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REVIEW ON EPIDEMIOLOGY OF PESTE DES PETITS RUMINANTES

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I. SUMMARY

Livestock is a major part of African agricultural sector and plays an important role in food and economic security through provision of a variety of products and services including hides, skins, meat, draught power, manure, fiber, and energy and capital accumulation. Peste des petits ruminants (PPR), is an acute contagious viral disease caused by a Morbillivirus. The disease affects small ruminants, especially goats, which are highly susceptible, and occasionally wild animals. It is clinically characterized by high fever, ocular and nasal discharge, pneumonia, necrosis and ulceration of the mucuous membrane and inflammation of the gastro-intestinal tract leading to severe diarrhoea and high mortality.At necropsy, characteristic zebra markings may occur in the large intestine, but are not a consistent finding. PPR is one of the most important economical diseases where clinical case is confirmed in Ethiopian goats, however, its circulation in other animals has never been describedThe collection of specimens at the correct time is important to achieve diagnosis by virus isolation and they should be obtained in the acute phase of the disease when clinical signs are still apparent. The specimens can be swabs of conjunctival discharges, nasal secretions, buccal and rectal mucosae, and unclotted blood.Rapid diagnosis is done by immunocapture enzyme-linked immunosorbent assay (ELISA), counter immunoelectrophoresis and agar gel immunodiffusion. Polymerase chain reaction may also be used. There is no treatment for PPR. However, mortality rates may be decreased by the use of drugs that control the bacterial and parasitic complications. Methods applied for rinderpest prevention may be appropriate for PPR. These include the following quarantine, slaughter, proper disposal of carcasses and contact fomites, decontamination of facilities and equipment and restrictions on importation of sheep and goats from infected areas. A homologous PPR vaccine is also now available and gives strong immunity.

Key words: Peste des petits ruminants, Morbillivirus, Shoats, Epidemiology

II. INTRODUCTION

Livestock is a major part of African agricultural sector and plays an important role in food and economic security through provision of a variety of products and services including hides, skins, meat, draught power, manure, fiber, and energy and capital accumulation. Besides its significant contribution to agricultural gross domestic product (GDP) and to food security in many countries, livestock is an intrinsic part of people's identity and way of life (Asfaw, 1997). Small ruminants are integral part of livestock keeping in Sub-Saharan Africa (SSA) that are mainly kept for immediate cash sources, milk, meat, wool, manure, and saving or risk distribution. Small ruminants also have various social and cultural functions that vary among different cultures, socio-economies, agro-ecologies, and locations in tropical and sub tropical Africa (Kosgey, 2004).

Ethiopia's ruminant livestock population is the largest in Africa and 10th in the world (ILCA, 1991). They are important components of the livestock subsector and are sources of cash income and play a vital role as sources of meat, milk and wool for smallholder keepers in different farming systems and agro-ecological zones of the country (Getahun, 2008). They are also sources of foreign currency (Berhanu*et al.*, 2007). However, the economic gains from these animals remain insignificant when it is compared to their huge number. There are various factors that contribute for low productivity: health constraints, feed shortage both in quality and quantity, poor feeding and health management (Sisay, 2006).

The sheep and goat industry is threatened by several diseases, of which peste des petits ruminants (PPR) is one of the most important. It is first described by Gargadennec and Lalane in 1942 from Ivory Coast in West Africa, PPR has been identified in many other countries of Africa, the Arabian Peninsula and various parts of the Middle East (EMPRES, 1999). *Peste des Petits Ruminants* (PPR) or goat plague is an acute and highly contagious viral disease of small ruminants such as sheep and goats (Asim*et al.*, 2009). This disease is characterized by high fever, ocular and nasal discharge, pneumonia, necrosis and ulceration of the mucous membrane and inflammation of gastro-intestinal tract leading to severe diarrhoea (Radostits*et al.,* 2000).

