**Abstract**

The present study was conducted in the Panchlaish Upazilla Veterinary Hospital of Chittagong district during the period of July to September, 2013 to find out the clinicopathological findings and prevalence of Peste des petits ruminants (PPR) in goats, and to observe the response of PPR-goats to different treatments. During the course of study 202 goats were examined of which 98 were PPR positive. Diagnosis of a PPR case was made following the case history, physical examination, clinical examination, hematological examination, and post mortem examinations. Fecal samples from 40 randomly selected PPR-goats were examined for the presence of parasitic eggs. The results showes that the median age of PPR-goats was higher compared with the healthy ones. The median per-rectum temperature, heart and respiratory rates were also higher in the the PPR-postive goats. No significant difference was seen on its prevalence in goats belonging to Black Bengal or Jamunapari breeds. Its occurrence was also evenly seen in each of the three months - July, August and Septmber. But, prevalence of PPR was significantly higher in the non-vaccinated goats. Some hematological parameters – Hb (%), PCV, TEC (million/mm3), TLC ( thousand/mm3), ESR (0 hour), Lymphocyte (%), Monocyte (%), Eosinophil (%), Basophil (%) were lower in PPR-goats; in contrast, neutrophil count was higher in PPR- goats. Eggs of 5 major parasites namely, *Haemonchus sp*., *Trichuris sp*. *Trichostrongylus sp., Strongyloides sp.* and *Oesophagostomum sp*were commonly seen in the PPR-goats.The recovery rate from clinical signs was higher if the goats were treated with parenteral administration of oxytetracycline.

**Key words:**PPR, Clinicopathological features, Prevalence, Haematological parameters, faecal examination, Post-mortem, Breeds of goats.

**Chapter one**

**INTRODUCTION**

Goat rearing provides a significant level of supplying animal protein in the form of meat (20%), and one of the important areas of earning foreign exchange through the exportation of skin. But there are several diseases of goat especially PPR, which cause higher mortality and great economic losses. PPR is an exotic disease of goats in Bangladesh (Debnath, 1995; Islam et al, 2012).

Peste des petits ruminants (PPR) is an acute or sub-acute viral disease of small ruminants, particularly in goats in Bangladesh (Islam et al, 2012, 2001). Other names commonly used includes, pseudo rinderpest of small ruminants; pest of small ruminants; goat plague; pest of sheep and goat; stomatitis pneumoenteritis syndrome; contagious pustular stomatitis and pneumoenteritis complex. It is a highly contagious, infectious and fatal viral disease of domestic and small ruminants. The disease is characterized by high fever, necrotic stomatitis, catarrhal inflammation of the ocular and nasal mucosae, pneumonia, diarrhoea and death (Islam et al, 2012;Fraser, 1986). It is a rinderpest-like contagion of goats and sheep characterized by erosive stomatitis, enteritis, pneumonia.

In epidemic areas, morbidity rate has been estimated from 80% to 90% accompanied by mortality rate range from 50% to 80% (Debnath, 1995).

PPR is caused by rinderpest-like virus called Peste des petits ruminants (PPRV) which mostly affects sheep and goats although goats are often more severely affected than sheep .(Albina *et al*, 2012). However, some other animals particularly, captive wild ungulates from three families; gazelline (Dorcas gazelle), caprine (Nubian ibex and Laristan sheep), Hippotraginae (gemsbox) can be infected. The American white tailer deer (Odocoileus virgininanus) has been infected experimentally (Saliki, 2008)**.** 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 (EMPERS, 1999)

It has been reported that the Black Bengal goats were 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). Animals become more susceptible to the infection during rainy season as compared to dry season (Samad,1996). The incubation period is 4-5 days but may range between 6-10 days. (Khan *et al,* 2005).

Transmission occur by close contact,(Mulindwa et al, 2011) inhalation of aerosol produced by sneezing and coughing of infected animals, direct contact with ocular, nasal, oral secretions, feces, fomites such as bedding, water and feed troughs (Ozkul, 2002;Durojaiye, 1980; Durtnell, 1972).

