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**ADDIS ABABA UNIVERSITY
FACULTY OF VETERINARY MEDICINE**

**SEROPREVALENCE OF PEST DES PETITIS RUMINANTS AND ITS
ASSOCIATIONS WITH POTENTIAL RISK FACTORS IN DOMESTICATED
SMALL RUMINANTS, CATTLE AND CAMEL IN JIJIGA AND SHINILLE
ZONE OF SOMALI REGION, ETHIOPIA**

BY

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A thesis submitted to the school of Graduate Studies of Addis Ababa University in the
partial fulfillment of the requirements for the degree of Master of
Veterinary Science in Tropical Veterinary Epidemiology

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LIST OF ABBREVIATION

AGID	Agar gel immunodiffusion test
C-ELISA	Competitive Enzyme Linked Immuno-Sorbent Assay
CFSPH	Center for Food Security and Public Health
CIEP	Counter Immunoelectrophoresis
CIRAD-BIOS	Centre de Coopération Internationale en Recherche Agronomique pour le Développement- Biological Systems Department
ELISA	Enzyme Linked Immuno Sorbent Assay
FVM	Faculty of Veterinary Medicine (Debrezeit)
GDP	Gross Domestic Product
H₂O₂	Hydrogen Peroxide
Kb	Kilobase
LCNRDB	Livestock, Crop and Natural Resource Development Bureau
MAbs	Monoclonal antibodies
MoARD	Ministry of Agriculture and Rural Development
NVI	National Veterinary Institute
OD	Optical densities
OIE	Office International des Epizooties
ORF	Open Reading Frame
PA	Peasant Association
PBS	Phosphate Buffer Saline
PCR	Polymerase Chain Reaction
PI	Percentage inhibition
PPR	Peste des Petits Ruminants
PPRV	Peste des Petits Ruminants Virus
RNA	Ribonucleic acid
RPV	Rinderpest virus
SNRS	Somali National Regional State
SPSS	Statistical Programme for Social Studies
TCID	Tissue Culture Infective Dose
TCRPV	Tissue Culture Rinderpest Virus
UK	United Kingdom

VNT	Virus Neutrization Test
Woreda	Administrative/geographic unit equivalent to district
H.H	House holds

ABSTRACT

Cross-sectional study was conducted in Jijiga and Shinille Zones of Somali Regional State from October 2008 to April 2009 to determine seroprevalence and associated risk factors. A multistage sampling was used in pastoral and agropastoral system. A total of (n=616) goats, (n=202) sheep, (n=110) cattle and (n=94) camel sera were collected for serological study. questionnaire survey was conducted in pastoral and agro-pastoral system. The seroprevalence of PPR in goats and sheep was 25.6% and 14.4% in the study area respectively. The seroprevalence was lower in camels and cattle, which was 8.5% and 6.63% respectively. Goats were found to be more susceptible than sheep, which was found to be statistically significant ($p=0.000$). However no significant difference was observed between cattle and camel ($P=0.558$). Seroprevalence of goats between Shinille Zone (pastoral) and Jijiga Zone (agro-pastoral) production system showed 44.09% and 18.9% in goats, 30.01% and 5.42 in sheep respectively. Sero prevalence increased from 21.7% to 28.9% in goats and from 8.1% to 20.4% in sheep from young to adults age respectively. There was significant difference between age groups in seroprevalence of both goats ($p=0.04$) and of sheep (0.013). In case of cattle ($p=0.062$) and camel ($p=0.249$) statistically no significance difference was observed between age groups in seroprevalence. Regarding sex, 26.4%, and 25.5% was found in goats ($p=0.851$), 15.6% and 14% in sheep's 8% and 5% ($p=0.795$) in cattle ($p=0.488$) and 8.2% and 9.5% in camel ($p=0.095$) in male and female respectively. There was statistically no significant difference between sexes in all species. According to the respondent the number of goats affected by CCPP (Sunbub) and PPR (*Kollere*) was 32.8%, 21.4% and 25.7%, 14.3% in Shinille and Jijiga Zone respectively. However more goats are affected by GIT parasite in Jijiga (40%) than Shinille zone (21.4%). Regarding susceptibility by age 64.28% and 60.7% of the respondent replied that young goats and sheep were more susceptible than adult animals. However, no response (0%) was made indicating susceptibility of goats and sheep less than 6 months of age.

Key words: goat, sheep, cattle, camel, , cELISA, Peste des petits ruminants, Seroprevalence, Risk factors, Jijiga Zone, Shinille Zone.

1. INTRODUCTION

Ethiopia has 44 million cattle, 23 million goats, 23 million sheep, 6 million equines, 2 million camels and 42 million chicken playing crucial roles in the livelihoods of the majority of rural people (Leforban, 2005). They provide milk, meat, manure, hides and skin, animal power and financial income. This sector is also important to the national economy, contributing 16 % of the total GDP, one-third of agricultural GDP, and eight percent of export earnings (Afewerk, 2000). An improvement in this sector, therefore, has the potential to contribute significantly to national income and to the welfare of the majority of rural families.

Given the large livestock population and distribution in the country and poor supply of veterinary services, infectious and parasitic diseases consequently cause death and debilitation to a significant number of animals. Animal health problems are generally exacerbated by drought, concentration of livestock at watering points and dry grazing grounds, combined with reduced resistance, intensifies the spread of communicable and parasitic diseases, which often cause higher losses than the forage or water shortages (The World Bank, 2001).

About 8-10% of cattle herd, and 14-16% and 11-13% of the national sheep and goats per annum are estimated to die from diseases. This is equivalent to an average live weight at death of 70 kg for cattle and 6 kg per sheep or goat, with an annual loss estimated at Ethiopian Birr 550 million (MOA, 1997). Indirect effect of diseases on live weight gain and production probably leads to an economic loss, which is greater than that from mortality. Altogether, economic losses from animal diseases were estimated at between Ethiopian Birr 1.5 and 2.5 billion (MOA, 1997), which accounts for 30 to 50% of total production value.

In most of the developing countries, small ruminant farming allows to cover the basic necessities of the majority of peasants. Sheep and goats are an essential source of subsistence. They determine the survival of the poor people in these countries. Unfortunately, in parts of Asia and Africa, the production of small ruminants is threatened by a highly contagious and fatal disease, Peste des Petits Ruminants (PPR). PPR is a transboundary disease and makes part of the group of economically important animal diseases in which outbreaks should be notified to the World

Organization for Animal Health (Office International des Epizooties). Because of the high importance of sheep and goats for the poor farmers, the control of diseases, which have a negative impact on their productions, is a major goal for programmes aimed at poverty alleviation (Gebreegziabhe, 2006).

Peste des petits ruminants (PPR) is an economically important viral disease of goats and sheeps, first described by Gargadennec and Lalane (1942) from Ivory Coast in West Africa. The disease is highly contagious causing varying degrees of morbidity and mortality in susceptible animals (Radostits *et al.*, 2000). 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 seldomly fatal but usually occurs as a subclinical or inapparent infection opening the door for other infections such as Pasteurellosis (Lefèvre and Diallo, 1990). The disease is characterized by high fever, ocular and nasal discharge, pneumonia, necrosis and ulceration of the mucous membrane and inflammation of gastro-intestinal tract leading to severe diarrhoea (Gibbs *et al.*, 1979). Morbidity and mortality rates can be as high as 100 and 90 per cent, respectively (Abu-Elzien *et al.*, 1990).

The causative agent of this economically important disease of small ruminants is a Morbillivirus, the Peste des petits ruminant's virus (PPRV), under the family Paramyxoviridae of order Mononegavirales (Murphy *et al.*, 1999). The virus is closely related to rinderpest virus (RPV), another member of Morbillivirus genus, which causes similar disease in large ruminants (Anderson *et al.*, 1990; Couacy-Hyman *et al.*, 1995). The virus is also serologically related to measles and canine distemper virus (Gibbs *et al.*, 1979). Like all members of the family, the PPR virus is an enveloped pleomorphic particle of size between 150 and 390 nm (Durojaiye *et al.*, 1985) containing non-segmented single stranded RNA genome of negative polarity. The genome of attenuated vaccine strain of PPRV (Nigeria 75/1) has entirely been sequenced and the physical map of the genome is the same as that of other morbilliviruses (Rima *et al.*, 1986; Diallo *et al.*, 1990).

In Ethiopia PPR was first suspected in goats in Afar region based on clinical signs in 1977 (Pegram and Tareke, 1981) and the presence of the virus was confirmed in 1990 from goats

purchased from southern Ethiopia for live export at holding site near Addis Ababa (Abraham *et al.*, 1991). However a comprehensive quantification of the (economic) importance of PPR, has never been attempted, mainly due to a paucity of reliable data on such important factors as the distribution and numbers of animals at risk, insufficient knowledge of the ethics of the disease on livestock production, and the difficulty in assessing the quantification of risk factors on livestock and their products in pastoral and mixed crop/livestock subsistence production systems.

After considering the aforementioned points, the present study was planned with the following objectives.

