COMPARATIVE STUDY OF SEROPREVALENCE OF PESTE DES PETITS RUMINANTS (PPR) IN SHEEP AND GOAT AT DINAJPUR DISTRICT OF BANGLADESH

A THESIS

BY

FARHAN ALI ABDI

REGISTRATION NO.: 1805179

SEMESTER: JANUARY-JUNE 2019

SESSION: 2018-2019

MASTER OF SCIENCE (M.S.)

IN

PATHOLOGY

DEPARTMENT OF PATHOLOGY AND PARASITOLOGY

HAJEE MOHAMMAD DANESH SCIENCE AND TECHNOLOGY UNIVERSITY, DINAJPUR-5200

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JUNE, 2019

DEDICATED TO MY

BELOVED PARENTS AND BROTHERS

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June, 2019 *The Author*

ABSTRACT

The study was conducted to compare seroprevalence of PPR in sheep and goat at Dinajpur during January to June, 2019. A total number of 77 blood sample (51 from goat and 26 from sheep) were collected by jugular venipuncture method considering different parameters such as age, sex of animal and then kept for an hour at room temperature, after centrifugation serum was separated and stored at -20^{0}C until use. The result showed that the seroprevalence was higher in goat (41.18%) than sheep (26.92%). Based on sex, there was no significant variation ($P>0.05$) in goat and sheep and it was found that seroprevalence of PPR was higher in female (45.71%) than male (31.25%) goat and male sheep (28.57%) had slightly higher prevalence of PPR than female (26.32%) sheep. Among breeds of goat and sheep, Black Bengal breed (45.16%) of goat and cross breed of sheep (36.35%) had the highest seroprevalence. According to the age group, it was observed that age had significant ($P<0.05$) effect on the prevalence of PPR in goat but had insignificant ($P>0.05$) effect on the prevalence of PPR in sheep. Seroprevalence was decreased in advanced of age in goat and sheep. According to the present study, it was revealed that prevalence of PPR was higher in poor hygienic condition of farm. The present study should be concluded that the seroprevalence was comparatively higher in goat than sheep.

CONTENTS

CHAPTER	TITLE	PAGE NO.
	ACKNOWLEDGEMENTS	**i**
	ABSTRACT	**ii**
	CONTENTS	**iii-v**
	LIST OF TABLES	**vi**
	LIST OF FIGURES	**vii**
CHAPTER 1	**INTRODUCTION**	**1-3**
CHAPTER 2	**LITREATURE REVIEW**	**4-24**
2.1	Peste Des Petits Ruminants	4
2.2	Etiology	5
2.2.1	Biology of the Virus	6
2.2.2	Structural proteins of the virus	6
2.2.3	Physiochemical properties of the virus	6
2.3	Pathogenesis and Clinical Signs	7
2.4	Epidemiological Situations	8
2.4.1	Geographical distribution of the disease	8
2.4.2	Molecular epidemiology of PPRV	8
2.4.4	Sero-prevalence of PPR virus	9
2.4.5	Transmission of the virus	12
2.4.6	Host range	13
2.4.7	Host determinants of the disease	13
2.4.8	Social ecology and seasonality of the PPR disease	14
2.4.9	Potential risk factors of PPR	15
2.4.10	Wild life Susceptibility to the disease	15
2.4.11	Pattern of the disease	16
2.5	Current Diagnostic Techniques	16
2.5.1	Serological detection	17
2.5.2	Antigen detection	17
2.5.3	Genome detection	18
2.6	Opportunities Presented Regarding PPR Eradication	18

CONTENTS (Contd.)

CHAPTER	TITLE	PAGE NO.
2.7	Socio-Economic Impact of PPR	19
2.8	Differential Diagnosis	21
2.9	Prevention and Control	21
2.9.1	Vaccination	21
2.9.2	Surveillance	23
2.9.3	Biosecurity	23
2.9.4	Stamping out	23
2.9.5	Public awareness	24
2.9.6	Treatment	24
CHAPTER 3	**METHODOLOGY**	**25-33**
3.1	Study area	25
3.2	Study population	26
3.3	Experimental layouts	26
3.4	Materials and Methods	28
3.4.1	Materials	28
3.4.1.1	Appliances used for blood collection	28
3.4.1.2	Material required used for ELISA test	28
3.4.1.3	Chemicals and reagents used for indirect ELISA	28
3.5	Antibody detection by indirect ELISA	29
3.5.1	ELISA kit	29
3.5.2	Reagent preparation	29
3.5.3	Sample preparation	29
3.5.4	ELISA plate layouts	29
3.5.5	ELISA testing procedure	30
3.5.6	Calculation of the result	31
3.6	Methods	31
3.6.1	Field survey	31
3.6.2	Blood collection and serum separation	31
3.7	Data analysis	32

CONTENTS (Contd.)

CHAPTER		TITLE	PAGE NO.
CHAPTER 4		**RESULTS**	**34-39**
	4.1	Seroprevalence of PPR in goat and sheep	34
	4.2	Sex related seroprevalence of PPR in goat and sheep	34
	4.3	Breed related seroprevalence of PPR in goat and sheep	35
	4.4	Age related seroprevalence of PPR in goat and sheep	35
	4.5	Seroprevalence of PPR based on hygienic condition	36
	4.6	Comparative study of seroprevalence of PPR in goat and sheep	37
CHAPTER 5		**DISCUSSIONS**	**40-41**
CHAPTER 6		**SUMMARY AND CONCLUSION**	42
		REFERENCES	**43-58**

LIST OF TABLES

TABLE NO.	TITLE	PAGE NO.
1	Seroprevalence of PPR in goat and sheep	34
2	Sex related seroprevalence of PPR in goat and sheep	35
3	Seroprevalence of PPRV based on breed in goat and sheep	35
4	Age related seroprevalence of PPR in goat and sheep	36
5	Seroprevalence of PPR based on hygienic condition in goat and sheep	36
6	Comparative study of seroprevalence of PPR in goat and sheep	37

LIST OF FIGURES

FIGURE NO.	TITLE	PAGE NO.
1	Study area at different pigeon farms in Dinajpur Sadar Upazila	25
2	Schematic representation for experimental lay out	27
3	Mucopurulent ocular and nasal discharge	32
4	Observed diarrhea	32
5	Blood collection from goat	33
6	Blood collection from sheep	33
7	After addition of Serum Sample	38
8	After addition of conjugate solution	38
9	After addition of stopping solution	39
10	After Positive and Negative Control	39

CHAPTER 1

INTRODUCTION

Agriculture and Livestock are very important for both the subsistence and economic development of Bangladesh. They provide a chain of essential food products throughout the year. In some countries, they are a major source of government revenue and export earnings. They also 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. In Nigeria, as in other African countries, small ruminants (sheep and goats) contribute a substantial proportion of the nation and meat supply (Brumby, 1990).

Livestock diseases are encountered to put some obstacles and constraints in the front of developing productivity of small ruminants, specially Peste Des Petits Ruminants (PPR) is considered the most important single cause of morbidity and mortality for sheep and goats, in Bangladesh (Md. Abu Yousuf *et all* 2017).

Sheep and goats contribute a major proportion of the nation and meat supply in Bangladesh and they are also a source of government revenue and export earnings. Peste des petits ruminant's virus (PPRV) results in an acute, highly contagious disease of small ruminants particularly in sheep and goat. It was first described in Cote d'Ivoire in West Africa in 1942. Gradually, it was realized that several clinically similar diseases was found in other parts of West Africa shared the same cause. The virus now called Peste des petits ruminant's virus (PPRV). Investigators soon confirmed the existence of the disease in Nigeria, Senegal and Ghana. For many years, it was thought that it was restricted to that part of the African continent until a disease of goats in Sudan, which was originally diagnosed as rinderpest in 1972, was confirmed to be PPR. The disease is endemic in Bangladesh since 1993 (Islam *et al.,* 1996). Generally 100% morbidity and 80- 90% mortality were recorded in goat (Hamdy *et al.*, 1976). After the first report of PPR from Ivory Coast, the disease has been reported in many countries like Middle East, the Arabian Peninsula, and most parts of Africa (Abu-Elzein *et al.*, 1990; Shaila *et al.*, 1996; Balamurugan *et al.*, 2014). Frequent outbreaks of PPR have also been recorded in south Asian countries like Pakistan, Bhutan, Nepal, Afghanistan, India and Bangladesh (Banik *et al.*, 2008). The PPRV was first identified by Sil *et al.* (1995) in Bangladesh during a severe outbreak in 1993. The PPR outbreaks caused 74.13% morbidity and

54.83% mortality in Black Bengal goats in this country (Islam *et al.*, 2001; Das *et al.,* 2007).

The etiology of PPRV is a member of the genus morbillivirus under the family of the paramyxoviridae. Other members of the genus are Rinderpest, measles, canine distemper, seal distemper and dolphin distemper viruses. The genome of this virus is a single linear molecule of approximately 4.5x106 DA with 1600 ribonucleotides which is encoded with six structural proteins; the Nucleoprotein (N), Matrix (M), Fusion (F), Haemagglutinin (H), Phospoprotein (P) and large protein (L). Replication occurs in cytoplasm and syncytium formation is a characteristic feature of cytopathology of PPR virus. It has low resistance in the environment, Chemical and PH (Paul *et al.*, 1979).

This virus is mostly spread by aerosol and probably only small amount of virus is required to infect susceptible individuals. Cattle may be infected by contact with the PPR virus, but will not exhibit any symptoms and respond to rinderpest vaccination (Anderson *et al.*, 1991). The clinical signs of PPRV infection are always associated with high fever (106°-107.7°F), discharges (nasal, ocular and oral), erosive stomatitis and excessive salivation. The oculo-nasal discharges become mucopurulent followed by pneumonia accompanied with coughing, pleural rales and abdominal breathing. in the later stage of infection A watery blood stained diarrhea is common, which is followed by death. During the cycle disease, immune-deficiency is common and result to contribute susceptibility to secondary infections that accounts for most of the mortality associated with PPR infection (Olaleye *et al.*, 1989 a, b).

PPR was clinically suspected for the first time in Ethiopia in 1977 in a goat herd from Afar region, eastern part of the country. Clinical and serological evidence of its presence has been reported by (Taylor 1984) and later confirmed in 1991 with cDNA probe in lymph nodes and spleen specimens collected from an outbreak in a holding near Addis Ababa. (Abraham *et al.* 2005) reported the overall seroprevalence of 9% in goats and 13% in sheep in different parts of Ethiopia, also reported that 14.6% of sheep sampled along 4 roads from Debre Berhan to Addis Ababa were seropositive. In 1999 national serosurveillance of PPR conducted in Ethiopia, the overall seroprevalence of 6.4% (95% CI: 6.0–6.8) in both goats and sheep ranging from 0% to 52.5% was estimated.

In Somalia, there were unconfirmed PPR outbreaks but sero-surveys conducted between 2006 and 2009 (SAHSP 2006a, b, 2009) revealed sero-prevalence of 6.5% in north-

western Somalia, 28.7% in north-eastern Somalia, 32.6% in central Somalia and 37.6% in southern Somalia.

Although these results are inconclusive on the frequency and distribution of PPR in Somalia, they are a good indicator of PPR virus circulation and hence PPR infection as reported by Waret Szkuta *et al.* (2008) in other studies.

