RISK FACTORS AND SOCIO-ECONOMIC EFFECTS ASSOCIATED WITH SPREAD OF *PESTE DES PETITS RUMINANTS* (PPR) IN TURKANA COUNTY, KENYA

A thesis submitted in fulfillment of the requirement for Doctor of Philosophy Degree of University of Nairobi.

(Applied Microbiology-Virology-option)

Dr. Simon Mwangi Kihu, BVM, MSc.

Department of Veterinary Pathology, Microbiology and Parasitology.

2014

Declaration

This thesis is my original work and has not been presented for degree in any other University

Dr. Simon Mwangi Kihu, BVM (UON), MSc (University of London).

Department of Veterinary Pathology, Microbiology and Parasitology, Faculty of Veterinary Medicine, University of Nairobi

Signed----------Date----------

This thesis has been submitted for examination with our approval as University supervisors

Signed----------Date----------
Dr. George Chege Gitao (BVM, MSc, PhD)

Signed----------Date----------
Prof. Lily Caroline Bebora (BVM, MSc, PhD)

Signed----------Date----------
Prof. Njenga Munene John (BVM, MSc, PhD)

Signed----------Date----------
Dr. Gidraph Gachunga Wairire (BSW, MA, PhD)

DEDICATION

I dedicate my thesis work to my family. A special feeling of gratitude to my loving wife, Nyambura whose words of encouragement and push for tenacity ring in my ears. My daughters, Watetu, Wanjiru and Wambui as well as my son Kihu who have never left my side and are very special.

ACKNOWLEDGEMENTS

I sincerely thank my supervisors, Dr G.C. Gitao, Prof. L. C. Bebora, Prof. Njenga M. J. and Dr G.G. Wairire for their guidance and encouragement from the development of the project proposal, through fieldwork and eventual preparation of this thesis.

I am greatly indebted to Forum for Capacity Building in Agriculture (RUFORUM) for the financial support they extended to my project through which this study was possible. Sincere thanks go to Vetworks Eastern Africa, who provided great logistical and material support. A lot of thanks to The University of Nairobi Institute of Tropical and Infectious Diseases (UNITID) and National Veterinary Research Centre of Kenya Agricultural Research Institute for allowing me use of their laboratories in molecular and serologic analysis.

I wish to sincerely thank the Catholic Missionaries of Turkana specifically Sr. A.W. Burugu and Sr Veronica of Lodwar, Sr L. Mwendwa of Kakuma; all of Assumption Sisters of Nairobi (ASN) for their support and provision of medical laboratories facilities for my use. I also extend my gratitude to Catholic Fathers and Brothers in Kaaleng and Kaeris for their brotherly welcome and accommodation at their premises.

I sincerely thank the Turkana community in all locations of this study (administrators, herders and traders) for their warm welcome, homely accommodation and invaluable

participation without which this project would have been incomplete. Indeed much thanks go to Dr. H. Hambe and P. T. Segut for their assistance with field laboratory equipment and space at a critical time when I least expected to access such facilities in the field.

My thanks are due to all colleagues who assisted me in one way or another in process of this study. Finally thanks to my family for their prayers; being patient, understanding and coping with problems that my study period may have imposed upon them particularly in the last days of this work.

TABLE OF CONTENT

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

AE	Acceptable Error
AGID	Agar Gel diffusion Test
AGID	Agar Gel Immuno-diffusion Test
ALRMP	Arid lands resource monitoring project.
AMREF	African Medical Research Foundation
ASAL	Arid and Semi Arid Lands
ASN	Assumption Sisters of Nairobi
BCA	Benefit cost analysis.
B-ELISA	Blocking Enzyme-linked immunosorbent assay
BSW	Bachelor of Social Work
BVM	Bachelor of Veterinary Medicine
CCPP	Contagious Caprine Pleuropneumonia
cDNA	complementary deoxyribonucleic acid
CDV	Canine distemper virus
C-ELISA	Competitive Enzyme-linked immunosorbent assay
CFSPH	Center for Food Security and Public Health
CI	confidence interval
CIEP	Counter immunoelectrophoresis
DPX	Distyrene Plasticizer Xylene
EPC	External positive control

FAO	Food and Agricultural Organization of United Nation
FFPE	Formalin-fixed, paraffin-embedded
FMD	Foot and Mouth Disease
GOK	Government of Kenya
GPS	Global Positioning System
HA	Haemagglutination test
HI	Haemagglutination Inhibition Test
IBM	International business machines
ICE	Immuno- Capture Enzyme-linked immunosorbent assay
IC-ELISA	Immuno-Capture Enzyme-linked immunosorbent assay
IFAT	Immunofluorescent Antibody Test
IHC	Immunohistochemistry
IIRR	International Institute of Rural Reconstruction
INR	Indian Rupee
IP	Immunoperoxidase Staining
IPC	Internal positive control
ITDG	Intermediate Technology Development Group
KES	Kenya Shilling
KMO	Kaiser-Mayer Olkin
KNBS	Kenya National Bureau of Statistics
KVAPS	Kenya Veterinary Association Privatization Scheme
LRT	Likelihood ratio test

LSI	*Laboratoire Service International*
MAM	March, April and May
MSc	Masters of Science
MSIER	Maternal, susceptible, infectious, exposed and recovered
MV	Measles Virus
n.d.	no date
n/a	not applicable
NC	Negative control
NCS	Negative control sample
NGN	Nigerian Naira
NGO	Non-governmental organization
NP	Nucleoprotein
NPV	Net Present Value
OD	Optical density
OIE	*Office International des Epizooties*
OND	October, November and December
PARC	Pakistan Agricultural Research Council
PDV	Phocine distemper virus
PE	Participatory Epidemiology
PhD	Doctor of Philosophy
PIT	Precipitinogen Test
PM	Post Mortem

PP	Proportion piling
PPR	Peste des petit ruminants
PPRV	Peste des petit ruminants virus
qRT-PCR	real time Reverse transcriptase Polymerase chain reaction
RF	Reference
RNA	ribonucleic acid
RP	Rinderpest
RPV	Rinderpest Virus
RT-PCR	Reverse transcriptase Polymerase chain reaction
RUFORUM	Regional Universities Forum for Capacity Building in Agriculture
SAARC	South Asian Association for Regional Cooperation
SD	Standard Deviation
SE	Standard Error
S-ELISA	Sandwich enzyme linked immunosorbent assay
SIR	Susceptible infectious and recovered
SPSS	Statistical Package for the Social Sciences
SSI	Semi structured interviews
TMB	Tetramethylbenzidine
UK	United Kingdom
UNITID	University of Nairobi institute of infectious diseases
UON	University of Nairobi
US$	United States Dollars

USA	United States of America
VIPR	Virus Pathogen Resource
VNT	Virus Neutralization test
VSFB	*Veterinaires Sans Fronteires* Belgium

ABSTRACT

Livestock keeping is the main source of livelihood for most pastoral households found in arid and semi arid lands (ASAL) of Kenya which are characterized by prevalence of diseases, extreme climatic features of drought, flooding, low investments, fragile ecosystems and high poverty levels as challenges to the pastoral livestock sector growth. Small stock keeping is one of the major livelihood activities for the pastoral communities which contribute heavily to pastoral household subsistence and market income. A major constraint to small stock keeping is emerging viral diseases including PPR that is a relatively new, highly contagious and often fatal disease of sheep and goats that has caused devastating losses in Kenya since it was first officially reported in 2007 in the Turkana County.

Peste des petit ruminants has since spread to almost all ASAL pastoral counties in Kenya. Efforts to control the disease in Kenya have been limited due to lack of epidemiological information while the risk factors and socio-economic effects associated with the spread of the disease in Turkana District are not fully known. As such it has not been clear how effective the control activities implemented had been in stemming the spread of the disease in Kenya.

The general objective of the study thus was to assess the risk factors and socio-economic effects associated with the spread of PPR in Turkana County, while the specific

objectives were to: (1) identify the risk factors influencing the patterns of disease spread: (2) Determine the level of herd immunity against the disease within the flocks: (3) Determine the disease socio economic impact and (4) Document and evaluate the control strategies of the disease in Turkana County of Kenya.

The risk factors associated with the spread of PPR in Turkana County were identified using participatory epidemiology (PE) methodologies. The data on community participatory appraisal of PPR disease was validated with field pathological samples that were collected and the PPR virus RNA analyzed with qRT-PCR both in fresh frozen samples and formalin fixed tissues. Histology samples were also examined for pathological lesions associated with PPR. Further participatory risk assessment questionnaires were used to determine community perception of PPR on the risk factors. The level of herd immunity was determined using serological methods namely cELISA tests to analyze in 969 serum samples (431 from sheep and 538 from goats) collected in six divisions of Turkana county that formed the study area. The socio economic impact of PPR in Turkana County was determined using data derived from PE methodologies, key informants interviews and secondary data. The current control strategies on PPR in Turkana Kenya were determined using participatory epidemiology methodologies and were subsequently documented. A stochastic PPR compartmental model comprising maternal antibody, susceptible, exposed, infectious and recovered was developed based on field parameters; it was then used to evaluate the appropriateness of vaccination

regimes applied in Turkana. The benefit cost analysis of vaccination was then determined from economic losses due to PPR and cost of vaccination in Kenya.

Results of PE exercises showed that the Turkana community was in agreement that PPR in sheep was associated with migration ($p<0.001$), herd mixing ($p<0.001$), raids ($p<0.001$), and dry season ($p<0.01$) while in goats PPR was associated with migration ($p<0.001$), herd mixing($p<0.001$), raids ($p<0.001$), mountain pastures ($p<0.001$) and dry season ($p<0.001$) . However the risk factors significantly associated with the spread of PPR in Turkana County were sharing of water points with odds ratio of 2.022 ($p<0.001$) particularly during wet season of 2009. Sick nursing mothers were also identified as risk factor during the wet season of 2010 with odds ratio of 1.621 ($p<0.049$). In a subsequent sero-epidemiological study species was significantly associated with presence of PPR antibodies in a mixed herd of sheep and goats and thus considered risk factors where goat had Odds ratio= 1.644; ($p<0.001$). Unvaccinated sheep had odds ratio of 4.24 ($p<0.000$) compared to the vaccinated sheep while adult goats had odds ratio of 2.381 ($p<0.003$) in relation to kids for association with presence of PPR antibodies.

The level of herd immunity within the flocks in Turkana was found to be 39.6% in goats which was significantly ($p<0.001$) higher than that of sheep at 31.6%. In both species, middle aged group between 6 months and 24 months were found to have low sero-positivity of between 14.2% and 18.2% rendering this group vulnerable during PPR outbreak. The sero-epidemiological survey established that high demographic changes in

small stock renders herds to lose herd immunity as new large numbers of weaned kids and lambs lose maternal immunity and become naïve and vulnerable to PPR infection.

Apart from drought, livestock diseases were the second most important factor that disrupt livestock livelihood with PPR being ranked as the disease with highest destructive impact on the small stock benefits to the Turkana community. The socio economic impact of PPR was found to be enormous in that it threatens to destroy sheep and goats that constitute the largest herd of livestock reared; their herd composition ranging between 42% and 64.4% of the total animal kept across the wealth groups. Lose of small stock would in essence destroy a source of animal food products which constitute 29.4% of all the food consumed by a Turkana household and is mainly consumed by the youthful workforce of morans and girl herders in the age group 13 to 18. The study established that the direct economic losses due to PPR in Turkana County alone for the year 2010 were in the tune of Kenya Shillings 11.1 billion.

The current control strategies on PPR in Turkana County, Kenya have been found to be mainly local methods of using herbs as well as reducing contact between sick herds and healthy herds. Local methods that reduced animal contacts such as running away from sick herds and local sanctions such as restricted movement or/and access to public water points and pastures were consistent with scientific control method of creating sanitary belts. Vaccination was perceived to be among the most effective control method but was not easily accessed and the community had little knowledge about it; thus was ranked lowly among other control methods.

Peste des petits ruminants disease maternal antibody, susceptible, exposed, infectious and recovered (MSIER) model established that sheep and goats have different disease dynamics due to among others species herd demographic differences. The study established PPR reproductive number (R_0) in sheep and goats to be different, though low than what has been reported by other studies. *Peste des petits ruminants* disease in sheep herds was seen to persist longer than was the case in the goats and thus may serve as the reservoir for virus in between outbreaks. A simulation of the model showed that vaccination coverage of 50% of combined sheep and goats herds was enough to curtail the spread of the PPR disease within 254 days. A spreadsheet model found vaccination was the most viable control method with a cost benefit of PPR vaccination being 35.

In conclusion there is need to utilize this wealth of indigenous knowledge on diseases of livestock that reside with pastoral communities, for purposes of understanding diseases in the community and setting up strong participatory surveillance systems that involve the communities as the basic element of disease surveillance intelligence gathering. The herd immunity against PPR in Turkana is low for both sheep and goats to allow for containment of spread of the disease with small stock in the middle aged group being the most susceptible to PPR infection because they were immunologically naïve. Since small stock are owned across Turkana and are a major livestock livelihood catering for both subsistence and market income, the community appreciate PPR control measures such as vaccination seen as effective control method despite having little knowledge

about it. Vaccination programs targeting 50% coverage of small stock herds will control and eventually eradicate the disease

It is thus recommended that indigenous livestock diseases knowledge form part of participatory surveillance systems that involve the communities as the basic element of disease surveillance intelligence gathering. A comprehensive PPR control strategy in the arid and semi arid pastoral are be crafted based on the key risk factors of PPR established from this study. Annual vaccination against PPR is carried out to improve the herd immunity to levels that can contain spread of the PPR disease with emphasis of vaccinating middle age group. The county government should come up with social protection measures for example livestock insurance, disaster fund and social safety nets that can cushion the pastoralists against the ravages of PPR. The central government, to provide adequate capacity for the support of the local community on requisite measures to safeguard themselves against the negative impact of PPR. *Peste des petits ruminants* disease control policy should envisage annual vaccination in risky areas focusing in 50% coverage with priority given to middle aged small stock.

CHAPTER 1: GENERAL INTRODUCTION

Peste des petit ruminants is a highly contagious often fatal disease of sheep, goats and wild small ruminants. The disease is caused by *Peste des petit ruminants* virus (PPRV) classified under genus Morbillivirus (Gibbs *et al*., 1979). The disease occurs in Middle East, South Asia, Central Asia, China and Africa. In the East African region the disease has been described in Sudan, Uganda, Tanzania, Ethiopia, Eritrea, Somalia and Kenya. In Kenya it was first suspected in 1992 (FAO, 2008) and confirmed in Turkana District in 2007 (*Office International des Epizooties* (OIE), 2014). The disease has since spread to almost 35 arid pastoral counties in Kenya.

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).

The disease is ranked among the top ten diseases of small ruminants by communities where the disease occurs or is endemic (Diallo, 2006). The direct economic losses caused by the disease are high and are aggravated by the sanitary measures imposed to control the disease. Control of the disease is through control of movement and vaccination of animals. Studies assessing benefits of vaccination against PPR in Niger revealed that the

program was highly beneficial (Diallo, 2006). It is estimated that 62.5% of global domestic small ruminant population is at risk of infection with PPR (FAO, 2009a). However data on economic losses due to the disease is scanty. This is complicated by confusion of PPR with other diseases like contagious caprine pleuropneumonia (CCPP) and helminthiasis which lead to underestimation of its economic impacts.

The disease is relatively new in Kenya and little is known about the factors that are responsible for the disease spread in the country with about 44,869,759 small stocks at risk of infection. However, efforts to control the disease in Kenya have been limited due to lack of epidemiological information, limited funding as well as inadequate technical support. It is not clear how effective the current control activities have been in stemming the spread of the disease in Kenya. This study aims at determining risk factors and socio-economic effects associated with the spread of PPR in Turkana County of Kenya, evaluating current control strategies and determining the socio-economic impacts of the disease.

1.1 Objectives of the Study

1.1.1 General objective

To identify risk factors and assess socio-economic effects associated with the spread of PPR in Turkana County, Kenya

1.1.2 Specific objectives

1. To identify risk factors influencing the patterns of PPR spread in Turkana County, Kenya.
2. To determine the level of herd immunity within the flocks.
3. To assess the socio economic impact of the disease.
4. To document and evaluate current control strategies used in Turkana, Kenya.

1.3 Justification of the study

The PPR disease in East Africa and more so in Kenya is not well understood since there are very limited studies that have been carried out in an attempt to elucidate the status of the disease in the region and country Kenya. The disease has been reported in Sudan and Ethiopia since the 1980s yet the PPR clinical disease was only identified in Kenya as from 2006. In Kenya PPR was first suspected in 1992 through serology however the country was considered free of the disease until 2006 when the PPR confirmed outbreak occurred in Turkana County. The disease caused massive losses on livestock assets of the Kenyan pastoral communities particularly the Turkana herders. This study is aimed at creating new knowledge on PPR in Kenya with a focus to informing the policy development in regard to control, prevention and eventual eradication of the disease. Further insights from the study will inform rehabilitation of affected communities so that they can regain meaningful livelihood through pastoralism.

CHAPTER 2: GENERAL LITERATURE REVIEW

The literature review highlights the PPR disease description and its social economic impact.

2.1 Disease definition and history

Peste des petits ruminants is an acute or sub-acute febrile, highly contagious and often fatal disease of sheep, goats and wild small ruminants (Furley *et al*., 1987). The disease is characterized by fever, erosive stomatitis, conjunctivitis, gastroenteritis, pneumonia and causes serious economic losses in small ruminant's production (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. *Peste des petits ruminants* is also known as goat plague, pseudo rinderpest of small ruminants, pest of small ruminants, pest of sheep and goats, Kata, stomatitis-pneumoenteritis syndrome, contagious pustular stomatitis, and pneumoenteritis 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).

2.2 Causative agent of the disease

Peste des petits ruminants disease is caused by PPRV which was first isolated in cell culture by Gilbert and Monnier in 1962. Despite the description and the isolation of PPRV, it was still believed that PPRV was a variant of rinderpest virus (RPV) that was adapted to small ruminants (Bourdin and Laurent, 1967; Laurent, 1968). This was based on the fact that cattle vaccinated with tissue culture of PPRV were protected against rinderpest (Gilbert and Monnier, 1962) while Bourdin (1973) also observed that rinderpest cell culture vaccine protected goats against PPR. The cross reactivity between the PPRV and RPV made it difficult for some diagnostic tests to distinguish between the two viral agents particularly when the tests were based on polyclonal antibody (Gopilo, 2005). However based on serological studies (Taylor, 1979; Gibbs *et al*., 1979), cDNA probes studies (Diallo *et al.,* 1989b) and biomolecular studies (Barret, 2001) PPRV was found to be different from RPV and was classified as the fourth member of Morbillivirus genus (Gibbs *et al*., 1979). Further studies carried out on the RPV and PPRV demonstrated that the two ruminants *Morbilliviruses* were distinctly different (Diallo, 2006). Moreover it has been established through gene sequencing and analysis that PPRV is not that closely related to RPV as initially thought (Diallo, 2006). In contrast RPV is more closely related to measles virus among other viruses in *Morbillivirus* genus (Diallo *et al.,* 1994).

2.3 PPR virus classification

Peste des petits ruminants virus is in the *Morbillivirus* genus of the order *Mononegavirales*, *Paramyxoviridae* family, subfamily *Paramyxovirinae*, which is divided into five distinct genera (Murphy and Parks 1999). Virions in the genus *Morbillivirus* are pleomorphic particles with a lipid envelope enclosing a ribonucleoprotein core that contains the genome, which is encapsidated by the nucleocapsid (Mahapatra *et al.,* 2006). *Morbiliviruses* have an identical genome organization (Barret *et al.,* 1991; Banyard *et al.,* 2005) with 15–16 kb in length and 200nm in diameter (Norrby and Oxman, 1990). The genus has general characteristics that include cytopathic effects in cell culture (syncytia, inclusions in the cytoplasm and nucleus), a common histopathological appearance (multinucleated giant cells) and a strong close antigenic relationship (high homology between proteins of different members of the group) (Dufour, 2010). One of the distinguishing characteristics of *Morbillivirus* genus from other genera of Paramyxovirinae is the lack of neuraminidase activity.

Morbillivirus genus has seven viruses that include PPRV*,* measles virus, RPV, canine distemper virus, phocine morbillivirus; porpoise distemper virus and dolphin morbillivirus (Barret *et al*., 1993). Sequencing studies of relatively well conserved proteins in this viral group of *Morbillivirus* genus have established relationships between the various viruses in this genus (Figure 2.1). On the basis of phylogenetic analysis of *morbilliviruses* an ancestral virus is believed to have evolved into other members of the morbillivirus at different times in history (Barrett and Rossiter, 1999; Westover and Hughes 2001).

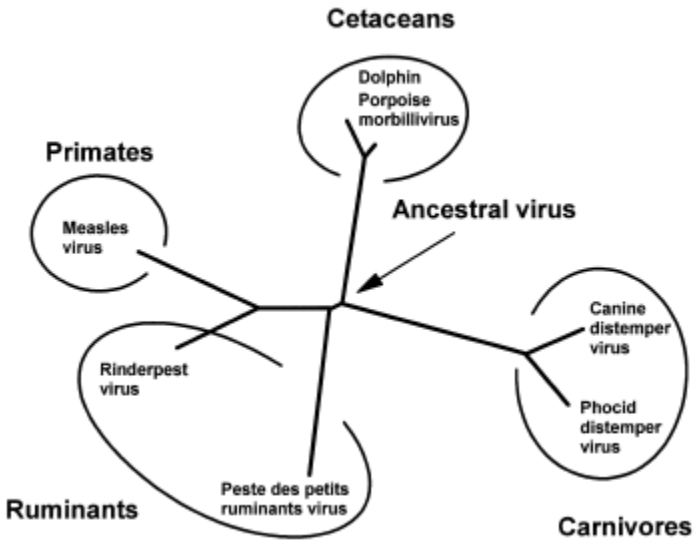


Figure 2.1: Phylogenetic tree showing the relationships of morbilliviruses of different species (Barrett, 1999)

Relationship between measles virus (MV) and RPV is much closer than between PPRV and RPV. Canine distemper virus (CDV) is closely related to phocine distemper virus (PDV) (Gopilo, 2005). Canine distemper and PDV are the most distantly related to MV and RPV among *morbilliviruses* (Figure 2.2) (Barret and Rossiter, 1999).

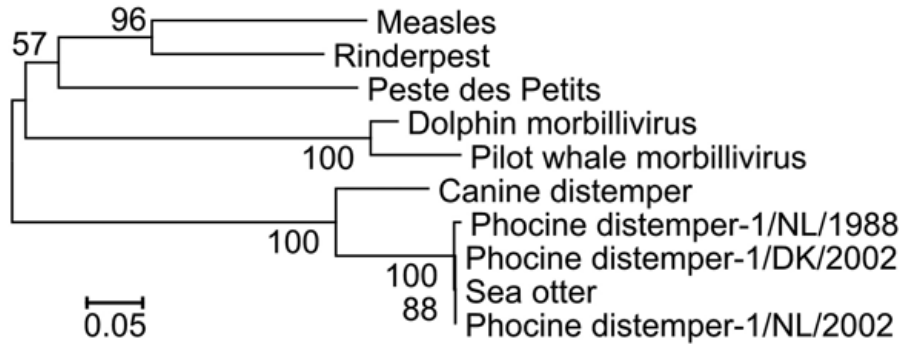


Figure 2.2: Neighbour-joining bootstrap tree showing morbillivirus relationships. (Goldstein *et al.,* 2009)

2.4 Physical chemical and biological characteristics of PPR virus

2.4.1 General morphology

Peste des petit ruminants virus, like other *Morbilliviruses*, is an enveloped, non-segmented negative-strand RNA virus (Qin *et al.*, 2012) (Figure 2.3). The PPRV genome encodes six structural proteins (nucleocapsid (N), phosphoprotein (P), matrix (M), fusion (F), hemagglutinin (H), and polymerase (L)), and two nonstructural proteins (C and V), which are in the order of 3′-N-P/C/V-M-F-H-L-5′ on the genome (Hu *et al*., 2012). Specifically the genome of PPRV is 15,948 kb (Bailey *et al.,* 2005) (Figure 2.4)

The matrix (M) protein is linked to the nucleocapsid and surface proteins F and H (Mahapatra *et al*., 2006). The three viral proteins (M, F and H) are associated with the host-derived envelope (Munir *et al*., 2013). The L protein acts as RNA-dependent RNA polymerase. The association of P protein to N and L is linked to viral cycle control, transcription and translation regulation (Munir *et al.*, 2013).

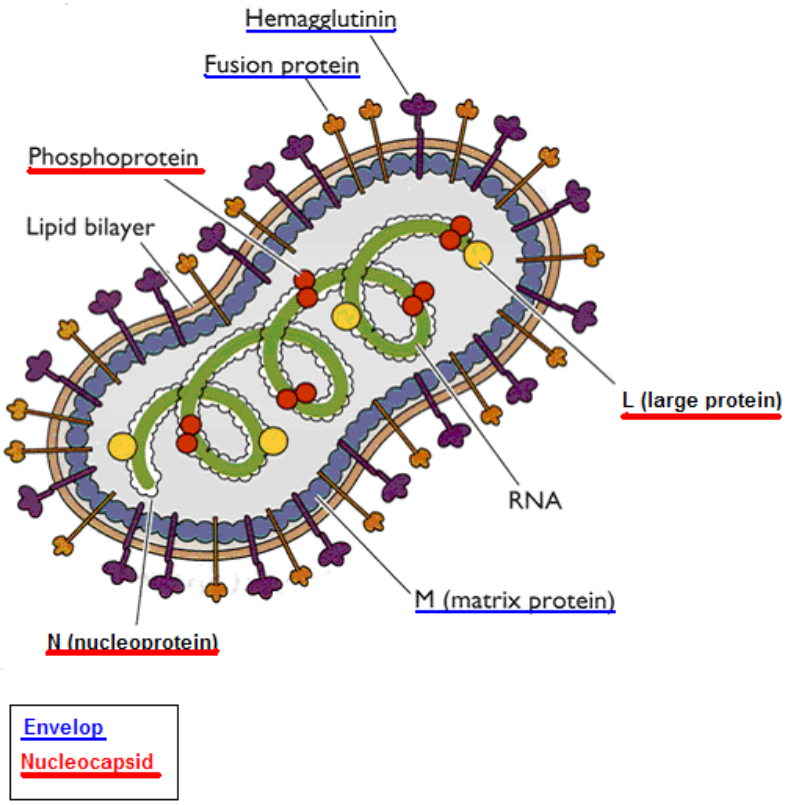


Figure 2.3: Structure of *Peste des petits ruminant virus* (Dufuor, 2010)

The sequencing of the genes encoding the viral proteins F (Shaila *et al.,* 1996) or N (Kwiatek *et al.,* 2007) has highlighted the genetic diversity of strains which were then classified into four genetically distinct lineages. This classification coincides well with the geographic distribution of different strains and has been very useful to speculate about the spread of the disease.

2.4.2 Physical characteristics

The *Peste des petits ruminants* virus has an envelope derived from the host-cell plasma membrane, containing two transmembrane glyocproteins surrounding a nucleocapsid.

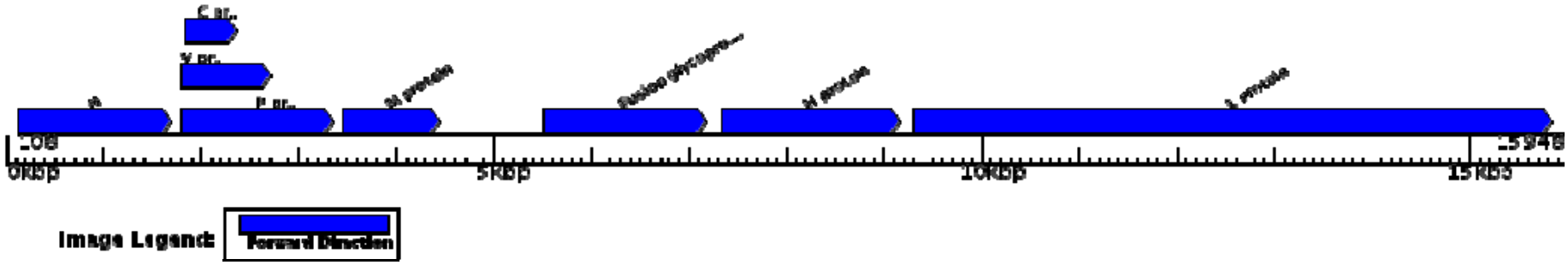


Figure 2.4: Genome Image Map of *Peste des petits ruminants (VIPR, 2014)*

The presence of the envelope renders virions sensitive to heat, lipid solvents or detergents, non-ionic detergents, formaldehyde and oxidizing agents. *Peste des petits ruminants* virus is also very sensitive to ultraviolet radiation and desiccation. Like all enveloped viruses, PPRV is very sensitive to heat. The half-life of the virus at 37°C was estimated at 2 hours, and at 50°C infectivity was destroyed in 30 minutes (Lefevre, 1982). Other studies have confirmed and clarified the thermal sensitivity of PPRV (Rossiter and Taylor, 1994; Diallo, 2000). *Peste des petits ruminants* virus has been shown to survive in lymph nodes for 8 days at 4°C (Lefevre, 1982).

The PPR virus is also sensitive to low pH, being destroyed after death of the animal by the low pH which accompanies rigor mortis. *Peste des petits ruminants* virus is stable at pH between 5.8 and 9.5 but rapidly loses activity at pH below 4 or above 11 at room temperature (Diallo, 1990). The optimum pH for survival of PPRV is between 7 and 8 (Dufuor, 2010).

2.5 Geographical distribution of the disease

From late 1980s onwards, the development of specific diagnostic tools has enhanced the, understanding of the geographical distribution of PPR disease globally (Diallo *et al.,* 1995). *Peste des petit ruminants* is found in Middle East, South Asia, Central Asia (Uzbekistan, Tajikistan, and Turkmenistan), China, and Africa (Abu Elzein *et al.,* 1990; Ozkul *et al*., 2002; Shaila *et al*., 1989, Nanda *et al.,* 1996; Kwiatek *et al.,* 2007; Wang *et al.,* 2009 Anees *et al.,* 2013). Recent data indicates the activity of PPRV in all countries

of Africa except those in Southern African region. In Africa, PPR disease is found in West, Central, East, and North African countries and currently the disease is heading to southern Africa countries (Barnyard *et al.,* 2010, Munir *et al.,* 2013). The disease has been reported in the Maghreb region of Africa where the disease was thought to be absent (Dufuor 2010; De Nardi *et al*., 2011). Countries in Central and southern Africa reporting their first ever PPR outbreaks include Gabon, Democratic Republic of Congo and Angola (Veterinary Record, 2012; Maganga *et al.,* 2013; OIE, 2012a). Within the Eastern Africa region, PPR disease has been reported in Sudan (Elhag and Taylor, 1984), Ethiopia (Roeder *et al.,* 1994), Eritrea (Sumption *et al.,* 1998; Cosseddu *et al.,* 2013), Uganda (Luka *et al.,* 2011) , Tanzania (Muse *et al.,* 2012) and in Kenya (Kihu *et al.,* 2012a). Figure 2.5 shows the global distribution of countries that have reported PPR disease.

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) (Figure 2.6). 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 (Figures 2.7) while Lineage 3 is responsible for PPR outbreaks in Tanzania (Figure 2.8) (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).

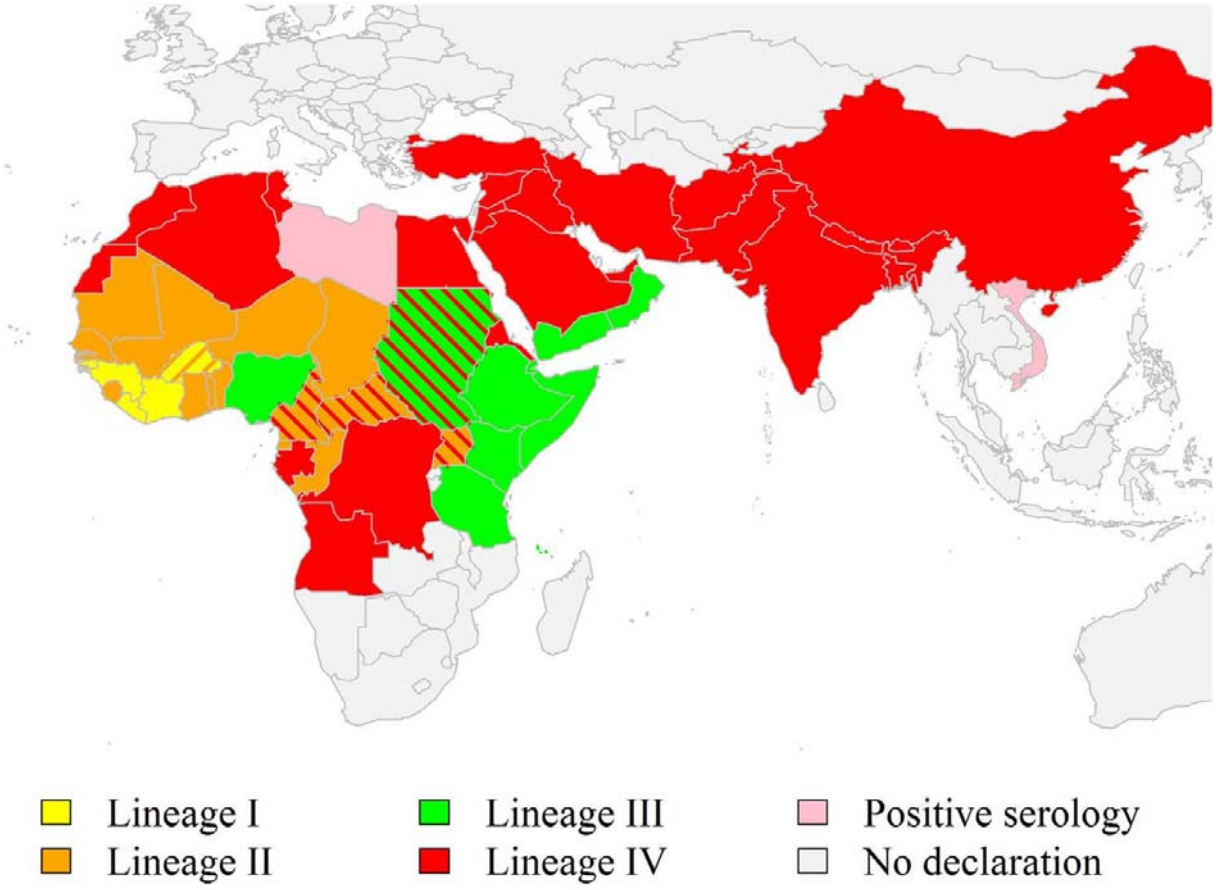


Figure 2.5: Map depicting the worldwide cumulative distribution of the four peste des petits ruminants virus (PPRV) lineages. (Libeau *et al.,* 2014)

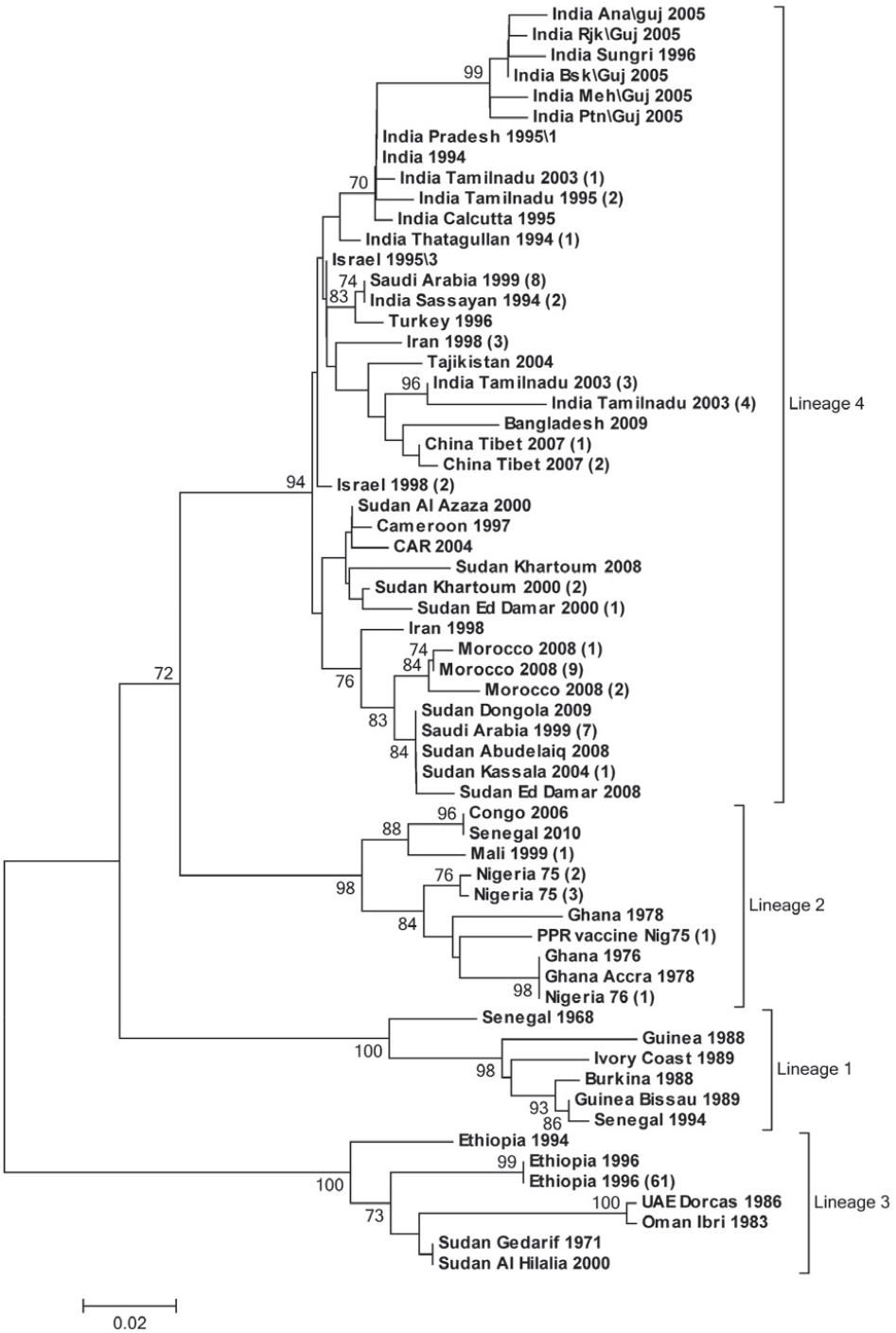


Figure 2.6: Phylogenetic tree representing the genetic diversity in peste des petits ruminants virus globally (Libeau *et al.,* 2014)

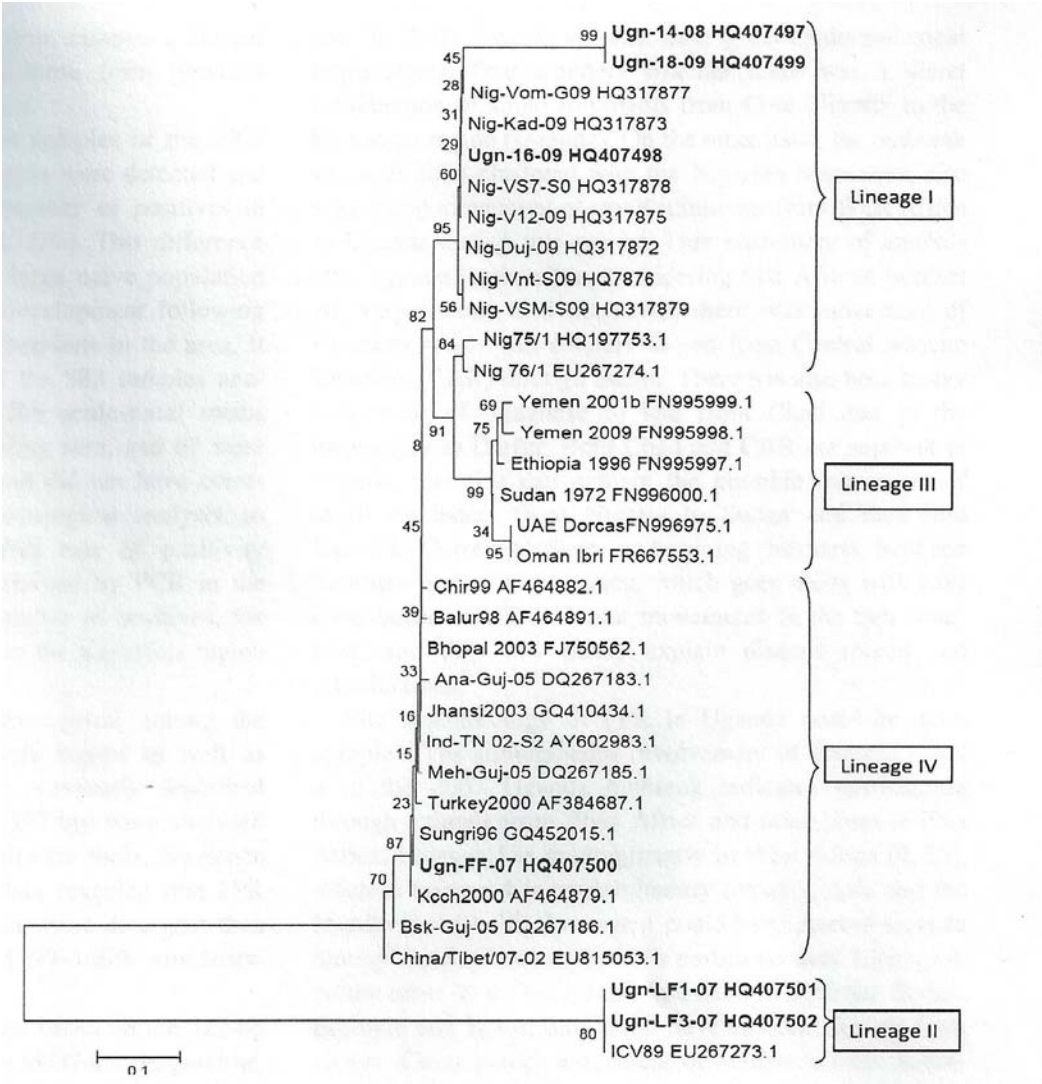


Figure 2.7: Phylogenic relationships between peste des petits ruminants virus detected in Uganda in 2007/2008 to the other virus isolates. (Luka *et al.,* 2012)

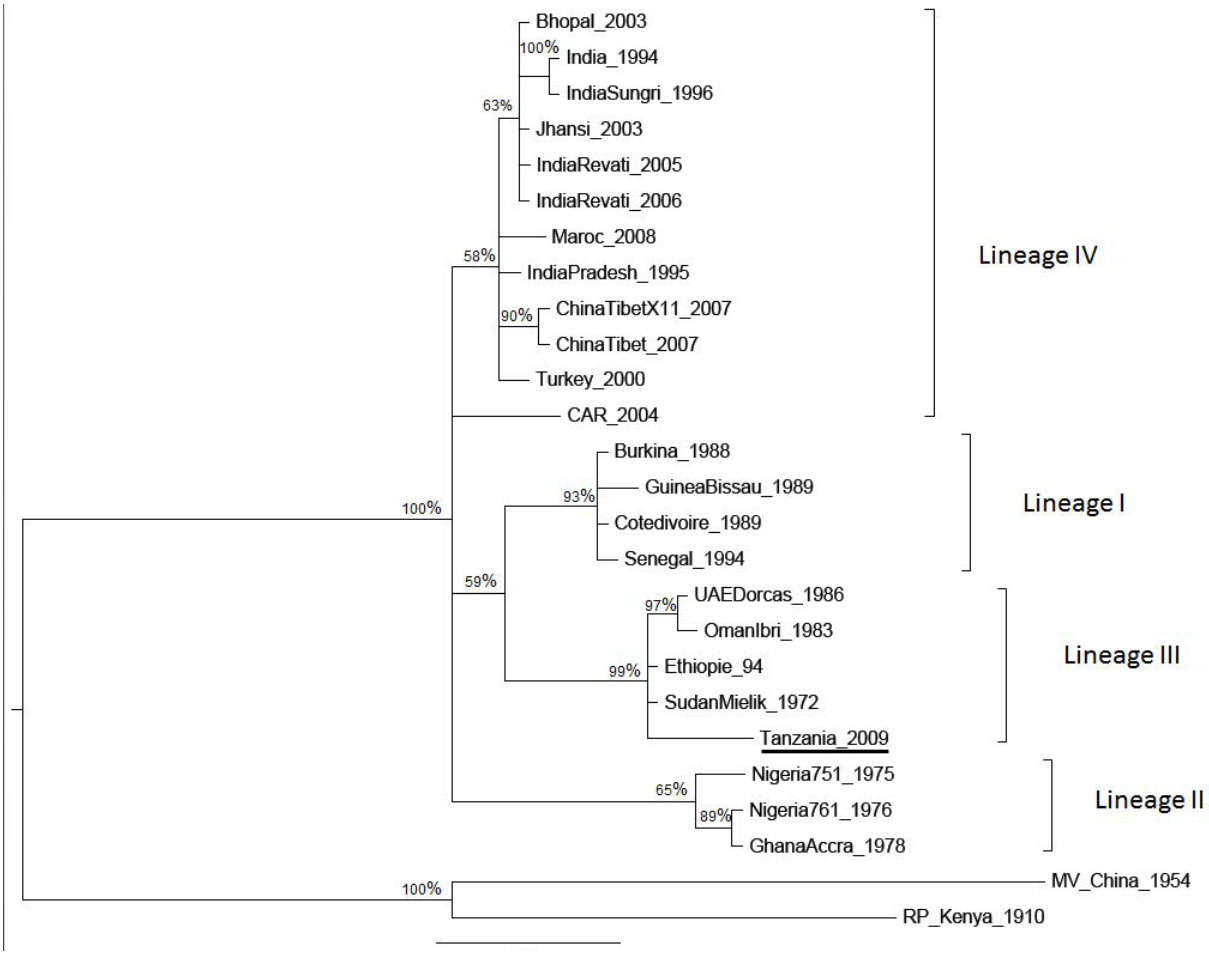


Figure 2.8: Phylogenetic tree based on the nucleoprotein gene of the peste des petits ruminants virus, positioning the Tanzania strain in lineage III (Kivaria *et al.,* 2013).

2.5.1 Peste des petits ruminants in Kenya

In Kenya PPR was first suspected in 1992 (FAO, 2008) with further serological reports being made by Wamwayi *et al.*, (1995). Clinical PPR disease in Kenya was first reported in Turkana County by the Director of Veterinary Services of Kenya to World Animal Health Organization (*Office International des Epizooties* (OIE)) in January 2007 (OIE, 2014). The PPR clinical disease in Kenya had first been suspected by pastoral

communities and local livestock NGOs in May 2006 in Oropoi and Lokichoggio divisions of Turkana County, Rift Valley province in Kenya (personal communication, Dr Irura and Dr Omori). Following these initial reports of PPR in 2006, and the advent of heavy short rains of 2006, the disease was reported to have spread to Kakuma, Loima and Kibish division of Turkana districts as depicted in a risk map reported by *Veterinaire San Fronteires* Belgium (VSFB) by January 2007 (Figure 2.9)

Disease assessments carried out in mid-2007 established that the PPR had managed to spread beyond Turkana district into the neighboring districts of Samburu, Pokot and Baringo (Kihu *et al.,* 2012a). The disease has since spread in most of all the arid and semi-arid pastoral districts in Northern and Southern Kenya (Figure 2.10) with several outbreaks reported to OIE as late as 2013 (FAO 2009; OIE, 2014).

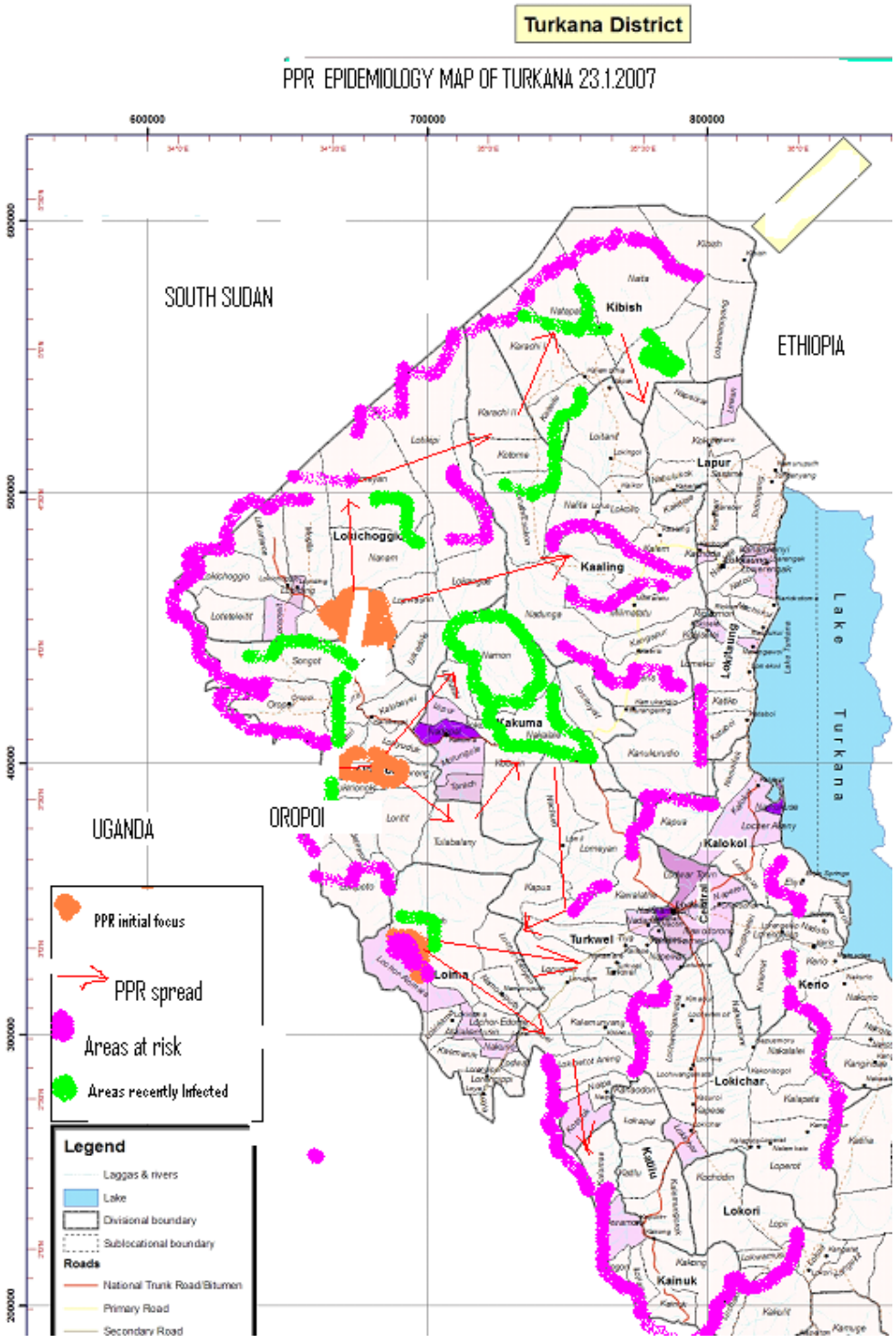


Figure 2.9: Initial PPR risk map of Turkana reported by VSFB in 2007

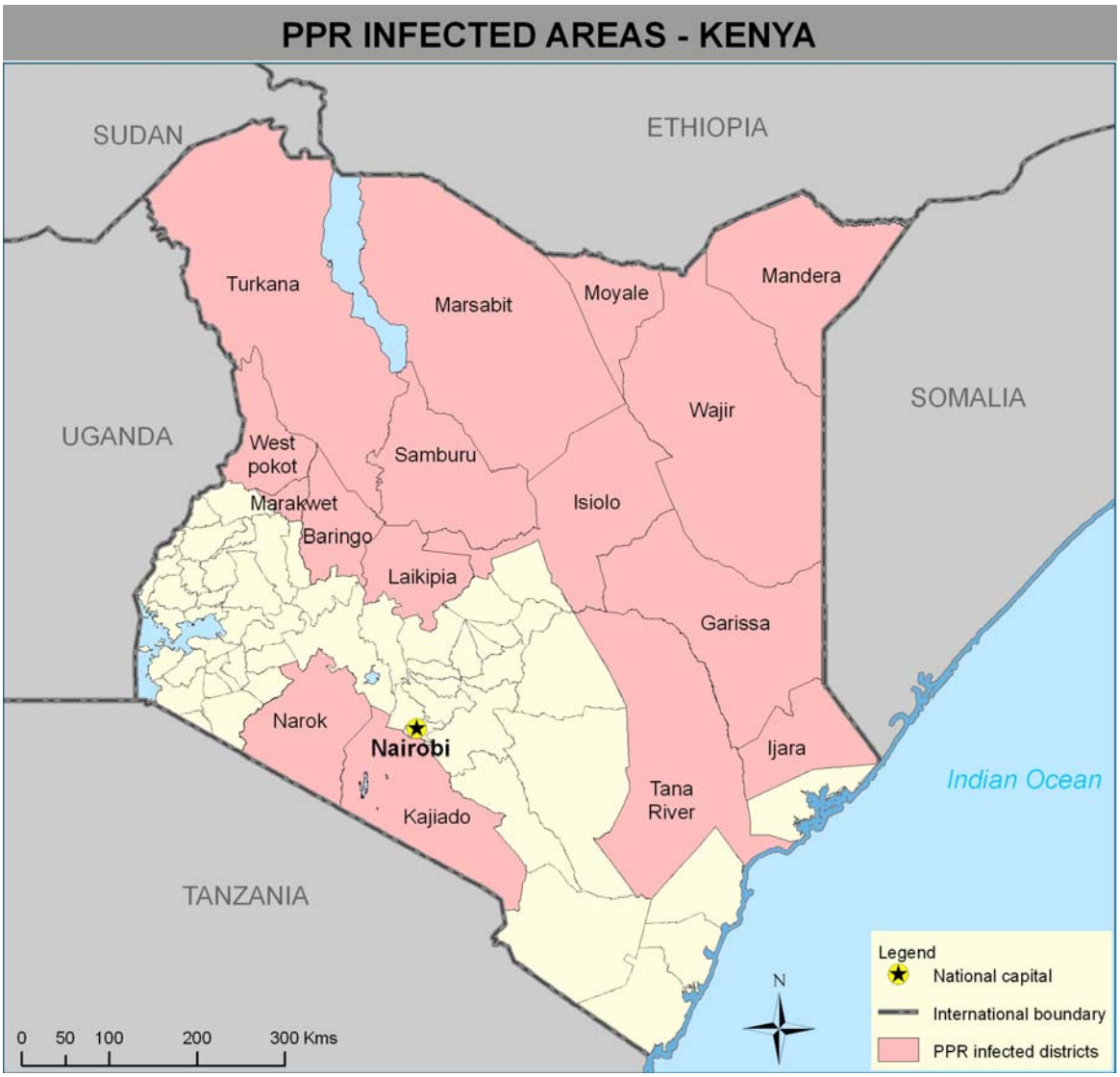


Figure 2.10: PPR status in Kenya by 2009 (FAO 2009)

2.6 Transmission of the disease

Peste des petits ruminants disease is transmitted by contact between infected animals in the febrile stage and susceptible animals (Gopilo, 2005; Munir *et al.,* 2013). Large

quantities of the virus are shed through ocula-nasal discharges as well as through the watery diarrhea (Center for Food Security and Public Health (CFSPH), 2008,). Secretions and excretions from an incubating animal contain virus 24 to 48 hours before the clinical diseases (Couacy-Hymann *et al.,* 2007; William and Barker, 2001; Abegunde and Adu, 1977). Sneezing and coughing by infected animal can spread infection, and transmission can occur through inhalation of droplets by animals in vicinity of 10 meters (CFSPH, 2008, Munir *et al.,* 2013). Fomites in contact with infected animals such as water, feed troughs and bedding could become additional sources of infection but for very short periods of time (Gopilo, 2005, CFSPH, 2008, Munir *et al.,* 2013). However, the PPR virus is very labile thus limiting its survival period outside the host to very short time (Lefevre and Diallo, 1990; Rossiter and Taylor 1994).

Maturation of meat lowers the pH to levels that deactivates the PPR virus thus meat from infected carcasses do not seem to present any risk of contamination though it has been reported that chilled lymph nodes from infected animal were infective after eight days (Lefevre*,* 1982; MacDiarmid and Thompson, 1997).

There is no carrier status for PPRV and the virus relies on a constant supply of new susceptible hosts for its maintenance (Munir *et al*., 2013; Dufour, 2010; Gopilo 2005).

2.7 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).

2.8 Host determinants of PPR

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).

2.9 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).

The increase in incidences of PPR outbreaks has been attributed more to an increased number of susceptible small ruminants recruited rather than seasonal upsurges in the viral activity (Taylor *et al.,* 1990). However 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). Climatic factors that affect availability of pasture and water, contribute to increased movements of small stock in search of better nutrition and shelter against the adverse climatic conditions; consequently, this aids spread of the PPR to susceptible groups (Singh *et al.,* 2004b). The seasonal epidemiologic patterns of the PPR disease differ in different ecological systems, geographical areas and are dependent on culture and livelihood patterns of small stock owners (Gopilo, 2005). In Pakistan, seasonal

outbreaks of PPR were alluded (Abubakar *et al.,* 2011; Abubakar *et al.,* 2009), suggesting that seasonal grazing patterns among nomadic livestock keepers during winter encourage diseases transmission (Munir *et al.,* 2008). Similar observations were made by Sarker *et al* (2011) who associated PPR outbreaks in Bangladesh to winter grazing. *Peste des petits ruminants* outbreaks among sheep and goats in India are described to occur any time of the year, but are most frequent during the wet (April to September / October) or cold dry (January and February) seasons (Balamurugan *et al.,* 2012; Singh *et al.,* 2004b).

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 (Gopilo, 2005, Waret-Szkuta *et al.,* 2008).

2.10 Pathogenesis of the disease

Peste des petits ruminants virus exhibits lympho-epitheliotropism (Scott, 1981). Lymphotropic nature, common to all Morbilliviruses, causes a severe leukopenia in infected animals, which promotes the development of secondary infections by bacterial agents or parasitic opportunists who take advantage of the induced immunosuppression and severe clinical pictures (Munir *et al*., 2013). Infection occurs primarily through naso-pharyngeal route via inhalation of virus particles (Dufuor, 2010). Then, the virus multiplies in lymphoid organs before regional spread through blood to the epithelial cells of respiratory and gastro-intestinal tract (Gopilo, 2005).

The *Peste des petits ruminants* virus causes cytopathic effect that is distinguished from that of other *Morbilliviruses*, by it characteristic appearance of multinucleated cells capable of forming round mini syncytia and intracytoplasmic inclusion bodies and

eosinophilic intra–nuclear inclusion bodies (Troung *et al*., 2014). The fusion between an infected cell and neighboring cells (syncytia) aided by viral fusion protein (F) which is expressed on the surface of infected cells is one way of spreading the virus. This process of spreading the infection from cell to cell through fusion allows the virus to continue the infectious process free of neutralizing antibodies as nucleocapsids migrate from cell to cell without passing through the external environment. The second process of spreading viral infection occurs when budding at the membrane of the infected cell takes place releasing virions (Figure 2.11). The virions are thus released into the outside environment and can spread the infection by binding to other cells.

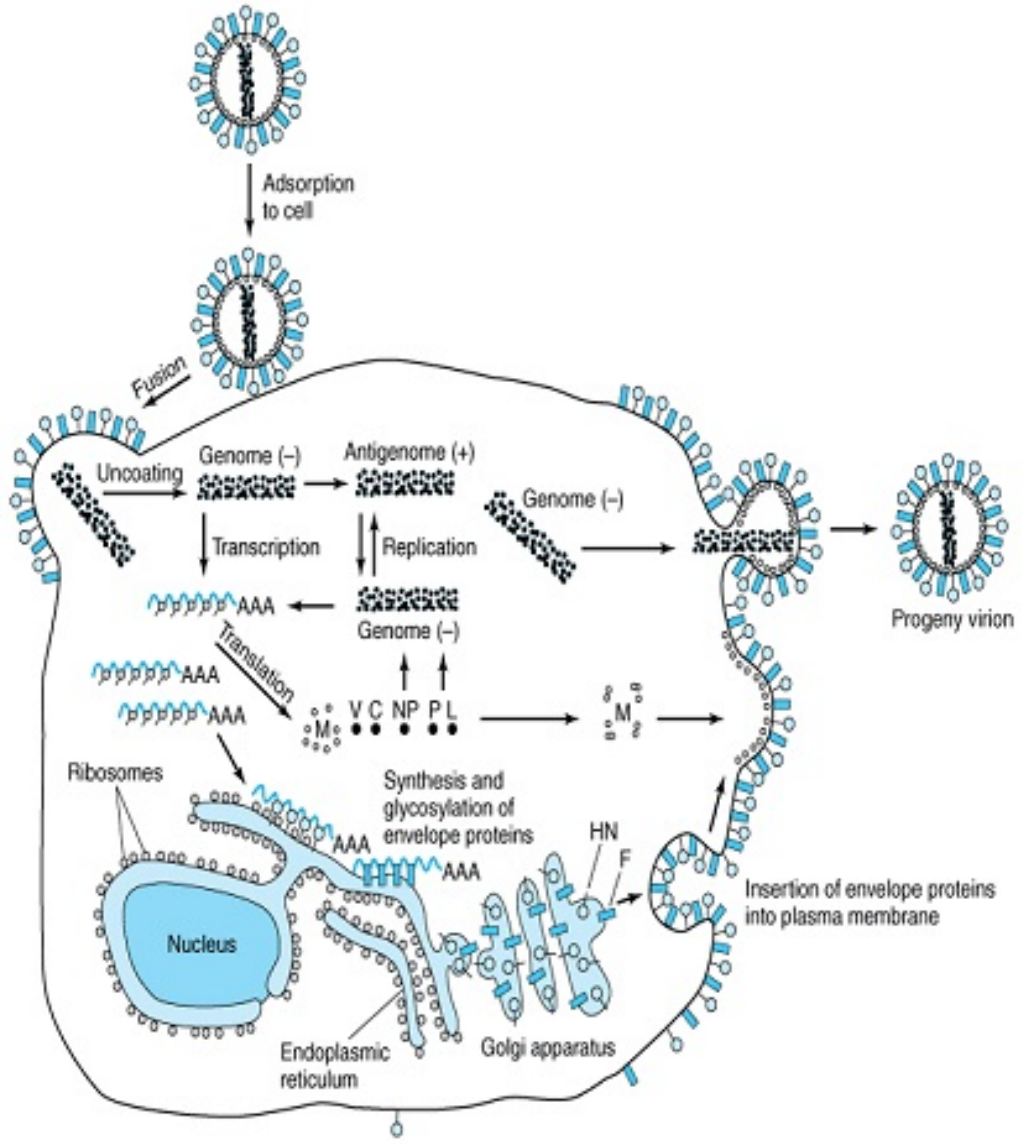


Figure 2.11: Showing the process of Morbillivirus virus replication (Brooks *et al.,* 2008)

2.11 Clinical Signs of the disease

The clinical presentation of PPR can be peracute, acute, sub-acute or subclinical as described by Munir *et al.,* (2013). The predominant form of the PPR disease

presentation is the acute form. Clinical signs of PPR have been documented by various reports (Hamdy *et al.,* 1976; Obi, 1984; Lefèvre, 1987; Taylor, 1984; Bundza *et al.,* 1988; Roeder *et al.,* 1994; Roeder and Obi, 1999). Following infection there is a 3–4 day incubation period during which the virus replicates in the draining lymph nodes of the oro-pharynx before spreading via the blood and lymph to other tissues and organs including the lungs causing a primary viral pneumonia. The salient clinical signs start with sudden rise in body temperature to 39.5 - 41°C. Affected animals breathe fast, show difficult and noisy breathing. A clear watery discharge starts to flow from the eyes, nose and mouth, later becoming thick and yellow as a result of secondary bacterial infection. Serous to mucopurulent nasal discharge crust over and occlude the nostrils causing sneezing and difficulty in breathing (Figure 2.12).

Figure 2.12: Mucopurulent nasal discharge and swollen upper lips (Roeder and Obi, 1999)

Serous to mucopurulent ocular discharges ensue causing matting together of the eyelids (Figure 2.13). One to two days after fever has set in, the mucous membranes of the

mouth and eyes become much reddened. Then, epithelial necrosis causes small pin-point greyish areas on the gums,

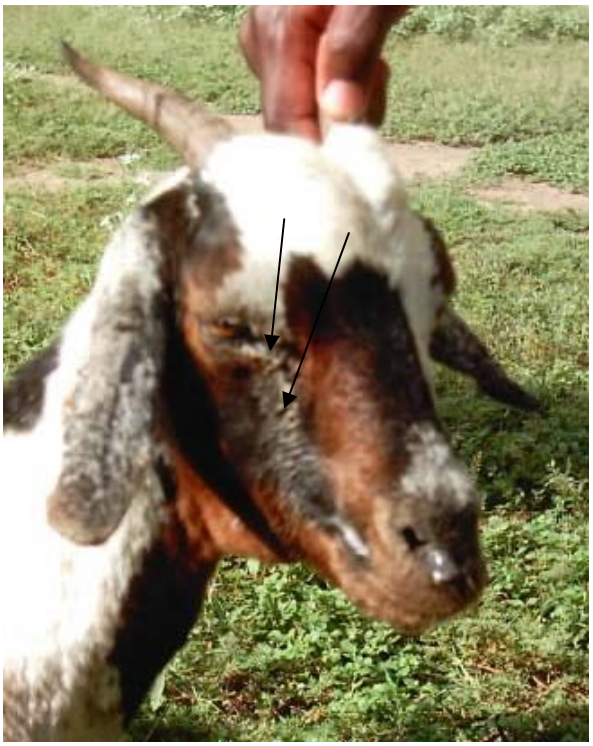


Figure 2.13: Mucopurulent ocular discharge matting hair from canthus of the eye (Kihu *et al.,* 2012a)

dental pad, palate, lips, inner aspects of the cheeks and upper surface of the tongue. These areas increase in number and size and join together. The lining of the mouth is changed in appearance. It becomes pale and coated with dying cells and, in some cases; the normal membrane may be completely obscured by a thick cheesy material. Underneath the dead surface cells, there are shallow erosions. Gentle rubbing across the gum and palate with a finger may yield a foul-smelling material containing shreds of epithelial tissue (Braide, 1981) (Figure 2.14)

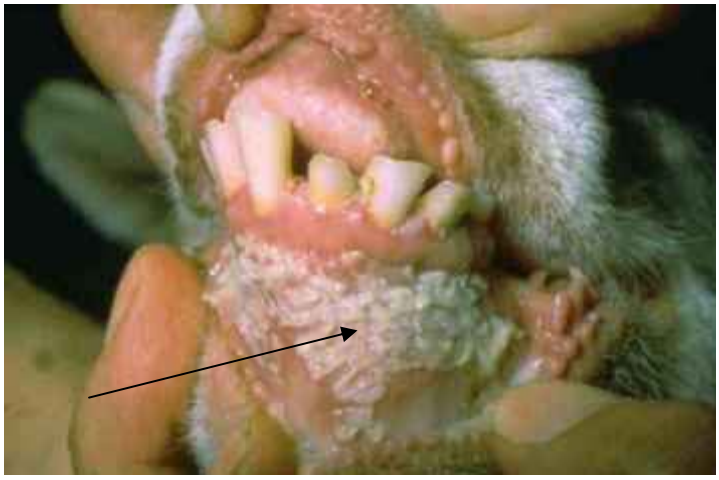


Figure 2.14: Erosive stomatitis with dead cells on the gums involving the inside of the lower lip (Berhe, 2006)

Two to three days after the onset of fever diarrhea appears though in early or mild cases, it may not be obvious. The feces are initially soft and then watery, foul-smelling and may contain blood streaks and pieces of dead gut tissue. *Peste des petits ruminants* cases presenting diarrhea eventually become dehydrated, emaciated with sunken eyeballs, and death often follows within seven to ten days from onset of the clinical reaction Munir *et al.*,2013 (Figure 2.15). Body temperature usually remains high for about 5-8 days, and then slowly returns to normal prior to recovery or drops below normal before death. Some animals will recover after a protracted convalescence. A common feature in later stages of the sub-acute disease is the formation of small nodular lesions in the skin on the outside of the lips around the muzzle (Muse *et al.,* 2012)

Figure 2.15: A case of PPR showing soiled behind, emaciated, depressed and nasal discharges (courtesy Dr Njiru of Moyale County)

2.12 Pathology of the disease

The carcass of a PPR affected animal is usually emaciated, the hindquarters soiled with soft/watery faeces and the eyeballs sunken. The eyes and nose contain dried-up discharges. Lips may be swollen and possibly scabs or nodules in late cases. The nasal cavity is **c**ongested (reddened) lining with clear or creamy yellow exudates and erosions. The pathology caused by PPR is dominated by necrotizing and ulcerative lesions in the mouth and the gastro-intestinal tract (Roeder *et al*., 1994). Erosion in the oral cavity is a constant feature affecting the gums, soft and hard palates, tongue and cheeks and into the oesophagus. Abomasum is congested with lining hemorrhages. The rumen reticulum and omasum rarely exhibit lesions. Occasionally, there may be erosions on the pillars of the rumen. The omasum is a common site of regularly outlined erosions often with oozing blood. Lesions in the small intestine are generally moderate, being limited to small streaks

of hemorrhages and, occasionally, erosions in the first portions of the duodenum and the terminal ileum. The large intestine is usually more severely affected, with congestion around the ileo-cecal valve, at the ceco-colic junction and in the rectum. In the posterior part of the colon and the rectum, discontinuous streaks of congestion "zebra stripes" form on the crests of the mucosal folds (Figure 2.16).