Necropsy of diseased goats revealed congested and consolidated pneumonic lungs, generalized enlargement of lymphnodes accompanied with necrosis and congestion of some lymphnodes, atrophied congested spleen and haemorrhagic gastroenteritis (Khan *et al*, 2005). Hemorrhagic ulceration is marked in the ileocecal region, colon and rectum where they produce typical “zebras stripes” (Radostits et al, 2005).

Collection of blood sample from animal is important for clinical prediction of the health status of a animal. Haematological examination is necessary because blood is a representative of the body tissue. The blood picture changes with the advancement of the animal age and also varies with certain conditions such as stress, bacteria/viral infections and intoxication.

The most important factor that leads to severity of PPR and mortality in goats is the concomitant infection that targets particularly the respiratory system, developing pneumonia in the immune-compromised goats. The choice of antibiotics is linked to the identity of the bacterial agents frequently exacerbate PPR in goats.

Control of PPR is based on a concerted effort of vaccination and sanitary measures. At present homologous PPR vaccine has been practiced against PPR to make up strong immunity in Bangladesh. PPR acts as one of the prime production limiting factors in goats all over the world. Effective disease management might play an integral part in goat’s development program to optimize the productivity of these animals. Infectious and contagious diseases are important impediments to the economical rearing of small ruminants (Radostits *et al,* 2000)**.** Among these diseases, Peste des petits ruminants (PPR) has become a concern because it causes heavy economic losses. In this study efforts have been made to estimate the prevalence of PPR in different breeds of goats in the Sea-belt areas of Bangladesh which would he help understand its intensity in this particular area to advocate proper control strategies, although the diagnosis of PPR was based on classical clinical findings. Hematological parameters in PPR-goats might also be indicative of necessary supportive treatments. With the background mentioned above this study was undertaken with the following specific objectives :

1. To determine the overall prevalence of PPR in relation with breed, age, sex, season and immune status in a coastal area in Bangladesh
2. To delineate the clinical signs shown in PPR affected goats
3. To perform detail postmortem on goats referred to Panchlaish Upazilla veterinary hospital, and to list predominate changes in those goats based on which a clinical case of PPR can be better understand.
4. To compare the haematological pictures of the PPR affected goats with healthy ones
5. To know the parasite eggs found in faecal sample of PPR affected goats in the study area.

**Chapter Two**

**REVIEW OF LITERATURE**

Several research works have been done throughout the world for measures the prevalence of PPR. Some research works have been done in Bangladesh and important research findings in these connections are reviewed in this section.

**History :-**

PPR was first described in Côte d’Ivoire (Gargadennec & Lalanne, 1942), but it occurs in most African countries from North Africa to Tanzania (Kwiatek et al, 2011; Swai et al, 2009; Lefevre & Diallo, 1990), and in nearly all Middle Eastern countries to Turkey (Ozkul et al, 2002; Perl et al, 1994; Lefevre et al, 1991; Taylor et al, 1990; Furley et al, 1987). PPR is also widespread in countries from a central Asia to South and South-East Asia. (Banyard et al, 2010; Wang et al, 2009; Shaila et al, 1989).

**Etiology of the Disease:**

The etiological agent, Peste des Petits Ruminants virus (PPRV) has been classified under Order Mononegavirales, family Paramyxoviridae and Genus Morbillivirus (Murphy *et al,* 1999). Like other Morbilliviruses, PPRV is fragile and it can not survive for long time outside the host. Its half life has been estimated to be 2.2 minutes at 560 C and 3.3 hours at 370C (Rossiter and Taylor, 1994). The virus is closely related to Rinderpest virus (RPV), another member of Morbillvirus genus, which causes similar disease in large ruminants (Anderson *et al,* 1990; Couacy-Hymann et al, 1995). The virus is also serologically related to Measles and Canine distemper virus (Gibbs et al,1979). Antibodies to PPRV and rinderpest are cross-protective, and vaccination for rinderpest can mask the presence of peste des petits ruminants. l

**Incidence of PPRV:-**

Higher incidence of PPR infection in goats than sheep , Sumit et al ., (2013) was reported that age wise analysis of data revealed higher incidence of PPR in young ones of both species than adults. The findings of present study are in agreement with other who also reported that the incidence of disease is more in young ones than adults and also the severity is more in young ones than adults. However the possible reason for higher incidence of PPR in young ones has been reported to be due to sub-clinical load of coccidial infection causing immune-suppressive effect or E.coli infection causing fimbrial adhesion with intestinal mucosa,thus enhancing the effect of PPR virus.

