University of Nairobi

MOLECULAR CHARACTERIZATION OF PESTE DES PETITS RUMINANTS IN CAMELS, SHEEP AND GOATS IN NORTHERN KENYA.

A THESIS SUBMITTED TO THE UNIVERSITY OF NAIROBI IN PARTIAL FULFILLMENT OF REQUIREMENTS OF THE MASTER OF SCIENCE DEGREE IN APPLIED MICROBIOLOGY (VIROLOGY OPTION)

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2018

DECLARATION

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

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DEDICATION

Mremboo, Mama Kaititi, Baba Eng'ina, Pebbles

and

the family at large.

We finally made it!

ACKNOWLEDGEMENTS

I wish to acknowledge my supervisors, Prof. George Gitao , Dr. John Gachohi and Prof. Peter Gathumbi for their academic advice, well-informed counsels, and professional direction and for their patience throughout the entire project work. To my colleagues, Dr. Vivian Chemweno; thank you for your support and advice.

My gratitude also goes to the Department of Veterinary Pathology, Microbiology and Parasitology at the University of Nairobi, the International Livestock Research Institute and KAVI institute of clinical research for providing laboratory facilities and technical assistance in handling and storage of samples and processing of samples. Special mention to Dr. Ahadi and Mr. George Warui who were influential in the processing of the samples.

Special appreciation goes to the County Government's veterinary departments, local leaders and the pastoralists in Mandera, Isiolo, Wajir and Marsabit counties for their collaboration and assistance during field work.

Last but not least, my acknowledgement goes to Kenya Camel Association (KCA) and Regional Pastoral Livelihood Resilience Project (RPLRP), for capably financing this project. It is with this backing that I was able to conduct my project efficiently.

Many thanks.

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LIST OF ABBREVIATIONS

ASALs – Arid and Semi-Arid Lands

CCPP- Contagious Caprine Pleuro-Pneumonia

cELISA- Competitive Enzyme-Linked Immunosorbent Assay (ELISA)

CSD- Camel Sudden Death

DNA- Deoxyribonucleic acid

EDTA- Ethylenediaminetetraacetic acid

ELISA- Enzyme linked immuno absorbent assay

FAO- Food Agricultural Organization

IC ELISA- Immunocapture Enzyme linked immuno absorbent assay

KAVI- Kenya Aids vaccine Institute

KCA- Kenya Camel Association

Kes- Kenya Shillings)

OIE- Organizational international Epizootic

PPR- Peste des Petits Ruminants

PPRV- Peste des Petits Ruminants Virus

RNA- Ribonucleic acid.

RPLRP- Regional Pastoral Livelihood Resilience Project

RT-PCR- Reverse Transcriptase Polymerase Chain Reaction

TADs- Trans-boundary Animal Diseases

ABSTRACT

Peste des petits ruminants (PPR) is a novel virus within the genus Morbillivirus and in the family Paramyxoviridae that has been reported in Sub-saharan Africa, the Arabian Peninsula, Middle Eastern countries and India. It is a notifiable trans-boundary disease of economic importance causing an extremely acute disease in sheep and goats which is manifested by fever, mouth lesions, diarrhoea and pneumonia that leads to death of the sick animal. Although a disease of primarily small ruminants, camels have been demonstrated to develop a clinical syndrome. This research was done to determine the presence of PPR disease in camels herded with sheep and goats in Marsabit, Wajir, Isiolo and Mandera counties of Kenya in the wake of an unknown camel disease syndrome that has been afflicting camel herds in the horn of Africa.

As from mid-February 2016 to the end of March 2016; disease field investigations were carried out in the study counties. Three hundred and ninety-two (392) camels from thirty-six (36) herds of camels were examined along the travel routes, watering points and as per the recommendations by the respective county governments. Ninety-three per cent (93%) of the sampled animals were not sick and were not clinically diagnosed as PPR diseased cases. Four (4) mixed herds of goats and sheep herded alongside the camel herds that we tested were also examined; only 13% of those identified presented with clinical PPR symptoms of fever coupled with nasal-ocular discharges and/or with diarrhea. Thirty-eight (38) samples, 15, 12, 8, and 3 samples, were collected from Wajir, Mandera, Isiolo, and Marsabit, respectively. Three key informant interviews with the county departments were held in Wajir, Mandera and Isiolo with the exception of Marsabit due to logistical problems and in addition; one focused group discussion was held in Mandera with the other counties missing on the same due to unavoidable

circumstances. PCR test and sequencing of the PPR genome were conducted; one goat in Wajir and one camel in Mandera were confirmed positive for PPR disease.

According to this study findings, camels present with a fatal sub-acute to acute syndrome of PPR disease loosely known as camel sudden death syndrome and often rarely noticed. Of the camel herds and sheep and goats herds examined only 6% and 12.5% presented with clinical PPR respectively. Only one camel in Mandera and one goat in Wajir were was confirmed to be suffering from the disease using laboratory diagnosis, RT-PCR. The analysis of sequences showed closest nucleotide identities of obtained sequences from both camel and goat to the lineage III of PPRV albeit with 60% of nucleotide identity.

In this research, it was established that camels suffering with PPR within the study area manifest clinical signs mainly manifested by anorexia, loss of body condition and general weakness terminally leading diarrhoea, ocular nasal discharges and conjunctivitis precedes death. These clinical signs were similar to those seen in small ruminants with slight variations of manifestations for example kerato- conjuctivitis and edema of the ventral surface of the abdomen. The clinical signs were also similar to those reported by other studies done to document the clinical signs of PPR in camels.

In conclusion, the study detected PPRV in both camel herds and mixed sheep and goat herds. This indicates that camels could be playing a role in the epidemiology of PPR disease in the region as well as that PPR disease virus could be responsible in the epidemics of Camel sudden death syndrome. There is therefore a need for resources to be dedicated in understanding the role camels play in the epidemiology of PPR disease and in understanding the role PPR diseases play in Camels Sudden death syndrome.

CHAPTER ONE

1. INTRODUCTION

Over two thirds of Kenyan lands mass constitute arid and semi-arid lands (Behnke and Muthami 2011). They are home to about 4 million pastoralists constituting more than 10% of Kenya‘s inhabitants including other rangeland users (Kirbride and Grahn., 2008). Arid and Semi- arid lands of Kenya are characterized by persistent drought which results in lack of pastures and water (Njoka *et al.,* 2016). Due to these climatic conditions, these areas mainly practice livestock keeping as opposed to crop farming as a main source of livelihood in pastoral set up consisting of mainly sheep, goats, cattle and camels (Makau *et al.,* 2005). The livestock sector is a big contributor to the Kenyan economy; the input was approximated to be Kes (Kenya Shillings) 356.217 billion in 2009 (Behnke and Muthami., 2011). From this amount, Kes 60billion is estimated to come from pastoral livestock keeping (Makau *et al.,* 2005). Of the species kept in these lands, camels have proven to be resilient; this species is able to survive on shrubs and go for days without water (Megersa *et al.,* 2008; Watson *et al.,* 2016). Camels are able produce milk even in periods of prolonged drought when other species have ceased to produce (Kuria., 2004). This makes camels important livelihood assets to the pastoral communities in Kenya where high poverty intensities persist (Megersa *et al.,* 2008; Kirbride and Grahn., 2008; Watson *et al.,* 2016)

Even though camels have been previously viewed as resilient animals and rarely predisposed to most of the diseases that affect other domestic animal species that are distributed in the same ecological zones, recent research developments have discovered that camels are vulnerable to a

large number of infectious agents whereby some are still unknown (Mohammed *et al.,* 2003; Abbas and Omer., 2005; Al-Ruwaili *et al.,* 2012). Of great concern is *Peste des petits ruminants* (PPR); a syndrome commonly affecting small domestic ruminants in sub-Saharan Africa and the Arabian peninsula has been reported to cause clinical disease in Camels. The disease has been reported in camels in outbreaks in Ethiopia (Roger *et al.,* 2001; Mergesa *et al.*,2012); Sudan (Khalafalla *et al.,* 2010; Kwiatek *et al.*,2011) and from Camels in Iran(Zakian *et al.,* 2016). PPR in camels is characterised by a respiratory syndrome with an exceedingly high morbidity rate with an irregular mortality rate that is independent on antibiotic treatment (Roger *et al.,* 2001; Kwiatek *et al.,* 2011). Often camels are reported to die suddenly of a per-acute syndrome (Zakian *et al.,* 2016), with other cases progressing into a syndrome presenting with fever, oral erosion, diarrhoea, respiratory distress, lymphadenopathy, dermatitis, and ulcerative keratoconjunctivitis (Khalafalla *et al.,* 2010; Zakian *et al.,* 2016). A syndrome, camel sudden death, with PPR like characteristics has been afflicting camel populations in Ethiopia, with information of its incidences in Djibouti, Somalia, Sudan, Eritrea and in Kenya since 1995/1996 (Roger *et al.,* 2001; Gluecks *et al.,* 2010; Gluecks and Younan 2010).

1.1 PROBLEM STATEMENT

To date, Camel Sudden Death Syndrome (CSD), continues to cause major production losses. In 2004 it resurged in Ethiopia and spread through the horn of Africa to include Somalia and Kenya causing sudden deaths of seemingly healthy animals (Gluecks *et al.,* 2010). The disease targets camels at reproductive ages including breeding bulls, dry and lactating females. Camel Sudden Death Syndrome CSD is a Trans-boundary animal disease (TAD) (Gluecks and Younan 2010), TADs create a grave danger to the international animal trade and endanger food Security (Domenech *et al.* 2006). Studies that have already been done have not been able to identify the causative agent and it has been presumed that a viral agent is involved (Gluecks *et al.,* 2010). In this perspective, a sudden death syndrome of apparently health animals was identified in Sudan as *Peste des Petis Ruminats* (PPR) in 2010 (Khalaffa *et al.,* 2010) and in Iran (Zakian *et al.,* 2016). Although PPR has been recorded as a clinical disease in camels in Sudan, Ethiopia and Iran, it has not been recorded in Kenya

Therefore, this study assessed the role of (*Peste des Petits Ruminants*) PPR disease in etiology of Camel Sudden death Syndrome through a field survey to characterize the clinical presentation of the disease and to collect diagnostic samples for PPR from sick camels. The overall aim of the project was to support the control strategies for the disease. The study focused on four administrative counties of Kenya; Mandera, Wajir, Marsabit and Isiolo since the disease has been reported in these counties that also host large herds of camels.

