THE EPIDEMIOLOGY OF PESTE DES PETITS RUMINANTS (PPR) IN GOATS IN ADAMAWA STATE

BY

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DECLARATION

I hereby declare that the work presented in this thesis is original. It was carried out by me under the supervision of Prof. J.U. Umoh, Dr. J.K.P. Kwaga and Dr. K.A. Majiyagbe in the Department of Veterinary Public Health and Preventive Medicine, Ahmadu Bello University, Zaria. Reference is made to work of other investigators and are duly acknowledged. No part of this thesis has been submitted elsewhere for a degree or diploma.

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CERTIFICATION

This thesis entitled **"The epidemiology of PPR in goats in Adamawa State"** by Qadeer Muhammad Abdul, meets the regulation, governing the award of Master of Science Degree of Ahmadu Bello University, Zaria and is approved for its contribution to Scientific knowledge and literary presentation.

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DEDICATION

To the Memories of Aishatu Aliyu and Abdulkadir (Faisal) who died during the course of my project work. May their Souls rest in peace, Amin.

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ABSTRACT

The point prevalence rate of antibodies to peste des petits ruminants (PPR) in 8 local government areas of Adamawa State was determined by competitive Enzyme linked immunosorbent assay. A total of 280 goat sera were sampled, out of which 176 (62.9%) tested positive to PPR antibodies. The antibodies to PPR were detected in all the local government areas of the State with Mubi having the highest prevalence rate of 76.0% and Jada had the least of 55.3%. There was no significant difference between the various local government areas ($P > 0.05$),The prevalence rate for males (53.6%) was significantly different from that of the females (69.0%). The age-specific rate for 0-6 months age group was 43.4%, for the 7-12 months, 54.2% and those greater than 12 months was 87.6%. There was a significant difference in age specific rate ($P < 0.05$). Males that aged 0-6 months had an age specific prevalence rate of 45.2%, whereas females of the same age had 40.0%. Males aged between 7-12 months had 44.7%, whereas females of the same category had 62.0%. Males above 12 months had an age specific prevalence rate of 77.4%, similarly females of the same age had 93.2%. There was a statistical significant difference in the age - sex specific rates. Red Sokoto goats had a breed specific rates of 52.6% and Sahelian had 52.3%. Even though there was no statistically significant difference between these breeds, there was a difference in breed specific rate between the pure breeds and their crosses; mixed breeds had a breed specific rate of 87.6%.

The prevalence rate of antigens to PPR in 9 reported outbreaks in the state was also determined using immunocapture enzyme linked immunosorbent (I (-F.LISA). Seventy five out of the 109 samples tested (68.8%) were positive to PPR antigens. About 82.7% (62 out of 75) of the "suspects" tested positive and 38.2% (13 out of the 34) of the apparently

healthy incontact goats tested positive. There was significant difference in the percentage positive tested from suspects and normals.(X^2= 5.64, ($p < 0.05$). The sex-specific rate for males was 75.9% and females 61.8%. The age group 0-6 months had the highest age-speeifie rate of 89.5% tested positive; 7-12 months had 61.7% and those greater than one year had 50.0%. There was a statistically significant difference in age-specific rates of detection rate of antigens of PPR. Breed specific rates to PPR antigens in the reported outbreaks indicated that red sokoto had 82.4% and sahelian breeds had 82.9%, whereas their mixed breeds had the least breed specific rate 45.0%. There was a statistically significant difference between the pure and cross breds ($P < 0.05$). There were four reported outbreaks in the rainy season and 5 in the dry season. More death occurred in the dry season (52.8%), than in the rainy season (47.2%). There was a statistical significant difference in mortalities between the two seasons.

The overall finding in this study indicated the presence of the disease (PPR) in Adamawa State with the conclusion that PPR may be a major cause of mortality limiting goats production.

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CHAPTER ONE

INTRODUCTION

Peste des petits ruminants (PPR) also known as stomatitis pneumoenteritis complex (SPC) or small ruminant plague is caused by an infectious PPR virus, a member of morbillivirus.

It is mainly a disease of sheep and goats that is of economic significance in Nigeria. It is probably the major cause of goat mortality in the country (Akerejola *et al*., 1979). The population of sheep and goats in Nigeria has been estimated at 22.104 and 34.495 millions respectively (FDLPCS, 1991).

Small ruminants provide sources of revenue to both government and people, employment to the rural populace, milk, meat, skin for leather industry and manure for crop production (Brumby, 1990). While sheep and goats suffer from many diseases, PPR is considered the most single cause of constraints to small ruminant production (Akerejola *et al*., 1979).

The disease PPR, alone can decimate whole flock or in combination with other diseases like pneumonia or gastrointestinal parasitism (Majiyagbe *et al*., 1992). PPR is a contagious and acutely devastating viral disease of sheep and goats endemic to the sub-humid region of West Africa. Current global distribution of the disease shows that it extends along a belt across Africa immediately South of the Sahara, extending into the Arabian Peninsula (Majiyagbe *et al*., 1992). PPR has been reported in the United Arab Emirate and Sultanate of Oman (Taylor *et al*., 1990). Majiyagbe *et al*., (1992), reported an overall prevalence rate of PPR in a clinically normal small ruminants in Camerouns and Nigeria to be 46%. Shamaki *et al*. (1996), found a prevalence rates of 38.4%, 40.6%, 40.0%, 7.7% in Oyo, Borno, Plateau and Abia States, respectively.

Literature on the epidemiology of PPR is very scanty in Nigeria and there are only few documented well-researched investigations into the occurrence of this disease in the country.

There have been field reports of PPR in the country and Adamawa State in particular but no concrete epidemiological data to support its occurrence.

It was against this background that the present study was conceived with the following objectives:-

(a) To establish an epidemiological data base of PPR in Adamawa State through serological detection of antibodies in goats sera.

(b) To study documented outbreaks of PPR through the detection of PPR antigens under field conditions.

(c) To determine infection rate among the incontact apparently healthy goats.

CHAPTER TWO

LITERATURE REVIEW

2.0 Definition

Peste des petits ruminants (PPR) is a highly contagious, infectious and fatal rinderpest like disease of small ruminants and some wildlife species (Furley *et al.*, 1987), caused by a virus that belongs to the morbillivirus genus of the family *Paramyxoviridae* (Gibbs *et al.*, 1979), which occurs in West Africa, the Middle East and the Indian subcontinent (Amjad *et al.*, 1996). The clinical signs and lesions of the disease are those of respiratory and alimentary systems.

2.1 Historical and geographical distribution of PPR

PPR was first observed in 1940 in sheep and goats in Ivory Coast (Gargadennec and Lalanne, 1942).

PPR was initially thought to be caused by a goat strain of rinderpest virus before the primary etiological agent of PPR was first elucidated by Gilbert and Monnier (1962) in Senegal. PPR has been described in Nigeria (Whitney *et al.*, 1967; Hamdy *et al.*, 1976; Mornet *et al.*, 1956), Sudan (El-Hag and Taylor, 1984), Ghana (Bonniwell, 1980), Sultanate of Oman (Hedger *et al.*, 1980). A PPR like disease in small ruminants which has not been confirmed by virus isolation has also been reported from other countries such as Benin Republic (Bourdin *et al.*, 1970; Chad Provost *et al.*, 1972); Saudi Arabia (Asmar *et al.*, 1980), Ethiopia (Roeder *et al.*, 1994). Niger (Taylor, 1984); Yemen Arab Republic (Taylor, 1984); Togo and Mali (Benazet, 1973); Mauritania and Upper Volta (Taylor, 1984) and Indian subcontinent, (Amjad *et al.*, 1996).

In Nigeria, the disease has been referred to by various names such as "Kata" (Whitney *et al.*, 1967; Rowland *et al.*, 1971), stomatitis pneumoenteritis complex (Johnson and Ritchie, 1968; Durtnell and Eid, 1973), goat catarrhal fever (McCulloch, 1951) and goat pneumonia (Campbell,

1958).

2.2 Etiology

Peste des petits ruminants (PPR) is caused by a PPR virus of *Morbillivirus* genus and of the family *Paramyxoviridae*.

2.3 Classification

The family *Paramyxoviridae* is divided into three genera (Mathews, 1982): *Paramyxoviruses* - include Parainfluenza type 1-4; Simian virus.

Morbilliviruses - measles and mumps virus, Canine distemper, rinderpest virus and PPRV.

Pneumoviruses - respiratory syncytia virus (RSV) and pneumonia virus of mice.

The PPR virus particle are spherical and have a diameter of 5,000 - 7,000 amstrong which is larger than Rinderpest virus. The virus particle consist of a core of ribonucleic acid (Mathews, 1982). The family *Paramyxoviridae* is divided into three genera; *Paramyxoviruses, Morbillivirus and Pneumovirus* (Mathews, 1982).

Taxonomic Structure of the Family

Order	-	*mononegavirale*
Family	-	*Paramyxoviridae*
Sub-family	-	*Paramyxovirinae*
Genus	-	*Paramyxovirus*
Genus	-	*Morbillivirus*
Genus	-	*Pneumovirus*

Adapted from Kingsbury (1990).

* The *Paramyxovirus* genus includes Newcastle Disease Virus, Parainfluenza types 1 - 4, Simian Virus 5 and Mumps Virus (Mathews, 1982).

* The *Morbillivirus* includes measles, Canine distemper PPR and Rinderpest Viruses whereas *Pneumovirus* genus consist of *Respirovirus* and *Pneumovirus* (Mathews, 1982).

2.5 Relationship with other Viruses

The PPRV is physically and chemically similar to rinderpest, canine distemper and measles viruses (Taylor *et al.*, 1979a), to which it is also serologically and immunologically related (Gibbs *et al.*, 1979).

Based on the reciprocal cross neutralization test, pathogenicity and degree of cross protection between rinderpest and PPRV, the latter has been classified as the fourth member of the morbillivirus genus of the family paramyxoviridae (Gibbs *et al.*, 1979).

2.6 Serological cross-reactivity of PPRV

The virus cross-reacts with other morbilliviruses of the paramyxoviridae. The serum neutralization test shows some level of cross-neutralization between RP and PPR viruses but parallel titration of sera against both viruses makes definitive diagnosis possible (Taylor, 1979a). PPR antigens have been shown to cross react with sera of animals infected with RP, Canine distemper and Measles virus (Gibbs *et al.*, 1979). It has been observed that during the period of resurgence of PPR in sheep and goats in endemic areas there was no report of rinderpest in cattle (Diallo *et al.*, 1989).

These diseases exhibit clinical signs, and PPR was thought to be caused by a mutant of rinderpest virus adapted to growing goats and sheep (Mornet *et al.*, 1956). Not only does PPRV differ from RPV, but the epidemiology of the two diseases are completely separate and neither virus depends on the other for a continued existence in nature (Taylor *et al.*, 1979a).

