ABSTRACT

The study was conducted at Thana Veterinary Hospital, Shyamnagar, Satkhira from 26th June to 25th August 2010, to find out the prevalence of PPR and its treatment in sheep and goat. During the study period a number of 223 diseased goats were examined of which 111 (49.78%) *peste des petits ruminants* (PPR) and 80 diseased sheep were examined of which 24 (30%) *peste des petits ruminants* (PPR); cases were diagnosed on the basis of clinical sings and gross pathological lesions. Dull and depression, rough hair coat, dyspnoea, diarrhoea, swollen and erosion of lip and occulonasal discharges were the common findings during clinical examination of PPR patients. The gross pathological lesions found during postmortem examinations were consolidated pneumonic lungs, hemorrhagic trachea, necrosis and hemorrhagic plugs in caecum and characteristics zebra striping in the mucosa of colon. Goats were more susceptible (49.78%) than sheep (30%). Male goats were more susceptible (58.52%) than female goats (41.48%). Mortality rate in goat is more (58.55%) than sheep (45.83). 7-12 months of age was found more prone (in sheep 66.67% and in goat 55.86%) to PPR than other age. Black Bengal goats were more susceptible (55.41%) than Jamunapari goats (36.36%). The combined therapy of atropine, antihistaminic aided antibiotic + fluid therapy is more (56.06%) effective than Atropine aided antibiotic + fluid therapy (33.33%) or Antihistaminic aided antibiotic + fluid therapy (29.17%).

Key words: PPR, prevalence, clinical signs, gross pathological lesion, treatment, vaccination.

Chapter I

INTRODUCTION

Goat is considered as the poor man's cattle. There is a significant population of goats (39.7 million) in Bangladesh **(DLS, 1999)**. There are several diseases in goat which cause higher mortality. In present conditions goats are affected with a serious exotic killer disease (**Debnath, 1995**). *Peste des petits ruminants (PPR) is* a highly contagious systemic disease of sheep and goats which is caused by a morbilli virus that is closely related to rinderpest of cattle (**Diop *et al.*, 200***5;*** **Roeder *et al.*, 1994**). There are several diseases of goat specially *peste des petits ruminants* (PPR), which cause higher mortality and great economic losses. PPR is an exotic disease of goats in Bangladesh (**Debnath, 1995; Islam *et al.*, 2001).**

Peste des petits ruminants (PPR) is an acute febrile viral disease of small ruminants, characterized by mucopurulent nasal and ocular discharges, necrotising and erosive stomatitis, enteritis and pneumonia (**Singh *et al.*, 2004**).The course of the disease might be per acute, acute, sub-acute or chronic and after an incubation period of 5-6 days, clinical sign develop, including fever, anorexia, necrotic stomatitis, gingivitis and diarrhea (**Lefever and diallo, 1990)**. *Pestides des petits ruminants* virus might lead to high economic losses where mortality in goats could reach 95% and in sheep only slightly less (**Murphy *et al.*, 1999**). PPR occurs in most African countries, the Middle East and the Indian subcontinent (**Abraham *et al.*, 2005; EI-Hag and Tylor**, **1984; Lefevre *at al.*, 1991**). *Pestedes petits ruminant* (PPR) is a contagious viral infection of small ruminants particularly in goats. PPR also called pseudo-rinderpest of small ruminants, goat catarrhal fever, pests of sheep and goats, kata, stomatitis-pneumoenteritis syndrome, Pneumoenteritis complex and goat plague (**Chakrabarti, 2003**). PPR was first described in Ivory Coast (**Gargadennec and Lalanne, 1942**) and then after, it has been recognized in many of the sub-saharian countries that lie between the Atlantic Ocean and the Red Sea (**Lefevre and Diallo, 1990**).

The affected area extends north to Egypt and south to Kenya, in Eastern-Africa, and to Gabon, in Western-Africa. PPR has not been recognized in most of Northern and Southern Africa. It is also widespread in India and southwest Asia (**Shaila *et al.*, 1989**). Presently, PPR occurs in most African countries situated in a wide belt between the Sahara and equator, the Middle East (Arabian Peninsula, Israel, Syria and Jordon) and the Indian subcontinent. Outbreaks of PPR are now known to be common in India, Nepal, Bangladesh, Pakistan and Afghanistan (**Abdollahpour *et al*, 2006**). In Bangladesh, the PPR virus was identified during a severe outbreak in 1993 (**Sil *et al.*, 1995**) which was further confirmed by World Reference Laboratory and found that the virus has a close relation with Indian isolates (West Bengal) of PPR virus at a cluster with Asian group (**Barrett *et al.*, 1997**). PPR being an exotic disease in Bangladesh, there is a very little knowledge about its epidemic nature, diagnostic techniques and control strategy (**Sil *et al.*, 2000-2001; Debnath, 1995).**

PPR in goat has been recorded in 1993 from the border belt areas of south western districts of Bangladesh and then spreads like epidemics fashion throughout the country. It has been reported that the Black Bengal goats are more susceptible (67.24%) to PPR than Jamunapari breed (32.76%). Morbidity varies from 40-95% and mortality as high as 80-85% (**Samad, 2000**). In Bangladesh, outbreak of Rinderpest like disease, later confirmed by a reference laboratory to be PPR during 1993 in border belts area of South Western districts (Sathkhira, Jessore and Barguna) of Bangladesh by Dr. Taylor PPR was not clearly recognizable up to 1972. But the true extend of the disease has become apparent in recent years and is still being clarified (**Reader and obi, 1999**). It still causes serious economic losses (**Diallo, 2003**) and remains a major constraint on the development of small ruminant farms in these countries.

Epidemics in sheep and goats, the mainstay of subsistence farming in the developing world can cause mortality rates of 50-80 % in naïve populations. Antelope and other small wild ruminant species can also be severely affected (**Abu Elzein *et al.*, 2004**). PPRV is considered as a serious predisposing factor for respiratory disease complex in sheep and particularly in goats, (**Taylor *et al.*, 1990; Baily *et al.*, 2005**). PPR virus, like other morbilliviruses, is lymphotropic and epitheliotropic (**Scott, 1981**).. The incubation period is about four days. The disease is particularly in young animals. The disease is characterized by mainle three symptoms like discharge (nasal, ocular and oral) diarrhea and death; hence it is called 3D disease. The other symptoms are high fever, eroded stomatitis, pneumonia and gastroenteritis (**Radostits *et al.*, 2000**). Erosions on the mucous membrane of the buccal cavity are accompanied by marked salivation. Ulcers develop in the mucosae of the alimentary, respiratory and urinary tracts. Conjuctivitis with ocular discharge is a feature of the disease. A profuse diarhhoea , which results in dehydration , develops within days of infection. Signs of tracheitis and pneumonia are common. There is asevere leucopenia which facilitates secondary bacterial infection.Pulmonary infections caused by Pasteurella species are common in the later stages of the disease.Pregnant animals may abort. Mortality rates in severe outbreaks often exceed 70 % and actually affected goats may die within ten days of exposure to the virus. (**Forsyth and Barret, 1995**).