- To study the seroprevalence of peste des petits ruminant's virus (PPRV) in small ruminants, cattle and camel in Jijiga and Shinille Zone of Somali region
- To assess potential risk factors associated with the disease in the area

2. LITERATURE REVIEW

2.1. The disease

The natural disease affects mainly goats and sheep, but it is usually more severe in goat's where, it causes severe morbidity and mortality and is only occasionally severe in sheep (Raghavendra *et al.*, 2000). It is generally admitted that cattle can only be infected subclinically. However, it is possible that cattle in poor health may develop lesions following PPRV infection, clinical signs of which would be ascribed to rinderpest. Nonetheless, PPRV was isolated from an outbreak of Rinderpest like disease in Indian buffaloes in 1995 (Govindrajan *et al.*, 1997). PPRV was also suspected to be involved in the epizootic disease that affected single humped camels in Ethiopia in 1995–1996 (Roger *et al.*, 2000, Roger *et al.*, 2001). PPRV antigen and PPRV nucleic acid were detected in some pathological samples collected during that outbreak, but no live virus was isolated. Both experimental and natural infections of PPRV have been reported in wild ruminants. Experimentally, subclinical infection in pigs has also been demonstrated (Tiwari, 2004).

2.2. Etiology

The causative agent of this economically important disease of small ruminants is a Morbillivirus, the Peste des Petits Ruminants Virus (PPRV), under the family Paramyxoviridae of order Mononegavirales (Murphy *et al.*, 1999). The virus is closely related to Rinderpest virus (RPV), another member of Morbillivirus genus, which causes similar disease in large ruminants (Anderson *et al.*, 1990; Couacy-Hyman *et al.*, 1995). The virus is also serologically related to measles and canine distemper virus (Gibbs *et al.*, 1979). A varying degree of cross protection *in vivo* and serological relationship is known to exist between PPR and RPV. Further, the PPR infection in sheep and goats is known to sero-convert and protect in-contact bovines from natural infection, and also may interfere in tissue culture rinderpest virus (TCRPV) vaccination response (Sudarshan *et al.*, 1995). PPRV isolates can be grouped into four distinct lineages on the basis of partial sequence analysis of the fusion (F) protein gene. Lineages 1 and 2 are found exclusively in West Africa while lineage 3 has been found in eastern Africa (Ethiopia), Arabia (Oman, Yemen)

and in southern India The fourth lineage is confined exclusively to the Middle East, Arabia and the Indian sub-continent (Shaila *et al.*, 1996).

2.2.1. Virus structure and genome organization

When viewed through electronmicroscope, morbilliviruses display the typical structure of Paramyxoviridae: a pleomorphic particle with a lipid envelope, which encloses a helical nucleocapsid (Gibbs *et al.*, 1979). The nucleocapsids have a characteristic herringbone appearance. Morbilliviruses are linear, non-segmented, single stranded, negative sense RNA viruses with genomes approximately 15–16 kb in size and 200 nm diameter. The genome is divided into six transcriptional units encoding two non structural (V and C protein) and six structural proteins, nucleocapsid protein, phosphoprotein matrix protein, fusion protein, haemagglutinin and large protein (Gebre-Egziabher, 2006).

2.3. Epidemiology

2.3.1. Transmission

Transmission requires close contact between infected animals in the febrile stage and susceptible animals 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. 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 solvents (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 (Abraham, 2005).

2.3.2. Host Range and pathogenicity

PPR is mainly a disease of small ruminants. It affects goats and sheep. PPR virus exhibits different levels of virulence between sheep and goats. Goats are severely affected while sheep generally undergo a mild form (Lefèvre and Diallo, 1990). Although infected, sheep rarely suffer clinical disease (Roeder *et al.*, 1994). An outbreak with a high mortality in sheep was reported by Taylor (1984) who hypothesised that sheep possessed an innate resistance to the clinical effects of disease, but occasional field strains could overcome this resistance and produce high mortality. Breed may affect the outcome of PPR virus infection and its epidemiology, the Guinean breeds (West African dwarf, Iogoon, kindi and Djallonke) are known to be highly susceptible (Lefèvre and Diallo, 1990). This is in agreement with the finding that British breed exhibited severe clinical reaction when infected experimentally while the Sudanese breeds failed to develop a characteristic clinical response (El Hag and Taylor, 1984). A more recent observation detected variations in breed susceptibility within goats in West Africa. The acute form of the disease was observed in WAD goats while WALL breed developed only mild form (Diop *et al.*, 2005).

2.3.3. Geographical distribution

PPR has comparatively a shorter history, which only dates back to 1940, when it was first described by Gargadennec and Lalane (1942) in Ivory Coast of West Africa. For a long time, its existence was associated with West African countries. After development of specific diagnostic tools in late 1980s onwards, our understanding of the geographical distribution of PPR has grown very quickly (Diallo *et al.*, 1995) and recent data indicates the activity of PPRV in all countries of Africa lying between Sahara and the Equator, Arabian peninsula and the Middle East with extension to Turkey, Pakistan, India, Bangladesh and Nepal (figure-1) (Shaila *et al.*, 1996; Dhar *et al.*, 2002; Taylor *et al.*, 2001). It has also been reported in Sudan (Ali and Taylor, 1984) Kenya, Uganda (Wamwayi *et al.*, 1995) and also in Ethiopia (Roeder *et al.*, 1994). Table 1 depicts the first report of PPR from different countries CFSPH (2004).



Figure 1: Geographical distribution of PPR,



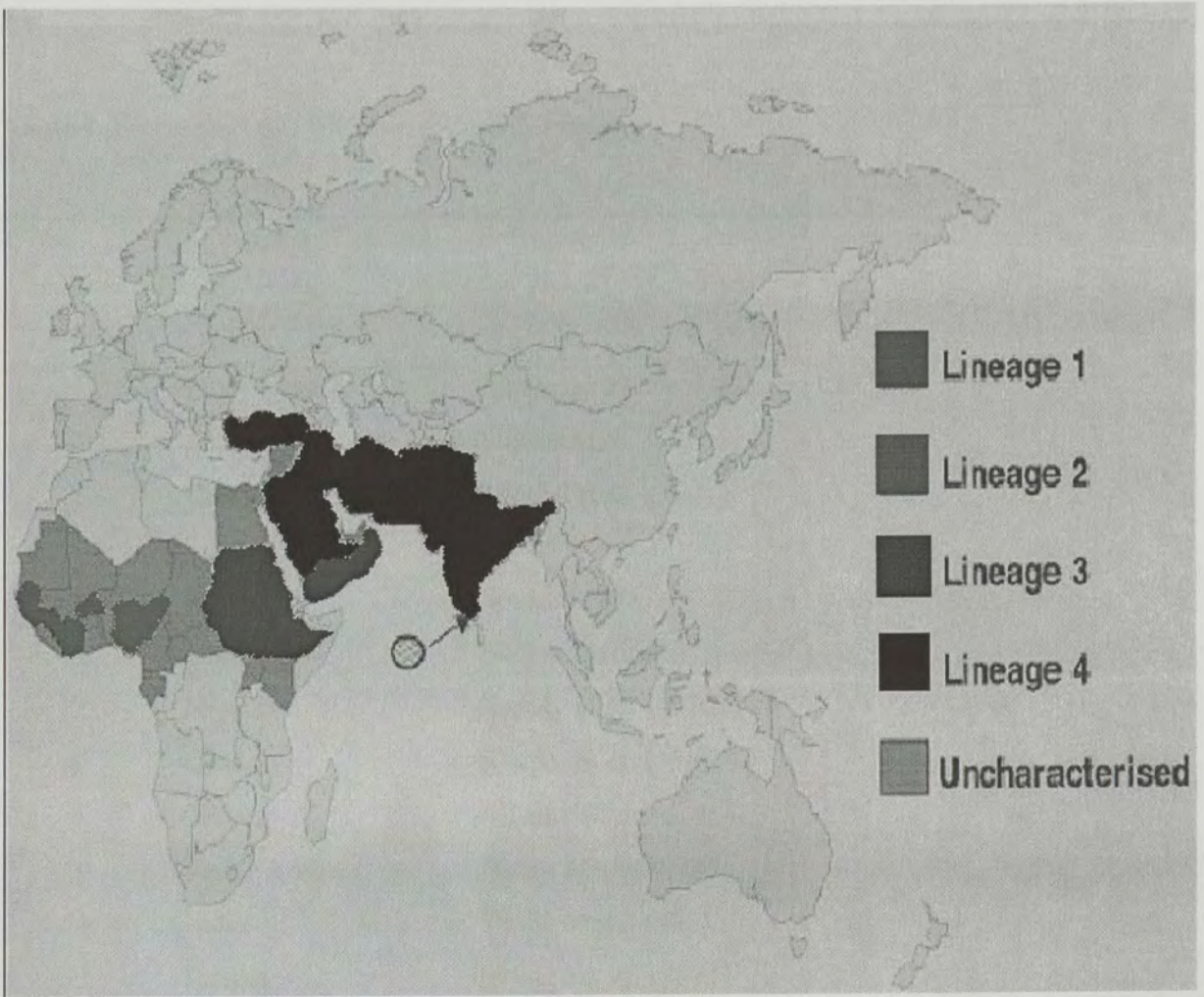


Figure 2: Geographic distribution of PPR. I, II, III & IV correspond to different lineages distributed around the world (Dhar *et al.*, 2002).

