Peste Des Petits Ruminants: An Update

ABSTRACT

Peste des petits ruminants (PPR) is a notifiable and reportable disease recognised by the Office Internationale Epizooties (OIE) and penned for eradication by 2030. It is an endemic and devastating disease of small ruminants in some countries of Africa, Asia, and the Middle East. The disease militates against small ruminant production and increased flock numbers in the region. Information and literature on the current review was especially obtained and compiled from CABI abstract data base, PubMed, Scopus, Elibrary USA and other online publications. This review highlights the viral structure, epidemiology, predisposing factors, clinical signs, pathology, immunoprophylaxis, diagnostic techniques and therapeutics of the disease with a view to generating increased scientific research interests on PPR. It is hoped that more robust scientific research breakthroughs could emerge particularly in the area of immunoprophylaxis, rapid penside diagnosis, and alternative therapeutics such as the use of probiotics. This will in no small measure boost small ruminant production, alleviate poverty and increase food security in countries endemic with the disease.

Keywords:Peste des petits ruminants, PPRV, Pathogenicity, Pathology, Immunoprophylaxis and epidemiology

INTRODUCTION

Peste des petits ruminants (PPR) is primarily a viral, highly contagious disease of small domestic ruminants (sheep and goats). Small wild ruminants and camels have also been reported to be susceptible to this viral infection [1]. The successful eradication of Rinderpest (RPV) starting in the early 90's and culminating in 2011 through the Global Rinderpest Eradication Program (GREP) and surveillance led to the recognition of *Peste des petits ruminants* as a potential threat to small ruminant production.

The disease was first reported in Africa, and then spread through to the Middle East and Asia [2-7]. The highly contagious, debilitating nature of PPR and associated fatality in naïve small ruminant populations has been shown to exacerbate poverty levels amongst the poor farmers who often rear them, threaten food security and has negative socio-economic impact in endemic countries [8, 9]. For these reasons, the rapid and continuous spread of this disease to previously non-infected countries has resulted to PPR being classified as a notifiable terrestrial animal disease by the World Organization for Animal Health (WOAH-OIE).

PPR is managed symptomatically as there is no specific treatment for the disease. Currently, there have been a lot of advances and efforts made in the diagnosis and prevention of PPR. Vaccination remains a successful method of control of the disease [10]. However, recrudescence of the disease still occurs in endemic areas [11-15]. Therefore, there is the need to enhance and improve on the diagnosis, immunoprophylaxis, and eradication of PPR in Africa, the Middle East, Asia, and other areas necessitating the need for global awareness of this disease (PPR).

This paper is a current review on the viral structure, epidemiology, predisposing factors, pathology, pathogenicity,clinical signs, and molecular diagnosis inter-alia of PPR disease. The need for increased scientific research and interests in this disease may drastically improve small ruminant production and increased flock yield of these species in affected countries.

1.0 HISTORY AND BACKGROUND

As far back as 1871 (Senegal) and 1927 (Guinea Bissau), Rinderpest-like lesions were observed in small ruminants and diagnosed as Rinderpest [16] even though in the same regions in Africa, Rinderpest was also reported not to cause clinical disease in sheep and goats [17]. This contradiction is suggestive of the fact that PPRV was most likely to be in existence before it was first reported and documented.

In 1942, two veterinarians, Gargadennec and Lalanne described PPR, as a disease of small ruminants similar to RP but was not transmitted to cattle even when they were in close contact with infected animals in Ivory Coast (Africa) [18].In 1979, the aetiology of this disease (PPRV) was established to be uniquely different from RPV that causes Rinderpest [19]

PPRV is of one serotype with four lineages (lineage I – IV). The lineage type is determined by a small region of the F gene (322nt) [20], the N gene (255nt) [21] and the H gene (298nt) [22]. The most reliable gene for the determination of lineage type is the N-gene. This is because it exhibits a wider variance between lineages or between isolates within lineages compared to the F and H genes [22].The numbering of the lineages was thought to be based only on the origin and order of spread of the virus. For example, lineages I-III were from Africa. Specifically lineages I and II from West Africa and lineage III from East Africa, while the Middle East and Asia predominantly reported lineage IV.

The present increasing virulence in lineage IV[23] is evidenced by its spread through endemic countries/regions, replacing existent lineages and resulting in several reported outbreaks in different parts of Africa [14, 24-27], European parts of Turkey, China, Kazakstan[11], and Tibet in Asia [9].

Due to better application and understanding of molecular techniques further light has being shed on characteristics of the virus. For instance, Bayesian phylogeneticstudies established that lineage III is the original common ancestor for PPRV, and the first root location for PPRV was Nigeria [24]. Secondly the full viral genome sequence data analysis also revealed that RPV was more closely related to Measles virus (MV) than PPRV [28,29].

2.0 GEOGRAPHIC DISTRIBUTION

In recent years PPRV has extended its boundaries southwards in Africa as far as southern Tanzania (2008), Zambia (2015) and the Democratic Republic of Congo and Angola (2012). PPR outbreaks have also been reported across North Africa including Tunisia (2006), Morocco (2008 and 2015) and Algeria (2011 and 2016). Alongside this, within the European parts of Turkey approximately twelve laboratories confirmed PPR outbreaks in sheep and

goats as reported in 2012 [30]. The first occurrence of PPR has been reported in Georgia between January and March, 2016 following an outbreak in sheep [31,32]. In East Asia, the virus spread to Tibet (2007) and has recently been reported all over China (2013–2014) [8]. According to the OIE World Animal Health Information System (WAHIS), PPR in small ruminants was found (reporting period: 2012- June 2013) in the Middle East, North Africa, West Africa, South Africa, East Africa, and parts of Asia. Countries that have reported PPR to the OIE in 2012-2013 include Algeria, Angola, Comoros, Egypt, Tajikistan, and Tunisia; the Republic of the Congo, Kenya, Mali, and Uganda suggesting that PPR is an endemic disease. Other countries that the OIE identified as having "disease presence" in 2012-2013 include (but are not limited to) Afghanistan, Bahrain, Bangladesh, Benin, Bhutan, Burkina Faso, Cameroon, Central African Republic, Chad, Eritrea, Ethiopia, Ghana, Guinea, Buinea-Bissau, India, Iran, Iraq, Israel, Kenya, Kuwait, Nepal, Nigeria, Saudi Arabia, Sudan, Tanzania, Turkey and Yemen [1, 10, 33, 34].