Environmental factors influence disease occurrence. Hegde et al., (2009) showed that incidences were highest during the rainy season and in the dry agro-climatic zones. The dusty and dry winds that characterize winter season of the year has been shown to enhance the spread of PPR (Obi, 1983).Aruni *et al.,* (1998)observed more than ten outbreaks of PPR in goats from Tamil Nadu. They observed that kids were susceptible than adult. An outbreak of PPRV in approximately 100 goats was diagnosed in Rawalpindi city, Pakistan in June 1997 by (Hussain *et al,* 1998) **.**The outbreaks of 74.13% morbidity and 54.83% mortality in Black Bengal goats in Bangladesh .(Islam *et al,* 2001; Das *et al,* 2007).

**Host Range of PPRV :**

Cattle, buffaloes, camels and pigs can also be infected but there is little or no evidence of disease associated with their infection. PPRV antigen has been detected in an outbreak of respiratory disease in camel and sick domestic buffaloes (Taylor *et al*, 1990; Scott, 2000; Abraham *et al*, 2005)Antelope and other small wild ruminant species can also be severely affected (Abu Elzein *et al*, 2004). A case of clinical disease has been reported in wildlife resulting in deaths of gazelles (Gazella dorcus), ibex (Capra ibex nubiana), gemsbok (Oryx gazelle) and Laristan sheep (Ovis orientalis laristanica). American white tailed deer (Odocoileus virginianus) can be infected experimentally (Hamdy and Dardiri, 1976). Changes in the allopatric speciation of lineages suggest that, when competing with indigenous strains, some strains have great power to spread because they are better adapted to the natural host and/or by switching to a new host.

**Transmission :-**

The transmission of virus requires close contact between susceptible and infected animals in the febrile stage (Braide, 1981). The discharge from eyes, nose, mouth and the loose feces contain large amount of the virus. Fine infected droplets are released into the air from these secretions and excreations, particularly when infected animals cough and sneeze (Bundza *et al*, 1988; Taylor, 1984). Animals in close contact inhale the droplets and are likely to become infected.

**Pathogenesis :**

The route of infection is respiratory and is spread by airborne droplets and it localizes first replicating in the pharyngeal and mandibular lymph nodes as well as tonsil. Viremia may develop 2-3 days after infection and 1-2 days before the first clinical sign appears. Subsequently viremia results in dissemination of the virus to spleen, bone marrow and mucosa of the gastro-intestinal tract and the respiratory system (Scott, 1981).PPRV has an established lymphatic and epithelial tropism. The signaling lymphocyte activation molecule (SLAM) is well recognized as the universal receptor for morbillivirus infection of immune cells, and this receptor tropism results in the leukopenia observed during infection ( Bao *et al*, 2012). Consequently, it induces the most severe lesions in lymphoid and epithelial tissues.

**Clinical Findings:-**

The predominant signs in natural infection of PPR includes dullness, high fever (41c or 106 F), anorexia, mucopurulent nasal discharge, eye discharge, lacrimation, congestion of mucous membrane, severe diarrhea, along with soiled perineal region with faeces, In addition a few animals also exhibited erosive rhinitis and dyspnoea. Erosive and ulcerative lesions on the tongue as well as the lips were also observed. (Islam et al, 2012; Harshad et al, 2011; Mirza et al, 2008; Das et al, 2007; Kwaitek et al, 2007; Anil et al, 2007; Ahmed et al, 2005; Radostits et al, 2000).

