using a formula at least to detect one seropositive animal as described by Martin *et al.* 1987 with confidence level of 95%, assuming the expected within herd prevalence of 20% and average herd size of 200 for sheep and goat flocks. Accordingly, 14 sheep and goats were sampled per flock.

$$k = \lceil [1 - (1 - \alpha)^{1/d}] \left[\frac{N - (d - 1)}{2} \right] \rceil = 14$$

Where k is the number of animals to sample within each herd, α the probability of observing at least one seropositive animal in the sample when the infection affects at least d/N of the animals in the herd, d the expected number of infected animals in a herd, and N is the average herd size.

Therefore, 22 PAs and 66 herds were the required sample size for the Afder and Liben zones of Somali cluster. However, due to the security issue we encountered we only included 19 PAs. Accordingly, 19 PAs, 3 villages per PA with a total of 57 herds were used to get a sample size of 798 (216 sheep, 582 goats).

Table 3: Zonal population and respective number of small ruminants sampled.

Zones	Zonal population		District selected	No. of sample	Kebeles (PAs) selected
	Sheep	Goat			
Afdar	592323	356503	Guradhamole	84	Harodube, Kundi
			Gorobekeksa	126	Hargedeb, Tour Hagermokor,
			Chereti/Weyib	42	Qorder
			Dolo Bay	84	Row-Row, Entoy
Liben	288932	329672	Dolo Ado	252	Bur Amino, Kole, Mulka
			Filtu	42	Dida, Suftu, Fiyo, Wedlehube
			Hudet/Udet	84	Bod
			Moyale	84	Kalehargessa, Dire dima
Total sample size				798	Darselam

Source: (CSA, 2009)

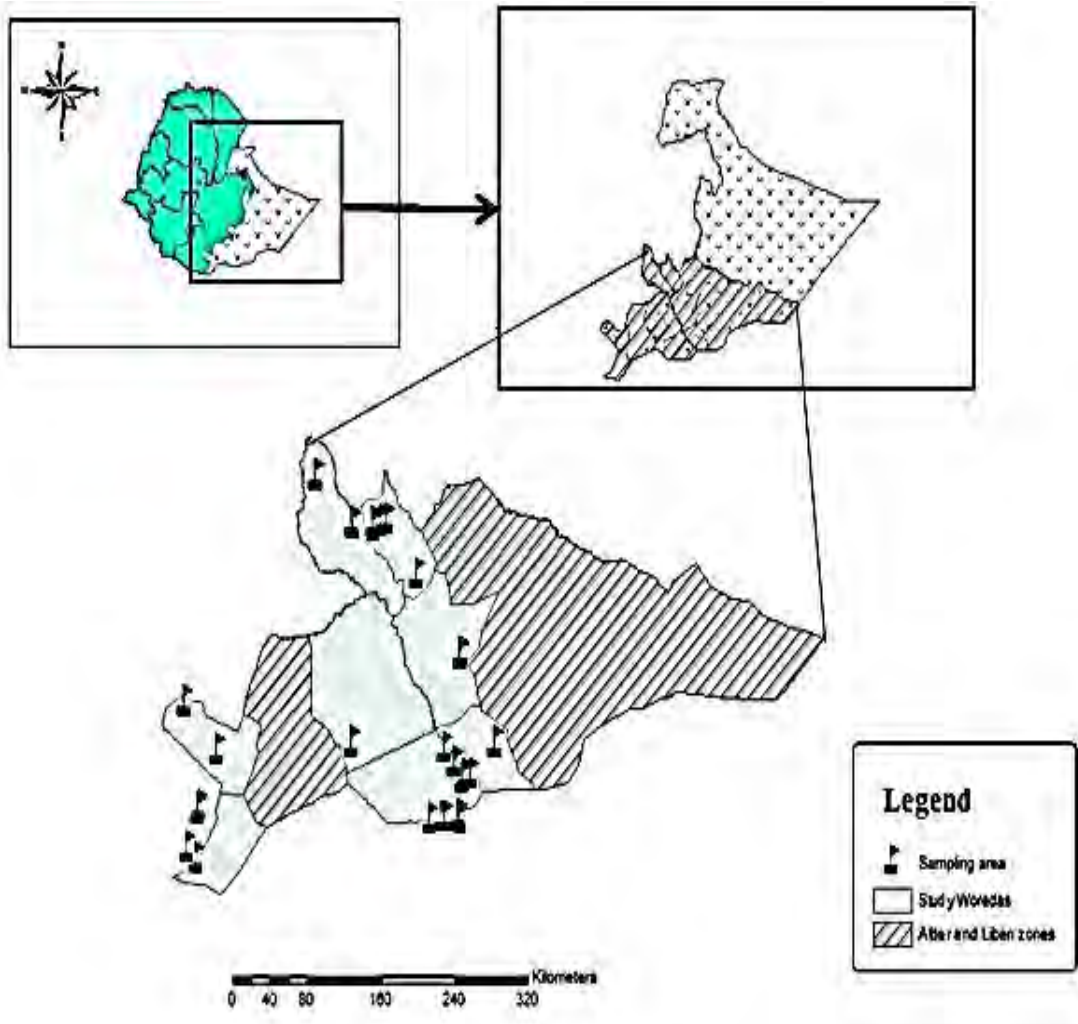


Figure 5: Map of Ethiopia with Study areas and region.

3.4. Inclusion Criteria

Small ruminants considered for sampling in this study were those animals that were not vaccinated against PPR before. This was to eliminate the possibility of sero-positivity due to vaccinal antibodies. Also, in order to avoid sero-positivity due to maternal antibodies, only sheep and goats greater than six months old were considered for sampling. Age was determined using dental pattern (dentition) (Payne, 1990 and ESGPIP, 2009).

3.5. Retrospective Epidemiological Analysis on PPR Outbreak

The data on PPR disease outbreaks used in the present study were obtained from the Ministry of Livestock and fisheries (MoLF). The data used were for a 10-year period (March 2006- August 2015) with the expectation that inferences based on these data would provide a snapshot of the current PPR disease situation in the country given that outbreaks occur every year. Also, PPR is a reportable disease in Ethiopia and as such all outbreaks of the disease must be reported to MoLF immediately. The information obtained this way therefore could reflect the disease situation in the country. The variables of interest included: time (year and month) and place (regions) where outbreaks occurred and the vaccine doses used for control following the disease outbreak.

3.6. Questionnaire Survey and Field Investigation

Verbal consent was obtained from the respondents, and the objectives of the survey were explained to them before the start of the interview. The interviews were conducted in the local language (Somali) using a translator. Two questionnaire formats, one for the serum sampled individual animal history and the other with a structured questionnaire format for the herders, were developed and used in this study. By doing so; risk factors that have possible association with the occurrence of PPR were investigated and used to support serological results.

The questionnaire was a pre-tested and adjusted in the field as required. In each village, flock owners were interviewed to reveal information regarding flock size, age, sex, and vaccination history.

Health status data was collected by recording history of disease outbreak or occurrence, its clinical signs, overall number of sick animals (used to compute morbidity) as well as overall and specific deaths associated with PPR. Flock management data included access to animal health and extension services (presence, type and frequency of services), inter-herd contact, animal movement, watering, grazing, live animal market visiting frequency and addition of new animals, and suspected source of the infection.

3.7. Serological Study

A total of 798 serum samples from 216 sheep and 582 goats were collected belonging to various districts as shown in Table 3. Initially, blood samples were collected from the jugular vein of each animal using plain Vacutainer tubes. The samples were labelled accordingly to allow identification of each animal and flock, labelled and kept in a slanted position overnight to allow serum separation from clotted blood samples. Serum was decanted and aliquoted into 1.5 mL cryovial and kept in an icebox before being transported and stored to the diagnostic laboratory. Finally, the serum samples were shipped in a cool box chilled on ice packs to the National Animal Health Diagnostic and Investigation Center (NAHDIC) at Sebeta, where serological analysis was carried out.

A monoclonal antibody (MAb) based competitive Enzyme Linked Immunosorbent Assay (cELISA) (Diallo *et al.* 1995) was used for the detection of antibodies in sera to PPR virus using commercial competitive ELISA kit as described by Libeau *et al.* (1995). According to the manufacturer, the specificity and sensitivity (for both animal and flock levels) of this cELISA are 99.4% and 94.5%, respectively. Briefly, the ELISA plates were coated with PPR antigen; the unbound antigen was washed away using buffer then samples were added; Rabbit anti-mouse-antibody horseradish peroxidase (HRPO) conjugate was added and incubated with constant agitation in each stage. Substrate solution (O-phenylenediamine dihydrochloride containing H₂O₂) was added allowing for a colour reaction to develop which was halted with the addition of an equal volume of 1 M H₂SO₄.

Finally, for each species and flock level, the apparent prevalence (AP) was estimated as: $Ap = y/n$, where y denoted the total number of animals positive for PPRV antibodies out

of the sample size, n. After adjusting to the specificity and sensitivity of the ELISA kit the true prevalence (TP) was calculated as:

$$\text{True prevalence (\%)} = \frac{\text{AP} + \text{Sp} - 1}{\text{Se} + \text{Sp} - 1} \times (100)$$

3.7.1. Principle of the test

The c-ELISA used in this study detects IgG type of antibody. The principle of the test is based on the inhibition of binding of the mouse monoclonal antibodies (Mab), directed against the nucleoprotein (NP) antigen of the PPR virus, in the presence of a positive serum. The presence of antibodies to PPR virus in the test serum blocks the reactivity of the monoclonal antibody resulting in a reduction in the color following the addition of enzyme labelled anti-mouse antibody and substrate (Anderson *et al.*, 1990).

3.7.2. Interpretation of the test

The ELISA micro plates were read with an immune-scan reader (Flow laboratories, UK) with an inference filter of 450 nm and connected to a computer loaded with ELISA Data Information (EDI) software for automated reading and calculation of the percentage inhibition (PI) values. The OD (Optical Density) values were converted to percentage inhibition using the following formula:

$$\text{PI} = 100 - \frac{(\text{Optical density of the test wells})}{\text{Optical density of the Mab control wells}} \times 100\%$$

The test serum samples showing PI value of 60 or above were taken as positive for PPR antibodies, PI value less than 50 were taken as negative while PI greater than 50 and less than or equal to 60 were taken as doubtful.

3.8. Data Analysis

Data obtained from both serological tests and questionnaire survey were stored in Microsoft excel spreadsheet (Microsoft Corp. 2016). These data were analyzed by descriptive statistics, univariate, multivariate and multilevel mixed-effect logistic regressions using Stata 13 (Stata Corp, Texas, USA).

Univariable models were first run to assess the relationship between PPR antibody seroprevalence and individual risk factors for PPR sero-positivity. The risk factors assessed included sex, age group, vaccination status and administrative division (PAs). A variable inclusion criterion in the regression model was set to be $P \leq 0.3$ to avoid loss of known disease predictor due to the random effects. Prior to logistic analysis, collinearity between exposure variables was checked. A multivariable logistic regression model was subsequently built using significant variables in the univariable analysis by extending the univariable model to include other risk factors. In the latter analysis, all the significant risk factors were initially included in the model. Model building used backwards elimination method to decide on the factors to exclude from the model using the likelihood ratio test ($P < 0.05$).

The strength of association between the risk factor and PPR sero-positivity was estimated using the odds ratios (OR) which were directly derived from the coefficient estimates from the logistic regression models. The odds ratio is a relative measure of risk that describes how much more likely it is that an animal which is exposed to the risk factor under analysis will develop the outcome as compared to an animal which is not exposed. If the odds ratio is 1, the risk factor is unlikely to be associated with the risk of PPR sero-positivity. For an odds ratio greater or less than 1, the likelihood that the risk factor is associated with risk of sero-positivity increases, and the stronger the association.

The relationship between PPR infection sero-status and the significant risk variables was finally evaluated by fitting mixed-effect models with the sub-location as a random effect. The latter step was carried out to provide, as much as possible, statistically unbiased estimates of sero-prevalence with associated uncertainty adjusted for clustering of PPR sero-positivity responses within sub-locations.

The villages where sampling took place were pointed out by taking GPS coordinates and elevations and a map was generated using Arc GIS version 10.2 showing the spatial distribution of the disease in region and specific Districts along with hierarchical PPR prevalence.

