SEROPREVALENCE AND RISK FACTORS OF PESTE DES PETITS RUMINANTS VIRUS INFECTION IN SHEEP AND GOATS IN GULANI LOCAL GOVERNMENT AREA, YOBE STATE, NIGERIA

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A DISSERTATION SUBMITTED TO THE SCHOOL OF POSTGRADUATE STUDIES AHMADU BELLO UNIVERSITY IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF MASTER DEGREE IN VETERINARY EPIDEMIOLOGY

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ZARIA, NIGERIA

NOVEMBER, 2019

DECLARATION

I declare that, the work in this Dissertation entitled "**SEROPREVALENCE AND RISK FACTORS OF PESTE DES PETITS RUMINANTS VIRUS INFECTION IN SHEEP AND GOATS IN GULANI LOCAL GOVERNMENT AREA, YOBE STATE, NIGERIA.**" has been carried out by me in the Department of Veterinary Public Health and Preventive Medicine, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, Nigeria. The information derived from the literature has been duly acknowledged in the text and a list of references provided. No part of this dissertation was previously presented for another degree or diploma at this or any other Institution.

Umaru BULAMA	________________	__________
Name of Student	Signature	Date

CERTIFICATION

This Dissertation entitled **"SEROPREVALENCE AND RISK FACTORS OF PESTE DES PETITS RUMINANTS VIRUS INFECTION IN SHEEP AND GOATS IN GULANI LOCAL GOVERNMENT AREA, YOBE STATE, NIGERIA.''** by **Umaru BULAMA** meets the regulations governing the award of **Master of Science** (M.Sc) degree of the Ahmadu Bello University, and is approved for its contribution to knowledge and literary presentation.

Prof. E. C. Okolocha Chairman, Supervisory Committee	________ Signature	________ Date
Dr. B. V. Maikai Member Supervisory Committee	________ Signature	________ Date
Prof. M. Bello Head of Department, Veterinary Public Health and Preventive Medicine	________ Signature	________ Date
Prof. Sani A. Abdullahi Dean, School of Postgraduate Studies, Ahmadu Bello University, Zaria	________ Signature	________ Date

DEDICATION

To my beloved parents, Late, Alhaji Bulama Kadafur and Fanna Umaru for their support and prayers at all times, may Allah in his infinite mercy reward them, and also to my compassionate wife (Habiba Aji Yerima) and my daughter Khadija Umar for their sacrifice, support and love throughout my study period.

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ABSTRACT

Peste des petits ruminants (PPR) is a severe, fast spreading disease of mainly domestic small ruminants (sheep and goats). It is characterized by sudden onset of depression, fever, discharges from the eyes and nose, sores in the mouth, disturbed breathing, coughing, foul smelling diarrhoea and death. This study was carried out between January and June 2018, to determine the seroprevalence and risk factors (age, sex, breeds and husbandry practices) of PPR in sheep and goats in Gulani Local Government Area (LGA) of Yobe State, Nigeria. A cross sectional study was conducted and a total of 331 sheep and goat sera were sampled and screened using Competitive Enzyme Linked Immunosorbent Assay (c-ELISA). An overall prevalence of 51.1% was obtained. There was higher seroprevalence of PPR in goats (53.9%) compared to sheep (47.7%), however, this was not statistically significant ($P > 0.05$). This work has also shown a higher seroprevalence of PPR in female (58.6%) than male (36.8%) goats and in female (50.5%) than male (41.7%) sheep. Only 18.4% of animals sampled were vaccinated against PPR, exposing a larger population of livestock to natural infections and possible PPR disease outbreaks. The seroprevalence of PPR antibody increases with age in both sheep (young 23.8%, adult 51.5%) and goats (young 42.5%, adult 57.2%). Location-based seroprevalence of PPR revealed the highest prevalence of 59.7% in Dutchi ward, while Kukuwa ward had the lowest prevalence rate of 34.5%. Breed-based seroprevalence revealed that the Sahelian goat had the highest prevalence of PPR (57.1%) while Udda breed of sheep had the lowest prevalence of 43.5%. Sokoto red goat, Yankasa and Balami sheep had 52.9%, 48.2%,

and 50% PPR prevalence rates respectively. In conclusion, there was high seroprevalence of PPR in the study area in sheep and goats which shows possible exposure to PPR in the study area. Therefore, further studies to isolate the virus and determine its strain and pathogenicity to livestock is recommended. Control measures should be instituted to monitor transboundary migration of animals for proper management of the disease and to minimize the loss associated with the disease.

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Abbreviation and definitions

PPR - Pestedes Petits Ruminants

TAD - Transboundary Animal Diseases

AU-IBAR – African Union Inter African Bureau for Animal Resources

USAID – United State Agency for International Development

SMP – Standard Method and Procedures

OIE – Office International des Epizootics

FAO – Food and Agriculture Organization

PPRV - Pestedes Petits Ruminants Virus

c-ELISA – Competitive enzyme linked immunosorbent assay

NPC - National Population Commission

YSG – Yobe State Government

NBS – National Bureau of Statistic

CHAPTER ONE

1.0 INTRODUCTION

1.1 Background of the Study

Peste des petits ruminants (PPR) is the most important singular cause of morbidity and mortality among small ruminants (Balamurugan *et al.,* 2010; King *et al.,* 2012). It is a contagious transboundary disease that is widely distributed across the Sub-saharan Africa, Middle East, Arabian Peninsula and the Indian subcontinent (El-Yuguda *et al.,* 2010; Emikpe and Akpavie, 2010; Woma *et al.,* 2016). The disease causes serious economic losses and remains a major deterrent to a successful development of small ruminant production in the countries where it occurs (El-Yuguda *et al.,* 2010; Emikpe and Akpavie, 2010; Woma *et al.,* 2016). It is one of the notifiable diseases of the OIE. Outbreaks of the disease have been reported in India (Balamurugan *et al.,* 2010), Southern Nigeria (Emikpe and Akpavie, 2010; Balogun *et al.,* 2017), Arabian peninsula (Abdelleti *et al*., 2016), Israel (Clarke et al., 2017), Ethiopia (Agga *et al*., 2019), Turkey (Atlan *et al*., 2019), Pakistan (Zahur *et al*., 2011), Iran (Shahriari *et al*., 2019), Iraq (Babashekh *et al*., 2014) and Tajikistan (Kwiatek *et al*., 2007) to mention but a few. Outbreaks of the disease are characterized by fever, erosive stomatitis, nasal and ocular discharges, pneumonia, diarrhoea and death. These signs may not all be exhibited by infected animals during an outbreak, as symptomless infections have been reported (Victor *et al*., 2017; El-Yuguda *et al*., 2010).

In Nigeria, sheep and goat production is constrained mainly by heavy burden of Peste des Petits des Petits Ruminant (PPR) which is a contagious viral disease of small ruminants that causes high morbidity and mortality, thereby reducing the number and productivity of the flock which impacts negatively on food security and the livelihoods of smallholder rural women and youth (Luka *et al.,* 2011). The disease is currently endemic in Nigeria and therefore disqualifies her from international trade in sheep and goats (Woma *et al.,* 2016; Victor *et al.,* 2017).

To address the negative impact of trans-boundary animal diseases (TADs) on livestock trade, African Union-Inter African Bureau for Animal Resource (AU-IBAR) together with the participating countries in the region, with financial support from the United States Agency for International Development (USAID), have developed a framework to support harmonization and coordination of the control of the diseases, referred to as the Standard Method and Procedures (SMP) Approach (AU-IBAR, 2015). The SMP approach involves strengthening capacities of member states for surveillance, epidemiology, laboratory diagnostics, disease control programs and communications. The fundamental aspect of the approach is the linking of disease prevention and control activities in a country, to a set of regional minimum standards and the procedures for TADs prevention and control in line with the World Organization for Animal Health (OIE) standards (AU-IBAR, 2015).

Spread of the disease to a number of countries in Africa and Asia with involvement of various lineage of PPR virus is a cause of global concern especially recent introduction of Asian lineage in some African countries and presence of PPR in Europe through Western Turkey (Banyard *et al.,* 2010; Luka *et al.,* 2011; OIE, 2019). Small ruminants play a very important role in Nigeria's livestock system, helping to meet the rising demand for animal protein in Nigeria's population of over 200 million people (Luka *et al.,* 2011; FAO-OIE, 2019). They provide small-holder farmers with a source of income, contributing manure for crop production and are part of the food and cash security available to many Nigerians (Woma *et al.,* 2016; Victor *et al.,* 2017). The animals are also used for sacrifices during religious and traditional festivals. Sheep and goats in Nigeria provide approximately 35% of the meat consumed and their skin support the leather industry and earn foreign exchange (Woma *et al.,* 2016; Victor *et al.,* 2017). The estimated annual direct financial losses as a result of deaths is as high as N50 billion (Fifty Billion Naira) at the rate of N10, 000/animal with indirect losses as high as N 65 Billion Naira (Fatiga *et al.,* 2013). The control of PPR is therefore expected to alleviate hunger, malnutrition and poverty, and solve associated societal problems such as youth and women unemployment and restiveness (IU-IBAR, 2015; FAO-OIE, 2019).

The first authentic and scientific description of the disease was reported in 1942 by Gargadennec and Lalanne who reported an epidemic disease in Ivory Coast (Cote d'ivoire) of West Africa, which was clinically similar to rinderpest (RP) but was

affecting only small ruminants, while in-contact cattle remained apparently healthy (Dundon *et al.,* 2020). In Nigeria, a successful PPR virus isolation was carried out in 1975 (Mantip *et al.,* 2019; Dundon *et al.,* 2020).

Peste des petit ruminant is characterized by sudden onset of depression, fever, discharges from the eyes and nose, sores in the mouth, disturbed breathing, coughing, foul smelling diarrhea and death (Balamurugan *et al.,* 2014; FAO-OIE, 2019). The causative agent (PPR virus) is an enveloped RNA virus which belongs to the genus *Morbillivirus* of the family *Paramyxoviridae* (sub family *Paramyxovirinae*) under the order *Mononegavirales* (Balamurugan *et al.,* 2010; King *et al.,* 2012) with other members of the genus, which include Rinderpest virus (RPV), Measles virus (MV), Canine distemper virus (CDV), Porcine distemper virus (PDV) and Dolphin and porpoise morbillivirus (DMV) (King *et al.,* 2012).

Clinically, the disease resembles Rinderpest. The virus is highly contagious and transmitted easily by direct contact with the secretions and/or excretions of infected animal (Luka *et al.,* 2011; King *et al.,* 2012). Outbreaks have continued to occur in the country during the last 40 years, despite the introduction of the tissue culture rinderpest vaccine (TCRV) against PPRV in the 1980s and the use of the homologous PPRV vaccine, since 1998. The National Veterinary Research Institute (NVRI) Vom, Nigeria produces a 50-dose PPRV vaccine vial using the Nigerian 75/1 strain, with the recommendation that small ruminants must be vaccinated from three months of age and thereafter every 12 months (Luka *et al.,* 2011; Woma *et al.,* 2016).

Peste des petit ruminant occurs in an enzootic form, it may have dramatic consequences with morbidity of 80-90% and mortality between 50-90% (Zahur *et al.,* 2011; Atlan *et al.,* 2019). Previously, PPR was reported in North-Eastern Nigeria on the basis of clinical signs by EL-Yuguda *et al.* (2008), serological by Woma *et al.* (2016) and another reported outbreak by El-Yuguda *et al.* (2008). A study conducted between 2010 and 2018 reported the PPR seroprevalence rates in some states of Nigeria at 20-55% in sheep and goats (El-Yuguda *et al.,* 2010; Luka et al., 2011; Woma *et al,* 2016; Victor *et al.,* 2017; Balogun *et al.,* 2017). A more efficient management and disease control in small ruminants is therefore warranted. To plan an effective strategy for the control of PPR, greater understanding of the current situation in the field is necessary.

1.2 Statement of Research Problem

In Nigeria, sheep and goat production is constrained mainly by heavy burden of Peste des Petits Ruminant (PPR) which is a contagious viral disease of small ruminants that causes high morbidity and mortality, thereby reducing the number and productivity of the flock which impacts negatively on food security and the livelihoods of smallholder rural women and youth (Shamaki, 2012; Fatiga *et al.,* 2013). The disease is currently

endemic in Nigeria and therefore disqualifies her from international trade in sheep and goats (Shamaki, 2012; Alimi *et al.,* 2013).

Livestock farming with emphasis on small ruminants (sheep and goats) is a major occupation of the inhabitants of Gulani Local Government Area and it has provided a source of animal protein, means of income, religious and cultural sacrifices.

A sero-epidemiological survey for PPR virus antibodies among sheep and goats in the Sahel region of North-East Nigeria reported a high prevalence of PPR among the two species in the area (El-Yuguda *et al.,* 2013; Bukar *et al.,* 2020). Yobe State is the central and share border with 4 out of the 6 states that form the North-East geographical zone giving it a hallmark of an economic and social disaster. Peste Des Petit Ruminants is poorly reported in Nigeria and is one of the epizootic diseases of small ruminants, which is highly infectious and causes high mortality (Shamaki, 2012; Alimi *et al.,* 2013). Information and knowledge on the prevalence, socio-economic impacts and risk exposure of the disease are negligible.

Insurgency in North-Eastern Nigeria caused by Boko Haram crisis has caused massive movement of both humans and livestock from one point to another with less or no disease surveillance in the area and this may cause a threat in the upsurge of PPR. The huge impact on small ruminant production has resulted in PPRV emerging as an

essential global animal health concern. Hence, PPR was proposed as the next animal disease to be eradicated after rinderpest (FAO, 2013).