Previous study demonstrated that small ruminants including goats can develop positive level of antibody titer against PPRV under natural situation. The seroprevalence of PPRV specific antibodies has been recorded from Bangladesh to be 49.17% in goats, 19.05% in cattle, and 36.0% in sheep (Razzaque *et al.*, 2004).

This study was carried out under the following objectives:-

i. To determine the seroprevalence of PPR virus in goat and sheep.
ii. To correlate the seroprevalence of affected goat and sheep in related factors such as, age, sex, breed, and lactation stage.
iii. To evaluate the management practice and hygienic status of household farmers

CHAPTER 2

LITREATURE REVIEW

2.1. Peste Des Petits Ruminants

Peste des petits ruminants (PPR), also known as Kata, pseudo rinderpest, pneumo enteritis complex or stomatitis-pneumo enteritis syndrome is a severe and highly infectious viral disease of small ruminants caused by PPR virus, a Mobilivirus of the Family Paramyxoviridae. The disease is characterized by fever, erosive stomatitis, conjunctivitis, gastroenteritis, and pneumonia. The clinical disease resembles Rinderpest in cattle, which is acute, and after an incubation period of 3-6 days, the clinical symptoms become apparent, and include high rise of temperature, oral, occular and nasal discharges, necrotic stomatitis, severe pneumonia, dyspnoea, coughing, enteritis, severe diarrhoea followed by death (Ezeokoli *et al.,* 1986; Pawaiya *et al.,* 2004).

The natural disease affects mainly goats and sheep, but is usually more severe in goats where it causes severe morbidity and mortality (Raghavendra *et al.,* 2000). Infection rates in sheep and goats increase with age, and the disease, which varies in severity, is rapidly fatal in young animals (Wosu, 1994). Generally, cattle are considered to be sub-clinically infected with the disease. However, in poor conditions it might be possible that cattle develop lesions following PPRV infection, clinical signs of which would be ascribed to rinderpest, because of the similarity of the two diseases clinically. Moreover, PPRV was isolated from an outbreak of rinderpest-like disease in buffaloes in India in 1995 (Govindarajan *et al.,* 1997). It was also suspected to be involved in the epizootic disease that affected one-humped camels in Ethiopia in 1995–1996 (Roger *et al.,* 2001). Indeed, PPRV antigen and PPRV nucleic acid were detected in some pathological samples collected during that outbreak (Roger *et al.,* 2001), but no live virus was isolated. Cases of clinical disease have been reported in wildlife resulting in deaths of gazelles in captivity (Elzein *et al.,* 2004).

Before the recognition of PPR as a disease entity in small ruminants, there were historical records of outbreaks of Rinderpest like diseases in sheep and goats that did not cause disease in cattle. Furthermore, diagnostic tests that are capable of differentiating between the two viruses only became available in the past 20 years. Therefore, it was likely that, in the past, many cases of PPR were ascribed to RPV (Baron, 2011). The idea

that PPR has been in existence as a distinct disease for a long time is supported by the phylogenetic tree of the morbilliviruses, which shows that both RPV and PPRV were a similar distance from their most a recent common ancestor. Assuming both viruses mutate according to the same evolutionary "clock", PPRV must have been in circulation for as long as RPV.

In all regions where PPR is endemic, it constitutes a serious threat to small ruminant production. The disease is said to be the fastest growing disease of small ruminants in developing countries (Baron, 2011). Therefore, it influences the livelihood of poor farmers, the main owners of sheep and goats. Hence its control is a major priority for programs aimed at poverty alleviation.

2.2. Etiology

The etiological agent, Peste des petitsruminants virus (PPRV) 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 0C and 3.3 hours at 37 ^{0}C (Rossiter and Taylor, 1994).

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, 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: the phosphoprotein (P) and the large protein (L) (Diallo, 2007).

The phosphoprotein is the cofactor of L, the viral RNA dependent 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 haemmagglutinin (H). By this position, M plays an important role in ensuring efficient incorporation of nucleocapsids into virions during the virus budding process. The haemagglutination 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. It has been used in the development of diagnostic tests (Diallo, 2007).

2.2.1. Biology of the Virus

Structure of PPR virus Peste des petits ruminant's virus is an enveloped, pleomorphic particle containing single stranded RNA, approximately 16 kb long with a negative polarity as a genome (Barrett *et al.,* 2005). The genome of PPR virus is so far the longest of all the mobiliviruses, consisting of about 15,948 nucleotides. Intact virion has a diameter of about 130-390nm with the thickness of the ribonucleoprotein measuring approximately 14-23nm (Durojaiye *et al.,* 1985). It is wrapped by a nuclear protein which is associated with two other proteins: the phosphoprotien (P) and the viral RNA dependant RNA polymerase (L). On the viral envelope are found two other viral proteins, Haemagglutin (H) and Fusion (F) proteins, which are very important for the induction of protective host immune response against the virus (Chauhan *et al.,* 2009).

2.2.2. Structural proteins of the virus

The virus encodes six structural proteins; nucleoprotein (N), phosphoprotein (P), matrix Protein (M), fusion protein (F), hemagglutinin protein (H), large polymerase protein (L), and non-structural proteins V and C (Bailey *et al.,* 2007). Among the structural proteins, N protein is antigenically the most conservative among the morbilliviruses and is highly immunogenic in spite of its internal location (Libeau *et al.,* 1995). The large (L) protein is the enzymatic component of the viral transcriptase and replicase. The L proteins are Multifunctional and, in addition to their polymerase activity, have methylation, capping and polyadenylation activities (Lamb and Kolakofsky, 2001). The Matrix (M) proteins are basic membrane associated molecules that interact with surface glycoproteins in the lipid envelope as well as the virion ribonucleoprotein. The F and H proteins are associated with the viral envelope where they are believed to play important roles in induction of protective immunity (Chauhan *et al.,* 2009).

2.2.3. Physiochemical properties of the virus

The molecular weight of the genome is 5.8 x 106 while the diameter of the virion measures about 150-300nm. The virion is very sensitive to heat, lipid solvents, non-

ionic detergents, formaldehyde and oxidising agents (Kingsbury, 1990). The virus is usually destroyed at 50°C for 60 minutes or 37°C for 2 hours. However, it survives for long periods in chilled and frozen tissues (OIE, 2009).

2.3. Pathogenesis and Clinical Signs

There is variation in the inherent resistance of different breeds of sheep and goats to PPRV (Couacy-Hymann *et al.,* 2007). There is anecdotal evidence that younger animals show higher mortality rates, but this has not been confirmed by experiments. Sheep and goats infected with PPRV show a similar, if slightly less severe, clinical picture to that seen in cattle infected with RPV. There is a rare per-acute form of the disease causing death four to six days after the onset of fever. The more frequent acute form is characterized by a sudden rise in body temperature, peaking at 2–2.5 °C above normal. The mucous membranes of the eyes and nose become congested and there is noticeable discharge from the eyes and nose (Lefevre and Diallo, 1990).

There is a marked and rapid loss of circulating white blood cells (leucopoenia) at this time, starting from two to three days post infection (dpi). The white blood cell count will remain low (about 20% of normal) and will return to normal only during the convalescent phase. As the disease progresses, congestion can be seen in the gums, and necrotic lesions appear in the epithelial tissue lining the mouth, first in the gums and the inside of the lower lip, and in severe cases can be seen on the top and sides of the tongue and in other parts of the buccal mucosa. The necrotic areas throughout the mouth and gums readily erode. As the disease progresses further, diarrhea develops, which is occasionally bloody (Lefevre and Diallo, 1990).

Many animals with PPR show abnormally rapid or labored breathing, and a productive cough. By this stage, the animal is apathetic, with labored breathing and an unwillingness to move. Convalescence, if it occurs, takes several weeks. Any animals that are pregnant at the time of infection will abort. The white blood cell count slowly returns to normal and the oral lesions heal over a period of two to three weeks. This transient loss of white cells, and the generalized immunosuppression that can go on for even longer, means that the animal is susceptible to activation of latent or chronic infections (e.g. with parasites) or to secondary infection by other pathogens. The virus infection, on the other hand, completely resolves in recovered animals, and there is no persistent infection or carrier state (Lefevre and Diallo, 1990).

In post-mortem examination, PPRV infection reveals significant lung pathology, with patches of congestion in the lung tissue and signs of pneumonia. Animals show extensive damage to mucous membranes of the digestive tract and to lymphoid organs. Immuno histological examination shows that the virus is primarily lymphotropic, with epithelial tissue involvement in only later stages of infection (Pope *et al.,* 2013). Further details on the pathology of PPR disease can be found in Wohlsein and Saliki (2006).

The morbidity and mortality rate varies enormously (up to 100 %) depending on the species infected, the age of the animals, the prevalence of secondary infectious agents and the PPRV lineage involved (Zahur *et al.,* 2009; Kivaria *et al.,* 2013; OIE, 2013; Chowdhury *et al.,* 2014).

2.4. Epidemiological Situations

2.4.1. Geographical distribution of the disease

Since it was first identified in the early 1940s in Côte d"Ivoire, the disease has spread throughout Africa, South Asia and China. In the last 15 years, it has expanded into previously non-infected regions. As a result, PPR is now endemic in large parts of the Middle East, Central Asia, South Asia and East Asia and is expected to spread into Southern Africa and Southeast Asia. Populations of the northern Mediterranean region are also at high risk. If left uncontrolled, and with the increasing global flow of livestock products to meet consumer demands, PPR will likely make inroads in Mongolia as well as to other countries in the Caucasus and Europe that have historically been free of the disease (OIE and FAO, 2015).

There are many gaps in current understanding about the epidemiology of PPR. There are many reports with different scenarios of animal species involved in the outbreaks: goats alone, sheep alone, or sheep and goats together. While large ruminants are believed to be relatively resistant, there have been reports indicating the involvement of PPRV in respiratory disease in camels (Roger *et al.,* 2000) in Africa or rinderpest-like disease in buffaloes in India (Govindarajan *et al.,* 1997).

2.4.2. Molecular epidemiology of PPRV

Peste des petits ruminants' virus has only one serotype with four distinct lineages (1, 2, 3 and 4) on the basis of partial sequence analysis of fusion protein (F) and (N) genes. These gene sequence analyses of PPR viral isolates have demonstrated the involvement

of each of the four PPRV lineages with specific geographical niches. The gene sequence analysis of nucleoprotein (N) has been found to be more precise map marker because of its conserved nature therefore allowing a more precise geographical distribution of different lineages concordant with the historic areas of trade or transhumance of small ruminants in some affected areas (Kwiatek *et al.,* 2007).

Lineage 1 and 2 are found exclusively in West Africa countries, Lineage 3 is found in Eastern Africa and Middle East while Lineage 4 is found in South Asian countries, Middle East and China (Dhar *et al.,* 2002). Sudan has lineage 3 and 4 circulating in the country (Saeed *et al.,* 2010) while recently lineage 4 has been found circulating in Morocco and other North African countries (De Nardi *et al.,* 2011). Lineage 1, 2 and 4 were confirmed circulating in Uganda while Lineage 3 is responsible for PPR outbreaks in Tanzania (Luka *et al.,* 2012; Kivaria *et al.,* 2013). However, the lineage of the PPRV circulating in Kenya has not been established (Banyard *et al.,* 2010).

