PESTE DES PETITS RUMINANTS IN GOATS IN KWALE COUNTY: CO-INFECTION WITH CONTAGIOUS CAPRINE PLEUROPNEUMONIA AND RESIDENTS' KNOWLEDGE, PERCEPTION, AND SOCIO-ECONOMIC ASPECT OF THE TWO DISEASES

A thesis submitted in partial fulfilment of requirements for Masters Degree of University of Nairobi (Applied Microbiology, Virology Option)

Lugonzo George Odongo, Bsc.

Department of Veterinary Pathology, Microbiology and Parasitology

AUGUST, 2023

DECLARATION

DECLARATION

I declare that this thesis is my original work and has not been submitted elsewhere for examination or award of a degree. Where other people's work has been used, this has been properly acknowledged and referenced in accordance with the requirements of the University of Nairobi.

Signature..... Date..... 18/08/2023 .....

George Lugonzo Odongo

J56/36140/2019

Department of Veterinary Pathology, Microbiology, and Parasitology

University of Nairobi

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

Prof. George Gitao,
Department of Veterinary Pathology, Microbiology and Parasitology
University of Nairobi, Kenya

Signature ..... Date.....18/08/2023

Prof. Lilly Bebora
Department of Veterinary Pathology, Microbiology and Parasitology
University of Nairobi, Kenya

Signature..... Date.....18/08/2023

Dr Harrison Osundwa Lutta
Biotechnology Research Institute
Kenya Agricultural and Livestock Research Organization

Signature..... Date..... 24/8/2023 .....

ii

DEDICATION

I dedicate this thesis to my mother, Marceline, and my siblings, Joy, Felix, and Lucy, for their unrelenting moral support during my studies. To my son Wein Barrack and my daughter Dancile Lugonzo for bearing with my lengthy absence. Finally, I dedicate this thesis to Stephen Hawking, a fellow scientist who died after battling a debilitating disease, for his contribution to science.

ACKNOWLEDGEMENTS

First, I thank God the Almighty for the strength, knowledge, ability, opportunity to undertake this research study, and perseverance to complete it satisfactorily. Without God's blessings, this achievement would not have been possible. Second, I deeply appreciate my supervisors, Prof. George Gitao, Prof. Lilly Bebora, and Dr Harrison Osundwa Lutta, for their invaluable guidance, encouragement, and support throughout my research work and writing of this thesis. Special appreciation to my family for the moral support they extended to me during my studies. Special thanks to the Biotechnology Research Centre, Kabete leadership, specifically Dr Catherine Taracha, for the material support in conducting research. Sincere appreciation to the leadership of the Kenya Climate Smart Agricultural Program under the Ministry of Agriculture, Kenya, for the scholarship and funding that facilitated this research. I extend my appreciation to several friends for contributing to this work as follows; Ruth Njeri for assisting in data collection, Abbu Ariko, Vincent Ondedo, Christopher Masaba, and Erick Too for assisting me in running laboratory assays. The able team of Dr Raphael Nyawa and Kevin Mkaumba, under the leadership of Dr Mohamed Umlai, many thanks for your assistance and coordination for sample collection and storage logistics.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICES

LIST OF ABBREVIATIONS

ADI:	Arginine deiminase.
ASAL:	Arid and Semi-Arid Lands.
BT:	Bluetongue.
CCPP:	Contagious Caprine Pleuropneumonia.
CD:	Classification determinant
CDV:	Canine distemper virus.
CeMV:	Cetacean morbillivirus.
CFR:	Case fatality rate.
CFT:	Complement fixation test.
CIE:	Counterimmunoelctrophoresis CIE
CPE:	Cytopathic effect.
DPI:	Days post-infection.
EDTA:	Ethylenediaminetetraacetic acid
ELISA:	Enzyme-linked Immunosorbent Assay.
ELISA:	Enzyme-linked Immunosorbent Assays.
FAO:	Food and Agricultural Organization
FAO:	Food and Agriculture Organization of the United Nations.
FMD:	Foot and Mouth Disease.

FMV: Feline morbillivirus.

HI: Haemagglutination Inhibition.

HRP: Horse reddish Peroxidase.

IFN: Inteferon.

IFN-β: interferon beta.

ILRI: International Livestock Research Institute.

KEVEVAPI: Kenya Veterinary Vaccines Production Institute.

KNBS: Kenya National Bureau of statistics.

LAMP: Loop-mediated isothermal amplification.

LAT: Latex Agglutination Test.

Mab: Monoclonal antibodies.

Mccp: *Mycoplasma capricolum* subspecies *capripneumoniae*.

Mmc: *Mycoplasma mycoides* sub sp. Capri.

Mmm: *Mycoplasma mycoides* subsp. *mycoides*.

mRNA: Messenger ribonucleic acid.

MV: Measles virus.

NCM: Nitrocellulose membrane.

NGN: Nigeria Naira.

NH4OAc: Ammonium acetate.

NP: Recombinant nucleoprotein.

OIE: World Organization for Animal Health.

Orf: Open reading Frame.

PDV: Phocine distemper virus.

PHT: Passive haemagglutination test.

PPR: *Peste des Petits Ruminants.*

REA: Restriction enzyme analysis.

RNA: Ribonucleic acid.

RNP: Ribonucleo-protein.

RP: Rinderpest.

RPV: Rinderpest virus.

RT-PCR: Reverse transcription PCR.

SPSS: Statistical Package for the Social Sciences.

TMB: 3,3′,5,5′-Tetramethylbenzidine

VNT: Viral Neutralization Test.

ABSTRACT

Goats are invaluable financial resources for small-scale farmers. Kenya is a developing country that depends immensely on goat production, mainly for food, skin, and income, among other socio-cultural uses. Meanwhile, goats are among the most important small ruminants affected by *Peste des Petits Ruminants* (PPR) and *Contagious Caprine Pleuropneumonia* (CCPP), the two most significant diseases worldwide that limit goat husbandry. Reports on joint CCPP and PPR prevalence in Kenya remain scanty, despite Kenya being among the countries practicing traditional and extensive goat husbandry. In addition, uncertainty exists regarding the elements that impact the disease pattern and dynamics, including the cost of vaccination, access to vaccines, and livestock trade. Herein, the seroprevalence of co-infection of PPR and CCPP and knowledge, perception, and goat husbandry practices that could have contributed to the spread and control of the two diseases in Kwale county were investigated. The PPR and CCPP antibodies in goat serum samples were detected using competitive ELISA and Latex Agglutination Test (LAT), respectively. Likely factors influencing PPR and CCPP control in the county were explored using a structured questionnaire. A total of 368 serum samples were collected in the four sub-counties of Kwale County (Kinango, Lungalunga, Matuga, and Msambweni) from goats of various ages and sexes showing signs of respiratory distress and tested for PPR and CCPP antibodies. A total of 101 goat farmers were also interviewed to assess their knowledge of PPR and CCPP. Of the 368 goats sampled, 259 (70.4%) were females, and 109 (29.6%) were males. Additionally, 126 (34.2%), 71(19.3%), 108 (29.3%), and 63(17.1%) were sampled from Kinango, Matuga, Lunga Lunga, and Msambweni, respectively. The overall PPR seropositivity was 179 (48.6%); for Kinango, Lungalunga, Matuga, and Msabweni, positivity rates were 70.6%, 29.6%, 49.3%, and 36.5%, respectively. The overall CCPP seropositivity was 45.4% (167/368); for Kinango, Lungalunga, Matuga, and Msabweni, the positivity rates were 51.6%, 49.1%, 36.6%, and 36.5%,

respectively. Notably, the seropositivity of PPR was higher in male goats (53.3%) than in female goats (46.7%). However, the PPR and CCPP seropositivity rates were not significantly different between male (44.0%) and female goats (45.9%). Regarding age, the PPR seropositivity rates were 45.9%, 55.8%, and 52.3% in adults, kids, and weaners, respectively. For CCPP, the seropositivity rates were 48.3%, 40. 4%, and 42.3% in adults, kids, and weaners, respectively. The co-infection rate for PPR and CCPP was 22.3% (82/3680). Regarding factors influencing PPR and CCPP control in Kwale county, of the 101 farmers interviewed, 61 (59.4%) were males, and 40 (40.6%) were females. Most of those interviewed Kwale county residents (92.1%) had heard of PPR/CCPP diseases and their associated symptoms; 68.3% had lost an animal to sickness, while 56.3% had had an animal that had died, exhibiting typical PPR/CCPP symptoms. Anaplasmosis, goat pox, rabies, and diarrhoea of unknown origin were among the other prevalent diseases in the county. Despite this, only 33.7% or less of the persons interviewed had vaccinated their animals against either or both infections, which was attributed to the high cost of vaccination and ineffective mass vaccination programs. Most farmers fed their goats by herding (63%), and all their animals slept in one shelter, which might have contributed to the disease spread among animals. Collectively, PPR and CCPP infections in Kwale county remain high. Accordingly, there is a need to intensify vaccination in the county and evaluate the need for joint vaccination of both diseases. Evaluation of the vaccine transport chain is key to understanding the quality of the vaccines at the point of vaccination. Further studies, including the characterization of PPR virus lineages circulating in the region, are needed in order to comprehend the evolution of the virus as well as the role of PPR in suppressing the immune system and increasing the susceptibility of the animal to other infections.

CHAPTER ONE: INTRODUCTION

1.1 Background information

Small ruminants are critical livestock production units worldwide, particularly in developing nations (Sherman, 2011). In low-income countries like Kenya, livestock husbandry significantly reduces poverty, promotes food security, and improves living standards (Herrero *et al*., 2013). Small ruminants are incredibly adaptive and may thrive in even the most adverse conditions. Sheep and goats are significant for smallholder farmers in Kenya in rural communities, reared for monetary value, food source, and social value, such as paying for bride price (Ojango *et al*., 2016). Despite their enormous contribution, optimal goat production is endangered by various factors, including inadequate nutrition, ineffective breeding, poor management, and diseases. Infectious diseases are a serious constraint on animal production worldwide; they result in both direct and indirect losses, including death, increased production costs, decreased productivity, and export restrictions (Kardjadj, 2017). Contagious caprine pleuropneumonia (CCPP) and *Peste des petits ruminants* (PPR) are two of the most serious illnesses affecting goat production in developing countries (Balamurugan *et al*., 2014b).

Peste des Petits Ruminants is a highly infectious contagious viral illness characterized by broncho-interstitial pneumonia, fever, fibrino-necrotic tracheitis, erosive-ulcerative stomatitis, diarrhoea, and mouth sores (Roeder *et al*., 1999). Although the disease is most prevalent in goats and sheep, the number of domestic and wild species that can be affected by PPRV, whether or not they show clinical symptoms, has been growing in the past few years (Dou *et al*., 2020). PPR was initially suspected in Kenya in 1992 (FAO, 2008) but only confirmed in the Turkana district of Kenya in 2007 (ProMed-Mail, 2007). Since then, the disease has spread to the entire pastoral communities in the Arid and Semi-Arid Lands (ASAL) of Kenya, including Narok county in 2008 and Kijiado county in 2010 (Gitao *et al*., 2014; Gitonga *et al*., 2016).

On the other hand, CCPP is a highly contagious goat disease that was first described in Algeria in 1873 and is caused by *Mycoplasma capricolum* subspecies *capripneumoniae* (Mccp) (Thomas, 1873). Goats are the primary hosts of CCPP, although there is evidence that sheep are also affected in goat-sheep mixed herds (Bölske *et al*., 1995). Close contact with affected materials and inhalation of infected respiratory droplets are the most common modes of CCPP transmission (Thiaucourt *et al*., 1996). Clinically, CCPP manifests with fever, difficulty in breathing, coughing, weakness, exercise intolerance, frothy nasal discharge, and lethargy (Sadique *et al*., 2012).

Account of two independent and separate studies demonstrated the simultaneous high prevalence of PPR and CCPP in some locations in Kenya. For instance, Kipronoh *et al*. (2016) reported the seroprevalence of CCPP in Turkana West and Pokot East to be 63.9 % and 29.2 %, respectively. Concurrently, Kihu *et al*. (2015) found PPR seroprevalence in goats in the same region to be 40%**,** demonstrating a possible relationship between the occurrence of the two diseases; combined, the two diseases constrain goat production in many rural communities in Kenya. In early 2021, an outbreak of a respiratory distress disease in Kwale county was reported. It was observed that the treatment outcome of the disease was ineffective; treatment with broad-spectrum antimicrobials was suboptimal, thus creating suspicion of a possible viral infection. This led to a prevalence survey conducted through the Directorate of veterinary services, which revealed a PPR-CCPP co-infection of around 29.25%. Consequently, it was critical to expand on the finding of the pilot study to uncover the actual extent of PPR-CCPP co-infection in the region.

1.2 Problem statement

PPR remains a significant threat to the establishment of sustainable agriculture and food security in areas where the disease is endemic, including Kenya. Outbreaks of PPR have been reported in many countries and regions, including China, South Asia, the Middle East, and sub-Saharan Africa (Shaila *et al*., 1996). In East Africa, serological evidence of PPR has been recorded in several countries (Kihu *et al*., 2015). In Kenya, PPR disease was initially suspected in 1992 (FAO, 2008), but the first officially documented cases in Turkana, Kenya, occurred in 2007 (Pro Med-Mail, 2007). Since then, the disease has spread to the entire pastoral districts in the Arid and Semi-Arid Lands (ASAL) of Kenya (FAO, 2009). Contagious caprine pleuropneumonia is another important and highly infectious disease of goats. The disease causes heavy economic losses in goat farming countries, particularly in Africa, Asia, and the Middle East (Wesonga *et al*., 2004). Mortality rates of 80% and morbidity rates of 100% have been reported in naive and native herds, respectively. The total yearly losses from CCPP are projected to be around US$507 million in endemic areas, highlighting the massive economic costs resulting from the disease. Despite PPR and CCPP being among the diseases notifiable to the World Organization for Animal Health (OIE), outbreaks of the disease have only been reported by a few countries (Liljander *et al*., 2019), which might potentially result from inadequate disease awareness and diminishing public funds which have led to inadequate surveillance and suboptimal diagnostics. The low documentation of PPR and CCPP may also be due to the possible confusion of the clinical signs of these diseases with those of other related respiratory diseases such as *Pasteurella* spp. infections (Peyraud *et al*., 2014).

PPR and CCPP share a few clinical symptoms, including respiratory distress and coughing. Accordingly, the two diseases can easily be confused with each other. Also, PPR-CCPP co-infection has previously been reported (Chota *et al*., 2019, 2020; Kgotlele *et al*., 2018).

1.3 Justification

Goats are invaluable financial resources for the small-scale farmer. Kenya, a developing country, immensely depends on goat production mainly for food, skin, and income, among other socio-cultural uses (Shiferaw *et al*., 2006). Incorporation and full exploitation of goat farming are hampered by a variety of factors, including diseases such as PPR and CCPP. Effective disease containment is achieved by rapid detection along with confirmation of outbreaks. Reports on CCPP and PPR prevalence in Kenya remain scanty, despite Kenya being among the countries practicing traditional and extensive goat husbandry. Besides regular vaccinations against the diseases, frequent outbreaks continue to be observed, particularly in arid and semi-arid areas of the country. PPRV damages the white blood cells causing immunosuppression which may increase the risk of infection and exacerbate the pathogenicity of other infections such as CCPP.

Current data on the prevalence of pathogenic and infectious bacteria, viruses, and parasites in many parts of Kenya are largely lacking. Separate studies have reported concurrent PPR and CCPP outbreaks in some parts of Kenya. However, reports of the diseases jointly are lacking. The findings of this research will create the rationale for making sound decisions in establishing specific control strategies against the diseases, with the primary aim of improving goat productivity in such areas. Findings on the perception and knowledge of Kwale farmers on PPR and CCPP are critical to designing sustainable production methods and practices that encourage and ensure optimal production in pastoral communities where the livelihood heavily relies on small ruminant husbandry. Further comprehension of the goat management methods that may limit PPR-CCPP control could indicate areas of attention for controlling the diseases.

1.4 Objectives

1.4.1 General objective

To determine the seroprevalence, knowledge, and perceptions of *Peste des petits ruminants* and contagious caprine pleuropneumonia co-infections in goats in Kwale county, Kenya.

1.4.2 Specific objectives

1. To determine the seroprevalence of PPR and CCPP co-infections in goats in Kwale county.
2. To evaluate the residents' knowledge and perceptions of PPR and CCPP co-infections in Kwale county.

1.5 Hypotheses

1. There is PPR and CCPP co-infection in goats in Kwale county.
2. Most residents in Kwale county have little knowledge of PPR and CCPP co-infections.

CHAPTER TWO: LITERATURE REVIEW

2.1 *Peste des petits ruminants*

2.1.1 History and geographical distribution of PPR

Since the first case of PPR in Ivory Coast in 1942, the disease has gained global distribution. To date, more than 70 countries in Africa, Asia, Europe, and the Middle East, have reported multiple outbreaks of the disease. In Africa, several countries, including Botswana, Lesotho, Namibia, Malawi, Mozambique, Zambia, South Africa, Zimbabwe, and Zambia, have yet to officially report PPR cases (Dundon *et al*., 2020). In Eastern Africa, PPR virus (PPRV) was first isolated in 1991 in Ethiopia (Roeder *et al*., 1994), although as early as 1977, ailing goat herds in Ethiopia were suspected of having PPR (Agga *et al*., 2019). In Kenya, PPR was detected for the first time in 2006 in Turkana county but was only communicated officially to OIE in 2007. The disease has since spread to neighbouring counties like Samburu and Baringo and has further extended to far-off counties. Although extensive vaccination programs have been rolled out, PPR has spread to several arid and semi-arid areas of Kenya (Kihu *et al*., 2012). Between 2006 and 2008, PPR spread fast, and over 2.5 million animals are believed to have succumbed to the disease within this period. The rapid transmission of the disease has mainly been accelerated by the transboundary movement of goats in search of water and pasture, and the search for better markets (Dundon *et al*., 2017). The disease is caused by viruses from different lineages distributed differently around the globe. Overall, four PPRV lineages have been described so far (Maganga *et al*., 2013). The initial cases of lineages I-III were exclusively reported in West African countries such as Senegal, Ivory Coast, Ghana, and Burkina Faso. Later on, the first case of lineage IV was reported in Asia, which has recently spread to several African countries, including Sudan, Cameroon, Uganda, Algeria, and Egypt (Balamurugan *et al*., 2014a). In Kenya, PPRV lineage III was identified from tissue samples obtained in the North-Western part of the country in May 2017 from goats suspected to have died

of PPR (Dundon *et al*., 2017, 2020). The global distribution of PPR lineages is shown in Figure 2.1.

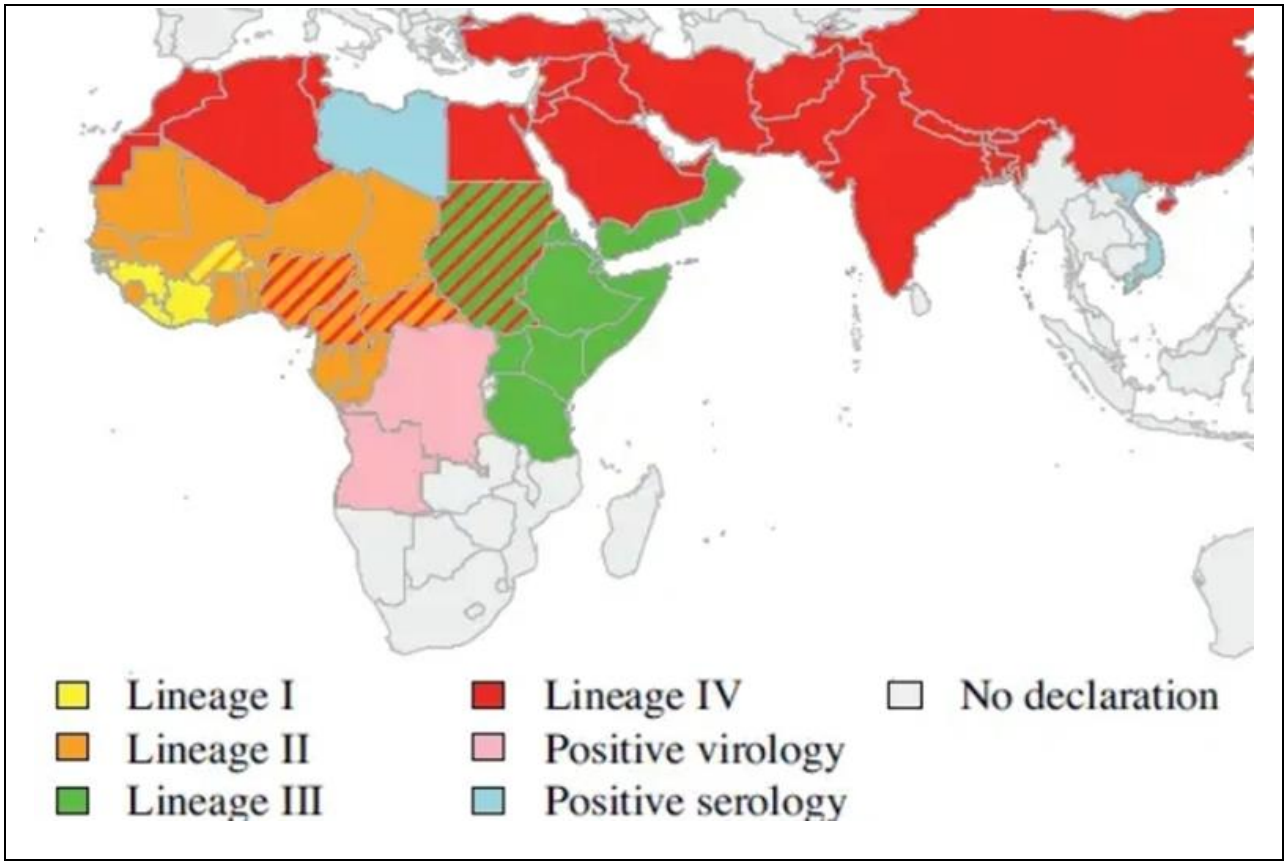


Figure 2.1: Distribution of the PPR virus in Africa and Asia (Maganga *et al*., 2013).

2.1.2 Phylogeny and genetics of PPRV

Peste des petits ruminants is a highly infectious and contagious rinderpest-like disease that is not transmissible to cattle (Gargadennec and Lalanne, 1942). The PPRV belongs to the Morbillivirus genus, Paramyxoviridae family, and in the order Mononegavirales (Gibbs *et al*., 1979). Other viruses in the Morbillivirus genus include the measles virus (MV), rinderpest virus (RPV), PPRV, canine distemper virus (CDV), phocine distemper virus (PDV), cetacean morbillivirus (CeMV), and feline morbillivirus (FMV) (Woo *et al*., 2012). Like other paramyxoviruses, the PPR virions are pleomorphic, enveloped, single-stranded, and non-segmented RNA. The genome of PPRV is

15,948 bp (Bailey *et al*., 2005), ranking just second among the morbilliviruses. The genome encodes six structural proteins, including the nucleoprotein (N), viral RNA-dependent polymerase (L), RNA-polymerase phosphoprotein co-factor (P), matrix protein (M), fusion protein (F), and hemagglutinin protein (H)), and two non-structural proteins (W and C/V) (Bellini *et al*., 1985; Cattaneo *et al*., 1989). The N gene is the most suitable for molecular characterization of closely related virus isolates (Kwiatek *et al*., 2007).

The N protein envelops the genomic RNA. The N and LP proteins form the ribonucleoprotein (RNP). This RNP core encapsulates and protects the PPR genome from endonuclease digestion. Since each molecule of N protein is in close association with the six nucleotides of the genome, 'the rule of six' is necessary for PPRV and other paramyxoviruses (Lamb and Kolakofsky, 2001). Contrary to the popular perception, it has been demonstrated that PPRV follows the rule of six but with some flexibility. The PPRV, like other morbilliviruses, has a phosphoprotein that is highly phosphorylated after translation (thus the tern phosphoprotein) due to high serine and threonine contents (Diallo *et al*., 1987). Though P protein plays an essential role in the synthesis of viral proteins, it is among the least conserved proteins, and a 51.4% amino acid identity between PPRV and RPV P protein has been demonstrated (Mahapatra, 2003). Furthermore, most unconserved residues are found between the 21st to the 306th amino acid position. Because the C-terminus of the P protein participates in the N–P interaction, this terminus is more conserved than the N-terminus of the P protein.