PPRV is transmitted by direct contact with secretions and excretions of infected animals. It is highly contagious and all discharges can carry virus. Substantial quantities of virus are found in ocular nasal or oral secretions of sick goats and in the faeces late in disease (**Abegunde and Abu, 1977**). PPR disease spread also depends upon season. Rainy season is more susceptible to spread the disease where as dry season is less susceptible. Cattle may be infected without showing any clinical signs on experimental inoculation (**Dardiri *et al.*, 1976; Taylor and Abegunde, 1979**). However, in poor conditions it might be possible that cattle develop lesions following PPRV infection, clinical signs of which would be ascribed to rinderpest. Indeed, in the 1950s, disease and death were recorded in calves experimentally infected with PPRV-infected tissue. (**Mornet *et al.*, 1956**).The carcass of an affected animal is usually emaciated, the

hindquarters soiled with soft/watery feces and the eyeballs sunken. The eyes and nose contain dried-up discharges. Lips may be swollen; erosions and possibly scabs or nodules in late cases. The nasal cavity is **c**ongested (reddened) lining with clear or creamy yellow exudates and erosions. The lung is dark red or purple with areas firm to the touch, hard mass like liver called hepatic liver, mainly in the anterior and cardiac lobes (evidence of pneumonia). Lymph nodes (associated with the lungs and the intestines) are soft and swollen. Abomasums congested with lining hemorrhages. The pathology caused by PPR is dominated by necrotizing and ulcerative lesions in the mouth and the gastro-intestinal tract (**Roeder *et al.*, 1994**). Erosion in the oral cavity is a constant feature. The rumen reticulum and omasum rarely exhibit less lesions in the small intestine are generally moderate, being limited to small streaks of hemorrhages and, occasionally, erosions in the first portions of the duodenum and the terminal ileum. The large intestine is usually more severely affected, with congestion around the ileo-cecal valve, at the ceco-colic junction and in the rectum. In the posterior part of the colon and the rectum, discontinuous streaks of congestion "zebra stripes" form on the crests of the mucosal folds. In the respiratory system, small erosion and petechiae may be visible on the nasal mucosa, larynx and trachea. Presumptive diagnosis made by knowledge of the history of outbreak, seasons of the year, recent purchase of animal from the markets or the return of unsold animal from the market, the nature of the clinical sign , degree of the morbidity and mortality indicates the cases of PPR (**Blood *et al.*, 1995**).

The outbreak of this disease causes a heavy economic loss in Bangladesh. Currently Bangladesh government has taken a national scheme of poverty alleviation through goat rearing. The total program will be null and void if the said disease will not control in Bangladesh. Though PPR is immunologically similar to rinderpest and produce solid immunity so prevention of the disease by vaccination is easier than any other method (**Sil, 2000**). DLS have developed a homologous cell culture attenuated PPR vaccine which is very potent against PPR. But vaccination program have not yet been covered total goat population of the country. In Bangladesh in field level the diagnosis of PPR infected goat is based on primarily owners complain, observing the signs and symptoms and physical examination. Supportive treatment of atropine aided antibiotic +fluid therapy and antihistaminic aided antibiotic + fluid therapy as well as combined therapy can save the life of PPR patient in field condition (**Scott, 2000).**

Considering all aspect described, the present study, therefore, undertaken with the following objectives-

1. To study the occurrence of PPR in terms of breed, age and sex.
2. Study on clinical sign and symptoms of PPR infected goat in field level.
3. Evaluation of supportive treatment in PPR infected goat.

Chapter II

REVIEW OF LITERATURE

Peste des petits ruminants (PPR) is a highly contagious systemic disease of sheep and goats. There are so many literatures about PPR diseases, from which some important research findings are listed below along with references.

2.1. History

The disease was first described in Ivory coast of west Africa ion 1940 (**Gargadennec and Lalanne, 1942**) and then after, it has been recognized in many of the sub-Saharan countries that lie between the Atlantic Ocean and the Red Sea (**Lefevre and Diallo, 1990**). The PPR virus was first isolated by Gilbert and Monnier in 1962 (**Sil, 2000**) in Senegal. PPR is present in nearly all Middle Eastern countries up to Turkey (**Furley *et al.*, 1987; Lefevre *et al.*, 1991; Perl *et al.*, 1994; Taylor *et al.*, 1990; Ozkul *et al.*, 2002)**. It is also widespread in India and southwest Asia (**Shaila *et al.*, 1989**). Presently, PPR occurs in most African countries situated in a wide belt between the Sahara and equator, the Middle East (Arabian Peninsula, Israel, Syria and Jordon) and the Indian subcontinent. Later it spread in Bangladesh at 1993 (**Sil, 1995; Debnath, 1995**). Outbreaks of PPR are now known to be common in India, Nepal, Bangladesh, Pakistan and Afghanistan (**Abdollahpour *et al.*, 2006**).

2.2. Geographical Distribution

The PPR virus was first recorded and isolated by Gilbert and Monnier (1962) in Senegal, although clinical description was reported from Ivory Coast during 1942. PPR virus circulates in belt lying across Africa and Saudi Arabia in 1981 and spread North wards from Saudi Arabia into Jordan, Syria, Iraq, Iran and Pakistan. PPR has also reported from Southern states of India in 1987 and later it spread in to other Indian States. A serious outbreak of PPR in goats has been recorded in 1993 from the border belt areas of Southwestern districts of Bangladesh and then spreads like epidemic throughout and the country. The etiological agent of PPR virus is a member of genus *morbillivirus* under the family of *Paramyxoviridae*. PPR virus is enveloped with helical pleomorphic shape containing single stranded, non-segmented RNA molecules.

