Original Research

Epidemiological Insights into the Occurrence of Peste Des Petits Ruminants Virus (PPRV) Among Sheep, Goats and Cattle in Western Uganda

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Abstract

Peste des Petits Ruminants (PPR) is a virulent trans-boundary disease caused by morbillivirus of the Paramyxoviridae family widespread in tropical and sub-tropical countries, particularly in sub-Saharan Africa, Middle East and western and southern Asia. Serum samples were collected from cattle, goats and sheep from Kasese and Rubirizi districts, western Uganda and subjected to competitive ELISA. Prior to this sero-epidemiological study in 2016, PPRV was known to be limited to Karamoja pastoral area in North-eastern Uganda due to the large numbers of goats and sheep in this region hence the outbreaks; and cattle were never implicated. In this study sero-prevalences in goats (6.65%), Sheep (5.88%) and cattle (38.1%) were detected in South-western Uganda. This showed that PPR virus was distributed throughout Uganda, hence a need to create awareness amongst the farmers and veterinary stakeholders and scale out control measures country wide.

Key words: cELISA, PPR virus, Ruminants, Sero-prevalence, Uganda

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Introduction

Peste des petits ruminants virus (PPRV) is an acute, highly contagious, and economically important transboundary disease, first described in Côte d'Ivoire in West Africa in 1942. The disease is now distributed in tropical and subtropical countries including sub-Saharan Africa, Middle East and western, southern Asia and European part of Turkey (Dhar *et al*., 2002). The genus Morbillivirus, subfamily Paramyxovirinae of the family Paramyxoviridae (Gibbs *et al*., 1979); consists of closely related

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morbilliviruses such as Peste des petits ruminants virus (PPRV), measles virus (MV), canine distemper virus (CDV) and rinderpest virus (RPV) (Gibbs *et al*., 1979). PPR virions are pleomorphic particles and are enveloped. The non-segmented, negative-strand genome encodes nine proteins: the nucleocapsid protein (N), the phosphoprotein (P), the matrix protein (M), the fusion protein (F), the haemagglutinin protein (H), the large polymerase protein (L) (viral RNA-dependent RNA polymerase, RdRP) and the three non-structural proteins, P, C and V. The diameter of PPR virions ranges from 400 to 500 nm. The genome is 15,948 nt in length (Michael, 2011; Munir *et al*., 2013). As with other morbilliviruses, it is only the P gene that encodes for the two or three non-structural proteins.

PPR is primarily a disease of sheep and goats. However, the disease also infects wild small ruminants. Cattle, buffaloes, camels, and pigs are also susceptible to infection but do not exhibit clinical signs and are unable to transmit the disease to other animals (EMPRES 1999). PPR is not infectious to humans. The virus is enveloped hence easily destroyed by means of lipid solvents and is very delicate, particularly outside the host. PPRV has the potential of eliminating large populations of goats and sheep once introduced into an area maliciously, hence a bioterrorism threat.

Materials and Methods

The study was conducted in western Uganda in Kasese (Latitude: 0.17 N, Longitude: 30.08 E) and Rubirizi (Latitude: -0.26 S, Longitude: 30.11 E) districts. Study animals included goats, sheep and cattle (ruminants), wild ruminants were not accessible much as our field work was conducted within and around Queen Elizabeth National Park this is because wildlife conservation is sensitive to invasive studies. A total of 752 goats, 85 sheep and 341cattle were sampled. The animals were bled via the jugular vein or the middle coccygeal artery and vein; 2.5 ml blood was collected into plain vacutainer tubes. Serum was separated from the blood cells by centrifugation at 2500 rpm for 15 min and stored at -20°C until use in a competitive enzyme-linked immunosorbent assay (cELISA). Commercial cELISA kits were used to analyze the sera for the presence of antibodies to PPRV as described by the manufacturer (ID vet innovative diagnostics, France; Libeau *et al*., 1995). The cut-off values for positive infections was 50% of competition percentage S/N%. S/N% = OD sample/OD NC X 100. Less than or equal to 50% are considered positive. Greater than 50% and less than or equal to 60% are considered doubtful. Greater than 60% are considered negative. A qualitative assessment of the risk of the spread and introduction of Peste des Petits ruminants in Uganda was undertaken using a questionnaire survey. Detailed history and clinical features of the exposure and outbreaks were recorded. Sampling of livestock was done with verbal consent from the herd owners who were informed about the purpose of the project.