The M protein of PPRV has its open reading frame (ORF) between position 3438–4442 of the genome. This ORF is translated into a protein of 335 amino acids with a molecular weight of 37.8 kDa. The M protein is hugely conserved, and a similarity of 92.5% and identity of 85.0% exist between the M protein of PPRV and RPV. This remarkable degree of conservation indicates the

critical role the M protein plays in cell membrane interaction and the generation of progeny viruses. The M protein of PPRV, RPV, and MV contains three ATG repeats (^{956}tctATGATGATGtca^{970}), which are lacking in the M gene of CDV, PDV, and DMV (Muthuchelvan, 2015).

The F protein (59.137 kDa) is highly conserved among PPRV and RPV all morbilliviruses. In all paramyxoviruses, the F protein is ingrained in the viral lipid bilayer envelope, sticking out as spikes on the surface of the virus. The virulence of paramyxovirus is mediated by the cleavage of the F protein. Since the majority of neutralizing antibodies are directed primarily against the HN protein, this section is constantly under high immunological pressure (Renukaradhya *et al*., 2002). The precise roles of the HN proteins in the attachment of the PPRV to the host cell and PPR progression are unknown. However, the discovery that the H protein is a primary factor for cell tropism in MV and cross-species pathogenesis in lapinized RPV (Yoneda *et al*., 2002) shows that this protein is a critical antigenic determinant of the morbilliviruses. However, the HN protein requires an F protein homolog for optimal replication of PPRV (Das *et al*., 2000).

All morbilliviruses have an L protein that acts as RNA-dependent RNA polymerase for the replication of viral genomic RNA. Also, the L protein carries out capping, methylation, and polyadenylation of viral mRNA, essential steps for effective viral replication. Although the functions of L proteins have not been investigated for PPRV, their role can be predicted based on the high sequence identity among morbilliviruses.

Meanwhile, the P gene is the only PPRV gene that encodes for multiple proteins, including the C protein through an alternative open reading frame and the V protein through RNA editing (Mahapatra, 2003). Apart from the involvement in viral replication, the C protein of RPV inhibits the synthesis of interferon beta (IFN-β) (Boxer *et al*., 2009). The molecular mechanism underlying the inhibition of the C protein is unclear, but it is thought that the C protein participates in blocking

the activation of IFN-β transcription factors. Whereas the C protein has been demonstrated to be a virulence factor in MV infection (Patterson *et al*., 2000) and the replication of RPV (Baron *and* Barrett, 2000), its function in PPRV remains unknown.

The V protein of PPRV has been predicted to have a molecular mass of 32.28 kDa and an iso-electric point of 4.68. By virtue of having the same initial gene frame, The V protein and P protein share the N-terminus since they originate from the same gene frame, but they have distinct C-terminus due to RNA editing (Mahapatra, 2003). Different from the C protein, the V protein undergoes very high phosphorylation in the serine residues in the N terminal, which cover about 60% of this segment (Blom *et al*., 1999). The interferon activity is inhibited by the V protein of most paramyxoviruses. More studies on the V protein of PPRV are required to determine its functions and how it is related to other morbilliviruses. Preliminary findings in an *in vitro* reporter system show that both C and V proteins are involved in regulating the expression of IFN at the ISRE level (Linjie *et al*., 2021). Nevertheless, the molecular mechanisms of inhibition across the two proteins might be distinct.

2.1.3 Hosts of PPRV

Previously, it was thought that PPRV only infects sheep and goats and that goats were more susceptible to the virus (Losos, 1986; Lefèvre and Diallo, 1990). However, further studies revealed that sheep possess intrinsic resistance to PPR (Shaila *et al*., 1989) and, therefore, hardly develop clinical infection (Roeder *et al.,* 1994). PPR has been reported in several wild animals, including the gazelles and deer, Nubian Ibex (Capra ibex Nubian), Dorcas gazelles (*Gazella dorcas*), Laristan sheep (*Ovis orientalis laristani*), Nagale (*Tragelaphinae*), and gemsbok (*Oryx gazella*) (Wang *et al*., 2009; Baron *et al*., 2016). Among domestic animals, PPR infects sheep, goats, calves, buffaloes, and camels (Khan *et al*., 2008; Abubakar *et al*., 2011). Cattle and pigs are believed to

be naturally infected, although experimental attempts to generate clinical illness in adult cattle have been unsuccessful despite the subsequent seroconversion (Gibbs *et al.*, 1979; Taylor, 1984).

Sero-neutralization test revealed a 4.2% positivity of PPR antibodies in 142 camels (Ismail *et al*., 1995). Furthermore, Roger *et al*. (2000) investigated a new camel disease in Ethiopia and found that PPRV was responsible for a spontaneous outbreak of an acute fever illness marked by respiratory distress. In Nigeria, peri-domestic rats have been thought to participate in the epizootiology of PPRV in goats. However, the experimental transmission of the virus from peri-domestic rats to goats was unsuccessful (Komolafe *et al*., 1987). Despite the failed transmission, non-natural hosts (as in the case of rats and camels) may contribute to the persistence of PPRV in the environment, consistent with Couacy-Hymann *et al*. (2005) findings. Cases of other ruminants like cows, buffaloes, and camels being seropositive for PPRV in numerous enzootic locations have been reported (Özkul *et al*., 2002). For instance, in one research, the seroprevalence of PPR in cattle and buffaloes reached 67% and 41%, respectively (Khan *et al*., 2008; Kumar *et al*., 2014).

2.1.4 Transmission of PPR

Peste des petits ruminants virus is transmitted between animals through direct contact with an infected animal or contaminated material. The virus is found in significant quantities in nasal, ocular, and mouth discharges as well as in faeces (Ezeibe *et al*., 2008). Infectious droplets are discharged into the air primarily when a sick animal sneezes or coughs (Abubakar *et al*., 2012). Infection between animals also occurs through numerous other means, including snuggling and licking. Also, while close contact is the primary mode of transmission, infected material can contaminate water, feed troughs, and bedding, creating rare sources of transmission (Bundza *et al*., 1988; Abubakar *et al*., 2011). Obi *et al*. (1983) linked the introduction of fresh stock to both disease outbreaks and transmission. Small ruminants and the trade of related products worldwide

participate in transmitting the disease globally. Besides this, nutritional deficiency weakens the immunity of the animals, increasing the risk of PPR infection and severe outbreaks. Limited research has been conducted on the survival of the *Peste des petits ruminants* virus outside the host animal. However, like rinderpest, it is not likely to persist outside the host for long durations (Rossiter and Taylor, 1994).

2.1.5 Occurrence of PPR

Peste des petits ruminants has a global distribution, especially in goat populations. Sheep and some goats are partly immune and may only present with milder illnesses. The epidemiology pattern of PPR varies greatly between ecological systems and geographical regions. The morbidity and mortality resulting from PPR can be up to 100% and 90% in some goat populations but with significant seasonal variation (Abubakar *et al*., 2009; Baron *et al*., 2016). PPR is rarely fatal in dry and semi-arid areas, where it is usually present as a subclinical infection but increases the risk for other infections, such as pasteurellosis (Lefèvre and Diallo, 1990). Although outbreaks of PPR in West Africa coincided with the wet season, the outbreaks were observed in two different regions during the dry season. According to Nigerian serological data, antibodies were present in all age groups between 4 and 24 months, suggesting continued viral circulation (Taylor, 1979 b). In Oman, the disease persists year-round in susceptible groups (Taylor *et al*., 1990).

2.1.6 Pathogenesis of PPR

The pathogenesis of PPR is comparable to that of rinderpest in cattle (Wohlsein *et al*., 1993; Brown *and* Torres, 1994). Research on the pathogenesis of PPR in ruminants has primarily relied on *in vivo* experiments using virulent forms of the virus (Pope *et al*., 2013; Truong *et al*., 2014). The virus enters through the nasal and oral passages, with initial replication occurring in the respiratory

epithelium. The virus then spreads to the neighboring lymphoid organs, where the second replication occurs before the virions spread to distant organs (Parida *et al*., 2015). A high tropism for lymphoid tissues is displayed in PPR, where the resultant killing of leukocytes induces immunosuppression (Bamouh *et al*., 2019). In particular, PPR replicates in immune cells, including lymphocytes, reticular cells, and macrophages.

Peste des petits ruminants virus infection activates apoptosis of immune mononuclear cells in peripheral blood; lesions in the spleen, thymus, and lymph nodes rusting from the infection have been reported. Apoptosis disrupts immune responses, predisposing the animal to other diseases and further exacerbating PPR pathogenesis (Truong *et al*., 2014). Baron *et al*. (2014) reported a continuous reduction of $CD4^+$ cells after PPR infection, and the depletion of $CD8^+$ cells is associated with a high viral load during the peak of viremia. The PPRV also depletes and impairs the activation of the T cells (Rojas *et al*., 2019). Secondary viral and bacterial infections resulting from leukopenia further weakens the natural defence system of the animal. In general, secondary infections complicate the progression of PPR disease, worsening the disease outcome. Even so, animals that recover from PPR remain immune to the disease for their entire lifetime (Parida *et al*., 2015).

2.1.7 Pathology of PPR

Pathological features of PPR include retrograde and necrotic alterations in lymphoid tissue and epithelial cells of the gastrointestinal and respiratory systems(Tripathi *et al*., 1996b; Kumar, 2001). Other common pathological characteristics of PPR infection include lung consolidation, changes in lung colour, and frothy mucus from lung tissues. The anterior part of the right lung is the most commonly affected site. In addition, subsequent bacterial pneumonia can induce lung lobe

consolidation and airway blockage (Kumar, 2001). Congested alveolar border is also very common.

According to most reports, about 90% of PPR deaths are caused by lung complications (Tripathi *et al*., 1996a, 1996b; Aruni *et al*., 1998). Bronchopneumonia, a chronic disease that can cause pleurisy, is also common. Lymph nodes involving the lungs (mediastinum) and intestine (mesentery) are severely affected through enlargement, swelling, and congestion. The engorged spleen is blocked, the surface of the capsule bleeds profusely, and petechiae may be seen. Severe constipation and gastrointestinal necrosis are common symptoms. Necrosis, hemorrhagic enteritis, or excessive congestion in the ileocecal valve where the colon and rectum meet have also been reported. Straight lines of hyperemia ("zebra pattern" or "zebra pattern") in the mucosal folds behind the colon and rectum are also common (Balamurugan *et al*., 2014b).

2.1.8 Clinical signs of PPR

The clinical manifestation of PPR is identical to that of Rinderpest (RP), except that for PPR, severe lung pathologies are common. Clinical signs of PPR infection include high fever, eye discharges, necrotizing and exfoliating stomatitis, gastroenteritis with diarrhea, followed by either death or recovery from the condition, depending on the severity of the disease. Compared to a normal infection, the clinical symptoms or signs are better characterized experimentally. The disease has an incubation period of 2 to 6 days in natural and experimental infections. Among infected kids, the first clinical symptoms of fever (40.6-44.2°C) are observed on day 5, followed by diarrhea (between day 5 to day 8), and death within 10 days. In addition, Kumar *et al*. (2014) observed an increase in temperature from the third day post-infection (dpi) (104.5 °F) to the 10th day after infection, and the oral mucosa of the affected goats was generally congested, eroded, and ulcerated.

Phases of PPR infection include: the incubation period, a febrile prodromal phase, the mucosal phase characterized by ocular and nasal discharge, erosions on the palate and tongue, and the recovery stage with non-fatal incidents. Regarding severity, PPR can present in several different ways, such as acute, mild and moderate, and this is highly influenced by the PPRV's virulence/lineage, the species (breed) of animal, age, and immunity. Sometimes, PPR can be mistaken for other infections that also present with fever and other clinical signs similar to those of PPR as Bluetongue (BT), contagious ecthyma (Orf), Foot and Mouth Disease (FMD), CCPP, Pasteurellosis, among others. Notably, PPR goat pox, and sheep pox (or Orf/BT, or Orf/Orf) are common co-infections that constrain correct diagnosis and control of the disease. In Nigeria, previous PPR and adenovirus co-infection outbreaks in goats have been confirmed, which might modify the classical clinical disease characteristics.

2.1.9 Control and prevention of PPR

Control of PPR requires avoiding the introduction of new animals from unknown areas, particularly those purchased at livestock markets. Unless the entire herd or flock has been vaccinated, animals brought back from markets without being purchased should also be isolated. The most efficient strategy to reduce the PPR epidemic is through vaccination. Vaccination in locations where the disease is rare is critical if sick animals are brought in from unknown regions. Strictly limiting the movement of animals from places where the disease is endemic is also another efficient way of controlling the spread of the disease. Since the outbreak of PPR in Turkana county in 2006, FAO has invested over USD20 million in response and preventative measures through various sources, saving over Ksh 45.58 billion in revenue by averting the loss of 10 million sheep and goats (FAO, 2021).

Attenuated vaccines that trigger a protective immune response lasting at least three years after vaccination are now commercially available (Diallo *et al*., 2007; Sen *et al*., 2010). Research shows that animals must be immunized at least every three years (Diallo *et al*., 2007; Saravanan *et al*., 2010). It is recommended that animals between the ages of 4 and 6 months should be vaccinated (Balamurugan *et al*., 2012). Animals that recover from the infection acquire a strong immunity that lasts at least four years and, in some goats, the entire lifetime of the animal. When PPR emerges in new locations, eradication is suggested (Enchery *et al*., 2019). Methods used to eradicate rinderpest in many locations could eradicate PPR, including slaughter, proper disposal of carcasses and contact fomites, disinfection, and limiting the immigration of sheep and goats from such areas (Saliki *et al*., 1994). Carcasses and materials that come in contact with products from infected animals should be buried or burned. Contact between wild animals such as gazelles, which are sensitive to the disease, and sheep and goats should be avoided (Yatoo *et al*., 2019).

2.2 Contagious Caprine Pleuropneumonia (CCPP)

2.2.1 Geographical distribution of CCPP

The first CCPP case was reported in 1873 in Algeria, in a shipment of Angora goats to South Africa (OIE, 2022)**.** The bacterium has since spread to over 40 countries worldwide (Yatoo *et al*., 2019), including Qatar in the Middle East and Turkey in Europe (Arif *et al*., 2007; G. E. Jones and Wood, 1988a), Yemen (Rurangirwa *et al*., 1987b), China in Asia (Li *et al*., 2007), and several countries in Africa (Eritrea, Ethiopia, Kenya, Mauritius, Niger, Sudan, Tanzania, Tunisia, and Uganda) (OIE, 2014)**.** In recent times, there have been frequent CCPP outbreaks in areas considered free of the disease. In Kenya, CCPP was confirmed in 1976 (MacOwan *et al.*, 1976) and has since spread to several pastoral regions, including Kajiado, Turkana, and Pokot (Kipronoh *et al*., 2016). The global distribution is shown in Figure 2.2

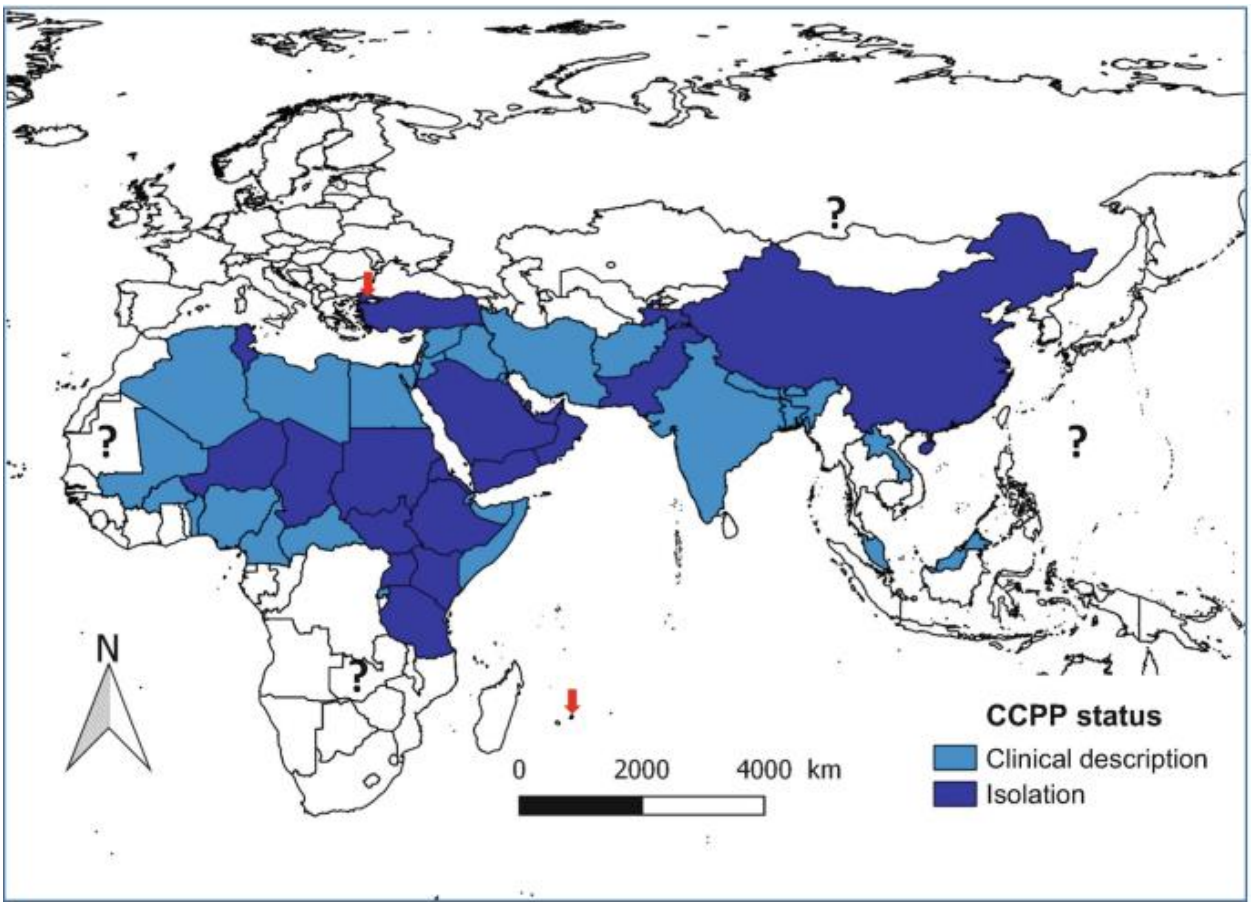


Figure 2.2: The global distribution of Contagious Caprine Pleuropneumonia.

2.2.2 The phylogeny of CCPP organism

Mycoplasma capricolum subspecies *capripneumoniae* (Mccp) is the common term for the causative agent of CCPP (Leach *et al*., 1993). Mccp was originally identified as *Mycoplasma* spp. type F38 because of taxonomic uncertainty, and each isolate was compared to the Kenyan F38 strain. To connect the new designation with the previous nomenclature, the authors abbreviated it as F38. These pathogenic bacteria belong to the Mollicutes class, characterized by the lack of a cell wall, the presence of galactan, and relatively short genomes of 0.58 to 1.35 Mb.

The four mycoplasma lineages are associated with distinct geographical areas. *The Mycoplasma capricolum* subsp. *capricolum* (Mcc) strain belongs to the *Mycoplasma mycoides* cluster, containing numerous species and subspecies, such as *Mycoplasma mycoides* subsp. capricolum

(Mcc), *Mycoplasma mycoides* subsp. capricolum (MmmLC), *Mycoplasma mycoides* subsp. capricolum (MmmSC), *Mycoplasma sp*. Some of these members cause similar diseases in sheep and goats that are manifested outside of the lungs. Due to the relatively recent discovery of Mccp, it is critical to consider the long-held notion that *Mycoplasma mycoides* subsp. Capri (Mmc), rather than Mccp, was to blame for CCPP (Leach *et al*., 1989). A strong taxonomic connection between mycoplasmas has been demonstrated using Tritium-labeled DNA-based hybridization linking *Mycoplasma pneumoniae* (Cottew *et al*., 1987; Bonnet *et al*., 1993).

2.2.3 Hosts of CCPP organisms

Among domestic animals, sheep and goats are both natural hosts for CCPP (Bölske *et al*., 1995). However, goats are more prone to infection than sheep (Lefèvre and Diallo, 1990). Animals in the Bovidae family (Manso-Silván *et al*., 2011) and several ungulates (Ockleford *et al*., 2017) are also vulnerable to CCPP. Several factors, including host density, livestock management systems, strain (Ahaduzzaman, 2020), and the host species and breed, influence the disease severity after infection (Fakri *et al*., 2017). Despite the fact that cattle, domestic buffaloes, and wild African buffaloes are not regarded as CCPP hosts, seroconversion has been reported in enzootic regions in 10% or more of these species, an indication of exposure to CCPP (Abraham *et al*., 2015).

Infections of CCPP in wild sheep, goats, gazelles, Tibetan antelopes, Arabian oryx, and desert gazelles have been reported. Though CCPP may infect other wild species, their sensitivity to Mccp is unknown (Yu *et al*., 2013). Furthermore, it has been hypothesized that CCPP might infect camelids, manifesting as a respiratory syndrome (Paling *et al*., 1988; Khalafalla *et al*., 2010). Attempts to replicate the illness in camels, on the other hand, have proved unsuccessful. Uncertainty surrounds the role of sheep as domestic reservoir species. Sick goats can transmit the disease to wild animals and sheep. Although wild ruminants are possible reservoirs for Mccp, their

significance as disease reservoirs or terminal hosts is unclear (Palmer, 2013). In recent years, CCPP infections in humans have been reported (Bölske *et al*., 1996).

2.2.4 Transmission of CCPP

Contagious Caprine Pleuropneumonia is mainly spread by aerosols and airborne transmissions up to a distance of 50 m (Lignereux *et al*., 2018). However, direct contact with infected animals is the main means of infection; outbreaks result from the introduction of infected lots to a group of vulnerable goats (OIE, 2009). Contagious Caprine Pleuropneumonia is highly infectious and can be transmitted through a brief encounter with infectious aerosols. Given that mycoplasma organisms are highly sensitive to environmental conditions, there is no evidence of indirect transmission (Thiaucourt and Bölske, 1996). It is not yet clear whether contaminated organisms, vectors, and animal products participate in the transmission of Mccp (Panel, 2017). The pathogens can survive for a long time in cold, wet, and crowded environments, causing serious outbreaks; however, the short survival of Mccp (3-14 days) in the external environment limits its spread. Temperatures over 56° C can quickly deactivate Mccp; also when exposed to 60^{o} C for two minutes (OIE, 2009).

2.2.5 Occurrence of CCPP

Contagious Caprine Pleuropneumonia outbreaks are often triggered by environmental factors, such as temperature and humidity. Outbreaks of the disease are common in North Africa during the winter months (Castelet, 1906). In Oman, high prevalences are often observed in January and July at the lowest and highest temperatures, respectively (Jones and Wood, 1988b). In India, CCPP outbreaks in goats have also been observed in winter, particularly during the long winter period of October to May. Although cold temperatures are considered an important predisposition factor, all

forms of stress, including transportation, feeding, and climatic stress, increase the risk of CCPP infection (Parray *et al*., 2019). In general, the introduction of sick animals is the most common way that CCPP enters a country or new region. Even though it is unclear whether there are long-term subclinical carriers, CCPP outbreaks have been reported in endemic regions upon importing completely healthy goats (OIE, 2022).