2.7 Transmission of PPRV

PPR Virus is disseminated among and between sheep and goats through contact. Movement of

infected animals may be the chief factor in the spread of the disease. Transmission occurs through inhalation of the infectious aerosol that is probably produced by a combination of sneezing and coughing at the height of the disease in an infected animal (Taylor *et al.*, 1979). Contact with infected fomites such as beddings may also contribute to the spread of the disease (Nduaka and Ihemelandu, 1975; Hamdy *et al.*, 1976).

2.8 Excretion of PPRV

PPRV is known to be excreted through various secretions including ocular, nasal, oral discharges and feces of the sick animals (goats and sheep) (Abegunde and Adu, 1977). All tissues and fluids from infected animals should be considered infectious throughout the period of clinical illness. Although PPRV is excreted by all routes at the onset of clinical signs (Abegunde and Adu, 1977), the primary route of virus dissemination are nasal secretions and fecal excretions (Leiss and Plowright, 1964). Viremia is eliminated several days before virus clearance occurs in the lymphoepithelial organs (Scott, 1955; Plowright, 1964).

2.9 PPRV in other animals and Host Range

Pigs could be sub-clinically infected with PPRV by inoculation or contact, but are unable to transmit the virus and are not regarded to be important in the epidemiology of PPR (Nawathe and Taylor, 1979). Cattle may be infected by contact with goats (Dardiri *et al.*, 1976), but also act as "dead end host" (Gibbs *et al.*, 1979).

Gargadennec and Lalanne (1942), did not observe clinical signs in cattle undergoing field exposure by contact with each sheep and goats. Similarly, many authors failed to reproduce the disease experimentally in adult cattle (Dardiri *et al.*, 1976; Taylor and Abegunde, 1977).

Mornet *et al.* (1956) observed both pyrexia and oral lesions in experimental calves. Experimental injection of PPRV into cattle was found to result in the stimulation of antibody production but

not viral replication (Gibbs *et al.*, 1979). The possible role of the camel in the epidemiology of PPR has not been established (Taylor, 1984), even though antibodies to PPR have been demostrated in these animals.

Peste des pestits ruminants (PPR) is an enzootic disease of goats and sheep in West Africa, which affects other wild animals in the Middle East. Cattle develop serological response without clinical signs as a result of inoculation. Contrary to the general observation in pigs and cattle, injection of PPR into white tailed deer, *Odocoileus virginiaus* induces clinical responses which could range from sub-clinical to fatal fulminating reaction and the virus is readily recovered from the deer (Hamdy and Dardiri, 1976).

Isolation of PPR virus from a **Dorcas gazelle** which died of natural infection in the United Arab Emirates has also been reported (Furley *et al.*, 1987). Other species of wild animals that were affected included **gemsbok Oryx gazella, Mouflon Ovis musimoh**, Laristan sheep **Ovis orientalis lariatanica** and the **Nubian ibex nutiana, Capra ibex nutian** (Furley *et al.*, 1987) and Indian Buffalo *Bubalus bubalis* (Govindarajan *et al.*, 1997). Sahu and Dardiri (1979) found that the mink, *Mustela vison*, was not susceptible to PPR.

2.10 Susceptibility based on Sex, Age and Breeds

There have been no reports of differences in prevalence and intensity of infection between breeds or sexes but these have been reported between varying ages and species of animals. PPR affects both sheep and goats (Taylor, 1979), the severity of the disease is greater in goats than in sheep (Lefevre, 1980); and Taylor (1984), hypothesized that sheep posses an innate resistance to the clinical effects of PPR and that occasional field strains can overcome this resistance to produce high mortality.

The morbidity and case mortality rates are both very high in young kids (Obi *et al.*, 1983b). These could range from 100% (Nduaka and Ihemelandu, 1975) to 62% morbidity rate. A case

fatality rate of 54.6% in five outbreaks of PPR involving 527 goats has been reported in Southern Nigeria (Obi *et al.*, 1983b). A morbidity and case fatality rates of 77% and 82% respectively amongst goats purchased at Vom, Northern Nigeria have also been reported (Appel *et al.*, 1981). Asmar *et al.* (1980) also found morbidity and case fatality rates of 90% and 60% respectively in sheep in Saudi Arabia. Obi *et al.* (1983b); found that PPR infections were common in animals 5 months to 2 years old. Opasina and Putt (1985) found that at 2 years old animals becomes immuned. At 24 months old, most animals have become immune in the absence of serious challenge (Obi *et al.*, 1983b). Ata *et al.*, (1989) found that maternally derived PPR antibodies remained for 3 to 4 months after birth and then decreased to an unprotective level.

2.11.0 Epidemiological Factors influencing the Prevalence of PPR

2.11.1 Seasonal Variations

Seasonal variations in the occurrence of PPR have been documented (Obi *et al.*, 1983b). For example in the Southern part of Nigeria, PPR is more frequent during wet and cold harmattan seasons (Obi *et al.*, 1983b; Opasina and Putt, 1985). Losses from mortality in young goats 4 - 12 months of age due to sporadic seasonal outbreaks have been reported (Opasina and Putt, 1985), in the humid and sub-humid West African region.

2.11.2 Kidding Pattern

There have been reports of PPR occurring throughout the year but with a tendency towards peak at the height of the wet season (June to August), (Obi *et al.* 1983b). This could be due to the kidding pattern in the country which occurs 3 - 4 months before the start of rainy season (Obi *et al.*, 1983b).

Opasina and Putt (1985) showed that in two different ecological zones where kidding peaked in June/July or August/September, PPR was reported in November and January respectively.

2.11.3 Ecological Factors - no work done on the effects of ecology on the PPR in goats and

sheep.

2.11.4 Stress

Bourdin and Doutre (1976) implicated stress associated with transhumance as one of the predisposing factors to PPR. The occurrence of PPR during harmattan period as a stress factor has also been reported (Obi *et al.*, 1983b; Opasina and Putt, 1985).

Introduction of new stock in the flock has been clearly linked with the occurrence of PPR (Asmar *et al.*, 1980; Bonniwell, 1980; Obi *et al.*, 1983b). Stress of new environment as in the case with traditional husbandry system, borrowing and renting of males for breeding purposes might also be responsible for introduction of PPRV into virgin flock (Obi *et al.*, 1983b).

2.12.0 Biology of Peste Des Petits Ruminants Virus

2.12.1 Physicochemical and physical properties

Pestes des petits ruminants virion molecular weight is > 500 X 10^6 and Buoyant density in sucrose is 1.8 - 1.20 g/cm^3 (Kingsbury, 1990). Virions are very sensitive to heat, lipid solvents, non-ionic detergents, formaldehyde and oxidizing agents (Kingsbury, 1990).

2.12.2 Nucleic acid and lipids

Virions contain a single molecule of linear, noninfectious (negative sense (kbp), single strand (SS) RNA. The genome size is about 16 kilobase-pairs and a molecular weight of 5 .8 x 10^6. The paramyxoviridae encode 5 to 7 major polypeptides including a transcriptase and neuraminidase (with the exception of morbilliviruses). The viruses exhibit hemagglutin (HN) glycoprotein (G) and fusion - protein (F). The virions measure up to 150 to 300 nm in diameter, the lipid envelop contains the coiled nucleocapsid of 12 to 17nm in diameter (Kingsbury, 1990).

2.12.3 Carbohydrate

Peste des petits ruminants virions are composed of 6% carbohydrate by weight, the composition being dependent on the infected host cell. Fusion and attachment proteins are glycosylated by

N-linked carbohydrate side chains (Kingsbury, 1990).

2.12.4 Antigenic properties

The attachment HN, or G and F proteins are of primary importance in inducing virus neutralizing antibodies and immunity against reinfection. Antibodies to N and variably to other viral proteins also are induced by infection. Various proteins have been reported to serve as antigens for cytotoxic or helper T-cell (Kingsbury, 1990).

2.12.5 Biological properties

Generally all the paramyxoviruses form inclusion bodies and syncytia in cell culture. Most infections are cytolytic, but temperate and persistent infections are also common.

All viruses in the *Morbillivirus* group are antigenically related with the fusion (F) and nucleoprotein (N) proteins being the most conserved across the group (Imagawa, 1968; Hall *et al*., 1980; Sheshbaradaran *et al*., 1986; Harder *et al*., 1992). Immunity induced to the F-proteins may be responsible for the strong cross protection seen after vaccination with heterologous virus.

2.12.6 Immunity to PPR Infection

Survivors from natural infections with PPRV develop life long immunity (Taylor, 1979a). Maternal antibody acquired from the dams last up to 8 weeks post parturition but it is unprotective at 3 months (Majiyagbe *et al*., 1991). Immunity conferred by TCRV is presumed to be cell mediated (Hamdy *et al*. 1976; Nawathe and Taylor, 1979).

2.12.7 Host factors response to infection and vaccination

The mechanism(s) responsible for early susceptiblity of the host to PPRV is not fully understood although neonatal tolerance and colostral transfer of soluble suppressor factors have been suggested (Herbert, 1970). In sheep, maternal antibody may also suppress the synthesis of endogenous T-cell and hence delay maturation of fetal immune system (Sterzl and Silverstein 1967).

2.12.8 Pathogenesis of PPRV

Discussion on the pathogenesis of PPRV infection is not well understood.

After attachment to cell receptors, Virus entry is achieved by fusion of the virus envelope with the cell surface membrane at neutral pH. Virus replication occurs in the cell cytoplasm and is thought to be independent of host nuclear functions. The genome is transcribed into 6-10 separate subgenomic viral complementary mRNAs progressively from 3^1 - 5^1 end by virion associated enzymes. The mRNA are capped and possess a 3^1 poly A tract. Nucleocapsids assemble independently in the cytoplasm. They are enveloped on the cell surface at site containing virus envelope proteins (Kingsbury, 1990).

2.13.0 Clinical Signs of Peste Des Petits Ruminants

The disease process occurs in phases: peracute, acute, chronic and recovery phases (Whitney *et al.*, 1967; Nduaka and Ihemelandu, 1975). Morbidity is often as high as 100%, whereas mortality rate can be up to 90% in most severe outbreaks (Obi *et al.*, 1983b).

The clinical manifestations of PPR include pyrexia, initial lacrimation, ocular and later mucopurulent nasal discharges, coughing, erosions of the gums, tongue, anorexia, lethargy, pneumonia and diarrhea (Whitney *et al.*, 1967; Nduaka and Ihemelandu, 1975; Obi *et al.*, 1983; Opasina and Putt, 1985).

The acute syndrome which is commonly seen in goats has an incubation period of five days with a course of 4 - 6 days terminating in death (Obi *et al.*, 1983b). The Peracute syndrome is characterized by sudden death without any premonitory signs (Obi *et al.*, 1983b). In goats population, the highest incidence occurs in adolescent animals (4 - 6 months of age) with losses up to 58% (Obi *et al.*, 1983b).