4. RESULTS

4.1. Serological Analysis of PPR Antibody Prevalence

4.1.1. Frequency Curves of Antibody Distribution

Among the samples considered negative for PPR (% color inhibition of less than 50%) the greatest number of samples had a percent color inhibition of between 30 to 39 and 20 to 35 for goats and sheep, respectively. Alternatively, the samples that were positive for PPRV in sheep and goats showed a peak frequency distribution between 80% and 96% in goats and between 90% and 95% in sheep (Figure. 6).

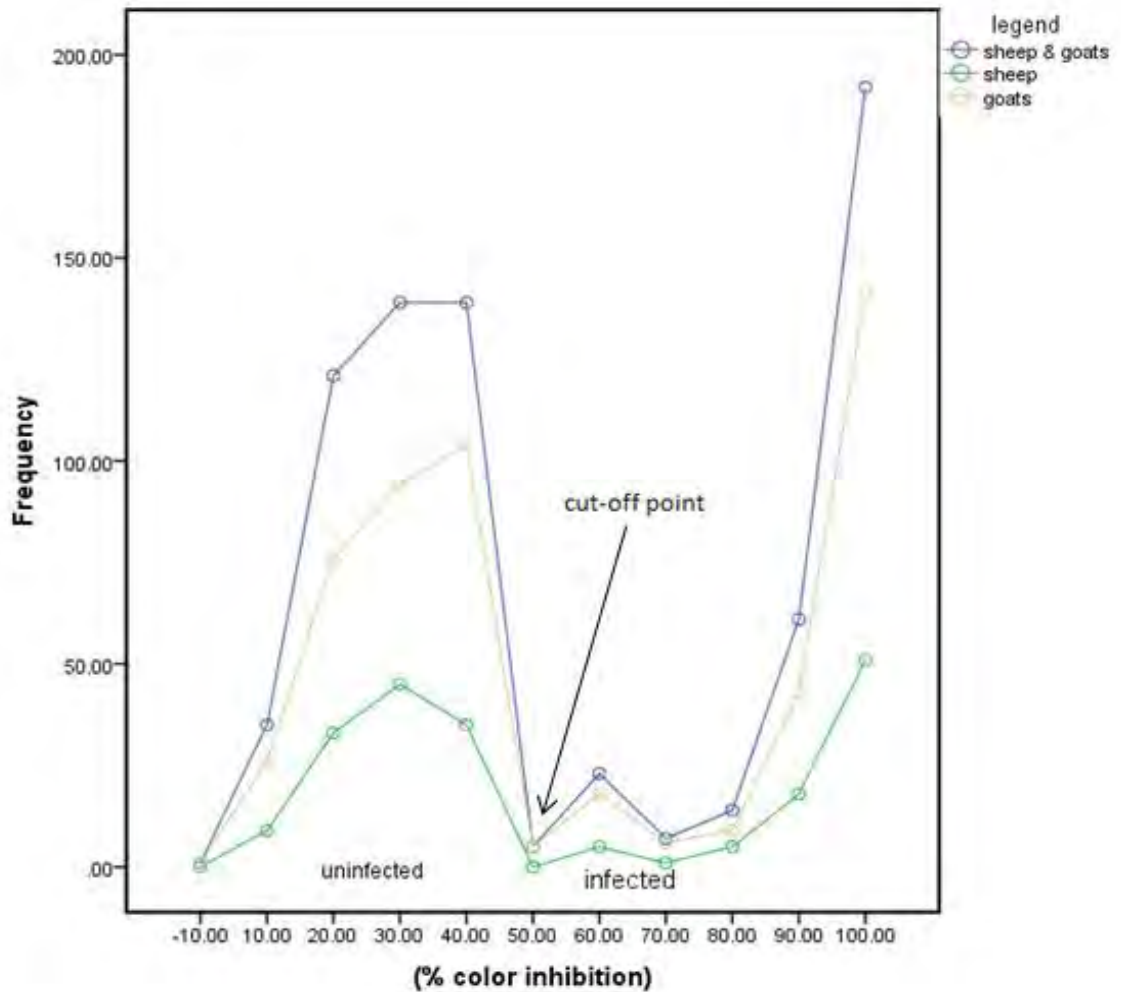


Figure 6: Percent inhibition of PPR sero-positive in sheep and goats

4.1.2. Individual and flock level prevalence

The individual animal apparent prevalence of PPR antibodies in sheep and goats was 39% and 42%, respectively. After adjusting to the PPR ELISA sensitivity and specificity, the true individual prevalence was 41% and 44% in sheep and goats, respectively. However, the apparent prevalence at flock level was 98%. The true flock prevalence was estimated to be 104% while the overall true prevalence of PPR disease was recorded as 43%.

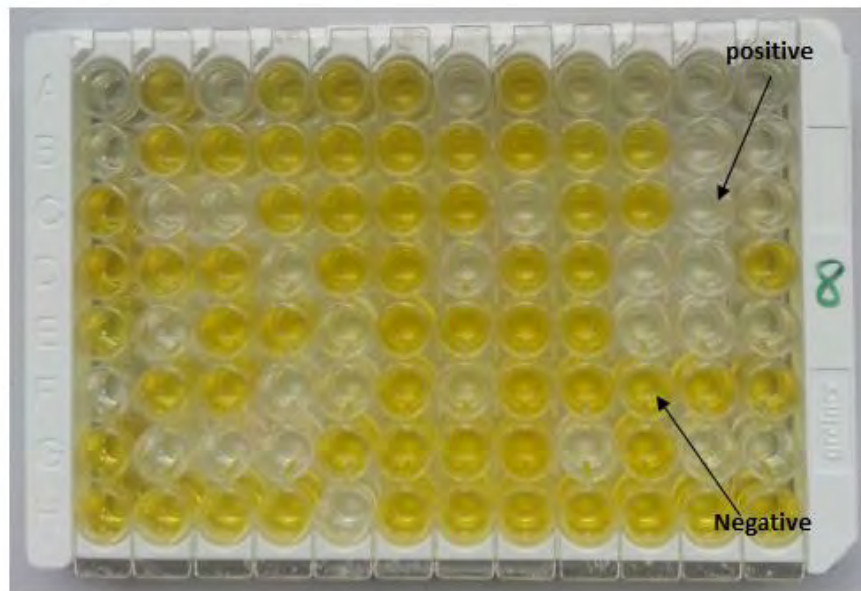


Figure 7: c-ELISA microplate showing positive and negative reactions

4.1.3. Spatial distribution of PPR virus antibody by geographical divisions

A total of 798 serum samples were obtained from small ruminants greater than six months old and with no history of vaccination against PPR. Out of 798, 326 sera tested positive with an overall PPR antibody prevalence of 41% while the herd prevalence was 98% (56 positive out of 57 herds). Antibodies to PPR were detected in small ruminants from all the Woredas (Districts) sampled. All flocks tested had at least one seropositive animal except in a village called Melkadida where all were negative. The highest seroprevalence result was found in Dolo Bay Districts (52%) followed in decreasing order of prevalence by Dolo Ado (42%), Hudet, Chereti, and Gorobekeksa districts have recorded

similar antibody prevalence of (40%), Guradhemole (38%), Filtu (36%) and Moyale (30%). The differences between sero-positivity across Districts were found insignificant (Table. 4).

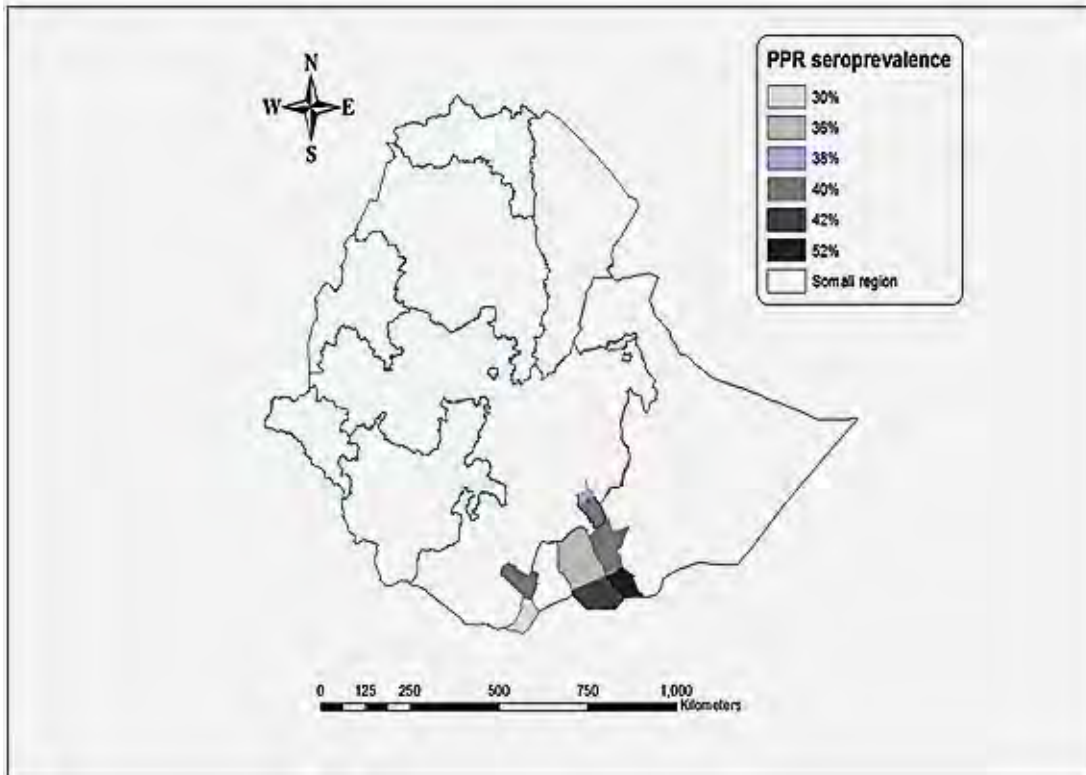


Figure 8: Geographical distribution of PPR sero-prevalence in selected Districts of Somali region.

Table 4: Prevalence of PPR in different Districts.

District	Sample tested	Sero-positives	% sero-prevalence (95% CI)
Guradhemole	84	32	38(28-49)
Gorobekeksa	126	51	40(31-50)
Chereti	42	17	40(26-57)
Dolo Bay	84	44	52(41-63)
Dolo Ado	252	105	42(36-48)
Filtu	42	15	36(22-52)
Hudet	84	34	40(30-52)
Moyale	84	25	30(20-41)
Total	798	326	41(37-44)

Chi square (X^2) = 7.46; p= 0.382

Kole PA had the highest sero-prevalence (64%) while the lowest sero-prevalence was recorded in Suftu PA (12%). There was a strong association between sero-positivity with the studied PA ($X^2= 53.3$; p= 0.000). All PAs had at least one sero-positive animal to PPR antibody (Table 5).

Table 5: Prevalence of PPR virus antibodies at PA level.