1.2 OBJECTIVES

1.2.1. Overall objective

To investigate Peste Des Petis Ruminants in Camels, sheep and goats from Wajir, Mandera, Marsabit and Isiolo counties in Kenya

Specific objective

1. To characterize the clinical presentation of PPR in Camels, sheep and goats in the study counties with regards to Camel Sudden Death Syndrome.
2. To detect PPR in camels in the study counties.
3. To characterize the virus through molecular techniques.

1.3 HYPOTHESES

1. PPR virus is circulating in Kenyan Camel herds.
2. Camels are involved in the transmission of *Peste Des Petis Ruminats* among sheep and goats herds.

1.4 JUSTIFICATION

Camels are important livelihood assets in Arid and Semi-arid lands of Northern Kenya where an estimated 1.06 million camels support livelihoods of poor communities in this region (Megersa *et al.,* 2008; Musinga *et al.,* 2008). They are able to prosper in the severest of Kenya's agro-ecological zones and are in general able to tolerate the frequent droughts which devastate other livestock populations while still continue to yield decent amounts of milk (Musinga *et al.,* 2008; Megersa *et al.,* 2008; Watson *et al.,* 2016). This places camels as conceivably as one of the most apt avenue through which livelihoods of communities living in these arid lands can be protected (Musinga *et al.,* 2008; Downie., 2011). Despite the massive importance of this species, they appear to be neglected by the research community. Few studies have been done to improve on their status in Kenya as diseases and climate change keep threatening their existence (Blackwell,. 2010). Among the diseases, CSD has a high impact on livelihoods of camel keepers as it causes death of camels within the reproductive brackets (Khalaffa *et al.,* 2010; Gluecks *et al.,* 2010; Zakian *et al.,* 2016).

The etiological agent of CSD is currently unknown; however, there is a strong indication that a viral agent could be involved based on previous studies (Gluecks *et al.,* 2010). The fact that the waves of this disease occur under temporal and spatial variations with propensity of disease spreading from one area to another suggests an extremely transmissible infectious disease, most undoubtedly a viral infection (Megersa *et al.,* 2012). With regards to the epidemiology of infectious diseases and history of transboundary diseases, many of the exceedingly contagious TADs identified to date have viral etiologies (Ote *et al.,* 2004).

In the year 2004, a syndrome with the same clinical presentation (death of apparently camels within reproductive ages) was identified as PPR in Sudan (Khalaffa *et al.,* 2010). During the mid1990s, a disease with sudden death of camels in camel-producing areas of Ethiopia occurred (Roger *et al.,* 2000). The clinical symptom and signs were; sero-mucopurulent nasal discharges, productive coughing, lacrimation and dyspnea (Roger *et al.,* 2000). These symptoms were coupled with abdominal breathing. PPR was detected using molecular techniques (Roger *et al.,* 2000). The current study therefore sought to determine whether such a clinical disease can be found in the main camel rearing countries in Kenya, the causative agent of such disease and whether it is found in other species reared together with camels.

CHAPTER TWO: LITERATURE REVIEW

2.1 CAMEL SUDDEN DEATH SYNDROME

2.1.1. Introduction

Camel Sudden Death Syndrome (CSD) is a trans-boundary camel disease of an unknown aetiology (Wernery *et al.* 2006; Dawo, 2010; Glueck *et al.,* 2010). It is an acute disease that affects camels with seemingly good body condition and often results in death of affected animals (Dawo, 2010; Gluecks *et al.,* 2010). Usually no clinical signs are observed with mortalities reported manly in adult female and male camels; the reproductive age groups (Dawo, 2010). The case fatality is high in pregnant camels trailed by breeding bulls, and lactating animals, inferring grave socioeconomic costs (Dawo, 2010; Glueck *et al.,* 2010). The disease has been reported for the first time in Ethiopia (2005), Somalia (2006) and Kenya (2007) (Gluecks *et al.* 2010).

The disease is a total new entity in the horn of Africa with the pastoralist communities lacking an indigenous name for the disease (Dawo, 2010). The disease is subsequently often referred to names that relate to its quick onset and the instant nature of death that results; the Somali ethnic group living in both Kenya and Somalia refer to the disease as '*Ha igu soo dhicin' (*don't fall on me); '*Babta'* (collapsed immediately) while the Borana ethnic group refers to the disease as '*Habaad* ' (Bullet); '*Maal oo dhaaf'* (Milk and abandon) (Dawo, 2010; Gluecks *et al.,* 2010).

2.1.2. Clinical signs

In most instances, there is no prior clinical course, but some cases show clinical signs before death (Gluecks *et al.,* 2010; Megersa *et al.,* 2012). These signs include; collapse, dyspnoea and rapid death within 1 hour after collapse, sometimes general initial symptoms that are observed in a span of less than 6hours (Dawo., 2010; Gluecks *et al.,* 2010). The animals generally have fatigue; Affected pregnant camels udders increase in size as if the animal is approaching parturition, and lactating females give a higher than usual milk yield before they die (Dawo., 2010; Gluecks *et al.,* 2010). In some instances affected camels manifest neurological signs and would vocalise a few minutes before death with dead camels extending their necks (Dawo., 2010; Gluecks *et al.,* 2010).

2.1.3. Pathological findings

When post mortem was done findings were; rupture of the pericardial sacs and in some instances the heart as well, blackening of the lungs, the small and large intestines would be inflamed with frothy gas in some parts of the large intestine (Dawo., 2010). Other findings found in post mortem included yellowish coloration of the intestines, liver and kidneys, the blood lacked clotting factors and there was no rigor mortis (Dawo., 2010). These signs were found consistent with Anthrax (Dawo., 2010) however vaccination and treatment with broad-spectrum antibiotics did not seem to reduce the mortalities. Post-mortems in Somalia and Kenya revealed; oedema and massive froth /foam in the lungs, severe haemorrhages on the tracheal mucosa, Petechial haemorrhages on the myocardium, shrunk or active spleen, diffuse haemorrhages in the intestines and delayed blood clotting (Gluecks and Yonan., 2010).

2.1.4. Laboratory findings

Laboratory tests done at Central Veterinary Laboratory, Kabete in Kenya and at the Department of Pathology in Hanover, Germany, for several aetiological including parasites, viruses and bacteria and post mortem findings did not confirm any commonly known animal disease agents (Gluecks and Younan 2010). Test done included tests on some human heart diseases but they were inconclusive (Gluecks and Younan 2010). .

The actual cause of death was determined to be left sided congestive heart failure (Gluecks and Younan 2010). Positive cytopathic effect was seen on three samples that were cultured on Vero cells at central veterinary laboratory, Kabete in Kenya (Gluecks and Younan 2010). This led to a conclusion that the disease has a viral in origin (Gluecks and Younan 2010).

2.1.5. The spread and dynamics of camel sudden death syndrome

As other diseases in pastoral zones; the disease spread dynamics in the horn of Africa appear to be linked with the pastoralists' nomadic way of life (Downie *et al.,* 2011; Megersa *et al.,* 2012). There is also a strong correlation to the trade routes used in animal trade (Megersa *et al.,* 2012). From the map (*Fig 1*) the syndrome was first reported in Afar regions of Ethiopia (November 2004), it then circulated to involve several other regions of the country (Bekele., 2006; Margesa *et al.,* 2012). Thereafter reports occurred in Somalia in 2006 with successive transmission to Kenya in 2007 (Gluecks and Younan 2010).

Figure 1 A distribution map of Camel sudden death syndrome

2.2. PESTE DES PETIS RUMINANTS DISEASE AND CAMEL SUDDEN DEATH SYNDROME

2.2.1. Pestes des Petis Ruminants

This is a notifiable disease that is highly contagious primarily affecting sheep and goats but has been detected in other domestic and wild ruminants (Abraham *et al.,* 2005; Abubakar *et al*., 2011; Munir., 2015). The disease is normally characterized by fever, mouth lesions, diarrhea and pneumonia which often result in death of the sick animals (Abraham *et al.,* 2005; Abubakar *et al*., 2011). The disease is caused by *Pestes des Petits Ruminants* virus from the genus Morbillivirus in the family Paramyxoviridae; order Mononegavirales (Parida *et al.,* 2015; Munir., 2015).

Pestes des Petits Ruminants (PPR) was first detected and documented in Ivory Coast of West Africa in the year 1942; nevertheless, it has extended its range to include other nations (Shaila *et al.,* 1996; Munir., 2015; Baron *et al.,* 2017). The disease commonly affects animals in the Arabian Peninsula, the Sub-saharan Africa, India and Middle Eastern countries (Kinne *et al*., 2010). The etiological agent is genetically categorized into four lineages based on the partial sequence of the fusion gene (Munir., 2015). Lineage I, II and lineage III is endemic in Africa whereas lineage IV is mainly present in Asia (Shaila *et al.,* 1996; Dhar *et al.,* 2002; Bailey *et al*., 2005) though recently lineage IV was isolated in Africa; Morocco and Sudan (FAO, 2009; Baazizi *et al.,* 2017). Despite the fact that no reported cases of the disease in Europe, a risk of introduction is eminent as recent outbreaks have been reported in the northern border of North Africa ((Banyard *et al.,* 2010; Baazizi *et al.,* 2017). The disease has also been expanding its territories with new regions reporting detection of the disease (*See fig 2 and 3*).