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2.13.1 Pathology of PPR

Gross Pathological Lesions associated with PPR are confined to the upper alimentary and

respiratory tracts (Whitney *et al.*, 1976; Nduaka and Ihemelandu, 1975; 1974; Ugochukwu, 1983). Degeneration and necrosis of the epithelia of the gastrointestinal and upper respiratory tracts lead to erosions, ulcerations and profuse serous and later mucopurulent exudate which forms the characteristics encrustation of the nares, lips and around the eyes (Whitney *et al.*, 1967). The peyers patches of the gastrointestinal tract where found to be necrotic, enlarged, hemorrhagic and edema of the lymph nodes throughout the body (Whitney *et al.*, 1967).

Other pathological lesions that have been observed include abscesses of the caudal lobes, pneumonia affecting the right cranial lobes (Nduaka and Ihemelandu, 1975; Abegunde and Adu, 1977), and also of the middle lobes. Nduaka and Ihemelandu (1975) reported generalized pneumonia characterized by diffuse consolidation of the caudal, middle and cranial lobes of both lungs in goats.

2.13.2 Histopathological lesions of PPR

Petechial hemorrhages and ulcers are occasionally found in the glandular mucosa of the abomasum (Whitney *et al.*, 1967). There is loss of villi in the small intestines with elongation of cryptic glands while large intestines show congestion and horizontal hemorrhages in the mucosa giving rise to the so-called "Zebra stripping" (Taylor *et al.*, 1979b). Eosinophilic intracytoplasmic inclusions were occasionally present as well as giant cells which contained intra nuclear inclusion bodies (Nduaka and Ihemelandu, 1975).

Lymphocytosis and necrosis of lymphoid follicles were also observed in the spleen and lymph nodes (Rowland *et al.*, 1971). Degenerative, necrotic and hyperplastic changes became metaplastic as the lesions progressed. These changes were observed in the cells of respiratory mucosae extending from the nasal turbinates to the terminal bronchioles (Whitney *et al.*, 1967). PPR lesions have been found to be complicated by secondary bacterial infections resulting in stomatitis, rhinitis and bronchopneumonia (Obi *et al.*, 1983a). The commonly isolated bacteria

from PPRV infected goats include *Pasteurella hemolytica, Staphylococcus pyogenes, Klebsiella* species from the lungs and, *Eschericia coli* and *Salmonella* from rectal swabs (Obi *et al.*, 1983b). Histopathological and ultra structural studies revealed a foci of mild interstitial pneumonia of the lungs with cell infiltration and oral lesions showed an areas of coagulative and liquefactive necrosis of the epithelia (Isitor *et al.*, 1984).

2.13.3 Hematological and biochemical changes associated with PPR

There have been scanty studies pertaining to the changes of blood parameter of PPR affected animals. Natural and experimental PPRV infections revealed that the virus induces leucopenia with consistent lymphopenia, eosinopenia and monocytosis (Obi *et al.*, 1983b).

Studies carried out on the changes of blood biochemical parameters of West African Dwarf goat challenged with PPRV showed a slight increase in total protein reflected especially in the globulin fraction. Other changes observed were icterus index and creatinine values consistent with decrease in hemoglobin, Blood Urea Nitrogen and Adenine/Guanine ratio which are indicative of slight reno-hepatic involvement (Daneji and Obi, 1994).

2.13.4 Radiographical findings:

Studies have been carried out on the radiographic findings of PPR infected animals. Kene (1995) carried out x-ray studies of the lateral and dorsoventral views of 60 WAD goats suffering from PPR. He found that 28 goats (46.7%), failed to show any appreciable radiographic changes, while the remaining animals revealed increased radio-opacity of the lungs due to alveolar and peribronchial infiltrations. The changes were more severe in younger goats than the adults and all the lobes were affected in sick goats.

2.14.0 Diagnosis of PPR

Presumptive diagnosis of PPR is normally based on post mortem examination and clinical findings. However, various confirmatory tests are available for the diagnosis of PPR either under

field or laboratory conditions. These techniques include viral isolation and characterization and serological tests.

2.14.1 Viral isolation and characterization

Viral isolation using tissue culture techniques including the use of primary fetal lamb, goat kidney cell culture and vero cells has been described (Gilbert and Monnier, 1962; Johnson and Ritchie, 1968; Hamdy *et al.*, 1976), although primary lamb or goat kidney cell cultures are preferable to primary bovine kidney cells. El-Hag and Taylor (1984), have shown that primary bovine kidney cells may be used for PPRV isolation.

2.14.2 Serology/serological diagnosis

Various serological tests have been described to detect PPR virus antigens and antibodies by many authors. These include:

Agar gel precipitation test (AGPT) (Hamdy *et al.*, (1976); Agar gel immunodiffusion test (Scott and Brown, 1961).

Counter immuno electrophoresis (CIE) Majiyagbe *et al.*, 1980; Dot-Enzyme immunoassay (Obi and Ojeh, 1989).

Complement fixation test (Hamdy *et al.*, 1976).

Serum Neutralization test (SNT) (Taylor, 1979a).

Microneutralization test (Rossiter *et al.*, 1985)

Monoclonal antibody based competitive ELISA (Anderson *et al.*, 1990)

Immunocapture ELISA (I-C ELISA) (Libeau *et al.*, 1994).

Microtitre Reversed Passive Hemagglutination test (RPHAT) and Solid Phase Aggregation of Coated erythrocytes Tests (SPACE) (Daneji, 1995), immunoperoxidase test (Daneji, 1995).

Generally, all the serological test are based on the antibody - antigen reactions. The agar gel precipitation and CIE have been found reliable, rapid, simple and feasible under field conditions.

Obi and Patrick (1984), showed the optimal time for sampling to be within one week from the onset of pyrexia for AGPT and CIE. CIE has been found to be twice as sensitive as AGPT (Obi *et al.*, 1983a). Microneutralization test was found to be associated with the problem of standardization as a result of slow rate of growth and low virus titre (Obi *et al.*, 1983a).

ELISAs (tests) are preferred to precipitation and neutralization tests because of their higher specificity, sensitivity and rapidity (Libeau *et al.*, 1994). The major disadvantage of ELISA based tests is cost (Obi *et al.*, 1983a). RPHAT and SPACE have been found to be more specific and cheaper than ELISA tests and more sensitive than AGPT and CIE. But RPHAT and SPACE are less sensitive than ELISA tests (Danclji, 1995). Jeremiah *et al.* (1994) found that IC-ELISA to be more sensitive than virus isolation using vero cells in detecting PPRV antigens.

2.14.3 Nucleic acid based detection of PPRV

A differentiating complementary DNA (cDNA) probe has been developed and used successfully to detect viral nucleic acid in postmortem specimens (Diallo *et al.*, 1989). A major limitation was that this test could only be carried out in well equipped laboratory and with freshly collected samples. A cDNA virus specific probe for PPR was used to detect and differentiate PPRV nucleic acids from in Ethiopian goats (Roeder *et al.*, 1994). The probe collectively distinguished PPRV from RPV (Roeder *et al.*, 1994).

The development of specific nuclei acid probes for hybridization studies and determination of nucleic acid sequences have confirmed that PPRV is quite distinct from RPV (Diallo *et al.*, 1989).

The development of non-radioactive methods for detection of hybridizing nucleic acid has increased the chances that they could be used in laboratories in developing countries (Pandey *et al.*, 1992). One of the most recent developments have been the combined use of polymerase chain reaction (PCR) with nucleic acid sequence analysis (Barrett *et al.*, 1991).

A universal morbillivirus PCR primer set has been identified which can amplify a cDNA fragment from all known morbilliviruses, which has been suggested to provide a more specific epidemiological tool for future studies (Barrett *et al.*, 1991). A disadvantage of the use of PCR and hybridization technology is that they are capital intensive and are currently unavailable in most developing countries.

2.15.0 Differential diagnosis of PPRV

PPRV should be differentiated from other diseases of sheep and goats. These include orf, goat pox, contagious caprine pleuropneumonia (CCPP), parasitic gastroenteritis complex (helminthiasis), coccidiosis and bacterial enteritis. In orf, the lesions are confined to peribuccal areas only. Goat pox lesions are generalized pocks on the mucus membranes, skin and also on the abdomen and hairless areas (Obi *et al.*, 1983a). In the case of CCPP, there is usually no diarrhoea and is manifested mainly by severe pneumonia with pyrexia. Coccidiosis is caused by a protozoan (*Eimeria spp*) and is characterised by blood tinged diarrhoea and without fever and no oral lesions. Helminthiasis is clinically manifested by greenish watery diarrhoea without fever. Bacterial enteritis is usually associated with mucoid shreds and melena in the feces and pyrexia (Obi *et al.*, 1983a).

2.16.0 Economic importance of PPR in Nigeria

PPR or stomatitis pneumoenteritis complex (SPC) is a viral disease of economic importance among sheep and goats in Nigeria, being probably the major cause of mortalities among goats in the country (Akerejola *et al.*, 1979). Hamdy *et al.* (1976) estimated the losses to the Nigerian economy due to PPR to be about $1,500.

Akerejola *et al.* (1979) claimed that PPR cause an annual loss of ₦30 - 60 million to the Nigerian economy. Opasina and Putt (1985) reported an average loss due to PPR in South-Western Nigerian villages to be ₦9.15 per goat in Badeku, ₦1.36 for Eruwa and ₦5.84 for Olukosi.

PPR is considered the single most important cause of morbidity and mortality in sheep and goats in Nigeria, because the disease is endemic in the humid and sub-humid zones of West African subregion, its economic effects are substantial (Majiyagbe, 1985). A major drawback of these reports cited is that they failed to provide the basis on which the calculations of the economic losses were made. Nevertheless the reports have highlighted the economic significance of PPR as a limiting factor to small ruminant production in the country's livestock industry (Obi *et al.*, 1983b).

2.17.0 Prevention and Control of PPR

The prevention and control of PPR can be effectively achieved by the use of appropriate vaccination techniques. Currently, the most effective form of protection against PPR is by vaccination using TCRV vaccine which has been shown to be effective in offering protection against PPR. A single vaccination with this vaccine confers immunity for at least 12 months (Bourdin *et al.*, 1970; Taylor, 1979b; Adu and Nawathe, 1981).

Generally most people keep only a few goats, therefore the usual methods of control such as quarantine, segregation and isolation are not applicable in the control of the disease. Therefore the only recourse left for PPR control is by vaccination using TCRV (Taylor, 1979b). It has been observed that the use of TCRV in control of PPR also causes adverse reactions.

Nduaka and Ihemelandu (1975), claimed that the use of TCRV against PPR led to abortions and clinical signs of the disease among goats purchased from markets in Southern Nigeria. Hill (1983) reported similar side reactions in goats and sheep bought from markets in Oyo state for International Livestock Centre for Africa, which were quarantined for 10 days before being vaccinated with TCRV. Okoye (1980) reported that TCRV gives excellent results though there could be vaccine breaks. However, contrary to what has been reported by some workers, Adu and Nawathe (1981) vaccinated 20 pregnant goats purchased from market around Vom in Plateau

observed no such side reactions.