Table 1: First report of PPR from different countries

Sr. No.	Location / Country	Reference
1	Ivory coast	Garagadennec and Lalane (1942)
2	Senegal	Mornet <i>et al.</i> (1956)
3	Chad	Provost <i>et al.</i> (1972)
4	Togo	Benazet <i>et al.</i> (1973)
5	Benin	Bourdin (1973)
6	Ghana	Boniwell and Gibbs (1976)
7	Nigeria	Hamdy <i>et al.</i> (1976)
8	Oman	Hedger <i>et al.</i> (1980)
9	Sudan	Ali and Taylor (1984)
10	Saudi Arabia	Furley <i>et al.</i> (1987)
11	India	Shaila <i>et al.</i> (1989)
12	Jordan	Lefevre <i>et al.</i> (1991)
13	Israel	OIE (1993)
14	Ethiopia	Roeder <i>et al.</i> (1994)
15	Kenya and Uganda	Wamwayi <i>et al.</i> (1995)
16	Pakistan	Amjad <i>et al.</i> (1996)

2.3.4. The epidemiology of PPR in Ethiopia

In Ethiopia PPR was first suspected in goats in Afar region based on clinical signs in 1977 (Pegram and Tareke, 1981). However, the presence of the virus was confirmed in 1990 from goats purchased from southern Ethiopia for live export at holding site near Addis Ababa (Abraham *et al.*, 1991). Moreover, antibodies of the virus were detected in sera sample collected from apparently healthy sheep and goats originating from Afar, North Shewa and Arsi (Gelagay, 1996; Roger and Bereket, 1996). The national sero-surveillance carried out in 1999 has indicated

a sero-prevalence of 5.7% in small ruminants from different parts of the country (MOARD, 2004). Dawit (2006) reported a sero-prevalence rate of 6.1% in goats in Gewane district Afar region while Fitsum (2006) indicated a sero-prevalence of 31% in goats, 26.1% in sheep and 20.2% in cattle in North Shewa. Getahun (2006) also reported a sero-prevalence rate of 1.05%, 0.88% and .74% in goats, sheep and cattle respectively. A report by Teshome *et al.*, (2008) indicated a sero-prevalence rate of 46.7% in goats and 32.7% in sheep from sample collected in Debrezeit Air Force Abattoir.

2.4. Disease economy

The PPR epidemics can cause mortality rates of 50–80% in naive sheep and goat populations (Kitching, 1988). Due to the confusion with other diseases, the economic impacts of PPR are probably underestimated, but it is believed that PPR is one of the major constraints of small ruminant farming in the tropics (Taylor, 1984). Based on assumption that goats experience an outbreak every 5 years, Opasina and Putt (1985) estimated an annual sum ranging from 2.47£ per goat at high loss and 0.36 £ per goat at lowest. The economic losses due to PPR alone in India have been estimated annually to 1,800 million Indian Rupees (39 millions US\$) (Bandyopadhyay, 2002).

2.5. Pathogenesis

PPR is inhaled with infected droplets and penetrates the retropharyngeal mucosa where multiplication in the tonsil and regional lymphnodes takes place. Then it enters into circulation in mononuclear cells, causing viremia, which disseminate the virus to other lymphoid organs, the lungs, epithelial cells and mucous membranes (Radostits *et al.*, 2000). PPR virus affects the alimentary, respiratory and lymphoid systems. Infected cell undergo necrosis, but in the respiratory mucosa and lungs there is remarkable proliferation of syncytia. Some virulent strains cause deaths from severe diarrhoea with subsequent dehydration and electrolyte imbalance, particularly in kids and lambs resulting in high mortality (Radostits *et al.*, 2000). In others, death

is hastened by concurrent diseases such as pneumonic pasteurellosis and coccidiosis. Like rinderpest virus PPR virus depresses humoral and cell mediated immunity (Antonye *et al.*, 1992).

2.6. Clinical sign

PPR is mostly characterized by three forms that is a per acute, an acute and a mild form. The per acute form is seen in young goats. The incubation period is two days on average. Then appears a strong hyperthermia (41 – 42 °C) quickly followed by an attack of the general state (prostration, pilo-erection, anorexia). The animal shows oral and ocular discharges. In the first days of the disease, constipation can appear. This will be followed quickly by profuse diarrhoea. In all cases, the disease leads to death within 5-6 days after the beginning of hyperthermia. The evolution of this per acute form is so fast that it does not allow the appearance of other evocative clinical signs of the disease (Gebre-Egziabher, 2006).

The acute form, most characteristically, resembles rinderpest. The incubation period is three to four days and the first clinical signs are identical to those of the per-acute form although they are less intense. The disease develops over a longer period. This allows the appearance of other symptoms absent in the preceding form: thus the congestive lesions of the oral mucous membrane are replaced by ulcers covered by white necrotic tissues. The pulmonary sign is manifested by dry cough, which quickly becomes purulent. The ocular and nasal discharges are serous at the beginning and later on, become mucopurulent. Breathing becomes difficult because of the pulmonary attack (broncho-pneumonia) and the partial nasal obstruction by thick mucosal secretions. These signs are in fact the results of bacterial complications, generally by infection with *Pasteurella multocida*. Because of this form, PPR was for a very long time confused with pasteurellosis. Other complications of parasitical origin, such as coccidiosis, can confuse the clinical diagnosis. Pregnant females abort in most cases. The evolution of the disease ends most often by death (40 - 60 %). Those animals surviving the disease remain immunized for the rest of their life (Gebre-Egziabher, 2006).

The mild forms are much more frequent than the per acute and acute forms and very often they are undetected clinically and are known during serosurveillance. At the moment of infection animals may have a slight and temporary hyperthermia. Sometimes less abundant ocular and

nasal discharges may appear. Dried-up purulent discharges around the nostrils of the animal can be observed and this symptom may lead to confusion with contagious ecthyma (Gebre-Egziabher, 2006).

2.7. Necropsy findings

Grossly, the carcasses are dehydrated with apparent swelling around the mouth. The lesions of respiratory tract included necrotic areas on the mucosa of nostrils and turbinates, severely congested tracheal mucous membrane and white froth in trachea. Lungs will be congested and consolidated especially involving antero-ventral parts. Hydrothorax is recorded in few cases. The oral cavity is full of white necrotic debris where oral lesions are severe. The lesions in gastrointestinal tract consisted of few erosions in the mucosa from where blood oozed out, large intestines shows congestion, especially at the caeco-colic junction with streaks of blood on mucosal crests (Zebra-stripes) though the zebra-stripes are not seen in all the carcasses. The lymph nodes, especially from mesenteries are severely oedematous, congested and enlarged. In some cases hemorrhages on the internal walls of gall bladder are observed. Spleen is slightly enlarged in few cases. In some animals liver is studded with necrotic foci. Vaginitis is observed in many animals and vulvar mucus membranes are inflamed and have erosive lesions. Though the types of lesions are more or less similar in all the animals, there is a variation in the severity and involvement of the organs. The vaccinated animals have less severe lesions on the tongue (Kataria *et al.*, 2007).



2.8. Diagnosis

PPR can be tentatively diagnosed by observing clinical signs and post mortem lesions, but other diseases that cause diarrhea and pneumonia may pose a diagnostic challenge. There fore PPR should be differentiated from rinderpest, bluetongue, orf, foot and mouth disease, sheep and goat pox, viral and parasitic pneumonia, pasteurellosis and CCPP. PPR can be confirmed by isolation of the agent or serological test (Gelagay, 1996). Rapid diagnosis is done by immunocapture enzyme-linked immunosorbent assay (ELISA) and counter immunoelectrophoresis and

Hyperimmune serum:

Standard antiserum is made by immunising sheep with 5 ml of PPR virus with a titre of 10^4 TCID₅₀ (50% tissue culture infective dose) per ml given at weekly intervals for 4 weeks. The animals are bled 5-7 days after the last injection. Standard RP hyperimmune antiserum is also effective in detecting PPR antigen.

Counter immunoelectrophoresis

Counter immunoelectrophoresis (CIEP) has the same principle as the AGID except that the gel is electrically charged to improve the sensitivity of the test.

ELISA for antigen detection

A monoclonal antibody-based sandwich ELISA was found to be highly sensitive in detection of antigen in tissues and secretions of infected goats. Another format of antigen ELISA which is more widely used is immunocapture ELISA. It utilizes MAb directed against the nucleocapsid protein (Libeau *et al.*, 1994). It can give a reliable result within two hours in precoated plates and from samples maintained at room temperature for a period of seven days with no more than 50% reduction in response (Libeau *et al.*, 1994). The immunocapture ELISA allows a rapid differential diagnosis of PPR or rinderpest viruses, and this is of great importance as the two diseases have a similar geographical distribution and may affect the same animal species. The detecting MAbs used in immunocapture ELISA are directed against two non overlapping domain of the N-protein of PPR and RP, but the capture antibody detects an epitope common to both RP and PPR. The test is very specific and sensitive, it can detect 100.6 TCID₅₀/well for the PPR virus and 102.2 TCID₅₀ for the rinderpest virus. This discrepancy between the two viruses in the assay may be due to a difference in the affinity of the detection of antibody for the different N proteins (Abraham, 2005). The main advantages of this assay are: rapidity, it can be performed in a precoated plate in less than 2 hours; specificity; robustness, it can be carried out on samples

which have not been kept under ideal conditions and where no viable virus is present; simplicity. The immunocapture ELISA is suitable for routine diagnosis of rinderpest and PPR from field samples such as ocular and nasal swabs.

cDNA probes:

cDNA ^{32}P labelled clones have been used to differentiate PPR and rinderpest, but their use in routine diagnosis is not recommended due to the short half-life of the ^{32}P and the need for special equipment to protect the users (OIE, 2004).