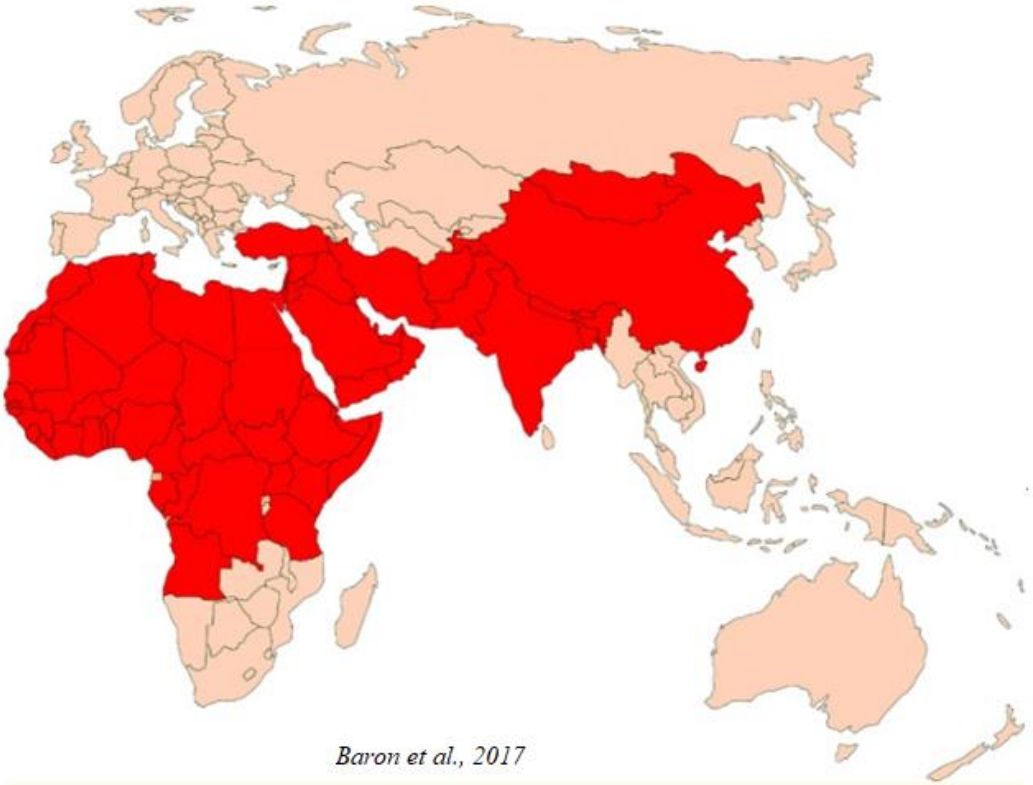

Figure 2 Global distribution of PPR as per OIE reports

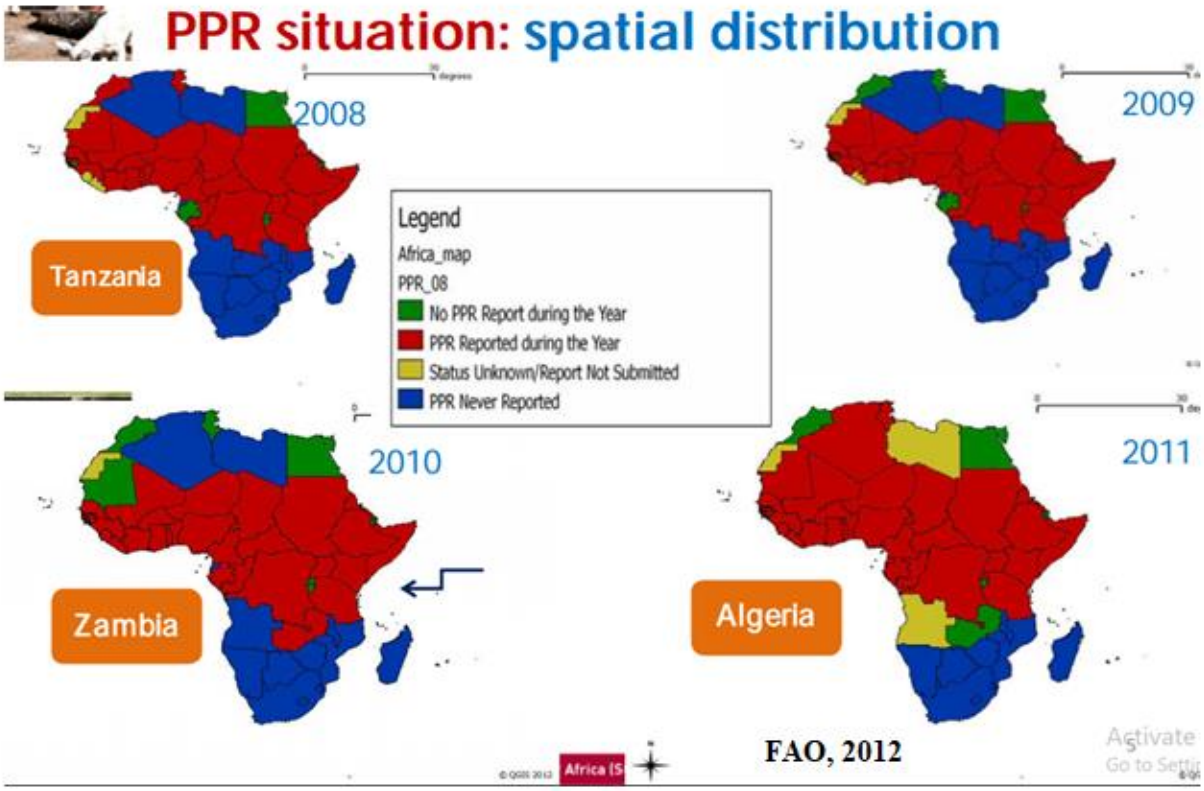

Figure 3 PPR situation in Africa with respect to recent outbreaks.

2.2.2. Clinical presentation

The disease may present in a per-acute, sub-acute or acute forms (Munir., 2015). Per-acute cases mainly occur in populations that have never been exposed to the disease (Dundon *et al.,* 2011; Torsson *et al.,* 2016). In sub-acute and acute cases, the first signs observed are usually high fever and depression preceding death (Albina *et al.,* 2013; Abubakar *et al*., 2011). Discharges from the nostrils and the eyes appear soon afterwards; these discharges are often serous in nature but eventually become mucopurulent due to opportunistic secondary bacterial infection (Munir., 2013; Munir., 2015). Affected animals appear to matt around the eyes and the nostrils as the nostrils become obstructed (Banyard *et al.,* 2010; Munir *et al.,* 2012).

Lesions appear on the lips and gums, a couple of days after the clinical signs are observed (Munir., 2013; Munir., 2015). The lesions are initially hyperaemic but eventually become necrotic (Munir., 2013). The lesions are often covered in a white cheesy material and may be widespread to cover the whole buccal cavity often giving the animal a bad breath (Roeder *et al.,* 1999). Some animals may develop diarrhea in the last stages. The material is fetid and may contain shreds of tissue. This is often coupled with respiratory distress (Munir., 2013).

In almost all terminal infections, death is initiated by severe dehydration and starvation caused by acute diarrhoea or by viral bronchopneumonia (Banyard *et al.,* 2010). Disease in pregnant animals, although rare, has been associated to abortion (Munir., 2015). The significance of the virus causing abortion and the process on how it occurs are at present not known (Abubakar *et al.,* 2008) although multiple infections with both PPR virus and other viruses in instances of abortions have been detected and described (Kul *et al.,* 2008).

The severity of PPR disease seems to differ depending on the strain involved, even though PPR has just one serotype (Couacy-Hymann *et al.,* 2005; Banyard *et al.,* 2010; Munir., 2015). The disease presentation and symptoms are frequently tangled with, and worsened by secondary contagions leading to PPR disease being classified as a complicated disease to describe, detect and treat (Kul *et al.,* 2008; Couacy-Hymann *et al.,* 2005; Munir., 2015). To further complicate its diagnosis the disease presents like many other common diseases including but not limited to contagious ecthyma, coccidosis, bluetongue , pasteurellosis, contagious caprine pleuropneumonia, mineral poisoning, heartwater, and foot-and mouth disease that are also distributed in the regions where the disease is has been detected (Banyard *et al.,* 2010; Fentahun and Woldie, 2012; Munir; 2015). Virulence also seems to differ amongst different species and even within species with sheep appearing more susceptible as compared to goats (Yesilbag *et al.,* 2005) and with different individuals of goats within the same breed experimentally showing different susceptibility and virulence (Couacy-Hymann *et al.,* 2017).

Several domestic and wild species including cattle (Lembo *et al.,*2013), buffaloes wildebeest, antelopes and gazelles (Elzein *et al.,* 2004; Abubakar *et al.,* 2011; Bao *et al.,* 2011; Munir.,2014; Mahapatra *et al.,* 2015; Orynbayev *et al.,* 2016) and recently even dogs (Ratta *et al.,* 2016) have been demonstrated to sero- convert PPR disease virus apart from sheep and goats. A number of species have been documented to get infected by PPR disease yet they remain asymptomatic (Bidjeh *et al.,* 1995; Banyard *et al.,* 2010) even though it is not clear on the modalities used by the virus as it mingles with the population in the absense of clinical disease (Banyard *et al.,* 2010). It has been suggested that PPRV may well circulate without clinical detection, intermittently causing periodic epidemics when the host population immunity levels drop (Yesilbag *et al.,* 2005; Banyard *et al.,* 2010). This has been generally accepted as how PPR

epidemics have given way to its endemicity in Asia and Africa (Banyard *et al.,* 2010). PPR is endemic in most parts of Africa with West, East and Central Africa being the endemic foci (FAO 2012). Of focus, the disease dynamics presentation has changed from acute disease with high morbidities and mortalities to a mild disease in nature with moderate morbidity and mortality as the case reported in Morocco in 2008 (Banyard *et al.,* 2010). The spread of PPRV is affected by both the density of susceptible hosts, the birth rate within the herds; nevertheless animals that survive the infection develop lifelong immunity (Couacy-Hymann *et al.,* 2007; Munir; 2015).

2.2.2.1. Disease in camels

The clinical disease of PPR in camels appears to be variable possibly attributed to the lineage of the virus involved (Munir., 2015). When lineage II and III are involved; PPR in camels is characterized by a respiratory syndrome with abdominal breathing, lacrimation and fever finally leading to depression and recumbency with an exceedingly high morbidity rate with an irregular mortality rate that is dependent on antibiotic treatment (Roger *et al.,* 2001; Khalafalla *et al.,* 2010; Kwiatek *et al.,* 2011). When lineage IV is involved camels die suddenly of a per-acute syndrome (Zakian *et al.,* 2016), with other cases progressing into a syndrome presenting with fever, oral erosion, yellow diarrhea, respiratory distress, lymphadenopathy, dermatitis, and ulcerative keratoconjunctivitis (Khalafalla *et al.,* 2010; Zakian *et al.,* 2016). Pregnant camels often abort when affected with the disease (Khalafalla *et al.,* 2010); in addition various mild signs of subcutaneous oedema, submandibular distension, occasional coughing, decreased milk production progressing to emaciation and dehydration persisted up to14 days (Khalafalla *et al.,* 2010). All age groups are affected however there is preference to pregnant and lactating animals (Khalafalla *et al.,* 2010).