Goats purchased from markets which might be invariably incubating the disease should be immunized with hyperimmune serum (Nawathe, 1984). PPR is highly endemic hence the fear of vaccinated goats transmitting the infection to susceptible animals as expressed (Gibbs *et al.*, 1979) appears to be theoretical. Many attempts have been made to develop a homologous PPR Vaccine, through various serial passages of PPRV. Benazet (1978) reported that 65 times serially passaged PPRV in lamb kidney cell culture was still pathogenic to susceptible kids.

Anon (1979) observed that 42nd serially passaged PPRV (Nigerian Isolate) in vero cells was also pathogenic to susceptible goats. Recently Adu *et al.* (1990), found that 80th serially passaged PPRV in vero cells was nonpathogenic to susceptible kids.

Heterologous TCRV has many set backs such as maintenance of cold-chains, annual vaccination, safety of the vaccines (vaccine breaks) and difficulty in administration of the vaccines.

These have led scientists to explore the development of a vaccine against PPR (Jones *et al.*, 1993).

Recently, a single vaccinia virus recombinant vaccine for Rinderpest has been developed (Yilma *et al.*, 1988). The single vaccinia virus recombinant expressing fusion (VRVF) or Haemagglutinin (VRVH) confers a lasting immunity against both diseases. Also a double recombinant vaccinia virus vaccine expressing both genes (VRVFH) of RPV have been developed (Giavedoni *et al.*, 1991). Since replication of the vaccinia virus is not affected by antibodies to PPRV, vRVFH can be used to immunise young animals with passive antibody to PPRV.

Unlike TCRV, VRVFH (Proteins) is safe, easy to propagate, simple to administer and heat stable, it is also possible to use serological test to distinguish between VRPVFH vaccinated goats from those exposed to the whole virus by either vaccination with TCRV or infection with PPRV

(Ahmad *et al.*, 1993). Since attenuation didn't affect immunogenecity, it might pose a potential threat to the widespread use of vaccinia virus. Nevertheless cattle and goats were 100% protected against RP and PPRV respectively (Yilma *et al.*, 1988; Giavedoni *et al.*, 1991).

Also there would be no transmission of vaccinia virus from the vaccinated group of animals to contact controls (Giavedoni *et al.*, 1991).

Further attenuation of the recombinant vaccinia virus by additional insertion of lymphokine genes such as interferon gamma or interleukin - 2 (IL-2) is in progress (Yilma *et al.*, 1988; Giavedoni *et al.*, 1991).

2.18.0 Optimal vaccination time using TCRV

Optimum vaccination time against PPR with the use of the heterologous TCRV in goats is at 3 months of age (Majiyagbe, 1991).

Ata *et al.* (1989) observed that maternally derived antibodies remained for 3-4 months after birth and then decreased. Since morbidity and mortality are highest in kids at 3-4 months of age, they are more susceptible to PPRV and hence should be vaccinated at 3 months (Obi *et al.*, 1983a). Lambs and kids between 1-3 months respond poorly to vaccination using TCRV because of immunologic immaturity and interference from maternally derived antibodies (Majiyagbe, 1991).

2.19.0 Vaccination trials

There have been few studies on vaccination trials. Goat adapted RP vaccine is lethal to goats (Scott, 1965). Gibbs *et al.* (1979) found that measle vaccine did not protect goats against PPR whereas canine distempter gave a partial protection based on the antigenic relationship between the morbiliviruses.

Nduaka and Ihemelandu (1975) produced chloroform inactivated vaccines (RPV) which didn't give good protection, shorter period of immunity because of the killed RPV do not evoke effective immune response as live vaccines.

2.20 Management and Treatment of PPR

As in the case of all viral diseases, chemotherapy has been instituted as a form of restraints on the inevitable opportunistic bacteria. Palliative and supportive treatments could also assist in handling cases of PPR (Obi *et al.*, 1983b).

PPR as one of the main cause of mortalities among small ruminants is complicated with frequent endo and ectoparasitism, pasteurellosis and salmonellosis, anaplasmosis and collibacillosis (Opasina and Putt, 1985; Falade *et al.* 1976; Adetosoye and Ojo, 1981; Ojo, 1976).

Treatment of such intercurrent infections as described by earlier workers would reduce severity and deaths in the flock, use of antibiotics, antidiarrheals, dewormers, ectoparasiticides and fluid therapy are indicated (Obi *et al.*, 1983b).

Use of hyperimmune serum as immunoprophylaxis (serovaccination) could assist in animals incubating the disease. Hygiene, sanitation, isolation and quarantine combined with chemotherapy would help in controlling PPR (Nawathe, 1984). Anene *et al.* (1987) using oxytetracycline (50 mg), chloramphenicol (25 % solution) and meta-merazine (20 %) recorded a recovery rate of 14.29 % in a 21 WAD PPR affected goats.

Wosu (1989) reported a recovery rate of 58.8 % using penicillin and streptomycin reinforced with chloramphenicol and lemon fruit in the treatment of labial lesions.

Survival rate of 80 % was recorded in 10 WAD PPR infected kids using a combination of oxytetracycline long acting 20 mg/kg superimposed on Advocin^{R} (danofloxacin 1.25 mg/kg), Ivax vet^{R} 2 ml/3 kg for three days and dextrose saline 5 % (60 mls/animal) twice for five days (Ajala *et al.*, 1997).

Results of antimicrobial sensitivity test to 30 bacterial isolates recovered from nasal discharges of PPR infected goats revealed a variable sensitivity of *Staphylococcus aureus*. Septrin^{R}, Erythromycin and novobiocin^{R} represent antimicrobial agents sensitive to most isolates of

S.aureus, some of which were insensitive to penicilin. *Neisseria catarrhalis*, *Pseudomonas aureginosa*, *Streptococcus* species were generally sensitive to septrin^{R}, gentamicin, tetracycline and kanamycin but *Streptococcus* species were insensitive to gentamicin and polymixin B (Ugochukwu, 1988).

CHAPTER THREE

MATERIALS AND METHODS

3.0 Study Area

Adamawa State has a total land area of 42,159 square kilometers. It is located between latitude 11° and 8° north of the equator to the south and longititude 11° 42" west and 13°34" east. The topography is a mountainous land transversed by big river valleys of Benue, Gongola and Yadzaram. The valleys of the Cameroun, Mandara and Adamawa mountains form part of its undulating landscape (Anon, 1992). The state is bordered in the northern part by Borno State, in the south by Taraba State, eastern part by Cameroun Republic while in the western part is bordered by Gombe State.

The climate is tropical and marked by dry and rainy seasons. Rainy season commences in May and ends in late October. The average rainfall is 759mm in the northern parts and 1011mm in the southern parts especially around Ganye Local Government area. The wettest months are August and September.

The Harmattan period usually begins in December and ends February. It is characterised by dust laden North-Eastern winds from the Sahara desert. The harmattan period is cold and dry with a relative humidity of 13%, the average temperature of 15.2°C, the maximum ambient temperature of 39.7°C (Anon, 1992).

MAP OF ADAMAWA STATE



ADAMAWA STATE OF NIGERIA

There are two marked vegetational zones; the subsudan zone which is marked by short grasses interspersed by short trees commonly found in the northern part and the northern Guinea savannah zone which is characterised with tall grasses and trees commonly found in the southern part of the state. The state comprises 16 local government areas.

Adamawa State has a goat population of 1,970,022, which consists of Red Sokoto, Sahelian goats and their crosses (Anon, 1992).

3.1 Animals

Red sokoto and sahelian bucks and does were used for the study. The approximate ages of the animals were determined through dentition and categorised as follows:

Class A	0 - 6 months
Class B	7 - 12 months
Class C	> one year

3.2 Sampling Procedure

Eight (8) of the sixteen (16) local government areas of Adamawa State were randomly selected without replacement, for sampling goat sera. All the local governments were listed alphabetically in pieces of papers and shuffled in a container, which was picked blindly without replacement, until eight of the sixteen local governments were picked.

The sampling frame was the local government areas and sampling units were the flocks. The local governments selected and the corresponding samples collected include Yola (29), Fufore (34), Maiha (37) Mayobelwa (36), Jada (36), Mubi (38), Numan (34) and Demsa (36).

3.3 Blood Collection (Bleeding)

Blood samples were collected from 280 goats into test tubes and allowed to clot at room temperature. Sera were seperated into bijou bottles and transported in ice packed coolers. The samples were stored at -20°C until analysed for PPRV antibodies.

3.4 Detection of Antibodies against PPRV using competitive ELISA

The principle of the test is the interruption of the reaction between PPRV antigen and monoclonal antibodies (mabs) against PPRV by the addition of test serum samples. The presence of antibodies to PPR in the test serum samples blocks the reactivity of the monoclonal antibody resulting in a reduction in the expected colour following the addition of enzyme labelled antimouse antibody and substrate (Anderson *et al.*, 1990).

3.4.1 Test Protocol

Stored serum samples were allowed to thaw at room temperature. Eight flat bottomed Nunc Maxisorb ELISA plates supplied in the standard ELISA Kit by Pirbright laboratory, U.K. and International Agency for Automic Energy (IAAE) Vienna were labelled (1-8). The ELISA plates were coated with PPR antigen as predetermined by the supplier and incubated overnight at room temperature in an incubator without shaking.

This was done for convenience and also to allow maximum absorption and avoidance of temperature fluctuation due to power failure.

PPR antigen was diluted to a pretitrated concentration of 1:100 in PBS (pH 7.2).

Fifty microlitres (50 µl) of the diluted PPR antigen was dispensed into all wells of the plates using multichannel micropipettes. After about 24 hours, all the coated plates were washed three times with PBS and blot dried on towels. The washing removed the unbound antigens.

Forty microlitres (40ul) of blocking buffer was added to all wells of the plates.

Ten microlitres (10ul) of blocking buffer was further added to the standard 0% competition control wells (G_1 G_2 H_1 H_2). In these wells were dispensed C77 mab controls, PPRV antigen, Mab plus conjugate. The mean of the optical density readings from this control represent the 0 % inhibition. The standard 0 % competition optical density is where most color was obtained and 100% competition level is where there was a total inhibition of the binding of antibody.

Sixty microlitres (60ul) of the blocking buffer was also added to the blank control wells (A_1 B_1]

which consisted of PPRV antigen and conjugate.

Ten microlitres (10 µl) of positive control serum was added to the control [C_1 C_2 D_1 D_2], these consists of PPRV antigen, PPR positive antiserum, mab and conjugate.

Ten microlitres (10ul) of negative control serum was added to the controls [A_2 B_2], which consists of PPR antigen negative serum, mab and conjugate.

Fifty microlitres (50ul) of mab diluted in blocking buffer to a predetermined concentration (1:100) was added to all the wells of the plates.

All the test plates (8) were incubated at 37°C for one hour with continuous shaking and washed three times

Plates were washed (3 times) with PBS tween and blot - dried.

Fifty microlitres (50 µl) of antimouse conjugate in blocking buffer was added to all wells of the plates.

Plates were incubated at 37°C for one hour with continuous shaking.

Plates were washed 3 times with PBS and blot dried on towels.