2.4.4 Sero-prevalence of PPR virus

PPR was first recorded by Gargadennac and Lalanne (1942) in Cote d'Ivoire, West Africa, and has since widely spread in other parts of Africa and Asia (Taylor *et al.* 1990; Banyard *et al.* 2010; Munir *et al.* 2013). In the Greater Horn of Africa region, PPR outbreaks were reported in Sudan, Ethiopia, Somalia and Kenya (Diallo 1988; Roeder *et al.* 1994; Karimuribo *et al.* 2008 and Kihu *et al.* 2012). In Somalia, there were unconfirmed PPR outbreaks but sero-surveys conducted between 2006 and 2009 (SAHSP 2006a, b, 2009) revealed sero-prevalence of 6.5% in north-western Somalia, 28.7% in north-eastern Somalia, 32.6% in central Somalia and 37.6% in southern Somalia. Although these results are inconclusive on the frequency and distribution of PPR in Somalia, they are a good indicator of PPR virus circulation and hence PPR infection as reported by Waret-Szkuta *et al.* (2008) in other studies.

PPR was clinically suspected for the first time in Ethiopia in 1977 in a goat herd from Afar region, eastern part of the country (FAO 2009). Clinical and serological evidence of its presence has been reported by (Taylor 1984) and later confirmed in 1991 with cDNA probe in lymph nodes and spleen specimens collected from an outbreak in a holding near Addis Ababa (P. L. Roeder *et al.,* 1994). Abraham *et al.* (2005) reported the overall seroprevalence of 9% in goats and 13% in sheep in different parts of Ethiopia. It was also reported that 14.6% of sheep sampled along 4 roads from Debre Berhan to Addis Ababa

were seropositive (W.-S. Agnes *et al.,* 2008). In 1999 national serosurveillance of PPR conducted in Ethiopia, the overall seroprevalence of 6.4% (95% CI: 6.0–6.8) in both goats and sheep ranging from 0% to 52.5% was estimated.

Previous study demonstrated that small ruminants including goats can develop positive level of antibody titer against PPRV under natural situation. The Sero-prevalence of PPRV specific antibodies has been recorded from Bangladesh to be 49.17% in goats, 19.05% in cattle, and 36.0% in sheep (Razzaque *et al.,* 2004). To develop disease control strategy against PPR, it is always necessary to have updated baseline seroprevalence data of the PPRV specific antibodies in the small ruminants.

Taylor *et al.* (1990) conducted an epidemiological investigation of PPR in the sultanate of Oman. In their investigation, virological and serological evidences were obtained to show that PPRV was widely distributed in sheep and goats in Oman, of 568 sheep and goats tested, 139(24.5%) was positive. Prevalence by region was 26.5% in Bating coast, 32% in Omani interior, 24.5% in sharqiyah and 4.8% in Salalah. There was no evidence for the concomitant presence of rinderpest virus in these species.

Lefevre *et al.* (1991) conducted a serological investigation of PPR in Jordan. During 1987/88, 8520 serum samples were collected from sheep and goats that were older than six months from 457 flocks in all the governorate of Jordan. Most of the flocks were from extensive types of farm and some flocks had migrated to and from neighboring countries. Of these samples, 548 showed a strong positive result but 64 showed an unclear negative result when tested by the virus neutralization test for antibodies against Rinderpest and PPR viruses. Thirty samples were negative against the two viruses, 32 were positive against PPR virus with titers ranging from 32 to 2048 and 8 samples showed higher titers against rinderpest virus than PPR virus.

Ekue *et al.* (1992) carried out a serological survey of antibodies against PPRV in small ruminants in Cameroon and reported that in micro-neutralization ELISA tests on 640 blood samples collected from sheep and goats in 1986-1988, 292 (46.5%) were positive for PPR virus antibodies with titers ranging from 1:32 to 1:8192. Comparative titration with rinderpest virus detected relatively low virus neutralizing (VN) antibodies from the same area. However 3 out of 111(2.7%) from West province were positive for VN antibodies against rinderpest virus with high titers ranging from 1:32 to 1:512. This indicated a possible latency of the virus in sheep and goat populations with a likely

environment of this species in the epidemiology of rinderpest in cattle in the country. They concluded with a recommendation that readily available and fairly cheap tissue culture rinderpest vaccine be used in a nation-wide campaign against PPR infection in small ruminants.

Saliki *et al.* (1994) stated that a simple and rapid double-antibody sandwich ELISA S-ELISA) was compared with a single-passage virus isolation procedure in Vero cell vultures for PPRV isolation. Eighty nine paired samples (heparinized blood; lachrymal, gal. and oral secretions; sonicated pokeweed mitogen-stimulated peripheral blood lymphocytes; and brain, tonsil, lung, mesenteric lymph nodes, liver, spleen, small intestine, colon, caecum and kidney homogenates) were obtained from 6 goats experimentally infected with PPRV. S-ELISA was positive for 64 (71.9%) samples, while virus isolation was positive for 58 (65.2%) samples. S-ELISA was significantly more sensitive.

Saudi Arabia is a major importer of livestock, the first recorded case of PPR was observed in 1990 (Abu Elzein *et al.*, 1990), ad later it was reported in Eastern central region of the country (Housawi *et al.,* 2004; AL-Afaleq *et al.*, 2004; Al-Dubaib, 2009; Boshra *et al.*, 2015). However, the disease has not been reported from all parts of the country.

Krishna *et al.,* (2001) carried out a serological survey on the prevalence of PPRV using differential neutralization tests with PPRV and RPV and an assessment was also made on the cross reacting antibodies to PPRV and RPV in small ruminants. A total of 672 (556 sheep and 116 goats) serum samples were collected from various districts of Andhra Pradesh and were analyzed simultaneously for the presence of neutralizing antibody to PPRV and RPV. Results revealed that 20 (3.6%) of the 556 sheep serum samples were positive to PPRV. Of these 20 positive cases, 5 (3.07%) were positive to PPRV alone and the 15 (9.2%) showed higher titres of PPRV and lower titres of RPV. None of the goat samples were positive to PPRV antibodies.

Roger *et al.* (2001) designed a serological survey to determine the antibody prevalence of RPV and PPRV in Ethiopian camels. A total of 90 dromedaries were distributed in-groups based on three epidemiologically-defined regions. The first group was from a non-affected area, the second from sick and contact animals and the third from convalescent animals. The sera were analyzed for antibodies to RPV and PPRV by

cELISA tests. Results showed a global seroprevalence of 7.8% for PPRV antibodies and 21.3% for RPV antibodies. None of the sera from the non-affected area were positive and the second and third groups had varying positive rates. In accordance with several authors, the receptivity of the camel to these viruses appears to be a reality. However, its susceptibility to RPV and PPRV, as well as its role as a potential reservoir of these viruses that cause two major diseases of ruminants, had never been confirmed. The hypotheses about the occurrence of an emerging infection in camels, caused by pathogens usually found in cattle, sheep and goats, are discussed.

According to sero-surveys, PPR virus had already been circulating in Kenya and Uganda during the 1980's but has been officially declared to the OIE as endemic in Kenya since 16 May 2007, and in Uganda since 10 Aug 2007. The first occurrence of PPR in Morocco in 2008 indicated that the virus had crossed the natural barrier of the Sahara with concomitant risks for North Africa. Following its spread into Tanzania in early 2009, the FAO published in November 2010 a warning about the potential spread of PPR to southern Africa, and emphasized that PPR is posing a mortal threat to more than 50 million sheep and goats in 15 countries. PPR has been reported annually in the Comoros islands since 2010. The disease is now (2013) recognized as also being present in the Democratic Republic of Congo and northern Angola. It is therefore clear that although PPR was formerly restricted to Africa, Asia and the Middle East, its distribution has expanded in the last 10 years.

2.4.5. Transmission of the virus

Transmission requires close contact between infected animals in the febrile stage and susceptible animals (Braide, 1981) because of the lability of the virus outside the living host. The discharges from eyes, nose and mouth, as well as the loose faeces, contain large amounts of the virus. Fine infective droplets are released into the air from these secretions and excretions, particularly when affected animals cough and sneeze (Bundza *et al.,* 1988; Taylor, 1984).

Animals in close contact inhale the droplets and are likely to become infected. Although close contact is the most important way of transmitting the disease, it is suspected that infectious materials can also contaminate water and feed troughs and bedding, turning them into additional sources of infection. These particular hazards are, however, probably fairly short-term since the PPRV, like rinderpest, would not be expected to

survive for long outside the host. Indirect transmission seems to be unlikely in view of the low resistance of the virus in the environment and its sensitivity to lipid solvent (Lefèvre and Diallo, 1990). There is no known carrier state for PPRV. Trade in small ruminants, at markets where animals from different sources are brought into close contact with one another, affords increased opportunities for PPR transmission, as does the development of intensive fattening units.

2.4.6. Host range

Peste des petits ruminants is a disease of sheep and goats. In general goats are more susceptible than sheep; with sheep undergoing a milder form of the disease (Lefevre and Diallo, 1990). Other domestic animals such as camels, cattle and pigs are known to undergo subclinical infection of PPR (Taylor, 1984). The disease has been reported in wild small ruminants in a zoo (Furley *et al.,* 1987) and those living in the wild (Ogunsanmi *et al.,* 2003; Sharawi *et al.,* 2010; Kinne *et al.,* 2010).

2.4.7. Host determinants of the disease

Host determinant factors of PPR spread have been reported in various studies, highlighting age, sex, breed and animal species (Munir *et al.,* 2013). Young animals are less likely to have developed protective antibody titers and therefore are more susceptible to PPRV (Luka *et al.,* 2011). This high susceptibility in the young has been reported in Ethiopia, Kenya, Pakistan, India and Turkey; thus, age of small ruminants is a key risk factor for susceptibility/resistance to the disease (Waret-Szkuta *et al.,* 2008, Abubakar et al 2009, Singh *et al.,* 2004b; Ozkul *et al* 2002). In Oman, the disease is reported to maintain itself in susceptible yearling population, with an increase in incidence being a reflection of increased number of susceptible young goats/sheep recruited (Taylor *et al.,* 1990). Sex has also been reported as a risk factor for susceptibility/resistance to the disease (Abdalla *et al.,* 2012; Sarker and Islam, 2011; Swai *et al.,* 2009; Waret-Szkuta *et al.,* 2008). The off-take of male small stock for social economic activities is higher and at an early age compared to females which end up staying in the herds for longer periods for productive purposes females (Singh *et al.,* 2004b). Therefore, females are more likely to demonstrate antibody titers than the males. The recruited young males, having been in the herds for a shorter period, are less likely to have been in contact with virus. Indeed, studies in Bangladesh have shown that male goats are significantly more prone to PPR than females (Sarker *et al.,* 2011). However,

studies from Pakistan have shown no significant difference between males and females, with respect to susceptibility (Munir *et al.,* 2008).

The influences of breeds of the small ruminants on susceptibility to the disease have also been studied by Munir *et al.* (2008), with results showing that there are insignificant differences between goat breeds but there are significant differences between sheep breeds. Breed differences to susceptibility to PPR have been reported in other studies (Lefevre and Diallo, 1990; El Hag and Taylor 1984; Diop *et al.,* 2005). Goat and sheep species" differences have been highlighted as major risk factor for PPRV susceptibility (Swai *et al.,* 2009, Munir *et al.,* 2008, Waret-Szkuta *et al.,* 2008). Though PPR has been described in other species of animals, the camel is emerging as a key risk factor in long distance transmission of the disease particularly those used in trade caravans (Libeau *et al.,* 2011).