**Post mortem findings :**

It was reported that the carcass of an affected animal is usually emaciated, the hindquarters soiled with soft/watery faeces and the eyeballs sunken. The eyes and nose contain dried-up discharges (Chauhan *et al,* 2009)**.** Lips may be swollen; erosions and possibly scabs or nodules in late cases. The nasal cavity is congested (reddened) lining with clear or creamy yellow exudates and erosions. Oral mucosa was congested in almost all cases with presence of minor erosions on lips and tongue in about 60% cases (Kumar *et al*, 2004). There may be dry with erosions on the gums, soft and hard palates, tongue and cheeks and into the esophagus. The lung is dark red or purple with areas firm to the touch, mainly in the anterior and cardiac lobes (evidence of pneumonia). Mesenteric lymph nodes were swollen and oedematous in more than 80% cases (Kumar *et al,* 2004). Abomasum congested with lining haemorrhages. The pathology caused by PPR is dominated by necrotizing and ulcerative lesions in the mouth and the gastro-intestinal tract (Roeder et al, 1994). The rumen, reticulum and omasum rarely exhibit lesions. (Aruni et al, 98; Pawaiya, 2004; Kumar et al, 2004).

Occasionally, there may be erosions on the pillars of the rumen. Lesions in the small intestine are generally moderate, being limited to small streaks of hemorrhages. 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. The haemorrhages recorded on the luminal wall of gall bladder in the present outbreak in some cases has not been reported to the best of our knowledge but presence of thick granular bile had been reported (Shaila et al, 1996). Though the types of lesions were more or less similar in all the animals but there was variation in the severity and involvement of the organs.

**Prevalance:**

**Breed**

The Prevalence of PPR disease was higher in Black Bengal goat (54.93%) than in Jamunapari goat (31.78%). (Islam et al, 2012; Samad, 2000).

**Age**

The prevalence of PPR was maximum 63.33% at age category of 7-12 months, in compare with 44.68%, 41.87%, 45.45% at age category of ≤ 6 months, 13-18 months and ≥ 19 months respectively. (Islam et al, 2012; Radostits, 2000).

**Immune Status**

Non-vaccinated goats were more susceptible (66.40%) to PPR infection than vaccinated goats (19.56%). (Islam et al, 2012; Gibbs et al, 1979).

The seroprevalence of PPR has been reported to be 36.0% in sheep, 49.17% in goats and 19.05% in cattle from Bangladesh ( Razzaque *et al*, 2004 ). Whereas Harshad et al., (2011) was recorded sero-prevalance for sheeps and goats was 64.10% and 60.00% ,respectively.

The overall sero-prevalence for female was 52.32% while that for males was 47.68%. (Iman et al, 2012).

**Morbidity and Mortality :-**

Because of high susceptibility of Black Bengal Goat to PPR virus, the morbidity rate has been estimated to be 100% accompanied by mortality rate of 50-90% ( Khan et al, 2005). Samad (2008) was reported that in unprotected animals the morbidity can be up to 100% and mortality may be 20 to 90% and in severe outbreaks with 100% case fatality particularly in goats. The outbreaks of PPR caused 74.13% morbidity and 54.83% mortality in Black Bengal goats in Bangladesh. (Das et al, 2007; Islam et al, 2001). In epidemic areas, morbidity rate has been estimated from 80% to 90% accompanied by mortality rate range from 50% to 80%. (Debnath, 1995). Kashem et al., (2011) reported that major causes of kids mortality (25.00%) was also due to PPR.

Parental (I/M) Oxytetracycline was more effective (64%) than parental (I/M) Sulphadimidine (44%) along with symptomatic treatment . PPR causes higher mortali ty and heavy economic losses in every year which may be reduced substantially by proper vaccination and other managemental approaches (Islam et al., 2012) .Taylor et al., (1984) reported that mortality rates may be decreased by the use of drugs that control the bacterial complications especially oxytetracycline and Chlortetracycline are recommended to prevent secondary pulmonary infections.