Reverse Transcription Polymerase Chain Reaction (RT-PCR)

Conventional serological techniques and virus isolation are normally used to diagnose morbillivirus infection in samples submitted for laboratory diagnosis. However, such techniques are not suitable for use on decomposed tissue samples. The polymerase chain reaction (PCR) has proved invaluable for analysis of such poorly preserved field samples. The method consists of repetitive cycles of DNA denaturation, primer annealing and extension by a DNA polymerase effectively doubling the target with each cycle leading, theoretically, to an exponential rise in DNA product. The replacement of the polymerase Klenow fragment by thermostable polymerase derived from *Thermus aquaticus* (Taq) has greatly improved the usefulness of PCR. Using this system, a rate of amplification up to 10^7 to 10^9 times has been reported (Abraham, 2005). This technique is very sensitive compared with other tests and results are obtained in 5 hours, including the RNA extraction (OIE, 2004).

Many tests have been used for the demonstration of PPR antibodies in serum: virus neutralization test, agar gel immuno diffusion test, immunoelectrophoresis and recently blocking and competitive ELISA.

Virus neutralisation

The virus neutralisation test (VNT) is sensitive and specific, but time-consuming and expensive. The standard neutralisation test is carried out in roller-tube cultures of primary lamb kidney cells or Vero cells when primary cells are not available. VNT is the most reliable test for detection of morbillivirus antibodies (Rossiter, 1994). Serum against either PPR or RP may neutralise both viruses, but would neutralize the homologous virus at a higher titre than the heterologous virus. Therefore, for differentiation purpose reciprocal cross neutralization is used (Abraham, 2005).

cELISA

The test uses a competitive enzyme-linked immunosorbent assay for detection of specific peste des petits ruminants (PPR) antibody in serum. The test depends on inhibition of the binding of a mouse monoclonal antibody (mAb) to a PPR-specific epitope in the presence of positive serum. Inhibition is detected as a reduction in the OD reading obtained with the mAb alone following the addition of peroxidase labelled anti-mouse conjugate and substrate/chromogen mixture. Sera can be tested at a final dilution of 1/20 or titred after serial dilution. A threshold value of 50% inhibition is adopted for routine screening. The mAb supplied is directed against the nucleoprotein (N) of the peste des petits ruminant's virus (CIRAD-BOIS, 2008). The overall specificity of c-ELISA test was 98.4% with a sensitivity of 92.2% when compared with VNT (Abraham, 2005).

2.9. Control and prevention

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.

2.9.1. Sanitary prophylaxis

Although it is obvious that strict sanitary measures are hardly possible in developing countries where the disease exists, the following measures have been recommended to be applied: Isolation of infected herds and sick animals for at least 45 days after recovery, slaughtering of infected herds (as far as possible), proper disposal of carcasses and products, stringent disinfection, quarantine before introduction into herds and animal and vehicle movement controls within the infected areas

2.9.2. Medical prophylaxis

In the absence of homologous vaccine, and taking advantage of the close antigenic relationship between RPV and PPRV, the attenuated tissue culture Rinderpest vaccine has been used for a long time to protect small ruminants against PPR. At the end of the 1980s, a PPRV strain was successfully attenuated by serial passages in Vero cells. Demonstrated to be very efficient in the protection of sheep and goats against a virulent challenge, this avirulent PPRV is now widely used in the control of PPR (Gebre-Egziabher, 2006).

3. MATERIALS AND METHODS

3.1. Study Area

3.1.1. Location

Jijiga Zone (Figure3) comprising six districts, is located at 630 km, east of Addis Ababa. Generally, Jijiga Zone represents parts of the vast lowland area (1500-2200m above sea level) of Somali National Regional State (SNRS). The Zone borders to the West with Oromia region, to the North with Shinile zone, to the East with North Somalia and to the Southeast with Fik and Degehabur Zones.

Shinile Zone is comprised of six districts, is located at 530km, East of Addis Ababa. It is an altitude range of 950-1350m above sea level. The annual rainfall varies from 500-700mm. The pastoral land covers the largest part of the Zone (Figure 3).

3.1.2. Climate

The climate is generally semiarid and arid with annual average rainfall of 560 mm and the annual daily minimum and maximum temperature ranges from 13 – 27⁰ C (Teshome, 2001). There are no permanent rivers running through the zone. The Fafan, Jerer and Dakhata rivers are all seasonal. The soil type is mostly black clay (fertile) except in Babile district where there is sandy soil (used for ground-nut cultivation). The Zone depends on the *Dira* rain (mid-March to mid-June) and the heaviest *Karan* rain (mid- July to mid-September), both of which are equally important for cultivation /maturation of crops, water availability and livestock pasture regeneration (Save The Children UK, 2003).



3.1.3. Land and water tenor

Land is inherited by many households but the household cultivates part of it. They also own some grazing land. Better-off households have the resources to cultivate wider areas. In a good year, there is enough water to support both human and livestock populations. Both the Fafan and Jerer valleys (running through the Western part of Jijiga zone) are grazing areas during dry season (Save the Children UK, 2003).

3.1.4. Crop production

The main crops cultivated with the *Dira* rains include long maturing varieties of sorghum and to a lesser extent maize. During the *Karan* rains short maturing varieties are cultivated (wheat, barley, maize) and some ground nuts in Babile *Wereda*; in addition small amount of chickpeas, flax, peas, oats, lentils, Irish and sweet potatoes, onions, garlic and chat are grown. *Guus* is the principal arrangement used to ensure all households are able to plough and cultivate some lands. The owner of land provides food and *chat* in exchange for day's farm-labor (Save the Children UK, 2003).

3.1.5. Livestock and migration

Pasture is supplemented by crop residues between May and July (primarily for milking cows and oxen) and from the second harvest; livestock eat stored fodder from January to March. March is the most difficult month in relation to pasture and fodder availability. In years of poor rainfall, households with livestock move to the closest grazing areas. Those from western and central areas go to the Fafan, Dhakata and Jerer valleys. Those in the North-East go to Borama in Somalia and Dambel in Shinile zone (Save the Children UK, 2003).

The Somali region has 3.7 million cattle, 8.5 million goats, 9 million sheep, 2.1 million equines, 2 million camels and 112,000 chicken playing crucial roles in the livelihoods of the majority of rural people. The total goat and sheep population in Jijiga and Shinile Zone in the study area are indicated in Table 2 (Save The Children UK, 2003).

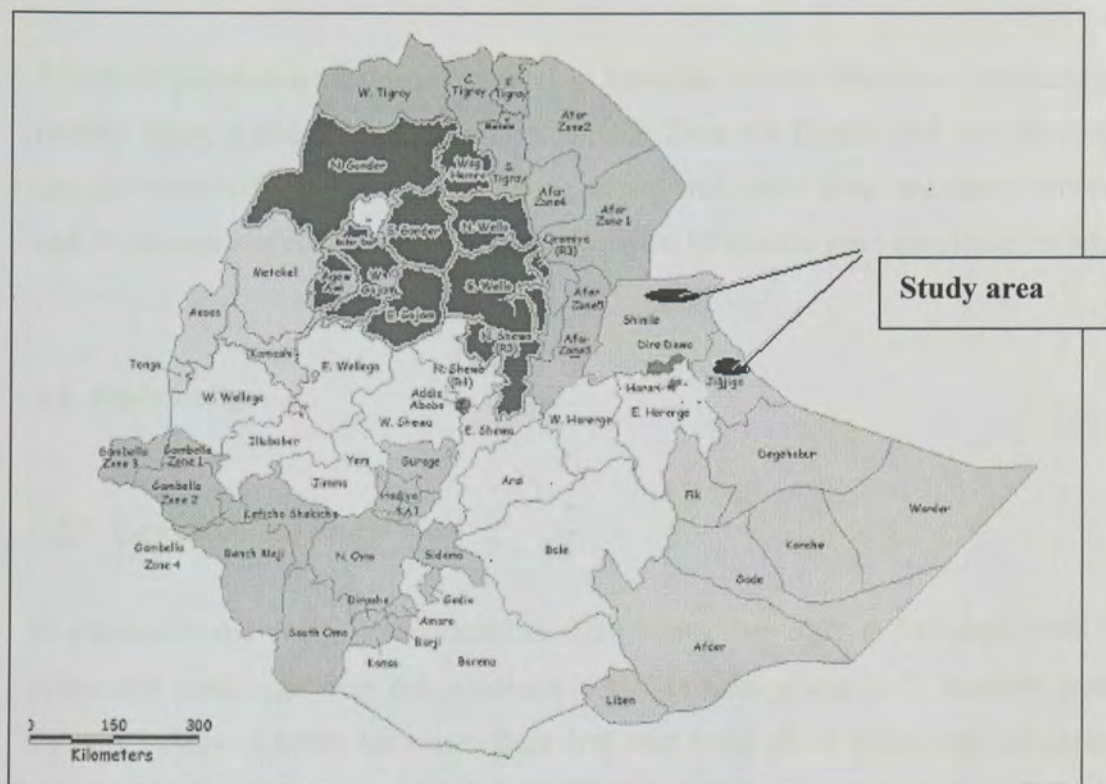


Figure 3: Map showing the study area

Table 2: Livestock population in Jijiga and Shinile Zones (study area)

Wereda	Cattle	Sheep	Goat	Camel	Equine	Total
Jijiga	139882	375970	156,629	8403	12116	693,000
Awbere	66504	178976	75,328		5739	330,464
Gursum	59986	162,391	64483	3944	5051	295,357
Shinile	47,880	191,730	162,960	15,120	9,660	427,350
Denbel	34,200	136,950	116,400	10,800	6,900	305,250
	489000	1316000	548,000	29000	42000	2,424,000

(Save the Children UK, 2003).