Camels have elicited a mild disease with experimental infection of camels with PPRV in Dubai and Saudi Arabia (Munir., 2015). Mild subclinical infection with a respiratory phase was elicited characterized with fever, nasal discharge and coughing (Munir., 2015). Other camels presented with a transient disease alongside goats but not sheep during this experiment (El-Hakim., 2006). Infection of a male camel with lineage IV did not elicit sero-conversion neither were clinical signs reported (Wohlsein and Singh., 2015).

2.2.3. Diagnosis of Peste des Petis Ruminants

Tentative diagnosis of PPR disease can be achieved through observation of clinical and pathological presentation of the disease in consideration to its epidemiology (Roeder *et al.,* 1999). This is very challenging as the disease is often complicated by secondary bacterial infections, co-infections with other viruses and for the fact that the disease presents like many other diseases that occur in the same localities further complicating the diagnosis (Kul *et al.,* 2008; Banyard *et al.,* 2010). Confirmation of the diseases is achieved by serology and molecular techniques which detect and identify PPRV antibodies and virus genetic material respectively (Balamurugan *et al.,* 2014).

The methods for detection, of PPRV antibodies differ extensively subject to local infrastructures available and systems adopted for veterinary service delivery (OIE 2008; Abera *et al.,* 2014). Competitive enzyme linked immunosorbent assay (cELISA) is the serological test recommended by OIE Reference Laboratories for PPR to test for PPR (OIE 2008). It is fast, highly accurate and equally sensitive and of importance, it can differentiate between PPR and Rinderpest a disease that is closely related to it (OIE 2008). However, several alternatives exist including immunofiltration, indirect ELISA, haemagglutination tests, novel sandwich ELISA and latex fixation test (Banyard *et al.,* 2010).

Antigen detection can be executed using a selection of diagnostic tests including counter immunoelectrophoresis, immunocapture (IC) ELISA or agar gel immunodiffusion (Banyard *et al.,* 2010). Of the three agar gel immunodiffusion test cannot differentiate between PPR and Rinderpest viruses (Banyard *et al.*, 2010); it is also comparatively less sensitive, hence successful detection of milder forms of PPR disease or samples with a small viral load may be difficult (Banyard *et al.,* 2010).

Identification of virus genetic material is done by the real time reverse transcriptase polymerase chain reaction (real time RT PCR) (OIE 2008) which is a molecular technique Abera *et al.*, 2014). The test is based on amplification of parts of either the nucleoprotein or fusion protein genes by use of primers and comparing it with a known positive sample of PPR (Deepak *et al.,* 2007). In spite of its high cost, it is now one of the technique that is highly used for PPR studies because of it is as its ability to detect all the four circulating strains of PPR (Maganga *et al.*, 2013). This test supersedes standard reverse transcriptase polymerase chain reaction (Deepak *et al.,* 2007; Couacy-Hymann *et al.,* 2002); however, the production of a standard reverse transcriptase polymerase chain reaction product is required in order to execute sequence analysis and successive phylogenetic description of new viral isolates (Banyard *et al.,* 2010).

Cell culture viral isolation can be achieved by a number of diverse cell lines; nevertheless virus recovery is often unsuccessful (Mahapatra *et al.,* 2006; Adombi *et al.,* 2011). Formerly, a marmoset-derived cell line was principally used in cell cultures (Mahapatra *et al.,* 2006; Adombi *et al.,* 2011); Vero cell cultures and primary lamb kidney have proven to achieve good results (Mahapatra *et al.,* 2006; Adombi *et al.,* 2011). In general, cultures are studied for cytopathic effect few days after suspicious material inoculation into the chosen cell line; virus identity is

thereafter using diagnostic techniques (Mahapatra *et al.*, 2006; Singh *et al.,* 2009; Adombi *et al.*, 2011).

2.2.4. Peste des petis Ruminants and its linkage to Camel Sudden Death Syndrome

Although antibodies had been found in sheep and goats a decade before; the disease was first reported in Kenya in the year 2007 (Luka *et al.*, 2012). This is the same time when the first incidence of camel sudden death syndrome had struck the country (Dawo, 2010). PPR disease has been reported to occur in camels before causing a serious disease (Wohlsein and Singh, 2015). In 2004, a syndrome with the same clinical presentation of Camel Sudden Death Syndrome (death of apparently camels within reproductive ages) was identified as *Peste des Petis Ruminats* in Sudan (Khalaffa *et al.,* 2010). In addition; disease in pregnant animals, although rare, has been associated to abortion (Munir., 2015). The significance of the virus causing abortion and the intricate on how it occurs are at present not known (Abubakar *et al.*, 2008) although multiple infections with both PPR virus and other viruses in instances of abortion has been detected and described (Kul *et al.,* 2008).

CHAPTER THREE: METHODOLOGY

3.1. STUDY AREA

The study was done in Mandera, Wajir, Marsabit and Isiolo Counties. Extensive pastoral livestock production is practiced in these counties with sheep, goats and camels being the species that are widely kept.

Marsabit, Mandera, Isiolo and Wajir Counties are largely classified on Agro ecological zone VI. This ecological zone is considered as a semi arid constituting the driest parts of the country with unreliable annual precipitation of 200-400 mm. The vegetation is characterised by Acacia among other shrubs with dispersed taller trees in addition to perennial and annual grasses (Kenya Soil Survey., 2008). A bigger proportion of the country's livestock population (camels, cattle, donkeys, sheep and goats) are kept in these areas (Kenya Soil Survey., 2008). These areas, which are also categorised as rangelands, are not suitable for rain dependent plant growing because of physical constraints such as aridity and poor vegetation subsequently extensive livestock grazing using vastly adapted livestock remains as the main livelihood alternative (Otolo and Wakhungu ., 2013).

The main communities in region are the Somali people and the Borana ethnic groups who constitute over 70% of the population of pastoralist. Other communities include the Rendile and the Gabbra. Camels are the main animals kept because of their adaptation to these arid lands and are kept alongside cattle sheep and goats (Watson *et al.,* 2016)

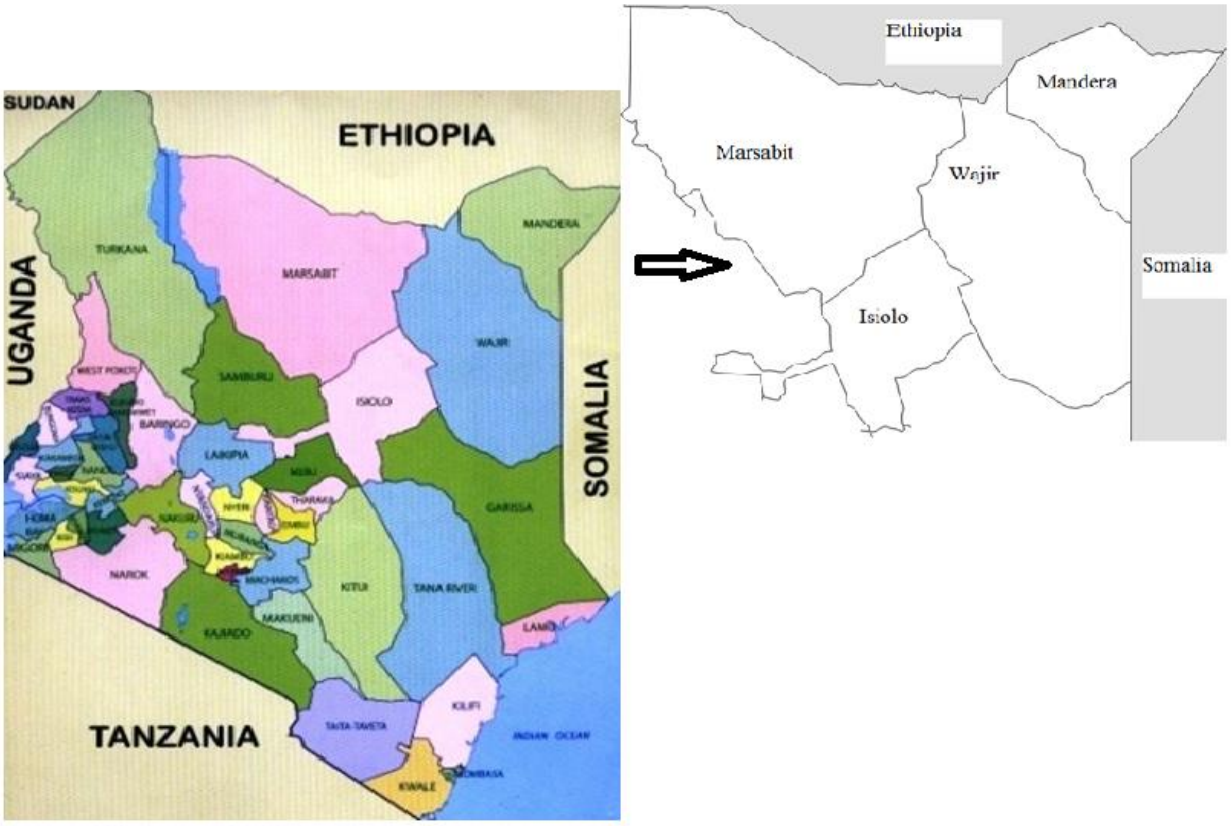

Figure 4: Study area

3.2. STUDY DESIGN AND SAMPLE COLLECTION

This was a cross sectional study in Isiolo, Marsabit, Mandera, and Wajir done in. February 2016 to March 2016. The study involved;

1. Administration of key informant interviews to senior veterinary officers in the study counties,
2. Disease field investigation
3. Focused group discussions
4. Collection of biological samples for disease diagnosis

The discussions and interviews were centred on understanding the

1. Current status of CSD in the county.
2. Clinical and pathological signs of the disease
3. Age group affected
4. Tentative diagnosis of CSD and
5. If there are sick sheep and goats during outbreaks of CSD

Three county directors of veterinary services (Mandera, Wajir and Isiolo) were interviewed after which disease field investigations were carried. No interview was conducted in Marsabit County due to logistical challenges. In addition to the above, one focused group discussion was conducted in Mandera County with other counties missing out on this due to unavoidable technicalities.