2.4.8. Social ecology and seasonality of the PPR disease

It has been reported that the recent PPR disease outbreaks have been attributed to the cessation of rinderpest vaccination and loss of antibody cross protection between the PPR and rinderpest, leaving the small ruminants fully exposed to PPRV (Libeau *et al.,* 2011). However, the spread of the PPR outbreaks has for a long time been associated with social, cultural and economic activities such as conflicts, disasters, livestock trade, cultural festivals, and change of husbandry practices, nomadism and seasonal climatic and environmental changes (FAO. 2009b, Libeau *et al.,* 2011). It has been reported that in Maghreb countries of North Africa, traditional sacrifices of sheep during major Islamic festivals provide a major opportunity for seasonal clustering of small ruminants of multiple sources whose health status is often unknown, thus creating a favorable environment for the transmission and dissemination of the PPR virus (Dufour, 2010). In the Sahel region, sero-prevalence of 75% is observed in pastoralist small ruminants and in most cases the disease is muted or subclinical (Grenfell and Dobson, 1995).

Clinical PPR is more prevalent in the humid and sub humid regions of West Africa with morbidity of 80 to 90% resulting in mortality of about 50 to 80% (Lefevre and Diallo, 1990). These epidemics in West Africa, which coincide with wet rainy seasons, have been associated with seasonal animal husbandry patterns and livelihood activities among the settled and pastoralist communities (Mai *et al.,* 2004; William and Barker, 2001).

However, Opasina and Putt (1985) have reported PPR disease outbreaks in South west Nigeria during dry season, in different ecological zones.

In Sudan, PPR outbreaks in camels coincided with the seasonal movement of animals towards autumn green pasture (Khalafalla et al, 2010), while other studies by Abdalla et al (2012) revealed significant association between prevalence of PPR and winter season. Seasonality of PPR in Ethiopia has been attributed to seasonal movement of small stock in search for water and pasture resources during dry seasons, social exchange of animals and livestock marketing which exhibit seasonal patterns with pick outbreaks being experienced in March-June and October-November (Abraham G, 2005, Waret-Szkuta *et al.,* 2008).

2.4.9. Potential risk factors of PPR

Kids over four months and under one year of age are most susceptible to the disease. Sahelian breeds of sheep and goats are believed to be more resistance than the dwarf breeds in the humid and sub-humid zones of West Africa. In a particular flock, risk of an outbreak is greatly increased when a new stock is introduced or when animals are returned unsold from livestock markets. Recovered animals have lifetime immunity (Radostitis *et al.,* 2007).

The disease is transmitted by direct and indirect contact (Carter *et al.,* 1993). Large amounts of the virus are present in all body excretions and secretions, especially in diarrheic faeces. Infection is mainly by inhalation but could also occur through conjunctiva and oral mucosa (Radostitis *et al.,* 2007).

2.4.10. Wild life Susceptibility to the disease

Wildlife susceptibility to PPR is a complicating factor, with infection and clinical disease reported in Dorcas gazelle (in captive groups), Thomson‟s gazelle, gemsbok and ibex (Wohlsein and Saliki, 2006; Gur and Albayrak, 2010), as well as in wild sheep such as bharals in Tibet and in wild goats in Kurdistan (Bao *et al.,* 2011; Hoffmann *et al.,* 2012). Recently, an outbreak of PPR in truly free-ranging Sindh ibex was confirmed by immunocapture ELISA and PCR in Pakistan in 2010 with 36 deaths, possibly associated with the sharing of water pasture with a presumed infected goat herd (Abubakar *et al.,* 2011).

The role of wild species as a reservoir has not been studied. However, considering the role of wildlife in the epidemiology of rinderpest (Shanthikumar*et al.,* 1985; Anderson *et al.,* 1990; Couacy-Hymann *et al.,* 2005; Kock *et al.,* 2006; Rossiter *et al.,* 2006), further research is needed about potential PPR spread through wild species. This may have serious repercussions in Ethiopia, where several wild ruminant species have the opportunity to contact during grazing and watering.

2.4.11. Pattern of the disease

In general, morbidity is common, particularly in fully susceptible goat populations. Mild forms of the disease may occur in sheep and partially immune goat populations. There are considerable differences in the epidemiological pattern of the disease in the different ecological systems and geographical areas. In the humid Guinean zone where PPR occurs in an epizootic form, it may have dramatic consequences with morbidity of 80%-90% accompanied with mortality between 50 and 80% (Lefèvre and Diallo, 1990). While in arid and semi-arid regions, PPR is seldom fatal but usually occurs as a subclinical or in apparent infection opening the door for other infections such as Pasteurellosis (Lefèvre and Diallo, 1990). Though outbreaks in West Africa coincide with the wet rainy season, Opasina and Putt (1985) observed outbreaks during the dry season in two different ecological zones. A high morbidity of 90% accompanied with 70% case fatality was reported from Saudi Arabia (Abu Elzein *et al.,* 1990).

Serological data from Nigeria revealed that antibodies occur in all age groups from 4-24 months indicating a constant circulation of the virus (Taylor, 1979b). In Oman the disease persisted on a year round basis maintaining itself in the susceptible yearling population (Taylor *et al.,* 1990). Therefore, an increase in incidence reflects an increase in number of susceptible young goats recruited into the flocks rather than seasonal upsurge in the virus activity, since its upsurge pend on the peak of kidding seasons (Taylor *et al.,* 1990). Moreover, the susceptibility of young animals aged 3 to 18 months was proved to be very high, being more severely affected than adults or unweaned animals (Taylor *et al.,* 1990).

2.5. Current Diagnostic Techniques

Earliest possible diagnosis of PPR is crucial in implementing control measures, to contain outbreaks and minimize economic losses. Initially, the majority of PPR outbreaks were diagnosed based on typical clinical signs. However, the signs of PPR are

often difficult to distinguish from those caused by a number of other diseases, such as foot-and-mouth disease and bluetongue disease (Munir *et al.,* 2013). This situation becomes even more complicated when these diseases are circulating in areas where PPR is endemic. Thus, it is necessary to confirm the clinical diagnosis through laboratory testing (Munir *et al.,* 2013). Currently, the diagnosis of PPRV is made based on demonstration of antibodies, which is a good indication because an animal infected with PPRV carries antibodies for life, with the development of a sustained antibody response.

2.5.1. Serological detection

Most of the available diagnostic assays have been developed based on the N protein. Owing to the presence at the 3′ end of the genome of PPRV, the N protein produced in quantities higher than any other structural proteins because attenuation occurs at each intergenic region between two genes (Lefevre *et al.,* 1991; Yunus and Shaila, 2012). The antibodies produced against the N protein don"t protect the animals from the disease. Due to abundance of the N protein it remains the most acceptable target for the design of PPRV diagnostic tools (Diallo *et al.,* 1994).

Moreover, because the HN protein is the most diverse among all the members of morbilliviruses, RPV and PPRV share only 50% similarity in their HN proteins. The HN protein determines cell tropism; most of the protective host immune response is raised against HN protein. Therefore, serological assays have also been developed targeting HN protein (Munir *et al.,* 2012a, 2013). Commercial ELISAs are available based either on the HN (Saliki *et al.,* 1993; Anderson and McKay, 1994; Singh *et al.,* 2004) or N proteins (Libeau *et al.,* 1995) for specific detection of antibodies against PPRV, in any susceptible host. The sensitivity and specificity of these assays can be as high as 90% and 99%, respectively.

2.5.2. Antigen detection

Immunocapture (Libeau *et al.,* 1994) and sandwich ELISAs (Saliki *et al.,* 1994) are available to efficiently detect antigens in the tissues and secretions of PPRV-infected animals. Both these assays utilize monoclonal antibodies (MAbs) directed against the N protein of PPRV. Both assays are rapid, sensitive and specific with a detection limit of 100.6 TCID50/well. Since the MAbs used in these assays are raised against the nonoverlapping domains of the N protein of PPR and RP viruses, this assay can be used to differentiate PPRV- from RPV-infected animals (Libeau *et al.,* 1994). The lateral flow

device (LFD)-based test for PPR using monoclonal antibody C77 recognizing the H protein of PPRV (Anderson *et al.,* 1990; Anderson and Mckay, 1994).

2.5.3. Genome detection

To overcome several shortcomings of the serological and antigen detections, such as the requirement of sera in well-preserved format, several PCRs have been developed for PPRV with wide range of sensitivities, specificities and detection limits (Munir *et al.,* 2013). Despite the high sensitivity and specificity of these assays, and their validity to detect both vaccine and field viruses, none of the assays is a formally approved OIE method. For this they need further extensive validation. None of the assays is field applicable since they require thermocycler and electrophoresis apparatus for RT-PCR, and real-time PCR for probe or SYBR Green-based assays. However, with the development of LAMP assay, on-site detection can be proposed. It is highly plausible tocombine the simple procedures for RNA extraction using Whatman FTA card (Munir *et al.,* 2012b, 2012c) and using the RT-LAMP assay for isothermal amplification. This could possibly be applied for field diagnosis of PPRV. Recently, a novel and nonamplification strategy was proposed in which two probes complementary to the target sequences (one conjugated to magnetic microparticles, the second to gold nanoparticles labelled with horseradish peroxidase) were used (Tao *et al.,* 2012). On specific binding to the target, the system allows magnetic separation and substrate detection. It was proposed to be quick (45 minutes), cheap and sensitive (17.6 ng/μl) for PPRV detection. This method holds great potential, especially when it is multiplexed for the detection of several pathogens in the same clinical sample.

2.6. Opportunities Presented Regarding PPR Eradication

The epidemiology and biology of the PPRV are very much similar to those of the RPV. Therefore, there are enough reasons to control and eradicate PPR very much in a similar way like rinderpest. Like RPV, there are several aspects that may favor eradication of PPR: (i) there is only one serotype of PPRV and it is believed that perfect cross protection appears to exist within strains from different lineages. (ii) Vaccine is considered to provide life-long immunity. (iii) There is no carrier state. (iv) A close contact between the animals is required for effective transmission of the disease. (v) Virus does not survive for a long period of time outside the host as it is readily destroyed by heat and sunlight and hence needs continuous source of susceptible animals for

survival. (vi) Appropriate diagnostic tools are available. However, unless the vaccine is used sufficiently, widely and thoroughly to stop transmission of the virus in the endemic areas, it may simply be wasting the public funds and at worst helping the virus to perpetuate (Kumar *et al.,* 2013).