2.2.6 Pathogenesis and pathology of CCPP

Contagious Caprine Pleuropneumonia pathogenesis involves the respiratory system, the proliferation and damage of epithelial cells in the oral cavity, and oxidative stress. The mycoplasma organism first attaches to the epithelial cells of mucous membranes (Sadique *et al*., 2012). After colonization, it induces inflammation, characterized by epithelial ciliates, serum fibrinous pleuropneumonia, vasculitis, and exudation of fibrin cells. Antigens from the mycoplasma, such as galactans, polysaccharides, and lipoproteins, stimulate inflammation and oxidative cascades that activate the immune system (Darzi *et al*., 1998; Maritim *et al*., 2018). When these pathogenic alterations occur, they promote inflammation and exudation of fluid, especially in respiratory organs such as the lungs, thoracic inserts, pleura, and the heart, but also in the liver and kidneys in rare instances (Yatoo *et al*., 2019).

In all cases of CCPP, there is lung consolidation, accompanied by exudation of the alveoli, accumulation of pleural fluid (91%), and adhesion of the pleura (73%). Microscopic examination of the lesions has indicated septal peri-bronchiolar fibrosis in 82% of the instances, as well as 64% in fibrinous pleuritis. (OIE, 2022). Furthermore, in young, immunocompromised animals, the pathogenesis is mainly acute, while in resistant, healthy animals, it can be chronic. Alveolar exudates predominantly comprise macrophages, neutrophils, and pulmonary fibrin deposits. Several histopathological changes in the lung, including fibrin deposits on alveoli and septal, and

peri-bronchial fibrous tissue, long filaments of the fibrous tissue, chronic pulmonary fibrosis, and pulmonary nodules, as well as alveolitis, broncho-interstitial pneumonia and hyperplasia of the bronchial lymph nodes have also been reported (Wesonga *et al*., 2004; Hussain *et al*., 2012; Sheikh *et al*., 2016)**.** Microscopic histopathological ulcerations, including alveolar atelectasis, interlobular septum thickening, inflammatory cell invasion, and buildup of proteins in the alveoli have been observed in Pashmina goats with CCPP (Parray *et al*., 2019; Yatoo *et al*., 2019)**.**

2.2.7 Clinical signs of CCPP

Clinically, CCPP manifests with inflammatory cascades, serofibrinous exudates in the lower respiratory tract (including the lungs), and pleural effusion. The organism bacterium primarily affects the upper respiratory tract and, less frequently, the eyes, chest, liver, kidneys, and joints. The symptoms of CCPP can be peracute, acute, or chronic. When the pathogen infects a herd that is completely susceptible, the peracute and acute stages of the disease frequently occur. Chronic disease occurs in endemic locations. In peracute cases, asymptomatic respiratory failure and sudden death usually occur within 24-72 hours (MacOwan and Minette, 1976a; Samiullah, 2013). Symptoms are present in acute infection or chronic forms and mild forms in relatively resistant animals with severe fibrin pneumonitis. The infection is characterized by loss of appetite, depression, shortness of breath, high temperature (44–41 °C), cough, runny nose, and growth retardation. In mildly afflicted or relatively resistant animals, typical signs include lying down, thoracic pain, and declining body condition (Rurangirwa *and* McGuire, 1996; OIE, 2014). Fever-related respiratory symptoms (40.3-41.1°C) usually appear after about 2-3 days (Radostits *et al*., 2009).

Clinical presentations commonly include snoring or wheezing and recurrent productive cough, accompanied by painful and rapid breathing (Wang *et al*., 2014; Shah *et al*., 2017). Clinical

symptoms of CCPP generally involve the respiratory system, mainly in the sensitive herds of goats of all ages and sex (Shah *et al*., 2017). Nevertheless, these clinical symptoms, whether peracute, acute, or chronic, might differ from animal to animal based on disease prevalence, type, and severity (Yiwen *et al*., 2021).

The disease's duration is determined by the environmental circumstances under which it occurs. This period can be as short as a few days for animals that have to deal with various stressors, such as malnutrition, parasitism, and moving large distances to get water, among others. The animals may battle the disease for more than a month and even recover with proper care and treatment. In areas where CCPP is endemic, subacute and chronic manifestations of the disease are common. In this situation, the symptoms resemble acute cases, although milder. Coughing is a common symptom after exertion, and nasal discharge may be missing. Without proper treatment, the mortality rates range from 60% to 100% (Kumar *et al*., 2014).

2.2.8 Prevention and control of CCPP

Disease control is possible by rigorous supervision of animal movement and restriction of live animal imports from contaminated regions (countries). Moreover, outbreaks in endemic regions have occurred after the introduction of healthy-looking goats into flocks, raising the question of whether subclinical carriers exist. Overall, outbreaks can be contained and controlled by quarantines, movement restrictions, slaughtering sick and exposed animals, and cleaning and disinfection of infected and exposed buildings. New animals must be introduced slowly into flocks in endemic regions because of the risk of spreading the disease. Broad-spectrum antibiotics, primarily tetracycline or macrolide antimicrobials, such as tylosin and spiramycin, are effective in treating sick animals, but early-stage treatment offers good results (Thiaucourt *et al*., 1996). Vaccination is an effective method of control and prevention of CCPP. In Kenya, inactivated Mccp

bacteria suspended in saponin adjuvant are used and protect goats for about a year (Rurangirwa *et al*., 1987b; Thiaucourt *et al*., 1996). In China and the USSR, aluminium-hydroxide and formalin-inactivated vaccines are used (Rurangirwa *et al*., 1987b).

2.2.9 Economic Importance of PPR and CCPP

Peste des Petits Ruminants has a substantial economic effect, impacting the income and food production of vulnerable and deprived populations (Elsawalhy *et al*., 2010). Small ruminants are valuable assets providing food, fertilizer, wool, fibre, and income in developing nations, particularly to low-income households. The total number of small ruminants globally is estimated to be 2.1 billion, and around 80% are in PPR prevalent regions. The virus causes an estimated $1.5 to 2 billion in financial losses each year (Bardhan *et al*., 2017). Small ruminants are an essential element in pastoralists' coping methods for re-building herds after environmental and political disasters (Elsawalhy *et al*., 2011). They are also given as presents or utilized as symbols in religious and traditional activities (FAO, 2009). According to the International Livestock Research Institute (ILRI) in Nairobi, Kenya, PPR is among the most important disease of animals that must be controlled to alleviate poverty (Perry *et al*., 2002; Kumar *et al*., 2014).

According to FAO estimates, 62.5% of the small ruminant population in the world, especially domestic animals, is at risk of infection with PPR. In India, the annual economic losses resulting from PPR in 2009 were approximated at US$39 million (Chauhan *et al*., 2009). In another report, the overall mortality was estimated at 5% in the same year, and direct economic losses due to PPR in India were estimated to be US $3.6 million. However, if mortality rates are set at 29% for goats and 17% for sheep, this figure rises to around US $13 million (Singh *et al*., 2009). 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) evaluated the annual losses

induced by PPR to be US $1.5 million. Subsequent studies in Africa have mainly focused on the cost-effectiveness of PPR treatment and control. In Nigeria, PPR treatment is between 0.27 and 1.83 Nigeria Naira (NGN) (US $0.30 to 2.03) per animal. A similar analysis conducted in Cameroon established that yearly vaccination against PPR in sheep and goats would improve the production of small ruminants and profits by two to threefold for the farmers (Taylor, 1979 b; Awa *et al*., 2000). In Kenya, a 2015 report by Kihu *et al*. (2015 b) revealed that the direct economic losses caused by PPR in Turkana in 2010 were Ksh. 2 billion (US$ 19 million). The effective global elimination of rinderpest impacted global efforts to eradicate PPR. In India, the ambitious PPR control campaign was initiated in 2010. The Food and Agriculture Organization of the United Nations (FAO) and the World Organization for Animal Health (OIE) launched two pilot programs in 2013 to manage PPR in Africa, funded by the European Commission and the Bill and Melinda Gates Foundation. To vaccinate 3.6 billion animals for three years would require 3.6 billion vaccine doses, each costing $1 and, thus, $3.6 billion.

Contagious Caprine Pleuropneumonia is one of the most serious and contagious goat infections. As a result, nations engaged in goat farming, particularly those in Africa, Asia, and the Middle East, suffer significant economic losses from CCPP infection (Jones and Wood, 1988a; Wesonga *et al*., 2004). For CCPP, fatality and mortality rates vary depending on the animals' age, breed, environment, health status, breed, location, and endemicity of the disease. Higher fatality and mortality rates are common in young and exotic animals and animals living in severe climates or under stress. The case fatality rate (CFR) for acute or peracute outbreaks is extremely high, especially when the goats are not treated (MacOwan, 1976; MacOwan and Minette, 1976a). The mortality rate can range from 9% (Hussain *et al*., 2012) to 100% (Panel, 2017), whereas the CFR ranges from 60% to 100% (Rurangirwa *and* McGuire, 1996; OIE, 2009).

Most often, pure breeds exhibit extremely high mortalities, which can reach 100% (OIE, 2014). The morbidity rate can also be as high as 100% (MacOwan, 1976; MacOwan and Minette, 1976a)**.** In endemic areas, CCPP outbreaks cause over US$507 million worth of losses annually, underlining the economic significance of the disease. Besides the direct costs of preventing, controlling, death, and treating the disease, the illness reduces the productivity of the animal Parray *et al*., 2019). Goat management is hampered by the prevalence of disease, the high cost of treatment, and the trade limitations imposed on animal products from the region. Direct losses result from the death of animals. In industrialized nations, diseased animals are often culled; this is not practicable in countries such as India (Yatoo *et al*., 2018) and Kenya, which are still developing.

2.3 Co-infection of PPR and CCPP diseases

Account of two independent and separate studies has demonstrated the simultaneous high prevalence of PPR and CCPP in some locations. For instance, Kipronoh *et al*. (2016) reported the seroprevalence of CCPP in Turkana West and Pokot East to be 63.9 % and 29.2 %, respectively. Concurrently, Kihu *et al*. (2015) found PPR seroprevalence in goats in the same region to be at 40%**.** Mondal *et al*. (2001) reported that PPRV is an immunosuppressive virus that damages lymphoid organs. Indeed *in vitro* studies have revealed that PPR induces apoptosis of peripheral blood mononuclear cells. Other damages include substantial destruction of thymol and splenic cells, Peter's gland, and lymph nodes (Kul *et al*., 2007; Pope *et al*., 2013). Surprisingly, challenging either vaccinated or unvaccinated goats with PPRV induces the overproduction of CD8+ T cells seven days after infection (Herbert *et al*., 2014), whereas CD4+ cells only increase in unvaccinated sheep (Rojas *et al*., 2014). In general, PPRV infection depletes and disrupts T-cell-mediated functions. Impairment of T cell functions in the early phase of PPR infection predisposes the

animal to other secondary infections such as CCPP. Given the integral role of T lymphocytes in mediating several key immune functions, it can be inferred that PPR exacerbates CCPP infection by modulating immune surveillance against the mycoplasma.

2.4 Diagnosis of PPR and CCPP

2.4.1 Diagnosis of PPR

Symptoms alone are insufficient to distinguish PPR from closely related diseases. Additionally, secondary infections alter the initial disease's clinical profile, thus reducing diagnostic precision. The most widely utilized viral-detection procedures for the diagnosis of PPR are direct PPR virus isolation and immunological approaches. To ensure high-quality clinical samples for these assays, particularly viral isolation, careful sample collection at the right stages of the disease is required. Clinical samples such as rectal swabs, ocular, nasopharyngeal, oropharyngeal, and lymphoid tissues with lesions such as the spleen, mesenteric, and bronchial lymph nodes) are collected from animals. Samples in the early stage of the illness, such as ocular and nasal swabs and blood in EDTA/heparin, are optimal for virus isolation (OIE, 2013). Maintaining the samples at a cold chain until they reach a diagnostic laboratory is essential for accurate and efficient virus detection.

2.4.1.1 Virus isolation

The gold standard test for diagnosis of PPR is virus isolation, which involves cultivating the virus in Vero cells which is the primary cell line obtained from goat or sheep kidneys (Sarkar *et al*., 2003), or marmoset B-lymphoblastoid (B95a) cell lines (Sreenivasa *et al*., 2006). Primary cell cultures have a high sensitivity for viral isolation, but they are not frequently utilized because of the concerns of contamination with other microorganisms, variation among batches, and animal ethics issues. The Vero cell line is frequently used to isolate viruses from clinical or field samples and conduct infectivity studies. B95a cells can maintain PPRV multiplication to a high titer and

are more susceptible to the virus (Kumar *et al*., 2013). However, it often takes many blind passages for a detectable cytopathic effect (CPE) to be observed when isolating the virus in Vero cells.

2.4.1.2 Immunological methods

Enzyme-substrate reactions are used in enzyme-linked immunosorbent assays (ELISAs) to evaluate antigen-antibody interactions. Various ELISA variants are available depending on the need for sensitivity and specificity. Monoclonal antibodies (MAbs) produced against the PPRV nucleoprotein are used in an immunocapture ELISA to detect antibodies (Libeau *et al*., 1994). With a diagnostic sensitivity of 100^6 for the medium tissue culture infectious dose (TCID50), the ELISA technique is ideal for detecting the disease even at the earliest stage. Sandwich ELISA uses polyclonal sera to capture antigens, while PPRV N protein MAb (4G6) is used as the detection antibody (Singh *et al*., 2004a). Compared to a commercial immunocapture ELISA kit, this sandwich ELISA has a diagnostic specificity of 92.8% and a sensitivity of 88.9%. (Singh *et al*., 2004b). Dot-ELISAs make use of anti-matrix protein and anti-nucleoprotein MAbs (Saravanan *et al*., 2006). Due to its lower sensitivity and specificity that give false-positive results, the dot-ELISA is best used together with both known positive and negative samples. The anti-nucleoprotein MAb 4G6 is used in the cell-ELISA, which is an indirect ELISA that detects antigens in infected cells (Sarkar *et al*., 2012). Compared to CPE observation-based virus infectivity titrations, these tests exhibit greater sensitivity and specificity of over 97%.

Immunochromatographic assays are quick, inexpensive, simple to perform, and rapid. Using a nitrocellulose membrane (NCM) as a platform, this assay can identify antigen-antibody interactions by entrapping the unknown antigen/antibody with a known antibody/antigen that has been pre-spotted on the NCM. According to Baron *et al*. (2014) and Brüning-Richardson *et al*. (2011), this method uses an anti-H-protein MAb (C77) as the detection and capture antibody on

the test line for detecting PPRV antigen. Compared with RT-PCR, this test has a relative sensitivity of 84% and specificity of 95%, and it can detect up to 10^3 TCID50/mL of PPRV. Additionally, it recognizes all four PPRV lineages from various geographic origins.

2.4.1.3 Nucleic-acid-based detection techniques

Reverse transcription PCR (RT-PCR) and its variations, including PCR-ELISA and LAMP, are extensively used nucleic-acid-based tools for the detection of PPRV. Morbillivirus-specific RT-PCRs target the fusion protein (F) and phosphoprotein (P) genes (Forsyth and Barrett, 1995). Additionally, RT-PCR targets the 3' end of the mRNA that encodes the nucleoprotein (N) of PPRV. However, conventional RT-PCR is very expensive for large sample sizes and routine diagnosis of clinical samples in laboratories with limited resources. Moreover, these methods are highly susceptible to false-positive results due to cross-contamination from previously collected, processed, and tested materials. Multiplex PCR, which employs numerous primer pairs to amplify multiple gene sections but with high specificity, overrides the false negatives limitation of RT-PCR occasioned by mutations in the primer-binding site. In one-step RT-PCR, Reverse transcription and PCR are carried out in the same microtube during single-step tests, which minimizes cross-contamination. Multiplex RT-qPCR can be used in detecting PPRV in goats and sheep and differentiating from other viruses in syndromic surveillance studies (Settypalli *et al*., 2016). Additionally, real-time (RT)-PCR based on the N gene (Kwiatek *et al*., 2010) and M gene (Balamurugan *et al*., 2010) using TaqMan and SYBR Green probes can be employed to detect PPRV. The PCR-ELISA method combines the amplification of nucleic acid and an ELISA assay for detection (Saravanan *et al*., 2004). For nucleic acid amplification, digoxigenin-labelled primers are utilized. The PCR amplicons are then mixed with biotinylated N-gene-specific probes and detected with ELISA (Kumar *et al*., 2007). Consequently, it can be utilized to diagnose PPR in

the early and late stages of the disease when sandwich ELISA (Singh *et al*., 2004b) may not be appropriate because of the low viral loads. The loop-mediated isothermal amplification (LAMP) assay uses at least four different primers to amplify a target gene sequence. Unlike PCR, LAMP employs a Bst polymerase that catalyzes strand displacement to amplify a target gene during denaturation (Notomi *et al*., 2015). Usually, PPR is diagnosed using the M and N genes of PPRV, which gives a higher sensitivity than the other genes (Li *et al*., 2010; Dadas *et al*., 2012)

2.4.1.4 Conventional PPR antibody detection assays

The OIE recommends the viral neutralization test (VNT) to be the gold-standard for detecting PPR antibodies. VNT involves combining 100 to 1000 TCID50 of PPRV with 100 lL of two-fold dilutions of serum) and incubating at 37° C before inoculating in a 96-well cell culture microplate (P. B. Rossiter *et al*., 1985). When a cytopathic effect (CPE) appears in wells with a particular antibody dilution, it implies that the virus has not been neutralized. Alternative to VNT, the haemagglutination inhibition (HI) test can be used to determine the amount of antiviral antibodies. Fresh RBCs are necessary for conducting HI, and it has been demonstrated that PPRV's HA characteristic varies depending on how it has been grown in cell culture (Dhinakar Raj *et al*., 2000). The counter-immunoelectrophoresis (CIE) approach has been created for the detection of antibodies from diseased animals, much like antigen detection (Tahir *et al*., 1998). Haemagglutination inhibition and CIE are both straightforward tests that might be employed in labs with limited resources. However, they are less sensitive than other methods for detecting antibodies. ELISAs are used for PPR sero-surveillance and sero-monitoring. Anti-H MAbs can be used in competitive and blocking ELISAs (Anderson and Mckay, 1994). In comparison to the VNT, the blocking ELISA showed sensitivity and specificity of 90.4% and 98.9%, respectively (Anderson and McKay, 1994). Similarly, a recombinant N protein antigen generated by a

baculovirus can be employed in a competitive ELISA that has both greater sensitivity (94.5%) and specificity (99.4%) (Libeau *et al*., 1995).

2.4.2 Diagnosis of CCPP

Mycoplasma capricolum subspecies *capripneumoniae* can be detected by microbiological, immunological, or molecular methods following a tentative clinical diagnosis. Based on Mccp's growth and metabolic features, such as its capacity to ferment glucose, digest serum and casein, or decrease in tetrazolium, Mccp can be identified (Soayfane *et al*., 2018). Complement fixation test (CFT) and ELISA are utilized to detect surface antigens, mainly glycolipids and proteins r (Peyraud *et al*., 2014). Molecularly, Mccp has been detected by targeting certain genes or loci, like CAP-21 genomic area (Hotzel *et al*., 1996), 16S rRNA genes (Bölske *et al*., 1996) and arginine deiminase (ADI) gene sequence (Woubit *et al*., 2004b).

2.4.2.1 Microbiological tests

Mycoplasma capricolum subspecies *capripneumoniae* isolation is seen as a technique to confirm a diagnosis, but the isolation is difficult, necessitating technical knowledge for accurate detection (Bölske *et al*., 1996). The pathogen is extremely picky and needs a long initial incubation at 37° C for about 45-47 days with 5% carbon dioxide under sterile experimental conditions that require an advanced laboratory facility (Awan *et al*., 2010).

2.4.2.2 Serological tests

According to earlier studies (Muthomi and Rurangirwa, 1983; Wambugu, 2005), the latex agglutination test (LAT), ELISA, and CFT are frequently utilized for the sero-diagnosis of CCPP. The most recent versions of enhanced LAT are acceptable for field use due to their specificity and sensitivity being comparable to ELISA and are much higher in CFT, CIE, and Passive

haemagglutination test (PHT). However, non-specificity has limited the use of serological testing (Bertin *et al*., 2015).

2.4.2.3 DNA or gene-based molecular tests

Deoxyribonucleic acid probe-based diagnostics have improved the diagnosis and confirmation of the existence of CCPP DNA. PCR-based diagnostic approaches have the potential to detect *M. mycoides* cluster members, particularly Mccp, early and accurately, as well as identify them precisely and specifically (Hotzel *et al*., 1996). *Mycoplasma capricolum* subspecies *capripneumoniae* can be diagnosed by a restriction enzyme analysis (REA) with *Pst1* after PCR (Pettersson *et al*., 1998). This technique works well for clinical field samples like lung tissue, pleural fluid, and discharge from the nose, throat, and ears. Woubit *et al*. (2004a) established a new Mccp-specific PCR approach that identified the *arcD* gene as a distinguishing DNA fragment. Lorenzon *et al*. (2008) established that qPCR was suitable for real-time detection and quantification of Mccp.

Consequently, Fitzmaurice *et al*. (2008) developed five RT-PCR assays standardized for differentiating Mccp from other members of the *Mycoplasma mycoides* clusters. However, the application of these PCR techniques to tissues is limited. He *et al*. (2014) utilized loop-mediated isothermal amplification (LAMP) to rapidly detect Mccp in tissue with high sensitivity based on the amplification of Mccp H2 gene sequences. Consequently, these PCR-based diagnostic tools are useful in both field and laboratory settings. Settypalli *et al*. (2016) developed a multiplex PCR that facilitates the simultaneous identification of Mccp (*Mycoplasma*), bacteria (*Pasteurella multocida*), and viruses (PPRV and capripoxvirus). Therefore, molecular gene-based methods such as PCR, DNA hybridization, and sequence analysis are preferred for determining the phylogenic links among the mycoides cluster (Bashiruddin, 1998). In addition to confirming the

proximity of these members, they have elucidated the contrasts between them (Bashiruddin, 1998). As a result, a quick diagnosis can be achieved using PCR-based DNA amplification and restriction fragment length polymorphism (RFLP) techniques (Bashiruddin, 1998). These assays have proven to be useful in the laboratory and in the field for identifying Mccp.

2.4.3 Factors affecting the control of animal diseases

Animal production is at the heart of farmers in developing countries, particularly those in the arid and semi-arid parts of Sub-Saharan Africa. However, due to the diversity and prevalence of many epidemic diseases in ASALs of Sub-Saharan Africa, animal production and food security at the household level are more constrained by diseases in this region than elsewhere (Frumence *et al*., 2021). A holistic approach that includes appropriate animal husbandry practices and control measures such as zoosanitary provisions that are enforced, vaccination, chemotherapy and chemoprophylaxis, and careful surveillance are required to successfully control and eradicate livestock diseases. For sustainability, these actions should be practical, cheap, and acceptable by farmers as appropriate and essential. Cooperation between livestock keepers and animal health experts is a critical ingredient to successfully and effectively control animal diseases. Unfortunately, this idea is rarely actualized in Sub-Saharan Africa, which is arguably the main reason why animal disease control is less effective in Sub-Saharan Africa region than in other parts (Penrith and Thomson, 2004).

Finance is an important constrain in disease control. First, a question arises as to whether there is return to investment in controlling animal diseases. Despite many studies showing a very highly positive return on investment in disease control (Morris, 1999), some studies show that the eradication of the 2001 foot and mouth disease in the United Kingdom cost more than it achieved, and a cheaper but time-consuming method could have been better (Eye, 2001). Who to pay for

the cost of disease control is another financial aspect that comes out strongly. In the current times, the beneficiary, who is the owner of the livestock, is expected to pay for the control or eradication of diseases. The problem is that in Sub-Saharan Africa, most farmers are very poor, and some depend solely on livestock. In addition, because the benefits of eradicating a disease do not only benefit the owner, the government should step in, but again, many countries in Sub-Saharan Africa can hardly fund large-scale control or eradication programs. In 1996/1997, Botswana undertook a remarkable exception of eradicating contagious bovine pleuropneumonia (CBPP) at a cost of 350 million USD (Amanfu *et al*., 1998). Currently, donors from the international community, such as Food and Agricultural Organization (FAO), provide significant financial aid in the control and eradication of animal diseases. In the case of epidemics, approaches and measures needed to control disease and prevent a transboundary spread are guided by international conventions based on what is accepted in developed countries. Unfortunately, it is not practically possible to apply the control measures in Sub Saharan Africa due to Financial and logistic reasons. Therefore, there is need for more research in practical and achievable control measures that are designed for Sub Saharan Africa in order to develop more specific strategies for good animal health realization. Other factors that impact the control and eradication of animal, including PPR and CCPP are discussed in detail in sections that follow.

