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ARTICLE

Peste de Petits Ruminants in Nigeria: A Review.

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SUMMARY

Peste de petits ruminants (PPR) is considered the most important single cause of high morbidity and mortality in sheep and goat eliciting great economic loss to small – holder farmers in Nigeria. It is a contagious rinderpest–like viral disease belonging to the *morbillivirus* group of *paramyxovirus* family of viruses. PPR infection has been recognized in many African countries that lie between the Atlantic Ocean and the red sea, and in recent years in near East and other Asian countries. Its presence close to European borders (western Turkey) makes it a threat to Europe. Confirmation of diagnosis requires the resources of a specialist laboratory since differentiating between PPR virus (PPRV) and rinderpest virus (RPV) can be difficult. Control of PPR outbreak relies on quarantine, ring vaccination and prophylactic immunization.

KEYWORDS: PPRV, control, economic loss, Nigeria.

INTRODUCTION

Nigeria is a large country of considerable diversity with a wide range of agroclimatic condition and corresponding varieties of vegetation suitable for sheep and goat production. The population of small ruminants (sheep and goat) was estimated to be a total of 51 million head throughout the country, with goats outnumbering sheep (FAO, 2006). Goats and sheep are highly important in tropical and sub-tropical livestock production system. They provide small-holder farmers with source of income, while their meat and milk constitute a major source of animal protein to low-income earners (Adamu *et al*., 2005). They sustain the employment and income of millions of people in rural areas, contribute draught energy and manure for crop production and are the only food and cash security available to many Africans (Majiyagbe *et al*., 1992). The animals are also used for sacrifices during religious and traditional festivals (Shamaki *et al.,* 2004). In Nigeria it has been estimated that sheep and goats provide over 35% of the whole animal protein consumed (Shamaki *et al*., 2004). Their skin supports the leather industry and also earns foreign exchange.

As in most developing countries, Nigeria records, high incidence of infectious diseases which constitutes a major impediment to livestock production (Brumby, 1990). Peste de petits ruminants (PPR) is considered the most important single cause of high morbidity and mortality in goats and sheep (Majiyagbe *et al*., 1992; Saliu *et al*., 2008). It is a highly devastating disease of sheep and goats in West Africa (Wosu, 1989). It is contagious rinderpest-like viral disease that is characterized by fever, necrotic stomatitis, gastroenteritis and pneumonia (Anon, 2006). Wherever PPR has been reported, it constitutes a major constraint to sheep and goat farming whether as a small scale backyard farming or on a large scale intensive farming, as periodic outbreaks of the disease literally wipe out almost all the stock of sheep and goats (Shamaki *et al.*, 2004; OIE, 2009). Considering the economic loss elicited by this disease, there is need for constant review of PPR for proper recognition, management and control.

HISTORY AND EPIDEMIOLOGY

Peste de Petits ruminants (PPR) was first described in Cote-d'ivoire, West Africa in 1942 (Radostits *et al*., 2000). Gradually, it was realized that several clinically similar diseases occurring in parts of West Africa shared the same cause (i.e. virus now called Peste de petits ruminants). Investigators soon confirmed the existence of the disease in Ethiopia, 1977 (Pegram and Tereke, 1981), Nigeria, Senegal and Ghana (Taylor, 1984). For many years, it was thought that it was restricted to that part of African continent until a disease of goats in Sudan, which was originally diagnosed as

rinderpest in 1972 was confirmed to be PPR (Shaila *et al*., 1996; FAO, 1999; Agnes *et al*., 2008). PPR infection has been recognized in many of the African countries (Wosu, 1994; Ashley *et al*., 2010), that lie between the Atlantic ocean and the Red Sea, i.e. to the North by Egypt and South to Kenya in the East, and Gabon in the West (FAO, 1999). In recent years, the disease has been reported in near East (Ashley *et al.,* 2010) Turkey (Kul *et al*., 2007; Albayak and Alkan, 2009), Arabian Peninsula (Abu-Elzein *et al*., 1990; Al-Dubaib, 2009), India (Shaila *et al*., 1989; Chavran *et al*., 2009), Pakistan (Zahur *et al*., 2009) and other Asian countries (Furely *et al*., 1986; Wang *et al.*, 2009). Recent reports of PPRV in areas close to European borders (Western Turkey) have increased its profile both scientifically and in the media. This makes PPRV a threat to Europe (Ashley *et al*., 2010).