Kebele (PA)	Animal tested	Sero-positives	% sero-prevalence (95% CI)
Harodube	42	14	33(20-50)
Kundi	42	18	43(28-59)
Tour	42	17	40(26-57)
Hargedeb	42	15	36(22-52)
Hagermokor	42	19	45(30-61)
Qorder	42	17	40(26-57)
Entoy	42	26	62(46-76)
Row-Row	42	18	43(28-59)
Bur-Amino	42	21	50(34-66)
Kole	42	27	64(48-78)
Melkadida	42	10	24(12-39)
Suftu	42	5	12(4-26)
Fiqo	42	24	57(41-72)
Wedlehube	42	18	43(28-59)
Bod-Bod	42	15	36(22-52)
Dire dima	42	23	55(39-70)
Kalehargesa	42	11	26(14-42)
Kojowa	42	15	36(22-52)
Darselam	42	13	31(18-47)
Total	798	326	41(37-44)

Chi-square(X^2) = 53.3 and p = 0.000

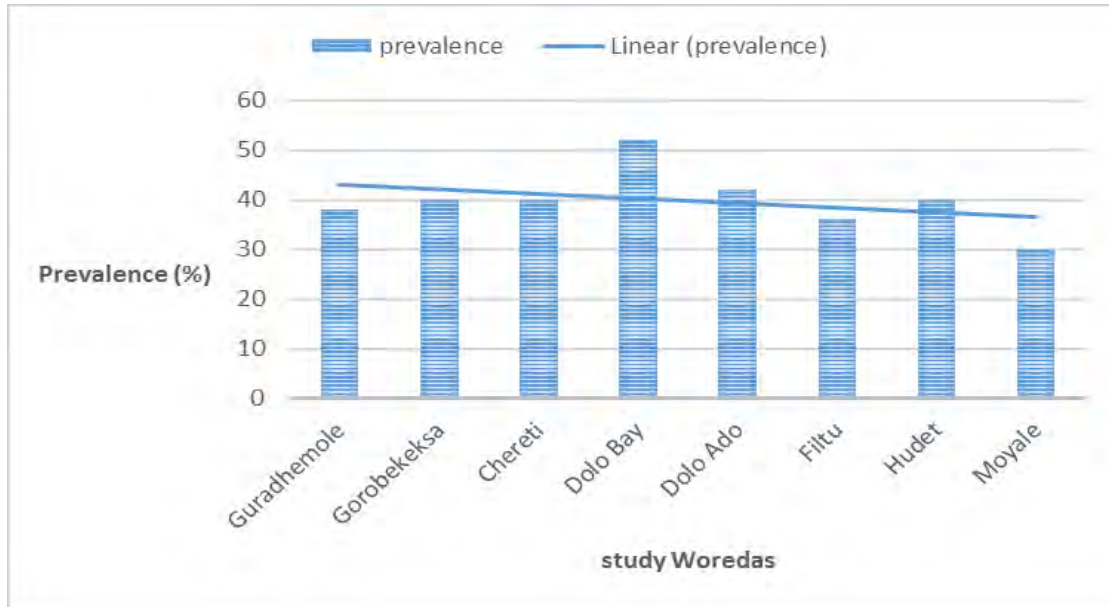


Figure 9: Distribution of PPR antibody across Districts

4.1.4. Species-wise PPR virus antibody distribution

Distribution of PPR sero-prevalence among sheep and goats is summarized in Table 6. Out of the 582 goats sampled, 242 (42%) were positive for PPR antibody. In the case of sheep, 84 (39%) out of the 216 sampled were positive for PPR virus antibodies. The seropositivity difference between the two species was not statistically significant. ($X^2 = 0.4920$; $P = 0.483$) as shown in Table 6 below.

Table 6: Prevalence and chi square test of PPR antibody in sheep and goats

variables	Animal tested	seropositive	Negative	Prevalence (%) (95%CI)
Species				
Sheep	216	84	132	39(32-46)
Goats	582	242	340	42(38-46)
Total	798	326	472	41(37-44)

Chi square (X^2) = 0.4920 and $P = 0.483$

4.1.5. Age-wise PPR sero-prevalence

Age wise distribution of PPR virus antibodies in small ruminants in the Districts has been determined in this study (Table 7). Out of the all age groups sampled, small ruminants aged 36-48 months had recorded the highest PPR sero-positivity rate of 58% while those aged 6-12 months had the least sero-prevalence of 24% with significant difference ($X^2 = 42.5533$; $P = 0.000$).

Table 7: Distribution of PPR in small ruminants.

variables	Animal tested	seropositive	Prevalence (%) (95%CI)
Age group			
6-12	167	40	24(18-31)
13-24	239	87	36(30-43)
25-36	248	117	47(41-54)
36-48	144	83	58(49-66)
Total	798	326	41(37-44)

Chi square (X^2) = 42.5533 and $P = 0.000$

Sheep and goats sampled in this study have been categorized into 4 distinct age groups: group 6-12month age group, 13-24month age, 25-36month age and 36-48month. Figure 11 below compares the sero-prevalence of small ruminants in different age groups and generally goats had a greater sero-prevalence than sheep in each age category. In goats, the risk of being positive to PPR increased as age increases while it decreases in sheep (Figure 11). The probability of being positive to PPR antibody increases when age increased in the small ruminants (Figure 10).

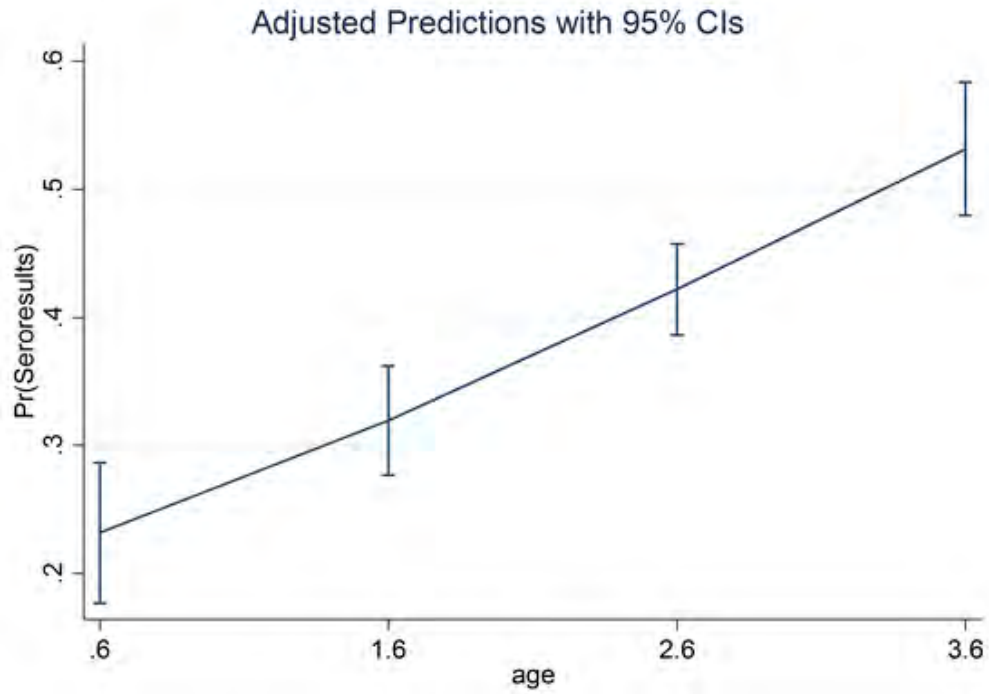


Figure 10: Probability of being sero-positive (95% CI) in line with age.

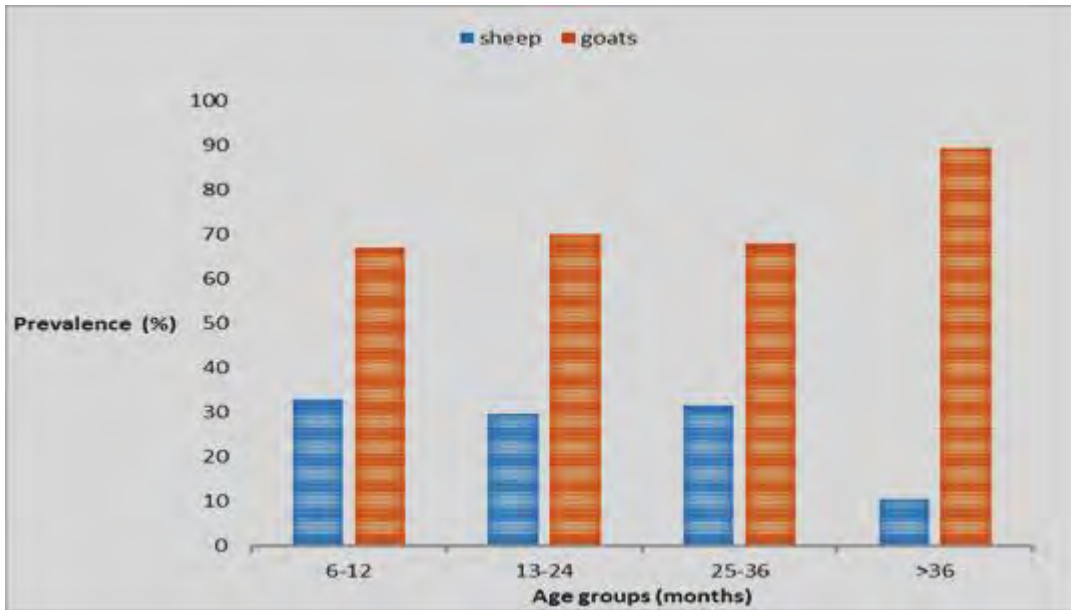


Figure 11: Sero-prevalence in different age categories of sheep and goats

4.1.6. Sex-wise sero-prevalence

Result for the sex- specific rates of PPR virus antibodies among small ruminants sampled is shown in Table 8. From the results, it is evident that females had a statistically greater sero-prevalence of 44% compared to the males whose sero-prevalence was estimated to be 26% (P=0.000). Females were 2.5 times more exposed than males in both sheep and goats (OR=2.5). Female and male sheep had recorded apparently higher prevalence of 42% and 41% respectively than female and male goats with 40% and 29% sero-prevalence respectively (Figure 12).

Table 8: Distribution of PPR virus antibodies in female and male small ruminants

Variables	Animal tested	Sero-prevalence (%) (95% CI)	Odds Ratio (OR)
Sex			
Male	143	26(19-34)	1*
Female	655	44(40-48)	2.5

Chi-square (X^2) = 16.4 and P= 0.000; CI: Confidence interval; 1*: Reference

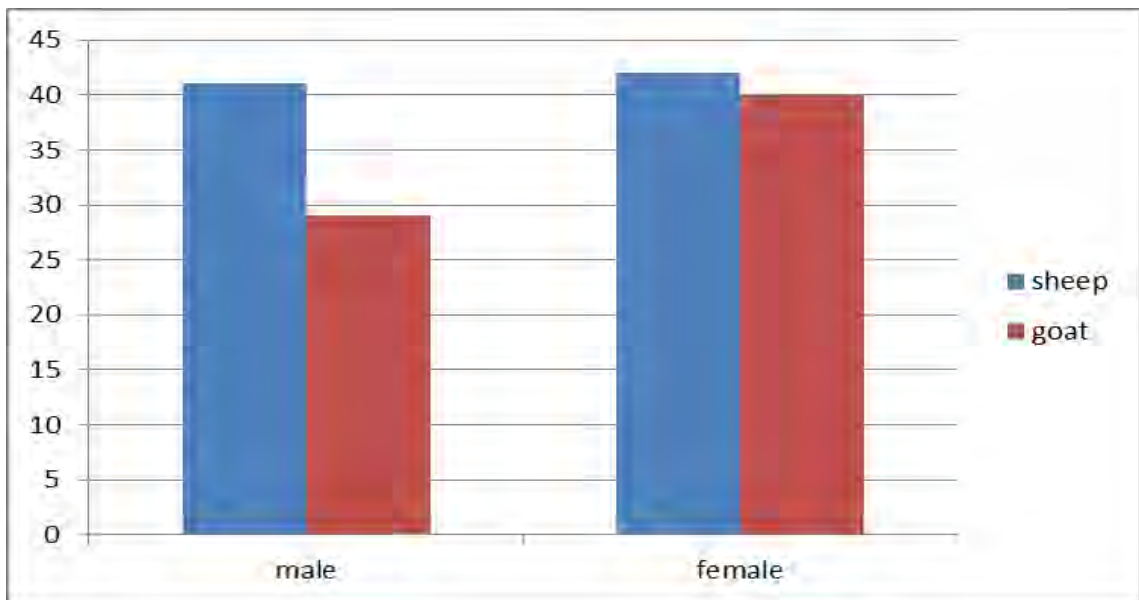


Figure 12: Sex-wise sero-prevalence (%) in sheep and goats.

4.2. Risk Factors for Sero-positivity

4.2.1. Univariate model for identification of potential risk factors in sheep & goats

For each species after running univariate logistic regression, the possible risk factors for PPR sero-positivity were identified. The significant risk factors recognized for sero-positivity in goats were sex, age group, altitude and source or origin of the animal (Table 9). While in Sheep mixed species rearing, grazing type, age groups and origin of the animal were found to be the significant risk factors (Table 10).

Table 9: Variables associated with the sero-positivity in univariable models of Goat data.