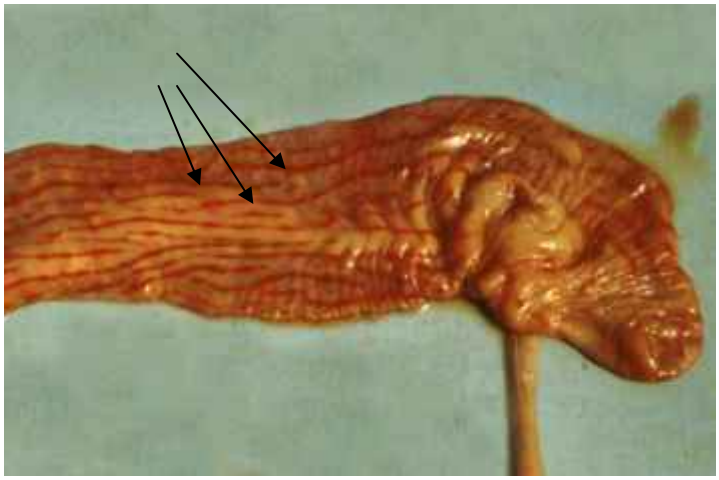


Figure 2.16: PPR in a goat: "zebra striping" in the large intestine (Roeder and Obi, 1999)

In the respiratory system, small erosion and petechiae may be visible on the nasal mucosa, turbinates, larynx and trachea. Bronchopneumonia may be present, usually confined to the antero-ventral areas, and is characterized by consolidation and atelectasis. The lung is dark red or purple with areas firm to the touch, mainly in the anterior and cardiac lobes show evidence of pneumonia (Figure 2.17). Lymph nodes associated with the lungs and the intestines are soft and swollen.

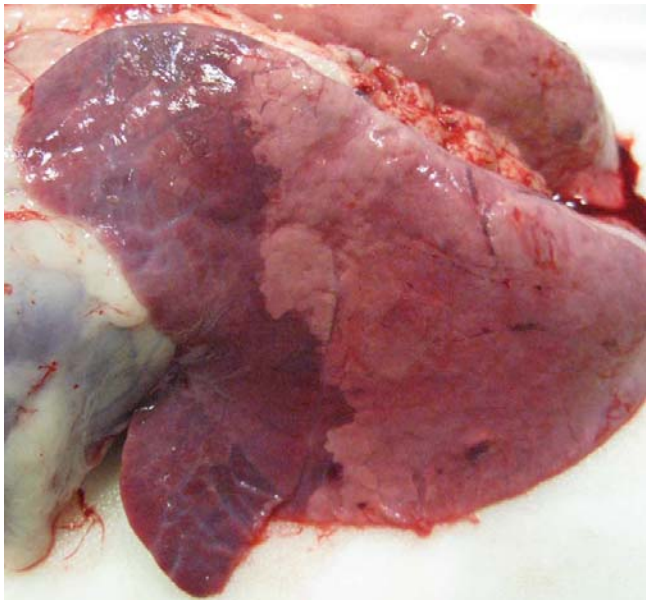


Figure 2.17: Bronchointerstitial pneumonia due to PPR infection (Troung *et al.,* 2014)

2.12.1 Histology of the disease

Peste des petits ruminants virus causes epithelial necrosis of the mucosa of the alimentary and respiratory tracts marked by the presence of eosinophilic intracytoplasmic and intranuclear inclusion bodies (Brown *et al*., 1991). Multinucleated giant cells (syncytia) can be observed in all affected epithelia as well as in the lymph nodes where there is severe depletion of lymphocytes (Troung *et al.,* 2014). In the lungs multifocal degeneration, ulceration and necrosis, followed by alveolar type II pneumocytes hyperplasia which mostly ends up with syncytial cell formation is a prominent feature (Munir *et al.,* 2013). Infiltration of the lymphocytes, plasma cells and histiocytes into the alveolar septae leads to its hypertrophy and desquamation with alveolar casts (Munir *et al.,* 2013). Intestinal lesions are characterized by blunted villi, degeneration of surface and crypt epithelial cells; expansion

of lamina propria by a primarily mononuclear infiltration with scattered syncytial cells (Troung *et al.,* 2014).

2.13 Immunity mounted towards the disease virus

The PPR virus is antigenically closely related to rinderpest virus (RPV), another member of the *Morbillivirus* genus. Antibodies against PPRV are both cross-neutralizing (Taylor 1979a) and cross-protective (Taylor 1979b) against RPV. The H protein and the F protein of RPV or the F protein of PPRV conferred protection against PPR disease in goats, but without production of PPRV-neutralizing antibodies (Romero *et al*., 1995). These suggest that cell-mediated immunity may play a major role in protection against PPR, in the absence of PPRV-neutralizing antibodies (Sinnathamby *et al*., 2001).

The protective immune response is usually elicited against the surface F and H proteins of PPRV which are highly immunogenic (Mahapatra *et al* 2006). The fusion proteins (F) elicit cellular immune response while hemagglutinin (H) elicits humoral immune response and thus both proteins are a major consideration in development of PPR vaccines (Dufour, 2010). The H protein binds to its cognate receptor on the host cell during the first step of the viral infection process; as such, it acts as a major antigen that stimulates a protective immune response in the host (Qin *et al*., 2012). Therefore among the viral proteins most of the neutralizing antibodies are directed against the H protein during PPRV infection (Diallo *et al.*, 2007). As H and F proteins are directly in contact

with antibodies in the external environment they are under constant pressure of the immune system and are therefore subject to frequent mutations, unlike the nucleoprotein (N), which itself is well preserved (Diallo, 2003). In all members of the genus *Morbillivirus* including PPRV, the N protein is the most abundant viral protein due to its presence at the extreme 3′-end of the viral genome (Parida *et al*., 2007). Owing to its high quantity during infection, the N protein is considered the most immunogenic, but the immunity produced against N protein does not protect the animals from the disease (Choi *et al*., 2005). By virtue of the nature of the H and N proteins, these remain the most acceptable targets for the design of PPRV diagnostic tools (Munir *et al*., 2012).

Most of the diagnostic assays for PPRV have been developed based on monoclonal antibodies (mAb) produced against the N protein (Libeau *et al.*, 1995). However there is an increasing need to design strategies which will differentiate between antibodies due to infection from those due to vaccination (DIVA strategies); these are vaccine strategies targeting the H protein of PPRV. The design of DIVA strategies for both vaccines and assays is informed by the fact that the current PPR homologous vaccines confer immunity to vaccinated animals that have a full range of immune responses to viral proteins; therefore, these vaccinated animals cannot be distinguished serologically from those that have recovered from natural infection (Maharaptra *et al*., 2006). This has led to difficulties in disease surveillance, as the sera from both vaccinated and naturally infected animals produce similar results in standard serological tests (Anderson and McKay, 1994).

Due to immunogenicity of PPRV, animals recovering from natural PPR infection or vaccination with PPR homologous vaccine acquire a protective immunity that is lifelong and thus cannot get infected with the disease again. This conferred PPR immunity is active against all strains of PPRV, despite genetic variability of the virus in the four lineages. Kids and lambs born to vaccinated or recovered mothers, acquire maternal immunity through colostrum. Maternally derived antibodies against PPR in young animals can be detected up to 6 months of age (Balamurugan *et al.,* 2012). However the maternal antibodies fall to below the protection threshold level at 3.5 and 4.5 months of age in lambs and kids, respectively (Awa *et al.,* 2003). Balamurugan *et al*., (2012b) notes that maternal antibodies in kids fall below protective titers by fourth month of age, while studies by Bodjo *et al* (2006) suggest that protective maternal antibodies against PPRV in lambs are depleted at the age of 75 days (two months and five days). Lambs tend to lose their protective immunity at an earlier age than kids, suggesting that lambs and kids borne of vaccinated or infected dams should be vaccinated at ages of between three to four months and between four to five months, respectively (Balamurugan *et al*., 2012; Awa *et al.,* 2003; Bodjo *et al* 2006; Ata *et al* 1989).

Studies on serum neutralization tests on PPRV have placed the protection threshold at PPR antibody titers of 10 (Diallo *et al.,* 1989a) and 8 (Rossiter *et al.,* 1985); OIE places it at 8 and/or above. In this regard serum samples with percentage inhibitions of more than 50% in PPR c-ELISA were considered to have protective antibody titers (Saravanan *et al*., 2010).

Since there is no carrier status in the PPRV infection (Munir *et al.,* 2013; Dufour, 2010; Gopilo 2005*)*, the virus is assumed to be perpetuated by the constant influx of new population of susceptible hosts.

2.14 Diagnosis of the disease

In the field, a presumptive diagnosis of PPR can be made on the basis of clinical, pathological, and epizootiological findings. However laboratory confirmation is an absolute requirement. Diagnosis of PPR may be performed through virus isolation, detection of viral antigens, nucleic acid isolation and sequencing; and detection of specific antibody in the serum (Gopilo, 2005).

2.14.1 Virus isolation

Detection of the virus is done by isolation of the PPR virus in cultured cells. This method of diagnosis can be very valuable as it provides live virus for biological characterization studies and the isolated viruses are stored for later studies (Roeder and Obi, 1999). Samples for virus isolation include heparinized blood, eye and nasal swabs (from live animals), tonsil, mesenteric lymph nodes, spleen, section of colon and lung from necropsied cases. For successful isolation, samples must be collected during the hyperthermic phase (Lefevre, 1987) and submitted to the testing laboratory in cold ice. The most widely used cell culture systems are primary lamb kidney and ovine skin (Gilbert and Monnier, 1962; Laurent, 1968, Taylor and Abegune, 1979; Adombi et al., 2011) and Vero cells (Hamdy *et al*., 1976).

2.14.2 Molecular techniques

2.14.2.1 Nucleic acid recognition methods

Reverse transcription polymerase chain reaction (RT-PCR) techniques based on the amplification of parts of the N and F protein genes has been developed for the specific diagnosis of PPR (Couacy-Hymann *et al.,* 2002; Forsyth & Barrett, 1995). This technique is 1000 times more sensitive than classical virus titration on Vero cells (Couacy-Hymann *et al.,* 2002) with the advantage that results are obtained in 5 hours, including the RNA extraction, instead of 10–12 days for virus isolation.

2.14.2.2 Specific cDNA Hybridization

Nucleic acid technology was applied to the detection of RP and PPR viruses by using cDNA probes corresponding to the nucleocapsid gene of each virus and labeled with [P^{32}] nucleotides (Diallo *et al.,* 1989b). This hybridization technique is used to clearly identify the virus involved in an outbreak (Taylor *et al.,* 1990). Unfortunately, this hybridization cannot be used widely because it requires fresh specimens and in addition to the short half life of [P^{32}], and there is constraints with the handling of isotopes.

2.14.3 Serological tests

2.14.3.1 Viral antigen detecting tests

2.14.3.1.1 Agar Gel Immunodiffusion Test

Detection of virus antigens by the agar gel immunodiffusion test (AGID) is a relatively simple, fast and cheap process. It is extremely useful as an initial test, but it does not

discriminate between PPR and RP viruses and further tests are needed to do this. Known hyperimmune antisera against PPR are used for testing the test antigen. Results are obtained in one day, but the test is not sensitive enough to detect mild forms of PPR due to the low quantity of viral antigen that is excreted (OIE, 2013).

2.14.3.1.2 Counter immunoelectrophoresis

Counter immunoelectrophoresis (CIEP) is in the same principle as the AGID except that the gel is electrically charged to improve the sensitivity of the test. Counter immunoelectrophoresis is most rapid test for detecting viral antigen. It is important to note that both the AGID and the CIEP are group-specific and may not distinguish between PPR and RP infections (Obi and Patrick, 1984).

2.14.3.1.3 Haemagglutination Test (HA)

Haemagglutination test is an easy, cheap and effective method for PPRV diagnosis that has advantage of the differentiating PPR from RPV (Johnson and Ritchie, 1968).

2.14.3.1.4 Immunofluorescent Antibody Test (IFAT)

The IFAT is simple and relatively quick, and has the advantage that facilities are available in most veterinary laboratories (Last *et al.,* 1994). The IFAT technique is reported to have 100% specificity in detection of PPR antigen in conjunctival smears from suspected cases of PPR collected from a field outbreak (Sumption *et al.,* 1998).

2.14.3.1.5 Immunoperoxidase Staining (IP)/Immunohistochemistry (IHC)

Histopathology combined with immunohistochemical staining (e.g. immunoperoxidase) is a useful procedure because it is performed on formalin–fixed material and can discriminate between PPR and rinderpest when performed with specific monoclonal antibodies. Specific IHC reaction is characterized by the presence of light to dark brown, fine to coarse granular area in cells and tissues (Kumar *et al.,* 2004).

2.14.3.1.6 Sandwich ELISA (S-ELISA)

PPR virus-specific neutralizing MAb are used in a simple and rapid double-antibody Sandwich ELISA for specific detection of PPRV antigen in goat tissues and secretions (Saliki *et al.,* 1994). Singh *at al.,* (2004a) described a Sandwich ELISA test using PPR specific MAb (clone 4G6) to N protein. The technique which is simple, convenient, rapid and cost effective is preferred for intensive clinical surveillance and routine diagnosis of the disease (Singh *et al.,* 2004a).

2.14.3.1.7 Immunocapture ELISA (IC-ELISA)

Virus antigens can also be detected by immunocapture ELISA (ICE) which is rapid and sensitive, and differentiates between PPR and rinderpest. The IC-ELISA allows a rapid differential identification of PPR or RP viruses, and this is of great importance as the two diseases have a similar geographical distribution and may affect the same animal species (Diallo, 2000; Diallo, 2004).

2.14.3 .2 Antibody detecting tests

2.14.3 .2.1 Agar Gel Diffusion Test (AGID)

Agar gel immunodiffusion test was used for the detection of antibodies against PPR in the sera of the affected goats (Durojaiye, 1982). Known PPR antigen is used for testing the test sera. This test is considered useful for field diagnosis of PPR.

2.14.3 .2.2 Precipitinogen Inhibition Test (PIT)

The principle of PIT is based on the ability of antibody in serum to inhibit diffusible virus antigen (precipitinogen) from developing a precipitin line against hyper immune serum in AGPT. It was observed that this test is more sensitive (33%) as compared to Neutralisation Test (NT) (28%) (Durojaiye, 1987).

2.14.3 .2.3 Virus Neutralisation Test (VNT)

The virus neutralisation test (VNT) is sensitive and specific, but time-consuming and expensive. Virus neutralization test is the most reliable test for detection of morbillivirus antibodies (Rossitter *et al.,* 1985). Serum against either PPR or RP may neutralise both viruses, but would neutralize the homologous virus at a higher titer than the heterologous virus. Therefore for differentiation purpose reciprocal cross neutralization is used (Taylor and Abegunde, 1979).

2.14.3 .2.4 Haemagglutination Inhibition Test (HI)

The technique is based on adsorbing out the cross reacting antibodies to rinderpest antigen from a PPR serum and leaving the specific antibody to PPR which is determined by haemagglutination-inhibition test (Wosu, 1985)

2.14.3 .2.5 Counter Immunoelectrophoresis (CIEP)

The CIEP is highly adaptable for use in titration of serum antibody and can be used for sero-epidemiological studies as well as experimental studies on PPR (Majiyagbe *et al.,* 1984).

2.14.3 .2.6 Competitive Enzyme-Linked Immunosorbent Assay (C-ELISA)

The C-ELISA is considered suitable for large scale testing due to its simplicity and availability of the recombinant antigen (Libeau *et al.,* 1995). Competitive Enzyme-Linked Immunosorbent Assay sensitivity is 99.4 % and specificity 94.5%. A competitive ELISA based on PPRV monoclonal antibodies specific for haemagglutinin (H) protein (Anderson *et al.,* 1991 ;) or nucleoprotein (N) (Libeau *et al.,* 1995) was developed for detection of antibodies to PPRV in serum samples of sheep and goats. The nucleoprotein (N) based diagnostic kit microplate wells are coated with purified recombinant nucleoprotein (NP). The samples to be tested and the control are added to the microwells. Anti-NP antibodies, if present, form an antibody-antigen complex which masks the NP epitopes. An anti-NP-peroxidase (Po) conjugate is added to the microwells. The Anti-NP-peroxidase monoclonal antibody conjugate competes with the serum antibodies in order to fix to the NP epitopes on the remaining coated antigen. Where no serum anti-NP antibodies are present, the anti-NP-peroxidase monoclonal antibody conjugate fixes the free NP epitopes forming an antigen-conjugate-peroxidase complex. After washing, in order to eliminate the excess conjugate, the substrate solution Tetramethylbenzidine (TMB) is added. The resulting coloration depends on the quantity of specific antibodies present in the sample to be tested. In absence of

antibodies, a blue solution appears which becomes yellow after addition of the stop solution. In presence of antibodies, no coloration appears. This test may be a useful tool for a standardized and accurate determination of the immune status of animals because of its superior sensitivity to conventional tests.

2.14.3 .2.7 Blocking ELISA (B-ELISA)

Blocking ELISA is proved to be simple, more rapid, sensitive and specific method for detection of PPR antibodies (Saliki *et al.,* 1993). Unlike the VNT, B-ELISA may be less affected by the quality of sera such as cytotoxicity and contamination (Saliki *et al.,* 1993).

2.15 Prophylaxis, control and eradication

Peste de Petit Ruminants disease has no treatment therefore the control of this disease is through the implementation of sanitary and veterinary prophylactic measures. The sanitary control measures of the disease in application include restriction of importation of sheep and goats from infected areas, quarantine, animal and vehicle movement controls within the infected areas, slaughter and disposal of carcasses, decontamination of contact fomites and affected premises in case of introduction (Berhe, 2006).

Veterinary prophylaxis entails immunization of the susceptible flocks. Vaccination of animals with RP attenuated virus has been practiced for a long time. The tissue culture rinderpest vaccine (TCRV) at a dose of $10^{2.5}$ $TCID_{50}$ protected goats against PPR for 12

months and the animals were not able to transmit the infection following challenge with PPR virus (Taylor, 1979a), although the antigen was detected in lachrymal swabs from vaccinated animals after challenge with virulent virus (Gibbs et al., 1979). However, it was reported that considerable residues of virulence were detected after 32, 42, even 65 serial passages in embryonic lamb kidney cells (Taylor, 1979a). This vaccine was successfully used to control PPR in some countries in West Africa (Bourdin, 1973) and is widely used in many African countries (Lefèvre and Diallo, 1990). It has been withheld from being used because of its interference with the Pan-African Rinderpest Campaign (PARC), since it is impossible to determine if sero-positive small ruminants have been vaccinated or naturally infected with RPV.

Currently PPRV homologous vaccine made from strain Nigeria PPRV 75/1 LK6 Vero 70 is in use to control of PPR (Diallo *et al.,* 1989a). Both of the above heterologous and homologous vaccines have two drawbacks: they require effective cold chain to conduct a vaccination campaign and the costs required in poor African countries are enormous and it is not possible to differentiate vaccination immunity from that of wild virus.

Efforts are being made to develop thermoresistant vaccine and PPR recombinant marker vaccines (Diallo, 2006; Berhe, 2006). The recombinant marker vaccines will make it possible to differentiate infected and vaccinated animals for sero-surveillance and sero-

monitoring purposes while thermoresistant vaccine will reduce the cost of vaccination by side-stepping the cold chain storage.

2.16 Socio-economic impacts of the disease

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). Small stock and mainly sheep and goats are the main farm animals owned by the poor in most developing countries. Goats, and sheep "considered as mobile banks", are reared as sources of not only milk and meat for family consumption, but also of income that can easily be mobilized for paying household expenditures, particularly in lean times. In addition to this economic role, sheep and goats have significant socio cultural roles. They are used as gifts or emblems for traditional rituals and religious purposes (FAO, 2009b. Elsawalhy *et al*. (2010) posit that goats and sheep provide an individual household with a high social status and also serve as the much envied symbol of wealth and respect amongst pastoral communities. Small ruminants are also an important means for rebuilding herds after environmental and political shocks thus, are a major component of pastoral coping mechanism

(Elsawalhy *et al*., 2010). By inflicting high losses to the small livestock, PPR is considered as a disease of major economic impact on the livelihoods and food security of the poor and marginalized segments of society as reported by the World Animal Health Organization (OIE) (Khalafalla *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). A lot of the PPR economic studies have been carried out in Indian subcontinent among the South Asian Association for Regional Cooperation (SAARC) member countries. Annual economic losses due to PPR in India were estimated at US$ 39 million (Chauhan *et al.,* 2009).Whereas in another report direct economic losses due to PPR in India were pegged at US$ 3.6 million if overall mortality was rated at 5%, but this figure rises to approximately US$ 13 million if mortality rates are set at 29% for goats and 17% for sheep (Singh *et al.,* 2009). Thombare and Sinha (2009) estimated the direct financial loss due to mortality occasioned by PPR in Pune District of Maharashtra state in India to be in tune of Indian Rupees (INR) 1,261,500 (US$ 21,254) for 83 affected farms studied. The disease-wise analysis of average losses for the 15-year period (1991-2005) revealed that PPR accounted for maximum (34.5%) of the total disease losses in India (Singh and Prasad, 2008). Economic losses due to PPR outbreak in herd of 1392 goats distributed in Dimla Thana of Nilfamari district in Bangladesh was estimated at US$ 14,520.49 (Islam *et al.,* 2011). While in Pakistan PPR causes economic losses of Pakistan Rupees (Pak Rs.) 20.5 billion (US$ 205 million) annually (PARC, 2007). In a meeting held in Kathmandu in 2011 to strategize PPR control, South Asian Association for Regional Cooperation (SAARC) member countries acknowledged that economic losses due to PPR within the member countries were in tune of US$180 million and that control programs would cost US$ 40 million over a period of 5 years with gross benefit to cost ratio of 4.83 (SAARC, 2011). Elsewhere in the Asian subcontinent the estimated

loss to the Iranian farmers due to mortality of sheep and goats affected by PPR was reported to be US$ 1.5 million (Bazarghani *et al.,* 2006).

In Africa, a few studies have provided a preview of what the economic impacts of PPR outbreaks portend. The earliest studies were reported in Nigeria where Hamdy *et al.,* (1976) had evaluated the annual losses induced by PPR to be in tune of US$ 1.5 million. Subsequent studies reported in Africa focused on the cost effectiveness of PPR treatment and control. Opasina and Putt (1985) evaluated three outbreaks in South west Nigeria and estimated an outbreak in every five years that would result in annual losses between Nigerian Naira (NGN) 0.27 to NGN 1.83 (US$ 0.30 to US$ 2.03) per animal in successful treatment. In neighboring Niger two economic analysis studies on PPR prevention were reported where the first study by Van Den Ende *et al*., (1988) concluded that vaccination of small ruminants was favorable to local herders and local economy. A sequel to this study was reported by Stem (1993) whose finding indicated that vaccination against PPR in Niger was highly beneficial, with an anticipated net present value (NPV) return in five years of US$ 24 million following an investment of US$ two million. Similar analysis conducted in Cameroon established that yearly vaccination against PPR in sheep and goats would improve small ruminants production and increase profits two to threefold for the farmers (Awa *et al.,* 2000).

CHAPTER 3: COMMUNITY APPRAISAL OF *PESTE DES PETITS RUMINANTS* AND VALIDATION OF THE COMMUNITY KNOWLEDGE IN TURKANA COUNTY

3.1 Introduction

The small ruminants (sheep and goats) are a very important livelihood asset for the Turkana pastoral community of Kenya. Sheep and goats are considered as mobile banks and are easily liquidated into cash in short notice to satisfy household requirements, with minimal consultation (Imana, 2008). General constraints facing small ruminants production in Turkana include poor husbandary, social conflict, lack of pasture and water; and livestock diseases. However a major constraint to production of small ruminants in Turkana and other pastoral areas of Kenya has been the recent entry of *Peste des petit ruminants* (PPR) disease into Kenya (ProMed-mail, 2007).

Pastoral communities in Kenya have had very rare encounters with *Peste des petit ruminants* disease prior to known outbreaks of 2006, thus indigenous knowledge on the disease is not as developed as in other small stock diseases. In Turkana community, PPR disease is much associated with descriptives and naming of rinderpest (Ohta, 1984; Kihu *et al,* 2012a). However, with increased occurrence of the classic cases of PPR disease in the Turkana herds, the Turkana herders in 2006 adapted the name *Lomoo* for the PPR disease (Bett *et al,* 2009). This study is a community appraisal of *Peste des petits ruminants* (*Lomoo)* disease in Turkana and validation of this community's knowledge

using scientific and laboratory analyses of cases and samples collected from appraised PPR cases by the community.

3.2 Material and methods

3.2.1 Study area

The study was carried out in Loima, Oropoi, Kakuma, Lokichoggio, Kaaling and Kibish administrative divisions of Turkana County (Figure 3.1). The county is located in the extreme north west of Kenya and is characterized by arid and semi-arid lands covered with grass and sparse thorny shrubs (Schilling *et al.,* 2012). The central eastern and southern part of the county consists of low-lying vast plains, with isolated rocky mountainous and hilly ranges surrounded by several seasonal rivers. The mountainous ranges are to the West, bordering Uganda and to the North, bordering Sudan; elevations which vary from 1800 – 2100 meters above sea level comprise the main grazing lands. The mountains are the sources of numerous seasonal streams, which feed into the Turkwell and Kerio rivers draining into Lake Turkana (Aemun, 2006). The county's woody vegetation is found on areas with the escarpments and mountains, and along the Turkwell and Kerio rivers and numerous seasonal water-courses (Amuyunzu, 1991). The rainfall pattern and its distribution are unreliable and erratic over the years. The long rains usually fall between April to June, and short rains in October – December with an annual rainfall approximately ranging between 100 mm to 500 mm. Temperatures range from a low of 24 ºC to a high of 38 ºC with a mean of 30 º C (ALRMP, 2009).

Turkana County has a human population of approximately 849,277 and an area of 77,000 km^2 with a small stock population of 3,517,151 sheep and 5,994,861 goats (KNBS 2010). Approximately 70% of the population in Turkana are nomadic or semi-nomadic pastoralists (Imana, 2008) deriving their livelihood from extensive livestock production (Figure 3.1).

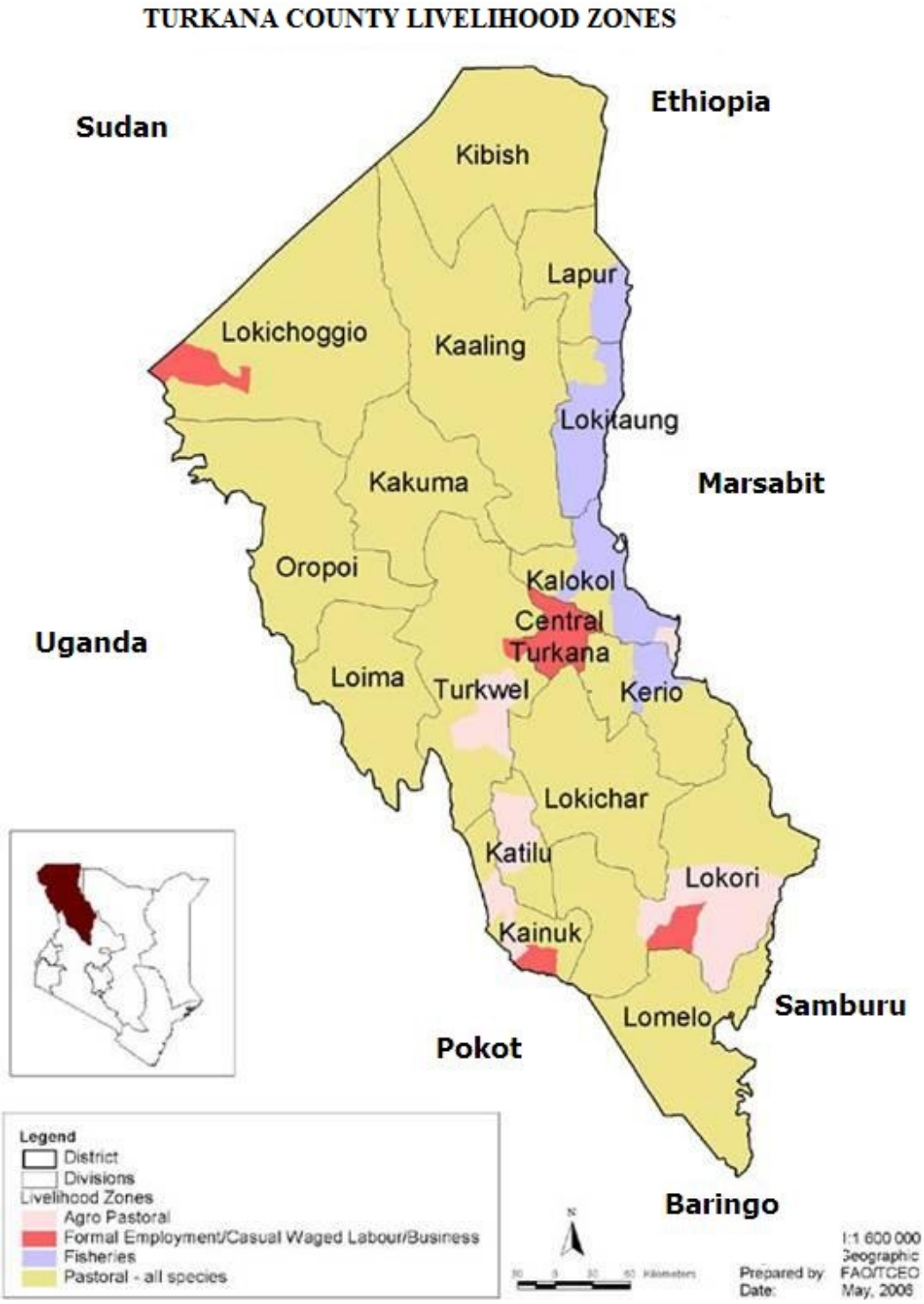


Figure 3.1: Map of Turkana district with administrative divisions adapted from FAO (2006)

3.2.2 Sampling unit and selection of study sites

The sampling unit was an *Adakar*. An *Adakar* entails a cluster of often-related Turkana households that pursue similar socio-economic activities such as search for pasture, water and security, under a trusted leader (Bett *et al.,* 2009). The study sites were selected purposively following a key informant's (government staff, local leaders and elders) interview conducted in a preliminary visit to the area. Informants from local *Adakars* provided information on *Adakars* that had high densities of small stock which became target for interview. However the general area for selection of study sites was heavily influenced by two other studies (Serum sample collection and Risk assessment survey) requiring probability sampling and ran concurrently with this study as shown in Figure 3.2. Therefore participatory epidemiology interviews on PPR case observations as well laboratory sample collections were done in the *Adakars* where serum samples were collected or where risk assessment questionnaire was administered.

3.2.3 Data collection

3.2.3.1 Key informants interviews

Before any interview was conducted, the research team introduced the purpose of the research to the local chiefs and elders and requested consent to work with the local community. To ensure the community did not bias their answers in favor of researcher's disease of choice (*Peste des petits ruminants)*, the introduction-brief to key informants and villagers was made in such way that it did not mention PPR disease but on generality of small stock diseases.

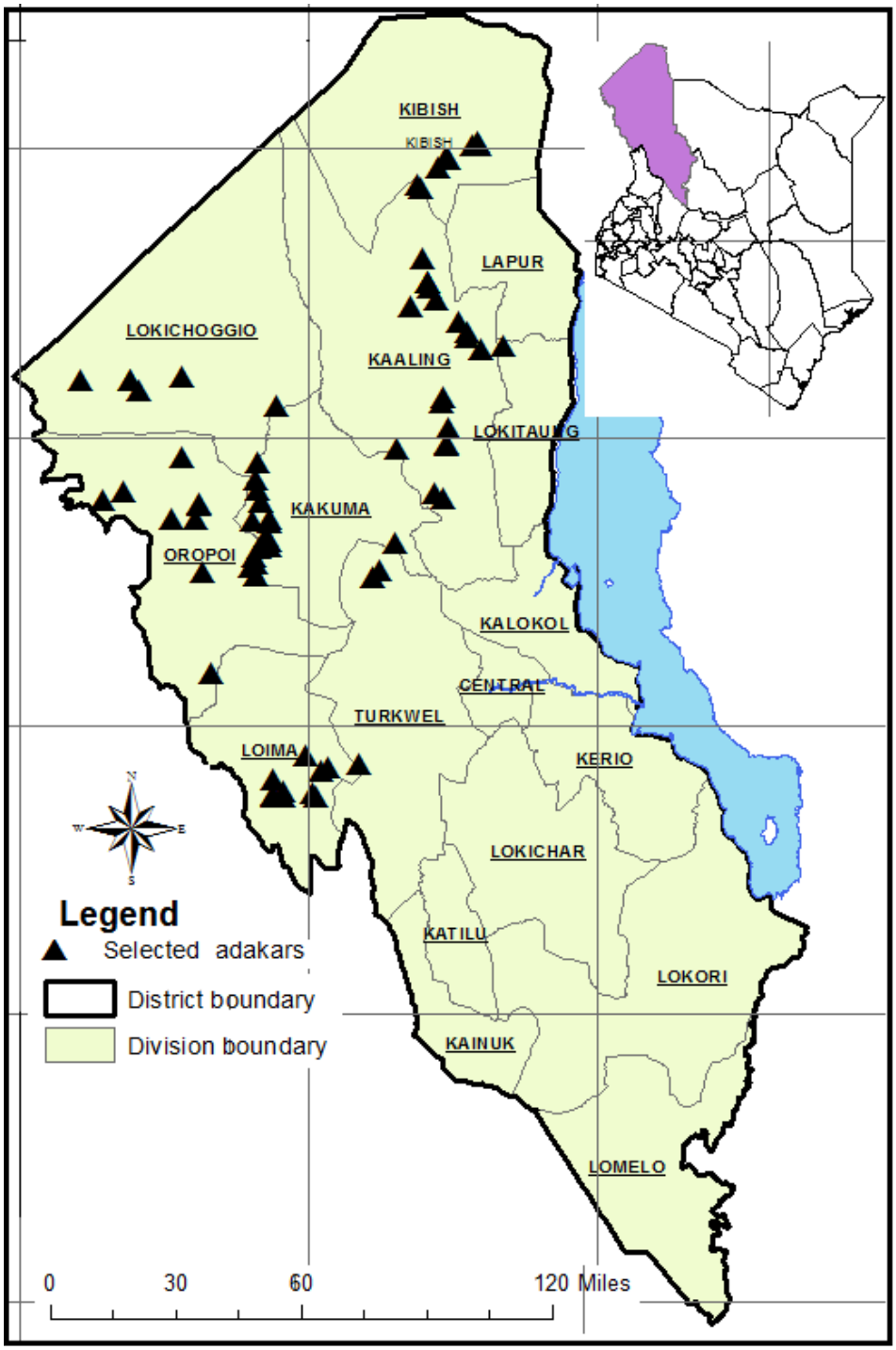


Figure 3.2: Map of Turkana sampling sites (Kihu et al., 2012b)

Key informants interviews included local administrative and opinion leaders, staff from Ministry of livestock development and local non-governmental development actors. The data collected from these interviews, which formed the main background of the study, covered information on sheep and goat diseases prevalent in the study area and the control activities that were ongoing. Since Turkana county experiences erratic insecurity incidences, the key informants' interviews were carried out in areas that were safe.

3.2.3.2 Focus group discussion

Focused group discussion were carried out with a group of 5 to 15 respondents. The discussions were led by a member of research team who was assisted by a local Turkana translator who helped translate between English and Turkana language. The focus group discussions employed various participatory epidemiology tools to elicit participation and triangulate various responses from the respondents. The key participatory epidemiology (PE) tools used were semi structured interview, simple ranking, matrix scoring and proportional piling, as described by Jost *et al*. (2010) and Catley *et al.,* (2012). A total of 60 focused group discussions were held in all the six administrative divisions

3.2.3.2.1 Semi Structured Interviews

Semi-structured interview (SSI) was the main tool used throughout the focused group discussions for collection and probing of general data on small stock diseases, epidemiological clinical and post mortem characteristic(s) of each disease as risk factors associated with the diseases. The SSI was guided by checklist of open-ended questions that provided for structured discussion with the respondents as described by Jost *et al*.

(2007) and Ameri *et al*. (2009). The checklist was pre-tested and adjusted prior to the initiation of the study. However, disease data collected through SSI was triangulated using other data collection methods that were also used to collect specific disease information. The SSI generated a list of small stock diseases including some general characteristics of the diseases of sheep and goats from the perception of Turkana herders.

3.2.3.2.2 Simple Ranking

Simple ranking method was used to arrange sheep and goat diseases listed by respondents in order of importance, based on mortality and morbidity of the diseases as described by Ameri *et al.,* (2009). The respondents were asked to give a list of diseases acquired by each of the small ruminant species over a 1-year period preceding the time of the interview. The respondents discussed among themselves and arranged the listed diseases according to the criteria. The respondents often used the local disease names to identify diseases. When the participants provided syndromes rather than specific names of diseases, probing using open-ended questions was done to characterize the syndrome whilst trying not to guide respondents. The names of the diseases and descriptions given by the pastoralists were later triangulated using matrix scoring and names validated at the local veterinary office. Two ranked lists of disease and their characterization were generated, one for sheep diseases and another for goat diseases. The simple ranking method was repeated in 20 *Adakars*.

3.2.3.2.3 Matrix scoring

To better understand the local characterization of the sheep and goat diseases and allocate meaning to local small stock disease names, a disease matrix scoring was used as described by Catley, (2005). The method allowed the herders to show their local perception on association between clinical, postmortem signs, risk factors; and the mentioned diseases. The top six ranked diseases of sheep and goats were used on x-axis of disease matrix scoring. Clinical, post mortem signs and risk factors generated for each disease during the semi-structured interviews by respondents were used in the y-axis of the disease matrix.

3.2.3.2.3.1 Matrix scoring for disease clinical characterization

Nine clinical signs were used in the sheep disease matrix of which five clinical signs (diarrhea, depression, ocular discharge, nasal discharge and death) formed the clinical case definition of PPR while each of the other four clinical signs was a common disease sign to any of the other diseases in the matrix. The process was repeated for goat diseases where six top ranked diseases of goats were used on the x-axis of disease matrix. Ten clinical signs and post mortem lesions were used on y-axis of goat disease matrix. Of the ten clinical signs used in goat disease matrix, seven were a case definition of PPR (depression, diarrhea, emaciation, coughing, nasal discharge, ocular discharge and death) while three other clinical signs were common to three other diseases in the matrix. Thirty counters were placed on each clinical sign and the respondents were asked to divide the counters to each of the disease on y-axis based on how much that

clinical sign is related with that disease. The counters for each clinical sign were counted and recorded in a matrix. The method was repeated in 12 *Adakars.*

3.2.3.2.3.2 Matrix scoring for disease risk factor characteristics

Two risk factor matrices; one for sheep diseases and another for goat diseases; were developed by the respondents. In a similar pattern, six top ranked diseases of sheep or goat were placed on x-axis and the risk factors on the y-axis of disease risk factor matrices. Thirty counters were placed on each risk factor and the respondents were asked to divide the counters to each of the disease on y-axis based on how much that risk factor is related with that disease. The counters for each risk factor were counted and recorded as a matrix. The method was repeated in 10 *Adakars*

3.2.3.2.4 Proportional piling

The Turkana perception of PPR impact on sheep and goat herds was demonstrated by relative morbidity and mortalities generated using proportional piling method as described by Ameri *et al.,* (2009) and Catley, (2005).

3.2.3.2.4.1 Proportional piling for age structure

Respondents provided names of four age groups in goats and sheep. Age categories in sheep were, new born lambs (*imethek*) up to age of two months, lambs in the age group between three and five months (*nanyang*), sheep in middle age group (*amethek nakale*) six months to 24 months; and adult sheep (*amethek naapolon*) with age above 24 months. Age categories in goats were new born kids up to two months age (*Ikale*); kids in the age

between three and five months (*namenaoei*); goats in middle age group (*akale*) with age between six months to 24 months, and adults (*Akine*) with ages more than 24 months. Hundred counters representing a herd of sheep in an *Adakar* were laid on ground. The respondents were asked to divide the counters into the four age groups of sheep to determine proportional age structure in sheep herds. The process was repeated for the goats. The proportional piling for age structure was carried out for both sheep and goats in 27 *Adakars*

3.2.3.2.4.2 Proportional piling for morbidity and mortality

The incidence and mortality of PPR (*Lomoo*), were determined relative to other five sheep and goat diseases and a category of "other diseases" that were considered important by the Turkana. These other five diseases of sheep were sheep pox (*Etune*), anaplasmosis (*Lonyang*), bottle jaw (*Loborbolio*), anthrax (*Lookot*) and foot and mouth (*Lojaa*). Similarly for the goats, five diseases evaluated alongside PPR (*Lomoo*) were contagious caprine pleuro-pneumonia (*Loukoi),* thin goat syndrome (*Loutogonyen*), diarrhea (*Naosin*), bottle jaw (*Loborbolio*) and pasteurellosis (*emany*). Using a pile of 100 counters to depict an age group in a herd, the respondents were asked to divide the counters into two piles to show the pattern of sick sheep and healthy sheep during the last one year (2010). The pile of counters representing sick sheep was then sub-divided by the respondents to show the pattern of sheep having each of the diseases listed above plus the "other diseases" category. Each pile of counters representing a disease category was then further sub-divided to show the pattern of sheep dying and surviving for each disease

category. When each of the diseases was scored, the counters were counted and recorded. This process was repeated for each age group in each species (sheep and goats) and all contents of piles generated were counted and recorded. The proportional piling for mortality and morbidity was carried out for both sheep and goats in 44 *Adakars.*

3.2.4 Validation of Turkana indigenous diagnostic knowledge

3.2.4.1 Clinical case observations

Following all sessions of participatory epidemiology and focused group discussions on disease characteristics, the respondents were asked to demonstrate by showing the study team small stock that had the various ailments discussed. General observation of the herds was done by walking amongst grazing goats. Sheep and goats that had one or more of clinical signs designated for clinical case definition of PPR were fully observed and examined. In one particular village herd (*Adakar*) named Lotakaa GPS position (N 03° 38 390; S 034° 50 987) in Kakuma, Turkana County, the respondents demonstrated several suspect cases of PPR *(Lomoo)*. The history narrated by the respondents showed that there were 21 kids, 24 adult goats, one sheep that had died from the suspect PPR infection in the previous two weeks. The suspect PPR cases that died had their carcasses examined for post mortem. The exercise helped to cross-check the information provided during the interview and scoring exercises.

3.2.4.2 Laboratory analysis of samples from suspected PPR cases

3.2.4.2.1 Laboratory sample collection

Laboratory samples were collected from carcasses of sheep and goats suspected to have died of PPR; those that died while on observation by the study team at the village herds. Postmortem samples collected from the carcasses were lung tissues, mesenteric and mediastinal lymph nodes and small intestines; each of them was divided into two sets which were placed in separate sample bottles. The first set of samples were prepared for histopathological examination and had formalin added to each tissue after collection. The second set of samples was stored at - 20°C in a mobile refrigerator during transportation to the laboratory. All collected samples were transported to University of Nairobi, Department of Veterinary Pathology, Microbiology and Parasitology laboratory where the frozen samples were stored at -30°C for two years before the observation were carried out.

3.2.4.2.2 Laboratory sample analyses

3.2.4.2.2.1 Histopathology slides preparation and staining

Tissues for histopathology were collected and preserved in 10% formalin. The formalin fixed tissues were prepared into slides for histological examination through the standard paraffin process that dehydrates, clears and infiltrates the tissue with paraffin wax. Embedding was done to allow orientation of the specimen in a block so that it could be easily handled, sectioned and stored. Sectioning was done using a microtome to produce very thin sections that were placed on a microscope slide ready for staining. The samples were stained with Hematoxylin and eosin stain **(**Gill, n.d.).

This protocol entailed dehydration of well trimmed tissue in three stations of alcohol with each station having alcohol strength of 80%, 90% and 96% and time spent in each station was four hours, two hours and two hours, respectively. The tissues were further dehydrated using Isopropanol in three stations and time spent in each station was one and half hours. The volume of alcohol used for dehydration was 50 times that of the tissue being dehydrated. Clearing of the tissues was done in three stations with the first station using Amyl Acetate for one hour. In the subsequent two stations, xylene was used for two hours in station two and two and half hours in station three. Finally the tissues were infiltrated with molten wax at 60°C in two stations, each station spending three hours. The volume of wax used for infiltration was 30 times the volume of the tissues infiltrated. Each of the waxed tissue was placed in a mould, molted wax poured on it and allowed to settle and solidify into a block. The block was left to cool slowly to form a surface skin and then immersed in cold water to cool it rapidly. Each individual block was placed on a wood block and clamped to attach.

The wax-wooden block was clamped onto a microtome and sections of 5 micrometer thickness cut. The cut sections were softened by floating in water bath at 40°C. Using a suitably sized microscope slide that was coated with an adhesive, the smoothened section was lifted out of the water bath, excess water drained, and dried on a hot plate at 60^{o}C. The drying firmly stuck the tissues to the slides so that they could be stained using Hematoxylin and Eosin staining.

The staining process involved dewaxing in xylene for three minutes followed by rehydration by placing in absolute alcohol for three minutes, in 80% alcohol for two minutes and in 50% alcohol for two minutes. The slides were washed in running tap water for one minute and then put in Harris hematoxylin for five to seven minutes. The slides were washed in running tap water for 30 seconds. Excess dye was washed-out in 1% acid alcohol by continuous agitation for 15 seconds. The slides were washed in running tap water for 30 seconds then dipped three times in ammonia water solution until tissue obtained blue color; they were again washed in running tap water for 30 seconds. The slides were counter stained with eosin for three to five minutes then washed in running tap water for 30 seconds. The tissues were dehydrated by keeping in increasing concentration of alcohol for two to three minutes in 50%, 70%, 95% and absolute alcohol respectively. Finally the tissues were cleared in xylene and mounted in Distyrene Plasticizer Xylene (DPX) and allowed to dry. Tissue slides were then examined under light microscope.

3.2.4.2.2.2 Ribonucleic acid (RNA) extraction and Real time reverse transcriptase polymerase chain reaction (RT PCR) analysis

A duplicate of field collected formalin fixed samples used for histology and frozen field samples were prepared for RNA extraction using two distinct protocols. The extraction of RNA from formalin fixed sample was based on RNeasy® FFPE kit protocol but was modified because the tissues were not paraffin embedded. The extraction of RNA from

the frozen samples was done based on RNeasy mini kit protocol. Both Kits were supplied by QIAGEN®.

3.2.4.2.2.2.1 RNeasy® FFPE kit protocol for extraction of PPR Virus RNA from formalin fixed tissue

The biosafety sample preparation cabin, working benches and all equipments used in preparation of the formalin fixed tissues were decontaminated before preparation of each sample using 1% Sodium hypochrolite, 70% alchohol and RNase Away® a decontamination reagent for RNase.

A total of nine formalin fixed tissues were used for the experiment and each of the samples was placed into RNase-free Petri dish container and 20 µm thickness section made using a new, sterile, and disposable scalpel blade made for a single reaction, discarding top three sections to avoid using the oxidized and or contaminated surface of tissue block. Each cut section was washed separately with 0.9%sterile sodium chloride (Liehr & Manvelyan 2009) and then DNase-RNase free water to remove excess unbound formalin (Putchler and Meloan, 1985).

The washed formalin fixed tissues were disrupted and homogenized in a clean DNase-RNase free mortar and pestle and tissue-minced very well. Each homogenized tissue was then put in a DNase-RNase free centrifuge tubes and labeled. The process was repeated until all samples were processed. The next section of the protocol was carried out as outlined by RNeasy® FFPE Protocol supplied by QIAGEN® (2011). Reagents in the

protocol such as buffer PKD, buffer RBC, buffer RPE are proprietary and confidential to QIAGEN® thus their contents and constitution is not in public domain.

In each centrifuge tube containing the homogenized tissue sample, 240 µl of buffer PKD was added and mixed by vortexing. In each sample tube, 10 µl of proteinase K was added and mixed gently by pipetting up and down. The sample tubes were incubated at 56°C for 15 min, then at 80°C for 15 minutes. The incubation in buffer PKD at this stage is for digestion of tissue by proteinase K and is also critical for reversal of formaldehyde cross-links modifying nucleic acids. After the incubation, the samples were transferred into a new 2 ml micro-centrifuge tube and incubated on ice for three minutes, followed by centrifuging for 15 minutes at 20,000 x g (13,500 rpm). The supernatant was transferred to a new micro-centrifuge tube, taking care not to disturb the pellet which contained insoluble tissue debris, including cross-linked DNA. In the new sample tube containing the supernatant, 25 µl DNase Booster Buffer and 10 µl DNase I stock solution were added and mixed by inverting the tube; it was then centrifuged briefly to collect residual liquid from the sides of the tube. These sample tubes were incubated at room temperature for 15 minutes; thereafter 500 µl Buffer RBC was added to adjust binding conditions, and the lysate mixed thoroughly. A volume of 1200 µl absolute ethanol (100%) was added to the sample, and mixed well by pipetting. Seven hundred (700) µl of the sample was transferred, including any precipitate that may have formed, to an RNeasy MinElute spin column placed in a 2 ml collection tube and centrifuged for 15seconds at ≥8000 x g (≥10,000 rpm). The flow-through was discarded and the collection tube reused.

The centrifugation process was repeated for the remaining sample until the entire sample was passed through the RNeasy MinElute spin column. A volume of 500 µl Buffer RPE was added to the RNeasy MinElute spin column and centrifuged for 15 seconds at ≥8000 x g (≥10,000 rpm) discarding the flow-through. Again 500 µl Buffer RPE was added to the RNeasy MinElute spin column centrifuged for two minutes at ≥8000 x g (≥10,000 rpm) to wash the spin column membrane. The RNeasy MinElute spin column was placed in a new 2 ml collection tube, with the lid open, and centrifuged at full speed for five minutes, to dry the spin column membrane. The RNeasy MinElute spin column was placed in a new 1.5 ml collection tube and 14–30 µl RNase-free water added directly to the spin column membrane and then centrifuged for 1 minute at full speed to elute the RNA. The eluted RNA in the collection tubes was stored at -20°C awaiting RT PCR processing.

3.2.4.2.2.2.2 RNeasy® mini kit protocol for PPR Virus RNA isolation from fresh frozen tissues

The biosafety sample preparation cabin, working benches and all equipments were decontaminated before preparation of each sample using 1% Sodium hypochrolite, 70% alchohol and RNase Away® a decontamination reagent for RNase.

A total of 18 frozen sample tissues was sliced using a sterile scalpel blade to a required size of 30 mg on a frozen sterile Petri dish. The sliced frozen sample tissue was placed in a clean DNase-RNase free mortar and pestle which was maintained cool in liquid

nitrogen. Some liquid nitrogen was poured into the mortar. The tissues were disrupted by grinding it by mortar and pestle until it was a very course powder, and further ground into fine powder. The powder was collected into a pre-cooled tube, labeled and stored at -20°C. The process was repeated until all samples were processed.

In each sample bottle containing the fine ground tissue, 600ul RLT buffer was added and homogenization done by passing the lysate five times through 20 gauge needle, fitted on an RNase free syringe. The lysate was centrifuged for three minutes at full speed and supernatant carefully pipetted into the tube that was labeled. In these tubes, containing the sample supernatant, 600 µl of 70% ethanol was added and mixed gently by pipette for 10 to 15 times. RNeasy mini columns (pink column) were identified and 700 µl of each sample applied to a column placed in a two ml collection tube, then centrifuged for 30 seconds at 10,000 g, followed by discarding the collection tube and keeping the sample column. The remaining 500 µl of each sample supernatant was transferred to a respective sample column placed in a new collection tube and centrifuged for 30 seconds at 10,000 g, followed by discarding the collection tube and keeping the column. The RW1 Buffer (700 µl) was added to each sample column fitted with a collection tube, then centrifuged for 30 seconds at 10,000 g, followed by discarding the collection tube keeping the column. For each sample column, 700 µl of the RPE Buffer (containing ethanol) was added; a collector tube was filled and then centrifuged for 30 seconds at 10,000g, followed by discarding the collection tube and keeping the column. Then 500 µl of the RPE Buffer was added to each sample column, fitted with collection tube,

centrifuged for 30 seconds at 10,000 g, followed by discarding the collection tube and keeping the sample column.

The sample columns were transferred to new collection tubes, centrifuged for one minute at 13000 g to dry the sample column. The dry sample column was transferred to a new labeled 1.5 ml DNase RNase free microtube and, 50 µl of DNase-RNase-free water applied directly onto the RNeasy silica-gel membrane. The tube was closed, gently incubated for one minute at room temperature, then centrifuged for one minute at 8 000 g to elute RNA into the microtube. The column was discarded and the microtube containing eluted RNA closed and stored at – 20°C awaiting real-time reverse transcription–polymerase chain reaction (qRT-PCR) to be carried out.

3.2.4.2.2.2.3 Real time reverse transcription polymerase chain reaction assay

A total of 21 RNA samples extracted from 21 samples from three kids suspected to have died of PPR were analyzed using a specific real-time reverse transcription polymerase chain reaction assay using a TaqVet™ Peste des Petits Ruminants Virus kit which had a set of primers/probes designed from the N-terminus of the Ngene in the Mix PPRV based on a protocol detailed in a report by *Laboratoire Service International* (LSI) (2011), supplier of the kit. The forward primer matched positions 483 > 508 in the (5-AGAGTTCAATATGTTRTTAGCCTCCAT-3); the TaqMan® probe positions 551 > 576 (FAM-5-CACCGGAYACKGCAGCTGACTCAGAA-3- MGB) and the reverse primer was located at positions 603 < 624 (5-TTCCCCARTCACTCTYCTTTGT-3) (Batten *et al.,* 2011). The kit also had a set of nucleotides for internal positive control

(IPC) forward and reverse primers and IPC probe TaqMan® labeled in VIC-MGB. The external positive control (EPC) in the kit was ready to use solution constituted of extracted PPRV Nucleic Acid labeled EPC PPRV. DNase/RNase free water, extracted as a sample, was used as a negative control sample labeled (NCS). A negative result for the target "PPRV" indicated that no contamination occurred during sample extraction and amplification process. PPRV mix was used as a negative control labeled (NC). A negative result indicated that no contamination occurred during the preparation..

A MicroAmp® Optical 96-well reaction plate with an adhesive cover was prepared noting that the qRT-PCR was to be performed in each one of the wells in the plate. The mix PPRV was briefly vortexed and centrifuged; then 20 µl of the Mix PPRV was added into each well of the Optical 96-well plate used for the assay. In each well of the plate used for the assay, 5 µl of the sample (RNA) or NCS or NC or EPC PPRV was added. The plate was covered with an adhesive cover and put in Abiprism® 7500 (Applied Biosystems) thermocycler. The amplification reaction was first programmed by creating a plate sheet for the qRT-PCR run. Passive reference was created as « ROX » and detectors for PPRV and IPC were created as FAM with no quencher and VIC with no quencher respectively. The qRT-PCR run had three steps.

- Step 1 : 45°C – 10 min – Repeat : 1
- Step 2 : 95°C – 10 min – Repeat : 1
- Step 3 : 95°C – 15 s ; 60°C – 1 min – Repeats : 45

The qRT PCR results were interpreted taking into account the threshold level which was the point of reading and analysis in real-time PCR. Threshold was set on the basis of the baseline variability, baseline being PCR cycles in which a reporter fluorescent signal was accumulating but was thus beneath the limits of detection of the instrument. A signal that was detected above the threshold was considered a real signal that can be used to define the threshold cycle (Ct) for a sample; thus Ct was the fractional PCR cycle number at which the reporter fluorescence (Rn) was greater than the threshold. This outcome was represented graphically with ΔRn values plotted against the cycle number; ΔRn being the increment of fluorescent signal at each point in time. Ct was thus plotted as the point of intersection between the sample amplification curve and the threshold line. Two graphs were produced: a log plot and a linear plot. Samples with Ct less than 45 with PPRV detector and IPC detector Ct of less than 45 were considered to have presence of the virus genome of *Peste des petits ruminants* while samples with Ct greater than 45 though IPC detector Ct was less than 45 were considered to be absent of the virus genome of *Peste des petits ruminants*. Samples that had PPRV detector Ct of greater than 45 and IPC detector Ct of equal or greater than 45were considered not validated and this could be due to presence of RT-PCR inhibitors. Internal positive control (IPC) detector whose Ct values were less than 45 validated negative results detected by PPRV detector (Table 3.1).

Table 3.1: Interpretation of the qRT-PCR results of TaqVet™ Peste des Petits Ruminants Virus kit (LSI, 2011)

Interpretation	PPRV « Detector »	IPC « Detector »
Detected PPRV	Ct < 45	Ct < 45
Non detected PPRV	Ct ≥ 45	Ct ≤ 45
Not validated	Ct ≥ 45	Ct ≥ 45

3.2.4 Data management and statistical analysis

Both qualitative and semi-quantitative data were collected in the study. The qualitative data were presented without being subjected to formal statistical analyses. The quantitative data was entered and cleaned in Microsoft Excel (Microsoft Corp., Redmond, WA). It was then exported to SPSS (2008) statistical software version 17.0 (IBM Corp., Armonk, NY) for analysis using non-parametric statistical tests. Analyses were undertaken using descriptive statistical procedures and data summarized using medians to determine central tendency while dispersion was expressed by 10^{th} and 90^{th} percentiles estimation. Analysis of disease ranks entailed conversion of the ranks into scores and summarized using Freidman test. To determine the significance of association between clinical signs and diseases; risk factors and diseases the Friedman test was used (Jost *et al*., 2010)

3.3 Results

3.3.1 Perception of Turkana herders on ranking and characterization of PPR

The respondents described and ranked the diseases of sheep and goats in their Turkana language. The frequency of sheep and goat diseases reported varied slightly between village herds (*Adakars)* though a trend emerged where PPR *(Lomoo*) featured prominently among the six top ranked diseases in all areas. Table 3.2 shows the six most highly ranked diseases of sheep from the first to the sixth out of 17 diseases reported. The diseases ranked first was Anthrax while PPR was ranked at position six among sheep diseases. The respondents'association of the sheep diseases with the ranks was statistically significant at $p<0.001$.

Table 3.2: Top six ranked diseases of sheep

Disease rank	**Disease in English**	***Disease equivalent in Turkana language***
1	Anthrax	*Lookot*
2	Anaplasmosis	*Lonyang*
3	Sheep pox	*Etune*
4	Bottle Jaw due to blood sucking Worm; Heamonchosis	*Loborbolio*
5	Foot and Mouth Disease	*Lojaa*
6	*Peste des petits ruminants*	*Lomoo,*

N=10, Freidman test statistic $\chi 2$ (16)=102.825; $p<0.001$

The top ranked six diseases of goats, out of 27 disease reported are sown in Table 3.3 with the most highly ranked disease being Contagious caprine pleuro pneumonia (CCPP) and sixth ranked disease being Acute helminthiasis (possibly heamonchosis). *Peste des petits ruminants* was ranked at position two after CCPP among goat diseases. The respondents' association of the goat diseases and with ranks was statistically significant ($p<0.001$).

Table 3.3: Top six ranked diseases of goats

Disease rank	**Disease in English**	***Disease equivalent in Turkana language***
1	Contagious caprine pleuro pneumonia	*Loukoi,*
2	*Peste des petits ruminants*	*Lomoo*
3	Pasteurellosis	*Emany*
4	Heavy worms load, constipation and soiling of anal area	*Naosin*
5	Thin sickly goat syndrome	*Loutogonyen,*
6	Bottle Jaw due to blood sucking Worm; Hemonchosis	*Loborboloi*

N=11, Freidman test statistic $\chi 2$ (26)=150.572; $p<0.001$

Local characterization of PPR by the Turkana herders showed that they were knowledgeable at recognizing clinical signs and risk factors associated with the PPR disease in both sheep and goats. The respondents consistently and correctly associated, through matrix scoring, the signs of clinical case definition of PPR as being indicative of PPR. Further, the respondents correctly and strongly associated appropriate disease signs with other diseases scored alongside PPR in the matrix scoring. Table 3.4 shows that the respondents scored highly for diarrhea, depression, ocular discharge, nasal discharge and death to associate these symptoms with PPR in sheep. The respondents' association of the symptoms with PPR was statistically significant ($p<0.001$).

Table 3.4: Sheep diseases characterization by clinical and postmortem signs using matrix scoring

	Median scores (10th and 90th percentiles in parenthesis)					
Clinical signs	Acute helminthiasis *(Loborbolio)*	FMD (*Lojaa)*	Anaplasmosis (*Loyang)*	Anthrax *(Lookot)*	PPR *(Loomo)*	Sheep pox *(Etune)*
Diarrhoea***	0 (0, 15.6)	0 (0,11.2)	0 (0,0)	0 (0,7)	16 (0,30)	0 (0,8)
Depresion***	1 (0, 6.6)	7 (.4, 11.2)	2 (0,7.8)	0 (0,21.2)	12 (4.4,22)	0 (0,7.6)
Pox lesions***	0 (0,0)	0 (0,0)	0 (0,0)	0 (0,0)	0 (0,0)	30 (30,30)
Bottle jaw ***	30 (19.6, 30)	0 (0,5.6)	0 (0,0)	0 (0,0)	0 (0,4.8)	0 (0,0)
Alopecia *	0 (0,0)	0 (0,0)	0 (0,0)	0 (0,0)	0 (0,7.2)	0 (0,30)
Yellow meat***	0 (0,0)	0 (0,0)	30 (6,30)	0 (0,0)	0 (0,0)	0 (0,0)
death***	3 (0,14.4)	2 (0, 3.8)	3 (0.2, 9)	6 (1.2,13.2)	10 (2.2,19.4)	3 (0.2,6.8)
ocular discharge***	0 (0,8.8)	14 (0,24.8)	0 (0, 4)	0 (0,3.2)	13 (5.2,30)	0 (0,6.8)
Nasal discharge***	0 (0, 13.8)	7 (0,23.6)	0 (0,7)	0 (0,4.8)	16 (5.4,30)	0 (0,4.0)

*P<0.05;**P<0.01; ***P<0.001. No asterix means P>0.05

The respondents' characterization of the diseases of goats included scoring post mortem Lissions. The respondents again scored highly for emaciation, depression, nasal discharge, ocular discharge, and diarrhea as signs strongly associated with PPR in goats. A further clinical sign associated with PPR in goats was cough, though it was not highly scored (Table 3.5). Just like for the sheep diseases, the respondents correctly and strongly associated appropriate disease signs with other diseases scored alongside PPR in goats in the matrix scoring. However, the respondents' association of the clinical signs with PPR was statistically significant ($p<0.001$).

Table 3.5: Goat diseases characterization by clinical and postmortem signs using matrix scoring

	Median scores (10th and 90th percentiles in parenthesis)					
Clinical/PM signs	Acute helminthiasis *(Loborbolio)*	Pasteurellosis (*Emany)*	CCPP *(Loukoi)*	Sick goat syndrome *(Loutogonyen)*	Helmimthiasis (*Naosin)*	PPR (*Lomoo)*
Emaciation ***	2.5 (0,6)	0 (0,5.4)	0.5 (0,4.7)	4 (0.6,9.1)	7.5 (3.3,12.4)	12 (7.9,19.1)
Bottlejaw***	27 (3.9,30)	0 (0,0)	0 (0,0)	0 (0,8.8)	0 (0,18.2)	0 (0,13.7)
Alopecia	0 (0,21)	0 (0,0)	0 (0,0)	0 (0,0)	0 (0,21)	0 (0,21)
Coughing***	0 (0,0)	0 (0,8.4)	24 (18.3,30)	0 (0,2.1)	0 (0,0)	4.5 (0,10.4)
Enlarged liver***	0 (0,6.3)	30 (13.1,30)	0 (0,16)	0 (0,8.4)	0 (0,0)	0 (0.0)
Death***	1 (0,3)	2.5 (.3,8.8)	7.5 (3.6,13.5)	3(1,6)	3 (.3,6)	12 (5.6,16)
Diarrhoea***	1 (0,10.9)	0 (0,1.4)	0 (0,0)	4 (0,9.7)	14.5 (5, 23.1)	10 (0,19.7)
Ocular Discharge***	0 (0,4.8)	0 (0,0)	0 (0,0)	10.5 (.9,23.1)	0 (0,6.4)	14.5 (1.8,24)
Nasal discharge***	2 (0,10.7)	0 (0,0)	0 (0,6.1)	6.5 (0,11.8)	1.5 (0,17.5)	16.5 (5,27.3)
Depression***	0 (0,7.1)	0 (0,8.1)	1 (0,8)	1 (0,7.4)	4 (0,9.4)	17.5 (11.6,30

*P<0.05;**P<0.01; ***P<0.001. No asterix means P>0.05; PM=post mortem

3.3.2 Characterization of sheep and goat diseases by risk factors.

Risk factors identified and scored by respondents for PPR in sheep were: long rain season, dry season, all age groups except new born lambs, migration, herd mixing and raids (Table 3.6). The respondent scored highly for migration, dry season, raids, herd mixing and all age groups except the newborn lambs. Wet season was scored marginally. The respondents associated seasonal risk factors with PPR in sheep; long rain season, dry season showed a statistical significance ($p<0.01$). The association of herd migration as a risk factor for PPR in sheep was statistically significant ($p<0.001$). Other risk factors that had statistical significance ($p<0.01$) for association as risk factors of PPR in sheep were raids and herd mixing. The respondents association of adult age group, middle age group and older kids age group as risk factors for PPR in sheep did not have any statistical significance. However association of newborn kids with the diseases as a risk factor was statistically significant ($p<0.001$).