2.3. Morphology

The etiological agent, *Peste des petits ruminant's* virus (PPRV) virus has been classified under family Paramyxoviridae, Order Mononegavirales and Genus Morbillivirus (**Tober *et al.*, 1998**). Similar to other morbilliviruses, PPRV is fragile and it cannot survive for long time outside the host. Its half life has been estimated to be 2.2 minutes at 56 0 C and 3.3 hours at 37 0C (**Taylor, 1994**). The virus is enveloped with helical pleomorphic shape containing sense single stranded non segmented RNA molecule. Like other members of the family Paramyxoviridae, PPR virus is an enveloped pleomorphic particle. The genome of PPRV is single stranded RNA, approximately 16kb long with negative polarity (**Haas *et al.*, 1995**). PPR virions, as other morbilliviruses, are enveloped, pleomorphic particles containing single strand RNA as the genome. It is composed of 15, 948 nucleotides,which is encoded with six structural protein, e.g. Necleocapside (N), Matrix (M), Fusion (F), Haemagglutinin (H), Polymerase (P), (**Sil, 2000**). The PPR virus identified in Bangladesh is under the lineage 4 of PPR phylogenetic tree based on the N gene analysis (**Barrett *et al.*, 1997**).

2.4. Agent

Peste des petits ruminants (PPR), also known as goat plague, are caused by a *Paramvxovirus* of the *Morbilliviras* genus **(Dhar, 2002; Barrett, 1994).** It was first described in 1942 in Ivory Coast, West Africa and is closely related to rinderpest virus, canine distemper virus, and human measles virus **(Barrett *et al.*, 1993).**

2.5. Hosts

The natural disease affects mainly goats and sheep, but it is usually more severe in goats where it causes heavy losses and is only occasionally severe in sheep **(Shaila *et al.*, 1989).** Black Bengal goats are more susceptible (67.24 %) to PPR than Jamunapari breed (32.76 %) **(Samad, 2000).** Susceptibility to infection rises with age; however, the disease is rapidly fatal in the young animals (60.87 %) especially at 7-12 months of age **(Blood *et al.*, 1995).** Cattle, buffaloes, camels, and pigs are also susceptible to infection but do not exhibit clinical signs and are unable to transmit the disease to other animals. It is generally admitted that cattle can only be infected subclinically **(Saliki, 1998; Ozkul, 2002; Lefevre and Diallo, 1990).**

2.6. Pathogenecity

The high morbidity (100%) and mortality (50-90%) rates in goats caused by PPR have been described in Bangladesh, followed by evaluation of ELISA as field diagnostic method and inactivated vaccine to control this disease (**Sil *et al*., 2000-2001 ab**). PPR virus is not pathogenic for cattle but rinderpest can also affect goat along with PPR (Gibbs et al., 1979). Case fatality rate is higher in goats (55-85%) compared to sheep (45-75%) (**Opasina *et al*., 1985**). When a susceptible population builds up, periodic epizootics (outbreak) occurs, some of which might lead to almost 100% mortality among affected goat and sheep at risk (**Taylor, 1984; Lefevre and Diallo, 1990**). PPR is a major constrain in the development of goat industry due to high morbidity (50-90%) and case fatality (50- 85%) rates. Kids over 4 months and under 1 year of age are at highest risk and cause huge economic loss (**Venkataramanan *et al.*, 2005**) per year.

2.7. Transmission

PPRV is transmitted by direct contact with secretions and excretions of infected animals. It is highly contagious and all discharges can carry virus. Substantial quantities of virus are found in ocular nasal or oral secretions of sick goats and in the faeces late in disease (**Abegunde and Abu, 1977**). The virus is present in eye, nose, and mouth discharges as well as feces. Faeces are the main spreading agent and through it, disease may occur in epidemic proportion. Newly purchased animal from market and wild animal have been suspected to play a role for spreadind a disease (**Radostits *et al*., 1994, Fraser, 1986**).

2.8. Clinical Features

Incubation period of PPR disease is 2-10 days, most commonly 4-5 **(Barrett, 1994).** After incubation period patients develop sudden high fever (40°C-41°C), remaining high for 5-8 days; will return to normal before recovery or drop below normal before death **(DEFRA, 2001; Sil, 2000).** Serous nasal discharge, becoming mucopurulent; can crust over and occlude nostrils. Purulent ocular discharge with congested conjunctiva and bronchopneumonia **(Lefevre and Diallo, 1990; Rowland *et al.*, 1969).** Necrosis and ulceration of mucous membrane, non haemorrhagic diarrhoea, respiratory distress, including dispread and sneezing, excessive salivation and anorexia, Severe dehydration and emaciation followed by

hypothermia. Death usually occurs after 5-10 days **(Ikede and Uzoukwu, 1983; Bundza *et al.*, 1968; Brown *et al.*, 1991).**

2.9. Pathology

PPR virus, like other morbilliviruses, is lymphotropic and epitheliotropic (**Scott, 1981)**. Pneumonia was not always a consistent feature of PPR in goat (Kulkarni ***et al.*, 1996**). Lungs were found highly consolidated in apical lobe, echymotic and brush paint hemorrhage were found in epicardium (**Rahman *et al.*, 2001**). Zebra stripes are formed by hemorrhages in the fold of rectum (**Sil, 2000**). PPR virus causes epithelial necrosis of the mucosa of the alimentary and respiratory tracts marked by the presence of eosinophilic intracytoplasmic and intranuclear inclusion bodies. Multinucleated giant cells (syncytia) can be observed in all affected epithelia as well as in the lymph nodes (**Brown *et al.*, 1991**). In the spleen, tonsil and lymph nodes, the virus causes necrosis of lymphocytes evidenced by pyknotic nuclei and karyorrhexis (**Rowland *et al.*, 1971**).

