ADDIS ABABA UNIVERSITY
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STUDY ON CONTACT POINTS FOR THE TRANSMISSION DYNAMICS OF PESTE DES PETITIS RUMINANTS (PPR) IN BASONA WERANA DISTRICT, NORTH SHEWA ZONE, ETHIOPIA

BY

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ABBREVIATIONS

AGID	Agar gel immunoduffussion
CCPP	Contagious Caprine Pleuro Pneumonia
C-DNA Probe	Complementary Deoxy ribonucleic acid
C-ELISA	Competitive Enzyme Linked Immunosorbent Assay
CIRAD-EMVT	International Research Center on Agronomy for developing countries
ELSMA	Ethiopian Livestock Marketing Authority
FAO	Food and Agriculture Organization
GPS	Global Positioning System
Ic-ELISA	Immunocapture Enzyme Linked Immunosorbent Assay
m.s.l.	Meters above sea level
Mabs	Monoclonal antibodies
MoARD	Ministry of Agriculture and Rural Development
OIE	Office International des Epizooties
PA(s)	Peasant Association(s)
PANVAC	Pan African Veterinary Vaccine Center
PPR	Peste des petits ruminants
PPRV	Peste des petits ruminants virus
RPV	Rinderpest virus
RT-PCR	Reverse Transcriptase Polymerase Chain Reaction

ABSTRACT

A study to establish contact points of Peste des Petits Ruminants (PPR) disease in small ruminants was conducted in Basona Werana District, North Shewa Zone, Ethiopia, from October 2006 to April 2007. This study was mainly designed for the identification of the contact of small ruminants at grazing and water points, on the way to the market and through *rebi* practice and contacts with wild ruminants. Further more, other supplementary data on the socioeconomic values of these animals were assessed. The Result of the study indicated that higher numbers of sheep are kept by the households than goats, suggesting that sheep are the dominant species raised in the study area compared to goats. In the present study, farmers sell small ruminants for a number of reasons. The cash obtained from the sale is used for acquiring goods and services which they do not produce themselves, in order to satisfy a variety of needs ranging from food items, clothing, medication, and schooling to the purchase of breeding stock and other production inputs and supplies. Out of the total herd owners interviewed, most farmers sold animals for purchasing clothes 303 (29.02%) followed by food for the family 224 (21%). Other owners used the cash for purchasing fertilizer 173(16.57%), animal feed 59 (5.65%) and for paying land taxation 143(13.7%), school fee (105(10.06%) and others. Out of 101 farmers who bought animals, 45 (44.55%) of the respondents used for own consumption followed by breeding reasons 42 (41.60%) and 14(13.86%) for profit. This shows that small ruminants are mainly used for immediate needs and investment.

Animal movement through the practice of *rebi*, herd contact at grazing and water points, the mixing of animals on the way to the market and at market site are the most important PPR risk factors identified. *Rebi* practice (the caring of animals by another person) is one of the traditional livestock rearing system practiced by farmers in the study area to fulfill the economic and social needs. The overall proportion of *rebi* practice in the study area was 36(4.5%). Animal movement from other districts to the study area by *rebi* practice might lead to the transmission of Peste des petitis ruminants (PPR) disease. Information recorded on such kind of animal movement was not available. The livestock production in the present study area predominantly depends on communal grazing lands. The finding of the study indicated more herd contact at communal grazing land (10.2%) compared to privately owned grazing points

(3.5%). Communal grazing increases the contact rate among animals and in cases where PPR out break has not been recognized in time and hence the disease will spread fast among the susceptible population. Contact of small ruminants at water points in the study area with herds from other PAs was identified in both permanent river and spring water 9.62% and 10.2%, respectively. The overall herd contact at water points was 10.34 %. The increased contact rate among animals at water points suggested that susceptible animals can therefore acquire infection from these points. Out of 737 farmers visited the markets, 390(53.0%) was found mixing their animals on the way to the market with herds of other Peasant associations or districts. Small ruminants trading at markets or their movement on the way to markets where animals from different sources are brought into close contact with one another, affords increased opportunities for PPR transmission. The role of wild life in the epidemiology of PPR in Ethiopia is not known. Bush Duiker and Klipspringer were the wild ruminants identified in the study area. About 36 (78.3) and 18(81.8%) of the respondents have identified Bush Duiker only, 6(13.0%) and 3(13.3%), Klipspringer only, and 4(8.7%) and 1(4.5%) both wild ruminants around their grazing and water points respectively. The overall proportion of these wild ruminants observation was higher at grazing point (57.5%) compared to water point (27.5%). The mean distance at which wild ruminants (Bush Duiker and Klipspringer) was observed from the water point and grazing point was 162.27$\pm$27.23 and 125$\pm$16.01, respectively. The mean frequency/week for the observation of these wild lives were 2.5 $\pm$0.16 and 2.55 $\pm$1.25 from the grazing and water points, respectively. In general, the identification of contact points for PPR transmission helps in adopting animal movement control in disease spread and in designing modeling of the disease and its control strategy.

Keywords: Contact points, sheep and goats, PPR transmission, Animal movements, wild ruminants.

1. INTRODUCTION

The domestic ruminant population in sub-Saharan Africa as estimated by Winrock International (1992) consists of 127 million sheep and 147 million goats, which make up 26% of the world's small ruminant population. Small ruminants are playing a significant role in the socio-economic development of the majority of African countries.

Unlike large animals, sheep and goats are advantageous interms of feasible income generation, easy manageability and remarkable adaptability to diversified environmental factors in different production systems. They are important sources of meat, milk, skin and wool and fibers and provide manure for maintaining soil fertility (Alemayehu and Fletcher, 1991).

Raising small ruminants is regarded as relatively easy with minimal input and low maintenance costs coupled with their ability to maximize available feed resources. In most cases it is basically a secondary activity that employs low levels of family resources (Nwafor, 2004).

Ethiopia is endowed with large livestock resources, with small ruminants assuming great share in the socio-economic activities of about 85% of the population (FAO, 1993). The highlands of Ethiopia, especially the central parts with altitudes of greater than 1500 meters above sea level are known for sheep production. They sustain 92% of the human, 75% of sheep and 28% of goat population of the country. In Ethiopia, sheep and goats cover 30% of domestic meat consumption and generate cash income from exports of meat, live animal and skin (Alemayehu and Fletcher, 1991). The small ruminant population of Ethiopia is estimated to be 24 million sheep and 18 million goats (ELSMA, 2002). However, the full exploitation of these resources is hindered due to a combination of factors such as drought, poor genetic potential of the animals, traditional system of husbandry and the presence of numerous prevalent diseases (Ibrahim, 1998).

Animal diseases have a significant impact on household food security. Some of these diseases wipe out the entire herds and threaten the livelihoods of the farmers. They contribute to a general decline in the productivity and have been determinant factors for poverty in rural

communities. The major causes of economic loss and poor productivity in livestock is the prevalence of wide ranges of diseases including peste des petits ruminants (PPR) PPR is one of the most important diseases of small ruminants in Africa, where poor farmers with subsistence agriculture rely on these animals for their livelihood and food security (Abraham, 2005).

Peste des petits ruminants (PPR) is a highly contagious animal disease responsible for high morbidity and mortality in sheep and goats and in some small wild ruminant species. The huge number of small ruminants, which are reared in the endemic areas, makes PPR a serious disease threatening the livelihood of poor farmers (Diallo, *et al.,* 2007).

It was first described in Cote d`Ivore in 1942 (Diallo, 1997) and since then it became endemic in several countries in Africa, the Arabian Peninsula, Middle East and India (Taylor, 1984; Wamwayi *et al.,* 1995; Shaila *et al.,* 1996; Govindarajan *et al.,* 1997).

PPR is caused by the genus *Morbillivirus* of the family Paramyxoviridae, which also includes measles, canine distemper and viruses of marine mammals. The virus is antigenically related to rinderpest virus, which infects cattle and other large ruminants (Barrett *et al.*, 1993).

PPR virus isolates can be grouped in to four distinct lineages (I, II, III, and IV) on the basis of partial sequence analysis of the fusion (F) protein genes; lineage III is the lineage found in Ethiopia.

The disease is transmitted by direct contact between animals and its incidence is associated with introduction of new animals into flocks and aggravated with close confinement (Taylor, 1984). The appearance of clinical PPR in the herd may be associated with the history of recent movements, gathering together of different species and ages of livestock and contact with trade or nomadic animals around grazing, watering and/or housing points (FAO, 1999).

Clinical disease is characterized by high fever, ocular and nasal discharge, pneumonia, necrosis and ulceration of the mucous membrane and inflammation of the digestive tracts

leading to severe diarrhea. Goats are affected severely but sheep undergo a mild form of the disease (Lefevre and Diallo, 1990) and cattle are only sub clinically infected. Man is not at risk (Hamdy, 1998). Recently, PPR antigen was detected in an outbreak of respiratory disease in camels (Roger *et al.*, 2000).

Peste des petits ruminants is considered to be one of the main constraints in improving productivity of small ruminants in the regions where it is endemic (Ilkede, 1983). It is of great economic importance on the basis of mortality, morbidity, losses through body wastage, poor feed efficiency, loss of meat, milk and milk products and offspring (Nawathe, 1984; Hamdy, 1998). Morbidity and mortality risks in small ruminants vary but can be as high as 100 and 90%, respectively.

In Ethiopia, PPR was suspected on clinical grounds to be present in goat herds in Afar region in 1977 (Pegram and Tareke, 1981). However, the presence of the virus was only confirmed in 1991 with cDNA probe in lymph nodes and spleen specimens collected from an outbreak in a holding land near Addis Ababa. The disease in this outbreak caused more than 60% mortality and it was characterized by ocular and nasal discharges, mouth lesions, pneumonia, gastroenteritis and diarrhea (Abraham *et al*., 1991; Roeder *et al*., 1994). In a serological survey carried out in 1997 at Debre Zeit abattoirs high prevalence of antibodies was reported (Yayehrade, 1997). The national serosurveillance carried out in 1999 has indicated seroprevalence of 5.7% in goats and sheep from different regions of the country (MoARD, 2004). The etiologic agent of PPR was isolated from tissue samples collected from suspected outbreaks of PPR in Arsi Zone of Ethiopia (Gelagay *et al.*, 2003). Abraham *et al.* (2005) have reported the overall seroprevalence of PPR as 3% in camels, 9% in cattle, 9% in goats and 13% in sheep in different parts of Ethiopia. Recent findings indicated an overall seroprevalence of the disease (26.0%) in Basona Werana and Kewet districts of North Shewa Zone, (Alemayehu, 2006), (0.94%) in Awash Fentale of Afar region (Bahiru, 2006) and (6.1%) in Gewane district of Afar region (Tesfaye, 2006).

Much of the investigations on PPR disease under Ethiopian condition was emphasized on the determination of its prevalence. The study on identifying the contact points of small ruminants

with each other from other areas and with wild ruminants is the first of its kind in Ethiopia in general and in Basona Werana district in particular.

The present study, therefore, discusses the following objectives:

OBJECTIVES:

1. To establish contact points of small ruminants for the transmission dynamics of PPR disease
2. To generate baseline data for modeling of PPR disease and its control strategies.

2. LITERATURE REVIEW ON PESTE DES PETITS RUMINANTS

2.1. Definition and History of PPR disease

Peste des petits ruminants (PPR) is an infectious, contagious viral disease of domestic and wild ruminants (Furley *et al.,* 1987). It is characterized by mucopurulent nasal and ocular discharges, necrotizing and erosive stomatitis, enteritis and pneumonia. It has a very high rate of morbidity and mortality and is one of the diseases registered by OIE under list "A" disease category (Lefvre and Diallo, 1990; Jones *et al.,* 1993). It was first identified in Ivory Coast by (Gardenne and Lalane, 1942, cited by Taylor, 1984) in West Africa where it used to be described with such varied name as Kata, Psuedo-rinderpest, Pneumoenteritis complex and stomatitis-pneumenteritis syndrome (Hamdy *et al.,* 1976, Braide, 1981).

2. 2. Aetiology

2.2.1. Classification

PPR virus is classified in the order Mononegavirales, the family Paramyxoviridae and the genus *Morbillivirus* which also includes rinderpest virus, canine distemper virus, human measles virus and viruses of marine mammals i.e. phocine distemper virus of seals and the cetacean *Morbillivirus* isolated from dolphins and porpoises (Gibbs *et al.,* 1979; Barrett *et al.,* 1993).

2.2.2. Viral structure and genome

Morbilliviruses genome is divided into six transcriptional units encoding two non-structural protein (V and C protein) and six structural proteins that include the two surface glycoprotein; hemagglutinin (H-protein) and fusion protein (F protein), the matrix protein (M), nucleoprotein (N) and the phoshoprotein (P) (Barret *et al.,* 1993, Haffar *et al.,* 1999). The

phosphoprotein (P) forms the polymerase complex with the large protein (L) of RNA genome with which is associated and functions in transcription and replication (Diallo *et al.,* 1994, Barret *et al.,* 1993, Haffar *et al.,* 1999). The gene order from 3' to 5' on the genome is N-P-M-F-H-L (Diallo, 1990; Mahapatra *et al*., 2003).

The sequencing of PPR N-protein revealed that PPR virus consists of 1662 nucleotide long. The N-protein encapsides and protects the RNA (Diallo *et al.,* 1987, Barret *et al.,* 1993). The M-protein appears to play a central role in the formation of the virions which are liberated from the infected cells by budding; defects in the M-protein leads to persistent, non productive virus infection that was observed in measles and CD (Haffar *et al.,* 1999, Barret *et al.,* 1993). Gene encoding the matrix protein of the PPR consists of 1466 nucleotide long with an open reading frame capable of encoding basic protein of 335 amino acids (Haffar *et al.,* 1999).

The hemagglutinin and fusion proteins are responsible for attachment and fusion of host cell membrane respectively. The H-protein determines host specificity because it recognizes receptors on the cells membrane and anchors the virus to them, so antibodies directed against it prevents attachment (Devireddy *et al.,* 1999, Meyer and Diallo, 1999). The F-protein fuses the virus envelope to the host cell membrane, releases the nucleocapsid and facilitates the spread of the virus from cell to cell by inducing synctia. Therefore, both F-protein and H-protein play a vital role in pathogenesis and cytopathology of virus and infection initiation (Barret, 1999, Devireddy e*t al.,* 1999, Meyer and Diallo, 1995, *Haffar et al.,* 1999). The H-protein of PPR induces neutralising antibodies and it is assumed to be the least antigenically cross-reactive protein (Barret *et al.,* 1993). With exception of MV H-protein, the PPRV H-protein is the only morbillivirus that hemagglutinates RBCs while the F-protein lysis chicken RBCs (Devireddy *et al.,* 1999).

PPR virus is sensitive to ether and acid and can easily be destroyed with lipid solvent (Lefevre and Diallo, 1990). It can be cultured and produce cytopathic effects with a clock faced syncytia in a variety of cell cultures including sheep and goats kidney, vero cells and bovine kidney (Gibbs, *et al.,* 1979). However difficulties in propagation and assessing of syncytia in bovine kidney cell culture was reported (Taylor and Abgunde, 1979). Sheep and goats cells

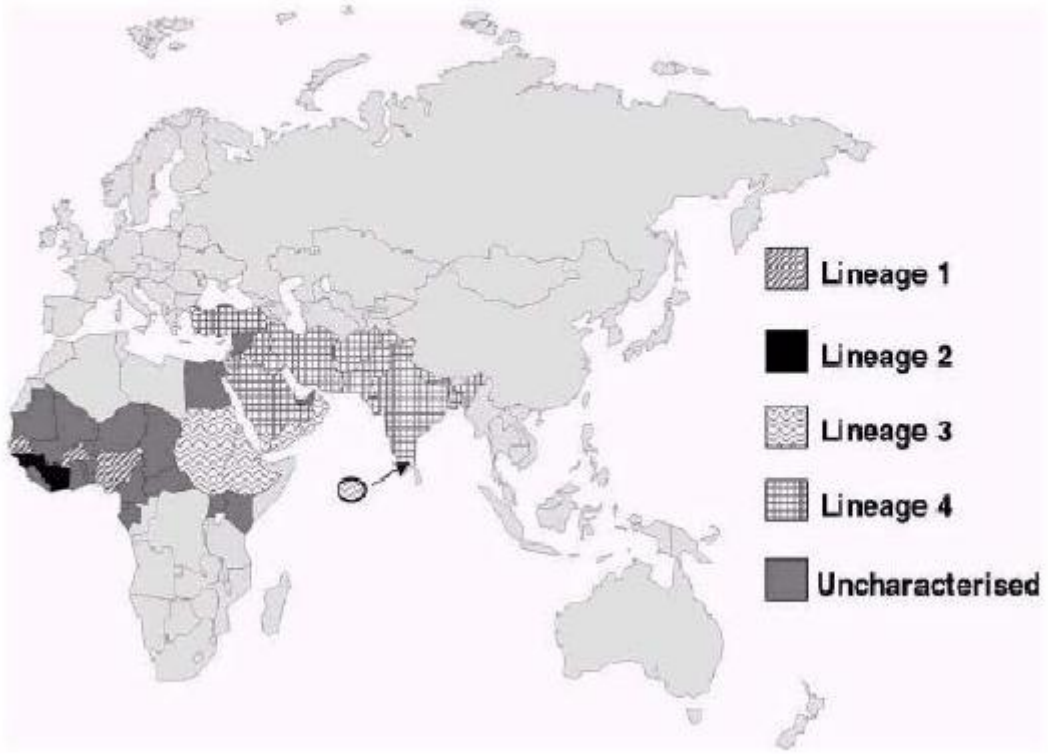
cultures were considered the most sensitive for isolation and assay of the virus (Gibbs *et al.,* 1979, Taylor and Abugunde, 1979). However, Rossiter *et al. (*1985) reported that CPE is much easier to detect in vero cell when used in micro-neutralization.