1.3 Justification of the Study

Peste des petits ruminant (PPR) is the most important singular cause of morbidity and mortality among small ruminants (Balamurugan *et al.,* 2010; King *et al.,* 2012). Small ruminant populations at risk of PPR are estimated to be 63% by Food and Agricultural Organization of the United Nation (FAO, 2013). Although various studies have been conducted especially on seroprevalence, virus characterization and assessment of the socioeconomic impact and risk factors in Nigeria, these are not reported in Gulani Local government Area, Yobe state, which calls for a study of the disease in Nigeria (Woma *et al*., 2016). Officially, the total number of reported outbreaks of PPR in Nigeria for the period 2010-2014 is 474; this represents a gross under-reporting of actual number of outbreaks which far outweighs this figure (Mantip *et al.,* 2016; Dundon *et al.,* 2020). Proper understanding of the actual disease prevalence is paramount for effective, evidence-based planning and implementation strategies for the control and eradication of PPR.

The country's total resource of 109,728,554 sheep and goats is spread across the six geopolitical zones; kept by both males and females in Nigeria. The full development of these animals will no doubt improve the socio-economic well-being of rural families, assure nutritional and food security, and achieve the objectives of

government in the area of providing gainful employment for the citizenry (FAO, 2013).

Gulani Local Government Area is one of the Local Government Areas in Yobe State that produces sheep and goats in large numbers and has several livestock market. These animals when infected with PPR can affect the economy of the area as the farmers may lose them and the money generated as revenue from the trade of these animals will be lost. This will subsequently affect the income of the farmers. Infection may also spread to other neighbouring states during trade of these animals. Hence, studies that will determine the prevalence of PPR in Gulani Local Government Area will provide useful information on the burden of the disease in small ruminant production in the area. Risk factors for PPR such as sex, age, breed, location and husbandry practices if identified will assist in the control and prevention of infection. Findings from this research will also provide information on the level of vaccination coverage against PPR in Gulani Local Government Area.

1.4 Aim and Objectives of the Study

1.4.1 Aim of the study

The aim of this study was to determine the seroprevalence and risk factors associated with Peste des Petits Ruminat (PPR) in small ruminants in Gulani Local Government Area of Yobe State.

1.4.2 Specific objectives of the study

The objectives of the study were to:

i. Determine the seroprevalence of peste des petits ruminant (PPR) in sheep and goats in Gulani Local Government Area of Yobe State, Nigeria.
ii. Identify risk factors (sex, breed, age, location and husbandry practices) associated with the prevalence of the PPR antibodies in sheep and goats in Gulani Local Government Area of Yobe State, Nigeria.
iii. Determine the rate of peste des petits ruminant (PPR) vaccination from previous vaccination exercises in sheep and goats in Gulani Local Government Area of Yobe State, Nigeria.

1.5 Research Questions

i. What is the seroprevalence of PPR in sheep and goats in Gulani Local Government Area, Yobe State, Nigeria?
ii. Risk factors like sex, breed, age, location and husbandary practices are associated with the prevalence of PPR in sheep and goats Gulani Local Government Area of Yobe State, Nigeria?
iv. What isthe rate of peste des petits ruminant (PPR) vaccination from previous vaccination exercises in sheep and goats in Gulani Local Government Area of Yobe State, Nigeria?

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1Definition, History and Geographical Distribution of Peste des PetitsRuminant (PPR)

Peste des petits ruminant (PPR) is a severe, fast spreading disease of mainly domestic small ruminants (sheep and goats), and characterized by sudden onset of depression, fever, discharges from the eyes and nose, sores in the mouth, disturbed breathing, coughing, foul smelling diarrhea and death (OIE, 2016; Dundon *et al.,* 2020). PPR was first recognized as a disease entity in 1942 in Cote d'ivoire by Gargadennec and Lalenne and in Senegal in 1962 by Mornet *et al.* (Banyard *et al.,* 2010; Torsson *et al.,* 2016), while the first reported isolation of the virus was in 1962 by Gilbert and Monier. Provost *et al.* (1972) reported the disease in Tchad, Bourdin (1983) reported a PPR-like disease in Benin, Benazet (1973) reported it in Togo, in Mauritania, Mali and Burkina Faso it was reported by Lefevre and cited by Taylor (1983), while Wamwayi *et al.* (1995) demonstrated the presence of PPR antibody in goat in East Africa (Banyard *et al.,* 2010; Torsson *et al.,* 2016).

PPRV has been identified as the cause of several serious epidemics in small ruminant populations over the last three decades and, since 1993, the Arabian Peninsula, the Middle East and major parts of the Indian subcontinent have reported major outbreaks and the virus is now considered endemic across this region (Khalafalla *et al.,* 2010; Torsson *et al.,* 2016). Lineage differentiation is determined by the sequence

comparison of a small region of the F gene or N gene, depending on the reaction components used by the testing laboratory (Munir *et al.,* 2013; OIE, 2016). Historically, African isolates of PPRV were numbered lineage I-III according to the proposed spread of the virus from West Africa to East Africa. The isolates derived from Ghana, Mali and Nigeria then formed lineage II and those detected in Ethiopia and Sudan were from lineage III. Data derived from the F gene material reversed the classification of lineage I and II isolates and historically this difference has been maintained (Banyard *et al.,* 2010). The current molecular characterization of PPRV virus isolates divides them into four genetically distinct lineages: lineage I being represented mainly by Western African isolates from the 1970s and recent isolates from Central Africa; lineage II by West African isolates from the Ivory Coast, Guinea and Burkina Faso; lineage III by isolates from Eastern Africa, the Sudan, Yemen and Oman; lineage IV includes all viruses isolated from recent outbreaks across the Arabian Peninsula, the Middle East, southern Asia and recently across several African territories. Data generated by PCR and sequencing is routinely used to construct phylogenetic trees for PPRV and ascribe different isolates to the different lineages (Munir *et al.,* 2013). It is unclear whether this lineage assortment has any relationship to pathogenicity or is just a result of geographical speciation.

In 2000, the presence of PPRV was analyzed in Saudi Arabia and it was concluded that the disease was not circulating in the country (Banyard *et al.,* 2010; OIE, 2016). However, by April 2002 an outbreak with a case mortality rate of 100 % was reported

in sheep and goats and since then further serological surveys and outbreaks have been reported (Al-Dubaib, 2009). A possible role of camels in the dissemination of PPRV to goats has also being suggested as in Ethiopia in 1995 although more recent surveys in Sudan have suggested this route of dissemination as being unlikely (Khalafalla *et al.,* 2010). Seroprevalence of both sheep and goats has also been reported in North Jordan and in the Lebanon with seroprevalence of up to 48.6 % being reported (Banyard *et al.,* 2010).

PPRV was first identified in West Africa in Nigeria in 1942. It is currently believed to be endemic across much of West Africa (Banyard *et al.,* 2010). However, virus outbreaks are often poorly characterized due to the lack of reporting systems and facilities within which to conduct molecular tests. West Africa includes 16 countries distributed over an area of approximately 5 million square km. A number of these countries have experienced significant outbreaks of PPRV. In recent years, material submitted to Regional Reference Laboratories (RRLs) has confirmed the presence of the either antibodies to the virus or the detection of viral nucleic acid in samples from Burkina Faso (2008), Ghana (2010), Nigeria (2007) and Senegal (2010) (Banyard *et al.,* 2010; OIE, 2016). PPRV strains from both lineages I and II are currently circulating across West Africa although undoubtedly many outbreaks are not characterized at the molecular level. Other cases of PPRV in sheep, goat and camel populations have also recently been described in Nigeria (Ibu *et al.,* 2008; El-Yuguda *et al.,* 2010) and a further Nigerian study used haemagglutinin tests with faecal matter

to detect PPRV excretion and suggested that healthy animals may serve as carriers for PPRV (Banyard *et al.,* 2010). In Burkina Faso, antibody prevalence to PPRV of 28.5 % has been reported in the north (Banyard *et al.,* 2010). According to the geographical distribution of PPR outlined above, it is not a worldwide disease. However, recent report of PPR in areas close to European borders such as Spain and Turkey have increased its profile both scientifically and in the media. Whilst this devastating disease of small ruminants has continued to plague agriculture across Africa and Asia for many years, the threat of spread in the developed world has greatly renewed interest in the virus (OIE, 2016).

2.2Classification and Characteristics of PPR Virus.

The causative agent, PPR virus (PPRV) is an enveloped RNA virus which belongs to the genus Moribillivirus of the family Paramyxoviridae (sub family Paramyxovirinae) under the order Monogavirales with other members of the genus, which include Rinderpest virus (RPV), Measles virus (MV) Canine distemper virus (CDV), Porcine distemper virus (PDV) and Dolphin and Porpoise moribillivirus (DMV) (Balamurugan *et al.,* 2010; Amarasinghe *et al.,* 2017).

PPRV is a negative-sense, single-stranded, non -segmented RNA virus. It is enveloped, pleomorphic, ranging from 150-700nm in size. The envelope is

approximately 8-15nm in diameter with spikes of 8.5-14.5 nm long (King *et al.,* 2012; Rima *et al.,* 2019). These characteristics show a close similarity with the viruses causing rinderpest, canine distemper and measles. PPRV is thermolabile but relatively resistant in a cool environment and can survive for months in frozen animal tissue (Shemaki, 2002). It is inactivated at a pH of 3.0 and has a half-life of about 22minutes at 56^0C and 3.3 hours at 37^0C. PPRV and RPV share serological relatedness but the viruses are not identical. Cross-protection trials showed that PPRV protected cattle against rinderpest (RP) and rinderpest virus (RPV) protected sheep and goats against PPRV challenge (Rossiter and Taylor, 1994). The virus is genetically grouped into four distinct lineages (I, II, III, and IV) on the basis of partial sequence analysis of the fusion (F) protein gene (Banyard *et al.,* 2010; Kwiatek *et al*., 2011; Kumar *et al.,* 2014). Despite the fact that only a single serotype has been reported. Lineages I and II are found exclusively in West Africa, whereas lineage III has been found in Eastern Africa and Arabia (Maganga *et al.,* 2011; Cosseddu *et al.,* 2015; Woma *et al.,* 2016). The fourth lineage is confined exclusively in the Middle East Arabia and India subcontinent (Cosseddu *et al.,* 2015).

The PPRV genome is organized into six contagious, non-overlapping transcriptional units. PPRV is a non-segmented, single, negative sense RNA virus with a genome of approximately 15-16kb in size (with a total 15, 948 nucleotides); it contains six transcription units encoding eight proteins (Kwiatek *et al.,* 2010; Abubakar *et al.,* 2011; Luka *et al.,* 2011; Dundon *et al*., 2014; Munir *et al.,* 2015). These are the nucleocapsid protein (N), phosphoprotein (P), matrix protein (M), fusion protein (F),

haemagglutinin protein (H), large polymerase (L) and two non-structural protein (V and C) which are translated from message-RNAs transcribed from the P gene (Kwiatek *et al.,* 2010; Abubakar *et al.,* 2011; Luka *et al.,* 2011; Dundon *et al*., 2014; Munir *et al.,* 2015).

2.3 Source of Infection and Transmission of Peste des Petits Ruminant(PPR) Virus

The source of infection is the sick or sub-clinically infected animal and the virus is discharged in milk, saliva, urine or faeces (Munir, 2014; Parida *et al.,* 2015a). The disease spreads primarily by inhalation but the virus can be acquired by ingestion and penetration through the conjunctival mucosa. Animals may acquire the infection by licking or muzzling each other. Bedding feed and water trough can be sources of infection. Goats are highly susceptible to PPR compared to sheep, and kids under one year are most susceptible (Balamurugan *et al.,* 2012; Truong *et al.,* 2014). In West Africa, the dwarf breeds of goats have been found to be more susceptible than the sahelian breeds (Balamurugan *et al.,* 2012). In West Africa, the incidence of PPR is reported to increase during the rainy season and during the cool harmattan winds (Balamurugan *et al.,* 2012).

On the field clinical assessment, moderately and minimally virulent isolates were described, allowing the recovery of infected sheep and goats, which were subsequently fully protected (Munir, 2014; Parida *et al.,* 2015a). Wild species are clinically affected by PPRV in a similar manner to domestic sheep and goats, the virus will hence have

little trouble spreading into this population should the opportunity arise (Bamouh *et al.,* 2019).

Transmission of PPR into a flock may be associated with introduction of newly purchased animals, change in husbandry management towards intensification, change of weather such as the onset of rainy season or harmattan as well as history of recent movement or gathering together of sheep and/ or goats of different ages with or without associated changes in housing and feeding (Munir, 2014; Parida *et al.,* 2015a, Bamouh *et al.,* 2019).

2.4Pathogenesis of Peste des Petits Ruminant (PPR) Virus

PPR has a direct life cycle, maintained by transmission of the virus from infected to susceptible animals, without involvement of carrier animals or vector (Parida *et al,* 2015b). The disease progresses with development of lacrimal, nasal and mucosal discharges and viral material can be detected in such excretions as early as 4 days after infection (Munir *et al*., 2013; Bamouh *et al.,* 2019). Viral antigens have also been observed by histochemical staining in the lymphoid organs, the respiratory and the gastro-intestinal tract (Munir *et al*., 2013; Bamouh *et al.,* 2019).

The pathogenesis of peste des petits ruminants has long been assumed to mirror that of rinderpest in cattle (Munir *et al*., 2013; Bamouh *et al.,* 2019). Pathogenesis studies in peste des petits ruminants have mainly been performed through experimental infection with a virulent form of the virus to develop a reliable and reproducible model (El

harrack *et al.*, 2012: Hammouchi *et al.*, 2012; Pope *et al.,* 2013; Baron *et al.,* 2014). Histological assessment during early infection showed an immune cell proliferation driven by the spread of the virus, similar to that caused by other morbilliviruses (Pope *et al.,* 2013). The initial site for virus replication is observed within the tonsillar tissue and lymph node draining the site of inoculations. It has been proposed that the virus infected immune cells within the respiratory mucosa would migrate to the local lymphoid tissue, where primary virus multiplication would occur, with the virus then entering the general circulation. Clinical signs usually develop 3-4 days later with established pyrexia and anorexia. Leucopoenia is observed from the fourth day post infection with considerable reduction of CD4 T cells (Baron *et al*., 2014; Herbert *et al.,* 2014). In the later stage of infection erosive lesions are formed in the oral cavity that also becomes necrotic. The severity of clinical signs generally peak between 6 and 8 days post infection and can continue for up to 14 days leading to death or recovery from infection.