Table 3.6: Sheep disease characterization by risk factors and seasonality using matrix scoring

	Median scores (10th and 90th percentiles in parenthesis)					
Risk Factors	Acute helminthiasis *(Loborbolio)*	Anthrax *(Lookot)*	PPR (*Lomoo)*	Sheep pox *(Etune)*	Anplasmosis *(Lonyang)*	FMD *(Lojaa)*
Wet Season *(Akiporo)***	3.5 (0,14.1)	11.5 (.8,28.7)	0.5 (0,12.7)	2 (0,8.6)	4 (0,9)	3 (0,14.1)
Start of dry season *(Ait)*	1 (0,14.6)	1.5 (0,28.3)	0 (0,12)	2 (0,14.2)	5 (0,17.9)	0.5 (0,27.7)
Dry season *(Akamu)***	9.5 (0,14)	0 (0,9)	8 (0.4,20.6)	6 (.2,9)	1 (0,6.8)	0.5 (0,8)
Pre- wet season *(Akicheres)*	0 (0,11.4)	4.5 (0,15.7)	0 (0,28.4)	6.5 (0,11.6)	2 (0,9.8)	3 (0,15.6)
Adult *(Namanjong Amethek)*	6.5 (2.1,13.8)	4.5 (.1, 12.6)	5.5 (0,15.7)	5.5 (2.1,7.8)	4 (.2,7.9)	2.5 (.1,8.5)
Young adults (*Nakale)*	4 (1.1,12.6)	4 (.2,7)	5.5 (0,14.4)	7.5 (3.1,12)	3.5 (.1,9.6)	3 (.1,8.8)
Older lambs *(Nanyang)*	2 (0,6.8)	3.5 (0.13.6)	3.5 (0,8.7)	5 (0,11.8)	4 (0,14.3)	2 (0,18.6)
New born lambs (*Imethek*)***	0 (0,3.9)	0 (0,10.4)	0 (0,20)	11 (.9,28.8)	1.5 (0,11.5)	0 (0,17.9)
Fleas	0 (0,30)	0 (0,3.6)	0 (0,27.8)	0 (0,27.8)	0 (0,16.7)	0 (0,3.6)
migration***	1 (0,7.8)	3.5 (0,12.6)	10 (2.1,14.8)	6 (2.1,17.1)	2 (0,4)	4.5 (3.1,14.9)
Herd mixing**	2 (0,4.9)	3.5 (0,8.6)	6.5 (0,15.4)	9.5 (3.2,16.7)	1 (0,5)	3 (0,11.4)
Raids**	2 (.1,5.9)	2.5 (0,5.9)	7 (.3,15.5)	6.5 (3.1,16.7)	1 (0,7.9)	7 (.3,16.6)
Ticks	0 (0,5.9	0 (0,21.3)	0 (0,2.79)	0 (0,5)	0 (0,22)	0 (0,27.3)
Toxic plants***	0 (0,2.7)	17.5 (0,30)	0 (0,4)	0 (0,10.5)	2 (0,13.7)	0 (0,3.8)
Mountain Pasture & water	5 (.1,18.5)	3.5 (0,12.9)	0 (0,8)	4.5 (0,7.9)	4.5 (0,7.9)	5.5 (1.2, 12.4)

*P<0.05;**P<0.01; ***P<0.001. No asterix means P>0.05

Table 3.7 shows that the respondents considered seasonality (all seasons), all age groups, livestock movements which entails herd migration, herd mixing and raids as well as environmental factors, such as toxic plants and mountainous pastures, as risk factors of PPR in goats. Dry season was the most highly scored risk factor followed by raids, young adults age group, herd mixing, start of dry season, mountainous pasture, adult age group, young adults age group, older kids age group, migration, long rainy season, short rainy season and toxic plants, in that order. Except for short rainy season all the other seasons were shown to have statistically significant (wet season and dry season $p<0.001$ while start of dry season $p<0.01$) association with the goat diseases. The association of goat age groups with diseases was statistically significant ($p<0.001$ for adults, young adults and older kids while $p<0.01$ for new born kids). The association of herd mixing, migration and raids with goat diseases was statistically significant at $p<0.001$. The respondents association of environmental factors with diseases was statistically significant with toxic plant having a significance of $p<0.05$ while mountainous pasture and water had a significance of $p<0.001$.

Table 3.7: Goat diseases characterization by risk factors and seasonality using matrix scoring

	Median scores (10th and 90th percentiles in parenthesis)					
Risk factor	Acute helminthiasis *(Loborbolio)*	Helminthiasis *(Naosin)*	Sick goat syndrome *Loutogonyen*	CCPP (*Loukoi)*	Pasteurellosis *Emany*	PPR (*Lomoo)*
Wet season *(Akiporo)***	1 (0,7.7)	4 (.1,14.4)	1 (0,2)	12 (.7,15.8)	4.5 (.2,11.7)	6 (0,12.7)
Start of dry season (*Ait*)**	1.5 (0,7.8)	9 (3.2,13.9)	3 (0,9.7)	4 (2.1,14.4)	1.5 (0,8.6)	7 (0,15.7)
Dry season (*Akamu*)***	4 (0,6.9)	5.5 (3,12.5)	4 (1.1,10.7)	4 (0,10.8)	0 (0,2.8)	9.5 (6,15.9)
Start of wet season (*Akicheres*)	3 (0,17.8)	3 (.1,12.5)	2 (0,6)	11 (0,20)	3 (0,9.7)	5.5 (0,10.8)
Adult (*Akine Naapolok*)***	3.5 (.1,6.9)	4.5 (2.1,7)	3 (2.1,4.9)	8.5 (7,11.9)	3.5 (1,9.5)	6.5 (.5,8.9)
Young adult (*Naakalei*)***	2 (0,6.7)	4 (2,9.7)	4.5 (1.1,7.8)	8 (5,15.8)	2.5 (0,7.5)	7.5 (5,10.9)
Older kids (*Nanyang*)***	1.5 (0,4)	2.5 (0,6.9)	3.5 (0,5)	6 (0,15.6)	0 (0,2)	6.5 (0,15.7)
New born kids (*Ikale*)**	0 (0,3.7)	6 (0,14.8)	4.5 (0,18)	9.5 (0,20.4)	0 (0,6.6)	6.5 (0,28)
Fleas	0 (0,6.6)	0 (0,3.6)	1.5 (0,30)	0 (0,3.6)	0 (0,4.5)	0 (0,22.1)
Migration***	1 (0,2.9)	4 (2,13.6)	3.5 (2,14.9)	10 (.7, 17.8)	3.5 (0,7.7)	6 (.4,9)
Mixing***	0 (0,3)	3 (0,5)	3 (0,5.9)	16 (9.1,20.7)	.5 (0,5.9)	7 (5,11.7)
Raid***	0 (0,5.7)	3 (0,4.9)	2.5 (0,5.9)	13 (9,29)	0 (0,5.9)	7.5 (.5,12.7)
Ticks	0 (0,3.6)	0 (0,0)	0 (0,29)	0 (0,27)	0 (0,0)	0 (0,27.6)
Toxic plants*	0 (0,2.9)	8.5 (0,19.7)	1.5 (0,4.9)	0 (0,14.7)	8 (0,17.4)	4 (0,14.3)
Mountain pasture and water***	1 (0,7.7)	0 (0,9.6)	0 (0,2)	13 (6.2,28.8)	3 (0,12.7)	7 (0,12.9)

*P<0.05;**P<0.01; ***P<0.001. No asterix means P>0.05

3.3.3 Turkana perception of PPR incidence and mortality in sheep and goats

The estimated morbidity, mortality and case fatality rate of PPR by sheep and goat age groups are illustrated in Tables 3.8 and 3.9.

In sheep, the relative morbidity of PPR varied from 19% (9.87, 40) in young adults (*Nakale*) to 25% (11.4, 40.8) in adults (*Amethek Naapolon*). The relative mortality ranged between 16% (7.3, 37.1) in young adults (*Nakale*) and 20% (7.8, 34.9) in adults (*Amethek Naapolon*). Case fatality rate was highest in young adults (*Nakale*) at 84.2% (68,100).

Table 3.8: Relative morbidity, mortality and case fatality due to PPR in sheep of Turkana

	Median scores (10th and 90th percentiles in parenthesis)			
	New born Lambs (*Imethek*)	Older lambs (*Nanyang*)	Young Adult (*Nakale*)	Adult (*Amethek Naapolon*)
Estimated morbidity of PPR	20 (7, 38)	20 (10, 40.4)	19 (9.87, 40)	25 (11.4, 40.8)
Mortality of PPR	17 (5.4, 31)	17 (6.5, 34.9)	16 (7.3, 37.1)	20 (7.8, 34.9)
Case fatality rate due to PPR	80.8 (59.2,98.8)	80 (60,100)	84.2 (68,100)	83.3 (62,96)

The estimated relative morbidity due to PPR in goats ranged between 17.8% (8.5,32.1) in new born kids (*Ikale*) and 20.9% (10.4,36) in adults (*Akine*). Relative mortalities

varied from 14.2% (6.1,28.6) in newborn kids (< 2 months of age *Ikale*) to 17% (5.5, 25.5) in older kids (>2 but <6 months *namenaoei*). Case fatality was highest in young adults (>6 but <24 months *Akale*) at 84.5% (58.6,100).

Table 3.9: Relative incidence, mortality and case fatality rate due to PPR in goats of Turkana

	Median % scores (10th and 90th percentiles in parenthesis)			
	New born kids (*Ikale*)	Older kids (*Namenaoei*)	Young adult (A*kale*)	Adults (*Akine*)
Estimated morbidity of PPR	17.8 (8.5, 32.1)	20.5 (8.4, 31)	20 (9.5, 30)	20.9 (10.4, 36)
Mortality of PPR	14.2 (6.1, 28.6)	17 (5.5, 25.5)	16.3 (6.5, 26.5)	16.66 (7.5, 35)
Case fatality rate due to PPR	83 (62.9,100)	79.1 (57.2,98.4)	84.5 (58.6,100)	82.8 (57.3,100)

3.3.4 Validation of Turkana local knowledge and perception of PPR disease

3.3.4.1 Clinical signs manifestations

The clinical signs observed from the goats presented from the herd were mainly in kids of four to six months old. One adult goat was also observed presenting some of the signs. The clinical signs presented included: depression, diarrhea, emaciation, difficult breathing, elevated body temperatures of 41 °C, serous and muco-purulent nasal discharges, and encrusted peri-nasal areas. Serous and mucopurulent ocular discharges matting the eye lids at the inner eye canthus and the hair below the eye was also observed (Figures 3.3, 3.4 and 3.5). Death due to this infection was observed in three kids and they were all necropsied for examination.

Figure 3.3: Muco-purulent ocular nasal discharges (arrow) from goat 1

Figure 3.4: Depressed goat 2 and goat 3 in the background

Figure 3.5: Diarrhoea in goat 2

3.3.4.2 Pathological lesions

3.3.4.2.1 Gross pathological lesions

All necropsied goats showed pneumonic lesions in the lungs. Inflammation of the apical lung lobes revealed red hepatization and congestion (Figure 3.6). The small and large intestines were empty (Figure 3.7) and showed moderate to severe hemorrhagic enteritis characterized by hemorrhagic intestinal mucosa (Figure 3.8). Intestinal blood vessels were congested with blood (Figures 3.7 and 3.9). The mesenteric lymph nodes were enlarged and swollen (Figures 3.7 and 3.9).

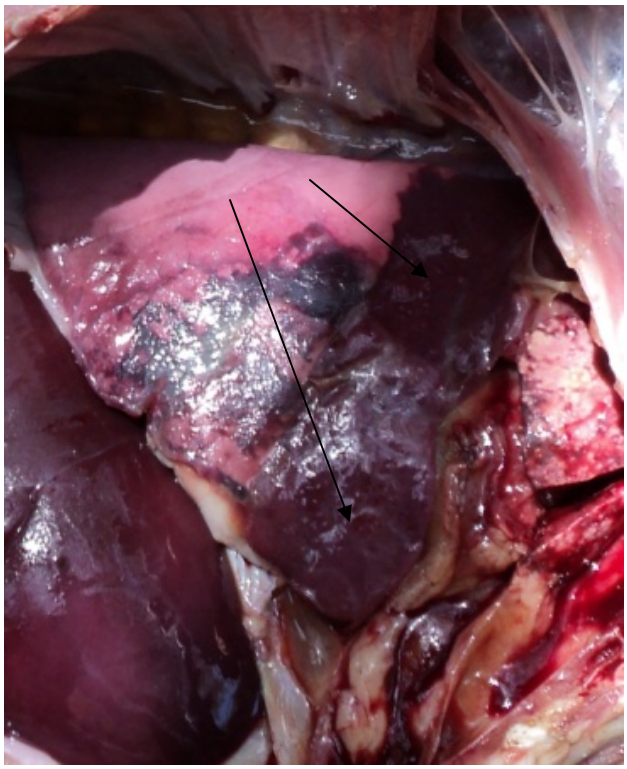


Figure 3.6: Hepatised apical lobe of goat 1

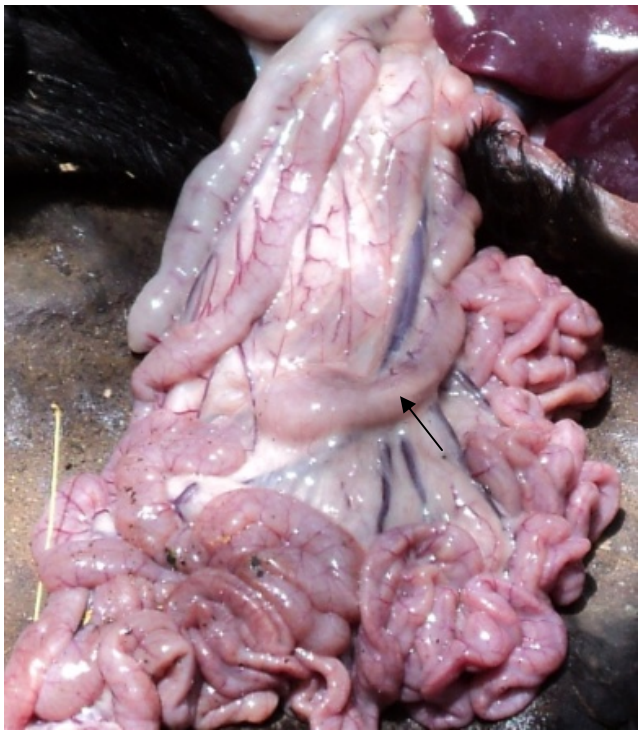


Figure 3.7: Swollen mesenteric lymph node (arrow) and hyperaemic intestines of goat 3

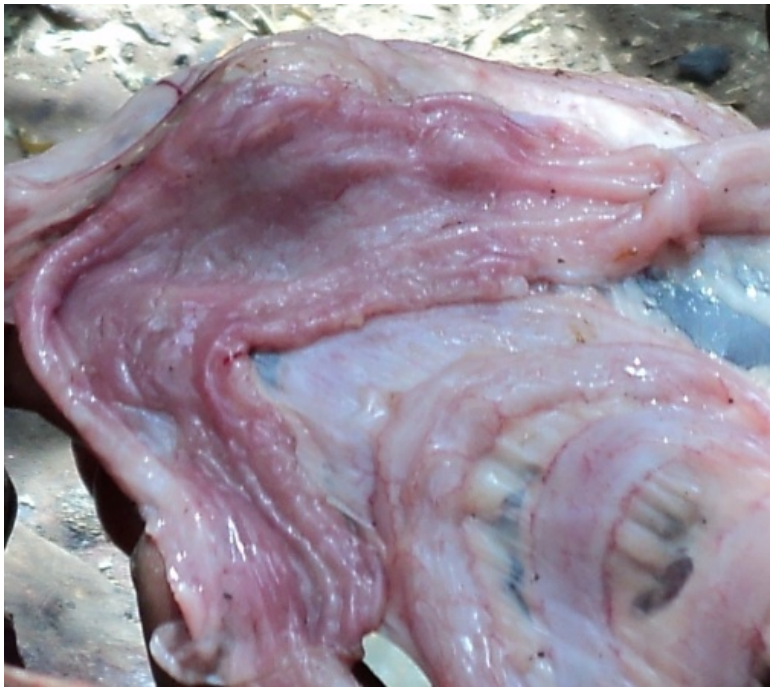


Figure 3.8: Hyperaemic intestinal mucosa (goat 3).

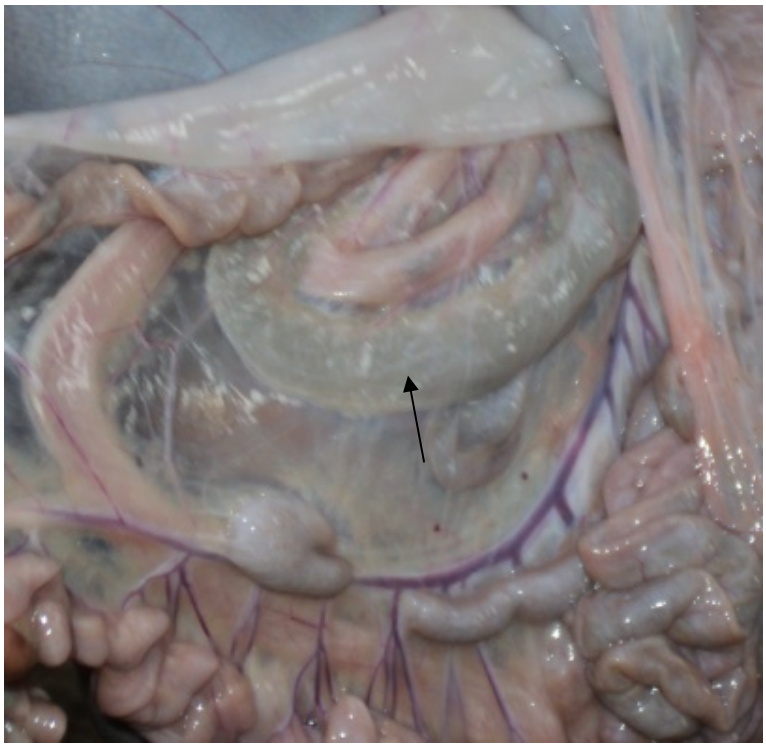


Figure 3.9: Swollen mesenteric lymph node (arrow) and congested mesenteric veins

3.3.4.2.2 Histopathological lesions

The lesions present in the lungs were characteristic of broncho-interstitial pneumonia (Figure 3.10). The lesions observed were alveolar collapse, thickening of the alveolar and interlobular septae. The bronchiolar epithelium was thickened and accumulation of exudates within the bronchioles. The blood vessels in the lungs were congested (Figure 3.11).

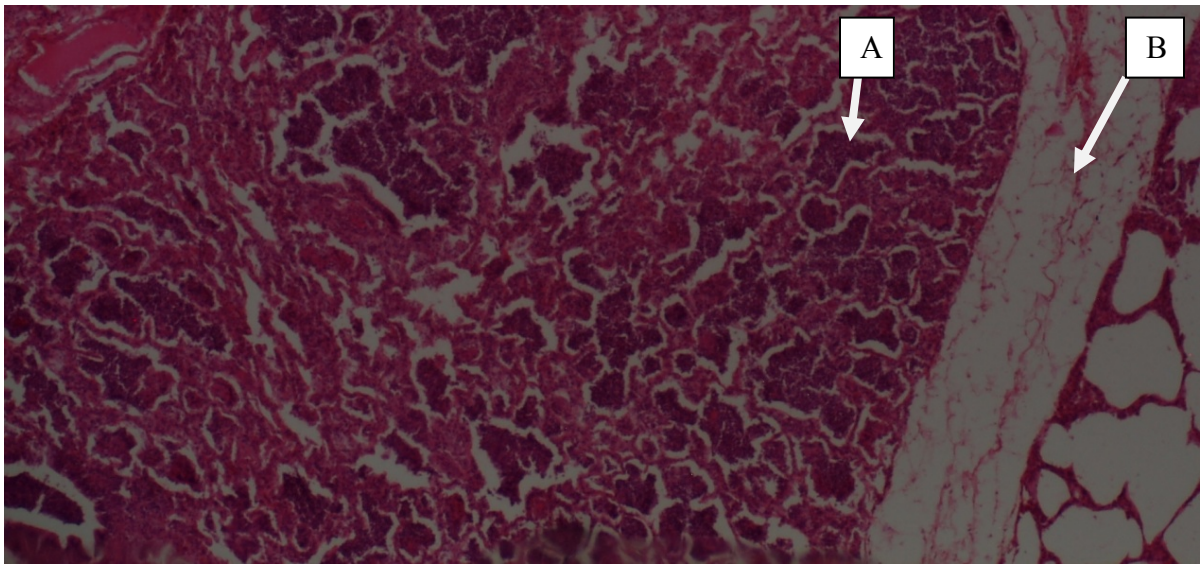


Figure 3.10: Lung of goat 1 showing collapsed alveoli (arrow A); thickening of the alveolar, interlobular septae (arrow B) X40

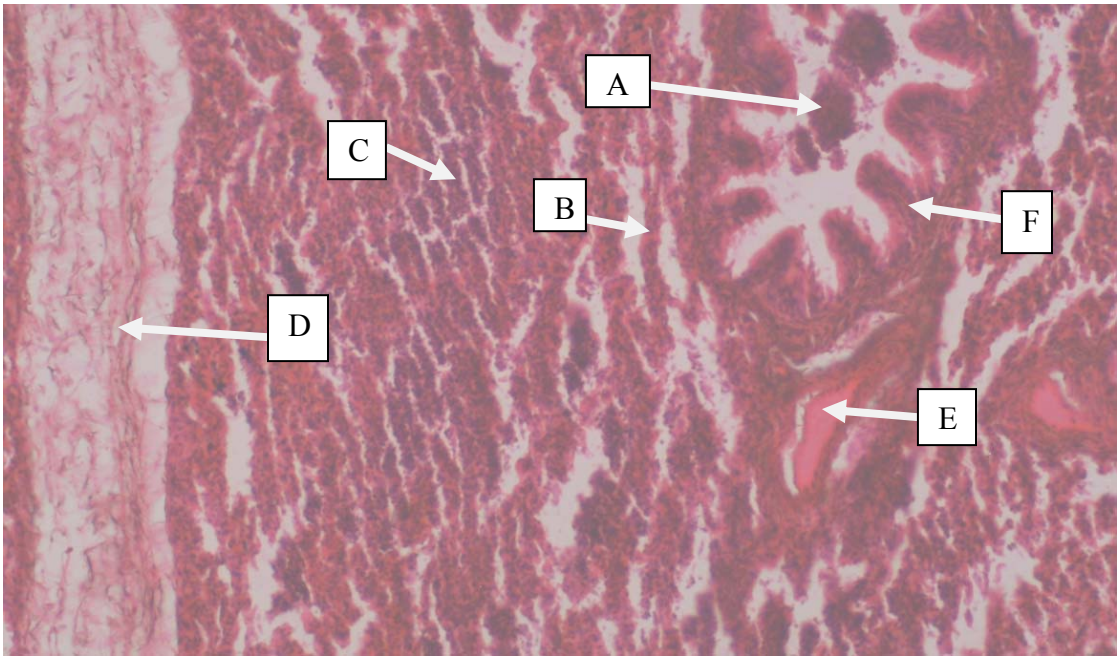


Figure 3.11: Lung of goat 1 showing collapsed alveoli (arrow C); thickening of the alveolar (arrow B), interlobular septae (arrow D) and bronchiolar epithelium (arrow F); accumulation of exudates within the bronchioles (arrow A) and congested blood vessels (arrow E) X100

Infiltration of the alveoli by mononuclear cells was evident in addition to multinucleated syncitia in the alveolar epithelium (Figure 3.12). Lesions in the large intestines revealed congestion of blood vessels, edema (Figure 3.13) and infiltration of lamina propria by inflammatory cells (Figure 3.14)

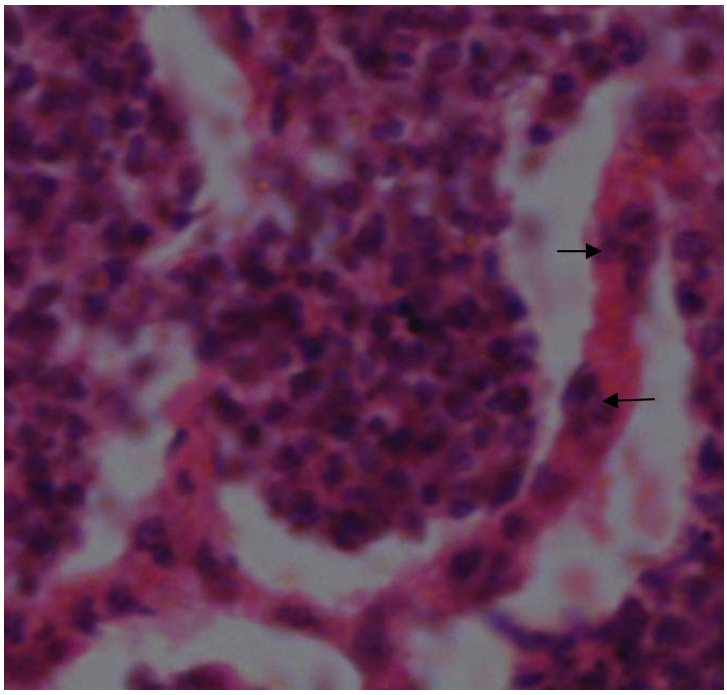


Figure 3.12: Lung of goat 1 showing infiltration with mononuclear cell in the alveoli and multinucleated syncitia (arrows) in the alveolar epithelium X400

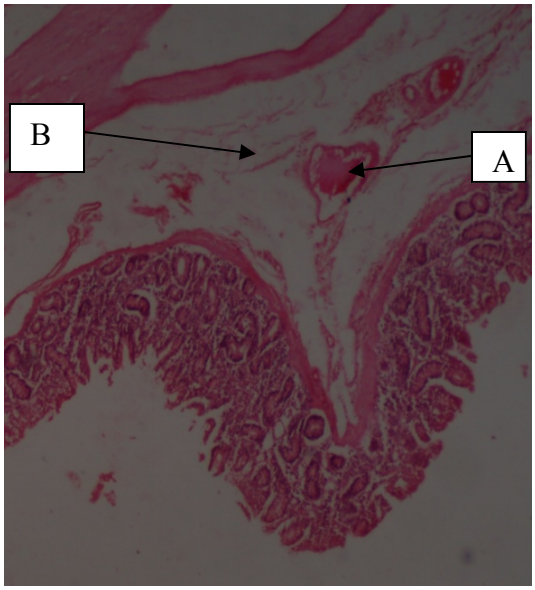


Figure 3.13: Large intestine of goat 3 showing oedema beneath sub mucosa (arrow B) and congestion of blood vessels (arrow A) X40

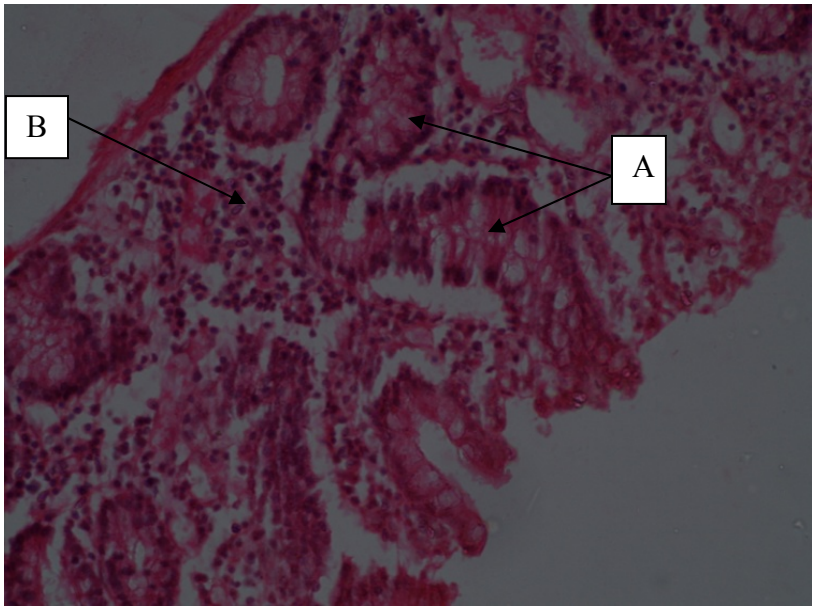


Figure 3.14: Large intestine of goat 3 showing infiltration of lamina propria (Arrow B), proliferation of goblet cells (arrow A) and accumulation of cell debris within the intestinal crypts X400

Two key lesions were observed in the lymph nodes: depletion of the lymphocytes in both mesenteric and mediastinal lymph nodes and congestion of blood vessels in the mesenteric lymph nodes (Figure 3.15).

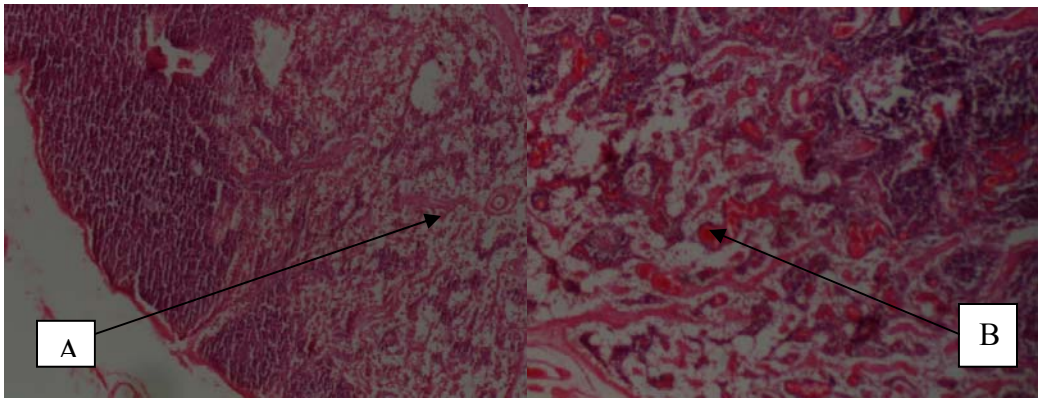


Figure 3.15: Mediastinal lymph node (arrow A) of goat 1 showing depletion of lymphocytes; Mesenteric lymph node (arrow B) of goat 3 showing congestion, haemorrhages and depletion of lymphocytes

3.3.5 Real time reverse transcriptase polymerase chain reaction analysis results

2.3.5.1 Formalin fixed samples

Formalin fixed samples were also analysed for presence of PPR virus RNA. A total of nine samples were analyzed (Table 3.10). Figures 3.16 and 3.17 show log plot and linear graph of results of PCR analysis of formalin fixed samples. Five out of nine samples gave positive results; these being mesenteric lymph node of goat one, lung of goat two, large intestine of goat two, mesenteric lymph node of goat three and mediastinal lymph node.

Table 3.10: qRT-PCR analysis results for the formalin fixed tissues from goats suspected of PPR

	Sample type	Formalin fixed tissues sample number	threshold cycle (Ct) positive samples	Results
Goat 1	Lung	PE37		negative
	Large intestine	PE 38		negative
	Mesenteric lymph node	PE 35	40.36	positive
Goat 2	Lung	PE36; F36	 40.10	negative positive
	Large intestine	PE 34 F34	44.25	positive negative
Goat 3	Mediastinal lymph node	PE 40	42.68	positive
	Mesenteric lymph node	PE 39	36.49	positive

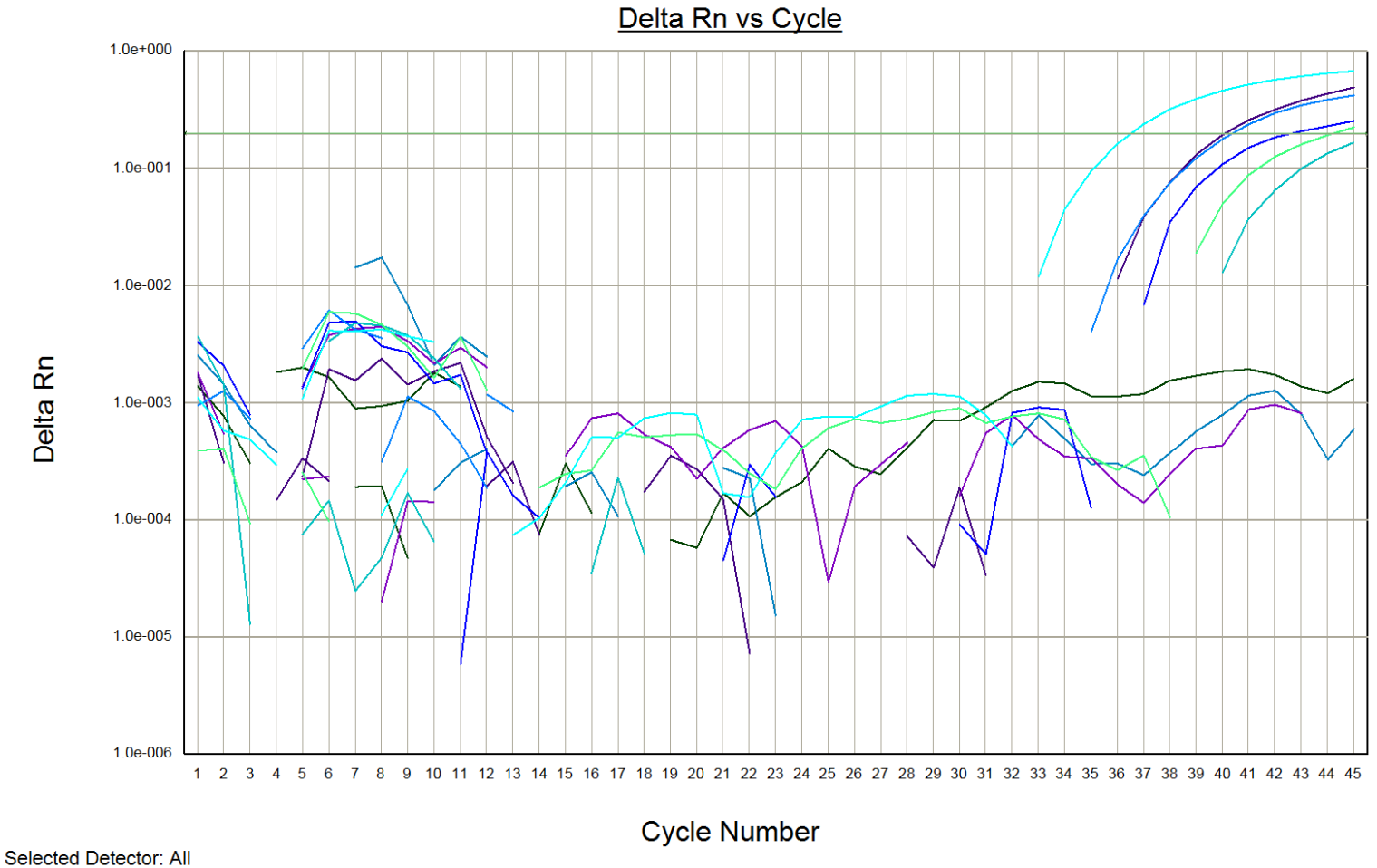


Figure 3.16: Log plot of Delta Rn against Cycle number for the formalin fixed tissue.

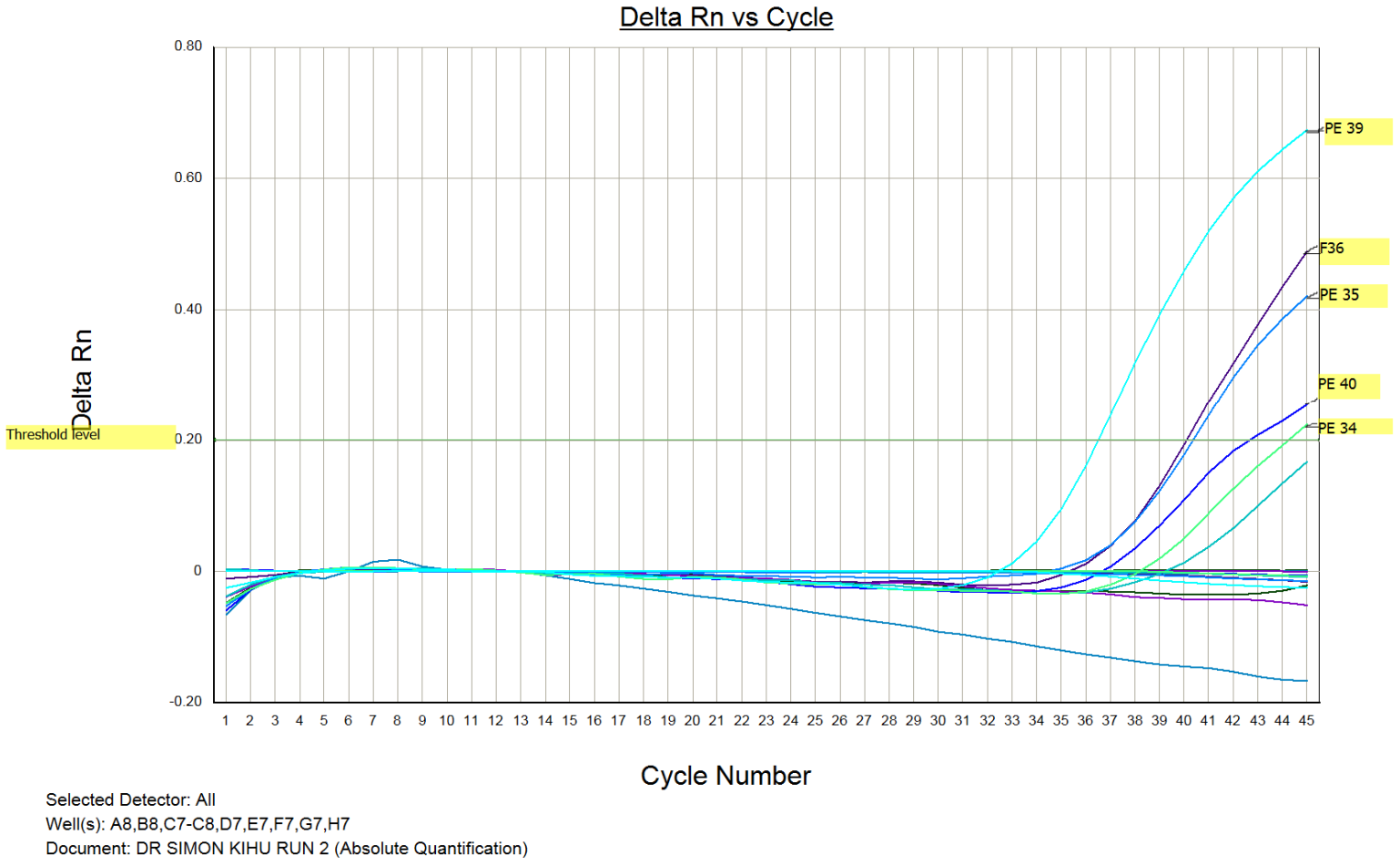


Figure 3.17: Linear plot of Delta Rn against Cycle number for the formalin fixed tissue

2.3.5.2 Frozen tissue

A total of 18 frozen samples were analysed for PPR virus RNA (Table 3.11). The samples were in duplicates comprising inocula prepared from original tissues for an experimental study and the original frozen samples. All original samples tested gave positive reaction for the presence of PPR virus RNA except inocula prepared from mediastinal lymph node from goat three (labeled I9).

The analysis of Ct values for the original frozen tissue samples shows that the cycle thresholds were in range of 20.46 to 29.17 symbolizing presence of large amounts PPR virus RNA in the samples (Table 3.11 and Figures 3.18 and 3.19). Inoculum samples of the same tissues had cycle threshold of ranges between 28.37 and 40.56, symbolizing reduction in PPR virus RNA.

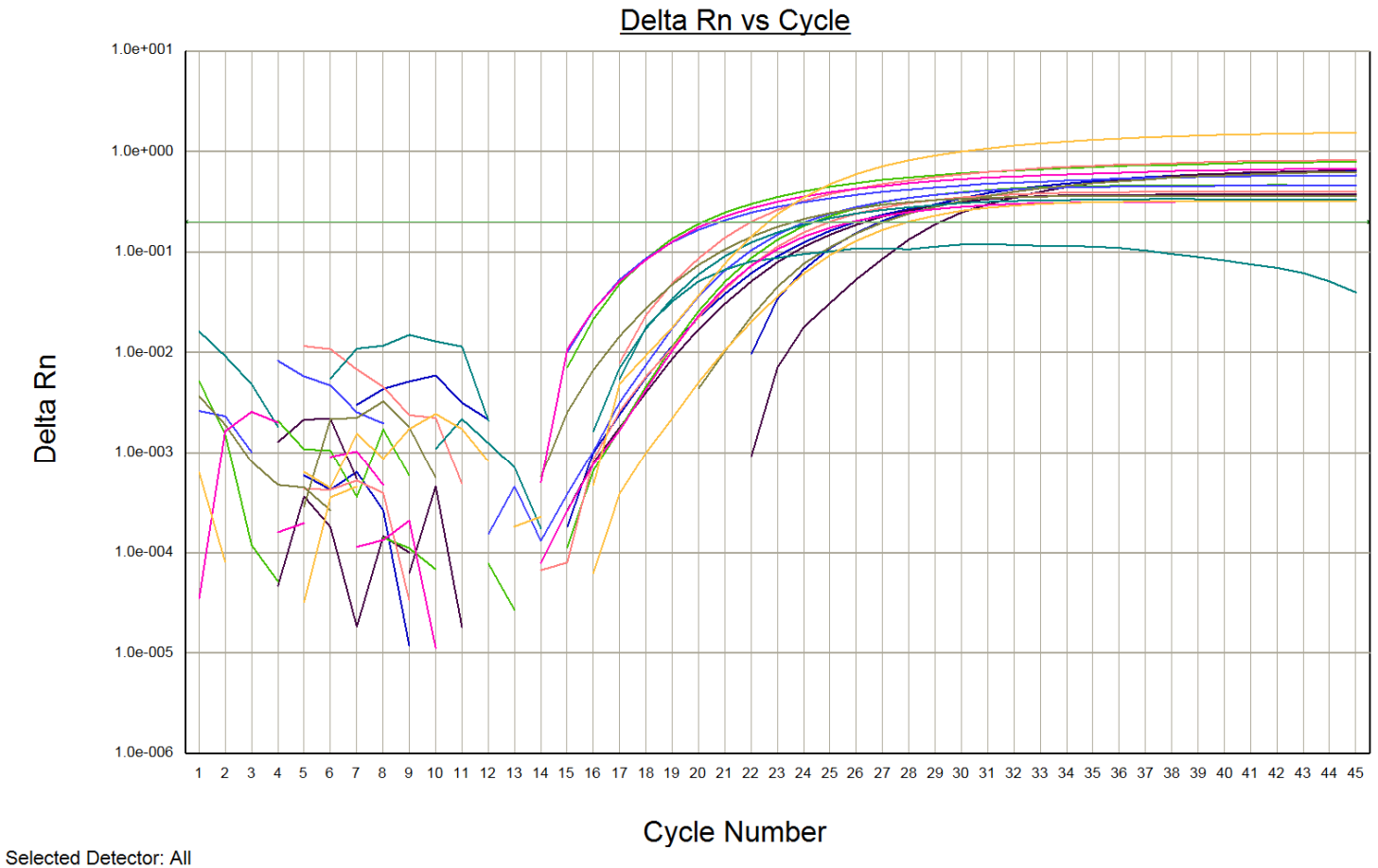


Figure 3.18: Log plot of Delta Rn against Cycle number for the frozen tissue samples

Table 3.11: qRT-PCR analysis results for the frozen tissues

Goat sampled	Sample tissue type	Frozen tissues samples			
		Tissues number	Inocula from frozen tissues	Ct positive samples	Results
Goat 1	Mediastinal lymph Node	1 F		29.17	positive
	Lung	2 F		26.89	positive
			I 2	37.95	positive
	Large intestine		I 3	38.65	positive
	Mesenteric lymph node	4 F		27.04	positive
			I4		negative
Goat 2	Mediastinal lymph Node	5		21.99	positive
			I 5	32.28	positive
	Mesenteric lymph node	6		20.14	positive
			I 6	29.29	positive
	Lung	7 F			negative
			I 7	28.37	positive
	Large intestine	8 F		20.46	positive
			I 8	31.28	positive
Goat 3	Mediastinal lymph Node		I9		negative
	Lung	10 F		22.6	positive
			I 10	40.56	positive
	Mesenteric lymph node	11 F		20.82	positive

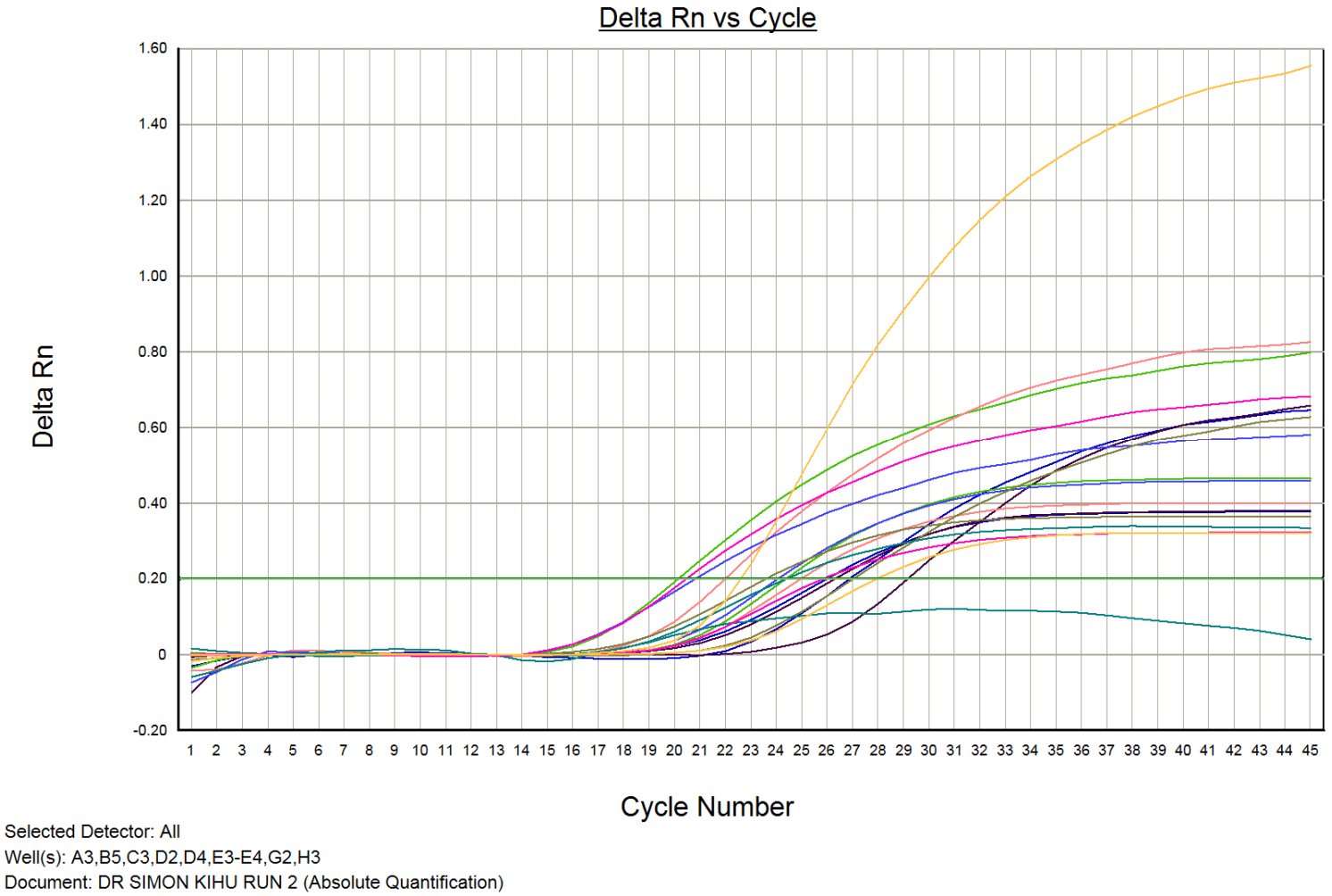


Figure 3.19: Linear plot of Delta Rn against Cycle number for the frozen tissue samples

3.4. Discussion

The Turkana community listed and described various diseases that affected their sheep and goats. A key disease that featured prominently in their description of diseases was PPR (*Lomoo). Peste des petits ruminants* was described as disease that made the sheep and goats depressed hence the *Lomoo* naming which etymologically is derived from action of looking sickly and depressed. Other names used to describe depression in goats and sheep, associated with PPR were *Ekitowo* and *Loutogonyen,* literally meaning sick goat syndrome though condition named *Loutogonyen* had other signs such as sunken eyes and emaciation (Kihu *et al*., 2012a; Ohta, 1984).

The PPR disease was associated more with goats where it was highly ranked after CCPP. In sheep, the PPR disease was ranked the sixth among other sheep diseases. 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). The Turkana community clearly characterized PPR by associating the disease with key clinical signs signifying that the community had witnessed and experienced the disease in their herds. In both sheep and goats, the Turkana community prominently associated PPR with migration, herd mixing and raids. This must have come from observation and realization that some goats and sheep, individually or as herds, introduced into new herds must have spread the PPR infection to the herds that hosted them. The community analyzed the seasonality of PPR where the disease in sheep and goats was

significantly associated with dry season. Activities associated with dry season such as migration and herd mixing, in formation of *adakars* for search of pastures and water, were seen to encourage the emergence of PPR in small stock. Livestock raids were known to be practiced throughout the seasons and could contribute to the emergence of PPR disease in all seasons. Key environmental features associated with PPR disease in goats were: toxic plants and mountainous pasture and water during dry season. However following probing of respondents, it was established that during dry season the small stock, particularly goats, were migrated to the high mountainous ranges that had some remaining pastures and water trapped between rocks. These dry season grazing areas in the mountains offered opportunity for extensive mixing of small stock from different herds during watering and grazing, providing for opportunity of infected small stock to make contacts with susceptible herd thus spread PPR infection. Such PPR infection, got during grazing in highlands, were thus blamed on the mountainous plants and water trapped in the rocks. Though the community's appraisal of PPR associated the disease with all age groups in goats and sheep, except lambs, it was interesting to note that discussion on morbidity and mortality due to PPR included all age groups in both sheep and goats. Despite PPR being ranked sixth in sheep diseases, the community perceived PPR to have higher morbidity, mortality and case fatality than any other sheep disease. Same appraisal of PPR morbidity, mortality and case fatality was observed in goats.

The appraisal of PPR by the Turkana community, through the various participatory epidemiology appraisal tools, yielded a description of the PPR disease that was

compatible with veterinarian scientific description of PPR. The *Lomoo* cases presented by the respondents during the study were examined on clinical and necropsy signs presented and tentatively diagnosed as PPR by the study team. Further laboratory analysis of tissue samples from the necropsied goats, through histological examination, revealed lesions in the lungs, intestines, mediastinal and mesenteric lymph nodes that were consistent with PPR infection. Two years after the samples were collected they were analyzed through real time reverse transcriptase polymerase chain reaction; *Peste des petits ruminants* virus genome was confirmed present in the sample tissues of the Turkana goats presented for analysis. It was worth noting that PPR genome was also detected by qRT-PCR in the formalin fixed tissues collected from the same goats and having been stored for two years in 10% formalin opening a possibility of using such tissues for PPR virus extraction for disease confirmation.

3.5. Conclusion

Peste des petits ruminants is a plague that has seriously affected the Turkana community. As such the disease could not escape community scrutiny since it touched on livestock, the core of Turkana community survival. The Turkana community has thus developed a very comprehensive description of PPR disease based on how they have observed the disease affect their small stock. In the arid pastoral areas of Kenya, where insecurity is prevalent alongside poor communication infrastructure, hard scientific and socio economic data is difficult to come by. However it has been observed from this study that pastoral communities who live in these marginal areas

have wealth of knowledge which can be tapped using appropriate participatory tools. *Peste des petits ruminants,* being a disease of major international concern, its control and eradication will depend partly on strong surveillance system and multi-country collaborative efforts. It is worth noting that this study has illuminated further the need to utilize the wealth of indigenous knowledge on diseases of livestock that reside with pastoral communities, for purposes of understanding diseases in the community and setting up strong participatory surveillance systems that involve the communities as the basic element of disease surveillance intelligence gathering.

CHAPTER 4: *PESTE DES PETIT RUMINANTS* (PPR) HERD IMMUNITY IN TURKANA COUNTY

4.1 Introduction

In Kenya the PPR was first suspected in 1992 (FAO, 2008) and confirmed in Turkana County in 2007 (ProMed-Mail, 2007). Serological data on PPR in Kenya was first reported in 1995 (Wamwayi *et al*., 1995). However published structured population based studies of PPR in Kenya are absent. Following the 2007 outbreak of PPR and subsequent vaccination responses in Turkana County a sero-monitoring exercise was carried out by the Director of Veterinary Services of Kenya and reported in unpublished report by VSFB (2007). Seroprevalence of 63% was reported by VSFB (2007) following the sero-monitoring exercise, of which 72% of positive animals were vaccinated and 27% of positive animals were unvaccinated. Since there was no pre vaccination sero monitoring, it was difficult to establish sero-conversion rates following the vaccination. In 2009, following a national vaccination exercise, a post-vaccination sero-monitoring exercise was carried out nationally by the Director of Veterinary Services of Kenya and reported in unpublished report by Kenya Veterinary Association Privatisation Scheme (KVAPS), (2009). There has also been other several *ad hoc* vaccination campaigns that have been carried out in the other regions of Kenya; however no pre- or post-vaccination sero-prevalence data is available. Any sero-prevalence data from Turkana and any other Kenyan regions will be likely to be a

mixture of vaccination immunity and immunity from wild virus, as well as maternal immunity.

This study aimed at establishing the sero-prevalence of PPR in the study area of North West Turkana.

4.2 Materials and methods

4.2.1 Study area

The study location was Turkana County that borders internationally with Ethiopia, Sudan and Uganda and internally borders Marsabit, Samburu, West Pokot and Baringo Counties previously described and depicted in section 3.2.1 and Figure 3.1 of Chapter 3.

4.2.2 Study community

Turkana is an arid and semi-arid area inhabited by Turkana people who are mainly pastoralists. Livestock keeping is the main livelihood among the Turkana people. The estimated populations of sheep and goats are 3,517,151 and 5,994,861 respectively (KNBS, 2010). Sheep and goats constitute 90% of livestock in Turkana region.

4.2.3 Study design

The sero-prevalence survey carried out was a cross-sectional one and aimed at determining the level of herd immunity in sheep and goats. The study design was based on a proportionate stratified random sampling while the sample frame was based on

sheep and goat populations in the six administrative divisions that formed the study area.

4.2.4 Sample size

The study focus was a region in Turkana County covered by six administrative divisions namely, Loima, Oropoi, Kakuma, Lokichogio, Kaaleng, and Kibish, being the international frontier bordering divisions that made initial reports on PPR disease outbreaks in 2006. The study population included all respective *Adakars* identified through local veterinary workers, key informants and population data. Proportional stratified random sampling was done using the locally determined small ruminants' herd structure, small ruminants' age groups, and vaccination status. The stratum sample size was proportional to small ruminants' population size in each administrative division. The strata considered in this study were five for each species investigated.

At the time of this study, PPR outbreaks had already occurred in the county and some vaccinations had also been carried out, and therefore,the seroprevalence was unknown. 50% sero-prevalence and a relative error of 10% were assumed when determining the desired sample size of sheep and goats to be sampled.The computed sample size therefore had a precision of ±0.05. In this case, 0.05 is the absolute error. We chose the 50% sero-prevalence because it provides the largest sample size (for given values of absolute error). Sample size for sero-prevalence sampling was determined using the formula described by Bennett *et al.,* (1991) in ProMESA (2011) a software programme for statistical sampling in animal populations.

$$n = \frac{\sum_{i=1}^{e}\left[\frac{(n_i)^2 \times p_i \times (1-p_i)}{N_i}\right]}{N^2 \times \frac{AE^2}{Z^2} + \sum_{i=1}^{e}\left[n_i \times p_i \times (1-p_i)\right]}$$

Where:

e	The number of strata.
n_i	The number of individuals in strata i.
p_i	The expected prevalence in strata i.
N	The total number of individuals in the population.
AE	The acceptable absolute error.
z	The value obtained from the standard normal distribution. To each value of confidence there is a correspondent value of z. The level of confidence used was in biological 95%. The value of z correspondent was 1.96.
w_i	A weighting factor of each strata, calculated as follows:

$$w_i = \frac{n_i \times \sqrt{p_i \times (1-p_i)}}{\sum_{i=1}^{e}\left[n_i \times \sqrt{p_i \times (1-p_i)}\right]}.$$ <=”” p=”” border=”0”>

Proportional stratified random sampling using the age groups, and vaccination status as the strata was done in relation to population size in each division. The strata considered in this study were five, developed from age groups and vaccination status of sheep and goats.

Since there is no serological test that can differentiate animals vaccinated with homologous PPR vaccine from animals that have recovered from natural PPR infection, it was conceived that in the prevailing circumstances, where vaccination and natural disease occurrences are common in Turkana district, the pastoral community were the best source of information as regards age groups and vaccination status of both sheep and goats. Distinction between vaccinated and recovered animals was done using community local knowledge on age and vaccination history; supported by markings such as ear notching and vaccination certificates.

4.2.4.1 Herd age structure in sheep and goats

Using semi-structured interviews and proportional piling as described in Chapter 3 section 3.2.3.2.1 and section 3.2.3.2.4.1 respectively the Turkana community divided the sheep and goats flocks into four groups based on age and provided the proportions of each respective age group in Tables 4.1.

Table 4.1: Distribution of sheep and goats by age categories in Turkana. n=27

Animal spieces and age Category	Local name	Percentage proportion of each age group in the population	
		Mean Score	SD (score±)
Sheep			
Up to 5 months	Imethek	15	5
6 to 12 months	Nanyang	21	6
13 to 24 months	Nakale	25	7
>24 months	Amethek Naapolon	39	8
Goats			
Up to 5 months	Ikale	18	5
6 to 12 months	Namenaoei	21	6
13 to 24 months	Akale	22	4
>24 months	Akale	39	10

However the final age groups per species were later reduced to three. The age group of 6 to 12 months was combined to age group 12 to 24 months to form a new group called middle age group as shown in Tables 4.2.

Table 4.2: Herd structure of sheep and goats in the study area.

	Up to 5 months age Young ones	6 to 24 months of age Middle age	older than 24 months Adult
Sheep Age groups %	15	47	38
Goat Age groups %	18	43	39

The last major vaccination campaign in Turkana was carried out in 2007 where 1,380,283 small stocks (sheep and goats) were vaccinated against PPR (unpublished report by VSFB, 2007). The total population of small stock in Turkana is 9,512,012 (KNBS, 2010). Therefore the proportion of small stock vaccinated was only 14% against 86% unvaccinated.

It is from this analysis of age groups and vaccination levels that the five strata; young ones (kids or lambs), middle aged vaccinated, middle aged non-vaccinated, adults vaccinated and adults non-vaccinated were conceived and population per strata established.

4.2.4.2 Parameters for sample size calculation

- Level of vaccination within the Turkana district is 14% vaccinated and 86% non-vaccinated (KNBS, 2010; VSFB, 2007).

- The herd structure was as given in Table 4.2 and derived by this study from Tables 4.1.

- Sheep and goat populations were as given in Table 4.3 (KNBS, 2010)

Table 4.3: The population of sheep and goats in the study area comprising six administrative districts. (KNBS, 2010)

	species	
Division	**Sheep**	**Goat**
Loima	158221	408722
Kakuma	75434	156685
Oropoi	272707	411676
Lokichoggio	236701	284546
Kaaleng	312056	509844
Kibish	212863	248983
Total population	**1,267,982.00**	**2,020,456.00**

- Expected prevalence was 0.5 being
- Confidence interval applied is 95% with z value of 1.96
- Number of strata are 5 (as discussed earlier)
- Acceptable relative error (Measure of precision) is 0.1

Using the formula described by Bennet et al., (1991)

$$n = \frac{\sum_{i=1}^{e}\left[\frac{(n_i)^2 \times r_i \times (1-p_i)}{w_i}\right]}{N^2 \times \frac{AE^2}{Z^2} + \sum_{i=1}^{e}\left[n_i \times p_i \times (1-p_i)\right]}$$

with W1 being $$W_i = \frac{n_i \times \sqrt{p_i \times (1 - p_i)}}{\sum_{i=1}^{e} \left[n_i \times \sqrt{p_i \times (1 - p_i)} \right]}.$$

and the ProMESA (2011) software programme for statistical sampling in animal populations, the sample size was determined as 384 samples per species. The 384 samples per each species were proportionately allocated to each stratum as given in Table 4.4.

Table 4.4: Proportionate stratification of 384 samples per species.

.	Young ones	Middle age vaccinated	Middle age non-vaccinated	Adult vaccinated	Adult non-vaccinated
sheep	58	25	153	21	127
goats	69	23	142	21	129

4.2.5 Sampling unit

The sampling unit was an individual animal of specific age and vaccination status belonging to an *Adakar.* An *Adakar* entails a cluster of often-related Turkana households that pursue similar socio-economic activities such as search for pasture, water and security, under a trusted leader (Bett *et al.,* 2009). The Turkana live in small households that consists a man, his wives, their children and possibly some dependent women. This social unit is referred to as household and is called *awi*. Household size varies considerably according to wealth, but averages about 20-25 people (McCabe, 1984). Each household chooses the *Adakar* that they wish to move with. Households

forming an *Adakar* range from 40 to 100 (Akabwai, 1992; AMREF, 2012). The majority of the people and most of the milking animals live in the major homestead which remains in the plains throughout the year but moves frequently as forage and water resources are depleted. In the wet season the milking and non-milking herds are mixed, with all the people and animals coming together in their *awi*. As the dry season progresses, non-milking herds may be split off from the *awi* to enjoin the *Adakar* of choice and often move to the foothills and slopes of the mountains where the vegetation lasts longer than it does on the plains.

For the purpose of this study the number of households per *Adakar* was taken to be 70 (being the average). Using the census population data of rural Turkana households (KNBS, 2010) in the study area of Loima, Oropoi, Kakuma, Lokichogio, Kaaleng, and Kibish, the number of *Adakars* was estimated to be 538. Sheep and goats population for each *Adakar* was estimated by dividing separately the population sheep and goats with number of *Adakars* estimated in each sub location. It was assumed that *Adakars* in any one sub location were of the same size. All *Adakars* in the sub-locations starting with those in Loima, Oropoi, Kakuma, Lokichoggio, Kibish and then Kaaleng divisions were allocated numbers 1 to 538 and strata population for each *Adakar* listed alongside the *Adakar*. Cumulative population per stratum for all *Adakars* was estimated with first animal in the stratum being from Loima and last being from Kaaleng. Using simple random sampling the sample for each stratum was selected from stratum population where each selected animal corresponded to its population number within an *Adakar* as indicated by cumulative population in that stratum. Therefore the *adakars* selected were

home of sampled animals. Out of the 538 *Adakars* estimated in the study area, animals in 142 *Adakars* were sampled. The animals not sampled were from *Adakars* located in some of the sub locations of Oropoi, Lokichogio, Kakuma and Kaaleng divisions that were experiencing livestock rustling insecurity, high mobility of the Turkana pastoralists and bad weather rendering roads impassable and inaccessible in certain areas.

A total of 969 serum samples were collected from sheep and goats (538 goats and 431 Sheep) in the six administrative divisions. The final number of samples collected for each species was slightly higher than the calculated stratum sample size and the final total samples for each species however the extra samples for each species were allocated to each stratum proportionately to stratum population. Of the 431 serum samples of sheep, 41 were from adults that were vaccinated, 156 from non vaccinated adults, 16 from middle aged vaccinated, 154 from middle aged non vaccinated, and 64 from newborn lambs. Out of the 538 serum samples from goats, 50 were from vaccinated adults, 177 from non vaccinated adults, 22 from vaccinated middle aged, 189 from non vaccinated middle aged and 100 from newborn kids. Study sites are shown in previous Figure 3.2.

4.2.6 Serum collection

Blood was collected by jugular-vein puncture using venoject needles and vacutainer tubes (Venoject, UK). The blood was transported to the field laboratories where it was left to clot overnight at room temperature in boxes. The serum was decanted into sterile

tubes and centrifuged to remove the remaining red blood cells before being transferred to 2 ml cryovials and stored at -20˚C.

4.2.7 Competitive Enzyme Linked Immunosorbent Assay (C-ELISA)

The PPR competitive enzyme linked immunosorbent assay (C-ELISA) test kit (ID Screen® PPRC, Montpellier, France) and a corresponding assay protocol was used in the analysis. The technology was developed by FAO reference laboratory CIRAD-EVMT, Montpellier France. Figure 4.1 presents the scheme for competitive ELISA technique

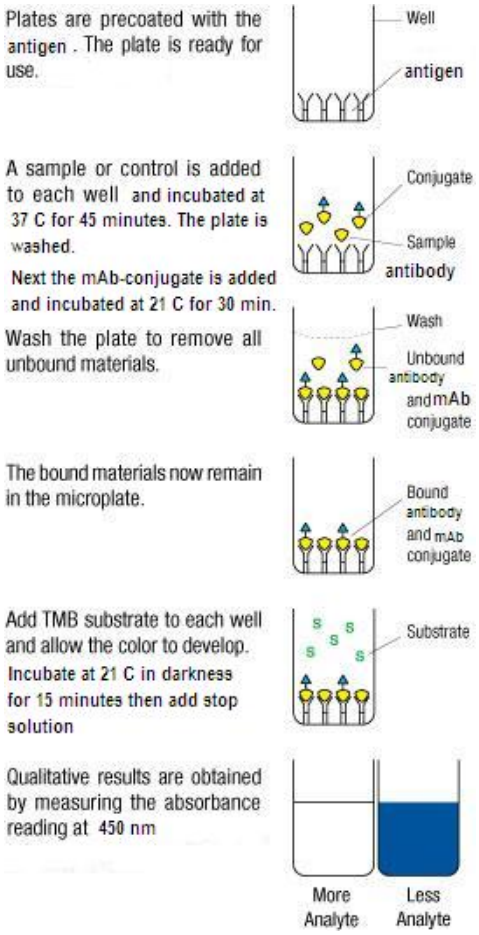


Figure 4.1: Competitive ELISA for antibody detection. Schema developed by Neogen Corporation, USA (Neogen, 2012) and modified by author.

The ELISA microplates were read with Thermo Scientific Multiskan® EX (Thermo Fischer Scientific, Finland) reader with inference filter of 450 nm. The reader was connected with a computer loaded with Thermo Scientific Ascent® Software for Multiskan (Thermo Fischer Scientific, Finland). The software was used to automate the reading where fifteen optical density (OD) were measured in kinetic mode per well and mean established as the final reading per well. With the software, the mean OD values per well were organised and the percentage competition calculated. For each sample, calculated competition percentage was based on the following formula:

Competition% = $(OD_{sample}/OD_{NC})*100$

The cut-off criteria provided by the manufacturer of the kit was as follows: competition percentage equal or less 50% were considered positive; samples with competition greater than 50% but equal or less than 60% were considered doubtful; samples with competition greater than 60% were considered negative. This criterion was inserted in the reader's software, and based on the competition percentage per well, the samples were organised into positives, doubtful and negatives.

According to the manufacturer the test should only be considered validated if the mean value of negative control optical density (OD_{NC}) is greater than 0.7 and the mean value of positive control (OD_{PC}) optical density is less than 30% of $OD_{NC.}$ The results of the competitive ELISA were plotted as a frequency and cumulative percentage of the percentage colour competition.

4.2.8 Statistical analysis

The data generated by the Ascent software was further arranged using Microsoft Excel, (Microsoft Inc USA). Statistical Package for Social Science (SPSS) statistical software version 17.0 (IBM Corp., Armonk, NY) was used to generate descriptive statistics between age groups, sex and administrative units and tested by Chi square statistic. Seroconversion status as the dependent variable was investigated as binary outcome variable in a logistic regression model against idependent variables that were species, sex, age group, vaccination status and administrative division. The analysis were ran using SSPS Backward elimination method of logistic regression with p-value < 0.1 at initial univariate analysis where the significant factors were were analysed in a final multivariable analysis with $p<0.05$. The maps were produced by ArcGIS version 9.1(ESRI, Redlands, California).

4.3 Results

4.3.1 Frequency Curves of antibody distribution

The samples that were positive for PPRV in sheep and goats showed a peak frequency distribution between 5% and 15% competition range in goats and between 5% and 10% in sheep. The peak frequency distribution of the negative samples in sheep and goats was in the range of between 60% and 95% competition for goats while that of sheep was between 65 and 90% (Figures 4.2 and 4.3).