The immunological relationship between PPR and RP was demonstrated by precipitating antibodies, compliment fixing antibodies and cross protection between the two viruses (Hamdy *et al.,* 1976, Gibbs *et .al,* 1979). However high neutralizing antibodies titers occur in the homologous system rather than in heterologous system (Taylor and Abugunde, 1979). A degree of cross protection between CD and PPR was reported while no cross protection existed between PPR and MV (Gibbs *et al.,* 1979).

2. 3. Epidemiology

2.3.1. Geographical distribution

PPR, being first described in Côte d'Ivoire in 1942, presently occurs in most African countries situated in a wide belt between the Sahara and Equator, the Middle East and the Arabian Peninsula, in countries including Iran, Iraq, Israel, Jordan, Kuwait, Lebanon, Oman, Saudi Arabia, the United Arab Emirates and Yemen. Outbreaks of PPR are now known to be common in India, Nepal, Bangladesh, Pakistan and Afghanistan (Taylor, 1984; Lefevre and Diallo, 1990; FAO, 1999). The virus was isolated from Senegal, Ivory Coast, Guinea (Shaila *et al.,* 1996), Nigeria (Taylor and Abugunde, 1979), Sudan (ElHag and Taylor, 1984), and Saudi Arabia (Abu Zein *et al.,* 1990).



Source: FAO, 1999; Dhar *et al.(* 2002)

Figure 1: Global distribution & Lineages of PPR

Serological evidence were detected in Syria, Niger and Jordan (OIE, 2000), while the virus presence was confirmed with cDNA probe and indirect fluorescent antibody technique in Ethiopia (Roeder *et al.,* 1994) and Eritrea (Sumption *et al.,* 1998) respectively.

2.3.2. Phylogeny of the virus

Peste des petitis ruminants virus is genetically grouped into four distinct lineages (I, II, III, and IV) on the basis of partial sequence analysis of the fusion (F) protein gene (Figure1). 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 (Tamil Nadu). The fourth lineage is

confined to exclusively the Middle East, Arabia and the Indian sub-continent, reaching east as far as Nepal and Bangladesh (Shaila *et al.,* 1996; Dhar *et al.*, 2002).

2.3.3. Disease pattern and season of occurrence

The disease may occur all round the year with high prevalence during the rainy season because of kidding pattern, which has no definitive season. Increase incidence during the rainy season is the reflection of large number of susceptible young kids, which had lost their maternal immunity, being recruited into the existing flocks during a period of adverse climatic conditions (Obi and Patrick, 1984).

2.3.4. Susceptible species

Peste des petits ruminants is primarily a disease of goats and sheep but they are not always affected simultaneously. For example, in Africa PPR is seen most commonly in goats, while in Western and South Asia sheep are usually the most noticeable victims. The disease can, however, strike both species with equally devastating consequences (Taylor, 1984; Lefevre and Diallo, 1990; Ozkul *et al.,* 2002). Sheep possessed an innate resistance to the clinical effects of disease, but occasional field strains could overcome this resistance and produce high mortality (Taylor, 1984).

Cattle and pigs are susceptible to infection with PPRV, but they do not exhibit clinical signs. Such subclinical infections result in seroconversion, and cattle are protected from challenge with virulent RPV. However, cattle and pigs do not play a role in the epizootiology of PPR because they are apparently unable to transmit the disease to other animals (Furley *et al.*, 1987).

Clinical disease has also been reported in wildlife resulting in deaths of *Gazellinae* (D*orcas gazelle*), *Caprinae* (Nubian ibex and Laristan sheep), and *Hippotraginae* (gemsbok). Experimentally, the American white-tailed deer (*Odocoileus virginianus*) is fully susceptible.

The role of wildlife on the epizootiology of PPR in Africa remains to be investigated (Furley *et al.,* 1987). The detection of antibodies against PPRV in African grey duiker (*Sylvicapra grimmia*) probably implicates the role of wildlife in the epizootiology of PPR in Nigeria (Ogunsanmi *et al.,* 2003; Elzei *et al.,* 2004). Subclinical involvement of Nigale (T*ragelaphinae*) was suspected .A report from Saudi Arabia indicated that PPR was suspected on clinical and serological base in Gazaelle and deer (Abu Zein *et al.,* 1990). Isolation of PPR virus from a domestic buffalo (*Bubalus bubalis*) in Nadu Tamil state in Southern India (Govidarajan *et al.,* 1997) extended the host range of PPR to include the domestic buffalo. According to Abraham (2005),camel was infected by the virus but without showing the clear disease. Serological evidences for the presence of PPR antibodies were detected in 4.2% of 142 healthy camels with sero-neutralization test (Ismail *et al.,* 1995).

2.3.5. Age and Breed factor

Age Factor

The prevalence of antibodies to PPRV was noted to differ between species (i.e. sheep versus goats) and age groups. Goats are exposed at an earlier age than sheep and more susceptible to infection with PPRV (Singh *et al.,* 2004a). Kids over four months and under one year of age are more vulnerable to the disease than adults (Radostitis *et al.,* 2000). Sero-prevalence of 23% and 24% in sheep and goats respectively was reported from Oman, with an increase in antibodies prevalence with age a thing that could be taken as indication of the endemicity of the disease (Taylor *et al.,* 1990). Serological data from Nigeria revealed that antibodies occur in all age groups from 4-24 months indicating a constant circulation of the virus (Taylor, 1984), a finding that was established in Oman where infection persisted on a year round basis maintaining itself in the susceptible yearling population (Taylor *et al.,* 1990). Therefore an increased in incidence reflects an increase in number of susceptible young goats recruited into the flocks rather than seasonal upsurge in the virus activity since its upsurge depend on the peak of kidding (Taylor *et al.,1990*). Moreover, the susceptibility of young animals aged 3 to 18 months was proved to be very high; being more severely affected than adults or unweaned animals (Taylor e*t al.,* 1990).

Breed Factor

Breed susceptibility among the hosts has also been reported. The Sahelian breeds of goats and sheep are believed to be more resistant than the Dwarf breeds of humid and sub-humid Africa (Nawathe, 1984; Radostitis *et al.,* 2000). 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 (ElHag and Taylor, 1984). No significant difference in the case fatality rate and attack rate between sex groups was observed (Opasina and Putt, 1985).

2.3.6. Mode of transmission

PPR infection spreads most often by direct contact (Braide, 1981). The discharges from eyes, nose and mouth, as well as the loose feces of sick animals contain large amounts of the virus (Bundza *et al.,* 1988). Fine infective droplets are released into the air from these secretions and excretions, particularly when affected animals cough and sneeze. Aerosol transmission would enable the disease to spread rapidly in large groups of animals. Successful transmission therefore requires close contact between sick and healthy animals (Hamdy, 1998; FAO, 1999). Indirect transmission seems to be unlikely in view of the low resistance of the virus in the environment and its sensitivity to lipid solvent (Lefevre and Diallo, 1990). No carrier state is known to exist (OIE, 2002).

2. 3.7. Predisposing factors

The appearance of PPR outbreaks in susceptible herds is facilitated by the following factors (FAO, 1999; Wous, 1995; Taylor, 1984, Braide, 1981).

- History of recent movement or gathering of sheep and /or goats of different ages with associated changes in housing and feeding.

- Contact with sheep and/or goats that had been sent to market but returned in a closed village flock.
- Introduction of recently purchased animals.
- Stress due to change in weather such as the onset of rainfall, changes in humidity or temperature.
- Contact with trade or nomadic animals near housing and/or at grazing, and watering sites.
- A change in the husbandry system because of the disease being more common in pastoral system than intensive type of husbandry system.

2.3.8. Economic significance

Peste des petitis ruminants is classified as an OIE notifiable disease and is considered to be one of the main constraints to improving productivity of small ruminants in the regions where it is endemic (Ilkede, 1983). It is of great economic importance on the basis of mortalities, morbidity, losses through body wastage, poor feed efficiency, loss of meat, milk and milk products and offspring (Nawathe, 1984).

A morbidity rate of 80-90 percent and a case fatality rate of 50-80 percent are not uncommon particularly in goats. Young animals 4 to 8 months of age have more severe disease, and morbidity and mortality are higher. When associated with other diseases, such as capri pox, mortality can approach upto 100% (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 the 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 million US$) (Bandyopadhyay, 2002).

Programs aimed at controlling PPR in ruminants have cost farmers, governments and aid agencies large sums of money for several years. A comprehensive quantification of the

economic importance of PPR, however, 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, and failure in the definitive clinical diagnosis (Nawathe, 1984).

There are considerable regional differences in the epidemiological pattern, which influence the methods of assessing the economic impact. In the Guinean zones of the African continent where PPR occurs in epizootic form, its negative economic consequence is severe because of the high morbidity and mortality. On the other hand, in endemic regions there may be possibility of concurrent bacterial infections and its economic impact on animal production is certainly high (Scott, 1981).

2.4. Pathogenesis

The causal virus has a particular affinity for lymphoid tissues and epithelial tissue of the gastrointestinal and upper respiratory tracts, in which it produces characteristic lesions (Hamdy, 1998). PPR virus inhaled with infected droplets penetrates the retropharyngeal mucosa and multiplies in the tonsils and regional lymph nodes. Then it enters the blood circulation through mononuclear cells causing viremia that disseminates the virus to other lymphoid organs, the lungs and epithelial cells of mucous membrane where it multiplies and causes the cytopathology responsible for the development of lesions and the disease (Bundza *et al.,* 1998; Radostitis *et al.,* 2000).

Some virulent strains cause death from severe diarrhea with subsequent dehydration and electrolyte imbalance, particularly in kids and lambs resulting in high mortality. In other cases, concurrent diseases like pneumonic pasteurellosis and coccidiosis facilitate the death of the animal (Radostitis *et al.,* 2000).

A study showed that PPR virulent virus causes marked immunosuppression as evidenced by leucopoenia, lymphopenia, and reduced early antibody response to both specific and nonspecific antigens. This observation was predominant particularly during acute phase of the disease, 4 to10 days post-infection (Rajak *et al.,* 2005).

2. 5. Clinical signs

The disease usually appears in the acute form, with an incubation period of 4 to 5 days followed by a sudden rise in body temperature up to 41°c (FAO, 1999). Affected animals appear ill and restless and have a dull coat, dry muzzle, and depressed appetite. From the onset of fever, most animals have a serous nasal discharge, which progressively becomes mucopurulent. The discharge may remain slight or may progress, resulting in a profuse catarrhal exudates, which crusts over and occludes the nostrils. At this stage, animals have respiratory distress, and there is much sneezing in an attempt to clear the nose. Small areas of necrosis may be seen on the visible nasal mucous membranes. As with the nose, there may be profuse catarrhal conjunctivitis resulting in matting of the eyelids (Hamdy, 1998).

Necrotic stomatitis is common. It starts as small, roughened, red, superficial necrotic foci on the gum below the incisor teeth. These areas may resolve within 48 hours or progressively increase to involve the dental pad, the hard palate, cheeks and their papillae, and the dorsum of the anterior part of the tongue. Necrosis may result in shallow irregular nonhemorrhagic erosions in the affected areas of the mouth and deep fissures on the tongue. Necrotic debris may collect at the oral commissures, and scabs may form along the mucocutaneous junction of the lips. There may be excessive salivation but not to the extent of drooling (Hamdy, 1998; FAO, 1999).

At the height of development of oral lesions, most animals manifest severe profuse diarrhea. As it progresses, there is severe dehydration, emaciation, and dyspnea followed by hypothermia, and death usually occurs after a course of 5 to 10 days. Bronchopneumonia, evidenced by coughing, is a common feature in the later stages of PPR. Pregnant animals may abort. Secondary latent infections may be activated and complicate the clinical picture (Hamdy, 1998; Taylor, 1984; FAO, 1999). According to Lefevre and Diallo (1990), the disease has three forms: Hyperacute, Acute, Sub clinical or inapparent form**s.**

2.5.1. Hyperacute form

It occurs frequently in goats in which elevated temperature is the first sign after two days incubation period, followed by apathy, anorexia, nasal and lachrymal discharges. There may be constipation during the first days that gives the way to profuse diarrhea. The prognosis is not good and it leads to death within 5-6 days.

2.5.2. Acute form

Incubation period is 3-4 days and the initial stages are identical with those of hyperacute form accompanied with sero-mucous nasal discharges which become mucopurulent. Erosive and ulcerative involvement and complication, which depend on the bacterial superinfection particularly P.haemoytica or P.multocida type A, may be observed. Also latent parasitic infection such as coccidiosis, piroplasmosis and trypanosomosis may be reactivated.

2.5.3. Sub clinical or inapparent forms

This form seems to be prevalent in a certain regions where the local breeds and animals have innate resistance. The disease course is 10-15 days with inconstant symptom. At the late stage pulmonary involvement usually occurs with a dry cough that rapidly become loose. Some papules and pustules similar to those of contagious ecthyma may appear, but the infection in general is complicated by respiratory diseases.

2. 6. Post mortem findings

The carcass of an affected animal is usually emaciated, the hindquarters soiled with soft/watery feces and the eyeballs sunken. The eyes and nose contain dried-up discharges. The lesions include dirty-white, false membranes of the buccal cavity, erosions on the lips, gums, soft and hard palates, tongue, cheeks and the esophagus. Erosive lesions may extend form the mouth to the reticulo- rumen junction. Characteristic linear hemorrhages or zebra stripes occur

in the large intestine, commonly at the caeco-colic junction, but they are not consistent features. Necrotic or hemorrhagic enteritis is usually present (FAO, 1999).

Lymph nodes are enlarged, the spleen may show necrotic lesions, and there is an apical pneumonia (FAO, 1999). Histopathologically, the pneumonocytes and epithelial cells of the ileum had eosinophilic cytoplasmic and nuclear inclusions and formation of giant cells (Bundza *et al.*, 1998).

2.7. Differential Diagnosis

Peste des petits ruminants is frequently confused with other diseases that present fever and grossly similar clinical signs, especially when it is newly introduced. Therefore, PPR must be differentiated from rinderpest, pasteurellosis, contagious echytma (orf), foot and mouth disease, and coccidiosis (FAO, 1999).

2.8. Laboratory Diagnosis

In the field, tentative diagnosis can be made on the basis of clinical, pathological, and epizootiological findings (FAO, 1999). However, laboratory confirmation is an absolute requirement particularly in areas or countries where PPR has not previously been reported.

2.8.1. Specimen collection

Specimen can be collected on live animals including swabs of the eye (conjunctival sac), nasal secretions, and mouth and rectal lining, clotted and whole blood with anticoagulant) or sampling during post-mortem examination of fresh and preserved samples of tonsil, tongue, spleen, lymph nodes, affected areas of the alimentary tract mucosa. All samples should be shipped fresh (not frozen) on ice within 12 hours after collection (FAO, 1999).

2. 8.2. Virus isolation

Peste des petits ruminants can be confirmed by virus isolation and identification. PPR virus can be grown on different type of cell culture but virus isolation for laboratory diagnosis is carried out routinely in primary lamb kidney cells culture and Vero cells. If cytopathic effect is evident the neutralization and Ic-ELISA are used to confirm the identity of the isolates (Furley *et al.,* 1987; Shaila *et al.,* 1996). Even when diagnosis has been carried out by rapid techniques, the virus should always be isolated from field samples in tissue cultures for further studies (Lefevre and Diallo, 1990). For successful isolation, samples must be collected during the hyperthermic phase and submitted to the testing laboratory in ice. The most widely used cell culture systems are primary lamb kidney (Taylor and Abegunde, 1979) and Vero cells (Hamdy *et al*., 1976).

The sensitivity of virus isolation technique could be increased when the virus is grown in lamb and goat kidney cells (Taylor, 1984). Vero cells are however, widely used for their continuity and low liability of contamination. PPRV has also been adapted to grow in other continuous cell lines including BHK-21 (Lefevre and Diallo, 1990). Vero cells, derived from African green monkey kidney are currently most widely used cell line for PPRV and RPV. Appearance of cytopathic effects (CPE) may require at least 8-10 days or several blind passages.

In Vero cells, the cytopathic effects (CPE) produced by PPRV consist of cell rounding, clumping into typical grape-like clusters, and formation of small syncytia and appearance of long fine often anastomosing "spindle cells". Like other *morbilliviruses*, PPRV produces eosinophilic intracytoplasmic and intranuclear inclusion bodies both in primary cells and continuous cell lines (Hamdy *et al*., 1976). Once isolated in cell culture, a candidate PPRV may be identified by one of the three procedures:

1. *Animal inoculation*: PPR causes clinical disease in goats and sheep but not in cattle (Diallo *et al*., 1995).