Most market goats harbor the virus which they transmit readily in close contact and some develop the disease when confined by housing (Radostits *et al*., 2000). The dry harmattan periods which coincides with the time when sheep and goats are allowed to graze in the fallows after crop harvest was recorded to be the highest period of incidence of PPR in Nigeria (Ezeibe *et al*., 2008). This view was observed earlier by Obi (1983) and Okoli (2003). It is the same period that the sahelian goats and sheep believed to have high innate resistance to the virus are moved south wards and commingles with the breeds in humid, sub humid tropics (Radostits *et al*., 2000). Trade in small ruminants, especially at markets where animals from different sources are brought into close contact with one another increases the opportunities of PPR transmission (FAO, 1999).

ETIOLOGY

The virus which causes PPR, the peste des petits ruminants virus (PPRV), belongs to the *morbillivirus* group of *paramyxoviridae* family of viruses (Anon, 2008). It is closely related to the rinderpest virus of cattle and buffaloes, the measles virus of humans, the distemper virus of dogs and some wild carnivores, and the *morbilliviruses* of aquatic mammals (Radostits *et al*., 2000). The PPR virus has now been group into four according to in-depth studies and its genetic characterization (Fosyth and Barrett, 1995). There are three groups from Africa and one from Asia. One of the African groups of PPRV is also found in Asia (Furley *et al*., 1986). Universal primer sets, which recognize all *morbilliviruses* have been developed, which are based on sequence within the *phosphoprotein* (P) and *nucleocapsid* protein (N) genes, both of which have regions that are highly conserved across the genus (Farooq *et al*., *2008*). PPR virions, as other *morbilliviruses, are* enveloped, pleomorphic particles containing single strand RNA as the genome. It is composed of 15,948 nucleotides (Chauhan *et al*., 2009), the longest of all *morbillivirus* genomes sequenced so far. This genomic RNA is wrapped by the nucleoprotein (N) to form the *nucleocapsid* into which are associated two other viral proteins (Chauhan *et al*., 2009); the *phosphoprotein* (P) and the large protein L, the viral RNA depend on RNA polymerase (RdRp). To the viral envelop which derives from the host cell membrane *are* associated three viral proteins: the matrix protein (M) which is located inside the envelope and serves as a link between the nucleocapsid and the two external viral proteins, the fusion protein (F) and the heammagglutinin (H). The heammaglutination allows the virus to bind to the cell receptor during the first step of the viral infection process. By their positions and their functions, both F and H are very important for the induction of protective host immune response against the virus. However, N the most abundant and also the most immunogenic among PPRV proteins does not induce protective immunity against the virus (Shamaki *et al*., 2004; George *et al*., 2006; Chauhan *et al*., 2009). It has been used in the development of diagnostic tests and in recent studies with very closely related lineage IV isolates. It has been suggested that the N gene is more divergent and therefore more suitable for phylogenetic distinction between close related circulating viruses (Ashley *et al*., 2010; Kwiatek *et al*., 2010). Sequence analysis of F and N gene sequence data revealed that the PPR viruses from distance geographical regions vary to a greater extent in their N gene sequences than in their F gene sequence (Kerur *et al*., 2008). In their report, Shamaki *et al*. (2004) showed that the sequence analysis of the various PPR viruses based on the nucleoprotein (N) gene study, as reported earlier on PPR viruses in Nigeria belong to the same group. The Nigeria PPRV isolates (PPRV 75/1, 75/2, and 75/3, 76/1) which were described by Taylor and

Abegunde (1975), were well studied by Shamaki *et al*. (2004) and found to fall within the same group as the isolate from Senegal and one isolate from Sudan. PPR virus can be destroyed at 50°C/60 minute, inactivated at PH<4.0 or >11.0 and can survive for long period in chilled and frozen tissues (OIE, 2009).

SUSCEPTIBILITY AND TRANSMISSION

Peste de petits ruminants (PPR) is a highly devastating disease of sheep and goats (Wosu, 1989; Shamaki *et al*., 2004). Natural disease may also occur in the gazelle (*Douces gazalla dorcas*), laristan sheep (*ovis orientalis laristanical*), gemsbok (*oryx gazelle*), ibex (*capra ibexnubiana*) and deer (Radostits *et al*., 2000). Cattle, buffaloes, camels and pigs can become infected but there is little or no evidence of the disease associated with their infection (FAO, 1999). Ogunsanmi *et al*. (2003), detected PPR virus antibodies in African grey duiker (*Sylvicapra grimmia*). In Nigeria, Shamaki *et al*. (2004) recovered PPR RNA from tissues of camels and warthog in addition to sheep and goats. Up to 100% of animals in a flock may be affected in a PPR outbreak with between 20-90% dying (FAO, 1999). The percentage of sheep and goats with antibodies to PPR increases with age (Majiyagbe *et al*., 1991; Luther *et al*., 2006). The disease, however, is more severe in goats than in sheep and is rapidly fatal in young animals of between 4-18 months of age, since maternal antibodies are lost at about 4 months of age (Radostits *et al*., 2000). Females were also reported to have higher risk of acquiring the infection than males (Pumov, 1984; Luther *et al*., 2006).