2.7. Socio-Economic Impact of PPR

Peste des Petits Ruminants virus has a widespread distribution spanning Africa and Asia (Nanda *et al.,* 1996; Shaila *et al.,* 1996). These areas encompass much of the developing world that relies heavily on subsistence farming to supply food or goods for trade, and small ruminants provide an excellent supply of both. Unfortunately, in many areas of Asia and Africa, small ruminant production and therefore the livelihoods of poor farmers is threatened by PPR among other trans-boundary animal diseases (TADs). With its associated high morbidity and mortality, PPRV constitutes one of the major obstacles to subsistence farming; a static and structured spreadsheet model was used to assess the costs of inaction on PPR in sheep and goats. The direct costs of this disease refer to the monetary values of physical losses due to the disease. Since PPR is more acute disease, these physical losses are only the results of mortality associated with disease. Mortality induces losses associated with the cost of dead animals. Disease burden is defined as the sum of direct costs of the disease, which include cost of mortality and the incurred costs of treatment and additional feed cost (Barnyard *et al.,* 2010).

The first step in this process was to determine the population at risk, which depends on the degree to which livestock population is protected by existing prophylactic measures. In that regard, background information on livestock across agro-ecological zones, vaccine availability, treatment availability and the degree to which disease surveillance programs are implemented are important. The data required are livestock population number, livestock production parameters, price/cost data and epidemiological parameters. The livestock population data are disaggregated by species, age and sex. Livestock production parameters are also collected by species and agro-ecological zones. The price/cost data to use as inputs in the spreadsheet model include cost of feed, price of live animals by species and age category, cost of treatment. The data used in this study are presented in Appendix part and are all for the year 2015/2016 or adjusted to that year when applicable. The epidemiological parameters involve disease incidence rate, affection rate (i.e., morbidity proportion and mortality proportion), rate of

vaccination coverage, extent of disease surveillance, disease treatment rate and impact of affection on productivity. These data were gathered from secondary sources, published studies and through interview.

In this exercise, data collected through questionnaire were compared and contrasted with data collected from secondary sources and judgments were made about the magnitude of the parameter estimates to use. Hence, the incurred costs of treatment and additional feed used to calculate the disease burden are elective, based on data collected through questionnaire, or from secondary sources, or from our assessment based on the two. The costs of treatment and feed were referred to as actual intervention costs and include for activities conducted by private public entities and The socio-economic losses associated with PPR mainly result from the high mortality rate that is characteristic of the disease. This negatively affects income from production and value addition in small ruminants marketing chains. Peste des Petits Ruminants disease is a constraint to international trade, although this impact is mitigated in local and regional markets due to wide geographic distribution of the disease at present (Elsawalhy *et al.,* 2010). However, the direct economic losses caused by the disease are aggravated by the sanitary measures imposed by authorities to control animal movement and by trade restrictions on animal by-products (Bailey *et al.,* 1999).

Because of the negative economic impact on countries affected by PPR, the disease is one of the priorities among international and regional livestock disease research and control programs (FAO 2012b; Baron, 2012; Soumare, 2013; Domenech, 2013). An international study conducted by Perry *et al.,* (2002) ranked PPR in the top ten diseases affecting small ruminants. The disease has also been ranked by pastoral communities as one of the top ten diseases of small ruminants (Diallo, 2006).

It is estimated that one billion small ruminants or about 62.5% of global domestic small ruminant population is at risk of infection with PPR (FAO, 2009a). However, there are very few economic studies related to the economic impact of the PPR and the data available on losses due to the disease is scanty (Diallo 2006; Munir *et al.,* 2013).

2.8. Differential Diagnosis

Other diseases cause diarrhea or pneumonia in sheep and goats may pose diagnostic challenge but a history of recent introduction of new stock and the clinical and postmortem findings of stomatitis, typical for PPR. Laboratory tests are requiring ruling out rinderpest (Radostitis et al, 2007 &LPP, 2006). In addition to rinderpest, other conditions that should be considered in differential diagnosis include: contagious caprine pleuropneumonia, bluetongue, pasteurollosis, contagious ecthyma, foot and mouth disease, heart water, coccidiosis and mineral poisoning (OIE, 2002).

Differential diagnosis of disease is to be made with Foot and Mouth Disease, Bluetongue and Peste Des Petits Ruminants (PPR) (Kitching, 2001). Orf virus has primarily affinity for oral and perilabial area to cause erythematous and ulcerative papules (Kumar *et al.*, 2015) but Peste Des Petits Ruminants (PPR) causes necrotic and erosive stomatitis (Abubakar *et al.*, 2015). Such type of similarity in site of lesions is responsible for incorrect diagnosis of orf as PPR in the field. This misdiagnosis of Orf, having zoonotic potential leads to transmission of disease from affected animals to humans (Koufakis *et al.*, 2014; Sanchez *et al.*, 1985; Mohr and Katz, 1989).

Disease transmission to human is also possible by having any abrasion and cutting infected meat (Maor, 2017). Veterinarian, farmers and butchers are the people who are at risk if they do not handle animals with care, considering Orf as PPR. In this outbreak, disease was tentatively diagnosed as PPR by veterinary assistant but later on was confirmed as Orf. So, objective of this report is to differentiate between Orf and PPR and stress for differential diagnosis of PPR.

2.9. Prevention and Control

2.9.1. Vaccination

There is no specific treatment against PPR. Antibiotics may prevent secondary pulmonary infections but this treatment is too costly in case of an outbreak. Therefore, the control of this disease is through the implementation of sanitary and medical prophylaxis measures (Berhe, 2006), although it is obvious that strict sanitary measures are hardly possible in developing countries (Berhe, 2006). Vaccination is the preferred method of control (Nawathe, 1984). The attenuated tissue culture Rinderpest vaccine has been used for a long time to protect small ruminants against PPR. This vaccine provides

protection for over one year and is tolerated by healthy goats of all breeds (Nawathe, 1984). Vaccination programmes do not always produce the desired benefits, at least in the short term (Reynolds *et al.,* 1988). High vaccination coverage and vaccine efficacy are required to prevent major epidemics (Leissel and Salama, 2003). An effective vaccine must (1) Induce right sort of immunity, (2) Be stable on storage, (3) Have sufficient immunogenecity (Peter, 2001). Effectiveness refers to the reduction in disease measured under conditions of use of the vaccine in ordinary clinical practice (Simon *et al.,* 1995). At the end of the 1980s, a PPRV strain was successfully attenuated by serial passages in Vero cells (Diallo *et al.,* 1989). The PPRV homologous vaccine was found to be safe under field conditions even for pregnant animals and it induced immunity in 98% of the vaccinated animals. A single injection and the induced immunity cover at least the economic life of the animals, around three years (Diallo *et al.,* 1995). Normally, homologous PPR vaccine attenuated after 63 passages in Vero cell produced a solid immunity for 3 years (Diallo *et al.,* 1995). The vaccine is harmless on pregnant sheep and goats at any stage of gestation and induces the production of colostral anti-PPR antibodies that have been found in kids up to 3 months old (Diallo, 2003). It is suggested that kids and lambs from immunized or exposed dams should be vaccinated at 4 and 5 months of ages, respectively (Awa *et al.,* 2002). Similarly, the antibodies due to naturally exposure to PPR infection might also interfere with the efficacy of vaccines. Therefore, monitoring of antibodies would be required before mass vaccination against PPR, especially in enzootic areas (Banik *et al.,* 2008).

To halt further spread of the disease, targeted vaccination of small ruminants based on critical control points such as livestock markets and transport routes used by traders and semi pastoralists is recommended. Animals recovered from PPR infection or immunized by 14 vaccination are not a PPRV carrier and do not play a role in maintaining virus circulation in an area. Animals that are not 'immunized' against the virus through natural infection or vaccination can be subclinically infected and constitute a high risk in maintaining and diffusing the virus without apparent clinical signs (Wakhusama *et al.*, 2011).

The advantages of vaccination as a control option are that the vaccine is readily available and very cheap. It confers immunity which lasts for 3 years and hence most animals will only need two vaccinations in their life time. However, annual vaccination is recommended due to the high reproductive rate of small ruminants. It will be important

to determine which strain(s) are circulating in newly infected countries. Control of PPR outbreaks relies on movement control (quarantine) combined with the use of focused (ring) vaccination (Roeder and Obi, 1999).

2.9.2. Surveillance

When analysing the surveillance options to employ, it is important to determine the objectives. Ideally, surveillance should aim to; define the extent of the disease, detect new outbreaks, establish disease free zones, monitor disease trend, and inform decision making at various disease control critical points (SADC, 2012b).

2.9.3 Biosecurity

The main mode of PPR virus transmission is through animal movement, which can either be illegal or under permit. With the advent of free movement of people and goods (including livestock and livestock products) as the SADC region moves ever closer to a borderless region, it is no longer enough for a country to rely on national animal movement controls of its neighbour to stem the spread of transboundary animal diseases (TADs) (SADC, 2012b).

When control of animal movement is used as one of the arms of an effective control of TADs, it should be implemented in a very careful and strict manner (Wakhusama *et al.* 2011).

All stakeholders such as the police, customs officials and farmers themselves must be engaged to support the movement restrictions from infected areas.

2.9.4 Stamping out

This option is favoured only in situations where the infected population is small and well defined and government has mechanisms in place to compensate the affected farmers.

Stamping-out programs involve the eradication of a disease by the destruction of all infected animals. When outbreaks occur, protection and surveillance zones are established around the outbreak area, and animals in the protected zone are destroyed. The level of surveillance in the area should be increased and the movement of animals from surveillance zone should be restricted. This has the ability to regain previous disease free status quickly and therefore be able to trade again as the biggest advantage (SADC, 2012b).

This option is best suited for high risk areas with low density of animals and for low risk areas such as Zambia. The main disadvantage of this option is that it is usually an expensive and therefore unattractive exercise for the State and as a result there is little political will to implement stamping out. It also has social and economically devastating consequences for affected communities even if they will be compensated. Other disadvantages linked to this option are loss of genetic material, diminishing the national herd andis difficult to carry out in light of lack of fences and zones to curtail movement in the event of an outbreak. In addition politically is very difficult (SADC, 2012b).

2.9.5 Public awareness

There should be need to create awareness through simple technical messages for farmers, traders, politicians, community leaders, the media, law enforcement officers, and the general public at large (SADC, 2012). There is also need to train field staff in the available control options such as vaccination, stamping out, zoning, and biosecurity. The government should 15 also provide the necessary material and financial resources to implement the available control options.

2.9.6 Treatment

Although there is no specific treatment for PPR, antibiotics and other supportive treatment are normally used to prevent secondary infections and decrease mortality (OIE 2012).

CHAPTER 3

METHODOLOGY

The present study was conducted in 5 different areas of Dinajpur district and the pathology laboratory, Department of Pathology and Parasitology, Faculty of Veterinary and animal Science, Hajee Mohammed Danesh Science and Technology University (HSTU), Dinajpur, Bangladesh for a period of 6 month from January to June 2019. The details outline of materials and methods are given below:

3.1 Study area

A cross-sectional study was undertaken in 5 different areas in Dinajpur.

Fig. 1: Study area at Dinajpur Sadar Upazila

3.2 Study population

A total of 79 blood samples from goats (58) and sheeps (21) were collected randomly according to the animal ages, sex, breed and lactation stage. After collection, the sample were kept for an hour at room temperature, serum was separated and stored at -20 °C until use.

3.3 Experimental layouts

Field survey was carried out and selected randomly in five different areas in Dinajpur district and blood samples were collected directly from jugular vein by vein puncture method from goats and sheeps which have no pervious history of peste des petits ruminants and rinderpest (RP) vaccination by using sterile 5 ml syringe. The collected samples were kept for 1-2 hours in slightly incline position after that sera were separated from blood in sterile screw capped serum tube (eppendorf tube) and the samples was placed in ice box and transported to the Pathology lab of HSTU, Dinajpur. The serum samples were subjected to indirect enzyme-linked immunosorbent assay (indirect ELISA) by the detection and measurement of antibody level against the PPR virus in the sera.