2. *Reciprocal cross neutralization (differential neutralization):* PPRV is neutralized by both PPR and RPV reference sera, but is neutralized at greater titer with the homologous serum (Taylor and Abegunde, 1979).
3. *Molecular techniques:* cDNA probe, (Pandey *et al*., 1992), electrophoretic profile in polyacrylamide gel (PAGE) (Diallo *et al*., 1987) and PCR (Barret *et al*., 1993; Forsyth *et al*., 1995; Couacy-Hymann *et al*., 2002).

2. 8.3. Detection of viral antigens and nucleic acid

PPR antigen can be detected by using immunocapture ELISA, Counter immunoelectrophoresis, Agar gel immunodiffusion test (AGID), cDNA probe and reverse transcription polymerase chain reaction (RT-PCR). Of these Ic-ELISA and PCR are the most sensitive and specific which avoids the problem of cross-reaction between rinderpest and PPR virus (Diallo *et al.*, 1995).

Immunocapture ELISA

The immunocapture ELISA allows a rapid differential identification 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 (OIE, 2000). 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 (Libeau *et al.*, 1995). 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.

Complementary DNA probe

Complementary DNA probe detects virus nucleic acid (Diallo *et al.*, 1995). The principle of this technique is the ability of two complementary single stranded nucleic acids to associate and form a stable duplex through the formation of a large number of hydrogen bonds. To detect this association, one of the nucleic acid strands (the probe) has to be lebelled in some

way. This technique has been used to differentiate rinderpest and PPR virus infections using cDNA clones corresponding to their respective N genes as probes. cDNA directed against the matrix protein, fusion protein and phosphoprotein gene were found to cross hybridise to a much greater extent and were not suitable for use as discriminating probe (Diallo *et al.,*1989). Unfortunately this hybridization can not be used widely because it can only be used for fresh specimens in addition to the short half life of [P^{32}] and the risk of handling isotopes (Diallo *et al.,* 1995, Pandey *et al.,* 1992). Therefore probes using non radioactive materials such as biotin (Pandey *et al*., 1992) or digoxin (Diallo *et al.,* 1995) as a label were developed. The biotin labeled cDNA was found to be as sensitive as the one use the radioactive label and more rapid in differentiation between PPR and RP (Pandey *et al.,* 1992). However it was reported that the expected sensitivity had never been obtained using non-radioactive labels (Diallo *et al.,* 1995).

Agar gel immunodiffusion test

Agar gel immunodiffusion test is widely used and can detect 42.6% of ante mortem specimens and necropsy specimens (Obi and Patrick, 1984). These techniques are based on the ability of antibody to form precipitin lines specifically with the antigen. After optimal development of precipitin patterns, the plate can be read directly or stained for considerable improvement in sensitivity (Talwar, 1983). Agar gel immunodiffusion (AGID) is a very simple and inexpensive test that can be performed in any laboratory and even in the field. It can be used to test the presence of both antigen and antibodies (OIE, 2004). One of the important advantages of this test is that it is highly specific (92%), though it cannot differentiate between PPR and RP (Obi and Patrick, 1984).

Indirect fluorescent antibody test (IFAT) was more sensitive than the staining for syncytia in the detection of viral antigen (Sumption *et al.,* 1998)

Sandwich ELISA

A monoclonal antibody-based sandwich enzyme-linked immunosorbent assay (S-ELISA) was developed for specific detection of peste des petits ruminants virus. The test employed monoclonal antibodies directed against the H-protein of PPRV was found to be highly

sensitive in detection of antigen in tissues and secretions of infected goats. It is a suitable alternative to virus isolation (Saliki *et al.,* 1994).

Reverse transcription Polymerase chain reaction (RT-PCR)

Reverse transcription-polymerase chain reaction test (RT-PCR) using phosphprotein (P) universal primer and fusion (F) protein gene specific primer sets to detect and differentiate between PPR and RP were described (Foryth and Barret, 1995). 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. It could be used for diagnosis once the RNA has been extracted from the sample. After extraction the RNA genome of PPR virus has first to be copied into cDNA using reverse transcriptase enzyme prior to amplification. Another set of primers has been prepared based on the sequence of the 3' end of the gene coding for the N protein (OIE, 2000). With these primers, specific amplification of 300 base pair fragment is possible. The specificity of the amplified fragments is confirmed by restriction enzyme digestion (Rsa 1 cuts only RP and PPR virus fragments) or by hybridization with a non-radioactive oligonucleotide used as a probe. The PCR is sensitive and results are obtained in 5 hour including the RNA extraction (OIE, 2000). PCR can be used to amplify the virus RNA in gum and lachrymal swabs transported in phosphate buffer containing penicillin, streptomycin and fugizone, whole blood and postmortem specimens.

RT-PCR has been shown to be useful for the rapid detection of *Morbillivirus*-specific RNA in samples submitted for laboratory diagnosis (Shaila *et al*., 1996).

Reverse transcription-polymerase chain reaction tests (RT-PCR) using phosphoprotein (P) universal primer and fusion (F) protein gene specific primer sets to detect and differentiate between PPR and RP The procedure is sensitive and rapid, taking only 5 hours to complete, including the RNA extraction (Diallo *et al*., 1995; Forsyth and Barret, 1995; Couacy- Hymann *et al*., 2002).

2.8.4. Antibody detection

The most commonly employed techniques are c-ELISA, AGID, and virus neutralization tests (Diallo *et al.,* 1995). Virus neutralization test is sensitive and specific, but it is 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 (OIE, 2000). Serum against either PPR or RP may neutralize both viruses, but would neutralize the homologous virus at a higher titer than the heterologous virus.

The c-ELISA test developed can easily replace virus neutralization tests (VNT) for sero-surveillance, sero-monitoring, diagnosis from paired sera samples and end-point titration of PPR virus antibodies (Singh *et al.,* 2004b). PPR and rinderpest can be differentiated by cross-neutralization test or by c-ELISA, using purified antigens and specific monoclonal antibodies (Renukaradhya *et al.,* 2003).

Because a blocking enzyme-linked immunosorbent assay (B-ELISA), using neutralizing Mabs proved to be nearly as sensitive and specific as the VNT while being simpler and more rapid; it would be an adequate substitute for the VNT for assessing herd immune status and for epidemiologic surveillance (Saliki *et al.,* 1994). Due to their simplicity, high sensitivity, and economy, several competitive enzyme-linked immunosorbent assays (C-ELISAs) and blocking-ELISA (B-ELISA) have been recognized as suitable systems for use for diagnosis and seroepidemiological surveillance. They target the hemagglutinin (H) protein (Singh *et al*., 2004b) or nucleocapsid (N) protein (Libeau *et al.,* 1995).

The C-ELISA procedure consists of at least four incubation steps, including adsorption of the antigen onto a solid phase, competitive binding of a serum-monoclonal antibody (MAb) mixture to the antigen, detection of the MAb bound to the antigen, and the substrate reaction. This rapid C-ELISA is a simple, fast, reliable, and inexpensive tool for diagnostic and epidemiological purposes (Diallo *et al*., 1995).

The relative sensitivity of this C-ELISA to VNT was 94.5%, while the specificity was 99.4%. Both blocking ELISA and C-ELISA detecting anti-H antibodies are based on competition between an anti-H monoclonal antibody (MAb) and serum antibodies, but in case of blocking ELISA the test sera are preincubated with antigen and then incubated with the MAb. The sensitivity and specificity of the H-blocking ELISA were found to be 90.4% and 98.9%, respectively (Saliki *et al*., 1994).

2. 9. The Nature of Immunity

PPRV harbors two major surface glycoproteins; the hemagglutinin (H) and the fusion (F) proteins, which are highly immunogenic and confer protective immunity. The immunized goats develop both humoral and cell-mediated immune responses. Antibodies generated in the immunized animals could neutralize both PPRV and RPV *in vitro* (Sinnathamby *et al.*, 2001). Maternal antibodies in young animals were detectable up to 6 months of age but fell below the protection threshold level at 3.5 and 4.5 months in lambs and kids, respectively (Awa *et al*., 2002).

Peste des petits ruminants (PPR) hyper immune serum was prepared in cattle and used to protect goats against PPR. A durable active immunity was conferred on goats when serum and virulent PPR virus were given simultaneously. Passive immunity lasted for 15 days (Adu and Joannis, 1984).

2. 10. Control and Eradication

2.10.1. Treatment

There is no specific treatment against the disease. However, valuable sick animals in the early stage of the disease should be isolated and given hyperimmune serum, which may be obtained from cattle hyperimmunized against rinderpest. Supportive treatment includes fluid therapy for dehydration (Radostits *et al*., 2000). In addition drugs that control bacterial and parasitic complications may be used to reduce mortality rates. Specifically, oxytetracycline and

chlortetracycline are recommended to prevent secondary pulmonary infections (Saliki *et al*., 1998; OIE, 2002).

2.10.2. Vaccination

Tissue culture rinderpest vaccine

The most practical vaccination against PPR was made using tissue culture rinderpest vaccine. The tissue culture rinderpest vaccine protects goats for at least 12 months against PPR. The vaccine is currently used in many African countries for vaccination against PPR (Taylor, 1979). Kids and lambs born to dams that have been immunized with RP virus tissue culture vaccine have heterologous colostral antibody to PPR virus for up to three months. The use of RP vaccine to protect small ruminants against PPR is now contraindicated because of the production of antibodies to RP, which compromise sero surveillance for RP, and there by the global rinderpest eradication programs (FAO, 1999).

Homologous PPR vaccine

The effective PPR homologous vaccine was developed in 1989. It was developed by serial passage on Vero cells with the PPR, Nigeria 75/1 isolate and it is now the main vaccine used to protect small ruminants against PPR in endemic zones (OIE, 2004). The homologous PPR vaccine developed recently is available at National Veterinary Institute, Debre Zeit, Ethiopia to be used for Africa through the Pan African Veterinary Center (PANVAC). (FAO, 1999). These vaccines can protect small ruminants against PPR for at least three years. The prospects of control using homologous attenuated vaccine are very important because PPR need to be differentiated from rinderpest, particularly as the Global RP Eradication Program proceeds towards the anticipated eradication of rinderpest by the year 2010 (OIE, 2002).

But the homologous PPRV vaccine is a thermo labile vaccine and it is necessary to maintain it in an efficient cold chain, a condition that is sometimes difficult to achieve in many of a developing countries where the disease is endemic. Thus a more heat-stable vaccine would be beneficiary for use in countries with hot climate (Berhe *et al*., 2003).

Dual recombinant vaccines

The thermostable Vero cell-adapted rinderpest vaccine and a recombinant capri poxvirus vaccine containing a cDNA of the peste des petits ruminants virus (PPRV) fusion protein gene protected goats against challenge with a virulent PPRV strain (Romero *et al.*, 1995; Berhe *et al.*, 2003; Diallo, 2003).

Marker vaccines

Taking advantage of the closely relationship between rinderpest and PPR viruses, the attenuated rinderpest vaccine was used in the control of PPR. It is now replaced by the homologous attenuated PPR vaccine. Unfortunately, they do not allow serological differentiation between animals that have been vaccinated and those that have recovered from natural infection With the advent of DNA recombinant technology, efforts are being made to develop effective PPR marker vaccines to enable such differentiation and which would allow countries to implement both vaccination and disease surveillance programmes at the same time (Diallo, *et al.,* 2007). The development of this dual-marker vaccine is the originality of RP/PPR MARKVAC project conducted by CIRAD-EMVT with other collaborators in Europe and Africa.

A work to protect small ruminants against both capripox and PPR using recombinant capripox vaccine where these two economic important diseases share the same geographical distributions is underway (Berhe *et al*., 2003).

2.10.3. Eradication

Eradication is recommended when PPR appears in new areas. Methods that have been successfully applied for RP eradication in many areas would be appropriate for PPR. These should include quarantine, slaughter and proper disposal of carcasses and contact fomites, decontamination, and restriction on importation of sheep and goats from affected areas (Saliki, 1998).

PPR has a very high rate of morbidity and mortality, and effective control of this disease is of economic importance in areas where it occurs. There is no specific treatment for PPR. However, mortality may be decreased by using drugs that control bacterial and parasitic complications (OIE, 2000).

Control of PPR outbreaks relies on movement control (quarantine) combined with the use of focused "ring" vaccination and prophylactic immunization in high-risk populations. The homologues vaccines can protect small ruminants against PPR for at least three years (FAO, 1999). The use of rinderpest vaccine to protect small ruminants against PPR is now contraindicated because its use produces antibodies to rinderpest, which compromises serosurveillance for rinderpest, and thereby the Global Rinderpest Eradication programmes (FAO, 1999). When PPR appears in new areas, eradication is recommended by quarantine, slaughter, proper disposal of carcasses and contact fomites, decontamination, and restrictions on import. The disease can be prevented by not introducing new stock from unknown sources, especially animals bought at livestock markets. In addition, animals returned unsold from markets should be segregated unless the entire herd or flock has been vaccinated (Radostits *et al*., 2000).

2.11. The Epidemiology of PPR in Ethiopia

2. 11.1.Disease History

The disease was probably introduced into Ethiopia in 1989 in the Southern Omo river valley from where it moved eastward to Borena region and then northwards along the Rift valley to Awash (Abraham *et al*., 1991; Roeder *et al*., 1994).

2. 11.2. Disease confirmation

Peste des petits ruminants was suspected on clinical grounds to be present in goat herds in Afar region of Eastern Ethiopia in 1977 (Pegram and Tareke, 1981). Moreover, serological

and clinical evidences were reported by Taylor (1984). However, the presence of the virus was only confirmed in 1991 with cDNA probe in lymph nodes and spleen specimens collected from an outbreak in a holding land near Addis Ababa (Abraham *et al*., 1991; Roeder *et al*., 1994).

In a serological survey carried out in 1997 at Debre Zeit abattoirs high prevalence of antibodies was reported (Yayehrade, 1997). The national serosurveillance carried out in 1999 has indicated seroprevalence of 5.7% in goats and sheep from different regions of the country (MoARD, 2004).

The etiologic agent of PPR was isolated from tissue samples collected from suspected outbreaks of PPR in Arsi Zone of Ethiopia (Gelagay *et al.*, 2003). Abraham *et al* (2005) have reported the overall seroprevalence of PPR as 3% in camels, 9% in cattle, 9% in goats and 13% in sheep in different parts of Ethiopia. The prevalence rate of PPR in Ethiopia reported by different researchers is summarized in (Table 1).

Table 1: Summary of prevalence of PPR in Ethiopia.

Region	Prevalence (%)		Type of Test	Reference
	Goats	Sheep		
Afar	Not Known	Suspected	-	Pegram and Tareke, 1981
Near Addis Ababa	Not Known	Confirmed	c-DNA probe	Abraham *et al.*, 1991
Arsi	53%	33%	Ic-ELISA	Roger and Bereket, 1996
Afar	67%	0%	Ic-ELISA	Roger and Bereket, 1996
North Shewa	17%	12%	C-ELISA	Gelagay, 1996
Konso	7%	0%	C-ELISA	Gelagay, 1996
Debre Zeit Abattoir	-	46.7%	Ic-ELISA	Yayehrade, 1997
North Wello	5.7%	3.0%	C-ELISA	Mohammed, 2001
East Shewa	13.9%	12.6%	C-ELISA	Mohammed, 2001
Arsi	Confirmed	-	Tissue culture, ELISA	Gelagay *et al.*, 2003
National survey		5.7%	C-ELISA	MoARD, 2004
Afar, Borena, E. Shoa, Gambella Jijiga	9%	13%	C-ELISA	Abraham *et al.*, 2005
Basona Werana	37.2%	-	C-ELISA	Alemayehu, 2006
Afar (Gewane)	-	6.1%	C-ELISA	Tesfaye, 2006
Afar(Awash Fentale)	0.88%	1.05%	C-ELISA	Bahiru, 2006

As part of the control activities, serological survey was undertaken in randomly identified areas of the country so as to determine the magnitude of PPR and identify areas of higher risk. A total of 13,849 sera samples were collected from 72 District of seven regional states through

the seven regional veterinary laboratories. The laboratory analysis of the sera samples using competitive ELISA has been undertaken at the National Animal Health Research Center (NAHRC), Sebeta. Accordingly, the serological prevalence of PPR in Ethiopia was estimated to be 5% in 1999 (MoARD, 2004).

In the year 2001, a total of two thousand eight hundred fifteen sera samples from camels, cattle, goats and sheep from different parts of the country were collected and tested. The animals had not been vaccinated against rinderpest or PPR. In this study the antibody prevalence was 10% in camels in Afar. In East Shewa the highest antibody prevalence was 16% in sheep. The overall PPR seroprevalence is 3% in camels, 9% in cattle, 9% in goats and 13% in sheep Antibody seroprevalence detected in camels, cattle, goats and sheep confirmed natural transmission of PPR virus under field conditions. This is the first report of PPR antibody detection in cattle and camels in Ethiopia (Abraham *et al.,* 2005).