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Result and Discussion

The serological results revealed 6.65% (n=50), 5.88% (n=5) and 38.1% (n=130) were sero-positive. Details were as shown in Table 1. Adult animals were more likely to survive infection and become seropositive when compared to the young.

Table 1: Serological-survey of PPRV in South-western Uganda

Summary	Total Collected	No. Positive	%Positivity
Goats	752	50	6.65
Sheep	85	5	5.88
Cattle	341	130	38.1

Originally, knowledge of PPR virus endemicity in Uganda was limited to Karamoja sub-region, but when compared with the records of National Animal Disease Diagnostics and Epidemiology Centre (NADDEC) between 2006-2016 (Table 2) it was found that there were numerous PPRV cases from passive detection coming from various districts throughout the country. Outbreaks of PPR were reported in several districts between 2006-2016 as shown in Fig. 1.

Table 2: Passive cases of PPRV captured by NADDEC (2006-2016) from various districts

Napak	Kamuli	Kumi
Kotido	Kaabong	Ngora
Kween	Pader	Abim
Abim	kitgum	Moroto
Amudat	Lamwo	Nakapiripirit
Wakiso	Kween	Katakwi
Bukedea		

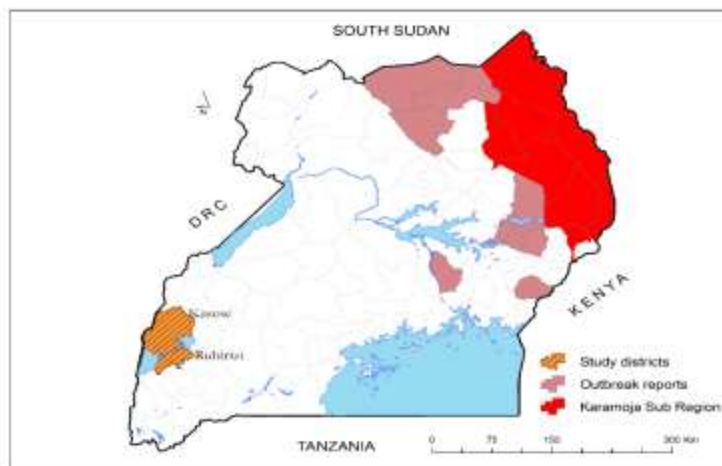

Fig. 1: Recorded PPRV outbreaks in several districts of Uganda; (MAAIF 2006-2016)

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Knowledge of PPRV endemicity in Uganda was limited to Karamoja sub-region possibly because this is a pastoral region with large number of sheep and goats hence large outbreaks of PPR virus. Hence PPRV control efforts in Uganda were limited to Karamoja sub-region. This study disapproved that and found PPRV in Kasese and Rubirizi districts in South-western Uganda (Table 1). Further screening of records from Ministry of Agriculture Animal Industry and Fisheries MAAIF and National Animal Disease Diagnostics and Epidemiology Centre (NADDEC) revealed passive surveillance of positive cases of PPRV by Veterinarians who brought samples to NADDEC for testing (Table 2). The positive cases captured by NADDEC from different parts of Uganda were as shown in Fig. 1. Large livestock density is also found in pastoral areas of western, southern and central Uganda known as the cattle corridor.

Previously, not much emphasis was put on PPRV in cattle; but this study has revealed a high serological-prevalence in cattle from South-western Uganda. The serological-prevalence in cattle was higher than that in small ruminants (Table 1). An outbreak of PPRV was reported in camels in Sudan in 2004 (Khalafalla *et al*., 2010). Serological evidence of PPRV exposure (2.6%) has been demonstrated from seemingly clinically healthy camels in Tanzania in 2010 (Swai *et al*., 2011). Other African countries, like Ethiopia and Nigeria have also reported antibodies to PPRV in camels (Abraham *et al*. 2005; Kgotlele *et al*., 2014). Sheep and goats are the natural hosts of PPR, with goats being the more susceptible of the two (Hussain *et al*., 2003; Abubakar *et al*., 2008; Aziz-ul-Rahman *et al*., 2016), however, cattle also develop subclinical infection and seroconvert. A study of wildlife in the Serengeti Ecosystem in Tanzania, have also been found to seroconvert (Mahapatra *et al*., 2015).