Discharges from the eyes, nose, mouth, and loose faeces contains large amounts of the virus is characteristic of the disease. Abengunde and Adu (1976), reported that infected goats excreted the PPR virus from their nasal and ocular routes at the onset of diarrhea. Gibbs (1979) also reported that infected goats excreted PPR virus in their saliva and urine. PPR virus was also confirmed in the faeces of clinically sick animals (Wosu *et al*., 1990) and recovered goats (Ezeibe *et al*., 2008). This led Ezeibe *et al*. (2008) to suggest carrier status which according to them may explain how the disease is maintained between season of low-incidence and periods of high incidence. Infective droplets are released in to the air from these secretions and excretions especially when affected animals cough and sneeze. Inhalation of these droplets by other animals leads to infection, although close contact (Nduaka and Ihemelandu, 1973; FAO,1999; Radostits *et al*., 2000) is the most important way of transmitting the disease. Ezeibe *et al*. (2008) described the possibility of spreading the infection to other farms with no history of recent contact, through soles of shoes and vehicle tyres when infected faeces are transported and used as manure to fertilize farms and pastures as is the practice in West Africa. It is also suspected that infectious materials can contaminate water and feed troughs and bedding, turning them into additional sources of infection (FAO, 1999). These particular hazards are, however, probably fairly short- term since the PPR virus, like its close relative rinderpest, would not be expected to survive for long outside the host (OIE, 2009).

CLINICAL FINDINGS

Incubation period is 4-6 days after natural infection. Disease severity depends on various factors i.e. PPRV lineage, species, breed, and immune status of the animals (OIE, 2009). The infection result in an acute, highly contagious disease characterized by sudden onset of fever with rectal temperature of at least 40 - 41.3°C (Radostits *et al*., 2000; Anon, 2006). Affected animals are markedly depressed and appear sleepy. Their hair stands erect giving them a bloated appearance (FAO, 1999). After this stage, there is anorexia, discharges from the eyes, nose and mouth. At the beginning, the discharges are watery, but later becoming thick, causing matting of the eyelids, obstruction of the nose and difficulty in breathing (Anon, 2006). Oral necrotic and erosive lesions in lips, gums, tongue, palate and cheeks are seen. Affected animals resist attempts to open their mouths because of pain (FAO, 1999). Two to three days (2-3) after the onset of fever, diarrhea commonly appears. It may be watery, foul-smelling and may contain blood and dead tissues (FAO, 1999; WHO, 2009).

Seriously affected animals show difficult and noisy breathing, marked by extension of head and neck, dilation of the nostrils, protrusion of the tongue (FAO, 1999) and painful coughs which may be complicated by secondary bacterial infection (Abdulkadir, 1989; Okoli,

2003; OIE, 2009). This eventually led to dehydration and death. Later stages are commonly followed by formation of small nodular lesions on the lips. Morbidity is up to 100% in a flock and mortality is 20-90%. Pregnant animals may abort (FAO, 1999).

LESIONS

Lesions associated with PPR are very similar to those observed in cattle affected with rinderpest (OIE, 2009). The carcass of an affected animal is usually emaciated, hindquarters are soiled with fluid faeces (FAO, 1999), and eye balls sunken.

Mouth & lips - Necrotic lesions are observed on the gums, soft and hard palates, the cheeks near the commissures, the tongue and pharynx. The erosions are shallow, with a red, raw base and later become pinkish white; they are bounded by normal epithelium that provides a sharply demarcated margin (Anon, 2006). The lips are swollen, with crusty scabs along the outer surfaces or nodules in late stages (FAO, 1999).

Respiratory Tract – The nasal cavity is congested with creamy exudates, and erosions. Petechiae may appear in the turbinates, larynx, and trachea (Anon, 2006). The lungs are covered with dark red or purple areas which are firm to touch (evidence of pneumonia).

Lymph Nodes – Are soft and enlarged. The entire patches of lymphoid tissue may be sloughed (Radostits *et al*., 2000).

Intestines – Abomasum and small intestines are congested. There are haemorrhages (or zebra stripes) seen in the large intestine, commonly at the caeco-colic junction (OIE, 2009). Streaks of haemorrhages, and less frequently erosions, may be present in the first portion of the duodenum and terminal ileum. Peyer's patches are severely affected (Anon, 2006).

DIAGNOSIS

Differentiating between rinderpest and PPR virus to obtain a definitive identification of PPR can be difficult. Confirmation requires the resources of a specialist laboratory e.g. National Veterinary Research Institute (NVRI), Vom-Nigeria, where confirmatory diagnosis of PPR is routinely performed.