According to the findings of Alemayehu (2006), the seroprevalence of PPR in Basona Werana and Kewet districts of North Shewa zone was 26.1% in ovine, 31.0% in caprine and 20.2% in Bovine species. Similar investigation in Gewane district in Afar region indicated the prevalence in caprine 6.1% and none in sheep and cattle (Tesfaye, 2006). Another report from the same region has shown the seroprevalence of PPR in caprine (1.05%), in ovine (0.88%) and in bovine (0.74%) in Awash Fentale district (Bahiru, 2006).

2. 11.3. PPR outbreak reports

As part of the livestock disease surveillance system of the country, disease outbreak reports are collected monthly from all s of the country, which helps to fully understand the epidemiology of the diseases.

From recent outbreaks of contagious diseases that occurred in Arsi, North Shewa (adjacent to Afar regional state) and Amhara regional state, clinical samples including lymph nodes, spleen and intestine were submitted to NAHRC for confirmatory diagnosis. The involvement of the virus in these outbreaks was confirmed by using PPR virus immuno-capture ELISA (MoARD,

2004). The highest PPR outbreak report was observed in 1998. The number of new outbreaks of PPR in Ethiopia according to the annual report of 1996-2004 was indicated in (Table 2). January, October and May were the seasons most outbreaks occurred.

Table 2: The number of new outbreaks of PPR in Ethiopia from 1996-2004

Year	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oc	Nov	Dec	Total
1996	0	1	1	1	1	2	1	1	0	0	4	0	12
1997	1	7	2	6	0	2	1	1	2	0	0	0	22
1998	1	1	2	0	6	1	0	0	0	8	3	2	24
1999	0	0	0	0	0	0	0	0	0	1	0	0	1
2000	0	0	0	0	0	0	0	0	0	1	0	0	1
2001	0	0	0	0	0	0	0	0	1	0	0	1	2
2002	0	0	2	1	0	0	0	0	2	0	0	0	5
2003	0	2	2	0	2	1	0	1	1	3	0	0	12
2004	2	3	0	1	5	1	0	2	0	2	2	3	21
Total	4	14	9	9	14	7	2	5	6	15	9	6	100

Source: OIE, 2005

3. MATERIALS AND METHODS

3.1. Study Area

The study was conducted in Basona Werana District of North Shewa zone, Amhara Regional State (Figure 2). The capital town of the Zone surrounded by the District is Debre Berhan, which is located in the central highlands of Ethiopia, 130 km northeast of Addis Ababa, at an altitude of 2780 m above sea level and at 9°36'N and 39°38'E.

Based on the information obtained from the District Ministry of Agriculture and Rural Development (MoARD) offices, the agro ecological zones of the District are classified as 50% Dega (Highland), 48% Woina Dega (Midland) and 2% Wurch (Frost). This indicates that the District is representative of the higher- altitude of the country.

The climate is characterized by a biannual rainfall, a long dry season, and relatively cool temperature. The annual rainfall ranges from 900–1200mm. The main (72%) rain occurs from July to September. The small (28%) rain occurs from February to April but the occurrence of this rain is variable. The long dry season extends from October to February. The average monthly minimum air temperature ranges from 2.5°C in November to 8.4°C in July. The average monthly maximum air-temperature ranged from 17.6°C in August to 22.5°C in June. The mean relative humidity recorded was 68.2%

Agriculture is the mainstay of the livelihood of peoples and the leading economic activity of the area. The farming system is a type of mixed crop-livestock production with extensive livestock management system. Livestock plays an integral role for agricultural activities, which also provide meat, milk, cash income, and transportation purposes. According to CSA (2003), the livestock population includes cattle 89,257; sheep 118,081; goats 41,973; horses 6,148; donkeys 25, 837; mule 469; poultry 97,209 and beehives 5117.

In Basona Werana district, the most common diseases recorded and frequently mentioned by the Veterinary department include pasteurellosis, fasciolosis, black leg, anthrax, foot rot, lungworm and ectoparasites.

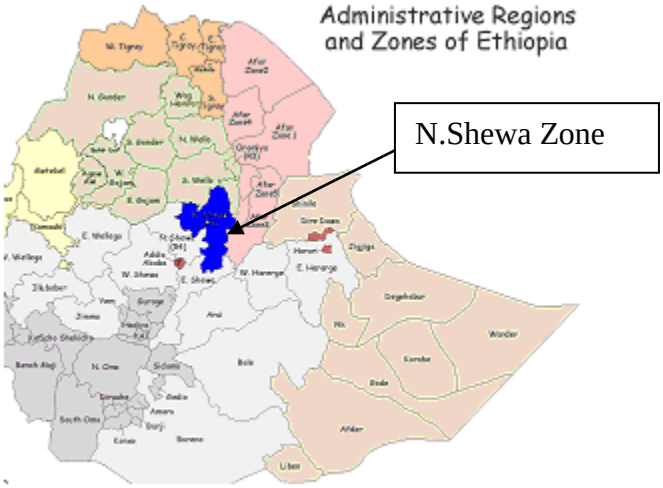


Figure 2: Map of Ethiopia showing North Shewa zone

3.2. Study Protocol

3.2.1. Data collection via questionnaire

Active data were generated by means of various questionnaires which include the number of small ruminants owned, practice of *rebi (caring of animals by another owner)*, market trekking routes and herd mixing on the way, number of sheep or goat sale and purchase, reason for selling and buying, contact points at grazing, and water points in the area.

3.2.2. Sampling method and strategy

Multistage sampling method was applied to conduct the survey. A structured questionnaire formats were developed for both the owners and herd keepers (Annex 9). The questionnaire survey was administered by taking the following assumptions and strategy.

1. The total number of PAs comprising Basona Werana district is 29.
2. On average each PA has 15 (2-25) villages.
3. Each village gathers on average 20 households (meaning families with small ruminants).

Ten PAs (Table 3) from the district have been selected randomly using the list provided by the district Ministry of Agriculture and Rural Development (MoARD) office. In each PA, all villages or 10 villages if there were more than 10 in the concerned PA, were visited. Villages were selected in collaboration with the respective Development Agents and the administrators of the PA after listing all the villages included in each PA. Then the villages were chosen randomly by allocating numbers to the villages and then picking folded papers with numbers in a box.

At the village level, all the households should be visited for the owners to be individually interviewed. If there were more than 10 households the following method was applied. Stand in the approximate center of the village and start walking north. Stop at every second household to interview the owner. When one corner of the village was arrived without having interviewed 10 owners, it was returned back to the center and the other direction (say West) was visited for interview. In the same way stop in one household out of two. If it is still not enough, the procedure was repeated taking the other direction until the 10 questionnaires have been filled on the "*Herd owners form"* (Annex 10).

In the evening there was a meeting with all the keepers of animals from the village to fill the questionnaire on the "Herd *keepers format"* (Annex 11) and the name of the herd keepers and their corresponding owner was recorded on the "Herd keepers attending the evening meeting*"* (Annex 11).

Table 3. Selected PAs for survey and their distance from the capital town

Name of the PAs	Distance (Km) from Debre Berhan
Bere Ager	25
Debele	20
Gudoberet	32
Keyit	18
Abamote	23
Angolela	10
Birbirsa	15
Goshe Bado	17
Metikoriya*	34
Koremargefiya*	12

** = Pilot study*

The questionnaires developed were first pre-tested in two PAs (Metikoriya and Koremargefia), which were finally excluded from the analysis. The outcome of the questionnaire testing was then used as a guide in the formulation of the final versions.

3.2.3. Recording georeference of study Village

Geographical coordinates and altitude of each villages visited were recorded on the "*PAs sheet form"* (Annex 9) by using a hand held receiver GPS (Garmin 48). These could be digitized and incorporated in to GIS database to produce a map indicating the villages surveyed.

3.3. Data Analysis

The Ms Excel spreadsheet computer program (Microsoft Corporation 2003) was used to store all study data). The data obtained from survey were analyzed using the SPSS Version 11.5 for widows system of statistical analysis. Descriptive statistics has been used to compute frequency of responses and percentage to summarize the data.

4. RESULTS

4.1. Mapping of the villages surveyed

The georeference of each village surveyed was recorded and the GPS data were then stored in Excel, exported to Microsoft Access and later linked to Map Info and GIS Arc View to produce the relevant Maps (Figure 3). The detail measurement of each village, altitude, latitude and longitude coordinates are indicated in (Annex 9).

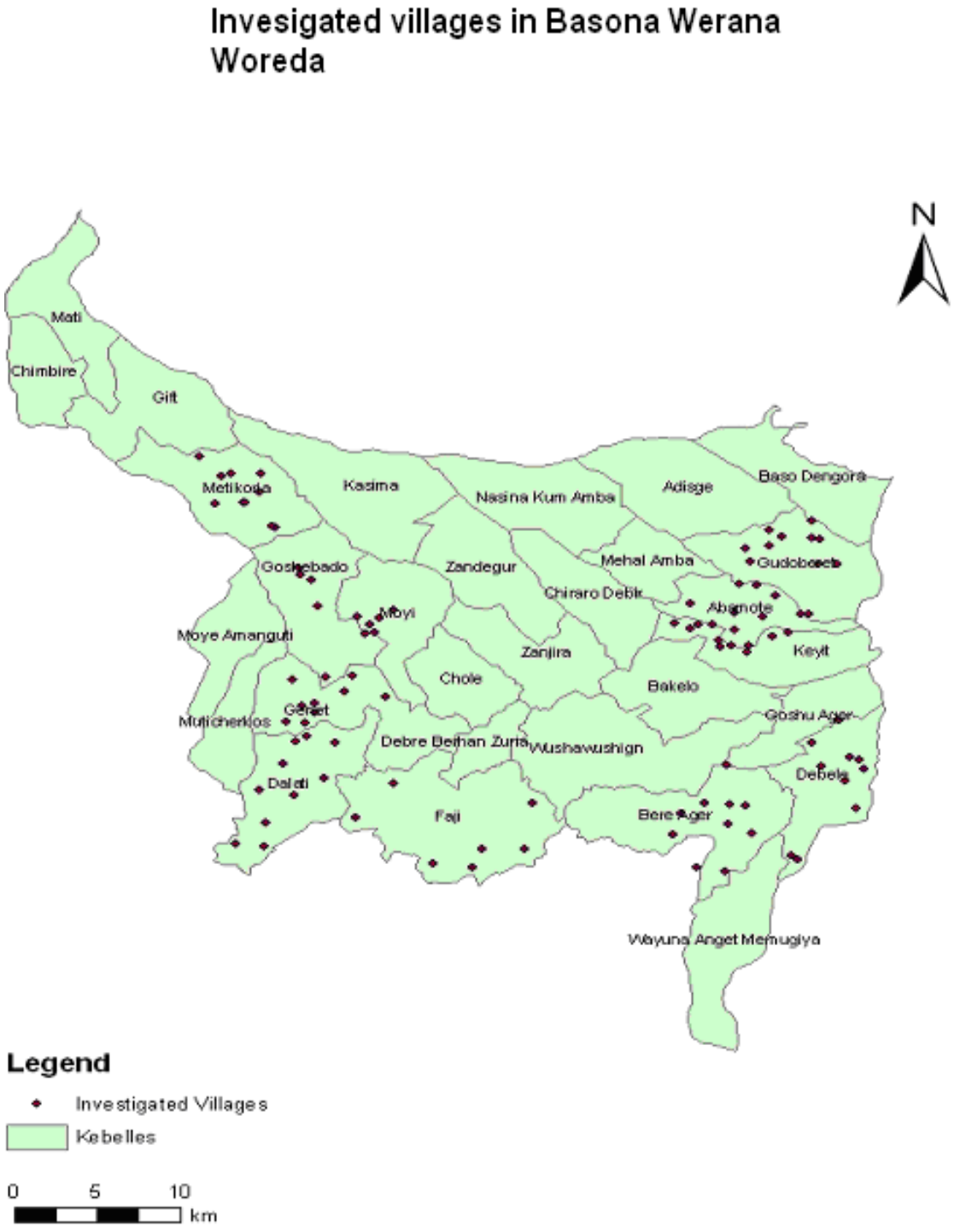


Figure 3: Map of villages surveyed in the study area (Adopted from GPS data)

4.2. Questionnaire results

Primary data were collected from 800 randomly selected smallholder households and 80 groups of herd keepers from the 80 selected villages through interviews with the aid of structured questionnaires during 2006/07. The questionnaire concentrated as yielding information on contacts occurring at grazing points, water points and market trek routes. The practice of *rebi* (which is the caring of animals by another owner) was also investigated.

4.2.1. Small ruminant population survey

The ownership of the small ruminants among the total 800 households studied is presented in (Table 4)**.** All respondents owned either sheep or goats or both flocks. About 68.1% of the sample households possessed at least a unit of sheep as compared with only 0.75% of the same sample households who owned goats. Also, the percentage of households owning any one of the two species of animal was 31.1%.

Out of all sheep owning households, 57.1% possessed between 1 and 10 sheep. In contrast, only 29.4% of all households with goats had less than 5 animals and 68.1% possessed no goats. The proportion of households that owned more than 20 units of sheep and greater than 5 units of goats was relatively smaller at 8.9 and 2.5%, for sheep and goats, respectively (Annex 1 and 2). Most of the farmers in the study area posses sheep than goats. Out of 800 farmers interviewed (68%) of them do not have goats (Table 4).

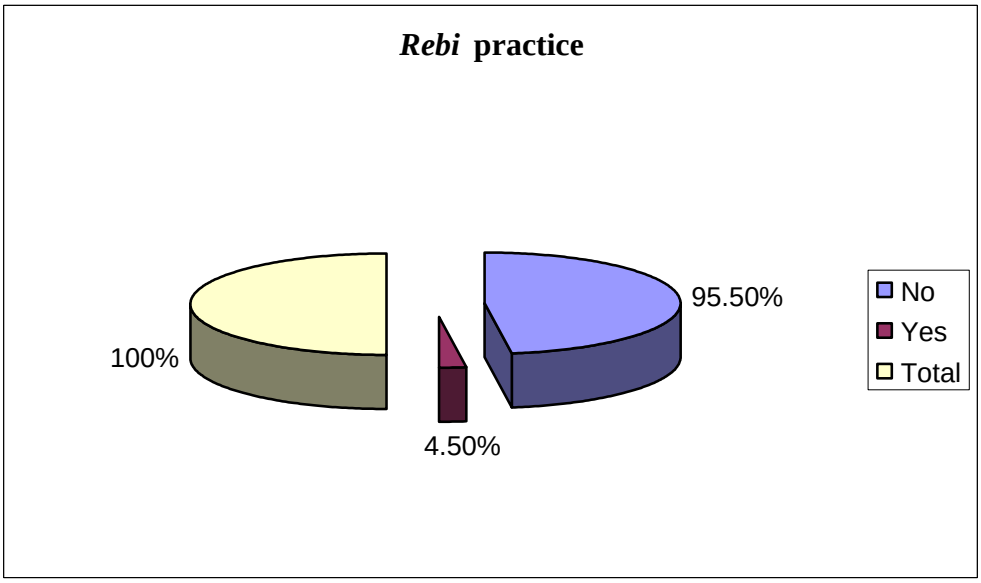
The average number of sheep owned across all households was 11.5(Annex 3) this varied greatly between the households as reflected in the standard deviation (SD = 7.32). Goat flock size also varied considerably among the households, ranging between 1 and 39 head with an average of only 0.98 (SD = 2.2). As regards total number of all ruminants per household, the average flock size was 6.24 with standard deviation of about 4.80 (Annex 3).

Table 4: Proportion of small ruminants possessed by owners in the study area

Species	**Frequency**	**Percent**
Sheep only	545	68.1
Goat only	6	0.75
Both species	249	31.1
Total	**800**	**100**

4.2.2. *Rebi* practice in the study area

The caring of animals by another farmer is locally called *rebi*. By taking animals for *rebi*, the taker equally share with the owner of the animals, the kids or lambs born either in kind (animal) or in the form of cash by selling the animals. The practice of *rebi* in different PAs and its overall proportion in the study area is indicated in (Figures 4).



(*n/N= 36/800*)

Figure 4: The overall proportion of *rebi* practice in the study area.

From the total herd owners interviewed (800) for *rebi* practice 36 (4.5%) of them were involved in such livestock caring and rearing economic activities as indicated in (Figure 4).

Small ruminants were taken for *rebi* from different PA in the study district or from other adjacent districts (Table 5).

Table 5: The origin of animals for *rebi* practice by district

Name of District	**Frequency**	**Percentage**
Basona Werana	16	2.0
Ankober	2	0.3
Angolelana Tera	16	2.0
Mendida	2	0.3
No animal for *rebi*	764	95.5
Total	**800**	**4.5**

In the study area the highest percentage of animals were taken from different PA that composes Basona Werana district (2.0%). The other District participated in giving animals for *rebi* include Angolelana Tera (2.0%), Ankober (0.3%) and Mendida (0.3 %). The result indicated that highest proportions of animals were taken for practicing *rebi* in long rainy season followed by long dry season (Annex 4). The number of animals taken for *rebi* was presented in (Annex 5)

4.2.3. Small ruminant contact at grazing point

Extensive type of animal husbandry is practiced in the study area and animals graze either on communal land or privately owned fallow lands irrespective of the species category. Herd mixing from other PAs or districts at a particular grazing point (communal/private) was mentioned by respondents (Table 6). The overall herd mixing proportion in the study area was 7.6%.