Fifty microlitres (50ul) of substrate/chromogen [orthophenylene diamine (OPD/H_2O_2) Solution] was added to all the wells of the plates.

Plates were left for 10 minutes without shaking at room temperature for colour development.

All reactions were stopped by the additon of 50 µl of $1MH_2So_4$ to all the wells.

Optical densities at 492 nanometer were read on an automatic ELISA reader which was connected to an IBM computer. Mean values of four standard 0 % competition wells were calculated. Percent Inhibition (PI) was calculated as

$$PI = 100 - \frac{\text{mean OD of test wells}}{\text{mean OD of standard (0 \%) wells}} \times 100$$

Inhibition values greater than 50% were considered positive.

3.5.0 PPRV Antigen Detection

3.5.1 Area of study

Field outbreaks of PPR in Adamawa state were studied based on the reports of suspected cases from different locations.

Specimens (nasal and ocular swabs) were collected using plastic cotton swabs from goats suspected of PPR. Nine (9) outbreaks were recorded during the course of the study in various districts of the local government areas and the number of the samples (swabs) collected were as indicated in Table 3.1:

Table 3.1: Specimens (Nasal and Ocular) collected from various districts of 9 Local Government Areas

Local Government Areas	District	No. of samples collected from suspects	No. of samples collected from apparently normal in contact animals
1. Numan	Lafia	10	5
2. Maiha	Belel	7	3
3. Ganye	Toungo	7	3
4. Song	Girei	7	3
5. Fufore	Ribadu	10	4
6. Yola	Namtari-Manga	10	5
7. Mubi	Fali	9	6
8. Jada	Jada	7	3
9. Gombi	Ga'anda	7	3

All specimens were labelled and identified on the basis of the area of collection, breeds, sex and approximate age.

Specimens were transported in an ice-packed cooler box and stored at -20°C until analysed for PPRV antigen and stored at primary health care cold chains in various local government areas.

3.5.2 Processing of Samples

All specimens were processed by eluting viral particles from the cotton wool swabs. Elution was done by adding 200ul of PBS to the tubes containing the cotton wool. The suspension was expressed into universal bottles and stored at -20°C until tested.

3.5.3 Immunocapture enzyme linked immunosorbent assay (ICE)

The assay was based on the test protocol described by Libeau *et al.* (1994).

Briefly (6), Microelisa plates were coated with anti PPR purified mab (capture antibody) diluted to 1:600 in PBS and used at 100ul per well. The plates were incubated at 37°C overnight without shaking.

All plates were washed 3 times with PBS 1:5, containing 0.05% Tween 20, and blot dried on towels.

Fifty microlitres (50ul) of samples in quadruple ($A_2B_2A_7B_7$) were added to each of the wells.

Fifty microlitres (50ul) of PPR ($G_5H_5G_{10}H_{10}$) and Rinderpest ($G_6H_6G_{11}H_{11}$) as reference antigens in quadruples were added to each well.

Fifty microlitres (50ul) of PBS containing 0.05% tween 20 and 0.5% negative serum (blocking buffer) were added to the wells of blank [A_1H_1 and $A_{12}H_{12}$].

Twenty five microlitres (25ul) of PPR detection antibody diluted 1:300 in blocking buffer were added to one duplicate of samples [A_2B_2] and the wells of PPR blank (A_1 to H_2)

Twenty five microlitres (25ul) of rinderpest detection antibody diluted 1:300 in blocking buffer were added to other duplicate of samples [A_7B_7] and the wells of RP blank [A_{12} to H_{12}].

Twenty five microlitres (25ul) of 1:100 conjugate were added to each well.

All the plates were incubated at 37°C for one hour with continuous shaking.

Plates were washed vigorously (with a pipette because samples are usually sticky in PBS and blot

dried on towels).

One hundred microlitres (100ul) of OPD substrate were added to each well (10ul OPD + 40ul H_2O_2).

All reactions were stopped after 10mins with 100ul $1MH_2SO_4$.

3.6 Interpretation of results

An ELISA reader connected to a microcomputer was used to read the Optical Densities (ODS) at 492nm with 15% of OD of the reference antigen as cut-off point for a positive reaction.

3.7 Statistical analysis of data

Data obtained in this study was statistically analysed using chi-square test (Snedecor and Cochran, 1980) and Odds ratio (OR) (Thrushfield and Hinxman, 1981) to see if associations exist between age, sex, breeds and the reactors.

CHAPTER FOUR

RESULTS

4.1 Detection of Antibodies to Peste des petits ruminants

Of a total of 280 goats sampled which had no history of vaccination, 176 (62.9%) tested positive to peste des petits ruminants antibodies. The infection rate varied from one area to another. Antibodies to PPR were detected in goats from all the local government areas of the state surveyed with Mubi having the highest prevalence rate of 76.0%, followed by Maiha 73.0% and the least was Jada 55.3%. There was no statistically significant difference in the prevalence rate of antibodies to PPR in various local government areas (Table 4.1).

The sex distribution of goats that tested positive to antibodies to PPR is shown in Table 4.2.

The sex specific prevalence rate for males (53.6%) was significantly different from the rate for females (69.0%) ($P < 0.05$). Females had higher infection rate than males (OR = 1.9).

Age distribution of antibodies to PPR is given in Table 4.3.

Age specific rate for the 0-6 months age group was 43.4%, for the 7-12 months age group 54.2% and for those greater than 12 months 87.6%. The difference between the age specific rates was statistically significant ($P < 0.05$). Those aged greater than one year were 4.06 times more likely to have antibodies to PPR than all other ages combined. Age and Sex distribution of PPR antibodies in goats is indicated in Table 4.4. Males that aged 0-6 months had an age-specific rate of 45.2%, whereas females of the same age had 40.0%. Males aged between 7-12 months had an age specific rates of 44.7% whereas females of the same category had 62.0% Males above 12 months had a specific rate of 77.4% while females of the same age had 93.2%. There was a statistically significant difference in the age-sex specific rates. Those greater than one year of age were 9.6 times more likely to acquire infection than other age group.

Breed distribution of antibodies to PPR in goats sampled is shown in Table 4.5.

Mixed breeds had the highest breed specific rates of 87.6%. Red sokoto had a breed specific rate of 52.6% and the sahelian breeds 52.3%. Even though there was a statistical significant difference among the breed specific rates ($P < 0.05$), there was no difference between Red Sokoto and Sahelian breeds.

Table 4.1 Prevelance of antibodies to Peste des Petits Ruminants among goats in 8 Local Government areas of Adamawa State.

Local Government Area	Total sampled	Number Positive	% positive
Maiha	37	27	73.0
Mubi	38	29	76.0
Jada	36	21	58.3
Mayobelwa	36	21	58.3
Numan	34	20	58.3
Demsa	36	22	61.1
Fufore	34	19	55.9
Yola	29	17	58.6
Total	280	176	62.9

Chi square (X^2) = 7.59

Degrees of freedom = 7 : NS

Table 4.2 Sex - distribution of peste des petits ruminants antibodies in goats in Adamawa State.

SEX	TOTAL NO SAMPLED	NO TESTED (+ ve)	NO TESTED (- ve)	SEX SPECIFIC RATE (%)	ODDS RATIO
MALES	112	60	52	53.6	0.7
FEMALES	168	116	52	69.0	1.3
TOTAL	280	176	104	62.9	1.0

alpha = 0.05

Chi square (X^2) = 7.75

Table 4.3 Age distribution of peste des petits ruminants antibodies among goats in Adamawa State

AGE	Total No Sampled	Total No. (+ve)	Total No. (-ve)	Age Specific rate (%)	OR (ODDS RATIO)
0 - 6 months	92	39	53	43.4	0.43
7 - 12 months	83	45	38	54.2	0.70
> 1 year old	105	92	13	87.6	4.06
Total	280	176	104	62.9	1.0

Calculated chi square (X^2) = 149.29, significant, $P < 0.05$

Table 4.4 Age and Sex distribution of peste des petits ruminants antibodies in Adamawa State.

Age	MALES			FEMALES			OR (in comparison to males (0 - 6 months)
	No of samples	No tested (+ ve)	Age Specific rate (%)	Total No sampled	No tested (+ ve)	Age specific rate (%)	
0 - 6 months	42	19	45.2	50	20	40.0	1.0
7 - 12 months	38	17	44.7	45	28	62.0	1.61
> 1 year old	32	24	77.4	73	68	93.2	9.62
Total	112	60	53.6	168	116	69.0	

chi square (X^2) = 46.73 df 2 significant with $P < 0.05$

Table 4.5 **Breed distribution of Peste Des Petits Ruminants antibodies among goats in Adamawa State.**

Breeds	Total No Sampled	Total No. (+ve)	Total No. (-ve)	Breed specific rate	OR
Sahelian	101	53	48	52.3	0.65
Red Sokoto	97	51	46	52.6	0.66
Mixed	82	72	10	87.6	4.35
Total	280	176	104	62.9	1.0

chi square (X^2) = 30.91, alpha = 0.05, degrees of freedom = 2 :

4.2 Peste Des Petits Ruminants Antigen Detection during outbreaks

The location, time, morbidity, mortality and case fatality rates, and clinical signs of the reported outbreaks of PPR in Adamawa State from which the samples were obtained are presented in Table 4.6. All cases involved in the outbreaks were characterised clinically by pneumonia, diarrhea and oculonasal discharges. In some cases pyrexia and lymphadenopathy were noted.

Outbreaks occurring in Gombi (Gaanda) had the highest mortality rate (40.0%) and case fatality rate of 66.7% and Yola had the least of the two rates. The distribution of antigens to PPR in 9 reported outbreaks in both suspects and apparently healthy incontact goats is shown in Table 4.7.

Overall, 75 out of the 109 samples tested 68.8% were positive to PPR antigens. About 82.7% of the suspects tested positive. There was a significant difference in the percentage positive between the suspects and apparently healthy incontact goats. Suspects had 7.7 times greater risk of having antigens than the incontact animals.

Sex distribution of PPR antigens in 9 reported outbreaks is presented in Table 4.8. The sex specific rate for males was 75.9% and for females 61.8%. About 75.9% of the 54 males tested positive. There was no significant difference in PPR antigen detection rates between males and females.

The age-distribution of PPR antigens in goats involved in the reported outbreaks is indicated in Table 4.9. The age-group 0-6 months had the highest age specific rate of 89.5% (34 out of the 38). Age group 7-12 months had 61.7% (29 out of the 47) and those aged greater than one year old had 50.0% (12 out of the 24). There was a statistical significant difference in age - specific rates of detection of PPR antigens (OR = 3.7).

Breed distribution of PPR antigens in the nine outbreaks is indicated in Table 4.10. About 82.4% of Red Sokoto breed tested positive. Sahelian breeds had breed specific rates of 82.9%. Mixed breeds

had the least breed specific rates of 45.0%. There was a statistically significant difference among the breeds ($P < 0.05$).