A total of 36 camel herds along transport routes were conveniently identified from which 392 camels were examined in the study counties. Additionally, 80 sheep and goats from 4 herds which were closely herded with camel herds that were examined (see *Appendix 2-7*).

Figure 5: A female camel being sampled at Banisa Mandera

The sampled animals had a fever and one or more of the following clinical signs; diarrhoea, ocular and nasal discharge. A total of 38 biological samples (See Table 1); 25 from camels and 11 goats and sheep were collected between mid-February 2016 and the end of March 2016 from the study areas. Of these samples, 15, 12, 8 and 3 samples were collected from Wajir, Mandera, Isiolo and Marsabit respectively. The samples were nasal and ocular discharges on viral transport media and EDTA blood. Samples were transported in a cool box with icepack and were stored in local laboratories in the field before transporting them to Kenya Aids Vaccine Institute (KAVI); Institute of Clinical Research molecular laboratory for further storage at negative 20°C awaiting laboratory analysis.

Table 1 Sample collected after field investigation

Samples	Mandera	Wajir	Isiolo	Marsabit	TOTALS
Camel blood	7	6	8	0	21
Camel nasal discharges	1	0	0	3	4
Camel ocular discharges	1	2	0	0	3
Sheep and goats blood	1	3	0	0	4
Sheep and goats nasal discharge	1	2	0	0	3
Sheep and goats ocular discharge	1	2	0	0	3
TOTALS	**12**	**15**	**8**	**3**	**38**

3.3. LABORATORY ANALYSIS

3.3.1. RNA extraction

RNA Extraction was done using QIAamp DSP Virus Kit ® (QIAGEN GmbH, 40724 Hilden, GERMANY) at KAVI; Institute of Clinical Research.The QIAamp process is appropriate for use with fresh or frozen whole blood and blood which has been preserved with EDTA. Twenty-five (25) µl of proteinase K was pipetted into the bottom of the lysis tube after which 200 µl of the sample was added. This mixture was incubated for at 65°c for 1hr for lysis of the cells to occur after which the mixture was briefly centrifuged to recollect the drops that had collected on the caps back into the lysis tube. Two hundred (200) µl of Buffer AL (containing 6.2 µg/ml carrier RNA) was then added into the lysis tube and blended by pulse-vortexing for ≥15seconds to produce a consistent solution. The resultant solution was then incubated at 56°C for 10 minutes then brief centrifuging was repeated to recollect the drops on the inside of the lid back into the lysis tube. Two hundred (200) µl of absolute ethanol was added to the sample in the lysis tube to precipitate the nucleic material in the sample and blended by pulse vortexing for ≥15 seconds this was tailed by brief centrifuging again.

The above mixture was cautiously transferred to the QIAamp Mini column with a 2 ml collection tube, the cap was tightened and the mixture centrifuged at 6000 x g for 1 minute. A clean 2 ml wash tube was attached to the QIAamp Mini column after the tube holding the filtrate was thrown away. Five hundred (500) µl Buffer AW1 was then added into the QIAamp Mini spin column. Care was taken to ensure the rim was not wetted. Centrifuging at 6000 x g was repeated at 1 minute after which the filtrate was thrown away and a new collection tube fitted to the Mini column.

Five hundred (500) µl of Buffer AW2 was added into the Mini column and centrifuging was done at 20,000 x g for 3 minutes. This step was repeated twice to ensure that ethanol is removed completely. The QIAamp Mini spin column was then placed in an elution tube and the collection tube holding the filtrate was thrown away. Two hundred (200) µl of elution Buffer was added into the Mini column and the mixture was incubate at room temperature (15–25°C) for 1 minute, and then centrifuge at 6000 x g for 1 minute. The resultant was the extracted RNA template from the sample. The quality and quantity of RNA after extraction was measured (see table in appendix 10).

3.3.2. Detection of PPRV by RT-PCR

Reverse transcription polymerase chain reaction was carried out in GeneAmp PCR System 9700 (Applied Biosystems, Carlsbad, USA). The master mix as shown in Table 2, was prepared using the AgPath-ID one-step RT-PCR kit (Applied Biosystems, Courtaboeuf, France) with PPRV specific primers from Eurogentec (Liège, Belgium) (Table 3). All reactions were run with Nigeria 75/1 vaccine strain as a positive control and nuclease-free water as the negative control.

Table 2. PCR reaction master mix components

No.	Component	Volume (µl)
1.	2X RT-PCR Buffer	12.5
2.	10µM Forward primer	1.0
3.	10µM Reverse primer	1.0
4.	Nuclease-free water	9.0
5.	25X RT-PCR Enzyme Mix	0.5
6.	Extracted RNA template	1.0
	Total volume per reaction	25.0

Cycling conditions for the NP3/NP4 primers were as described by Ularamu *et al*., 2012 (Figure 6) where ' represents minutes and " represents seconds and ∞ represents infinity.

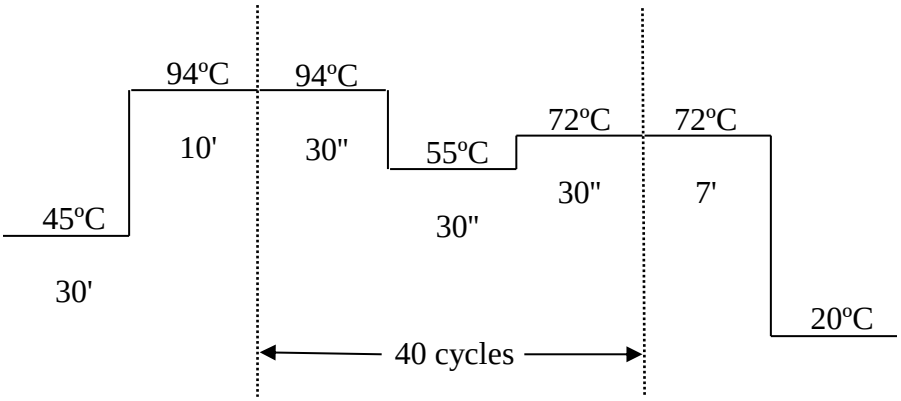

Figure 6. Reverse transcription PCR cycling conditions for the amplification

Table 2. List of primers used in the study.

Name	Gene	Location	Sequence	Amplicon Size	Reference
NP3-forward	Nucleoprotein	1232-1255	5'-TCTCGGAAATCGCCTCACAGACTG-3'	351bp	Ularamu *et al.*, 2012; Couacy-Hymann *et al*, 2002
NP4-reverse	Nucleoprotein	1583-1560	5'-CCTCCTCCTGGTCCTCCAGAATCT-3'	351bp	

3.3.3. Gel electrophoresis and visualization

Reverse transcription polymerase chain reaction (RT-PCR) products were separated by electrophoresis on a 1.5% agarose gel in 0.5% TAE buffer (SERVA Electrophoresis, Heidelberg, Germany) stained with GelRed nucleic acid stain (Phenix Research Products, Candler, USA). Each well was loaded with 5 µl of the PCR product and 1 µl of blue/orange 6X DNA loading dye (Promega, Madison, USA). Samples were separated along with a 1000bp DNA ladder (Promega, Madison, USA) at 100 volts for 30 minutes. The agarose gel was visualized by ultraviolet fluorescence light using a transilluminator (Sigma-Alderich, St. Louis, USA).

3.3.4. Sequencing

Samples that tested positive for PPR virus using the N gene primer set were sequenced. Sequencing of the RT-PCR amplified products was done by Inqaba Biotechnical Industries (Pty) Ltd in Pretoria, South Africa. Four partial N gene sequences from Dakawa (KF939643.1), Ngorongoro (KF939644.1) in Tanzania, Kenya (KP100649.1) and Uganda (KJ867543.1) were aligned using MEGA 7® and found to be 100% similar. The BLAST online tool was used to search in the GenBank for homologous gene sequences in the NCBI database with one of the sequences.

CHAPTER 4: RESULTS

4.1. KEY INFORMANT INTERVIEWS

4.1.1. Key informant one; County Veterinary Official Wajir County.

"Camels that had died from the disease were reported to have a good body condition; in all the outbreaks he had encountered; adult females were mostly affected especially the lactating and the in-calf ones. Key informant one further stated that clinical signs were hardly detected but when observed they were often vague and may include; dullness, bloat, halitosis, swollen lymph nodes (sub-mandibular lymph nodes) before an animal proceeds to sternal recumbency and eventually death occurs.

Those that clinical signs were observed and reported to the county's office were treated with antibiotics (Long Acting Oxytetracycline/Penicilllin & Streptomycin combination) had better prognosis. The progression of the disease before death was between 2-3 days (shortest) and longest at 10-14 days.

Most of the camels that had gone recumbent had been slaughtered & meat consumed. The meat had not caused any ham to human being. During the outbreaks in camels, goats had been suffering from PPR but he stated that he couldn't link the two together."

4.1.2. Key informant two; County Veterinary Official Mandera County

Key informant two stated that the disease was taking a different shape than it used to occur in the previous outbreaks as there was much misrepresentation that the disease was actually sudden. The informant stated that clinical signs were often observed especially when the herder is experienced in herding camels and patience was exercised in probing him for more clues. Often the camels would show clinical signs that are often missed or diagnosed as the usual diseases the herders are accustomed to. Camels that died showed the following signs before dying: nasal discharge, weakness, cycling, staggering gait, halitosis, coughing, diarrhoea in some camels, anorexia, enlarged lymph nodes, swollen neck region. The respondent stated that in his office they had observed two variants of the disease, one which his team had diagnosed as Heart water since the animals present with a neurological syndrome and another where their tentative diagnosis was PPR. During the duration this exercise was being carried there was an active diseased herd which had been diagnosed as PPR.

4.1.3. Key informant three; County Veterinary Official Isiolo County

According to key informant three, the county had not recently reported cases of CSD, the last outbreak was in October to December, 2015, and during the outbreak adult camels were the most affected with no sex predilection. The clinical signs observed before death included; swollen mammary glands, inappetence and recumbency. The key informant was quick to inform the research team that pastoralists sought advice from animal health experts after trying treating animals on their own using antibiotics and this mask the true picture of the clinical disease. There were no cases of sick sheep and goats during Camel Sudden Death outbreaks that occurred in 2015. The major diseases reported in Isiolo were; Camel pox, Trypanosomiasis, mange, camel

flu, mastitis and abscesses. Tryponosomiasis and diseases affecting the respiratory system were suspected to cause Camel Sudden Death.