After the clinical signs and epidemiology of the disease have been considered, the samples of choice for confirmation of diagnosis of PPR are the tears, gum debris, tissues of mediastinal and mesenteric lymph-nodes, portions of spleen and lungs, unclotted blood and clotted blood or serum (FAO, 1999; Anon, 2006; OIE, 2009). The international office of epizootic (OIE) manual of standards for diagnostic tests and vaccines contains guidelines on the collection of samples and the diagnostic technique for diagnosis of PPR infection (FAO, 1999). Detection of viral antigens by complement fixation or agar-gel precipitin tests does not differentiate the disease from rinderpest (FAO, 1999; Anon, 2006).

Histopathology combined with immuno-histochemical staining (e.g. immunoperoxidase) is a useful procedure because it is performed on formalin-fixed material and can discriminate between PPR and rinderpest when performed with specific monoclonal antibodies (Brown *et al*., 1991; Sumption *et al*., 1998; FAO, 1999). Virus antigens can also be detected by immunocapture ELISA (ICE). It is rapid and sensitive, and differentiates between PPR and rinderpest. Standardized reagent kits for ICE are commercially available (Libeau *et al*., 1994; FAO, 1999; OIE, 2009).

Reverse transcriptase polymerase chain reaction (RT- PCR) for detection of virus genetic material requires a specialist facilities and expertise (FAO, 1999). Despites its high cost, it is now one of the tests used most frequently in reference centres, together with enzyme linked immunosorbent assay (ELISA), because it is rapid, accurate, highly sensitive and can discriminate between PPR and rinderpest (Forsyth and Barreth 1995; Couacy–Hymann *et al*., 2002; Saravannan *et al*., 2004; Kumar *et al*., 2007). Counter immunoelectrophoresis (CIEP) is the most rapid test for viral antigen detection (Majiyagbe *et al*., 1984; OIE, 2009). It is carried out on a horizontal surface using a suitable electrophoresis bath, which consists of two compartments connected through a bridge. The presence of 1-3 precipitation lines between pairs of wells is a positive reaction.

Wosu, (1985) demonstrated the heamagglutinin or PPR homogenate antigen, to porcine erythrocytes. With this knowledge, Wosu and Ezeibe (1992) demonstrated that it is possible to

make a definitive serological diagnosis of PPR specific antibodies by haemagglutination – inhibition test using the adsorption technique.

DIFFERENTIAL DIAGNOSIS

Frequently, PPR is confused with other diseases which have grossly similar clinical signs. These diseases include rinderpest, foot-and-mouth disease, bluetongue, contagious ecthyma (Orf), Pneumonic pasteurellosis, contagious caprine pleuropneumonia (CCPP), and gastro-intestinal helminth infestations (FAO, 1999; Radostits, 2000; Anon, 2006).

The most frequent sources of confusion are:- The mouth lesions, which could be due to rinderpest, foot and mouth disease, bluetongue or contagious ecthyma; difficult breathing, which could be due to Pneumonic pasteurellosis or Contagious caprine pleuropneumonia; or diarrhea which could be due to coccidiosis or gastro-intestinal helminth infestations (FAO, 1999).

Rinderpest –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 (FAO, 1999). Specialist laboratory is required to differentiate the two.

Pneumonic pasteurellosis – This is a purely respiratory disease of sheep and goats caused by the bacterium *Pasteurella haemolytica*. It is a secondary infection of PPR, a consequence of the immune depression that is induced by the causal agent of PPR, (PPRV) (OIE, 2009). In primary pneumonic pasteurellosis, the numbers of affected and dead animals are usually lower than for PPR except under exceptional conditions of stress and overcrowding (Okoli, 2003). There are no oral lesions or diarrhea in primary pasteurellosis (FAO, 1999). Diagnostic tests for detecting PPRV should be carried out in all suspected cases of Pneumonic pasteurellosis where there is a risk of PPR.

***Contagious Caprine Pleuropneumonia* (CCPP)**
This is a disease of goats caused by *Mycoplasma sp*. Like PPR, it is characterized by fever, difficult or abnormal breathing and coughing, but there are no mouth lesions or diarrhea. At post mortem examination, the lung lesions in CCPP are more diffuse and a fibrinous fluid is found in chest cavity. Fibrin deposits cover the lungs (Anon, 2008). Laboratory testing can rule out PPR in PPR high –risk areas.

Foot and Mouth Disease (FMD)
Commonly seen in sheep than goats. The most important distinguishing features of FMD, other than the appearance of lesions, are the absence of breathing problems and diarrhoea, and the presence of lameness (FAO, 1999; Anon, 2006).

***Contagious ecthyma* (Orf):** This is often confused with PPR because of the nodules and thick scabs sometimes seen on lips in the late stages of PPR. In uncomplicated Orf, there is usually no oral necrosis, diarrhea or pneumonia (FAO, 1999; Radostits *et al*., 2000).