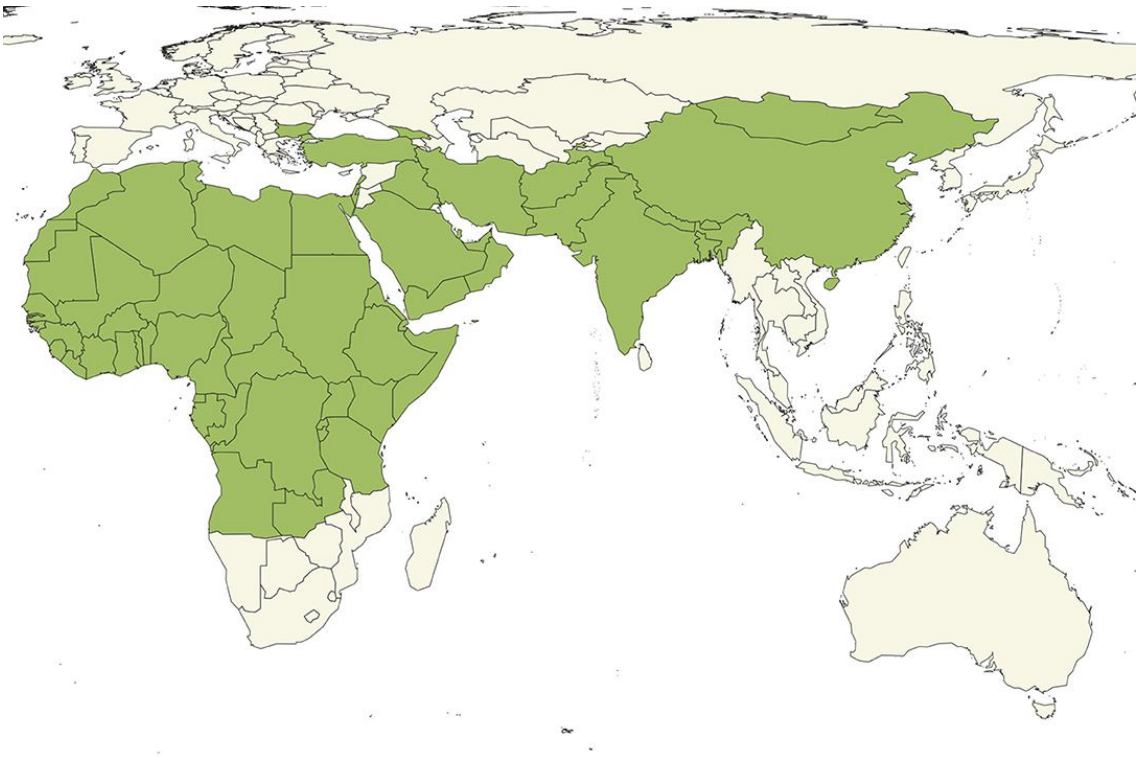


Fig 1: Coutries (green) reporting the presence of PPR to OIE as at April 2019. Adopted from http://www.oie.int/wahis_2/public/wahid.php/Wahidhome/Home (accessed April 19, 2019).

3.0 ECONOMIC IMPORTANCE

Small ruminant farmers have been identified as predominantly women and the youths [35-37]. Women keep goats for traditional milking and for family sustenance as small holding ventures. Livestock farming is carried out by 68% of households in developing coutries [38]. They are kept for various reasons; financial, social, cultural and nutritional benefits. Sheep and goats are kept and sold for cash to provide other needs for the family, and with larger herds, it enables for gainful employment opportunities. Socially, they are presented as gifts at weddings, slaughtered during festivities and nutritionally, the meat is a good source of animal protein while their milk is also good for young children, pregnant women, in the aged and also useful for the malnourished [39, 40].

Endemic regions of the disease are Africa, Asia and Middle East and they account for 80% of the global small ruminant population [39]. Africa and Asia also account for 72.9% of the world population living in poverty [41, 42]. The losses associated with this disease are multidimensional. This could be due to mortality, reduced production, poor quality of animal in the live market, culling and restrictions that disallow transboundary movements[8, 42-44].

In 1976, Nigeria was reported to have lost US$1.5 million to the disease [43]. Diallo also reported that the OIE was notified of over 750 million cases of sheep and goats infected with PPR. Kenya reported a loss of US$23.6 million from the death of 1.2 million small ruminants and a drop in milk production by 2.1million litres following an outbreak between 2005 and 2006 which raised poverty level by 10%. During the same period, Tanzania reported the culling of 64,661 small ruminants due to PPR [39]. In Cote d'Ivoire following an outbreak, animals were sold at half the market price as a result of the effect of this disease. In these countries, households affected recorded 28 – 68% decrease in income within the first two years following an outbreak of PPR. Between 2014 and 2015, India reported a loss of US$180 million due to PPR [39], suggesting colossal economic waste on affected Nations and family income.

4.0 AETIOLOGY AND STRUCTURAL BIOLOGY OF THE VIRUS

Peste des petits ruminants virus (PPRV) is classified in the genus Morbillivirus, subfamily Paramyxovirinae, family Paramyxoviridae and order Mononegavirales [19, 45, 46] genus Morbillivirus plays host to other important viral diseases of human and non-human primates (Measles virus- MV); canines (Canine distemper virus- CDV); wild and domestic cats (feline morbillivirus) [45] marine animals (Phocine distemper virus); marine animals and mammals (Phocine distemper virus & Cetacean morbilliviruses) [46, 47]; as well as morbilli-like viral diseases of rodents & bats[48, 49].

PPRV like CDV are non-specie specific, this differentiates them from all other members of this genus. For example, PPRV has been implicated in the mortality of camels [50-52] another un-corraborated incident was reported in a lion [53]. CDV on the other hand has been described in non primates, felids (tigers, hyenas and lions), and polar bears [54].

The genome of PPRV is a linear, non-segmented, single-stranded, negative sense, RNA molecule which is 15 948 nucleotides long[55]. The genome is said to conform to the 'rule of six' as it carries six transcriptional units/genes [56]. The six structural proteins encoded by the transcriptional units are: the nucleocapsid (N) protein, the matrix (M) protein, the polymerase or large (L) protein, the phosphoprotein (P), surfaceglycoproteins:haemagglutin-neuraminidase (HN) and fusion (F) protein. In addition to the six proteins, there are two non-structural proteins drived from the P open reading frame. They were detected using alternate start codons and RNA gene editing [57], namely the C and V proteins. Therefore the transcriptional unit order of the PPRV genome is 3'-N-P/C/V-M-F-HN-L-5' [55, 58].

The N protein is 525 amino acids in length, weighs 58kDa and it is in close proximity to the 3' location and is produced in abundance by morbilliviruses [57, 59]. This is characteristic of all morbilliviruses. It is made up of a conserved N structural core domain (NCORE) and a highly unstable C terminal domain (NTAIL) [60, 61].The N protein forms a helical nucleocapsid around the PPRV genome andit is resistant to RNase degradation [62] but whether this suggests any stability to the virus remains unknown.

The P protein of the PPRV is the longest with 509 amino acids compared to other morbilliviruses, it is the least conserved protein [63] and has a predicted size of 60kDa [64]. It is the least conserved protein and is heavily phosphorylated [65]. It uses alternate expression strategies to code for two non-structural proteins viz., V□ and C□ [59]. Phosphoprotein (P), the active form of P protein is a tetramer and it is a multi-functional protein which interacts with both the N and L protein as N-P and P-L complexes [66]. Hence the P protein helps regulate N protein self assembly, and by maintaining a soluble state, helps in the formation of the nucleocapsid structure[66].

Large (L) / polymerase protein (RdRp), is the largest PPRV protein (247 KDa) and is encoded by a gene (2,183 amino acids) located at the 5' proximal end of the genome. It is also the protein produced in the least amount in an infected cell as it is required in catalytic amounts [67]. The L gene accounts for 40% of the total genome length. The L protein though less significantly produced is required in polymerase enzymatic activity. Its combination with P protein is essential in the replication and transcription, capping and polyadenylation of viral mRNAs [67-69]. L protein sequences and their different functions are highly conserved

across the paramyxoviruses with clearly identified protein domains. There are three conserved domains with different and connected by variable hinge regions [70, 71]. The first N-terminal domain is involved in interactions with both the N and P proteins and serves as an RNA binding element.The second domain carries out phosphodiester bond formationduring polymerase activity (RdRp activity) and it is flanked by two hydrophobic regions [72]. Finally, the third domain has an ATP binding function and kinase activity [72].

The N, P and L proteins which form the ribunucleoprotein complex (RNP) are further protectedfrom degradation by the RNases. They consist of either positive antigenome strains or negative genome sense strains [30].

The Matrix (M) protein, lines the inner surface of the virus envelope [1]. It is 335 amino acids long, 38 kDa in molecular weightand the most conserved protein among PPRV isolates. It serves as an important link between the envelope glycoproteins and the RNP complex [60, 73]. Recombinant viruses lacking the M protein are unable to assemble and bud efficiently reiterating its role in these elements of the viral life cycle [74]. Recently, the co-expression of the PPRV M, H and N proteins in Sf9 insect cells has been reported to enable generation of PPRV virus-like particles [75].

The Fusion (F) protein is 456 amino acids in length and has a predicted molecular weight of 59.137kDa. It is a type I transmembrane glycoprotein that appear as spikes on the viral envelop.F proteins are synthesized on ribosomes of the rough endoplasmic reticulum (RER). Unlike other paramyxoviruses, PPRV requires only the F protein for virus-host cell fusion and infected cell-cell fusion [76]. They have also been shown to possess haemolytic factors [77] and induce autophagy [78].