Table 6: Herd mixing status at grazing points in different PAs

Name of PA	No of grazing point mentioned	Herd mixing response	Proportion (%)
Bere Ager	37	1	2.7
Debele	31	0	0
Angolela	42	3	7.1
Keyit	40	3	7.5
Birbirsa	38	4	10.5
Goshebado	35	0	0
Abamoti	40	6	15.0
Gudoberet	26	5	19.2
Total	**289**	**22**	**7.6**

The result on type of grazing point and the possibility of herd contact shows that high percentage of herd contact was observed in communal grazing point (10.2%) compared to private grazing points (3.5%) as indicated in (Table 7).

Table 7: Status of herd mixing with type of grazing point

Type of grazing point	No grazing point	Response to herd mixing	Proportion (%)
Communal	176	18	10.2
Private	113	4	3.5
Total	**289**	**22**	**7.6**

4.2.4. Small ruminant contact at water point

Contact of small ruminants at water points in the study area with herds from other PAs was identified in both Permanent River and spring water (9.62% and 10.2%), respectively. In contrast, no herd mixing at artificial pond was reported by the respondents in the study area. The overall herd contact at water points was 10.34 % (Table 8)

Table 8: Small ruminant herd contact by type of water point in different PAs

Type of water Point	**No of water points**	**No respondents**	**Proportion of herd mix (%)**
Permanent river	135	13	9.62
Spring water	49	5	10.2
Artificial pond	8	0	0
Total	**174**	**18**	**10.34**

4.2.5. Small ruminant markets and marketing system

Table 9: Marketing system for selling small ruminants in the study area

Marketing system	**Count**	**Percent**
Village	5	0.7
Different markets	625	85.0
Traders	107	14.5
Total	**737**	**100**

Farmer's sale their animals at the village level to farmers or traders; or by taking their animals to different markets found within the District or adjacent District (Table 12). This is based on the accessibility of the market or the preference of the farmers to fetch higher prices on their animals.

The most dominant market for small ruminant in the study area was Debre Berhan 352(58%) followed by Gudoberet 92 (15.1%). Farmers also take or bring their animals to/from Ankober and Angolelana Tera District which are found outside of the study area (Table 10).

Table 10: Identification of different small ruminant markets in the study area

Market name	**District Location**	**Proportion of farmers sold/bought animals**	
		Count	**Percent**
Debre Berhan	Basona Werana	352	58.0
Arb Gebeya	Basona Werana	73	12.0
Rob Gebeya	Basona Werana	67	11.0
Ankober	Ankober	3	0.5
Chacha	Angolelana Tera	20	3.3
Gudoberet	Basona Werana	92	15.1
	Total	**607**	**100**

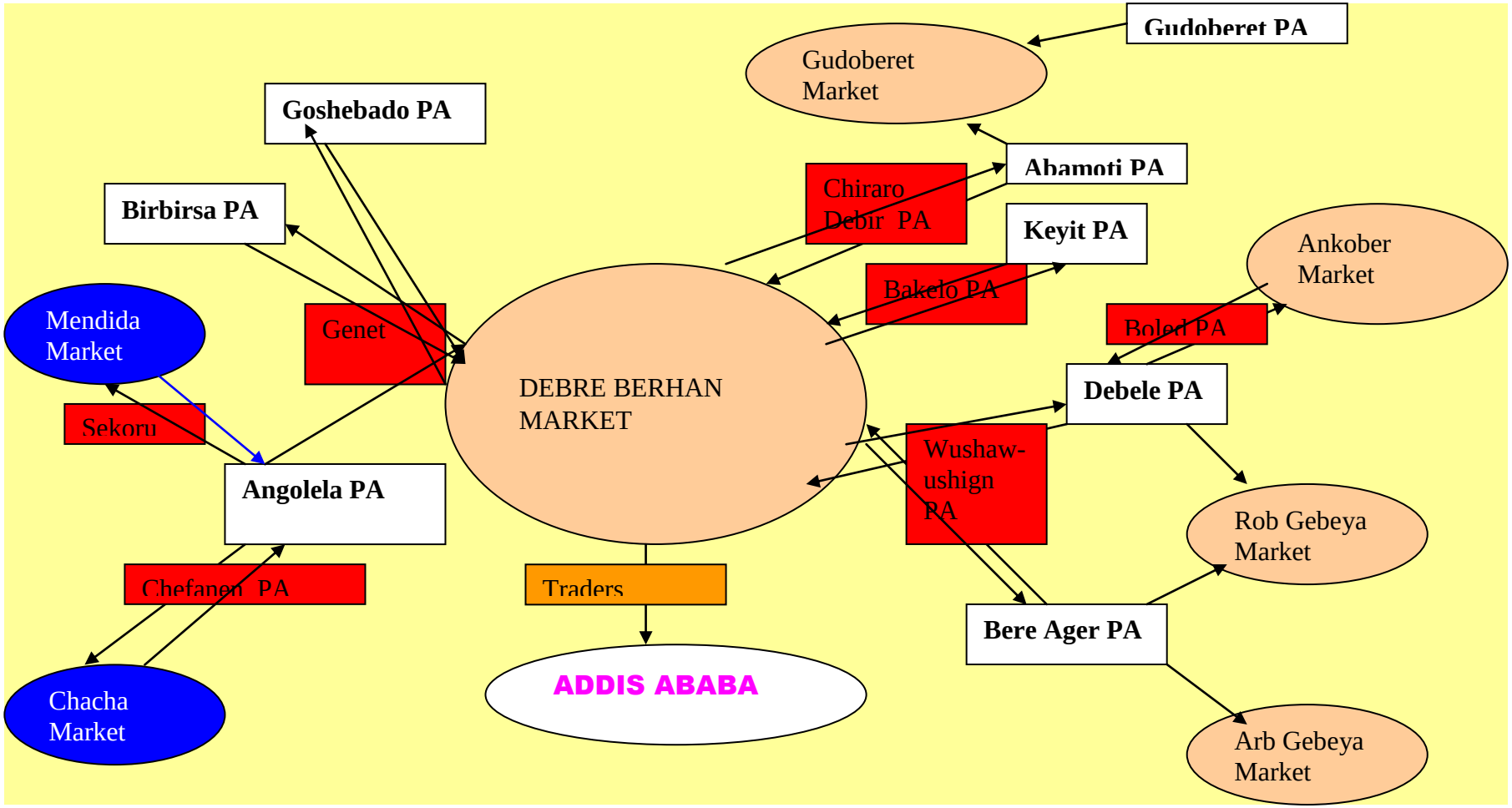


Figure 5: Small ruminant market trek routes and herd mixing while crossing PAs on the way to the Market

Key: PAs selected for study PAs crossed to market (PPR risk points)

Small ruminant markets Markets outside of study district

Herd owners in the study area sell their animals to different markets, which are found in the, study district or neighboring districts (Figure 5). On the way to the market they cross other PAs by mixing their animals with herds from that particular PA or other sources. At these points animals acquire PPR infection. The mean proportion of animals sold or purchased indicated in (Annex 6).

4.2.6. Reasons for selling or buying of small ruminants

Herd owners in Basona Werana district sell small ruminants for a number of reasons. Among these, purchasing of clothes and human food, expense on fertilizers, animal feed, security and school fee are the one mentioned by farmers. The details of socio-economic reasons for marketing small ruminants are indicated in (Annex 7 and 8).

Farmers interviewed in the area buy animals for breeding purpose, own consumption and profit (Annex 8). Out of 101 farmers who bought animals, 45 (44.55%) were used for own consumption followed by breeding reasons 42 (41.60%) for constituting the flock and 14(13.86%) for profit.

4.2.7. Herd contact on the way to the market

When farmers take their animals to the market or on return back, they cross some of the PA until they reach the destiny market. During this time they mix their stock with other herds of the PA (Figure 5 and Table 11). Out of 737 farmers who visited the markets 390 (53.0%) was found mixing their animals with herds of other Peasant associations or districts.

Table 11: Proportion of herd mixing on the way to the market in each PAs.

Name of PA	No of respondents	Mixing status (Frequency/Percent)	
		No	Yes
Bere Ager	121	92 (76.0%)	29 (24.0)
Debele	77	73 (94.5)	4 (5.2)
Angolela	123	28 (22.8)	95 (77.2)
Keyit	84	15 (84)	69 (82.1)
Birbirsa	78	14 (17.9)	64 (82.1)
Goshebado	79	11 (13.9)	68 (86.1)
Abamoti	109	49 (45.0)	60 (55.0)
Gudoberet	66	65 (98.5)	1 (1.5)
Total	**737**	**347 (47.1)**	**390 (53.0)**

Table 12: Summary of herd mixing response on the way to the market

Herd Mix	Frequency	Percentage
Yes	390	53.0%
No	347	47.1%
Total	**737**	**100**

4.2.8. Small ruminant contact with wild ruminants at grazing point

Wild ruminants that wonder around the grazing and water points were identified by herd keepers (Table 13). The local names of these animals as they call it are *Midako and Ses*. The common name and scientific name of these animals is Bush Duiker (*Syvicapra grimmia*) for *Midako* and Klipspringer (*Oreotragus oreotragus)* for *Ses*, respectively. The contact of these wild ruminants with sheep and goats at grazing and water points is indicated in (Table 13).

Table 13: Identification and contact of Wild ruminants at water and grazing points

Wild ruminants	Contact at water point	Contact at grazing point (n/%)
Bush Duiker *(Midako)*	18 (81.8%)	36 (78.3%)
Klipspringer *(Ses)*	3 (13.3%)	6 (13.0%)
Both	1 (4.5%)	4 (8.7%)
Total (N=80)	**22 (27.5%)**	**46 (57.5%)**

The observation of wild ruminants at grazing or water point is almost similar with higher proportion of Bush Duiker than Klipspringer. The overall proportion of these wild ruminants observation was higher at grazing point (57.5%) compared to water point (27.5%) (Figure 6).

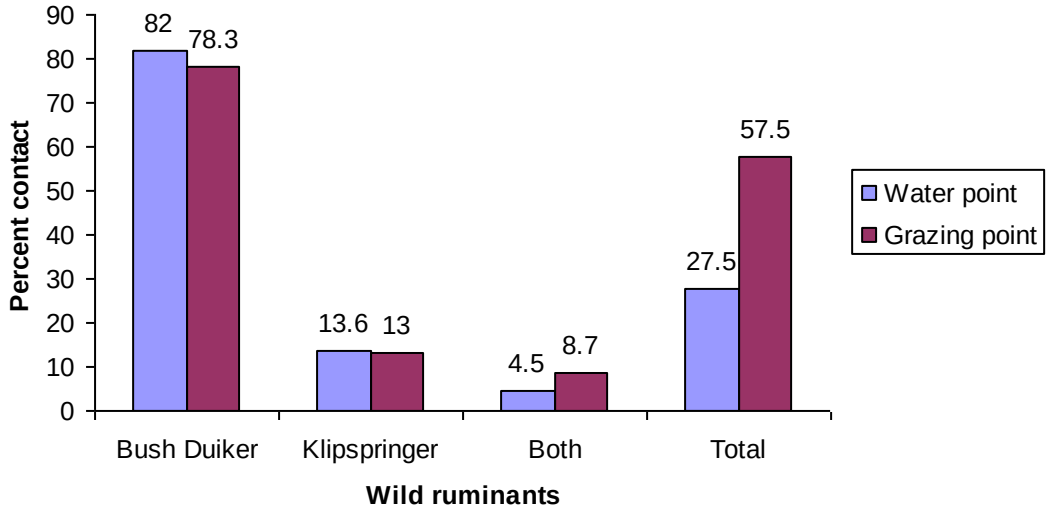


Figure 6: Comparison of the contact of small ruminants at both grazing and water points

Table 14: Identification and proportion of wild ruminants at grazing point in each PAs

Name of PA	**No grazing point**	**Name of wild ruminants/count (%)**		
		Bush Duiker	**Klipspringer**	**Both**
Bere Ager	0	0	0	0
Debele	16	12 (75.0)	1 (6.3)	3 (19.0)
Angolela	5	3 (60.0)	1 (20.0)	1 (20.0)
Keyit	7	7 (100.0)	0	0
Birbirsa	4	3 (75.0)	1 (25.0)	0
Goshebado	1	0	1 (100.0)	0
Abamoti	8	7 (87.5)	1 (12.5)	0
Gudoberet	5	4 (80.0)	1 (20,0)	0
Total	**46**	**36 (78.3)**	**6 (13.0)**	**4 (8.7)**

4.2.9. Small ruminant contact with wild ruminant at water point

Table 15: Identification of Wild ruminants at water point in each PAs of the study area

Name of PA	**No. Water point**	**Wild ruminants/count (%)**		
		Bush Duiker	Klipspringer	Both
Bere Ager	1	1 (100)	0	0
Debele	7	7 (100)	0	0
Angolela	6	4 (66.7)	2 (33.3)	0
Keyit	3	2 (66.7)	0	1 (33.3)
Birbirsa	1	1 (100)	0	0
Goshebado	0	0	0	0
Abamoti	4	3 (75.0)	1 (25.0)	0
Gudoberet	0	0	0	0
Total	**22**	**18 (81.8)**	**3 (13.6)**	**1 (4.5)**

4.2.10. Distance and frequency observation of wild ruminants

Table 16: Observed distance of wild ruminants from grazing point

Name of wild ruminants	**No of respondent**	**Observed distance (meter)**	
		<125m	**>125m**
Bush Duiker	36	27 (75.0)	9 (25.0)
Klipspringer	6	2 (33.3)	4 (66.7)
Both	4	3 (75.0)	1 (25.0)
Total	**46**	**32 (70.0)**	**14 (30.0)**

(Mean = 125meter)

Table 17: Observation of the frequency of wild ruminants at grazing point

Name of wild ruminants	**No. grazing point for response**	**Frequency/week(%)(Mean=2.55)**	
		<3 times	**>3 times**
Bush Duiker	36	29 (80.6)	7 (19.4)
Klipspringer	6	6 (100.0)	0
Both	4	4 (100.0)	0
Total	**46**	**39 (85.0)**	**7 (15.0)**

The mean distance and mean frequency/week at which wild ruminants (Bush Duiker and Klipspringer) was observed from the grazing point was 125.43 $\pm$16.01 and 2.5 $\pm$0.16 and from the water point was 162.27 $\pm$27.23 and 2.55 $\pm$1.25, respectively.

Table 18: Distance of wild ruminants from water point

Name of wild ruminants	**No of respondents**	**Distance (meter) (Mean=162meter)**	
		<162m	>162m
Bush Duiker	18	10 (55.6)	8 (44.4)
Klipspringer	3	0	3 (100)
Both	1	0	1 (100)
Total	**22**	**10 (45.5)**	**12 (54.5)**

Table 19: Frequency observation of wild ruminants at water point

Name of wild ruminants	No respondents	Frequency per week (%) (Mean =2.55 times)	
		< 3 times	> 3 times
Bush Duiker	18	11 (61.1)	7 (38.9)
Klipspringer	3	2 (66.7)	1 (33.3)
Both	1	0	1 (100)
Total	**22**	**13 (59.1)**	**9 (40.9)**

Table 20: Observed distance of wild ruminants from grazing and water points

Distance from grazing point (n=46, N=80)		Distance from water point (n=22, N=80)	
<125m	>125m	<162m	>162m
32 (70.0%)	14 (30.0%)	10 (45.5%)	12 (54.5%)

Table 21: Mean distance and frequency of wild ruminants from grazing and water points

Criteria	Grazing point	Water points
	Mean	Mean
Distance (meters)	125.43 $\pm$16.01	162.27$\pm$27.23
Frequency / week	2.5 $\pm$ 0.16	2.55$\pm$1.25

Table 22: Summary of small ruminant contact structure in Basona Werana district.

Herd contact	Total response	Proportion (%)
At grazing point	22/289	7.6
At water points	18/174	10.34
On the way to the market	390/737	53.0
Through *rebi* practice	36/800	4.5
With Wild ruminants at grazing point	46/289	20.0
With Wild ruminants at water point	22174	13.0

5. DISCUSSION

The Result of the study indicated that higher numbers of sheep are kept by the households than sheep, suggesting that sheep are dominant species in the study area compared to goats. The difference in the flock size of both species might be attributed to the higher adaptation potential of sheep to the prevailing harsh cooler climatic conditions in the area than that of goats. Furthermore, goat rearing in the area is not encouraged by the farmers from the religious and cultural taboos.

In the present study, farmers sold small ruminants for a number of reasons but most of them sold animals for purchasing clothes 303 (29.02%) followed by food for the family 224 (21%). Other owners used the cash for purchasing fertilizer 173(16.57%), animal feed 59 (5.65%) and for paying land taxation 143(13.7%), school fee 105 (10.06%) and for security. This shows that small ruminants are used for immediate needs and investment. Other researchers indicated that marketing provides the mechanism whereby producers exchange their livestock and livestock products for cash. The cash is used for acquiring goods and services which they do not produce themselves, in order to satisfy a variety of needs ranging from food items, clothing, medication, and schooling to the purchase of breeding stock and other production inputs and supplies (Bekure and Nigussie, 1983; Gryseels, 1988).