4.2. DISEASE FIELD INVESTIGATION

Thirty six (36) herds of camels were examined along the travel routes, watering points and as per the recommendations by the county governments. The herds consisted a few camels as less as three to more than 100 per herd. Ninety three per cent 93% of the camels examined although reported to be sick they were not clinically diagnosed healthy. Four (4) herds of sheep were also examined that were found herded alongside the camel herds that we examined. Only 13.75% of those identified appeared to present with clinical PPR; a fever coupled with nasal-ocular discharges and or with diarrhea (See *table below*).

Table 3. Percentage of those clinically sick

Species	Number examined	Number sampled	Percentage
Camels	397	25	**6.329**
Sheep and goats	80	11	**13.75**

4.2.1. Focused group discussion

Following the recommendation of the County Director of Veterinary services, Mandera; a camel herd was tracked to an area called Banisa in Mandera County to assess the situation of the active disease process. A focused group discussion was held with the herders in the area to gather more information about what they knew about the disease (see figure 9).

Figure 7 herds of camels and flocks of goat and sheep at a watering point in Wajir.

According to the herder whose camels were affected, the first case of the camel sudden death disease in their community occurred eight years ago; 2007/2008. There was sudden death of apparently healthy animals at times even during feeding hours. Over 50% of the herders who were present during this discussion had experienced this disease in their herds. The recent outbreak beginning in November 2015 had even the camels aged less than 4 years succumbing to the disease unlike in the past where the majority who would succumb were the reproductive ages.

Figure 8: Interactive session to elucidate on the community knowledge about the disease

The unknown disease affected camels after the rain season and animals die suddenly without any prior clinical signs. They were however quick to point out that when the herder is experienced with herding camels; clinical signs are observed. The affected camels appeared to be losing weight, listless, had a cough and ruffled hair coat few days before the disease progresses rapidly. The animal then appeared like it had some joints problems, would start to diarrhoea, prefer or fall into recumbency and then it would eventually die. There was no knowledge on the causative of the disease though during the rainy season, sheep and goats would also get sick at the same time with some respiratory syndrome identified as contagious Caprine Pleuro Pneumonia and *Peste des petits ruminants*. Conventional medicines and herbal remedies had not been able to protect their camels from this disease compared to other disease where there was generally a good prognosis after treatment.

Figure 9. Suspected Case of a male calf showing emaciation and diarrhoea.

The camel (*Figure* 9) was anorexic, emaciated, had a ruffled hair coat and had matted the perineum showing signs of diarrhoea. The camel was sneezing, had nasal discharges and had a fever of 40°C. The animal was weak and couldn't move in the same pace with the rest of the herd.

Figure 10 Suspect case of a goat in Wajir

This goat (figure 10) was found amongst a herd of camels in a watering point in Wajir (*Figure* 8) above. It was anorectic, had a fever of 39.8°C with mucopurulent nasal discharges. The goat had not been treated.

4.3. PCR RESULTS

Two of the 38 samples collected turned out positive for PPR virus. Using the same primers the two samples, one from Mandera (Camel) and another from Wajir (Goat) were amplified using the same primers to optimize the PCR conditions with gradient PCR machine by changing the concentration of the cDNA and the other master mix contents to yield amplicons as seen in figure 11 and 12 below.

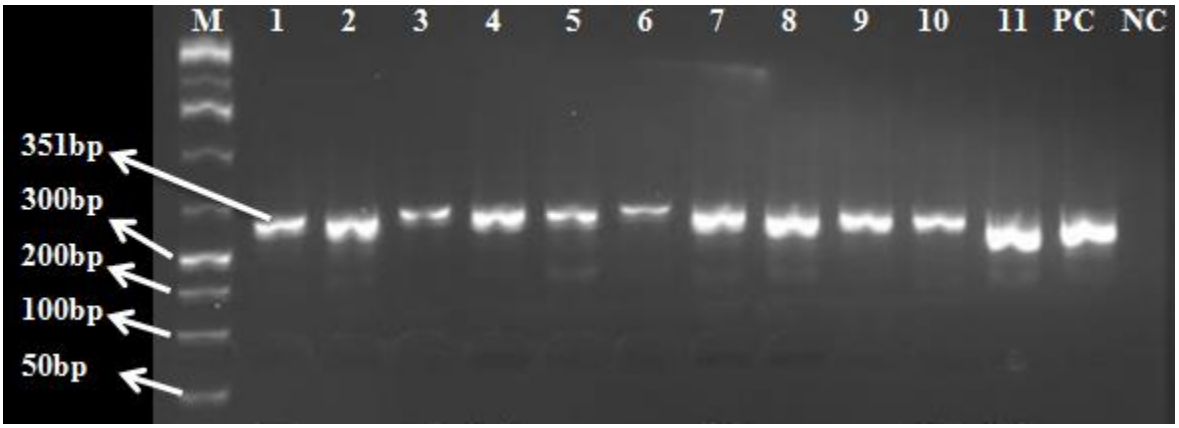

Figure 11 RT-PCR products visualized under UV transilluminator

Figure 11 shows the nucleoprotein gene amplicons where M is the DNA marker, 1-11 samples on gradient PCR. 1-6 is Kenya_ PPRV_ Goat _Wajir; 7-11 is Kenya_ PPRV_ Camel _Mandera, NC is negative control and PC is p.

>Kenya_PPRV Goat Wajir 2017__nucleocapsid (N), partial cds
CCTTCCTCCAGCATAAAATAGGAGAGGGAGAGTCACATGCATCGGCGACCGGGGAAGAAGTCAAAGCTGC
GACCCCAAATGGGCCCGACGAAAAGGACAAAACTCGGGCGCGCTCAGGAAAGCCAAGAGGAGGAACCCCT
GACCAACTGCTCCTGGAAATCATGCCTGAAGACGAGGTCCCGCGAGGGTCTGGACAAAAGCCTCGAGAGG
CTCATTGAT

>Kenya_PPRV Camel Mandera 2017__Ngene_PPR
TCTCGGAAATCGCCTCACAGACTGGGGACGAAAGGACCGCTAGAGGGACCGGGCCCAGACAGGCGCAGGT
TTCCTTCCTCCAGCATAAAATAGGAGTGGGAGAGTCACATGCATCGGCGACCAGGGAAGAAGTCAAAGCT
GCGACCCCAAATGGGCCCGACGAAAAGGACAAATCTCGGGCGCGCTCAGGAAAGCCAAGAGGAGGAACCC
CCGACCAACTGCTCCTGGAAATCATGCCTGAAGACGAGGTCCCGCGAGGGTCTGGACAAAACCCTCGTGA
GGCTCAAAGATCGGCCGAGGCACTCTTTAGACTGCAGGCCATGGCCAAGATTCTGGAGGACCAGGAGGAG

Figure 12. Nucleotide sequences of the two samples that tested positive

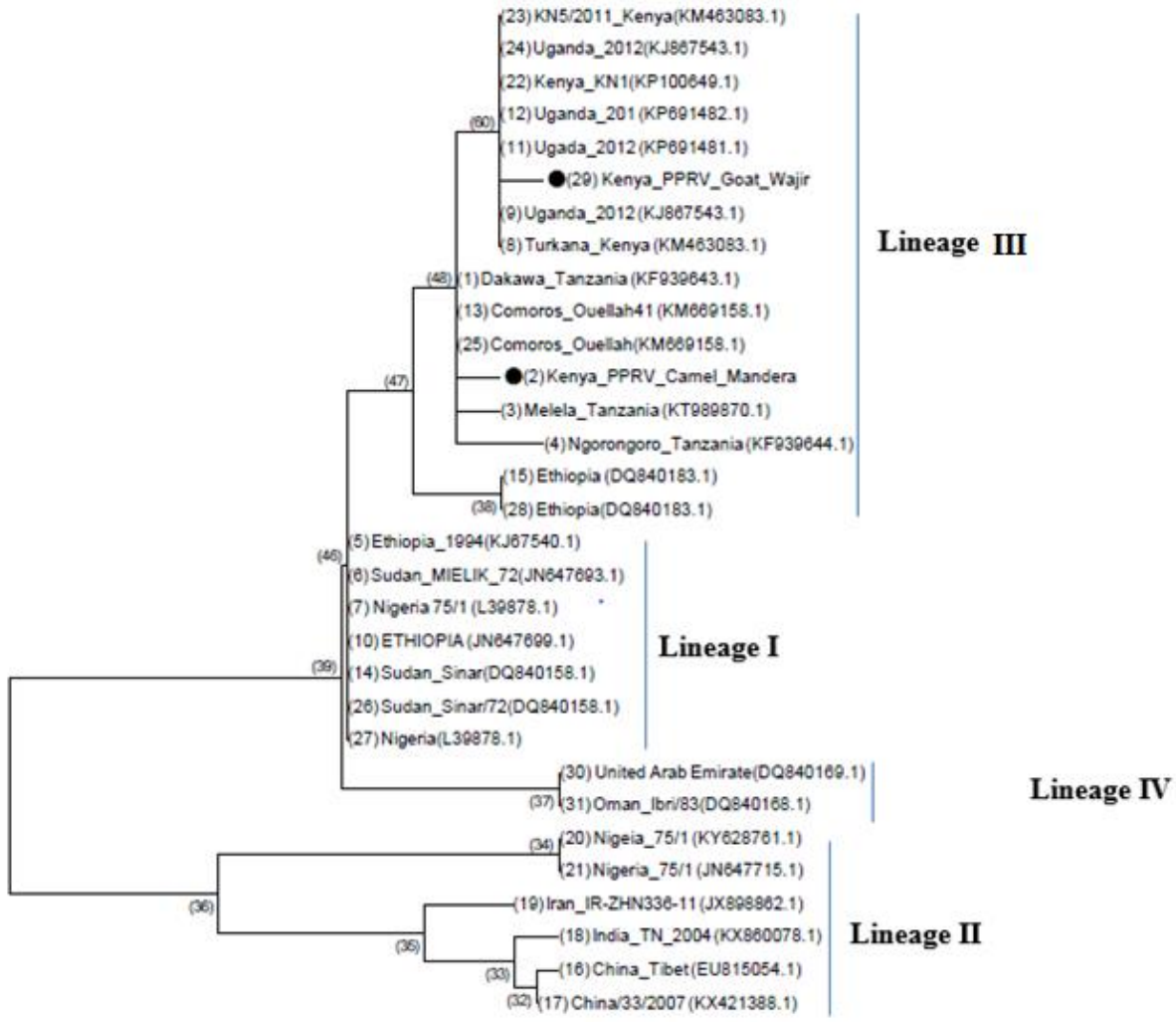

Figure 13. Phylogenetic tree of PPR viruses based on the N gene constructed from MEGA 7®software.