Haemagglutinin (Hemagglutinin-Neuraminidase -HN) protein, consists of 609 amino acids, and has a predicted molecular weight of 67kDa [79]. Unlike other Morbilliviruses H proteins, the PPRV H displays both haemagglutination activity (agglutination of erythrocytes) and neuraminidase activity (cleaves sialic acid residues from the carbohydrate moieties of glycoproteins) [76, 80] and as such is often termed the HN protein. HN is a type II glycoprotein, because it posseses an outer Cterminaland an inward N-terminal transmembrane domain in relation to the vial membrane. The extent of glycosylation of H protein after its synthesis on ribosomes of the RER determines the virulence and antigenicity of the virus [72].

The non-structural V and C proteins are non-structural or accessory proteins produced from the P open reading frame (ORF) by alternative translational and transcriptional mechanisms. The C protein is unphosphorylated, located either within the cytoplasm and nucleus in

measles virus [81] or found restricted to the cytoplasm in Rinderpest virus [82]. It plays important roles in: blocking induction of interferon (Type I) [83] RNA synthesis [84] modulation of P protein activity [85], viral replication in peripheral blood cells [86] and determination of virulence of the virus [87]. Recently it has been reported that the C protein is immunogenic and can be targeted for the development of marker vaccines [88].

The V protein is phosphorylated unlike the C protein. It binds to the L and P proteins therefore it is reported to regulate RNA synthesis [82]. They are also implicated in the blockage of interferon signalling [89]. It is clearly obvious that the structural activity relationship inherent in this PPR virus has enabled it to maintain its house keeping agents and perpetuate pathogenicity, immune reactions and modulations.

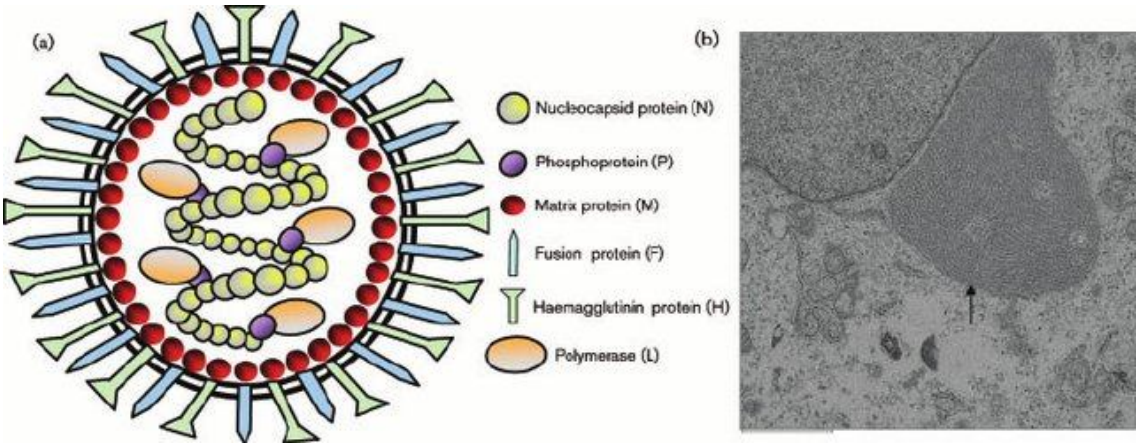


Fig. 2 (a) Schematic structure of Morbillivirus virion structure; (b) Electron micrograph of viral ribonucucleoprotein. Adopted from Banyard *et al.,* (2010).

5.0 EPIDEMIOLOGY OF PPR

There are various factors that predispose animals to PPRV. These factors include: species, age, sex, immune status, seasonal variations and geographical location.

5.1 Species

Small ruminants are more susceptible with higher incidence rates in goats than sheep [90-92]. The reason for this susceptibility is not fully understood, but genetic variations and the distribution of the host might play an important role. Pawar*et al*., (2008) suggested that varying levels of Signalling Lymphocyte Adhesion molecule (SLAM) of mRNA influences viral replication in different species [93]. In comparing the susceptibility of goats and buffaloes to PPRV, Dhanasekaran (2014) demonstrated that the level of Toll-Like Receptors

(TLR3, TLR7) and downstream signalling molecules correlated with the susceptibility of species [94].

5.2 Age

Younger animals are more at risk of the disease than adults [95-99]. This could be due to the fact that kids and lambs of about 5-6 months old lack immunity due to the loss of maternal antibodies at this age and poor nutrition [100-102].

5.3 Sex

Female animals have been widely reported to be more affected by PPRV than male animals. This may be due to the physiological factors associated with the female animal such as stress associated with infections arising from pregnancies and milk. Also because of the significant reproductive capacity of the female animals, they are maintained for a longer time compared to the male. Hence, they are exposed to the PPRV over time. [95, 103-111]. This however contradicts other reports that the male animals are more susceptible to PPRV than the female [64, 112, 113]. The likelihood of male goats sniffing of the female'sperineum during oestrus, exposes them to the virus especially from infected animals with diarrhea since the feces is known to be infected with virus [114, 115].Sarkar and Islam (2011) also argued that this could be due to genetic variation between males and females [102].

5.4 Management practices and Seasonal variation

These are diverse variables which influence the occurrence of livestock infectious diseases. Some of the important factors that enhance the transmission of PPRV from infected animals to susceptible ones include: biological factors (pathogen related), weather patterns, socio-cultural activities, and herd management practices [101, 103, 106, 116-120].

Areas with high rainfalls and high wind speed have been shown to aid the spread of the virus as; the rains cool the weather while the wind helps in disseminating the virus [95, 121-123]. Animals reared under the transhumance and normadic system are also more affected than animals reared under intensive management system [95, 124] because the unrestricted movement and gathering of animals especially due to seasonal and climatic changes which affect forage and water availability exposes susceptible herd to infected herds aiding the spread of the virus [90]. Also, during festive seasons, animals are moved across states for

meat purposes, given as gifts, or even serve as collateral for loans collection, hence further encouraging mixing of susceptible and infected animals [107].

5.5 Immune status

Introduction of infected animals to an immunologically naïve population, has resulted in very high morbidity and mortality rates (almost 100%), causing severe economic setbacks to livestock keepers [43]. Kids and lambs kidded/lambed by naïve dams are also at risk of PPRV if left to suckle from sick dams [43].

6.0 TRANSMISSION

PPRV is transmitted mainly via close contact between infected and susceptible animal populations [125, 126]. The disease is also transmitted through: contaminated fomites (e.g feeding and water troughs, water, and bedding) [5]. The principal port of entry is the respiratory route and the dissemination of the virus is primarily by inhalation. The virus is shed in the air [10, 125] and detected in the body secretions (ocular, nasal, saliva, urine, faeces and milk) of infected animals [127]. Although studies have also described the detection of PPRV antigen in clinically recovered goat (at about 1-3 months post recovery), the virus is not known to survive in the environment for long periods of time [5, 128]. Aerosol transmission over long distances has also not been reported. Transmission of PPR has also been associated with seasonal changes, with the disease occurring more frequently in the rainy or cold and dry seasons as well as during periods of increased local trade of goats [41, 101, 129].

The similarities between PPR and Rinderpest as well as the enveloped nature of the virus lends credence to the fact that the virus can be inactivated by: ultraviolet light, changes in pH (<4 or >11); destroyed by temperature (50°C/60 minutes), strong acids, alkaline solutions, alcohol, ether, common detergents and mostdisinfectants (e.g. phenol, sodium hydroxide 2%/24 hours); desiccation occurs within 3-4 days [130, 131]. The virus however is resistant to cold and can survive for a month at 2- 4□C in blood; and for several months in frozen or salted meat [131].