2.10. Prognosis

The prognosis of acute PPR is usually poor; especially when lesions do not resolve within 2 to 3 days or when extensive necrosis and bacterial infection hive the animal's breath an unpleasant, fetid odor **(Dhar, 2002).** Lymphocytes percentage declines (up to 21 %) with onset of duration of diseases increases at the same time neutrophilia (up to 72 %) occur **(Sil, 2000).** Young animals (7 to 12 months) often have more severe disease. Morbidity and mortality can be as high as 100% and 90%, respectively. When associated with other diseases such as Capri pox, mortality can be 100 % **(Saliki, 1998).** The recovery percentage in initial stage (prodromal and erosive) was 100% in 1^{st} group and 75% in 2^{nd} group and in later stage (Pneumonia and diarrhea) was 50% in 1st ' group and 25% in 2^{nd} group were almost similar to the result published in BLRI report**, (1999).**

2.11. Outbreak Control

Methods applied for rinderpest eradication may be appropriate for PPR. These include the following quarantine, slaughter, proper disposal of carcasses **(Saliki, 1998)** and contact

fomites, decontamination of facilities and equipment, restrictions on importation of sheep and goats from infected areas **(Nanda *et al.*, 1996).**

2.12. Treatment and vaccination

PPR being an exotic disease in Bangladesh, there is a very little knowledge about its epidemic nature, diagnostic techniques and control strategy (**Sil *et al.*, 2000-2001)**. A control programme using locally produced tissue culture live attenuated rinderpest vaccine (TCV), as well as imported rinderpest vaccine were adapted against PPR in this country, but failed (**Sil *et al.*, 1995**). To overcome the problem, a homologous PPR live vaccine was developed, but the main disadvantage of this vaccine, like other *Morbillivirus* vaccine, is its poor thermal stability (**Diallo, 2002**).

To solve this problem, a thermostable PPR vaccine has been developed experimentally by scientists of BLRI and DLS (**Chowdhury *et al.*, 2004**). Therefore, the present study was undertaken for the first time in Bangladesh, to determine the efficacy of the thermostable PPR vaccine incubated at room temperature for 14 days. Two candidates of homologous vaccine have already been developed in Ethiopia of Africa & another in Bangladesh. Hyper immune serum can be used successfully along with long acting antibiotic (Antibiotic combined hyper immune serum therapy) to limit the spread of virus and recover those animals which are under incubation and in early stage of infection. Good nursing, symptomatic treatment with broad spectrum antibiotic or sulphar drugs can safe life of sick animal and also can improve the immunosuppressive condition of the affected goat, (**Sil, 2000; Scott, 2000**).

Chapter III

Materials and Methods

3.1. Study period

The study was conducted over 8 weeks from 26th June to 25th August, 2010.

3.2. Place of study

Thana veterinary hospital (TVH), Shyamnagar and Kaligong in Satkhira district, Bangladesh.

3.3. History

Information about the diseases and clinical signs exhibited by the animal during illness were recorded in detail provided by the owner.

3.4. Population and tools used for data collection

The study was conducted on natural PPR infected sheep and goats of various age, sex and breed that were brought to the hospital over the study period. A number of total cases of 365 were recorded in internship period in District Veterinary Hospital, Satkhira, from 26th June to 25th August, 2010 of those PPR were detected 135 cases both sheep (26) and goat (109).

In both cases the total samples were divided into several groups such as species, age, and sex.

3.5. Diagnosis of PPR cases

Diagnosis was made by mean of history, clinical signs and physical examinations of the goats and sheep.

3.6. Anamnesis

History of the cases were taken from the owner and carefully recorded in each case individually.

3.7. Clinical examination

The flowing clinical examinations were done carefully and the findings were recorded.

3.7.1. Close inspection

Close inspection were performed properly in order to observe the presenting signs such as a sharp rise of temperature of 104^0F – 106^0F, occulonasal discharge, diarrhea, respiratory distress and off feed.

3.7.2. Temperature recorded

Per rectal temperature were recorded with the thermometer in every cases.

3.7.3. Indirect auscultation

Respiratory distress was identified with the help of stethoscope and observed the lung and tracheal sound and recorded.

3.7.4. Skin fold test:

In diarrheal case, dehydration was measured by skin fold test. Skin fold test was performed to rough estimation of the degree of dehydration.

3.8. Clinical signs

The clinical signs were found during clinical examination of PPR patients are:

- ⇒ Markedly depressed and sleepy appearance.
- ⇒ Rough hair coat and clear watery discharge from anus and thick purulent discharge from the eyes and nose.
- ⇒ Sudden high fever (104^0 – 106^0 F), remaining high for 5-8 days, will return to normal before recovery or drop below normal before death.
- ⇒ Anorexia, severe dehydration and emaciation followed by hypothermia.
- ⇒ The mucous membrane of the mouth and eyes become much reddened and small pinpoint grayish areas appeared on the gum, dental pad, palate, lips and upper surface of the tongue and characteristic foul smell came out from mouth.

⇒ Faces were soft, watery foul smelling and contain blood streaks and pieces of dead gut tissue.

⇒ In severe cases, difficulty in breathing marked by extension of head and neck, dilation of nostrils, protrusion of the tongue and soft painful coughs.

3.9. Disease other than PPR

Major diseases of goats other than PPR were recorded based on gross findings & clinical signs and symptoms.

3.10. Follow up treatment

As being viral disease only supportive therapy was applied to the PPR infected goats. Antibiotics, Sulphar drugs, antihistaminic and fluid therapy were given to the study population. The therapy regime was –

3.10.1. Sulphadimidine (Salidone ® ACI) @ 0.2g per kg body weight. (3 ml per 5 kg body wt.) in the 1st dose & ½ of the initial dose in subsequent days. IM.

3.10.2. Oxytetracycline (Renamycin ® Reneta) @ 10mg per kg body wt. (1 ml per 10 kg body wt.) IM daily.

3.10.3. Promethazine hydrochloride (Dellergen ® Reneta) @10 mg per kg body wt. (2ml per 10 kg body wt.) IM daily.

3.10.4. Oral fluid therapy.

3.11. Analysis of data

A descriptive analysis was carried out to express the result in frequency (F) and percentage (%) and risk ratio, χ^2 – value, p-value, significant interpretation was analyzed.

CHAPTER IV

Results and Discussion

Table No-1: Comparison of occurrence of different goat diseases with PPR in the study periods

Disease	Case	Prevalence (%)
PPR	135	44.55
Enteritis	22	7.26
Pneumonia	15	4.95
Arthritis	9	2.97
Cyst	2	.66
Cataract	8	2.64
Worm Infestation	70	23.1
Anorexia	42	13.86
Total Cases	303	100

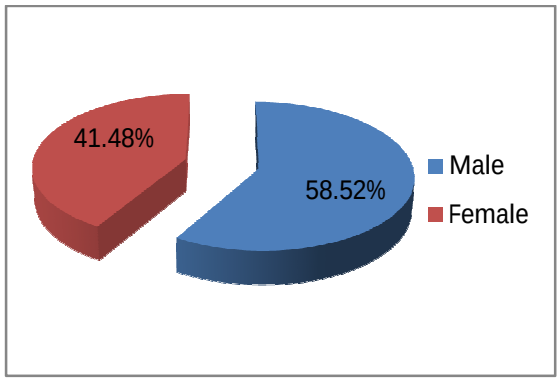
The above table shows that the prevalence of PPR is higher (44.55%) than any other diseases. These results are in agreement with the result of Taylor *and* Baily (**Taylor 1990; Baily, 2005).**