Ultimately the value was calculated optical density (OD) that was taken from the indirect ELISA plate reader machine, during the survey questionnaire was collected about the hygienic and management practice, diagnostic history, clinical sign of the animal and then finally data recorded and analyzed.

Layout of the experiment

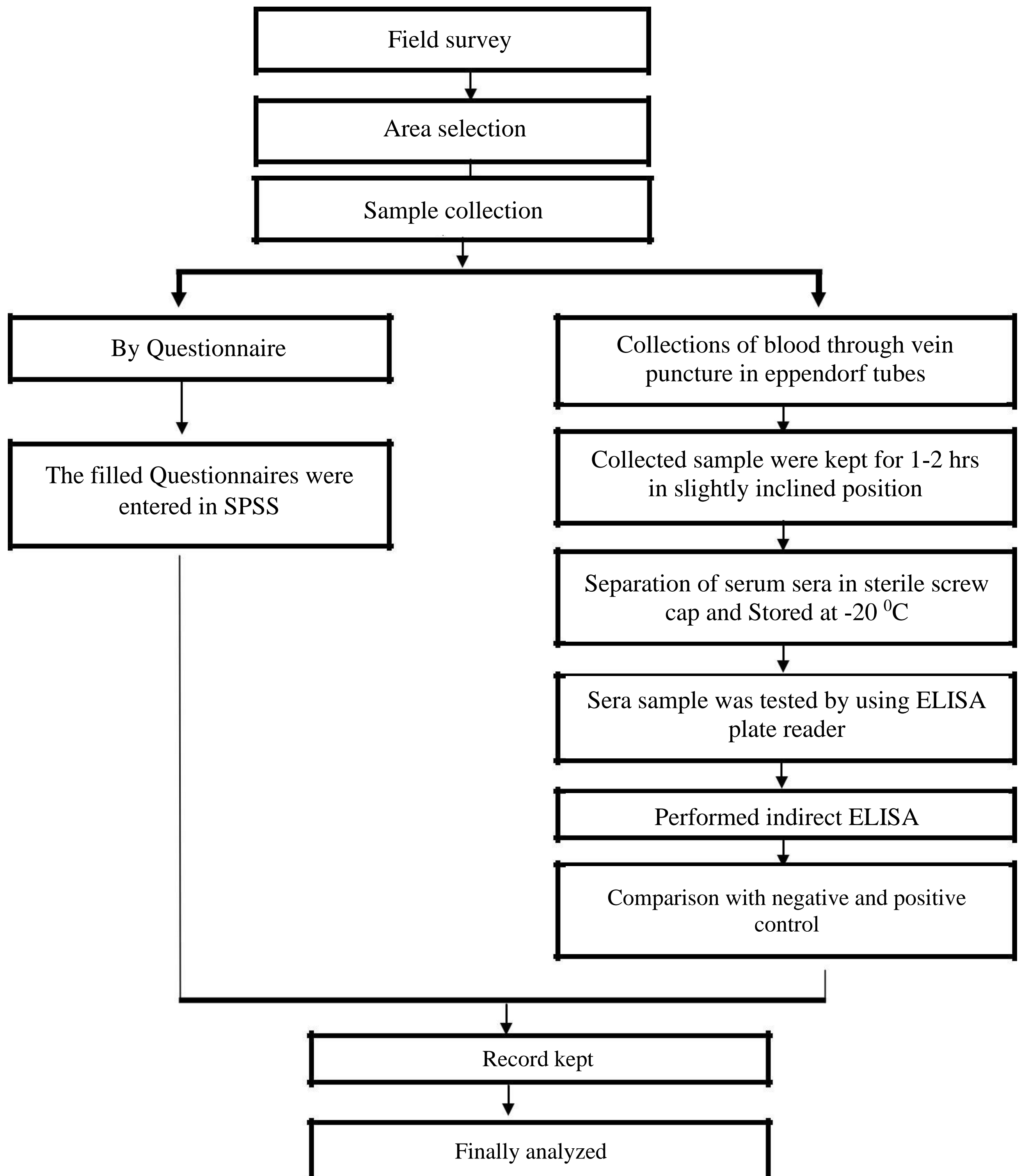

Fig. 2: Schematic representation for experimental lay out

3.4 Materials and Methods

3.4.1 Materials

3.4.1.1 Appliances used for blood collection

- ✓ Sterile Syringes (Vol. 5 ml)
- ✓ Ice box
- ✓ Cotton,
- ✓ Hand gloves and Marker pen
- ✓ Scissors,

3.4.1.2 Material required used for ELISA test

- ✓ ELISA plate Reader,
- ✓ ELISA Kit, Manufactured by ID. Vet, CIRAD-EMVT, Montpellier, France.
- ✓ Mono or multi-channel Micropipettes cable different delivering volume like 10µ l50µl 100µl, 200µl) and Disposable tips of varying categories.
- ✓ Distilled or de-ionized water.
- ✓ Foil paper
- ✓ Falcon tube
- ✓ Beaker (500 ml), Racks
- ✓ Falcon tubes (10ml, 50ml), Dish, Ependorf® tubes (Vol. 1.5 ml),
- ✓ Cotton and Tissue paper
- ✓ Waste disposal container
- ✓ Wash system

3.4.1.3 Chemicals and reagents used for indirect ELISA

The competitive ELISA kit was developed by ID. Vet. Innovative Diagnostics, CIRAD EMVT, Montpellier, France.

- ✓ Microplate coated with recombinant nucleoprotein
- ✓ Anti-NP-HRP concentrated conjugate (10X)
- ✓ Positive control
- ✓ Negative control
- ✓ Dilution buffer 4
- ✓ Dilution buffer 13

- ✓ Wash concentrate (20X)
- ✓ Substrate solution
- ✓ Stopping solution (0.5M)
- ✓ Alcohol

3.5 Antibody detection by indirect ELISA

3.5.1 ELISA kit

Serum sample were applied indirect ELISA kit manufactured by ID. vet Innovative Diagnostics, CIRAD-EMVT, Montpellier, France. to detect of anti-PPRV nucleoprotein antibodies in goat serum.

3.5.2 Reagent preparation

Preparation of wash solution

300ml Wash solution (1X) was prepared by diluting the wash concentrate (20X) in double distilled water as per follows

- ✓ Double distilled water : 290 ml
- ✓ Wash solution : 10 ml

Preparation of conjugate

10 ml Conjugate (1X) was prepared by diluting Conjugate (10X) in Dilution Buffer 4 as per follows

- ✓ Dilution buffer 4 : 10 ml
- ✓ Concentrate conjugate: 1 ml

3.5.3 Sample preparation

96-well plate were added sample test and control sample, also avoided in different incubation time then transferred them into an ELISA microplate that was used multichannel pipettes

3.5.4 ELISA plate layouts

1. Positive control (Pc): Wells A1, B1, was the positive control. They contain dilution buffer 13, positive control, conjugate, substrate and stop solution.

2. Negative Control (Nc): Wells C1, D1 was the negative control, they contain dilution buffer 13, negative control, conjugate, substrate and stop solution.

3. Serum Sample: 79 tested sera were added in the remaining wells (A3 to H12).

They contain dilution buffer 13, conjugate, substrate and stop solution.

3.5.5 ELISA testing procedure

All the reagents were kept and allowed at room temperature before use and homogenized all the reagents by inversion or Vortex.

1. Addition of

- ✓ 25 μl of Dilution Buffer 13 were added to each well.
- ✓ 25 μl of the Positive Control were added to well A1and B1.
- ✓ 25 μl of Negative control were added to well C1 and D1.
- ✓ 25 μl of each sample were tested main to the remained wells.

2. 45 min ± 4 min at 37 0C (± 3 0C) were Incubated.

3. Three times were washed each well with a proximately 300μl of the washing solution. Avoid drying of the well between washings.

4. 100 μl of the Conjugate 1X were added to each well.

5. 30 min ± 3 min at 21 0C (± 5oC) were incubated.

6. Three times were washed each well with a proximately 300μl of the washing

Solution. Avoid drying of the well between washings.

7. 100 μ1 of the Substrate solution were added to each well.

8. 15 min ± 2 min at 21 0C (± 5oC) were Incubated in the dark.

9. 100 μl of the Stoping solution were added to each well in order to stop the reaction.

10. Read and recorded the O.D at 450 nm.

3.5.6 Calculation of the result

For each sample were calculated by the competition percentage (S/N %).

$$S/N\% = \frac{\text{OD sample}}{\text{ODNC}} \times 100$$

Samples were presented S/N %:

- ✓ Less than or equal to $\leq 50\%$ were considered Positive,
- ✓ Greater than 50 % and less than or equal to 60 % (50-% < SN % $\leq$ 60 %) were considered doubtful,
- ✓ Greater than 60 % (SN % > 60%) were considered Negative.

Validity test

The test is validated if

- ✓ The mean of the Negative control O.D. (ODNC) is greater than 0.7.

$$ODNc > 0.700$$

- ✓ The mean value of the positive control (ODPC) is less than 30% of the ODNC.

$$ODPCODNC < 0.3$$

3.6. Methods

3.6.1. Field survey

A field survey was carried out and questionnaire was used to collect the data about the risk factors associated with the PPRV disease outbreak from owner of goat and sheep the stakeholders of veterinary hospitals in Dinajpur district.

3.6.2 Blood collection and serum separation

Blood samples were collected from jugular vein of goats and sheeps through venipuncture method by using sterile 5 ml syringe and test tube without any anticoagulant.

Collected blood was kept at least 1 hour at room temperature in a slightly inclined position (45° angle) to facilitate clotting and separation of serum. Then clotted blood

samples with sera were transferred to refrigerator at 4°C and kept overnight. After this period, the collected sera were decanted into eppendorf tubes and remaining cells were centrifuged at 2500 rpm for 10 minutes. The collected sera were stored at -20°C until use.

3.7. Data analysis

Data was recorded and stored in Microsoft Excel a spreadsheet and the collected data was analyzed with Statistical Package for Social Sciences (SPSS) version 22.