**Haematological analysis**

Khan et al., (2005) was reported that haematological profiles are important indices of the physiological state of the individual. The ability to interpret the state of blood profile in normal and in diseased condition is among many primary objectives of haematological studies. Definite changes occur in the profile of blood cells throughout life due to physiological and pathological condition. **(**Fasuyi, 2007).

Blood with its myriad of constituents provides a valuable medium both for clinical

investigations and nutritional evaluation of the organism. However the ingestion of numerous dietary components has measurable effects on blood constituents. (Aderemi, 2004; Church, 1984; Kerr, 1982).

Babatunde et al., (1992) was also reported that blood sampling for the assay of haematological traits are frequently employed in nutritional studies. Changes in the constituent components of blood when compared to normal values could be used to interpret the metabolic state of the animal.

**Haematological changes in PPRV affected goats:**

Kaneko et al., (1997) stated that, haematological and blood biochemical measurements may vary depending on factors such as sex, age, weather, stress, season, pregnancy status and physical exercise.The severe dehydration in the affected animals was evidenced by increased viscosity and specific gravity which led to polycythaemia (Bhikane et al, 1997). Severe leucopoenia could have been due to the inhibition of peripheral blood lymphocytes proliferation by PPR virus (Heaney et al, 2002). A marked lymphocytopoenia, monocytopoenia, neutrophilia and eosinopoenia in present investigation could have been due to the combined effect of virus infection and stress as evidenced by elevated cortisol levels (Kataria and Kataria, 2004).

**Concomitant infection with PPR :**

Obi *et al*., (1983) showed that, the most significant bacteria associated with PPR infected goats were *Pasteurella haemolytica, Klebsiella* sp., *Pseudomonas aeruginosa* and *Staphylococus pyogenes* from the lungs, *Salmonella sp.* and *E. coli* from the faeces, *Moraxella bovis* from the eyes and *Staphylococcus pyogenes* from the oral cavity. Pneumonia is usually a very obviously presented sign in PPR. Pneumonic pasteurellosis is a purely respiratory disease of sheep and goats caused by the bacterium *Pasteurella haemolytica.*

**Faecal Examination :**

The prevalence of parasitic infestation depends on ecology, geographical and climatic condition prevailing in Bangladesh (Hossain et al, 2004). Gastrointestinal nematodes (*Haemonchus, Trichostrogylus* and *Strongylus)* causes impaired digestion and also affect the absorption of minerals particularly the Calcium and Phosphorus (Speedy, 1992). Jugessur et al, 1998 conducted a survey and reported that 35.3% of the infected goats had above 300 epg in faeces.

Hassan et al., (2010) was observed that the overall prevalence of gastrointestinal and ectoparasites were 63 and 57.72% (N= 317), respectively. Of all the endoparasites *Strongyloides* spp. was 51.74%, *Haemonchus* spp. was 41.79%, *Paramphistomum* spp. was 39.30%, *Trichostrongylus* spp. was 36.32%, *Oesophagostomum s*pp., *Bunostomum* spp., *Fasciola* sp. and *Cooperia* spp. remained between 10.95 and 12.94% and the rest of the species (*Capillaria* spp., Moniezia spp.and *Chabertia* spp.) were just in between 1.5 and 2.0%. The percentage of gastrointestinal parasitic load was variable.

Hassan et al., (2010) was observed that the older and heavier goats were more prone to infection (52.24%) than younger and lighter goats (47.76%) (p < 0.05) . The infestation rate is 1.61 times higher in older and heavier goats than younger and lighter goats.

**Differential Diagnosis:**

The disease must be differentially diagnosed from Foot and Mouth disease, Heart water, Bluetongue, Contagious ecthyma, Pneumonic Pasteurellosis, Contagious caprine pleuropneumonia, Nairobi sheep disease, Coccidiosis, Plant and Mineral poisoning etc. (Radostits et al, 2000; Appel et al, 1981).