Blue tongue: Like PPR, blue tongue is characterized by fever, discharges and oral lesions. It however differs from PPR by presence of edema of the head region, bluish discoloration of the oral cavity, the coronary band of the hooves and less hairy parts of the body and lameness (Radostits *et al*., 2000).

Coccidiosis: This is a disease caused by protozoa of the genus Eimeria. It is characterized by bloody diarrhea. Breathing problems and oral lesions are absent in coccidiosis. Laboratory identification of the causative agent of coccidiosis is confirmatory (Radostits *et al*., 2000).

MANAGEMENT AND CONTROL

There is no specific treatment; however, treatment for bacterial and parasitic complications decreases mortality in affected flocks or herds (Anon, 2008). Fluid replacement therapy is essential for valuable animals (Ogunsanmi *et al*., 2003). But OIE, (2009) also 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 (Anon, 2006; OIE, 2009). 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 (OIE, 2009). In Nigeria, various

research findings recommend ethno-veterinary treatment as an acceptable means of managing PPR disease. The use of such preparations along with PPR vaccine to improve the efficacy of the treatment is acceptable among rural farmers (Okoh, 2003; Saliu *et al*., 2007; Saliu *et al*., 2008).

Requirements for vaccines – Until recently, the most practical vaccination against PPR made use of tissue culture rinderpest vaccine. But its' use to protect small ruminants against PPR is now contraindicated because it produces antibodies to rinderpest which compromise serosurveillance for rinderpest and thereby the Global Rinderpest Eradication Programme (OIE, 2009). Strategic ring vaccination and / or vaccination of high-risk population are usually considered in outbreak situations. A homologous PPR vaccine is available. In 1998, the OIE international Committee endorsed the use of this vaccine in countries that have decided to follow the 'OIE path way' for epidemiological surveillance for rinderpest in order to avoid confusion when serological surveys are performed (OIE, 2009). The PPRV vaccine which is a Nigerian strain (Nigeria 75/1), is a live vaccine cultured in vero cells. The original strain of the virus was isolated in Nigeria in 1975 (Taylor and Abegunde, 1975). The vaccine is stored in freeze-dried form at -20^{0}C. It is available in Nigeria, produced by National Veterinary Research Institute (NVRI) Vom, Nigeria.

CONCLUSION

PPR is regarded as the most important disease of goats and sheep in Nigeria, where these animals are a major source of animal protein. It is also the bane of entrepreneurs just going into small ruminant production but later suffer heavy losses. Even at a give-away price of one thousand five hundred naira for a small ruminant, the total financial value of small ruminant industry in Nigeria could be put at about 84.9 billion naira based on the estimated population of 56.6 million sheep and goats. This is why PPR is considered the most important single threat to small ruminant industry in Nigeria due to its periodic devastating outbreaks and subsequent effect on sheep and goats population.

The choice of diagnostic test among the various tests enumerated is dependent on availability, sensitivity, specificity, rapidity and economic factors. Therefore, it is paramount to embark on the eradication of PPR through mass vaccination, quarantine, mass slaughter of infected and carrier animals. If practiced properly, it will surely sustain employment and income to the rural people, contribute to source of manure for crop production and also provide raw materials to the leather industries.

RECOMMENDATIONS

Sheep and goats should be vaccinated as from four (4) months of age. Government should embark on vaccination campaign programmes, aimed at whole village flocks instead of individual or household flocks.

Government should also recognize the vital role of these animals and encourage or assist in the timely provision of PPR vaccines to the public. Governments should collaborate with each other within West Africa to set up a sub-regional programme to control and eradicate the disease. Research institutes such as NVRI Vom and Universities should intensify research geared towards the production of vaccines that are specific to the viral strain present in Nigeria.

REFERENCES

ABENGUNDE, A. A and ADU, F.D (1976): Excretion of the virus of peste des petits ruminants by goats. *Bull of Epizoo. Dis. Afric.* **25**: 307-311.

ABDULKADIR I. A. (1989): Infectious diseases of livestock in Nigeria: An Outline, Ahmadu Bello University press Limited. 173-174

ABU-ELZEIN, E.M.E., HASSANIEN M.M., AL-AFALEG A.I., ABUDELHALI M.A., HONSAWI F.M.J. (1990): Isolation of peste despetits ruminants from goats in Saudi Arabia. *Vet. Rec.* **27**: 309

ADAMU S; EKUNDAYO, O.S; USEH, N.M; NEILS J.S; BISSALLAH, M; and ESIEVO K.A.N (2005): Serum levels of some Electrolytes in diarrhoeic Savanna Brown goats in Zaria. 42nd congress of Nigerian Vet. Medical Asso. Nov. 14 -18, 2005,Maiduguri, Nigeria.