The causative agent of this economically important disease of small ruminants is a *Morbillivirus*. These viruses are enveloped, non segmented negative strand RNA viruses and constitute a genus within the family *Paramyxoviridae*and the order Mononegavirales. Morbilliviruses are a pleomorphic particle with a lipid envelope which encloses a helical nucleocapsid. Nucleocapsids are usually filamentous with a herring-bone appearance; 600-800(-1000) nm long; 18 nm in diameter. The total genome length is 15200-15900 nucleotides. Full length genome sequences of Morbilliviruses are available. The genome is divided into six transcriptional units encoding two non structural (V and C protein) and six structural proteins: the nucleocapsid protein (Np), phosphoprotein (P), L for large protein, matrix (M) protein, t fusion (F) and haemagglutinin (H) (Barrett, 1999).

The virus is closely related to Rinderpest virus (RPV), another member of *Morbillivirus*genus, which causes similar disease in large ruminants. The virus is also serologically related to Measles and Canine distemper virus. The virus may survive at 60^{o}C for 60 minutes, remain stable from pH 4.0 to 10.0 and is killed by most of the disinfectants but have long survival time in chilled and frozen tissues (Abraham, 2005). PPR virus is transmitted between animals such as sheep, goats and other small ruminants through inhalation of aerosols and direct contact with ocular/nasal secretions, faeces, contaminated water and feed troughs (Intazir*et al.,* 2009). Specific clinical signs of PPR include sudden pyrexia (40-41oC), purulent ocular/nasal discharge with congested conjunctiva, erosions, respiratory distress, sneezing in an attempt to clear nose, ulceration of mucous membranes and gastroenteritis. Morbidity and mortality vary considerably, depending on the susceptibility of the small ruminants' population in an area, animal husbandry, breed and age (Intizar, 2009).

Clinically, PPR is similar to Rinderpest (RPV). Initial diagnoses were made using Agar Gel Immunodiffusion (AGID), Counter-Immunoelectrophoresis (CIEP), Enzyme Linked Immunosorbent Assays (ELISA) and Virus Neutralization assays (VNT). These assays were time consuming and laborious, rapid, sensitive molecular techniques were subsequently developed (Couacy- Hymann*et al*., 2002).

In order to control the disease, various options had been identified as control measures including; efficient disease reporting, emergency response to outbreaks, restriction of animal movement, quarantine and vaccination. Vaccination has remained the only feasible option because of the inability to afford the zoo-sanitary control measures. Attenuated rinderpest tissue culture vaccine has been used to confer immune protection of up to one year to susceptible small ruminant's population because of its cross reactivity with other members of the genus *Morbillivirus*(Intizar*et al.,* 2009).

PPR was clinically suspected for the first time in Ethiopia in 1977 in a goat herd in the Afar region, east of the country. Clinical and serological evidence of its presence has been reported by Taylor (1984) and later confirmed in 1991 with cDNA probe in lymph nodes and spleen specimens collected from an outbreak in a holding near Addis Ababa (Roeder *et al*, 1994).

Generally PPR is one of the most important diseases that seriously hinder sheep and goat production in Ethiopia and other part of the world. Knowledge of the disease in terms of the symptoms observed and prevention methods is important in combating the disease and consequently improve sheep and goat productivity.

So the objective of this seminar paper is:-

- To review the epidemiology of Peste des petits ruminants
- To highlight the economic impact of the disease

III. PESTE DES PETITS RUMINATS

A. Definition

Peste Des Petits ruminants (PPR) is highly acute contagious viral disease of shoats characterized by high fever, occular and nasal discharge, pneumonia, necrosis and ulceration of the mucous membrane and inflammation of the gastro-intestinal tract which leads to severe diarrhea and high mortality. It affects small ruminants, especially goats, which are highly susceptible, and occasionally wild animals (Misbah*, et al.,* 2009).

B. Etiology

Peste des petits ruminants (PPR) also known as goat plague, is caused by a *Paramyxovirus of the Morbillivirus* genus. It is closely related to rinderpest virus, canine distemper virus, and human measles virus. The virus has the following characteristics: may survive at 60^{o}C for 60 minutes , stable from pH 4.0 to 10.0 , killed by alcohol, ether, and detergents as well as by most disinfectants (eg, phenol, sodium hydroxide), long survival time in chilled and frozen tissues (OIE, 2002). Morbilli-viruses are highly contagious pathogens that cause some of the most devastating viral diseases of humans and animals worldwide. They include measles virus

(MV), canine distemper virus (CDV), rinderpest virus (RPV), and peste des petitsruminants virus (PPRV). They cause fever, coryza, conjunctivitis, gastroenteritis, and pneumonia in their respective host species. All members produce both cytoplasmic and intranuclear inclusion bodies. The major sites of viral propagation are lymphoid tissues. The acute diseases are usually accompanied by profound lymphopenia and immunosuppression, leading to secondary and opportunistic infections (Murphy *et al*., 1999).