Goats n = 582		p ≤ 0.3				
Variable	Animal tested	Sero-positive (n)	% sero-prevalence (95% CI)	Crude Odds ratio (95% CI)	p-value	
Sex						
Male	105	26	25(17-34)	1*		
Female	477	216	45(41-50)	1.7(1.04-2.6)	0.032**	
Age (month)						
6-12	127	4	3(0-8)	1		
13-24	162	40	25(18-32)	3.6(1.2-11)	0.023**	
25-36	162	59	36(29-44)	4.5(1.5-14)	0.007**	
>36	131	117	89(83-94)	4.6(1.5-13)	0.006**	
Herd size						
Small	214	80	37(31-44)	1*		
Medium	234	84	39(30-42)	0.9(0.6-1.4)	0.744	
Large	134	56	42(33-51)	1.2(0.8-1.9)	0.412	
Mixed Species						
No	105	41	39(30-49)	1*		
Yes	477	179	38(33-42)	0.9(0.6-1.4)	0.771	
Production						
Sedentary	-	-	-	-	-	
Agro-pastorals	310	119	38(33-44)	1*		
Pastorals	272	101	37(31-43)	0.9(0.7-1.3)	0.755	
Grazing						
Zero	-	-	-	-	-	
Fenced	-	-	-	-	-	
Communal	274	108	39(34-45)	1*		
Migratory	308	112	36(31-42)	0.9(0.6-1.2)	0.449	
Water source						
On farm	129	45	35(27-44)	1*		
Shared	453	175	39(34-43)	1.2(0.8-1.8)	0.439	
Origin						
Born in	464	110	24(20-28)	1*		
Brought in	118	110	93(87-97)	44(21-94)	0.000**	
Altitude						
High	158	49	31(24-39)	1*		
Medium	175	70	40(33-48)	1.1(1.07-2.7)	0.908	
Low	249	101	41(34-47)	1.7(1.07-2.6)	0.052**	

OR: Odds ratio; CI: Confidence Interval; 1*: Reference; **Significant

Table 10: Prevalence (%) and univariable analysis of the potential risk factors for seropositivity of sheep to PPR

Sheep n = 216		P ≤ 0.3			
Variable	Animal tested	Sero-positive (n)	% sero-prevalence (95% CI)	Crude Odds ratio (95% CI)	p-value
Sex					
Male	39	11	28(15-45)	1*	
Female	177	73	41(34-49)	1.1(0.52-2.13)	0.877
Age (month)					
6-12	109	38	35(26-45)	1*	
13-24	3	2	67(9-99)	1.7(0.8-4)	0.190**
25-36	103	51	50(40-60)	2.8(1.3-6)	0.012**
>36	1	0	0	1.5(0.5-5)	0.468
Herd size					
Small	38	12	32(18-49)	1*	
Medium	143	65	45(37-54)	1.8(0.8-3.9)	0.127**
Large	35	14	40(24-58)	1.4(0.5-3.8)	0.454
Mixed Species					
Yes	167	56	34(26-41)	1*	
No	49	35	71(57-83)	5(2.5-10)	0.000**
Production					
Sedentary	-	-	-	-	-
Pastorals	91	35	38(28-49)	1*	
Agro-pastorals	125	56	45(36-54)	1.25(0.8-1.7)	0.352
Grazing					
Zero	-	-	-	-	-
Fenced	-	-	-	-	-
Migratory	99	32	32(23-42)	1*	
Communal	117	59	50(41-60)	2(1.25-3.3)	0.008**
Water source					
On farm	29	12	41(24-61)	1*	
Shared	187	79	42(35-50)	1.04(0.5-2.3)	0.930
Origin					
Born in	160	45	28(21-36)	1*	
Brought in	56	46	82(70-91)	12(5.5-25.3)	0.000**
Altitude					
High	40	12	30(17-47)	1*	
Medium	48	21	44(29-59)	1.1(0.5-2)	0.853
Low	128	58	45(36-54)	2(0.9-5)	0.089**

OR: Odds Ratio; CI: Confidence Interval; 1*: Reference; **Significant

4.2.2. Multivariate logistic model for individual & joint data of sheep and goats

After running in the univariate logistic regression for sheep and goats separately, those variables found significant ($P \leq 0.3$) were subjected into multivariate logistic model. In the final multivariate logistic regression age group, origin and altitude were risk factors for sero-positivity in goats while origin of the animal and mixed species rearing were significant risk factor in case of sheep as shown for both in Table 11.

In goat herd, 13-24month old were 10.7 times more likely to have PPR antibody compared to 6-12month old age ($P= 0.002$). 25-36month old goats were 9.9 times more likely to have PPR antibody compared with 6-12month old ($P= 0.003$). 36-48month old goats were 9.2 times at a higher risk for PPR infection compared to 6-12month old (Table 11). Goats brought from outside a herd were 55.7 times at a higher chance of being seropositive compared with goats born in the herd ($P= 0.000$). Small ruminants in lower altitude were 2.5 times at higher risk to PPR sero-positivity than animals in higher altitude ($OR = 2.5$; $P= 0.000$).

In the individual multivariate logistic model, sheep brought from different location or flocks were 12.2 times more likely to have PPR antibody than a sheep born in a flock. Sheep reared with goats were 3.3 times at higher risk compared with sheep reared together with goats, camels and cattles ($OR= 3.3$ $P= 0.001$) (Table. 11).

Table 11: Multivariable logistic regression model for goats and sheep separately.

Variables	Sheep		Goats	
	Odds Ratio (95% CI)	Likelihood P-value	Odds Ratio (95% CI)	Likelihood P-value
Age group(month)				
6-12			1*	
13-24			10.7(2.3-49.5)	0.002**
25-36			9.9(2.2-44.9)	0.003**
36-48			9.2(2.1-40.1)	0.003**
Origin				
Brought in	12.2(5.5-27)	0.000**	55.7(25.2-123)	0.000**
Altitude				
High			1*	
Medium			1.4(0.01-0.02)	0.155
Low			2.5(0.02-0.05)	0.000**
Mixed species				
No	3.3(1.6-10)	0.001**		

**Significance; CI: Confidence interval; 1*: Reference

Table 12: Combined multivariable logistic regression model for both sheep and goats.

Variables	Number of animals tested	Number infected	Adjusted Odds ratio (95% CI)	Std. Err	p-value
Age group(month)					
6-12	236	39	1*		
13-24	165	87	1.7 (1.07-2.7)	0.39	0.023**
25-36	265	117	2.7 (1.7-4.3)	0.62	0.000**
36-48	132	83	4.2 (2.5-6.9)	1.07	0.000**
Origin					
Brought in	174	156	3.8 (2.6-5.5)	0.71	0.000**
Altitude					
high	198	61	1*		
Medium	223	91	1.25(0.8-2)	0.14	0.181
low	377	159	2(1.25-2.5)	0.11	0.002**

Std. Err: Standard error; **Significance; CI: Confidence interval; 1*: reference

In the combined multivariable logistic regression, age group, origin (source) of the animal and altitude were returned to be risk factors for PPR sero-positivity in both sheep and goats. Both sheep and goats aged 13-24month old were 1.7 times at higher risk of getting PPR disease infection when compared with the 6-12month respectively. Those sheep and goats aged 25-36month were 2.7 times more likely than with those aged 6-12months to become PPR sero-positives. 36-48month old sheep and goat were the most likely to have PPR infection compared with 6-12month olds. Small ruminants brought from outside the flock were 3.8 times at higher risk for the PPR disease while low altitude rearing was 2 times at higher risk than higher altitude rearing (OR= 2) in both species (Table 12).

4.2.3. Multilevel mixed-effect logistic regression model

In the mixed effect logistic regression all variables were run for sheep and goats without considering collinearity between the variables in to one model after adjusting the cluster effect of PAs and Herds. The two models, mixed logistic and standard logistic regression models were also tried to compare with each other. Presence of sub-location random effect resulted in widening of confidence intervals for the sheep and goat data (Table. 11).

Accordingly, the likelihood ratio test in the multilevel mixed effect logistic regression model showed that inclusion of sub-location random effect provided a substantially better fit ($P= 0.000$) than the fixed effect logistic regression model for both sheep and goats.

When mixed effect logistic regression used after including the effect of sub-location for goats and sheep, age group and origin were returned to be the risk factors while altitude, production system and water source were newly included predictors for PPR sero-positivity. 13-24month age was 1.4 times more likely to have PPR antibody compared with 6-12months. 25-36month age was found 2.2 times at higher risk than the 6-12month ages for PPR disease. The highest likelihood of being seropositive was recorded in 36-48month ages sheep and goats (OR= 3.7; $P= 0.000$). Newly brought animals were found 3.7 times more likely to have PPR antibody than animals born in the herd (Table 13).

Small ruminants reared at lower altitude were 2.5 times at higher risk (OR= 2.5; P= 0.038) compared with sheep and goats at higher altitude (Table 13). Those sheep and goats reared by pastorals were 2.4 times more likely to have PPR disease as compared to agro pastorals (OR= 2.4; P= 0.012). Sharing water source was found 1.7 times more likely to cause PPR disease compared with animals that used water source at farm level.

Table 13: Multilevel mixed-effect logistic regression for sheep and goat

Risk factor	Category	LRT X² p= value	Coefficient	Adjusted odd ratio (95%)
Age group	6-12			1*
	13-24	0.023**	0.33	1.4(0.8-2.3)
	25-36	0.002**	0.79	2.2(2.4-3.6)
	36-48	0.000**	1.30	3.7(2.1-6.5)
Origin	Born in			1*
	Brought in	0.000**	0.9	3.7(2.5-5.7)
Altitude	high			1*
	medium	0.41	-0.31	1.4(0.7-3.3)
	low	0.038**	-0.84	2.5(1.1-5)
Production	sedentary			
	Agro-pastorals			1*
	pastorals	0.012**	0.86	2.4(1.2-4.6)
Water source	On farm			1*
	shared	0.053**	0.58	1.7(0.99-3)

Log likelihood ratio (p= 0.000): 1*: reference; **: Significance; CI: confidence interval

4.3. Questionnaire Survey Analysis

Herd owners were asked about the condition of their animals. A total of 65 herd owners who provided their animals for sampling were interviewed. Out of the respondents, 64% replied that their herds were affected by a disease with a clinical sign of diarrhea, nasal discharge and sore mouth in the last one year. According to the finding of the

questionnaire survey, when animals become sick, 45% of the respondents either consult community animal health workers or treat their animals by themselves while 34% of the respondents treat their animals based on their own home grown knowledge towards the disease by purchasing drugs either from animal health post or markets. Only 21% of the interviewee replied, when get diseased their animals, they consult animal health officials to get the treatment.

Among the respondents, there was a practice to keep sheep and goats either with camels or with cattle or both. The mixing of the different species during migration, at watering or in night enclosures (resting), between camels and cattles with small ruminants was recorded. Besides, all respondents put their animals at night in enclosure, with a fence from thorny woods which was not built well, so that animals could escape from or wild animals could cross in.

75% of the respondents use markets to sale their live animals while 8% sale either directly to nearby neighbor or to middle men. 17% of the interviewee used both selling in the market and nearby village or neighbor. Out of the respondents, 82% buy live animals from local nearby markets, 13% purchase from nearby village or neighbor. The remaining 5% use both markets and nearby villages for purchasing animals. When we come to the immunization status of the sampled animals against PPRV, 65% of the respondents replied that their animals have never had vaccination against PPR where as 17% of the interviewee doesn't exactly differentiate between vaccine and ordinary drug they had taken. 18% of the respondents replied they had vaccine against PPR before 4years.

All respondents interviewed in all Districts knew PPR disease and its typical clinical symptoms. Based on the nature of the disease different names were given to PPR in the study areas (Table. 14).

Table 14: Different local names given by pastorals and agro-pastorals to PPR disease in the study areas

Zonal region	District	Local name given for PPR
Liben	Hudet	Merchekes
	Moyale	Merchekes
	Dolo Ado	Debahare, Har
	Filtu	Deefhare
Afder	Gorobekeksa	Afgurfo, malberer
	Chereti	Har
	Dolo Bay	Rihwayne

Source: (Local communities)

Table 15: Last date of outbreak and vaccination history in line with the sero-status based on questionnaire survey.

Sample collection date	District	Kebele (PA)	Last vaccination	last outbreak	Sero-positives		
					Ages group (months)		
					6-12	13-24	25-48
10-12/3/2016	Guradhemole	Harodibe	2011	Sep-2015	1	4	9
		Kundi		2013	2	16	
13-15/3/2016	Gorobekeksa	Hargedeb	2012	-	4	6	5
		Hagermokor		2	3	14	
		Tour		5	5	7	
17/3/2016	Chereti	Qorder	2012	Feb-16	5	5	7
19-20/3/16	Dolo Bay	Entoy	2013	Apr-2015	1	10	15
		Row-Row		1	5	12	
22-26/3/16	Dolo Ado	Bur amino	2012	2012/2014	5	7	9
		Kole			2	9	16
		Melkadida			-	4	6
		Suftu		1	2	2	
		Fiqo		2012	Jan-2016	5	1
28/3/2016	Filtu	Wedlehubbe	2005	Oct-2014	3	6	9
30-31/3/2016	Hudet	Bod-Bod			-	3	12
1-2/4/2016	Moyale	Diredima	2011	Feb-2016	-	5	18
		Kalehargessa		Feb-2016	-	2	9
1-2/4/2016	Moyale	Kojowa	2011	Feb-2016	3	3	9
		Darselam		Feb-2016	1	-	12
Total					39	82	205

N.B: Districts and PAs with biased information were left empty.