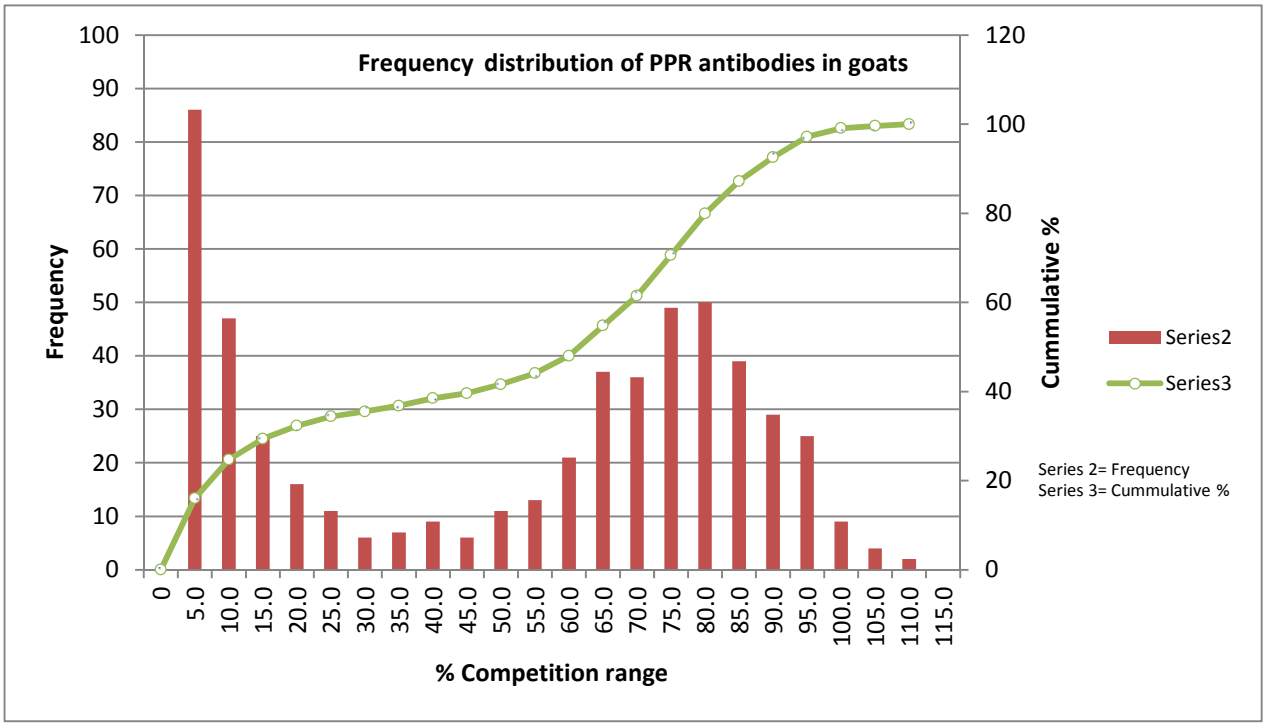


Figure 4.2: Frequency distribution of antibodies against PPR in Goats

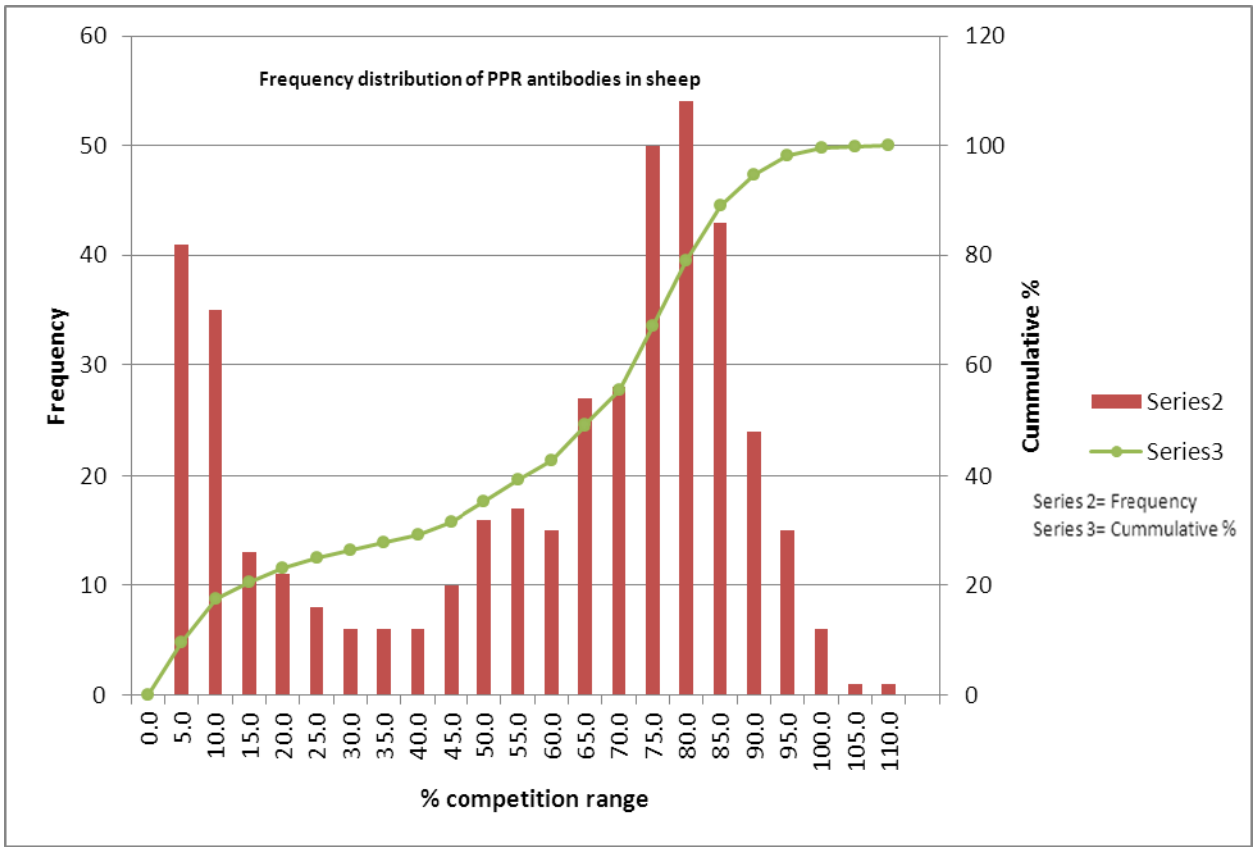


Figure 4.3: Frequency distribution of antibodies against PPR in sheep

4.3.2 PPR antibody status by species

The sero-prevalence for sheep and goats combined (n=969) in the six administrative divisions was 36% (95% confidence interval (CI): 33.00% to 39.1%). The goats (n=538) had mean sero-positivity of 39.6% (95% CI: 35.5% to 43.9%) which was significantly higher than that of sheep (n=431), at 31.6% (95% CI: 27.2% to 36.2%) χ^2 (p=0.000).

4.3.3 PPR antibody by sex in each species

For the sheep, the mean sero-positivity in females was 33.3% (27.7% to 39.5%) while in males, it was 28.8% (95% CI: 22.3% to 36.3%), with no significant difference p=0.389. In goats mean sero-positivity in females was 44% (95% CI: 38.5% to 49.6%); it was significantly higher than that of males which was 33% (95% CI: 26.9% to 39.8%) χ^2 (p=0.023).

4.3.4 PPR antibody status by age in each species

The mean sero-prevalences were significantly different between the age groups in sheep with that of adults being 39.6% (95% CI: 32.8% to 46.8%), that of middle aged group being 18.2% (95% CI: 12.9% to 25.0%), while that for lambs were 42.2% (95% CI: 30.2% to 55.2%) χ^2 (p=0.000). Mean sero-prevalences for goats agegroups were: adults 63.4% (95% CI: 56.8% to 69.6%), middle aged 14.2% (95% CI: 10.0% to 20.0%), kids 39.6% (95% CI: 29.6% to 49.4%) χ^2 (p= 0.000). Table 4.5 gives the prevalence profiles for the sheep and goats.

.

Table 4.5: Prevalence profiles of antibody against PPRV and proportion of sheep and goats investigated by variables

Variable	Sheep n=431			Goats n=538		
	sera Examined	sera Positive	% sero-positivity [95% CI]	sera Examined	sera Positive	% sero-positivity [95% CI]
Species	431	136	31.6[27.20,36.20]	538	213	39.6[35.46,43.88]
Sex						
Male	170	49	28.8[22.27,36.34]	251	71	33.0[26.87,39.80]
Female	261	87	33.3[27.71,39.45]	323	142	44.0[38.50,49.57]
Age						
Adult	197	78	39.6[32.78,46.81]	227	144	63.4[56.77,69.63]
Middle age	170	31	18.2[12.9,25.04]	211	30	14.2[10.04,20.01]
Young	64	27	42.2[30.15,55.15]	100	39	39.0[29.56,49.30]
Sex and age						
Male Adult	71	23	32.4[22.05,44.66]	77	36	46.8[35.42,58.41]
Male middle age	68	12	17.6[9.83,29.19]	90	12	13.3[7.38,22.52]
Male young	41	14	34.1[24.26,45.53]	84	23	27.4[20.93,34.89]
Female Adult	126	55	43.7[34.92,52.76]	150	108	72.0[63.98,78.87
Female middle age	102	19	18.6[11.86,27.81]	121	18	14.9[9.29,22.76]
Female young	33	13	39.4[27.82,52.2]	52	16	30.8[22.29,40.69]
Vaccination status						
Adult Vac*	41	27	65.9[49.33,79.44]	50	33	66.0[51.14,78.41]
Adult NVac**	156	51	32.7[25.52,40.72]	177	111	62.7[55.10,69.76]
Mid age Vac*	16	9	56.3[30.55,79.24]	22	7	31.8[14.73,54.88
Mid age NVac**	154	22	14.3[9.36,21.04]	189	23	12.2[8.03,17.90]

*Vac= Vaccinated; **NVac= Non vaccinated

4.3.5 PPR antibody status by vaccination status in each species

The PPR antibody profiles in the sheep within the specific age group were: vaccinated adults 65.9% (95% CI 49.3% to 79.4%), non vaccinated adults 32.7% (95% CI: 25.5% to 40.7%), vaccinated middle aged 56.3% (95% CI: 30.6% to 79.2%), non vaccinated middle aged 14.3% (95% CI: 9.4% to 21.0%) χ^2 (p=0.000) and lambs 42.2% (CI 30.2 to 55.2). The PPR antibody profiles in goats, taking into account vaccination status, were; vaccinated adults 66%(95% CI: 51.1% to 78.4%) , non-vaccinated adults 62.7%(95% CI: 55.1% to 69.8%) , vaccinated middle aged 31.8%(95% CI: 14.7% to 54.9%), ,non vaccinated middle aged 12.2%(95% CI: 8.0% to 17.9%) χ^2 (p=0.000) and new born kids 39% (CI 29.6% to 49.3%).

4.3.6 PPR antibody by geographical divisions

Sero-prevalence results by geographical divisions showed that, PPR antibody prevalence in sheep within Kaaleng division had lowest seropositivity of 15.4% (95% CI: 6.4% to 31.2%); the highest sero-prevalence was recorded in Oropoi at 68.3% (95% CI: 51.8% to 81.4%) (p=0.00) (Figure 4.4). Kakuma recorded the lowest sero-prevalence in goats of 22.1% (95% CI: 15.75% to 30.1%) while Oropoi had highest sero-prevalence at 63.2% (95% CI: 50.62% to 74.35%) with (p=0.000) (Figure 4.5).

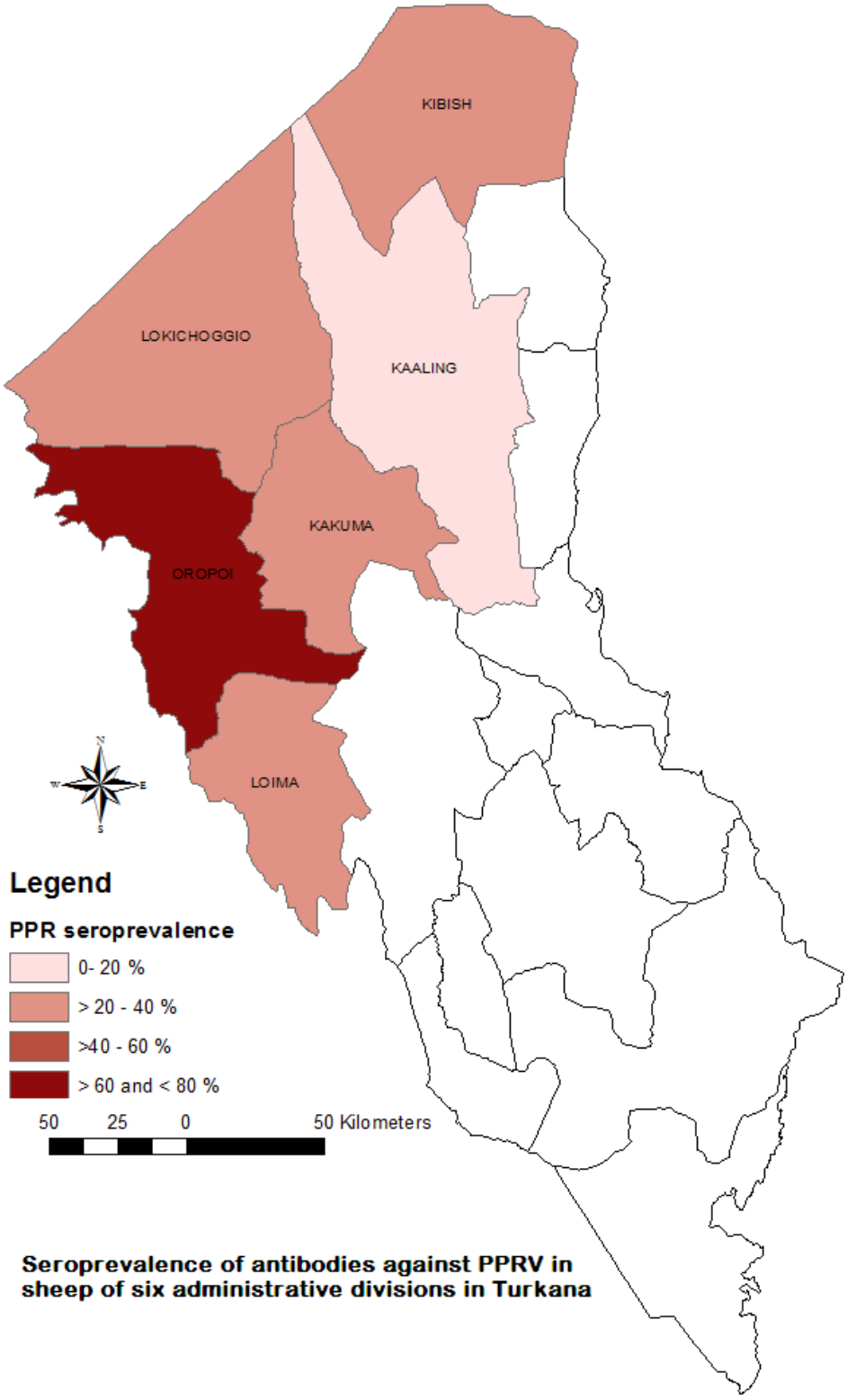


Figure 4.4: Geographical distribution of PPR sero-prevalence in sheep

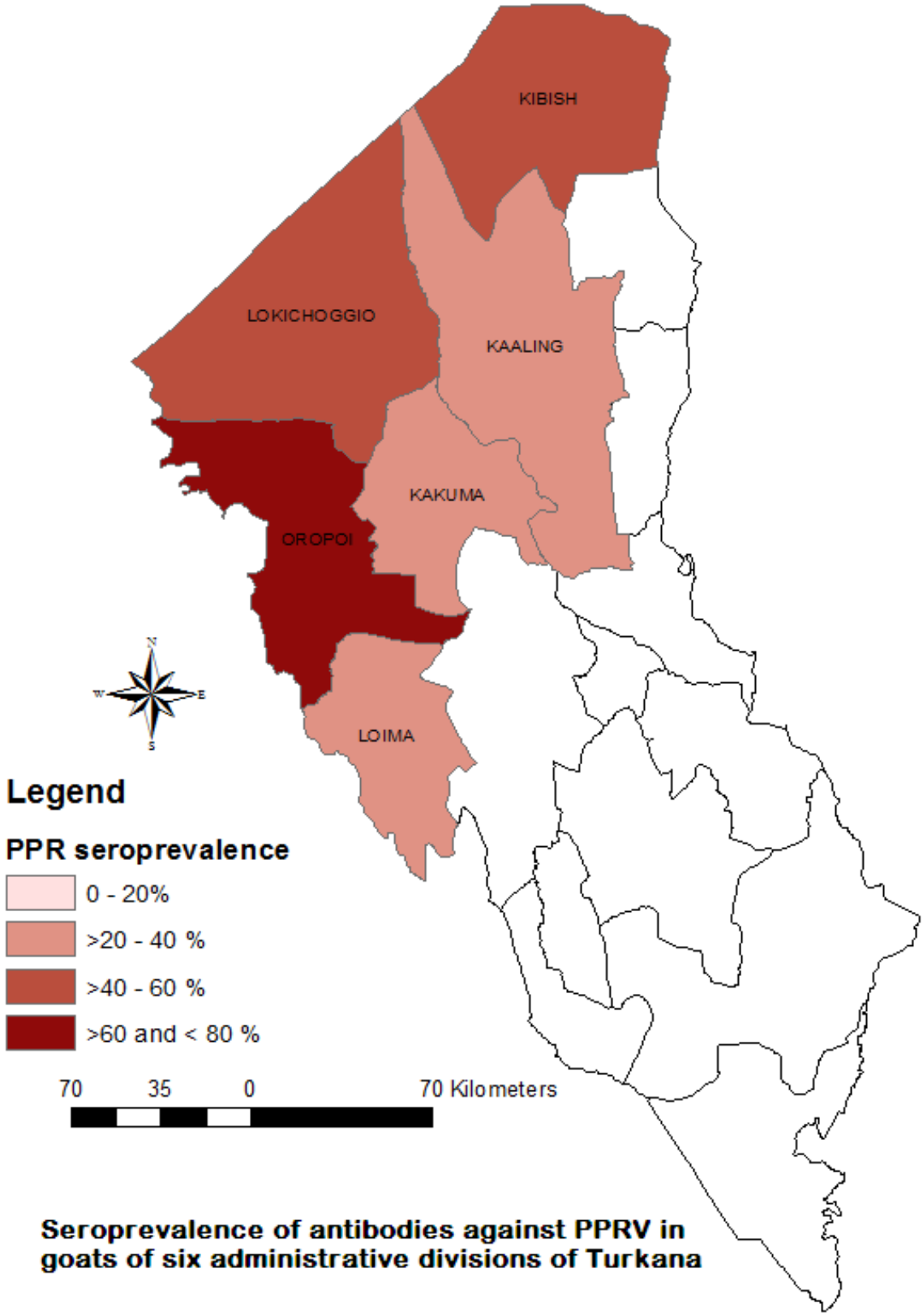


Figure 4.5: Geographical distribution of PPR sero-prevalence in goats

4.3.7 Risk factors for sero positivity

In the initial univariate logistic regression models that were ran for each species, the significant risk factors identified for seropositivity in sheep were sex, vacination status, age and division (Table 4.6). While in goats the risk factors identified were vaccination status, age and division (Table 4.7).

Table 4.6: Variables associated with seroposity in Univariate models of goat data

								95% C.I.for EXP(B)	
	Variable	**B**	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**Lower**	**Upper**
Model 1	Male	-.410	.183	5.047	1	.025	.664	.464	.949
	Constant	-.255	.112	5.176	1	.023	.775		
Model 2	Unvaccinated	.609	.249	5.987	1	.014	1.839	1.129	2.996
	Constant	-.504	.096	27.542	1	.000	.604		
Model 3	Age			94.274	2	.000			
	Adult	.896	.245	13.327	1	.000	2.450	1.514	3.963
	Middle Age	-1.433	.283	25.617	1	.000	.239	.137	.415
	Constant	-.364	.203	3.204	1	.073	.695		
Model 4	Div			43.925	5	.000			
	Kakuma	-1.800	.324	30.938	1	.000	.165	.088	.312
	Kaaleng	-1.427	.371	14.786	1	.000	.240	.116	.497
	Loima	-1.028	.361	8.091	1	.004	.358	.176	.726
	Loki	-.892	.321	7.732	1	.005	.410	.219	.769
	Constant	.542	.252	4.650	1	.031	1.720		

Reference for vaccination status was Vaccinated, for age groups was Young, for administrative Divisions was Oropoi and for sex was Female.

Table 4.7: Variables associated with seropositivity in Univariate models of sheep data

								95% C.I.for EXP(B)	
	variable	**B**	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**Lower**	**Upper**
Model 2	unvaccinated	-1.547	.298	26.874	1	.000	.213	.119	.382
	Constant	.539	.275	3.853	1	.050	1.714		
Model 3	Age			22.169	2	.000			
	Middle Age	-1.185	.322	13.575	1	.000	.306	.163	.574
	Constant	-.315	.253	1.550	1	.213	.730		
Model 4	Div			37.400	5	.000			
	Kakuma	-2.113	.423	24.954	1	.000	.121	.053	.277
	Kibish	-1.257	.394	10.185	1	.001	.285	.132	.616
	Kaaling	-2.472	.556	19.737	1	.000	.084	.028	.251
	Loima	-1.243	.428	8.432	1	.004	.289	.125	.668
	Loki	-2.020	.414	23.851	1	.000	.133	.059	.298
	Constant	.767	.336	5.226	1	.022	2.154		

Reference for vaccination status was Vaccinated, for age groups was Young, for administrative Divisions was Oropoi and for sex was Female.

Species was identified as a risk factor for seropositivity when combined data of sheep and goat was analysed in univariate model (Table 4.8)

Table 4.8: Combined sheep and goats data in a univariate model testing species as a risk factor

								95% C.I.for EXP(B)	
	Variable	**B**	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**Lower**	**Upper**
Model 1	Goats	.360	.136	6.987	1	.008	1.433	1.097	1.870
	Constant	-.774	.104	55.812	1	.000	.461		

Reference species was sheep

The final multivariate logstic regression model for goat returned age and division as risk factors for sero-positivity in goats (Table 4.9). Adult goats were 2.381 times more likely to have PPR antibodies compared to kids p=0.003. Middle aged goats were 0.808 less likely to have PPR antibodies compared to kids (Odds ratio =0.191; p=0.000). Risk factors identified for seropositivity in sheep in the final multivariate model were vaccination status, age and division (Table 4.10). The sheep that were not vaccinated were 4.24 times likely to have PPR antibodies compared to the vaccinated sheep p=0.000. Similarly the middle aged sheep were 0.818 less likely to have PPR antibodies compared to lambs (Odds ratio=0.182; p=0.000). The sheep and goats in the other five administrative divisions were less likely to have PPR antibodies compared to goats and sheep in Oropoi division.

Table 4.9: Variables associated with seroposity in final multivariate models of goat data

							95% C.I.for EXP(B)	
Variable	**B**	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**Lower**	**Upper**
Age			88.591	2	.000			
Adult	.868	.291	8.863	1	.003	2.381	1.345	4.216
Middle Age	-1.656	.319	27.003	1	.000	.191	.102	.356
Division			46.385	5	.000			
Kakuma	-2.217	.379	34.280	1	.000	.109	.052	.229
Kaaleng	-1.706	.463	13.558	1	.000	.182	.073	.450
Loima	-1.564	.436	12.890	1	.000	.209	.089	.492
Loki	-1.373	.426	10.411	1	.001	.253	.110	.583
Constant	1.077	.375	8.226	1	.004	2.936		

Reference for age groups was Young and for administrative Divisions was Oropoi

Table 4.10: Variables associated with seropositivity in final multivariate models of sheep data

							95% C.I.for EXP(B)	
Variable	**B**	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**Lower**	**Upper**
Unvaccinated	1.444	.381	14.359	1	.000	4.240	2.008	8.949
Age			26.387	2	.000			
Middle Age	-1.701	.368	21.405	1	.000	.182	.089	.375
Div			35.001	5	.000			
Kakuma	-2.287	.478	22.863	1	.000	.102	.040	.259
Kaaling	-2.201	.625	12.418	1	.000	.111	.033	.376
Loima	-.994	.490	4.113	1	.043	.370	.142	.967
Loki	-1.754	.485	13.091	1	.000	.173	.067	.448
Constant	1.255	.492	6.513	1	.011	3.508		

Species were also retained as a risk factor in final multivariate model of combined data of sheep and goats where it was established that the likelihood of presence of PPR antibodies was higher in goats than in sheep (Odds ratio= 1.644; $p<0.001$).

Table 4.11: Combined sheep and goats data in a multivariate model testing species as a risk factor

							95% C.I.for EXP(B)	
Variable	**B**	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**Lower**	**Upper**
Goat	.497	.155	10.253	1	.001	1.644	1.213	2.228

Reference was the sheep

4.4 Discussion

Peste de petits ruminants is considered a recent disease of small stock having been clinically observed in Kenya for the first time in Turkana district in 2007 (Kihu *et al.,* 2012a). The outbreaks experienced in Turkana were dramatic with high mortality causing panic in the livestock sector. The response to the outbreak was mass vaccination that was sponsored by donors (VSFB, 2007). However the numbers of small stock vaccinated by the donor sponsored exercise in 2007 was 1,380,283; constituting 14% of the total population of 9,512,012 small stock in Turkana district (KNBS, 2010). The current study has, however, established a vaccination prevalence rate of 7.84%. This is probably due to lots of the vaccinated small stock leaving the herds; i.e. during the period between last vaccination (year 2008) and the time of the study (year 2011). It could also be due to the actual vaccination coverage having been very narrow (low). This, therefore, means that the PPR antibody profile in the study area could be attributed to both wild virus and vaccination. By inference, however, the presence of antibodies was more likely to reflect infection as demonstrated by vaccination status of the sheep which indicated that non vaccinated sheep were 4.2 times ($p=0.000$) more likely to have PPR antibodies. This observation can be explained by the fact that non vaccinated sheep were more likely to show an increase in antibody prevalences.

The frequency distribution of the percentage color competition shows that large populations of the sheep (69.4%) and goats (61.4%) did not have sufficient antibody titers to mount a strong humoral antibody response to PPRV infection. Overall, sero-prevalence for the small stock for this study was 36%. It was lower than 55.26% reported by Luka *et al*. (2011) in Karamoja Uganda; an area that shares common boundary and social, cultural and environmental similarities with Turkana County. A study by Swai *et al*. (2009) noted a sero-prevalence in Northern Tanzania of 49.5% in goats and 39.8% in sheep. Whereas the sero-prevalence in the Kenyan study is lower compared to Uganda and Tanzania, the difference could be attributed to various factors (Gur and Albayrak 2010). The difference in sero-prevalence between Karamoja Uganda and Turkana Kenya can be attributed, in part, to the difference in level of coverage and intensity of PPR vaccinations over time in the two areas; the two areas experienced the initial outbreaks at the same time, in 2007. Other factors that can be eluded in causing the differences in sero-prevalence between these studies include socio-economics of small stock husbandry management, diagnostic tests used and sampling procedures (Swai *et al.,* 2009, Waret-Szkuta *et al.,* 2008).

Goats were found to have a significantly higher percentage of sero-positivity (at 39.6%) compared to sheep (at 31.6%) (p=0.000). However, looking at the seroprevalence by age and sex, for both species, adult goats and more so females contributed to the elevated sero-positivity in goats. This can be attributed to the fact that female goats, being the main source of breeding stock, rarely leave herds, thus those female goats that

survive PPR outbreaks or were vaccinated are likely to remain in herds in their productive life for a long period of time. This also explains the significantly lower sero-positivity in male goats (33%) compared to females (44%). Male goats are often sold in markets as the main source of immediate household income or sacrificed in various cultural ceremonies, thus large numbers of male goats that would have been exposed to wild virus or vaccinations are removed from the herds. This, therefore, means that most of the remaining male goats in herds are usually newly recruited and may not have had contacts with vaccine or wild virus. The sheep are less considered for economical purposes and thus both female and male sheep often remain in herds for almost similar periods of times. It was thus observed that, although the sero-positivity in male sheep was 28.8% compared to female sheep (at 33.3%), there was no significant difference between the two ($p = 0.389$).

There was a considerably significant difference in sero-positivity between age groups in both sheep and goats. The middle aged group in sheep had sero-positivity of 18.2% while middle aged goats had seropositivity of 14.2%. The small stocks in the middle age group sampled in this study were generally born in the period between 2009 and 2010 when no major vaccination was carried out. By the time of this study only Oropoi division and a couple of few other locations had conducted a new round of vaccination in Turkana district for the year 2011 and this meant most of the sheep and goats in this category of middle aged group had not been vaccinated. The data on vaccination status shows that vaccinated sheep and goats in middle aged group were but a small

proportion of the overall middle age group population. The vaccinated sheep in middle aged group had sero-positivity of 56.3% while goats in the similar group had 31.8%. The difference in sero-positivity in this age group is attributed to retention of sheep within herd compared to early disposal of goats, particularly the males, through market. However the non-vaccinated middle aged group had sero-positivity of 14.3% in sheep and 12.2% in goats; explanation for this difference being similar to that of the vaccinated middle aged group - that it had limited exposure to vaccination and wild virus. Therefore, this group remained the most risky since it had no antibody protection (Haque *et al.,* 2004).

The category of the young small stock (lambs and kids) sampled were tested for their levels of seropositivity which was in evidently due to maternal antibodies against PPR. This is evidenced by 42.2% of lambs being sero-positive, against that of 43.7% for female sheep. On the same vein, 39% of the kids were sero-positive, against that of 72% for female goats. The difference in sero-positivity between adult females and kids could be explained by the fact that some kids could have been born by female goats in the middle aged groups who had no PPR antibodies.

As noted earlier, some vaccinations were carried out in early 2011 in Oropoi division, three weeks prior to this study. Consequently the sero-prevalence in Oropoi division was significantly higher for both sheep (68.3%) and goats (63.5%) compared to sero-

prevalence in other administrative divisions of Kakuma, Loki, Kaaleng, Kibish and Loima. Sero-positivity for sheep was lowest in Kaaleng division at 15.4%.

Variables that appeared to be risk factors for sero-positivity were species, vaccination status, age groups and geographical administrative areas. Age was not significant risk factor in sheep while vaccination status was not a significant risk factor in goats.

4.5 Conclusion

In conclusion, this study has established that, despite the early vaccination after initial PPR outbreaks, the two year break (2009/2010) without vaccination created a pool of small stock in the middle aged group that were most susceptible to PPR infection because they were immunologically naïve. This was confirmed, in this study, by demonstrating that age was a risk factor for sero-positivity. Type (species) of small stock was also found to be a risk factor; this was due to the differential socio - economic importance accorded to sheep and goats, separately. Animals that remained within herds for longer period were also more likely to have PPR antibodies and remain so in the herd. Animals that were non-vaccinated were found to be more likely to produce PPR antibodies mainly due to wild virus infection; thus non vaccination status was identified as risk factor for sero-positivity. Areas that vaccinated against PPR had high level of sero-positivity. This study has established that the wild virus has continued to infect the immunologically naïve small stock and is widespread in Turkana district. However due the endemic status of the disease that has been established in Turkana; as well as the resultant high antibody production against PPRV,

dramatic outbreaks are not likely to occur; the many infections that occur at sub-clinical levels, constantly stimulate the middle-aged group to produce higher titers, which are protective. These findings are important in informing disease control managers particularly in managing the consistency and efficiency of PPR vaccination process.

CHAPTER 5: RISK FACTORS INFLUENCING THE PATTERNS OF *PPR* SPREAD IN TURKANA COUNTY

5.1 Introduction.

The epidemiology of PPR in Eastern Africa, and more so in Kenya, is less clearly understood (William and Barker, 2001). The link between the disease pattern and factors that could influence the disease dynamics, including socio-cultural and economic factors such as nomadism, transhumance, livestock trade or livestock rustling, has yet to be fully established. However, it is becoming evident that the human factors, more so the cultural and livelihood activities, play a great role in the emergence of the animal diseases (Robbins, 2012; Newcastle University, 2012). In pastoral societies, where the livelihood survival strategies develop around the use and accumulation of animals, culture plays a particularly important role in livestock disease spread. Some of the cultural activities, such as livestock raids and transfer of animals in marriage ceremonies, among other activities, increase probability of susceptible herds getting infection from incoming animals (Sollod and Knight, 1982). Therefore, understanding social and cultural aspects of small stock management practices, that could pose as risk factors for PPR is, for a large a part, a socio-ecological solution to the epidemiology of PPR in Turkana and other areas (Cumming, 2010). It would entail carrying out a PPR disease risk analysis that focuses on risk identification and risk assessment (MacDiarmid 1991). This study, therefore, attempted to evaluate the small ruminant management practices by Turkana herders, as predictors of PPR outbreaks; it

was done through integration of risk assessment with participatory methodologies (Grace *et al.,* 2008). The results of this study aim at aiding future designing of contextual and specific disease control policies. The objective of this study was to evaluate the small ruminant management practices by Turkana herders as risk factors of PPR outbreaks and spread in Turkana.

5.2 Material and Methods

5.2.1 Study area

The study location was Turkana County that borders internationally with Ethiopia, Sudan and Uganda and internally borders Marsabit, Samburu, West Pokot and Baringo Counties previously described and depicted in section 3.2.1 and Figure 3.1 of Chapter 3.

Generally, the County experiences both temporal and spatial rainfall variability, as well as frequent droughts and famines (Oba, 1992). Results from times series plot indicate Turkana District as having two distinct rainfall seasons: long rains (*Akiporo*) March, April and May (MAM) rainfall season and short rains (*erupe*) October, November and December (OND) rainfall season (Savatia, 2011). The annual rainfall ranges between 100 to 500 mm per year (Figure 5.1).

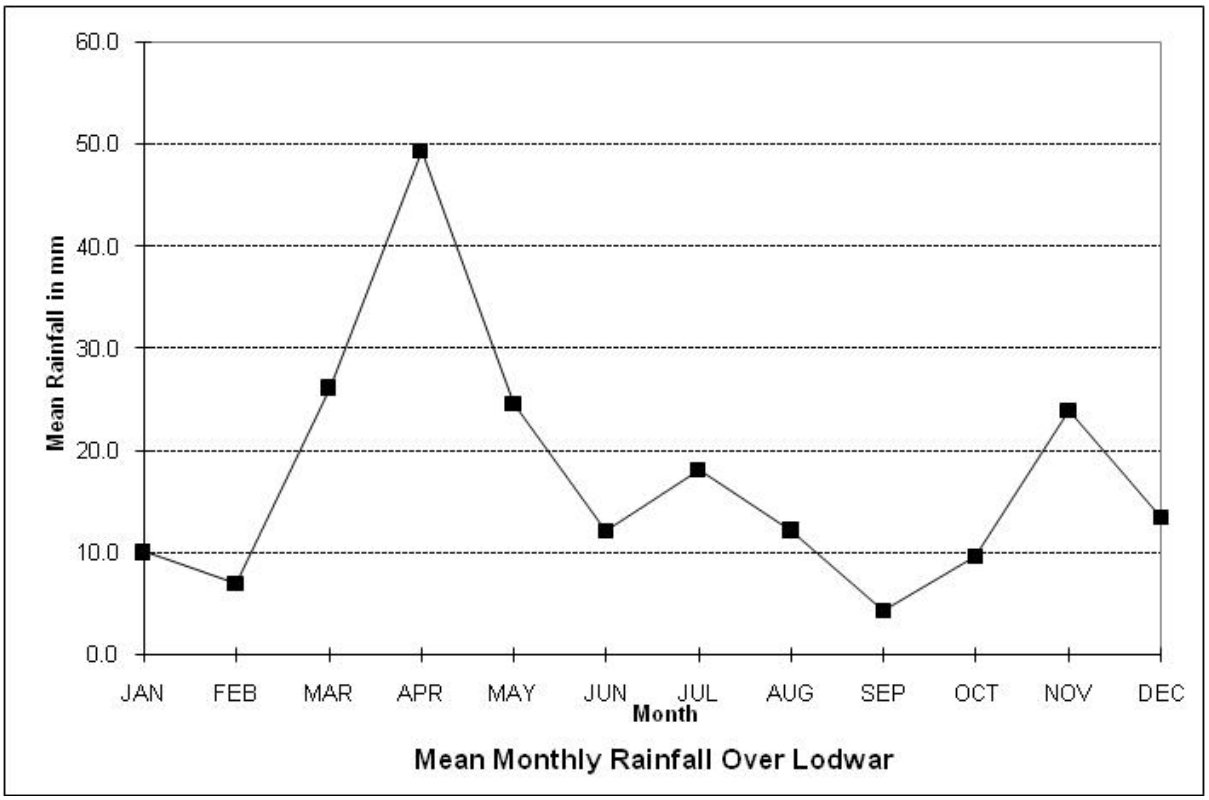


Figure 5.1: Mean Monthly Rainfall over Lodwar Showing MAM and OND Seasons (Savatia, 2011)

Four climatic seasons are identified in Turkana, though description of two key seasons is more prevalent; the wet season (*akiporo*) expected in April to June and dry season (*akamu*) expected in October to January. The other non-conspicuous seasons are early rainy showers (*Akicheres*) expected in February and March; and the end of wet season (*Ait*) expected in July to September. Temperatures range from a low of 24 ºC to a high of 38 ºC with a mean of 30 º C (ALRMP, 2009).

5.2.2 Sampling Unit and sample size

The sampling unit was an *Adakar* as described in section 3.2 of Chapter 4 titled. All the *Adakars* in study area were allocated numbers and using a random number generator (Microsoft Excel) the study sample of 142 *Adakars* (Hatcher, 1994; Kim, 2008; Pallant,

2005) was selected by simple random sampling proportionate to population size of each administrative division (Table 5.1). Sample sites were same as those shown in Figure 3.2.

Table 5.1: The sample size proportionate to households' population of each administrative division

Division	Location	Households	*Adakars*	Number of sampled *Adakars*
Loima		***5288***	***76***	***20***
	Loima	2550	36	6
	Lorengipi	974	14	6
	Lokiriama	1764	25	8
Oropoi		***8265***	***118***	***32***
	Letea	4957	71	19
	Kalobeyei	2577	37	12
	Loreng	731	10	1
Kakuma		***6060***	***87***	***23***
	Kakuma	2763	40	14
	Peleckech	1863	27	9
	Nakalale	1434	20	
Lokichoggio		***8505***	***121***	***33***
	Lokichoggio	554	8	1
	Songot	1077	15	3
	Lorao	1985	28	9
	Mogila	2194	31	11
	Nanam	1828	26	5
	Loteteleit	867	12	4
Kibish		***2935***	***42***	***11***
	Kibish	724	10	3
	Naita	360	5	2
	Natapar	1851	26	6
Kaaleng		***6392***	***91***	***24***
	Kaikor	1853	26	7
	Yapakuno	1738	25	6
	Loruth	847	12	4
	Kaeris	1954	28	6
Adakars			535	142

5.2.3 Data collection

5.2.3.1 Risk assessment questionnaire

The study primarily examined the complex interrelationship between various variables describing sheep and goat husbandry, related small stock production and socio-cultural activities in the pastoral set up of Turkana community that may lead to direct or indirect exposure of small stock to possible PPR infected animals or livestock herds. The Turkana herders identified the four age groups of the small stock based on age (young kids and lambs < 2 months, older kids and lambs >2 but < 5 months, young sheep and goats > 6 months but < 24 months and adults > 24 months). Through key informant interviews, it was established that each age received varied managerial attention in terms of herding care and disposal, based on socio-cultural significance of the age group. Therefore, variables for the risk assessment were developed for each of the four age groups looking at herding care, off take and restocking patterns for each age group. The questionnaire was developed into three sections with section one dwelling on informants' data, section two queried the PPR herd history in the study location and the third section explored on the possible PPR exposure variables (Appendix 9.3). The data collected on section two of the questionnaire was both qualitative and quantitative. The section three of risk assessment questionnaire consisted 62 variables and was developed as a Likert scale based on summated rating scale format as described by Spector (1992). The variables in the survey questionnaire were rated by scale of five items that were assigned risk scores. In all the frequency structured scales, a high score indicated high risk while in the agreement structured scales, high score indicated low risk. At analysis

level, all the agreement structured scales were reverse-coded so that high score depicted high risk (DeCoster, 2005). The questionnaire (Appendix 9.3) was pretested and relevant adjustment made prior to final study.

5.2.3.2 Focus group discussion

The participatory risk assessment entailed administration of the risk questionnaire orally to a small focused group of about five to 15 respondents, being representatives and key informants of each *Adakar* interviewed (Catley and Mariner 2002; Mariner and Paskin 2000; Grace *et al*., 2008). The scale items were translated in local Turkana language for ease of scoring the respondent responses. In all the frequency and agreement structured scales the translation was depicted and recorded on cards (section 5.2.3.2.1 and 5.2.3.2.2) that were used for interview:

5.2.3.2.1 Agreement scale

Kichamakin Kire	*Kichamakin*	*Nakidingos*	*Nyikichamakin*	*Kingeer*
Strongly agree	agree	neutral	disagree	strongly disagree.
1	2	3	4	5

5.2.3.2.2 Frequency scale

Maam	*Maam Cha*	*Achepak*	*Ngicherua*	*Jiik*
Never	seldom	sometimes	often	always
1	2	3	4	5

The interviewer with help of local Turkana language interpreter led a discussion on each question following which an agreed scoring was pointed out and recorded for each variable based on agreement reached between the respondents in their group discussion.

5.2.4 Data management and analysis

The data collected from the field was entered, cleaned and constructed in Microsoft Excel and Word (Microsoft Corp., Redmond, WA). The quantitative data was then exported to SPSS statistical software version 17.0 (IBM Corp., Armonk, NY) for descriptive statistics, exploratory factor analysis and logistic regression analysis. The qualitative data was recorded in Microsoft word and analyzed. The qualitative data provided for the Turkana description of annual seasons, their characteristics, livestock related activities undertaken during the seasons and corresponding months of the season. Descriptive analysis generated descriptive statistics of distribution of PPR disease outbreaks by administrative divisions and disaggregation analysis of PPR disease outbreaks in each season. Exploratory factor analysis using maximum likelihood method of common factor analysis was used for the extraction of the latent factors. Common factor analysis assumes all factors are related to some degree and that those that share same dimensions (latent factors) are highly correlated compared to those that do not share dimensions thus yielding low correlations (Basto and Pereira, 2012). Therefore, common factor analysis uncovers the latent factor structure of a set of variables and explains the correlations among the variables (Kim 2008; Basto and Pereira, 2012), The factorability of the variables was assessed by correlation matrix where some correlations had to be > 0.3 (Tabachnick & Fidell, 2007). Anti-image

correlation matrix diagonals were examined to ensure they were > 0.5 while anti-image correlation matrix diagonals that were < 0.5 were considered for exclusion from analysis (Field, 2009). Finally a measure of sampling adequacy Bartlett's test of sphericity (Bartlett, 1954) was checked for significant and Kaiser-Mayer Olkin (KMO) (Kaiser, 1970) measure of sampling adequacy checked to be > 0.6. The initial extracted factors were then rotated using orthogonal factor rotation (varimax rotation) so as to obtain results that had a simplified structure that was easier to interpret (Tabachnick and Fidell, 2007). Rotated factors with factor loading >0.40 were retained for interpretation (Berghaus *et al.,* 2005). In determining the number of factors to retain for interpretation, four criteria were used. First, retaining factors with eigen value greater >1 (Kaiser rule); second, identifying the break point (elbow) on graph plot of factors and eigen values and select factor above elbow (Scree method) (Cattell, 1966), third, parallel analysis based on Monte Carlo random simulated eigen values which form a criterion for comparison with actual eigen values from raw data (O'Connor, 2000). Raw data eigen values larger than criterion are retained in parallel analysis. The final and also important criterion is the selection of those factors whose interpretation based on variables in them makes sense (Boklund *et al.,* 2004). After selection of a final factor model, standardized factor scores with an approximately zero mean and unit variance were calculated for *Adakars* that were interviewed (DiStefano *et al.,* 2009). These scores were subsequently evaluated as predictors in a model-based logistic regression analysis to determine whether they were associated with the observation of PPR outbreaks during 2009 and 2010. Treating the management factor scores as

longitudinal repeated data by season univariate and multivariate logistic analyses was carried out with the presence or absence of PPR outbreaks as dependent variable. The data was further disaggregated by season in each study year and univariate and multivariate logistic analyses was carried out with the presence or absence of PPR outbreaks as dependent variable analyzed per season. In the univariate logistic analysis for each season a total of eight models were derived with p-value being $P \leq 0.1$, giving a total of 32 models per year. Variables that were significant at $P \leq 0.1$ were further analysed in a multivariate logistic analysis. Likelihood ratio test (LRT) was done to test the significance of a variable in a multivariable regression analysis. The LRT significance of a variable in the multivariable regression analysis was carried out using Wald test ($p<0.05$).

5.3 Results

5.3.1 Seasonal characteristics

Treating data as longitudinal data, the study covered 142 Adakars (villages) in 6 administrative divisions (Loima, Oropoi, Kakuma, Lokichoggio, Kaaling, and Kibish) of Turkana district. The data was collected for two years 2009 and 2010 and each year was divided into four seasons The Turkana community described four seasons in their local calendar year namely *Akicheres, Akiporo, Ait* and *Akamu* (Table 5.2). Each of these seasons had a binary outcome of PPR outbreak, i.e. whether it occurred or not. An outbreak was defined as an observation, by herders, of PPR clinical signs in several small ruminants (more than two) in a household herd (*awi*) or *Adakar* herd within the

study period. In each year studied there was 142 *Adakars* making observations for presence of PPR in each of the 4 seasons, thus, a total of 568 observations were made. Similarly in 2010 138 *Adakars* made the observations.

Table 5.2: Turkana description of season, months and their characteristics

Turkana Season	**English Equivalent of seasons**	**Turkana months in a season**	**Turkana Interpretation of the month**	**English Equivalent months**	**Seasonal climatic vegetation characteristics**
Akicheres	Early rains Season	*Lodung'e*	To put off; as of fire: The dry season ends	February	Very hot and dry, no pasture and water. First signs of long rains clouds. Some areas get early showers. Most small stock grazing on mountain ranges within *Adakar* herd
		Lomaruk	Cloud formation: Life comes back with formation of clouds	March	
Akiporo	Long Rains Season	*Titima*	Growth: Growth of grasses and greening of trees.	April	Long rains, plenty of green pasture and water, shrubs are green and seasonal rivers flowing with flood water. Small stock grazing on the plains dispersed as household *(awi)* herd.
		Yelyel	Flowering process: crops (millet) and plants flower	May	
		Lochoto	Mud/cow dung: The colours of grasses turn to dark green	June	
Ait	Start of Dry season Season	*Losuban*	Rituals: Ceremonies. Grass begins to whither.	July	Start of dry season. Water available in pans and sand dams. The pasture are maturing and drying into standing hay. Small stock in household (*awi*) herd graze along the river beds to access both pasture and water.
		Lotiak	To divide: Separation of rains and dry season.	August	
		Lolongu	Hunger starts to bite. Trees shed leaves	September	
Akamu	Dry season Season	*Lopo*	Cook wild foods	October	It's dry and hot with extreme high temperatures. Pastures and water in plains are depleted. Community mining water from dry river beds. Trees shed leaves and fruits Scare pastures and water available only on mountain ranges and river beds near mountains. Small stock grazed within *Adakar* herd.
		Lorara	Fall: Wild berries and pods drop	November	
		Lomuk	Cover. Shrubs may green due to short rain	December	
		Lokwang'	White: Bare rangeland	January	

5.3.2 Distribution of PPR outbreak observations by divisions

In 2009, 131 observed PPR outbreaks (23.1% [95% CI 19.9%, 26.7%]) were reported to have occurred throughout the four seasons. There was no significant difference among divisions. In 2010 there were 133 (23.4% [95% CI 20.1%, 27.1%]) observed PPR outbreaks that were reported to have occurred throughout the four seasons; however, there was no significant difference among divisions. Therefore it was noted that distribution of the reported observed disease outbreaks among the divisions was similar within each of the two years (Table 5.3).

Table 5.3: Distribution of PPR outbreak observations by divisions in 2009 and 2010

	Outbreak present		Total
Division	n	%	n
2009			
Kaaleng	32	24.2	132
Kakuma	30	23.4	128
Kibish	17	25.0	68
Loima	23	24.0	96
Loki	18	18.0	100
Oropoi	11	25.0	43
Total	131	23.1	568
2010			
Kaaleng	34	25.8	132
Kakuma	32	25.0	128
Kibish	17	25.0	68
Loima	22	22.9	96
Loki	17	17.0	100
Oropoi	11	25.0	44
Total	133	23.4	568

5.3.3 Disaggregation analysis of PPR outbreaks in each season

When occurrences were compared per season, there was significant difference in outcomes between seasons in each division for five divisions, in year 2009 (Table 5.4). Most outbreaks were reported in dry season (*Akamu*) followed by wet season (*Akiporo*). The first early season (*Akicheres*) recorded the lowest outbreak. For year 2010, there was also significant difference in outcomes between seasons in all divisions (Table 5.5). Outbreaks were more likely to be reported in season 4 (*Akamu* or dry season) for 2010 in all divisions.

Table 5.4: PPR disease outbreak disaggregated by season in 2009

Division	N	*Akicheres* **Early Rains** n	*Akiporo* **Long rains** n	*Ait* **Start of dry season** n	*Akamu* **Dry season** n
Kaaleng***	33	1 (3.0%)	8 (24.2%)	4 (12.1%)	19 (57.6%
Kakuma***	32	0 (0%)	6 (18.8%)	5 (15.6%)	19 (59.4%)
Kibish***	17	0 (0%)	9(52.9%)	0 (0%)	8 (47.1%)
Loima***	24	3 (12.5%)	1(4.2%)	5 (20.8%)	14 (58.3%)
Loki	25	2 (8.0%)	5 (20.0%)	3 (12.0%)	8 (32.0%)
Oropoi**	11	0 (0%)	2 (18.2%)	3 (27.3%)	6 (54.5%)
Total	142	6 (4.0%)	31 (21.2%)	20 (14.1%)	74 (52.1%)

N=Total reported observations; n = the number of observations reported as outbreaks in a season; *** P≤0.001; ** P≤0.05; *P≤0.1

Table 5.5: PPR disease outbreak disaggregated by season in 2010

		Akicheres **Early Rains**	*Akiporo* **Long rains**	*Ait* **Start of dry season;**	*Akamu* **Dry season**
Division	N	n	n	n	n
Kaaleng***	33	0 (0%)	7 (21.2%)	1 (3.0%)	26 (78.8%)
Kakuma***	32	1 (3.1%)	2 (6.3%)	2 (6.3%)	27 (84.4%)
Kibish***	17	1 (5.9%)	5 (29.4%)	0 (0%)	11 (64.7%)
Loima***	24	0 (0%)	1 (4.2%)	2 (8.3%)	19 (79.2%)
Loki***	25	1 (4.0%)	1 (4.0%)	1 (4.0%)	14 (56.0%)
Oropoi**	11	1 (9.1%)	2 (18.2%)	1 (9.1%)	7 (63.6%
Total	142	4 (2.8%)	18 (12.7%)	7 (5.0%)	104 (73.2%)

N=Total reported observations; n = the number of observations of reported as outbreaks in seasons; *** P≤0.001; ** P≤0.05; *P≤0.1

5.3.4 Factor analysis of small ruminants' pastoral management practices in Turkana

Of the 62 variables entered in the factor analysis model 49 were retained and were further reduced into seven factors that were established as major small ruminants' pastoral management practices (Table 5.7).

Table 5.7: The seven factors extracted from 49 variables of PPR risk assessment

Factor variables		Factor 1	Factor 2	Factor 3	Factor 4	Factor 5	Factor 6	Factor 7
Factor 1: Indiscriminate mixing of vulnerable group with high risk group within herds								
Q3.25	Sick adults stock watered on same troughs with older kids/lambs	.860						
Q3.23	Older kids/lambs share the same watering troughs with older animals	.718						
Q3.18	Older kids/lambs graze alongside wild herbivores.	.708						
Q3.38	Extent of watering young goats/sheep at separate water holes	-.684		.446				
Q3.41	Sick young goats/sheep watered in communal water holes	.657						
Q3.26	Sick adult stocks grazed along with older kids/lambs	.625						
Q3.36	Frequency young sheep and goats graze along with wild herbivores	.609						
Q3.40	Sick young sheep/goats separated from other	-.526						
Q3.62	Traders graze their animals alongside herds on their way to the markets	.468						
Q3.39	Young goats/sheep share the same watering troughs with older animals	.447						
Factor 2: Introduction of new animal into the herds								
Q3.44	Frequency of young goats/sheep returned home after failed market sale		.873					
Q3.61	Frequency of adult goats/sheep returned home from failed market sale		.769					
Q3.47	Extent of goats/sheep sourced from markets used to restock herds		.650					
Q3.29	Extent of young goats/sheep bought from markets used to restock herds		.570					
Q3.45	Extent of introduction into herds goat/sheep gifts from ceremonies		.538					
Q3.30	Extent of young goats/sheep got through raids used to restock herds		.520			.469		
Q3.13	Extent of kids/lambs bought from markets used to restock herds		.502	-.405				
Q3.33	Young goats/sheep grazed in common pasture		-.465		-.445			
Q3.48	Extent of adult goats/sheep got from raids used to restock herds		.465					
Factor 3: Share watering source leading concentration of young stock in one point								
Q3.21	Extent of watering older kids/lambs at separate times			.681				
Q3.7	Extent of watering young kids/lambs at separate times from other stock			.582				
Q3.22	Extent of watering older kids/lambs at separate water holes	-.436		.544				
Q3.37	Extent of watering young goats/sheep at separate times			.528				
Q3.8	Extent of watering young kids/lambs at separate water holes			.506				
Q3.14	Extent of older kids/lambs got through raids used to restock herds			-.478				
Factor 4: Foreign stock from across international borders grazing in local pastures								
Q3.53	Extent of herds from neighboring countries graze in local pastures				.923			
Q3.54	Extent of herds from neighboring countries watering in local pastures				.871			
Factor 5: Restocking through raids								
Q3.43	Frequency of young goats/sheep lost through raids returned back					.830		
Q3.60	Extent of young goats/sheep got through raids used to restock herds					.686		
Q3.5	Young kids/lambs moved with other animals during transhumance					.582		
Q3.19	Older Kids/lambs moved with other animals during transhumance					.436		
Factor 6: Local culture of borrowing and loaning of livestock								
Q3.28	Extent of exchange of young goats/sheep on loans						.665	
Q3.46	Frequency of exchange of adult goats/sheep/ on loan						.615	
Factor 7: Sick dams left to nurse their young kids/lambs								

Q3.11	Sick adult stock are watered on same troughs with young kids/lambs							.887
Q3.9	Young kids/lambs share the same watering troughs with older animals							.476
Q3.12	Sick adults stocks are grazed along with young kids/lambs				-.408			.444

Based on the variables in each factor and considering the variables with heavy loading on the factor a description representing the general theme of the factor was generated. The list of factors with their descriptive theme titles in the final factor model are as listed below.

- Factor 1 Indiscriminate mixing of vulnerable group with high risk group within herds (Indiscriminate herd mixing)
- Factor 2 Introduction of new animal into the herds (introducing new animals in herds)
- Factor 3 Share watering source leading concentration of young stock in one point (Share water points)
- Factor 4 Foreign stock from across international borders grazing in local pastures (Share grazing with foreign animals)
- Factor 5 Restocking through raids (Raids)
- Factor 6 Local culture of borrowing and loaning of livestock (Loaned animals)
- Factor 7 Sick dams left to nurse their young kids/lambs (Sick dams nursing)

Final analysis based on observation of factors and what made biological sense confirmed the selection of seven factors that accounted for 45.3% of the variance. Most variables loaded highly on single factor in a simplified structure that was easier to interpret as shown in Table 5.7, though there were five variables Q 3.12, Q3.13, Q3.22, Q3.30, Q3.33 and Q3.38 which loaded on more than one factor each. The

double loading of variables was an indication that these variables were moderately related to both factors (Berghaus et al., 2005). It is also observed that variables Q3.22, Q3.38 and Q3.40 load negatively on factor one. Assessment of Q3.22 and Q3.38 "Extent of watering older kids and lambs at separate water holes" and "Extent of watering young goats and sheep at separate water holes" shows that if these actions happened, there would be reduced contacts and possibly reduced risk thus the negative loading, while the opposite would have been "watering goats in common hole", thus increasing contacts and risk. Similarly Q 3.40 "Sick young sheep and goats are separated from other stock" would reduce the risk, thus negative loading. Other variables loading negatively were Q3.33 on factor two, Q3.13 and Q3.14 on factor 3, Q3.12 and Q3.33on factor 4 but variable loadings were low and their description did not fit the general theme of the factors they loaded on.

5.3.5 Analysis of small ruminants' pastoral management factors as predictors of PPR outbreaks

Treating the management factor scores as longitudinal repeated data by season, a univariate logistic regression analysis, was carried out to check the association between outcome (observed PPR outbreaks occurrence or not) on one hand and the factors, season and divisions on the other. For the two years studied, the result showed that there was no management factors (Indiscriminate herd mixing, Introduction new animals in herds, Sharing water points, Sharing of pasture with foreign animals, Raids, Loaned animals and Sick nursing dams) that was significantly associated with outcome (Tables 5.8 and 5.9). In addition, accounting for correlation of responses by division showed that inclusion of division random

effect did not provide a better fit than the standard logistic regression (p=1). However seasons were significantly associated with the outcome.

Table 5.8: Logistic regression analysis of repeated 2009 data by season

Variable	**Odds Ratio**	**[95% Conf.Interval]**	**P-value**
Indiscriminate herd mixing	0.94	[0.76, 1.15]	0.55
Introduction new animals in herds	1.04	[0.85, 1.29]	0.66
Sharing water points	1.04	[0.85, 1.29]	0.65
Sharing of pasture with foreign animals	0.98	[0.81,1.20]	0.90
Raids	1.01	[0.82, 1.24]	0.91
Loaned animals	1.00	[0.80, 1.24]	0.98
Sick nursing dams	1.01	[0.82, 1.24]	0.88
Wet season	6.33	[2.5,15.7]	0.000
Start dry season	3.71	[1.4, 9.5]	0.006
Dry Season	24.66	[10.2, 59.5]	0.000
Kakuma	0.95	[0.54, 1.69]	0.87
Kibish	1.04	[0.52, 2.05]	0.90
Loima	0.98	[0.53, 1.82]	0.96
Loki	0.68	[0.35, 1.31]	0.25
Oropoi	1.04	[0.47, 2.29]	0.91

Start of wet season is reference season; Kaaleng is the reference division

Table 5.9: Logistic regression analysis of repeated 2010 data by season

Variable	Odds Ratio	[95% Conf.Interval]	P-value
Indiscriminate herd mixing	0.938	[0.534,1.147]	0.534
Introduction new animals in herds	1.068	[0.867,1.316]	0.536
Sharing water points	1.047	[0.850,1.289]	0.665
Sharing of pasture with foreign animals	0.999	[0.821, 1.216]	0.991
Raids	1.035	[0.840, 1.274]	0.749
Loaned animals	0.959	[0.771, 1.191]	0.703
Sick nursing dams	1.001	[0.816, 1.229]	0.99
Wet season	5.008	[1.65,15.200]	0.004
Start dry season	1.789	[0.512,6.251]	0.362
Dry Season	94.421	[32.672,272.872]	0.000
Kakuma	0.961	[0.549,1.680]	0.888
Kibish	0.961	[0.49,1.884]	0.907
Loima	0.857	[0.463,1.586]	0.623
Loki	0.59	[0.308,1.133]	0.113
Oropoi	0.961	[0.438,2.109]	0.921

Start of wet season is reference season; Kaaleng is the reference division

Accounting for correlation of responses by division: The likelihood ratio test (P=1) showed that inclusion of division random effect did not provide a better fit than the standard logistic regression.

Standard univariate and multivariate logistic regression with data disaggregated by season was carried out. Results from univariate logistic analysis for year 2009 is shown in Table 5.10 restocking through raids and share watering source are positively associated with observing PPR outbreaks in start of wet seasons and wet season respectively. Indiscriminate herd mixing, Kakuma, Loima and Loki divisions were inversely related to the outcome in wet season. Share watering source and division Loki were also inversely related to outcome in dry season.

Table 5.10: Univariate logistic analysis (P≤0.1) of 2009 data

Season	**Variable**	**Odds Ratio**	**[95% Conf.Interval]**	**P-value**
1	Raids	3.515	[1.084, 11.395]	.036
2	Indiscriminate herd mixing	.651	[.436, .973]	.036
2	Sharing water points	2.022	[1.308, 3.126]	.002
4	Sharing water points	.681	[.470, .987]	.042
2	Kakuma	.218	[.042, 1.140]	.071
2	Loima	.161	[.018, 1.413]	.099
2	Loki	.155	[.018, 1.352]	.092
4	Loki	.343	[.109, 1.081]	.068

* Division of reference was Kaaleng (division arranged alphabetically)

Table 5.11 shows the results of univariate logistic regression analysis for year 2010. Loaned animals was positively associated with outcome in start of wet season while sick dam nursing and division Kibish were also positively associated with outcome in

wet season. Division Loima was inversely related to outcome in wet season. Loaned animals was inversely associated with outcome in start of dry season while sick nursing dams, indiscriminate herd mixing and division Loki were inversely related to outcome in dry season.

Table 5.11: Univariate logistic analysis (P≤0.1) for 2010 data

Season	Variable	Odds Ratio	[95% Conf.Interval]	P-value
1	Loaned animals	4.204	[1.072, 16.476]	.039
2	Sick nursing dams	1.621	[1.027, 2.559]	.038
3	Loaned animals	.277	[.101, .763]	.013
4	Sick nursing dams	.656	[.451, .955]	.028
4	Indiscriminate herd mixing	.692	[.450, 1.064]	.093
2	Kibish	3.516	[1.016, 12.165]	.047
2	Loima	.136	[.016, 1.172]	.069
4	Loki	.347	[.117, 1.029]	.056

* Division of reference was Kaaleng

In the final multivariate logistic analysis with P-value set at ≤0.05 for 2009 reports, sharing water points was positively associated with PPR outbreaks in wet season. However the same sharing of water points was inversely associated with PPR outbreak in dry season, as shown in Table 5.12. It was also noted that factors significant in the univariate analysis such as raids in start of wet season and indiscriminate herd mixing in wet season became insignificantly associated with PPR outbreak in the multivariate logistic model.

Table 5.12: Final multivariate logistic regression analysis for 2009 data

Season	Variable	Odds Ratio	SE	[95% Conf.Interval]	P-value
2	Sharing of water points	2.022	0.45	[1.308 3.126]	0.0005
LRT P=0.0011 for season 2					
4	Sharing of water points	0.681	0.129	[.470, .987]	.0321
LRT P=0.04 for season 4					

Table 5.13 shows the final multivariate logistic analysis results for year 2010. Sick nursing dams were positively associated with PPR outbreaks in wet season. Kibish division was positively associated with PPR outbreak in dry season. Indiscriminate herd mixing, loaned animals, Kakuma, Loima, Loki, Oropoi divisions were inversely associated with PPR outbreaks in dry season . Factors that were significant in the univariate logistic models but were dropped off in multivariate logistic model were loaned animals, start of wet seasons and start of dry seasons; indiscriminate herd mixing and sick nursing dams in dry season.

Table 5.13: Final multivariate logistic regression analysis for 2010 data

Season	Variable	Odds Ratio	SE	[95% Conf.Interval]	P-value
2	Sick nursing dams	1.621	0.377	[1.027, 2.559]	.049
LRT P=0.04 for season 2					
4	Indiscriminate herd mixing	.529	0.138	[.318, .882]	.020
4	Sick nursing dams	.662	0.142	[.434, 1.010]	.045
4	Kakuma	0.253	0.225	[.044, 1.449]	
4	Kibish	1.018	0.746	[.242, 4.282]	
4	Loima	0.162	0.133	[.032, .816]	
4	Loki	0.608	0.436	[.149, 2.482]	
4	Oropoi	0.186	0.131	[.046, .744]	
LRT P=0.04 for season 4					

5.4 Discussion

The seven factors extracted as potential risk variables for PPR are assessed in relation to pastoral livestock management as practiced by the Turkana pastoral herders. Turkana community livelihood is hinged on pastoralism practiced in some of the harshest and most arid land of Northwest Kenya (Weinpahl, 1985). Turkana pastoralists are thus a very mobile community in search of pasture and water for their livestock. Decisions relating to livestock management in Turkana are made based on labor

availability, herd size, social obligations and perceptions of the environment in terms of security, forage and water availability (McCabe, 1984). In examining the factors in the model, indiscriminate herd mixing is strongly loaded by variables highlighting the most vulnerable groups of older kids and lambs; young sheep and goats making contacts with high risk groups such as wildlife and sick adults through sharing of water holes, troughs and grazing. The indiscriminate mixing of sick animals and wildlife during grazing poses a health risk to the herd. The deliberate decision of the pastoral Turkana herder to allow sick animals to mingle with healthy ones can be explained from the point of labor shortage; households with abundant labor segregate their herds of livestock into herding groups based on species, age, production status and health status (Ohta 1982). Wildlife such as dikdik (*Madoqua guentheri*) is common in Turkana dry land savannah and graze along with small stock in the savannah shrubs; they could thus pose as a risk considering similar wild small ruminants, grey duiker (*sylvicrapa grimmia*) have been reported with PPR sero-positivity (Ogunsanmi *et al.,* 2003). Introduction of new animals in the herd strongly loaded on variables highlighting introduction of new animals into a herd. Such introduction may come from market purchases, gift from cultural ceremonies and raids (de Vries, 2002). Variables depicting unsold small stock returned home from market sale yard loaded highly on introduction of new animals in the herd. At the market sale yard, animals from various locations are concentrated in closed pens therefore creating a high risk environment for contracting of diseases; these diseases can be spread to herds where bought animals are destined to go. Source of animal gifts, as well as raids, may be herds that were infected. It is told that, in last

serious PPR outbreak in Turkana in 2006, on realizing the immense danger they faced of losing their small stock through PPR disease, the pastoral herders rushed to settle their socio-cultural obligations that required giving out of small stock to other clan members. This action aided in increasing the risk of PPR spread to other herds. Sharing water points loaded on variables that highlighted on watering of the younger groups of sheep and goats. Water is a scarce commodity in Turkana and a single water source may serve several herds during dry season. In situations where water and labor are abundant, animals of different age groups are watered at different times and places; the very young kids and lambs being watered at home. However, in the very dry seasons, all animals may be seen crowding in a single water hole waiting their turn to drink, consequently increasing possibility of making infective contacts. Similarly, during wet season, the small stock will drink indiscriminately from the puddles scattered in the plains. Several herds may share common puddles particularly along the herding pathways. Sharing pasture with foreign animals loaded highly on variables mentioning invasion of local pastures by foreign herds from across international borders. During the severe drought, even the communities who are adversaries will grant each other passage to pasture and water. It is at this time that pastoralists will cross international borders in search of pasture and water. Turkana community expressed their concern that foreign animals brought disease into their pastures. Such concerns were based on the fact that the Turkana community had little knowledge of whether the visiting foreign animals had received adequate protective animal health care before getting into their pasture. Raids had high correlations on livestock raids and transhumance focusing

on older lambs and kids as well as young sheep and goats. As previously mentioned Turkana are pastoralists and thus are very mobile in search for pasture and water to sustain their livestock (Fry and McCabe, 1986). It is this mobility that exposes their livestock to share pastures with herds that could be exposed to diseases. Livestock raids are common cultural activities among the Turkana and the neighboring communities. Despite the perceived gains from raiding, Turkana herders avert that raided animals are also known to spread disease to herds they end up to. Loaned animals was correlated with loaning of livestock. In Turkana begging livestock is an accepted normal and people negotiate to be given animals by their clansmen or age mates (Renfrew 1990; Sakumichi 1997; de Vries, 2002). In such circumstances animals given out are those that are of less benefit to the owner. Therefore it is high risk to borrow animals from a sickly herd because the owner will readily hand them over to borrower who will end up with liability of paying back whether the animals survive or not. Sick nursing dams, key variables loading on the factor have general theme of sick adult goats and sheep sharing grazing and water with kids and lambs. From the interviews, it was emphasized that sick livestock were left to graze with kids and lambs around the homestead; this included sick dams that were allowed to continue nursing their young ones.