2.4.3.1 Uncontrolled movement of animals

Management of contagious animal diseases such as CCPP and PPR requires strict enforcement of restricted animal movement measures such as quarantine. However, controlled movement of livestock in most parts of sub-Saharan Africa is difficult due to the open borders between most countries. In some countries, members of the same ethnic group live on either side of the border, like the Maasai of Kenya and Tanzania, and the individuals and their livestock move between

either side in search of pasture and water. Notably, the practice of pastoralists to move from one place to the other, covering a long distance in search of better grazing land and water, irrespective of country boundaries, is a common practice. Uncontrolled intra-country movement of animals takes place in the same manner as that of inter-countries. In most cases, the border is a road or a river that cuts through settlements where the residents are related or have formed cross friendships, making is difficult to restrict people and animal movement. Furthermore, the officials responsible for enforcing quarantine may compromise their work if offered bribes (Penrith and Thomson, 2004).

2.4.3.2 Animal husbandry practices

Livestock owners and the general public find it challenging to embrace the idea of "stamping out" in both impoverished nations and more developed ones. This method of disease control was successfully used in Ghana and Côte d'Ivoire to control ASF and in Botswana to eradicate CBPP. To ensure smooth control of CCPP and PPR by stamping out, governments are usually required to pay market-related compensation for the animal slaughtered. When mandatory slaughter does occur, it is essential to closely monitor the proper disposal of carcasses. Additionally, the carcasses must be processed in a way that renders them unusable, including burying and burning (Johansen and Penrith, 2009). Due to the high level of poverty, it is challenging to destroy large amounts of meat that would otherwise have been utilized for food.

2.4.3.3 Availability and affordability of aaccines

Prevention of CCPP and PPR epidemics requires either vaccination and chemoprophylaxis, or both. Vaccines can be provided by the government or privately by the farmer. There is limited availability of vaccines in rural areas located far from the cities and towns. The situation is further

complicated because most vaccines require an unbroken cold chain to retain viability. In the event of a disease outbreak, ring vaccination could be done to prevent the spread of the disease. Ring vaccination is expensive, and in developing countries, the government may not be able to afford the expenses. Even when vaccines and funds are available, a lack of infrastructure, such as roads and serviceable vehicles, may delay the delivery of vaccines, or the cold chain might be broken. The state may also not have enough resources to maintain a sufficient number of livestock experts in the disease outbreak area to administer the vaccines (Penrith and Thomson, 2004).

2.4.3.4 Surveillance and monitoring of disease

Adequate surveillance is important for the early detection of disease incidences or an increase in the prevalence of an economically important disease for early and effective control. However, maintaining an effective country-wide surveillance system may be expensive. Additionally, the benefits are long-term, and given that the funders may not appreciate this, they may want to allocate funds to projects that bring instant results. In the event of a disease outbreak, surveillance and monitoring are vital during and after the outbreak to determine the effectiveness of the control method used and establish the degree of the outbreak. Therefore, it is important to set a budget for surveillance and monitoring by the veterinary service of a country to monitor disease outbreaks before and after the outbreak. Additionally, trained personnel, good infrastructure, and funds are needed for effective monitoring and surveillance of animal disease, especially CCPP and PPR (Toma *et al*., 1999).

2.4.3.5 Livestock production systems

Disease control is easily applied in commercial and more intensive production systems than in extensive systems. It is crucial to control diseases where animals are confined, particularly where

a large number of animals are contained in a small area because it favours the spread of contagious pathogens such as Mccp and PPRV. Furthermore, the high mortality of animals in a commercial farm may trigger panic selling of animals which in turn can reach a wide market and contribute to the rapid spread of the disease. Control of disease in an intensive system is easy because the number of animals is usually known, records on births, deaths, purchases, and sales are also kept, and, thus, a rapid response to an emergency is possible. In intensive livestock systems, compliance with the management practices to control a disease depends on the surety of return on the investment. Controlling animal diseases in the traditional production system is challenging because most communities do not have ready access to veterinary services (Penrith and Thomson, 2004). The animal may represent wealth, but the owner may not have ready cash to cater for the animal treatment at that moment. Due to low production levels in these systems, there is a tendency to maintain a large population of animals to compensate for higher mortality and lower reproduction rates. In such cases, the value of the herd is often assessed based on the number of animals rather than their quality. In traditional systems of animal production, because animals are sold through informal outlets, tracing the source of a purchased animal is difficult (Penrith and Thomson, 2004).

The principles of disease control are identical regardless of the production system. Ultimately, however, the aim would be to promote transformation from traditional to commercial systems of production. Historically, development has depended upon the commercialization of agriculture, and there is no doubt that, given the opportunity, sub-Saharan Africa can achieve the necessary level of disease control for optimal animal production.

CHAPTER THREE: MATERIALS AND METHODS

3.1 Study area

The protocol for this study was approved by the University of Nairobi faculty of veterinary medicine. Kwale county, with a total land area of 8,270.2 square km, is located on the Southern Coast of Kenya and borders Tanzania to the Southwest (Figure 3.1). The 2019 population census estimated that Kwale county has a population of 858,748 people (KNBS, 2019). The county lies between Latitude 4° 10' 25.50" S and Longitude 39° 27' 7.42" E.

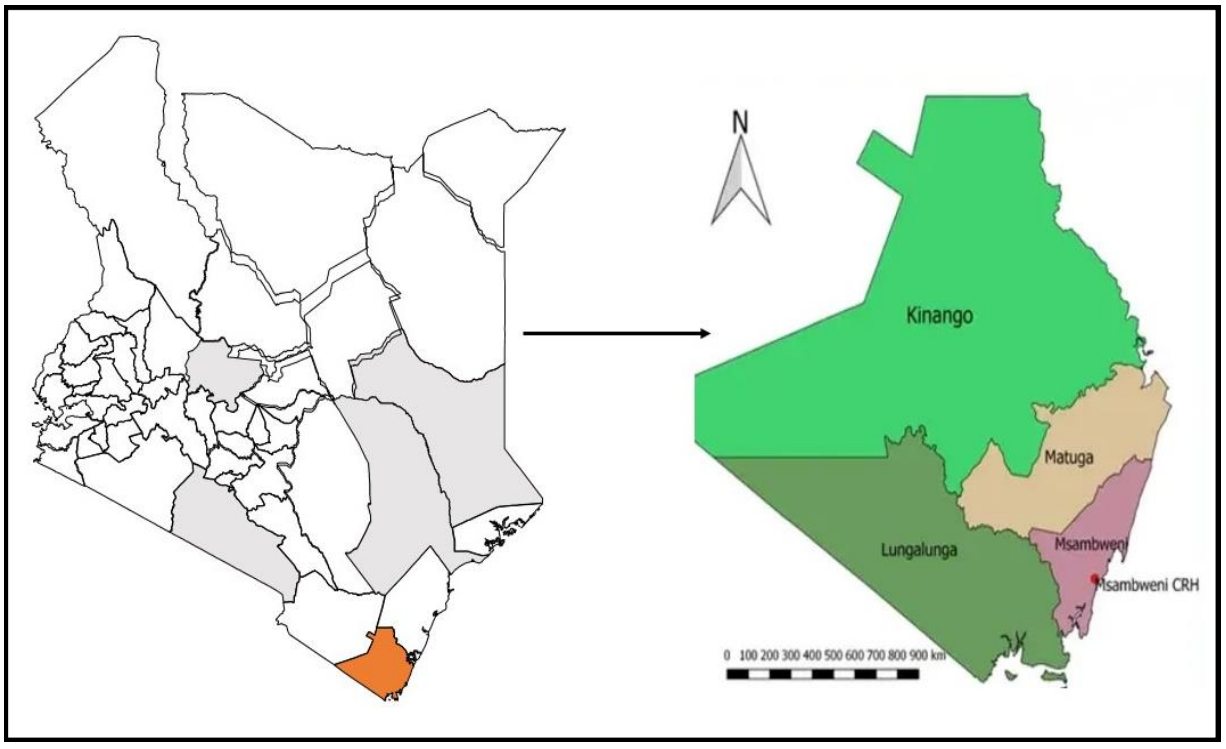


Figure 3. 1: Map of Kenya showing the location of Kwale county

3.2 Study design

This was a cross-sectional study conducted in Kwale county from August 2021 to September of the same year. The target population was goats of all ages, sex, and breed from various farms in the county. The specific target goats were those with respiratory distress and typical PPR and

CCPP symptoms. The occurrence of the two diseases was assessed by serological testing. PPR antibodies were screened using competitive ELISA, while CCPP antibodies were detected using LAT.

3.3 Sample size calculation

Since there was no data on the seroprevalence of PPR in Kwale county, the sample size was calculated using an estimated PPR-CCPP co-prevalence of 29.25%, Z value of 1.96 (at 95% CI), and precision of 5%. The 29.25% was obtained from a pilot study of 106 samples conducted in March 2021. The sample size was determined using the formula $n = Z^2 P (1- P)/ (d^2)$ where n is the sample size, Z is the statistic for the level of confidence, P is predicted prevalence (based on similar studies or a pilot study), and d is precision (based on the size of the effect) (Pourhoseingholi *et al*., 2013). This results in $n = 1.96^2 \times 0.2925(1- 0.2925)/ (0.05^2)$, giving a total of 318 goats.

3.4 Sampling design

Purposive sampling was employed in this study in which goats with respiratory distress were sampled from the four sub-counties of Kwale county (Matuga, Msambweni, Kinango, and Lungalunga). The sub-counties formed the first sampling units, with wards forming the second sampling units. Animals were then sampled from villages in the wards. Only unvaccinated goats were sampled. The history of the goats was obtained from the farmer. Newly bought goats were not sampled because the current farmer could not have an accurate vaccination history of the animal.

Regarding the socioeconomic and goat husbandry factors related to the spread and control of PPR-CCPP infection, 101 goat farmers within the four sub-counties in Kwale county were selected randomly and interrogated using questionnaires. To be included in the survey, one had to have

fulfilled the following: lived in the county for at least five years, reared a goat or goats for at least three years, and had closely participated in goat rearing within the period of their stay in the county.

3.5 Sample collection, handling, and transportation

Blood (10 ml) for screening of PPR and CCPP antibodies was drawn from the jugular vein of goats into non-heparinized vacutainer tubes after cleaning the injection sites using alcohol. The samples were stored in cooler boxes in the field and transported to the Kilifi veterinary laboratories for temporary storage. Serum was extracted from the blood samples after overnight storage at +4° C and stored in 2 ml cryovials. The samples were then transported to the Kenya Agricultural and Livestock Research Organization (KALRO) Kabete biotechnology center to screen for PPR antibodies.

3.6 Screening of anti-PPR antibodies using competitive ELISA (c-ELISA)

Screening of anti-PPR antibodies in the serum samples was performed using a c-Elisa kit (Grabels, France), following the manufacturer's instructions. The wells of the ELISA plates were coated with recombinant nucleoprotein (NP) of the PPR virus. Briefly, 25 ul of the diluent buffer was added to each well of the microtitre plate. Equal volumes of positive controls were added to wells A1 and B1, whereas equal volumes of negative controls were added to wells C1 and D1. Equal volumes of serum samples were added to the rest of the wells. The plates were then incubated for 45 minutes ±5 minutes at 37^{o} C ± 3^{o} C and rinsed three times using 300 µl of wash buffer. Thereafter, 100 µl of 1x conjugate buffer was added to each well (anti-NP-peroxidase; HRP), followed by an incubation of 30 minutes. The wells were rinsed three times using wash buffer before adding 100 µl of substrate solution (TMB). After incubation for 15 minutes, a stop solution was added. The optical densities (ODs) were then obtained at 450 nm using an ELISA reader.

3.7 Screening for anti-CCPP antibodies using Latex agglutination test (LAT)

3.7.1 Preparation of F38-polysaccharide-coated polystyrene beads

3.7.1.1 Mycoplasma culture and confirmation using PCR

Mycoplasma capricolum subsp. *capripneumoniae* culture was obtained from the Kenya Veterinary Vaccines Production Institute (KEVEVAPI) and confirmed using PCR. For DNA extraction, 20 ml of the bacterial culture at the Logarithmic growth phase (1–3 $x10^{10}$ cells) was transferred into a centrifuge tube and centrifuged at 4,100 rpm for 1 hour at 4° C. The supernatant was discarded, and the pellet was washed three times using 5 ml TES buffer (10mM Tris-HCl, pH 7.5, 140mM NaCl, 1mM EDTA) at 4000 rpm for 20 minutes at 4° C. The pellet was re-suspended in 100µL TE buffer (10mM Tris-HCl, pH 7.5, 1mM EDTA, and pH 8.0) and transferred to 2 ml Eppendorf tube. GES buffer (500µL) was added and mixed through vortexing. The mixture was incubated for 5–10 minutes at room temperature (a clear lysate forms), cooled on ice for 3–5 minutes before adding 250µL ice-cold 7.5 M NH4OAc of pH 7.7, mixed by inverting the tube several times and incubated on ice for 10 minutes. PCIA (Phenol: CHCl3Isoamylalcohol=49.5:49.5:1; 500µL) was then added, and the mixture was centrifuged at 14,000 rpm for 15 minutes at 4° C. The aqueous phase was transferred into a new Eppendorf tube, and the PCIA process was repeated three times. Isopropanol was then added to the solution and mixed well (850µL aqueous phase added to 600µL isopropanol). The solution was then incubated at -20° C for at least 20 minutes before centrifugation at 14,000rpm for 15 minutes at 4° C. The supernatant was discarded, and the pellet was washed three times with cold (-20°C) 80% EtOH (without mixing). Ethanol (800µL of 80% ethanol) was added and centrifuged at 14,000rpm for 15 minutes at 4° C. The supernatant was discarded, and the DNA pellets were dried at room temperature for 3 hours in the BSC before re-suspension in 100µL TE (pH 8.0). The DNA concentration was measured using a

Nanodrop/QUBIT; it was then stored at -20° C until used. For confirmation, the 316 bp *arcD* gene was amplified using F, 5′-ATCATTTTTAATCCCTTCAAG-3′, and Mccp-spe-R, 5′-TACTATGAGTAATTATAATA-TATGCAA-3′ primers as previously described by Woubit *et al*. (2004).

3.7.1.2 Extraction of F38 polysaccharide from the mycoplasma culture

The F38 polysaccharide of Mccp, mainly consisting of four neutral sugars (glucose, galactose, mannose, and fructose) and two amino acid sugars (glucosamine and galactosamine), was extracted from the Mccp culture as previously described by Rurangirwa *et al.* (1987). Briefly, the pH of the MCCP culture supernatant was adjusted to 5.0 with glacial acetic acid before boiling for one hour and filtering through a Whatman coarse filter paper. The filtrate was mixed thoroughly with two volumes of ethyl alcohol and then incubated at 4^{o} C for 24 hours. The mixture was centrifuged at 1000 xg for 15 minutes, and the resultant supernatant was suspended in distilled water and stirred for 2 hours at 37^{o} C. Thereafter, the mixture was spun at 3000 xg for 30 minutes at 4°C. The supernatant was collected for subsequent processes. An equal volume of aqueous phenol (6 g of phenol in 1.0 ml of distilled water) was added, and the mixture was incubated at 68^{o} C for 1 hour in a water bath. The mixture was then incubated at 4^{o} C overnight before centrifugation at 3000 xg for 30 minutes at 4^{o} C. The aqueous phase was separated and dialyzed for two days in running tap water to eliminate all the phenol. Two volumes of ethyl-alcohol were added to the dialysate, mixed thoroughly, and incubated overnight at 4°C to precipitate the carbohydrate. The mixture was centrifuged at 1000 xg for 15 minutes to fully precipitate the carbohydrate, which was suspended in a small volume of distilled water. The suspension was dialyzed further for two days with at least four changes of water per day. Finally, the carbohydrate was precipitated with two

volumes of ethyl alcohol and dissolved in 50 ml of distilled water. The dialysate was aliquoted and stored at -20 ^{o}C until further analysis.

The concentration of the polysaccharide was determined using the sulphuric acid method. Glucose was used as the standard. All the reagents used were of analytical grade. Several dilutions of the extracted polysaccharide were prepared alongside D-glucose standards of known concentrations (20, 40, 60, 80, 100, 120, 140, 160, 180, and 200 µg/ml). Phenol solution was added to 0.5 ml of the D-glucose of each concentration in clean labeled test tubes, mixed, and pre-chilled on ice. Concentrated sulphuric acid (2.5 ml) was added, followed by quick vortexing. The tubes were then incubated immediately at 68 ° C for 15 minutes in a water bath. Test samples and water were treated equally. The tubes were cooled at room temperature, and optical densities were determined at 490 nm using an ELISA reader (MR 400 microtiter plate reader, Dynatech instruments, Torrance, CA). The concentration of the polysaccharide was determined using a standard calibration curve drawn from D-glucose readings generated automatically by an ELISA reader. The polysaccharide concentration was then adjusted to 1 mg/ml and stored at -20 °C.

3.7.1.3 Coating of polystyrene beads

The polysaccharides isolated from the Mccp culture supernatants were used utilized for sensitizing latex beads. Polystyrene latex beads (1.08 µl) purchased from Sigma Aldrich (St. Louis, Missouri, USA) were used. To sensitize the beads, 1 ml latex beads were incubated with 1 .0 mg of polysaccharide in 1 ml phosphate buffered saline, supplemented with 0.2 sodium EDTA and 0.01% sodium azide. The mixture was stirred and incubated for 1 hour at 37°C before adding 8 ml of the same buffer to make a final volume of 10 ml. Before conducting the LAT test, the sensitized beads were vortexed for 5 seconds.

3.7.2 Latex agglutination test (LAT)

Lates agglutination test was carried out as described by Rurangirwa *et al*.(1987). In a clear latex agglutination slide, approximately 10 µl of serum was mixed with 10 µl of polysaccharide-sensitized latex beads containing the antigen. The slide was swirled for 1 minute and observed visually against a black background between 1-2 minutes for agglutination. The test was done with a known serum concentration as the positive control and PBS without serum as the negative control. Development of agglutination, as shown in the supplementary Figure in Appendix 6.3, was taken as a positive reaction.

3.8 Questionnaire administration

The questionnaires were filled out through in-person interviews. Farmers that were interviewed were selected randomly from the four counties of Kwale and were guided through challenging sections, including translating the technical terms into the local dialect or language. Numerous factors, including the size of the flock, general knowledge of PPR and CCPP, and goat husbandry, among others, were investigated. Some information, such as the size of the flock and the number of animals with PPR/CCPP symptoms, were verified through observation.

3.9 Data analysis

Data were analyzed using SPSS software, version 25. Grouped data, including disease rate and the distribution of goats of different ages, were summarized into percentages. Differences in PPR and CCPP infection rates among kids, weaners, and adults were analyzed using analysis of variance (ANOVA). The relationship between age and sex and PPR and CCPP infection was investigated using correlation analysis. Statiscal significance was set at $P<0.05$.

CHAPTER FOUR: RESULTS

4.1 Overall serological results for PPR and CCPP

A total of 368 serum samples from goats in Kwale county were analyzed for both PPR and CCPP antibodies. Of these, 259 (70.4%) were from females and 109 (29.6%) from males. With respect to sub-counties, 126 (34.2%), 71(19.3%), 108 (29.3%), and 63 (17.1%) were sampled from Kinango, Matuga, Lunga Lunga, and Msambweni, respectively. The serological results are shown in Table 4.1. Kinango had the highest seroprevalence for both PPR and CCPP; Msambweni had the lowest for the two diseases. A proportion of 22.3% of the tested goats gave positive reactions to both PPR and CCPP (Table 4.2), indicating co-infection by the two diseases.

Table 4. 1: Overall seroprevalence results for PPR and CCPP in Kwale county.

Subcounty	PPR – ELISA No. of positive reactions (%)	CCPP – LAT No. of positive reactions (%)
Kinango	65 (51.6%)	89 (70.6%)
Lungalunga	53 (49.1%)	32 (29.6%)
Matuga	26 (36.6%)	35 (49.3%)
Msambweni	23 (36.5%)	23 (36.6%)
Overall positivity	167 (45.4%)	178 (48.6%)

4.2 The relationship between PPR and CCPP infection

Spearman's correlation coefficient analysis of the two diseases revealed no interdependence between the occurrence of PPR and CCPP infections in the goats (r=0.0084, P=0.8724) (Table 4.2). Univariate logistic regression analyses for the associations between the sex and age of the goat and PPR/CCPP diseases indicated no correlation between the sex and age of the goat and either of the two diseases (Tables 4.3 and 4.4).

Table 4. 2: Correlation between occurrences of PPR and CCPP in goats in Kwale county

			CCPP-LAT		Total
			Negative (Neg)	Positive (Pos)	
PPR-ELISA	Neg	Count	104	85	189
		% within PPR-ELISA	55.0%	45.0%	100.0%
		% within CCPP-LAT	51.7%	50.9%	51.4%
		% of Total	28.3%	23.1%	51.4%
	Pos	Count	97	82	179
		% within PPR-ELISA	54.2%	45.8%	100.0%
		% within CCPP-LAT	48.3%	49.1%	48.6%
		% of Total	26.4%	22.3%	48.6%
Total		Count	201	167	368
		% within PPR-ELISA	54.6%	45.4%	100.0%

Table 4. 3: Relationship between sex and age of the goat and PPR infection.

	Neg. n (%)	**Pos. n (%)**	**Crude Odds Ratio**	**[95% Conf. Interval]**		**P**
Gender						
F	138 (53.28)	121 (46.72)	Ref			
M	51 (46.79)	58 (53.21)	1.297035	0.8282794	2.031076	0.256
Age						
A	113 (55.12)	92 (44.88)	Ref			
K	23 (44.23)	29 (55.77)	1.548677	0.8393203	2.85755	0.162
W	53 (47.75)	58 (52.25)	1.344135	0.8459029	2.135822	0.211

F: Female; M: Male; A: adult goat; K: kid (young one of a goat); W: weaner goat.

Table 4. 4: Relationship between sex and age of the goat and CCPP infection

	Neg. (%)	Pos. (%)	Crude Odds Ratio	[95% Conf. Interval]		P
Gender						
F	140 (54.05)	119 (45.95)	Ref			
M	61 (55.96)	48 (44.04)	0.925747	0.590135	1.452224	0.737
Age						
A	106 (51.71)	99 (48.29)	Ref			
K	31 (59.62)	21 (40.38)	0.725318	0.390973	1.34558	0.308
W	64 (57.66)	47 (42.34)	0.786301	0.493599	1.252574	0.312

F: Female goat; M: Male goat; A: adult goat; K: kid (young one of a goat); W: weaner goat.

4.3 Socio-demographic characteristics of the study participants

A total of 101 permanent residents of Kwale county participated in the survey. Of these, 60 (59.4%) were males, whereas 41 (40.6%) were females. About 64.4% of the interviewed farmers had mixed herds comprising goats, sheep, cows, or other domestic animals, whereas only 35.6% of the interviewed farmers practiced pure goat husbandry.

4.4 Number and categories of goats affected

A total of 231 goats from the 101 farmers interviewed showed typical PPR-CCPP symptoms (Table 4.5). Weaners (52.81%) and females (80.52%) were the most affected categories. The infections were, however, not significantly different between kids (22.94%) and adults (24.24%).