Morbilliviruses are enveloped, non-segmented negative strand RNA viruses and constitute a genus within the family *Paramyxoviridae*and the order Mononegavirales. Morbilliviruses are a pleomorphic particle with a lipid envelope which encloses a helical usually filamentous with a herring-bone appearance; 600-800(-1000) nm long; 18 nm in diameter. The total genome length is 15200-15900 nucleotides. Full length genome sequences of Morbilliviruses are available. The genome is divided into six transcriptional units encoding two non-structural (V and C protein) and six structural proteins: the nucleocapsid protein (Np), which encapsulates the virus genomic RNA, the phosphoprotein (P), which associates with the polymerase (L for large protein), the matrix (M) protein, the fusion (F) and the haemagglutinin (H). The matrix protein, intimately associated with the internal face of the viral envelope, makes a link between the nucleocapsid and the virus external glycoproteins: H and F, which are responsible for the attachment and the penetration of the virus into the cell to be infected (Barrett, 1999).

Structurally, PPRV is composed of a helicoidalnucleocapsid surrounded by a lipoproteic envelope. Owing to the presence of this envelope, the virus is easily destroyed by means of lipid solvents and is very delicate, particularly outside the host. The nucleocapsid is formed by a genome surrounded by three viral proteins, the most important of which is the nucleoprotein (N protein). The viral genome is a simple, negative RNA fragment and therefore it cannot be translated directly into proteins and has to be transcribed into messenger RNA. This stage is accomplished by an RNA-dependent polymerase complex, formed by two other nucleocapsid proteins: a polymerase-associated protein (P) (a phosphorylated protein) and a large polymerase protein (L). N Protein**:** This is the major viral protein and possibly plays an important role in inducing antiviral immunity. Currently, the great interest in this protein is the use of its cDNA as a potential specific diagnostic probe. The lipoprotein envelope is coated with spicules measuring about 10 nm and is composed of two glycoproteins. Haemagglutinin (H protein): Despite its name, this protein has no haemagglutinating activity and is thus called because it is the equivalent of the haemagglutinin of the measles virus. It enables the virus to become attached to the membrane of the target cell. Fusion protein (F)has the ability to fuse: either the viral membrane to that of the target cell, enabling the nucleocapsid to be liberated into the cytoplasm; or the membrane of an infected cell to that of an adjoining cell, this being the basis of syncytium formation and of the spread of the virus without previous extracellular release. H and F proteins enable the virus to become attached to the target cell and to release its nucleocapsid into the cytoplasm. The neutralising antibodies produced by the infected host are directed against these proteins.

Therefore, the genes of these two proteins might be of use in producing a PPR vaccine, with the help of genetic engineering. In addition to the H and F proteins, there is a third viral envelope protein which coats its inner surface: the membrane protein (M). This provides the link between both external glycoproteins and the nucleocapsid and plays an important part in virus formation. There is a seventh viral protein (C protein) which differs from the others in being non-structural; it is found only in cells infected by the virus. Its exact function is not yet known (Lefevre and Diallo, 1990).

Although, there is only one serotype of the virus, PPRV isolates on the basis of partial sequence analysis of the fusion (F) protein gene, can be grouped in to four distinct lineages. Lineage 1 and 2 are found exclusively in West Africa, whereas lineage 3 has been found in eastern Africa and Arabia. The fourth lineage is confined exclusively in the Middle East Arabia and Indian subcontinent (Shaila*et al.,* 1996, and Dhar*et al.,* 2002).

C. Epidemiology

1) Geographic distribution of PPR

PPR was first described in Côte d'Ivoire in 1942 and then after, it has been recognized in many of the sub-saharian countries that lie between the Atlantic Ocean and the Red Sea (Lefevre and Diallo, 1990). PPR has been found in parts of sub-Saharan Africa for several decades and in the Middle East and southern Asia since 1993. It has been reported in Sudan, Kenya, Uganda, and Ethiopia. In Africa and Asia, the disease is particularly devastating, as these countries often use small ruminants as components of agricultural food production (EMPRES, 1999).