From the assessment of results of the factor analysis model it was found that the seven factors describe some of the livestock management decisions made by Turkana herders at household level and *Adakar* level in their management of small ruminants. The management decisions were made in response to constraints experienced by the

herders, such as labor shortage, pasture and water availability, socio-cultural obligations, herd health, need to expand herd size and prevailing security situations. To overcome these constraints Turkana herders have developed strategies which constitute, among others, the seven management factors extracted from factor analysis model.

The results of initial logistic regression model of repeated data in both years 2009 and 2010 (Tables 5.8 and 5.9) had identified only seasons as risk factors for PPR outbreak. It was observed that PPR was 24.66 times more likely to occur in or dry season (*Akamu*) of 2009 relative to start of rains season (*Akicheres*), whereas PPR was 94.42 times more likely to occur in dry season of 2010 relative to start of the rains season in 2010. The wet season (*Akiporo*) in both years 2009 and 2010 was the second riskiest period that PPR outbreaks were predicted would occur relative to start of wet season. Start of dry season (*ait*) was only significantly risky for year 2009. These outcomes are consistent with seasonal reports made by respondents during the interviews.

Raids in start of wet season and sharing of water point in wet season of 2009 were significantly and positively associated with PPR outbreak in the univariate model. Variables that loaded highly on raids as a factor such as raids, transhumance and nomadism, are known as major risk factors in spread of infectious diseases including PPR. Loaned animals in start of wet season, sick nursing dams in wet season and Kibish division in wet season were significantly and positively associated with PPR outbreak in 2010 in a univariate model. Loaning of animals as a factor had variables that described dominant Turkana culture of borrowing and loaning of animals which to

some extent is also blamed for disease spread between herds. All the significant factors and divisions in univariate logistic analysis were further analyzed in multivariate logistic analysis with subsequent rendering of raids in 2009 and loaning of animals in 2010 insignificant to association with PPR outbreak.

In the final multivariate logistic regression model where data was disaggregate into seasons the management factors were evaluated and the factor found to be significantly associated PPR outbreak in 2009 was share watering source leading concentration of young stock in one point. The significant factors in 2010 were Sick dams left to nurse their young kids/lambs and divisions.

Factor scores for sharing water points in wet season (*akiporo*) of 2009 was significantly higher for *Adakars* that observed PPR. During the wet season, the small ruminants migrate from the mountains to the plains since there is abundant fresh grass and water puddles everywhere. The small ruminants are unrestricted in their movements and all age groups allowed to graze together since the movement within the pastures are close to the homesteads. During these periods other *Adakar* herds may share the same grazing pastures in the plains necessitating the transient mixing of herds (Ohta, 1982). Mixing of herds, particularly during watering, and sharing of watering sources lead to concentration of young stock at one point (risk factor sharing water points) and was found to have an odds ratio of 2.022, meaning that the odds of having PPR outbreaks during wet season (*Akiporo*) of 2009 increased by 2.022 times for every unit increase (1 standard deviation) in the factor sharing water points score.

On the other hand, scores for sharing pasture with foreign animals in dry season (*Akamu*) of 2009 was significantly lower in *Adakars* that reported PPR outbreaks. During dry season the pastures and water availability in the plains is diminished forcing *Adakar* herds to migrate from the plains to the mountain ranges or to the neighboring countries. The drought in 2009 was severe and thus no foreign herds could have been attracted as there were no pastures to share within Turkana plains and mountains (ALRMP 2009a; Zwaagstra *et al.,* 2010). Foreign stock from across international borders grazing in local pastures had an odds ratio of 0.681, meaning that the odds of having PPR outbreak in dry season (*Akamu*) of 2009 decreased by 0.319 times for every increase in (1 standard deviation) in the sharing of pasture factor score.

In wet season (*Akiporo*) of 2010, the factor scores for sick nursing mothers were significantly higher for *Adakars* that reported PPR outbreaks. During wet season, small stock grazes close to homestead and there is general mixing of all groups and sometimes herds. As discussed earlier, sick mothers are allowed to nurse their young ones due to lack of alternative management, occasioned by lack of labor. Kids and lambs, born by immunologically naïve mothers, and older lambs and kids that are above five months may not have maternal immunity or could have lost protective maternal antibodies against PPR; they are thus at risk when they nurse from their sick mothers or graze along with other sick adults. Therefore, sick dams left to nurse their young kids/lambs had an odds ratio of 1.621; meaning that the odds of having PPR

outbreak in wet season (*Akiporo*) of 2010 increased by 1.621 for every unit increase in sick nursing dam factor score.

Indiscriminate herd mixing and sick nursing dams in dry season (*Akamu)* of 2010 demonstrated an inverse association for outcome of PPR outbreak. Indiscriminate mixing of vulnerable group with high risk group within herds had odds ratio of 0.529 while sick dams left to nurse their young kids/lambs had an odd ratio of 0.662. The odds of PPR outbreak in dry season decreases by 0.471 for every increase in factor score of indiscriminate herd mixing. Similarly the odds of PPR outbreak in dry season decreased by 0.338 for every increase in factor score of sick nursing dams. Consistently, the indiscriminate mixing of livestock is common during wet season when the animals are in the plains and there is less competition for pasture and water. During the dry season the animals are herded into *Adakar* herds which are more likely to keep by themselves, away from others, as competition for pasture and water intensifies. As the dry season becomes intense most of the new born kids and lambs do not survive and even the older kids and lambs succumb earlier to vagaries of drought. Such situations were witnessed from August 2010 to December 2010 (ALRMP, 2010).

It is inexplicable that some of the management practices that would have been expected to increase risk were not captured by the final logistic regression model. Therefore, several of the factors and individual variables that we evaluated were not significantly associated with the observation of PPR outbreaks; this does not necessarily imply that they are not important in the transmission of PPR. Factors associated with the

observation of clinical cases during outbreaks may be quite different from those associated with the prevalence of infection. As previously noted the PPR disease is relatively new in Kenya and to the Turkana pastoralists. The clearest picture of the PPR disease the Turkana pastoralist can describe is that of dramatic epidemic that killed their sheep and goats in 2006 and 2007. However as the disease became endemic, such dramatic PPR outbreaks affecting both species and all ages of small ruminants may not appear again. This would mean that there are possibilities of misreporting of the disease as it is likely to present a different epidemiological picture from their original experience with the disease.

It was also observed that similar factors were not significant in the same season among the years. Thus it cannot be generalised that significant factors in one year are similar in other similar seasons of other years. This temporal heterogeneity of significant factors can be explained from the fact that, though the cultural practices of Turkana may be consistent overtime; these cultural practices may be practised in varied geographical places. Turkana community is highly mobile in search of pasture, water and security for their livestock. Thus, in process of herding their animals, the Turkana community interact with various other communities and environments. It is also observed that in 2010, spatial factors –i.e. establishment of the divisions - become a significant factor in PPR outbreak. Though spatial heterogeneity was not fully investigated, it can be deduced that it plays critical factor in evolvement of temporal changes of key risk factors to PPR. It thus follows that understanding the spatial temporal heterogeneity of

significant factors associated with PPR outbreak has significant impact on the understanding of the PPR disease spread and the disease control measures.

5.5 Conclusion

it has been observed that seasons, geographical locations and some seasonal livestock management activities are risk factors to PPR disease out breaks. However, it cannot be generalised that risk factors in one year are similar in other similar seasons of other years considering livestock are in constant move in search of pasture and water. Understanding this spatial-temporal heterogeneity of risk factors will greatly improve design of disease control measures against PPR. However further studies in the neighboring pastoral communities need to carried out to elucidate more on the social ecology of PPR.

CHAPTER 6: SOCIO-ECONOMIC IMPACT OF PESTE DES PETITS RUMINANTS IN TURKANA COUNTY

6.1 Introduction

Peste des petits ruminants occur as epidemics that can cause mortality rates as high as 100% in immunologically naive sheep and goat populations resulting in significant negative impact to the economies in the inter-tropical regions of Africa, in the Arabian Peninsula, the Middle East and Asia (Munir *et al.,* 2013).

At the height of PPR outbreaks in Kenya, estimates were made as regards the losses the pastoral communities had incurred from mortalities of sheep and goats occasioned by PPR. The Government of Kenya estimated the annual losses due to PPR to be over US$ 15 million (GOK, 2008; Nyamwea *et al.,* 2009). It was estimated that 16.1 million small stocks in arid and semi arid areas of Kenya were at risk of infection with PPRV. These small ruminant flocks contribute significantly to the main source of livelihood for the majority of the rural population and especially in the arid and semi-arid (ASALs) districts (GOK, 2008). The PPR outbreaks in Turkana had devastating effects on the Turkana community livelihood following the high mortalities in small stock. A study carried out in Turkana by FAO (2012a) found that PPR infection resulted in more households slipping into poverty, while the poor and very poor became destitute. The estimated livestock asset losses due to PPR ranged from 65 to 100% within the

traditional wealth categories resulting in losses in livestock income to the tune of 21 to 99% (FAO 2012a).

This chapter examines the socio economic impacts of *Peste des petit ruminants* and presents a model of socio economic losses occasioned by the disease in Turkana Kenya.

6.2 Material and Methods

6.2.1 Study area

The study location is Turkana County that borders internationally with Ethiopia, Sudan and Uganda and internally borders Marsabit, Samburu, West Pokot and Baringo Counties. The area has previously been described and depicted in section 3.2.1 and Figure 3.1 of Chapter 3 and section 5.2.1 of Chapter 5.

6.2.1.1 Turkana pastoral livelihood economy

Turkana people belong to the Ateker group of the Eastern Nilotes and are nomadic pastoralists occupying the Turkana County (McCabe 1996). The human population of Turkana County is 849,277 with approximately 93% of the populations being nomadic or semi-nomadic pastoralists deriving their livelihood from extensive livestock production (Imana, 2008; KNBS, 2010).

Livelihood patterns identified in Turkana County are pastoralism, agro pastoralism, fisheries and; formal/informal employment and trade. Within the study area key livelihood patterns are pastoralism and; partly formal/informal employment and trade in Lokichoggio and Kakuma divisions in Figure 3.1 The Turkana depend on five species

of livestock, camels, cattle, donkey, sheep, and goats for their subsistence as primary assets and income source. The donkeys are also used as food animals alongside transportation of household goods during migrations. Turkana live exclusively from the products of their livestock including milk, meat, blood skin and dung. The key animals used as meat source are sheep and goats. However, during major cultural ceremonies cattle, camel and donkeys are slaughtered (Ohta, 2007). Since cattle are always in the mountainous highland pastures away from access to most households in the plains, most milk for consumption in the plains is sourced from camels, sheep and goats (Degen, 2007). Some of the livestock are sold in local markets and money acquired from these sales is used for purchase of household items. Sheep and goats are the most sold livestock and the income used for food and consumer items such as maize meal, beans, sugar, tobacco, tea leaves rubber tire sandals, beads and cloth.

Turkana is a polygamous society and largely patriarchal. Bride-price is unusually high among the Turkana; a typical bride-price payment might include 30 to 60 large stock, and 100 to 200 small stocks (Boinski and Garber, 2000; Ohta, 2007). There is no other events in which so many animals are given out in bulk like the payment of bride price. This indirectly implies that a man may not marry until his father has died and he has inherited livestock (McCabe, 1996). The high bride-price also requires that the prospective groom collect livestock from all his relatives and friends, thus reinforcing social ties through the transfer of livestock (McCabe, 1996; Ohta, 2007). Incase animals are scarce, the prospective groom may result to livestock raids to raise requisite number of animals needed for bride price (Finke, 2003; Okumu, 2013).

The Turkana live in small households consisting a man, his wives, their children and possibly some dependent women. Household size varies considerably according to wealth, but averages about 20-25 people (McCabe, 1984). This basic household unit of Turkana social organization is called *awi*. Most herd owners live and travel with two to five other herd owners and their households, forming what is referred to as an *awi apolon*, or large *awi*. The composition of this unit changes frequently, as individuals and families leave to join other homesteads, or others come to join the *awi apolon*. During the wet season and insecure situations, many homesteads congregate into temporary associations *Adakar*. (McCabe, 1996). An *Adakar* entails a cluster of often-related Turkana households that pursue similar socio-economic activities such as search for pasture, water and security, under a trusted leader (Bett *et al.,* 2009).

Within a household (*awi*) all livestock are owned by male head of the household but within the *awi* they are allocated to women (wives) depending on number of children to be fed (Ohta, 2007). Sheep, goats and camels are commonly grazed in the plains while cattle are grazed on the mountainous ranges where there is plenty of grass in most seasons. Decisions relating to livestock management are made by men though level of consultation on decision making issues by both women and men at household level is high (Omolo, 2010). The climate imposes constraints on the available options of livestock management and due to these constraints the Turkana have developed highly flexible survival strategies that include mobility, herd splitting, re-distribution of surplus livestock within networks, livestock loans and gifts; livestock raids and reliance

on relief foods (Weinpahl 1985; Peperkamp and Remie 1989; Renfrew 1990; de Vries 2002; Omolo, 2010).

Livestock wealth among other factors such as age, wisdom, and oratorical skill is a major contributor to gaining political influence within the Turkana. Turkana social organization is based on territorial rights to pasture and water; as well as kinship, relationships among individuals, and rights in livestock and labor (McCabe 1996). In the recent times the Turkana political influence and social organization has come under undue and severe pressure from various natural and man-made factors. Natural disasters such as floods, droughts, famine; infectious livestock and human diseases have become cyclic in occurrence affecting the resilience of Turkana people. Worse still the man-made disasters such as vicious livestock raids, tribal conflicts, high population pressure from settled refugee camps and environmental degradation make Turkana County one of the harshest places to live in. The consequence of compromised Turkana political and social organization is dropping out of pastoralism and thus seeking sedentalization in mushrooming urban centers. The Turkana pastoralist dropouts have limited livelihood options and end up being destitute. However, some who are able bodied mainly rely on petty trade, manual labor, borrowing and relief hand outs. Wealth ranking in Turkana which is based on livestock and family size depicts a picture of the rich, middle rich, poor and very poor categories (Arasio 2004; OXFAM, 2006; FAO, 2012a)..

6.2.1.2 Livestock population

Small ruminants' population is 9,512,012 with 3,517,151 sheep and 5,994,861 goats and constitutes 75.5% of livestock in Turkana County. The study area of the six divisions has a population of 3,288,438 small ruminants with 2, 020, 456 goats and 1,267,987 sheep (KNBS 2010).

6.2.2 Sampling unit and selection of study sites

The sampling unit was an *Adakar* as described in section 3.2.2 of Chapter 3 titled "Community appraisal of *Peste des petits ruminants* and validation of the community knowledge in Turkana County". The study sites were well distributed in the six administrative divisions that were a focus for the study as shown in figure 3.2.

6.2.3 Data collection

Data collection used participatory rural appraisal techniques for gathering of socio economic and disease epidemiological data. The data collection techniques used were: Key informants interviews, semi-structured interviews, proportional piling, matrix scoring and disease impact matrix scoring. At the study sites the research team introduced the purpose of the research to the local chiefs, elders and community and requested consent to work with the local community. The introductory brief to community respondents was broad on community livelihood issues and general small ruminants' diseases prevalent in the area rather than mentioning PPR disease as the disease of interest. This ensured that the community respondents did not bias their answers in favor of researcher's disease of research focus (*Peste des petits ruminants*).

6.2.3.1 Key informants' interviews and secondary data

Key informants interviews were conducted with 20 local administrative and opinion leaders, 30 livestock traders, 10 staff from Ministry of livestock development and 5 local non-governmental development actors. Key informants provided information on sheep and goat diseases prevalent in the study area, milk and meat production and losses due to diseases and the sale value of meat, milk, and live animals of all ages. The data from these key informants informed the development of parameters values for spreadsheet modeling of direct economic losses due to PPR. Some of data from these interviews formed the background of introducing the focus group discussions carried out with *Adakar* respondents in this study.

6.2.3.2 Focus group discussion

The focused group discussions were carried out as described in section 3.2.3.2 of Chapter 3.

6.2.3.2.1 Semi Structured Interviews

Throughout the focused group discussions the semi-structured interviews (SSI) were carried out as described in section 3.2.3.2.1 of Chapter 3.

6.2.3.2.2 Proportional piling

Proportional piling method was used extensively in this study to generate ranks and proportions.

6.2.3.2.2.1 Proportion piling for wealth ranking

After establishing the wealth groups which were identified as the rich, middle rich, poor and very poor categories through SSI, the respondents were asked to rank the wealth groups using a pile of hundred counters The respondents were asked to divide the hundred counters among the list of wealth groups. The most prevalent wealth group received most counters while the least prevalent wealth group received few counters. The wealth groups in that *Adakar* were arranged from most prevalent to the least prevalent. The counters in each wealth group were counted and recorded.

6.2.3.2.2.2 Proportion piling for livelihood analysis

For each wealth group a list of livelihood activities were established by the respondents through SSI. In each wealth group proportion piling was carried out by the respondents to establish the proportion contribution of each livelihood activity in each wealth group. Taking one wealth group at a time, one hundred counters were given to the respondents and asked to divide the counters among the livelihood activities in one wealth group. The activity that provided the largest share of livelihood received most counters while livelihood activity that contributed least to livelihood was allocated few counters. The counters for each livelihood activity were counted and recorded.

6.2.3.2.2.3 Proportion use of livestock income

For each wealth group a list of commodities and services that were purchased using the livestock income was established in SSI for the *Adakar*. The respondents were asked to show the proportion expenditure of livestock income used in the listed household commodities and services. One hundred counters were used for each wealth group

where respondents were asked to divide the counters with respect to the purchased commodity/service; the commodity/service with large expense taking more counters while the one with least expense taking few counters. The counters for each expense were then counted and recorded.

6.2.3.2.2.4 Proportion of food sources

Sources of foods in the Turkana community were established through SSI and the community respondents asked to show proportion contribution of each food to total food basket in the *Adakar.* The respondents were asked to divide one hundred counters among the type of food source listed (Catley *et al.,* 2008). The food source contributing large share of food for the *Adakar* received more counters. The counters were counted and recorded.

6.2.3.2.2.5 Proportion herd composition

Types of animals by species reared by respondents from each *Adakar* interviewed were listed. Proportion of each species in the herd was established by dividing and allocating the a hundred counters to each species. The higher kept species in the herd got more counters while smaller proportion of counters were allocated proportionately to species with fewer animals. The counter were counted and recorded.

6.2.3.2.2.6 Decrement proportions by species and age group

This exercise was carried out as a process to determine small stock herd dynamics for sheep and goats where each species age group structure was first established through SSI. The age structure of sheep was generated by respondents as follows, lambs

(*imethek*) 0 to 3 months, older lambs (*nanyang*) 4 to 6 months, young adults (*amethek nakale)* 6 to 24 months and old adults (*amethek naapolon*). The goat age groups were kids *(ikale*) 0 to 3 months, older kids (*namenaoei*) 4 to 6 months, young adults (*akale*) 6 to 24 months and older adults (*akine*). The respondents were then asked to show the proportion of sheep and goats that left the sheep and goat herds separately for the year 2010. The respondents were given one hundred counters for each species and asked to divide the counters into proportions of those that left the herd and those that remain in the herd for the study year. The respondents were then asked to show the proportion of sheep and goat for each age group that left the herds respectively. Of the proportion of sheep and goats that left the herds, the respondents were further asked to subdivide respective counters and allocate them according to the age categories so as to show how each age category contributed in totals of those that left the herds. For each age group and species, separately the counters were counted and recorded.

6.2.3.2.2.7 Proportion of pregnant and twinning females

To understand the proportion of females that got pregnant in last one year the respondent were given hundred counters to represent all females of either sheep or goats. The respondents were asked to divide the counters between females that got pregnant and delivered and those that did not get pregnant. The results were recorded. The respondent were again given 100 counters to represent pregnant females that delivered and asked to divide between those that twinned and those that gave birth to one kid/lamb. The results were recorded.

6.2.3.2.3 Matrix scoring

6.2.3.2.3.1 Matrix scoring of livestock related food allocation in a household

The matrix scoring was used to establish how livestock-derived food is distributed within the members of a household. The respondent generated a list of livestock derived foods mainly from small ruminants. The list included meat (*Akiring*), milk (*Akile*), blood (*Akot*), fat (*Akuring*) and ghee (*Akimet*). The members of the household were categorised age-wise as follows; children below five years, children above five but less than 12 years, teenagers above 12 but less than 18 years, Women and men over 18 years. This categorisation of household members was acceptable to respondents as it reflected labour groups in the households. Children below five years are always left at home with their mother nursing them. Children above five years and less than twelve are involved in herding calves, kids and lambs as well as the small milking herd left with small household (*awi).* The teenagers above 12 years and young adults below 18 years are the main group that look after all livestock herds in the large *Adakar*. Adult move with animals depending on their age and give livestock management decisions to the young herders. The matrix was constructed on the ground with x-axis representing household members' categories. The y-axis represented the livestock foods. For each type of food the respondents were given hundred counters that represented the total amount of that type of food in the household. The respondents were then asked to allocate the specific type of food to each of the household members as normally happens in household in a normal year. The age group category that consumes most of that type of food was allocated more counters while the group consuming less of the

food were allocated less counters. The process was repeated for all the five types of livestock-derived foods. The scores allocated to each age category for each food were counted and recorded.

6.2.3.2.3.2 Matrix scoring for herd dynamics

A list of factors that led to sheep and goat leaving the herd was generated through SSI. The respondents were asked to show proportional contribution of each factor in the decrement of sheep and goats. For each small ruminant species, the age group structure was generated by respondents during the interviews as described in proportional piling for off-take. A matrix was constructed on the ground for each species where the x-axis represented the decrement factors being drought, raid, predators, disease, dowry, slaughter and sales. The y-axis represented the age structures of the small ruminants. For each age group the earlier proportion that had been shown to have left the herd in the decrement analysis was subdivided to show proportional contribution of each factor to the dequisition or off-take. Starting with sheep, each age group was allocated counters equal to proportion that left the herd that year and respondent asked to score the decrement factors based on their contribution to total decrement of that age group during that year. The procedure was repeated for all age groups in both species of small ruminants. The scores were counted and recorded.

6.2.3.2.4 Disease impact matrix scoring

Disease impact matrix scoring was used to rank diseases of sheep and goats. The disease ranking was based on the negative impact the disease has on the household use

of the benefits derived from the sheep and goats. The respondents were asked to provide a list of benefits derived from sheep and goats separately during an SSI session. In the matrix scoring exercise described in section 3.2.3.2.3.1 of Chapter 3 the respondents listed the diseases prevalent in the study area and prioritized six of them as most important diseases. The sheep diseases and conditions prioritized were Anaplasmosis (*Lonyang),* PPR *(Lomoo),* Foot and mouth Disease *(Lojaa),* Anthrax *(Lookot),* Sheep pox *(Etune)* and Bottle Jaw *(Loborbolio)* while the conditions and diseases of goat prioritized were Contagious Caprine Pleuropneumonia (*Loukoi),* thin goat syndrome *(Loutogonyen),* PPR *(Lomoo),* Pasteurellosis *Emany,* Diarrhoea *(Naosin)* and Bottle jaw (*Loborbolio)* as outlined in Tables 3.2 and 3.3. For each small ruminant species a matrix was constructed on the ground, with livestock-derived benefits along the *y* axis and diseases on the *x* axis. The respondents were given hundred counters and asked to divide the counters among the benefits according to the relative importance of each benefit, with the most important benefit receiving the highest number of counters and the list important benefit receiving fewer counters. The allocation of the counters to the benefits by respondents provided the weighting of the benefits since the benefits were not of equal importance to the community. A benefit may be of least importance to the community but it is heavily impacted negatively by diseases. The counters for each benefit carrying the weight of the benefit were then further subdivided and allocated across to each disease, based on how each disease relatively impacted negatively on achievement of that benefit by the household of that *Adakar*. The disease having the greatest impact received the highest number of

counters. The number of counters allocated for each disease was totaled to give a measure of the overall impact of that disease on small ruminant-derived livelihood benefit. However this impact measure was probed through semi structured interviews to triangulate the results.

6.2.3.3 Spreadsheet modeling of direct economic losses due to PPR

Peste des petits ruminants disease caused both direct and indirect economic losses due to high mortality and morbidity rates in the infected sheep and goats for the year 2010. Unavailability of recorded data on PPR occurrence and its impact on small stock productivity in Turkana County constrained the quantification and assessment of indirect losses associated with the occurrence of the PPR (Kivaria, 2006; Singh and Prasad, 2008). In order to estimate the direct economic losses associated with PPR, goat and sheep productivity parameters in Turkana County were estimated using participatory epidemiological methods and some variables were drawn from literature. The key direct losses were calculated based on PPR mortality, milk and body weight losses. The PPR mortality and morbidity data was generated in a participatory epidemiological study of PPR disease reported in section 3.3.3 of Chapter 3 titled "Community appraisal of *Peste des petits ruminants* and validation of the community knowledge in Turkana County". Milk losses in sheep and goats occasioned by PPR death and increased inter-kidding/inter-lambing period as well as its market value were estimated from key informants interviews in Turkana The direct economic impact of PPR in Turkana was estimated in the spreadsheet mathematical model (Bennet and Kitching, 2000; Kivaria; 2006):as follows:-

Total direct loses (T^L) for the model herd are depicted by the formula

$$T^L = L+M+W+O \quad \ldots\ldots 1$$

However the model herd costs are converted to field herd model cost by the formula as described by Barasa *et al.,* (2008).

$$T^L = (L+M+W+O) \times (N/100) \quad \ldots\ldots 2$$

Where

L = Mortality losses due to PPR.

M = Milk losses associated with PPR

W = Body weight losses associated with PPR

O = Opportunity cost of managing surviving animals.

N = Population of sheep and goats in Turkana County

The key variables used in spreadsheet model are summarized in Table 6.1.

The model was subjected to sensitivity analysis on selected input variables. Sensitivity analysis for the model was carried out by adjusting upwards by 5%, 10% and 20% disease parameter of mortality, morbidity, milk loss per PPR case and proportion weight lose per PPR case.

6.2.3.3.1 Mortality losses due to PPR

Mortality losses included small stock dying of PPR and the kids and lambs lost due to dying pregnant does and ewes. The participatory methodologies used in estimating the variables in this analytical model assumed herd of 100 sheep and 100 goats therefore the results were further extrapolated to cover actual field population of Turkana County (Barasa *et al.,* 2008)

6.2.3.3.1.1 Losses due to PPR mortality =L^d

Direct mortality losses due to PPR were worked out as the product of the proportion of the sheep/goats in age groups young to adult (*Imethek* to *Amethek* in sheep and *Ikale* to *Akine* in goats) denoted as ($G^{a..y}$); PPR mortality in sheep/goats in age groups young to adult ($H^{y\ldots a}$) and the sale value in Kenya Shillings (KES) of sheep/goats for each age groups young to adult ($C^{y\ldots a}$) (Barasa *et al.,* 2008)

$$L^d = G^{y...a} \times H^{y\ldots a} \times C^{y\ldots a} \quad \ldots\ldots 3$$

6.2.3.3.1.2 Losses of kids/lambs due to doe/ewe dying of PPR =L^y

Losses from expected kids and lambs from pregnant dams was derived as a product of proportion of pregnant sheep/goats (P^f), PPR mortality in sheep/goats in age group adult (H^a) and the sale value in KES of sheep/goats for age group young (C^y)

$$L^y = P^f \times H^a \times C^y \quad \ldots\ldots 4$$

Total Mortality losses (Barasa *et al.,* 2008)

$$L = L^d + L^y \quad \ldots\ldots 5$$

6.2.3.3.2 Milk losses due to PPR

Milk losses included those occasioned by PPR deaths of lactating and pregnant dams, reduced milk yield from recovering PPR cases of lactating dams and long term losses due to increased inter-kidding / lambing interval.

6.2.3.3.2.1 Direct milk losses due to PPR deaths M^d

Direct milk loses included those due to PPR mortality of lactating and pregnant dams they were calculated as a product of sum of proportions of lactating sheep/goat (L^f) and pregnant in sheep/goat (P^f); PPR mortality in sheep/goats in age group adult (H^a), daily volume (litres) of milk produced per healthy sheep/ goat (V^a), lactation period (days) (T^l) and sale value of milk (Kenya shillings/litre) (C^m).

Proportion of lactating sheep/goat (L^f) was derived from proportion of the sheep/goats in age groups young (G^y) and proportion of male and female in the herds ($S^{m..f}$).It was assumed that for every young kid or lamb there was a lactating mother. Thus proportion of lactating sheep/goat (L^f) equals proportion of the sheep/goats in age groups young (G^y) divided by proportion of females in the herds ($S^{.f}$) (Barasa *et al.,* 2008).

$$M^d = (L^f+P^f) \times H^a \times V^a \times T^l \times C^{m...} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 6$$

6.2.3.3.2.2 Direct losses due to reduction in milk yield in recovering cases M^r

Losses due to reduced milk yield in convalescing lactating dams was calculated as a product of proportion of lactating sheep/goat (L^f) multiplied by the difference of prevalence of PPR in lactating sheep/goats (I^{af}) and mortality of lactating sheep and goats (H^a), volume of milk loss (litres) per day per PPR case (V^l), duration (days) of reduced milk production per acute PPR case (T^r) and sale value of milk (KES/litre) (C^m). Key informants indicated that milk loss (V^l) due to PPR disease was drastic. Using their milking calabash the women who milk demonstrated losses estimated at 60% in sheep and 50% in goats they said "The sheep and goats are literally dry; their milk production cannot feed the herdsboys and lambs for them to spare anything for the young children at home". Only kids and lambs would be left suckling yet milk

production in some cases would not be enough resulting in malnutrition and death in newborns. Duration of disease in convalescing animal informed duration of reduced milk production (T^r) (Barasa *et al.,* 2008).

$$M^r = L^f \times (I^{af}-H^a) \times V^l \times T^r \times C^m \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 7$$

6.2.3.3.2.3 Milk losses due to increased inter-kidding/lambing period M^y

The problem of non-conception caused by PPR increases the inter-kidding period and thus lower number of animals that would be in milk at any given time. A recovering sheep/goat lost a season equivalent to 5.5 months delay to the next conception. The loss of milk was calculated as the reduction in proportion of lactating animals in any year being a product of sum of proportions of lactating and pregnant animals, recovering animals, lost kiddings/lambings, volume of milk production per sheep/goat, lactation period of sheep/goat and cost of milk per litre (Singh and Prasad, 2008).

$$M^y = (L^f + P^f) \times (I^a - H^a) \times (12/K-12/(K+Q)) \; V^a \times T^l \times C^m \ldots\ldots\ldots 8$$

Total milk losses

$$M=M^d + M^r + M^y \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 9$$

6.2.3.3.3 Body weight losses

6.2.3.3.3.1 Direct losses due to body weight losses W^d

The surviving convalescing sheep/goats lost weight and market value due to PPR disease. The weight losses were estimated as a product of proportion of sheep/goats surviving the disease multiplied by estimated proportion of weight loss per animal,

estimated weight of sheep/goats in various age groups and cost of liveweight per kilogram in Kenya shillings (Singh and Prasad, 2008).

$$W^d = G^{y...a} \times (I^{y...a} - H^{y...a}) \times W^l \times W^{y...a} \times C^{me} \quad \ldots\ldots\ldots\ldots\ldots\ldots 10$$

6.2.3.3.3.2 Body weight losses due to increased inter kidding W^y

The body weight losses due to increased inter-kidding was estimated by calculating the lost kiddings and lambings due to PPR disease multiplied by proportion of surviving lactating and pregnant sheep and goat females, birth weight of kids and lambs and sale value of meat locally in Kenya shillings (Singh and Prasad, 2008)

$$W^y = (L^f + P^f) \times (I^a - H^a) \times (12/K\text{-}12/(K+Q)) \times B \times C^{me} \ldots\ldots\ldots 11$$

Total weight losses

$$W = W^d + W^y \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 12$$

6.2.3.3.4 Opportunity cost

Use of conventional veterinary medicine in rural Turkana is rare. Most herdsmen will generally gather herbs to treat ailments on their small stock. Veterinary medicines are expensive and most herders access them through relief handouts from local development agencies. However at times herders buy capsules and tablets of human medicine and apply them on sick small stock. In light of this it was assumed that the Turkana people will put effort to care for the PPR surviving small stock at cost equivalent to 2.5% sale value of sheep and goats (Singh and Prasad, 2008).

$$O^c = G^{y...a} \times (I^{y...a} - H^{y...a}) \times (.025 * C^{y...a}) \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots 13$$

Table 6.1: Summary of parameters and methodology for estimating losses due to PPR in Turkana pastoral herds (Barasa et al.,2008)

Parameter description	Methodology for estimation of parameter
Herd age structure	
$G^{y \cdots a}$ = proportion (%) of Sheep/goats in ages group young to adult	Proportional piling (PP) of sheep and goat herds by age group(n=27)
Losses due to mortality	
$H^{y \cdots a}$ = PPR mortality (%) in sheep/goats age groups young to adult	PP Sheep (n=43) / goat (n=44) mortality by disease
$C^{y \cdots a}$ = sale value (KES) of sheep/goats in age groups young to adult	Informal interviews with livestock traders
Losses from reduced milk production	
$S^{m..f}$= proportion (%) of male and female sheep/goats in the herds	PP Sheep (n=6) / goat (n=6)
L^{f} = proportion (%) of lactating sheep/goats	Derived from Gy, and Pf
P^{f}= proportion (%) of pregnant in sheep/goat	PP Sheep (n=6) / goat (n=6)
$I^{y \cdots a}$= prevalence (%) of PPR in sheep/goats in ages group young to adult	PP for disease prevalence
I^{af}= prevalence (%) of PPR in lactating sheep/goats	PP for disease prevalence Iaf equals Ia in adults
V^{l} = volume of milk loss (litres) per day per PPR case	Key informant interviews with livestock keepers
T^{r} = duration (days) of reduced milk production per acute PPR case	Key informant interviews with livestock keepers (Budza 1988)
V^{a} = daily volume (litres) of milk produced per healthy sheep/ goat	Key informant interviews & literature (Njanja 1991)
T^{l} = lactation period (days)	(Njanja 1991, Marete, 2011)
C^{m} = sale value of milk (KES/l)	Key informant interview with milk sellers
C^{me}=sale value of meat in locally (KES/kg)	Meat price from meat traders
Losses due to weight losses	
W^{l} = proportion (%) of body weight loss	Key informant interviews with livestock officials & keepers
$W^{a \cdots y}$ = Average body weight (kg) of sheep/goat in ages group young to adult	Key informant interviews with livestock officials & keepers
K = Inter-kidding/lambing interval in days	Key informant interviews Literature (Njanja, 1991)
B = Birth weight (kg)of Kid/Lamb	Literature (Njanja, 1991)
N = Sheep/Goat population in study area in Turkana County	(KNBS, 2010) Kenya population and livestock census data
Q=Delay in conception in months due to disease	Key informant interviews and Livestock keepers

6.2.4 Data management and statistical analysis

Both qualitative and semi-quantitative data were collected in the study. The qualitative data were presented without being subjected to formal statistical analyses. The quantitative data was entered and cleaned in Microsoft Excel (Microsoft Corp., Redmond, WA). It was then exported to SPSS (2008) statistical software version 17.0 (IBM Corp., Armonk, NY) for analysis using non-parametric statistical tests. Analyses were undertaken using descriptive statistical procedures and data summarized using medians to determine central tendency while dispersion was expressed by 10^{th} and 90^{th} percentiles estimation. To determine the significance of association between livelihood activities and wealth categories; species in herd structure and wealth categories; livestock income expenditure items and wealth categories; livestock food products consumption and household members; livestock decrement factors and small stock age groups and finally small stock benefits and small stock diseases as reported Turkana respondents, the Friedman's test was used (SPSS, 2008; Jost *et al.,* 2010)

The data to be used in spread sheet model was maintained in Microsoft Excel and model outputs computed following the insertion of the formulas (Bennet and Kitching, 2000)

6.3 Results

6.3.1 Turkana pastoral socio economy

6.3.1.1 Livestock keeping and social satisfaction among Turkana

Livestock keeping and social satisfaction was interrogated from the respondent groups. The analyses of responses show that Turkana livestock keeping is a way of life where a person with livestock is admired, feared and revered. Men respondents were heard saying *'Brave men end up being rich"*. The people with livestock are able to provide for their families and can access income to purchase any consumer products. They easily build friendship relations usually based on exchange of livestock gifts. These friendship networks allow them to participate in social and political activities that are major decision making fora for the Turkana community. Respondent averred that owners of livestock are seen as creditworthy; since they have ability to repay their loan obligations and help other members of their networks in community to settle their loans by provision of required animals. On the contrary people without livestock are looked down with disdain and are seen as lazy. Other sections of the community that own livestock do not bother with people without livestock unless when engaging them as herd laborers. People without livestock are not involved or invited in meetings, discussions or community ceremonies; they remain in the periphery begging for leftovers. People without livestock find it hard to marry or participate in any socio-cultural activities that require livestock as a settlement for the social obligation. It is for this reason that ownership of livestock can be attempted at any cost. The respondents

reported that in situation where livestock herds are not productive enough to feed the households, women ridicule their men particularly those without livestock portraying them as people who cannot provide for their families. The young men are not spared either with reminders through ridicule songs that survival of their community is through livestock. It is through such pressure and influence (Schilling *et al.,* 2012) that men old and young organize themselves to go raid livestock from other neighboring community so that they can provide for their women, families and sustain their relevance in Turkana society.

6.3.1.2 Wealth ranking and livelihood analysis at village level

The Turkana pastoralist identified a list of indicators of wealth within their community that included: livestock, children, wives, farm, close relatives, extended family, friends, elders, money, rain, business, peace, hard-work, health and creditworthiness. The key indicators mentioned by all villages were livestock, children and wives as indicated in Figure 6.1. Using the list of wealth indicators the respondents categorized the wealth groups in their communities into four, which were identified as being the very rich (*Ekabaran)*, rich *(Lokindingos)*, poor (*Ekibotonit)* and very poor or destitute *(Ekalokanit* or *Ekulokit)*.

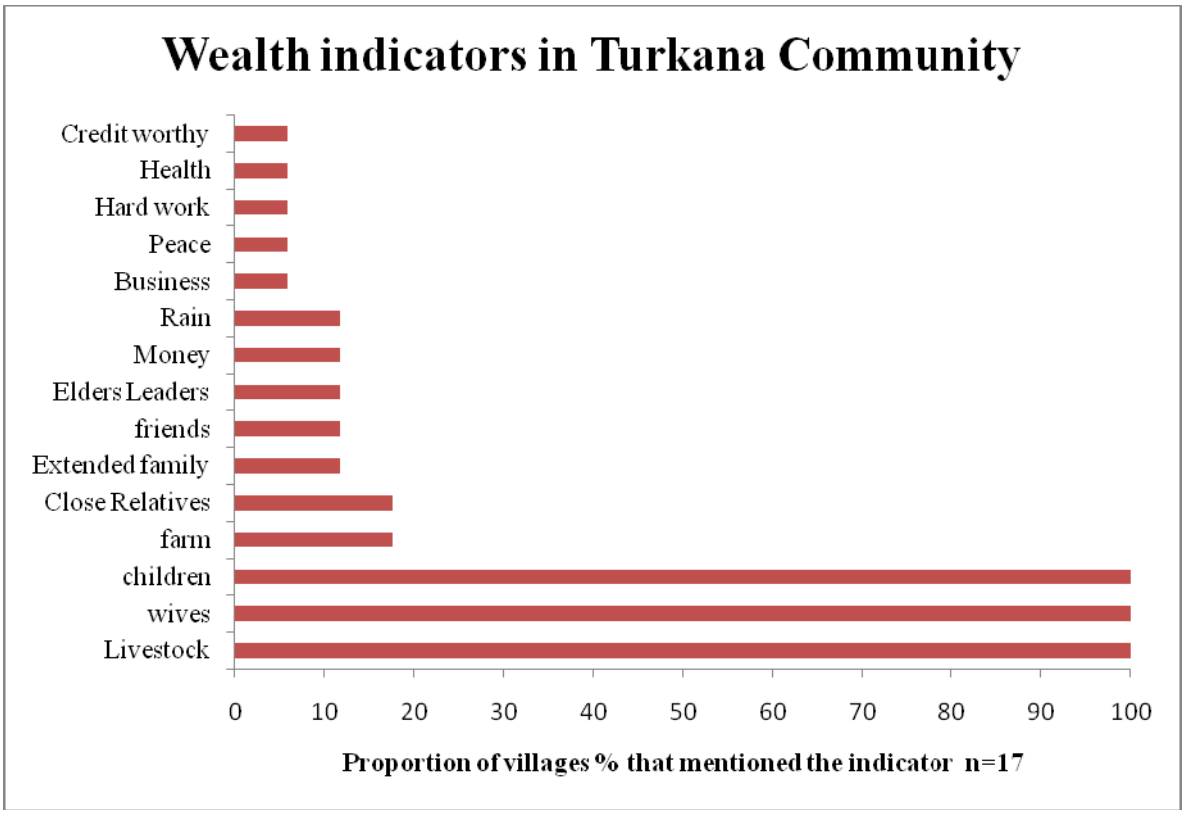


Figure 6.1: Wealth indicators in Turkana Community

The very rich *Ekabaran* were described as members of the Turkana community that had large herds of all livestock (camel, cattle, goats, sheep and donkeys), several wives and numerous children. The *Ekabaran* rely almost wholly on livestock for their livelihoods and live a nomadic life with their families tending their livestock. The rich *Lokindingos (*meaning middle rich*)* were described as having medium size herds of all livestock, two to three wives and a number of children. The poor *Ekibotonit,* were described as members of the community who had a handful of small stock and possibly two to three camels or cattle. *Ekibotonit* has small family of wife and few children. The very poor *Ekalokanit / Ekulokit* were described as section of Turkana community that generally did not own any livestock. These people lived lonely lives and some were destitute;

they were normally found at the market centers. They have few or no relatives and friends that are concerned with them. They fend from portfolio of activities that include borrowing hand outs, relief and petty trade. The Turkana community acknowledged that calamites such as armed livestock raiding, drought and livestock and human diseases could reduce an *Ekabaran* to any of the lower wealth categories depending on the impact and extent of the disaster. Loss of livestock could lead an *Ekabaran* to be a destitute (*Ekalokanit / Ekulokit).* It is the most feared situation because it essentially means a man could not fend for his wives and children and the consequence is to lose them as they go out to fend for themselves.

Using proportional piling (N=16) the four wealth categories were proportionally allocated to the Turkana pastoral community as median scores with 10th and 90th percentiles where the very rich *Ekibaran* were the highest proportion at 28.7% (2.1, 59.7%) the rich *Lokidingos* had proportion of 20.9% (8.8, 35.6%), the poor *Ekibotonit* at 19.1% (5.7, 52.5%) while the very poor *Ekalokanit / Ekulokit* proportion was 24.2% (7, 62.5%). The association of percentage proportions with the wealth categories was statistically insignificant (p=0.843).

For each of the wealth categories a livelihood analysis was conducted to establish key livelihood activities that each wealth category pursued for its subsistence as presented in Table 6.2.

Table 6.2: Key livelihood activities pursued by each wealth category (N=13)

	Median scores (10th and 90th percentiles)			
Livelihood activity	Very Rich (*Ekibaran*)	Rich (*Lokidingos*)	Poor (*Ekibotonit*)	Very Poor (*Ekulokit/Ekalokanit*)
Livestock***	100 (100,100)	42.0 (18.3, 67.1)	11.1 (5.0, 48.6)	0 (0,0)
Borrowing	0 (0,0)	14.0 (6.4, 24.4)	0 (0,0)	0 (0,0)
Craft work	0 (0,0)	15.2 (5.0, 30.0)	14.0 (3.8, 27.9)	12.9 (3.2, 20.6)
Trade***	0 (0,0)	22.0 (8.1, 56.3)	11.9 (2.8, 27.1)	6.9 (1.2, 27)
Charcoal	0 (0,0)	0 (0, 0)	22.2 (4.4, 44.0)	19.0 (5.5, 30.4)
Building poles/rafts*	0 (0,0)	0 (0, 0)	17.0 (4.8, 21.2)	10.0 (4.6, 25.9)
Wage Labour	0 (0,0)	0 (0, 0)	12.0 (4.8, 21.2)	9.1 (2.8, 22.0)
Hunter/gatherer	0 (0,0)	0 (0, 0)	0 (0, 0)	21.2 (9.2, 55.4)
Employment	0 (0,0)	0 (0, 0)	0 (0, 0)	7.6 (4.0, 23.2)

*P<0.05;**P<0.01; ***P<0.001. No asterix means P>0.05

The very rich (*Ekibaran*) relied entirely 100% on their livestock for subsistence while the rich (*Lokindingos*) rely 42% (18.3, 67.1%) on livestock alongside other subsistence activities such as borrowing from friends, craft works and petty trade. The poor (*Ekibotonit*) have allay of activities they depend on for their subsistence key ones being charcoal burning, building poles and raft harvesting for sale, craft work, wage labor, petty trade and livestock in that order. For the *Ekibotonit,* livestock contribute 11.1% (5.0, 48.6%) of their livelihood. Livestock as an enterprise was associated with the well off wealth categories (p<000). The very poor (*Ekulokit/Ekalokanit)* were shown to have

a larger portfolio of livelihood activities that they engage in for subsistence purposes. The very poor did not have livestock and relied heavily on hunting and gathering, charcoal burning, craft work, building poles and raft harvesting, wage labour, employment as herds' people in return for food and petty trade. The analysis of livelihood activities in each wealth category and wealth category proportions indicated that only 76% of the population relied on livestock as part of their subsistence in Turkana. Further analysis was carried out to establish the herd structures in terms of species reared by each wealth group for subsistence Table 6.3.

Table 6.3: Herd structure by species within the wealth groups categories N=11

	Median scores (10th and 90th percentiles)		
Species	Very Rich (*Ekibaran*)	Rich (*Lokidingos*)	Poor (*Ekibotonit*)
Camel***	24.0 (17.6, 34.4)	17.0 (12.3, 34.0)	3.0 (0.0, 9.7)
Cattle***	20.0 (10.4, 23.3)	18.0 (7.4, 24.0)	6.5 (0.0, 13.9)
Goat**	25.0 (16.1, 37.7)	29.3 (17.6, 39.6)	41.4 (1.6, 67.7)
Sheep	17.0 (12.3, 34.0)	30.5 (13.3, 45.9)	23.0 (7.2, 40.6)
Donkey	8.1 (3.7, 14.4)	9.1 (6.5, 16.7)	6.0 (0.0, 43.4)
Poultry***	0.0 (0.0, 5.1)	0.0 (0.0, 3.1)	5.4 (0.0, 51.6)

*P<0.05;**P<0.01; ***P<0.001. No asterix means P>0.05

The very rich wealth category *Ekebaran* had well balanced herds with goats 25.0% (16.1%, 37.7%) and camels 24.0% (17.6%, 34.4%) constituting the larger portions of the herds. The rich *Lokidingos* herds were approximately 60% constituted of sheep 30.5% (13.3, 45.9%) and goats 29.3% (17.6, 39.6%). The poor wealth group,

Ekebotonit herds were almost entirely composed of goats and sheep at 41.4% (1.6, 67.7%) and 23.0% (7.2, 40.6%) respectively. Poultry was one of the livestock owned by the poor constituting 5.4% (0, 51.6) of their herds. Cattle camel and poultry proportions in herds were well associated with the wealth categories ($p<000$). Similarly the goats' proportion and composition within the herds for each wealth group was well associated with the wealth status ($p<01$). Sheep and donkey proportion and composition between the herds of the different wealth groups were not statistically different across the wealth groups.

An analysis was carried out to establish how the subsistence income generated from livestock and livestock products sales was used within each of the wealth categories Table 6.4.

Table 6.4: Uses of livestock income N=11

	Median scores (10th and 90th percentiles)		
Expenditure	Very Rich (*Ekibaran*)	Rich (*Lokidingos*)	Poor (*Ekibotonit*)
Food *	23.0 (13.5, 42.4)	23.6(13.0, 43.7)	31.0 (23.8, 51.7)
Clothes	11.6 (7.0, 19.2)	11.1 (4.5, 23.7)	15.1 (6.3, 26.3)
health	9.1 (3.6, 17.2)	10.1 (2.6, 20.1)	12.8 (4.2, 22.0)
Veterinary*	7.0 (2.6, 18.1)	9.1 (4.6, 15.2)	14.4 (4.9, 26.9)
dowry	17.1 (7.9, 36.6)		
Trade	10.1 (3.4, 18.6)	13.0 (7.3, 30.8)	
School	15.2 (9.3, 34.4)	17.5 (7.9, 36.6)	0(0, 26.1)
Gifts		5.5 (2.8, 11.0)	
Utensils			13.6 (6.5, 19.4)

*P<0.05;**P<0.01; ***P<0.001. No asterix means P>0.05

The greatest share of livestock income was spent on food for all the three wealth groups with association of food with wealth groups showing statistical significance of $p<0.05$. The only other expenditure which had marked association with wealth group with statistical significant ($p<0.05$) was veterinary service. Expenditure items shared between all the three wealth groups included cloths, school, health and veterinary services. Some of the expenditure items such as dowry, trade, gift and utensil were specific to one or two wealth groups.

Food being a major livestock income expenditure item was further analyzed to identify food type composition consumed by the Turkana community. The community identified three sources of food being animal, plants and relief as well as commercial-processed food products. The animal products included milk (*Akile),* meat (*Akiring),* ghee (*Akimet),* fat (*Akuring)* and blood (*Akot)*. The plant products included farmed crops such as maize, sorghum and exotic vegetables as well as the wild fruits, seeds and vegetables such as *edapal, eglae, nakalalio, edum, elamash, epat, emeyen, ng'alam, edome, ng'apong'a, eng'omo, ng'iminae* and *ng'tit* among other that could not be identified with their biological names. The relief sand commercial products included sugar, tea, oil, cereals flour and pulses The animal products constitute 29.4% (7.7, 46.6%), plant products 21.6% (11.8, 38.2%) while relief and commercial processed foods constitute 47.0% (28.6, 73.6%). The Turkana association of percentage proportions with food type showed a marked statistical significance ($p<0.011$).

The consumption of animal products was analyzed looking at proportion allocation of each type of animal product to each age group in the household Table 6.5.

Table 6.5: Allocation of food products from animal within a Turkana household N=14

	Median scores (10th and 90th percentiles)				
			Youth		
	Children <5yr	Children >5yr<12yr	>13yr<18yr	Women	Men
Ghee *Akimet* ***	10.7 (4.0, 36.7)	15.1 (5.5, 21.6)	25.3 (15.1, 37.7)	21.6 (11.6, 54.0)	15.8 (10.0, 36.0)
Fat *Akuring****	10.6 (3.0, 34.0)	14.1 (5.0, 21.6)	23.6 (7.4, 36.8)	27.1 (13.5, 40.5)	21.7 (13.9, 41.5)
Milk *Akile* ***	12.4 (4.5, 27.6)	21.4 (10.6, 34.8)	29.5 (16.9, 50.2)	11.0 (6.0, 15.5)	21.0 (9.0, 31.0)
Meat *Akiring****	5.0 (1.0, 10.0)	17.9 (9.5, 30.5)	31.5 (20.1, 49.3)	15.5 (5.5, 24.1)	28.0 (16.6, 42.7)
Blood *Akot****	3.5 (0.0, 10.5)	19.0 (9.1, 23.1)	39.0 (28.1, 65.2)	11.5 (4.0, 16.1)	26.5 (9.6, 37.5)

*P<0.05;**P<0.01; ***P<0.001. No asterix means P>0.05

Proportions allocated for consumption of different animal products associated to different age groups were statistically significant (p<001) for all animal products across all age groups. The youth in the age group >13yr<18yr consumed the largest share of all animal products in the household.

6.3.2 Small stock herd dynamics and production parameters

Parameters relating to dynamics in the small stock herds in sizes and composition were analyzed. Key among the factors analyzed were kidding and lambing per year, twining rates, proportion of pregnant and non-pregnant females in a herd, proportion of males and females in a herd and average age of males and females at disposal time. In an analysis of responses from interviewed *Adakars,* [villages herds (N=6) for goats and N=7 for sheep (Table 6.6)] it was established that pregnant and non pregnant female goats constitute 42.0% (5.0, 81.0%) and 58.0% (19.0, 94.0) respectively while pregnant and non pregnant female sheep constitute 54.0% (4.0, 75.0) and 46.0% (25.0, 96.0%) respectively of all female sheep in the herd (Table 5.6). Both sheep and goats produced on average two off-springs per year. Twining was common in goats 13.5% (2.0, 26%) as compared to sheep 7.0% (4.0, 12.0%).

Table 6.6: Small stock herd parameters on productivity status in Turkana

	Median scores (10th and 90th percentiles)			
	Pregnant	Non pregnant	young per year	Twining
Sheep N=7	54.0 (4.0,75.0	46.0 (25.0, 96.0)	2.0 (1.0, 2.0)	7.0 (4.0, 12.0)
Goat N=6	42.0 (5.0, 81.0)	58.0 (19.0, 94.0)	2.0 (1.0, 2.0)	13.5 (2.0, 26.0)

The age structure analysis showed that the females in both sheep and goats remained in the herds for longer periods (in years) before they were disposed [4.0 (3.0, 6.0) in sheep and 4.0 (3.0, 5.0)] in goats respectively. The males in both sheep and goats had a shorter lifespan in years [3.5 (1.5, 4.0) and 4.0 (2.0, 4.0)] respectively. This is because the males in both sheep and goats have more ceremonial, ritual rite utilities and significance in several aspects of the Turkana cultural lives. Further the adage that small stock are mobile banks is borne out the quick nature of market disposal of male small stock for income to be used in the households. The sex structure revealed that the proportion of males in both sheep and goats was approximately a quarter of the herd at 23.0% (16.0, 37.0%) for sheep and 28.5% (19.0, 46.0%) for goats. Table 6.7 present small stock herd parameters.

Table 6.7: Small stock herd parameters on age and sex structure in Turkana

	Median scores (10th and 90th percentiles)			
	Maxi Age Female	Max Age Male	Sex Structure Male	Sex Structure Female
Sheep N=7	4.0 (3.0, 6.0)	3.5 (1.5, 4.0)	23.0 (16.0, 37.0)	77.0 (63.0, 84.0)
Goat N=6	4.0 (3.0, 5.0)	4.0 (2.0, 4.0)	28.5 (19.0, 46.0)	71.0 (53.0, 80.0)

Increase in size of small stock herds as described by the respondents was influenced by various factors. Good seasonal rains favored abundant pasture growth and increase in presence of water sources was described as the major factor that helped build the herds through improved lambing and kidding. Local livestock exchanges and herd splitting were also encouraged and practiced heavily by the Turkana as ways of securing the family herds and offered opportunity for the herds to expand. A major source of

animals into the herds mentioned by the respondents was the animals paid in bride price (dowry) and other cultural obligations. Livestock raids were also considered as significant source of animal into the Turkana herds. During the interviews the respondents would say *"A man with many wives will have a big family of girls he will marry off to get more wealth while his boys are a source of labor to look after the livestock"*. Of little significance was building herds through market and project restocking done by development agencies.

In-depth analyses of factors influencing decrement in small stock was carried out. Within the sheep and goat herds the community described the losses in small stock for every age group. The proportions of various age groups of small stock that were lost from the herd within the study period of 2010 are presented as percentage median score with 10^{th} and 90^{th} percentiles in parenthesis. Among the sheep N=14, those lost from the herds accounted for 79.38% (60.5, 94.25%) of all sheep. The lambs (*imethek*) lost from the herds accounted for 27.78% (21.18, 32.99%) of all new born lambs up to those with age of 2 months. Lambs in the age group between 3 and 5 months (*nanyang*) had a general decrement of 11.91% (9.08, 14.14%). The middle age group (*amethek nakale)* 6 months to 24 months had an decrement of 23.81% (18.15, 28.28%), while the adults (*amethek naapolon)* age above 24 months had an decrement of 15.88% (12.10, 18.85%). The association of percentage proportions of lost sheep with each age group was statistically significant ($p<0.000$).

Analysis of goat decrement for the same study period shows a similar tread. Among the goat N=13, those lost from the herds accounted for 74.50% (52.65, 90.95%) of all goat.

The new born kids and up to 2 months age (*Ikale*) had a decrement of 28.02% (19.89, 33.72%); kids within the age between 3 and 5 months (*namenaoei*) had a decrement of 9.84% (6.99, 11.85%); middle age group (*akale*) age between 6 months to 24 months had a decrement of 24.24% (17.20, 29.16%) while the adult age more than 24 months had a decrement of 13.64% (9.68, 16.40%). The association of percentage proportions of lost goats in each age group was statistically significant ($p<0.000$).

The different factors that influence depletion of small stock from the herds were categorized by respondents as drought, livestock raids, predators, diseases, and bride price (dowry), home slaughter and market sales. Droughts were characterized as prolonged drought seasons that resulted in depletion of pasture and drying up of water sources. Small stock going through drought season became wasted and dehydrated due to lack of food nutrients and water and consequently, if the weather does not improve to provide for pasture regeneration most weak animals die. Livestock raid was socio-cultural activity practiced by the Turkana against their neighbors as way to accumulate livestock wealth. However all Turkana neighbors (Samburu, Pokot, Karamojong, Toposa, Jie and Merrille) are also known to viciously raid the Turkana for livestock. With so many enemies Turkana herds are in constant threat of depletion. Livestock raids were a show of military might and were unsettling to the raided community if successful. The raided community loses their livestock, their family members and constantly lives in fear and frustration as try to build their security by rearming themselves or taking refuge. In normal times raids are carried out by warriors who are impatient of being laborers and waiting for inheritance from their fathers who could

still be strong and healthy and are not ready to relinquish their wealth to their sons. Such warriors were in such of their own identity and space in society. If they successfully carried out a raid it rewarded them with enough animals to marry and start a family. Irrespective of their fathers wealth such warriors were seen as men of means and earned space in society leadership for their prowless in creating new wealth. During abnormal times particularly after extended drought where animals are lost, communities would organize to restock lost animal through raids irrespective of their military might as this was seen as a matter of life or death. The raided communities were usually devastated particularly if able men are killed and animals are lost. The raided households rarely recovered and would have to diversify to new sources of livelihoods including destitution. Thus raiding was abhorred and revered in equal measure.

Disease threat to small stock was characterized by long list of diseases identified by the Turkana respondents. Among the diseases some were fatal while others induce reproductive losses. Predator threat to small stock was characterized by wild carnivores such as hyenas, wild dogs and snakes that prey on the unattended small stock. Dowry payment consisted about 100 up to 150 small stock; It is a Turkana culture which constitutes a significant large amount of livestock. Only the very rich people could afford to marry more than one wife without depleting their herds. Other cultural gifting and payments were done with livestock and contribute to small stock depletion. Home

slaughter and market sale of small stock contribute to herd depletion but to lesser degree.

Among the goats (Table 6.8) drought was the major factor affecting goat depletion in the herds. New born kids and young adults in middle age group were heavily affected by drought with association between drought and all the age groups being statistically significant ($p<0.000$). Disease were recorded as the second most important factor influencing depletion of goats in the herds with some statistical significance of $p<0.000$ for association of age groups and disease. New born kids were the most affected by diseases followed by young adults in the middle age group. Raids were the third major factor that depletes goats from the herds. Animals paid as dowry were listed as the fourth factor that depleted goat most from the herds. Slaughter and sales were mainly carried out for the middle age group (*Akale*) and the adult goats (*Akine*) and contributed to the lowest percentage of the goats lost in the herd.

Table 6.8: Proportion contribution of different factors influencing loss of goats in herd N=13

	Median scores (10th and 90th percentiles)			
	New born kids (*Ikale*)	Older kids (*Namenaoei*)	Young adult (A*kale*)	Adults (*Akine*)
Drought***	8.92 (4.64, 13.18)	3.01 (1.57, 4.87)	5.67 (2.90, 8.48)	3.69 (1.38, 7.12)
Raids***	3.79 (0, 7.22)	1.48 (0.48, 2.72)	3.20 (1.85, 5.95)	1.71 (1.35, 3.28)
Predators***	3.15 (1.20, 7.83)	1.08 (0.38, 2.15)	2.41 (0.60, 4.21)	1.05 (0.34, 2.81)
Disease***	8.21 (3.53, 12.60)	1.78 (0.76, 3.83)	6.00 (1.47, 8.47)	2.77 (1.18, 4.50)
Dowry§***	4.74 (0, 5.99)	1.55 (0.56, 2.85)	2.88 (1.91, 7.11)	1.35 (0.34, 2.81)
Slaughter***	0	0 (0, 0.66)	1.83 (0.32, 3.17)	0.88 (0.28, 1.80)
Sale***	0	0 (0, 0.86)	1.45 (0.49, 2.44)	0.99 (0.61, 2.19)

*P<0.05;**P<0.01; ***P<0.001. No asterix means P>0.05; §Dowry represent bride price and other cultural animal gifts

Drought was the major cause of sheep depletion (Table 6.9 ;) from the herds with the association of drought with the different age groups showing statistical significance ($p<0.000$). The young adult sheep in the middle age group and the new born lambs were the most affected by drought. The second most important factor affecting depletion of sheep from the herd was diseases and there was statistical significant association ($p<0.000$) between diseases and different age groups. The most affected age group by disease was new born lambs and young adults in middle age group. Livestock raids were the third factor affecting sheep losses from the herd. Animals paid as dowry were listed as the fourth factor depleting sheep from the herds. Predators were the fifth

factor affecting the depletion of sheep flocks. The lambs in the age group between 3 and 5 months (*Nanyang*) were the most preyed by the predators. Slaughter and sale of lambs was not done as this activity was confined to middle age group and adults above 24 months. All the decrement factors in sheep herds were significantly associated with age groups ($p<0.000$).

Table 6.9: Proportion contribution of different factors influencing herd dynamics in sheep N=14

	Median scores (10th and 90th percentiles)			
	New born Lambs (*Imethek*)	Older lambs (*Nanyang*)	Young Adult (*Amethek Nakale*)	Adult (*Amethek Naapolon*)
Drought***	8.39 (5.65, 13.53)	3.10 (1.53, 5.11)	5.59 (2.50, 10.91)	4.80 (2.86, 8.03)
Raids***	3.47 (0, 13.04)	1.78 (0.27, 4.85)	3.81 (2.21, 7.36)	2.56 (1.10, 4.96)
Predators***	3.63 (0.96, 9.68)	1.90 (0.80, 4.48)	2.55 (1.23, 7.27)	1.43 (0.44, 4.45)
Disease***	4.80 (3.03, 10.20)	2.44 (0.75, 3.72)	3.77 (1.63, 6.32)	2.37 (1.08, 4.52)
Dowry§***	3.08 (0, 8.25)	1.49 (0.69, 3.99)	3.91 (1.20, 7.89)	1.61 (0.75, 3.00)
Slaughter***	0 (0, 1.68)	0 (0, 0.88)	1.04 (0, 2.86)	0.86 (0.27, 1.70)
Sale***	0	0 (0,0.40)	1.45 (0, 2.92)	0.67 (0, 1.83)

*P<0.05;**P<0.01; ***P<0.001. No asterix means P>0.05; §Dowry represent bride price and other cultural animal gifts

6.3.3 Disease impact on small stock livelihood benefits

6.3.3.1 Benefits derived from sheep and goats

The community listed meat, milk, sale income, dowry, skin, gifting, high reproduction, goat survival ability in adverse conditions and prestige bestowed to owners of herds of

goats as the benefits that are derived from goat rearing by the Turkana community. Women had an interesting explanation of importance of some of the benefits as one woman respondent said,

"Milk can stay edible for a long period of time and can support several visitors. It can be in the morning and evening. Milk can also be sold to get sugar or tobacco. You can also get oil from milk. A person cannot eat meat on a daily basis."

Analysis of proportion piling results of each benefit, ranked goat milk as the most important benefit as tabulated in Table 6.10. There was statistical significance in association of the perceived proportions to the benefits derived from the goats ($p<0.000$).

Table 6.10: Proportion contribution of the benefits derived from goats

	Mean Rank	Median score (10th and 90th percentile)
milk	7.15	16.0 (8.8,24.4)
meat	6.97	14.0 (8.6, 24.2)
dowry	5.91	12.0 (8.0, 22.8)
survival	5.62	11.0 (2.0, 23.0)
production	5.21	10.0 (6.0, 21.0)
sale	4.68	9.0 (4.8, 15.2)
skin	3.88	8.0 (3.8, 12.2)
prestige	3.41	7.0 (3.0, 13.2)
gift	2.18	5.0 (1.6, 9.8)

N=17, Freidman test statistic χ^2 (8) =50.007; $p<0.000$

Analysis of benefits derived from sheep showed that community respondents identified meat, milk, skin, fatty oil for ointment, dowry, gift, production and prestige (Table 6.11). Meat was ranked as the most important benefit and gifts the lowest ranked benefit. There was statistical significance in the association of proportional allocation to the benefit derived from sheep ($p<0.000$).

Table 6.11: Rank and proportion contribution of benefits derived from sheep

	Mean Rank	Median Score (10th and 90th percentile)
meat	6.24	14.0 (8.0, 32.0)
milk	5.85	14.0 (9.8, 25.8)
oil	5.62	14.0 (7.6, 25.0)
production	5.56	15.0 (6.4, 36.2)
dowry	4.79	13.0 (4.8, 21.2)
skin	3.44	8.0 (4.0, 13.0)
prestige	2.79	6.0 (4.6, 15.4)
gift	1.71	7.0 (2.0, 11.4)

N=17, Freidman test statistic $\chi^2(7) = 55.069$; $p<0.000$

6.3.3.2 Disease impact on small stock benefits

The six most important diseases in sheep and goat were identified and reported in section 3.3.1 of Chapter 3 titled "Community appraisal of *Peste des petits ruminants* and validation of the community knowledge in Turkana County". Analysis of how the goat disease impacted on the small stock benefits showed that Peste des petit ruminants

(PPR) (*Lomoo*) had the highest disease impact score of 34 and thus was ranked number one disease that destroyed the benefits derived from goats. The disease PPR was described as one that easily finished herds of goats and thus was feared by the community. Other diseases ranked in order of importance were contagious caprine pleura-pneumonia (CCPP) (*Loukoi)* with score 19, Thin goat syndrome (*Loutogonyen*) with score of 14, Diarrhea (*Naosin*) characterized with soiling of the anal region had a score of 9, Bottle jaw (*Loborbolio*) had a score of 2.9 while Pasteurelosis (*emany*) was lowly scored at zero (Table 6.12). The association of goat diseases and perceived contribution of negative impact was statistically significant ($p<0.000$) for each of the benefit.

Table 6.12: Goat disease impact matrix scoring

	Median scores (10th and 90th percentiles)					
Benefits	CCPP (*Loukoi*)	PPR (*Lomoo*)	Thin Syndrome (*Loutogonyen*)	Pasteurellosis (*Emany*)	Diarrhea (*Naosin*)	Bottle Jaw (*Loborbolio*)
Meat***	3.0 (0.0, 6.8)	5.0 (1.8, 10.2)	3.0 (0.8, 7.2)	0.0 (0.0, 2.9)	2.0 (0.8, 5.0)	1.9 (0.0, 3.6)
Milk***	3.0 (0.0, 7.2)	5.0 (2.4, 10.5)	3.0 (0.8, 7.1)	0.0 (0.0, 1.3)	2.0 (0.8, 5.3)	1.0 (0.0, 3.2)
Survival ability***	2.0 (0.0, 9.0)	4.0 (0.8, 11.8)	2.0 (0.0, 5.0)	0.0 (0.0, 1.6)	1.0 (0.0, 3.4)	0.0 (0.0, 2.2)
Sale income***	2.0 (0.0, 6.3)	3.0 (1.0, 7.3)	1.2 (0.0, 4.0)	0.0 (0.0, 1.2)	1.0 (0.0, 3.2)	0.0 (0.0, 2.2)
Prestige/honour***	3.0 (0.0, 6.1)	3.9 (1.0, 5.6)	1.0 (0.0, 3.4)	0.0 (0.0, 0.0)	0.0 (0.0, 2.0)	0.0 (0.0, 0.2)
Production***	2.0 (0.0, 5.2)	4.0 (1.6, 11.2)	2.0 (0.0, 4.8)	0.0 (0.0, 1.8)	2.0 (0.0, 5.2)	0.0 (0.0, 2.0)
Dowry***	3.0 (1.0, 5.8)	5.0 (2.8, 8.2)	1.9 (0.0, 7.5)	0.0 (0.0, 2.12)	1.9 (0.0, 4.21)	0.0 (0.0, 2.2)
Skin***	0.0 (0.0, 2.4)	3.0 (0.0, 10.6)	1.0 (0.0, 4.4)	0.0 (0.0, 0.9)	0.9 (0.0, 4.0)	0.0 (0.0, 1.0)
Gifting***	1.0 (0.0, 3.3)	2.0 (0.0, 6.6)	0.0 (0.0, 1.4)	0.0 (0.0, 0.0)	0.0 (0.0, 1.4)	0.0 (0.0, 0.2)
disease impact	**19**	**34**	**14**	**0**	**9**	**2.9**

*P<0.05;**P<0.01; ***P<0.001. No asterix means P>0.05

As shown in Table 6.13, the six sheep diseases identified by respondents to have major impact on sheep derived benefits were PPR (*Lomoo*) with a score of 31, sheep pox (*Etune*) with a score of 21.7, Anaplasmosis (*Lonyang*) score of 9, Bottle jaw (*Loborbolio*) with score of 7, anthrax (*Lookot*) with score of 6.9 and Foot and mouth (*Lojaa*) with score of 5. The disease PPR was ranked as the most destructive disease of the sheep derived benefits. The sheep disease association with perceived contribution to negative impact was statistically significant ($p<0.000$) for each of the benefit. Lose of livelihoods benefits from livestock are not only occasioned by death of livestock but also by debilitating nature of the disease that can render the animal unproductive in all it aspect despite surviving.