4.4. A Retrospective Epidemiological Analysis of PPR outbreaks

4.4.1. Temporal distribution

A total of 1282 outbreaks were reported between March 2006 and August 2015 from different agro-ecological regions of Ethiopia. The highest number of outbreak was reported in 2010 despite with the lowest case fatality. The highest case fatality of the disease was recorded in 2009.

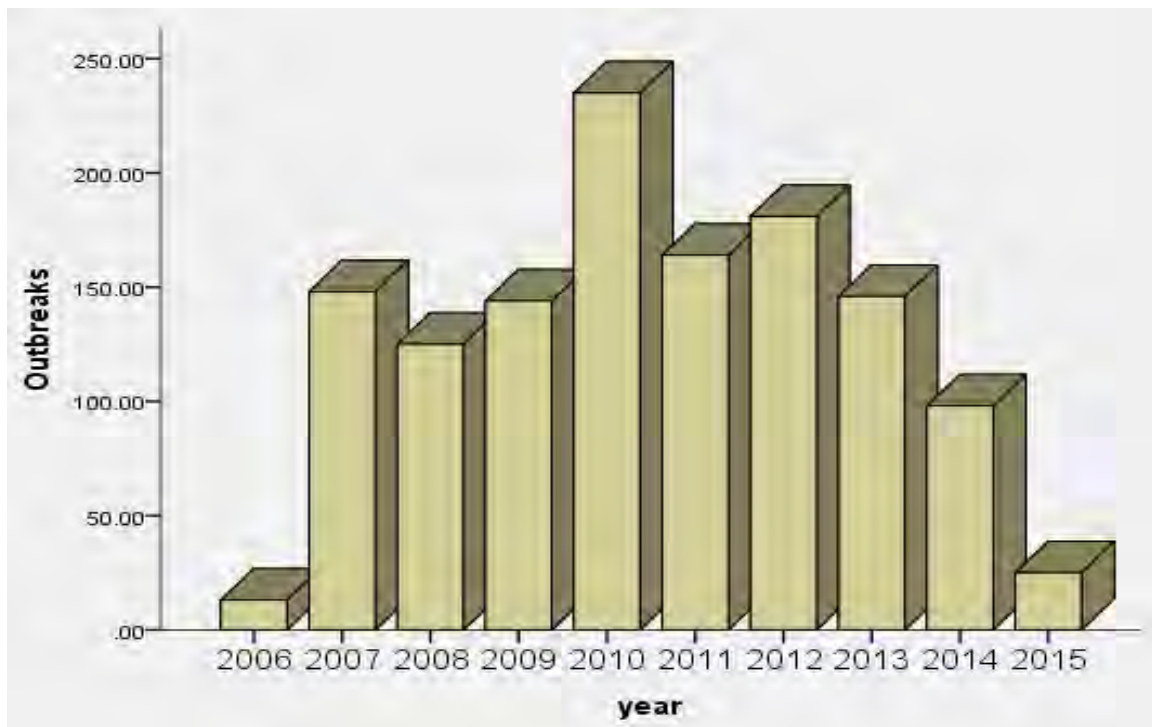


Figure 13: Year wise number of PPR outbreaks from 2006-2015

To observe the temporal pattern of the disease in a detailed manner, we filtered the average monthly outbreak of the disease for the decade (Figure 16). The monthly overall average outbreak pattern of the disease for the study time is shown in Figure.16. The incidence of PPR was lowest in June and gradually increases in July. The frequency of the disease was peak in September gradually decreases towards November (Figure. 15).

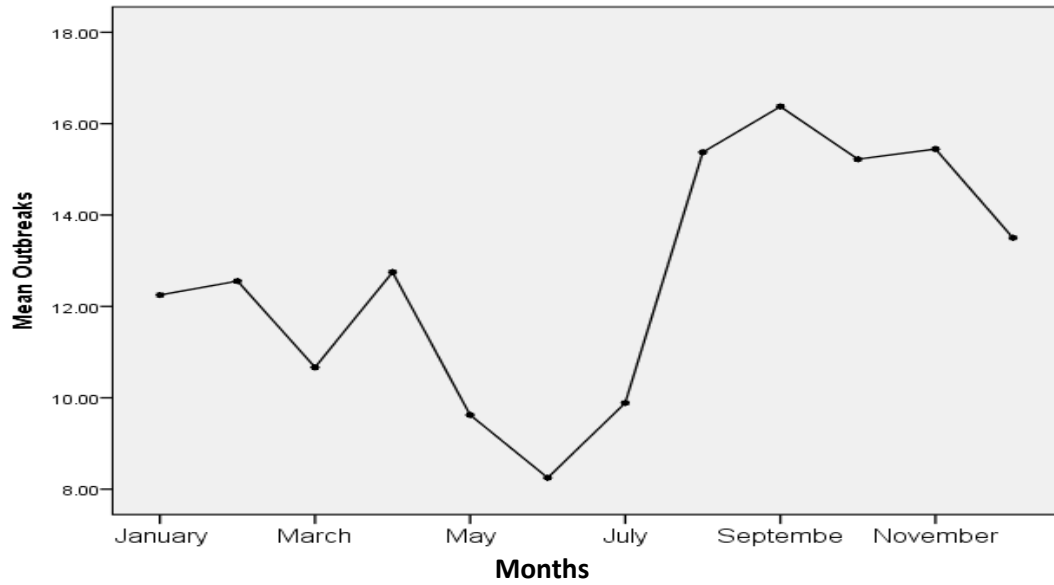


Figure 14: Average monthly outbreak of PPR

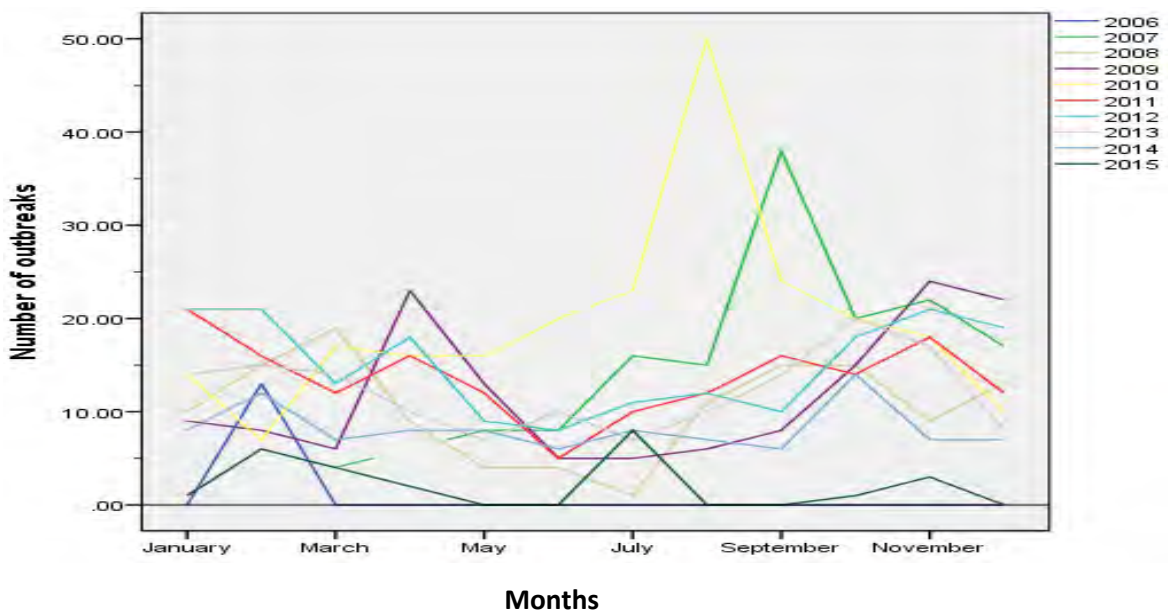


Figure 15: PPR seasonal disease pattern during 2006-2015

4.4.2. Spatial distribution of PPR disease outbreaks across different regions

The highest number of PPR outbreaks were recorded in Oromia region (68.80%) followed by Amahara (16.99%), SNNPR (4.71%), Tigray (3.32%), Somali (2.78), Afar (1.31), Benishangul Gumuz (1%), Gambella (0.46%), Dire Dawa (0.39%) and Ababa

(0.23%). The distribution and number of outbreaks of PPR in different regions is present in Figure.16.

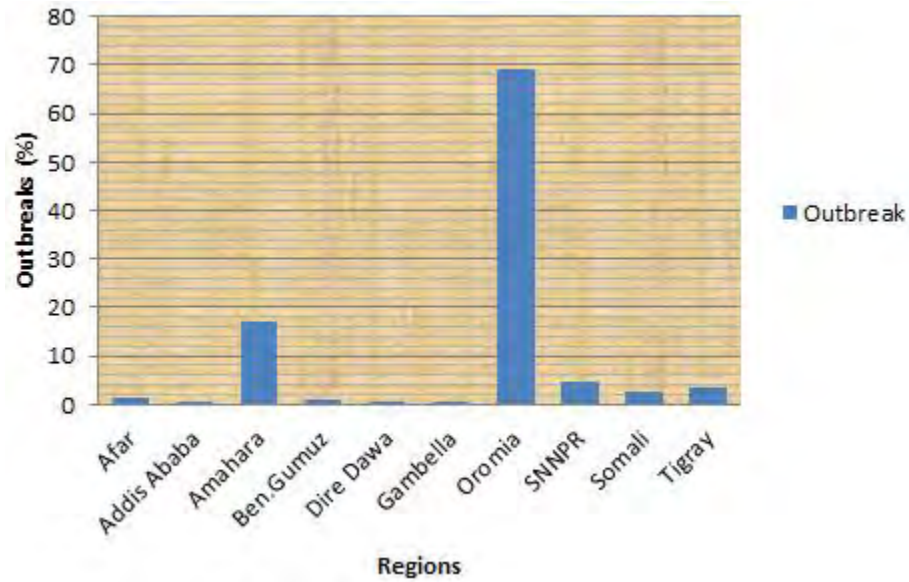


Figure 16: Region wise number of PPR outbreak reports.

4.4.3. Epidemiological parameter estimates of PPR

An overall morbidity and mortality rate of 2.3% and 0.31% was recorded during the study period. The highest morbidity and mortality of the disease was reported in 2006 and gradually decreases in 2015.

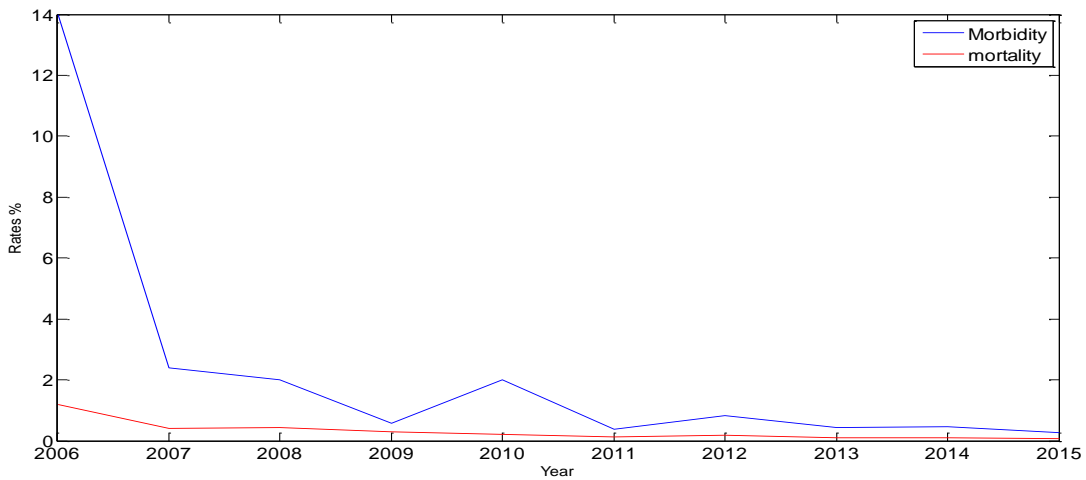


Figure 17: Overall mortality and morbidity of PPR

4.4.4. Species-wise case-fatality of PPR

A total case of 51551 and 61734 were reported in sheep and goats during the study time, respectively. The highest case fatality in sheep and goats was reported in 2011 and 2009, respectively. While the lowest case fatality was reported in 2010 for goats and 2006 for sheep. Case fatality due to PPR in sheep and goats between 2006 and 2015 is summarized in Figure. 19.