AGNES W., FRANCIOS, R., CHAVERNAC, D., MARIAM, L., GENEVIEVLIBEAU, Y., PFEIFFER, D. U. and GUITIAN, J. (2008): Peste de Petits Ruminants (PPR) in Ehtiopia: Analysis of National Serological Survey. MBC *Vet. Res.* **4**:34 doi:10.1186//1746-6148-4-34.

ALBAYAK, H. and ALKAN, F. (2009): PPR virus infection of sheep in black sea region of Turkey: epidemiology and diagnosis by RT – PCR and virus isolation. *Vet. Res. Commun.* **33**: 241 – 249.

AL-DUBAIB, M. A. (2009): Peste des petits ruminants morbillivirus infection in lambs and young goats at

Qassim region, Saudi Arabia. *Trop. Anim. Hlth. Prod.* **41**: 217 – 220.

ASHLEY, C., PARIDA, B. S., BATTERN, C., OURA, C., KWIATEK, O., and LIBEAU, G. (2010): Global distribution of Peste des petits ruminants virus and prospects for improved diagnosis and control. *J. Gen. Virol.* **91**: 2885

ANONYMOUS (2006): Mercks Veterinary Manual, 2008. 18th ed. Merck Publishing, Merck & Co., Inc. NJ, USA.

BROWN C.C, MARINER J.C. and OLANDER H.J. (1991): An immunohisto-chemical study of peste des petits ruminants virus. *Vet. Pathol*. **28**: 166-170.

BRUMBY, P. (1990): System and Livestock development in tropical Africa. In: Animal Disease as a factor limiting economic development in Africa. *Cornell Vet*. **80**:17-25.

CHAUHAN, H.C., CHANDEL, B.S., KHER, H.N., DADAWALA A.I. and AGRAWAL., S.M. (2009): Pestes des petits ruminants virus infection in animals. *Vet. Wrld.* **2(4)**: 150 – 155

CHAVRAN, V., DIGRASKAR, S., and BEDARKAR, S. (2009): Seromonitoring of peste des petits ruminants virus (PPR) in goats (*capra hircus*) of parbhani region of maharastra. *Vet. Wrld.* **2**: 299 – 300.

COUACY – HYMANN, R.F., HURARD, C., GUILLOU, J.P., LIBEAU, G. and DIALLO, A. (2002): Rapid and sensitive detection of peste des petits ruminants virus by a polymerase chain reaction assay. *J. virol. Methods*, **100**: 17-25.

EZEIBE, M.C.O., EZE, I.C., and NGENE, A.A. (2008): Persistent Detection of Pestes de Petits Ruminants Antigen in the faeces of Recovered Goats. *Trop Anim Health Prod* **40**, 517–519.

F.A.O. (1999): Recognizing peste Des Petits Ruminants. A Field manual. Prepared by Dr. P.L Roeder and Prof. T.U. Obi of food and Agriculture Organizations of the united Nations (FAO). http://www.fao.org//DOCREP/003/-X1703E/X1703E00.HTM

FAO (2006): FAOSTAT database. Food and Agricultural Organisation, Rome, Italy. www.fao.org/.../ess/-publications-studies/...yearbook/**fao**-statistical-yearbook-2005-**2006**/en

FAROOQ, U., KAN, Q.M. and BARRETT, T. (2008): Molecular based diagnosis of rinderpest and peste des petits ruminants virus in Pakistan. *Int J. Agric. Biol.* **10**: 93 – 96.

FORSYTH, M.A. and BARRETT, T. (1995): Evaluation of Polymerase chain reaction for the detection and characterization of rinderpest and peste de petit ruminants viruses for epidemiological studies. *Virus Res.* **29**:151-163.

FURELY, C.N; TAYLOR, W.P; OBI, T.U. (1986): An outbreak of PPR in a Zoological collection, *Vet. Rec.* **121**: 443-447.

GEORGE, A., DHAR, P., SREENIVASA, B.P., SINGH, R.P. and BANDYOPAHYAY, S. K. (2006): The M and N genes based simplex and multiplex PCR's are better than the F or H gene simplex PCR for peste des petits ruminants virus. *Acta Virol.* **50**: 217-222.

GIBBS, E.P.J., TAYLOR, W.P., LAWMAN, H.P., BRYANT, J. (1979): Classification of Peste des Pestits ruminant's virus as a fouth member of Genus morbillivirus. *Inter. Virol.* **11**: 268-278.

KERUR, N., JHALA, M.K. and JOSHI C. G. (2008): Genetic characterization of Indian peste des petits ruminants virus (PPRV) by sequencing and phylogenetic analysis of fusion protein and nucleoprotein gene segments. *Res. Vet. Sci.* **85**: 176 – 183.