The analysis of sequences revealed closest nucleotide identities of obtained sequences from both goat and camel to the lineage III of PPRV found in East Africa and Ethiopia (Figure 13). The sequences identified in this study are marked with a round black dot (●). Phylogeny was inferred following 1000 bootstrap replications

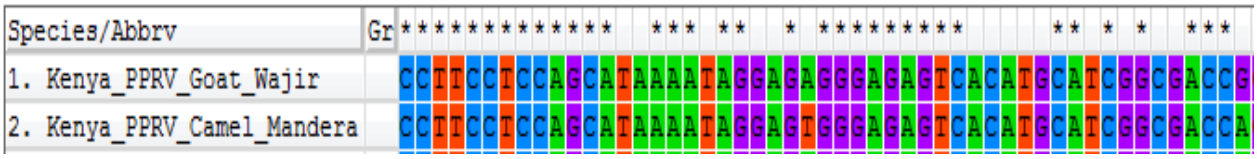

Figure 14.Comparison between the PPRV from camel and goat in a 51bp nucleotide sequence fragment

		1	2
Kenya_PPRV_Goat_Wajir	1	100.00	60.29
Kenya_PPRV_Camel_Mandera	2	60.29	100.00

Figure 15. Pairwise comparison of the isolates

There was 60.29% of nucleotide identity between the PPRV isolate from goat in Wajir district compare to camel isolate in Mandera in Kenya in a 351bp nucleoprotein fragment see figure 14 and 15 above.

CHAPTER FIVE. DISCUSSION

The study has demonstrated that camels display both clinical and laboratory detected *Peste des Petits Ruminants* (PPR). PPR Virus is spread through contact among prone animal's species through respiratory droplets, predominantly when an infected animal coughs or through excretions of clinical infective material such as nasal, ocular and oral discharges (Muniir., 2013; Nwoha *et al.,* 2013;*Abubakar et al.,* 2016). Based on these dynamics; the virus may be spread over areas especially through movement of infected animals especially for trade and/or during relocation for water and pastures (Banyard *et al.,* 2010; Sharma *et al.,* 2015; Taylor., 2016). This is so especially for animals that incubate the disease without overt clinical signs (Balamurugan *et al.,* 2014; Parida *et al.,* 2016).

Several domestic and wild species including cattles (Lembo *et al.,* 2013), buffaloes wildebeest, antelopes and gazelles (Abubakar *et al.,* 2011; Bao *et al.,* 2011; Munir.,2014; Mahapatra *et al.,* 2015; Orynbayev *et al.,* 2016) and recently even dogs (Ratta *et al.,* 2016) have been demonstrated to sero- convert PPR disease virus however, there is limited study on their contribution in maintaining the disease in the animal population. This is the case with camels who continue to be kept with small ruminants in extensive farming systems in the Asian and African continent (Sharma *et al.,* 2003; Dror *et al.,* 2015; Marshall *et al.,* 2016; Gifford-Gonzalez., 2017). Camels have been proven to be susceptible to PPR virus disease both in this study and in literature, nevertheless, there is limited information on camels developing active and fatal disease as their small ruminants' counterparts not until recently (Roger *et al.,* 2001; Khalafalla *et al.,* 2010; Maganga *et al.,* 2013; Zakian *et al.,* 2014; Cosseddu *et al.,* 2013).

This study's finding is in agreement with studies carried out in Sudan and Iran which demonstrated that camels are able to develop an active clinical syndrome caused PPR virus albeit not with the same typical PPR virus disease clinical presentation in small ruminants (Khalafalla *et al.,* 2010; Zakian *et al.,* 2016). According to these findings, the affected camels present with a fatal sub-acute to acute syndrome loosely referred to '*Camel Sudden Death syndrome'* which is rarely noticed and if noticed is nonspecific and characterized by in-appetence, loss of body condition and general weakness, diarrhoea, conjunctivitis oculo-nasal discharges and finally recumbency preceding death of the affected animals. These notable clinical signs that were encountered in this study have been encountered in camels that have tested positive for PPR in Iran as well as Sudan (Khalafalla *et al.,* 2010; Zakian *et al.,* 2016). Of notable significance; these clinical signs are more similar with those that have been observed in sheep and goats manifesting with PPR disease albeit with slight variations of manifestations such as kerato-conjuctivitis as well as oedema of the ventral surface of the abdomen that was observed in this study as well other previous studies (Khalafalla *et al.,* 2010; Zakian *et al.,* 2016). The sudden onset and the fatal nature of PPR disease in camels in the study areas and in previous literature (Khalafalla *et al.,* 2010; Zakian *et al.,* 2016) also shows some similarities with the disease in sheep and goat where sudden onset and high mortalities have been reported more especially in herds that have not been exposed before (Albina *et al.,* 2013).

Of the total animals examined, 6% of those presented as sick had clinical signs similar to those of PPR and, only one (1) of the twenty five (25) camels had a clinical disease that was detected as PPR using laboratory signs representing only 4% of the sick population. This also occurred in sheep and goats herds where only (12.5%) of the eighty (80) individuals presented showed a clinical disease. One camel from Mandera and one goat from Wajir tested positive through RT-PCR. This was despite the fact that all animal sampled were showing clinical signs of a disease identified as PPR. The detection of clinical *Peste des Petits Ruminants* is a challenge as there are other diseases that present with clinical signs that appear as PPR (Couacy-Hymann *et al.,* 2005). Furthermore, PPR is often found as a mixed infection and also there are other diseases that present with fever and with wholly comparable clinical signs, more particularly when it is newly introduced in a herd (Ozmen *et al.,* 2009; Balamurugan *et al.,* 2014; Mondal and Yamage; 2014). Despite the low detection probability, results of on a single individual in a herd can be used to build inferences on the way the disease behaves in a herd (FAO., 1999). Nevertheless, the low detectability of the disease through RT- PCR may have been attributed to the sampled animals having very low viral load hence not detected by the assay, or the RNA might have been lost during sample processing (Mantip, *et al.,* 2016). In addition, the low detectability could also have been attributed to the sampled animals suffering from other diseases that present in the same way as PPR (Couacy-Hymann *et al.,* 2005; Banyard *et al.,* 2010; Balamurugan *et al.,* 2014).

Peste des Petits Ruminants (PPR) virus from Lineage III of was isolated from the samples that were detected positive. This coincides with the lineage that affects goats and sheep and that has been found to be distributed in Kenya as well as other countries that are bordering Kenya including Tanzania and Uganda (Wamwayi *et al.,* 1995; Dundon *et al.,* 2017; Muniraju *et al.,* 2014; Kgotlele, *et al.,* 2014; Luka *et al.,* 2012). In addition the same lineage has been isolated before in other camels that have presented with seemingly similar clinical conditions (Rogers *et al.,* 2001; Khalafalla *et al.,* 2010; Kwiatek *et al.,* 2011, Munir., 2015). This reinforces the geographic and species stability of this virus lineages as stated in earlier studies that sought to understand the genetic variation and geographical clustering of the virus (Libeau *et al.,* 2014).

Despite the above stated geographic stability of the virus lineages, recently lineage IV has been isolated in the host range that Lineage III has been known to occur with the same occurring for lineage II being isolated in lineage I geographical range (Munir *et al.,* 2012; Banyard *et al.,* 2010). Like all the RNA viruses, PPR virus exhibits variable genetic attributes that are known to occur in RNA viruses (Volz *et al.,* 2013). This study findings show that although the two strains were both from lineage III, there were slight differences between the two viruses strains based on the comparative gene sequences of the two sequences obtained. Nevertheless, there was a 60% of nucleotide identity between the two isolates. It is possible that the comparative different gene sequences noted in this study could be as a result of the interaction of PPR virus among the camels and goats in this ecosystem followed with subsequent natural selection that may have made the virus to develop ability to cause atypical clinical signs that have been noted in this study (Munir., 2015). It was not however possible to determine if there was actual transmission of the disease between the different hosts as the sample size was limited and temporally

dispersed. The study was also carried out post epidemic, hence there were fewer animals presenting with clinical signs.

This study findings elucidates that there is possible interaction of this virus between small ruminants and camel population in Kenya subsequently necessitating the evaluation of the role enjoyed by different strains that differences in virulence and their transmission potential (Libeau *et al.,* 2014). The clinical findings as well as the molecular tests confirm this and these correlate with the other studies that have been undertaken in some pastoral regions where small ruminants and camels share the same ecosystem (Zakian *et al.,* 2016; Maganga *et al.,* 2013).

In this study it was not clear on the role PPR plays in the epidemiology of Camel Sudden death syndrome. However, literature and local knowledge demonstrate that PPR is involved in one way or another in camel disease with a sudden onset and unexplained deaths with few or no conceivable clinical signs (Roger *et al.,* 2001; Khalafalla *et al.,* 2010; Kwiatek *et al.,* 2011; Zakian *et al.,* 2016). Herds were demonstrated to succumb to a disease that even though referred to as Camel Sudden Death Syndrome, was clinically diagnosed as PPR by the County Veterinary Team and this was further confirmed as PPR through laboratory diagnosis (PCR). It was also not clear if the clinical picture and sudden death of camels was due to PPR or PPR alongside other co-infections. Even though this study did not isolate other organisms, more so bacteria that have been isolated in camels presenting with PPR (Munir., 2015).