2.5 Immuno-suppression and Immunological Features

Peste des petits ruminants virus is highly lymphotrophic and infection often leads to a profound immune-suppression that causes leuocopoenia and reduced antibody responses (Jagtap *et al.,* 2012; Pope *et al.,* 2013). Immuno-suppression by peste des petits ruminants virus has been observed in both vaccinated and infected animals (Jagtap *et al.,* 2012; Harrak *et al.,* 2012; Rojas *et al.,* 2017). Virulent strain of the virus causes marked immune-suppression, whereas vaccination only induces a transient

leucopoenia with no significant effect on the immune response (Jagtap *et al.,* 2012; Harrak *et al.,* 2012; Rojas *et al.,* 2017).

Maternal antibodies against the virus can be detected in young animals and remain able to neutralize virus for three (3) to four (4) months enabling a level of protection in newborn animals (Pope *et al.,* 2013; Rojas *et al.,* 2017). Therefore, vaccination of new born animals is not necessary until that age (Truong *et al.,* 2014; Rojas *et al.,* 2017).

2.6 Clinical Signs of Peste des Petits Ruminant (PPR)

Although goats and sheep are the primary hosts of the virus, goats seem to be more susceptible to the disease than sheep, with some breeds of goats considered to be more susceptible than others (Balamurugan *et al.,* 2012; Truong *et al.,* 2014). The incubation period of the disease is typically 4-6 days, although it may range between 3-14 days (OIE, 2012; Parida *et al.,* 2015a). During the acute stage of disease, animals show pyrexia (up to 41^0C) that may last for 3-5 days and that can be accompanied by depression, anorexia and dryness of the muzzle, watery nasal and lacrymal discharges gradually become mucopurulent with excessive salivation (Munir *et al.,* 2013). Erosive lesions formed in the oral cavity may become necrotic. In severe cases of the disease, these necrotic lesions progress with the appearance of a deposit of fibrin (caseous deposit) on the tongue. In later stages of the disease, the animal develops diarrhea and coughing with labored abdominal breathing (Chowdhury *et al.,* 2014; OIE, 2016). Finally, the animal may become dyspnoeic, suffering progressive weight

loss and emaciation ultimately leading to death. In mild cases, the animal may recover. The mortality rate can reach 100% with a high case fatality rate in the acute form of disease (Pope *et al.,* 2013; OIE, 2016). The above described clinical signs and mortality can vary considerably depending on the virulence of the viral strain and the immunological state of the affected animal (OIE, 2012). Pregnant animals may abort. The clinical disease may be complicated by many secondary pathogens. The most important pathogens include *Pasteurella* spp causing pneumonic pasteurellosis and *Mycoplasma* which causes severe pneumonia. In addition, infections with Escherichia coli produce a heat-labile enterotoxin which will increase the severity of the clinical signs and the morality rate (Chowdhury *et al.,* 2014; Bamouh *et al.,* 2015).

2.7Pathological Features of Peste des Petits Ruminant (PPR)

The gross pathological pictures include an emaciated and dehydrated carcass, soiled hind quarters, matted eyelids and nostrils blocked with exudates (Kumar *et al.,* 2013; Emikpe *et al.,* 2013). Focal necrotic lesions in the oral mucosa, pharynx, upper oesophagus, abomasum and small intestines are evident. Zebra stripping of the large intestinal mucosa, oedema and congestion of mesenteric lymph nodes occur (Emikpe *et al.,* 2013). The nasal cavity and larynx are filled with mucoprulent exudates and presence of froth in the trachea and pulmonary oedema are common features, secondary bacterial infections results in bronchitis, tracheitis, atelectasis and interstitial pneumonia (Pope *et al.,* 2013; Parida *et al.,* 2015b).

The histopathological picture includes lymphocyte depletion in lymphoid tissues, picnosis and karynorrhesis of lymphocytes in the cortex of lymph nodes (Emikpe *et al.,* 2013). Multinucleated giant cells, eosinophilic intracytoplasmic and internuclear inculsion bodies can be demonstrated in epithelial cells which also show hydropic degeneration and necrosis (Emikpe *et al.,* 2013). The lamina propria of the small intestine is infiltrated with lymphocytes, macrophages and eosinophils (Pope *et al.,* 2013). There is also necrosis of the epithelium of the abomasal and intestinal glands, villous atrophy and accumulation of necrotic debris in glandular pits. The alveoli and terminal bronchi are infilterated with giant cells containing intracytoplasmic and intranuclear inculsion bodies. There may be hepatic necrosis and glomerulonephritis. The clinical pathology of PPR is dominated by leucopenia and hymphopaenia (Emikpe *et al.,* 2013; Pope *et al.,* 2013).

2.8 Susceptibility of Peste des Petits Ruminant (PPR) Virus

Sheep and goats are the primary hosts for peste des petits ruminants virus with few reports of disease outbreak in other animals (Khalafalla *et al.,* 2010; Kwiatek *et al.,* 2011 b). Cattle, Buffalo and pigs develop subclinical infection but are not capable of excreting the virus, and thus are not considered to be important in the epidemiology of the virus (Banyard *et al.,* 2010; Lembo *et al.,* 2013; Schulz*et al.,* 2018). Infection of

various wildlife species, mainly living under semi-free range conditions, has been reported, although their exact role in the epidemiology of the disease needs to be studied (Kinne *et al.,* 2010). Other host factors e.g. age, sex, breeds or season, may also play a role in disease development.

The isolation of PPRV from Dorcas gazelle which died of a natural infection in the United Arab Emirates has broadened our understanding of the host range of the virus (Kinne *et al.,* 2010). Other wildlife that were affected during previous outbreaks included Gemsbok (*Oryx gazelle*), Mouflon (*Ovis musimon*), lavistan sheep (*Ovis orientalis laristanica*), Nubian Ibex (*Ovis ibex nutiane*) and free-living bharals (*Pseudois nayaur*) (Guer and Albayrak, 2010); FAD PReP, 2014). In cattle PPR was not shown to be pathogenic although there was sero-conversion (Rahma *et al.,* 2020). In Nigeria, the natural disease is known only in sheep and goats. Result of experimental transmission indicates that cattle and pigs do not suffer from clinical disease (Mantip *et al.,* 2019; Rahma *et al.,* 2020). Oludairo*et al.* (2016) and Woma *et al.* (2015) reported the probable role of wildlife (warthog) and camel in the epidemiology of PPR in Nigeria.

2.9 Diagnosis of Peste des Petits Ruminant (PPR)

In the diagnosis of PPR, other diseases that cause diarrhea or pneumonia may pose diagnostic challenge (Santhamani *et al.,* 2016). However, a history of recent

introduction of new stock, the clinical and post-morterm findings of stomatitis, enteritis and pneumonia are typical for PPR (Banyard *et al.,* 2010; Balamurugan *et al.,* 2014; Santhamani *et al.,* 2016). However, some laboratory techniques have been useful to confirm the diagnosis of PPR infection under various conditions.

The differential diagnosis include, pasteurellosis, contagious ecthyma, contagious caprine pleuropnemonia (CCP), blue tongue, heart water, coccidiosis, mineral poisoning and foot and mouth disease (Malik *et al.,* 2011; Albina *et al.,* 2013; Santhamani *et al.,* 2016) . Samples required for laboratory diagnosis of PPR include: whole blood in heparin or EDTA, serum, lymph nodes, tonsils, spleen, lungs and swabs of secretions (FAO, 2015; Santhamani *et al.,* 2016).

Clinical diagnosis of PPR is confirmed by various methods. The most commonly used are: Counter immune electrophoresis (CIEP), Agar gel imunodifussion (AGID), Indirect or passive haemogglutination, Immunoflourescence or immunoperoxidase staining, Single radial haemolysis, Immunocapture ELISA, Competitive ELISA, (c-ELISA), latex agglutination, virus neutralization (VN), Complement fixation and virus isolation in cell culture (Balamurugan *et al.,* 2010; Balamurugan *et al.,* 2012; Kumar *et al.,* 2013; Munir *et al.,* 2013; Baron *et al.,* 2014; Ishaq *et al.,* 2015).

For molecular detection of PPR, conventional RT-PCR test is available. Real-time-PCR assays and loop-mediated isothermal amplification techniques have been

developed (Kwiatek *et al.,* 2010; Santhamani *et al.,* 2016). However the gold standard for definitive diagnosis is virus isolation from tissue samples.

The OIE recommends the use of competitive PPRV - specific anti-H monoclonal based ELISA and virus neutralization test (Kwiatek *et al.,* 2010; Santhamani *et al.,* 2016). The virus neutralization test is time-consuming (requiring 10 to 12 days for a result), requires tissue culture and sterile test samples, is labour intensive and of low throughput. Meanwhile, the competitive ELISA to PPR has a sensitivity of 94.5% and a specificity of 99.4% (Balamurugan *et al.,* 2012; Santhamani *et al.,* 2016). The ease with which ELISA can be performed, the large number of samples that can be tested in a short time and the high sensitivity of the test, have all contributed to its adoption as at routine technique in a disease diagnosis.

2.9.1 Competitive enzyme linked immunosorbent assay (c-ELISA)

The commercially available c-ELISA kit (IDVET, France) for PPR was used for this study. This diagnostic kit detects antibodies directed against the nucleoproteins of PPRV and was developed by FAO Reference Laboratory (CIRAD-EMVT, Montpellier, France). The test was performed according to the Manufacturers instructions (Zhan *et al.,* 2013). The ELISA micro plate was read with an immunoscan reader (Flow laboratories, UK) with a filter of 450nm. The optical density (OD) was recorded and the test was validated when the mean value of the negative control OD (ODNC) was greater than 0.7 and the mean value of the positive control OD (ODPC)

was less than 0.3 of the ODNC. The OD values were converted to competition percentage (CP) using the following formula; CP = OD sample / ODNC * 100.

2.10 Differential Diagnosis of Peste des Petits Ruminant (PPR)

The differential diagnosis include rinderpest (although many reports of rinderpest among small ruminants may have been PPR), blue tongue, contagious ecthyma, foot and mouth disease, heart water, coccidiosis and mineral poisoning. The respiratory signs can resemble contagious caprine plueropneumonia (CCPP) or pasteurellosis, Pasteurellosis can also be a secondary complication of peste des petits ruminants (Dundong *et al.,* 2011; Malik *et al.,* 2011; Albina *et al.,* 2013; Santhamani *et al.,* 2016).

2.10.1 Rinderpest

This is a disease of ruminants characterized by fever, erosive stomatitis, severe diarrhea or dysentery, dehydration and death (Parida *et al.,* 2015). It is caused by a rinderpest virus of the genus Morbillivirus and family Paramyxoviridae which closely resembles the PPR virus. (Munir, 2014; Parida *et al.,* 2015). Differentiating rinderpest from PPR is difficult because their clinical signs closely resemble each other. However, it should be borne in mind that clinical disease caused by rinderpest in small ruminants is a relatively rare event, even in Asia (Bantard *et al.,* 2010; Parida *et al.,* 2015).

2.10.2 Contagious ecthyma

Contagious ecthyma (Contagious pustular dermatitis or orf) is a highly infectious disease of goats and sheep characterized by pustular and scabby lessions on the muzzle, commissures of the lips and nostrils (Flemming *et al.,* 2015; Karki*et al.,* 2019). The disease is caused by a contagious ecthyma virus of the genus Parapox virus and family Poxviridae and it also affects man (Flemming *et al.,* 2015; Adedeji *et al.,* 2018). The nodules seen in this disease resembles the thick scabs observed on the lips in PPR infection. However pneumonia, diarrhoea and sometimes oral lessions are not usually observed in uncomplicated orf (Flemming *et al.,* 2015; Karki*et al.,* 2019).

2.10.3 Blue tongue

This is an arthropod-borne disease of ruminants particularly sheep which is characterized by catarrhal stomatitis, haemorrhages, enteritis, cyanosis of the oronasal cavity, laminitis oedema of the head and neck, and torticollis (Maclachlan*et al.,* 2011). Blue tongue (BT) is caused by a blue tongue virus of the genus orbivirus and family Reoviridae (Maan *et al.,* 2011; Flemming, 2015). In this disease, fever, nasal discharge and oral lessions similar to those seen in PPR are observed. However, unlike PPR, blue tongue is characterized by edema of the head region as well as bluish discoloration of the oral cavity (Maclachlan*et al.,* 2011).

2.10.4 Foot and mouth disease

This is a highly infectious disease affecting cloven-hoofed animals caused by a foot and mouth disease virus of the genus Aphthovirus and family Picornaviridae and characterized mainly by fever, stomatitis, lamness and fall in production (OIE, 2019; Muthukrishnan *et al.,* 2020). The most important distinguishing features between FMD and PPR are the absence of breathing problems and diarrhea, and the presence of marked lameness in FMD (Atuman *et al.,* 2020). In addition, the foul smelling odour that exudates from the mouth of PPR infected animals is usually not found in FMD (OIE, 2016; Flemming, 2015; Atuman *et al.,* 2020).

2.10.5 Pneumonic pasteurellosis

This is purely a respiratory disease. No diarrhea or oral lesion is found. In cases where PPR presents without oral lesions, diagnosis can only be confirmed by detecting PPRV from tissues (FAO, 2010; Flemming, 2015).