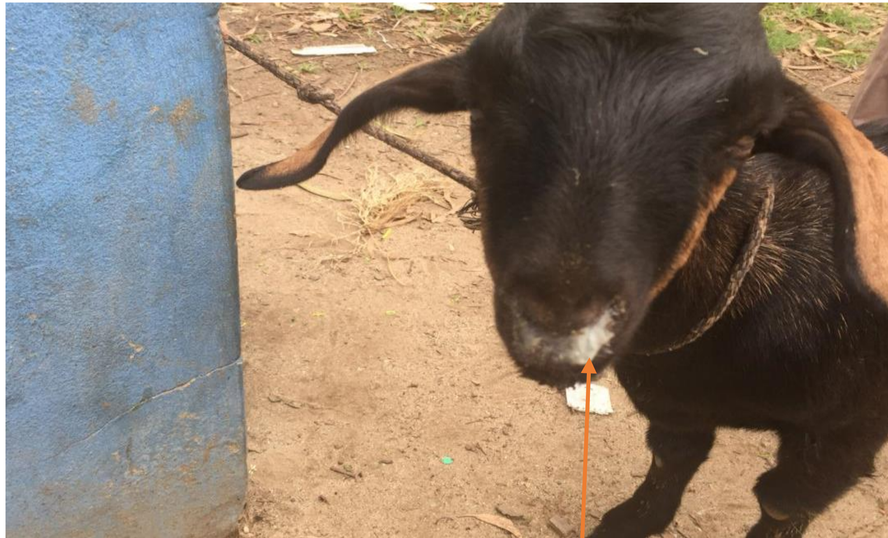

Fig. 3: Mucopurulent ocular and nasal discharge

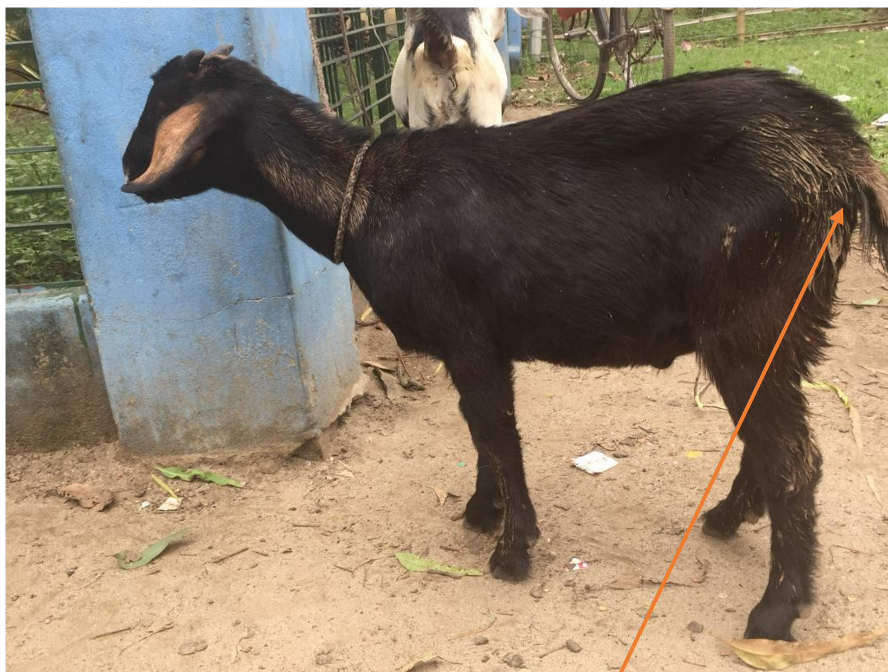

Fig 4: Observed diarrhea

Fig 5: Blood collection from goat

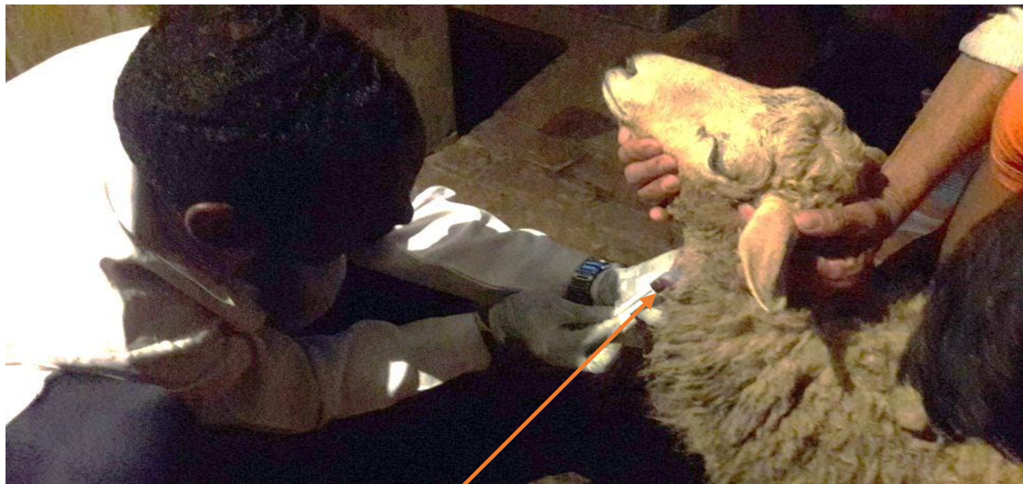

Fig 6: Blood collection from sheep

CHAPTER 4

RESULTS

The present study was conducted to determine seroprevalence of PPR in goats and sheep at Dinajpur district of Bangladesh by Enzyme Linked Immunosorbent Assay (ID.vet Innovative Diagnostic in France) and finally the result are presented below:

4.1 Seroprevalence of PPR in goat and sheep

The seroprevalence of PPR in goat and sheep is presented in Table. The present study revealed that the seroprevalence was higher (P>0.05) in goat (41.18%) compared to sheep (26.92%).

Table 1: Seroprevalence of PPR in goat and sheep

Species	Number of tested sample	Number of positive sample	Prevalence (%)	Chi-square (P-value)
Goat	51	21	41.18	1.512 (0.219) (NS)
Sheep	26	7	26.92	

NS = Non significant

4.2 Sex related seroprevalence of PPR in goat and sheep

The Table shows sex related seroprevalence of PPR in goat and sheep. It was revealed that sex had no significant (P>0.05) effect on the prevalence of PPR in goat and sheep. In case of goat, female (45.71%) had insignificantly higher prevalence of PPR than male (31.25%). In case of sheep, male (28.57%) had slightly higher prevalence of PPR than female (26.32%).

Table 2: Sex related seroprevalence of PPR in goat and sheep

Species	Sex	No. of tested sample	No. of positive	Prevalence (%)	Chi-square (P-value)
Goat	Male	16	5	31.25	0.948 (0.330) (NS)
	Female	35	16	45.71	
Sheep	Male	7	2	28.57	0.031 (0.91) (NS)
	Female	19	5	26.32	

NS = Non significant

4.3 Breed related seroprevalence of PPR in goat and sheep

Breed related seroprevalence of PPR in goat and sheep is presented in Table . The present study revealed that prevalence of PPR in goat and sheep was not significantly (P>0.05) influenced by breed. Black Bengal breed (45.16%) of goat had higher prevalence than cross breed (35.0%). In sheep, cross breed (36.35%) had higher prevalence than indigenous (20.0%).

Table 3: Seroprevalence of PPRV based on breed in goat and sheep

Species	Breed	No. of tested sample	No. of positive	Prevalence (%)	Chi-square (P-value)
Goat	Black Bengal	31	14	45.16	0.518 (0.472) (NS)
	Cross	20	7	35.0	
Sheep	Indigenous	15	3	20.0	0.640 (0.350) (NS)
	Cross	11	4	36.35	

NS = Non significant

4.4 Age related seroprevalence of PPR in goat and sheep

Age related seroprevalence of PPR in goat and sheep is shown in Table . It was found that age had significant (P<0.05) effect on the prevalence of PPR in goat but had insignificant (P>0.05) effect on the prevalence of PPR in sheep.In case of goat, the seroprevalence of PPR was significantly (P<0.05) higher in Group 1 (<1.5 years) (64.29%), followed by Group 2 (1.5-3 years) (34.78%) and Group 3 (>3 years) (16.67%) respectively. In sheep, Group 1 (<1.5 years) (33.33%) was slightly highest prevalence compared to Group 2 (1.5-3 years) (30.77%) and Group 3 (>3 years) (14.29%).

Table 4: Age related seroprevalence of PPR in goat and sheep

Species	Age group	No. of tested sample	No. of Positive case	Prevalence (%)	Chi-square (P-value)
Goat	Group 1 (<1.5 years)	14	9	64.29[a]	8.884 (0.012)
	Group 2 (1.5-3 years)	23	8	34.78[b]	
	Group 3 (>3 years)	24	4	16.67[c]	
Sheep	Group 1 (<1.5 years)	6	2	33.33[a]	0.791 (0.673) (NS)
	Group 2 (1.5-3 years)	13	4	30.77[a]	
	Group 3 (>3 years)	7	1	14.29[a]	

[abc] Superscript letters in same column different significantly (P>0.05). NS = Non significant.

4.5 Seroprevalence of PPR based on hygienic condition

According to the present study, it was revealed that poor hyginic condition (55.0%) of goat was significantly (P<0.05) highest prevalence of PPR than medium (30.77%) and good condition (13.33%) respectively. Good hygienic condition (14.29%) of sheep had lower prevalence rate in comparison to medium (22.22%) and poor condition (40.0%) respectively (Table 13).

Table 5: Seroprevalence of PPR based on hygienic condition in goat and sheep

Species	Hygienic condition	No. of tested sample	No. of Positive case	Prevalence (%)	Chi-square (P-value)
Goat	Good	15	2	13.33[c]	6.86 (0.032)
	Moderate	26	8	30.77[b]	
	Poor	20	11	55.0[a]	
Sheep	Good	7	1	14.29[a]	1.538 (0.463) (NS)
	Moderate	9	2	22.22[a]	
	Poor	10	4	40.0[a]	

[abc] Superscript letters in same column different significantly (P>0.05). NS = Non significant.

4.6 Comparative study of seroprevalence of PPR in goat and sheep

The table shows comparative study of seroprevalence of PPR in goat and sheep. The comparative study revealed that there was no significant variation on the prevalence of PPR among goat and sheep in case of age, sex and hygienic condition. But the prevalence of PPR was higher in goat than sheep in almost all cases except good hygienic condition.

Table 6. Comparative study of seroprevalence of PPR in goat and sheep

Traits	Category	Prevalence (%)		Chi-square (P-value)
		Goat	Sheep	
Overall		41.18	26.92	1.512 (0.219) (NS)
Age	Group 1 (<1.5 years)	64.29	33.33	1.22 (0.269) (NS)
	Group 2 (1.5-3 years)	34.78	30.77	0.060 (0.806) (NS)
	Group 3 (>3 years)	16.67	14.29	0.023 (0.880) (NS)
Sex	Male	31.25	28.57	0.017 (0.898) (NS)
	Female	45.71	26.32	1.95 (0.163) (NS)
Hygienic condition	Good	13.33	14.29	0.04 (0.952) (NS)
	Moderate	30.77	22.22	0.239 (0.625) (NS)
	Poor	55.0	40.0	0.600 (0.439) (NS)