7.0 INCUBATION PERIOD

The incubation period of the disease varies. It can range from 2-10 days, 3-4 days [132, 133], with 2-6 days being typical (OIE). OIE Terrestrial Animal Health Code reported the incubation period as 21 days. The variation in the incubation period may be associated with the fact that higher doses of innoculum are used in experimental infection when compared to natural disease, therefore incubation takes longer in the latter [194]. Peste des petits ruminants has a high mortality rate (70–80 %), especially in naïve populations [43, 135] with most animals dying within the 10–12 days following the onset of disease. Mortality reaches 100% when PPR is associated with other diseases [113, 136, 137].

8.0 PATHOGENESIS

The pathology of a viral infection generally encompasses four distinct components of disease:
(i) aetiology,
(ii) mechanisms of development (pathogenesis),
(iii) structural alterations of cells (morphologic changes)
(iv) and the consequences of changes (clinical manifestations) [138].

Presently, the pathogenesis of PPRV is still poorly understood and is based on the closely related morbillivirus infections like Rinderpest virus [139, 140]. Also, very few studies have focused specifically on the pathogenesis of PPRV. PPRV like other morbilliviruses show affinity for epithelial (epitheliotropic) and lymphoid (lymphotropic) cells. Lymphoid and epithelial cell receptors like the signalling lymphocyte activation molecule (SLAM) and Nectin-4 are well-established receptor for morbillivirus infection [93,141-143]. The respiratory tract is the main portal of entry, the virus is picked by the antigen presenting cells (APCs) and transported to the oropharynx, local lymphnode (pharyngeal and mandibular), and tonsils where replication occurs [144-147]. The virus is amplified in the lymphnode. Viremia develops 2-3 days before the onset of clinical disease and is disseminated to other lymphoid tissues and organs (lungs, spleen, lymphnode, bone marrow, and mucosa of gastro-intestinal tract) via blood and lymphatics [145, 147-150]. The virus continues to replicate in the endothelial, epithelial and monocytic cells of these organs [150]. This secondary viremia is associated with the appearance of initial symptom of the disease (prodromal phase), while the pneumonic phase is associated with high viral load during the later stages of infection [149]. The replication, widespread destruction of lymphoid tissues (>25% circulating peripheral blood leucocytes) and induction of apoptosis (programmed cell death) of peripheral blood mononuclear cells by the virus is also responsible for the

immunosuppression associated with PPRV disease [149, 151, 152]. This immunosuppression predisposes the animal to secondary bacterial infection which is the cause of death associated with PPRV [153, 154]. Animals that recover from acute disease develop life-long immunity to re-infection [155], however goats that recover from the disease shed the virus in their feces for up to 12 weeks [115]. Pyrexia (39.5 to 41□C) is observed 2-7 days following incubation and clinical signs may vary, depending on other inherent or extraneous factors.

9.0 PATHOGENICITY, VIRULENCE AND/ EARLY VIRAL SHEDDING

A few experimental models which are reliable and reproducible have been developed to test the pathogenicity, virulence and replication of PPRV strains in the host cell [156]. These reports would also play a role in evaluating efficacy of vaccines [148, 149, 157-159]. The nucleic acids of PPRV may be detected before clinical observations in saliva and ocular discharges at 5 days post inoculation [149, 160], viral isolation can also be achieved from conjuctival swabs at 7dpi and convalescent goats continue to shed the virus in their feces for up to 12 weeks [115]. A reverse genetics approach recently developed may shed more light on interaction between different hosts and molecular pathways of the virus, replication of the virus and its pathogenesis [161, 162]. Pope *et al*. (2013) developed a clinical scoring system that enabled disease symptoms to be graded and scored in order to enable ethical euthanasia of animals during late stage of the disease [134, 149].

10.0 CLINICAL SIGNS

The severity of disease is dependent on many factors, namely; PPRV lineage, age of animal, breed [1], species, nutritional and immune status of the animal [90, 163]. Clinical signs appear in 3-6 days [10, 164, 165] following exposure to the virus. These signs are mostly observed in younger animals than adults [97-99]. However, the disease can be mild, inapparent (hence, may be missed or misdiagnosed), or obvious as observed in most outbreaks.

10.1 Sheep and goats

PPRV infection is more severe in goats than in sheep [90, 92, 166-168].

Peracute form

This is observed more in goats, especially naïve populations that have been newly exposed to the circulating virus. In this form, the clinical signs are generally limited to high fever (40□-

41□C), severe depression and mortality is higher. [169, 170]. Death occurs in infected animals within 4-5 days following a diarrhoeic phase [142].

Acute form

Initial signs include a sudden high fever (40□-41□C) which may last as long as 3-5 days. This is accompanied by inappetence, marked depression, dry muzzle, dull haircoat and somnolence [30]. Serous oculonasal discharges appear soon after the onset of clinical signs; these discharges later become mucopurulent (from secondary bacterial infections) causing hairs around the eyelids to become matted [30, 91, 113]. In the respiratory tract, this may progress to profuse cattarahal discharges which form crusts and occlude the nasal airways causing severe respiratory distress [5, 170]. Evidence of bronchopneumonia such as: Coughing, rales, rapid respiration and dyspnea are also common findings [171, 175].

The gums become hyperemic, and small, gray, necrotic foci, covering shallow erosions begin to appear in the mouth. If these lesions are difficult to find, rubbing a finger across the gums and palate may recover foul-smelling exudates and shreds of tissue [167, 172]. The oral lesions are painful, and animals may resist opening their mouths. Salivation is usually increased. In some cases, the mouth lesions resolve rapidly. In others, they enlarge, spread and coalesce. While lesions are most common on the lips and gums, they can also be found on the dental pad, palate, cheeks and their papillae, and tongue [30]. In severe cases, the mouth may be completely covered in thick, cheesy material. The lips are often swollen, cracked and crusted, and the breath of animals with severe stomatitis is fetid [171]. Necrotic lesions may also be detected on other mucous membranes, including those of the nasal cavity, vulva and vagina [20, 174].

Many animals also develop profuse diarrhea, which may be watery, fetid and/ or blood-stained, and sometimes contains shreds of tissue. Severely affected animals can become dehydrated and emaciated [20, 113, 174]. In addition, some animals may abort. [99, 168].

Subacute form

Incubation period is about 10-15 days. Disease manifests at about 6th day post infection, and is commonly seen in experimentally infected animals [158, 175]. It is also frequent in endemic areas where the local breeds are susceptibile. The signs are inconsistent and include: fever, serous ocular and nasal discharges. The onset of diarrhoea is associated with a decline in temperature. Severe and prolonged diarrhoea may result in dehydration, loss of electrolytes, and prostration[158, 171, 175].

In the late stages of the disease, small nodules resembling contagious ecthyma or sheep/goat pox can appear in the skin around the muzzle. The cause of these lesions is unknown [176] Deaths are usually the result of dehydration and/or pneumonia[172, 175]

11.0 BREED PREDISPOSITION AND CLINICAL MANIFESTATION TO PPR.

11.1 Cattle

Cattle are usually asymptomatic, and do not play any role in the persistence of the virus [105, 164, 177]. However, clinical signs have been reported in experimentally infected calves[178]. and it is possible that some cattle in poor condition might become symptomatic. If this occurs, the syndrome would probably resemble rinderpest. However, reports by Li *et al*., (2018; 2014) suggested that due to the social interaction between cattle, yaks and other domesticated ruminants, there is the risk of cross transmission of PPRV between species [103, 126].

11.2 Pigs

Although PPRV caused subclinical disease in experimentally inoculated pigs, and pigs exposed to infected goats, they did not exhibit clinical signs of the disease neither was an onward transmission of the virus to other species reported[179].

11.3 Camels

Respiratory disease was the predominant syndrome in PPRV-infected camels during one outbreak in Ethiopia[180]. Concurrent infections with PPRV and other respiratory pathogens were found in the lungs of apparently healthy camels sampled from abattoirs in Sudan [50].