Seasonal distribution of the reported outbreaks of PPR antigens detection is indicated in Table 4.11. There were four reported outbreaks in the rainy and five in the dry seasons. More deaths were recorded in the dry than in the rainy season. There was a statistically significant difference in the number of deaths between the two seasons.

Table 4.6 Characteristics of 9 reported outbreaks of PPR in goats in Adamawa state (January 1995-December, 1996).

	Location	Period	Morbidity rate (%)	Mortality rate (%)	Case fatality rate (%)	No. of animals in flock	Clinical Signs	Samples taken
1	Ganye (Toungo)	5th Aug, '95	100.0 (100)	40.0 (40)	40.0	100	Pneumonia, Diarrhoea	10
2	Gombi (Ga'anda)	10th Dec, '95	60.0 (48)	40.0 (32)	66.7	80	Diarrhoea, Stomatitis	10
3	Numan (Lafiya)	23rd Dec, '95	50.0 (30)	17.0 (5)	16.7	60	Pneumonia, Stomatitis	15
4	Maiha (Belel)	4th Jan, '96	70.0 (46)	31.8 (20)	43.5	65	Diarrhoea, Stomatitis	10
5	Yola (Namtarimanga)	20th Feb, '96	80.0 (44)	14.5 (8)	18.2	55	Ocular discharge, Diarrhoea	15
6	Song (Girei)	10th Jun, '96	75.0 (53)	17.0 (12)	22.6	70	Pneumonia, Stomatitis,	10
7	Fufore (Ribadu)	15th Jul, '96	85.0 (81)	15.8 (15)	18.5	95	Pneumonia, Stomatitis,	14
8	Jada (Jada)	15th Aug, '96	65.0 (49)	26.7 (20)	41.8	75	Diarrhoea, Stomatitis	10
9	Mubi (Fali)	2nd Jan, '96	55.0 (66)	25.0 (30)	45.5	120	Ocular discharge, Diarrhoea	15

Table 4.7 **Distribution of peste des petits ruminants antigens in reported outbreaks in both suspects and apparently healthy incontact goats.**

		Suspects (Cases)	Control (Apparently Healthy incontact goats)	% Test Results	Odds ratio
Test Result	+	62 (82.7 %)[a]	13 (38.2 %)[b]	68.8	7.7
	-	13 (17.3 %)	21 (61.8 %)	31.2	1.0
Total		75	34		

Chi square (X^2) = 5.64 > 3.841

alpha 0.05; degrees of freedom = 1

∴ Significant

a % of cases positive to the test

b % of normals positive to the test

Table 4.8 Sex distribution of peste des petits ruminants antigen among goats in Adamawa state.

Sex	Total No Sampled	Total No. tested +ve	Total No. tested -ve	Sex specific rates (%)	OR (Odds ratio)
Males	54	41	13	75.9	1.4
Females	55	34	21	61.8	0.7
Total	109	75	34		

Chi square (X^2) alpha = 0.05; degrees of freedom = 1

$X^2 = 1.91$

∴ Not Significant

Table 4.9 Age distribution of Peste Des Petits Ruminants antigen in 9 outbreaks of Adamawa state.

Age Group	Total No sampled	Total No +ve	Total No -ve	Age specific rates (%)	Odds ratio
0 - 6 months	38	34	4	89.5	3.7
7 - 12 months	47	29	18	61.7	0.7
> 1 year old	24	12	12	50.0	0.5
Total	109	75	34	68.8	1.0

Chi square (X^2) at 2 degrees of freedom .: significant

alpha 0.05

$X^2 = 12.81$

Table 4.10 Breed distribution of peste des petits ruminants antigens among goats in reported outbreaks in Adamawa state

Breeds	Total No sampled	Total No +ve	Total No -ve	Breed specific rate (%)	OR
Red Sokoto	34	28	6	82.4	2.1
Sahelian	35	29	6	82.9	2.2
Mixed	40	18	22	45.0	0.4
Total	109	75	34	68.8	1.0

alpha 0.05 degrees of freedom = 2

Chi square $X^2 = 16.69$ is significant.

Table 4.11 Seasonal distribution of outbreaks (January, 1995 - December, 1996)

Seasons	No. of Outbreaks	No. Dead	No. Recovered (survivors)	Total morbidity
Rainy	4	83	200	283
Dry	5	93	135	228
Total	9	176	335	n = 511

Chi square (X^2) = 78.02 Significant

degrees of freedom = 1

alpha = 0.05

CHAPTER FIVE

DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

5.0 Discussion

The overall point prevalence of antibodies to peste des petits ruminants in 8 local government areas of Adamawa State as determined by competitive ELISA in this survey was 62.9%. This is higher than the results obtained in previous studies in other parts of the country (Shamaki *et al.*, 1996). In that study prevalence rates of 38.4 %, 7.7 %, 40.6 % and 40.0 % were determined for Oyo, Abia, Borno and Plateau States respectively. This could be explained due to variations in sampling techniques and different locations. It might also suggest that PPR is on the increase.

The antibodies to PPR were detected throughout the local government areas of the state investigated in the current study, which suggests that the disease is widely distributed in the state.

There appears to be a paucity in the literature, on the susceptibility and prevalance of PPR amongst goats on the basis of breeds and sex. In the present study, the sex specific rate for males 53.6 % was significantly different from that of the females, 69.0 % ($P < 0.05$). This may be related to the number of females being kept and sampled during the course of the study. Females were found to have a higher risk of acquiring an infection than males (OR = 1.9). This shows that bucks may therefore be less susceptible than does.

This observation is similar to the previous findings of Pumov (1984), who reported that any prevalence recorded were mainly amongst the females which is related to the system of management whereby males are weaned at 4-6 months of age and possibly sold off while the females remain in

the breeding flock. This will give a flock population whereby female dominate, giving them, greater exposure experience than males.

Goats older than one year tended to have higher antibody frequency than other age groups. This shows that a solid immunity acquired by such age groups which were onced infected and subsequently recovered from infection. The results obtained agree with the previous findings of Obi *et al.* (1983b), who reported that animals at one to two years must have already become immune in the absence of any serious field challenge. The animals aged less than one year old had a lower frequency of antibodies to PPR, which means that they could be more susceptible than the other age groups.

In this study, females above one year old were found to have a higher chance of having acquired protective antibodies to PPR than their male counterparts. Females were more likely to have antibodies to PPR than the males (OR = 9.6). To our knowledge there is no documented report on the age-sex relationship of PPR in goats in the literature.

We found that mixed breeds have higher breed specific rates of 87.6%, which was followed by red sokoto with 52.3%. Although there was a statistically significant difference between mixed and pure breeds, there was no significant difference between red sokoto and sahelian breeds. Mixed breed goats appeared to be more resistant than either of the breeds.

The clinical signs observed during the 9 reported outbreaks of PPR were consistent with the previous findings (Obi *et al.*, 1983b; Opasina and Putt, 1985). These included pneumonia, diarrhea, stomatitis, fever and anorexia.

Gombi (Ga'anda) local government area of the state had the highest reported mortality rate (40.0%), which had a case fatality rate of 66.7% and morbidity rate of 100%.

These values are much lower than those reported by Appel *et al.* (1981), who reported a case fatality rate of 82.0% and morbidity rate of 77.0% during an outbreak of PPR in goats at Vom. The variation could be due to difference in techniques used, number of animals involved and environmental conditions. Opasina and Putt (1985), reported that harsh environmental conditions (cold) could act as a predisposing factor to PPR infection. It is however, higher than that of Obi *et al.* (1983b), who reported a morbidity rate of 62.0 %, case fatality rate of 54.6 % in southern Nigeria.

Yola local government area (Namtarimanga), had the least mortality rate of 14.5 % and case fatality rate of 18.2 %. Opasina and Putt (1985) also reported case fatality rates of 86.9 %, 41.0 %, and 63.9% in three villages in south-western Nigeria. The results might suggest that PPR infection is on the increase and an increased in the virulence of the field strains in recent outbreaks.

High frequency of apparently healthy incontact animals testing positive indicates a high rate of inapparent infection and incubators of the disease. This might be explained in part by the fact that the apparently normal animals were in the same flock during the outbreaks or could be due to the fact that the normal goats were also infected and were incubating the PPRV. Sixty-two (82.7%), out of 75 goats which tested positive were from those showing clinical signs - penumonia, diarrhea, stomatitis, anorexia and fever. Those that were showing clinical signs of PPR were more likely to be positive to PPR antigens (OR = 7.7) than those apparently normal.

The sex specific rates of antigens to PPR in males was 75.9% and for females, 61.8%, there was no statistically significant difference between antigen detection in males and females. This agreed with the findings of the previous workers. Opasina and Putt (1985), reported that there was no sex difference in the susceptibility to PPR infection.

The age specific rates for the occurrence of antigens to PPR was 89.5% amongst those aged 0 - 6

months, 61.7% in the age group of 7 - 12 months and 50.0 % for those greater than one year old. As the age increases, fewer animals tested positive for PPR antigen.

The younger and adolescent animals were thus more susceptible to PPR infection during outbreaks than the older ones. This could be due to the fact that the older animals had greater exposure potential and had acquired solid immunity to PPRV. The results agree with those of previous workers (Obi *et al.,* 1983b; Opasina and Putt, 1985) reported similar findings.

Although, there was no statistically significant difference in the breed specific rates between red sokoto (82.4%) and sahelian goats (82.9%) to PPR antigens, the differences in breed specific rates of mixed breeds (45.0%) and the pure breeds of either red sokoto or sahelian goats was statistically significant ($P < 0.05$). This is possibly explained by the fact that mixed breeds tend to be hardier to environmental stresses than the pure breeds and therefore likely to be more resistant to diseases. The pure breeds were found to be more likely to come down with PPR infection than their mixed breeds (OR = 2.0).

From the outbreaks there were more deaths during the dry season and fewer for those occurring during the rains. This is not in agreement with the report of Obi *et al.* (1983b) who reported that in southern Nigeria PPR outbreaks occurred throughout the year there were higher peaks during the mid rainy season.

In the northern Nigeria, the dry season is characterised by windy, low relative humidity, harsh environmental conditions and transhumance. Together, these may contribute to the dissemination of PPRV infection and might have contributed to higher deaths recorded in this study. This was also noted by Bourdin and Doutre (1976), who suggested that transhumance and environmental factors during dry season could lead to higher deaths.

5.2 Conclusions and Recommendations

The present study revealed a point prevalence of antibodies to PPR in goats in Adamawa state using C-ELISA test to be 62.9%. Antibodies to PPR were detected in all the 8 local government areas of the state surveyed. The prevalence of PPR infection as determined by the serological survey was found to vary by local government and flocks. From the results of the survey in the study areas PPR infection in goats may be widely distributed in the state.

The investigation into the reported outbreaks of PPR using IC-ELISA also revealed 68.8% tested positive to PPR antigens. The reported outbreaks were distributed throughout the local government areas of the state. This shows that PPR infection is widely distributed in all parts of the state. Data obtained in this study could be helpful in planning strategies for the prevention and control.