Table 4. 5: Categories of goats affected

Category	No. affected (%)
Kids	53 (22.94%)
Weaners	122 (52.81%)
Adults	56 (24.24%)
Total	231

4.5 Knowledge and perception of PPR and CCPP

The vast majority of respondents (99%) believed that infectious diseases were the most common cause of mortality among goats in the region, with only 1% believing that internal parasites significantly impact goat husbandry in the region. Also, 92.1% of the 101 respondents interviewed had heard of PPR and CCPP diseases compared to 7.9% of the interviewed persons who had little knowledge of the disease and related symptoms.

4.6 Loss of a goat to a disease

Approximately 68% of the respondents had lost animals to diseases. Over half of the lost animals (55.4%) exhibited typical PPR/CCPP symptoms before death, demonstrating the impact of the two diseases on goat husbandry. Unfortunately, the vaccination rate of goats against the two diseases was quite low, with only 21.8%-33.7% of respondents having vaccinated their goats against PPR, CCPP, or both. Other diseases prevalent in the region, based on the history of the animals and the accounts of local veterinary officers, were rabies, goat pox (2%), and anaplasmosis. Loss of goats to injuries from fights, leg wounds, and loss of limbs (1%) was reported. Also, significant losses (36.63%) were caused by non-specific illnesses. The breakdown of the major causes of the loss of animals is shown in Table 4.6.

Table 4. 6: Causes of goat deaths in Kwale county

Cause of death	No. of goats affected (%)
Anaplasmosis	1 (1)
FMD	1(1)
Goat pox	2 (2)
Injury from fight	1 (1)
Leg wounds and loss of limbs	1 (1)
None specific illness	37 (36.63)
Rabies	2 (2)
Typical PPR/CCPP	56 (55.4)
Total	101 (100)

FMD: Foot and mouth disease; CCPP: Contagious caprine pleuropneumonia; PPR: *Peste des petits* ruminants.

4.7 Goat sales and pricing

Data on goat sales and pricing were collected to understand the economic significance of goats, the general goat trade, and how this would impact the spread of PPR and CCPP. The price of a goat ranged from Kshs. 3,000 ($30) to Kshs. 10 000 ($100), averaging 6 000 ($60). About 15.8% of the respondents sold more than seven goats a year. The majority (42.6%) sold an average of 1-3 goats in a year. However, 22.8% of the respondents had not sold a single goat in the past year.

4.8 Goat farming practice

Results showed that goat farming was the main source of income and main farming practice for most Kwale residents, mostly on a small scale, owing to the dry nature of the region. Herding was the most common method for goat feeding, practised by more than half of the respondents (63%).

Free-range (25.7 %) and tethering (9.9 %) came second and third, respectively. None of the interviewed farmers practised zero-grazing of goats. For most of the interviewed persons (68.3%), the goats slept in one cage or were confined in other ways at night. For 14.9% of the farmers, the goats slept in different cages based on age.

CHAPTER 5. DISCUSSION, CONCLUSIONS, AND RECOMMENDATIONS

5.1 Discussion

5.1.1 Co-infections of *Peste des Petits ruminants* (PPR) and Contagious Caprine Pleuropneumonia (CCPP) in Kwale county

Peste des Petits ruminants and CCPP are highly contagious respiratory diseases that threaten goat husbandry. Losses from PPR and CCPP arise from direct and indirect effects, including mortality, morbidity, decreased productivity (increased production costs), costs of vaccination, sanitation measures, control, and trade restrictions (OIE, 2014; Kardjadj, 2017; Parray *et al*., 2019). In endemic countries, economic losses from the two diseases are upwards of US$ 2 billion annually (FAO, 2015; OIE, 2015). In exotic breeds, CCCP morbidity and mortality rates in exotic breeds can reach 100% (DaMassa *et al*., 1992) and up to 80% in disease-naive herds. *Peste des Petits ruminants* can inflict 100% mortality in goats; the disease can also occur in sheep and wild small ruminants (OIE, 2015).

Peste des Petits ruminants was first reported in West Africa in 1942 (Libeau *et al*., 1994) but have since spread to over 40 countries globally. In Kenya, PPR was initially reported in 1992 but later confirmed in the Turkana district in 2007 (ProMed-Mail, 2007; FAO, 2008) through serological and molecular assays. Since then, the disease has spread to all pastoral districts in the Arid and Semi-Arid Lands (ASAL) of Kenya (FAO, 2009), reaching the southern part of Narok County in 2008 and Kijiado County in 2010 (Gitao *et al*., 2014; Gitonga *et al*., 2016). On the other hand, CCPP was first reported in Algeria in 1873 (McMartin *et al*., 1980; Castelet, 1906) but has since spread to many other countries worldwide. The first case of CCPP in Kenya was reported in 1976 (MacOwan and Minette, 1976b), but the disease is primarily confined to pastoral regions, particularly in Kajiado, Turkana, and Pokot (Kipronoh *et al*., 2016).

Peste des Petits ruminants and CCPP continue to be reported in several countries, including Tanzania and Uganda (Gitao *et al*., 2015). *Peste des Petits ruminants* seroprevalence in both sheep and goats has ranged from 2.5–21.3% in Uganda (Ruhweza *et al*., 2010) and 27.4% in Tanzania (Kgotlele *et al*., 2018). Abdalla *et al*. (2012) found that in Sudan, the PPR seroprevalence was 45.6%, relatively higher than in other countries. Contagious Caprine Pleuropneumonia has also been reported in Ethiopia (8.5%) (Abrhaley *et al*., 2019) and Uganda (17.7%-23.3%) (Sheikh *et al*., 2016). In Pakistan, CCPP seroprevalence was reported to be at 32.5% (Hussain *et al*., 2012). In Kenya, however, there are few studies on the prevalence of PPR and CCPP, despite Kenya being among the countries practising traditional and extensive goat husbandry. Besides regular vaccinations against PPR and CCPP, frequent outbreaks continue to be observed, particularly in arid and semi-arid areas of the country (personal observation). Also, no study has assessed the relationship between PPR and CCPP infections despite the infectious nature of PPR for immune cells and the latent infectiousness of *Mycoplasma*, of which the CCPP causative agent is a member. *Peste des Petits Ruminants* virus damages the white blood cells, inducing immunosuppression which may increase the risk or exacerbate the pathogenicity of other infections, including CCPP. The present study was prompted by an outbreak of an unknown respiratory disease in Kwale County, Kenya, in which the treatment response of goats in the region to antibiotics was unsatisfactory (regional veterinary observation). Based on the symptoms, PPR was highly suspected. This led to a serological investigation for the presence of PPR and CCPP in goats in the four Kwale sub-counties. The findings of the current study will provide insight into PPR and CCPP prevalence in the county, one of the country's pastoral regions. Data on PPR is particularly important; it will enable the formulation of measures aligned with the disease eradication plan by 2030. Also, the findings of this research will create the rationale for making sound decisions in

establishing specific control strategies against the diseases, with the primary aim of improving goat productivity in endemic areas. While the PPR and CCPP seropositivity in Kwale county was 41.7% and 47.5%, respectively, there was wide regional variation across the four sub-counties. The CCPP seropositivity in Kwale County was higher than previously reported in Ethiopia (8.5%) (Abrhaley *et al*., 2019), Uganda (17.7%-23.3%) (Atim *et al*., 2016), and Pakistan (32.5%) (Hussain *et al*., 2012). The differences in the seroprevalence may be attributed to disparities in the climatic factors as well as sampling methods and time among these studies.

5.1.2 Relationship between PPR and CCPP infections in goats in Kwale

Univariate analysis revealed no relationship ($p<0.05$) between PPR and CCPP infections despite a high (22.3%) PPR-CCPP co-infection. This might be because the two diseases are endemic in the region, and the exposure risks are comparable. Despite the lack of association, the high seropositivity implies that mass vaccination campaigns against PPR and CCPP in Kwale County are needed. Annual booster vaccinations may be necessary to ensure sustained immunity against the PPRV and CCPP infection. To comprehend the evolution of the PPRV and Mccp circulating in the county, further studies, such as the characterization of these pathogens, are needed. Also, vaccines that induce antibodies unique from natural exposure are needed for accurate monitoring of disease cases; at present, surveys tend to rely on the memory of the farmers, which may be inaccurate.

5.1.3 Relationship between age and sex of goat and PPR and CCPP infections

Further analyses revealed no significant difference in PPR seropositivity among kids, weaners, and mature goats ($p<0.05$), inconsistent with separate findings in Sudan (Salih *et al*., 2014), Ethiopia (Dubie *et al*., 2022), India (Mahajan *et al*., 2012), and Bangladesh (Sarker and Islam,

2011). The inconsistent results could be attributed to the smaller sizes in the first two studies (42 for the first and 21 for the second study, and the difference in the prevalence of PPR between the present and the Sarker and Islam study. Similar to PPR, no significant difference in CCPP seropositivity among kids, weaners, and mature goats ($P<0.05$), consistent with previous findings in Pakistan (Hussain *et al*., 2012) and Tanzania (Nyanja *et al*., 2013). Conversely, a related study in Nigeria (Bello *et al*., 2018) revealed a higher PPR seropositivity in young goats. The cause for this discrepancy is unknown, although it could be due to underlying infections, nutrition, stress, and even climate, which may be harsher to kids than mature goats.

In this study, the gender difference in PPR and CCPP seropositivity was not statistically significant ($p<0.05$). Higher CCPP seropositivity in females has, however, been reported in Tanzania (Nyanja *et al*., 2013), and this might be because female goats are kept longer for reproduction (Abrhaley *et al*., 2019), and their immunity may weaken as they get older, increasing their risk for contracting diseases. Both PPR and CCPP were highest in Kinango sub-county, and this may be because the three other sub-counties are relatively remote, with lesser goat trade which increases the risk of disease transmission. A previous study identified no association between the PPR virus and environmental factors such as stormy weather and strong winds (Acharya *et al*., 2018). However, it showed that regular animal movement and nutritional or climatic stressors increased the susceptibility of goats to CCPP (Parray *et al*., 2019).

5.1.4 Factors affecting the control of *Peste des petits ruminants*-Contagious Caprine Pleuropneumonia infection in Kwale County, Kenya

Uncertainty exists regarding the elements that impact the disease pattern and dynamics, comprising socio-cultural and economic effects such as the disease's importance on goat production, cost of vaccination, access to vaccines, and livestock trading. Several factors,

including animal husbandry, cultural practices, and economic activities, significantly impact animal disease emergence, transmission, and prevalence in some geographical regions. It is, therefore, critical to design sustainable production methods and practices that encourage and ensure optimal production in pastoral communities where the livelihood heavily relies on small ruminant husbandry. Comprehension of the social and cultural components of small stock management methods that may limit PPR-CCPP control could indicate areas of attention for controlling the diseases.

Livestock is the primary source of revenue for local farmers in Kwale County, Kenya. However, optimal goat farming in the region is constrained by numerous factors, with diseases and drought being the most important among them. For diseases, losses arise from morbidity and mortality, as well as treatment and prevention costs. In the present study, 101 goat farmers were interviewed so as to understand the socioeconomic factors affecting goat farming in the region; 59.4% of them were males, whereas 40.6% were females. About 64.4% of the interviewed farmers had mixed herds comprising goats, sheep, cows, or other domestic animals. Among domestic animals, PPR affects mostly goats; sheep are rarely infected (Lembo *et al*., 2013), while subclinical cases in cattle have been reported (Asil *et al*., 2019). This implies that, in mixed herds, the other animals may serve as sources of PPR for goats. Results of this study further showed that most goat farmers in Kwale County were knowledgeable about PPR and CCPP, the major diseases constraining goat farming in the region.

The financial burden associated with CCPP-PPR infection primarily results from the mortality of the animals. According to FAO, research on livestock diseases and management strategies should focus on *Peste des Petits Ruminants* and CCPP, which are two of the leading ten diseases affecting small ruminants in pastoral communities (Diallo, 2006). In the present study, more than

half of the respondents on the socioeconomic factors associated with CCPP and PPR spread and control had lost at least one goat to the diseases. *Peste des Petits Ruminants* and CCPP severely reduce income in terms of death and less trade of goats by limiting the movement of goats and their products to and from diseased areas (Gitao *et al*., 2014; Kihu *et al*., 2015).

Besides being a major source of revenue, goats are also reared for food and cultural practices such as dowry settlement (Manirakiza *et al*., 2021). About 47% of the respondents in the present survey had sold at least one goat in the last year. A goat fetches Kshs. 3,000 - Kshs. 10,000, averaging Ksh. 4 000. *Peste des Petits Ruminants* and CCPP control is primarily through vaccinations. Although Kwale County performs routine vaccination against the two diseases, the coverage is insufficient. PPR and CCPP share several clinical signs, including respiratory distress. Accordingly, goats with PPR maybe be treated with antimicrobials, an inappropriate treatment, which causes the disease to worsen unchecked (Malik *et al*., 2011; Balamurugan *et al*., 2014). *Peste des Petits Ruminants* and CCPP cause significant losses in goats of all ages, and the diseases might be asymptomatic in healthy animals, but they may persist in weak and sick kids (Panel, 2017). Studies conducted in Kenya and Ethiopia showed that young animals are more sensitive to the PPR virus and have a lower chance of developing protective antibodies. Vaccination against the two diseases ensures herd immunity, and more emphasis should be put on the weaners.

To combat CCPP-PPR, the Kenyan government launched biannual (Kyotos *et al*., 2022) and annual vaccination programs (Zhao *et al*., 2021) against CCPP and PPR, respectively, in the disease-endemic areas. However, this program faces numerous challenges, including financial constraints and uncontrolled goat trade. Lack of knowledge on the role of vaccination in

controlling PPR and CCPP is another factor constraining high vaccination uptake in Kwale county, consistent with previous findings reported among women in Machakos county (Kyotos *et al*., 2022). Individual vaccination for fewer herds is also very expensive, with the higher cost falling on facilitating the movement of the veterinary officers rather than the cost of the vaccine itself. With the majority of the farmers in Kwale doing small-scale goat rearing, it is very expensive to undertake individual PPR-CCPP vaccination. Integration of the herd-health disease control strategy could presumptively reduce the cost of vaccination, reduce shock resulting from disease outbreaks and preserve the family source of income, as demonstrated in Ethiopia (Mekonnen *et al*., 2021).

Goats in Kwale County are mainly fed through grazing, given the scarcity of feeds and the large flock size, which may be over 200 goats in some homesteads. Herding increases contact between animals from different villages, increasing disease transmission risk (Yatoo *et al*., 2019; Herzog *et al*., 2020). It was noted that most goat farmers in Kwale County house their goats in one cage, regardless of the disease status.

Peste des Petits Ruminants and CCPP directly impact goat health, lowering productivity and leading to a loss of livelihood and nutritional sources. CCPP and PPR control in Kenya's ASAL areas is constrained by numerous factors, including poor farming practices, low vaccination uptake, uncontrolled trade, and high vaccination cost. Accordingly, the county governments in the disease-risk regions need to upscale the vaccination coverage and subsidize the vaccination cost. If widespread vaccination is not possible, "ring" vaccination and preventive immunization can be explored in high-risk populations. Quarantining diseased animals, removing contact fomites, and limiting imports of sheep and goats from vulnerable areas are other strategies. Infected animals should be slaughtered, and the carcasses buried in deep grounds (Saliki, 1998). Cleaning and

disinfecting affected areas should be done with lipid solvent solutions of low or high pH, disinfectants (sodium hydroxide, phenol, ether alcohol), and detergent (Coetzer *et al*., 1995). Additionally, PPR outbreaks must be monitored closely. Animal handlers must be educated on reactive methods after the identification of these diseases and control measures. These measures include restricting the movement of infected animals, adhering to fundamental hygiene standards, and biosecurity levels within the flocks, which are crucial to limiting the severity and spread of PPR outbreaks (Balamurugan *et al*., 2010).

5.1.5 Knowledge and Perception of PPR and CCPP

By virtue of working and living closely with their livestock, members of pastoralist communities have traditional knowledge of animal health, including animal diseases. However, the terms used on diseases by the locals do not distinguish diseases in a similar manner as the globally recognized names, as they are only based on the observed symptoms rather than the etiological agents causing the disease. In some instances, communities vie different symptoms of a single disease as different diseases (Catley *et al*., 2012). Moreover, because the description of the symptoms varies with local dialects, a given disease in the same location can be referred to by different names(Jones *et al*., 2020). In the present study, the vast majority of respondents (99%) believed that infectious diseases were the most common cause of mortality among goats in the region, with only 1% believing that internal parasites significantly impact goat husbandry in the region. Also, 92.1% of the 101 respondents interviewed had heard of PPR and CCPP diseases compared to 7.9% of the interviewed persons who had little knowledge of the disease and related symptoms.

A study on knowledge and perception of PPR among sheep owners and herders was done in Sudan by Shuaib *et al.* (2016). The herders and sheep owners confirmed to know the clinical symptoms presented by PPR, including fever, depression, dullness, coughing, weight loss, loss of appetite,

lacrimation, diarrhea, and death. Furthermore, half of the participants in Shuaib *et al.* study claimed to have observed the PPR symptoms in their herds. Wifag (2009) reported a similar finding where almost half of the herders and sheep owners identified clinical symptoms of PPR in their livestock. (Wifag, 2009) also reported that 20.9% of owners stated that morbidity was higher than mortality, while 18.6% reported that mortality was higher than morbidity. However, 60.0% of the farmers had no knowledge of either of the parameters. The two studies in Sudan could not verify if the participants had really observed the reported symptoms or if they had heard about them from fellow farmers. In Kenya, pastoralists have reported being aware of PPR-like disease, and their description of clinical symptoms matched the internationally recognized descriptions(Kihu *et al*., 2012).

A good number of farmers in ASALs of Kenya are aware of the clinical symptoms presented by CCPP. A study by (Ouya *et al*., 2022) showed that the majority of farmers in Kajiado and Taita Taveta County were aware of coughing (84%), weakness (50%), respiratory distress (43%), nasal discharge (42%) and nasal discharge (42%) to be CCPP symptoms in goats. Few farmers (17%) reported to know foamy salivation as a CCPP symptom in some goats. Mbyuzi *et al*., (2015) also reported that a high percentage of farmers (62.0%) in Southern Tanzania are aware of clinical symptoms of PPR and CCPP, including coughing, dyspnea, nasal discharge, and rough hair coat as the major symptoms. Diarrhoea, skin nodular lesions, stomatitis, lameness, abortion, and sudden death have also been reported by farmers (Mbyuzi *et al*., 2015).

Poor identification of small ruminant diseases from their clinical symptoms is a major challenge to goat and sheep owners, and it leads to the use of wrong disease control methods. Awareness and knowledge of animal disease is the foundation of technology adoption and uptake of control methods like vaccination and isolation. Due to the declining number of livestock experts and

extension officers in Kenya, coupled with the nomadic nature of pastoralists, animal owners treat their own livestock (Lamuka *et al*., 2017; Kingiri, 2021). Therefore, it is important to create awareness among animal keepers about animal disease identification, prevention and control, and even when to report an outbreak to relevant authorities. In that line, veterinary doctors and other livestock experts should be equipped with simplified training manuals that farmers can understand. Public and private participation in farmer training should be prioritized. Collectively, the livestock sector will be protected against animal diseases by upscaling awareness of control measures at the community level.

5.2 Conclusions

1) The co-infection proportion of PPR and CCPP was 22.3% (82/3680), not significantly higher than the 29.5% rate detected in an earlier pilot study. Despite the high co-infection, there is no relationship between PPR and CCPP infections ($P<0.05$).
2) The majority of Kwale County residents (99%) believed that infectious diseases are the most common cause of mortality among goats in the region.
3) Most Kwale County residents (92.1%) have heard of, or their animals had shown, typical PPR or CCPP symptoms at the time of this study.
4) Only about 33.7% of Kwale County goat farmers had vaccinated their animals against either or both PPR and CCPP infections attributed it to the high cost of vaccination and ineffective mass vaccination programs.
5) Most farmers in Kwale County fed their goats by herding (63%), and all their animals slept in one cage, which may contribute to disease transfer among the animals.

5.3 Recommendations

1) Given the high (22.3%) PPR-CCPP co-infection in Kwale County, mass vaccination campaigns against PPR and CCPP in Kwale County are needed.
2) Annual booster vaccinations may be necessary to ensure sustained immunity against the PPRV and CCPP infection.
3) For the purpose of comprehending the evolution of the PPRV and Mccp circulating in the county, further studies, such as the characterization of these pathogens, are needed.
4) Farmers in Kwale County should be educated on good goat farming husbandry practices, including the need for vaccination, isolating sick animals from healthy ones, and avoiding buying goats during the PPR/CCPP outbreak seasons.
5) Finally, vaccines that differentiate between infected and vaccinated animals (DIVA) are needed for accurate monitoring of disease cases; at present, surveys tend to rely on the memory of the farmers, which might be inaccurate.

REFERENCES

Abdalla, A., Majok, A., Malik, K., and Ali, A. (**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*, ***4***(**3**), 59–64.

Abera, T., Thangavelu, A., Joy Chandran, N. D., and Raja, A. (**2014**). A SYBR Green I based real time RT-PCR assay for specific detection and quantitation of Peste des petits ruminants virus. *BMC Veterinary Research*, ***10***, 22. https://doi.org/10.1186/1746-6148-10-22

Abraham, S., Asha, T. T., Julie, B., Prathiush, P. R., Nandakumar, S., and Prasad, P. M. (**2015**). Pathological and molecular characterization of contagious caprine pleuropneumonia (CCPP) outbreak in Kerala. *Indian Journal of Veterinary Pathology*, ***39***, 121. https://doi.org/10.5958/0973-970X.2015.00028.0

Abrhaley, A., Ejo, M., and Fentie, T. (**2019**). Seroprevalence and Risk Factors Associated with Contagious Caprine Pleuropneumonia in Western Amhara, Northwest Ethiopia. *Journal of Veterinary Medicine*, ***2019***, 9878365. https://doi.org/10.1155/2019/9878365

Abubakar, M., Arshed, M. J., Zahur, A. B., Ali, Q., and Banyard, A. C. (**2012**). Natural infection with peste des petits ruminants virus: a pre and post vaccinal assessment following an outbreak scenario. *Virus Research*, ***167***(**1**), 43–47. https://doi.org/10.1016/j.virusres.2012.03.018

Abubakar, M., Jamal, S. M., 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***(**7**), 1197–1202. https://doi.org/10.1007/s11250-008-9300-9

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 Ruminant Research*, ***96***(**1**), 1–10. https://doi.org/10.1016/j.smallrumres.2010.10.006

Acharya, N., Poudel, S. P., and Acharya, K. P. (**2018**). Cross-sectional sero-prevalence study of Peste des Petits Ruminants (PPR) in goats of Syangja and Kaski districts of Nepal. *VirusDisease*, ***29***(**2**), 173–179. https://doi.org/10.1007/s13337-018-0449-1

Agga, G. E., Raboisson, D., Walch, L., Alemayehu, F., Semu, D. T., Bahiru, G., Woube, Y. A., Belihu, K., Tekola, B. G., Bekana, M., Roger, F. L., and Waret-Szkuta, A. (**2019**). Epidemiological Survey of Peste des Petits Ruminants in Ethiopia: Cattle as Potential Sentinel for Surveillance. *Frontiers in Veterinary Science*, ***6***. https://www.frontiersin.org/articles/10.3389/fvets.2019.00302

Ahaduzzaman, M. (**2020**). Peste des petits ruminants (PPR) in Africa and Asia: A systematic review and meta-analysis of the prevalence in sheep and goats between 1969 and 2018. *Veterinary Medicine and Science*, ***6***(**4**), 813–833. https://doi.org/10.1002/vms3.300

Amanfu, W., Masupu, K., Adom, E., Raborokgwe, M., and Bashiruddin, J. (**1998**). An outbreak of contagious bovine pleuropneumonia in Ngamiland district of north-western Botswana. *Veterinary Record*, ***143***(**2**), 46–48.