An outbreak among camels in Sudan [181] was characterized by sudden death in some animals, and a more prolonged course in others. The most prominent clinical signs in the latter cases were yellowish diarrhea, which later became bloody, and abortions [182]. Other reported signs included, subcutaneous edema, submandibular swelling, “chest pain,” infrequent coughing, decreased milk production, weight loss and increased water consumption. Although all ages were affected, fatal cases were most common in animals that were pregnant or had recently given birth [50, 181].

In Kenya, clinical signs of sudden death, emaciation, general malaise, pneumonia, diarrhoea, oculonasal discharges and fever <40□C, has been reported. The clinical manifestation differs from those associated with goats and sheep hence was named "Camel sudden death syndrome" [180]. Keratoconjunctivitis and oedema of the ventral surface of the abdomen have also been reported [180].

In Iran, clinical signs of the affected camels included sudden death, fever, oral erosions, and ecthyma like lesions, dermatitis, ulcerative keratitis, conjunctivitis, enlarged lymphnodes, yellowish diarrhea, pneumonia and respiratory distress, severe dehydration [182].

Woma*et al*., (2016) reported only a transient infection of camels by PPRV in Nigeria [12].

11.4 Water buffalo

A highly fatal outbreak was reported in Indian buffaloes. Signs observed included: depression, profuse salivation and conjunctival congestion; however, the animals were afebrile [3]. Experimentally infected 3-5-month old water buffalo calves developed a fever but no other clinical signs, and died in 30- 35 days [183, 184]. Gastrointestinal lesions were found in these calves at necropsy [185, 186].

Wild ungulates

Clinical signs have been described in a few exotic species like the Tibetan antelope, bharal and gazelle [63, 187]. Deers can have signs similar to sheep and goats, but subclinical infections have also been reported. Captive gazelles became severely ill during one outbreak. The initial signs were anorexia and depression, followed by fever, lacrimation, congested mucous membranes, nasal discharges, erosions on the tongue, salivation and diarrhea[188, 189]. All affected animals died. Similar signs were reported in Ibexes [190]. Elevated respiratory rates, lacrimation, congested mucous membranes, ocular and nasal discharges, sneezing and ocular lesions (ulcerative keratitis and conjunctivitis) were documented in wild goats[191, 192]. Clinical descriptions of live animals were not available for one outbreak in the United Arab Emirates. However, necropsy findings indicated involvement of the lower gastrointestinal tract (e.g., catarrhal to hemorrhagic colitis) and lungs (congestion, subacute bronchointerstitial pneumonia with occasional suppurative or fibrinopurulent pneumonia). However, no lesions were found in the upper digestive and respiratory tracts, including the oral mucous membranes, during this outbreak. Lameness has been reported in some wild ruminants, but not definitively linked to PPR. [188]. The 2016 – 2017 introduction of *Peste*

des petits ruminant virus (PPRV) into livestock in Mongolia was followed by mass mortality of the critically endangered Mongalia saiga antelope and other wild ungulates [193].

The diverse occurrence of PPR infection across species may suggest some epidemiological significance in disease spread.

12.0 PATHOLOGY

12.1 Clinical pathology

Leukopenia characterised by marked lymphopenia is a frequently reported finding in goats infected with PPRV [158, 194-197]. However, Balogun *et al*., (2017) reported Leukocytosis, this he explained was probably due to regenerative changes after the acute phase of infection [200]. Leucocytosis characterised by neutrophilia and lymphopenia was also a consistent finding in sheep. This was attributed to the inflammation in tissues and lymphocytolysis caused by the virus [198]. Eosinopenia, monocytosis and a rise in the packed cell volume, presumably due to haemoconcentration resulting from the diarrhoea, have also been reported [199]. Microcytic, Hypochromic anemia observed in PPRV have been attributed to the necrotizing, debilitating (dehydration, emaciation) nature of disease [200-202]. Increased Alkaline Phosphatase (ALP) and Aspartate Aminotransferase (AST) are common findings confirming liver damage [202, 203] and there is an associated hypoglycaemia due to difficulty in glycogenolysis [167, 200, 204].

12.2 Gross Pathology

The gross lesions are commonly evident in digestive, respiratory and lymphoid tissues and has been well documented [99, 153, 175, 205-213]

The carcass may be emaciated and/or dehydrated with sunken eyes, but appear in good body condition in peracute cases [208]. Serous or mucopurulent oculonasal discharges, crusts around the eyes and nostril [209, 214]. The Perineum and hind quarters is soiled with liquid reddish, brownish, or greenish colored feces in diarrhoeic cases [99, 211, 212]. Distinct gross features in this disease are the erosive and necrotic lesions in the oral and gastrointestinal tract: crusted scabs on the lips, and necrotic stomatitis; erosions which are sharply demarcated from normal epithelium, may be found on the gums, hard palate, tongue, cheeks, sometimes in the pharynx and upper esophagus, vulva and vaginal mucous membranes of

some animals [153, 173, 175, 208]. Erosions and hemorrhages are common in the abomasum, but the rumen, reticulum and omasum are not significantly involved (although erosions are occasionally found on the pillars of the rumen) [205-208].

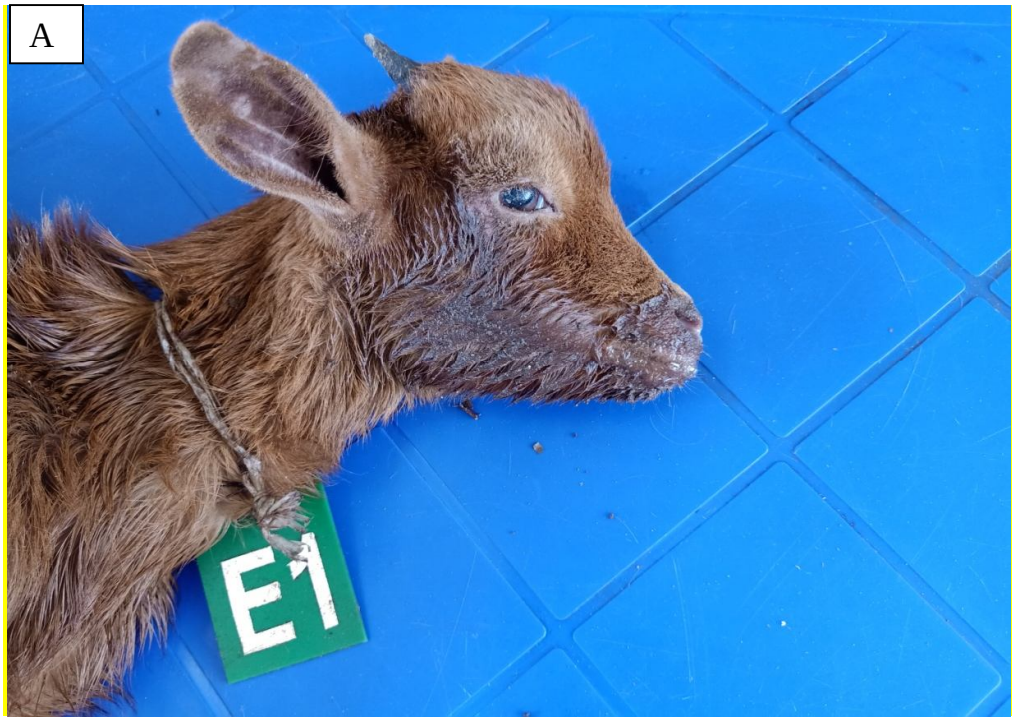
Hemorrhagic streaks and erosions may also occur in the duodenum and the terminal ileum, but other segments of the small intestine are generally spared [153, 173, 175, 209, 210, 214]. The Peyer's patches often have extensive necrosis, which can lead to ulceration. The large intestines are more significantly affected evidenced by marked congestion at the ileoceacal valve, ceaco-colic junction and the rectum. The alternating of the necrotic zone and congested zones are sometimes found in the posterior part of the colon on the mucosal folds giving the characteristic zebra stripes reported in this disease [205-208]. Intussusception has also been reported in natural PPR infections [215]. Respiratory lesions include: small erosions and petechiae in the nasal mucosa, turbinates, larynx and trachea; frothy exudation (which may be blood tinged) in the lumen of the trachea with congestion of tracheal mucosa; varying degrees of congestion, haemorrhages, emphysema, hepatisation and consolidation of the lungs (bronchopneumonia) [99, 153, 173, 175, 205, 206, 209-214]. Blood-tinged, frothy exudates have been reported in the tracheas of some experimentally infected goats [175]. The lymphnodes, are generally oedematous, enlarged, hemorrhagic and necrotic, especially the respiratory tract associated and mesenteric lymphnodes [215]. Splenic atrophy has also been reported in animals that died following a prolonged illness [208]. The liver and spleen may have hemorrhagic and necrotic lesions. In peracute cases, the lesions may be limited to congestion of the ileocecal valve and bronchopneumonia.