Table No-2: Disease incidence of goat according to sex

Sex	No. of cases	Percentage (%)
Male	79	58.52
Female	56	41.48
Total	135	100

The above table shows that the male goats are much more affected than female. This result is in agreement with the result of samad **(Samad 2001).**

Figure No-1: Disease incidence of goat according to sex



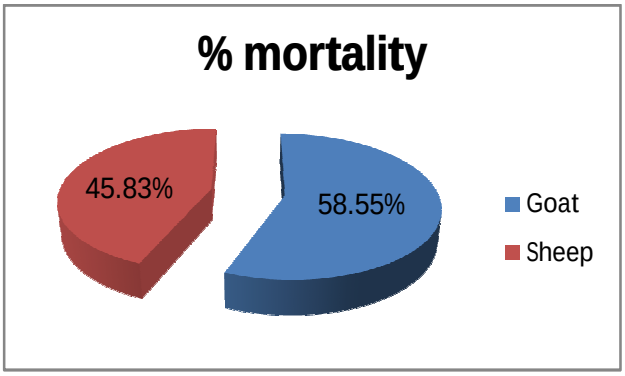
The above figure shows that the incidences of PPR in male and female goat are 58.52% and 41.48% respectively. This result is in agreement with the result of samad **(Samad 2001).**

Table No-3: Incidence of case mortality

Species	Total case	No. of case died	Mortality (%)
Goat	111	65	58.55
Sheep	24	11	45.83

The above table shows that the mortality percentage of goat and sheep are 58.55% and 45.83% respectively. These results are in agreement with the result of Opasina and Abu Elzein **(Opasina, 1985 and Abu Elzein, 2004).**

Figure No-2: Incidence of case mortality



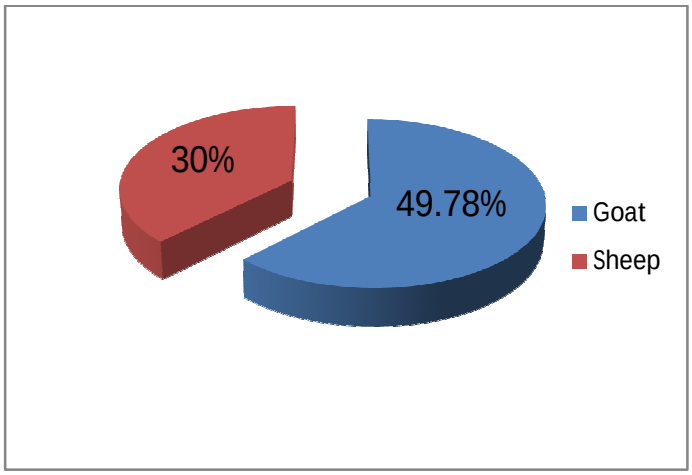
The above figure shows that the case mortality of PPR in goat and sheep are 58.55% and 45.83% respectively.

Table No-4: Total study of PPR cases in sheep and goat

Species	No. of Samples	No. of Affected	No. of Non affected	χ^2 – value	p-value	Interpretation
Goat	223	111 (49.78%)	112	9.32	0.0032	insignificant
Sheep	80	24 (30%)	56			
Total	303	135				

Prevalence of PPR in goat and sheep is presented in Table-4. The goats are highly susceptible than sheep. The total number of PPR affected goat were 111and sheep were 24. These results are in agreement with the result of Shaila **(Shaila, 1989).** The statistical analysis shows that p-value is 0.0032 and the difference was not significant.

Figure No-3: Total study of PPR cases in sheep and goat



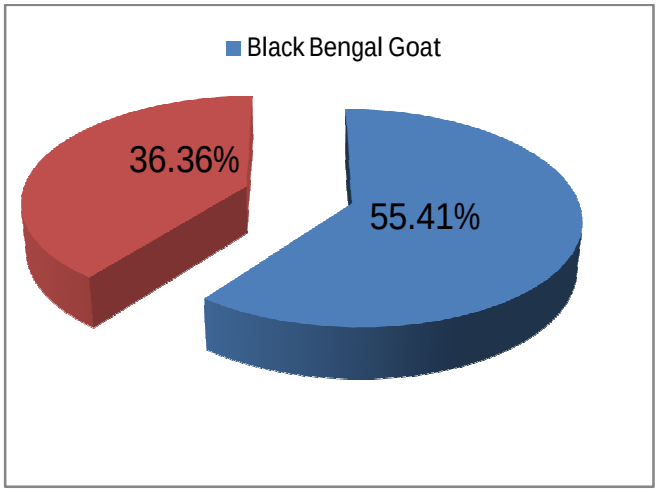
The above figure shows that the incidences of PPR in sheep and goat are 30% and 49.78% respectively. These results are in agreement with the result of Shaila **(Shaila, 1989).**

Table No-5: Analysis of prevalence of PPR diseases in goat in breed variation

Breed	No. of Samples	No. of Affected	No. of Non affected	χ^2 – value	p-value	Interpretation
Black Bengal Goat	157	87 (55.41%)	70	6.75	0.0094	insignificant
Jamunapari Goat	66	24 (36.36%)	42			
Total	223	111				

Analysis of prevalence of PPR disease in Black Bengal goat and Jamunapari goat is presented in Table-5. Black Bengal goats are more prone in PPR 87(55.41%) than Jamunapari goats 24(36.36%). These results are in agreement with the result of samad (**Samad 2000- 2001**). The statistical analysis shows that that p-value is 0.0094 and the difference was not significant.

Figure No-4: Analysis of prevalence of PPR diseases in goat in breed variation



The above figure shows that the prevalence of PPR in Black Bengal and Jamunapari goat are 55.41% and 36.36% respectively.