KUL O, K., KABAKCI, N., ATMACA, H. T. and OZHUL, A. (2007): Natural peste des petits ruminants virus infection: novel pathologic findings resembling other morbillivrus infection. *Vet. Pathol.* **44**: 479 – 486.

KUMAR, C.S., RAJ, G.D., THANGAVELU, A. and SHAILA, M.S. (2007): Performance of RT- PCR- EUSA for the detection of pest petits ruminants virus. *Small Rumin. Res*, **72**: 200-208.

KWIATEK, O., KEITA, D., GIL, P., FERNANDEZ – PINERO, J., JIMENEZ CLAVERO, M.A., ALBINA, E. and LIBEAU, G. (2010): Quantitative one-step real-time RT –PCR for the fast detection of the four genotypes of PPRV. *J. Virol. Methods*, **165**:168 – 177.

LIBEAU, G., DIALLO, A., COLAS, F. and GUERREL, L. (1994):Rapid differential diagnosis if rinderpest and peste des petits ruminants using an immunocapture ELISA. *Vet. Rec*. **134**: 300-304.

LUTHER, N.J., UMOH, J.U., MAJIYAGBE, K.A., SHAMAKI, D.,NWOSU, C.I., DOGO, G. (2006): Studies on the prevalence of antibodies to peste de petits Ruminants virus (PPRV) Among Goats in Bauchi State. *Nig. Vet. J.* **27(I)**: 17-22.

MAJIYAGBE, K.A., NAWATHE, D.R. and ABEGUNDE, A. (1984): Rapid diagnosis of PPR infection, application of immuno- etectro-osmophoresis (IEOP) technique. *Rev. Elev. Med. Vet. Pays Trop*, **37**: 11-15

MAJIYAGBE, K.A., AYENI, A., IDOWU, S.O. and SHAMAKI, (1991): Effect of Age on Immune Response of West African Dwarf (WAD) sheep to Tissue Culture Rinderpest Virus (TCRV) vaccine. (Submitted to *Nigerian Journal of Immunology)*.

MAJIYAGBE, K.A., SHAMAKI, D., KULU, D. and UNDEANI, T.K.C. (1992): Peste des Petite ruminants (PPR) and Rinderpest (RP) antibodies in clinically normal small ruminants in Cameroon and Nigeria. SMALL RUMINANT RESEARCH AND DEVELOPMENT IN AFRICA. Proceedings of the second Biennial Conference of the African small ruminant research network, AICC, Arusha, Tanzania.

NDUAKA, O. and IHEMELANDU, E.C. (1973): Observation of Pneumo-enteritis
complex in Dwarf goats in Eastern States of Nigeria, preliminary report. *Bull.*
Epiz. Dis. Afr. **21**:87-89.

OBI, T.U. (1983): Studies on the epidemiology of PPR in southern Nigerian. *Trop. Vet.*
1: 209-217.

OGUNSAMI, A.O., AWE, E.O., OBI, T.U. and TAIWO, V.O. (2003): Peste des petits Ruminants (PPR) virus antibodies in African grey Duiker (Sylvicapra grimmia). Afr. J. Biomed. Res. **6(1)**: 59-61.

OKOH, A.E.J, (2003): Ethno veterinary Medicine and Small scale livestock farming in Nigeria In: Obime. C.P, B.A. Kalu and J.E. Umeh (Eds), Indegenous Knowledge and sustainable Agricultural Development in Nigeria. Proceedings of the National conference on indigenous knowledge and development held in October 22-25, 2003, at University of Agriculture, Makurdi, Benue State.

OKOLI, I.C. (2003): Incidence and modulating effects of environmental factors on trypanosomosis, Peste des Petits Ruminants (PPR) and bronchopneumonia of

West African dwarf goats in Imo State, Nigeria. *Liv. Res. Rul. Dev.*, **15 (9)**: article 6. http://-www.utafoundation.org lrrd159/okoli159.htm

PEGRAM R.G and TEREKE F. (1981): Observation on the health of Afar Livestock, *Ethopian Vet.* **5**:4-11.

PUMOV, G. (1984): Goat production within the growing system of small holders in Northern Batha, Brazil. In small ruminant Collaborative research support programme, *Tech.l Rep. Series*, **365**:16

RADOSTITS, O.M., BLOOD, D.C., and GAY, C.C. (2000): Veterinary Medicine (A textbook of the diseases of cattle, sheep, pigs, goats and horses), 9th ed., W.B. Saunders, London, UK

SALIU, O.J., AUDU, S.I. and OLAOLU, M. (2007): Comparative attitudinal disposition on sheep/goat farmers to the adoption of vaccination and Ethnoveterinary treatments of PPR Disease in Ijumu, L.G.A of Kogi State. In; Olufajo, O.O., D.F. Omokore, G.N. Akpa and S.A. Sanni (Eds), Reviving Agriculture for sustainable National Growth and Stable democracy. Proceeding of the 41st Annual conference of the Agricultural Society of Nigeria (ASN) held at IAR, Samaru ABU Zaria, Nigeria, 553-567.