3.2. Study population

The study population was based on random selection of sites from three districts of Jijiga Zone namely Jijiga, Awbere and Gursum. In Shinille Zone the Denbel and Shinille districts. Serum samples were collected from goats, sheep, camel and cattle. Goat and sheep between 6 months and 12 months was considered as young and above 12 months were considered as adult.

3.3. Study design

3.3.1. Cross sectional study

A cross-sectional study was undertaken from September 2008 to February 2009. The ages of sheep and goats, age were categorized into two groups, young (6-12 months) adult (above 12 month). In case of cattle and camel those less than 5 and above years were categorized as young and adult, respectively. Sex was also considered and categorized as male and female. Possible risk factors were considered and compared with respect to PPR positivity.

3.3.2 Questionnaire survey

A questionnaire was administered to livestock owners to collect data on management of goats and sheep, history of PPR in the area, traditional ways of handling PPR once reported in a herd or neighboring herds, individual animal data and vaccination schedules and types of treatment used. A total of 140 livestock owners were interviewed. Both open-ended and closed type questionnaires were prepared. The questionnaires were pre-tested before administrating to the study population. The format was coded for computer database entry and analyzed.

3.4. Sampling method and sample size determination

Five districts, two from pastoral areas (Shinille, Denbel) and three from agro-pastoral areas (Jijiga, Gursum and Awbere) were purposively selected based on husbandry and PPR status to get a better picture of the area. From each district 2 'Kebele' were selected purposively. In turn within the 'Kebele' each 2 villages and 2 herds were selected purposively. Individual animals were selected by simple random technique.

The sample size required for this study was determined depending on the expected prevalence of PPR and the desired absolute precision according to the formula Thrusfield (2005) as follows:

$$n = \frac{1.96^2 P_{exp} (1 - P_{exp})}{d^2}$$

Where:

n = required sample size

P_{exp} = expected prevalence

d = desired absolute precision

A previous study on PPR in Jijiga recorded a prevalence of 15% in goats, 7% in sheep, 5% in cattle and camels (Abraham, 2005). Therefore, 95% confidence interval, 5% precision and expected prevalence 15% in goats, 7% in sheep and 5% in cattle and camel were used to compute the sample sizes. Using these proportions, 195 goats, 100 sheep, 73 cattle and 73 camels were obtained (Table 3). To increase the precision 616 goats, 202 sheep, 110 cattle and 94 camels were used.



Table 3: Distribution of Serum sample collected by production system Woreda, PA, altitude and animal types.

Production system	woreda	PA	Altitude	Total	Serum samples			
					Goat	Sheep	Cattle	Camel
Agro-pastoral	jijiga	Karamara	1765	70	65	5	-	-
		Dibley	1717	97	80	12	-	-
		Dudumas	1709	100	94	6	30	43
Agro-pastoral	Gursum	Fafem	1530	92	78	30	20	-
		Bombas	1800	86	32	33	-	-
Agro-pastoral	Awbere	Gedeb	1600	95	68	22	-	-
		Wojaale	1555	49	32	22	-	-
Pastoral	Shinille	Shinille -1	1200	98	65	19	30	20
		Shinille - 2	1100	42	27	20	-	-
Pastoral	Denbel	sendelol	1050	48	34	18	30	31
		Arabi	1100	49	30	16	-	-
Total					616	202	110	94

3.5. Specimen collection and laboratory procedure

3.5.1. Blood samples collection

Blood was collected from the jugular-vein using Venoject needles and vacutainer tubes (Venoject, UK). The blood was left to clot overnight in cold boxes or small field refrigerators. The serum was collected into sterile tubes and kept in ice for transportation to the laboratory. In the laboratory, the serum was centrifuged at 2000 rpm for five minutes to remove the remaining red blood cells before being transferred to 2-ml cryovials and stored at -20°C until analyzed.

3.5.2. Laboratory procedure

PPR c-ELISA kit for PPRV antibody detection along with the user manual was obtained from CIRAD/EMVT 2008. The contents of the kit were listed in Table 4



Table 4: Contents of the PPR c-ELISA kit

Serial no	Item
1	Manual with fact sheet
2	Distilled water (10 ml) 1 RT*
3	PBS powder (Sigma 1 L) 2 RT
4	Tween-20 (10 ml) 1 RT
5	ELISA plate (Nunc Maxisorp) 15 (5 x 3) RT
6	Anti-mouse HRPO conjugate (100 µl) 1 40C
7	OPD tablet (30 mg) 2 40C
8	H ₂ O ₂ (3%) 10 ml 1 40C
9	Antigen (1 ml) 1 -200C
10	Negative serum (1 ml) 2 -200C
11	Strong positive serum (1 ml) 1 -200C
12	Strong positive serum (1 ml) 1 -200C
13	Monoclonal antibody 1 -200C

RT* - Room Temperature

The cELISA was performed strictly as per the protocol outlined in the user manual supplied with the kit, as follows.

Preparation of reagents

- a) PPRV Antigen: One ml of sterile distilled water was added to the freeze-dried contents of the vial and mixed till completely dissolved and stored at -20°C till used.
- b) Monoclonal Antibody (MAb): One ml of sterile distilled water was added to the freeze-dried contents of the vial and mixed till completely dissolved and stored at -20°C till used.
- c) Anti-mouse HRPO-conjugate: supplied in liquid form.
- d) Serum Controls (strong positive, weak positive and negative serum): One ml of sterile distilled water was added to each of the freeze-dried contents of the vial and well mixed until dissolved and stored at -20° C till used.

All immunoreactants (antigen, MAb and control sera) after reconstitution were kept at 4°C or on ice while performing the test.

Diluents

- a) Phosphate-buffered saline (PBS, pH 7.4): Contents of one liter PBS pouch was dissolved in 100 ml of fresh glass distilled water to make 10X PBS. The stock was diluted 1 in 10 depending on requirements and stored at 4^oC.
- b) Blocking buffer: PBS containing 0.1% Tween-20 and 0.2% negative serum. It was prepared fresh every time when needed.
- c) Chromogen-substrate solution: One tablet (30 mg) of OPD was dissolved in 75.0 ml fresh glass distilled water and stored at -20^oC till used. H₂O₂ solution (3%) was added just before use at the rate of 4.0 µl H₂O₂ per ml of OPD solution.
- d) Stopping solution (1N sulphuric acid): To make 100 ml of 1N sulphuric acid, 5.45 ml of concentrated sulphuric acid (not supplied with the kit) was added to 94.5 ml of distilled water, mixed well and stored in amber colored tight stoppered bottle.

Test Protocol

- a) The reconstituted stock antigen of PPRV was diluted at the ratio of 1:100 in 1X PBS, mixed well and added (50 µl) to all the wells of a 96-well ELISA plate supplied with the kit. The plate was tapped gently to ensure that the fluid settled at the bottom of the wells. The plate was covered with a lid and incubated at 37^oC for one hour on an orbital-shaker with continuous shaking at moderate speed.
- b) At the end of the incubation period, the antigen was discarded from the plate by inverting the plate over the sink and tapping or jerking it down with a single motion of the hand. The plate was washed three times by filling up the wells with the washing buffer (PBS diluted four times with distilled water) and then discarding the buffer by inverting the plate over the sink and tapping it over a piece of filter paper.
- c) The following reagents were then added very carefully step by step:
 - 40 µl of blocking buffer in all the wells.
 - 20 µl of additional blocking buffer to monoclonal antibody control (Cm) wells.
 - 60 µl of additional blocking buffer to each of the conjugate control (Cc) wells.
 - 20 µl per well of each test serum sample in a set of two wells using a separate tip for each sample (vertical duplicates as per the template provided).