Furthermore, farmers bought small ruminants for breeding, own consumption, or fattening and sale to get profit. Out of 101 farmers who bought animals, 45 (44.55%) of the respondents used for own consumption followed by breeding reasons 42 (41.60%) to constitute the flock and 14(13.86%) for profit. This consumption was related most often with the celebration of New Year and a number of other local and national holidays. According to Gryseels (1988), sheep supplies the dominant type of meat consumed in rural areas and an average of three head of sheep is slaughtered per year per family in the central highlands of Ethiopia.

Rebi practice (the caring of animals by another person) is one of the traditional livestock rearing system engaged by farmers in the study area to fulfill the economic and social needs. The overall proportion of *rebi* practice in the study area was 36(4.5%). Farmers take these animals irrespective of the season according to the availability of the *rebi* animals and

willingness of the owner giving his animal for *rebi.* It seems that animals taken during dry season will breed during the rainy season where the food is available for the lambs/kids born.

Herd owners had taken animals for *rebi* from other PAs within Basona Werana district and neighboring areas. The practices of *rebi* constitute another source of risk because animal movement from other districts to the study area through rebi practicing might lead to the transmission of PPR disease. Information recorded on such kind of animal movement was not available. However, Mariner *et al.* (2006) reported that cattle exchange is of particular interest in the epidemiology of diseases particularly in contagious bovine pleuropneumonia.

The movement of animals between premises is considered to be the main risk factors for the introduction of infectious diseases to farms. Animal movement is not a risk in itself but a mechanism for bringing infected animals in contact and as a result introducing or spreading disease (Ortiz-Pelaez, *et al.* 2006).

Extensive type of animal husbandry is practiced in the study area and animals graze either on communal land or privately owned lands irrespective of the species category. Similar observation was reported by in mixed farming systems in the Ethiopian highlands. According to Dibissa and Peters (1999) the livestock production in the present study area predominantly depends on communal grazing lands.

The finding of this study indicated more herd contact at communal grazing land (10.2%) compared to privately owned grazing points (3.5%). Privately owned lands become communal during dry season. During this time, crops are not cultivated and animals graze everywhere they get pasture. The Peasant associations in the study area mix their animals at grazing points with other PAs within the same study site or with others in other adjacent districts. Communal grazing increases the contact rate among animals and in cases where a PPR out break has not been recognized in time and appropriate movement control put in place, the disease will spread fast among the susceptible population (FAO, 1999).

Contact of small ruminants at water points in the study area with herds from other PAs was identified in both permanent river and spring water (9.62% and 10.2%), respectively. In contrast, no herd mixing at artificial pond was reported by the respondents in the study area. This is due to the fact that artificial ponds are constructed by individuals near his/her home or by groups within the same villages and animals could not come from other PAs to this point. The overall herd contact at water points was 10.34 %. The increased contact rate among animals at water points suggested that susceptible animals could therefore pick up infection from these points.

The role of wild life in the epidemiology of PPR in Ethiopia is not known. Bush Duiker and Klipspringer were the wild ruminants identified in the study area. About 36 (78.3) and 18(81.8%) of the respondents have identified Bush Duiker only, 6(13.0%) and 3(13.3%), Klipspringer only, and 4(8.7%) and 1(4.5%) both wild ruminants around their grazing and water points respectively.

The mean distance at which wild ruminants (Bush Duiker and Klipspringer) was observed from the water point and grazing point is 162.27 +27.23 and 125 +16.01, respectively. The mean frequency/week for the observation of these wild lives were 2.5 +0.16 and 2.55 +1.25 from the grazing and water points, respectively. A similar work done by Bates *et al.* (2001) on owners of large beef herds to investigate the transmission of foot and mouth disease (FMD), indicated 55%(16/29) of the owners observed deer or elk within 150m of livestock at least once per month. It was suggested that the estimate and probability functions that represent contact rates are prerequisite elements of models aimed at predicting the geographic spread of foot and mouth disease.

PPR outbreak was reported in captive wild ungulates from three families: Gazellinae (Dorcas gazelle), Caprinae (Nubian ibex and Laristan sheep), and Hippotraginae (gemsbok). Experimentally, the American white-tailed deer (Odocoileus virginianus) is fully susceptible. The role of wildlife on the epizootiology of PPR in Africa remains to be investigated (Furley *et al.,* 1987). The prevalence rate of antibodies to peste des petits ruminant's virus (PPRV) and rinderpest virus (RPV) antigens was studied using 38 sera samples collected from African

grey duiker (Sylvicapra grimmia). Out of the total, 4 (10.5%) were positive for antibodies to PPRV which probably implicates the role of wildlife in the epizeotiology of PPR in Nigeria. The results also indicated PPRV may have gained access to this region through wildlife, especially wild small ruminants which are prominently hunted animals in this environment (Ogunsanmi *et al.*, 2003). The isolation of virus from an outbreak in Indian buffalo (Bubalus bubalis) has been reported (Govindarajan *et al*, 1997) to occur at a District Livestock Farm in Orathanadu, Tanjore District, and Tamil Nadu where 50 of the 385 buffaloes were affected.

Livestock marketing in Ethiopia follows a three-tier system: the primary, secondary and terminal markets through which animals go into the hands of small traders and then to large traders. Final buyers, which include butchers, meat-processing factories, fattening farms or live animal exporters, purchase livestock at any stage (ELSMA, 2002).

In this study, small ruminant markets used in the study area were identified. These markets include Debre Berhan, Rob Gebeya, Arb Gebeya and Gudoberet. The other two markets (Chacha and Ankober), even though outside the study area were mentioned and used by farmers. Farmers sell their animals to farmers in the village, traders or by taking them to different markets found in the area. Most farmers sold their animals to Debre Berhan market (58%) followed by Gudoberet market (15.1%). These markets were the potential area for transmission of contact diseases. Trade routes where some of the animals mix on their way to the market with other animals contribute much for the transmission of PPR disease. Farmers cross one or more PAs may stop for some time in the PA, and mix their herds creating a possibility of PPR and other contagious diseases transmission.

Trade in small ruminants, at markets or their movement on the way to markets where animals from different sources are brought into close contact with one another, affords increased opportunities for PPR transmission. In a particular flock, the risk of an outbreak is greatly increased when a new flock is introduced or when animals are returned unsold from livestock markets where they may have readily contracted the disease (FAO, 1999; Radostits *et al.*, 2000).

6. CONCLUSION AND RECOMMENDATIONS

The Result of this study indicated that higher numbers of sheep are kept by the households than goats, suggesting that sheep are dominant species raised in the study area compared to goats. The principal functions of small ruminants are security and investment.

Animal movement through the practice of *rebi*, herd contact at grazing and water points, the mixing of animals on the way to the market and at market site and contact with wild ruminants are the most important PPR risk factors identified. There is a possibility of disease transmission through the practice of *rebi* as animals might be taken from other Peasant association or neighboring districts.

The finding of the study indicated more herd contact at communal grazing land compared to privately owned grazing lands. In another finding, the contact of small ruminant herds at spring water was higher than the contact at permanent river. Small ruminants trading at markets or their movement on the way to markets where animals from different sources are brought into close contact with one another, affords increased opportunities for PPR transmission.

Even though, the role of wild ruminants in the epizootology of PPR disease is not known in the study area, the lesser the distance and the more frequent wandering of them at watering and grazing points revealed greater possibility for the transmission and spread of PPR and other contagious disease to domestic ruminants and the vice versa. The identification of contact points for PPR transmission helps in adopting animal movement control and in modeling of PPR disease and its control strategy. The present finding on contact structure can also be adopted for other contagious diseases of small ruminants.

In line with the above conclusions, the following recommendations can be forwarded:

- Further research has to be conducted on the epidemiology of PPR in wild ruminants in the study area in order to determine the prevalence of PPR, its effects on wildlife conservation and the possible role of these species in the transmission cycle or carrier nature of PPR Vius.
- Control of animal movement policy should be adopted.
- Education of farmers about ways of disease transmission by mixing of animals through *rebi* practice, with other herds on the way to the market, at grazing and water points.
- Animals returned unsold from markets or purchased for breeding should be isolated unless the entire herd or flock has been vaccinated.
- Intervention should done at the identified potential contact risk points to reduce herd to-herd transmission of PPR disease.
- Further work on modeling of PPR disease and its control strategy should be done.

7. REFERENCES

Abraham, G. (2005): Epidemiology of peste des petits ruminants virus in Ethiopia and molecular studies on virulence. PhD Thesis, National Polytechnique Inistitute of Toulouse, Toulouse, France.

Abraham, G., Roeder, R L., Kinfe, G. (1991): Peste des petitis ruminants as cause of goat mortality. In: Institute of Agricultural Research proceeding of the 4th Livestock improvement conference; 13-15 Nov., Addis Ababa, Ethiopia. Pp 276-279.

Abraham, G., Sintayehu, A., Libeau, G., Albina, E., Roger, F., Laikemariam, Y., Abayneh, D., Awoke, K.M. (2005): Antibody seroprevalences against peste des petits ruminants (PPR) virus in camels, cattle, goats and sheep in Ethiopia. *Prev Vet Med.,* **70:** 51-57.

Abu ElZein, E. M. E., Hassanien, M. M., Alfaleg, A. I. A, Abd Elhadi, M. A., Housawi, F.M.T. (1990): Isolation of PPR virus from goats in Saudi Arabia. *Vet. Rec.*, **127:** 309-310.

Adu, F. D., Joannis, T. E. (1984): Serum-virus simultaneous method of immunization against peste des petits ruminants. *Trop. Anim. Hlth. Prod.,* **16:**119-122.

Alemayehu, F. (2006): Seroprevalences study of PPR in North Shewa, Ethiopia. Debre Zeit: Addis Ababa University, Faculty of Veterinary Medicine, DVM Thesis.

Alemayehu, Z.' and Fletcher, I (1991): Small ruminant productivity in the central Ethiopian mixed farming systems. Institute of Agricultural Research proceeding. 4th NLIIC. Pp 141-147.

Awa, D. N., Ngagnou, A., Tefiang, E., Yaya, D., Njoya, A. (2002): Post vaccination and colostral peste des petitis ruminants antibody dynamics in research flocks of Kirdi goats and Foulbe sheep of north Cameroon. *Prev Vet Med.,* **55:** 265-271.

Bahiru, G. (2006): Seroprevalence study of PPR in pastoral area in Awash Fentale district, Afar Regional state, Ethiopia. Debre Zeit: Addis Ababa University, Faculty of Veterinary Medicine, DVM Thesis.

Bandyopadhyay, S. K. (2002): The economic appraisal of PPR control in India. In 14th annual conference and national seminar on management of viral diseases with emphasis on global trade and WTO regime, Indian Virological Society, 18-20 January, Hebbal Bangalore.

Barrett, T., Visser, I. K. G., Mamaev, L., Goatley, L., Bressem, M. F., Van Osterhaus, A. D. M. (1993): Dolphin and porpoise morbilliviruses are genetically distinct from phocine distemper virus. *Virology* **193:** 1010-1012.

Barrret, T. (1999): Morbillivirus infection, with special emphasis on morbillivirus of carnivores. *Vet. Microbio.*, **69**: 3-13.

Bates T. W., Thurmond, M. C., and Carpenter, T. E (.2001): Direct and indirect contact rates among beef, dairy, goat, sheep, and swineherds in three California countries, with reference to control of potential FMD disease transmission. *AJVR***, 62:** 1121-1129.

Bekure, S. and Nigussie, T. (1983): Livestock marketing studies. In: Pastoral systems Research in Sub Saharan Africa. Proceedings of the IDRC/ILCA workshop held at ILCA, Addis Ababa, Ethiopia, 21-24 March 1983. ILCA, Addis Ababa.327-360.

Berhe, G., Minet, C., Le Goff, C., Barret, T., Ngangnou, A., Grillet, C., Libeau, G., Fleming, M., Black, D. N., Diallo, A. (2003): Development of a dual recombinant vaccine to protect small ruminants against peste des petits ruminants virus and capripoxvirus infections. *J Virol.*, **77:** 1571-1577.

Braide, V. B. (1981): Peste des petits ruminants. *World Anim. Review*, **39:** 25-28.

Bundza, A., Afshar, A., Dukes, T. W., Myers, D. J., Dulac, G. C., and Becker, S. A. W .E. (1988): Experimental peste des petits ruminants (goat plague) in goats and sheep. *Can. J. Vet. Res.*, **52:** 46-52.

Couacy-Hymann, E., Roger, F., Hurard, C., Guillou, J.P., Libeau, G. and Diallo, A. (2002): Rapid and sensitive detection of peste des petits ruminants virus by a polymerase chain reaction assay. *J. Virol. Methods.* **100**: 17-25.

CSA (2003): Ethiopian Agricultural sample enumeration 2001/2002. Central Statistics Authority, Addis Ababa, Federal Democratic Republic of Ethiopia.

Devireddy, L. R., Raghavan, R., Ramachandran, S., Shaila, M. S. (1999): The fusion protein of PPR virus is a hemolysin. *Arch. Virol*., **144:**1241-1247.

Dhar, P., Sreenivasa, B. P., Barret, T., Corteyn, M., Singh, R. P., Bandopadhyay, S. K. (2002): Recent epidemiology of peste des petits ruminants virus (PPRV). *Vet. Microbiol.*, **88:**153-159.

Diallo, A. (1990): Morbillivirus group: genome organization and proteins. *Vet. Microbiol*., **23:** 155-163.

Diallo, A. (1997): Peste des petitis ruminants: An overview. FAO EMPRESS FMD and Contingency Planning Workshop, held from July 28 to August 01, 1995, Hanoi

Diallo, A. (2003): Control of peste des petits ruminants: classical and new generation vaccines. *Dev Biol (Basel*), **114:** 113-9.

Diallo, A., Barret, T., Barbon, M., Shaila, M. S., Taylor, W. P. (1989): Differentiation of rinderpest and peste des petits ruminantss viruses using specific cDNA clones. *J. Virol. Methods,* **23**:127-136.

Diallo, A., Barret, T., Lefevre, P. C. and Taylor, W. P. (1987): Comparison of proteins induced in cells infected with RP and PPR viruses. *J. Gen. Virol,* **68**:2033-2038.

Diallo, A., Barrett, T., Barbron, M., Meyer, G. and Lefevre, P. C., (1994): Cloning of the nucleocapsid protein gene of peste-des-petits-ruminants virus: relationship to other morbilliviruses. *J. Gen. Virol.,* **75:** 233–237.

Diallo, A., Libeau, G., Couacy-Hymann, E., Barbron, M. (1995): Recent development in Diagnosis of RP and PPR. *Vet. Microbiol*., **44:** 307-317.

Diallo, A., Minet, C., Le Goff, C., Berhe, G., Albina ,E., Libeau, G., Barret, T. (2007): The threat of peste des petits ruminants: progress in vaccine development for disease control.*Vaccine,* .2007*(Articles in Press).*

Dibissa, N. and Peters, K. J. (1999): Livestock species composition and herd size in relation to household farm resources in mixed farming systems in the Ethiopian highlands. *Outlook on Agriculture*, **28**: 51-54

ElHag, Ali, B., Taylor, T. W. (1988): An investigation on Rinderpest virus transmission and maintenance by sheep and goats. *Bull. An. Heath. Prod. Afr*. **36**, 290-294

Elzei, E .M., Housawi, F. M., Bashareek, Y., Gameel, A. A., Al-Afaleq, A. l., Anderson, E. (2004): Severe PPR infection in gazelles kept under semi-free range conditions. *Vet Med Infect Dis.*, **51:** 68-71.

FAO, (1993): Agrostata data, Statistical Division. Vol 39. Rome Italy.

FAO, (1999): Animal health manual; recognizing Peste des petitis ruminants. A field manual. Food and Agriculture Organization of the United Nations, Rome, Italy. Pp 2-25.

Forsyth, M. A. and Barrett, T. (1995): Evaluation of polymerase chain reaction for the detection and characterisation of rinderpest and peste des petits ruminants viruses for epidemiological studies. *Virus Res.,* **39**: 151-163.

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

Gelagay, A. (1996): Epidemiological and serological investigation of multifactorial ovine respiratory disease and vaccine trial on the high land of North Shoa, Ethiopia. Debre Zeit: Addis Ababa University, Faculty of Veterinary Medicine, DVM Thesis.

Gelagay, A., Hailu, W., Rufael, T., Esayas, G. and Aschalew, Z. (2003): Isolation and identification of PPR virus in Arsi Zone of Oromia region, Ethiopia. In: Proceedings of the 17th Annual conference of the Ethiopian Veterinary Association held from June 5-6: Addis Ababa, Ethiopia. Pp 91-99.

Gibbs, E. P .J., Taylor, W. P., Bryant, J. (1979): Classification of peste des petits ruminants virus as the fourth member of genus Morbillivirus. *Intervirology*, **11***:* 268–274.