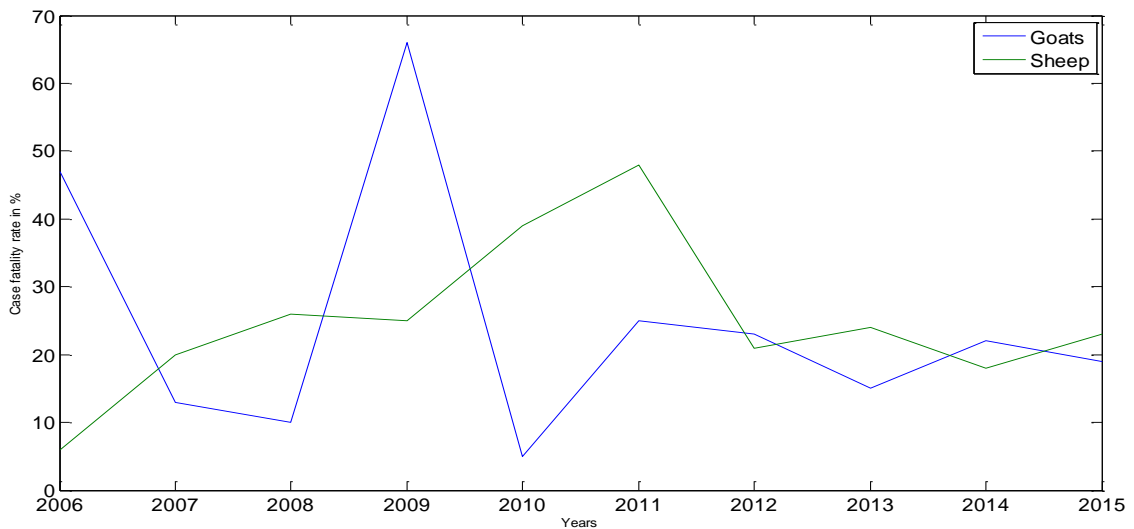


Figure 18: Case fatality of PPR in sheep and goats

4.4.5. Vaccine intervention following PPR outbreaks in different regions

During the disease outbreak, Oromia region used the highest percentage (68%) of PPR vaccine doses followed by Amhara (15%), SNNPR (5%), Somali (4%), Benshangul Gumuz (4%), Gambella (2.2%), Tigray (2%), Dire Dawa (0.7) and Addis Ababa (0.01%) to control the outbreak. Regions where high number of outbreaks occurred were high livestock abundance like Oromia used the greatest quantities of PPR vaccine. Majority of outbreaks as well as the greatest percentage of vaccine doses were used in Oromia region while the lowest cases of the disease and vaccine dose were recorded in Dire Dawa administrative. No vaccine intervention was recorded in Afar despite 1.3% of PPR outbreak reported during the study period.

Table 16: Usage of control vaccines following PPR outbreaks

Region	Total no of outbreaks	Control vaccine (Dose)
Addis Ababa	3	2480
Afar	17	-
Oromia	879	1744086
Benshangul Gumuz	12	100,000
SNNPR	61	125,000
Somali	36	96440
Amahara	220	379113
Diredawa	5	20000
Tigray	43	51677
Gambella	6	56492
Total	1282	2575288

Yearly distribution of PPR outbreaks in different regions from 2006-2015

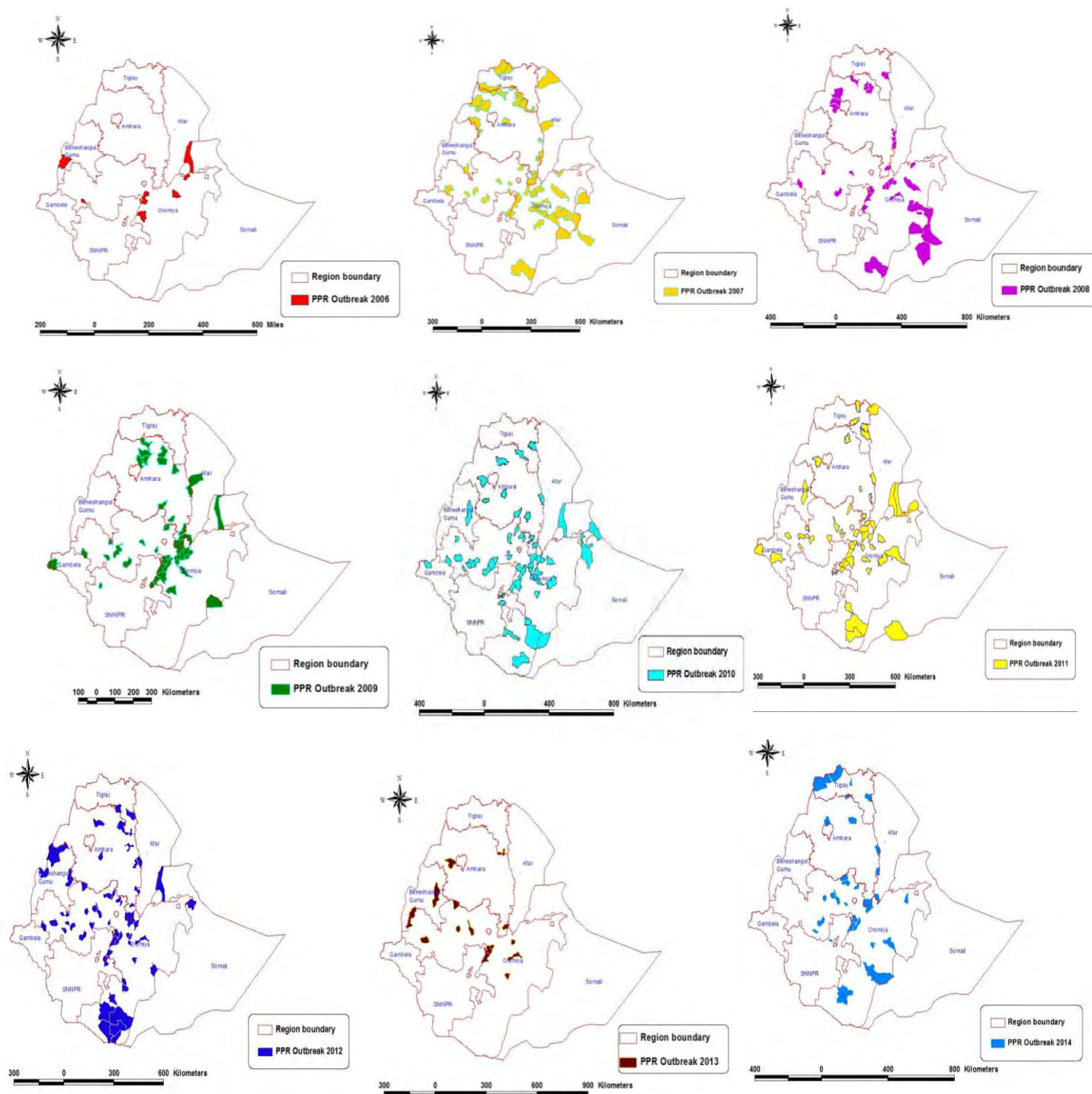


Figure 19: Maps depicting the distribution of PPR outbreak reports 2006-2014

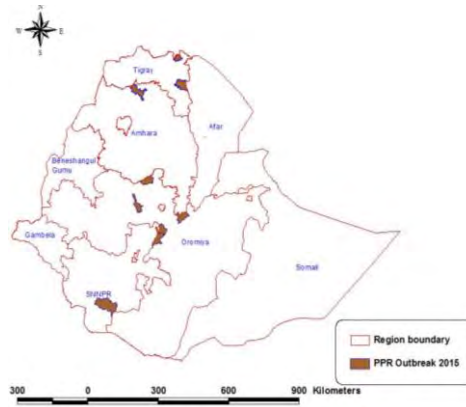


Figure 20: Distribution of PPR outbreak report in 2015

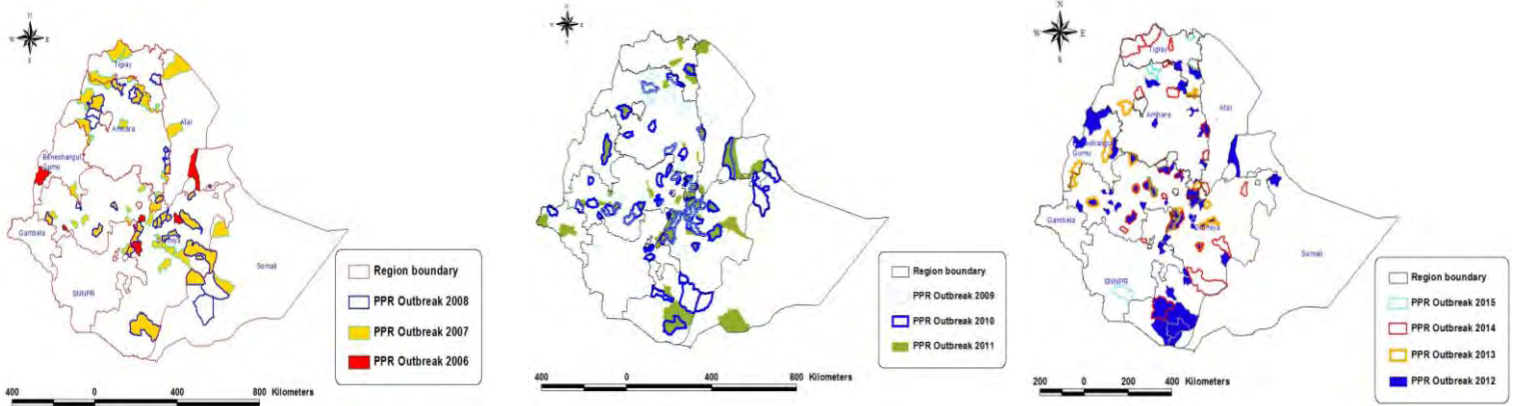


Figure 21: PPR outbreak pattern adjoining consecutive years

5. DISCUSSIONS

5.1. Serological Analysis of PPR Antibodies

For effective control of PPR, accurate diagnostic techniques and timely vaccination of susceptible populations are necessary. Accordingly, a full understanding of the disease epidemiology is imperative. PPR eradication depends on rapid and accurate diagnosis, and carrying out of prompt control measures. Due to the immense economic impact of PPR, it is absolutely necessary to implement epidemiological surveys of this disease.

In Ethiopia, studies on PPR disease are very few compared with the impairment up on the livelihood of pastorals and agro pastorals. After being recognized a couple of years back PPR has been the adversary of the poor pastorals who solely rely on their small ruminants who act like cash in a bank. This study was undertaken in one of the pastorals areas of Somali regional state of Ethiopia bordering Kenya and Somalia and the result of this study could give a snap shot on the status of the neighboring border regions as well.

In this study, an overall individual and flock level prevalence of 43% and 104% was recorded respectively in the selected districts of Somali region. Hence, a true prevalence of 41% and 44% was found in sheep and goats, respectively. The finding was higher when compared with the result noted by Megersa *et al.* (2011) in Afar and Gambella regions of Ethiopia. In this finding, goats were found to have higher prevalence compared with sheep though the difference was not significant ($P=0.483$). This could be due to the fact that sheep and goats are equally exposed to the PPR risk factors.

Similar findings were reported showing a higher prevalence of PPR in goats than sheep (Abubakar *et al.*, 2011; Zahur *et al.*, 2011; Kazeem, 2001). However, Singh *et al.*, (2004); Abraham *et al.* (2005); Mehmood *et al.* (2009) had reported a higher PPR prevalence in sheep than goats which disagrees with our finding. The difference in prevalence could be due to the difference in the proportion of sampled animals. Besides, since goats were used for meat and milk compared with sheep that were less considered for economical purpose, in return pastorals were intense to keep more goats than sheep. Possessing more goats is a symbol of wealth and provides respect in most communities.