SALIU, O.J., AUDU, S,I., SANDA, M.E., ARIBIDO, S.O. and OLAOLU, M. (2008): Adoption of vaccination and Ethnoveterinary treatment for peste des petits ruminnats (PPR) among sheep and goat farmers in Ijumu local Government area of Kogi State. *Nig. Agric. J.* **3(5)**: 404-408.

SARAVANAN, P., SINGH, R.P., BALAMURUGAN, V., DHAR, P.,SREENIVASA, B. P, MUTHU CHELVAN, D., SEN, A., ALEYAS, A.G., SINGH, R.K. and BANDYOPADYAY, S.K. (2004): Development of an N gene-based PCR-ELISA for detection of peste-despetits- ruminants virus i n chinical samples. *Acta virol*. **48**: 249-255.

SHAILA, M.S., PURUSHOTHAMAN, V., BHAVASAR, D., VENUGOPAL, K. and VENKATESAN, R. A. (1989): Peste des petits ruminants of sheep in India. *Vet. Rec.* **125**: 602.

SHAILA, M.S., SHAMAKI, D., FORSYTH, M., DIALLO, A., GOATLEY, L. and KITCHING, P. (1996): Geographic distribution and epidemiology of peste des petits ruminants virus. *Virus Res.* **43**:149-153.

SHAMAKI, D., OLALEYE, O.D., OBI, T. U., DIALLO, A., MAJIYAGBE, K.A., LOMBIN, L.H., BARRETT, T. (2004): Peste Des petits Ruminants (PPR) in Nigeria: Serological and Molecular Epidemiology. *Vom J. Vet. Sci.* **1**: 18 – 27.

SUMPTION, K.J., ARADOM, G., LI BEAU, G. and WILSMORE, A.J. (1998): Detection of peste des petits ruminants antigen in conjunctiral smears of goats by indirect immuno fluorescence. *Vet. Rec*. **142**: 421-424

TAYLOR, W.P. and ABEGUNDE, A. (1975): The Isolation of peste des petits ruminants virus from Nigerian sheep and goats. *Res. Vet. Sci*. **26**: 94-96.

TAYLOR W.P. (1984): The distribution and epidemiology of peste des petits ruminants. *Prev. Vet. Med.* **2**:157-166.

WANG, Z., BAO, J., WU, X., LIU, Y., LI, L., LIU, C., SUO, L., XIE, Z., ZHAO, W. and other authors (2009): Peste des petits ruminants virus in Tibet, China. *Emerg. Infect. Dis*.**15**: 299 – 301.

WHO(2009):WHO,TechnicalDiseasecards.http://www.cfsph.iastate.edu/FactSheets/pdfs/pes_des_petits_ruminants.pdf

OIE, (2009): OIE manual of Diagnostic Tests and vaccines for Terrestrial Animals. http://www.oie.int/eng/-normes/MMANUAL/2008/pdf/0.01.1b_FOREWORD.pdf

WOSU L.O. (1985): Agglutination of Red Blood cells by PPR Virus. *Nig. Vet. J.* **14**: 54 – 58.

WOSU, L.O. (1989): Management of clinical cases of PPR disease in goats. *Beitr trop Land Wirtecl: Vet. Med.* **27**: 342-352.

WOSU; L.O; OKIRI, J.E; and ENWEOZOR, A.P. (1990): Optimal time for vaccination against PPR disease in goats in Humid Tropical Zone in Southern Nigeria. *Arch. Roum Path. Exp. Mcrobial Res.* **49**: 283-291.

WOSU, L.O. and EZEIBE, M.C.O. (1992): Haemagglutination– inhibition technique for definitive diagnosis of peste des petits ruminants virus specific antibody. *Bieitr trop. Landwirtch Vet. Med.* **30 (30)**: 299-304.

WOSU L.O (1994): Current status of peste des petits ruminants PPR disease in small ruminants. A review article. *Stud. Res. Vet. Med.* **2**: 83 – 90.

ZAHUR, A. B., ULLAH, A., IRSHAD, H., FAROQ, M. S., HUSSAIN, M. and JAHANGIR, M. (2009): Epidemiological investigations of a peste des petitis ruminants (PPR) outbreak in Afghan sheep in Pakistan. *Pakistan Vet. J.* **29**: 174 – 178.