- 20 µl of strong positive serum control (C++) in each of the four designated wells in the plate.
- 20 µl of weak positive serum control (C+) in each of the four designated wells in the plate.
- 20 µl of negative serum control (C-) in each of the two designated wells in the plate.
- 40 µl of diluted monoclonal antibody in each well of the plate except the conjugate control wells (Cc).

d) Contents of the wells were mixed by gently tapping the sides of the plate. The plate was covered with a lid and incubated at 37° C for one hour on an orbital-shaker with continuous shaking at moderate speed.

e) At the end of the incubation, the plate was taken out of the incubator and repeated the discarding and washing procedures as in Step (b).

f) Diluted anti-mouse conjugate (50 µl) was added in all the wells of the plate. Contents of the wells were mixed by gently tapping the sides of the plate. The plate was covered with a lid and incubated at 37° C for one hour on an orbital-shaker with continuous shaking at moderate speed.

g) At the end of the incubation, the plate was taken out of the incubator and discarding and washing procedures as in Step (b).

h) Freshly prepared OPD-substrate mixture (50 µl) was added in each well of the plate. Also added 50 µl of the same in each well of the blank 8-well module supplied with the kit.

i) The plate was incubated and the blank module for about 10 to 20 min at 37°C without shaking or till visible color developed in Cm wells.

j) Once visible color developed in Cm wells, 50 µl of stopping solution (1M sulphuric acid) was added to each well of the plate and the blank module. The plate was tapped by sides and read at 492 nm in ELISA plate reader.

k) The blanking plate/module (supplied in the kit) was put in the ELISA plate reader followed by the plate containing the test proper and the instructions as prompted by the computer was followed.

Interpretation of test results

The test sera samples showing more than 50 per cent inhibition (PI) of mean OD values of the Cm wells were taken as positive for PPR antibodies provided other controls fell within this range. The plate reading was rejected if the PI in the control panel did not fall within the expected range indicated below.

- Conjugate control: 91 to 105%
- Strong positive serum (C++): 81 to 100%
- Weak positive serum (C+): 51 to 80%
- Negative control serum (C-): -25 to 25%

3.6. Data management and analysis

All data were entered into a Microsoft Excel spreadsheet and checked for accuracy. After validation, data were transferred to SPSS release 15.0 (SPSS, 2006) for analyses. Sero-prevalence estimation of PPR in goats and sheep was determined using standard formulae (i.e. the number of positive animals/samples divided by the total number of animals/samples examined). Difference between proportions of groups with certain determinant factor was determined by Chi-square (χ^2) test, confidence interval. Odds ratio was calculated to estimate strength of association between risk factors and prevalence of PPR. Descriptive statistics such as percentages (proportion) and frequency distributions were used to describe the characteristics of the data.

4. RESULTS

4.1. Seroprevalence of PPR by species

The seroprevalence of PPR in goats and sheep was 25.6% and 14.4% in the study area respectively (Table 5). The seroprevalence was lower in camels and cattle, which was 8.5% and 6.63% respectively. Goats showed more seropositivity (odd ratio 2.05) were found to be more susceptible than sheep, which was found to be statistically significant ($\chi^2 = 27.566$, $p=0.000$). However no significant difference was observed between cattle and camels ($\chi^2 0.343$, $P=0.558$).

Table 5: Distribution of seroprevalence of PPR by animal Species

Animal species	Sera screened	Seropositivity n (%)	95 %Confidence interval of seropositivity	
			Lower limit	Upper limit
Goat	616	158 (25.6)	22.36	29.24
Sheep	202	29 (14.4)	10.19	19.86
Cattle	110	7 (6.63)	3.11	12.55
Camel	94	8 (8.51)	4.38	15.9

4.2. Seroprevalence of PPR by location

Serum samples were collected from a total of 5 districts in two different agro-ecologically Zones namely Jijiga, Awbere, Gursum from Jijiga Zone and Shinille and Denbel in Shinille Zone. Seroprevalence of goats and sheep in Shinille Zone (pastoral) and Jijiga Zone (agro-pastoral) production system were 44.09% and 18.9% (goats), 30.01% and 5.42 (sheep) respectively (Table 6). There was statistically significant ($\chi^2 =4.3$ $P= 0.002$) difference between seroprevalence of goat and sheep in Jijiga and Shinille Zone (table 6). Among cattle and camel statistically

significant difference ($p=0.043$) was observed between the two agro-ecological zonal seroprevalence.

Among the districts in Jijiga Zone, the highest seroprevalence of PPR (25.2%) was observed in goat population in Gursum District followed by Jijiga (18.8%) and Awbere (12%) (Table 7). Gursum seroprevalence was statistically significantly different from Jijiga and Awbere ($p<0.05$). As for the sheep, highest seroprevalence was found in Gursum District (7.7%), followed by Awbere (4.5%) and Jijiga (4.35%). However, there were no statistically significant ($p= 0.177$) difference was observed among these districts wise seroprevalence.

In Shinille Zone, higher seroprevalence of PPR was observed in goat population in Denbel Districts (48.3%) compared to Shinille Districts (41.23%) (Table 8). But, no significant difference was observed between these seroprevalence ($p=0.156$). As for sheep high seroprevalence of PPR was observed in Shinille Districts (33.3%) compared to Denbel District. However, no significant ($P>0.101$) difference was found between these seroprevalence.

Table 6: Agro-ecology wise sero-prevalence by species

Agro-ecology by district	Species	No of sera tested	Seropositivity n (%)	95% confidence interval	
				Lower limit	Upper limit
Jijiga	Goat	455	86 (18.9)	15.57	22.75
	Sheep	130	8 (6.15)	3.15	11.67
	Cattle	50	2 (4)	4.41	31.89
	Camel	43	2 (4.65)	4.98	34.64
Shinile	Goat	161	71 (44.09)	36.66	51.82
	Sheep	72	22 (30.01)	21.13	41.96
	Cattle	60	5 (8.33)	3.6	91.806
	Camel	51	6 (11.76)	5.5	23.38

Table 7: Sero-prevalence of sheep and goat in Jijiga zone by Districts

District	Species	No of sera tested	Seropositivity n (%)	95% Confidence interval		p-value
				Lower limit	Upper limit	
Jijiga	Goat	239	45 (18.8)	14.38	24.26	0.051
	Sheep	23	1 (4.35)	4.54	32.12	
	Cattle	50	2 (4)	1,1	13.46	
	Camel	43	2 (4.7)	1.28	17.05	
Gursum	Goat	115	29 (25.2)	18.17	33.87	0.001
	Sheep	63	5 (7.7)	1.63	13.09	
Awbere	Goat	100	12 (12)	7.76	20.98	0.052
	Sheep	44	2 (4.5)	0.4	11.8	

Table 8: Sero-prevalence of sheep and goat in Shinille zone by Districts

District	species	No of sample tested	Seropositivity n (%)	95% Confidence interval	
				Lower limit	Upper limit
Shinille	Goat	107	45 (42.06)	33.14	51.53
	Sheep	28	8 (28.85)	15.25	47.06
	Cattle	60	5 (8.3)	5.5	23.46
	Camel	51	6 (11.76)	1.28	17.05
Denbel	Goat	54	26 (48.3)	35.4	61.16
	Sheep	44	14 (31.8)	31.82	46.56

4.3. Seroprevalence of PPR by Age

The Table 9 shows that seroprevalence increased from 21.7% to 28.9% in goats and from 8.1% to 20.4% in sheep from the young to adults, respectively. There was significant difference between

age groups in seroprevalence of both goats ($p=0.04$) and of sheep (0.013). In both cattle and camels the seroprevalence of young and adults were not different ($p>0.05$; Table 9).

Table 9: seroprevalence of sheep and goat in the study area by age

Species	Age/sex	No of sample tested	Seropositivity n (%)	95% Confidence interval		p-value
				Lower limit	Upper limit	
Goat	Young	277	60 (21.7)	17.21	26.88	0.04
	Adult	339	98 (28.9)	24.34	33.95	
Sheep	Young	99	8 (8.1)	4.15	15.14	0.013
	Adult	103	21 (20.40)	13.74	29.17	
Cattle	Young	36	4 (11.1)	4.41	31.89	0.062
	Adult	74	3 (4.1)	1.39	11.25	
Camel	Young	21	3 (14.28)	4.98	34.64	0.249
	Adult	73	5 (7.5)	2.96	15.05	

4.4. Seroprevalence of PPR by sex

Regarding sexes, 26.4%, and 25.5 % ($p=0.851$) was found in goats, 15.6% and 14% ($p=0.795$) in sheep's 8% and 5 % ($p=0.488$) in cattle ($p=0.488$) and 8.2% and 9.5% ($p=0.095$) in camel in male and female respectively. Therefore there were statistically no significant difference by sex specific seroprevalence. (Table 10).