2.10.6 Contagious caprine pleuro-pneumonia (CBPP)

Contagious caprine pleuro-pneumonia (CBPP) like PPR, is characterized by fever, dypsnea and coughing, but however, no mouth lesions and diarrhea are found (FAO, 2010; Flemming, 2015). In addition, at post mortem, fibrin deposits are found on the lungs and there are also adhesions of the lungs to the chest cavity in CCPP (FAO, 2010; Flemming, 2015).

2.11 Risk Factors Associated with Peste des Petits Ruminants (PPR)

Factors that have been reported to affect the prevalence of PPR include acquisition of animals from local markets (Abubakar *et al.,* 2011; Rahman *et al.,* 2016). Mass transportation of animals during major festivals, local husbandry and customary practices of renting males, and all year round kidding and constant introduction of new stock leading to an increased number of susceptible animals (Elsawalhy*et al.,* 2010;Dundong *et al.,* 2011). Age is also one of the risk factors which affect the occurrence of PPR in small ruminants (Dundong *et al.,* 2011; Victor *et al.,* 2017). As such, the disease is more fatal in goats of about 4-8 months of age when maternal antibodies might have weaned as the passive immunity becomes progressively lost, susceptibility to PPR infection increases (Victor *et al.,* 2017). PPR is more common in the rainy season or dry cold season. Weather related seasonality and livestock morbidity cause stress and can compromise immune response (OIE, 2013). The presence of naive (unprotected) populations within an infected region is a major risk factor in epidemics. Young and non-vaccinated animals are groups that are at high risk of infection (Victor *et al.,* 2017). Malnutrition, parasitism and bacterial infections are predisposing factors that aggravate the clinical disease (Dundong *et al.,* 2011; AU-IBAR, 2014).

2.12 System of Small Ruminant Management in Sub-saharan Africa

Small ruminant management system in sub-saharan Africa can be grouped into two broad categories, that is, traditional and modern systems (OIE, 2013). The traditional management system can be further subdivided into groups namely the extensive (pastoral), semi intensive (agropastoral) and intensive (agriculture or village) systems (OIE, 2016).

2.13 Seroprevalence of PPR in Nigeria

A number of studies have been carried out to describe outbreaks of PPR in Nigeria since 1970, but the virus was first isolated in 1975 and 1976 for the first time (Mantip *et al.,* 2016). The current live-attenuated PPRV vaccine was derived from one of those isolates, Nigeria 75/1 (Luka *et al.,* Woma *et al.,* 2016). The full genome sequence of another lineage II PPRV isolate from Nigeria in 1976 was generated by Chard et al. in 2008 (Luka *et al.,* 2011). In 2007 and 2009 samples were collected during outbreaks in Kaduna and Plateau States which were identified lineage II viruses (assigned to lineage I by the authors according to the classification used at the time) (Luka *et al.,* 2011). Similarly, an outbreak of peste des petits ruminant (PPR) among unvaccinated Sahel goats was later reported in a farm in Maiduguri, Nigeria in 2007 by El-Yuguda *et al.* (2009), with a morbidity rate of 63% and mortality rate of 17%, giving a case fatality rate of 27%. PPR virus neutralizing antibodies was also detected in 75% of goats and 60% of in-contact sheep (El-Yuguda *et al.*, 2009). In 2013, a serological study was performed in semi-arid region of North-eastern Nigeria using VNT and c-ELISA, an overall seroprevalence of 57% and 55% were revealed using VNT and c-

ELISA, respectively, with the highest seroprevalence of 76.5% in sheep followed by goat 51.6%, camels 27.8% and cattle 16.7% (El-Yuguda *et al.,* 2013).

In Makurdi, Benue State and Yobe State, Nigeria, Victor *et al.* (2017) and Bukar *et al.,* (2019) found an overall prevalence of PPR to be 51.3% and 55.4% respectively in goats using competitive enzyme-linked immunosorbent assay (c-ELISA). Vicotor *et al*. (2017) showed that the incidence of PPR was very high in dry season than rainy season with animals less than 12 months of age and mostly female were affected by PPR. (Victor *et al.,* 2017; Bukar *et al.,* 2019). Woma *et al.* (2016) performed a molecular epidemiological study of 140 clinical samples from sheep and goats collected in agro-ecological zones of Nigeria between 2010 and 2013, and revealed that viruses from lineages II and IV were circulating in the country and that the lineage IV isolates grouped into two clades (IV-NigA and IV-NigB) (Woma *et al.,* 2016). They discovered that one of these viral sequences (GenBank no. KJ124767) was identical to the vaccine strain Nigeria 75/1.

Mantip *et al.* (2016) performed a meta-analysis of the PPR virus isolation in molecularepidemiology studies and confirmed the presence of both lineage II and lineage IV PPRV (PPRV 75/1) in Nigeria. The isolates PPRV 75/1 was dominant and is been used in the scientific study to understand the taxonomy, molecular dynamics, lineage differentiation of PPRV and the development of vaccine seeds for immunization against PPR.

2.14 Population and Distribution of PPR Susceptible Species

The country's total resource of 109,728,554 small ruminants is made up of 41,147,464 sheep and 68,581,090 goats and spread across the six geopolitical zones (Dilli *et al.,* 2011). Currently, sheep contribute 5% while goats contribute 16% of the 833,000 tonnes of meat production in the country per annum (Dilli *et al.,* 2011). The full development of these animals will no doubt improve the socio-economic well-being of rural families, assure nutritional and food security, and achieve the objectives of government in the area of gainful employment for the citizenry.

Sheep and goats are the primary hosts for peste des petits ruminants virus with few reports of disease outbreak in other animals (Khalafalla *et al.,* 2010; Kwiatek *et al.,* 2011 b). Cattle, Buffalo and pigs develop subclinical infection but are not capable of excreting the virus, and thus are not considered to be important in the epidemiology of the virus (Banyard *et al.,* 2010; Lembo *et al.,* 2013; Schulz*et al.,* 2018). Infection of various wildlife species, mainly living under semi-free range conditions, has been reported, although their exact role in the epidemiology of the disease needs to be studied (Kinne *et al.,* 2010).

El-Yuguda *et al.* (2013) performed the seroprevalence of peste des petits ruminants among domestic small and large ruminants in the semi-arid region of North-eastern Nigeria. The discovered the highest seroprevalence of 76.5% in sheep followed by goat (51.6%), camels (27.8%) and cattle (16.7%) (El-Yuguda *et al.*, 2013). Victor *et al.* (2017) on the other hand showed that the West African Dwarf (WAD) breed of g oat was found to have higher PPR prevalence (69.7%) than the Sokoto red (67.5%) an d Sahel (22.2%) breeds of goat. Similarly, the Yankasa breed of sheep was found to have a higher prevalence (30.8%) than the West African Dwarf (WAD) breed sheep (29.2%) (Victor *et al.* 2017). Similarly, Woma *et al.* (2016) found the seroprevalence of PPRV antibodies in each animal category to be similar (20.44% in male adult sheep, 22.29% in female adult sheep, 21.64% in young male sheep, 23.7 8% in young female sheep, 23.89% in adult male goats, 22.09% in adult female goats, 26.21% in young male goats and 24.02% in young female goats) (Woma *et al.,* 2016).

2.15Economic Impact of Peste des Petits Ruminant (PPR)

The presence of PPR can have a serious impact on the economics of a region which has a widespread distribution spanning Africa and Asia (Ahaduzzaman, 2020). Economic losses are due to loss of production, death and abortion. The presence of disease can limit trade, export, import of new breeds, and the development of intensive livestock production. PPR is a major constraint on the availability of protein for human consumption as well. In addition to this economic role, sheep and goats have significant socio-cultural roles. They are used as gift or emblems for traditional rituals

and religious purposes (Banyard *et al.,* 2010; Ahaduzzaman, 2020). Based on a 15-year programme with total discounted costs of US$2.26 billion, Jones *et al.* (2020) estimated discounted benefits of US$76.5 billion, yielding a net benefit of US$74.2 billion. This suggests a benefit cost ratio of 33.8, and an internal rate of return (IRR) of 199%. As PPR mortality rates are highly variable in different populations, we conducted a sensitivity analysis based on lower and higher mortality scenarios. All the scenarios examined indicate that investment in PPR eradication would be highly beneficial economically. Furthermore, removing one of the major constraints to small ruminant production would be of considerable benefit to many of the most vulnerable communities in Africa and Asia (Jones *et al.*, 2020).

The total numbers of dead sheep and goats are 154,977 in the Sudan Savannah, 325,679 in the Northern Guinea Savannah, 53,772 in the Subhumid Zone and 74,621 in the Humid Zone. About 3.3% of the total population of sheep and goats is lost each year as a result of PPR (FAO and OIE, 2018). The total value of losses caused by PPR amounted to NGN 4.3 billion nationwide with the Northern Guinea Savannah Zone accounting for 52%, followed by the Sudan Savannah Zone (28%), the Humid Zone (12%) and the Subhumid Zone (8%). There are additional costs of the disease that pertain to treatment, vaccination, and surveillance. However, secondary data and expert opinions revealed a lack of any credible surveillance program for PPR and despite the absence of any effective treatment against PPR clinical cases, producers still provide various forms of treatment to their stricken flocks. On average, nearly 50% of the flock holders affirmed having carried out treatment against PPR.

Moreover, while a vaccine against PPR is available, seldom did producers utilize it as an option to protect their flocks despite its lower cost (NGN 225, on average) compared to the treatment options (NGN 374, on average). Information collected through PE coupled with compiled secondary data, including a 2008 AU-IBAR report and expert opinions, indicated not more than 2% of sheep and goats were vaccinated across all agroecological zones.

Total expenditure on treatment was evaluated at NGN 2.602 billion nationwide of which 16% was incurred in the Subhumid Zone, 50% in the Northern Guinea Savannah Zone, 12% in the Sudan Savannah Zone and 23% in the Humid Zone. Total expenditure on vaccination was evaluated at NGN 64 million of which 44% was incurred in the Northern Guinea Savannah Zone, 25% in the Sudan Savannah Zone, 13% in the Subhumid Zone and 17% in the Humid Zone. Total PPR burden amounts to NGN 6.779 billion with the Northern Guinea Savannah accounting for 51%, the Sudan Savannah for 22%, the Subhumid Zone for 11% and the Humid Zone for 16% (FAO, 2013).

2.16Prevention and Control of PPR

The methods for prevention and control of PPRV vary widely depending on local facilities, techniques adopted and the provision of veterinary services and vaccine, respectively (Singh, 2012; Dundon *et al.,* 2020). OIE recommended that exposed or infected animals should be slaughtered and the carcasses should be burned or buried.

In endemic areas, the most commonly approved control mechanism is vaccination (Singh, 2012; FAO-OIE, 2015). Other general control measures include sanitary prophylaxis, strict quarantine and control of animal movements, monitoring of wild and captive animals i.e avoiding contact with sheep and goats (Dilli *et al.,* 2011; Singh, 2012). In Nigeria, various research findings recommend ethno-veterinary treatment as an acceptable means of managi-
ng PPR disease. The use of such preparations along with PPR vaccine to improve the efficacy of the treatment is acceptable among rural farmers (Dilli *et al.,* 2011; Luka *et al.,* 2011; Singh and Bandyopadhyay, 2015; Woma *et al.,* 2016).

In endemic areas PPR is controlled through administration of a live attenuated vaccine such as the Nigeria 75/1 strain (Luka *et al.,* 2011; Woma *et al.,* 2016). In India live-attenuated vaccines are currently licensed for use; Sungari 96, Arasur 87 and coimbatore 97 (Saravanan *et al.,* 2010; Singh and Bandyopadhyay, 2015). Historically, live attenuated rinderpest vaccines were also used to protect small ruminant against PPRV due to the antigenic cross-reactivity between the two viruses (Singh and Bandyopadhyay, 2015; Mantip *et al.,* 2019). However, use of the later was haltered to avoid false positive detection of RPV during the final stages of the eradication campaign and to enable a country to seek OIE recognition for freedom from rinderpest (OIE, 2013). The vaccine in current use in Nigeria is thermolabile, and it is necessary to maintain it in an effective cold chain, a condition that is sometimes difficult to achieve in many rural areas where the majority of susceptible animals are

kept. It is envisaged that this vaccine could be improved upon by using Xerovac technology to increase its thermostability. The thermolabile nature of the vaccine may increase the likelihood of low sero-conversion following vaccination with the vaccine (Luka *et al.,* 2011; Woma *et al.,* 2016; Mantip *et al.,* 2019). Faris *et al.,* (2012) reported a low conversion rate of 61%, indicating weak herd immunity in the flocks of sheep and goats vaccinated using the homologous PPR vaccine. Thus there is a need to increase the thermostability of the vaccine so as to boost the sero-conversion rate following vaccination with vaccine (Singh and Bandyopadhyay, 2015).

Control strategies that have been successful in ensuring the eradication of RPV are also valid for PPR (Banyard *et al.,* 2010; Dundon *et al.,* 2020) although small ruminant structures differ greatly to that of cattle. In areas where PPR is not endemic, outbreaks are controlled most efficiently through a number of methods including slaughter of infected herds, good sanitation, import control, movement restrictions and quarantine. However, lack of suitable facilities, including the provision of veterinary services, often preclude effective outbreak movement (FAO-OIE, 2015). Regardless of the level of viral presence within an area, sero-monitoring through surveillance initiatives, as used successfully during the different RPV eradication programme remain a critical tool in combating PPRV infection and preventing further spread (Dundon *et al.,* 2020).