Table No-6: Analysis of age group

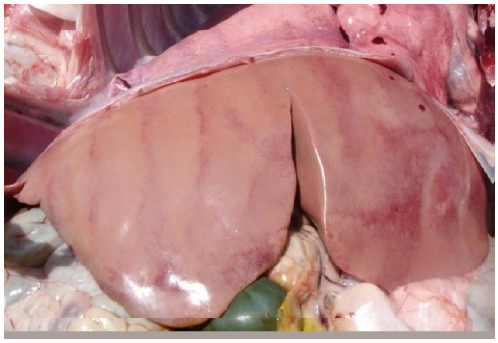
Species	0-6 Months	7-12 Months	>12 Months	Total	χ^2 – value	p-value	Interpretation
Goat	17 (15.32%)	62 (55.86%)	32 (28.83%)	111	1.19	0.55	insignificant
Sheep	02 (8.33%)	16 (66.67%)	6 (25%)	24			

Susceptibility of PPR disease in age group is presented in Table-6. Data shows that, 7-12 months age groups both sheep and goat are more significantly affected in PPR. In goat it was 62(55.86%) and in sheep it was 16(66.67%) both were more than 50 percent. Young kids of below one year are much more susceptible than adult one. These results are in agreement with the result of samad (**Samad 2001**). Then >12 months age group were significant in PPR disease, in goat it was 32 (28.83%) and in sheep it was 6(25%). Finally 0-6 month of age groups were less susceptible in PPR disease, in goat it was 17(15.32%) and in sheep it was 02(8.33%), because kids get immunity from vaccinated does. The statically analysis shows that the p-value is 0.55 and the difference were not significant.

Table No-7: Efficacy of therapeutic response in PPR of goat in different therapeutic methods

SL.No.	Therapy	Total no of case treated	No of cured cases	Percentage of cured cases
1	Atropine aided antibiotic + fluid therapy	45	15	33.33
2	Antihistaminic aided antibiotic + fluid therapy	24	7	29.17
3	Combined therapy	66	37	56.06

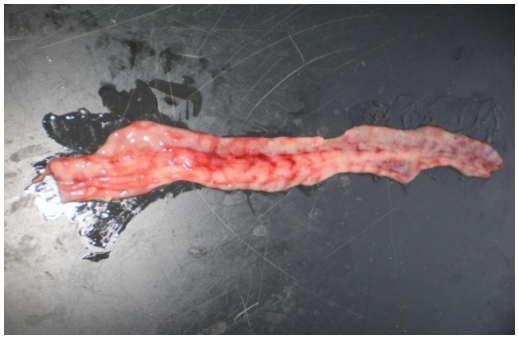
The combined therapy of atropine, antihistaminic aided antibiotic and fluid therapy can save the life of PPR patients in the field condition. The results are in agreement with the author Scott (**Scott 2000**).



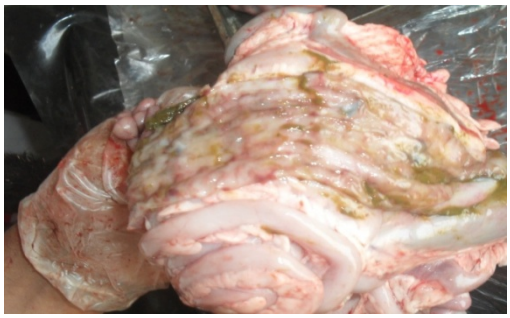
Haemorrhages on Liver

Erosions on the inner side of upper

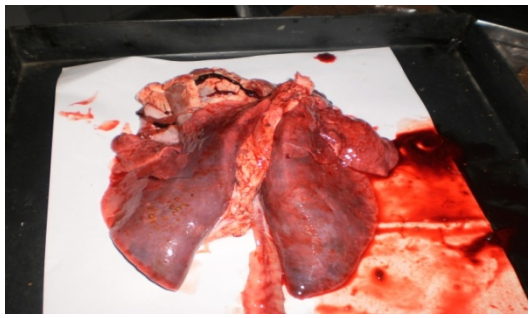
Haemorrhages on Lung



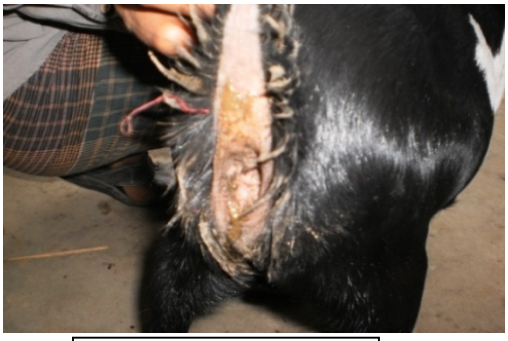
Zebra Stripes in the mucosa of Colon



Haemorrhages on Intestine



Consolidated Pneumonic Lung



Diarrhoea in Goat

Occulonasal discharge in Goat

Chapter v

CONCLUSION

From the study, it is observed that Black Bengal goats were more susceptible to PPR than Jamunapari goats. Young goats about 7-12 months were more prone to PPR than adult. Female are more susceptible than male. Goat is more susceptible than sheep. Vaccination of goats markedly reduces the chances of infection. Immunosuppression occurs due to destruction of lymphocytes and lymphoid tissues. The chance of other bacterial infection raises especially pneumonia and gastroenteratitis with duration of disease increases; it may be due to immunosuppressive nature of PPR disease. PPR causes heavy economic losses in every year especially in rainy seasons and also markedly decreases the production performances of the patient. This study was made to discover the present situation of PPR disease in major goat rearing area of Satkhira district. The district situated on border area, so for control of this disease, first to need restriction of movement in border area and border area must be under ring vaccination program. Regular vaccination against PPR and proper surveillance and monitoring can eradicate this disease from our country.

Chapter VI
REFERENCES

Abdollahpour, G.; Roofi, A.; Najafi, J.; Sasani, F. and Sakhaie, E. (2006). Clinical and para-clinical findings of a recent outbreak of Peste des petits ruminants in Iran. J. Vet. Med. B, 53: 14-16.

Abraham, G.; Sintayehu, A.; Albina, G. E.; Roger, F. (2005). Antibody seroprevavence aginst *Peste des petits ruminants (PPR)* virus in came, cattle, goat and sheep in Ethiopia Prev. Vet. Med., 70: 51-7.

Alam, J.S.M.A.; Rahman, A.; Sayeed, M.A. (1998). A study on Livestock Credit in Rural Bangladesh. Bangladesh Journal of Livestock Research1 (1): 15-18.

Abegunde A.; Taylor W.P. (1979). The isolation of *peste des petits ruminants* virus from Nigerian sheep and goats. Res. vet. Sci., **26**: 94-96.

Abegunde , A.A. and Abu, F.D. (1977). Excretion of the virus of *peste des petits ruminants* by goats. Bull. Anim. Health Prod. Afr. 25(3): 307-311.