Govindarajan, R., Koteeswaran, A., Venugopalan, A. T., Shyam, G., Shaouna, S., Shaila, M. S., Ramachandran, S. (1997): Isolation of peste des petits ruminants virus from an outbreak in Indian buffalo (*Buballus bubalus). Vet. Rec*., **141:** 573-574.

Gryseels, G. (1988): The role of livestock in the generation of smallholder farm income in two Vertisol areas of the central Ethiopian highlands. In: Jutiz S C, Haque I, McIntire J and Stares J E S (eds), Management of Vertisols in sub-Saharan Africa. ILCA, Addis Ababa, Ethiopia. Pp. 345-358.

Haffar, A, Libeau, G., Moussa, A., Cècile, M., Diallo, A. (1999). The matrix protein gene sequence analysis reveals close relationship between PPR virus and dolphin morbillivirus. *Virus Research*, 64**:** 69-75.

Hamdy, F. M. (1998): Peste des petitis ruminants. In: The Merck Veterinary Manual. 8th ed. New Jersy: Merck and Co., Inc. Whitehouse Station. Pp 539-541.

Hamdy, F. M., Dardiri, A. H., Nduaka, O, Breese, S. S., Ihemelandu, E. C.(1976) Etiology of the stomatitis pneumoenteritis complex in Nigerian dwarf goats. *Can.J.Comp.Med.* **40**, 276-284.

Ibrahim, H., (1998): Small ruminant production techniques. ILRAD, Manual No.3, ILRI, Nairobi, Kenya. Pp 1-36.

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

Ismail, T. H., Yamanaka, M. K., Saliki, J. K., Elkhoy, A., Mebus, C., Yilma, T. (1995): Cloning and expression of the nucleoprotein of PPR virus in baculovirus for use in serological diagnosis. *Virol.,* **208**: 776-778.

Jones, L., Giavedoni L., Salik., J. T. Brown, C., Mebusand, Yilma T. (1993): Protection of goats against peste des petits ruminants with a vaccinia virus double recombinant expressing the F and H genes of rinderpest virus. *Vaccine,* **11:** 961-964.

Kitching, R. P. (1988): The economic significance and control of small ruminant viruses in North Africa and West Asia. In: Thompson, F.S. (Ed.), Increasing Small Ruminant Productivity in Semi-arid Areas. ICARDA. Pp 225–236.

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

Libeau, G., Prehaud, C., Lancelot, R., CoLas, F., Guerre, L., Bishop, D. H., Diallo, A. (1995): Development of a competitive ELISA for detecting antibodies to the PPR virus using a recombinant nucleoprotein. *Res.Vet.Sc.,* **58**, 50-55.

ELSMA (2002): Socio-economic and Policy Research Working Paper 58. ILRI (International Livestock Research Inistitute), Nairobi, Kenya. Pp.47

Mahapatra, M., Parida, S., Egziabher, B. G., Diallo, A., Barrett, T. (2003): Sequence analysis of the phosphoprotein gene of peste des petits ruminants (PPR) virus: editing of the gene transcript. *Virus Res.,* **96:** 85–98.

Mariner, J. C., McDermot, J., Heesterbek, J. A. P., Thomson, G., Roeder, P. L., Martin S. W. (2006): A heterogeneous population model for contagious bovine pleuropneumonia transmission and control in Pastoral communities of East Africa. *Prev.Vet.Med.,* **73:** 75-91.

Meyer, G. and Diallo, A. (1995): The nucleotide sequence of the fusion protein gene of the PPR virus: The long untranslated region in the 5-end of the F-protein gene of morbilliviruses seems to be specific to each virus. *Virus Research*, **37**: 23-38.

MoARD, (2004): CCPP and PPR control strategy Document. Department of Animal Health, Ministry of Agriculture and Rural Development, Addis Ababa. Pp 5-30.

Mohammed, A. E. B. (2001): Contribution to the knowledge of the epidemiology of peste des petitis ruminants in Wallo and East Shoa Zones of Ethiopia. Debrezeit: Addis Ababa University, Faculty of Veterinary Medicine, Msc Thesis.

Nawathe, D. R. (1984): The control of peste des petits ruminants in Nigeria. *Prev Vet Med*., **2:** 147-155.

Nwafor, C U., (2004): Small ruminant livestock marketing in the Gambia: a socio-economic perspective. *Livestock Research for Rural Development **16**:*1-12.

Obi, T. U. and Patrick (1984): The detection of PPR virus antigen by agar gel precipitation test and counter-immunoelectrophoresis. *J. Hyg*., **93:** 579-586.

Ogunsanmi, A. O., Awe, E. O., Obi, T. U and Taiwo, V. O. (2003): Peste des petits ruminants (PPR) virus antibodies in African Grey Duiker (*Sylvicapra grimmia). Afr. J. Biomed. Res*., **6:** 59-61.

OIE, (2000): Peste des petitis ruminants. In: manual of standards for diagnostic and vaccines, ed. 4. Paris, France. Pp.2-15.

OIE, (2002): Peste des petitis ruminants. Techinical disease card database. Paris, France.

OIE, (2004): Manual of diagnostic tests and Vaccines for Terresterial Animals. Paris, France.

OIE, (2005): World Animal Health HANDISTATUS ii prototype: OIE Central Bureau database. Paris, France.

Opasina, B. A., Putt, S. N. H. (1985): Outbreaks of peste des petits ruminantss in village goat flocks in Nigeria. Trop. Anim.Hlth. Prod., 17:219 -224.

Orttiz-Pelaez, A., Pfeiffer, D U., Soares-Magalhaes, R. J., Guitian, F. J. (2006). Use of social network analysis to characterize the pattern of animal movement in the initial phases of the 2001 foot and mouth disease (FMD) epidemic in the UK *Pev.Vet.med., (Article in Press).*

Ozkul, A., Akca, Y., Alkan, F., Barret, T., Karaoglu, T., Dagalp, S. B., Anderson, J., Yeilbag, K., Cokcaliscan, C., Gencay, A., Burgu, I. (2002): Prevalence, distribution, and host range of peste des petits ruminants virus, Turkey. *Emerg. Infect Dis.* **8:** 708-712.

Pandey, K. D., Baron M. D. and Barrett T. (1992): Differential diagnosis of rinderpest and PPR using biotinylated cDNA probes. *Vet. Rec.* **131**:199-200.

Peacock C (1987): Measures for assessing the productivity of sheep and goats. *Agricultural systems* **23**: 197-210.

Pegram, R G. and Tareke, F. (1981): Observation on the health of Afar livestock. *Ethiopian Vet. J.*, **5:**11-14.

Radostits, O. M., Gay, C. C., Blood, D. C., Hinchcliff, K. W. (2000.) Peste des petitis ruminants. In: Veterinary Medicine: A Textbook of cattle, sheep, pig, goats and horses, 9th ed. London: Bailliere Tindall. Pp 1077- 1079

Rajak, K. K., Sreenivasa, B. P., Hosamani, M., Singh, R. P., Sigh, S. K., Sing, R. K. (2005): Experimental studies on immunosuppressive effects of peste des petits ruminants (PPR) virus in goats. *Comp Immunol Microbiol Infect Dis.,* **28:** 287-296.

Renukaradhya, G. J., Suresh, K. B., Rajasekhar, M., Shaila, M. S. (2003): Competitive enzyme-linked immunosorbent assay based on monoclonal antibody and recombinant hemagglutinin for serosurveillance of rinderpest virus. *J Clin Microbiol***., 41:** 943-947.

Roeder, P.L., Abraham, G., Kinfe, G., Barrett, T. (1994): Peste des petits ruminants in Ethiopian goats. *Trop. Anim. Hlth. Prod*., **26:** 69–73.

Roger, F., and Bereket, Z. (1996): Contagious caprine pleuropneumonia in Ethiopia. Laboratory investigation, station and field studies in 1994.

Roger, F., Diallo, A., Yigezu, L. M., Hurard, C., Libeau, G., Mebratu, G. Y., Fuye, B. (2000): Investigation of a new pathological condition of camels in Ethiopia. *J. Camel Pract. Res*., **2:** 163-166.

Romero, C. H., Barret, T., Kitching, R. P., Bostock, C., Black, D. N. (1995): Protection of goats against peste des petits ruminants with recombinant capripoxviruses expressing the fusion and haemagglutinin protein genes of rinderpest virus. *Vaccine,* **13:** 36-40.

Rossiter, P. B., Jessett, D. M., Taylor, W. P., (1985): Microneutralisation systems for use with different strains of Peste des petits ruminantss virus and Rinderpest virus. *Trop. Anim.Hlth. Prod*., **17**: 75-81.

Saliki, J T., Libeau, G., House, J. A., Charles, A., Mebus, D., Edward, J. (1993): Monoclonal antibody-based blocking ELISA for specific detection and titration of PPR virus antibody in caprine and ovine sera. *J.clin.Micro.* **31**:1075-1082.

Saliki, J T., Libeau, G., House, J. A., Charles, A., Mebus, D., Edward, J. (1994): Comparison of monoclonal antibody-based sandwich enzyme-linked immunosorbent assay and virus isolation for detection of peste des petits ruminants virus in goat tissues and secretions. *J Clin Microbiol.,* **32:** 1349-1353.

Saliki, J. T. (1998): Peste des petits ruminants. In: US Animal Health Association, Committee on Foreign animal diseases: The gray book 6th, Richmond. Pp 1123-1125.

Scott, G. R. (1981): Rinderpest and peste des petits ruminants. In: Gibbs, E. P. J. (Ed.), Virus Diseases of Food Animals, Vol. II, New York: Academic Press. Pp 401–425.

Shaila, M. S., Shamaki, D., Forsyth, M. A., Diallo, A., Goatley, L., Kitching, R. P., Barrett, T. (1996): Geographic distribution and epidemiology of peste des petits ruminants viruses. *Virus Res.*, **43:** 149-153.

Singh, R. P., Saravanan, P., Sreenivasa, B. P, Sing, R. K., Bandyopadhyay, S. K. (2004a): Prevalence and distribution of peste des petits ruminants virus infection in small ruminants in India. *Rev. Sci. Tech. Off. Int. Epiz*., **23:** 807-819.

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

Sinnathamby, G., Renukaradhya, G. J., Rajasekahar, M., Nayak, R., Shaila, M. S.(2001): Immune responses in goats to recombinant hemagglutinin-neuraminidase glycoprotein of Peste des petits ruminants virus: Identification of a T cell determinant. *Vaccine,* **19:** 4816- 4823.

SPSS (2003). SPSS for Windows, Version 11.5 SPSS Inc., 1989-2002.

Sumption, K. J., Aradom, G., Libeau, G., Wilsmore, A. J. (1998): Detection of peste des petits ruminants virus antigen in conjunctival smears of goats by indirect immunofluorescence. *Vet Rec.*, **142:** 421- 424.

Talwar, G. P. (1983): A handbook of Practical Immunology. New Delhi Vikas Publishing House Pvt. Ltd. Pp 122-133.

Taylor, W. P. (1979): Protection of goats against peste-des-petits-ruminants with attenuated rinderpest virus. *Res. Vet Sci.*, **27:** 321-324.

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

Taylor, W. P. Abusaidy, S., Barret, T. (1990): The epidemiology of PPR in the sultanate of Oman. *Vet. Micro.*, **22**:341-352.

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

Tesfaye, D. (2006): Seroprevalences study of PPR in Gewane district, Afar Regional state, Ethiopia. Debre Zeit: Addis Ababa University, Faculty of Veterinary Medicine, DVM Thesis.

Wamwayi, H. W., Fleming, M., Barrett, T. (1995): Characterization of African isolates of rinderpest virus. *Vet. Microbiol.,* **41:** 151-163.

Winrock International (1992): Assessment of Animal Agriculture in Sub-Saharan Africa. Winrock International Institute for Agricultural Development, Morrilton, Arkansas, USA. 162 Pp.

Wous, L O. (1995): Seasonality of occurrence of PPR disease in Nigeria. A Preliminary report. *Mysore Journal of Agricultural Science* **29:** 158-162.

Yayehrade, T. F. (1997): Epidemiological survey of peste des petits ruminants and contagious caprine pleuropneumonia in selected areas of Ethiopia. Debre Zeit: Addis Ababa University, Faculty of Veterinary Medicine, DVM Thesis.

8. ANNEXES

Annex 1: Number of ovine possessed by interviewed farmers in selected PA

Number of Sheep	**No owners**	**Percentage**
No sheep	6	0.8
1-10	457	57.1
11-20	266	33.25
>20	71	8.9
Total	**800**	**100**

Annex 2: Number of caprine possessed by interviewed farmers in selected PA

Number of goats	**No owners**	**Percentage**
No goat	545	68.1
Less than 5	235	29.4
Greater than five	20	2.5
Total	**800**	**100**

Annex 3: Mean distribution of the ownership of small ruminants in the study area

Species	**Mean**	**SD**
Sheep	11.5 $\pm$ 0.26	7.32
Goats	0.98$\pm$0.08	2.20
Total	**6.24 $\pm$0.17**	**4.80**

Annex 4: Season of *rebi* practice

Period of rebi practice (N=800)	**Owners practicing rebi**	
	Number	Percent
Long rainy season	16	2.0
Short rainy season	4	0.5
Long dry season	15	1.9
Short dry season	1	0.1
Total	**36**	**4.5**

Annex 5: Summary of proportion of owners who has taken *rebi* animals

No. of *rebi* animals (N=800)	**No. owners practicing *rebi***	**Percent**
<5 animals	31	3.9
> 5 animals	5	0.6
Total	**36**	**4.5**

Annex 6: The mean number of small ruminants sold/purchased in the Study area

Variables	**Mean**	**SD**
No. Sheep sold	2.24 $\pm$ 0.09	2.84
No. Goats sold	0.12 $\pm$0.02	0.55
No Sheep bought	0.32$\pm$0.04	1.30
No. Goats bought	0.01$\pm$0.00	0.10

Annex 7: Ranking of reasons for selling small ruminants in the study area

Reasons	**Frequency**	**Percent**	**Ranking**
Fertilizer	173	16.57%	3
Clothes	303	29.02%	1
Human food	224	21.46%	2
Land tax	143	13.70%	4
School fee	105	10.06%	5
Animal feed purchase	59	5.65%	6
Security and other expenses	37	3.54%	7
Total	**1044***	**100%**	

*= *One farmer may have more than one reason for selling his animals*

Annex 8: Ranking of reasons for purchasing small ruminants in the study area

Reasons	**Frequency**	**Percent**	**Ranking**
Breeding	42	41.60%	2
Own consumption	45	44.55%	1
Profit	14	13.86%	3
Total	101	100%	

Annex 9: Locations of the different villages in the present study in terms of 3D GPS (Geographical position system)

Name of PA	Name of the Village	Altitude (masl)	GPS Coordinates (center of the village) In Degrees		Number of HH in the village	Estimated number of HH with small ruminants	Number of owners interviewed in the village	Number of Keepers interviewed in the village
			N	E				
Debele	Tinishu Amba	3335	09°40.592'	039°42.833'	42	37	10	13
Debele	Debele Aysofe Amba	3159	09°39.697'	039°41.964'	33	24	10	12
Debele	Tachignaw shengo	3278	09°39.155'	039°43.189'	20	14	10	15
Debele	Feres Amba	3092	09°38.754'	039°42.279'	26	21	10	10
Debele	Difdif	3304	09°39.055'	039°43.499'	42	33	10	10
Debele	Tach Betekirstian Amba	3228	09°37.137'	039°43.404'	33	26	10	6
Debele	Nefaso 1	3340	09°38.671'	039°43.634'	31	23	10	15
Debele	Layignaw madegidegiya	3216	09°38.225'	039°43.042'	27	20	10	12
Debele	Sengaberet	2966	09°35.261'	039°41.285'	22	17	10	13
Debele	Mitak	2943	09°35.127'	039°41.457'	26	19	10	10
Goshebado	Goshebado	2816	09°43.996	039°27.344	30	21	10	9
Goshebado	Goch Amba	2773	09°44.045	039°27.645	39	33	10	7
Goshebado	Kirite	2803	09°44.645	039°27.119	27	24	10	10
Goshebado	Akale Woyira	2807	09°45.078	039°25.808	33	28	10	8
Goshebado	Hudad Amba	2793	09°44.341	039°27.521	39	34	10	6
Goshebado	Workegur	2783	09°46.571	039°25.185	34	26	10	6
Goshebado	Key Afer	2790	09°46.320	039°25.238	24	17	10	9
Goshebado	Shinet	2744	09°46.105	039°25.588	42	37	10	11
Goshebado	Kimo	2676	09°44.903	039°28.274	25	20	10	8
Goshebado	Lay Kelem Amba	2681	09°44.591	039°27.815	29	22	10	8
Keyit	Kufir	3115	09°.43.539'	039o39.867'	42	42	10	6
Keyit	Tach Amba	2955	09°.43.706'	039°38.918'	52	43	10	7
Keyit	Lay Amba	2945	09°43.477'	039°38.954	48	37	10	6
Keyit	Dinkuan	3048	09°43.528'	039°39.337'	31	28	10	11
Keyit	Kombol	3025	09°43.276'	039° 39.817'	26	23	10	6
Keyit	Genet	2960	09° 44.193'	039° 37.986'	29	28	10	13
Keyit	Limad Amba	2932	09° 44.361'	035° 38.217'	32	27	10	11
Keyit	Libash Amba	2861	09°44.379'	039°37.452'	27	21	10	8
Keyit	Amer Ager	3278	09°43.855'	039° 40.664'	38	32	10	12
Keyit	Andwork Ager	3293	09°44.010'	039° 41.196'	34	26	10	4
Gudoberet	Kelkay Amba	3335	09°46.688'	039° 42.777'	38	38	10	12
Gudoberet	Ansas	3015	09°46.801'	039^0 39.922'	45	45	10	11
Gudoberet	Aradana Amba	3293	09°46.712'	039^0 42.125'	47	47	10	10
Gudoberet	Mewukeriya Ager	3208	09°47.665'	039° 42.215'	52	43	10	9
Gudoberet	Tosign Amba	3046m	09°47.457'	039° 40.532'	20	11	10	11
Gudoberet	Salasifa	2965m	09° 47.319'	039o 39.802'	39	39	10	9
Gudoberet	Misag	3014m	09o 48.025'	039° 40.573'	39	25	10	8