Table 10: seroprevalence of pest des petitis ruminants in the study area by Sex

Species	Sex	No of sample tested	No sera positive	Percent Of positive	95 %confidence interval		p-value
					Lower limit	Upper limit	
Goat	Female	510	130 (25.5)	25.5	21.90	29.45	0.851
	Male	106	28 (26.4)	26.4	18.96	35.53	
Sheep	Female	157	22 (14)	14	9.44	36.68	0.795
	Male	45	7 (15.6)	15.6	7.75	28.79	
Cattle	Female	60	3 (5)	5	1.71	13.70	0.488
	Male	50	4 (8)	8	3.15	18.84	
Camel	Female	21	2 (9.5)	9.5	2.65	28.91	0.095
	Male	73	6 (8.2)	8.2	3.82	16.8	

4.5. Questionnaire Survey result

A total of 70 individuals in Shinille and 70 in Jijiga were interviewed (Annex 3). The responses obtained per each disease (Table 11 and 12) were CCPP (*Sunbub*), PPR (*Kollere*) and GIT parasite (*aal*) ranked first, second and third, respectively in shinille zone while in jijiga zone GIT parasite (*aal*), CCPP (*Sunbub*) and tick (*Shillin*) were ranked first, second and third, respectively. According to the respondents the proportion of goats affected by CCPP (*Sunbub*) and PPR (*Kollere*) were 32.8%, 21.4% and 25.7%, 14.3% in Shinille and Jijiga Zone respectively. However more goats are affected by GIT parasites in Jijiga (40%) than in Shinille Zone (21.4%). No associations were observed between presence of ticks and occurrence of pox disease. However, more goats were affected by GIT parasite in Jijiga (40%) than Shinille. The percentage of response for CCPP and PPR was higher in Shinille compared to jijiga Zone.

A proportion of 29.62% of the interviewees (Table 13) said they knew PPR (*Kollere*). Regarding species susceptibility 69.28 % respond that goat was more susceptible than sheep. Further (30.72%) responded that sheep were more susceptible than goats. Concerning season, 67.85% responded that PPR occurs in the dry season and 32.15 replied that the disease occurs in the rainy season. All of (100%) the pastoralists and agro-pastoralists indicated the need for vaccination out of which 86% agreed to pay vaccination fee while 14% were not interested. Regarding susceptibility by age 64.28% and 60.7% of the respondent replied that young goats and sheep were more susceptible than adult animals. However, no response (0%) was made indicating susceptibility of goats and sheep less than 6 months of age (Figure 4).

Table 11: Frequency ad proportion disease in Jijiga Zone in goats

Disease		Frequency (response)	Proportion (%)
Local name	Conventional name		
<i>aal</i>	GIT parasite	28	40
<i>Sunbub</i>	CCPP	15	21.4
<i>Shillin</i>	Tick	12	17.2
<i>Kollere</i>	PPR	10	14.3
<i>Gedanod</i>	Pox disease	5	7.1
Total		70	100

Table 12: Frequency ad proportion disease in Shinile Zone in goats

Disease		Frequency (response)	Proportion (%)
Local name	Conventional name		
<i>Sunbub</i>	CCPP	23	32.8
<i>Kollere</i>	PPR	18	25.7
<i>aal</i>	GIT parasite	15	21.4
<i>Shillin</i>	Tick	11	15.8
<i>Gedanod</i>	Pox disease	3	4.3
Total		70	100

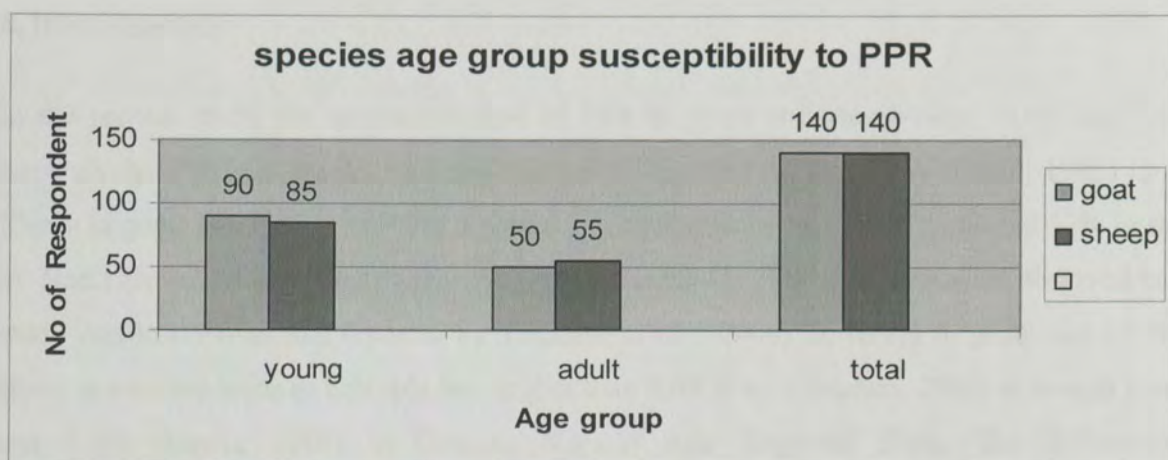


Figure 4: species-age group susceptibility to PPR

Table 13: Result of percentage of respondent to various questions

No of person interviewed	Type of questionnaire	Percentage of respondent
140	Knowledge about disease with oral & muzzle lesion, watery diarrhea & cough, nasal & lachrymal discharge and PPR	29.62% said the disease is present
140	Season of occurrence	67.85% said the disease occur in dry season and 32.15 said it to occur in rainy season
140	Payment for vaccine	All of them both pastoralist and agro-pastoralist indicated the need for vaccination and 86% agreed to pay vaccination fee while 14% did not
140	Species susceptibility	69.28 % said goat is more susceptiple than sheep and 30.72 said sheep is more susceptible than goat
No of person interviewed	Type of questionnaire	Percentage of respondent

5. DISCUSSION

In the present study the seroprevalences of PPR in goats and sheep were 25.6% and 14.4% respectively. Previous studies reported a seroprevalence of 16.7 % (Yayehyirad, 1997) in East Shewa in goats and Gelagay (1996) reported a seroprevalence of 17% in goats and 12% in sheep in North Shewa which closely agree with the present study. The seroprevalence observed in this study was lower than that reported by Teshome *et al.*, (2008) of 46.7% in goats and 32.7% in sheep in selected areas in Ethiopia but higher than 0.94 % by (Getahun, 2006) in Awash Fentale and 6.1% (Dawit, 2006) in Gewane *Woreda* Afar Regional State. The difference in seroprevalence within Ethiopia could be due to difference in ecology, production system, study design features and the temporal and spatial distribution of the disease.

In this study, goats were found to be more susceptible than sheep. In general sheep are considered to be more resistant to PPR than goats. The disease occurs in goats without affecting sheep living in close proximity (Lefevre and Diallo, 1990). In Ethiopia goats react more severely to PPR virus exposure compared to sheep and they exhibit overt clinical signs while sheep undergo a mild form of the disease (Abraham *et al.*, 2004). In Africa, PPR is more prevalent in goats while it is common in sheep in South Eastern Asia.

In contrast the variation of the seroprevalence was lower in camels and cattle, which were 8.5% and 6.63%, respectively. Seroprevalence in cattle was comparable with that reported in Jijiga (5%) (Abraham *et al.*, 2004) but higher than those reported in West Africa of 1.78 and 4.5 in Mali and Cameroon, respectively (Tounkara *et al.*, 1996). This may be attributed to high population densities of sheep and mixed grazing resulting in increased contact, between small ruminant and cattle. Regarding camel comparable seroprevalence of 10% was observed in Afar (Abraham *et al.*, 2004). Antibody seroprevalence detected in camels, cattle, goats and sheep confirmed natural transmission of PPR virus under field conditions.

Seroprevalence of goats between Shinille (pastoral) and Jijiga Zone (agro-pastoral) production systems showed 44.09% and 18.9% in goats, and 30.01% and 5.42 in sheep respectively. These were significantly different. Among cattle and camel differences were observed between the two agro-ecological zones. These results were in agreement with the report by Teshome *et al.*, (2008).

Pastoralist inhabits the lowland and in contrast to agro-pastoralist move in search of feed and water for animals. This allows continuous mixing of different herds especially at watering points. Thus diseases like PPR that need close contact for transmission are highly prevalent in such type of production system (Ezeokoli *et al.*, 1986)

Among districts in Jijiga Zone (agro-pastoral), the highest seroprevalence of PPR (25.2%) was observed in goat population in Gursum District followed by jijiga (18.8%) and Awbere (12%). The variation in seroprevalence of Gursum, Jijiga and Awbere Districts that are located in the in the same agropastoral area could be due to the migration of small ruminants in search of water from Denbel District to Gursum Jerer River.

In Shinille Zone, high seroprevalence of PPR was observed in goat population in Denbel Districts (48.3%) compared to Shinille Districts (41.23%). Regarding sheep higher seroprevalence of PPR was observed in Shinille District (33.3%) compared to Denbel District. This is in agreement with a prevalence rate of 53% in goats and 33% in sheep reported in Arsi (Gelegay, 1996). The lack difference in seroprevalence between the two districts may be explained by the fact that they share the same agro-ecological conditions..

Sero prevalence increased from 21.7% to 28.9% in goats and from 8.1% to 20.4% in sheep from young to adults respectively. Similar findings were reported by Singh *et al* (2004) in small ruminants in India and Teshome *et al.*, (2008) in selected areas of Ethiopia. Among small ruminants above 4 months of age the seroprevalence increases with age (Obi, 1984; Radostits *et al.*, 2000) which supports the findings of this study.