The affected area extends north to Egypt and south to Kenya, in Eastern- Africa, and to Gabon, in Western-Africa. PPR has not been recognized in most of Northern and Southern- Africa. In 1998, serological survey in the United Republic of Tanzania did not detect any antibodies to PPR suggesting that infection has not extended that far to the south. PPR is present in nearly all Middle Eastern countries up to Turkey (Ozkul*et al.* 2002). It is also widespread in India and southwest Asia (Shaila*et al.* 1989). Presently, PPR occurs in most African countries situated in a wide belt between the Sahara and equator, the Middle East (Arabian peninsula, Israel, Syria and Jordon) and the Indian subcontinent. It still causes serious economic losses and remains a major constraint on the development of small ruminant farms in these countries (Diallo, 2003).

PPR is considered to be one of the main constraints to improve productivity of small ruminants in the regions where it is endemic. It has 4 lineages, the four virus lineages are found in different geographic regions. Lineages 1 and 2 occur in parts of Africa, and lineage 3 has been reported from parts of Africa, the Middle East, and southern India. It is not certain whether lineage 3 has persisted in India; one study reports that there is no evidence for this virus after 1992. Lineage 4 has been found in the Middle East and the Indian sub-continent, but as of 2008, this virus has not been reported from Africa (OIE, 2008).

2) Distribution of PPRV

Africa

West Africa: PPRV was first identified in West Africa in Nigeria in 1942.It is currently believed to be endemic across much of West Africa. However, virus outbreaks are often poorly characterizeddue to the lack of reporting systems and facilities within whichto conduct molecular tests. West Africa includes 16 countriesdistributed over an area of approximately 5 million square km.A number of these countries have experienced significant outbreaksof PPRV. In recent years, material submitted to Regional ReferenceLaboratories (RRLs) has confirmed the presence of the eitherantibodies to the virus or the detection of viral nucleic acidin samples from Burkina Faso (2008), Ghana (2010), Nigeria (2007)and Senegal (2010). PPRV strains from both lineagesI and II are currently circulating across West Africa althoughundoubtedly many outbreaks are not characterized at the molecularlevel. Other cases of PPRV in sheep, goat and camel populationshave also recently been described in Nigeria (El-Yugudaet al.,2010; Ibuet al., 2008). In Burkina Faso, antibody prevalence to PPRVof 28.5 % has been reported in the north (Sow *et al.*, 2008).

East Africa: East Africa is generally used to specifically refer to the areanow comprising the countries of Kenya, Tanzania and Uganda butoften includes Somalia, Djibouti, Ethiopia and Eritrea. PPRVis endemic across the majority of these countries with genetictyping of the virus in 1996 determining a virus circulatingin Ethiopia as belonging to lineage III. Molecular tools have characterized, where appropriatesamples were available, some of these viruses as belonging tolineage III with isolates being characterized in Sudan (2000),Uganda (2007), and most recently in Tanzania (2008 and 2010). Lineage IV viruses have also been isolated fromthe Sudan in 2000, 2004, 2008 and 2009 (Khalafallaet al., 2010). Clearly both lineages III and IV are circulatingin the Sudan and further serological reports from the countryhave confirmed outbreaks of PPRV in Sudan (Osman *et al.*, 2009; Saeedet al., 2010). Swaiet al. (2009) recently confirmednatural transmission of PPRV and circulation of virus withinherds in Tanzania. Somalia was also affected by PPRV in 2006 with the central regionsbeing most seriously affected. Fortunately, the geological topologyof Somalia prevented the spread of disease across the entirecountry. Nevertheless, ring vaccination was implemented in 2009in Somalia to prevent further spread (Nyamweyaet al., 2009).

Asia

Pakistan: PPRV has been reported in Pakistan since 1991 with initial epidemicsin the Punjab region being characterized by using PCR in 1994(Amjadet al., 1996). Since then both the spread of the virusand an increase in reporting has meant that PPRV has been documentedon several occasions. Serum samples from healthy animals ina goat flock following a suspected outbreak in 2005 were seropositivefor PPRV antibodies with further reportsin the north of the country. Currently, only lineage IV virus has been identified in Pakistan (Ahmad *et al.*, 2005).