CHAPTER SIX. CONCLUSIONS AND RECOMMENDATIONS

6.1. CONCLUSIONS

1. Camels in the study area suffering with PPR manifest clinical signs that are mainly characterized by inappetence, loss of body condition and general weakness terminally leading diarrhoea, conjunctivitis and ocular nasal discharges preceding death. These clinical signs were similar to those observed in small ruminants with slight variations of manifestations such as kerato- conjuctivitis as well as edema of the ventral surface of the abdomen. The clinical signs were also similar to those reported by other studies done to document the clinical signs of PPR in camels.
2. *Peste des Petits Ruminants* (PPR) was detected in camels both through clinical presentation of the disease and via laboratory confirmation through PCR. The data available for this study was limited and it was not possible to build inferences of the detection probabilities of the disease in the population.
3. Lineage III of the PPR virus was identified through molecular characterization. The virus was in the same lineage as that of goats albeit with 60.29% of nucleotide identity.

6.2. RECOMMENDATIONS

1. During outbreaks of PPR disease in the study counties, camels should be herded separately from the flocks of sheep and goats.
2. More research should be conducted to document to clinical, pathological and laboratory presentation of PPR in Camels.
3. There should be a move to consider vaccination of camels as a strategy of protecting camel keepers from production losses associated with the disease in camels. This would also limit the spread of the disease to other regions where the disease has not been reported.
4. Investigation should be done to find out if there is transmission of PPR among sheep goats and camels.
5. Further research should be conducted to expound on the role PPR plays in Camel Sudden Death.

Conflict of interest statement

The authors declare that they have no competing interests.

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8. APPENDICES

8.1. Appendix1: Key informant Interview

University of Nairobi

College of Agriculture and Veterinary sciences

Faculty of Veterinary Medicine

Department of Veterinary Pathology, Microbiology and Parasitology

Key informant Interviews (Veterinary health workers)

- The current status of CSD in the county.
- Clinical and pathological signs of the disease
- Age group affected
- Tentative diagnosis of CSD

  If there are sick sheep and goats during outbreaks

8.2. Appendix 2 Sample collected from tentative cases of PPR

Blood samples	Lab code	Species	Sample type
Mn/29/01	1	Camel	Blood
Mn/16/01	2	Camel	Blood
Mn/ 65/01	3	Camel	Blood
Mn/08/01	4	Camel	Blood
Mn/15/01	5	Camel	Blood
Mn/14/01	6	Camel	Blood
Mn/81/01	7	Camel	Blood
Wj /goat/01	8	Goat	Blood
Wj /goat/02	9	Goat	Blood
Wj /shp/01	10	Sheep	Blood
Mn/65/01	11	Camel	Nasal
Wj/goat/02	12	Goat	Nasal
Mn/65/01	13	Camel	Ocular
Wj/goat/01	14	Goat	Ocular
Wj/goat/01	15	Goat	Nasal
Wj/goat/04	16	Goat	Ocular
Mn/goat/01	17	Goat	Nasal
Mr/13/01	18	Camel	Nasal
Mr/63/01	19	Camel	Nasal
Mr/29/01	20	Camel	Nasal
Wj/shp/03	21	Sheep	Nasal

V/revival/G	22	Goat Kid	Nasal
Migwi/G3	23	Goat Kid	Nasal
Is/42/1	24	Camel	Blood
Is/35/1	25	Camel	Blood
Is/40/1	26	Camel	Blood
Is/15/1	27	Camel	Blood
Is/25/1	28	Camel	Blood
Is/12/1	29	Camel	Blood
Is/37/1	30	Camel	Blood
Is/36/1	31	Camel	Blood
Is/23/1	32	Camel	Blood
Is/18/1	33	Camel	Blood
Is/31/1	34	Camel	Blood
Is/41/0	36	Camel	Blood
Is/19/0	37	Camel	Blood
Is/27/1	38	Camel	Blood

8.3. Appendix 3. Camels examined and Sampled in Mandera

Herds	Location	Animal examined	Animal sampled
1	**Eskrito**	**10**	**0**
2	**Eskrito**	**12**	**3**
3	**Sanbur**	**10**	**0**
4	**Sanbur**	**10**	**1**
5	**Sanbur**	**10**	**2**
6	Banisa	**8**	**0**
7	Banisa	**10**	**2**
8	**Lulis**	**5**	**0**
9	**Lulis**	**5**	**1**
10	**Lulis**	**5**	**0**
	Total	**85**	**9**

8.4. Appendix 4. Sheep and goats sampled in Mandera County

Herds	Location	Animal examined	Animal sampled
1	**Eskrito**	**15**	**0**
2	**Eskrito**	**7**	**2**
3	Banisa	**9**	**0**
4	**Lulis**	**1**	**1**
		32	**3**

8.5. Appendix 5. Camels Sampled in Marsabit County

Herds	**Location**	**Animal examined**	**Animal Sampled**
1	Ngurunit	**35**	**2**
2	Melgis	**25**	**0**
3	Melgis	**14**	**1**
4	Palgis	**12**	**0**
	Total	**86**	**3**

8.6. Appendix 6. Camels examined and sampled in Wajir County.

Herds	Location	Animal examined	Animal Sampled
1	Irigani	**5**	**0**
2	Irigani	**4**	**1**
3	Irigani	**5**	**0**
4	Bojigaras	**4**	**0**
6	Bojigaras	**7**	**2**
7	Bojigaras	**7**	**0**
8	Bojigaras	**6**	**0**
9	Ibrahim Ule	**8**	**1**
10	Ibrahim Ule	**5**	**0**
11	Ibrahim Ule	**6**	**0**
12	Leheley	**11**	**2**
13	Leheley	**13**	**2**
14	Leheley	**10**	**0**
	Total	**90**	**8**

8.7. Appendix 7. Sheep and goats sampled in Wajir County

Herds	Location	Animal examined	Animal Sampled
1	Irigani	**12**	**3**
2	Irigani	**17**	**0**
3	Bojigaras	**9**	**1**
4	Leheley	**10**	**3**
	Total	**48**	**7**

8.8. Appendix 8. Camels and examined in Isiolo County

Herds	Location	Animal examined	Animal sampled
1	Kulamawe	10	0
2	Kulamawe	12	1
3	Kulamawe	10	0
4	Kulamawe	10	1
5	Kulamawe	15	0
6	Kulamawe	14	1
7	Kina	26	2
8	Kina	24	3
	Totals	**131**	**8**

8.9. Appendix 9: RNA content quantification in sample after sample RNA extraction

Sample ID	Species of animals	Nucleic A Conc./ ng/µl	A260	A280	260/280	260/230	Sample Type	Factor	
Mn/29/01	Camel	-0.2	-0.006	-0.014	0.41	0.4	RNA	40	Blood
Mn/65/01	Camel	1.2	0.03	0.017	1.73	0.3	RNA	40	Blood
Mn/08/01	Camel	0.3	0.007	-0.003	-2.07	0.02	RNA	40	Blood
Mn/15/01	Camel	0	0.001	-0.006	-0.18	0.01	RNA	40	Blood
Mn/14/01	Camel	1.2	0.029	0.01	2.91	0.09	RNA	40	Blood
Mn/81/01	Camel	1.2	0.029	0.016	1.79	0.08	RNA	40	Blood
Wj /goat/01	Camel	10.1	0.254	0.131	1.93	0.1	RNA	40	Blood
Wj /goat/02	Goat	0	-0.001	0	1.14	0	RNA	40	Blood
Wj/sheep/01	Goat	0.5	0.013	0.002	8.47	0	RNA	40	Blood
Mn/65/01	Sheep	0.7	0.017	0.005	3.13	0.02	RNA	40	Blood
Wj/goat/02	Camel	0.3	0.008	-0.008	-0.97	0	RNA	40	Nasal
Mn/65/01	Goat	0.5	0.014	-0.002	-8.07	0.02	RNA	40	Nasal
Wj/goat/01	Camel	0.6	0.015	-0.002	-7.38	0.01	RNA	40	Ocular
Wj/goat/01	Goat	0.2	0.005	-0.004	-1.17	0.04	RNA	40	Ocular
Wj/goat/04	Goat	0.3	0.006	-0.008	-0.84	0.01	RNA	40	Nasal
Wj/goat/01	Goat	3.4	0.085	0.097	0.87	0.08	RNA	40	Ocular
Mr/13/01	Goat	-15.7	-0.392	-0.205	1.91	-0.06	RNA	40	Nasal
Mr/63/01	Camel	-13.8	-0.346	-0.181	1.9	-0.04	RNA	40	Nasal
Mr/29/01	Camel	-10	-0.25	-0.151	1.66	-0.02	RNA	40	Nasal
Wj/sheep/03	Camel	-14.3	-0.357	-0.197	1.81	-0.03	RNA	40	Nasal
V/revival/G	Sheep	3.6	0.089	0.05	1.76	0.01	RNA	40	Nasal
Migwi/G3	Goat Kid	1.2	0.03	0.014	2.18	0.05	RNA	40	Nasal
Is/42/1	Goat Kid	1	0.025	0.004	6.5	0.04	RNA	40	Nasal
Is/35/1	Camel	1.2	0.03	0.022	1.34	0.02	RNA	40	Blood
Is/40/1	Camel	2.2	0.056	0.031	1.8	0.02	RNA	40	Blood
Is/15/1	Camel	1.4	0.036	0.009	3.9	0.01	RNA	40	Blood
Is/12/1	Camel	1.3	0.034	0.02	1.66	0.01	RNA	40	Blood
Is/37/1	Camel	7	0.174	0.04	4.39	0.01	RNA	40	Blood
Is/36/1	Camel	-1.1	-0.028	-0.025	1.13	0.06	RNA	40	Blood
Is/23/1	Camel	-1.3	-0.033	-0.019	1.72	0.07	RNA	40	Blood
Is/18/1	Camel	18.1	0.453	0.325	1.39	0.33	RNA	40	Blood
Is/31/1	Camel	17.4	0.435	0.288	1.51	0.24	RNA	40	Blood
X(k)	Camel	14.6	0.365	0.252	1.45	0.65	RNA	40	Blood
Is/41/0	Camel	46.2	1.155	0.861	1.34	0.24	RNA	40	Blood
Is/19/0	Camel	36.3	0.908	0.647	1.4	0.18	RNA	40	Blood
Is/27/0	Camel	38.3	0.957	0.691	1.38	0.21	RNA	40	Blood
Y(L)	Camel	22.2	0.556	0.376	1.48	0.39	RNA	40	Blood

8.10. Appendix 10. Homologous gene sequences in the NCBI database

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