Anderson, J., and Mckay, J. A. (**1994**). The detection of antibodies against peste des petits ruminants virus in cattle, sheep and goats and the possible implications to rinderpest control programmes. *Epidemiology and Infection*, ***112***(**1**), 225–231. https://doi.org/10.1017/S0950268800057599

Anderson, J., and McKay, J. A. (**1994**). The detection of antibodies against peste des petits ruminants virus in cattle, sheep and goats and the possible implications to rinderpest

control programmes. *Epidemiology and Infection*, ***112***(**1**), 225–231. https://doi.org/10.1017/s0950268800057599

Arif, A., Schulz, J., Thiaucourt, F., Taha, A., and Hammer, S. (**2007**). Contagious Caprine Pleuropneumonia Outbreak in Captive Wild Ungulates at Al Wabra Wildlife Preservation, State of Qatar. *Journal of Zoo and Wildlife Medicine*, ***38***(**1**), 93–96.

Aruni, A. W., Lalitha, P. S., Mohan, A. C., Chitravelu, P., and Anbumani, S. P. (**1998**). Histopathological study of a natural outbreak of Peste des petits ruminants in goats of Tamilnadu. *Small Ruminant Research*, ***28***(**3**), 233–240. https://doi.org/10.1016/S0921-4488(97)00095-3

Asil, R. M., Ludlow, M., Ballal, A., Alsarraj, S., Ali, W. H., Mohamed, B. A., Mutwakil, S. M., and Osman, N. A. (**2019**). First detection and genetic characterization of peste des petits ruminants virus from dorcas gazelles "Gazella dorcas" in the Sudan, 2016-2017. *Archives of Virology*, ***164***(**10**), 2537–2543. https://doi.org/10.1007/s00705-019-04330-w

Atim, S. A., Ayebazibwe, C., Mwiine, F. N., Erume, J., and Tweyongyere, R. (**2016**). A Survey for Contagious Caprine Pleuropneumonia in Agago and Otuke Districts in Northern Uganda. *Open Journal of Veterinary Medicine*, ***6***(**1**), Article 1. https://doi.org/10.4236/ojvm.2016.61002

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 Animal Health and Production*, ***32***(**6**), 391–403. https://doi.org/10.1023/A:1005233703331

Awan, M. A., Abbas, F., Yasinzai, M., Nicholas, R. A. J., Babar, S., Ayling, R. D., Attique, M. A., Ahmed, Z., Wadood, A., and Khan, F. A. (**2010**). First report on the molecular

prevalence of Mycoplasma capricolum subspecies capripneumoniae (Mccp) in goats the cause of contagious caprine pleuropneumonia (CCPP) in Balochistan province of Pakistan. *Molecular Biology Reports*, ***37***(**7**), 3401–3406. https://doi.org/10.1007/s11033-009-9929-0

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***(**1–2**). https://doi.org/10.1016/j.virusres.2005.01.013

Balamurugan, V., Hemadri, D., Gajendragad, M. R., Singh, R. K., and Rahman, H. (**2014**a). Diagnosis and control of peste des petits ruminants: a comprehensive review. *VirusDisease*, ***25***(**1**), 39–56. https://doi.org/10.1007/s13337-013-0188-2

Balamurugan, V., Hemadri, D., Gajendragad, M. R., Singh, R. K., and Rahman, H. (**2014**b). Diagnosis and control of peste des petits ruminants: a comprehensive review. *Virus Disease*, ***25***(**1**), 39–56. https://doi.org/10.1007/s13337-013-0188-2

Balamurugan, V., Sen, A., Venkatesan, G., Yadav, V., Bhanot, V., Bhanuprakash, V., and Singh, R. K. (**2010**). Application of semi-quantitative M gene-based hydrolysis probe (TaqMan) real-time RT-PCR assay for the detection of peste des petits ruminants virus in the clinical samples for investigation into clinical prevalence of disease. *Transboundary and Emerging Diseases*, ***57***(**6**), 383–395. https://doi.org/10.1111/j.1865-1682.2010.01160.x

Balamurugan, V., Sen, A., Venkatesan, G., Yadav, V., Bhanot, V., Bhanuprakash, V., and Singh, R. K. (**2012**). A rapid and sensitive one step-SYBR green based semi quantitative real time RT-PCR for the detection of peste des petits ruminants virus in the clinical samples. *Virologica Sinica*, ***27***(**1**), 1–9. https://doi.org/10.1007/s12250-012-3219-z

Balamurugan, V., Sen, A., Venkatesan, G., Yadav, V., Bhanuprakash, V., and Singh, R. (**2010**). Isolation and identification of virulent peste des petits ruminants viruses from PPR outbreaks in India. *Tropical Animal Health and Production*, ***42***, 1043–1046.

Bamouh, Z., Fakri, F., Jazouli, M., Safini, N., Omari Tadlaoui, K., and Elharrak, M. (**2019**). Peste des petits ruminants pathogenesis on experimental infected goats by the Moroccan 2015 isolate. *BMC Veterinary Research*, ***15***(**1**), 452. https://doi.org/10.1186/s12917-019-2164-6

Bao, J., Li, L., Wang, Z., Barrett, T., Suo, L., Zhao, W., Liu, Y., Liu, C., and Li, J. (**2008**). Development of one-step real-time RT-PCR assay for detection and quantitation of peste des petits ruminants virus. *Journal of Virological Methods*, ***148***(**1–2**), 232–236. https://doi.org/10.1016/j.jviromet.2007.12.003

Bardhan, D., Kumar, S., Anandsekaran, G., Chaudhury, J. K., Meraj, M., Singh, R. K., Verma, M. R., Kumar, D., Kumar P T, N., Ahmed Lone, S., Mishra, V., Mohanty, B. S., Korade, N., and De, U. K. (**2017**). The economic impact of peste des petits ruminants in India. *Revue Scientifique Et Technique (International Office of Epizootics)*, ***36***(**1**), 245–263. https://doi.org/10.20506/rst.36.1.2626

Baron, J., Fishbourne, E., Couacy-Hyman, E., Abubakar, M., Jones, B. A., Frost, L., Herbert, R., Chibssa, T. R., Van't Klooster, G., Afzal, M., Ayebazibwe, C., Toye, P., Bashiruddin, J., and Baron, M. D. (**2014**). Development and testing of a field diagnostic assay for peste des petits ruminants virus. *Transboundary and Emerging Diseases*, ***61***(**5**), 390–396. https://doi.org/10.1111/tbed.12266

Baron, M. D., and Barrett, T. (**2000**). Rinderpest Viruses Lacking the C and V Proteins Show Specific Defects in Growth and Transcription of Viral RNAs. *Journal of Virology*, ***74***(**6**), 2603–2611.

Baron, M. D., Diallo, A., Lancelot, R., and Libeau, G. (**2016**). Peste des Petits Ruminants Virus. In M. Kielian, K. Maramorosch, and T. C. Mettenleiter (Eds.), *Advances in Virus Research* (Vol. 95, pp. 1–42). Academic Press. https://doi.org/10.1016/bs.aivir.2016.02.001

Bashiruddin, J. B. (**1998**). PCR and RFLP Methods for the Specific Detection and Identification of Mycoplasma mycoides subsp. mycoides SC. In R. Miles and R. Nicholas (Eds.), *Mycoplasma Protocols* (pp. 167–178). Humana Press. https://doi.org/10.1385/0-89603-525-5:167

Bellini, W. J., Englund, G., Rozenblatt, S., Arnheiter, H., and Richardson, C. D. (**1985**). Measles virus P gene codes for two proteins. *Journal of Virology*, ***53***(**3**), 908–919.

Bello, M. B., Kazeem, H. M., Oladele, S. B., Fatihu, M. Y., Tambuwal, F. M., and Jibril, A. H. (**2018**). Seroprevalence of peste des petits ruminants among unvaccinated small ruminants in Sokoto State, northwestern Nigeria. *Comparative Clinical Pathology*, ***27***(**5**), 1141–1146. https://doi.org/10.1007/s00580-018-2711-8

Bertin, C., Pau-Roblot, C., Courtois, J., Manso-Silván, L., Tardy, F., Poumarat, F., Citti, C., Sirand-Pugnet, P., Gaurivaud, P., and Thiaucourt, F. (**2015**). Highly dynamic genomic loci drive the synthesis of two types of capsular or secreted polysaccharides within the Mycoplasma mycoides cluster. *Applied and Environmental Microbiology*, ***81***(**2**), 676–687. https://doi.org/10.1128/AEM.02892-14

Blom, N., Gammeltoft, S., and Brunak, S. (**1999**). Sequence and structure-based prediction of eukaryotic protein phosphorylation sites. *Journal of Molecular Biology*, ***294***(**5**), 1351–1362. https://doi.org/10.1006/jmbi.1999.3310

Bölske, G., Johansson, K. E., Heinonen, R., Panvuga, P. A., and Twinamasiko, E. (**1995**). Contagious caprine pleuropneumonia in Uganda and isolation of Mycoplasma capricolum subspecies capripneumoniae from goats and sheep. *The Veterinary Record*, ***137***(**23**), 594.

Bölske, G., Mattsson, J. G., Bascuñana, C. R., Bergström, K., Wesonga, H., and Johansson, K. E. (**1996**). Diagnosis of contagious caprine pleuropneumonia by detection and identification of Mycoplasma capricolum subsp. capripneumoniae by PCR and restriction enzyme analysis. *Journal of Clinical Microbiology*, ***34***(**4**), 785–791. https://doi.org/10.1128/jcm.34.4.785-791.1996

Bonnet, F., Saillard, C., Bové, J. M., Leach, R. H., Rose, D. L., Cottew, G. S., and Tully, J. G. (**1993**). DNA relatedness between field isolates of Mycoplasma F38 group, the agent of contagious caprine pleuropneumonia, and strains of Mycoplasma capricolum. *International Journal of Systematic Bacteriology*, ***43***(**3**), 597–602. https://doi.org/10.1099/00207713-43-3-597

Boxer, E. L., Nanda, S. K., and Baron, M. D. (**2009**). The rinderpest virus non-structural C protein blocks the induction of type 1 interferon. *Virology*, ***385***(**1**), 134–142. https://doi.org/10.1016/j.virol.2008.11.022

Brown, C. C., and Torres, A. (**1994**). Distribution of antigen in cattle infected with rinderpest virus. *Veterinary Pathology*, ***31***(**2**). https://doi.org/10.1177/030098589403100206

Brüning-Richardson, A., Akerblom, L., Klingeborn, B., and Anderson, J. (**2011**). Improvement and development of rapid chromatographic strip-tests for the diagnosis of rinderpest and peste des petits ruminants viruses. *Journal of Virological Methods*, ***174***(**1–2**), 42–46. https://doi.org/10.1016/j.jviromet.2011.03.016

Bundza, A., Afshar, A., Dukes, T. W., Myers, D. J., Dulac, G. C., and Becker, S. A. (**1988**). Experimental peste des petits ruminants (goat plague) in goats and sheep. *Canadian Journal of Veterinary Research = Revue Canadienne De Recherche Veterinaire*, ***52***(**1**), 46–52.

Castelet. (**1906**). La pleuropneumonie de la chèvre en Algérie. *Prog Vét.*, ***27***, 324–329.

Catley, A., Alders, R. G., and Wood, J. L. (**2012**). Participatory epidemiology: approaches, methods, experiences. *The Veterinary Journal*, ***191***(**2**), 151–160.

Cattaneo, R., Kaelin, K., Baczko, K., and Billeter, M. A. (**1989**). Measles virus editing provides an additional cysteine-rich protein. *Cell*, ***56***(**5**), 759–764. https://doi.org/10.1016/0092-8674(89)90679-x

Chauhan, H. C., Chandel, B. S., Kher, H. N., and Dadawala, S. M. (**2009**). Pesti Des Petits ruminants virus infection in animals. *Veterinary World*, 150. https://doi.org/10.5455/vetworld.2009.150-155

Chota, A., Shirima, G., and Kusiluka, L. (**2019**). Risk factors associated with Mycoplasma capricolum subspecies capripneumoniae and morbillivirus infection in small ruminants in Tanzania. *Tropical Animal Health and Production*, ***51***(**7**), 1807–1815. https://doi.org/10.1007/s11250-019-01981-4

Chota, A., Shirima, G., and Kusiluka, L. (**2020**). Detection of Contagious Caprine Pleuropneumonia and Concurrent Diseases in Outbreaks Presenting with Respiratory

Signs in Small Ruminants in Tanzania. *International Journal of TROPICAL DISEASE and Health*, 70–83. https://doi.org/10.9734/ijtdh/2020/v41i730302

Coetzer, J., Thomson, G., and Tustin, R. (**1995**). Infectious diseases of livestock with special reference to Southern Africa. *Journal of the South African Veterinary Association*, ***66***(**2**), 106.

Cottew, G. S., Breard, A., DaMassa, A. J., Ernø, H., Leach, R. H., Lefevre, P. C., Rodwell, A. W., and Smith, G. R. (**1987**). Taxonomy of the Mycoplasma mycoides cluster. *Israel Journal of Medical Sciences*, ***23***(**6**), 632–635.

Couacy-Hymann, E., Bodjo, C., Danho, T., Libeau, G., and Diallo, A. (**2005**). Surveillance of wildlife as a tool for monitoring rinderpest and peste des petits ruminants in West Africa. *Revue Scientifique Et Technique (International Office of Epizootics)*, ***24***(**3**), 869–877.

Dadas, R., Chaudhary, D., Bhadouriya, S., Dhanavelu, M., K, R., Sudhakar, S. B., Venkatesan, G., and Pandey, A. B. (**2012**). *Development of N-gene based single step reverse transcription loop mediated isothermal amplification (RT-LAMP) for rapid detection of peste des petits ruminant virus (PPRV) in clinical samples*.

DaMassa, A. J., Wakenell, P. S., and Brooks, D. L. (**1992**). Mycoplasmas of goats and sheep. *Journal of Veterinary Diagnostic Investigation: Official Publication of the American Association of Veterinary Laboratory Diagnosticians, Inc*, ***4***(**1**), 101–113. https://doi.org/10.1177/104063879200400126

Darzi, M. M., Sood, N., Gupta, P. P., and Banga, H. S. (**1998**). The pathogenicity and pathogenesis of Mycoplasma capricolum subsp. capripneumoniae (F38) in the caprine mammary gland. *Veterinary Research Communications*, ***22***(**3**), 155–165. https://doi.org/10.1023/a:1006017120500

Das, S., Baron, M., and Barrett, T. (**2000**). Recovery and Characterization of a Chimeric Rinderpest Virus with the Glycoproteins of Peste-des-Petits-Ruminants Virus: Homologous F and H Proteins Are Required for Virus Viability. *Journal of Virology*, ***74***, 9039–9047. https://doi.org/10.1128/JVI.74.19.9039-9047.2000

Dhinakar Raj, G., Nachimuthu, K., and Mahalinga Nainar, A. (**2000**). A simplified objective method for quantification of peste des petits ruminants virus or neutralizing antibody. *Journal of Virological Methods*, ***89***(**1–2**), 89–95. https://doi.org/10.1016/s0166-0934(00)00206-8

Diallo, A. (**2006**). Control of Peste des Petits Ruminants and Poverty Alleviation? *Journal of Veterinary Medicine Series B*, ***53***(**s1**), 11–13. https://doi.org/10.1111/j.1439-0450.2006.01012.x

Diallo, A., Barrett, T., Lefevre, P. C., and Taylor, W. P. (**1987**). Comparison of proteins induced in cells infected with rinderpest and peste des petits ruminants viruses. *The Journal of General Virology*, ***68 (Pt 7)***, 2033–2038. https://doi.org/10.1099/0022-1317-68-7-2033

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***(**30**), 5591–5597. https://doi.org/10.1016/j.vaccine.2007.02.013

Dou, Y., Liang, Z., Prajapati, M., Zhang, R., Li, Y., and Zhang, Z. (**2020**). Expanding Diversity of Susceptible Hosts in Peste Des Petits Ruminants Virus Infection and Its Potential Mechanism Beyond. *Frontiers in Veterinary Science*, ***7***, 66. https://doi.org/10.3389/fvets.2020.00066

Dubie, T., Dagnew, B., Gelo, E., Negash, W., Hussein, F., and Woldehana, M. (**2022**). Seroprevalence and associated risk factors of peste des petits ruminants among ovine and caprine in selected districts of Afar region, Ethiopia. *BMC Veterinary Research*, ***18***(**1**), 429.

Dundon, W. G., Diallo, A., and Cattoli, G. (**2020**). Peste des petits ruminants in Africa: a review of currently available molecular epidemiological data, 2020. *Archives of Virology*, ***165***(**10**), 2147–2163. https://doi.org/10.1007/s00705-020-04732-1

Dundon, W. G., Kihu, S. M., Gitao, G. C., Bebora, L. C., John, N. M., Oyugi, J. O., Loitsch, A., and Diallo, A. (**2017**). Detection and Genome Analysis of a Lineage III Peste Des Petits Ruminants Virus in Kenya in 2011. *Transboundary and Emerging Diseases*, ***64***(**2**), 644–650. https://doi.org/10.1111/tbed.12374

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*, ***58***(**3**), Article 3. https://doi.org/10.4314/bahpa.v58i3.64206

Elsawalhy, A., Mariner, J., Chibeu, D., Wamwayi, H., Wakhusama, S., Olaho-Mukani, W., and Toye, P. (**2011**). Pan African Strategy for the Progressive Control of Peste des Petits Ruminants (Pan African PPR Strategy). *Bulletin of Animal Health and Production in Africa*, ***58***(**3**). https://doi.org/10.4314/bahpa.v58i3.64206

Enchery, F., Hamers, C., Kwiatek, O., Gaillardet, D., Montange, C., Brunel, H., Goutebroze, S., Philippe-Reversat, C., Libeau, G., and Hudelet, P. (**2019**). Development of a PPRV challenge model in goats and its use to assess the efficacy of a PPR vaccine. *Vaccine*, ***37***(**12**), 1667–1673.

Eye, P. (**2001**). Not the foot and mouth report. *London: Pressdram Ltd*.

Ezeibe, M. C. O., Okoroafor, O. N., Ngene, A. A., Eze, J. I., Eze, I. C., and Ugonabo, J. a. C. (**2008**). Persistent detection of peste de petits ruminants antigen in the faeces of recovered goats. *Tropical Animal Health and Production*, ***40***(**7**), 517–519. https://doi.org/10.1007/s11250-008-9128-3

Fakri, F. Z., Elhajjam, A., Bamouh, Z., Jazouli, M., Boumart, Z., Tadlaoui, K., Fassi-Fihri, O., and Elharrak, M. (**2017**). Susceptibility of Moroccan sheep and goat breeds to peste des petits ruminants virus. *Acta Veterinaria Scandinavica*, ***59***, 1–6.

FAO. (**2009**). *Peste des Petits Ruminants [PPR]. A challenge for small ruminant production*. ww.fao.org/docs/eims/upload/258701/ak065e00.pdf

FAO. (**2021**). *Kenya receives two million doses of PPR vaccine*. https://www.fao.org/kenya/news/detail-events/ar/c/1372928/

Fitzmaurice, J., Sewell, M., Manso-Silván, L., Thiaucourt, F., McDonald, W. L., and O'Keefe, J. S. (**2008**). Real-time polymerase chain reaction assays for the detection of members of the Mycoplasma mycoides cluster. *New Zealand Veterinary Journal*, ***56***(**1**), 40–47. https://doi.org/10.1080/00480169.2008.36803

Food and Agriculture Organization of the United Nations (FAO). (**2008**). *Peste des petits ruminants (PPR) in Morocco*. https://www.fao.org/3/aj120e/aj120e.pdf

Food and Agriculture Organization of the United Nations (FAO). (**2009**). *Trans-boundaryDisease* (Bulletin 33). ftp://ftp.fao.org/docrep/fao/011/i0919e/i0919e00.pdf.

Food and Agriculture Organization of the United Nations (FAO). (**2015**). *Global control and eradication of peste des petits ruminants* (Investing in Veterinary Systems, Food Security and Poverty Alleviation). https://www.fao.org/3/i4477e/i4477e.pdf

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***(**2–3**), 151–163. https://doi.org/10.1016/0168-1702(95)00076-3

Frumence, G., Mboera, L. E. G., Katale, B. Z., Sindato, C., Kimera, S., Durrance-Bagale, A., Mshana, S. E., Clark, T. G., Rweyemamu, M. M., Legido-Quigley, H., and Matee, M. (**2021**). Policy actors and human and animal health practitioners' perceptions of antimicrobial use and resistance in Tanzania: A qualitative study. *Journal of Global Antimicrobial Resistance*, ***25***, 40–47. https://doi.org/10.1016/j.jgar.2021.02.027

Gargadennec, L., and Lalanne, A. (**1942**). La Peste des Petits Ruminants. *Bull. Serv. Zoo. A. O. F.*, ***5***, 15–21.

Gibbs, E. P., Taylor, W. P., Lawman, M. J., and Bryant, J. (**1979**). Classification of peste des petits ruminants virus as the fourth member of the genus Morbillivirus. *Intervirology*, ***11***(**5**), 268–274. https://doi.org/10.1159/000149044

Gitao, G., D.G, I., R., G., and G.R, I. (**2014**). The confirmation of Peste des petit ruminants(PPR) in Kenya and perception of the disease in West Pokot. *Research Opinions in Animal and Veterinary Sciences*, ***4***, 312–317.

Gitonga, P., Gachene, C., Njoroge, E., and Thumbi, S. M. (**2016**). Small ruminant husbandry practices amongst Kajiado and Marsabit pastoralists and their effects on Peste des petits ruminants control strategies. *Livestock Research for Rural Development*, ***28***(**26**). http://www.lrrd.org/lrrd28/2/gito28026.htm

Hamdy, F. M., Dardiri, A. H., Nduaka, O., Breese, S. S., and Ihemelandu, E. C. (**1976**). Etiology of the stomatitis pneumoenteritis complex in Nigerian dwarf goats. *Canadian Journal of Comparative Medicine*, ***40***(**3**), 276–284.

He, Y., Zhang, N.-Z., ZHAO, P., Chu, Y., GAO, P., Jian-jun, Z., LIU, X., and Lu, Z. (**2014**). Sensitive and rapid detection of Mycoplasma capricolum subsp. capripneumoniae by Loop-mediated isothermal amplification. *African Journal of Biotechnology*, ***13***, 2113–2118. https://doi.org/10.5897/AJB12.2960

Herbert, R., Baron, J., Batten, C., Baron, M., and Taylor, G. (**2014**). Recombinant adenovirus expressing the haemagglutinin of peste des petits ruminants virus (PPRV) protects goats against challenge with pathogenic virus; a DIVA vaccine for PPR. *Veterinary Research*, ***45***(**1**), 24. https://doi.org/10.1186/1297-9716-45-24

Herrero, M., Grace, D., Njuki, J., Johnson, N., Enahoro, D., Silvestri, S., and Rufino, M. C. (**2013**). The roles of livestock in developing countries. *Animal*, ***7***(**s1**), 3–18. https://doi.org/10.1017/S1751731112001954

Herzog, C. M., de Glanville, W. A., Willett, B. J., Cattadori, I. M., Kapur, V., Hudson, P. J., Buza, J., Swai, E. S., Cleaveland, S., and Bjørnstad, O. N. (**2020**). Peste des petits ruminants Virus Transmission Scaling and Husbandry Practices That Contribute to Increased Transmission Risk: An Investigation among Sheep, Goats, and Cattle in Northern Tanzania. *Viruses*, ***12***(**9**), E930. https://doi.org/10.3390/v12090930

Hotzel, H., Sachse, K., and Pfutzner, H. (**1996**). A PCR scheme for differentiation of organisms belonging to the Mycoplasma mycoides cluster. *Veterinary Microbiology*, ***49***(**1–2**), 31–43. https://doi.org/10.1016/0378-1135(95)00176-x

Hussain, R., Auon, M., Khan, A., Khan, M. Z., Mahmood, F., and Ur-Rehman, S. (**2012**). Caprine pleuropneumonia in Beetal goats. *Tropical Animal Health and Production*, ***44***(**3**), 477–481. https://doi.org/10.1007/s11250-011-9922-1

Ismail, T. M., Yamanaka, M. K., Saliki, J. T., el-Kholy, A., Mebus, C., and Yilma, T. (**1995**). Cloning and expression of the nucleoprotein of peste des petits ruminants virus in baculovirus for use in serological diagnosis. *Virology*, ***208***(**2**), 776–778. https://doi.org/10.1006/viro.1995.1210

Johansen, M. V., and Penrith, M.-L. (**2009**). Has culling been properly assessed as a valid and justified control intervention measure for zoonotic diseases? *PLoS Neglected Tropical Diseases*, ***3***(**10**), e541.