**Treatment of PPR :**

There is no specific treatment for PPR, however hyperimmune PPR serum produced in goats reverses the disease process if administered at the onset of fever . The affected animals were given antibiotics to control secondary bacterial infections along with anti-inflammatory drugs. Specifically, oxytetracycline and chlortetracycline are recommended to prevent secondary pulmonary infections (OIE, 2000). Sil et al.,(2006) reported that, the use of combined antibiotic hyper immune serum therapy (ACHST) for PPR helpful to overcome the condition. For diarrhoeal conditions, they suggested that 10 ml hyperimmune serum intravenous route per animal three doses every 3 days interval. Long acting Oxytetracycline tabs 1ml/10 kg body weight 2nd dose after 72 hours of 1st dose. A mixture of Oxytetracycline tabs and Metranidiozol (1:1) oral doses twice daily until diarrhea subsides.

**Prevention and control of PPR :**

Control of PPR outbreaks depends on movement control combined with the use of vaccine. Although vaccination against PPR is being practiced in Bangladesh and other countries, PPR is still causing major constraints to the productivity of small ruminants. Therefore, development of effective prophylactic procedures along with rapid, specific and sensitive diagnostic methods is extremely important for effective control of the disease. Singh et al., (2009) said that, the availability of an effective vaccine, accurate diagnostic tests for PPR and an experienced infrastructure prompt us to propose a national project for a Peste des Petits Ruminants eradication programme on the lines of National Project on Rinderpest Eradication. To control peste des petits ruminants (PPR) in Bangladesh a live attenuated conventional PPR vaccine was developed by Bangladesh Livestock Research Institute (BLRI) and currently being used in the country (Rahman et al, 2011). This would greatly enhance the prospects of PPR eradication not only on a national level but also from the Asian continent, alleviate poverty and, in turn, contribute to the national economy.

**Chapter Three**

**MATERIALS AND METHODS**

**Study area:**

The study was conducted at Panchlaish Thana under the district of Chittagong.

**Duration of the study:**

The duration of the study was from July to September, 2012 during my DVM internship placement in the Upazilla Veterinary Hospital, Panchlaish .

**Case definition:**

Diagnosis of a PPR case was made by means of anamnesis and clinical signs. A PPR case was initially suspected if an animal showed signs of fever in the initial stage followed by pneumoenteritis evidenced by nasal and ocular discharges, conjunctivitis, erosion in oral mucosa, dyspnoea, diarrhea, dehydration and generalized weakness. The degree of dehydration was estimated by conventional skin fold test. All the clinical signs were properly noted in the record sheet. Sometimes the tentative diagnosis was supported with hematological findings and post mortem examinations.

**History**

Information about the diseases and clinical signs exhibited by the animal during illness

were recorded in detail provided by the owner. Data were recorded by interviewing

the owners regarding the breed/sex/age of the animals.

**Population and tools used for data collection**

The study was conducted on natural PPR infected goats of various age, sex and breed that were brought to the hospital over the study period. A total of 202 goats were registered to Upazila Veterinary Hospital, Panchlaish for treatments of which 98 were diagnosed with PPR

Close inspection was performed properly in order to observe the presenting signs.A case of PPR was diagnosed on the combination of following clinical signs: 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 (1040 – 1060 F), remaining high for 5-8 days then returned to normal; anorexia, severe dehydration and emaciation followed by hypothermia; the mucous membrane of the mouth and eyes became 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; feces 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.

In diarrheal case, dehydration was measured by skin fold test.

**Post-mortem Findings:**

Three dead animals were subjected for postmortem examination. A PPR case was tentatively diagnosed if postmortem examinations reveal the presence of changes that included severely dehydrated carcass, soiled hind quartet matted with fluid feces, exudates crusts around the eyes, lips and nose, discrete or extensive areas of erosion, necrosis and ulceration in the oral mucosa, pharynx and upper esophagus, mucopurulent exudates extending from the nasal opening to the larynx, hyperemic trachea and bronchi containing froth, consolidated lungs, congested and haemorrhagic abomasum and small intestine, haemorrhagic ulceration in large intestine particularly on the ileocecal region, colon, rectum producing the “Zebra-stripe” lesions and enlarged regional lymph nodes.