The most prominent lesions in an outbreak among camels in Sudan included pulmonary congestion and consolidation of primarily the apical lobes, gastrointestinal haemorrhages, gastritis and enteritis, lymphadenopathy, pale and friable liver [50]. Oral lesions (swelling of the lips and hemorrhagic ulcers on the tongue) were reported in one animal. Zakian *et al*., 2016, also reported keratoconjunctivitis, congestion and consolidation of the lung, paleness of the liver, and enlargement and edema of lymph nodes in camels that died in Iran [182]. The gross lesions in wild small ruminants and water buffalo are generally reported to be similar to those in sheep and goats [3]. However, hemorrhagic and edematous gastroenteritis was found to involve the abomasum and all segments of the intestines in infected water buffalo [3]. The presence of oral lesions might also be inconsistent in some wild species. Small erosions were found on the tongue of gazelles in one outbreak, and the oesophagus contained thick mucoid deposits along the walls [192]. However, oral lesions and erosive mucosal lesions were absent from the upper intestinal and respiratory tracts of affected ungulates during another

outbreak [192]. In addition, congestion has been reported in visceral organs such as the liver, kidney, pancreas, spleen and brain of wild ruminants [53].

12.3 Histopathology

The pleura, interlobular septae may reveal thickening with fibrin, this fibrin network may be infiltrated by mononuclear cells. The lung tissue show congested blood vessels, and in some cases congestion of the alveolar walls. The alveolar may also be dilated with evidence of emphysema. The bronchioles/bronchi lumen, alveolar lumen and interstitium show predominantly mononuclear cellular infiltration [198, 205, 206, 214, 216]. The bronchiolar epithelium may show evidence of necrosis (sloughing of epithelium into the lumen) and intraepithelial eosinophilic inclusions and proliferation of type II pneumocyte in the alveolar wall. There may also be formation of syncytia and giant cells [134, 151, 154, 171, 217, 218, 219, 220]. Lymphoid organs show depletion of lymphoid cells and necrosis [151, 154, 198, 205, 206, 220]. In the intestine, there is congestion of blood vessels, mononuclear infiltration of the lamina propria and submucosa as well as loss of intestinal villi [134, 198, 205, 216]. The mucosa and submucosa of the abomasum, urinary bladder and uterus also show congestion and inflammatory cellular infiltration (mononuclear and neutrophilic) [134]. In camels, degeneration and acute hyperemia of the lungs, hepatic fatty deneration, foci of hepatic nectrosis, tubular necrosis in the kidneys, and necrotic dermatitis were also reported [182].



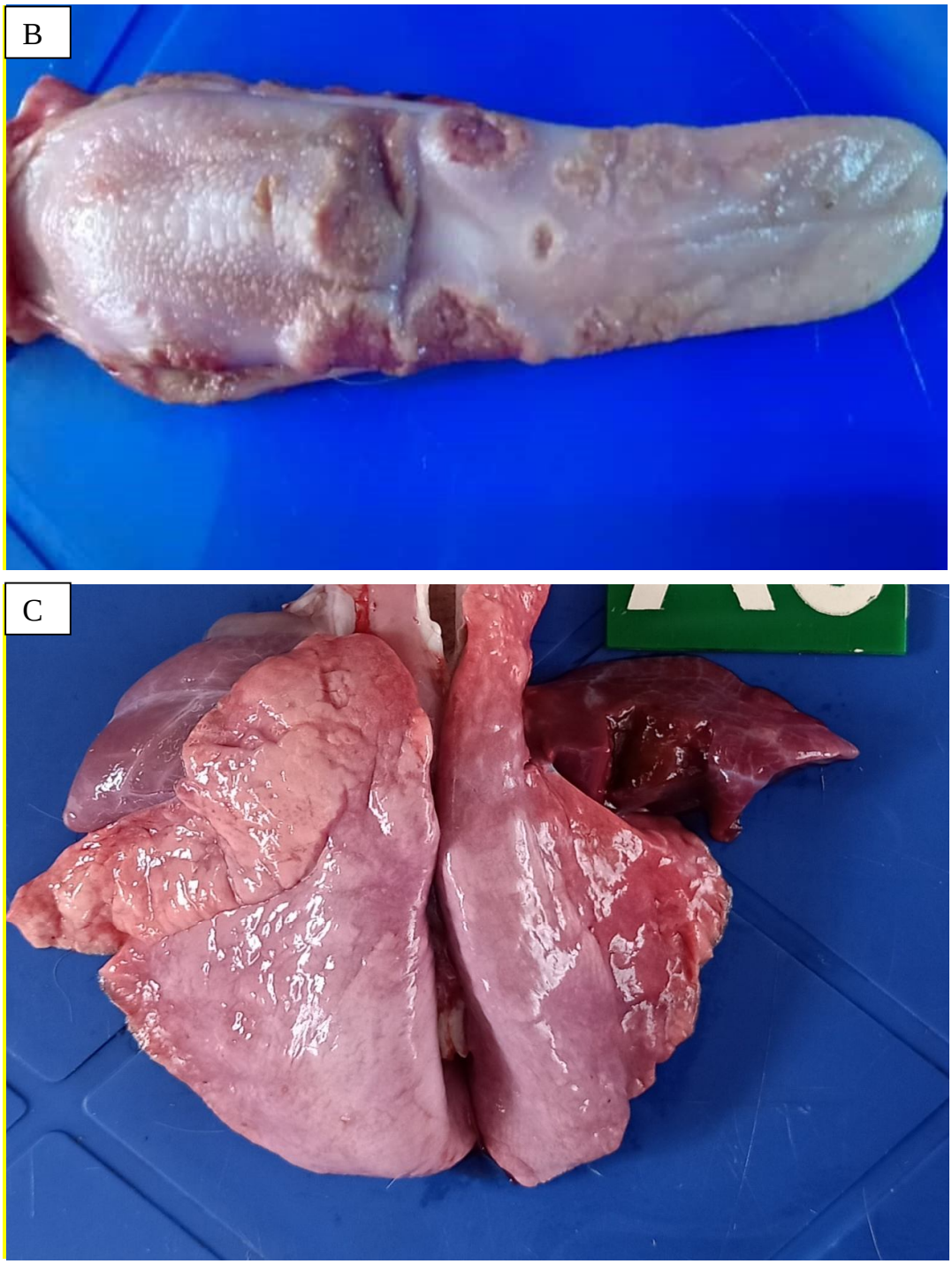


Fig 3: Clinicopathological observations of a goat infected with PPR virus during the present study.

A) Carcass of a goat showing serous nasal discharge

B) Dorsal aspect of the tongue showing ulcerations (black arrows) and erosions

C) Right cranial lobe of the lung showing red hepatisation.