Complete genome sequences of the fusion (F), nucleocapsid (N) and hemagglutinin (H) genes of four PPR viruses indicated that lineage III is endemic in Uganda. However, further research needs to be conducted to establish whether PPR virus belonging to any of the other three lineages (I, II and IV) does not occur in the country. Molecular evolutionary studies and emergence of PPR virus in Uganda, and the lineages of PPR virus circulating in other domestic and wild small and large ruminants need to be conducted (Muniraju *et al*., 2014). Uganda borders with Kenya in the East, Tanzania in the South and Sudan in the North. Lineage III has been characterized in Sudan (2000), Lineage IV viruses have also been isolated from the Sudan in 2000, 2004, 2008 and 2009 (Khalafalla *et al*., 2010). Clearly both lineages III and IV are circulating in the Sudan. An outbreak in Kenya in 2006 in the Turkana district where it rapidly spread to 16 districts, Uganda (2007) and most recently in Tanzania (2008 and 2010). On the other hand, Uganda borders which Democratic Republic of Congo DRC in the West, DRC harbors lineage III and IV. Phylogenetic analysis has shown that lineage IV viruses are circulating across Central Africa. Therefore, there is a need to further characterize the lineages circulating in Uganda. PPRV exists in one serotype separated into four lineages (I-IV) based on the genetic comparison of a fragment of the nucleoprotein or the fusion protein (Banyard *et al*., 2010). Lineage III is the lineage most commonly found in eastern Africa, Arabian and Southern India

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whereas lineage I and II are most commonly found in western and Central Africa (Banyard *et al*., 2010). Lineage IV was historically regarded as an Asian lineage, in the Middle East and Asia subcontinent but since the 1990s it has spread in northern and eastern Africa replacing the other lineages (Kwiatek *et al*., 2011; Misinzo *et al*., 2015; Khalafalla *et al*., 2010; Cosseddu *et al*., 2013; Maganga *et al*., 2013). In West Africa, a similar scenario has occurred in detections after 2005, with lineage II replacing I that was dominant. This trend of mixed lineages present in a single country occurs in countries like Uganda (Libeau *et al*., 2014) and Tanzania. So far the PPRV confirmed in Tanzania belongs to lineages II-IV. Lineage II (Mahapatra *et al*., 2015; Woma *et al*., 2015), III (Misinzo *et al*., 2015), IV (Woma *et al*., 2015) DRC reported PPRV in 2012 and is now endemic in some areas (FAO 2012).

In Kenya and Uganda, PPR was recognized in 2007, although PPRV antibodies had been detected several years earlier (Luka *et al*., 2012; Wamwayi *et al*., 1995; Dundon *et al*., 2015). PPRV control involves use of a live attenuated culture vaccine based on Nigeria75/1 strain. Much as four lineages of PPRV virus exist, vaccination using vaccine prepared from any of the lineage will provide protection against all the lineages. The immunity lasts up to 3 years and the vaccine is safe for pregnant dams inducing immunity in at least 98% of the vaccinated animals (Kihu *et al*., 2015). In Uganda, PPR virus is prevalent in the districts of Abim, Kaabong, Kotido, Moroto, Nakapiripirit Kapchorwa and Kitgum, districts (FAO, 2008). Goats and sheep mortalities result in loss of livelihoods, nutrition, income and employment. According to Mulindwa *et al*. (2011), the overall prevalence of antibodies against PPR virus in Karamoja sub-region was 57.6 %. No single study has been undertaken to establish the occurrence of the virus in cattle. More studies need to determine sero-positivity of PPRV in cattle country wide.

Conclusion

PPRV was distributed throughout Uganda. Control efforts for PPR in Uganda should be focused country wide, not just in Karamoja sub-region. The serological-prevalence was highest among cattle; this has implications to control of the disease. Cattle could serve as a reservoir to the disease hence a challenge to disease elimination. Active surveillance and vaccination against PPR especially in younger animals should be done on annual basis by using lyophilized vaccine.

Conflict of Interest

The authors have not declared any conflict of interest

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