Jones, B. A., Muhammed, A., Ali, E. T., Homewood, K. M., and Pfeiffer, D. U. (**2020**). Pastoralist knowledge of sheep and goat disease and implications for peste des petits ruminants virus control in the Afar Region of Ethiopia. *Preventive Veterinary Medicine*, ***174***, 104808.

Jones, G. E., and Wood, A. R. (**1988**a). Microbiological and serological studies on caprine pneumonias in Oman. *Research in Veterinary Science*, ***44***(**1**), 125–131.

Jones, G. E., and Wood, A. R. (**1988**b). Microbiological and serological studies on caprine pneumonias in Oman. *Research in Veterinary Science*, ***44***(**1**), 125–131. https://doi.org/10.1016/0034-5288(88)90022-7

Kardjadj, M. (**2017**). An epidemiological overview of small ruminant diseases in Algeria. *Revue Scientifique Et Technique (International Office of Epizootics)*, ***36***(**3**), 997–1006. https://doi.org/10.20506/rst.36.3.2731

Kgotlele, T., Chota, A., Chubwa, C. C., Nyasebwa, O., Lyimo, B., Torsson, E., Karimuribo, E., Kasanga, C. J., Wensman, J. J., Misinzo, G., Shirima, G., and Kusiluka, L. (**2018**). Detection of peste des petits ruminants and concurrent secondary diseases in sheep and goats in Ngorongoro district, Tanzania. *Comparative Clinical Pathology*, ***28***(**3**), 755–759. https://doi.org/10.1007/s00580-018-2848-5

Khalafalla, A. I., Saeed, I. K., Ali, Y. H., Abdurrahman, M. B., Kwiatek, O., Libeau, G., Obeida, A. A., and Abbas, Z. (**2010**). An outbreak of peste des petits ruminants (PPR) in camels in the Sudan. *Acta Tropica*, ***116***(**2**), 161–165. https://doi.org/10.1016/j.actatropica.2010.08.002

Khan, H. A., Siddique, M., Sajjad-ur-Rahman, null, Abubakar, M., and Ashraf, M. (**2008**). The detection of antibody against peste des petits ruminants virus in sheep, goats, cattle and buffaloes. *Tropical Animal Health and Production*, ***40***(**7**), 521–527. https://doi.org/10.1007/s11250-008-9129-2

Kihu, S. M., Gachohi, J. M., Ndungu, E. K., Gitao, G. C., Bebora, L. C., John, N. M., Wairire, G. G., Maingi, N., Wahome, R. G., and Ireri, R. (**2015**). Sero-epidemiology of Peste des petits ruminants virus infection in Turkana County, Kenya. *BMC Veterinary Research*, ***11***, 87. https://doi.org/10.1186/s12917-015-0401-1

Kihu, S. M., Gitao, G. C., Bebora, L. C., John, N. M., Wairire, G. G., Maingi, N., and Wahome, R. G. (**2015**). Economic losses associated with Peste des petits ruminants in Turkana County Kenya. *Pastoralism*, ***5***(**1**), 9. https://doi.org/10.1186/s13570-015-0029-6

Kihu, S. M., Njagi, L. M., Njogu, G. N., Kamande, J. N., and Gitao, C. G. (**2012**). Peste des petits ruminants in Kenya; Pastoralist knowledge of the disease in goats in Samburu and

Baringo Counties. *Research Opinions in Animal and Veterinary Sciences*, ***2***(**11**), 544–553.

Kipronoh, A. K., Ombui, J. N., Kiara, H. K., Binepal, Y. S., Gitonga, E., and Wesonga, H. O. (**2016**). Prevalence of contagious caprine pleuro-pneumonia in pastoral flocks of goats in the Rift Valley region of Kenya. *Tropical Animal Health and Production*, ***48***(**1**), 151–155. https://doi.org/10.1007/s11250-015-0934-0

KNBS. (**2019**). *Kenya Population and Housing Census 2009*. https://www.knbs.or.ke/publications/.

Komolafe, O. O., Ugwu, H. O., and Ebirim, R. A. (**1987**). Investigation into the possible role of peridomestic rats in the epizootiology of peste des petitis ruminants in goats. *Bulletin of Animal Health and Production in Africa*, ***35***(**3**), 207–2010.

Kul, O., Kabakci, N., Atmaca, H. T., and Ozkul, A. (**2007**). Natural peste des petits ruminants virus infection: novel pathologic findings resembling other morbillivirus infections. *Veterinary Pathology*, ***44***(**4**), 479–486. https://doi.org/10.1354/vp.44-4-479

Kumar, N., Chaubey, K. K., Chaudhary, K., Singh, S. V., Sharma, D. K., Gupta, V. K., Mishra, A. K., and Sharma, S. (**2013**). Isolation, identification and characterization of a Peste des Petits Ruminants virus from an outbreak in Nanakpur, India. *Journal of Virological Methods*, ***189***(**2**), 388–392. https://doi.org/10.1016/j.jviromet.2013.03.002

Kumar, N., Maherchandani, S., Kashyap, S. K., Singh, S. V., Sharma, S., Chaubey, K. K., and Ly, H. (**2014**). Peste des petits ruminants virus infection of small ruminants: a comprehensive review. *Viruses*, ***6***(**6**), 2287–2327. https://doi.org/10.3390/v6062287

Kumar, P. (**2001**). *Pathological studies on Peste des petits ruminants (PPR) in goats and sheep* [Dissertation]. Indian Veterinary Rsearch institute.

Kumar, S. C., Dhinakar Raj, G., Thangavelu, A., and Shaila, M. S. (**2007**). Performance of RT-PCR-ELISA for the detection of peste des petits ruminants virus. *Small Ruminant Research*, ***72***(**2**), 200–208. https://doi.org/10.1016/j.smallrumres.2006.09.004

Kwiatek, O., Keita, D., Gil, P., Fernández-Pinero, J., Jimenez Clavero, M. A., Albina, E., and Libeau, G. (**2010**). Quantitative one-step real-time RT-PCR for the fast detection of the four genotypes of PPRV. *Journal of Virological Methods*, ***165***(**2**), 168–177. https://doi.org/10.1016/j.jviromet.2010.01.014

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. *Journal of Comparative Pathology*, ***136***(**2–3**), 111–119. https://doi.org/10.1016/j.jcpa.2006.12.002

Kyotos, K. B., Oduma, J., Wahome, R. G., Kaluwa, C., Abdirahman, F. A., Opondoh, A., Mbobua, J. N., Muchibi, J., Bagnol, B., Stanley, M., Rosenbaum, M., and Amuguni, J. H. (**2022**). Gendered Barriers and Opportunities for Women Smallholder Farmers in the Contagious Caprine Pleuropneumonia Vaccine Value Chain in Kenya. *Animals: An Open Access Journal from MDPI*, ***12***(**8**), 1026. https://doi.org/10.3390/ani12081026

Lamb, R. A., and Kolakofsky, D. (**2001**). Paramyxoviridae: the viruses and their replication. In *Fields Virology* (Vol. 1, pp. 1305–1340). Lippincott Williams *and* Wilkins. https://doi.org/10.1016/bs.aivir.2016.02.001

Lamuka, P. O., Njeruh, F. M., Gitao, G. C., and Abey, K. A. (**2017**). Camel health management and pastoralists' knowledge and information on zoonoses and food safety risks in Isiolo County, Kenya. *Pastoralism*, ***7***, 1–10.

Leach, R. H., Costas, M., and Mitchelmore, D. L. (**1989**). Relationship between Mycoplasma mycoides subsp. mycoides ("large-colony" strains) and M. mycoides subsp. capri, as indicated by numerical analysis of one-dimensional SDS-PAGE protein patterns. *Journal of General Microbiology*, ***135***(**11**), 2993–3000. https://doi.org/10.1099/00221287-135-11-2993

Leach, R. H., Ernø, H., and MacOwan, K. J. (**1993**). Proposal for designation of F38-type caprine mycoplasmas as Mycoplasma capricolum subsp. capripneumoniae subsp. nov. and consequent obligatory relegation of strains currently classified as M. capricolum (Tully, Barile, Edward, Theodore, and Ernø 1974) to an additional new subspecies, M. capricolum subsp. capricolum subsp. nov. *International Journal of Systematic Bacteriology*, ***43***(**3**), 603–605. https://doi.org/10.1099/00207713-43-3-603

Lefèvre, P. C., and Diallo, A. (**1990**). Peste des petits ruminants. *Revue Scientifique et Technique (International Office of Epizootics)*, ***9***(**4**). https://doi.org/10.20506/rst.9.4.532

Lembo, T., Oura, C., Parida, S., Hoare, R., Frost, L., Fyumagwa, R., Kivaria, F., Chubwa, C., Kock, R., Cleaveland, S., and Batten, C. (**2013**). Peste des Petits Ruminants Infection among Cattle and Wildlife in Northern Tanzania. *Emerging Infectious Diseases*, ***19***(**12**), 2037. https://doi.org/10.3201/eid1912.130973

Li, L., Bao, J., Wu, X., Wang, Z., Wang, J., Gong, M., Liu, C., and Li, J. (**2010**). Rapid detection of peste des petits ruminants virus by a reverse transcription loop-mediated isothermal amplification assay. *Journal of Virological Methods*, ***170***(**1–2**), 37–41. https://doi.org/10.1016/j.jviromet.2010.08.016

Li, Y., Zhang, J.-H., Hu, S.-P., Wang, L., and Xin, J.-Q. (**2007**). Reclassification of the four China isolated strains of the pathogen for contagious caprine pleuropneumonia. *Wei Sheng Wu Xue Bao = Acta Microbiologica Sinica*, ***47***(**5**), 769–773.

Libeau, G., Diallo, A., Colas, F., and Guerre, L. (**1994**). Rapid differential diagnosis of rinderpest and peste des petits ruminants using an immunocapture ELISA. *The Veterinary Record*, ***134***(**12**), 300–304. https://doi.org/10.1136/vr.134.12.300

Libeau, G., Préhaud, C., Lancelot, R., Colas, F., Guerre, L., Bishop, D. H., 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*, ***58***(**1**), 50–55. https://doi.org/10.1016/0034-5288(95)90088-8

Lignereux, L., Chaber, A.-L., Saegerman, C., Manso-Silván, L., Peyraud, A., Apolloni, A., and Thiaucourt, F. (**2018**). Unexpected field observations and transmission dynamics of contagious caprine pleuropneumonia in a sand gazelle herd. *Preventive Veterinary Medicine*, ***157***, 70–77. https://doi.org/10.1016/j.prevetmed.2018.06.002

Liljander, A., Sacchini, F., Stoffel, M. H., Schieck, E., Stokar-Regenscheit, N., Labroussaa, F., Heller, M., Salt, J., Frey, J., Falquet, L., Goovaerts, D., and Jores, J. (**2019**). Reproduction of contagious caprine pleuropneumonia reveals the ability of convalescent sera to reduce hydrogen peroxide production in vitro. *Veterinary Research*, ***50***(**1**), 10. https://doi.org/10.1186/s13567-019-0628-0

Linjie, L., Xiaoling, S., Xiaoxia, M., Xin, C., Ali, A., and Jialin, B. (**2021**). Peste des petits ruminants virus non-structural C protein inhibits the induction of interferon-β by potentially interacting with MAVS and RIG-I. *Virus Genes*, ***57***(**1**), 60–71. https://doi.org/10.1007/s11262-020-01811-y

Lorenzon, S., Manso-Silván, L., and Thiaucourt, F. (**2008**). Specific real-time PCR assays for the detection and quantification of Mycoplasma mycoides subsp. mycoides SC and Mycoplasma capricolum subsp. capripneumoniae. *Molecular and Cellular Probes*, ***22***(**5–6**), 324–328. https://doi.org/10.1016/j.mcp.2008.07.003

Losos, G. (**1986**). *Infectious tropical diseases of domestic animals*. International Development Research Centre.

MacOwan, K. J. (**1976**). A Mycoplasma from chronic caprine pleuropneumonia in Kenya. *Tropical Animal Health and Production*, ***8***(**1**), 28–36. https://doi.org/10.1007/BF02383362

MacOwan, K. J., and Minette, J. E. (**1976**a). A mycoplasma from acute contagious caprine pleuropneumonia in Kenya. *Tropical Animal Health and Production*, ***8***(**2**), 91–95. https://doi.org/10.1007/BF02383376

MacOwan, K. J., and Minette, J. E. (**1976**b). A mycoplasma from acute contagious caprine pleuropneumonia in Kenya. *Tropical Animal Health and Production*, ***8***(**1**), 91–95. https://doi.org/10.1007/BF02383376

MacOwan, K., and Minette, J. E. (**1976**). A mycoplasma from acute contagious caprine pleuropneumonia in Kenya. *Tropical Animal Health and Production*, ***8***, 91–95.

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***(**1**), 82. https://doi.org/10.1186/1743-422X-10-82

Mahajan, S., Agrawal, R., Kumar, M., Mohan, A., and Pande, N. (**2012**). Risk of seroconversion to peste des petits ruminants (PPR) and its association with species, sex,

age and migration. *Small Ruminant Research*, ***104***(**1–3**), 195–200. https://doi.org/10.1016/j.smallrumres.2011.10.009

Mahapatra, M. (**2003**). Sequence analysis of the phosphoprotein gene of peste des petits ruminants (PPR) virus: editing of the gene transcript. *Virus Research*, ***96***(**1–2**), 85–98. https://doi.org/10.1016/S0168-1702(03)00176-X

Malik, Y. S., Singh, D., Chandrashekar, K. M., Shukla, S., Sharma, K., Vaid, N., and Chakravarti, S. (**2011**). Occurrence of Dual Infection of Peste-Des-Petits-Ruminants and Goatpox in Indigenous Goats of Central India. *Transboundary and Emerging Diseases*, ***58***(**3**), 268–273. https://doi.org/10.1111/j.1865-1682.2011.01201.x

Manirakiza, J., Moula, N., Detilleux, J., Hatungumukama, G., and Antoine-Moussiaux, N. (**2021**). Socioeconomic assessment of the relevance of a community-based goat breeding project in smallholding systems. *Animal*, ***15***(**1**), 100042. https://doi.org/10.1016/j.animal.2020.100042

Manso-Silván, L., Dupuy, V., Chu, Y., and Thiaucourt, F. (**2011**). Multi-locus sequence analysis of Mycoplasma capricolum subsp. capripneumoniae for the molecular epidemiology of contagious caprine pleuropneumonia. *Veterinary Research*, ***42***, 86. https://doi.org/10.1186/1297-9716-42-86

Maritim, N., Ngeiywa, M., Siamba, D., Mining, S., and Wesonga, H. (**2018**). Influence of Worm Infection on Immunopathological Effect of Mycoplasma Capricolum Capripneumoniae Pathogen in Goats. *Advances in Animal and Veterinary Sciences*, ***6***. https://doi.org/10.17582/journal.aavs/2018/6.6.234.241

Mbyuzi, A., Komba, E., Cordery-Cotter, R., Magwisha, H., Kimera, S., and Kambarage, D. (**2015**). Descriptive survey of Peste des Petits Ruminants and Contagious Caprine

Pleuropneumonia outbreaks in traditional goat flocks in Southern Tanzania: producers concerns, knowledge and attitudes. *Livestock Research for Rural Development*, ***27***(**4**), 71.

McMartin, D. A., MacOwan, K. J., and Swift, L. L. (**1980**). A century of classical contagious caprine pleuropneumonia: from original description to aetiology. *The British Veterinary Journal*, ***136***(**5**), 507–515. https://doi.org/10.1016/s0007-1935(17)32196-6

Mekonnen, M., Alemu, B., Desta, H., Wieland, B., and Knight-Jones, T. J. D. (**2021**). Integrated herd health approach to reduce impact of respiratory diseases. *SmaRT Ethiopia Brief*. https://cgspace.cgiar.org/handle/10568/117275

Mondal, B., Sreenivasa, B. P., Dhar, P., Singh, R. P., and Bandyopadhyay, S. K. (**2001**). Apoptosis induced by peste des petits ruminants virus in goat peripheral blood mononuclear cells. *Virus Research*, ***73***(**2**), 113–119. https://doi.org/10.1016/S0168-1702(00)00214-8

Morris, R. (**1999**). The application of economics in animal health programmes: a practical guide. *Revue Scientifique et Technique (International Office of Epizootics)*, ***18***(**2**), 305–314.

Muthomi, E. K., and Rurangirwa, F. R. (**1983**). Passive haemagglutination and complement fixation as diagnostic tests for contagious caprine pleuropneumonia caused by the F-38 strain of mycoplasma. *Research in Veterinary Science*, ***35***(**1**), 1–4.

Muthuchelvan, D. (**2015**). Peste-Des-Petits-Ruminants: An Indian Perspective. *Advances in Animal and Veterinary Sciences*, ***3***(**8**), 422–429. https://doi.org/10.14737/journal.aavs/2015/3.8.422.429

N. Kingiri, A. (**2021**). Agricultural advisory and extension service approaches and inclusion in reaching out to Kenyan rural farmers. *African Journal of Science, Technology, Innovation and Development*, ***13***(**7**), 797–806.

Notomi, T., Mori, Y., Tomita, N., and Kanda, H. (**2015**). Loop-mediated isothermal amplification (LAMP): principle, features, and future prospects. *Journal of Microbiology (Seoul, Korea)*, ***53***(**1**), 1–5. https://doi.org/10.1007/s12275-015-4656-9

Nyanja, P. M., Kusiluka, L. J. M., and Mellau, S. B. (**2013**). Prevalence of Contagious Caprine Pleuropneumonia in Goats in Musoma District of Mara Region, Tanzania. *Tanzania Veterinary Journal*, ***28***(**1**), Article 1. https://doi.org/10.4314/tvj.v28i1

Obi, T. U., Ojo, M. O., Durojaiye, O. A., Kasali, O. B., Akpavie, S., and Opasina, D. B. (**1983**). Peste des Petits Ruminants (PPR) in Goats in Nigeria: Clinical, Microbiological and Pathological Features. *Zentralblatt Für Veterinärmedizin Reihe B*, ***30***(**1–10**), 751–761. https://doi.org/10.1111/j.1439-0450.1983.tb01900.x

Ockleford, C., Adriaanse, P., Berny, P., Brock, T., Duquesne, S., Grilli, S., Hougaard, S., Klein, M., Kuhl, T., Laskowski, R., Machera, K., Pelkonen, O., Pieper, S., Smith, R., Stemmer, M., Sundh, I., Teodorovic, I., Tiktak, A., Topping, C. J., … Hernandez-Jerez, A. F. (**2017**). Scientific Opinion of the PPR Panel on the follow-up of the findings of the External Scientific Report 'Literature review of epidemiological studies linking exposure to pesticides and health effects.' *EFSA Journal*, ***15***(**10**). https://doi.org/10.2903/j.efsa.2017.5007

OIE. (**2014**). *World Organisation for Animal Health – contagious caprine pleuropneumonia* (In Manual of Diagnostic Tests and Vaccines for Terrestrial Animals).

OIE (World Organization for Animal Health). (**2009**). *Contagious caprine pleuropneumonia* (Terrestrial Animal Health Code, p. 690). https://scholar.google.com/scholar_lookup?title=Terrestrial+Animal+Health+Code*and*publication_year=2009*and*

OIE (World Organization for Animal Health). (**2013**). *Manual of diagnostic tests and vaccines for terrestrial animals.* (Chapter 2.7.11, pp. 1–4). Office International des Epizootie.

OIE (World Organization for Animal Health). (**2015**). *Global strategy for control and eradiction of Peste des Petits Ruminants* (Peste Des Petits Ruminants). Office International des Epizootie.

OIE (World Organization for Animal Health). (**2022**). *Chapter 14.3 Contagious Caprine Pleuropneumonia*. https://www.woah.org/fileadmin/Home/fr/Health_standards/tahm/3.08.09_PPR.pdf

Ojango, J. M. K., Audho, J., Oyieng, E., Recha, J., Okeyo, A. M., Kinyangi, J., and Muigai, A. W. T. (**2016**). System characteristics and management practices for small ruminant production in "Climate Smart Villages" of Kenya. *Animal Genetic Resources/Ressources Génétiques Animales/Recursos Genéticos Animales*, ***58***, 101–110. https://doi.org/10.1017/S2078633615000417

Ouya, F. O., Bett, E., Nguhiu, P., Makokha, S., and Mwirigi, M. K. (**2022**). Agro pastoralists' awareness and knowledge on contagious caprine Pleuropneumonia in two selected counties in Kenya. *Cogent Food and Agriculture*, ***8***(**1**), 2140472.

Özkul, A., Akca, Y., Alkan, F., Barrett, T., Karaoglu, T., Dagalp, S. B., Anderson, J., Yesilbag, K., Cokcaliskan, C., Gencay, A., and Burgu, I. (**2002**). Prevalence,

Distribution, and Host Range of Peste des petits ruminants virus, Turkey. *Emerging Infectious Diseases*, ***8***(**7**), 709–712. https://doi.org/10.3201/eid0807.010471

Paling, R. W., Waghela, S., Macowan, K. J., and Heath, B. R. (**1988**). The occurrence of infectious diseases in mixed farming of domesticated wild herbivores and livestock in Kenya. II. Bacterial diseases. *Journal of Wildlife Diseases*, ***24***(**2**), 308–316. https://doi.org/10.7589/0090-3558-24.2.308

Palmer, M. V. (**2013**). Mycobacterium bovis: characteristics of wildlife reservoir hosts. *Transboundary and Emerging Diseases*, ***60 Suppl 1***, 1–13. https://doi.org/10.1111/tbed.12115

Panel, E. (European F. S. A. (**2017**). Scientific Opinion of the PPR Panel on the follow-up of the findings of the External Scientific Report 'Literature review of epidemiological studies linking exposure to pesticides and health effects.' *EFSA Journal*, ***15***(**10**), e05007. https://doi.org/10.2903/j.efsa.2017.5007

Parida, S., Muniraju, M., Mahapatra, M., Muthuchelvan, D., Buczkowski, H., and Banyard, A. C. (**2015**). Peste des petits ruminants. *Veterinary Microbiology*, ***181***(**1–2**), 90–106. https://doi.org/10.1016/j.vetmic.2015.08.009

Parray, O. R., Yatoo, Mohd. I., Muheet, Ahmed Bhat, R., Ullah Malik, H., Bashir, S. T., and Nabi Magray, S. (**2019**). Seroepidemiology and risk factor analysis of contagious caprine pleuropneumonia in Himalayan Pashmina Goats. *Small Ruminant Research*, ***171***, 23–36. https://doi.org/10.1016/j.smallrumres.2018.12.004

Patterson, J. B., Thomas, D., Lewicki, H., Billeter, M. A., and Oldstone, M. B. A. (**2000**). V and C Proteins of Measles Virus Function as Virulence Factors in Vivo. *Virology*, ***267***(**1**), 80–89. https://doi.org/10.1006/viro.1999.0118

Penrith, M., and Thomson, G. (**2004**). Special factors affecting the control of livestock diseases in sub-Saharan Africa. *Infectious Diseases of Livestock*, ***1***, 171–177.

Perry, B. D., Randolph, T. F., McDermotT, J. . J., Sones, K. R., and Thornton, P. K. (**2002**). *Investing in Animal Health Research to Alleviate Poverty*. International Livestock Research Institute.