Age wise prevalence of PPR antibody in small ruminants in the study area revealed a statistically significant difference among the four age groups considered in this study ($P=0.000$) (Table. 7). Small ruminants aged between 36-48 months had recorded the highest PPR virus antibodies (58%) followed by small ruminants aged 25-36month (47%), while the least prevalence was in small ruminants aged 6-12month (25%). In this investigation, PPR prevalence was increased as age increases in both sheep and goats (Figure. 10). This finding is agreement with Majiyagbe *et al.* (1992) who showed that PPR sero-prevalence increases with age. Dams infected with PPR virus can passively transfer maternal antibodies to their young ones. Although the maternal antibodies progressively decay, they remain above the protective threshold for up to 4-5 months after which PPR vulnerability increases with age (Abubakar *et al.*, 2009). This increased PPR susceptibility with age after five months in small ruminants may explain the relatively lower sero-prevalence rates obtained in small ruminants aged between 6-12 months when compared with those aged between 36-48 months in this study.

The high PPR antibody prevalence in the age group of 36-48 could be attributed due to the high proportion of sampled animals in this age group. This finding however disagrees with Al-Majali *et al.* (2008) who reported that PPR prevalence to be high in age group of above 4 month and below 2 year. Higher PPR sero-positivity in 36-48month age could be due recurrent drought in the study area which mostly affected the young ones (6-12month), so during our study pastorals were unwilling to bled the left over for sampling. Throughout our sampling there was drought in the study area, which was very difficult for the young small ruminants to sustain the scarcity of feed and water in the region. Since small ruminants aged greater than 36 months can sustain and endure the severe environment condition and pastorals were highly relying on these animals during the condition.

Age group distribution of PPR virus antibodies in different species (sheep and goats) is shown in figure 11. Goats aged above 36 had the highest sero-prevalence (59%) followed by goats aged 25-36months, while the least prevalence was recoded in goats with age group of 6-12month. Different pattern was also observed in sheep. When age of the sheep increases the PPR prevalence decreased.

Sex wise, female sheep and goats were 2.5 times more exposed than males (OR= 2.5). From the current study females had a statistically greater sero-prevalence rate of 44% compared to the male counterpart whose sero-prevalence was estimated to be 26% (p= 0.000). This reflection agrees with the findings by Luther *et al.* (2007); Khan *et al.* (2008); Waret-Szkuta *et al.*, (2008) who reported a significantly higher sero-prevalence rate of PPR virus antibodies in females than in male goats. This could be majority of male animals are not usually kept in a flock for a long period of time. They are often sold out for meat at approximately 1-2 years of age, while the females remain in the flock for breeding purposes, which may indirectly account for the high prevalence of PPR antibodies in females.

Comparing both species" sex, female sheep had a higher prevalence (42%) than female goats (40%). And this finding is in accordance with (Bello, 2013), who reported a higher PPR antibody prevalence in female sheep compared with female goats. Similar picture was observed in male sheep which recorded a higher prevalence (41%) compared with male goats (29%) (Figure 11). This increase PPR prevalence in sheep in both sexes could most probably attributed due the difference in the proportion of sampled animals. The other reason might be the variation of recovery rate on sheep and goats. Sheep"s recovery is usually higher than goats (Khan *et al.*, 2008).

Area wise, the highest overall prevalence of 52% was found in Dolo Bay district followed by Dolo Ado (42%) while the lowest antibody prevalence was noted in Ethiopian Moyale district (30%). This finding agrees with Waret-Szkuta *et al.*, (2008) who finds a higher prevalence in Dolo Bay and Dolo Ado (52%). Hence, an overall apparent flock prevalence of 98% was found amongst 57 focks sampled across the districts. The difference in prevalence among the districts was found insignificant (P=0.382). Even though the kebeles (PAs) selected between districts were not proportional in number, the prevalence among districts was not significantly different. This suggests remarkable contagious nature of the disease on wide geographic areas and infecting perhaps most of the susceptible animals in affected villages.

Dolo Bay and Dolo Ado were districts who registered the highest overall prevalence (52% and 42% respectively) compared with other selected districts (Figure 8). These districts border share with the South West to Kenya and South East to Somalia where there is uncontrolled movement of livestock for grazing and marketing between the pastorals of neighboring nations. When drought strikes in one country, they will move their ruminants through the pores borders for the safety of their ruminants as well as their livelihood and stay for some year until the drought pacifies.

Therefore, the increase in prevalence in these districts could be attributed from the movement of animals that plays an important role in the transmission and maintenance of PPRV in nature (Abubakar *et al.*, 2009). This is in accordance with the work done by Al-Majali *et al.* (2008) which recorded a higher sero-prevalence of PPR in regions with free animal movement than other areas in Jordan. In addition, these districts share a common market place, which could play a key role for the contact of infected animals triangulating with the three countries. And those unsold will be brought back and join with the naïve flocks which could contribute to the wide endemecity of the disease.



Figure 22: Inter-species mix up at Weyib River Gorobekeksa district of Afder Zone.

Out of the 57 flocks sampled in the study area, a total prevalence of 98% was found. Similar finding was found by Taylor, (1997) on Syria with 96% flock prevalence. An equal aggregate of animals was taken from all the selected flocks. Since animals in a village share similar scenario, we considered one flock to represent one village in this study. A flock with at least one seropositive was taken to represent the whole flock as a positive. All flocks except a flock in Mulkadida PA had got at least one seropositive animals. These findings suggest that the disease movement within herds is higher compared between herds. If one animal get the infection the probability of contracting the virus in the herd is much higher. The pastorals in Moyale and Hudet district called PPR the name “Merchekes” to mean the disease can be transmitted only by voice to show the degree of transmissibility within the herd.

Using multivariate logistic regression, we had identified that age group, origin and altitude to be predictors of PPR. However, since the study was undertaken in a multilevel cluster sampling, we implemented mixed-effect models for the random effects (clustering by PA and by herd). After adjusting the effect of the sub-locations; age group, altitude, production system and water source became the most likely predictors of PPR in this study.

Animals brought from outside the flock were 3.8 times at higher risk of becoming PPR seropositive compared with those born in the herd. Superior male goat was borrowed among the pastoral community in the areas for days and return to the parent flock which could attribute as a bridge for the transmission of the disease among different flocks.

Living in the higher altitude (>1000m) was found to be at lower risk for PPR compared with the low altitude (<500m) (Table 13). This study observed the finding that districts found in the low altitude like Dolo Bay has the highest PPR sero-positivity compared with districts at a higher altitude such as Moyael. This could be attributed in the low altitude areas which mostly strike by the recurrent drought, and face shortage of water and grazing, the animal owners will often travel long distances during the dry season in search of fodder and water. Consequently, a considerable proportion of the population

becomes infected during this period, giving rise to the establishment of disease endemicity and continued, year-round circulation of the virus.

In our finding, the odds of being positive to PPR virus was 2.4 times higher if animals were raised in pastoralism production system. And this was in agreement with Kivaria *et al.*, (2013) that reported pastoral districts appeared to have suffered more from PPR infection. Pastoralism is a system which solely depends on the livestock. This system is prone to the recurrent climate change due to El Nino, so the pastorals move for search of water and pasture in order to keep their livelihood. In this circumstance different flocks will mix-up during grazing and in water points where a lot number of animals gather. Besides, we witnessed sharing of the water point with the wild ruminants animals which might play a role in the transmission of disease though it needs further study to substantiate the suggestion.

5.2. Spatio-temporal and Epidemiological Measures from Retrospective Data

PPR which is an endemic disease in most parts of Ethiopia and has been misdiagnosed from other similar diseases such as blue tongue, Foot and mouth disease, Contagious caprine pleuro pneumonia for some time in the case of the disease outbreak. Due to the development of diagnostic kits for field and in vitro and hence the awareness buildup of field and community animal health professionals could be some of the reasons for the decline of the disease in recent years.

The study of annual occurrence of PPR in Ethiopia showed that the highest number of PPR outbreak was reported in 2010 and the lowest in 2006. Out of the total event reported in 2010, 65% was from Oromia regional state. This could possibly be due to the drought that occurred in most pastoral regions of Oromia and Somali in the same year (AU, 2011). The climate change that has been occurring due to El Nino in the past years could have played a great role in the distribution and frequency of the disease outbreaks. When there is lack of rainfall most pastorals move their animals long distance or search of pasture this will result in the mix up of herds, and due to shortage of feed the animals' immunity weaken and predispose for infectious disease like PPR.

The lowest incidence of the disease was reported in 2006. The reports were all based on suspicions; the awareness during the early time of 2006 for the disease was low compared to early days. The lack of awareness for the disease might have played a role for the low number of reports. Nowadays professionals are becoming familiar with the disease condition through time. The report in 2006 might not be a representative of the whole status of that year.

Meanwhile, after filtering all the months in the entire years and observe at the average number of outbreak per month the highest and lowest was recorded in September and June, respectively. Analysis of the average monthly pattern of the disease showed a slight increase between March and April and gradually decreases and measures lowest at June. The number of outbreaks started to increase slowly from then until August, with a peak in September.

The months where PPR is peak (see Figure 14) coincides with the time Ethiopian celebrate Easter and New Year. A large amount of livestock is collected for the central market before the festive which could help flocks to mix up and distribute the disease. The high amount of rain during the end of the rainy season (August) may be a factor for the increase. In rainy seasons animals graze to homestead where there is contact of different flocks. Rainfall causes animals to clump together, enhancing close contact, and high relative humidity values are related with virus survival in aerosols (Hegde *et al.*, 2009). Similar studies have been reported on occurrence of the disease in wet season (Obi *et al.*, 1983).

Lack of pastures and water due to long dry spells or winter results in poor livestock nutrition; consequently, small ruminants become weak and dilapidated with lowered immunity against PPR (Abubakar *et al.*, 2009; Munir *et al.*, 2008).

The month June has recorded the lowest outbreak report of all. In June it is the time that the wet season starts and pasture and water accessibility eased in most part of the country. During these time animals reduce to go far distances for search of food and also the frequency to use water shares will decline. This will avoid the contact of naïve flocks with infected ones.

The overall morbidity, mortality and case fatality were higher in goats than sheep during the study. This is in agreement with Abubakar *et al.*, (2008c) and Khan *et al.*, (2007) who reported the disease is more severe in goats mostly. This could be attributed due to the high proportion of goats reared compared to sheep in lowland pastoral areas of Ethiopia. This also could indicate the difference in susceptibility between the two species.

Region wise, the highest number of outbreaks was recorded in Oromia region followed by Amhara. While the list case of the disease was reported from Addis Ababa city municipal followed by Dire Dawa. Afar and Somali region who are dominantly pastorals and consist the majority of the small ruminants has reported a few compared with Tigray and SNNPR (Southern Nations Nationalities People Region). This could be attributed from poor disease reporting system due to lack of trained manpower, migratory behavior of the pastorals which is difficult to monitor the status of the animals and miss-diagnosis of PPR disease from other disease. The other reason for the variation in the number of outbreaks could most probably be due to the different production system implemented in the different regions.

From the above reports, regions with the highest and lowest disease outbreak doesn't mean the disease is high or low, on those areas respectively. This could probably have associated with regularity of disease reporting. The more a region reports outbreak the more will be the number and vice versa. The other reason might be the differential number of sheep and goat populations among the regions. So to avert this, improved disease reporting system should be established such as by participatory disease surveillance (PDS) based on participatory rural appraisal (Mariner and Paskin, 2000).

The spatial pattern of PPR control vaccine consumption and the outbreak frequency were almost supplementary (Table 16). However, there were variations considering the disease outbreak and vaccine consumption across regions. This could be associated with the differential number of sheep and goat populations among the regions. From the regions listed, nothing was reported from Afar region regarding the intervention of PPR outbreaks using vaccines during the entire study period. Even though there is no history of report in Afar, vaccination against PPR outbreak was given (personal communication).

This is instigated due to the poor reporting system from regional and district officials and lack of awareness whether to report vaccination activities. Most officials consider only reporting disease outbreaks (personal communication) even though they did use vaccines.

Despite the usage of vaccines to control the outbreaks, peak of the disease was reported from Oromia region (Table 16). This could be attributed largely to such factors like lack of maintenance of cold-chain, faulty administration and handling of the vaccines and hence application of the vaccine on the aftermath of the disease could be among the reasons.

5.3. Questionnaire Survey Analysis

Most of the time pastorals were the front targets when an emerging disease such as PPR overtakes. Due to the effect of most diseases they were forced to know the behavior of most diseases. In the field we had noticed the abysmal perception of the pastorals about PPR disease.

Out of the interviewed pastorals more than half percent experienced the outbreak of the disease within a year. And all of them were able to explain the typical clinical sign of the disease; diarrhea, nasal, and ocular discharge with sore mouth. The reason that they developed the awareness about the disease could be most probably due to the year round recurrent of outbreaks, the intermittent training that was given to community animal health workers (CAWS) by the government or non-governmental organizations could some of the reasons for building up of their knowledge.