Several studies have involved the development of new generation of marker vaccines that will enable the differentiation of infected or vaccinated animals (DIVA). The development of a rinderpest-based chimeric vaccine against PPRV has been attempted by swapping the glycoprotein gene (F and H) between PPRV and RPV. This RPV – PPRV FH recombinant virus grew poorly in cell culture (Kwiatek *et al.,* 2010; Mantip *et al.,* 2019). In other to improve the virus growth in the cell culture, M, F, and H genes from RPV were replaced with those from PPRV and a higher virus yield was obtained with the resulting chimera. In a pilot study, this chimeric virus protects goats from challenge with a virulent strain of PPRV and it was possible to differentiate between vaccinated and infected animals using the competitive c-ELISA and C-N-ELISA (Kwiatek *et al.,* 2010; Mantip *et al.,* 2019). Furthermore, development of a multivalent vaccine is essential in countries where political and economic infrastructures are unable to support concerted vaccination programmes (Kwiatek *et al.,* 2010; Gilli *et al.,* 2011).

2.17Management of Peste des Petits Ruminants

There is no specific treatment for PPR, however, treatment for bacterial (long acting oxytetracycline, chlortetracycline) and parasitic complications decreases mortality in affected flocks or herds (Dilli *et al.,* 2011; Balamurugan *et al.,* 2014). Fluid replacement therapy (B-complex and Dextrose saline) for 5-7 days and anti-diarrhoeal medicines are essential, which may be useful to reduce the severity of the disease. Treatment and management of clinical cases of PPR or in the event of outbreaks in

sheep and goats is necessary in order to minimize the economic losses to farmers (Dundon *et al.,* 2020).

The use of chemotherapeutic agents combined with hygiene, sanitation, isolation and quarantine of the infected animals has been observed to help in managing the disease (Balamurugan *et al.,* 2014; Dundon *et al.,* 2020).

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 Study Area

Yobe State is located in North-Eastern Nigeria created on August 27, 1991, with capital in Damaturu (Fig. 3.1). The state borders four states: Bauchi, Borno, Gombe, and Jigawa. It borders to the north the Diffa and Zinder Regions of Niger (YSG Diary, 2010). Because the state lies mainly in the dry savanna belt, conditions are hot and dry for most of the year, except in the southern part of the state which has more annual rainfall.It has a land area of 45,502 km^2, with human population of over 2,757,000 according to 2006 National Census (NPC, 2011; NPC, 2010; NBS, 2013).

Yobe state is an agricultural state; it also has rich mineral deposits, including gypsum and kaolin in Fune Local Government and very rich agricultural resources as well. The state's agricultural produce include gum arabic, groundnuts, beans, and cotton. The state also has one of the largest cattlemarkets in West Africa, located in Potiskum. The livestock population is 3.45million and 2.8million for goat and sheep respectively (YSG Diary, 2010).

Gulani is a Local Government Area in Yobe State with headquarters in Bara (Fig. 3.2). It lies within Coordinates: 11^0 00'N 11^0 43'E with headquarters is Bara town. It has an area of 2,090km^2 and a population of 103,510 as at the 2006 census (YSG Diary, 2010; NPC, 2010).

Figure 3.1: Map of Nigeria Showing Yobe State (coloured red).

Source; Research Gate (www.reserachgate.net)

Figure 3.2: Map of Yobe State Indicating Gulani Local Government Area.

Source: Research Gate (www.researchgate.net)

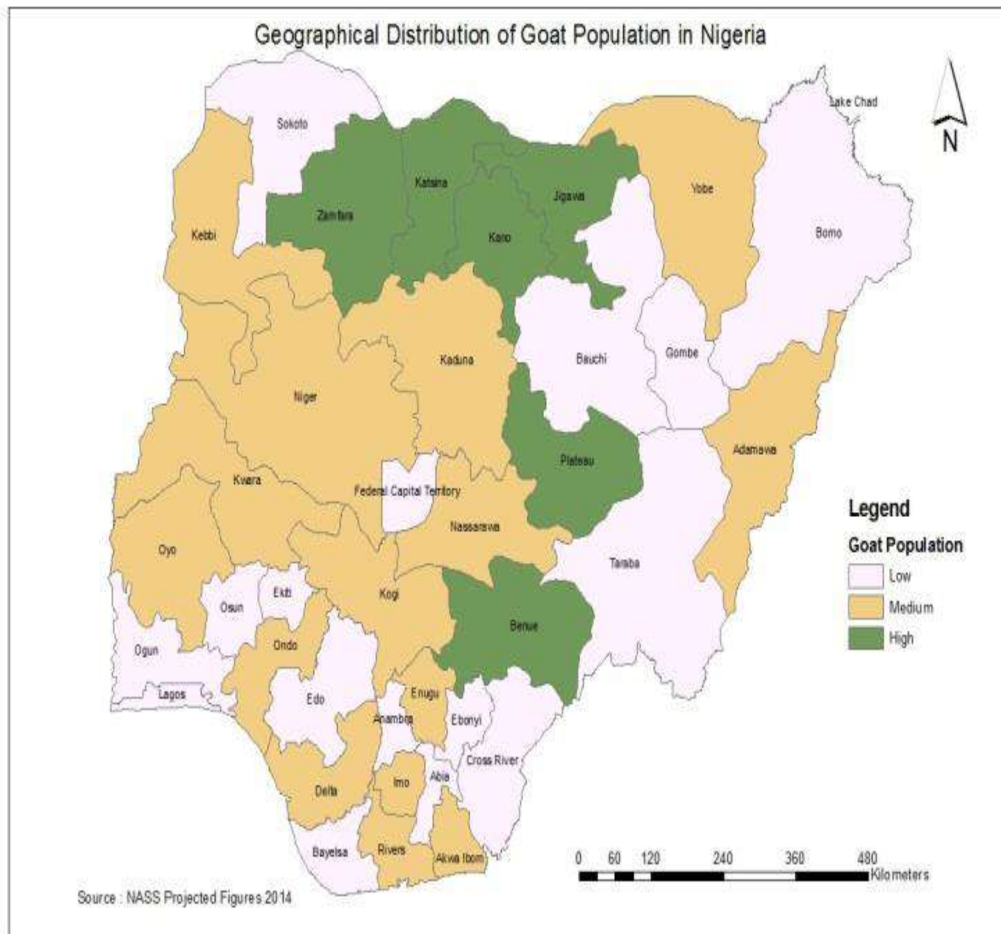

Figure 3.3: Map of Nigeria Showing the Distribution of Goat.

Source: NASS Projected Figures, 2014

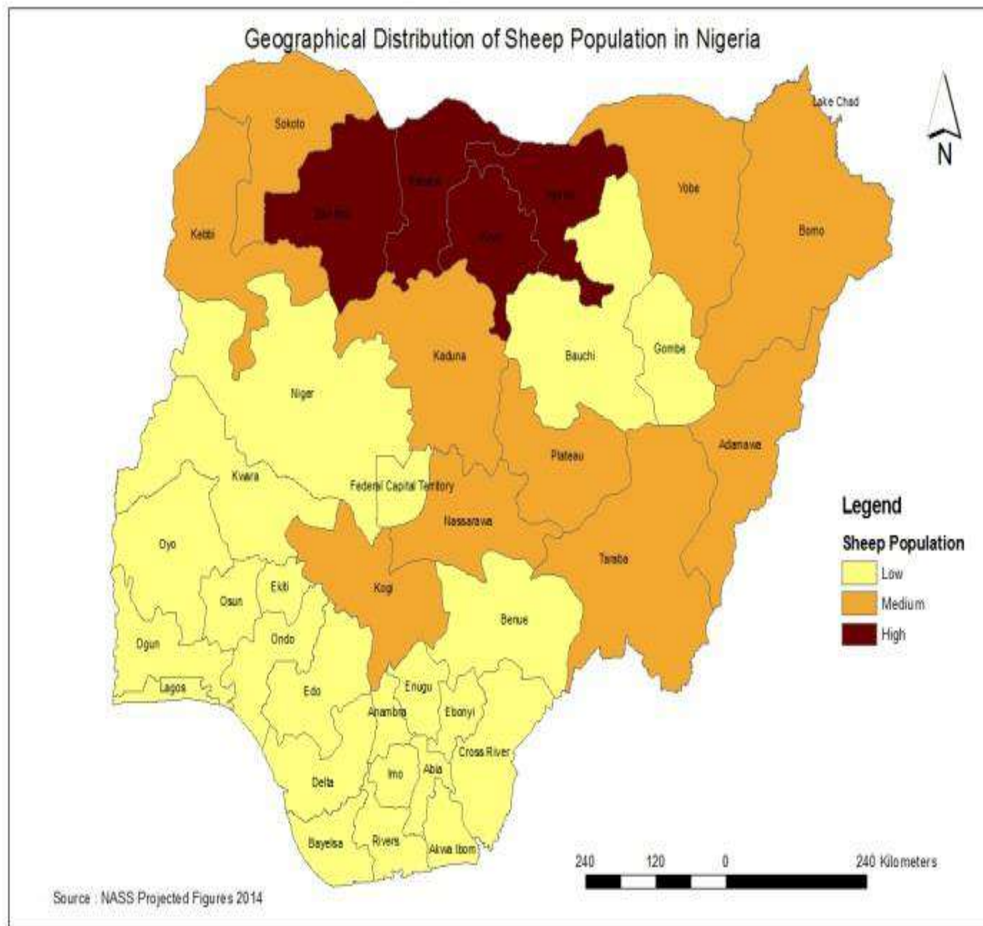

Figure 3.4: Map of Nigeria Showing the Distribution of Sheep

Source: NASS Projected Figures, 2014

3.2 Study Design

A cross sectional study was carried out in sheep and goats in Gulani Local Government Area, Yobe State during which six (6) villages namely; Kukuwa, Bularafa,Bumsa, Dutchi, Manawaji and Bursari were covered. The number of samples taken from each sampling unit depends on the flock size. One animal was sampled randomly out of every three animals for both sheep and goat. Therefore, a total of 153 sheep and 178 goats were sampled.

3.3 Sample Size Determination

Sample size was calculated using the formula by Thrushfield (2005) at 95% confidence level

$$n = \frac{Z^2PQ}{d^2}$$

Where,

n = sample size, Q = 1-P,

P = Prevalence (23.61 % in sheep and goats) (Woma *et al.,* 2016)

Z = confidence interval at 95% and d = level of significance (precision 0.05)

$$n = \frac{1.96^2 \times 0.2361 \times (1-0.2361)}{(0.05)^2}$$ = 271 (samples from sheep and goat).

Therefore, n = 271, using the prevalence rate of 23.61 %, in sheep and goats from different agro-ecological zones of Nigeria (Woma *et al.,* 2016). However, the sample size was increased by 9% to 331 to increase precision and for convenience.

3.4 Sampling Unit

The sampling units of the study are mainly household flocks and animals (sheep and goats) on free range within Gulani Local Government Area. Small ruminants considered for sampling in this study were those with no recent history of vaccination against PPR. This was to eliminate the possibility of seropositivity due to vaccinal antibodies. Also, in order to avoid seropositivity due to maternal antibodies, only sheep and goats of not less than six months old were considered for sampling in this study. The famer's and the flock owner's consents were sorted before the sample collection.

3.5 Sampling Procedure

A multi stage sampling technique was applied. Six wards were selected using simple random sampling base on the availability of sheep and goats from the 12 wards in Gulani Local Government Area. One village was further selected from each ward using convenient sampling method based on the availability of sheep and goats and also road accessibility to the study area.Therefore, a total of 6 villages were selected

for this study. Within each selected village, sheep and goats were sampled. The sampling units included household flocks, livestock markets and slaughter slabs. For each animal selected, variables such as species, sex, age, location, husbandry system, vaccination history, and breed were recorded.

3.6 Sample Collection

Three hundred and thirty one (331) serum samples were collected from both sheep and goats. Using a 10ml syringe, 6ml of blood was drawn from the jugular vein and transferred to a sampling bottle and allowed to stand for 24hrs to clot. Sera were then decanted and separated into sterile plain sample bottles containing well labeled sample number, flock location, age and sex. All samples collected were placed in cooler boxes and transported to the Virology Laboratory of the Department of Veterinary Public Health and Preventive Medicine, Ahmadu Bello University Zaria. Samples were then centrifuged at 1500 revolution for 10 minutes, and stored in serum tubes at -20^{0}C prior to the serological tests.

3.7 Laboratory Procedure

3.7.1 Competitive enzyme linked immunosobent assay (c-ELISA)

A commercially available c-ELISA kit (IDVET, France) for PPR was used for this study. This diagnostic kit detects antibodies directed against the nucleoproteins of PPRV and was developed by FAO Reference Laboratory (CIRAD-EMVT, Montpellier, France) (Kwiatek *et al.,* 2012; Comtet *et al.,* 2013). The test was

performed according to the Manufacturer's instructions (Kwiatek *et al.,* 2012). The ELISA micro plate was read with an immunoscan reader (Flow laboratories, UK) with a filter of 450nm. The optical density (OD) was recorded and the test was validated when the mean value of the negative control OD (ODNC) was greater than 0.7 and the mean value of the positive control OD (ODPC) was less than 0.3 of the ODNC. The OD values were converted to competition percentage (CP) using the following formula;

$CP = OD\ sample / ODNC \times 100.$

3.7.2 Description and principle

The procedure for the c-ELISA kit was done according to the manufacturer's (IDVET, France) instructions as described below. The wells are coated with purified recombinant PPR nucleoprotein (NP). The samples to be tested and the controls are added to the microwells. Anti-NP antibodies, if present, form antibody-antigen complex which mask the NP epitopes. An anti-NP-peroxidase (HRP) conjugate is added to the microwells. It fixes to the remaining free NP epitopes, forming an antigen-conjugate-HRP complex. Washing was done in other to eliminate the excess conjugate. The substrate solution tetramethyl benzidine (TMB) was added. The resulting coloration depended on the quality of specific antibodies present in the sample to be tested: In the presence of antibodies, a bluish solution appears which becomes yellowish after addition of the stop solution. In the absence of antibodies, no

coloration appears. The microplate is read at 450nm (Kwiatek *et al.,* 2012; Comtet *et al.,* 2013).