**Medication :**

For observing the treatment efficacy the PPR goats were divided into three groups:

**Group I:** treated with Diadin ( Sulphadimidine-Na) + Antihista vet (Pheneramine meleate) + Renalyte(ORS);

**Group II:** treated with Renamycin-100 (Oxytetracycline) + Antihista vet (Pheneramine meleate) + Renalyte (ORS) and

**Group III**: treaed with orall or gut acting sulphonamide + Renalyte (ORS).

A response to a treatment was measured by recovery from the clinical illness.

**Haematological examinations:**

Approximately 3 ml of blood was collected aseptically from the jugular vein of a PPR-goat and then transferred to a sterile vials containing Na EDTA at 2 mg/ ml to assess certain hematological parameters. Haematological analysis of a sample was performed on an autohemolyzer within 24 hours of collection of a blood sample, in the Department of Physiology,Biochemistry & Pharmacology, CVASU. In total blood was collected from 20 PPR-goats whose hematological parameters were compared with the values of those from 20 healthy goats. The hematological parameters assessed included Total Erythrocyte Count (TEC), Total Leukocyte Count (TLC), Differential Leukocyte Count (DLC), Hemoglobin Content (Hb), Packed Cell Volume (PCV) and Erythrocyte Sedimentation Rate (ESR). TEC and TLC were determined by Haemocytometer method and Hb level was assessed by Hellige-Shali method. All differential counts of leukocytes were from prepared thin blood smear, stained by Wright’s method. A stained blood was examined under a microscope and different types of leukocytes were counted applying tally method.

**Coproscopic examinations:**

About 5 g of fresh feacal sample from each PPR goat was directly collected from the rectum with aseptic condition (using hand gloves or sometimes thermometer). Collected samples were kept in vials containing 2% formalin. Each vial was marked with the unique identification number and basic demographic information (age, sex and body weight). The vials were kept in a refrigerator at 4°C temperature until further examination. The direct smear, flotation and sedimentation methods were performed to screen parasitic eggs in feces of PPR-goats. The age of a goat was determined on the basis of dentition, sex was detected by examining the external genitalia.

**Coproscopic examination - Direct smear method**

A few drops of water plus an equivalent amount of faeces were mixed on a glass slide. The slide was tilted which allowed the lighter eggs to flew away from the debris. A cover slip was placed on the fluid and the preparation (slide with cover slip) was examined under a microscope. Detection of most eggs or larvae was possible by direct Smear Method.

**Coproscopic examination – Floatation method**

About 2g feces from a PPR-goat was added to floatation solution and following thorough mixing the suspension was poured into a test tube and more floatation solution was added to fill the tube to the top. A cover slip was placed on the top of the surface of the liquid and the tube and then a coverslip was left standing for 10 to 15 minutes. Then the cover slip was removed vertically and was placed on a slide and examined under a microscope.

**Coproscopic examination - Sedimentation method**

About 3 g feces was homogenized with water, and passed the suspension through a coarse mesh sieve. The material was thoroughly washed and retained on this screen using a fine water jet, and discarded the debris. The filtrate was transferred to a conical flask, allowed to strand for 2 minutes; the supernatant was removed and the remainder was transferred to a flat-bottomed tube. After sedimentation for a further 2 minutes the super mutant was again drawn off, and a few drops of 5% methylene blue was added and the sediment was examined under a microscope.

**Statistical analysis**

All data were collated and stored into a spreadsheet (Microsoft office excel-2007, USA). The Excel spread sheet was imported to SAS version 9.2 for further analysis. Categorical variables; sex, breed, vaccination status, season were tested with Chi square test to estimate the effect of the variables on the outcome (PPR) and continuous variables; age, temperature, heart rate, respiratory rate were tested with t-test where the null hypotheis was that there was no difference in mean between positive and negative groups of animals. Descriptive analysis was done by means of histogram and boxplot. An association was regarded as significant if the p value was <0.05.