NS = Non significant

Fig 7: After addition of Serum Sample

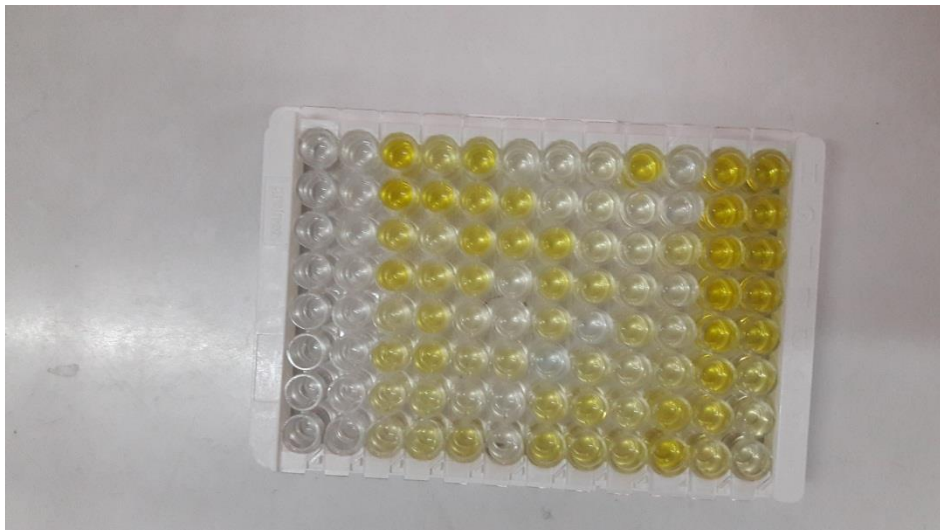

Fig 8: After addition of conjugate solution

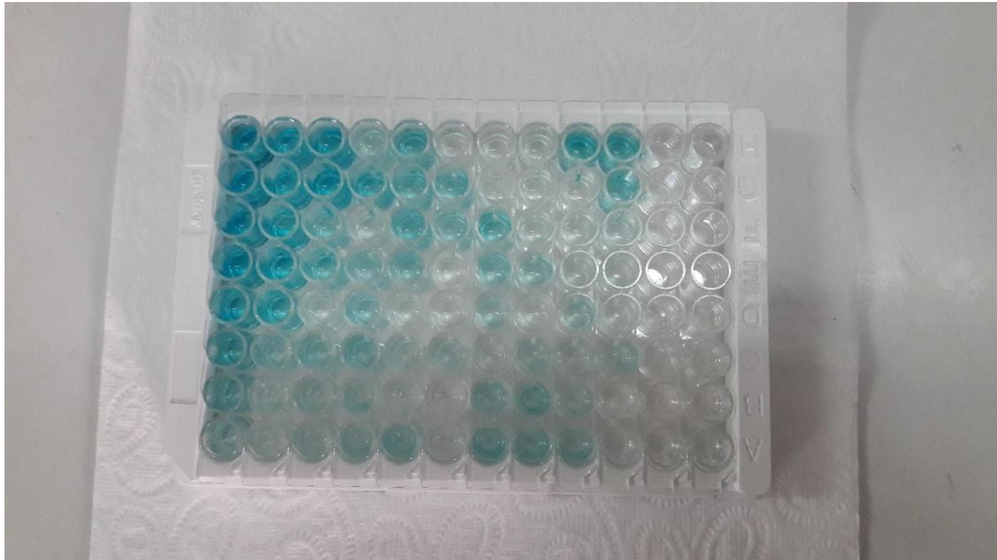

Fig 9: After addition of stopping solution

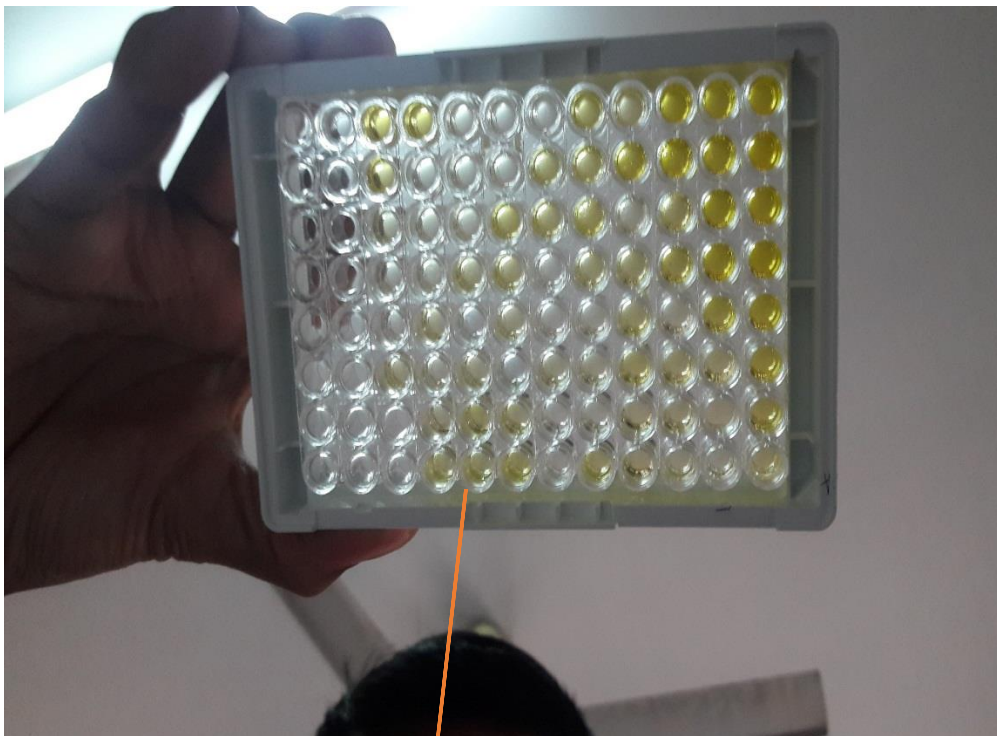

Fig 10: After Positive and Negative Control

CHAPTER 5

DISCUSSIONS

The present study was conducted to compare the seroprevalence of PPR in goat and sheep. In the present study, it was revealed that the seroprevalence was higher in goat (41.18%) than sheep (26.92%). Similar findings were reported by Afera *et al.* (2014) who reported that the overall seoprevalence rate was 46.53%. Shamaki *et al.* (1995) recorded prevalence rates of 40%, 40.6%, 7.7% and 38.4% from Plateau, Borno, Abia and Oyo States respectively. The present findings are higher than the findings of Rahman *et al.* (2015) and Luther *et al.* (2007) who reported that the overall seoprevalence rate was 31.5% and 24.7% respectively. Obi *et al.* (1983) reported seroprevalence of 47.2% of PPR neutralizing antibodies in sheep which is higher than the present findings. Bello (2013) also stated the overall prevalence of PPR virus antibodies in small ruminants in the study area to be 45.50%. Abraham *et al.* (2005) reported the overall seroprevalence of 9% in goats and 13% in sheep in different parts of Ethiopia which is lower than the present findings.

Female (45.71%) goat had higher prevalence of PPR than male (31.25%) but male sheep (28.57%) had slightly higher prevalence of PPR than female (26.32%). Similar results were reported by Bello *et al.* (2016) and found that PPR seroprevalence was higher in females 70.4% as compared to male 51.4% goat. The present study is closely related to the earlier study of Munir *et al.,* 2008 who found no significant difference between males and females, with respect to susceptibility in Pakistan. The present result is also in agreement with the earlier result of Shuaib, (2011), Abdalla *et al.* (2012) and Bello (2013) who indicated that females had a statistically greater seroprevalence rate (62.61%) than the male goats whose seroprevalence rate was 39.58%. Sarker and Islam (2011) also reported that the influence of sex on PPR outbreaks was found to be higher in male (28.52%) than female which is disagreed with the present findings. Another reports by Luther *et al.* (2007) and Khan *et al.* (2008) who reported a high seroprevalence in the females than in the males. Similar results were reported by Bello *et al.* (2016) and observed that PPR seroprevalence was insignificantly higher in male 50.4% as compared to female 47.4% in sheep.

According to the present study, it was revealed that black Bengal breed (45.16%) of goat was more prone to PPR than cross breed (35.0%) and cross breed (36.35%) sheep had higher prevalence than indigenous (20.0%). The present study is in line with the result of Sarker and Islam (2011) who observed that the disease affected mostly (27.13%) indigenous Black Bengal goats. This observation was supported by that of Mondal *et al.* (1995), where they found that the prevalence of PPR was higher in indigenous Black Bengal (27.13%) goats than Jamunapari (11.81%) and exotic breeds (9.68%). Higher incidence of PPR in indigenous Black Bengal goats may be due to immunosuppression and irregular vaccination compared to cross breeds (Mondal *et al.,* 1995). Similar results were found by (Islam *et al.,* 2012; Hasan, 2012). Munir *et al.* (2008) observed that there are insignificant differences between goat breeds but there are significant differences between sheep breeds.

In goat and sheep, the seroprevalence of PPR was higher in <1.5 years, and lower in >3 years age. The present result is similar with the result of Sarker and Islam (2011) who found that PPR was significantly higher in young (31.06%) compared to sucklers (13.14%) and adult (10.15%). This, to some extent agrees with the findings of Mahajan *et al.* (2012) who observed a significantly higher prevalence of PPR virus antibodies in animals aged above 12 months compared to those aged between 8-12 months. Khan *et al.* (2008) reported a higher seroprevalence of PPRV antibodies in the >2 years age group in both sheep and goats. However, Majiyagbe *et al.* (1992) showed in their study that PPR seroprevalence increases with age which is dissimilar with the present study. Dams infected with PPR virus can passively transfer maternal antibodies to their young ones. Although the maternal antibodies progressively decay, they remain above the protective threshold for up to 4-5 months after which PPR vulnerability increases with age (Abubakar *et al.,* 2009). This increased PPR susceptibility with age after five months in small ruminants may explain the relatively lower seroprevalence rates obtained in small ruminants aged between 6-12 months when compared with those aged between 13-24 months in this study. However, low seroprevalence of PPR was observed in those small ruminants aged above 24 months in this study.

During the present study, it was found that poor hyginic condition of goat and sheep had highest prevalence rate of PPR than medium and good condition respectively. There is no relevant data in the review of literature.

CHAPTER 6

SUMMARY AND CONCLUSION

Peste des petits ruminants (PPR) are an acute and highly contagious viral disease of small ruminants caused by a Morbillivirus under paramyxoviridae family. PPR is highly spreadable, transponder, and economic important disease in the world.The study was conducted at the Department of Pathology lab, Faculty of Veterinary Science, Hajee Mohammed Danesh of Science and Technology University (HSTU), Dinajpur for a period of 6 month since January to June 2019 for comparative study of seroprevalence of PPR in goat and sheep. A total number of 77 blood sample (51 from goat and 26 from sheep) were collected by jugular venipuncture method considering different parameters such as age, sex of animal and then kept for an hour at room temperature, after centrifugation serum was separated and stored at -20 ^{0}C until use. The result showed that the seroprevalence was higher in goat (41.18%) than sheep (26.92%). Based on sex, there was no significant variation ($P>0.05$), in goat and sheep and it was found that seroprevalence of PPR was higher female (45.71%) goat and male (28.57%) sheep.

Among breeds of goat and sheep, Black Bengle breed (45.16%) of goat and cross breed of sheep (36.35%) had the highest seroprevalence. According to the age group, it was observed that age had significant ($P<0.05$) effect on the prevalence of PPR in goat but had insignificant ($P>0.05$) effect on the prevalence of PPR in sheep. Seroprevalence was decreased in advanced of age in goat and sheep. According to the present study, it was revealed that prevalence of PPR was higher in poor hygienic condition of farm.

The present study should be concluded that the seroprevalence was comparatively higher in goat than sheep. Finally the following recommendations were suggested:

- To develop good management practices like hygienic condition, vaccination program to minimize the risk of the disease.
- To carry out further studies on isolation, morphology and characterization of peste des petits ruminants virus (PPRV).
- To study on pathological investigation to find out gross and microscopic lesions of the disease.

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