In view of the growing awareness of the importance of goats as one of the major source of protein guided by the increasing economic losses suffered by subsistence and traditional livestock producers, the need for an official nationally co-ordinated action against PPR becomes inevitable.

REFERENCES

Abegunde, A. and Adu, F.D. (1977). Excretion of the virus of peste des petits ruminants by goats. *Bulletin Animal Health Production Africa.* **25**: 307-311.

Adetosoye, A. I. and Ojo, M. O. (1983). Characteristics of E.coli isolated from goats suffering from peste des petits ruminants and detection of enterotoxin in isolation from other cases of diarrhoea. *Tropical Veterinarian* **1**: 102-110.

Adu, F.D. and Nawathe, D.R. (1981). Safety of tissue culture rinderpest vaccine for pregnant goats. *Tropical Animal Health Production.* **13**:61.

Adu, F.D., Joannis, T., Nwosu, E., Abegunde, A., (1990). Pathogenecity of attenuated peste des petits ruminants in sheep and goats. *Revue d' elevage et de Medicine Veterinaire des Pays Tropicaux.* **43**:23-26.

Afshar, A., Wright, P.F., and Dulac, G.C. (1986). Dot EIA for visual detection of atibodies to pseudorabies in swine serum. *Journal Clinical Microbiology.* **23**:563-567.

Ahmad, S., Bassii, M., Benerjee, A.K. and Yilma, T. (1993). Immunologic characterisation of the VSV nucleocapsid (N) protein expressed by recombinant baculovirus in *Spodoptera exiqua* larva: use in differentiating betweeen vaccinated and infected animals *Virology* **192**:207.

Ajala, O. O., Daneji, A. I., Kumshe, H. A. and Oyeyemi, M. O. (1997). Treatment of peste des petits ruminants in preweaned WAD kids. *Tropical Veterinarian.* **15**: 39-42.

Akerejola, O.O., Schillhorn Van Veen, T.W. and Njoku C.O. (1979). Ovine and caprine diseases in Nigeria: Review of economic losses. *Bulletin Animal Health Production Africa.* **27**:65-70

Amjad, H.Q.; Forsyth, M.; Barrett, T. and Rossiter, P.B. (1996). Isolation of peste des petits in goats in Pakistan. *Veterinary Records.* **139**: 118-119.

Anderson, J., McKay, J.A. and Butcher, R.N. (1990). The use of monoclonal antibodies in competetive Enzyme linked immunosorbent assay for the detection of antibodies to rinderpest and peste des petits ruminants virus. In: Jeggo "The seromonitoring of rinderpest throughout Africa phase one: *The proceedings of a final research co-ordinated meeting of the FAO/IAEA/SIDA/IBAR/PARC Coordinated research programme Ivory coast IAEA - TECDOC.* **623**:43-53.

Anene, B.M., Ugochukwu, E.I. and Omamegbe, J.O. (1987). The appraisal of three pharmaceutical regimes for the treatment of naturally ocurring in goats. *Bulletin Animal Production Africa.* **35**: 1-3.

Anon. (1979). Annual report of virology Division National veterinary Research Institute, Vom.

Anon. (1992). Nigerian livestock resources, Resource inventory management limited report, *National Synthesis*.

Appel, M.J.G., Gibbs, E.P.J., Martin S.J., Termeulen, V., Rima, B. K., Stephenson, J.R. and Taylor, W.P. (1981). Morbillivirus Diseases of Animals and Man. In: E. Kurstak (eds.), comparative diagnosis of viral disease Vol. IV, Vertebrate Animal and related viruses part B - RNAViruses, PP. 235-297.

Asmar, J.A., Rawdwan, A.I., Abi Assi, H. and AlRashied, A. (1980. A PPR like disease in sheep in central Saudia Arabia: Evidence of its immunologic relationship to RP; Prospects for a control method. In: *Proceedings of 4th Conference on the Biological aspects of Saudi Arabia. Riyah.* pp325-337.

Ata, F.A., Al-sumry, H.S., King, J.G. Ismail, S.I. and Ata, A.A. (1989) Duration of maternal immunity to peste des petits ruminants. *Veterinary Records*. **124**: 590-591.

Barrett, T., Subbarao, S.M., Belsham, G.J. and Matty, B.W.J.M. (1991). The molecular biology of the morbilliviruses. In: The *paramyxoviruses*. pp83-102. ed. by D.W. Kingsburry Plenum Press, N.Y.

Benazet, B.G.H. (1978). La peste des petits ruminants. Etude experimentale de la vaccine'. Cited by Taylor, W.P. (1984). Epidemiology of peste des petits ruminants. *Preventive Veterinary Medicine*. **2**: 157-166.

Beutin, L., Bode, L., Ritcher, T., Peltre, G. and Stephen, R. (1984). Rapid visual detection of *Escherichia coli* and *Vibrio cholerae* heat labile enterotoxins by Nitro-cellulose EIA. *Journal Clininical Microbiology*. **19**:371-375.

Beyer, C.F. (1984). A dot Enzyme immuno assay on Nitrocellulose membrane for the determination of Immunoglobulin class of mouse monoclonal antibodies. *Journal Immunological Methods*. **67**: 79-87.

Bonniwell, M.A. (1980). The use of Tissue cultured rinderpest vaccine to protect goats and sheep against peste des petits ruminants in the Ashanti region of Ghana. *Bulletin Office International Epizootics*. **92**:1233-1238.

Bourdin, P. and Doutre, M.P. (1976). Peste Des Petits Ruminants en Senegal: Donnees novelles. *Revue d' elevage et de Medicine Veterinaire des Pays Tropicaux*. **29**:199-204.

Bourdin, P., Rioche, M. and Laurent, A. (1970). Emploi d'ul Vaccine antibovi-pestique produit sur cultures cellulaires dans la prophylaxie de peste des petits ruminants au Dahomey. *Revue d' elevage et de Medicine Veterinaire des Pays Tropicaux*. **23**:295-300.

Brumby, P. by Dauda, K.J. (1990). System and livestock development in Tropical Africa cited in Animal disease as a factor limiting economic development in Africa. *Cornelle Veterinary*. **80**: 17-25.

Campbell, A.D. (1958). Goats pneumonia, In: Eastern region annual report. Veterinary Department, 8-9.

Daneji, A.I. and Obi, T.U. (1994). Changes in selected blood biochemical parameters of peste des petits ruminants infected West African Dwarf goat. *Tropical Veterinarian*. **12**: 47-52.

Daneji, A.I. (1995). **Comparative analysis of RPHAT and SPACE and other Immunodiagnostic tests to detect peste des petits ruminants virus antigens.** Ph.D Thesis, University of Ibadan, Nigeria.

Dardiri, A.H., Deboer, C.J. and Hamdy, F.M. (1976). Response of American goats and cattle to peste des petits ruminants virus. In: **Proceedings of the 19th Annual meeting of American Association of Veterinary Laboratory diagnosticians**. pp 344-377.

Diallo, A., Barbron, M., Barrett, T., Subbarao, S.M. and Taylor, W.P. (1989). The differentiation of rinderpest and peste des petits ruminants virus using specific cDNA clones. *Journal Virological Methods*. **23**:127-136.

Diallo, A., Barrett, T., Lefevre, P.C. and Taylor, W.P. (1990). Comparison of proteins in cells infected with Rinderpest and Peste des Petits Ruminants Viruses. *Journal General Virology*. **68**:2033.

Durtnell, R.E. and Eid, F.I. A. (1973). Preliminary notes on diseases of goats resembling peste des petits ruminants in Sokoto province, Northwestern state, Nigeria. *Nigerian Veterinary Journal*. **2**: 18-21.

EL-Hag, A.B. and Taylor, W.P. (1984). Isolation of peste des petits ruminants virus from the sudan. *Research Veterinary Science*. **36**:1- 4.

Falade, S. (1976). Isolation of *Salmonella poona* from diarrhoeic Nigerian goats. *Veterinary Rocords*. **99**: 419.

Federal Department of Livestock and Pest Control Services (1991). Nigerian Livestock Survey, FDLPCS, Ministry of Agriculture, Abuja. Vol. **2**: 89.

Furley, C.W., Taylor, W.P. and Obi, T.U. (1987). An outbreak of peste des petits ruminants in a Zoological collection. *Veterinary Records*. **121**: 443-447.

Gargadennec, L. and Lalanne, A. (1942). La peste des petits ruminants. Bull. des service zootech. et des. epiz. de L' Afrique occidentale franaise. **5**:16.

Giavedoni, L., Jones, L., Mebus, C. and Yilma, T. (1991). A *Vaccinia Virus* double recombinant expressing the F. and H. genes of RPV protects cattle against RP and causes no pock lesions. **In : Proceedings of Natural Academic Sciences**. **88**: pp 8011-8015.

Gibbs, E.P.J., Taylor, W.P., Lawman, M.J.P. and Bryant, J. (1979). Classification of peste des petits ruminants as member of genus *morbillivirus*. *Intervirology* **II**: 268-274.

Gilbert, Y. and Monnier, J. (1962). Adaptation du virus de la peste des petits ruminants aux cultures cellulaires: Note preliminaire. *Revue d' Elavage et de Medicine Veterinaire des pays Tropicaux*. **15**: 321-335.

Govindarajan, R., Koteeswaran, A. T., Venugopalan, A. T., Shyam, G., Shaounas, S., Shaila M.S. and Ramachandran, S. (1997). Isolation of peste des petits ruminants virus from an outbreak in Indian Buffalo *(Bubalus bubalis)*. *Veterinary Records*. **141**: 573-574.

Hall, W.W., Lamb, R.A and Choppin, P.W. (1980). The polypeptides of canine distemper virus. Synthesis in infected cells and relatedness to the polypeptides of other *Mobilliviruses*. *Virology*. **100**: 433-444 .

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

Hamdy, F.M. and Dardiri, A.H. (1976). Response of white tailed deer to infection with peste des petits ruminants virus. *Journal Wild Life Disease*. **12**: 516-522.

Harder, T.C., Klusmeyer, K., Frey, H.R., Orvell, C. and Leiss, B. (1992). Intertypic differentiation and detection of intra typic variants among canine and phocid *morbillivirus* isolates by kinetic nutrialization using a novel immunoplaque assay. *Journal General Virology*. **4**: 430-433.

Hedger, R.S., Barnett, I.T.R. and Gray, D.F. (1980). Some viral diseases of domestic animals in the sultanate of Oman. *Tropical Animal Health Production*. **12**: 107-114.

Herbert, W.J. (1970). Neonatal immunity. Veterinary Immunology. Blackwell Scientific Publications. Oxford and Edinburgh. pp247-260.

Hill, D.H. (1979). Highlights from ILCA'S humid Zone programme in Hill, D.H. (ed.) peste des petits ruminants: 12-13 p (ILCA - Adis Ababa).

Imagawa, D.T. (1968). Relationship among measles, canine distemper and rinderpest viruses. *Progress Medicine Virology*. **10**:160-193.