3.7.3 c-ELISA kit components

Microplates coated with PPR recombinant nucleoproteins

Anti-NP-HRP concentrated conjugate (10 ×)

Positive Control

Negative control

Dilution Buffer 13 {Phosphate Buffer Saline (PBS) 0.01 M, pH 7.4-7.6}

Dilution Buffer 4 {Phosphate Buffer Saline (PBS) 0;1 M at pH 7.4 with 0.01% between 20 and 0;3% negative sheep and goats serum supplied with the kit}

Wash Concentrate (20×),), 3 M Nacl, 0.005 M, Kcl, 0.13 M Na_2HPO_4, Polysorbate (Tween) 20, 0.005% pH 7.2

Substrate Solution

Stop solution (0.5 M H_2SO_4).

3.7.4 Sample preparation

In order to avoid differences in incubation times between samples, a 96-well plate containing the test and control samples was prepared, before transferring them into an ELISA microplate using a multi-channel pipette.

3.7.5 Wash solution preparation

The wash concentrate (20×) was brought to room temperature (21ºC ± 5ºC) and mixed thoroughly to ensure that the wash Concentrate was completely solubilized. The wash solution (1×) was prepared by diluting the wash Concentrate (20×) in distilled water.

3.7.6 Testing procedure

The reagents were brought to room temperature (21ºC) before use. The reagents were then homogenized by inversion.A volume of25 µl of Dilution Buffer 13 was added to each well and 25 µl of the Positive Control to well A1 and B1. Then25 µl of the Negative Control was added to well C1 and D1**.** 25 µl of sample to be tested was added to the remaining wells.The samples were then incubated for 45 min at 37ºC.Each of the wells was washed 3 times with approximately 300 µl of the wash solution. (Drying of the wells between washings was avoided).The conjugate × 1 was then prepared by diluting the conjugate 10× to 1/10 in Dilution Buffer. 100µl of the Conjugate 1× were added to each well.Then it was incubated for 30 min at 21ºC (Kwiatek *et al.,* 2012; Comtet *et al.,* 2013).

Each of the wells was washed 3 times with approximately 300µl of the wash solution. Drying of the wells between washings was avoided. Then 100µl of the Substrate

solution was also added to each well. Then it was incubated for 15 min at 21ºC in the dark, 100 µl of the Stop solution was also added to each well in order to stop the reaction. The result was then read and the O.D recorded at 450nm (Kwiatek *et al.*, 2012; Comtet *et al.*, 2013).

3.7.7 Validation of results

The test was acceptable if;

-The mean value of the Negative control O.D. (OD_{ND}) of a test already carried out was greater than 0.7 ($OD_{NC} > 0.700$)

-The mean value of the Positive control (OD_{PC}) of a test already carried out was less than 30% of the OD_{NC}. ($OD_{PC} / OD_{NC} < 0.3$)

3.7.8 Interpretation of results

For each sample of a test already carried out, Competition Percentage (S/N %) were calculated using

$$S/N\% = \frac{OD_{sample}}{OD_{NC}} X\ 100$$

Less than or equal to 50 % of a test already carried out were considered positive.

Greater than 50 % and less than or equal to 60 % of a test already carried out were considered doubtful.

Greater than 60 % of a tests already carried out were considered negative.

Summary of results

If the Competition Percentage (S/N %) is ≤ 50 %, the test was considered positive. If 50 % <the Competition Percentage (S/N %) ≤ 60 %, the test was considered doubtful. If the Competition Percentage (S/N %) is > 60 %, the test was considered negative.

3.8 Data Analysis

All data the collected were summarized and entered into Microsoft Excel 7 spreadsheet (Microsoft Corp.) and stored.

The prevalence of PPR was calculated using the formula;

Prevalence = Positive samples /Total samples analyzed × 100

Percent PPR prevalences were presented using descriptive statistics and in tables.

These data were analyzed using a Statistical Package for Social Sciences (SPSS) version 20.0. Pearson's Chi-square test was used to determine association between prevalence of PPR and risk factors such as species, breeds, sex, age, locations and husbandry management practices. A P ≤ 0.05 was considered significant.

CHAPTER FOUR

4.0 RESULTS

4.1 Specie-Based Seroprevalence of PPR in Sheep and Goats in Gulani LGA, Yobe State, Nigeria.

A total of 153 sheep were sampled and 73 were positive for PPR indicating a prevalence rate of 47.7%. A total of 178 goats were sampled and 96 were positive for PPR indicating a prevalence rate of 53.9%. An overall seroprevalence of 51.1% for both sheep and goats was obtained. However, there was no significant association (χ^2 =1.0372; P-value= 0.308) between seroprevalence of PPR and the two species (sheep and goats) (Table 4.1).

4.2 Seroprevalence of PPR Based on Sex and Age of Goats in Gulani LGA Yobe, State,Nigeria

A total of 178 sera were sampled with 38 male and 140 females' goats. The study showed higher prevalence of PPR antibodies in female goats 58.6% than male goats

36.8%. The prevalence in adult goats was 57.2% while in young goats, it was 42.5%. There was no association between seroprevalence of PPR and sex (χ^2 =4.839; p-value= 0.028) as well as age (χ^2 =2.1530; p-value= 0.142) of goats sampled. (Table 4.4).

4.3 Seroprevalence of PPR Based on Sex and Age of Sheep in Gulani LGA Yobe, State,Nigeria

A total of 153 sera were sampled comprising 48 male and 105 female sheep. The study showed higher prevalence of PPR antibodies in female (50.5%) than male sheep (41.7%). Similarly, out of 153 sheep that were sampled, 21 were young and 132 were adult sheep. Result showed higher prevalence in adult (51.5%) than young sheep (23.8%). There was no association between seroprevalence of PPR and sex (χ^2 =0.702; p-value= 0.402) as well as age (χ^2 =4.519; p-value= 0.034) of sheep sampled (Table 4.3).

4.4Seroprevalence of PPR Based on Previous Vaccination History in theflock of Sheep and Goats in Gulani LGA, Yobe State, Nigeria (n=331)

According to the previous vaccination history in the flock, 61 animals (sheep and goats) representing 18.4% of the total number of animals sampled (331) were vaccinated for PPR, while 270 animals representing 81.6% were not vaccinated (Table 4.4). A total of 23 (37.7%) animals (sheep and goats) from the vaccinated ones were seropositive for PPR antibodies, while 146 (54.1%) animals (sheep and goats) from the unvaccinated ones were seropositive for PPR antibodies (Table 4.4).

4.5 Seroprevalence of PPR Based on Previous Vaccination History in the flock of Sheep in Gulani LGA, Yobe State, Nigeria (n=153)

Results from Table 4.5 show the previous vaccination history for sheep with seroprevalence rate of 40.8% in the vaccinated population and 58.7% in the unvaccinated populations for PPR.

4.6 Seroprevalence of PPR Based on Previous Vaccination History in the flock of Goats in Gulani LGA, Yobe State, Nigeria (n=178)

Results from Table 4.6 show the previous vaccination history for goats with seroprevalence rate of 25.0% in the vaccinated population and 51.2% in the unvaccinated populations for PPR.

Table 4.1: Species-based seroprevalence of PPR in sheep and goats in Gulani LGA,Yobe State, Nigeria

Species	No. of animals sampled	No. of animals positive for PPR	Prevalence (%)
Sheep	153	73	47.7
Goats	178	96	53.9
Total	**331**	**169**	**51.1**

Chi square (χ^2) =1.0372; p-value= 0.308

Table 4.2: Seroprevalence of PPR based on sex and age of goats in Gulani LGA, Yobe State, Nigeria (n=178)

Variables	Frequency	Sero-positive (n)	% Sero-prevalence	χ^2	P-values
Sex				4.839	0.028
Male	38	14	36.8		
Female	140	82	58.6		
Age				2.153	0.142
Young	40	17	42.5		
Adult	138	79	57.2		
Total	**178**	**96**			

Table 4.3: Seroprevalence of PPR based on sex and age of sheep in Gulani LGA, Yobe State, Nigeria (n=153)

Variables	Frequency	Sero-positive (n)	% Sero-prevalence	χ^2	P-values
Sex				0.702	0.402
Male	48	20	41.7		
Female	105	53	50.5		
Age				4.519	0.034
Young	21	5	23.8		
Adult	132	68	51.5		
Total	**153**	**73**			

Table 4.4: Seroprevalence of PPR based onprevious vaccination history in the flock of sheep and goats in Gulani LGA, Yobe State, Nigeria (n=331)

S/No	Previous vaccination in the flock	No. of animal sampled	No. of animal positive for PPR	% of animal positive for PPR
1.	Yes	61	23	37.7
2.	No	270	146	54.1
Total		331	169	51.1

Table 4.5: Seroprevalence of PPR based on previous vaccination history in the flock of sheep in Gulani LGA, Yobe State, Nigeria(n=153)

S/No	Previous vaccination in the flock	No. of animal sampled	No. of animal positive for PPR	% of animal positive for PPR
1.	Yes	49	20	40.8
2.	No	104	61	58.7
Total		153	81	52.9

Chi square (χ^2) =0.425 p-value= 0.514

Table 4.6: Seroprevalence of PPR based on previous vaccination history in the flock of goats in Gulani LGA, Yobe State, Nigeria(n=178)

S/No	Previous vaccination in the flock	No. of animal sampled	No. of animal positive for PPR	% of animal positive for PPR
1.	Yes	12	3	25.0
2.	No	166	85	51.2
Total		178	88	49.4

Chi square (χ^2) =5.674; p-value= 0.017

4.7 Seroprevalence of PPR in sheep and goats based on locations in Gulani LGA, Yobe State, Nigeria

Out of the six villages that were sampled for this research work, Dutchi village had the highest prevalence rate of PPR (59.7%) while Kukawa had the lowest prevalence rate of PPR (34.5%). There was no association between seroprevalence of PPR and the locations sampled (P-value $\leq$ 0.05) (Fig. 4.1).

4.8 Breed-based Seroprevalence of PPR in Sheep in Gulani LGA, Yobe State,Nigeria

The highest seroprevalence of 50% was seen in Balami breed while Udda breed had the lowest seroprevalence rate of 43.5 %. There was no association between seroprevalence of PPR and breeds of sheep sampled (P-value $\leq$ 0.05). (Fig. 4.2)

4.9 Breed-based Seroprevalence of PPR in Goats in Gulani LGA of Yobe State, Nigeria

Sahelian breed showed higher seroprevalence rate of 57.1% compared with Sokoto Red with seroprevalence of 52.9%. There was no association between seroprevalence of PPR and breeds of goats sampled (P-value $\leq$ 0.05). (Fig. 4.3)

4.10 Seroprevalence of PPR Based on Husbandry Practices of Sheep and Goats in GulaniLGA, Yobe State, Nigeria

Prevalence (PPR) of 52.8%, 48.5% and 47.6% for semi-intensive, extensive and intensive system of husbandry practices respectively were obtained. There was no statistical significance for PPR based on husbandry practice (P-value $\leq$ 0.05). (Fig. 4.4);

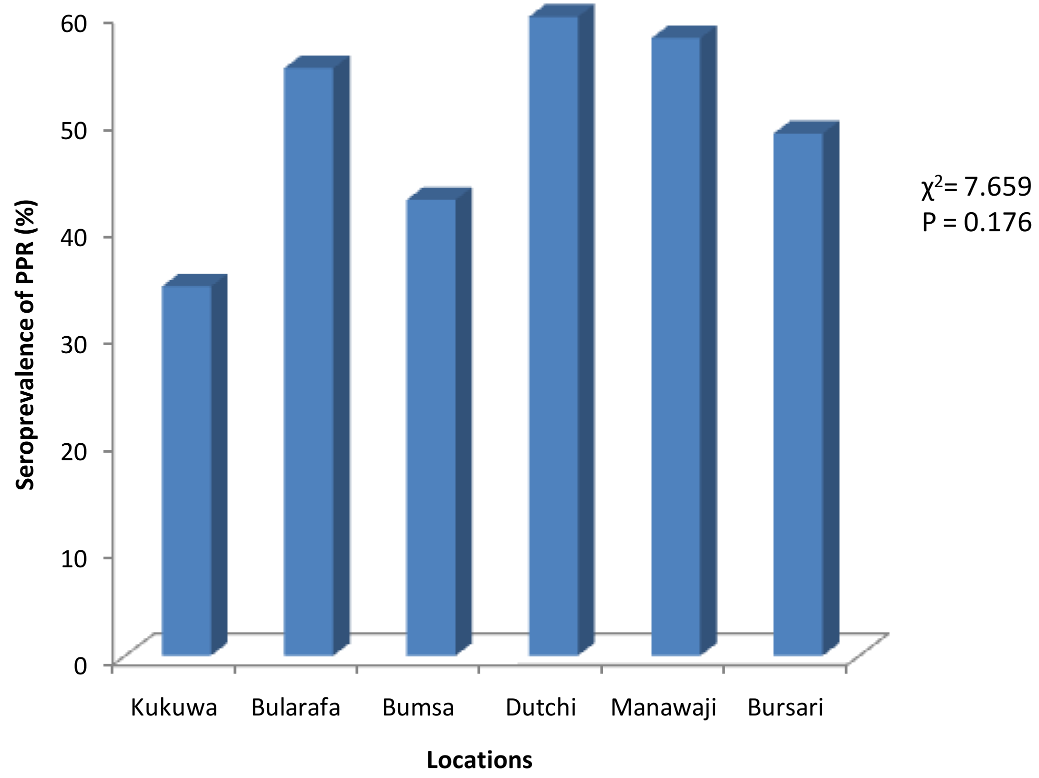

Figure 4.1: Seroprevalence of PPR in sheep and goats based on different locations in Gulani LGA of Yobe State, Nigeria