Gudoberet	Gibi	3078	09° 47.814'	039° 40.967'	24	21	10	7
Gudoberet	Mehal Ginaberet (Gosh)	3336	09° 47.732'	039° 41.947'	27	24	10	6
Gudoberet	Ginaberet	3183	09° 48.416'	039° 41.947'	30	23	10	11
Birbirsa	Dubisa	2772	09°41.734	039°26.697	21	15	10	7
Birbirsa	Ketanit	2817	09°42.293	039°26.077	26	17	10	12
Birbirsa	Shotele	2778	09°42.342	039°26.969	17	14	10	5
Birbirsa	Wobi	2818	09°41.517	039°28.016	28	22	10	11
Birbirsa	Tach Amba	2787	09°40.527	039°24.772	18	13	10	14
Birbirsa	Gendetuma	2757	09°40.450	039°25.423	20	17	10	9
Birbirsa	Koshim	2813	09°41.272	039°25.704	22	16	10	7
Birbirsa	Birbisa	2816	09°41.125	039°25.312	27	23	10	8
Birbirsa	Ketera	2753	09°42.181	039°24.989	19	15	10	5
Birbirsa	Abraham guba	2822	09°40.875	039°25.733	22	20	10	7
Abamoti	Abamote	3003	09° 45.933'	039° 39.593'	54	42	10	11
Abamoti	Jilo Bado	3081	09° 45.873'	039° 40.135'	41	34	10	7
Abamoti	Tinishu Bura	2975	09° 44.120'	039° 39.444'	38	26	10	10
Abamoti	Mamire Amba	3115	09° 44.657'	039° 40.342'	48	40	10	11
Abamoti	Tereter	2932	09° 44.804'	039° 39.422'	30	25	10	11
Abamoti	Lay Legeyida	2905	09°44.355'	039° 38.682'	39	28	10	6
Abamoti	Tach Legeyida	2849	09° 45.157'	039° 37.962'	37	23	10	9
Abamoti	Gishoch Amba	3243	09° 45.499'	039° 40.761'	39	31	10	12
Abamoti	Tikurit Bado	3293	09° 44.760'	039° 41.857'	54	47	10	12
Abamoti	Zebtina Qech	3314	09° 44.768'	039° 41.587'	39	30	10	13
Angolela	Gari Amaba	2761	09° 38.897'	039° 24.659'	53	37	10	10
Angolela	Daloch Amba	2738	09° 37.826'	039° 23.906'	34	27	10	7
Angolela	Abadale Beret	2663	09° 38.331'	039° 26.024'	32	15	10	13
Angolela	Misireta	2780	09° 37.646'	039°25.018'	29	20	10	12
Angolela	Mar Midir	2770	09° 39.977'	039° 25.467'	32	30	10	13
Angolela	Lay Tofit	2781	09° 39.737'	039° 25.070'	43	35	10	6
Angolela	Dalati	2655	09° 39.698'	033°26.363'	32	28	10	9
Angolela	Ejara Chacha	2805	09° 36.554'	039°24.092'	39	33	10	11
Angolela	Woinabadera	2830	09° 35.647'	039°24.066'	39	32	10	17
Angolela	Yasede	2770	09° 35.748'	039°23.159'	16	12	10	14
Bere Ager	Dibut	3112	09°37.263'	039° 39.271'	24	21	10	12
Bere Ager	Bedoager	3065	09°34.660'	039° 39.092'	23	23	10	10
Bere Ager	Ayalfush 2	3091	09°36.483'	039° 39.195'	34	34	10	13
Bere Ager	Lay Arsi Amba	3134	09°38.823'	039° 39.176'	27	22	10	15
Bere Ager	Belgmidir	3028	09°34.791'	039° 38.193'	36	32	10	14
Bere Ager	Qetema	3117	09°37.225'	039° 39.763'	48	43	10	8
Bere Ager	Layborebor	3063	09°36.130'	039° 40.012'	18	14	10	8
Bere Ager	Boraberet	3061	09°36.107'	039° 37.386'	33	30	10	11
Bere Ager	Alenga Washa	3052	09°36.928'	039° 37.651'	18	17	10	13
Bere Ager	Tach Arsi Amba	3065	09°37.349'	039° 38.443'	31	27	10	10

Koremargefia	Bolo	2751	09°37.317'	039° 32.836'	42	35	10	11
Koremargefia	Wanashete	2686	09°35.511'	039° 32.591'	40	37	10	10
Koremargefia	Dibe	2755	09°38.093'	039° 28.305'	43	43	10	15
Koremargefia	Weregen	2765	09°34.955'	039° 29.602'	30	24	10	19
Koremargefia	Anasi	2743	09°34.799'	039° 30.855'	35	32	10	13
Koremargefia	Gendedhe	2794	09° 35.531'	039o 31.151'	35	30	10	8
Koremargefia	Kabi Gizaw	2724	09° 49.137'	039° 23.417	30	30	10	17
Koremargefia	Wobi	2680	09°35.511'	039° 32.591'	36	34	10	23
Koremargefia	Girar Amba	2725	09°36.776'	039°.27.029'	35	31	10	13
Koremargefia	Lay Meda	2800	09°38.094'	039°.28.307'	45	38	10	11
Metikoriya	Enchelele	2728	09° 50.245'	039°.22.971'	44	31	10	6
Metikoriya	Enat Guya	2419	09°50.518'	038o 45.443'	25	21	10	10
Metikoriya	Ferego	2735	09°49.139'	039o 23.426'	39	30	10	9
Metikoriya	Arada	2394	09°48.132'	039o 24.428'	43	34	10	5
Metikoriya	Ganabet	2415	09°48.213'	039° 24.321'	39	37	10	7
Metikoriya	Gay Meda	2238	09°49.528'	039o 23.909'	42	40	10	5
Metikoriya	Enatori	2280	09°49.081'	039° 22.447'	37	33	10	12
Metikoriya	Kulkualber	2656	09°50.149'	039° 22.672'	29	22	10	8
Metikoriya	Atamober	2572	09° 50.247'	039° 23.972'	41	39	10	11
Metikoriya	Key Gedel	2575	09°50.933'	039° 21.940	31	24	10	8

Annex 10: Questionnaire format (Herd owners form)

Name of the person interviewed:
PA
Village

Herd Size

1. How many sheep do you possess? _____

2. How many goats do you possess? _____

Practice of Rebi

3. Do you practice *REBI* with animals from other PA? Yes ☐ No ☐

If yes fill the following table please, otherwise complete table for Market Issues. You can tick one or more boxes for period of Rebi

Name(s) of the concerned PA(s) and their corresponding district	**How many small ruminants in Rebi?**	**Period of Rebi**
		☐ Long rainy season ☐ Short rainy season ☐ Long dry season ☐ Short dry season

Market Issues

Which market or village do you go to, to sell or buy small ruminants? *(you can fill in one or more market boxes, forget the other purposes)*	How many times did you go to this market last year with small ruminants to sell or that you had effectively bought?	On average how many animals did you bring *each time* to this market?	How many animals did you sell last year?		What was your reason for selling? *(Please rank the answers from 1 most important)*		How many animals did you buy last year?		What was your reason for buying? *(please rank the answers from 1 most important)*	
			Sheep	Goat			Sheep	Goat		
					Fertilizer				Fattening to sell	
					Clothes				Breeding	
					Human food				Profit	
					Land tax				Own consumption	
					School fee				Other	
					Animal food					
					Security					
					Other					
Trader *(please also answer to*					Fertilizer				Fatenning to sell	
					Clothes				Breeding	

question 5)					Human food				
					Land tax				Profit
					School fee				Own consumption
					Animal food				Other
					Security				
					Other				
TOTAL									

If the answer "Trader" has been given above:

5. Which markets do the traders take your animals to? *(Please put the name of the markets and the PA and district of their location)*

☐ Don't know

If the answer "Market" has been given above, go orally with the farmer through all markets <u>one by one</u> to ask:

6. Do you pass through other PA on your way to the <u>first market</u> or coming back from it?

Yes ☐ No ☐

6.1 If yes, please give the corresponding names: _____, _____, _____

6.2 If yes, do you stop in these PA for some time and then proceed? Yes ☐ No ☐

6.3 Do you mix with herds from these PA when you stop? Yes ☐ No ☐

7. Do you pass through other PA on your way to the <u>second market</u> or coming back from it?

Yes ☐ No ☐

7.1 If yes, please give the corresponding names: _____, _____

7.2 If yes, do you stop in these PA for some time and then proceed? Yes ☐ No ☐

7.3 Do you mix with herds from these PA when you stop? Yes ☐ No ☐

Annex 11: Herd keepers form (all keepers of the village)

Date: ----------

A. General information

District
PA
Village
Number of keepers attending

1. Could you list grazing points and watering points, along with their names?

2. List name of wild ruminants found around your grazing and water points.

(Proceed to the detail description of these points)

B. Small ruminant contacts at the grazing point

Name of the grazing points	**Type of grazing point**	**Period of use (you can tick one or more boxes)**	**Does your herd mix with herds of other PA at this grazing point?**	**If yes, give the names of the concerned PA**
	☐ Communal ☐ Private	☐ Long rainy season ☐ Short rainy season ☐ Long dry season ☐ Short dry season	☐ Yes ☐ No	
	☐ Communal ☐ Private	☐ Long rainy season ☐ Short rainy season ☐ Long dry season ☐ Short dry season	☐ Yes ☐ No	
	☐ Communal ☐ Private	☐ Long rainy season ☐ Short rainy season ☐ Long dry season ☐ Short dry season	☐ Yes ☐ No	

C. Small ruminant contacts at the watering point

Name of the water points	Type of watering point	Period of use (you can tick one or more boxes)	Does your herd mix with herds of other PA at this water point?	If yes, give the names of the concerned PA
	☐ Permanent river ☐ Spring water ☐ Artificial pond	☐ Long rainy season ☐ Short rainy season ☐ Long dry season ☐ Short dry season	☐ Yes ☐ No	
	☐ Permanent river ☐ Spring water ☐ Artificial pond	☐ Long rainy season ☐ Short rainy season ☐ Long dry season ☐ Short dry season	☐ Yes ☐ No	
	☐ Permanent river ☐ Spring water ☐ Artificial pond	☐ Long rainy season ☐ Short rainy season ☐ Long dry season ☐ Short dry season	☐ Yes ☐ No	

D. Small ruminant contacts with wild ruminants at water point

Do you see wild ruminants when you are at the water points you use? Yes ☐ No ☐

Please fill the table below for the ones where wild ruminants are seen:

Name of the water point where wild ruminants are seen	Local name of the wild animal(s)	Distance (in meters)	Frequency (per week)
1			
2			
3 etc.			

E. Small ruminant contacts with wild ruminants at grazing point

Do you see wild ruminants when you are at grazing points? Yes ☐ No ☐

Please fill the table below for the ones where wild ruminants are seen:

Name of the grazing point where wild ruminants are seen	**Local name of the wild animal(s)**	**Period/ season**	**(Distance (in meters)**	**Frequency (per week)**
1				
2				
3 etc.				

F. Herd keeper attending evening meeting sheet

Please fill the table underneath after having identified the people whose corresponding owner has been interviewed during the day. Only those persons are concerned by this sheet.

No	**Name of the Keeper**	**Name of the corresponding owner**
1		
2		
3 etc.		

Number of Keepers attending and corresponding to owners interviewed during the day: ____

Total number of herd keepers interviewed: _____

Annex 12: PAs Sheet for recording and summarizing GPS coordinates and other data

Date: _____Name of the PA_____

N°	Name of the village	Altitude (m.a.s.l.)	GPS coordinates (centre of the village) In degrees		Number of household	Estimated number of household with small ruminants	Number of owners interviewed in the village	Number of Keepers interviewed in the village
			N	E				
1								
2								
3								
4								
5								
6								
7								
8								
9								
10 etc.								

Numbers picked in the box:

m. a.s.l. = metres above sea level

Annex 13: Slide show on the field work in Basona Werana District 2006/07

Slide 1 Communal grazing point (Koremargefiya PA)

Slide 2. Water points (Angolela River)

Herd keepers at Dibe village *Herd keepers at Ganabet Village*

Slide 3. Interviewing herd keepers

Gay meda Village *Wanashete Village*

Slide 4. Representative of a typical highland village

Farmer at Bolo Village *Farmer at Ganabet village*

Slide 5 Interviewing herd owners

Slide 6. Small ruminant at Debre Berhan Market (Photo by Alma Yirjo, 2007)

9. CURRICULUM VITAE

PERSONAL DATA

Full Name: Desta Beyene Ensermu

Birth Date: 26/02/1974

Place of Birth: Bako, West Shoa, Ethiopia

Marital status: Single

Nationality: Ethiopian

Membership: Ethiopian Veterinary Association (EVA) since 1999

EDUCATIONAL BACKGROUND

2005-2007: MVSc in Tropical Veterinary Epidemiology at Addis Ababa University (AAU), Faculty of Veterinary Medicine, Debre Zeit, Ethiopia.

1992-1997: Doctorate degree in Veterinary Medicine at Addis Ababa University (AAU), Faculty of Veterinary Medicine, Debre Zeit, Ethiopia.

1990-1991: Ethiopian School Leaving Certificate Examination (ESLCE), at Gedo senior secondary school (11-12), West Shoa, Ethiopia

1988-1989: Transcripts at Hareto senior secondary school (9-10), East Wollega, Ethiopia.

1986-1987: National examination certificate and transcripts at Bebella Junior Secondary school (7-8), East Wollega, Ethiopia.

1980-1985: National examination certificate and transcripts at Goben elementary school (1-6), East Wollega, Ethiopia.

WORK EXPERIENCE

1998-1999: Veterinary Division Head, importing and distributing veterinary Drugs and instrument at Ethio-Falcon Trading (PLC), A.A, Ethiopia.

2000-2001. Team Leader, Animal Health department,OBA, Arsi Zone, Merti district agriculture Development Office, Abomsa.

2001-2002: Head, Animal and Fisheries Resource Development and Health Department at OBA, Arsi Zone, Digalu and Tijo district, Sagure.

2002-2005: Senior Instructor at Alage Agricultural and technical and Vocational Education training (TVET) college lecturing on different Animal health courses

LANGUAGE ABILITY

Fluent in Listing, Speaking, Writing and Reading, Oromiffa, Amharic and English

PAPERS AND PUBLICATION

1995/96: Senior student seminar on livestock development in Ethiopia; CCPP: Epidemiology and Diagnostic Techniques, AAU,FVM, Debre Zeit, Ethiopia

1996/97: DVM Thesis: Research on Sero-epidemiological investigation of CBPP in South Western Ethiopia with laboratory works at National Veterinary Institute (NVI).

2005/06: Seminar on Current Topics: A review on Peste des Petits Ruminants (PPR) and its epidemiological status in Ethiopia.

2006/07: MSc Thesis: Study on Contact Points for the transmission dynamics of Peste des petitis ruminants (PPR) in Basona Werana District, North Shewa Zone, Ethiopia

WORKSHOP AND TRAININGS

18 April - May 10, 2005: Certificate on Advanced Veterinary Public Health, Addis Ababa University, Faculty of Veterinary Medicine, Debre Zeit, Ethiopia.

August 2004: Training on Curriculum Development for 5 days at Holleta ATVET College

February 2004: Training on Pedagogy for 5 days at Kotebe Teachers Training College.

ADDITIONAL SKILLS

Computer literacy: Microsoft words, Excel, Access and data entry and Analysis.

REFERENCES

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4. Dr. Bayisa Tesfaye, Team Leader, Animal Health Training Section, MoARD, TVET Department, Addis Ababa, Ethiopia.

10. SIGNED DECLARATION SHEET

I the under signed, declare that the thesis is my original work and has not been presented for a degree in any university.

Name: Desta Beyene Ensermu

Signature: _____

Date of submission 25/06/ 2007

This thesis has been submitted for examination with my approval as university Advisor

Dr. Yilkal Asfaw _____

Dr. Berhe G/Egizabiher _____