13.0 IMMUNOLOGY& IMMUNITY

13.1 Immune Response

PPRV like other morbilliviruses induce immunosuppression in their hosts due to the lymphotropic nature of the virus [149, 197]. The innate immune responds to various pathogens through pathogen recognition receptors (PRRs). Examples of PRRs are, toll like receptors (TLRs), retinoic acid-inducible gene-I (RIG-I) like receptors (RLRs) such as RIG-I, melanoma differentiation antigen 5 (MDA-5) and Nod-like receptors (NLRs) [221]. These PRRs detect and interact with pathogen associated molecular pattern (PAMPs) [221-225]. This interaction leads to the induction and activation of complex signalling pathways and transcription of several cytokines [196, 226, 227]. Interferons (IFN) are one of the major groups of cytokines. Their role is to modulate the adaptive immune response. The binding of the virus to type I IFN α/β results in the establishment of a host antiviral state. The engagement of the viral PAMPs with the PRRs leads to the activation of a complex network of intracellular signaling pathways, which ultimately results in transcription of several cytokine genes to produce an antiviral state in the host [228, 229].

However, the morbillivirus through its V protein has been shown to block IFN type I, through its interaction with MDA-5, thereby inhibiting phosphorylation of STAT-1, STAT- 2, and interferon receptor kinase (Tyk. 2) [230, 231], but The V protein has varying abilities to block type II interferons [231]. Morbilliviruses also commonly target dendritic cells and monocytes/macrophages due to their lymphotropism. The DC expresses RIG-1 & MDA-5 (PRRs) which detect the virus and present them to the bronchial associated lymphoid tissue (BALT) where they are amplified and dissemination could lead to viremia [232]. The paramyxovirus C protein also inhibits the synthesis of mRNA and dsRNA during polymerase complex formation [233].

The adaptive immune response targets the viral proteins which are: Hemaglutinin (H), Fusion protein (F) and Nucleoprotein (N) [234-237], by interacting with L protein thereby interfering with host cell protein synthesis which is PKR mediated [238]. Although viral N protein is the most abundant of the viral proteins and is reported to induce strong cell-mediated immune response, anti-N antibodies does not protect against PPRV [234, 239, 240]. In one study [241], it was reported that blood collected from PPRV infected animals showed no changes in circulating WC1 + γ/δ T- cells and CD14+ monocytes/macrophages; a decrease in circulating CD4+ cells at day 4 post challenge (p.c), while CD8+ remained unchanged in both naïve and vaccinated animals. At day 7p.c however both naïve and vaccinated animals showed a mild

increase in CD8+ T-cells. This change was attributed to CTL responses by PPRV infection [242].

In vaccinated animals, antibodies are the most likely immune effectors that provide protective immunity [243, 244] but they are insufficient to act alone [219]. Anamnestic immune response following challenge with a live virus in cattle which had recovered from RPV after 8 years and was almost antibody negative has also proven important in protective role of the immune system [245].

Another important factor to note is the passive acquired immunity by kids from maternal antibodies, this starts to decline by the third month and protective titres are maintained through the 4^{th} month. Therefore immunization has been suggested to best be carried out after the 4^{th} month [244, 245].

14.0 DIAGNOSIS

Over the years, several methods have been employed to effectively diagnose this disease albeit with difficulties.

PPRV disease can be tentatively diagnosed using clinicopathological signs and lesions, however this method is unreliable because of its subjectivity as well as the fact that lesions/signs are similar to other viral diseases like Blue tongue, Foot and mouth disease, Contagious ecthyma, and Contagious caprine pleuropneumonia (CCPP), mixed infections of PPRV and goat pox, adenovirus or Pasteurellosis have also been reported [164, 246-250].

Conventional methods available to confirm the diagnosis of PPRV involves the detection of PPRV antigen in clinical (conjuctival smears, nasal swabs, tissue) and post mortem formalin fixed tissue samples using PPRV antibodies [251-253]. These tests/assays include: Viral neutralization test (VNT), Agar gel diffusion test (AGDT), Agarose gel immunodiffusion (AGID), Counter immune electrophoresis, Indirect Flourescent antibody technique (IFAT), Immunoperoxidase test (IPT), MAb, and viral isolation in cell culture. The major limitations to these methods include the need for a laboratory, expensive equipment/reagent, and expertise. These tests are generally time consuming, lack the required sensitivity and specificity in viral detection [114, 173, 255-256]. Although viral isolation remains the 'Gold standard' in PPRV detection [257)], it can only be undertaken in few OIE reference laboratories, it is time consuming, cumbersome and not as sensitive as the molecular techniques now available [258].

In seromonitoring or prevalence studies with large sample sizes, monoclonal and polyclonal antibody based Enyme-linked immunosorbent assay (ELISA) has been used as a simpler and rapid assay in serological profiling. Improvement on ELISA has been very successful with

current assays reported to be more sensitive and specific than previously developed ones. Competitive ELISA (c-ELISA) and blocking ELISA (B-ELISA) have been reported to have higher sensitivity and specificity compared to Viral Neutralization Test [119, 252, 259, 260, 261]. Polyclonal antibody based indirect ELISA showed even higher specificity and sensitivity to PPRV antibody detection than c-ELISA [119]. Immunocapture ELISA and Sandwich ELISA have also successfully detected PPRV antigens in clinical samples [119, 262].

More sensitive and specific nucleic acid based assays were developed following a better understanding of the viral genome and molecular biological techniques. Two sensitive Nucleic acid hybridization tests like Real time or gel based Reverse transcription – polymerase chain reaction (RT-PCR) [27, 143, 263, 264,265)and cDNA hybridization techniques have been used to diagnose PPRV infection. One advantage of RT-PCR is that diagnosis can be made from putrefied or semi putrefied tissue samples from the field. The limitation however, was that it was cumbersome and time consuming with large sample sizes [266, 267]. cDNA hybridization technique was also reported to directly differentiate PPRV from RP without viral isolation, however this method, had a short half life and required only fresh specimen and sample [267].

An improvement on PCR is the recent Loop mediated isothermal amplification (LAMP) [33, 50, 268] which is a simpler and rapid test in the diagnosis of PPRV. Its advantages are that it can be carried out in the field; highly sensitive and specific, requires less equipment, and can be conducted without Agarose gel analysis.

Furthermore, advancement in gene expression technology has led to the production of more efficient diagnostic assays using recombinant viral proteins [269, 270] advantages of this method includes; utilization of PPRV antigen which is safer compared to the live PPR antigen used in ELISA, can be produced in large quantities, the test may be rapid and PPRV antibodies can be detected in shorter period of time (30 minutes) after incubation [271], as well as possessing high specificity and sensitivity as VNT [270]. Restriction fragment length polymorphism method has also been used following RT-PCR; it has an added advantage of differentiating between PPRV lineages [272].

However, the need for penside tests cannot be over emphasised. These tests are suitable for field application, useful in screening large clinical samples, are rapid, highly sensitive and specific for PPRV, cheap, and user friendly. A few pen side test have been developed over the years, they include; Simple dot ELISA [273, 274], Lateral flow tests [157, 275], Dipsticks [164], Immunofiltration & antigen-competitive ELISA [276], Immunochromatographic test

[157] and SDS-PAGE [277, 278]. The recent penside diagnostic tool using lateral flow assay developed at the Pirbright institute (United Kingdom) has been proven to provide 84% sensitivity (279), hence indicating the need for more field evaluations in affected regions.

The limitation to these tests however are that; results produced may be subjective requiring other tests/equipment to confirm a false negative result (which increases cost), and sometimes results are unreliable [157].

15.0 PREVENTION AND CONTROL

15.1 Vaccines and Vaccination

For policies targeted at poverty alleviation/PPRV eradication to be effective, control of debilitating ruminant diseases like PPR is important. The most important method of controlling this disease is through vaccination. One of the earliest attempts at PPRV control was the use of formalized Rinderpest spleen to boost the production of antibodies against PPRV, but the results were generally inconclusive [280]. Lapinised Rinderpest vaccine (LRPV) did not prevent vaccinated animals from mortality even though some level of success was recorded [178]. Tissue culture Rinderpest virus vaccine (TCRPV) was used in small ruminants to control PPRV in Benin republic and Senegal for several years [281]. The heterologous vaccine proved successful, and was found to protect animals for up to one year [244]. Its success was attributed to the similarities in antigenic properties of the viruses (PPR & RPV). This led to the OIE recommending its usage in African countries and other affected countries around the world for prophylaxis [282]. The launch of Rinderpest eradication program targeted at a worldwide Rinderpest free status by the OIE led to the ban of the use of TCRPV [261].