Regarding sex, 26.4%, and 25.5% was found in goats 15.6% and 14% in sheep 8% and 5 % in cattle and 8.2% and 9.5% in camel in males and females respectively. This was in agreement with Teshome *et al.*, (2008). This shows that sex has no influence PPR seroprevalence.

CCPP (*Sunbub*), PPR (*Kollere*) and GIT parasites (*aal*) were ranked first, second and third, respectively in Shinille Zone while in jijiga Zone GIT parasites (*aal*), CCPP (*Sunbub*) and ticks (*Shillin*) were ranked first, second and third, respectively. According to the respondent the



number of goats affected by CCPP (*Sunbub*) and PPR (*Kollere*) was 32.8%, 21.4% and 25.7%, 14.3% in Shinille and Jijiga Zone respectively. However, more goats were affected by GIT parasites in Jijiga (40%) than in Shinille Zone (21.4%). This indicates that CCPP and PPR need more attention in Shinille Zone while GIT parasite and CCPP in Jijiga Zone.

As for the knowledge of PPR (*Kollere*) 29.62% of the interviewees said they know it. Previous report showed that 17.9 % experience the disease in Awash Fentale Woreda (Getahun, 2006). Regarding species susceptibility, 69.28 % responded that goats were more susceptible than sheep and 30.72% responded that sheep was more susceptible than goats. This was in agreement with serological test findings.. Regarding susceptibility by age 64.28% and 60.7% of the respondents replied that young goats and sheep were more susceptible than adult animals. This is in agreement with Abraham *et al.*, (2004) who reported that goats react more severely to exposure to PPR than sheep.

6. CONCLUSIONS AND RECOMMENDATIONS

A wide occurrence and distribution of PPR infection in both pastoral and agropastoral production system in these two Zones was observed in goats, sheep, cattle and camel. Species, agro-ecology and age of goat and sheep was significantly associated, where young ones were found to be more susceptible. PPR was found to occur in dry season compared to wet season. There was a gap regarding awareness of PPR with in the pastoralist.

Based on the above conclusions the following recommendations are forwarded:

- The presence of seroprevalence of PPR in different species highlight the need for further epidemiological investigations, designing, implementing, prevention and control strategies
- Prevention should be centered around improving young goat and sheep susceptibility by vaccinating age groups above 6 months
- Attention must be given for cattle and camel regarding their role in the epidemiology of PPR
- Vaccination programme should be strategically designed by taking in to consideration the dry season
- Awareness creation of pastoralist about transmission and control PPR should to be strengthened

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8. ANNEXES

Annex 1: PPR case investigation format (Individual animal record)

1. Background information:

- 1.1. Region _____
1.2. Zone _____
1.3. District _____
1.4. PA _____
1.5. Village name _____
1.6. Geo. Reference
1.6.1. _____ N
1.6.2. _____ E
1.6.3. Altitude _____
1.7. Owner s name _____

2. Animal identification

2.1. Species

Caprine

Ovine

2.2 Age group

2.1.1. between 4 and 12 month _____

2.1.2. above 12 month _____

2.3 Sex:-

2.1.3. Male:

2.1.4. Female

2.4 Animal origin:

2.4.1. Born:

2.4.2. Introduced

If introduced, from which District _____

3. Clinical signs observed

Dyspnea	Oral lesion	Diarrhea	Fever	Lacrimation	Nasal mucoid discharge	Respiratory distress

Additional clinical signs
observed _____

4. Tentative diagnosis (based on clinical sign and epidemiological information)

4.1. -----

4.2. -----

4.3. -----

5. Possible source of infection

5.1. Newly introduced animal

5.2. Common greasing \watering point

5.3. Market place

5.4. Other sources (mention) _____

6) Sample taken

6.1. -----

6.2. -----

6.3. -----

If sample is taken during sero-survey, please indicate the code and serial number.

Code _____ S.N. _____

6. Date of last PPR case in the area _____

7. Date of last PPR vaccination _____

Investigated and completed by

Name _____

Date _____

(Day/month/year)

Signature _____

Désignation _____

Annex 3: PPR questionnaire (pastoralist s ' observation)

Code _____

1. REFERENCES

REGION: _____

WEREDA:

ZONE: _____

Date of Interview: E.C. G.C.

Name of interviewer

Season: Wet Dry

Village:

PA:

Geo. reference

1.1. _____ N
1.2. _____ E
1.3. Altitude _____

2. Livestock species raised by the community (rank by population size 1-5)

1. Cattle 4. Camel
2. Sheep 5. Equine
3. Goat

3. HUSBANDRY SYSTEM

pastoral:
Agro-pastoral:

4. DISEASE PROBLEMS

4.1. List major diseases of goat and sheep in the area (in order of importance).

	LOCAL NAME	SCIENTIFIC NAME
1	_____	_____
2	_____	_____
3	_____	_____
4	_____	_____
5	_____	_____

4.2 Have you ever observed an oral lesion, diarrhoea, nasal discharge, respiratory distress and lacrimation in goat and sheep? Yes No

4.3 a) Do you know a disease called PPR? Yes No

4.3b) If yes, list the clinical symptoms observed.

4.4) When did you observe PPR for the first time?

Before 5 years

Before 2 years

Less than a year

5.5) How frequently you have observed a PPR outbreak?

Each year

Twice a year

4.6) Suspected sources of PPR outbreak?

Seasonal herd movement for grazing and watering

Newly introduced animal (trade, gift)

Unknown

4.7) Give the number of goat and sheep that died of the disease in the last 12 months

4.8) Is your herd affected by PPR now? Yes No

4.9) If yes, give the number of sick and dead goat and sheep in the last 12 months

	Sick	dead		sick	dead
Buck	<input type="checkbox"/>	<input type="checkbox"/>	ram	<input type="checkbox"/>	<input type="checkbox"/>
Doe	<input type="checkbox"/>	<input type="checkbox"/>	ewe	<input type="checkbox"/>	<input type="checkbox"/>
Kid	<input type="checkbox"/>	<input type="checkbox"/>	lamb	<input type="checkbox"/>	<input type="checkbox"/>

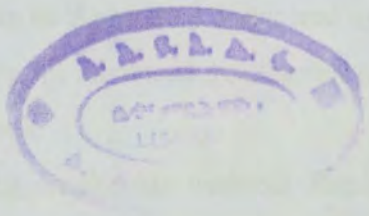
4.10) Do you treat a PPR sick animal? Yes No

4.11) If yes give the cost of treatment per animal in birr.

4.12. How many times you treated your shoats for PPR in the last 12 months?.....

4.13) There are different PPR control approaches like vaccination, quarantine (animal movement control) .

- Do you agree to pay for vaccination? Yes No
- Do you practice PPR control by quarantine (not buy goat and sheep from unknown origin or not sell) if you are told to do so? Yes No



9. CURRICULUM VITAE

A. PERSONAL INFORMATION

Name	Teffera Sime
Date of Birth	23 September 1969
Place of birth	Jijiga
Sex	Male
Nationality	Ethiopian
Marital status	Married
Address	Jijiga Tel 0912169612 off. 025-775-6950 Email-teferasime@yahoo.com

B. EDUCATION AND QUALIFICATION

1978 – 1987G.C	Jijiga Secondary School
1988 – 1993G.C	Addis Abeba University Faculty of Veterinay Medicine Debreziet Award Degree of Doctor of Veterinary Medicine.
2008-2009 G.C	MVSc in Tropical veterinary Epidemiology, from Addis Ababa University Faculty of Veterinary Medicine, (will be awarded in June 20, 2009)

Other Trainings

- Training on sheep and goat management by FARM Africa January 2000
- Training on Apiculture Sponsored by UNICEF Agriculture, June1995 Wolayta Sodo
- Training on advanced goat health management sponsored by FARM Africa July 2001 Debrezeit
- Training on Disease outbreak Reporting System Sponsored by Sponsored By National PACE Coordination Office,, Feb 1998 Awassa.
- Training on participatory disease management in Addis Ababa University, Faculty of Veterinary Medicine, by Participatory epidemiology course.

Additional Skills

- ❖ Computer Knowledge in Window, Microsoft Word, Microsoft Excel, and Microsoft Power Point.

- ❖ Good oral and written English communication skill and good oral Somali communication skill.

C. WORK EXPERIENCE

1994-1997G.C Head of Animal and Fishery Resources Development Department of Angacha Woreda, Kembata and Tambaro zone, SNNPR state

1998-2000G.C Head of Animal and Fishery Resources Development and FARM AFRICA Dairy goat project contact person In Angacha Woreda Kembata and Tambaro zone SNNPR state

2001- 2008 G.C. Head of Animal Production And Health Desk

2008-2009 G.C Epidemiologist at Jijiga Regional Veterinary Diagnostic, Investigation and Research Laboratory (JRVDIRL)

D. PAPPERS

May 1992G.C Camel Trypanosomiasis(Literature Review)

July,1993 prevalence of sheep and goat lung worm in Kombolcha and Dessie.

Currently I am working my MVsc thesis on sero-prevalence of pest des petitis ruminants in shoats ,cattle and camel in Jijiga and Shinille zone of Somali region.

References

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10. DECLARATION LETTER:

This thesis is my original work, has not been presented for a degree in any other University and that all sources of material used for the thesis have been duly acknowledged.

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