Anderson, J.; Mckay L.A.; Butcher, R.N. (1991). IAEA Technical document 623, Proceedings of a final research coordination meeting of the FAO/IAEA/SIDA/OAU coordinated research programme. Bingerville, Ivory Coast. November 19-23, 1990.

Abu elzein E.M.E.; El Naeem A.; Al Afaleq A.I. (2004). Epizootiological aspects of PPR and rinderpest in sheep and goats in Saudi Arabia. Revue sci. tech. Off. int. Epizoot., **19**: 855-858.

Barrett, T. (1996). Geographic distribution and epidemiology of *peste des petits ruminants* virus. *Virus Res.* 43 (2):149-53.

Barrete, E. (1994). Rinderpest and distemper viruses. In: R.G Webster and A. Granoff(eds). Encvci. Virol, 3: 1260-1268.

Barrete, T.; Pronab, D.; Sreenivasa, B.P. and Corteyn, M. (1997). Recent epidemiology of *peste des petits ruminants* virus (PPRV). Vet. Microbiology. 88: 125-130.

Bailey, D. (2005). Full genome sequence of *peste des petits ruminants* virus, a member of the Morbillivirus genus. *Virus Res.* 110: 119-24.

Blood, D.C.; Radostits & Hunderson, J.A. (1995). Veterinary medicine, 6th ed. London: Bailliere Tindall.

Bundza, A.; Afshar, A.; Mayers, D.J.; Dulac, G.C. and Dukes, T.W. (1968). Experimental *peste des petits ruminants* (goat plague) in goats and sheep. Canadian Journal of Veterinary Research. 52: 46-52.

Brown, C.C.; Mariner, J.C. and Olander, H.J. (1991). An immune-histological study of pneumonia caused by *peste des petits ruminants* virus. Veterinary Pathology 28: 166-170.

BLRI. (1999). Research achievement and activities, P. 27-28.

Barrett, T. (1993). The molecular biology of rinderpest and *peste des petits ruminants*. *Ann. Med. Vet.* 137 (2): 77-85.

Cokcaliskan, A.; Gencay and Burgu, I. (2002). Prevalence, distribution and hosts range of *Peste des petits ruminants* virus in Turkey. Emerging Infec. Dis., 8(7): 708-712.

Chakrabarti, A. (2003). *A Textbook of Preventive Veterinary Medicine*. 3rd edn., Kalyani Publishers, New Delhi, India.

Chakrabarti, A. (2004). A Text Book of Preventive Veterinary Medicine. P. 72-76.

Chowdhury, S.M.Z.H.; Shukur, A.; Nasiruddin, M.; Ara, M.R.; Ferdous, K.S.; Sobhan, Z.; Habib, S.; Das, B.K. and Litamoi, J.K. (2004). Molecular characterization of PPR virus: Experiential development of PPR thermostable vaccine. Annual Research Review Workshop-2004, Bangladesh Livestock Research Institute, Savar, Dhaka, 9-10 May 2004.

DLS, (2009). Directorate of Livestock Services, Khamer Bari Road, Dhaka. Personal Communication.

Debnath, N. C. and Sil (1995). *Peste des petits ruminants* (PPR). An overview, proceeding of the BSVER Symposium of Eradication of Rinderpest and Related disease. Second BSVER Annual Scientific Conference held on 2nd December, 1995, Dhaka. P.9-13.

Diap, M.; Sarr J. and Libeeau, J. (2005). Evaluation of novel diagnostic tools for *Peste des petits ruminants* virus in naturally infected goat herds. Epidemiol. Infect 133: 711-717.

Dardiri, A. H. and Hamdy, F. M. (1976). Response of American goats and cattle to PPR virus. Proceedings of the 19th. Annual Meeting, the American Association of Veterinary Laboratory Diagnosticians, November 7-9 1976. P. 337.

Dimri, V.; Mishra, S.; Mishra, N.; Shinde, A. K.; Bhagwan, P.S.K. and Sharma, M. C. (2002). An outbreak of *peste des petits ruminants* in goats in an organized farm of semi-Arid India. *Intas Polivet* 3 (1): 96-100.

Diallo A. (2002). Control of PPR. Classical and new generation vaccines. Vaccines for OIE List-A and emerging animal diseases. Proceeding of a symposium. Ames, Iowa, USA. September 16-18, 2002.

Dhar, P.; Singh, R.P.; Sreenivasa, B.P.; Shah, L.C. and Bandyopadhyay, S.K. (2002). Development of a monoclonal antibody based competitive ELISA for detection and titration of antibodies *to peste des petits ruminants* (PPR) virus. *Vet. Microbiol.*, 98: 3-15.
Diallo, A. (2003). *Peste des petits ruminants*. Featured ar ticle published in the website: http:// www.indiveterinarycommunity.com (Accessed 12th August, 2004).

Diallo, A. (2006). Control of *Peste des petits ruminants* and poverty alleviation. J. Vet. Med., 53: 11-13**.**

DEFRA (Department for Environment Food and Rural Affairs). Animal health and welfare. *Peste des petits ruminants*; May 24, 2005.

El hag ali b.; Taylor, W.P. (1984). Isolation of PPR virus from the Sudan. Res. vet. Sci., **36**: 1-4.

Fraser, C.M. (1986). *Peste des petits ruminants*(PPR). The Merck Veterinary Manual. 6th edition, Merck and co.Inc.U.S.A. 402-403.

Furley, C.W.; Taylor, W.P. (1987). An outbreak of *peste des petits ruminants* in a zoological collection. Vet. Rec., **121**: 443-447.

Forsyth, M.; Amjad, H.; Barrett, T. and Rossiter P. B. (1995). *Peste des petits ruminants* in goat in Pakistan. Vet. Rec. 139: 118-119.

Gargadennec, L. and Lalanne, A. (1992). Bull des services zoo techniques et des epizootics de 1, afrique occidentale franaise, 5:16.

Gargadennec, l.; Lallane A., (1942). La *peste des petits ruminants*. Bull. Serv. Zool. (AOF), **5:** 15-21.

Hass, S.S.; Horst, P. and Islam, A.B.M.M. (1995). Effects of different factors on pre-weaning survivality of Black Bengal Kids. *Small Ruminant Research* 18: 1-5.

Islam, M.R.; Shamsuddin, M.; Das, P.M. and Dewan, M.L. (2001). An outbreak of *Peste des petits ruminants* in Black Bengal goats in Mymensingh, Bangladesh. The Bangladesh Veterinarian. 18(1): 14-19.