Despite the knowledge they have got a few (21%) were able to take their animals to a nearby clinic when they suspect PPR disease. This might be supported by different thoughts such as lack of veterinary service at each corner where pastorals could easily accessed, insufficient drug supply and training to those established health posts because we had witnessed pastorals buying drugs from markets and treating by themselves (34%). However, around 45% of livestock owners we interviewed had the practice of to consult CAWS and give treatment based on that.

All pastorals replied that they keep more than one species. We noticed that all species came in contact mostly at water point (Figure 22) and sometimes during herding with camels and cattle. Even though Camels and cattle were not regarded as possible hosts for PPRV some studies had reported PPRV sero-conversion in these animals. Roger *et al.*, 1996 had reported the first documented outbreak of PPR in Camels of Ethiopia. In a study conducted in Sudan PPRV was isolated from Camels that belongs to lineage 4 (Khalafalla *et al.*, 2010). Studies also reported that PPRV sero-conversion in Cattle in Ethiopia and Nigeria by (Abraham *et al.*, 2001; and El-Yuguda *et al.*, 2013), respectively.

6. CONCLUSION AND RECOMMENDATIONS

The current sero-prevalence of PPR in sheep and goats in the selected districts of Afer and Liben zones was 43% while the flock level prevalence was 98%. A flock with at least one positive animal was considered a positive flock for PPR. This shows the transmissibility of the virus within herds is very fast when compared between herds. The fact that antibodies of PPR virus were detected in the whole PA and districts suggests the endemicity of the disease in the studied districts. Hence, the absence of vaccinations in some districts created a pool of young stocks that were susceptible to PPR infection because they were immunologically naïve. The antibodies to PPR virus observed in this study were neither due to vaccination nor maternal antibodies. They must have therefore been due to natural infection with field PPR virus. Remarkably, mixed species rearing was found to be a low disease predictor in sheep population only when being analyzed individually on multivariate logistic regression without considering the cluster effect. Age group, origin of the animal, altitude, production system and utilization of common water source were epidemiological variables that affect the outcome of PPR disease after adjusting the cluster effect of PA and herds. Even though it is important to use cluster sampling when the list of sampling units are not available, it will omit necessary disease predictors unless the random effect is not taken into consideration. From the questionnaire survey, 64% of the respondents were affected by PPR in the last one year starting from the study period. This shows the active circulation of the virus in study areas which needs a strategic vaccination to control the spread of the disease. Dolo Ado, Dolo Bay and Moyale are districts that border with Kenya and Somalia. Therefore, the sero-status of the mentioned districts could give a reflection towards the bordering regions of the neighboring country. Based on the retrospective data, the disease frequency of PPR was high in 2010 that is the middle of the 10 year report. However, it showed a decrease pattern until 2015. This may show the attention that was given towards the disease regionally and nationally through time. A region with no PPR outbreak report doesn't mean there is no PPR. Since outbreak reporting needs awareness towards the disease, harmonization, and dedication of all parties (region, district and field professionals).

Based on this study, the following recommendations were forwarded

- ❖ Based on the sero-results, it is necessary to plan out strategic vaccination not only in the studied regions but also in other regions with a history of recurrent disease outbreaks in order to prevent the circulation of the virus.
- ❖ It would be a milestone if heat resistant (thermo tolerant) vaccine is developed and outreach the remote pastorals where it was very difficult for field veterinarians to sustain the currently used attenuated live PPR vaccine.
- ❖ Further research should be undertaken on the development of DIVA (Differentiating infection from vaccinated animals) vaccine which is the out most important measure in the control and then eradication of PPR.
- ❖ It needs harmonization in the control and eradication of the disease between the study districts and the neighboring countries specially Kenya and Somali where there is active movement of livestock across the poros border.
- ❖ It is necessary to train field veterinarians on the basis of participatory disease appraisal so that they couldn't misdiagnose it and hence could take necessary measure to at field level.
- ❖ To confirm the effect of mixing different species on PPRV transmission further study need to be conducted whether large ruminants like Camels and Cattles play a role in the epidemiology of PPR or they are only dead end host for the disease.

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8. APPENDIXES

Appendix 1: Age and dentition

Number of pairs of permanent incisors	Sheep	Goat
0	Less than one year	Less than one year
1	1 to 1.5 years	1 to 2 years
2	1.5 to 2 years	2 to 3 years
3	2.5 to 3 years	3 to 4 years
4	More than 3 years	More than 4 years
Broken mouth (teeth missing or worn down)	aged	aged

Source: ESGPIP (2009) and Payne, W.J.A. (1990)

Appendix 2: Age determination with figure

0	1	2	3	4	4*
no permanent incisors, only temporary (milk) teeth	one pair of permanent incisors (or two incisor teeth)	two pairs of permanent incisors (or four incisor teeth)	three pairs of permanent incisors (or six incisor teeth)	four pairs of permanent incisors (or eight incisor teeth)	"broken mouth", four pairs of permanent incisors, but very worn, or some fallen out.
					

Source: AU-IBAR-STSD and VS

Appendix 3 : Questionnaire format for PPR risk factor investigation and for individual serum sampled.

Herd number		Name of livestock keeper	
Date		Name of village/location	
GPS coordinats	Lat :	Administrative area 1	
	Long :		
Elevation		Administrative area 2 & higher admin divisions	
Distance to main roads	Km		

1. Herd size : how many animals of different species and ages are in the herd

species	Young (<1 year sheep, goats, pigs. <2 years cattle and camels)		Mature (1 year or more - sheep, goats, pigs. 2 years or more - cattle and camels)		Total
	Male	Female	Male	Female	
Cattle					
Sheep					
Goats					
Pigs					
Camels					

1. Herd dynamics: *in the past year, how many animals have been added to the herd-
In the past year, how many animals have left the herd- death, sale, slaughter, gifts
etc*

species	No. born	No. died	No. bought	No. sold	No. slaughtered	No. gifted or loaned	No. gifts received or borrowed	Other reasons for leaving herd (stolen, lost, predator)	Other reasons for joining herd (stray animal, breeding bull)
Cattle									
Sheep									
Goats									

Species	3. Production type: what is the Main reason for keeping each species			4. Production system: what is the main farming system?			5. Housing: <i>are the animals enclosed?</i>	
	Dairy	meat	Multi-purpos e	Sedentary mixed farming	Agro-pastoralis t	Pastoralis t	Enclosed at night	Enclosed during day
Cattle								
Sheep								
Goat								
camel								

species	6. Grazing: what type of grazing is practiced?				7. Water source: <i>where do the animals drink?</i>	
	Zero-grazed	Fenced	communal	migratory	On the farm	Shared water source
Cattle						
Sheep						
Goats						
Camels						

8. Selling live animals: *in the past one year, what methods have you used to sell animals?*

species	Livestock market	Direct local sale to neighbor or nearby village	Direct sale to middle man/trader	Other
Cattle				
Sheep				
Goats				
Camels				

9. Buying live animals: *in the past one year, what methods have you used to buy animals?*

species	Livestock market	Direct local sale to neighbor or nearby village	Direct sale to middle man/trader	Other
Cattle				
Sheep				
Goats				
Camels				

10. What diseases have affected the herd in the past one year?

Local disease name	Species affected	Number affected	Main clinical signs	Season(s) when occurred	Suspected diagnosis

11. Current disease affecting the herd (using the local terms for PPR, CCPP, ORF- like disease syndromes)

Local disease name	Species affected	Number affected	Main clinical signs	Season(s) when occurred	Suspected diagnosis

12. If an animal becomes sick, what do you usually do?

Do nothing	Treat it myself (or a family member)	Consult a traditional healer	Consult a community animal health worker	Consult an extension officer	Consult a private vet	Consult a government vet	Consult an NGO

13. Where do you get medicines from?

Collect or make myself	Traditional healer	Community animal health worker	Pharmacy	General shop or market stall	Private vet	Government vet	NGO

14. When did you last have vaccinations against the following diseases?

Species	Vaccine	Date of vaccination	Source of vaccine
	PPR		
	CCPP		
	FMD		

Appendix 4: Different studies of PPR in IGAD regions

Sero-prevalence	Study details	Reference
Overall 61.8% (28.6-69.3% variation by State) Sheep 62.9% Goats 59.7%	600 sera (399 sheep, 201 goats) from Sudan, 2005-6 – Blue Nile, Gadarif, North Kordofan States – convenience sample within States. cELISA	Abdella et al., 2012
Overall small ruminants 11.6% (sheep 13%, goats 9%). Variation by Region 0.9% - 22.5%. Cattle 9% Camels 3%	2815 sera from unvaccinated sheep (835), goats (442), cattle (910) and camels (628) in Ethiopia – Afar, Addis, Somali, Borena, Gambela. 276 randomly selected villages. 2001 c-ELISA	Abraham et al., 2015
Overall 1.7%, sheep 0.3%, goats 2.3%	1239 sera (360 sheep, 879 goats) from Awash Fentale District, Afar Region, Ethiopia 2006-7. C-ELISA. Multi-stage random sample.	Delil et al., 2012
Overall 23.1 (sheep 26%, goats 19%) Village prevalence 0-59.1%	254 sheep (164) and goats (90) from 14 villages in Marawi Province, Northern State, Sudan 2008. C-ELISA. Survey design not described.	Enan et al., 2011
Overall 52.5%, sheep 51.9%, goats 57.6% Variation by district – 20-78%	316 sera (106 sheep, 210 goats) Karamoja region of Uganda 2009 cELISA. Area had recent outbreaks and vaccination. Sampling method not described.	Luka et al., 2011
Overall 30.9%, sheep 29.5%, goats 31.3% Variation by Region 27.3-38.3% 100% villages in Afar positive, 97% villages in Gambela.	1163 sera (251 sheep and 912 goats) from Gambela and Afar Regions in Ethiopia 2009-10 cELISA. 32 randomly selected villages.	Megersa et al., 2011
Overall 57.6% Variation by district 1.6-85%	280 goats from northeastern districts of Uganda 2009, cELISA. Convenience sample.	Mulinda et al., 2011
Overall 50.7% Variation by State 39.5-66%	519 sera from sheep (313) and goats (206) – River Nile, Darfur, Blue Nile, Khartoum, Kordofan and Southern Sudan, Sudan 20013. Study design not described.	Osman et al., 2009

Overall 9.4% Variation by district 0-21.3%	960 sheep and goats unvaccinated from 11 districts neighbouring Karamoja in northeast Uganda, 2009, cELISA. Purposive – farms with recent history of disease.	Ruhweza et al., 2010
Overall 62.8% (by area ranged from 51.4-80.6%) 67.2% sheep (by area ranged from 53.3 – 93.8%) 55.6% goat (by area ranged from 0%, 41.7-63.6%) 0.3% camel (1/391)	1198 sera (500 sheep, 306 goats, 392 camels) from 11 States in Sudan (no sampling strategy described), cELISA, 2008	Saeed et al., 2010
Overall 45.6% (sheep 43.7%, goats 47.9%) Variation by State 34.9-57.2% Variation by location 10-91.7%	480 sera from sheep (261) and goats (219) – Sennar, Gedarif, River Nile and North Kordofan States, Sudan 2012-13. C-ELISA. Study design not described.	Salih et al., 2014
Overall 11.4% Variation by district 9.2-13.3% Variation by IDP camp 0-33.3%	474 sera from sheep (8 [^]) and goats (388) in Amuru and Gulu districts – 39 IDP camps	Sande et al., 2011
overall 70.2% (95% ci 67-73%) - Kassala 66.2% (61.7-70.7) - North Kordofan 74.5% (70.278.8) - by locality 57%-88%	820 sheep sera, purposive selection of 2 regions, random selection of one state per region, random selection of 50% localities from each state. No indication of how herds were selected. Unvaccinated herds selected. C-ELISA. 48.7% owners had vaccinated in the past of which 68.4% had vaccinated in 2011	Shuaib et al., 2014
Overall 0.9% Variation by area 0-13%	1025 sera from sheep and goats – north, west, south and central areas of Kenya 1987-1991. Targeted sample from border areas for rinderpest sero-surveillance.	Wamwahi et al., 1995
Overall 6% Variation by Region 1.7% - 21.3% Variation by District 0-53%	13651 sera from unvaccinated sheep and goats, 7 regions of Ethiopia. C-ELISA. Multistage cross-sectional national survey. 1999-2002.	Waret-Szkuta et al., 2008

Source: AU-IBAR