Table 6.13: Sheep disease impact matrix scoring

	Median scores (10th and 90th percentiles)					
Benefits	Anaplasmois (*Lonyang*)	PPR (*Lomoo*)	Foot & mouth (*Lojaa*)	Sheep pox (*Etune*)	Anthrax (*Lookot*)	Bottle jaw (*Loborbolio*)
Oil**	2.0 (0.0, 6.3)	5.0 (0.0, 7.5)	1.0 (0.0, 7.5)	2.0 (0.0, 5.0)	1.9 (0.0, 3.4)	2.0 (0.0, 7.4)
Meat***	3.0 (0.0, 5.0)	4.0 (1.6, 11.3)	2.0 (0.0, 4.0)	3.0 (0.0, 5.0)	2.0 (0.0, 5.6)	2.0 (0.0, 5.5)
Milk***	2.0 (0.0, 3.2)	4.0 (2.1, 10.7)	2.0 (0.0, 5.1)	2.7 (0.0, 5.6)	1.0 (0.0, 3.0)	1.0 (0.0, 6.5)
Skin***	0.0 (0.0, 5.2)	0.0 (0.0, 5.3)	0.0 (0.0, 0.4)	6.0 (1.6, 10.2)	0.0 (0.0, 0.2)	0.0 (0.0, 0.2)
Productivity***	1.0 (0.0, 4.1)	5.0 (0.8, 11.9)	0.0 (0.0, 5.6)	6.0 (1.4, 13.1)	1.0 (0.0, 6.5)	1.0 (0.0, 6.1)
Dowry***	1.0 (0.0, 4.0)	5.0 (1.6, 12.2)	0.0 (0.0, 2.2)	1.0 (0.0, 4.4)	1.0 (0.0, 3.6)	1.0 (0.0, 4.2)
Gift***	0.0 (0.0, 2.2)	4.0 (0.0, 5.1)	0.0 (0.0, 1.0)	1.0 (0.0, 2.0)	0.0 (0.0, 2.2)	0.0 (0.0, 3.3)
Prestige***	0.0 (0.0, 2.2)	4.0 (1.6, 10.0)	0.0 (0.0, 1.0)	0.0 (0.0, 3.0)	0.0 (0.0, 3.2)	0.0 (0.0, 2.4)
Disease impact	**9**	**31**	**5**	**21.7**	**6.9**	**7**

*P<0.05;**P<0.01; ***P<0.001. No asterix means P>0.05

6.3.4 Direct economic losses associated with PPR disease in Turkana County

In order to estimate the direct economic losses due to PPR disease small stock productivity parameters in Turkana pastoral production system were estimated through participatory epidemiology and secondary data. Table 6.14 provides the estimates of the parameter values used in the calculation of direct losses due to PPR in Turkana County. The losses due to mortality of sheep and goats occasioned by PPR were in the tune of 5.6 billion Kenya shillings accounting for 52.5% of the total losses. Proportion of sheep mortality losses were placed at 2.3 billion while goat contributed 3.3 billion Kenya shillings in mortality losses. Milk losses due to PPR were estimated at 4.4 billion Kenya shillings for both sheep and goats accounting for 38.9% of the total losses. Sheep proportion of milk losses was 1.3 billion while milk losses in goats were 3.1 billion Kenya shillings. Weight losses attributed to PPR disease caused an estimated loss of 0.9 billion Kenya shillings in both sheep and goats. Major weight losses were recorded in goats at 0.7 billion Kenya shillings while sheep weight losses were in the tune of 0.2 billion Kenya shillings. Opportunity cost of local treatments of PPR in Turkana accounted for 25 million Kenya shillings.

Table 6.14: Parameter values for estimating direct loses due to Peste des petit ruminant in Turkana

Parameter description	Parameter values							
	Sheep				Goat			
Herd age structure	New born Lambs (Imethek)	Older lambs (Nanyang)	Young Adult (Amethek Nakale)	Adult (Amethek Naapolon)	New born kids (Ikale)	Older kids (Namenaoei)	Young adult (Akale)	Adults (Akine)
$G^{y \cdots a}$ = proportion (%) of Sheep/goats in ages group young to adult	15	21	25	39	18	21	22	39
Losses due to mortality								
$H^{y \cdots a}$ = PPR mortality (%) in sheep/goats age groups young to adult	18	20	20	22	16	17	16	19
$C^{y \cdots a}$ = sale value (KES) of sheep/goats in age groups young to adult	700	1500	2500	5000	700	1500	2500	5000
Losses from reduced milk production								
L^{f} = proportion (%) of lactating sheep/goats				33				42
P^{f}= proportion (%) of pregnant in sheep/goat				18				17.5
$I^{y \cdots a}$= prevalence (%) of PPR in sheep/goats	21	24	22	27	19	20	20	22
I^{af}= prevalence (%) of PPR in lactating sheep/goats				27				22
V^{l} = volume of milk loss (litres) per day per PPR case				0.3				0.4
T^{r} = duration (days) of reduced milk production per acute PPR case				20				20
V^{a} = daily volume (litres) of milk produced per healthy sheep/ goat				0.49				0.73
T^{l} = lactation period (days)				120				120
C^{m} = sale value of milk (KES/l)				50				50
C^{me} =sale value of meat in loca/ shops (KES/kilogram)				320				320
Losses due to weight losses								
W^{l} = proportion of body weight loss				0.3				0.3
$W^{a \cdots y}$ = Average body weight (kilo grams) of sheep/goat	2.5	4.5	10	30	2.5	5	12.5	35
K = Inter-kidding period (days)				252				344
B = Birth weight (kilograms) of Kid/Lamb	1.9				2.1			
N = Sheep/Goat population in study area in Turkana County	3,517,151				5,994,861			
Q=Delay in conception due to disease months				5.5				5.5

The total estimated direct economic losses associated with PPR for sheep and goats in Turkana County that were derived from the spreadsheet model were in tune of 11.1 billion Kenya shilling (Table 6.15). Production losses in sheep were placed at 3.9 billion Kenya shillings accounting for the 35% of total direct economic losses. The production losses in goat herds were 7.2 billion accounting for 65% of the total direct economic losses.

Table 6.15: Calculated production losses associated with PPR in sheep and goats.

	Losses in Kenya shillings	
	Sheep	Goat
Mortality		
$L^d = ((G^{y\ldots a} \times H^{y\ldots a} \times C^{y\ldots a}) \times (N/100)$	2,236,556,321	3,190,524,973
$L^y = (P^f \times H^f \times C^y) \times (N/100)$	97,495,426	139,530,390
$L = L^d + L^y$	**2,334,051,747**	**3,330,055,363**
Milk losses		
$M^d = ((L^f + P^f) \times H^a \times V^a \times T^l \times C^m) \times (N/100)$	1,160,195,566	2,968,409,378
$M^r = (L^f \times (I^{af} - H^a) \times V^l \times T^r \times C^m) \times (N/100)$	17,409,897	30,214,099
$M^y = ((L^f + P^f) \times (I^a - H^a) \times (12/K-12/(K+Q)) V^a \times T^l \times C^m) \times (N/100)$	146,243,139	158,229,925
$M = M^d + M^r + M^y$	**1,323,848,602**	**3,156,853,402**
Losses due to weight losses		
$W^d = (G^{y\ldots a} \times (I^{y\ldots a} - H^{y\ldots a}) \times W^l \times W^{y\ldots a} \times C^{me}) \times (N/100$	230,967,086	693,485,520
$W^y = ((L^f + P^f) \times (I^a - H^a) \times (12/K-12/K+Q) \times B \times C^{me}) \times (N/100)$	30,243,479	24,276,372
$W = W^d + W^y$	**261,210,565**	**717,761,893**
Opportunity cost		
$O^c = (G^{y\ldots a} \times (I^{y\ldots a} - H^{y\ldots a}) \times (.025 * C^{y\ldots a})) \times (N/100)$	11,057,043	14,047,458
$O = O^c$	**11,057,043**	**14,047,458**
Total direct economic losses for field herd in Turkana		
$T^L = L + M + W + O$	**3,930,167,957**	**7,218,718,116**
Combined total loss	**11,148,886,072**	

Sensitivity analysis for the model herd was carried out by adjusting upwards mortality, morbidity, milk loss per PPR case and proportion weight loss per PPR case by 5%, 10%

and 20%. The result of sensitivity analysis model showed a similar increase in proportion of the total cost for all the parameters altered (Table 6.16).

Table 6.16 Sensitivity analysis for direct economic losses due to PPR for the model herd

	Increment on total cost (KES) % in parenthesis as the effect of increasing parameter by		
Disease Parameter	5%	10%	20%
Mortality & Morbidity	+11,607.96 (5%)	+23,215.96 (10%)	+46,431.96 (20%)
Milk loss per PPR case	+49.96 (0.022%)	+99.96 (0.043%)	+199.96 (0.086%)
Proportion weight loss due to PPR	+906.96 (0.391%)	+1,813.96 (0.781%)	+3626.96 (1.562%)

6.4 Discussion

Following the onset of PPR outbreaks in Turkana County in 2006, the disease triggered relatively large economic losses to Turkana pastoral herders. The PPR disease entrenched itself and became endemic in Turkana County thus causing cyclic outbreaks that perpetually resulted in continuous economic losses to the herders. At the county and national level, data on livestock productivity in Turkana is scarce and rarely updated. Turkana County being extreme rural pastoral area with insecurity, poor infrastructure and communication, has provided very little incentive for the successive governments to collect data on livestock productivity in the region. This study marks a useful start in developing a system for the economic assessment of PPR based on parameters derived from participatory epidemiological approaches for the mathematical model.

This study has established that the Turkana community relies heavily on their livestock and all their socio- cultural activities revolve around livestock as earlier indicated by FAO (2012a). Wealth and a person's standing in the community are judged based livestock owned and what livestock has contributed particularly for the number of wives married and children born by the wives (McCabe, 1984). Approximately a quarter of the Turkana people without livestock are perceived to be poor. Small stock that is sheep and goats constitute the largest herd of livestock reared by Turkana with herd composition ranging between 42 to 64.4% of the total herds across the wealth groups. Livestock-keeping remains the major livelihood activity for the livestock keepers contributing significantly to food, cloth/ornaments and other consumer services (Imana, 2008). Of all the food consumed by the Turkana 29.4% consists animal products which are well distributed in the household with the major youthful workforce of morans and girl herders in the age group 13 to 18 consuming most of the animal products.

The Turkana identify diseases as the second most important small stock herd decrement factor. Diseases deprive the community from accessing livestock generated benefits. Based on this premise *Peste des petit ruminants* was ranked as the disease with highest destructive impact on the small stock benefits to the Turkana community.

The losses due to PPR disease that impact on small stock benefits were mainly due to disease mortality, morbidity, accompanying milk and weight losses. These losses

encompass the direct losses that were incorporated in the calculation of the economic loses due to PPR in the spread sheet model. Early estimates of economic losses due to PPR reported by government of Kenya were in tune of US$ 15 million equivalent to KES 1.275 billion (GOK, 2008; Nyamwea *et al.,* 2009). However this study has established that the direct economic losses due to PPR in Turkana County alone for the year 2010 were in the tune of KES 11.1 billion. This shows that earlier national estimates of PPR disease loses had been under- estimated.

6.5 Conclusions

PPR remain a major economic disease affecting the Turkana herders. As the wealth groups lose their small stock due PPR they begin a perilous journey of joining the poor categories resulting in disruption of cultural set up and economy. The disease has the potential of destroying livelihoods and reducing most herders to destitution consigning them into the ever growing internally displaced camps of economically challenged people.

CHAPTER 7: PERCEPTION OF TURKANA COMMUNITY ON CONTROL OF PESTE DES PETITS RUMINANTS AND MODELING VACCINATION OF PPR IN TURKANA COUNTY KENYA.

7.1 Introduction

Peste des petits ruminants is characterized by high morbidity and mortality of immunologically naïve sheep and goats. *Peste des petits ruminants* has caused major outbreaks in East Africa more so in Kenya (Kihu *et al.,* 2012a; Muse *et al.,* 2012; Luka *et al.,* 2011). Currently there are major international efforts being undertaken by several animal health institutions to develop sound control and eradication strategies (FAO, 2014; FAO, 2012b; Baron, 2012; Elsawalhy *et al.,* 2010).

A number of features of PPR virus make it easy to control or eradication being that it is transmitted by direct contact between the infectious and healthy animals, has one sero-type divided into four lineages, and there is cross immunity between them (Berhne, 2006). Sanitary control measures and medical prophylaxis (vaccination) has been in application to control and eradicate the disease.

Implementation of these PPR control measures in Kenya such as quarantine, livestock movement restrictions and vaccination have been going on since the first outbreak in 2006 in to stop the spread of PPR. However these measures have not stemmed the spread of the PPR because they are implemented haphazardly due to lack of

epidemiological information. Various vaccination exercises that have been carried out particularly during the period of 2007 and 2008 were viewed as reactionary due to their lack of strategic implementation. Chapter three of this thesis "Herd immunity in Turkana County" reports that the first round of vaccinations carried out between 2007 and 2008 in Turkana resulted in 1,380,283 small stock vaccinated. The initial thinking was that 50% of the small stock was covered. However, following the livestock census of 2009, it was established that small stock population in Turkana County was 9,512,012 and therefore the initial vaccination of the small stock herds had only coverage of 14% of small stock population in Turkana County due to prior lack of comprehensive population data.

The effectiveness of PPR control measures is thus dependent on host of epidemiological information available to the implementers of disease control programs (Mariner *et al.,* 2005). In this study Turkana community perception on PPR control options are discussed. The study also suggests that a better understanding of the transmission dynamics of PPR virus within herds could help improve effectiveness of PPR control. In this regard a simple participatory compartmental disease model is described with disease parameters derived from participatory methods, serology and secondary data.

7.2 Material and Methods

7.2.1 Study area

The study location is Turkana County that borders internationally with Ethiopia, Sudan and Uganda and internally borders Marsabit, Samburu, West Pokot and Baringo Counties previously described and depicted in section 3.2.1 and Figure 3.1 of Chapter 3.

7.2.2 Sampling unit and selection of study sites

The sampling unit was an *Adakar* as described in section 3.2 of Chapter 3 titled "Community appraisal of *Peste des petits ruminants* and validation of the community knowledge in Turkana County". The study sites were well distributed in the six administrative divisions that were a focus for the study as shown in Figure 3.2.

7.2.3 Data collection

7.2.3.1 Key informants interviews

Key informants interviews were conducted as described in section 3.3.1 of Chapter 3 titled "Community appraisal of *Peste des petits ruminants* and validation of the community knowledge in Turkana County"

7.2.3.2 Focus group discussion

The focused group discussion was carried out as described in section 3.3.2 of Chapter 3.

7.2.3.2.1 Semi Structured Interviews

The semi-structured interview (SSI) was carried out described in section 3.3.2 of Chapter 3.

7.2.3.2.2 Sustainability matrix of PPR local control methods

Turkana community analysis of local PPR control methods was carried out using a sustainability matrix. The several local PPR control methods practiced by Turkana community were debated by the respondents and later listed as follows: tradition hot stone (*Amoru*); local medicinal plants *Egis (Cissus guadrangularis), Emus (Euhorbia Uhligiana), Eichuchuka (Aloe Turkanensi);* Human antibiotic capsules, traditional quarantine/herd isolation and conventional PPR vaccination. Similar livestock disease control methods have been reported in other studies (Ohta, 1984; ITDG and IIRR, 1996; Msafiri, 1996; Bosch 2006). The focus group discussion respondents were then provided with disease control sustainability criteria that was initially generated and agreed upon in a key informants' interview. The disease control sustainability criteria composed of the following: accessibility of the control measure, its effectiveness in disease control, affordability, ability to use with ease, local knowledge of control method, commitment to provide labor in participation and commitment to finance the control measure. The respondents were given hundred counters and asked to score the sustainability criteria based on their importance. The criterion with more importance got more counters and thus carried more weight. After weighting the criteria for sustainability of local disease control methods, a matrix was drawn on the ground where the x-axis was represented by PPR control methods while the y-axis had

sustainability criteria. Each criterion had counters that represented its weight. The respondents were asked to score the local PPR control methods using counters in each weighted criterion based on its prominence on the control methods. This was repeated for the whole criteria. The scores for each PPR control method were then totaled thus providing a measure of sustainability. The higher the score the more sustainable the disease control method was perceived by local community.

7.2.3.3 Peste des petits ruminants transmission model

7.2.3.3.1 Model structure

Peste des petits ruminants virus elicits permanent immunity on the recovered animals. The recovered mothers will confer passive maternal immunity against PPR to their young ones by transfer of IgG antibodies across the placenta and through colostrum. The maternal antibodies remain in the body up to four and half months. Considering that farm life expectancy of sheep and goats is on average four years, the four months of passive immunity before it decays translates into 8.33% of small stock life thus the need to include the passive maternal immunity class in the compartmental transmission model.

A state-transmission model was developed on Berkeley Madonna™ (2000) software version 8.0.1. The model has five compartments depicting temporal immunity M, susceptible S, exposed E, infectious I and recovered R states for a population size N (Figure 7.1).

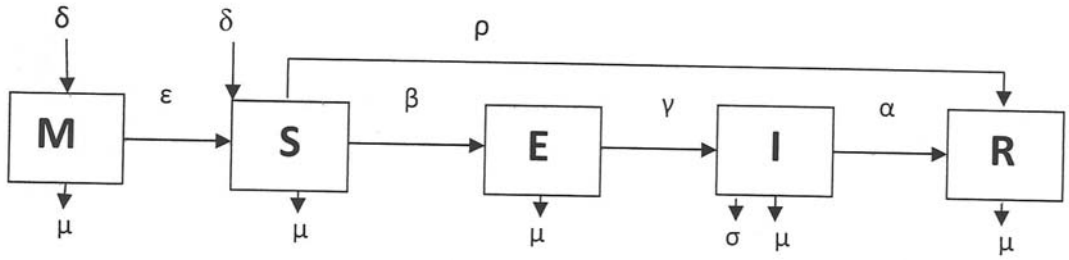


Figure 7.1: Peste des petits ruminants MSEIR model structure for sheep or goats (Hethcote 2000).

Assumptions in developing the model are that there is homogenous mixing of all states compartments of both species in an open population, the non-specific mortality was set equal to birth rate and there is no age structure in this model. In the initial stages of the model development each species (sheep and goats) were treated as separate populations each with its own parameters and each model ran separately (Figure 7.1).

The parameters within the composite PPR MSEIR model transfer diagram are described based on descriptive made by Mariner *et al.,* (2005) for rinderpest model.

Non-specific mortality (μ) occurs in all five states. All births rates (δ) enter in the susceptible (S) as well as temporal immune (M) state. Temporal immunity is lost at a rate (ε) in (M) for an animal to become susceptible. Only infectious (I) animals experience PPR mortality at rate (σ). The rate at which exposed animals (E) become infectious is described by γ respectively. The rate at which infectious (I) become resistant (R) is described by α. Vaccination is modeled as a transition from the susceptible state directly to the resistant state at the immunization rate (ρ). The rate at which animals move from the susceptible to exposed state (E), is governed by the effective contact rate (β).

At any given time (t) the homogeneous population of size N(t) is categorized into disease status of five compartments; M(t) S(t), E(t), I(t) and R(t) as passively immune newborn, susceptible, exposed, infectious and the immune individuals, where t represents the time. If β is the average number of adequate contacts (i.e., contacts sufficient for transmission) of a sheep or goat, so that the force of infection β (I/N) is the average number of contacts with infectives of one susceptible per unit time; then the incidence (the number of new cases per unit time) of the S susceptibles is βSI/N.

The model is formulated using mass action differential equations however the deterministic representation of the model is as follows:-

$$dM/dt = \delta (N - S) - (\varepsilon + \mu)M, \quad (1)$$

$$dS/dt = \delta S + \varepsilon M - \beta SI/N - \mu S, \quad (2)$$

$$dE/dt = \beta SI/N + \beta SI1/N1 - (\gamma + \mu)E, \quad (3)$$

$$dI/dt = \gamma E - (\sigma + \mu)I, \quad (4)$$

$$dR/dt = \alpha I + \rho S - \mu R, \quad (5)$$

$$dN/dt = (\delta - \mu)N. \quad (6)$$

However the two species are herded together and mixed freely in reality and each species could infect the other thus a final composite model consisting of the sheep and goat models combined was developed (Figure 7.2). The parameters with a suffix **1** are for the goat component of model.

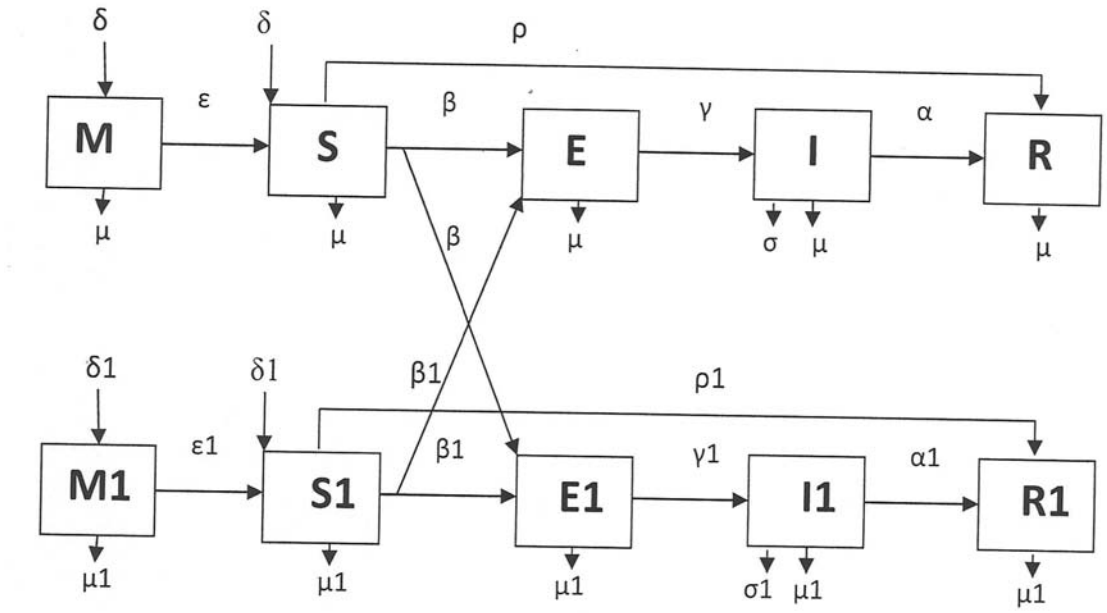


Figure 7.2: Peste des petits ruminants MSEIR model structure of sheep and goats in mixed herd.

Non-specific mortality (μ) and (μ1) occurs in all five states. All births rates (δ) and (δ1) enter in the susceptible (S) and (S1) as well as temporal immune (M) and (M1) state. Temporal immunity is lost at a rate (ε) and (ε1) in (M) and (M1) respectively for an animal to become susceptible. Only infectious (I) and (I1) animals experience PPR mortality at rate (σ) and (σ1) respectively. The rate at which exposed animals (E) and (E1) become infectious is described by γ and γ1 respectively. The rate at which infectious (I) and (I1) become resistant (R) and (R1) is described by α and α1. Vaccination is modeled as a transition from the susceptible state directly to the resistant state at the immunization rate (ρ) and (ρ1). The rate at which animals move from the susceptible to exposed state (E), is governed by the effective contact rate (β) and (β1). The differential equations for composite model are shown below:

$$dM/dt = \delta (N - S) - (\varepsilon + \mu)M, \quad (7)$$

$$dS/dt = \delta S + \varepsilon M - \beta SI/N - \mu S, \quad (8)$$

$$dE/dt = \beta SI/N + \beta SI1/N1 - (\gamma + \mu)E, \quad (9)$$

$$dI/dt = \gamma E - (\sigma + \mu)I, \quad (10)$$

$$dR/dt = \alpha I + \rho S - \mu R, \quad (11)$$

$$dN/dt = (\delta - \mu)N. \quad (12)$$

$$dM1/dt = \delta 1 (N1 - S1) - (\varepsilon 1 + \mu 1)M1, \quad (13)$$

$$dS1/dt = \delta 1 S1 + \varepsilon 1 M1 - \beta 1 S1I1/N1 - \mu 1 S1, \quad (14)$$

$$dE1/dt = \beta 1 S1I1/N1 + \beta 1 S1I/N - (\gamma + \mu)E, \quad (15)$$

$$dI1/dt = \gamma 1 E1 - (\sigma 1 + \mu 1)I1, \quad (16)$$

$$dR1/dt = \alpha 1 I1 + \rho 1 S1 - \mu 1 R1, \quad (17)$$

$$dN1/dt = (\delta 1 - \mu 1)N1. \quad (18)$$

Determination of the success (persistence and spread) or extinction of any infectious organism in a host population is fundamentally dependent on reproductive number, R_0, which measures the potential for spread of infection. It is defined as the average number of new infections caused when an infection enters an entirely susceptible population (Anderson & May 1991). Basic reproductive number has an average value that can change from population to population or over time, depending on the patterns of contact or biological influences at play (Kivaria *et al.,* 2013). The reproductive number has a threshold value of 1 to ensure persistence of the infection, <1 for infection to die out or >1 for infection to spread exponentially.

The basic reproduction number R_0 for this MSEIR model is given by $R_0 = \beta \gamma / (\alpha + \sigma + \mu)(\gamma + \mu)$ (Swinton *et al.,* 1998). This R_0 is the product of the contact rate β per unit

time, the average infectious period $1/(\alpha + \sigma + \mu)$, and the fraction $\gamma/(\gamma + \mu)$ of exposed sheep or goats surviving the latent class E (Hethcote 2000; Mariner *et al.,* 2005).

An estimated range of R_0 was determined from the data of PPR outbreak in Turkana in 2007 (OIE 2014), participatory epidemiology data on morbidity and mortality reported in Tables 3.8 and 3.9 of Chapter 3 and serology data reported in Table 4.9 of Chapter 4 of this thesis. The R_0 values determined serology and mortality data in this study were used to estimate the distribution of values used for β which were directly incorporated in the model

7.2.3.3.2 Estimation of initial model parameters

7.2.3.3.2.1 Deterministic estimation of reproductive number, R0

In this study an estimate of R_0 was also determined from three types of data using two methods:-

1. As described by Heffernan and Wahl (2005), and using reported morbidity and case fatality risk associated with PPR according to the formula:

   $R_0 = 1 + r_0/a_0$

   where r_0 is the reported PPR morbidity and a_0 is the average PPR case fatality risk.

Estimates of *Peste des petit ruminants* relative incidences, mortality and case fatality rates reported in Tables 3.8 and 3.9 were reorganized and presented in Table 7.1 for use in calculating R_0.

Table 7.1: Estimated PPR incidence, mortality and case fatality by proportional piling method of participatory epidemiology

	Median scores (10th and 90th percentiles)	
	Goats herd	Sheep herd
Estimated Incidence of PPR	19.6 (10.5, 30.0)	20.3 (10.1, 31.5)
Mortality of PPR	15.9 (6.6, 27.6)	17.1 (7.7, 29.4)
Case fatality due to PPR	79.7 (63.3, 96.8)	81.7 (64.1, 94.3)

Based on proportional piling PPR mortality and morbidity data generated from participatory epidemiology in Turkana, the R_0 was estimated to be 1.25 for both sheep and goats.

The initial PPR disease outbreak data from Turkana was reported to the World Organization for Animal Health (OIE) and included location of the outbreak, susceptible population of sheep and goats, cases of PPR observed, mortality, morbidity and case fatality (OIE, 2014). The PPR outbreak data was been reorganized and presented in Table 7.2 and 7.3 and used for determination of the R_0.

Table 7.2: Outbreak data of PPR in goats in Turkana reported to OIE in 2007 (OIE 2014)

Division	Location	Susceptible	cases	Deaths	Morbidity	Mortality	Case fatality
Oropoi	Loreng	11816	566	525	0.05	0.04	0.93
Oropoi	Moruarengan	10000	600	450	0.06	0.05	0.75
Oropoi	Letea	13747	2474	1590	0.18	0.12	0.64
Loki	Nanam	16759	2709	1827	0.16	0.11	0.67
Lorae	Lokangae	20000	770	507	0.04	0.03	0.66
Kibish	Lokamarinyang	28072	1437	1179	0.05	0.04	0.82
Lapur	Meiyan	13399	804	482	0.06	0.04	0.60
Kainuk	Kaputir	10506	3502	3152	0.33	0.30	0.90

Table 7.3: Outbreak data of PPR in sheep in Turkana reported to OIE in 2007 (OIE 2014)

Division	Location	Susceptible	cases	Deaths	Morbidity	Mortality	Case fatality
Oropoi	Natira	3500	1800	890	0.51	0.25	0.49
Oropoi	Songot	3000	1600	800	0.53	0.27	0.50
Oropoi	Loreng	3184	152	142	0.05	0.04	0.93
Oropoi	Letea	6253	1126	723	0.18	0.12	0.64
Loki	Nanam	13241	2141	1443	0.16	0.11	0.67
Kibish	Lokamarinyang	21928	1123	921	0.05	0.04	0.82
Lapur	Meiyan	11601	696	418	0.06	0.04	0.60
Kainuk	Kaputir	4494	1498	1348	0.33	0.30	0.90

Using the above data from PPR outbreak of 2007, the R_0 was estimated to be in range of 1.05 and 2.07 in sheep while that of goat was in the range 1.05 and 1.28.

2. R_0 was also estimated from the serology data. The deterministic critical herd immunity threshold (h) required to interrupt disease transmission can be back calculated from R_0 using the simple relationship (Anderson and May, 1991) as indicated below.

   $h=1-1/R_0$

   Derived formula to calculate R_0 was

   $R_0=1/(1-h)$

Serology data from the herd immunity chapter of this study also provided useful data that were used in deriving some of the model parameters. The goats had sero positivity of 39.6% (95% CI: 35.46% to 43.88%) while the sheep were at 31.6% (95% CI: 27.20% to 36.20%).

The R_0 estimated from serological data from Turkana sheep and goats in method earlier reported by Mariner *et al.*, (2005) was 1.46 for sheep and 1.66 in goats.

7.2.3.3.2.2 Estimation of latent, infectious and passive immunity period for PPR infection

The latent, infectious and passive immunity periods in sheep and goats were estimated from secondary data as reported in Table 7.4.

Table 7.4: Some of the estimated model parameters and their source

Parameter	duration	Reference
Latent period	2 to 3 days	Gopilo (2005)
	3 days	Coucy-Hymann et al. (2007)
	5 to7days	Osman et al. (2009)
	3days	Balamurugan et al. (2010)
	1 day	Truong et al. (2014)
Infectious period	12 to 17 days	Coucy-Hymann et al. (2007)
	13 to 15 days	Osman et al. (2009)
	14 days	Balamurugan et al. (2010)
	11 days	Truong et al. (2014)
Maternal antibody decay	Kids 4 months	Balamurugan et al. (2012)
	Kids 4.5 months	Awa et al. (2003)
	Lambs 75 days to 90 days	Bodjo et al. (2006)
	Lambs 3.5 months	Awa et al. (2003)

In several other reports it was indicated that the course of the disease is 20 days (Scott et al., 1986; Bundza et al., 1988; Truong et al., 2014). The infectious period was thus calculated taking into account that the disease resolve at 20^{th} day post infection.

7.2.3.3.2.3 Estimation of sheep and goats herds structures and productive parameters

Proportion piling was also used to establish proportion of productive females and birth rates as reported in Table 6.7 of Chapter 6 of this thesis (Table 7.5). However some of the data on reproductive rates of sheep and goats was estimated from secondary data

(Njanja 1991). Reproductive rates in sheep and goats of Turkana were reported to be 1.44 and 1.1 young ones per year respectively (Njanja, 1991).

Table 7.5: Proportional piling estimate of herd composition in terms of proportion of females, productive females and age they leave the herd.

	Median scores (10th and 90th percentiles)			
	Pregnant	Non pregnant	Age Female leaves herd	Sex Structure Female
Goat	42.0 (5.0, 81.0)	58.0 (19.0, 94.0)	4.0 (2.0, 4.0)	71.0 (53.0, 80.0)
Sheep	54.0 (4.0,75.0)	46.0 (25.0, 96.0)	3.5 (1.5, 4.0)	77.0 (63.0, 84.0)

7.2.3.3.3 Derived parameters from estimated parameters

Derived parameters (Table 7.6) were calculated from the estimated parameters described above .

- Rate of decay of passive immunity.

The maternal passive immunity periods in sheep and goats were denoted by E and they varied between sheep and goats. In sheep passive maternal immunity is the range of 75 days to 105 days (Bodjo *et al.,* 2006; Awa *et al.,* 2003) while in goats it was in the range of 120 days to 135 days (Balamurugan *et al.,* 2012; Awa *et al.,* 2003). The mean transition rate from passive immunity to susceptible status denoted by $\varepsilon = 1/E$ and determined be in range of 0.0095 and 0.03 for sheep and 0.007 and 0.008 for goats.

- The rate of loss of latency.

The latent period is defined as the period between the time PPR virus is introduction into the animal and the time the animal start shedding the virus and as such it becomes infectious. The latent period was denoted by (L). The latent period was determined to be 1 to 7 days (Truong *et al.,* 2014; Balamurugan *et al.,* 2010; Osman *et al.,* 2009; Coucy-Hymann *et al.,* 2007; Gopilo, 2005). The rate of loss of latency to animal becoming infection was denoted by $\gamma=1/L$ and determined to be in a range of 0.14 and1 in both sheep and goats.

- Peste des petits ruminants disease specific mortality rate

Peste des petits ruminants disease specific mortality rates in Turkana were determined from two sets of data being the PPR outbreak data reported to OIE in 2007 and participatory epidemiology conducted during this study in 2011. Case fatality rate was derived from PPR mortality rates divided by PPR incidence. The case fatality rate was denoted as (m) and was determined to be within the range of 60% to 93% in goats and 49% to 93% in sheep (OIE, 2014). The case fatality rate determined from participatory epidemiology was 79.7% (63.3%, 96.8%) in goats and 81.7% (64.1%, 94.3%) in sheep and was within the range of case fatality rates determined from OIE derived Turkana data. The PPR infectious animals experienced mortality at rate $\sigma=m/D$ where D was infectious period. Thus σ in sheep was in range of 0.045-0.055 and in goats was 0.055

- Rate of recovery

The transition from PPR infectious state to recovery state in sheep and goat was determined by the infectious period denoted by D. The infectious period was determined to be 11 to 17 days (Truong *et al.,* 2014; Balamurugan *et al.,* 2010; Osman

et al., 2009; Coucy-Hymann *et al.,* 2007). The animal cease to become infectious at recovery rate denoted by $\alpha=(1-m)/D$ and was estimated to be range of 0.004 and 0.046 for sheep and 0.004 0.036 for goats

- Birth rate and non specific mortality rate

Birth rate denoted by (δ) and was estimated from the proportion of pregnant females in the herd and reproductive rates of each species. The birth rate in sheep was determined to be 60% and goats 33% in Turkana small stock. It was assumed that birth rate were equal to non specific death rates (μ). Birth rate δ per day was equal to percentage divided by 365.

- Disease transmission rate

From the calculated R_0 the PPR disease transmission rate denoted as effective contact rate (β) was estimated to be equal to R_0/D (Swinton et al., 1998). The determined value for (β) in sheep was 0.054 and 0.213 while that of goats was 0.063 and 0.152.

Table 7.6: Estimated and derived parameters for PPR MSIER model

Estimated parameter		**symbol**	**value**	
			sheep	goat
	Death rate	μ	0.6	0.33
	Maternal passive immunity in days	E	120-135	75-135
	Latent period days	L	1-7	1-7
	Infectious period days	D	11-17	11-17
	Case mortality	m	0.49-0.93	0.6-0.93
Derived parameter			**sheep**	**goats**
	Rate of decay of passive Immunity	ε	0.0095-0.03	0.007-0.008
	Latency loss	γ	0.14-1	0.14-1
	recovery rate	α	0.004-0.046	0.004-0.036
	PPR mortality rate	σ	0.045-0.055	0.055
	Generation time	G	12-24	12-24
	Transmission	β	0.054- 0.213	0.063-0.152
	Basic reproduction ratio	R_0	1.05-2.07	1.05-1.66
	Birth rates	δ	0.00164	0.000904

7.2.3.3.4 Running the model

7.2.3.3.4.1 Simulation process

The model formulae were checked for consistency and all parameters entered for preparation to make initial verification runs that were carried out for sheep and goats models separately and then the composite model combining both sheep and goat models. The stochastic nature of the model was achieved in the model by have a range of values for each parameter among the β, ε, γ, α and σ. The models were evaluated with different values of each parameter (β, γ, α and σ) while holding other parameters at fixed known values to show how the models behaved and whether changes in the parameters had corresponding changes in proportion of infectious (Ramanathan *et al.,* 2012). In the initial runs the total population (N) was maintained 10,000 for sheep and 15,600 for goats to maintain field ratio of sheep to goat at 1 to 1.56; exposed and infection at 0.1% each while recovered and maternal immunity was initially at zero. The model was run for period of 1460 days (4 years) (unless specified elsewhere) being the average life span of a sheep and goats. This analysis of the models also helped to establish conditions under which the epidemic may manifest and gauge which parameters were most critical in driving the epidemic (O'Neill, 2010). With model parameters (β, γ, α and σ) set at field values listed in table 6, sensitivity analysis was carried out for the maternal antibody, exposed, infectious, recovered, population size and vaccination coverage where each of these compartmental parameters was varied incrementally at constant level while holding other compartmental parameters at fixed known values for

model period of 1460 days. The exposed and infectious were increased in equal numbers from 1 to 40 in steps of 5 with recovered fixed at zero and total population set at 10,000 in sheep and 15,600 in goats. The sensitivity analysis of recovered was done by increasing recovered from zero to 100% at increments of 10%. Impact on increase of population size was completed with increments of 10,000. Under field conditions the inter-epidemic periods and lengths of epidemics were analyzed. Vaccination was analyzed by increasing percent coverage from 0 to 100% at interval of 10% while maintaining a population size of 10,000, recovered at zero, exposed and infectious at 10.

7.2.3.4 Benefit cost analysis of PPR vaccination

Vaccination against PPR remain the most viable control measure against PPR virus since immunity against PPR in lifelong. A Benefit cost analysis of PPR vaccination was developed based on data generated from the modeled economic losses and reported in Table 6.15 of Chapter 6 of this thesis. The key direct losses were calculated based on PPR mortality, milk and body weight losses due to the disease and were estimated at Kenya shillings 11,148,886,072. The estimation of costs for PPR vaccination in Turkana County comprised the costs of the vaccine purchase and importation into Kenya, transportation of vaccine into Turkana County, vaccination equipment and cold chain, training of veterinary workers, supervision costs, and NGO overheads and administrative costs. Annual vaccination using homologous PPR vaccine was assumed, with 100% vaccination coverage in Turkana. The costs associated with mass vaccination were estimated from unpublished report (Peste des petits ruminants (PPR)

emergency control final report 2009) retrieved from office of Director of Veterinary Services in Kenya. From this report 12,621,390 small stock were vaccinated nationally in Kenya at cost of Kenya shillings 420,000,000 in 2009. From these figures vaccination cost per small stock was estimated and was used to estimate the cost of vaccinating all small stock in Turkana County. The benefit–cost analysis (BCA) of PPR vaccination was therefore estimated to be:-

Benefit-cost = estimated economic loss/PPR vaccination costs

The benefit–cost model described above was developed in an MS Excel spreadsheet.

7.3 Results

7.3.1 Sustainability matrix of PPR local control methods

The focused group discussions established that the Turkana community in their own way and based on their local knowledge have attempted to put measures of controlling PPR in their herds. The Table 7.7 lists six approaches of PPR control practiced by the Turkana community each scored against the sustainability criteria. Human antibiotic capsules had a score of 15.92, Aloe is the second highly scored control method with a score of 15.74, traditional massage hot stone (*Amoru*) is third with 13, *Cissus guadrangularis (Egis)* has a score of 12.79, PPR vaccination has a score of 12, *Euhorbia Uhligiana (Emus)* a score of 11.91 and local quarantine/self restriction or community sanctions 9.83 points (Table 7.7).

Table 7.7: Sustainability matrix of PPR control methods as perceived by Turkana community

	Runaway/self or community sanctions	*Cissus guadrangularis (Egis)*	*Euhorbia Uhligiana (Emus)*	Hot stone	Antibiotic Capsules	Aloe *(Aloe Turkanensi)*	Vaccine
Access	2 (.99, 5.52)	2.88 (1, 5.2)	2.88 (.8, 6.2)	3 (.96, 5.95)	2 (0, 5)	3 (1,5.2)	1 (0, 2,91)
effectiveness	2 (0, 4.8)	2 (0, 3.2)	1.02 (0, 3)	2 (0, 6.7)	2 (0, 9.2)	2.04 (.77, 5.2)	3 (1, 6)
Affordability	1 (0,2.93)	2 (0.3.22)	2 (0, 3.1)	2 (0,5.03)	2 (0, 4.4)	2 (0, 3.2)	1 (0, 3)
Easy to use	1.83 (.99, 5.36)	2 (.98, 5.2)	3 (.74, 5)	3 (.95, 5.4)	2 (0, 5.4)	3.7 (1.02, 6)	1 (0, 2)
Local Knows	2 (.99, 4.2)	2 (.8, 4.02)	2 (.73, 3.45)	2 (.99, 5)	2 (0, 6.2)	3 (1, 5)	1 (0, 3.2)
Labour Commit	1 (0, 2.37)	1.9 (0, 3.01)	1 (0, 3.01)	1 (0, 3.32)	1.92 (0, 4)	1.92 (0, 4)	2 (0, 4.48)
Finance commit	0 (0, 2.23)	0 (0, 2.18)	0 (0, 2.52)	0 (0, 2.12)	4 (0, 6)	0 (0, 4.2)	3 (0, 6)
Final aggregate score	**9.83**	**12.79**	**11.91**	**13.00**	**15.92**	**15.74**	**12.00**

7.3.2 Model output

7.3.2.1 Model verification and analysis of beta β values on the impact of infectious as used in the model

The developed MSEIR sheep and goats composite model was ran and produced characteristic picture of the susceptible infectious and recovered (SIR) epidemiological models (Figures 7.3 and 7.4).

Impact of varying model parameter beta β, as shown in Figures 5 to 10 indicates that model infectious curves changed consistently and proportionally to the changes in the β in all the models (sheep, goat and composite models). Similar changes in the other parameters γ, α and σ were also made on the models returning a consistent and proportional changes on model outputs of the infectious curves.

The characteristic of the infectious curves of sheep model (Figure 7.5) showed near complete fade out of the infectious by 366^{th} day for all values of β following the initial outbreak. High values of β above 0.3 generated secondary epidemic peaks which emerged as from 630^{th} day. The β values calculated from field data of sheep indicated that they ranged from 0.054 to 0.213 and at this range of β the infectious faded out within the range of 15.8 to 50 days with no re-emergence of the infection.

The infectious curves of goat model (Figure 7.6) showed one peak of epidemic for each β value. However the outbreaks lasted for longer periods. The β values calculated from field data of sheep indicated that they ranged from 0.063 to 0.152. Within this range of β it was found that the infectious faded out at 112 days for β=0.06 and at 860 days for

β=0.09 days. For all the other values of β from field data the infectious did not fade out by end of model period at 1460 (four years).

The composite model of sheep and goat produced four infectious curves outputs. The first output was sheep infectious curve derived from range of β values of sheep applied to the composite model while maintaining the model to pick random β values for the goat portion of composite model (Figure 7.7). This first output being sheep infectious curves has one epidemic peak and the infectious do not fade out for all values of sheep β by the end of the model period of 1460 days. However the second output a goat infectious curve is derived while maintaining the conditions

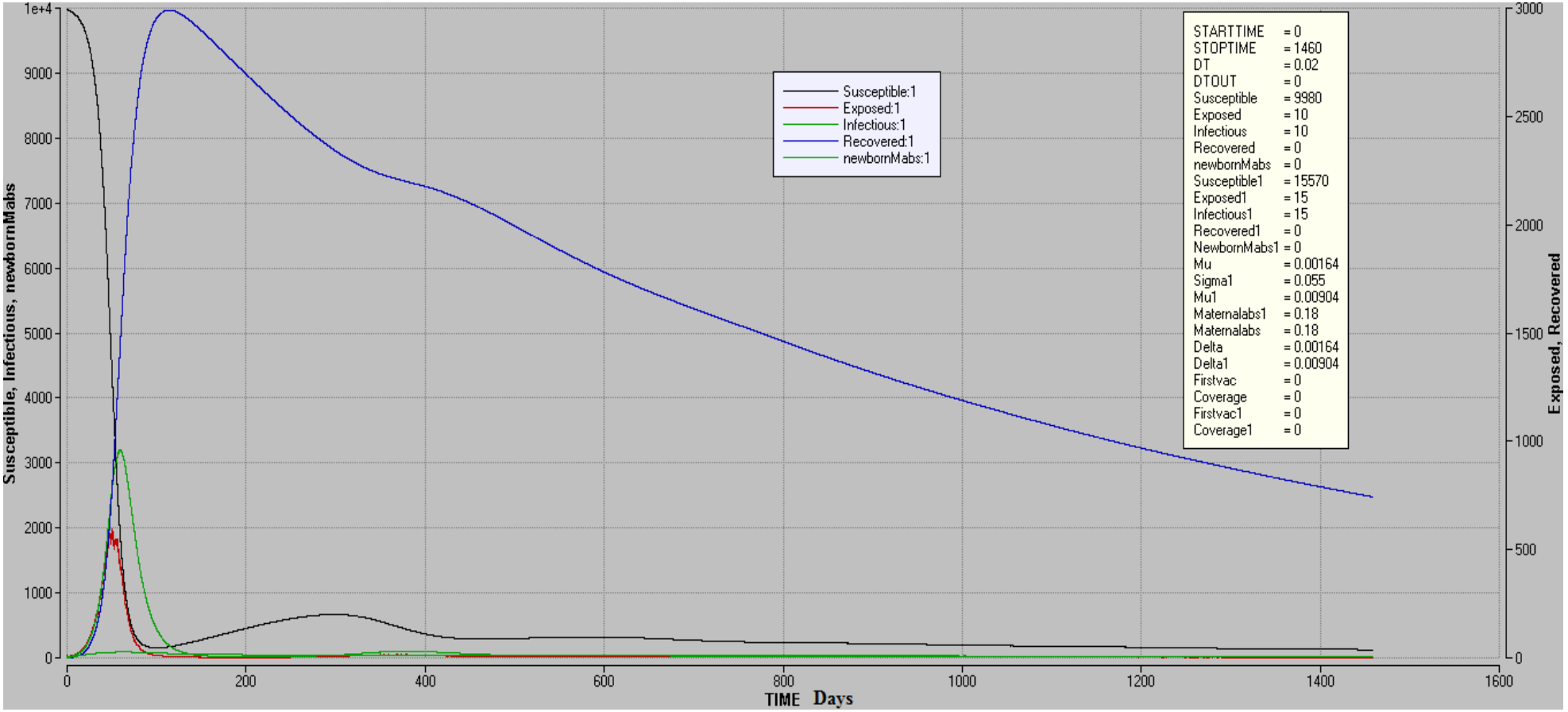


Figure 7.3: Plot of sheep epidemiological parameters changes in Sheep/Goat composite MSIER model over 4 year period

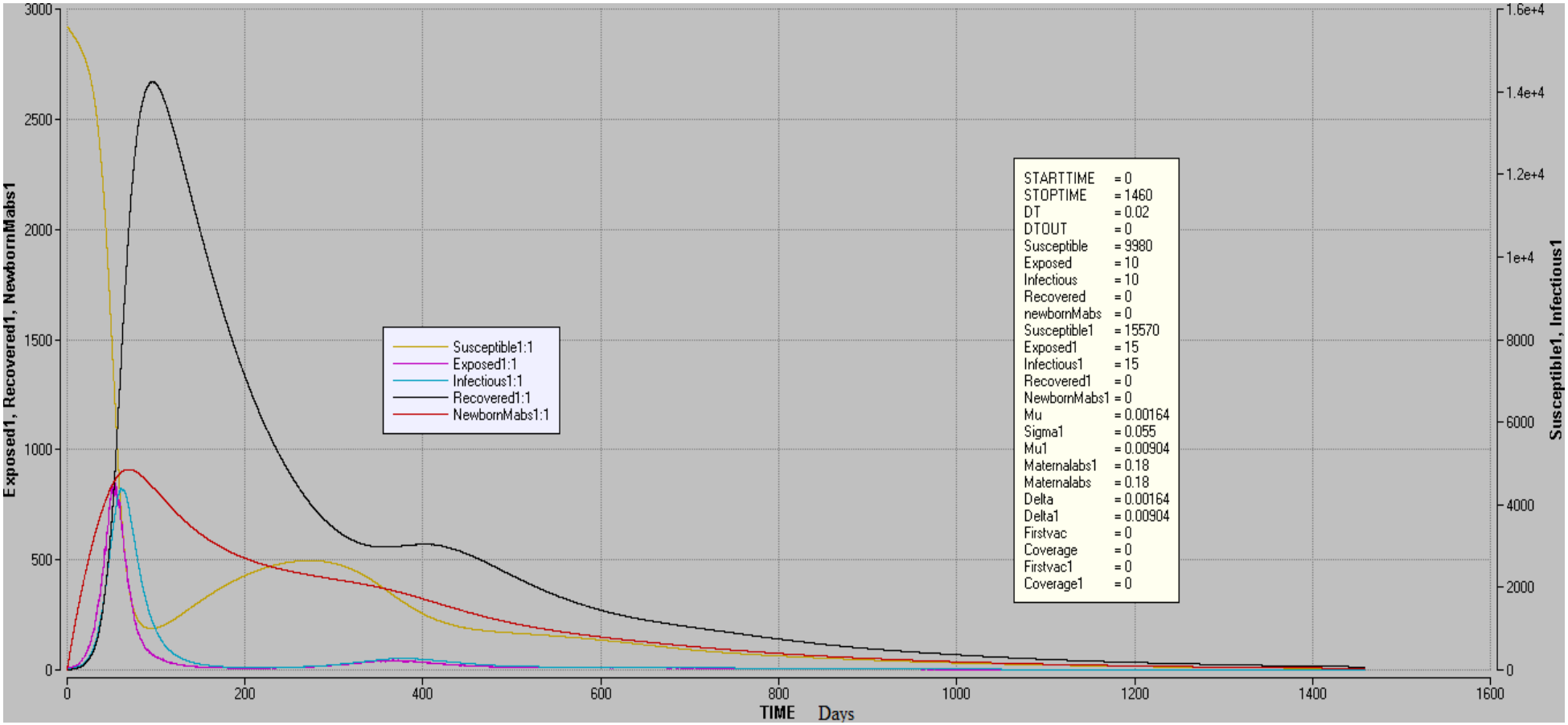


Figure 7.4: Plot of goat epidemiological parameters changes in Sheep/Goat composite MSIER model over 4 year period

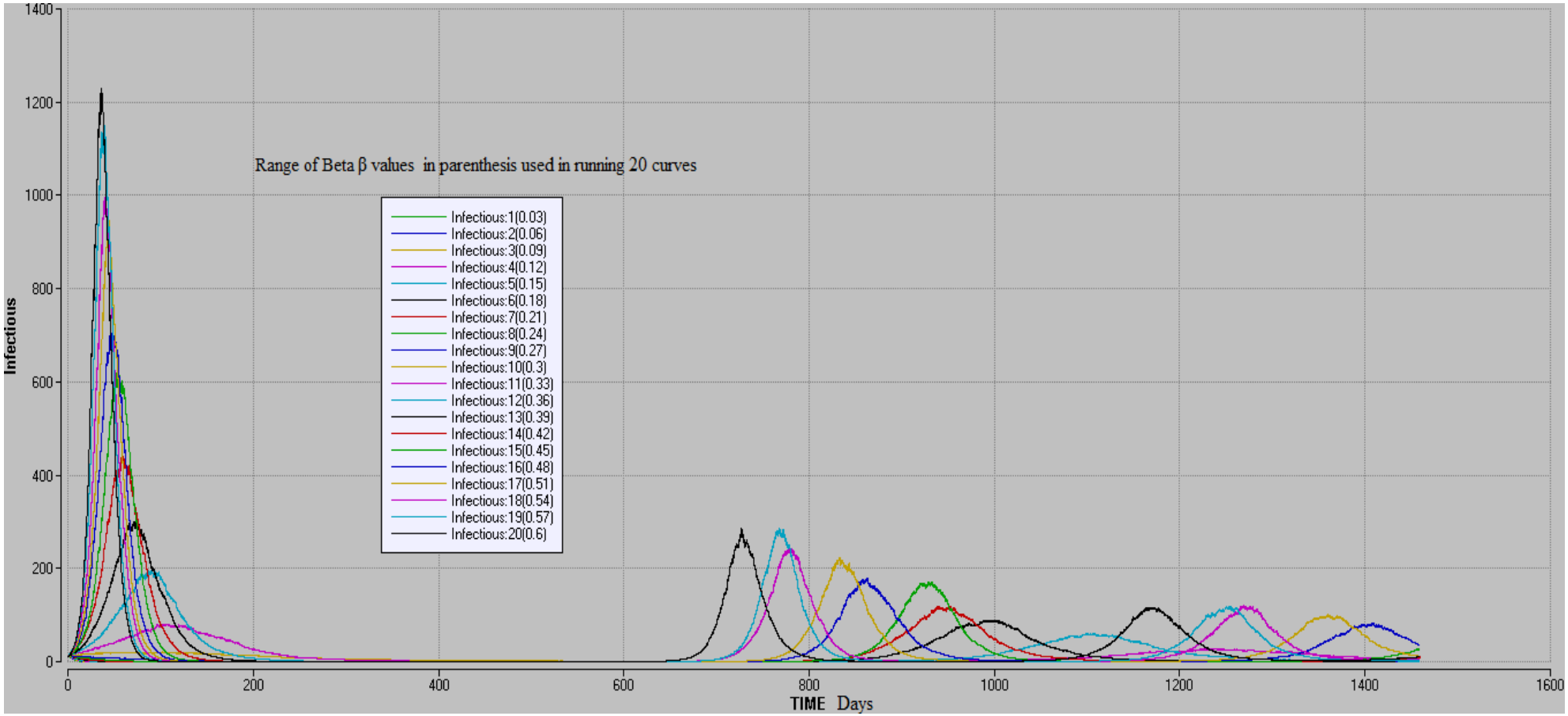


Figure 7.5: Infectious curves from 20 β values in the sheep model.

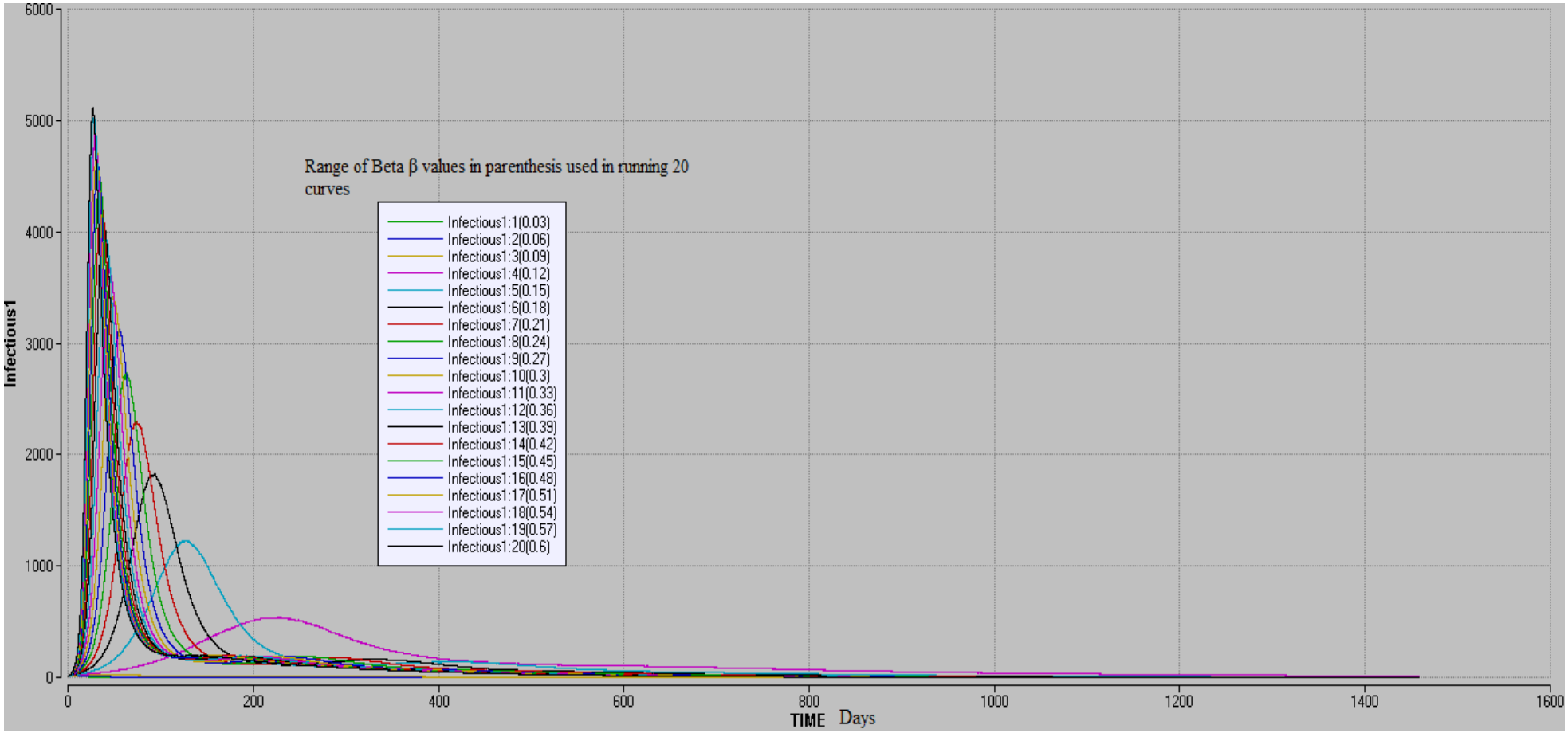


Figure 7.6: Infectious curves from 20 β values in the goat model.

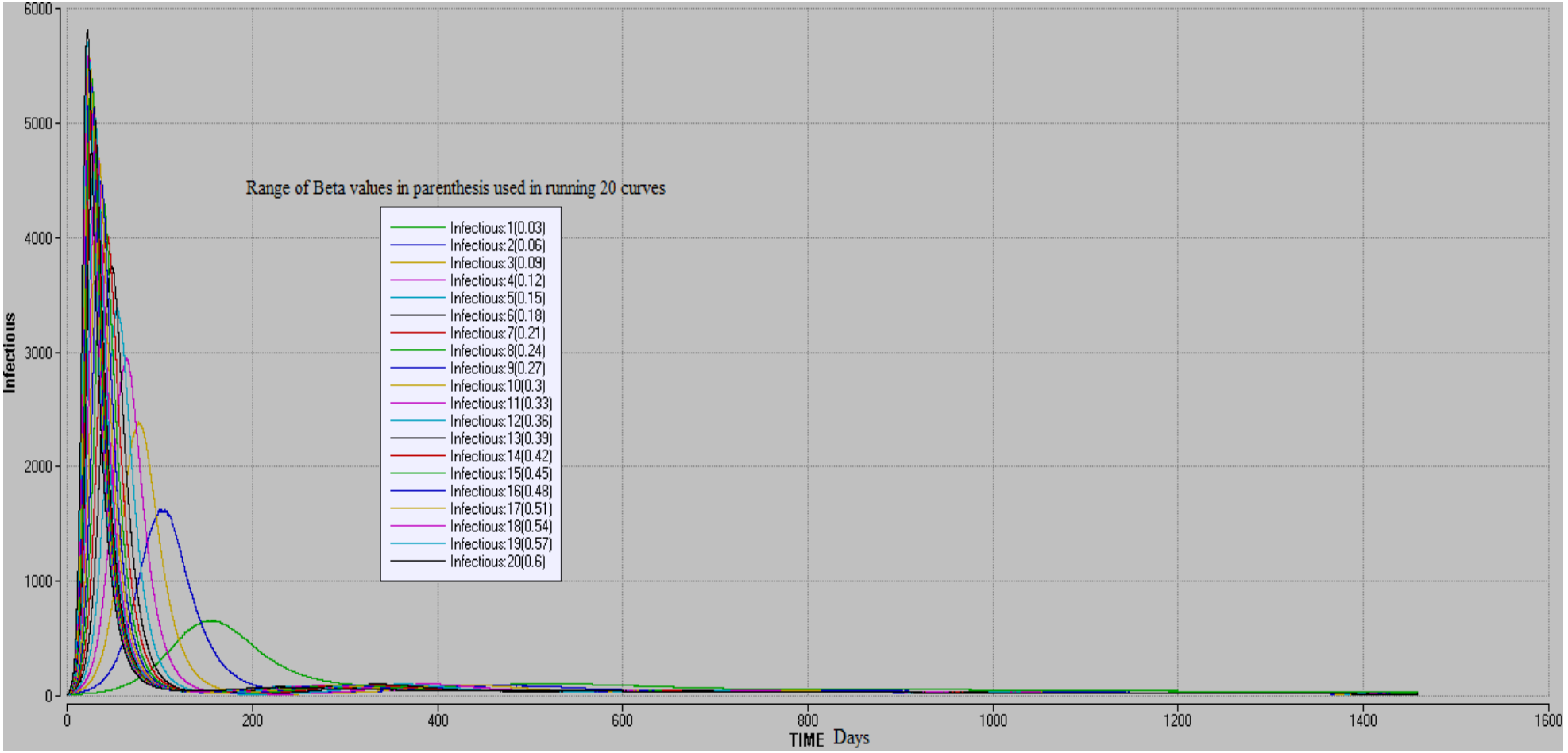


Figure 7.7: Sheep infectious curves from 20 β values of sheep in the composite model of sheep and goats.

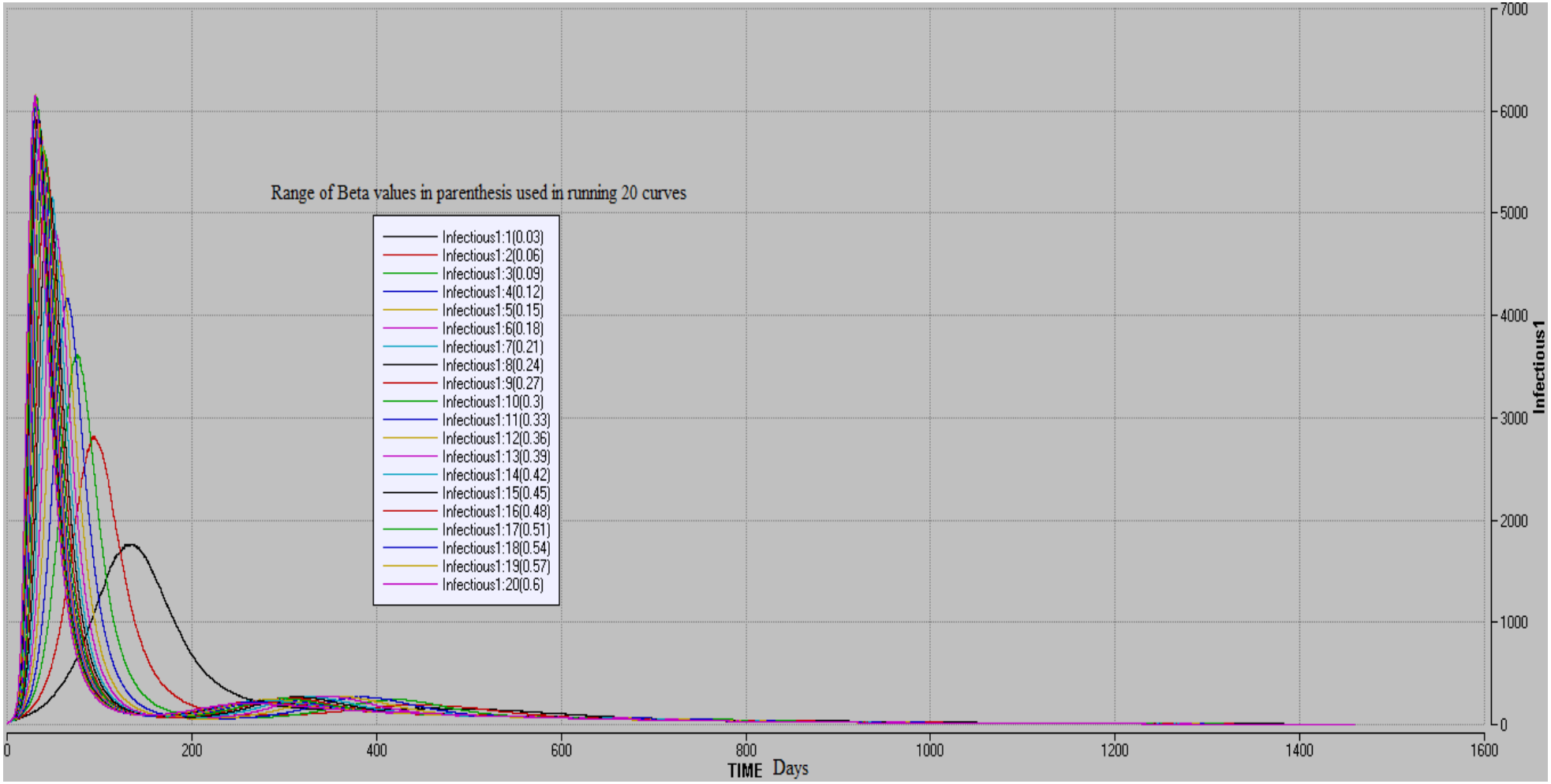


Figure 7.8: Goat infectious curves from 20 β values of sheep in the composite model of sheep and goats.