Isitor, G.N., Ezeokoli, C.D. and Chineme, C.N. (1984). Histopathological and Ultrastructural study of lesions of PPR in Red Sokoto goats. *Tropical Veterinarian*. **2**: 151-158.

Jeremiah, T.S., James, A.H., Charles, A.M. and Edward, J.D. (1994). Comparison of monoclonal antibodies based Sandwich Enzyme linked immunosorbent assay and Virus Isolation for detection of peste des petits ruminants virus in goats tissue and secretions *Journal Clinical Microbiology*. Vol. **32(5)**: pp 1349-1353.

Johnson, R.H. and Ritchie, J.S.D. (1968). A virus disease associated with pseudorinderpest in Nigerian dwarf goats. *Bulletin Epizootics Africa*. **16**: 411.

Jones, L., Giavedoni, L., Saliki, J.T., Brown, C., Mebus, C. and Yilma, T. (1993). Protection of goats against PPR with a *Vaccinia* virus double recombinant expressing the F and H genes of RPV. *Vaccine* **II**: 961-964.

Kamata, H., Tsukiyama, K., Sugiyama, M., Kamata, Y., Yoshikawa, Y. and Yamanouchi, K. (1991). Nucleotide sequencing of cDNA to the rinderpest virus mRNA encoding the nucleocapsid protein. *Virus Genes* **5**:5-15.

Kene, R.O.C. (1995). Radiographic changes in the lower respiratory tract of West African Dwarf goats suffering from peste des petits ruminants. *Tropical Veterinarian*. **13**:125-134.

Kingsbury, D.W. (1990). *Paramyxoviridae* and their replication. In: Fields BN, Knipe, J.C. *et al.*, (eds) Virology 2nd edition Raven Press NY. pp 945-962.

Lefevre, P.C. (1980). La peste des petits ruminants (synthese bibliographique) *Institute de Elevage et de Medicine Veterinaire des pays Tropicaux* 94700 maisons - ALFort, France 48p.

Libeau, G., Diallo, A., Colas, F. and Guerre, L. (1994). Rapid differential diagnosis of rinderpest and peste des petits ruminants viruses using Immunocapture Enzyme linked immunosorbent assay *Veterinary Records*. **134**: 300-304.

Liess, B. and Plowright, W. (1964). Studies on the Pathogenesis of Rinderpest in Experimental cattle I. Correlation of Clinical Signs, Vireamia and virus excresion by various routes. *Journal Hygiene Campbridge*. **62**: 81-100.

Limo, M. and Yilma, T. (1990). Molecular cloning of rinderpest matrix (M) gene: Complementary Sequence analysis with other paramyxoviruses. *Virology* **175**:323-327.

Mc Culloch, J. (1951). Goat catarrhal fever. In: Eastern Nigeria Annual Report Veterinary Department (1949-1951): 27.

Mathews, R.E.F. (1982), Immunochemistry of viruses, II, the basis for serodiagnosis and vaccines: *Paramyxoviruses, Intervirology* **17**:1-1181.

Mornet, P., Orue, J., Gilbert, Y., Thiery, G. and Sow, M. (1956). La peste des petits ruminants en Afrique occidentale Francaise: ses rapports avec la peste bovine. *Revue Elevage Medicine de Pays Tropicaux*. **9**:313-342.

Nawathe, D.R. and Taylor, W.P. (1979). Experimental infection of domestic pigs with the peste des petits ruminants virus *Tropical Animal Health Production*. **11**:120-122.

Nawathe, D.R. (1984). Control of peste des petits ruminants in Nigeria. *Preventive Veterinary Medicine*. **2**: 147-155.

Nduaka, O. and Ihemelandu, E.C. (1975). The control of pneumonia enteritis complex in dwarf goat of Eastern States of Nigeria by the use of chloroform inactivated tissue vaccine. *Bulletin Animal Health Production Africa*. **23**:341-348.

Obi, T.U., Ojo, M.O., Duroajaiye, O.A., Kasali, O.B., Akpavie, S. and Opasina, D.B. (1983a). Peste des petits ruminants of goats in Nigeria Clinical, epizootiology, Pathogical and Microbiological features. Zbl. *Veterinary Medicine*. **30**: 751-761.

Obi, T.U., Ojo, M.O., Taylor, W.P. and Rowe, L.W. (1983b). Studies on the epidemiology of peste des petits ruminants in Southern Nigeria. *Tropical Veterinarian*. **1**: 209-217.

Obi, T.U. and Ojeh, K.C. (1989). Dot enzyme immunoassay for visual detection of peste des petits ruminants virus antigen from infected caprine tissues. *Journal Clinical Microbiology*. **27**: 2096-2099.

Obi, T.U. and Patrick, D. (1984). The detection of peste des petits ruminants virus antigen by Agar gel precipitation test and counter immuno electrophoresis *Journal Hygiene* (Cambridge). **93**: 579-586.

Ojo, M.O.K. (1976). Caprine pneumonia in Nigeria I. Epidemiological and bacterial flora of normal and diseased tracts. *Tropical Animal Health Production*. **8**: 85-89.

Okoye, J.O.A. (1980). Use of Tissue Culture Rinderpest Vaccine in goat. In discussion during international workshop on PPR held at Ibadan.

Opasina, B.A. and Putt, S.N.H. (1985). Outbreaks of peste des petits ruminants in village goat flocks in Nigeria *Tropical Animal Health Production*. **17**: 219-224.

Pandey, K.D., Baron, M.D. and Barrett, T. (1992). Use of biotinylated probes in differential diagnosis of rinderpest and peste des petits ruminants virus *Veterinary Records*. **131**:199-200.

Plowright, W. (1964). Studies on the pathogenesis of rinderpest in Experimental cattle II: Proliferation of the virus different tissues following intranasal infection. *Journal Hygiene* (Cambridge) **62**: 261-281.

Plowright, W.(1967). Rinderpest virus, Viral monographs. **3**: 25-110.

Provost, A., Maurice, V. and Bourdin, C. (1977). La peste des petits ruminants existe-t-elle en Afrique centrale? cited by Taylor, W.P. (1984). In: The distribution and epidemiology of peste des petits ruminants. *Preventive Veterinary Medicine*. **2**: 157-166.

Pumov. G. (1984). Goat production within the growing system of small holders in Northern Batha. Brazil. In: Small ruminant collaborative research support programme. Technical report series **35**:116.

Roeder, P.L., Abraham, G., Kenfe, G. and Barrett, T. (1994). Peste des petits ruminants in Ethiopian goats. *Tropical Animal Health Production*. **26**:69-73.

Rossiter, P.B., Jesset, D.M. and Taylor, W.P. (1985). Microneutralisation systems for use with different strains of Peste des petits ruminants and rinderpest viruses. *Tropical Animal Health Production*. **17**:75-81.

Rowland, A.C., Scott, G.R., Ramachandran, S. and Hill, D.H. (1971). A Comparative study of peste des petits and Kata in West African Dwarf goats. *Tropical Animal Health Production*. **3**:241-247.

Sahu, S.P. and Dardiri, A.H. (1979). Susceptibility of mink to certain viral animal diseases foreign to the U.S. *Journal Wildlife Diseases*. **15**: 489-494.

Scott, G.R. (1955). Life expectancy of rinderpest virus. *Bulletin Epizootics diseases Africa*. **3**:117-118.

Scott, G.R. and Brown, R.D. (1961). Rinderpest diagnosis with special reference to agar gel double diffusion test. *Bulletin Epizootics diseases Africa*. **9**:83-125.

Scott, G.R. (1965). Peste des petits ruminants and rinderpest viruses. In: Virus Disease of food animal. A world geography of Epizootics and control cited by Gibbs **2**: pp 401-432. **Academy Press London**.

Shamaki, D., Ibu, J.O., Aba-Adulugba, E.P., Oyetunde, I.L., Majiyagbe, K.A., Abegunde, A and Udeani, T.K.C. (1996). Studies on the control of peste des petits ruminants, Parainfluenza type III and Pastereullosis in small ruminants in Nigeria. REFILS in house Review Workshop, N.V.R.I.,

463646

Vom.

Sheshberaderan, H., Norrby, E. McCullough, K.C. Carpenter, W.C. and Orvell, C. (1986). The antigenic relationship between measles, canine distemper and rinderpest viruses studies with mabs. *Journal General Virology*. **67**: 1381-1392.

Snedecor, G.W. and Cochran, W.G. (1980). Statistical Methods. Iowa State University Press Ames. Iowa.

Sterzl, J. and Silvestein, A.M. (1967). Developmental aspects of immunity. In: **Advances in immunology**. Frank *et al*., (eds) Academic press. **6**: 337-384.

Taylor, W.P. (1979a). Serological studies with peste des petits ruminants virus in Nigeria. *Research Veterinary Science*. **26**:236-242.

Taylor, W.P. (1979b). Protection of goats against peste des petits ruminants with attenuated rinderpest vaccine. *Research Veterinary Science*. **26**: 94-96

Taylor, W.P. (1984). The distribution and epedimiology of peste des petits ruminants. *Preventive Veterinary Medicine*. **2**: 157-166.

Taylor, W.P., Al Busaidy, S. and Barrett, T. (1990). The epidemiology of peste des petits ruminants in the Sultanate of Oman. *Veterinary Microbiology*. **22**: 34-352.

Thrushfield M.V. and Hinxman, A.I. (1981). A computerised system for storing and querying clinical case record summaries. *Journal Small Animal Practice*. **22**: 669-679.

Tsukiyama, K., Sugiyama, M., Yoshikawa, Y. and Yamanouchi, K. (1987). Molecular cloning and sequencing analysis of the rinderpest virus mRNA encoding the Haemagglutinin (H) protein. *Virology*, **160**:48-54.

Ugochukwu, M. (1983). The pathology of peste des petits ruminants Caprine Pneumoenteritis. Cited by Hill, D.H. (1983): In the ILCA'S peste des petits ruminants report, Adis Ababa.

Ugochukwu, E.I. (1988). Antibiogram of bacterial isolates from nasal discharges of goats suffering from peste des petits ruminants. *Bulletin Animal Health Production Africa*. **36**: 156-160.

Whitney, J.C., Scott, G.R. and Hill, D.H. (1967). Preliminary observation on a stomatitis and enteritis of goats in southern Nigeria. *Bulletin Epizootics Disease Africa*, **15**:31-41

Wosu, L.A. (1989). Management of Clinical Cases of peste des petits ruminants in goats. Beitr. Tropical, Landwirtsch. *Veterinary Medicine*, **27**: 357-361.

Yamaka, M., Hsu, D., Crisp, T., Dale, B., Grubman, M. and Yilma, T. (1988). Cloning and equence analysis of the Haemagglutinin (H) gene of the virulent strain of rinderpest virus. *Virology*, **366**:251-253.

Yilma, T. Hsu, D., Jones, L., Owens, S., Grubman, M., Mebus, C., Yamanaka, M. and Dale, B. (1988). Protection of cattle against rinderpest with vaccinia virus recombinants expressing HA or F genes. *Science*, **242**: 1058-1061.