Pettersson, B., Bölske, G., Thiaucourt, F., Uhlén, M., and Johansson, K. E. (**1998**). Molecular evolution of Mycoplasma capricolum subsp. capripneumoniae strains, based on polymorphisms in the 16S rRNA genes. *Journal of Bacteriology*, ***180***(**9**), 2350–2358. https://doi.org/10.1128/JB.180.9.2350-2358.1998

Peyraud, A., Poumarat, F., Tardy, F., Manso-Silván, L., Hamroev, K., Tilloev, T., Amirbekov, M., Tounkara, K., Bodjo, C., Wesonga, H., Nkando, I. G., Jenberie, S., Yami, M., Cardinale, E., Meenowa, D., Jaumally, M. R., Yaqub, T., Shabbir, M. Z., Mukhtar, N., … Thiaucourt, F. (**2014**). An international collaborative study to determine the prevalence of contagious caprine pleuropneumonia by monoclonal antibody-based cELISA. *BMC Veterinary Research*, ***10***(**1**), 48. https://doi.org/10.1186/1746-6148-10-48

Pope, R. A., Parida, S., Bailey, D., Brownlie, J., Barrett, T., and Banyard, A. C. (**2013**). Early Events following Experimental Infection with Peste-Des-Petits Ruminants Virus Suggest Immune Cell Targeting. *PLoS ONE*, ***8***(**2**), e55830. https://doi.org/10.1371/journal.pone.0055830

Pourhoseingholi, M., Vahedi, M., and Rahimzadeh, M. (**2013**). Sample size calculation in medical studies. *Gastroenterology and Hepatology from Bed to Bench*, ***6***(**1**). https://pubmed.ncbi.nlm.nih.gov/24834239/

ProMed-Mail. (**2007**). *Peste des petits ruminants - Kenya (Rift Valley)*. OIE. 20070117.0226. http://www.oie.int/wahis_2/public/wahid.php/Reviewreport/Review?page_refer=MapEventSummary*and*reportid=4526.

Radostits, O. M., Gay, C. C., Constable, P. D., and Hinchcliff, K. W. (**2009**). *A textbook of the diseases of cattle, sheep, pigs, goats and horses*.

Renukaradhya, G. J., Isloor, S., and Rajasekhar, M. (**2002**). Epidemiology, zoonotic aspects, vaccination and control/eradication of brucellosis in India. *Veterinary Microbiology*, ***90***(**1–4**), 183–195. https://doi.org/10.1016/s0378-1135(02)00253-5

Roeder, P. L., Abraham, G., Kenfe, G., and Barrett, T. (**1994**). Peste des petits ruminants in Ethiopian goats. *Tropical Animal Health and Production*, ***26***(**2**), 69–73. https://doi.org/10.1007/BF02239901

Roeder, P. L., Obi, T. U., Taylor, W., and Diallo, A. (**1999**). Recognizing peste des petits ruminants. A field manual. FAO's Emergency System for Transboundary Animal and Plant Pests and Diseases (EMPRES). *FAO Animal Health Manual (FAO)*.

Roger, F., Yigezu, L., Hurard, C., Libeau, G., Mebratu, G. Y., Diallo, A., and Faye, B. (**2000**). Investigation of a new pathological condition of camels in Ethiopia. *Journal of Camel Practice and Research*, ***7***, 163–165.

Rojas, J. M., Moreno, H., Valcárcel, F., Peña, L., Sevilla, N., and Martín, V. (**2014**). Vaccination with Recombinant Adenoviruses Expressing the Peste des Petits Ruminants Virus F or H Proteins Overcomes Viral Immunosuppression and Induces Protective Immunity against PPRV Challenge in Sheep. *PLOS ONE*, ***9***(**7**), e101226. https://doi.org/10.1371/journal.pone.0101226

Rojas, J. M., Rodríguez-Martín, D., Avia, M., Martín, V., and Sevilla, N. (**2019**). Peste des Petits Ruminants Virus Fusion and Hemagglutinin Proteins Trigger Antibody-Dependent Cell-Mediated Cytotoxicity in Infected Cells. *Frontiers in Immunology*, ***9***, 3172. https://doi.org/10.3389/fimmu.2018.03172

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*, ***17***(**2**), 75–81. https://doi.org/10.1007/BF02360775

Rossiter, P., and Taylor, W. (**1994**). *Peste des petits ruminants. In infectious diseases of livestock, vol II*.

Ruhweza, S. P., Ayebazibwe, C., Mwiine, F., Muhanguzi, D., Mulindwa, B., and Olaho-Mukani, W. (**2010**). Seroprevalence of Peste des petits ruminants (PPR) virus antibodies in goats and sheep in North-Eastern Uganda. *Bulletin of Animal Health and Production in Africa*, ***58***, 141–146.

Rurangirwa, F. R., and McGuire, T. C. (**1996**). *Contagious caprine pleuropneumonia: Diagnosis and control*. 3. Biennial Conference of the African Small Ruminant Research Network on Small Ruminant Research and Development in Africa, Kampala (Uganda), 5-9 Dec 1994.

Rurangirwa, F. R., McGuire, T. C., Kibor, A., and Chema, S. (**1987**a). A latex agglutination test for field diagnosis of contagious caprine pleuropneumonia. *The Veterinary Record*, ***121***(**9**), 191–193. https://doi.org/10.1136/vr.121.9.191

Rurangirwa, F. R., McGuire, T. C., Kibor, A., and Chema, S. (**1987**b). An inactivated vaccine for contagious caprine pleuropneumonia. *The Veterinary Record*, ***121***(**17**), 397–400. https://doi.org/10.1136/vr.121.17.397

Sadique, U., Chaudhry, Z. I., Rana, M. Y., Anjum, A. A., Zahoor-Ul-Hassan, Sajid, A., and Mushtaq, M. (**2012**). Pathogenesis and Immunohistochemical Studies of Caprine Pleuropneumonia in Experimentally Infected Goats. *Pakistan Veterinary Journal*, ***32***(**3**), 427–431.

Salih, H., Elfadil, A., Saeed, I., and Ali, Y. (**2014**). Seroprevalence and risk factors of Peste des Petits Ruminants in sheep and goats in Sudan. *Journal of Advanced Veterinary and Animal Research*, ***1***. https://doi.org/10.5455/javar.2014.a12

Saliki. (**1998**). Peste des petits ruminants. In : US Animal Health Association, Committee on Foreign Animal Disease. In *The Gray Book.* (6th Part IV, pp. 344–352).

Saliki, J. T., Brown, C. C., House, J. A., and Dubovi, E. J. (**1994**). Differential Immunohistochemical Staining of Peste Des Petits Ruminants and Rinderpest Antigens in fOrmalin-Fixed, Paraffin-Embedded Tissues Using Monoclonal and Polyclonal Antibodies. *Journal of Veterinary Diagnostic Investigation*, ***6***(**1**), 96–98. https://doi.org/10.1177/104063879400600117

Samiullah, S. (**2013**). Contagious caprine pleuropneumonia and its current picture in Pakistan: a review. *Veterinární Medicína*, ***58***(**No. 8**), 389–398. https://doi.org/10.17221/6977-VETMED

Saravanan, P., Balamurugan, V., Sen, A., Sahay, B., and Singh, R. K. (**2006**). Development of Dot-ELISA for Diagnosis of Peste des petits ruminants (PPR) in Small Ruminants. *Journal of Applied Animal Research*, ***30***(**2**), 121–124. https://doi.org/10.1080/09712119.2006.9706600

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: Journal of the International Association of Biological Standardization*, ***38***(**4**), 479–485. https://doi.org/10.1016/j.biologicals.2010.02.003

Saravanan, P., Singh, R. P., Balamurugan, V., Dhar, P., Sreenivasa, B. P., Muthuchelvan, D., Sen, A., Aleyas, A. G., Singh, R. K., and Bandyopadhyay, S. K. (**2004**). Development of a N gene-based PCR-ELISA for detection of Peste-des-petits-ruminants virus in clinical samples. *Acta Virologica*, ***48***(**4**), 249–255.

Sarkar, J., Belaineh, G., Bp, S., and Singh, R. P. (**2012**). Development of a cell-elisa using anti-nucleocapsid protein monoclonal antibody for the titration of peste des petits ruminants (PPR) vaccine virus. *Indian Journal of Comparative Microbiology, Immunology and Infectious Diseases*, ***33***, 18–20.

Sarkar, J., Sreenivasa, B. P., Singh, R. P., Dhar, P., and Bandyopadhyay, S. K. (**2003**). Comparative efficacy of various chemical stabilizers on the thermostability of a live-attenuated peste des petits ruminants (PPR) vaccine. *Vaccine*, ***21***(**32**), 4728–4735. https://doi.org/10.1016/S0264-410X(03)00512-7

Sarker, S., and Islam, M. (**2011**). Prevalence and Risk Factor Assessment of Peste des petits ruminants in Goats in Rajshahi, Bangladesh. *Veterinary World*, 546. https://doi.org/10.5455/vetworld.2011.546-549

Sen, A., Saravanan, P., Balamurugan, V., Rajak, K. K., Sudhakar, S. B., Bhanuprakash, V., Parida, S., and Singh, R. K. (**2010**). Vaccines against peste des petits ruminants virus. *Expert Review of Vaccines*, ***9***(**7**), 785–796. https://doi.org/10.1586/erv.10.74

Settypalli, T. B. K., Lamien, C. E., Spergser, J., Lelenta, M., Wade, A., Gelaye, E., Loitsch, A., Minoungou, G., Thiaucourt, F., and Diallo, A. (**2016**). One-Step Multiplex RT-

qPCR Assay for the Detection of Peste des petits ruminants virus, Capripoxvirus, Pasteurella multocida and Mycoplasma capricolum subspecies (ssp.) capripneumoniae. *PloS One*, ***11***(**4**), e0153688. https://doi.org/10.1371/journal.pone.0153688

Shah, M., Sadique, U., Ahmad, S., Iqbal, A., Ali, A., Shahzad, W., Khan, M., Khan, H., Rahman, H., Ali shah, S., and Israr, M. (**2017**). Molecular Characterization of Local Isolates of Mycoplasma capricolum Sub Specie Capripneumoniae in Goats (Capra hircus) of Khyber Pakhtunkhwa, Pakistan. *Pakistan Veterinary Journal*, ***37***, 90–94.

Shaila, M. S., Purushothaman, V., Bhavasar, D., Venugopal, K., and Venkatesan, R. A. (**1989**). Peste des petits ruminants of sheep in India. *The Veterinary Record*, ***125***(**24**), 602.

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 viruses. *Virus Research*, ***43***(**2**), 149–153. https://doi.org/10.1016/0168-1702(96)01312-3

Sheikh, F. D., Wani, S. A., Fatima, K., and Masoodi, K. H. (**2016**). *Contagious caprine pleuropneumonia: a major threat to pashmina farming in Ladakh*.

Sherman, D. M. (**2011**). The spread of pathogens through trade in small ruminants and their products. *Revue Scientifique Et Technique (International Office of Epizootics)*, ***30***(**1**), 207–217. https://doi.org/10.20506/rst.30.1.2036

Shiferaw, G., Tariku, S., Ayelet, G., and Abebe, Z. (**2006**). Contagious caprine pleuropneumonia and Mannheimia haemolytica-associated acute respiratory disease of goats and sheep in Afar Region, Ethiopia. *Revue Scientifique Et Technique (International Office of Epizootics)*, ***25***(**3**), 1153–1163.

Shuaib, Y. A., El-Fadil, A. A. M., and Abdel-Rahman, M. E. (n.d.). *Knowledge and Perceptions of Sheep Owners and Herders on Peste Des Petits Ruminants in North Kordofan and Kassala States, the Sudan*.

Singh, R. K., Balamurugan, V., Sen, A., Saravanan, P., and Yadav, M. P. (**2009**). Possible control and eradication of peste des petits ruminants from India: technical aspects. *Vet Ital*, ***45***, 15.

Singh, R. P., Sreenivasa, B. P., Dhar, P., and Bandyopadhyay, S. K. (**2004b-11**). A sandwich-ELISA for the diagnosis of Peste des petits ruminants (PPR) infection in small ruminants using anti-nucleocapsid protein monoclonal antibody. *Archives of Virology*, ***149***(**11**), 2155–2170. https://doi.org/10.1007/s00705-004-0366-z

Singh, R. P., Sreenivasa, B. P., Dhar, P., Shah, L. C., and Bandyopadhyay, S. K. (**2004**). Development of a monoclonal antibody based competitive-ELISA for detection and titration of antibodies to peste des petits ruminants (PPR) virus. *Veterinary Microbiology*, ***98***(**1**), 3–15. https://doi.org/10.1016/j.vetmic.2003.07.007

Soayfane, Z., Houshaymi, B., and Nicholas, R. A. J. (**2018**). Mycoplasma capricolum subsp. capripneumoniae, the cause of contagious caprine pleuropneumonia, comprises two distinct biochemical groups. *Open Veterinary Journal*, ***8***(**4**), 393–400. https://doi.org/10.4314/ovj.v8i4.7

Sreenivasa, B. P., Singh, R. P., Mondal, B., Dhar, P., and Bandyopadhyay, S. K. (**2006**). Marmoset B95a Cells: A Sensitive System for Cultivation of Peste des Petits Ruminants (PPR) Virus. *Veterinary Research Communications*, ***30***(**1**), 103–108. https://doi.org/10.1007/s11259-005-3200-5

Tahir, M. T., Ahmad, R., Hussain, I., and Hussain, M. (**1998**). Counter-immunoelectrophoresis - A rapid technique for the diagnosis of Peste-des-petits ruminants. *Pakistan Veterinary Journal (Pakistan)*.

Taylor, W. P. (**1979**). Serological studies with the virus of peste des petits ruminants in Nigeria. *Research in Veterinary Science*, ***26***(**2**), 236–242. https://doi.org/10.1016/S0034-5288(18)32924-2

Taylor, W. P. (**1984**). The distribution and epidemiology of peste des petits ruminants. *Preventive Veterinary Medicine*, ***2***(**1–4**), 157–166. https://doi.org/10.1016/0167-5877(84)90059-X

Taylor, W. P., Al Busaidy, S., and Barrett, T. (**1990**). The epidemiology of peste des petits ruminants in the Sultanate of Oman. *Veterinary Microbiology*, ***22***(**4**), 341–352. https://doi.org/10.1016/0378-1135(90)90021-M

Thiaucourt, F., and Bölske, G. (**1996**). Contagious caprine pleuropneumonia and other pulmonary mycoplasmoses of sheep and goats. *Revue Scientifique Et Technique (International Office of Epizootics)*, ***15***(**4**), 1397–1414. https://doi.org/10.20506/rst.15.4.990

Thiaucourt, F., Bölske, G., Leneguersh, B., Smith, D., and Wesonga, H. (**1996**). Diagnosis and control of contagious caprine pleuropneumonia. *Revue Scientifique Et Technique (International Office of Epizootics)*, ***15***(**4**), 1415–1429. https://doi.org/10.20506/rst.15.4.989

Thomas, P. (**1873**). *Rapport médical sur le Bou-Frida: Vol. Publ. Gouv*. Général Civ.

Toma, B., Dufour, B., Sanaa, M., Benet, J., Moutou, F., Louza, A., and Ellis, P. (**1999**). *Applied veterinary epidemiology and the control of disease in populations.* Association pour l'Etude de l'Epidémiologie des Maladies Animales, 7 Avenue ….

Tripathi, B. N., Chattopadhyay, S. K., and Parihar, N. S. (**1996**a). *Clinical and pathological studies on peste des petits ruminants-an emerging disease in Indian goats* (Proceeding of International Conference on Goats, pp. 742–744).

Tripathi, B. N., Chattopadhyay, S. K., and Parihar, N. S. (**1996**b). Clinicopathological studies on an outbreak of peste des petits ruminants in goats and sheep. *Indian Journal of Veterinary Pathology*, ***20***(**2**), 99–103.

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**). https://doi.org/10.1371/journal.pone.0087145

Wambugu, A. N. (**2005**). *Development and evaluation of an improved assay for detecting specific antibodies post contagious caprine pleuropneumonia exposure* [PhD Thesis]. Kenyatta University.

Wang, H., Ni, L., Yang, H., Xu, L., Ma, N., and Ding, H. (**2014**). Isolation and identification of Mycoplasma mycoides cluster strains from goats in Chongqing, China. *Bulletin of the Veterinary Institute in Pulawy*, ***58***. https://doi.org/10.2478/bvip-2014-0002

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***(**2**), 299–301. https://doi.org/10.3201/eid1502.080817

Wesonga, H., Bölske, G., Thiaucourt, F., Wanjohi, C., and Lindberg, R. (**2004**). Experimental Contagious Caprine Pleuropneumonia: A Long Term Study on the Course of Infection and Pathology in a Flock of Goats Infected with Mycoplasma capricolum subsp. capripneumoniae. *Acta Veterinaria Scandinavica*, ***45***(**3**), 167. https://doi.org/10.1186/1751-0147-45-167

Wifag, A. (**2009**). Survey and Serological Investigations on Peste des Petits Ruminants (PPR) in the White Nile State, the Sudan. *MVSc Thesis, Faculty of Veterinary Medicine, University of Khartoum, the Sudan*.

Wohlsein, P., Trautwein, G., Harder, T. C., Liess, B., and Barrett, T. (**1993**). Viral Antigen Distribution in Organs of Cattle Experimentally Infected with Rinderpest Virus. *Veterinary Pathology*, ***30***(**6**), 544–554. https://doi.org/10.1177/030098589303000608

Woo, P. C. Y., Lau, S. K. P., Wong, B. H. L., Fan, R. Y. Y., Wong, A. Y. P., Zhang, A. J. X., Wu, Y., Choi, G. K. Y., Li, K. S. M., Hui, J., Wang, M., Zheng, B.-J., Chan, K. H., and Yuen, K.-Y. (**2012**). Feline morbillivirus, a previously undescribed paramyxovirus associated with tubulointerstitial nephritis in domestic cats. *Proceedings of the National Academy of Sciences of the United States of America*, ***109***(**14**), 5435–5440. https://doi.org/10.1073/pnas.1119972109

Woubit, S., Lorenzon, S., Peyraud, A., Manso-Silván, L., and Thiaucourt, F. (**2004**a). A specific PCR for the identification of Mycoplasma capricolum subsp. capripneumoniae, the causative agent of contagious caprine pleuropneumonia (CCPP). *Veterinary Microbiology*, ***104***(**1–2**), 125–132. https://doi.org/10.1016/j.vetmic.2004.08.006

Woubit, S., Lorenzon, S., Peyraud, A., Manso-Silván, L., and Thiaucourt, F. (**2004**b). A specific PCR for the identification of Mycoplasma capricolum subsp. capripneumoniae,

the causative agent of contagious caprine pleuropneumonia (CCPP). *Veterinary Microbiology*, ***104***(**1–2**), 125–132. https://doi.org/10.1016/j.vetmic.2004.08.006

Yatoo, I. M., Parray, R. O., Bashir, T. S., Muheet, Ahmed Bhat, R., Gopalakrishnan, A., Karthik, K., Dhama, K., and Vir Singh, S. (**2019**). Contagious caprine pleuropneumonia – a comprehensive review. *Veterinary Quarterly*, ***39***(**1**), 1–25. https://doi.org/10.1080/01652176.2019.1580826

Yatoo, M. I., Parray, O., Mir, M. S., Qureshi, S., Kashoo, Z., Nadeem, M., Fazili, M., Tufani, D. N., Kanwar, M., Chakraborty, S., Dhama, K., and Rana, R. (**2018**). Mycoplasmosis in Small Ruminants in India: A Review. *Journal of Experimental Biology and Agricultural Sciences*, ***6***, In Press. https://doi.org/10.18006/2018.6(2).264.281

Yiwen, C., Yueyue, W., Lianmei, Q., Cuiming, Z., and Xiaoxing, Y. (**2021**). Infection strategies of mycoplasmas: Unraveling the panoply of virulence factors. *Virulence*, ***12***(**1**), 788–817. https://doi.org/10.1080/21505594.2021.1889813

Yoneda, M., Bandyopadhyay, S. K., Shiotani, M., Fujita, K., Nuntaprasert, A., Miura, R., Baron, M. D., Barrett, T., and Kai, C. (**2002**). Rinderpest virus H protein: role in determining host range in rabbits. *The Journal of General Virology*, ***83***(**Pt 6**), 1457–1463. https://doi.org/10.1099/0022-1317-83-6-1457

Yu, Z., Wang, T., Sun, H., Xia, Z., Zhang, K., Chu, D., Xu, Y., Xin, Y., Xu, W., Cheng, K., Zheng, X., Huang, G., Zhao, Y., Yang, S., Gao, Y., and Xia, X. (**2013**). Contagious caprine pleuropneumonia in endangered Tibetan antelope, China, 2012. *Emerging Infectious Diseases*, ***19***(**12**), 2051–2053. https://doi.org/10.3201/eid1912.130067

Zhao, H., Njeumi, F., Parida, S., and Benfield, C. T. O. (**2021**). Progress towards Eradication of Peste des Petits Ruminants through Vaccination. *Viruses*, ***13***(**1**), 59. https://doi.org/10.3390/v13010059

APPENDICES

Appendix 6. 1: The questionnaire used in exploring the factors affecting control of PPR and CCPP in Kwale County

SECTION A: General data

1. Date: …… No.: …….
2. Name and contact of farmer:

   …….
3. Longitude…….Latitude…….
4. Sub-county and Ward: ……
5. Composition of herd:

   …….

   - Goat
   - sheep
   - cows

SECTION B: Knowledge and perceptions of PPR and CCPP

1. Which of the following do you believe is the main cause of diseases in this area?
   - external parasites ☐ internal parasites ☐infections☐ toxic plants ☐ nutrition ☐
   - other ☐ [specify]
2. Have you ever heard of the following diseases?
   - PPR: Yes ☐ No ☐
   - CCPP: Yes ☐ No ☐
3. Name any common symptom (S) for;
   - PPR

- CCPP

4. Have you vaccinated your goats against

   - PPR: Yes ☐ No ☐
   - CCPP: Yes ☐ No ☐

5. Have you ever lost animals to a disease? Yes ☐ No ☐

If yes, state the disease symptoms

6. On a scale of 0–10 (0 = no importance, 10 = highest importance), how do you rate the importance of diseases to sheep and goat farming in your area?

SECTION C: socioeconomic aspects of PPR-CCPP in Kwale County

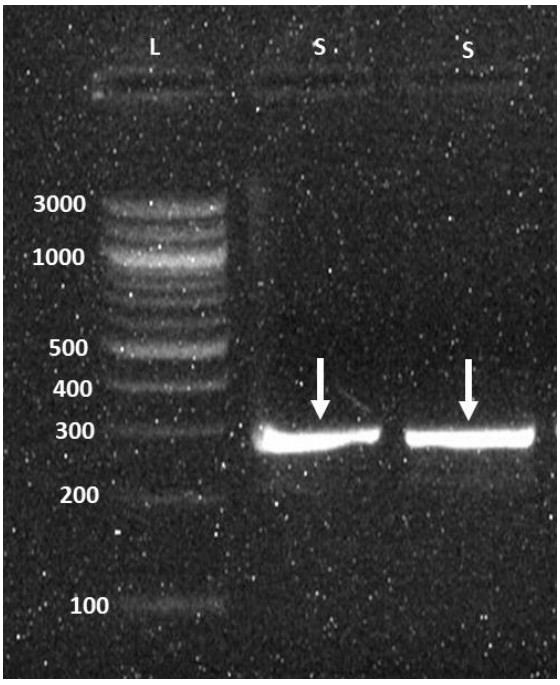
1. How many goats do you sell in a year?
2. How much, on average, does a mature goat sell at?
3. How do you feed your goats?

Zero grazing ☐ Free range (in personal farm) ☐ Tethering ☐ Herding ☐

4. Where do your goats sleep?

Outside ☐ All in one cage ☐ In several cages depending on age and other conditions ☐

Appendix 6. 2: Agarose gel showing the size of *Mycoplasma capricolum* Subsp. *capripneumoniae (Mccp)* polymerase chain reaction (PCR) products



L = 100 bp ladder (Invitrogen); S = PCR products for Mccp gene, 316 bp loaded in duplicate (white arrows).

Appendix 6. 3: Sampled LAT test results for (A) positive and (B) negative results