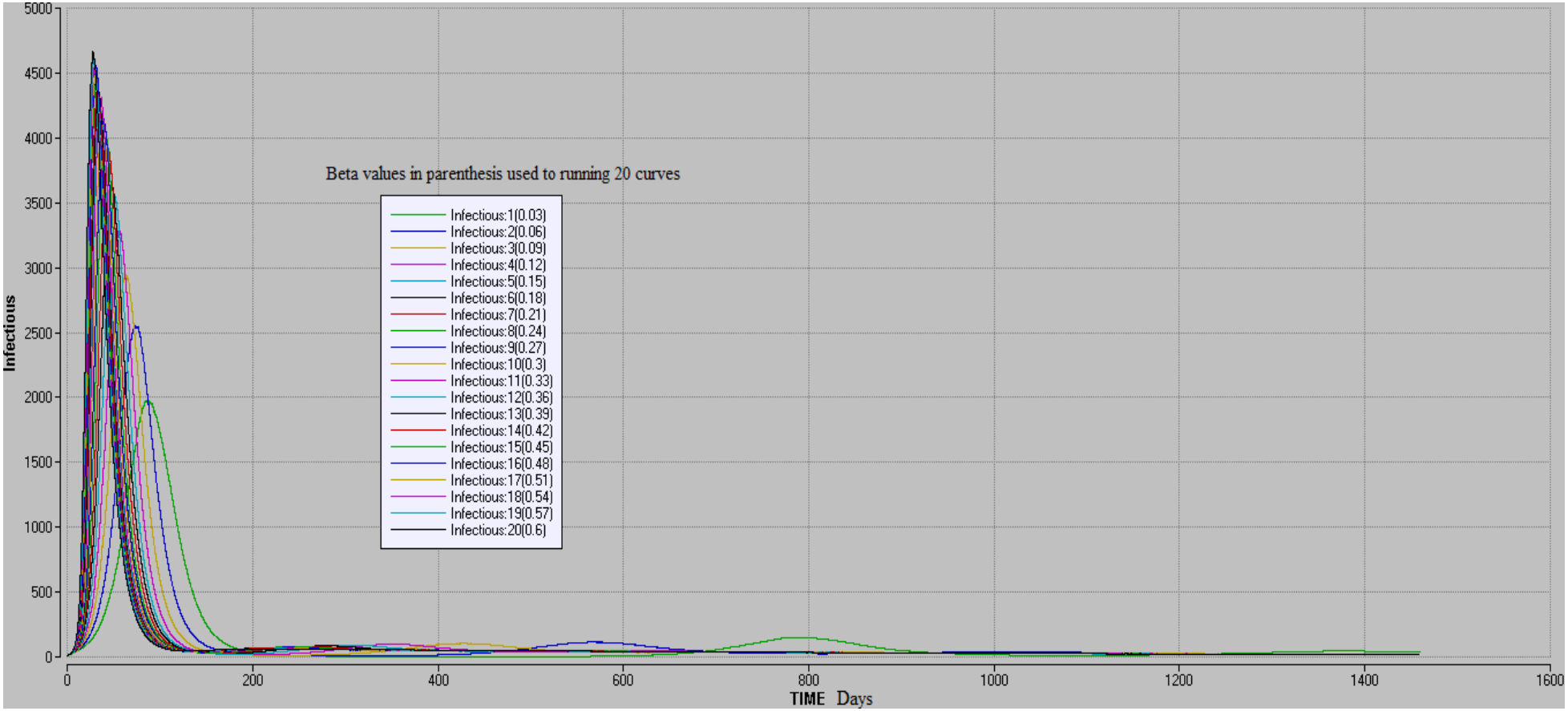


Figure 7.9: Sheep infectious curves from 20 β values of goats in the composite model of sheep and goats.

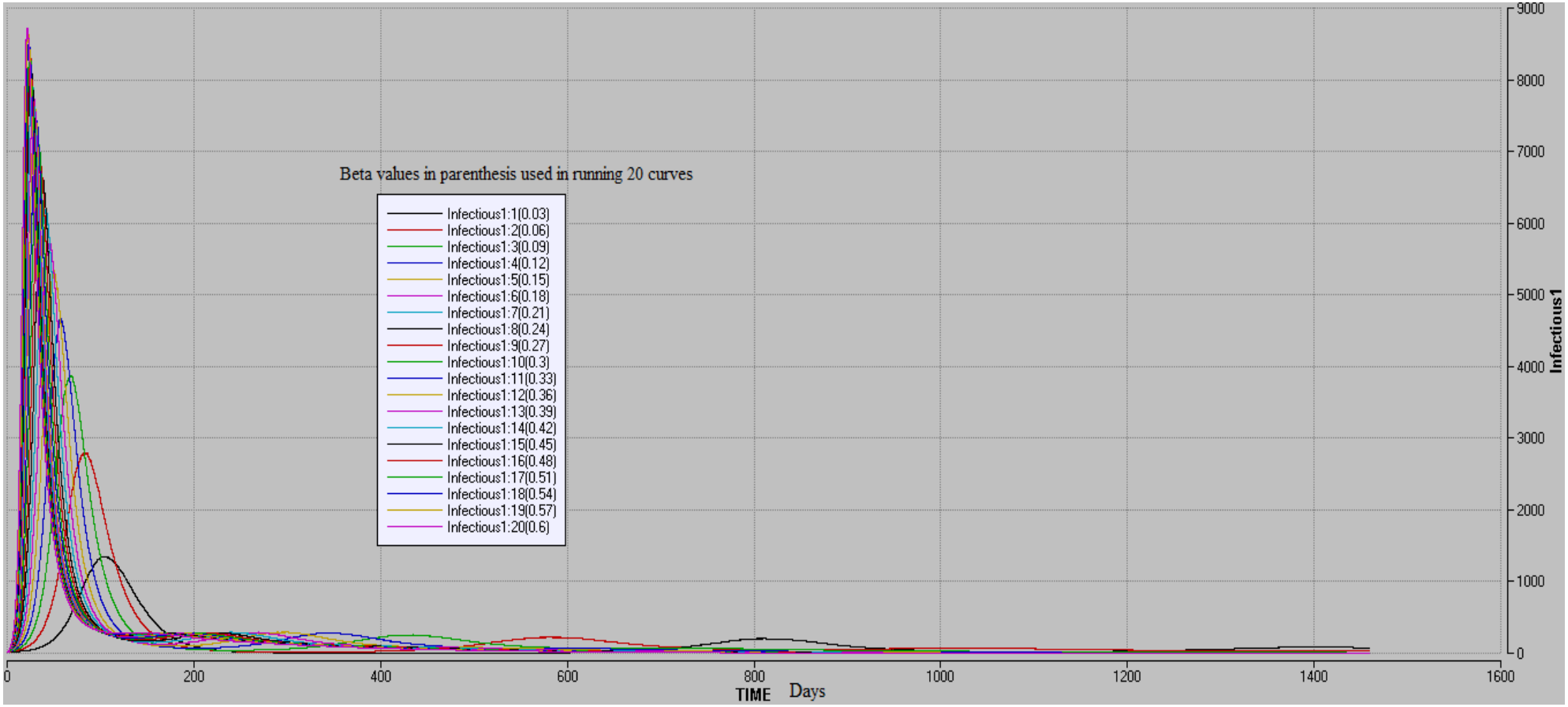


Figure 7.10: Goat infectious curves from 20 β values of goats in the composite model of sheep and goats.

set out as in the first output of sheep infectious curves and shows two epidemic peaks (minor and major) for β values above 0.06 (Figure 7.8). For the range of β values of goats derived from field data (0.063 to 0.152) the minor epidemic outbreaks comes first though intense and have high peaks of infectious (range 1783 to 6240) lasts between 197 days to 290 days however the major outbreaks picks up from fade-out trough and are less intense, have lower peaks of 280 infectious and do not fade out to zero for the rest of the model period.

The third output infectious curves is derived from applying a range of β values of goat to the goat portion of composite model while maintaining the model to pick random β values for the sheep portion of composite model (Figure 7.9). These infectious curves of the sheep shows several epidemic peaks and the infectious do not fade to zero for the model period. The fourth output curve is a goat infectious curve derived while maintaining the conditions set out in the third output goat infectious curves with each having more than one epidemic peaks (Figure 7.10). Infectious for β values of goat in the range of 0.06 and 0.152 do not fadeout to zero by end of model period of 1460 days.

7.3.2.2 Sensitivity analysis of varying initial population contents of exposed, infectious and recovered compartments

Regardless of initial number of exposed and infectious the major outbreaks were uniform in shape height and duration. However increasing the number of recovered reduced the peak of infection for the minor and major outbreaks. At zero recovered, infectious in sheep had not faded out by end of model period of 1460 days (Figure

7.11). However extension of the model to 8 years shows the infectious in sheep fades out completely at 2020 days (Figure 7.13) . The infectious in goats faded out by end of model period 1420 days (Figure 7.11 and 7.13). Subsequent increase in of recovered numbers to 50% increased the outbreak period for goats beyond the model period (Figure 7.12).

At population size of 15,600 goats' infectious population of goats faded out at the fourth year while at the population size of 10,000 sheep infectious population of sheep faded out at 5.5 years (Figure 7.13) and these were construed to be the critical community size that would allow PPR to persist.

7.3.2.3 Analysis of inter- epidemic periods and epidemic length

Data generated by the composite model simulation depicted in Figure 7.13 was analyzed to identify the characteristic of epidemic and inter epidemic lengths. In the model period of 8 years both sheep and goat exhibited 2 outbreaks, minor and major outbreaks. The first minor epidemic outbreak for sheep lasted 183 days and second major outbreak lasted1780 days. Inter – epidemic period between sheep outbreak peaks was 57 days. Goat's first minor outbreaks lasted 206 days and second major outbreak lasted 1291 days. Inter-epidemic period between first and second goat epidemic was 23 days.

7.3.2.4 Analysis of vaccination

In simulating infectious of sheep and goat separately it was found that infection of PPR in sheep would fade out completely by itself with one year (Figure 7.5). If vaccination at 20% coverage was applied to flock of 10,000 sheep raised separately from goats, PPR epidemic would resolve in 15 to 37 days. However in goats the PPR was self perpetuating up to three years (Figure 7.6) before it resolves completely. Simulation of vaccination of PPR in goats herded separately from sheep shows that PPR persist beyond model period of 1460 days and there is no complete fadeout of infectious group even at high vaccination level of 90%.

When the vaccination in mix flocks of sheep and goat was simulated in the composite model and analyzed separately for each species, a different picture of disease dynamic was obtained. It was established that, when only sheep herd was vaccinated, 60% coverage of sheep flock was enough to make the disease fade out in both sheep and goats in 42 days after vaccination at 180^{th} day. When vaccination was applied to goat alone, 70% coverage of goat flock would make the epidemic fade out in both species in 62 days after vaccination at 180^{th} day. Vaccination of both sheep and goat with a coverage of 50% resulted in the infectious fade out by 74 days after vaccination at 180^{th} day (Figure 7.14).

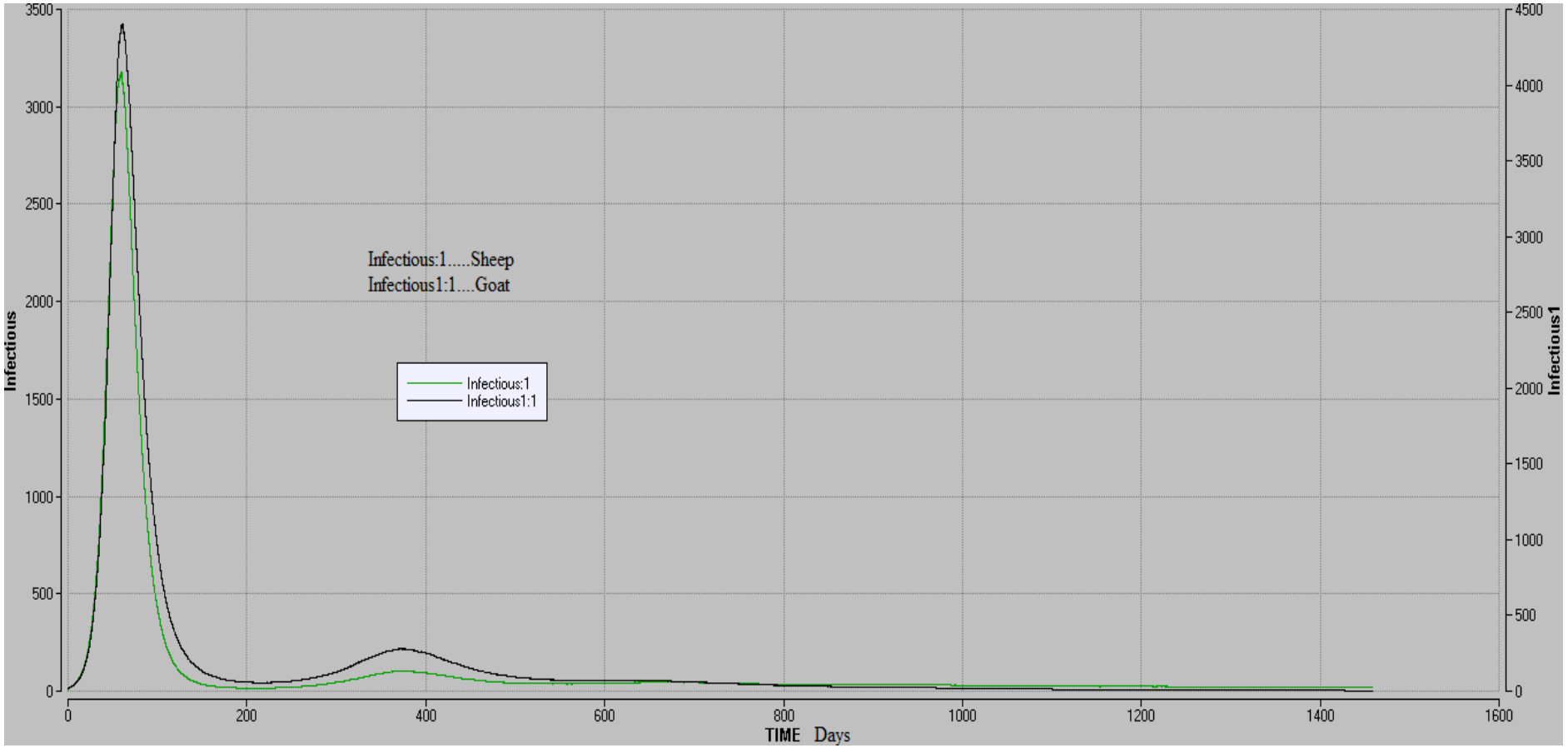


Figure 7.11: At zero initial recovered (R) the infectious goats fadeout before model period.

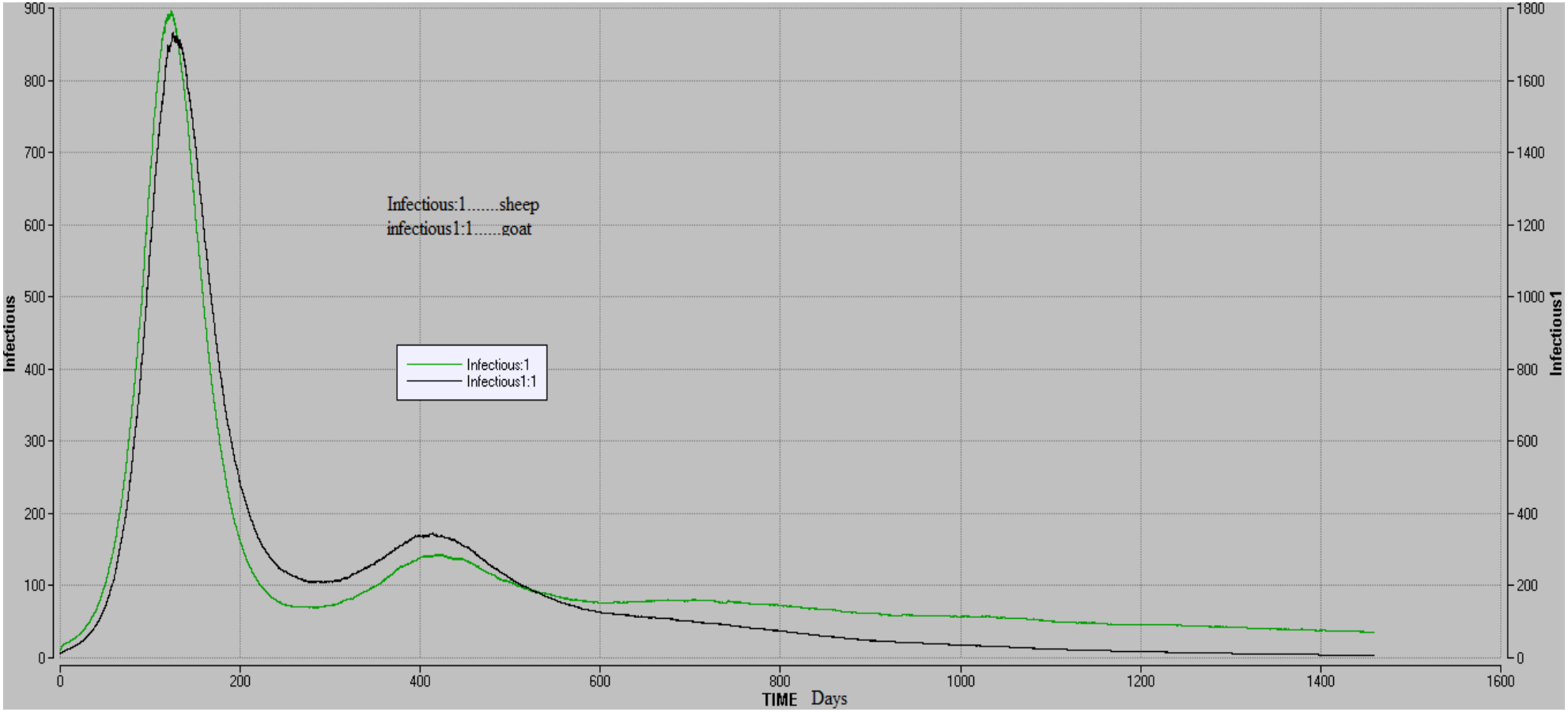


Figure 7.12: At 50% initial recovered both goat and sheep infectious remain beyond model period

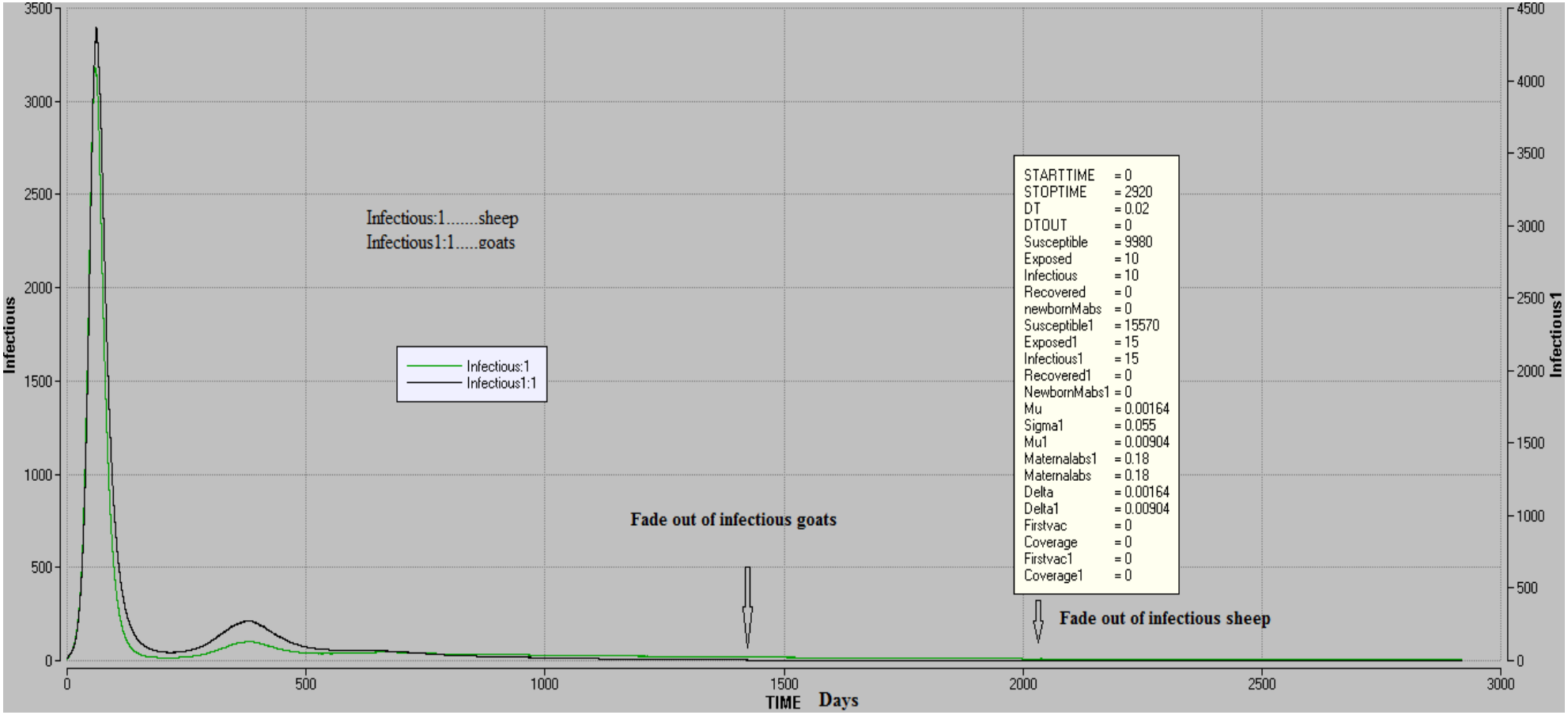


Figure 7.13: Time at the complete fade out of infectious for goats and sheep

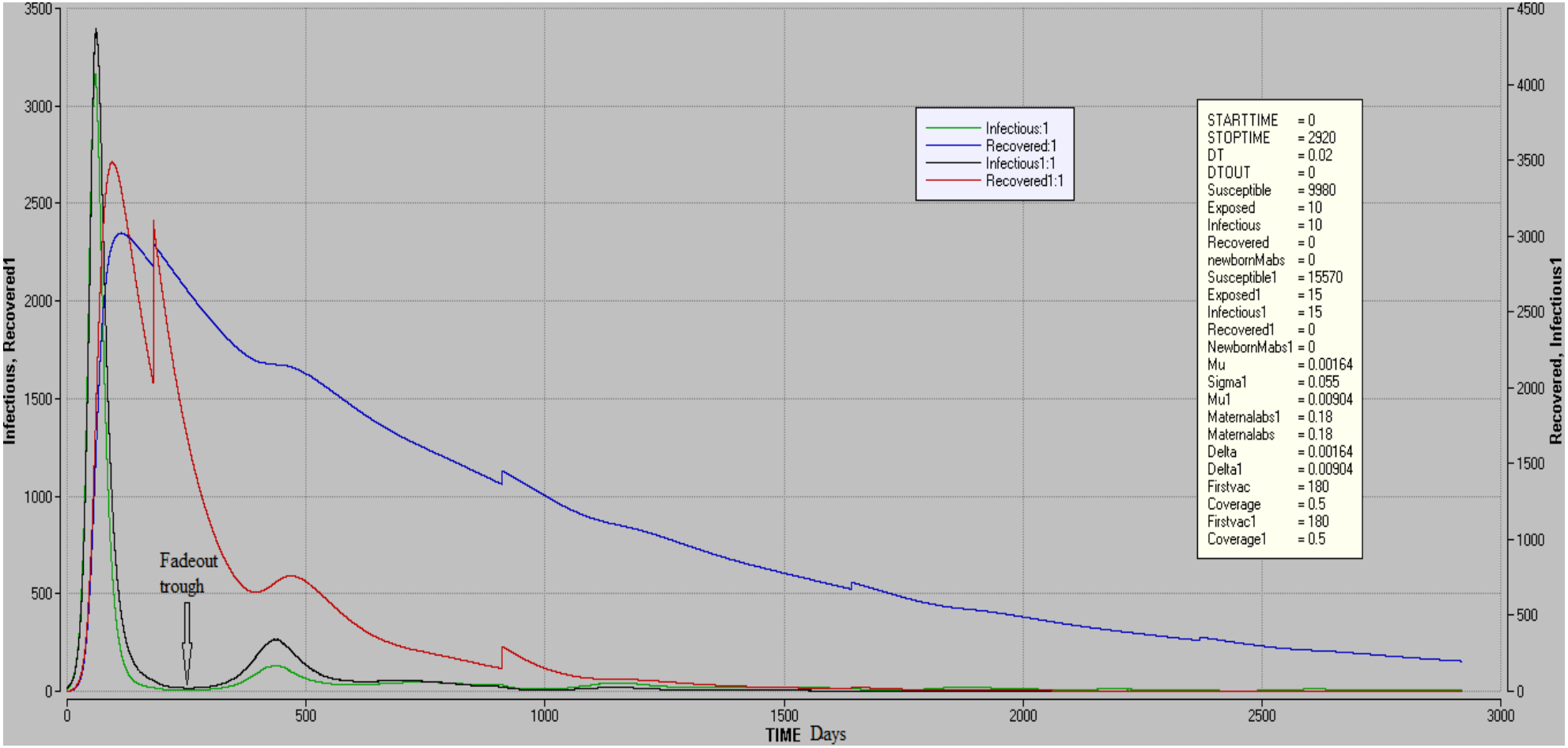


Figure 7.14: Vaccination coverage at 50% in both sheep and goats showing sheep and goat infectious group fade out.

7.3.3 Benefit cost analysis of PPR vaccination

With 12,621,390 small stock vaccinated nationally in Kenya at cost of Kenya shillings 420,000,000 as reported in 2009 by Director of Veterinary Services in Kenya the vaccination unit cost translates into Kenya shillings 33.28 per small stock vaccinated nationally. In Turkana a total population of small stock is 9,512,012. If they were all to be vaccinated the total vaccination cost is estimated at Kenya shillings 316,559,759. The total economic losses were previously estimated at Kenya shillings 11,148,886,072.

Benefit-cost ratio = estimated economic loss/PPR vaccination costs

Benefit-cost ratio = 11,148,886,072/316,559,759.4

Benefit-cost ratio = 35

7.4 Discussion

The Turkana community has devised ways of controlling the rampaging PPR disease. Their approach to disease control is based on local knowledge of what works best either for PPR or other diseases. The local herbs and hot stones used by Turkana such *Cissus guadrangularis (Egis), Euhorbia Uhligiana (Emus)* and Aloe *(Aloe Turkanensi)* are also used for treatment of other livestock disease and therefore are not specific treatment for PPR. Human antibiotic capsules are commonly used by local community in treatment of contagious caprine pleura-pneumonia and other bacteria ailments with varying success. The Kenyan law prohibits use human drugs and veterinary prescription drugs on animals without assistance from qualified animal health provider.

However veterinary services are scare or non existent in marginal pastoral areas like Turkana county. It is from this experience that community experimented and treated against PPR with the herbs, capsules and hot stones. It was observed that the community had recognized two scientifically proven control measures being community imposed quarantines to keep away sick herds and create sanitary cordon as well as vaccination. However these two control measure were ranked lowly in the sustainability matrix. The reason for ranking community imposed quarantines lowly was established to be the high cost of policing and ensuring sanctioned herds were kept away. Enforcing the sanctions at times resulted to insecurity as sections of community with sick herds felt marginalized and curtailed from accessing grazing resources due to a disease that was not their fault. Vaccination though scored highly on effectiveness and community being ready to commit financial resources in undertaking vaccination; was overall scored poorly on other criteria. It was noted that the community had little knowledge about the PPR vaccination process and it was perceived as high technology that was only available from Nairobi on request by local veterinarian. Experience with forced vaccination particularly in dry season when community was more bothered by drought has also created negative perception of vaccination.

It is thus important that local development actors, county government and national government should strengthen local structures in enforcing community lead quarantines and movement control because when government institutes quarantines a lot of criticism is generated from civil societies that support livestock market development

accusing government of creating artificial market barriers for pastoral livestock. Similarly livestock extension services should be enhanced to demystify and create positive perception of PPR vaccination process particularly now that community appreciate that vaccines are effective and are ready to commit resources towards vaccination process.

Development of PPR model for Turkana has elicited various issues relating to PPR transmission dynamics in sheep and goats. Data from the field has shown that PPR transmission in sheep and goats is varied and treating sheep and goats as one species would be a mistake that would lead to wrong conclusions. From the basic parameters it was established that there is a great demographic difference in the way the sheep and goat leave and get into Turkana herds. This demographic dynamics though not fully captured in the model except in birth rate and death rate; influences how the PPR disease is transmitted in the herd. Other parameters such as maternal antibody decay and PPR specific morbidity and mortality showed a varied difference between sheep and goats in Turkana. It follows that transmission factors such as the reproductive number, R_0 are different for sheep and goats. However in other studies (Zahur et al., 2009; Kivaria et al., 2013) have reported R_0 for sheep and goat as if they were single species. This study established R_0 for sheep in the range of 1.05 to 2.07 while that of goats was 1.05 to 1.66. The R_0 from this study was triangulated from three sources of data collected independently and that is why it was deemed that the R_0 values were representative of what was happening in Turkana sheep and goat herds. It is worth noting that other factors that could affect PPR transmission and therefore interfere with

calculated R_0 are that *Peste des petits ruminant*s is not a single species disease as it has been reported in camel, cattle and wildlife; however transmission dynamics in these other species has not been studied. Another key factor that could influence the calculated R_0 is that only data from intra herd was used in its calculation. Taking in mind the inter herds transmission dynamics may provide a more different picture.

Based on transmission parameters of PPR transmission in Turkana (specifically R_0) within sheep herds alone, simulation process established PPR outbreak would self extinguish without any vaccination process. However in goat herds only PPR was seen to persist for three years before outbreak faded away. The true picture of Turkana small stock herd is that it is a mixed herd of sheep and goat at a ratio of 1 to 1.56. So the mixed model of sheep and goat previously referred to composite model was found best suited to analyze transmission dynamics of PPR. Output from this mixed model showed that in mixed herd, infectious with PPR virus persisted for longer period in the sheep than goats. Though initial sheep only model had shown that sheep cannot sustain PPR virus transmission in Turkana beyond a year, the mixed model established that sheep were the main drivers of PPR transmission in mixed herds. Further observation noted that the most plausible and best option to control the spread of PPR was to vaccinate at least 50% of sheep and goats in the mixed herd. It is envisaged that these finding will provide guidance in PPR control and inform vaccination process. Currently PPR control programs are inference from control programs of other morbillivirus such as rinderpest, measles and canine distemper.

On economic grounds the vaccination of PPR was grounded by the benefit cost analysis that returned a BCA of 35. It should be noted the BCA included only the direct loses and could be higher if the indirect benefits are included in the analysis. This economic analysis echoes previous report by Van Den Ende *et al*., (1988), Stem (1993), Awa *et al.,* (2000) and SAARC (2011);

7.5 Conclusion

This study has provided an economical, scientific and social basis for PPR control through vaccination as well giving guidance at minimum level vaccination needed to stamp out the disease. It has also been established local community appreciate vaccination as effective control method of PPR and are ready to commit themselves financially in control process if guided well and provided necessary information.

CHAPTER 8: SUMMARY, GENERAL CONCLUSIONS AND RECOMMENDATION

8.1 Introduction

This chapter provides a summary of the discussions, gives general conclusion and recommendations from this study.

8.1.1 Risk factors influencing the patterns of PPR spread in Turkana County

Peste des petits ruminants in Kenya was first reported in Turkana, one of largest, peripheral, remote, insecure county with poor infrastructure. These characteristics of Turkana County are disincentive for any official data collection. Official livestock disease surveillance is scanty and may not reflect the true picture at the *Adakar* level. For investigation on risk factors in Turkana to be meaningful it was necessary to engage the herders in data collection as they were the main livestock disease information repository, though officially unrecognized. Turkana love for their livestock will draw them to narrate details of experiences and observations their animals have gone through to any willing listener. Using participatory epidemiology methods and approaches, focused group discussions on sheep and goat diseases helped generate organized and useful information that was not officially available in any

veterinary office. A key feature of this data collection method is respect and trust building between respondents and interviewer.

Throughout the discussion it was established that Turkana people were very knowledgeable of livestock diseases prevalent in their region. A key disease that featured prominently in their description of diseases was *Peste des petits ruminants* (PPR) (*Lomoo). Peste des petits ruminants* was described as disease that made the sheep and goats depressed hence the *Lomoo* naming which etymologically is derived from action of looking sickly and depressed. Other names used to describe depression in goats and sheep, associated with PPR were *Ekitowo* and *Loutogonyen,* literally meaning sick goat syndrome though condition named *Loutogonyen* had other signs such as sunken eyes and emaciation.

The PPR disease was associated more with goats where it was highly ranked after contagious caprine pleuro -pneumonia. In sheep, the PPR disease was ranked the sixth among other sheep diseases. The Turkana community clearly characterized PPR by associating the disease with key clinical signs signifying that the community had witnessed and experienced the disease in their herds. In both sheep and goats, the Turkana community prominently associated PPR with migration, herd mixing and raids. This must have come from observation and realization that some goats and sheep, individually or as herds, introduced into new herds, must have spread the PPR infection to the herds that hosted them. The community analyzed the seasonality of PPR where

the PPR in goats was associated with all seasons while the PPR in sheep was associated with dry season only. However, even in the goats, PPR disease was significantly noted to be associated with dry season. Activities associated with dry season such as migration and herd mixing, in formation of *Adakars* for search of pastures and water, were seen to encourage the emergence of PPR in small stock. Livestock raids were known to be practiced throughout the seasons and could contribute to the emergence of PPR disease in all seasons. Key environmental features associated with PPR disease in goats were: toxic plants and mountainous pasture and water during dry season. However following probing of respondents, it was established that during dry season the small stock, particularly goats, were migrated to the high mountainous ranges that had some remaining pastures and water trapped between rocks. These dry season grazing areas in the mountains offered opportunity for extensive mixing of small stock from different herds during watering and grazing, providing for opportunity of infected small stock to make contacts with susceptible herd thus spread PPR infection. Such PPR infection, got during grazing in highlands, were thus blamed on the mountainous plants and water trapped in the rocks. Though the community's appraisal of PPR associated the disease with all age groups in goats and sheep, except lambs, it was interesting to note that discussion on morbidity and mortality due to PPR included all age groups in both sheep and goats. Despite PPR being ranked sixth in sheep diseases, the community perceived PPR to have higher morbidity, mortality and case fatality than any other sheep disease. Same appraisal of PPR morbidity, mortality and case fatality was observed in goats.

The appraisal of PPR by the Turkana community, through the various participatory epidemiology appraisal tools, yielded a description of the PPR disease that was compatible with veterinarian scientific description of PPR and was validated by further laboratory analysis that confirmed presence of PPR virus in Turkana.

During the participatory focused group discussion for local appraisal of PPR, the Turkana respondents mentioned factors associated with the disease. This was followed up in a further investigation of pastoral livestock management as practiced by the Turkana pastoral herders through participatory risk assessment; seven risk factors related to some of the livestock management decisions made by Turkana herders at household level and *Adakar* level in their management of small ruminants were identified. The management decisions were made in response to constraints experienced by the herders, such as labor shortage, pasture and water availability, socio-cultural obligations, herd health, need to expand herd size and prevailing security situations. To overcome these constraints Turkana herders have developed strategies which constitute, among others, the seven management factors extracted from factor analysis model.

Indiscriminate herd mixing is strongly associated with variables highlighting the most vulnerable groups of older kids and lambs; young sheep and goats making contacts with high risk groups such as wildlife and sick adults through sharing of water holes, troughs and grazing. The indiscriminate mixing of sick animals and wildlife during grazing poses a health risk to the herd.

The deliberate decision of the pastoral Turkana herder to allow sick animals to mingle with healthy ones can be explained from point of labor shortage; households with abundant labor segregate their herds of livestock into herding groups based on species, age, production status and health status. Wildlife such as dikdik (*Madoqua guentheri*) is common in Turkana dry land savannah and graze along with small stock in the savannah shrubs; they could thus pose as a risk considering similar wild small ruminants, grey duiker (*sylvicrapa grimmia*) have been reported with PPR sero-positivity (Ogunsanmi *et al.,* 2003).

Introduction of new animals in herds strongly associated with variables highlighting introduction of new animals into a herd. Such introduction may come from market purchases, gift from cultural ceremonies, raids and unsold small stock returned home from market sale yard. At the market sale yard animals from various locations are concentrated in closed pens therefore creating a high risk environment for contracting of diseases; these diseases can be spread to herds where bought animal are destined to go.

Sharing water points was associated with variables that highlighted on watering of the younger groups of sheep and goats. Water is a scarce commodity in Turkana and a single water source may serve several herds during dry season. In situations where water and labor are abundant, animals of different age groups are watered at different times and places; the very young kids and lambs being watered at home. However, in the very dry seasons, all animals may be seen crowding in a single water hole waiting their turn to drink, consequently increasing possibility of making infective contacts.

Similarly, during wet season, the small stock will drink indiscriminately from the puddles scattered in the plains. Several herds may share common puddles particularly along the herding pathways.

Sharing of local pasture with foreign animals was associated variables mentioning invasion of local pastures by foreign herds from across international borders. During the severe drought, even the communities who are adversaries will grant each other passage to pasture and water. It is at this time that pastoralists will cross international borders in search of pasture and water.

Raids were associated with livestock raids and transhumance focusing on older lambs and kids as well as young sheep and goats. Turkana community mobility exposes their livestock to share pastures with herds that could be exposed to diseases. Livestock raids are common cultural activities among the Turkana and the neighboring communities. Despite the perceived gains from raiding, Turkana herders avert that raided animals are also known to spread disease to herds they end up to.

Loaned animals were associated with loaning of livestock. In Turkana begging livestock is an accepted normal and people negotiate to be given animals by their clansmen or age mates. In such circumstances animals given out are those that are of less benefit to the owner. Therefore it is high risk to borrow animals from a sickly herd because the owner will readily hand them over to borrower who will end up with liability of paying back whether the animals survive or not.

Sick nursing dams were associated with variables mentioning sick adult goats and sheep sharing grazing and water with kids and lambs. From the interviews, it was

emphasized that sick livestock were left to graze with kids and lambs around the homestead; this included sick dams that were allowed to continue nursing their young ones.

Of the above seven factors it was established that Factor 3 was significantly associated PPR outbreak in 2009 while Sick nursing dams and administrative divisions where *Adakars* were located were the significant factors associated with PPR outbreak in 2010.

In a subsequent sero-epidemiological study variables that were significantly associated with presence of PPR antibodies and thus considered risk factors were small stock species, vaccination status, age groups and geographical administrative areas. Age was not significant risk factor in sheep while vaccination status was not a significant risk factor in goats.

Whereas some of the management practices that would have been expected to increase risk were not captured by the final analysis this did not necessarily imply that they are not important in the transmission of PPR. Factors associated with the observation of clinical cases during outbreaks may be quite different from those associated with the prevalence of infection. As previously noted the PPR disease is relatively new in Kenya and to the Turkana pastoralists. The clearest picture of the PPR disease the Turkana pastoralist can describe is that of dramatic epidemic that killed their sheep and goats in 2006 and 2007. However as the disease became endemic, such dramatic PPR outbreaks affecting both species and all ages of small ruminants may not appear again. This would mean that there are possibilities of misreporting of the disease as it is likely to

present a different epidemiological picture from their original experience with the disease.

It was also observed that similar factors were not significant in the same season among the years. Thus it cannot be generalised that significant factors in one year are similar in other similar seasons of other years. This temporal heterogeneity of significant factors can be explained from the fact that, though the cultural practices of Turkana may be consistent overtime; these cultural practices may be practised in varied geographical places. Turkana community is highly mobile in search of pasture, water and security for their livestock. Thus, in process of herding their animals, the Turkana community interacts with various other communities and environments and this will impact on their livestock based livelihood management decisions.

8.1.2 Levels of herd immunity within the flocks.

The response to the outbreak was mass vaccination that was sponsored by government of Kenya and development partner donors. However the numbers of small stock vaccinated by the donor sponsored exercise in 2007 was 1,380,283; constituting 14% of the total population of 9,512,012 smallstock in Turkana district with the current study having established a vaccination prevalence of 7.84%. This, therefore, means that the PPR antibody profile in the study area could be attributed to both wild virus and vaccination.

Goats were found to have a significantly higher percentage of sero-positivity (at 39.6%) compared to sheep (at 31.6%) ($p=0.000$). However, looking at the seroprevalence by

age and sex, adult goats and more so females contributed to the elevated antibody prevalence in goats. It was thus observed that, although the antibody prevalence in male sheep was 28.8% compared to female sheep (at 33.3%), there was no significant difference between the two.

There was a considerably significant difference in antibody prevalence between age groups in both sheep and goats. The middle aged group in sheep had antibody prevalence of 18.2% while middle aged goats had antibody prevalence of 14.2%. The small stocks in the middle age group sampled in this study were generally born in the period between 2009 and 2010 when no major vaccination was carried out. The vaccinated sheep in middle aged group had antibody prevalence of 56.3% while goats in the similar group had 31.8%. The difference in antibody prevalence in this age group is attributed to retention of sheep within herd compared to early disposal of goats, particularly the males, through markets. However the non-vaccinated middle aged group had antibody prevalence of 14.3% in sheep and 12.2% in goats; explanation for this difference being similar to that of the vaccinated middle aged group - that it had limited exposure to vaccination and wild virus. Therefore, this group remained the most risky since it had no antibody protection.

The category of the young small stock (lambs and kids) sampled were tested for presence of respective antibody which were due to maternal antibodies against PPR. The antibody prevalence in kids and lambs matched the antibody profiles in the adult females. The difference in sero-positivity between goat adult females and kids could be

explained by that fact that some kids could have been born by female goats in the middle aged groups who had no PPR antibodies.

As noted earlier, some vaccinations were carried out in early 2011 in Oropoi division, three weeks prior to this study. Consequently the sero-prevalence in Oropoi division was significantly higher for both sheep (68.3%) and goats (63.5%) compared to sero-prevalence in other administrative divisions of Kakuma, Loki, Kaaleng, Kibish and Loima. Antibody prevalence for sheep was lowest in Kaaleng division at 15.4%. Spatially, the sero-positivity of sheep in other divisions was in the range of 20 to 40%. Spatial distribution of antibody prevalence in goats show Kibish and Loki with sero-prevalence range between 40 and 60% while Kakuma, Loima and Kaaleng had a sero prevalence range between 20 and 40%.

The sero-prevalence for the small stock in Turkana for this study was 36% with an overall picture of large populations of the sheep (69.4%) and goats (61.4%) that did not have sufficient antibody titers to mount a strong immunity to PPRV infection.

8.1.3 The socio-economic impact of Peste de petit ruminants.

Following the onset of PPR outbreaks in Turkana County in 2006, the disease triggered relatively large economic losses to Turkana pastoral herders. The PPR disease entrenched itself and became endemic in Turkana County thus causing cyclic outbreaks that perpetually resulted in continuous economic losses to the herders. This study has established that the Turkana community relies heavily on their livestock and all their socio- cultural activities revolve around livestock. Wealth and a person's standing in

the community are judged based on amount of livestock owned and what the livestock has contributed to - mostly measured by the number of wives married and children born by the wives. Approximately a quarter of the Turkana people without livestock are perceived to be poor. Small stock sheep and goats constitute the largest herd of livestock reared by Turkana with herd composition ranging between 42 to 64.4% of the total herds across the wealthy groups. Livestock-keeping remains the major livelihood activity for the livestock keepers contributing significantly to food, cloth/ornaments and other consumer services. Of all the food consumed by the Turkana 29.4% consists animal products which are well distributed in the household with the major youthful workforce of morans and girl herders consuming most of the animal products.

The Turkana residents identify diseases as the second most important that diceminate their herds. Diseases deprive the community from accessing livestock generated benefits. Based on this premise *Peste des petit ruminants* was ranked as the disease with highest destructive impact on the small stock benefits to the Turkana community.

The direct losses due to PPR disease that impact on small stock benefits were mainly due to disease mortality, morbidity, accompanying milk and weight losses. This study has established that the direct economic losses due to PPR in Turkana County alone for the year 2010 were in the tune of KES 11.1 billion. Compared to previous early estimates of economic losses due to PPR of KES 1.275 billion reported by government of Kenya in 2007 it shows that earlier national estimates of PPR disease losses had been under- estimated.

8.1.4 Documentation and evaluation of current control strategies in Turkana, Kenya.

The Turkana community has devised ways of controlling the rampaging PPR disease. Their approach to disease control is based on local knowledge of what works best either for PPR or other diseases. The local ethno-veterinary knowledge is applied for treatment of PPR among other livestock disease. Human antibiotic capsules are commonly used by local community in treatment of contagious caprine pleuro-pneumonia and other bacteria ailments with varying success. It is from this experience that community experimented and treated against PPR with the herbs, capsules and hot stones. It was observed that the community had recognized two scientifically proven control measures community imposed quarantines to keep away sick herds and create sanitary cordon and vaccination. However these two control measures were ranked lowly in the sustainability matrix. The reason for ranking community imposed quarantines lowly was established to be the high cost of policing and ensuring sanctioned herds were kept away. Enforcing the sanctions at times resulted to insecurity as sections of community with sick herds felt marginalized and curtailed from accessing grazing resources due to a disease that was not their fault. Vaccination, though scored highly on effectiveness and community being ready to commit financial resources in its undertaking; was overall scored poorly on other criteria. It was noted that the community had little knowledge about the PPR vaccination process.

Development of PPR model for Turkana has elicited various issues relating to PPR transmission dynamics in sheep and goats. Data from the field has shown that PPR

transmission in sheep and goats is varied and treating sheep and goats as one homogenous group would be a mistake that would lead to wrong analytical conclusions. From the basic parameters it was established that there is a great demographic difference in the way the sheep and goat leave and get into Turkana herds. This demographic dynamic influences how the PPR disease is transmitted in the herd. Other species intrinsic parameters such as maternal antibody decay and PPR specific morbidity and mortality showed a varied difference between sheep and goats in Turkana. It follows that derived transmission parameters such as the reproductive number, R_0 are different for sheep and goats. The R_0 from this study was triangulated from three sources of data collected independently and that is why it was deemed that the R_0 values were representative of what was happening in Turkana sheep and goat herds. It is worth noting that other factors that could affect PPR transmission and therefore interfere with calculated R_0 are that *Peste des petits ruminant*s is not a single species disease as it has been reported in camel, cattle and wildlife which are abundant in Turkana County; however transmission dynamics in these other species has not been studied. Another key factor that could influence the calculated R_0,in this case is that only data from intra herd was used in its calculation. Consideration of the inter herds transmission dynamics in calculation of R_0 may provide a more different picture.

Using PPR transmission model in Turkana within sheep herds alone, simulation process established that PPR outbreak would self extinguish within a year without any vaccination process. However in goat herds only, PPR was seen to persist for three years before outbreak faded away.

The true picture of Turkana small stock herd is that it is a mixed herd of sheep and goat at a ratio of 1 to 1.56. So the mixed transmission model of sheep and goat that was previously referred to composite model was found best suited to analyze transmission dynamics of PPR. Output from this mixed model showed that infections with PPR virus persisted for longer period in the sheep than goats. Though initial sheep only model had shown that sheep cannot sustain PPR virus transmission in Turkana beyond a year, the mixed model established that sheep were the main drivers of PPR transmission in mixed herds. Further observation noted that the most plausible and best option to control the spread of PPR was to vaccinate with coverage of at least 50% of sheep and goats in the mixed herd.

On economic grounds the vaccination of PPR was grounded by the benefit cost analysis that returned a BCA of 35. It should be noted the BCA included only the direct loses and could be higher if the indirect benefits are included in the analysis. This economic analysis echoes previous reports by Van Den Ende *et al*., (1988), Stem (1993), Awa *et al.,* (2000) and SAARC (2011);

8.2 General conclusions

- Risk factors of PPR
    - The Turkana community has developed a very comprehensive description of PPR disease overtime thus is a repository of livestock disease information for their locality. There is need to utilize this wealth of indigenous knowledge on diseases of livestock that reside with

pastoral communities, for purposes of understanding diseases in the community and setting up strong participatory surveillance systems that involve the communities as the basic element of disease surveillance intelligence gathering.

- Field pathological tissue samples, collected and stored at -30°C, as well as those stored in formalin 10% for up two years, could be used for PPR virus RNA extraction for disease confirmation during local knowledge validation process.
- In both sheep and goats Turkana community associated PPR disease with herd migration, mixing and livestock raids.
- Seasons, geographical locations and seasonal livestock management activities were also identified as risk factors to PPR disease out breaks.
- Risk factors associated with presence of PPR antibodies in small stock were species, age group, geographical administrative areas and vaccination status.
- Similar risk factors were not significant in the other seasons of other years. Understanding this spatial-temporal heterogeneity of risk factors will greatly improve design of disease control measures against PPR.
- More in-depth understanding of socio-ecology PPR in Turkana community and neighboring communities will refine the risk factor associated with socio cultural and economic activities of the involved pastoral communities.

- Herd immunity against PPR
    - The herd immunity in Turkana is low for both sheep and goats low to allow for containment of spread of the disease.
    - Vaccination breaks of two years created a pool of small stock in the middle aged group that was most susceptible to PPR infection because they were immunologically naïve. In such situation the wild virus has continued to infect the immunologically naïve small stock and is widespread in Turkana district. Due to the endemic status of the disease in Turkana dramatic outbreaks are not likely to occur. However muted or sub-clinical levels infections persist.
- Socio economic effect of the disease
    - Small stock is the common livestock owned by merely all wealth groups across the Turkana community.
    - Small stock is the major livestock livelihood catering for both subsistence and market income for the Turkana community.
    - Turkana community recognises PPR as a major economic disease affecting the Turkana herders and has the potential of disrupting cultural set up and local economy.
- Documentation and evaluation PPR control in Turkana
    - Turkana practice several PPR control measures such as herbs and local hot massage stones however the scientific basis for their use as well as efficacy remain unknown.

- Other PPR control measures used by Turkana based on local knowledge include community imposed quarantines and restrict livestock movements and mixing all in effort to keep away from infected herds and create sanitary cordons.
- Local Turkana community appreciate vaccination as effective control method of PPR and are ready to commit financially in vaccination control process if guided well and provided necessary information.
- Vaccination programs targeting 50% coverage of small stock herds will control and eventually eradicate the disease.

8.3 Recommendations

- There is need to incorporate indigenous knowledge on livestock diseases for purposes of understanding diseases in the communities and setting up strong participatory surveillance systems that involve the communities as the basic element of disease surveillance intelligence gathering.
- The key risk factors of PPR in Turkana highlighted by this study to provide a good basis for crafting a comprehensive PPR control strategy in the pastoral communities.
- There is need to carry out further studies on social ecology of PPR covering other pastoral communities in Kenya so as to uncover and refine the risk factors for improved design of disease control measures against PPR.

- This study recommend the collection of samples fixed in formalin 10% for PPR virus RNA extraction for disease confirmation in cases where cold chain is a constraint.
- Vaccination against PPR should be carried out regularly preferably on annual basis to improve the herd immunity to levels that can contain spread of the PPR disease.
- The greater emphasis of vaccination should target middle age group (>6 months to <24 months) being the group that is most susceptible to PPR infection in Turkana.
- Considering that goat and sheep are the backbone of the socio-economic livelihoods of the Turkana, the county government should come up with social protection measures for example insurance, disaster fund and social safety nets that can cushion the pastoralists against the ravages of PPR.
- The central government should ensure that the veterinary offices and the social development office within the county have adequate human resource to advice the local community on requisite measures to take to safeguard themselves against the negative impact of PPR.
- *Peste des petits ruminants* disease control policy should envisage annual vaccination in risky areas focusing in 50% coverage with priority given to middle aged small stock.
- Local pastoral structures need to be strengthened aimed at enforcing sanitary disease control measures in a sustainable manner.

- Veterinary extension services should be enhanced to demystify and create positive perception of PPR vaccination process.

REFERENCES

Abdalla A.S., Majok A.A., El Malik K.H. and **Ali A.S., (2012):** Sero-prevalence of Peste des petits ruminants virus (PPRV) in small ruminants in Blue Nile, Gadaref and North Kordofan States of Sudan Journal of Public Health and Epidemiology, 2012, **4**(3), 59-64.

Abegunde A.A. and **Adu F.D. (1977):** Excretion of the virus of peste des petits ruminants by goats. Bulletin of Animal Health and Production in Africa. **25**(3): 307-311.

Abu Elzein E.M.E., Hassanien M.M., Alfaleg A.I.A, Abd Elhadi M.A., and **Housawi F.M.T. (1990):** Isolation of PPR virus from goats in Saudi Arabia. Veterinary Record, **127**: 309-310.

Abubakar M, **Khan H.A., Arshed M.J., Hussain M.,** and **Ali Q, (2011):** Peste des petits ruminants (PPR): Disease appraisal with global and Pakistan perspective. Small Rumminant Research, **96**: 1–10.

Abubakar, M., **Jamal M.S., Arshed M.J., Hussain M.,** and **Ali Q. (2009):** Peste des petits ruminants virus (PPRV) infection: Its association with species, seasonal variations and geography. Tropical Animal. Health and Production, **41**: 1197-1202.

Adombi C.M., Lelenta M., Lamien C.E., Shamaki D., Koffi Y.M., Traoré A., Silber R., Couacy-Hymann E., Bodjo S.C., Djaman J.A., Luckins A.G., and **Diallo A. (2011):** Monkey CV1 cell line expressing the sheep-goat SLAM protein: a highly sensitive cell line for the isolation of peste des petits ruminants virus from

pathological specimens. Journal of Virology Methods. 2011 May;**173**(2):306-13. doi: 10.1016/j.jviromet.2011.02.024. Epub 2011 Mar 1.

Aemun E. P. (2006): Re-evaluation of Drought Early Warning System (EWS); Time-series Data and the Role of Indigenous Knowledge for Drought Management in Turkana District, Kenya. *Master's thesis* in Management of Natural Resources and Sustainable Agriculture. Department of International Environment and Development Studies, Noragric Norwegian University of Life Sciences, UMB

Akabwai D. (1992): Extension and livestock development: Experience from among the Turkana pastoralists of Kenya. Pastoral Development Network Paper, **33**b ODI (Overseas Development Institute), London, UK, 14 pp.

ALRMP (2009): Drought monitoring bulletins, Turkana North, Central, West, East, South and Loima Districts, 2009. http://www.aridland.go.ke/bullentins/2009/july/turkana.pdf. Accessed on 30th May 2011

ALRMP (2010): Drought monitoring bulletins, Turkana North, Central, West, East, South and Loima Districts 2009

http://www.aridland.go.ke/bullentins/2010/august/turkana.pdf

http://www.aridland.go.ke/bullentins/2010/sept/turkana.pdf

http://www.aridland.go.ke/bullentins/2010/oct/turkana.pdf

http://www.aridland.go.ke/bullentins/2010/nov/turkana.pdf

http://www.aridland.go.ke/bullentins/2010/dec/turkana.pdf. Accessed on 30th May 2011

Ameri, A.A., Hendrickx, S., Jones, B., Mariner, J., Mehta, P., and C. Pissang. (2009): Introduction to Participatory Epidemiology and its Application to Highly Pathogenic Avian Influenza Participatory Disease Surveillance: A Manual for Participatory Disease Surveillance Practitioners. Nairobi: ILRI

AMREF (2012): AMREF, Ngâ'Anadakarin Bamocha -Integrated Health and Social DevelopmentProgramme, website: http://www.amref.org/whatwe-do/nganadakarin-bamocha--integrated-healthand-social-development-programme/ Accessed on 17th February 2012

Amuyunzu, C.L., Oba, G., Turkana Resource Evaluation and Monitoring Unit, and UNESCO (1991): Vegetation Resources of Central Turkana District, Kenya. TREMU technical report. UNESCO, 1991

Anderson J and **Mackay JA (1994):** The detection of antibodies against Peste des Petits Ruminants virus in the cattle, sheep and the goats and the possible implication to rinderpest control programmes. Epidemiology and Infection **112** : 225– 231.

Anderson, J., Mckay, J. A. and **Butcher, R. N.(1991):** The use of monoclonal antibodies in competitive Elisa for detection of antibodies to rinderpest and peste des petite ruminants viruses. In: Seromonitoring of rinderpest throughout Africa, Phase one .Proceedings of final Research Coordinating Meeting of the FAO/IAEA/ SIDA/OAU/PARC coordinated Research Programme, Bingerville, Cote d'Ivoire, 19-23 November 1990. IAEA-TECDOC-623. pp. 43-53.

Anderson, R.M. and **May, R.M., (1991):** Infectious Disease of Humans. Oxford University Press, New York.

Anees M., Shabbir M.Z., Muhammad K., Nazir J., Shabbir M.A., Wensman J.J., and **Munir M. (2013):** Genetic analysis of peste des petitsruminants virus from Pakistan. BMC Veterinary Research 2013 9:60. doi:10.1186/1746-6148-9-60

Arasio L. R., (2004): Participatory assessment and analysis of Livestock markets, off take and marketing Constraints in Loima division, Turkana district,Kenya. MSc Thesis. Faculty of Veterinary Medicine , University of Nairobi

Ata, F. A., Al Sumry, S., King, G. J. J., Ismaili, S. I. and **Ata A. A. (1989):** Duration of maternal immunity to peste des petits ruminants. Veterinary Record **124**: 590-591.

Awa D. N., Ngagnou A., Tefiang E., Yaya D. and **Njoya A. (2003):** Post vaccination and colostral Peste des petits ruminants antibody dynamics in research flocks of Kirdi goats and Fulbe sheep of North Cameroon. Jamin J.Y., Seiny Boukar L., Floret C. (éditeurs scientifiques), 2003. Savanes africaines : des espaces en mutation, des acteurs face à de nouveaux défis. Actes du colloque, mai 2002, Garoua, Cameroun. Prasac, N'Djamena, Tchad -Cirad, Montpellier, France.

Awa D.N., Njoya A., and **Ngo Tama A.C. (2000):** Economics of Prophylaxis against Peste des Petits Ruminants and Gastrointestinal Helminthosis in Small Ruminants in North Cameroon. Tropical AnimalHealth and Production, 32 (2000) 391 403#2000Kluwer Academic Publishers. Printed in theNetherlands

Bailey, D., Banyard A., Dash P., Ozkul A. and **Barrett T. (2005):** Full genome sequence of peste des petits ruminants virus, a member of the Morbillivirus genus. Virus Research 110: 119–124.

Bailey, D., Barrett C.B., Little P.D. and **Chabari F., (1999):** Livestock markets and risk management among East African pastoralists: a review and research agenda. GL-CRSP Pastoral Risk Management Project Technical Report No. 03/99. Utah State University, Logan. 46 pp.

Balamurugan V., **Saravanan P., Sen A., Rajak K. K., Venkatesan G., Krishnamoorthy P., Bhanuprakash V.,** and **Singh R.K., (2012 a):** Prevalence of Peste des petits ruminants among sheep and goats in India. Journal of Veterinary Science. (2012), **13**(3), 279-285 http://dx.doi.org/10.4142/jvs.2012.13.3.279.

Balamurugan V., Sen A., Venkatesan G., Rajak K. K., Bhanuprakash V. and **Singh R. K. (2012b):** Study on passive immunity: Time of vaccination in kids born to goats vaccinated against Peste des petits ruminants Virologica Sinica August 2012, Volume **27**, Issue **4**, pp 228-233

Balamurugan V., Sen A., Venkatesan G., Yadav V., Bhanuprakash V., and **Singh R.K. (2010):** Isolation and identification of virulent peste des petits ruminants viruses from PPR outbreaks in India. Tropical Animal Health and Production. 2010 Aug;42(6):1043-6. doi: 10.1007/s11250-010-9527-0. Epub 2010 Mar 5.

Banyard A. C., Parida S., Batten C., Oura C., Kwiatek O., and **Libeau G.(2010):** Global distribution of peste des petits ruminants virus and prospects for improved

diagnosis and control. Journal of General Virology (2010), **91**, 2885–2897 doi:10.1099/vir.0.025841-0.

Banyard, A.C., Baron, M.D., and **Barrett, T., (2005):** A role for virus promoters in determining the pathogenesis of rinderpest virus in cattle. Journal of General Virology 86: 1083-92.

Barasa M., Catley A., Machuchu D., Laqua H., Puot E., Tap Kot D., and **Ikiror D. (2008):** Foot-and-Mouth Disease Vaccination in South Sudan: Benefit–Cost Analysis and Livelihoods Impact. Transboundary and Emerging Diseases. 55 (2008) 339–351

Baron M. D., (2012): Towards the eradication of Peste des petits ruminants. Global PPRV Research Alliance Inaugural meeting March 9th 2012 Wellcome Collection Conference Centre, London . http://www.iah.ac.uk/research/Paramyxo/docs/GPRA_meeting_report.pdf Accessed on November 5, 2013

Barrett T. (1999): Morbillivirus infections, with special emphasis on morbilliviruses of carnivores Veterinary Microbiology **69** (1999)

Barrett T. (2001): Morbilliviruses, dangers old and new, *In* : *New challenges to health: the threat of virus infection*, SMITH G.L., McCAULEY J.W., and ROWLANDS D.J. (editors), Society for General Microbiology, Symposium 60, Cambridge, United Kingdom : Cambridge University Press, 155-178

Barrett, T., Rossiter P.B. (1999): Rinderpest: the disease and its impact on humans and animals. Adv. Virus Res. 53: 89-110.

Barrett, T., Subbarao,S.M., Belsham G.J., and **Mahy B.W.J., (1991):** The molecular biology of the morbilliviruses, pp. 83-102. In D.W. Kingsbury (ed.) The paramyxoviruses. Plenum Press . New York.

Barrett, T., Visser, I.K.G., Mamaev, L., Goatley, L., Bressem, M.F., and **Van Osterhaus, A.D.M. (1993):** Dolphine and porpoise morbilliviruses are genetically distinct from phocine distemper virus. Virology, **193:** 1010 – 1012.

Bartlett, M.S. (1954): A note on the multiplying factors for various chi square approximations. Journal of Royal Statistical Society, **16**(Series B), 296-298.

Basto, M. and **Pereira, J.M. (2012):** An SPSS R-Menu for Ordinal Factor Analysis. Journal of Statistical Software January 2012, Volume **46**, Issue 4. http://www.jstatsoft.org/

Batten C.A., Banyard A.C., King D.P., Henstock M.R., Edwards L., Sanders A., Buczkowski H., Oura C. A. L. and **Barrett T. (2011):** A real-time PCR assay for the specific detection of peste des petits ruminants virus. J. Virol. Methods, **171** (2), 401–404.

Bazarghani, T. T., Charkhkar, S., Doroudi, J. and **Bani Hassan, E. (2006)**. A review on peste des petits ruminants (PPR) with special reference to PPR in Iran. Journal of Veterinary Medicine Series B-infectious Diseases and Veterinary Public Health vol. **53**, no. s1, pp. 17-18, 2006

Bennett R M. and Kitching A. S. (2000): Estimating the costs associated with livestock diseases - A study of 30 diseases of livestock in Great Britain open access content International Symposia on Veterinary Epidemiology and Economics

proceedings, ISVEE 9: Proceedings of the 9th Symposium of the International Society for Veterinary Epidemiology and Economics, Breckenridge, Colorado, USA, Economics & livestock production session, p 72, Aug 2000

Bennett S., Woods T., Liyanage W.M. and **Smith D.L. (1991):** A simplified general method for cluster-sample surveys of health in developing countries. World Health Statistics. Quarterly, 44 (3), 98–106.

Berghaus, R.D., Lombard J.E., Gardner I.A., and **Farver,T.B. (2005):** Factor analysis of a Johne's disease risk assessment questionnaire with evaluation of factor scores and a subset of original questions as predictors of observed clinical paratuberculosis. Preventive Veterinary Medicine, **72** (2005) 291–30

Berhe G. (2006) Development of Dual Vaccines for the Control of Peste des Petits Ruminants and Capripox Infections of Small Ruminants. *PhD thesis*. Institut National Polytechnique de Toulouse

Bett, B., Jost C., Allport R. and **Mariner, J. (2009):** Using participatory epidemiological techniques to estimate the relative incidence and impact on livelihoods of livestock diseases amongst nomadic pastoralists in Turkana South District, Kenya. Preventive. Veterinary. Medicine, **90**:194-203.

Bodjo S. C., Couacy-hymann E, Koffi M. Y. and **Danho T. (2006):** Assessment of the duration of maternal antibodies specific to the homologous peste des petits ruminant vaccine “Nigeria 75/1" in Djallonké lambs Biokemistri, Vol. **18**, No. 2, December, 2006, pp. 99-103

Boinski S. and **Garber P. A., (2000).** On the Move: How and Why Animals Travel in Groups. University of Chicago Press, May 15, 2000 - Nature - 811 pages. ISBN 0 226 06339 9 and ISBN 0 226 06340 2 http://books.google.co.ke/books?isbn=0226063399 Accessed on November 5, 2013

Boklund A., Alban L., Mortensen S., and **Houe H. (2004).** Biosecurity in 116 Danish fattening swineherds: descriptive results and factor analysis. Preventive Veterinary Medicine, **66** (2004) 49-62.

Bosch, C.H., (2006). Aloe turkanensis Christian. In: Schmelzer, G.H. & Gurib-Fakim, A. (Editors). Prota 11(1): Medicinal plants/Plantes médicinales 1. [CD-Rom]. PROTA, Wageningen, Netherlands. http://database.prota.org/PROTAhtml/Aloe turkanensis_En.htm accecesed on 30th December 2013.

Bourdin P. and **Laurent A. (1967):** Note sur la structure du virus de la peste des petits ruminants, Rev. Elev. Med. vét. Pays trop., **20** (3), 383-386.

Bourdin, P. (1973): La peste des petits ruminants (PPR) et sa prophylaxie au Sénégal et en Afrique de l'Ouest. Rev.Elev Méd.vét. Pays trop. **26** :71-74.

Braide, V.B. (1981): Peste des petits ruminants. World animal. Review.**39**: 25-28.

Brooks G.F., Butel J.S., and **Morse S.A. (2008):** "Jawetz, Melnick, & Adelberg's Medical Microbiology", **24**.th edition, McGraw-Hill Companies inc., USA

Brown, C.C., Mariner, J.C., and **Olander, H.J. (1991):** An immunohistochemical study of the pneumonia caused by PPR virus. Veterinary Pathathology. **28**: 166-170.

Bundza A., Afshar A., Dukes T.W., Myers D.J., Dulac Susi G. and **Becker, A.W.E. (1988):** Experimental PPR (goat plague) in Goats and sheep. Canadian J. Vet. Res. 52, 46-52.

Catley A., Burns J., Abebe D., and **Suji O.,(2008):** Participatory Impact Assessment A Guide for Practitioners. Feintein International Centre, Tufts University, 2008

Catley, A., (2005): Participatory Epidemiology: A guide for Trainers. African Union / Interafrican Bureau for Animal Resources, Nairobi. http://www.participatoryepidemiology.info/PE Guide electronic copy.pdf *accessed on 12/9/2008*

Catley, A., Alders, R.G., and **Wood, J. L. N., (2012):** Participatory epidemiology: approaches, methods, experiences. The Veterinary Journal **191**, 151-160

Catley, A. and **Mariner, J. (2002):** Where there is no data: participatory approaches to veterinary epidemiology in pastoral areas of the Horn of Africa. IIED. Issue paper No. **110**.

Cattell, R.B. (1966). The scree test for the number of factors. Multivariate Behavioural Research, **1**:245-276.

CFSPH (2008). Peste des Petits Ruminants – CFSPH Technical Disease Fact Sheets . DPR_A2008 ©2008 CFSPH. Website: www.ivis.org/advances/Disease_Factsheets/peste_des_petits_ruminants.pdf . Accessed on 21st May 2009

Chauhan H. C., Chandel B. S., Kher H. N., Dadawala A. I., Agrawal S. M., (2009). Pesti des petits ruminants virus infection in animals. Veterinary World, Vol.**2** (4):150-155

Chauhan, H.C., Lambade, P.S., Sen, A., Dadawala, A.I., Ranaware, P.B., Chandel, B., Joshi, D.V., Patel, S.S., Pankaj, K., Shah, N.M. and **Kher, H.N. (2011):** The use of pathological and histopathological techniques in the diagnosis of Peste des petits ruminants in India. Veterinaria Italiana, **47(**1): 41-47.

Choi, K.S., Nah, J.J., Ko, Y.J., Kang, S.Y., Yoon, K.J. and **Jo, N.I. (2005):** Antigenic and immunogenic investigation of B-cell epitopes in the nucleocapsid protein of peste despetits ruminants virus. Clinical and diagnostic laboratory immunology. **12(1),** 114-21.

Cosseddu GM, Pinoni C, Polci A, Sebhatu T, Lelli R, and **Monaco F. (2013):** Characterization of peste des petits ruminants virus, Eritrea, 2001–2011. Emerging Infectious Diseases. 2013 Jan. http://dx.doi.org/10.3201/eid1901.121072

Couacy-hymann E., Bodjo S. C., Danho T., Koffi M. Y., Libeau G., and **Diallo A. (2007):** Early detection of viral excretion from experimentally infected goats with peste des petits ruminants virus. Preventive Veterinary Medicine*,* **78***:85-88*

Couacy-Hymann E., Roger F., Hurard C., Guillou J.P., Libeau G., and **Diallo A. (2002):** Rapid and sensitive detection of peste des petits ruminants virus by a polymerase chain reaction assay. Journal of Virological Methods 100: 17-25.