Ikede, B.O. (1983). Hisopathology of natural cases of PPR, characteristic lesions and changes occurring during the disease *peste des petits ruminants* (PPR) in sheep and goats. Proceedings of the International workshop held at IITA Ibadan, Nigeria. 24-26 September. D.H. Hill (eds). ILCA, Addis Ababa. Ethiolpia.

Kulkarni, D.D.; Bhikane, A.U.; Shaila, M.S.; Varalakshmi, P.; Apte, M.P. and Narladkar, B.W. (1996). *Peste des petits ruminants* in goats in India. *Vet. Rec.* 138 (8): 187-188.

Lefevre, P.C. and Diallo, A. (1990). *Peste des petits ruminants* virus. *Rev. Sci. Tech. Off. Int. Epiz.* 9: 951-965.

Murphy, F.A.; Gibbs, E. P. J.; horzinek, M. C. and M. J. Studdert, (1999). Veterinary Virology. 3rd edition, Academic Press, San Diego, California, USA.

Mornet, P. Gilbert, Y. (1956). La *peste des petits ruminants* en afrique occidentale francaise, sesrapports avec la peste bovine. Revue Elev. Med. Vet. Trop. 9: 313-342.

Narladkar, B.W. (1996). *Peste des petits ruminants* in goats in India. Veterinary Record. 138:187-188.

Nanda, Y. P.; Chatterjee, A.; Sharma, R. N. and Taylor, W. P. (1996). The isolation of *peste des petits ruminants* virus from Northern India. Veterinary Microbiology, 51: 207-212.

Opsina, B.A. (1985). Tropical Animal Health Production. 17:217.

Ozkul, A.; Akea, Y.; Alkan, F. (2002). Prevalence, distribution, and host range of *peste des petits ruminants* virus, Turkey. Emerge Infect Dis. Jul; 8(7): 708-712.

Perl, S.; Alexander, A.; Yakobson, B.; Nyska, A.; Harmelin, A.; Shekhat, N.; Shimshony, A.; Davidson, N.; Abramson, M. and Rapoport, E. (1994). *Peste des petits ruminants* (PPR) of sheep in Israel. Isr. J. vet. Med., **49**: 59-62.

Roeder, P. L.; Abraham, G.; Kenfe, G. and Barrett, T. (1994). *Peste Des petits ruminants* in Ethiopian goats. Trop. Anim. Health prod., 26: 69-73.

Rahman, M.B.; Razzaque, M.A.; Kafi, M.A.; Islam, M.R. and Amin, K.M.R. (2005). Antibody responses in ruminant's species following vaccination with tissue culture adapted PPR

vaccine. Proceedings of 21st Annual Conference of Bangladesh Society of Microbiologist. 1-2 July 2005, BINA, Mymensingh, Bangladesh.

Rwland, A. C.; Scott, G. R. and Hill, D. H. (1969). The pathology of an erosive stomatitis and enteritis in West African dwarf goats, Journal of pathology, 98 : 83-87.

Radostits, O. M. (1994). Vet Medicine 8th edition.

Radostits, O.M.; Gay, C.C. and Blood, D.C. (2000). Veterinary medicine. 9th edn. W. B. Saunders Company Ltd. London.

Reader, P.L. and Obi, T.U. 1999. Recognizing *Peste des Petitis Ruminants*: A Field Manual, Food and Agricultural Organization. Rome. Animal Health Manual pp: 5-28.

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.

Samad, M. A. (2001). Poshu palan o chikitsa vidya LEP publication BAU Mymensingh, P-693.

Samad, M. A. (2000). An overview of livestock research reports published during the twentieth century in Bangladesh. Veterinary Journal 34: 53-194.

Saliki, J.T.; House, J.A. and Dubovi, E.J. (1998). Comparison of monoclonal antibody based sandwich enzyme-linked immunosorbent assay and virus isolation for detection of *Peste des petits ruminants* virus in goat tissue and secretions. J. Clin. Microbiol., 32: 1349-1353.

S**il B.K.; Rahman M.M.; Taimur M.J.F.A. and Sarker A.J. (1995).** Observation of outbreaks of PPR in organized goat farms and its control strategy. Presented at the Annual Conference of the Bangladesh Society for Veterinary Education and Research. December 3, 2005, BARC, Dhaka.

Scott, G. R. (2000). *Peste des petits ruminants* and rinderpest. In: Martin W. B., and I. Aitken (ed), Diseases of Sheep, 3rd edn, p. 398. Blackwell Publishing, Oxford, UK.

Singh, R.P.; Sreenivasa, B.P.; Dhar, P.; Shah, L.C. and Bandyopadhyay, S.K. (2004). Development of a monoclonal antibody based competitive ELISA for detection and titration of antibodies to *peste des petits ruminants* (PPR) virus. *Vet. Microbiol.,* 98: 3-15.

Sil, B.K. and Taimur, M.J.F.A. (2001). Final report of the: Contract research project on production of *peste des petits ruminants* (PPR) vaccine and its field application. BARC funded project, BLRI, Savar, Dhaka, June 2001.

Sil, B.K.; Taimur, M.J.F.A.; Hossain, K.M.; Giasuddin, M.; Haque, M.E.; Chowdhury, E.R.; Alam, M.R. and Sarker, A.J. (2000- 2001). Preliminary study towards the development of inactivated PPR vaccine. *Bangladesh Journal of Livestock Research* 7- 8: 1-6.

Taylor, W.P.; Abegunde, A. (1979). 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 PPR. Prev. vet. Med., **2**: 157-166.

Taylor, W.P.; Albushaidy, S. and Barrett, T. (1990). The epidemiology of *peste des petits ruminants* in the Sultanate of Oman. Vet. Microbiol., **22**: 341-352.

Tober, and Taylor, W.P. (1998). The epidemiology of *peste des petits ruminants* in the Sultanate of Oman. *Vet. Microbiol.* 22 (4): 341-352.

Uzoukwu, M. (1983). The pathology of *peste des petits ruminants* (PPR caprine pneumoenterits) in sheep and goats. Proceedings of the International Workshop held at IITA. Ibadan, Nigeria. 24-26 September 1980 D, H. Hill (Eds) .ILCA. Addis Ababa. Ethiopia.

Venkataramanan, R.; Bandyopadhyay, S. K. and Oberoi, M. S. (2005). Present status and strategies for the control of transboundary and other economically important animal diseases in India: A review. *Indian J. Animal Sci.* 75(4): 456-464.