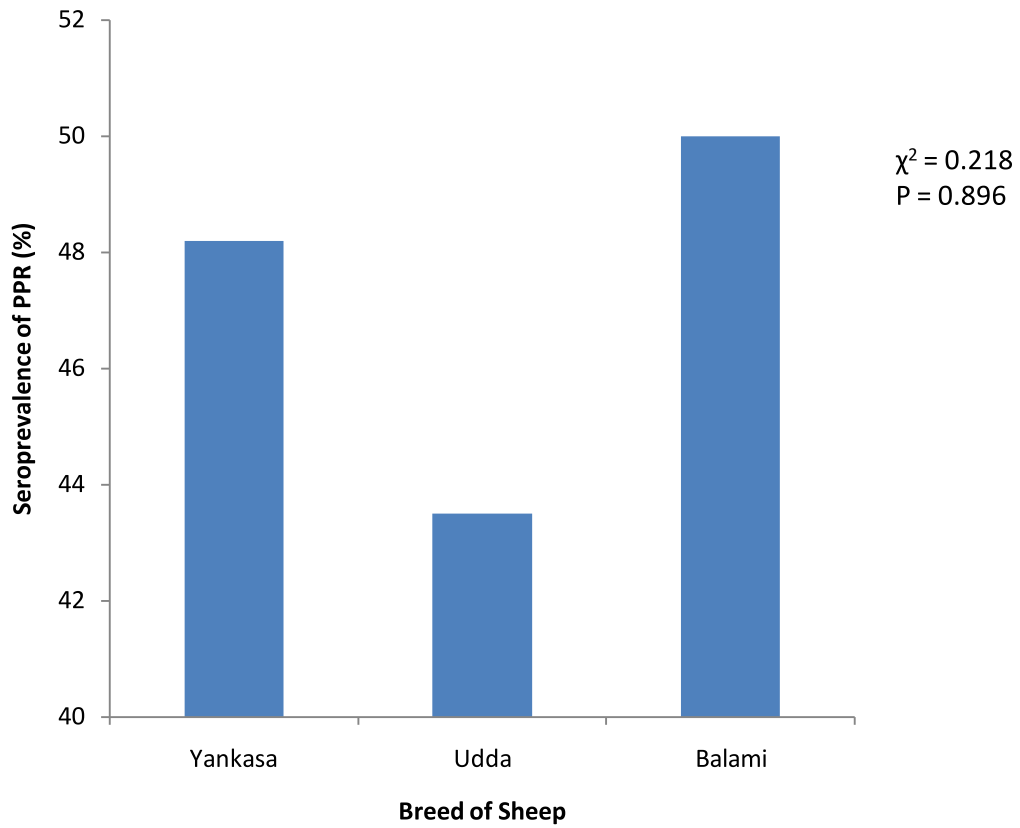

Figure 4.2:Breed-based seroprevalence of PPR in sheep in Gulani LGA of Yobe State, Nigeria

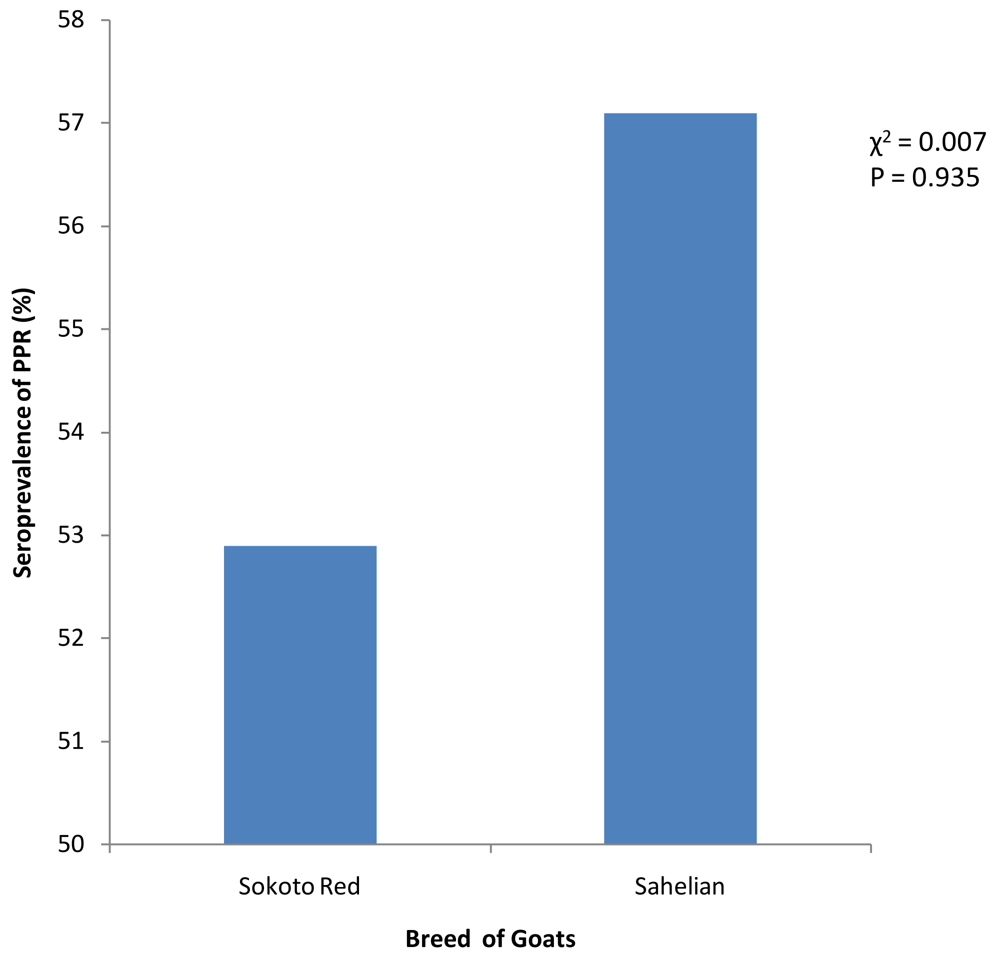

Figure 4.3:Breed-based seroprevalence of PPR in goats in Gulani LGA of Yobe State, Nigeria

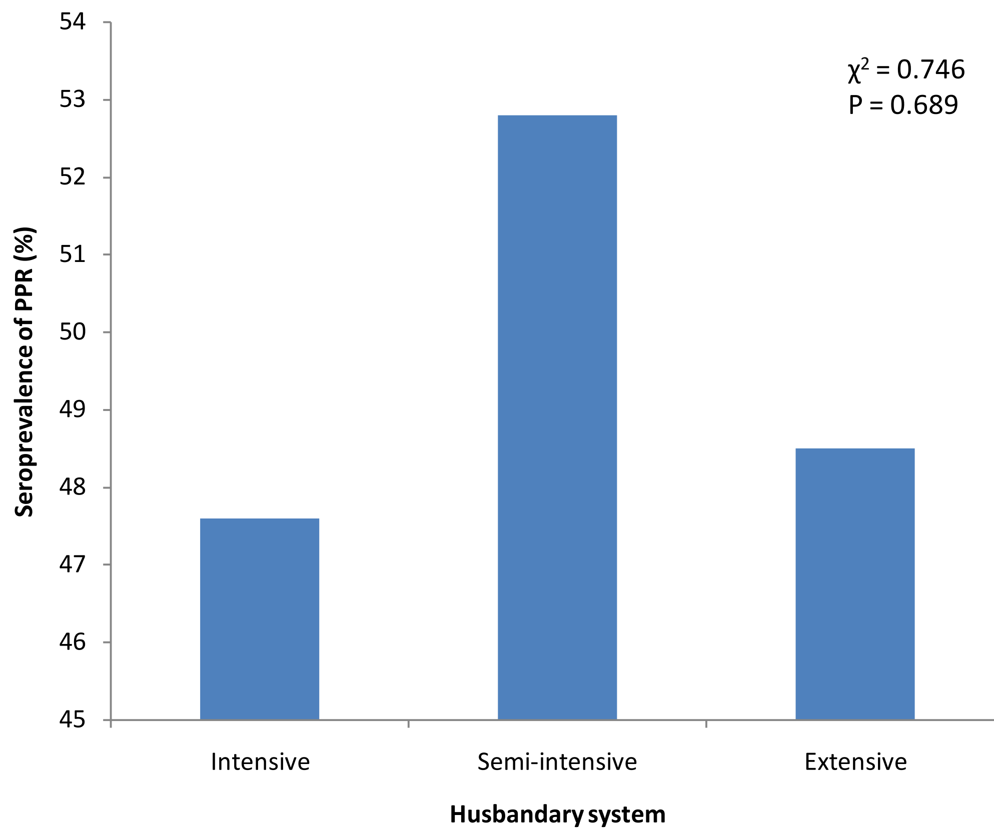

Figure 4.4: Seroprevalence of PPR based on husbandry practices of sheep and goats in Gulani LGA of Yobe State, Nigeria

CHAPTER FIVE

5.0 DISCUSSION

Peste des Petits Ruminants (PPR) is an acute and highly contagious viral disease that affects both domestic and wild small ruminants (Parida *et al.,* 2015; OIE, 2019). PPR is one of the economically important diseases of small ruminants with variable prevalence in several parts of Asia and Africa, and is increasingly becoming important where small ruminants contribute in no small measure to food security (Banyard *et al.,* 2010; OIE, 2016). In Nigeria, PPR is regarded as the most important disease of small ruminants because the economic losses due to the disease are enormous. In addition, the disease is considered as a militating factor against livestock production in Nigeria (Emikpe and Akpavie, 2010; Balogun *et al.,* 2017).

Serological results from this study showed the overall seroprevalence of PPR is 51.1% in sheep and goats in selected areas of Gulani Local Government Area of Yobe State (Table 4.1). The high prevalence of PPR virus antibodies recorded in this study could be attributed to the geographic location of the state. Yobe state shares a common border with Niger republic which is also a PPR endemic country (Dundon *et al.,* 2020). There is cross border nomadic movement and migration of small ruminants from Niger Republic into the state, which could increase the chances of contact with local sheep and goats thereby facilitating the transmission of PPR. Also, Yobe state has high population of livestock, which may result in over-crowding of animals during grazing or feeding, and subsequently increases chances of PPR transmission (Bukar *et*

al., 2019. The results may suggest very high activity of PPRV among the animals and indirectly pointing how vulnerable the small ruminants of Gulani Local Government Area could be to other secondary infections, because PPRV, like most other morbilliviruses, is reported to induce immune-suppression in its natural hosts (Bukar *et al.,* 2019). This finding is in agreement with the 51.3% reported by Victor *et al.* (2017) in Makurdi, Benue state, but lower than the 55% by reported by El-Yuguda *et al*. (2013) in semi-arid region of North-eastern Nigeria, 55.4% by Bukar *et al.* (2019) in Yobe state and 63.2% reported by Mulindwa *et al.* (2011) in Karamoja subregion of Uganda. However, the result is higher than the 23.16% observed by Woma *et al.* (2016) in agro-ecological zones of Nigeria, 24.5% reported by Balarumugan *et al.* (2012) in India and 38.2 % observed by Mahmoud *et al.* (2016) in Hail, Saudi Arabia.. The differences in the seroprevalence of the PPR may be due to differences in locations, differences sampling frames, seasonal variation, differences in sampling techniques and laboratory tests used. Another reason could also be due to the fact that the previous studies might have collected samples during outbreaks, during which most of the animals are habouring the virus.

According to locations showed the highest seroprevalence rate of PPR was observed in Dutchi (59.7%), followed by Manawaji, Bularafa and the least was Kukawa (34.5%) (Figure 1). There was no association between seroprevalence of PPR and the locations sampled (P-value ≤ 0.05). Dutchi is the smallest town among the different locations that samples were taken, but with a large population of livestock. This

encourages over-crowding especially and sharing of common water drinking points at the village entrance, which may subsequently increase the transmission and spread of the PPR virus among the animals. However, Kukuwa town which is a larger town among the different locations has low population density of livestock recorded, hence the low seroprevalence of PPR in that area.

The results from this study also showed that the prevalence of PPR is higher in goats (53.9%) than sheep (47.7%), although there was no significant difference ($P \leq 0.05$) (Table 4.1). These varying results show that the sensitivity to PPR is not necessarily linked to the given species of small ruminants, but rather to rearing conditions and other individual factors. This rate of variation among species may be related to the fact that sheep succumb easily to drought and other environmental stresses, and may mean that goats are more susceptible to PPR than Sheep as has been reported by several authors. Also genetic variation may be a factor in the high prevalence in goats which make them more susceptible to the PPR virus than sheep. In Nigeria goats are affected more severely to PPR virus exposure compared to sheep and they exhibit striking clinical sign while sheep undergo mild form of the disease (Victor *et al.,* 2017; Bukar *et al.,* 2019). The seroprevalence in goats (53.9%) was not significantly higher than sheep (47.7%), which may resulted from equal exposure of sheep and goat because they are herded together and communal grazing. The specie variation in the prevalence of PPR among goats and sheep was also reported by El-Yuguda *et al.* (2009) in Maiduguri (75% in goats and 60% in sheep), El-Yuguda *et al.* (2013) in semi-arid

zones of North-eastern Nigeria ((goats-64.8% and sheep-63.8%) and Gari *et al.* (2017) inEast Shewa and Arsi Zones, Oromia Region, Ethiopia (sheep-50.85% and goats-46.68%). Elsewhere in India, Hail, Saudi Arabia and AgroclimaticZones of Odisha, Balarumugan *et al.* (2012), Mahmoud *et al.* (2016) and Hota *et al.* (2017) found goats to be more infected with PPRV than goats with a prevalence of 24.5% in sheep and 38.2% goats, 33.2% in sheep and 62.9% in goats and 44.7% in sheep and 45.74% in goats respectiv ely. The presence of PPR antibodies in the sheep may be because sheep only suffer mild to inapparent infection with the virus or this may be attributed to a higher recovery rate and a greater longevity of sheep verses goats which is similar to the serological profile reported earlier by some workers (El-Yuguda *et al.,* 2013).