Cumming, G. S. (2010): Risk mapping for avian influenza: a social–ecological problem. Ecology and Society **15**(3): 32. [online] URL: http://www.ecologyandsociety.org/vol15/iss3/art32/

De Nardi M., Lamin Saleh S. M., Batten C., Oura C., Di Nardo and **A. Rossi D. (2011):** First Evidence of Peste des Petits Ruminants (PPR) Virus Circulation in Algeria (Sahrawi Territories): Outbreak Investigation and Virus Lineage Identification Transboundary and Emerging Diseases. Volume **59**, Issue 3, pages 214–222, June 2012.

DeCoster, J. (2005): Scale construction notes. Website:http://www.stat-help.com/notes.html. Accessed on 2nd January 2011

Degen A. A., (2007): Sheep and goats milk in pastoral societies Small ruminants research **68** (2007) 7-19

De-Vries D.H. (2002): Livestock redistribution dynamics in nomadic pastoralist herd demography: A case study among the ngisonyoka Turkana of south Turkana, Kenya. *Master thesis.* Department of Anthropology, University Of North Carolina Chapel. Chapel Hill 2002.

Dhar B.P., Barrett T., Corteyn M., Sing R.P. and **Bandyopadhyay S.K. (2002):** Recent epidemiology of peste de petits ruminants virus. Veterinary Microbiology **88**: 153-159.

Diallo A. (1990): Morbillivirus group : genome organisation and proteins, *Vet Microbiol*., **23**, 155-163.

Diallo A. (2000): Peste des petits ruminants. In: OIE Manual of Standards for Diagnostic Tests and Vaccines, 4tth edition .Chapter 2. 1. 5. Office international des Epizooties, Paris, pp. 114-122.

Diallo A. (2003): Control of peste des petits ruminants: classical and new generation vaccines. Developmental Biology (Basel) 114, 113–119.

Diallo A. (2004): Peste des petits ruminants. In: OIE Manual of Standards for Diagnostic Tests and Vaccines for Terrestrial Animals, 5th edition. Chapter 2.1.5. Office international des Epizooties, Paris, pp. 153-162**.**

Diallo A. Taylor, W.P. Lefèvre P.C . and **Provost, A. (1989a):** Atténuation d'une souche de virus dela peste des petits ruminants: candidat pour un vaccin homologue vivant. Revue D'élevage et de Médecine Vétérinaire Des **Pays** Tropicaux **42**: 311-319.

Diallo A., (2006): Control of Peste des petits ruminants and poverty alleviation. Journal Veterinary Medecine, **53**: 11-13

Diallo, A., Barret T., Barbon M., Shaila M., Subbarao T. and **William P. (1989b):** Differentiation of rinderpest and peste des petits ruminantss viruses using specific cDNA clones. J. Virol. Methods. **23**: 127-136.

Diallo, A., Barrett, T., Barbron, M., Meyer, G. and Lefevre, P.C. (1994): Cloning of the nucleocapsid protein gene of peste des petits ruminants virus: relationship to other Morbilliviruses. Journal of General Virology **75**: 233-237.

Diallo, A., Minet, C., Le Goff, C., Berhe, G., Albina, E., Libeau, G. and **Barrett, T. (2007):** The threat of peste des petits ruminants: progress in vaccine development for disease control. Vaccine **25**, 5591–5597.

Diop, M., Sarr, J. and **Libeau, G. (2005):** Evaluation of novel diagnostic tools for Peste des petits ruminants virus in naturally infected goat herds. Epidemiology and infection **133**(4), 711-7.

DiStefano, C, **Zhu, M.,** and **Mîndrilă, D. (2009):** Understanding and Using Factor Scores: Considerations for the Applied Researcher. Practical Assessment, Research & Evaluation, **14** (20).: http://pareonline.net/getvn.asp?v=14&n=20. Accessed on 16 November 2011

Domenech J. (2013): Peste des Petits Ruminants Development of a global PPR Control Strategy Introduction Workshop on PPR prevention and control in the SADC Region 10-12 June 2013 Dar es Salam Tanzania . http://www.rr-frica.oie.int/docspdf/en/2013/PPR/DOMENECH2.pdf Accessed on November 5, 2013

Dufour L. (2010): The plague of small ruminants: Moroccan outbreak of 2008, a danger to Europe? *PhD thesis.* The Faculty of Medicine, Creteil, National Veterinary School of Alfort.

Durojaiye O. A. **(1982):** Precipitating antibody in sera of goats naturally affected with peste des petits ruminants. Tropical Animal Health and Production, **14**:98-100.

Durojaiye, O. A. **(1987):** Application of a precipitinogen inhibition test in the detection of antibody to peste des petits ruminants virus . Revue D'Elevage et de Medecine Veterinaire des Pays Tropicaux, **40**(1):17-20.

Elhag, A. B. and **Taylor W. P. (1984):** Isolation of Peste des petits ruminants virus from the Sudan. Research in Veterinary Science, **36,** 1–4.

Elsawalhy A., Mariner J. C. , Chibeu D., Wamwayi H., Wakhusama S., Olaho-Mukani W., and **Toye P. (2010):** Pan African strategy for the progressive control of Peste des petits ruminants (Pan African PPR strategy) Bulletin of Animal Health and Production in Africa, (2010), Volume **58**. No. 3. 185-193.

FAO (2006): Turkana livelihood map 2006. Geographic FAO/TCEO May 2006

FAO (2008): Peste des petits ruminants (PPR) in Morocco. EMPRES WATCH August 2008. Website: ftp://ftp.fao.org/docrep/fao/011/aj120e/aj120e00.pdf. Accessed on 21st may 2009.

FAO (2009a): Peste des Petits Ruminants [PPR]. A challenge for small ruminant production. ww.fao.org/docs/eims/upload/258701/ak065e00.pdf. *Accessed on 12th November 2009.*

FAO (2009b): Transboundary Disease. Bulletin 33. ftp://ftp.**fao**.org/docrep/**fao**/011/i0919e/i0919e00.pdf . Accessed on 12th November 2009.

FAO (2012a): FAO supports study on the socioeconomic impacts of peste de petits ruminants in Kenya.

http://www.fao.org/ag/againfo/home/en/news_archive/2010_FAO_supports_study.html Accessed on 21st July 2013

FAO (2012b): FAO and partners develop joint strategies for PPR control in East Africa. 24th October 2012. http://www.fao.org/ag/againfo/programmes/en/empres/news_241012b.html Accessed on 14th November 2013

FAO (2014): E-Conference to launch Global Research and Expertise Network on PPR. EMPRES Animal Production and Health Division FAO. http://www.fao.org/ag/againfo/programmes/en/empres/news_030214b.html accessed on 4th April 2014

Field, A.P. (2009): Discovering statistics using SPSS, London, England : SAGE.

Finke J (2003): Turkana -Livestock (and Agriculture) Traditional Music & Cultures of Kenya. Copyright Jens Finke, 2000-2003. http://www.bluegecko.org/kenya/tribes/turkana/livestock.htm#cattle Accessed on November 5, 2013

Forsyth, M.A. and **Barrett, T. (1995):** Evaluation of polymerase chain reaction for the detection and characterisation of rinderpest and peste des petits ruminants viruses for epidemiological studies. Virus Research **39**: 151-163.

Furley, C.W., Taylor, T.W. and **Obi, T.U. (1987):** An outbreak of PPR in a zoological collection. Veterinary Record **121**: 443-447.

Gibbs, P.J.E., Taylor, W.P. Lawman, M.P. and **Bryant, J. (1979):** Classification of the peste des petits ruminants virus as the fourth member of the genus Morbillivirus. Intervirology, **11**: 268 – 274.

Gilbert Y and **Monier J. (1962):** Adaptation du virus dela PPR aux cultures celluaire. Rev. Elev. Med Vet pays trop **15**: 321 335

Gill G.W. (n.d.): CT(ASCP); Chapter 13 , H&E Staining: Oversight and Insights; http://www.dako.com/08066_12may10_webchapter13.pdf accessed on 20 January 2014

GOK (2008): Emergency project for the control of pestes des petit ruminants (PPR) in Kenya; Situation analysis and concept note, June 2008. www.hackenya.org/index.php?option=com_docman&task=doc_download&gid=474&Itemid=376. *Accessed on 21st May 2009.*

Goldstein T, **Mazet J.A.K., Gill V.A., Doroff A.M., Burek K.A.,** and **Hammond J.A. (2009):** Phocine distemper virus in northern sea otters in the Pacific Ocean, Alaska, USA. Emerg Infect Dis [serial on the Internet]. 2009 Jun [*date cited*]. Available fromhttp://wwwnc.cdc.gov/eid/article/15/6/09-0056.htm

Gopilo A. (2005): Epidemiology of Peste des Petits Ruminants virus in Ethiopia and molecular studies on virulence. *PhD thesis*. Institut National Polytechnique de Toulouse

Grace D., Randolph T., Olawoye J., Dipelou M. and **Kang'ethe E. (2008):** 'Participatory risk assessment: a new approach for safer food in vulnerable African communities', Development in Practice, **18**:4,611 — 618

Grenfell B. T. and **Dobson A. P. (1995):** Ecology of infectious diseases in natural populations. Edition: illustrated, reprint Published by Cambridge University Press, 1995 ISBN 0521465028, 9780521465021, 521 pages.

Gur S. and Albayrak H. (2010): Seroprevalance of Peste des Petits Ruminants (PPR) in Goitered Gazelle (Gazella subgutturosa subgutturosa) in Turkey. Journal of Wildlife Diseases, **46(2),** 2010, pp. 673–677

Hamdy F. M., Dardiri A. H., Nduaka D., Breese S. S., and **Ihemelandu E. C. (1976):** Etiology of stomatitis pneumocomplex in Nigeria dwarf goats. Canada. Journal of Comparative. Medicine. **40**, 276–284.

Haque M.E., Habib S., Islam M.R., Khan K.A., Hannan A.S.M.A., Anowar A.K.M.M. and **Nadir E.U.A. (2004):** Sero-monitoring of Peste Des Petits Ruminants (PPR) Antibodies in Small and Large Ruminants in Bangladesh. Journal of Animal and Veterinary advances 2004 **3(7)**: 453-458.

Hatcher L., (1994): Exploratory factor analysis. A Step by- Step Approach to Using the SAS System for Factor Analysis and Structural Equation Modeling, SAS Institute, Cary, USA, pp: 57–127.

Heffernan, J.M. and **Wahl, L.M. (2005)**: 'Monte Carlo estimates of natural variation in HIV infection', Journal of Theoretical Biology **236**, 137–153.

Hethcote H.,W. (2000): The Mathematics of Infectious Diseases. SIAM REVIEW, Vol. **42**, No. 4, pp. 599–653 (2000) Society for Industrial and Applied Mathematics.

Hu Q., Chen W., Huang K., Baron M. D., and **Bu1 Z. (2012):** Rescue of recombinant peste des petits ruminants virus: creation of a GFP-expressing virus

and application in rapid virus neutralization test. Veterinary Research 2012 **43**:48. doi:10.1186/1297-9716-43-48

Imana C. A. (2008): Goat rearing as a livelihood strategy of Turkana Pastoralists in north west Kenya. A dissertation submitted to the Centre for Development Support in partial fulfillment of the requirements of the degree of Master of Arts in Development Studies of the Centre for Development Support, UFS. University of the Free State, Bloemfontein, South Africa.

Islam A., Singha A., Islam M. A., and **Majunder S., (2011):** Peste des petit ruminant of goats, outbreak and economic losses: Case study. Pakistan journal of scientific industrial research. Serial B. Biological sciences. 2011 **54** (2) 76-82

ITDG and **IIRR (1996):** Ethnoveterinary medicine in Kenya: A field manual of traditional animal health care practices. Intermediate Technology Development Group and International Institute of Rural Reconstruction, Nairobi, Kenya,

Johnson R. H. and **Ritchie J.S.D. (1968):** A virus associated with pseudorinderpest in Nigerian Dwarf goats. Bulletin of Epizootic Diseases of Africa **16**:411-417.

Jost C. C., Nzietchueng S., Kihu S., Bett B., Njogu G., Swai E. S. and **Mariner J. C. (2010):** Epidemiological Assessment of the Rift Valley Fever Outbreak in Kenya and Tanzania in 2006 and 2007 Am J Trop Med Hyg 2010 vol. **83** no. 2 Suppl 65-72 doi: 10.4269/ajtmh.2010.09-0290

Kaiser H.F. (1970): A second generation little jiffy. Psychometrika, **35**, 401–415.

Khalafalla A.I., Saeed I.K., Ali Y.H., Abdurrahman M.B., Kwiatek O., Libeau G., Obeida A.A. Abbas, Z. (2010): An outbreak of peste des petits ruminants (PPR) in

camels in the Sudan Acta Tropica, Volume **116**, Issue 2, November 2010, Pages 161-165.

Kihu S.M., **Gitao C.G., Bebora L.C., Njenga M.J., Wairire G.G., Maingi N.** and **Wahome R.G. (2012b):** Participatory risk assessment of Peste des petit ruminants: Factor analysis of small ruminants pastoral management practices in Turkana district, Kenya. Research opinions in animal & veterinary sciences,, **2(9),** 503-510.

Kihu S.M., Njagi **L.M., Njogu G.N., Kamande J.N.** and **Gitao C.G., (2012a):** Peste des petits ruminants in Kenya; Pastoralist knowledge of the disease in goats in Samburu and Baringo Counties. Research opinions in animal & veterinary sciences, **2(11),** 544-553.

Kim, H. (2008): Common Factor Analysis Versus Principal Component Analysis: Choice for Symptom Cluster Research. Asian Nursing Research, March 2008 Vol **2** No 1

Kinne J., Kreutzer R., Kreutzer M., Wernery U. and **Wohlsein P. (2010):** Peste des petits ruminants in Arabian wildlife. Epidemiology and Infection **138**(8):1211–1214

Kivaria F.M., (2006): Estimated direct economic costs associated with tick-borne diseases on cattle in Tanzania. Tropical Animal Health and Production (20060 **38**:291-299. DOI 10.1007/s 11250-006-4181-2

Kivaria, F.M., Kwiatek, O., Kapaga, A.O., Swai, E.S., Libeau, G., Moshy, W., Mbyuzi A. O. and, **Gladson J. (2013):** 'The incursion, persistence and spread of peste des petits ruminants in Tanzania: Epidemiological patterns and predictions',

Onderstepoort Journal of Veterinary Research **80**(1), Art #593, 10 pages. http:// dx.doi.org/10.4102/ojvr. v80i1.593

KNBS (2010): 2009 Kenya population and housing census Volume 1 A, Population distribution by administrative units. Kenya National Bureau of Statistics, August 2010

Kumar P., Tripathi, B .N. Sharma, A. k., Kumar, R., Sreenivasa, B .P. ,Singh, R. P., Dhar, P. and **Bandyopadhyay, S. K . (2004):** Pathological and immunohistochemical study of experimental peste des petits ruminants virus infection in goats. Journal of Veterinary Medicine B, Infectious Diseases Veterinary Public Health, **51:** 153-195.

KVAPS (2009): Peste des Petit Ruminants (PPR) Post Vaccination Report, August 2009

Kwiatek O., Minet C., Grillet C., Hurard C., Carlsson E., Karimov B., Albina E., Diallo A. and **Libeau G.. 2007:** Peste des petits ruminants (PPR) outbreak in Tajikistan. J. Comp. Pathol. **136**:111–119.

Last R. D., Jardine J. E., Smit M. M. and **van der Lugt, J. J. (1994):** Application of immunoperoxidase techniques to formalin –fixed brain tissue for the diagnosis of rabies in Southern Africa. Onderstepoort Journal of Veterinary Research, **61**(2):183-187.

Laurent A. **(1968):** Aspects biologiques de la multiplication du virus de la peste des petits ruminants ou PPR sur les cultures cellulaires*, Rev. Elev. Méd. Pays trop*., **21** (3), 297-308.

Lefevre P.C. (1982): *Peste des Petits Ruminants et infection bovipestique des ovins et des caprins*, Etudes et synthèses de l'IEMVT, **5**, 99p.

Lefèvre, P.C. (1987): Peste des petits ruminants et infection bovipestique des ovins et caprins (Synthèse bibliographique), Institute d'Elevage et de Médecine vétérinaire des pays tropicaux, 94704 Maison-Alfort, France, 99pp.

Lefèvre, P.C. and **Diallo, A. (1990):** Peste des petites ruminants. Revue Scientifique Office International des Epizooties, **9**: 951-965.

Libeau G., Kwiatek O., Lancelot R., and **Albina E. (2011):** Peste des petits ruminants, growing incidence worldwide. 2011, *the OIE and its partners* 2011 (**2**) page 52-54. Animal Health Information Department-OIE. www.oie.int/doc/ged/D10850.PDF

Libeau G., Prehaud C., Lancelot R., Colas F., Guerre L., Bishop D. H. L. and **Diallo A. (1995):** Development of a competitive ELISA for detecting antibodies to the peste des petits ruminants virus using a recombinant nucleoprotein Research in Veterinary Science *1995* ***58****, 50-55*

Libeau G., Diallo A. and **Parida S. (2014):** Evolutionary genetics underlying the spread of peste des petits ruminants virus Animal Frontiers January 2014 **4**:14-20; doi:10.2527/af.2014-0003

Liehr T. and **Manvelyan M. (2009):** Characterization of Archived Formalin-Fixed/Paraffin-Embedded or Cryofixed Tissue, Including Nucleus Extraction DOI: 10.1007/978-3-540-70581-9_15; Page Range: 147-156

Editor(s): Thomas Liehr Fluorescence In Situ Hybridization (FISH) Application Guide ISBN: 978-3-540-70580-2

Luka P.D., Erume J., Mwiine F.N. and **Ayebazibwe C. (2011):** Seroprevalence of Peste des petits ruminants Antibodies in Sheep and Goats after Vaccination in Karamoja, Uganda: Implication on Control. International Journal of Animal and Veterinary Advances **3(1):** 18-22, 2011. ISSN: 2041-2908

Luka P.D., Erume J., Mwiine F.N. and **Ayebazibwe C. (2012):** Molecular characterization of peste des petits ruminants virus from the Karamoja region of Uganda (2007-2008). Archives of Virology. 2012 Jan;**157**(1):29-35. doi: 10.1007/s00705-011-1135-4. Epub 2011 Oct 7.

MacDiarmid S. C. (1991): Risk analysis and the importation of animals. Surveillance **18**(5): 8 - 11.

MacDiarmid S.C. and **Thompson E.J. (1997):** The potential risks to animal health from imported sheep and goat meat, *Rev. Sci. Techn. OIE*, **16** (1), 45-56.

Maganga, G.D., Verrier D., Zerbinati R.M., Drosten C., Drexler J.F. and **Leroy E.M.. (2013):** Molecular typing of PPRV strains detected during an outbreak in sheep and goats in south-eastern Gabon in 2011. Virology Journal **10**:82.

Mahapatra, M., Parida, S., Baron, M. D. and **Barrett, T. (2006):** Matrix protein and glycoproteins F and H of peste-des-petits-ruminants virus function better as a homologous complex. The Journal of general virology **87**, 2021–2029.

Mai H.M., Saidu I., Obasi O.L. and **Iliyasu M.A. (2004):** Effects of Vaccination on the Prevalence of Peste Des Petits Ruminants (PPR) in Small Ruminants in Taraba

State, Nigeria. Pertanika Journal of Tropical. Agricultural Science. **27**(2): 101 - 105 (2004).

Majiyagbe K. A., Nawathe D. R. and **Abegunde A.(1984):** Rapid Diagnosis of peste des petite ruminants (PPR) infection, application of Immunoelectroosmophoresis (IEOP) technique. Revue d'Elevage et de Médecine Vétérinaire des pays tropicaux **37**(1):11-15.

Marete A. (2011): Sheep. http://www.infonet-biovision.org/print/ct/269/livestockSpecies accessed on 22 January 2014

Mariner J.C., McDermott J. , Heesterbeek J.A.P. , Catley A. and **Roeder P. (2005):** A model of lineage-1 and lineage-2 rinderpest virus transmission in pastoral areas of East Africa. Preventive Veterinary Medicine **69** (2005) 245–263

Mariner, J. and **Paskin R., (2000):** FAO Animal Health Manual. Manual on Participatory epidemiology. Rome 2000. http://www.fao.org/docrep/003/X8833E/X8833E00.htm *Accessed on 19/5/2005*

McCabe J.T. (1984): Food and the Turkana in Kenya. Nomads: Stopped in Their Tracks? Cultural Survival Quarterly Back Issues 8.1 (Spring 1984) Website: http://www.culturalsurvival.org/publications/cultural-survival-quarterly/81-spring-1984-nomads-stopped-their-tracks. *Accessed on 25th June 2012*

McCabe, J. (1996): "Turkana." Encyclopedia of World Cultures. 1996. Encyclopedia.com. http://www.encyclopedia.com/topic/Lake_Turkana.aspx *Accessed on 25th June 2012*

Merck Sharp and **Dohme Corp.,(2009):** The Merck Veterinary Manual. A subsidiary of Merck & Co., Inc. Whitehouse Station NJ, USA. Downloaded on 27th September, 2009. http://www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/56100.htm Accesed on 27th September 2009

Msafiri F. (1996): Inventory and conservation of economic plant genetic resourcesin Kenya rangeland: a case of Turkana district. Proceedings of the XIV th EATFAT congress Wageningen, The Netherlands. pp 220–223**.**

Munir M, Zohari S, and **Berg M. (2013):** Molecular Biology and Pathogenesis of Peste des Petits Ruminants Virus. 2013:**151**. ISSN 2211-7504, ISBN 978-3-642-31450-6, DOI 10.1007978-3-64231451-3 Springer Briefs in Animal Sciences; http://www.springer.com/978-3-642-31450-6.

Munir M., Abubakar M., Zohari S. and **Berg M. (2012):** Serodiagnosis of Peste des Petits Ruminants Virus. Serological Diagnosis of Certain Human, Animal and Plant Diseases, 2012 Eds Moslih Al-Moslih p. cm.ISBN 978-953-51-0370-7

Munir, M., Siddique M., Shehzad A., Zohari S. and **Stahl K. (2008):** Sero-prevalence of antibodies to Peste des Petits ruminants at various governmental livestock farms of Punjab, Pakistan. Asian Journal of Epidemiology, **1**, 82-90.

Murphy S.K. and **Parks G.D. (1999):** RNA replication for the paramyxovirus simian virus 5 requires an internal repeated (CGNNNN) sequence motif. Journal of Virology **73**: 805-9

Muse E.A., Matondo R.B., Karimuribo E.D., Misinzo G., Albano M.O. and Gitao **G.C. (2012):** Clinico-pathological findings of the 2011 outbreak of Peste des Petits

Ruminants (PPR) in Tandahimba district, southern Tanzania. Research Opinion in Animal and Veterinary Science **2**(4), 256-262.

Nanda, Y. P., Chatterjee, A., Purohit, A. K., Diallo, A., Innui, K., Sharma, R. N., Libeau, G., Thevasagayam, J. A. and **Bruning, A. (1996):** The isolation of Peste des petits ruminants virus from northern India. Veterinary Microbiology **51**, 207–216.

Neogen (2012): ELISA Assay Procedure website: http://www.neogen.com/LifeSciences/Assay_Principle.html. *Accessed on 24th November 2012.*

Newcastle University (2012): Animal disease research misses the human perspective, say researchers. Science Daily. Retrieved October 11, 2012, from http://www.sciencedaily.com/releases/2012/05/120515070353.htm

Njanja, J.C. (1991): Livestock ecology in Central Turkana. United nation educational Scientific and Cultural organisation TREMU Technical Report No. E - 1

Norrby, E. and **Oxman M.N., (1990):** Measles virus, PP; 1013-1044. In B.N. Fields et al., Virology, 2nd edition, Vol. **I**, Raven Press, New York 1: 1013 – 1044.

Nyamweya, M., **Otunga, T., Regassa, G.** and **Maloo, S,. (2009):** Technical brief of peste des petits ruminants virus. ELMT Livestock Services Technical Working Group.

O'Connor, B.P. (2000): SPSS and SAS programs for determining the number of components using parallel analysis and Velicer's MAP test. Behavior Research Methods, Instruments & Computers, **32** (3): 396-402

Oba G. (1992): Ecological factors in land use conflicts, land administration and food insecurity in Turkana, Kenya. *ODI* Pastoral Development Network Paper **33a**. Overseas Development Institute, London.

Obi T . U . and **Patrick, D . (1984):** The detection of peste des petits ruminants (PPR) virus antigen by agar gel precipitation test and counter-immunoelectrophoresis. Journal of Hygiene, Cambridge, **93**: 579-586.

Ogunsanmi A.O., Awe E.O., Obi T.U. and **Taiwo V.O. (2003):** Peste des petits ruminants (PPR) virus antibodies in african grey duiker (sylvicapra grimmia). African Journal of Biomedical Research, Vol. **6** (1); 59 – 61 (2003)

Ohta I. (1984): Symptoms Are Classified into Diagnostic Categories: Turkana's View of livestock Diseases. Faculty of Science, Kyoto University. African Study Monographs, Supplementary Issue **3**: 71-93, March 1984

Ohta I. (2007): Marriage and bridewealth negotiations among the Turkana in northwestern Kenya. African Study Monographs, Suppl. **37**: 3-26, December 2007 Center for African Area Studies, Kyoto University

Ohta, I. (1982): Man-animal interaction complex in goat herding of the pastoral Turkana. African Study Monographs, Supplementary Issue, (**1**): 13-41.

OIE (2012): Peste des petits ruminants [PPR], Angola. Weekly Disease Information 2012; 25(41) [edited]. France, OIE, WAHID (World Animal Health Information Database). weekly disease information 2012; 5(41) 1. http://www.oie.int/wahis_2/public/wahid.php/Diseaseinformation/statusdetail/popu

p?diseaseid=5&country=AGO&y=2012&m=7&admin1=113&detail=2&sdid=536226 Accessed on 28th December 2012.

OIE (2014): Disease outbreak summary, Kenya http://www.oie.int/wahis_2/public/wahid.php/Diseaseinformation/statusdetail/popup?diseaseid=5&country=KEN&y=2006&m=8&admin1=1590&detail=2&sdid=97395 http://www.oie.int/wahis_2/public/wahid.php/Diseaseinformation/statusdetail/popup?diseaseid=5&country=KEN&y=2007&m=2&admin1=1590&detail=2&sdid=220067 http://www.oie.int/wahis_2/public/wahid.php/Diseaseinformation/statusdetail/popup?diseaseid=5&country=KEN&y=2007&m=3&admin1=1590&detail=2&sdid=220069 Accessed on 15th January 2014

OIE (2013): Version adopted by the World Assembly of Delegates of the OIE in May 2012; Ch a p t e r 2 . 7 . 1 1 .Peste des petits ruminants. http://www.oie.int/fileadmin/Home/fr/Health_standards/tahm/2.07.11_PPR.pdf Accessed in 27th July 2013.

Okumu W. (2013): From Ethnic Violence to Seeds of Peace: Pastoralists, Youth, and Intercommunity Peace building in Northwestern Kenya. http://forums.ssrc.org/kujenga-amani/2013/02/11/from-ethnic-violence-to-seeds-of-peace-pastoralists-youth-and-inter-community-peacebuilding-in-northwestern-kenya/#sthash.hc91LyD3.dpuf Accessed on November 5, 2013

Omolo N.A., (2010): Gender and climate change-induced conflict in pastoral communities: Case study of Turkana in northwestern Kenya Vol African Journal on Conflict Resolution **10**, No **2** (2010) http://dx.doi.org/10.4314/ajcr.v10i2.63312

O'Neill P.D. (2010): Introduction and snapshot review: relating infectious disease transmission models to data. Stat Med. 2010 Sep 10;29(**20**):2069-77. doi: 10.1002/sim.3968.

Opasina B.A. and **Putt S.N.H. (1985):** Outbreaks of peste des petits ruminants in village goat flocks in Nigeria. Tropical Animal Health and Production **17**: 219-224

Osman N. A., Ali A. S., Me A. R. and **Fadol M. A. (2009):** Antibody seroprevalences against Peste des petits ruminants (PPR) virus in sheep and goats in Sudan. Tropical Animal Health and Production **41**, 1449–1453.

OXFAM 2006: Household Economy Assessment of North East Turkana. HEA baseline NE Turkana, 2006. https://googledrive.com/host/0B5kizcQrKucOTml5VS1rUFZQSEE/OxfamTurkanareport.pdf .Accessed on 21st July 2013

Ozkul A., Akca Y., Alkan F., Barrett T., Karaoglu T., Dagalp S.B., Anderson J., Yesilbag K., okcaliskan C., Gencay A. and **Burgu I**. **(2002):** Prevalence, distribution, and host range of Peste des petits uminants virus, Turkey. Emerging infectious Diseases, **8** (7), 708-712.

Pallant, J. (2005): SPSS Survival Manual: a step by step guide to data analysis using SPSS version 12. 2nd Edition.

PARC, **(2007):** Pakistan Agricultural Research Council. (PARC) Animal health http://www.parc.gov.pk/1SubDivisions/NARCASI/ah.html Assessed on 20th July 2013.

Parida S, Mahapatra M, Kumar S, Das SC, Baron MD, Anderson J., and **Barrett T. (2007):** Rescue of a chimeric rinderpest virus with the nucleocapsid protein derived from peste-des-petits-ruminants virus: use as a marker vaccine. Journal of General Virology. 2007 Jul;**88 (Pt 7**):2019-27.

Peperkamp G., and **Remie C. (eds) (1989):** The Struggle for Land World-wide**.** The struggle for land and livestock among theTurkana, (ex)-nomadic pastoralists of north-west Kenya by Rutten M. M. E. ,M. NICCOS 1989 Nijmegen Page 48 – 71. http://hdl.handle.net/1887/9012

Perry B.T., **Mcdermott J., Ones S., K.,** and **Tornton P., K. (2002):** Investing in Animal Health Research to alleviate Poverty, International livestock research institute (ILRI), Nairobi, Kenya, and p.148.

ProMed-Mail (2007): Peste des petits ruminants - Kenya (Rift Valley): OIE. 20070117.0226: http://www.promedmail.org/pls/otn/f?p=2400:1001:4263237816442653::::F2400_P1001_BACK_PAGE,F2400_P1001_ARCHIVE_NUMBER,F2400_P1001_USE_ARCHIVE:1202,20070117.0226,Y. Accessed on 21st May 2009.

PROMESA (2011): PROMESA is a programme for statistical sampling in animal populations. Website: http://www.promesa.co.nz/ProMESA_Home.htm. *Accessed on 18/5/2011*

Puchtler H., Meloan S.N., (1985): On the chemistry of formaldehyde fixation and its effects on immunohistochemical reactions. Histochemistry. 1985;82(3):201-4.

QIAGENiagen (2010): RNeasy® Mini Kit ,For purification of total RNA from animal cells, animal tissues, bacteria, and yeast, and for RNA cleanup. http://www.genome.duke.edu/cores/microarray/services/rna-qc/documents/RNeasy_Mini_Handbook.pdf accessed on 30th July 2013.

QIAGENiagen (2011): RNeasy® FFPE Handbook For purification of total RNA from formalin fixed, paraffin-embedded tissue sections. http://www.algimed.by/download/EN-RNeasy-FFPE-Handbook.pdf accessed on 30th July 2013.

Qin J, Huang H, Ruan Y, Hou X, and **Yang S, (2012):** A Novel Recombinant Peste des Petits Ruminants-Canine Adenovirus Vaccine Elicits Long-Lasting Neutralizing Antibody Response against PPR in Goats. PLoS ONE **7(5):** e37170. doi:10.1371/journal.pone.0037170

Ramanathan, A., Steed, C.A. and **Pullum, L.L., (2012):** "Verification of Compartmental Epidemiological Models Using Metamorphic Testing, Model Checking and Visual Analytics," BioMedical Computing (BioMedCom), 2012 ASE/IEEE International Conference on , Dec. 2012 pp.68,73, 14-16 doi: 10.1109/BioMedCom.2012.18 IEEE Computer Society Washington, DC, USA©2012 ISBN: 978-0-7695-4938-5

Renfrew, M.P. (1990): "Lopae: geographical dispersal of friendship in Turkana" Working paper no: **472**. Institute for Development Studies. University of Nairobi.

Robbins J. (2012): The Ecology of Disease New York times July 14, 2012 http://www.nytimes.com/2012/07/15/sunday-review/the-ecology-of-disease.html?pagewanted=all&_r=0 retrieved on 7th August 2012

Roeder, P. L., Abraham G. Kenfe G. and **Barrett T. (1994):** Peste des Petits Ruminants in Ethiopian goats. Tropical Animal Health and Production. **26**: 69-73.

Roeder, P.L. and **Obi, T.U. (1999):** Recognizing peste des petites ruminants: A field manual, FAO Animal Health Manual (**5**), 28 pp.

Romero, C.H., Barrett, T., Kitching, R.P., Bostock, C., and **Black, D.N. (1995):** Protection of goats against peste des petits ruminants with a recmbinant capripox viruses expressing thefusion and haemagglutinin protein genes of rinderpest virus. Vaccine **13** : 36-40.

Rossiter P.B., Jessett D.M., and **Taylor W.P. (1985):** Microneutralisation systems for use with different strains of peste des petits ruminants virus and rinderpest virus. Tropical Animal Health and Production 1985;**17(2):**75–81.

Rossiter, P.B and **Taylor, W.P. (1994):** Peste des petits ruminants. In infectious diseases of livestock. pp758. Eds. Coezter, J.A.W. Vol.II.

SAARC (2011): Workshop to develop a regional road map for progressive control of Peste des petits ruminants (PPR) for South Asian countries. 7-9 December 2011, Kathmandu, Nepal. http://www.saarc-rsu-hped.org/content/reports/2011-12-07_Workshop_to_develop_Regional_Roadmap_for_Progressive_Control_of_PPR.pdf Accessed on 20th July 2013.

Saeed K I., Ali **Y. H., Khalafalla A I.** and **Rahman-Mahasin E. A. (2010):** Current situation of Peste des petits ruminants (PPR) in the Sudan. Tropical Animal Health and Production (2010) **42**:89–93

Sakumichi S. (1997): Coping with illness in Turkana: A preliminary report .African Study Monographs, **18**(3,4): 229-240, December 1997. Hirosaki University

Salik J.T., House J. A., Mebus C.A. and **Dubovi E.J.(1994):** Comparison of monoclonal antibody –based sandwich enzyme-linked immunosorbent assay and virus isolation for detection of peste des petits ruminants virus in goat tissues and secretions. Journal of Clinical Microbiology, **32**(5):1349-1353.

Saliki J.T., Libeau G., House J.A., Mebus C.A. and **Dubovi E.J.(1993):** Monoclonal antibody-based blocking enzyme-linked immunosorbent assay for specific detection and titration of peste- des- petits- ruminants virus antibody in caprine and ovine sera. Journal of Clinical Microbiology, **31**(5):1075-1082.

Saravanan, P., Sen, A., Balamurugan, V., Rajak, K. K., Bhanuprakash, V., Palaniswami, K. S., Nachimuthu, K., Thangavelu, A., Dhinakarraj, G. Hegde R. and **Singh R.K. (2010):** Comparative efficacy of peste des petits ruminants (PPR) vaccines. Biologicals **38**, 479–485.

Sarker S. and **Islam. H. Md. (2011):** Prevalence and Risk Factor Assessment of Peste des petits ruminants in Goats in Rajshahi, Bangladesh. Veterinary. World, 2011, Vol.**4**(12):546-549

Savatia, V. (2011): Impacts of climate change on water and pasture resulting in cross-border conflicts: a case study of Turkana and Pokot pastoralists of northwestern Kenya. Journal of Meteorology and Related Sciences, **5**.

Schilling J., Opiyo F. E. O. and **Scheffran J. (2012):** Raiding pastoral livelihoods: motives and effects of violent conflict in north-western Kenya. Pastoralism: Research, Policy and Practice 2012, **2**:25 http://www.pastoralismjournal.com/content/2/1/25

Scott G . R ., **Taylor, W . P.** and **Rossitter, P. R. (1986):** Manual of the diagnosis of Rinderpest. FAO Animal Health Manual-1, 1st edition. Food and Agriculture Organization of the UnitedNations, Rome, Italy, No.**23**, pp.99.

Scott G.R., (1981): Rinderpest and peste des petits ruminants. In Gibbs, E.P.J. (Ed.). Virus Diseases of Food Animals. Vol. II Disease Monographs. Academic Press, New York. PP.401 – 425.

Shaila M. S., Shamaki D., Forsyth M. A., Diallo A., Goatley L., Kitching R. P. and **Barrett T. (1996):** Geographic distribution and epidemiology of peste des petits ruminants virus. Virus Res **43**, 149–153.

Shaila M.S., Purushothaman V., Bhavasar D., Venugopal K. and **Venkatesan R.A. (1989):** Peste des petits ruminants of sheep in India. Vet. Rec. **125**:602.

Sharawi S.S., Yousef M.R., Al-Hofufy A.N. and **Al-Blowi, M.H. (2010):** Isolation, serological and real time PCR diagnosis of Peste Des Petites Ruminants virus in naturally exposed Arabian Gazelle in Saudi Arabia. Veterinary World, (**11**):489-494

Singh B. and **Prasad S.,(2008):** Modelling of Economic losses due to some important diseases in goats in India. Agricultural Economics Research Review Vol **21** July December 2008 pp 297-302

Singh R. P., Sreenivasa B. P., Dhar P., Shah L. C. and Bandyopadhyay S. K. (2004a) Development of a monoclonal antibody based competitive-ELISA for detection and titration of antibodies to peste des petits ruminants (PPR) virus Division of Virology, Indian Veterinary Research Institute, Mukteswar, Nainital 263 138, Uttaranchal,

Singh R.K., Balamurugan V., Bhanuprakash V., Sen A., Saravanan P. and **Yadav M.P. (2009):** Possible control and eradication of peste des petits ruminants from India: technical aspects. Veterinaria. italia., **45**, 449–462.

Singh, R.P., Saravanan P., Sreenivasa B.P., Singh R.K. and **Bandyopadhyay S.K., (2004b):** Prevalence and distribution of *peste des petits ruminants* virus infection in small ruminants in India. Revue scientifique et technique (International Office of Epizootics), **23**: 807-819.

Sinnathamby, G., **Renukaradhya G.J., Rajasekhar M., Nayak R.** and **Shaila M.S. (2001):** Immune responses in goats to recombinant hemagglutinin-neuraminidase glycoprotein of peste des petits ruminants virus: identification of a T cell determinant. Vaccine **19**: 4816-4823.

Sollod A. E. and **Knight J. A. (1982):** Veterinary anthropology: a herd health study in central Niger. Proceedings of the 3rd International Symposium on Veterinary

Epidemiology and Economics, 1982 Available at www.sciquest.org.nz downloaded on 11th October 2012

Soumare B. (2013): Pan African Program for the Control & Eradication of PPR: A Framework to Guide & Support the Control and Eradication of PPR in Africa AU-IBAR and AU-PANVAC Joint Proposal 5th Pan African CVOs Meeting,Abidjan, Cote d'Ivoire, 13-15, 2013. www.au-ibar.org/component/jdownloads/finish/64/1423 Accessed on November 5, 2013

Spector P.A. (1992): Summated Rating Scale Construction: An Introduction. Newbury Park, CA, Sage. VandeWalle, D.

Stem C. (1993): An economic analysis of the prevention of peste des petits ruminants in Nigerien goats* Preventive Veterinary Medicine, 16 (1993) 141-150 141 Elsevier Science Publishers B.V., Amsterdam strategy) *Bullettin of Animal. Health and Production in Afria,**58**-3-2 (2010), 185-193.*

Sumption K . J ., **Aradom G ., Libeau G .** and **Wilsmore A . J . (1998):** Detection of peste des petits ruminants virus antigen in conjunctival smears of goats by indirect immunofluorescence. Veterinary Record, **142**:421-424.

Swai E. S., Kapaga A., Kivaria F., Tinuga D., Joshua G. and **Sanka P. (2009):** Prevalence and distribution of Peste des petits ruminants virus antibodies in various districts of Tanzania. Veterinary Research Communications (2009) **33**:927–936

Swinton J., Harwood J., Grenfell B. T. and **Gilligan, C. A. (1998):** Persistence thresholds for phocine distemper virus infection in harbour seal Phoca vitulina metapopulations. Ecology 67, 54–68.

Tabachnick, B.G. and **Fidell, L.S. (2007):** Using multivariate statistics (5th ed.). Boston: Allyn and Bacon.

Taylor W.P. (1979a): Protection of goats against Peste des Petits Ruminants with attenuated Rinderpest virus. Research in Veterinary Science **27** : 321 – 324.

Taylor W.P. (1979b): Serological studies with the virus of peste des petits ruminants in Nigeria. Research in Veterinary Science 1979 Mar;**26(2):**236-42

Taylor, W. P. (1984): The distribution and epidemiology of PPR. Preventive Veterinary Medicine, **2**: 157-166.

Taylor, W. P., Busaidy S.A. and **Barret T. (1990):** The epidemiology of peste des petits ruminants in the sultanate of Oman. Vet. Microbiology, **22**(4): 341-352.

Taylor, W.P., and **A. Abegunde (1979):** The isolation of peste des petits ruminants virus from Nigerian sheep and goats. Res. Vet. Sci. 26 : 94-96.

Thombare, N.N and **Sinha, M. K.. (2009):** Economic implications of peste des petits (PPR) disease in sheep and goats: a sample analysis of district Pune of Maharastra. Agricultural Economics Research Review 22:319-322

Truong T., Boshra H., Embury-Hyatt C., Nfon C., Gerdts V., Tikoo S. Babiuk L. A., Kara P., Chetty T., Mather A., Wallace D. B. and **Babiuk S. (2014):** Peste des Petits Ruminants Virus Tissue Tropism and Pathogenesis in Sheep and Goats following Experimental Infection. PLoS ONE **9**(1): e87145. doi:10.1371/journal.pone.0087145

van den Ende M, Stem E.C., Sollod A.E., and **Salifou S. (1988):** Peste des petits ruminants and cost effectiveness of its prevention in Niger open access content

International Symposia on Veterinary Epidemiology and Economics proceedings, ISVEE 5: Proceedings of the 5th International Symposium on Veterinary Epidemiology and Economics, Copenhagen, Denmark (published as Acta Veterinaria Scandinavica Supplementum 84), Animal health & production management session, pp 126-128, Jul 1988. http://www.sciquest.org.nz/node/61424 Accesed on 20th July 2013.

Veterinary Record (2012): News and Reports; Transboundary Diseases ;FAO responds to PPR epidemic in the Democratic Republic of the Congo *Veterinary Record* 2012; **171**:7 doi:10.1136/vr.e4525

VIPR (2014): Strain Details for *Peste-des-petits-ruminants* virus Strain ICV89Virology, 2nd edition, Vol. I, Raven Press, New York 1: 1013 – 1044. http://www.viprbrc.org/brc/viprStrainDetails.spg?strainName=ICV89&decorator=paramyxo

VSF B (2007): Pestes des petits ruminants sero monitoring in Turkana district; Final report, October 2007

Wamwayi, H. M., **Rossiter, P. B.; Kariuki, D. P.; Wafula, J. S.; Barrett, T**. and **Anderson, J. (1995):** Peste des petits ruminants antibodies in east Africa. Veterinary Record, **136**: 199-200.

Wang Z., Bao J., Wu X., Liu Y., Li L., Liu C., Suo L., Xie Z., Zhao W., Zhang W., Yang N., Li J., Wang S. and **Wang J. (2009):** Peste des petits ruminants virus in Tibet, China. Emerging Infectious Diseases **15**: 299–301.

Waret-Szkuta A., Roger F., Chavernac D., Yigezu L., Libeau G., Pfeiffer D. U., and **Guitián J. (2008):** Peste des Petits Ruminants (PPR) in Ethiopia: Analysis of a national serological survey. *BMC Veterinary Research*, **4:**34doi:10.1186/1746-6148-4-34

Westover, K. M. and **Hughes, A. L. (2001):** Molecular evolution of viral fusion and matrix protein genes and phylogenetic relationships among the paramyxoviridae. Mol. Phylogenet. Evol. **21**, 128–134. (doi:10.1006/mpev.2001.0999)

Wienpahl, J. (1985). Commision on Noomadic peoples."Turkana herds under environmental stress"

William, E.S. and **Barker, I.K. (2001):** Infectious diseasesof wild mammals. Edition: 3, illustrated Publishedby Wiley-Blackwell, 2001, ISBN 0813825563,9780813825564; 558 page

Wosu, L. O.**(1985):** Agglutination of red blood cells by peste des petite ruminants (PPR)virus. Nigerian Veterinary Journal ,**14**:56-58.

Zahur A. B., Ullah A., Irshad H., Farooq M. S., Hussain M. and **Jahangir M.(2009):** Epidemiological investigations of a peste des petits ruminants (ppr) outbreak in afghan sheep in Pakistan. Pakistan Vet. J., 2009, **29**(4): 174-178.

Zwaagstra, L., Sharif, Z., Wambile, A., de Leeuw, J., Said, M.Y., Johnson, N., Njuki, J., Ericksen, P. and **Herrero, M. (2010):** An assessment of the response to the 2008 -2009 drought in Kenya. A report to the European Union Delegation to the Republic of Kenya. ILRI, (International Livestock Research Institute) Nairobi, Kenya, 108 p.

APPENDICES

Appendix 9.1 *Peste des Petits Ruminants* (PPR) participatory epidemiology (PE) study in Turkana animal health checklist

Check list for the PE livestock keepers focused group interview target 5-15 livestock keepers

Presentation of appraisal team and the objectives of the project (Minimum 2 people; recorder & interviewer)

I Livestock health information

1. General information about livestock keepers:-

- Respondent's personal data

1.1. Bio data (participants name, Average age, Proportion of each gender; Type of group representing the Adakar- boys and Girls, young men, men and women, old folks)

No.	Name	Age group	Gender
	Extend the table as necessary		

1.2. Administrative area and Village/Adakar

District	Division	Location	Sub location	Adakar	Coordinates

2. Disease clinical signs and associated factors as perceived by local community (25 adakars)

2.1. List the diseases of sheep and goats you have observed in last one years

2.2. Historical timeline of diseases of sheep and goats observed in last 30 years

2.3. Rank five most important disease for sheep and goat separately species

- Pair wise comparison for disease characterisation.

2.4. This exercise establishes disease indicators/factors associated to five key diseases by comparing all the diseases.

- Key questions

2.4.1. Which of these two diseases is most important?

2.4.2. Why is that disease more important than the other?

2.4.3. How do you tell the difference between the two diseases?

2.4.4. Where does the disease originate from?

2.4.5. What are the causes of these diseases?

2.4.6. Have you ever seen the disease?

2.4.7. How do the diseases get into your flocks?

2.4.8. When does the disease get in to the flocks?

2.4.9. How does the disease affect the flocks?

2.4.10. What ages are affected by the diseases?

2.4.11. How does the disease affect individual goat/sheep?

2.4.12. What signs are seen on sick sheep or goat?

2.4.13. What signs are seen on goat or sheep dead from the diseases?

2.4.14. What are livelihood activities are associated with causes of these diseases?

2.4.15. What cultural activities are associated with causes of these diseases?

From the above questions develop a list of disease indicators and characteristics of the five diseases

- *Species affected*
- *Ages affected*

- *Clinical signs*
- *Morbidity and mortality*
- *Seasonality*
- *Other factors associated with the diseases (risk factors)*

- Disease Matrix scoring exercises with group discussion.

2.5. Establish that the respondents can associate clinical signs, and disease risk factors indicators to diseases under investigation.

2.5.1. Characterisation of clinical signs with the diseases thus confirmation of PPR disease case definition (stomatitis-pneumo-enteritis syndrome).

Goats	**Clinical sign 1**	**Clinical sign 2**	**Clinical sign 3**	**Clinical sign 4**
PPR				
Disease 1				
Disease 2				
Disease 3				
Disease 4				

sheep	**Clinical sign 1**	**Clinical sign 2**	**Clinical sign 3**	**Clinical sign 4**
PPR				
Disease 1				
Disease 2				
Disease 3				
Disease 4				

2.5.2. To associate risk factors with the diseases. Two matrices separate for sheep and goats.

Goats	**Risk factor 1**	**Risk factor 2**	**Risk factor 3**	**Risk factor 4**
PPR				
Disease 1				
Disease 2				
Disease 3				

Disease 4				

Sheep	**Risk factor 1**	**Risk factor 2**	**Risk factor 3**	**Risk factor 4**
PPR				
Disease 1				
Disease 2				
Disease 3				
Disease 4				

3. **Spatial distribution of the diseases; with focus on PPR and stomatitis-pneumo-entritis syndrome (6 maps 1 per division)**

3.1. PPR disease participatory mapping to show spatial distribution in relation to natural resources and infrastructure.

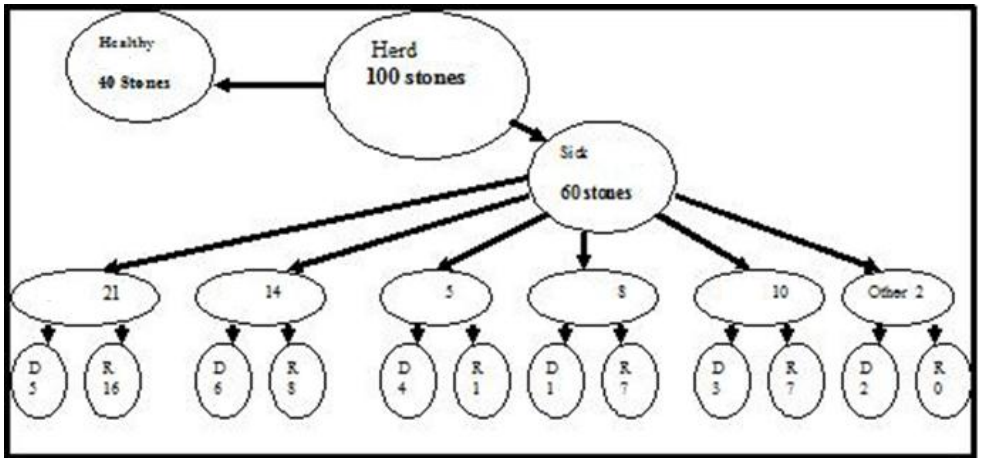
4. **Disease incidence (morbidity and mortality and of sheep and goat diseases) (60 adakars sampled)**

- Proportional piling exercise and group discussion on livestock diseases in the preceding year:-

4.1. Establish age group structure per species -sheep and goats.

Goat and/or sheep				
Age set 1	Age set 2	Age set 3	Age set 4	Age set 5

4.2. For age group establish relative incidence of the key diseases and PPR or stomatitis-pneumo-entritis syndrome included.. Disease categories must have category “other disease” to establish relative incidence



4.3. Establish mortality and fatality rates per each age group

5. Community analysis of PPR control methods (25 adakars sampled)

- Focused group discussion on

5.1. Current PPR control methods

- Key question

5.1.1. What do the community do when the animals get sick with PPR?

5.1.2. What are the PPR treatment and control methods that your Adakar/Village use?

5.1.3. Whom do you get treatment and disease control support services from?

5.1.4. Develop a sustainability indicators of control methods

Access, effectiveness, low cost, easy to use, builds on local knowledge, commitment to contribute Finance and labour, Individual action, group action.

Proportional piling to give a weight of importance to each indicator

5.2. Develop a matrix of PPR control methods against sustainability indicators

The weight for each indicator is allocated in the PPR control methods

For each control method, sum up the ranks to get the most sustainable control method.

	Weight of	Control 1	Control 2	Control 3	Control 4

	Indicator (100)				
Access					
Effectiveness					
Low cost					
Easy to use					
Local Knows					
Commitment labour					
Commitment finance					
Individual acts					
Group acts					
Total scores					

Appendix 9.2: Peste des Petits Ruminants (PPR) participatory epidemiology (PE) study in Turkana Socio-Economics Checklist

Check list for the PE livestock keepers focused group interview target 5-15 livestock keepers

The questionnaire to be administered to 25 Adakars in the six divisions

II. Socio Economics of PPR

1. **Presentation of appraisal team** and the objectives of the project. Reference period will be previous 12 months (2010) unless stated. (Minimum 2 people; recorder & interviewer)
2. **General information about livestock keepers:-**

- Respondent's personal data

- Biodata (name, age, gender)

No.	Name	Age group	Gender
	Extend this table to fit attendance		

- Administrative area and Village/Adakar

District	Division	Location	Sub location	Adakar	Coordinates

3. **Wealth ranking and livelihood analysis at village level**

- Focused Group discussion to :-

- Establish wealth definition criteria

- Key questions

i. What is wealth?

Note down all the definitions of wealth as presented by villagers

ii. What are the indicators of wealth?

List the indicators of wealth

iii. What are the indicators of lack of wealth?

List the indicators of lack of wealth

- Proportional piling exercise and semi structured interview to establish:-

- Wealth categories using wealth indicators.

  - Key question

i. What are the wealth categories in your village/Adakar based on indicators of wealth?

List the wealth categories

Wealth Category	***Wealth category***	***Wealth category***	

- Livelihood analysis for each wealth category

  - Key question

i. What are the livelihood activities for each wealth category?

For each wealth category establish list of livelihood activities and Proportional piling livelihood activity incomes

Wealth category	***Wealth category***	***Wealth category***	***Wealth category***	***Wealth category***	

ii. List of uses of livestock income. Proportional piling of expenditure of livestock incomes

Wealth category	***Wealth category***	***Wealth category***	***Wealth category***	***Wealth category***	

- Sources of food in the household to establish livestock contribution.
    - Key questions

i. What are the main sources of food in the household?

List the main sources of food in the household

ii. What is the contribution of livestock to household food?

Proportion piling of all sources of food in a household

iii. How the livestock based food allocated among the household members?

Matrix scoring of Livestock related food against household members

	Milk	Meat
Children		
Young boy and girls		
Young Men and Women		
Women		
Men		

4. **Livestock keeping and social satisfaction**

- In what ways does procession of animals build ones social standing in the village?
- Does the lack of a livestock in the village affect ones social standing in the village?

i. Explain Yes or No

5. **Herd structure and livelihood contribution**

- Proportional piling exercise to establish herd structures:-

- Type's livestock reared in the village.

- Key question

i. What are the proportions of different livestock species in herds for different wealth groups?

Livestock species	Proportion in the herd			
	Very poor	poor	rich	Very rich
Sheep				
Goat				
Camel				
Cattle				
Poultry				

- Sheep and goat herds dynamics

- Key question

i. How do you build your herds?

List ways herds are accumulated and increase in sizes

ii. How do your sheep and goats leave the herd?

List ways small stock leaves herds making your herd small

iii. What are age structure of sheep and goats?

List of age sets of Sheep and goats

iv. In last one year what proportion of goats per age category left your herd?

	Age set 1	**Age set 2**	**Age set 3**	**Age set 4**
Left herd				
remained				

v. In last one year what proportion of sheep per age category left your herd?

	Age set 1	**Age set 2**	**Age set 3**	**Age set 4**
Left herd				
remained				

vi. What proportions of the ewes were pregnant in a year?

vii. What proportions of the does were pregnant in a year?

viii. How many lambs/kids are borne by an ewe or doe in a year?

ix. What is proportion of twining in goats?

x. What is the proportion of twining in sheep?

xi. What is the reproductive life span of males/females?

xii. Matrix scoring for herd dynamics (Sheep and goat separate)

Goats	**Proportion that left**	**herd change factor 1**	**herd change factor 2**	**herd change factor 3**	**herd change factor 4**	**herd change factor 5**
Age group 1						
Age group 2						
Age group 3						
Age group 4						

sheep	**Proportion that left**	**herd change factor 1**	**herd change factor 2**	**herd change factor 3**	**herd change factor 4**	**herd change factor 5**
Age group 1						
Age group 2						
Age group 3						
Age group 4						

- Proportion importance of each species to the contribution of livelihood for each wealth group

Livestock species	Proportion of importance in livelihood contribution			
	Wealth A	Wealth B	Wealth C	Wealth D
Sheep				
Goat				
Camel				
Cattle				
Poultry				

- Key questions

i. What are the benefits derived from livestock kept by each wealth group?

List the benefits of each species kept

ii. Rank by proportional piling the benefits of sheep and goats.

species	Benefit 1	Benefit 2	Benefit 3	Benefit 4	Benefit 5	Benefit 6	Benefit 7	Benefit 8	Benefit 19
Goats									
Sheep									

6. **Disease impact on the livelihoods per wealth group**

- Matrix scoring and probing. Two matrices separate for sheep and goats.

- Establish disease impact on the benefits for each discussed diseases of the small ruminants

i. What is the effect of disease on benefit (Sheep and goat separate)

Goats	**benefit 1**	**benefit 2**	**benefit 3**	**benefit 4**	**Other benefit**
Weights of the benefits					
PPR					
Disease 1					
Disease 2					
Disease 3					
Disease 4					

sheep	**benefit 1**	**benefit 2**	**benefit 3**	**benefit 4**	**Other benefit**
Weights of the benefits					
PPR					
Disease 1					
Disease 2					
Disease 3					
Disease 4					

Appendix 9.3: Peste des petit ruminants participatory risk assessement -- survey questionnaire

1. Site details

Date…… District:…… Division:……

Location:…….. Adakar Name:…… Adakar size ……

Adakar coordinates…… Sample size 200 sub Adakars

Focus Group members, gender composition, average age.

1 5
2 6
3 7
4 8

2. Herd history of Peste des Petit Ruminant

2.1 Breed:

Small Eastern Africa	Galla

Local sheep	Black head Persian

2.2 When was the last time the herd had PPR cases in the last two years?

	Akicheres	Akiporo	Ait	Akamu
2009				
2010				

An outbreak is a sudden occurrence of the PPR disease within a health flock of small sock

2.3 How many PPR outbreaks were seen in the herd in last 2 years?

	Akicheres	Akiporo	Ait	Akamu
2009				
2010				

2.4 How many PPR outbreaks were seen in the herd in the last 12 months?

	Akicheres	Akiporo	Ait	Akamu
2010				

m

2.5 How many times have the herd been vaccinated in the last 12 months?

	Akicheres	Akiporo	Ait	Akamu
2010				

2.6 What proportion of the Adakars herd was vaccinated in the last 12 months?

2.7 Which age sets of the small stock were vaccinated in the last 12 months?

2.10 How many times have the herd been vaccinated in last 2 years?

	Akicheres	Akiporo	Ait	Akamu
2009				
2010				

2.11 What proportion of the Adakars herd was vaccinated in the last 2 year?

2.12 Which age sets of the small stock were vaccinated in the last 2 years?

3. Variables on PPR exposure

3.01 Which is the kidding period in a year?

Akicheres	Akiporo	Ait	Akamu

3.02 What period are major cultural ceremonies?

Akicheres	Akiporo	Ait	Akamu

3.03 What period of the year are livestock raids common?

Akicheres	Akiporo	Ait	Akamu

3.04 What period of the year different herd graze separately?

Akicheres	Akiporo	Ait	Akamu

3.05 What period of the year do different herds graze together?

Akicheres	Akiporo	Ait	Akamu

3.06 When is transhumance common?

Akicheres	Akiporo	Ait	Akamu

3.07Do you cross into neighboring countries in such of pasture and water?

Akicheres	Akiporo	Ait	Akamu

3.08When do you cross into neighboring countries to trade with livestock?

Akicheres	Akiporo	Ait	Akamu

3.09When do you cross into neighboring countries to restock your herds (raid)?

Akicheres	Akiporo	Ait	Akamu

3.010 When do herds from other countries enter for grazing to your land?

Akicheres	Akiporo	Ait	Akamu

3.011 When do herds from other countries enter you land to share your water sources?

Akicheres	Akiporo	Ait	Akamu

3.012 When are small stock mostly sold?

Akicheres	Akiporo	Ait	Akamu

Pre weaned kids/lambs (less than two months)

Grazing pattern

3.1 Mothers of pre two month kids/lambs are milked

strongly agree	agree	neutral	disagree	strongly disagree.
1	2	3	4	5

3.2 Are kids/lambs housed with older animals?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.3 Are the kids/lambs grazed with older animals?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.4 Kids/lambs graze alongside wild herbivores.

o

strongly agree	agree	neutral	disagree	strongly disagree.
1	2	3	4	5

3.5 Are kids/lambs moved with other animals during transhumance?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.6 How often do kids/lambs stray off for days into other *Adakars* herds?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.7 What is the extent of watering kids/lambs at separate hours from other small stock?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.8 What is the extent of watering kids/lambs at separate water holes?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.9 Kids/lambs share the same watering troughs with older animals?

strongly agree	agree	neutral	disagree	strongly disagree.
1	2	3	4	5

3.10 Are sick dams separated from kids/lambs?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.11 Sick older small stock are watered on same troughs with kids/lambs?

strongly agree	agree	neutral	disagree	strongly disagree.
1	2	3	4	5

3.12 Are sick older small stocks are grazed along with kids/lambs?

Never	seldom	sometimes	often	always
1	2	3	4	5

Pre weaned kids/lambs (more than two months but less than five months)
Restocking pattern

3.13 What extent are kids/lambs bought from markets used to restock herds?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.14 What extent are kids/lambs got through raids introduced into flocks?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.15 How often are kids/lambs received as gifts?

Never	seldom	sometimes	often	always
1	2	3	4	5

Grazing pattern

3.16 Are kids/lambs housed with older animals?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.17 Are the kids/lambs grazed with older animals?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.18 Kids/lambs graze alongside wild herbivores.

strongly agree	agree	neutral	disagree	strongly disagree.
1	2	3	4	5

3.19 Are kids/lambs moved with other animals during transhumance?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.20 How often do kids/lambs stray off for days into other *Adakars* herds?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.21 What is the extent of watering kids/lambs at separate times?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.22 What is the extent of watering kids/lambs at separate water holes?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.23 Kids/lambs share the same watering troughs with older animals?

strongly agree	agree	neutral	disagree	strongly disagree.
1	2	3	4	5

3.24 Are sick dams separated from kids/lambs?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.25 Sick older small stocks are watered on same troughs with kids/lambs?

strongly agree	agree	neutral	disagree	strongly disagree.
1	2	3	4	5

3.26 Are sick older small stocks are grazed along with older animals kids/lambs?

Never	seldom	sometimes	often	always
1	2	3	4	5

Post weaned 6 months up to two years

Restocking pattern

3.27 What extent are young goats/sheep received as gifts in cultural ceremonies?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.28 What is the extent of exchange of young goats/sheep on loans?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.29 What extent are young goats/sheep bought from markets used to restock herds?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.30 What extent are young goats/sheep got through raids used to restock herds?

Never	seldom	sometimes	often	always
1	2	3	4	5

Grazing pattern

3.31 Young goats/sheep are housed with older animals?

strongly agree	agree	neutral	disagree	strongly disagree.
1	2	3	4	5

3.32 Are the young goats/sheep grazed with older animals?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.33 Young goat are grazed in common grazing pasture within *Adakar* locations

strongly agree	agree	neutral	disagree	strongly disagree.
1	2	3	4	5

3.34 Are young goats/sheep moved together with other small stock during transhumance?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.35 How often do young goats/sheep stray off for days to other *Adakars* herds?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.36 How often do young goats/sheep graze alongside wild herbivores?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.37 What is the extent of watering young goats/sheep at separate times?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.38 What is the extent of watering young goats/sheep at separate places?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.39 Young goats/sheep share the same watering troughs with older animals?

strongly agree	agree	neutral	disagree	strongly disagree.
1	2	3	4	5

3.40 Are sick young goats/sheep separated from other animals?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.41 Are sick young goats/sheep watered in communal water holes?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.42 Are sick small stocks grazed along with young goats/sheep/sheep?

Never	seldom	sometimes	often	always
1	2	3	4	5

Off-take pattern

3.43 How often are young goats/sheep/sheep lost through raids returned back?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.44 How often are young goats/sheep/sheep returned home after failed market sale?

Never	seldom	sometimes	often	always
1	2	3	4	5

Adults above two years

Restocking pattern

3.45 What is the extent of introduction of goat gifts from ceremonies into herds?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.46 How often is the exchange of adult goats/sheep/sheep on loan?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.47 What extent are goats/sheep/sheep sourced from markets used to restock herds?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.48 What extent are goats/sheep/sheep got from raids used to restock herds?

Never	seldom	sometimes	often	always
1	2	3	4	5

Grazing pattern

3.49 How often do different herds mix during grazing?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.50 How often do adult goats/sheep/sheep stray off for days into other herds?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.51 How often is transhumance in search of pasture and water?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.52 Transhumance involves crossing international borders?

strongly agree	agree	neutral	disagree	strongly disagree.
1	2	3	4	5

3.53 What extent do herds from neighboring countries graze in your pastures?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.54 What extent do herds from neighboring countries water in your water sources?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.55 What is the extent of herds mixing during watering?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.56 How often do different herds share same watering holes?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.57 Do different herds sharing the same watering troughs?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.58 What extents are sick herds grazed along with health herds?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.59 What extent do sick herds share with health herds same watering source?

Never	seldom	sometimes	often	always
1	2	3	4	5

Off-take pattern

3.60 How often are goats/sheep/sheep lost through raids returned?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.61 How often are goats/sheep/sheep taken to the market returned home?

Never	seldom	sometimes	often	always
1	2	3	4	5

3.62 Traders graze and trek their animals alongside your herds on their way to the markets.

Never	seldom	sometimes	often	always
1	2	3	4	5

Appendix 9.4: Checklist of questions on the PPR control and small stock production in Turkana

Institutions and agencies involved in PPR control

1. **Interview site**

Division_____Location_____Sub Location_____

Adakar_____GPS coordinates_____Adakar size_____

Service provider details

Name: Service offered:

2. **Disease of small ruminants**
    a. The most important diseases affecting goats in your area in last one year?
    b. The most important diseases affecting sheep in your area in last one year?
3. **PPR disease control**
    a. What are the methods that are used to control Peste de Petit ruminants (PPR)?
    b. Which institutions are involved in PPR control services?
    c. What are the different components of PPR disease control?
    d. What do each of the institution involved provide as disease control service?
    e. What are the costs of disease control components?

#	component	organisation	cost

f. When are these control services offered?

4. Small stock trade routes

a. Which are the markets of livestock in the division?

b. Which are transit (Primary & Secondary) markets?

c. Which are terminal markets?

d. Which trade stock routes where livestock are trekked to markets?

e. Which are the traditional seasonal migration routes in search of pasture/water?

f. Which routes are used for trucking livestock to markets?

g. How are these livestock routes and markets affected by PPR disease?

5. Small stock production and products

a. What are the products of from the small stock?

b. What is the average number of Kids per kidding per year?

c. What is the average number of lambs per lambing per year?

d. What is the kidding interval?

e. What is the lambing interval?

f. What is average milk yield per goat per year?

g. What is average milk yield per sheep per year?

6. Market for livestock and livestock products

a. What are the prices of each product in the local market?

i. Meat per Kg

ii. Milk per Litre

iii. Skins per piece

b. What are the prices of small stock for each different age group and sex?

c. What is the seasonal variation (average through put per season) in livestock availability in the market?

d. What is the seasonal variation (average through put per season) in livestock products in the markets? Milk, Meat Skins

e. What is the average live body weight for each age group and sex?

1. Kihu SM, JM Gachohi, CG Gitao, LC Bebora, JM Njenga, GG Wairire, N Maingi and RG Wahome, 2013. Analysis of small ruminants' pastoral management practices as risk factors of Peste des petits ruminants (PPR) spread in Turkana District, Kenya. Res. Opin. Anim. Vet. Sci., 3(9), 303-314.

2. Kihu SM, Gitao CG, Bebora LC, Njenga MJ, Wairire GG, Maingi N, Wahome RG, 2012. Participatory risk assessment of Peste des petit ruminants: Factor analysis of small ruminants pastoral management practices in Turkana district, Kenya. Res. Opin. Anim. Vet. Sci., 2(9), 503-510.