This however informed a call for production of homologous vaccines aimed at protecting against PPRV. Several of these vaccines were produced and are still in existence. The first of its kind developed was done using the live attenuated PPRV 75/1 Nigerian strain isolated in Nigeria from the carcass of a PPRV infected goat [242]. The vaccine was considered safe as vaccinated animals did not come down with disease (even in pregnant animals), effective (protected 98% of vaccinated animals), provided solid immunity in small ruminants for a period of 3 years [267, 283] and was also reported to be protective against RP in cattle [284]. The thermo-stability of the vaccine was also reported as the freeze dried vaccine could withstand temperatures as high as 45□C without reducing/losing its potency. It was subsequently used in Africa, Middle East and Southern Asia. In 2016, the Nigerian 75/1 vaccine was also reported to be safe to use in camels and at an optimal dose of 10^2 TCID50;

high antibodies were detected for up to 6 months [285]. Concerns about this vaccine arose however due to the existence of different Lineages of PPRV in other affected countries in the world, there was the risk of introducing a new live virus to a country where it was previously nonexistent. Therefore other countries like India produced vaccines (CBE, 1997; Sungri, 1996; Arasur, 1987) using their own isolates of sheep and goat origin [286-289]. These vaccines were also reported safe, protected against other lineages of the virus as well, and provided adequate immunity in small ruminants; thermostable and mild lymphopenia associated with these vaccines was insufficient to cause immunosuppression [197, 239, 290].

Although, till date vaccination remains very relevant and is widely used in the control of PPRV, its limitations include: (i) the difficulty associated with differentiating between vaccinated from naturally infected animals in sero-epidemiological surveys due to similar antibody responses in both cases. (ii) in rural areas the need to maintain cold chain remains a challenge, due to poor electricity. This is risky as viral strains used in the vaccines are labile when exposed to high temperatures and there are no reports on the intrinsic thermostability of PPRV. To alleviate this challenge, live attenuated thermostable (thermo-adapted) PPR vaccines were produced. These vaccines have been proven successful in clinical trials and may be better suited for use in tropical countries [10, 291]. Other studies have attempted adapting the vaccine to high temperatures using stabilizers like Deuterium water (D2O) [292], Trehalose dehydrate [293] or by reducing the moisture content through freeze drying [294] and the ultra rapid method (Xerovac) [295].

A study conducted in 2013 using the Nigerian 75/1 PPR vaccine, showed that the intranasal route of vaccination produced a significant increase in the number of bronchial associated lymphoid tissues (BALT) as well as the number of lymphocytes in BALT (nodular) than subcutaneous vaccination which is the norm [217]. This finding substantiates the fact that intranasal vaccination induces protection efficiently and can be used in PPR endemic countries.

Recombinant vaccines also have been produced and are also considered successful. These vaccines make use of the F and H external glycoproteins of the virus in sub-unit vaccines. Examples include the F/H protein inserted into the genome of attenuated capripox vaccine conferred immunity at 0.1 and 10 plaque forming units respectively [92, 295]; an edible vaccine using viral HN gene inserted into Agrobacterium tumefaciens strain GV3 101; H & F genes of RPV which developed antibodies to only RPV (however completely protected against animals challenged with virulent strain of PPRV) [296, 297]. Other examples include,

capripox vaccine containing RPV [298, 299], and Recombinant adenovirus containing PPRV proteins [299]. An advantage to the recombinant vaccines is that recombinant marker vaccines for detection of infection in vaccinated animals (DIVA) can be produced to aid serosurveillance using the recombinant technology [300] by easing difficulties associated with differentiating natural infection in animals from vaccine-induced immunity [128]. Generally recombinant vaccines have proven successful and the combination of several vaccines for the control of different diseases like Orf, Blue tongue, Sheep and goat pox especially in endemic areas has also eased the cost of vaccination, reduced stress on vaccinated animals and saved time [164, 299, 301]. However, the booster doses required for sustaining level of immunity in recombinant vaccines contributes to increasing the cost associated with its use especially in large flocks.

Other conventional methods of control include: purchasing animals from recognised breeders instead of the market, short quarantine periods prior to stocking, better checks of transboundary animals and improving intensive systems and better flock management.

15.2 Treatment

PPRV like other viruses have no specific treatment. In 1989, lemon fruit and citrus aurantium were demonstrated to increase the recovery rates of PPR infected animals marginally [302]. Hyper immune immune serum and fluid therapy were also employed but only yielded positive results in the early stages of infection [135]. A study reported only 10% full recovery in a herd of goats after treatment [208]. Animals are managed symptomatically with the use of anti diarrhoeal drugs, antihistamines, astringent, antibiotics (Long acting Oxytetracyclin, Chlortetracycin) to prevent secondary bacterial infections that occur and are a common cause of mortality associated with PPRV infection [208, 303]. Fluid therapy (Dextrose saline) is also recommended to alleviate electrolyte losses following diarrhoeic phase of infection [113, 208, 303]. Vitamin B complex is also given as part of the supportive therapy [164]. Newer therapeutic alternatives such as the mucosal delivery of Probiotics should be investigated. The probiotic have been shown to activate dendritic cells thereby producing type 1 interferons required to eliminate viruses [304] and enhance the cellular immunity of the upper respiratory tract [305] and mucosal delivery of drugs has been reported to be protective against pathogens that interact directly with host mucosal surfaces [306-308].

The use of antibiotics in the treatment of a viral disease like PPR should however be discouraged due to the development of antimicrobial resistance which is likely to occur.

16.0 RECOMMENDATIONS

The OIE & FAO have inaugurated a joint strategic intervention plan to eradicate Peste des petits ruminants globally by the year 2030. Pending this period, the only option left to endemic countries to control the disease is through vaccination and restriction of livestock transboundary movement. Therefore, efforts to mitigate the effects of the virus should be geared towards:

i. Easier methods of detection of the virus with the use of pen-side tests that are readily available, sensitive, specific, affordable and possibly possess DIVA markers to appropriately distinguish infected from vaccinated animals and early antibody detection. Hence, scientists should strive and explore research on the structural viral genome so as to produce subunit or DNA vaccines that may be more specific in enhancing protective antibodies to PPR.

ii. The parenteral route of administration of PPR vaccine (which is the recommended practice) does not always elicit the specific response to the virus as expected due to interference from concurrent infections and/or maternal factors. Hence, a mucosal route of vaccine delivery could be explored as this has been shown to induce stronger respiratory mucosal responses when compared to the intramuscular route of vaccination. Its administration would also be easier and less technical compared to intramuscular injections during vaccine delivery.

iii. The intranasal use of probiotics which is currently being widely studied and tried in many diseases and animal models should also be explored in PPR prophylaxis as they have been demonstrated to possess immunomodulatory and other anti-inflammatory properties.

iv. Global attention should refocus on alleviating poverty on small holder small ruminant farmers especially women, through public enlightment on PPR, financial assistance or provision of aid agencies to poor African and other countries endemic with PPR.

17.0 CONCLUSION

Peste des petits ruminants is endemic in some countries of the world particularly Africa, Middle East and Asia. It is an emerging and re-emerging disease in some high risk areas. There is need to intensify vaccination programmes as well as fortify the food security system. Hence, this paper emphasizes the need for more research geared towards penside tests for rapid diagnosis and improvement in immunoprophylaxis by producing DIVA vaccines. Interactions between Government/Agencies and small holder peasant farmers especially the women would also shed light on barriers associated with access to information about PPR,

the need for annual vaccination, and ultimately the isolation of sick animals from apparently healthy ones. Government should build better equipped laboratories that will efficiently handle the diagnosis of disease. It is hoped that the current review would stimulate more research interest to meet the aforementioned goals.

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