Base on the sex of the animal examined, the seroprevalence of PPR was observed to be significantly higher ($P < 0.05$) in female goats (58.6%) than male goats (36.8%) (Table 4.2). The result is also similar with the sheep in which female sheep recorded a higher prevalence of 50.5% than the males with 41.7% (Table 4.3). The reason for the higher prevalence in females could be due to the fact that females are usually kept longer in the flock for the breeding purposes while males either sold out or slaughtered for meat purposes. Another possible reason is that when females become pregnant, and then lambed/kidded, their immune status becomes lowered as a result their ability to resist the challenges of infection is lowered. This predisposed them to infection by PPR virus. The seroprevalence across sex variation in the current study is comparable

to the report by Woma *et al.* (2016) in agro-climatic zones of Nigeria, Gari *et al.* (2017) InEast Shewa and Arsi Zones, Victor *et al.* (2017) in Makurdi, Benue state, Oromia Region, Ethiopia, Hota *et al.* (2018) in different agroclimatic zones of Odisha and several others who showed that females small ruminants are more susceptible to PPR virus than their male counterparts.

The seroprevalence of PPR across age showed that both adult sheep and goats had higher seroprevalence of PPR antibodies compared to the young sheep and goats with 79% and 17% for adult goats and young goats; and 68% and 5% for adult sheep and young sheep respectively (Tables 4.2 and 4.3). This can be attributed to waning of the maternal immunity in young animals older than 4 months of age and the absence of PPR antibodies in the serum of older animals never exposed to or vaccinated against the disease. Low prevalence was found in kids and lambs ageing 1 to 3 months. This can be attributed to the presence of maternal colustral antibodies. This finding is in agreement with the observation of Victor *et al.* (2017) in Makurdi, Benue state who observed that animals older than 12 months of age (adults) had the highest prevalence (60%) than those from 4 to 12 months of age (young ones). It is also similar to the report by Gari *et al.* (2017) who showed that adult goats and sheep had a seroprevalance of 74.5% and 56.0% higher than the young goats and sheep with 31.6% and 38.1% respectively. However, the present study contradict those of Woma *et al.* (2016) who showed that even though the adults animals (age > 1 year) had higher seroprevalnce of 22.55% compare to 24.40% seroprevalnce in young animals

(age 3 and 12 months), there was no significant difference between the seroprevalence of PPRV in small ruminants over 1 year of age and those between 3 and 12 months of age (p = 0.323). The present study contradict that of Bukar *et al.* (2020) who reported the age-wise distribution of the PPRV seroprevalence to be 65.2% for Sahel goats less than 18 months of age (young animals) and 35.3% for Sahel goats older than 18 months (adults). On the other hand, El-Yuguda *et al.* (2009) observed a similar pattern in goats with the current study with the PPR antibodies among goats > 2 years old (adults) and 1-2 years old(adults) higher with (72% and 83%) than goats of < 1 year old (young) with 58% seroprevalence; but the sheep showed higher prevalence of 80%among the < 1 year old (young), than 60% and 40% recorded by sheep of >2 years old (adults) and those of 1-2 years old (adults).

Base on the different breeds of sheep and goats examined, the highest seroprevalence of 50% was seen in Balami breed of sheep, followed by Yankasa breed with 48.2%, while the Udda breed had the lowest seroprevalence rate of 43.5 % (Fig. 4.2). While in goats, the Sahelian breed showed higher seroprevalence rate of 57.1% compared with Sokoto Red with seroprevalence of 52.9%. There was no association between seroprevalence of PPR and breed of goats sampled (P-value $\leq$ 0.05) (Fig. 4.3). Genetic factors such as breed predisposition may be the major factors that cause the variation in the seroprevalence rates among different breeds observed in this study. It is possible that Balami breeds of sheep and the Sahelian breed of goats are genetically more susceptible to PPR infection than the other breeds examined this study. A

comprehensive animal trial should be performed to further elucidate the susceptibility of different breeds of sheep and goats to virulent PPR purposes in the study area. The finding from this study on seroprevalence differences among different breeds is similar to that reported by Victor *et al.* (2017) in Makurdi, Benue state. Their report showed that the West African Dwarf (WAD) breed of goat had higher PPR prevalence (69.7%) than the Sokoto red (67.5%) and Sahel (22.2%) breeds of goat, while, the Yankasa breed of sheep was found to have a higher prevalence (30.8%) than the West African Dwarf (WAD) breed sheep (29.2%). Also, Bello *et al.* (2018) in Sokoto state found the Red Sokoto goats with 47.52% to have significantly greater seroprevalence of PPR virus antibodies than other breeds (Sahelian and Mixed). A study by Bukar *et al.* (2017) showed high prevalence of PPRV antibodies among sahel goats in Yobe state, which was attributed to the high population of small ruminants in the state. This encourage crowding of animals and subsequent disease transmission and spread. It also showed high activity of PPRV among the sahel goats, and indirectly pointed at how vulnerable the small ruminant population of Yobe state could be to other secondary infections, because PPRV, like most other morbilliviruses, is reported to induce immune-suppression in its natural hosts. The distribution of PPR seroprevalence in Yobe State, Nigeria as observed in this study shows that the disease is endemic in this part of the country.

In this study animals reared under semi-intensive and extensive husbandry practices had higher seroprevalence of 52.8% and 48.5% when compare with intensive system

of husbandry practices with 47.6% (Fig. 4.4). Though, there was no statistical significance for PPR based on husbandry practice (P-value $\leq$0.05) (Fig. 4.4). Livestock owners in Gulani L.G.A mostly practice semi-intensive system of husbandry practice where livestock are allowed to graze in the open farms and bushes during the day time and taken home in the evening. In most cases, livestock are given supplementary feed in the evening while at home. A usual practice of taking animals to a common drinking point during day time and introduction of new animals in to the flock without quarantine is believed to increase the risk of the disease. Seasonal calendar such as onset of rainy season, cold dry harmatan is also believed to be associated with higher prevalence. This trend could be attributed to the restricted movements of the animals kept under the intensive system of husbandry practices as well as to the vulnerability of small ruminant herds in semi-intensive and extensive husbandry practices to infected herds in pastures and water points. This was similar findings of Victor *et al.* (2017) in Makurdi, Benue state who observed that animals reared under open grazing and pastoralist systems had higher prevalence, with prevalence rates of 71.2% and 63.6% respectively. This finding is also in agreement with the observations of Saeed *et al.* (2010). Their study found seroprevalence of PPR to differ significantly between housing categories. Among the animal housing types, the animals without housing had the highest PPR prevalence (70.4%) followed by animals in barbed wire/wire mesh, shrub and scrap fences with prevalence rates of 52.9%, 58.3% and 52.4%. The prevalence rate in animals kept in modern houses with

fences was found to be low. Animals kept in mud and brick houses had the lowest prevalence rates of 27.9% and 20% respectively.

According to the previous vaccination history in the flock, 61 animals (sheep and goats) representing 18.4% of the total number of animals sampled (331) were vaccinated for PPR, while 270 animals representing 81.6% were not vaccinated (Table 4.4). It was also observed that 23 (37.7%) of the vaccinated animals (sheep and goats) were seropositive for PPR antibodies, while 146 (54.1%) animals (sheep and goats) from the unvaccinated ones were seropositive for PPR antibodies (Table 4.4).The previous vaccination history for sheep showed that 40.8% of the vaccinated population was seroppositive, while 58.7% of the unvaccinated population was seropositive for PPR. Likewise, the previous vaccination history for goats showed that 25.0% of the vaccinated population was seropositive, while 51.2% of the unvaccinated populations for PPR were also seropositive (Table 4.6).However, the seroprevalence in the vaccinated populations cannot be further verified as the c-ELISA kit does not differentiate between antibodies due to vaccination or natural infection. The lack of vaccination exposes a larger population of livestock to natural infections and possible outbreaks. Since the greater percentage of the animals was not vaccinated against PPR, the antibodies could only have come as a result of subclinical infection as earlier observed by El-Yuguda *et al.* (2009). This outcome is consistent with the report in Markudi, Benue state in which vaccinated animals with medication history have lower

rate of PPR (43.6%) compare to those with no vaccination history but have some medication history (73.3%) (Voctor *et al.,* 2017).

Mostly endemicity of PPR in small ruminants pertains to transmission of infection, either through close interaction of healthy animals with the affected ones or due to nomadic grazing and Transboundary movement of affected animals. Spread of infection also occurs via infected materials, fomites, contaminated water and feed troughs and bedding etc. (Banyard *et al.,* 2010). The disease causes a high morbidity (100%) and mortality (20-90%) among small ruminants (Balamurugan *et al.,* 2010; King *et al.,* 2012). The matter of concern is about the dependence of millions of small, marginal, landless farmers upon these animals for their livelihood and daily earnings, for which these animals are considered as poor man's cow in India. The predisposing factors towards the prevailing infection can be linked with poor nutritional management, environmental stress, concurrent parasitic and/or bacterial infection, ultimately enhancing the severity of the disease (Elsawalhy*et al.,* 2010;Dundong *et al.,* 2011). Frequent economic losses in Nigeria have been claimed by several authors due to this disease (FAO, 2013; Jones *et al.*, 2020). In order to check further losses, it is necessary to take preventive and control measures in endemic areas. Also trade limitation is another constrain to the country's economy, calls for a global eradication program which has already been started by OIE and FAO (FAO, 2015; OIE, 2016). Implementation of extensive vaccination programme using a thermostable vaccine and

mass awareness camps could be the ideal approach to minimize the infection and losses.

CHAPTER SIX

6.0 CONCLUSION AND RECOMMENDATIONS

6.1 Conclusion

From this study, it was concluded that small ruminants in Gulani Local Government Area of Yobe state are highly infected with PPR virus with an overall seroprevalence of 51.1%. This is of great concern to livestock industry owing to the economic importance of the disease in Nigeria. Although, all breeds of the small ruminants were observed to have been infected significantly with the PPRV, but the Balami breed of sheep and the Sahelian breed of goats are the most infected ruminants. Similarly, the

infection with PPRV was higher in goats than in sheep, higher in the females than males, and also higher in unvaccinated animals than vaccinated ones. The detection of PPRV antibodies among the unvaccinated small ruminants in Gulani local Government Area of Yobe state is a clear indication of the active circulation of the virus, and confirms the endemicity of the disease in Gulani local Government Area of Yobe state. Epidemiologic variables such as age, sex, species and breeds may serve as risk factors that determine the outcome of the disease in the study area. Therefore, control efforts for PPR particularly vaccination with PPR homologous vaccine should be intensified in the study area. The antibodies to PPR virus observed in this study were neither due to vaccination nor maternal antibodies. They must have therefore been due to natural infection with field PPR virus.

Because of the virulent nature of PPR in the majority of infected sheep and goats, most especially in unvaccinated animals, PPR is still considered a disease of higher economic importance than other small ruminant diseases in PPR endemic areas of Gulani local Government Area of Yobe state. Active surveillance for PPR is not carried out and the economic impact of the disease on the Gulani local Government Area of Yobe state livestock industry is thus difficult to precisely quantify. Nevertheless, not all farmers choose to vaccinate their animals and outbreaks of PPR amongst unvaccinated sheep and goats continue to occur on an annual basis. Clinical disease amongst unvaccinated sheep and goats in the field can be severe, and it is not uncommon for sheep and goat farmers in Gulani local Government Area of Yobe state

to suffer significant losses amongst their flocks as a result of seasonal outbreaks of PPR.

6.2 Recommendations

1. Stamping out of the exposed and the infected animals, and the carcasses burnt or buried due to the high costs associated with compensation as recommended by OIE should be adopted.
2. A strategic vaccination campaign should be adopted across Gulani Local Government Area and Yobe state at large. This can be achieved by the use of a Nigeria 75/1 live PPR vaccine produced by Nigerian Veterinary Research Institute (N.V.R.I) Vom that is highly efficacious in protecting animals against all PPRV strains.
3. An epidemiological participatory technique, to assess livestock owners' perceptions of vaccination success and serological surveys at a defined time period after vaccination to determine vaccinated animals' immune response, should be adopted.
4. A periodic surveillance to understand the epidemiological situation to help define the status of PPR Gulani Local Government Area and Yobe state at large should be adopted. The purpose of this is to determine the early detection of the appearance of the disease or virus incursion; to demonstrate the absence of clinical disease or infection with PPRV; and to subsequently determine and monitor the prevalence, distribution and occurrence of the disease or infection across Gulani Local Government Area and Yobe state at large.

5. Monitoring of transboundary migration and movements of livestock across the Niger Republic border or strict quarantine of such livestock where applicable to limit transmission and spread of the PPRV.
6. Proper and effective husbandry practices, including proper sanitary practice and hygiene should be adopted.

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APPENDICES

Appendix I

Sample Bottles, Cooler and Ice Pack for collection and transportation of samples

Appendix II

c-ELISA Kit for PPR

Appendix III

Sheep and Goat on Free Range in Gulani LGA

Appendix IV

Blood sample collection from jugular vein of goat

Appendix V

Pastoralist in Gulani LGA

Appendix VI

Micro-Titre Plate Reader

Appendix VII

Micro-Titre Plate

Appendix VIII

Blood Samples Collected in Syringe in Gulani LGA