India: PPRV is endemic across much of India and an improvement in veterinaryservices, reporting networks and diagnostic capabilities acrossIndia has led to an increase in awareness of the disease. Thevirus was first reported in southern India in 1987 (Shailaetal., 1989) where it seemed to remain for several years

beforespreading across the entire country and surrounding regions.Molecular characterization of virus isolates from India showthat virtually all isolates analysed belong to lineage IV, whichuntil recently had been thought to be restricted to the ArabianPeninsula, Middle East and India. One exception to this is avirus detected in the Tamil Nadu region of India in 1992. ThisIndian isolate is the only case of a lineage III virus beingpresent in India and no further lineage III viruses have beendetected in India since its discovery. It is thought that theintroduction of the Tamil Nadu isolate was an importation throughtrade in small ruminants and that the virus was unable to spreadand establish itself in the area. Alternatively, it may havebeen replaced by lineage IV virus that swept across the Arabianpeninsula, the Middle East and the Indian subcontinent between1993 and 1995.More recent epidemiological studies of PPRV in India have characterizeda number of closely related lineage IV viruses being present(Dhar*et al.*, 2002).

Europe

Recent reports of PPRV in areas close to European borders haveincreased its profile both scientifically and in the media.Whilst this devastating disease of small ruminants has continuedto plague agriculture across Africa and Asia for many years,the threat of spread into the developed world has greatly renewedinterest in the virus. The detection of PPRV in European Turkeyin 1996 raised initial awareness of the virus and questionedthe potential for PPRV to spread across the rest of Europe (Ozkul*et al.*, 2002). Indeed, there have been numerous reports of PPRVin Turkey having now also been reported in Western Turkey, Bursaprovince (Yesilbag*et al.*, 2005) and Mugla and Aydin provinces(Toplu, 2004) in the Aegean district. Throughout 2005, 78 separateoutbreaks of PPRV were recorded across Turkey with quarantineand vaccination being used to prevent further spread of thedisease (Tufan, 2006).

3) Risk factors of PPR

The following are factors that could potentially contribute to the spread of infection following introduction into certain area: unsatisfactory levels of bio-security along the sheep/goats value chain (production to consumption), production system, complex marketing chains (involves exchange of sheep/goats from the farms through middle men and finally sold at the live markets), transport mode, quality of inspection of sheep/goat processing (monitoring and surveillance), interactions between domestic and wild ruminants and cultural factors, geographical and environmental factors,animal demographics and trade, wild ruminants' role, mechanical transmission and trade (legal/illegal/imports) (Somalia contingency plan, 2009).

4) Host Range

The natural disease (PPRV) primarily affects mainly goats and sheep, but it is usually more severe in goats, although both cattle and pigs are susceptible to infection, but do not contribute to the epidemiology as they are unable to excrete virus. The existence of sylvatic reservoirs for PPRV has been reported with infections and deaths in captive wild ungulates from several species having been described previously (Abu-Elzein*et al.*, 2004, Kinne*et al.*, 2010).Epidemics in sheep and goats, the mainstay of subsistence farming in the developing world can cause mortality rates of 50-80% in naïve populations. Antelope and other small wild ruminant species

can also be severely affected (Abu Elzein*et al*, 2004). A case of clinical disease has been reported in wildlife resulting in deaths of gazelles (Gazelladorcus), ibex (Capra ibex nubiana), gemsbok (Oryx gazelle) and Laristan sheep (Ovisorientalislaristanica). The American white tailed deer (Odocoileusvirginianus) can be infected experimentally (Saliki, 2002). Cattle, buffaloes, camels and pigs can become infected but there is little or no evidence of disease associated with their infection and is unable to transmit the disease to other animals. PPRV antigen has been detected in an outbreak of respiratory disease in camel and sick domestic buffaloes. PPRV was also suspected to be involved in the epizootic disease that affected one humped camels in Ethiopia in 1995-1996. PPRV antigen and PPRV nucleic acid were detected in some pathological samples collected during that outbreak, but no live virus was isolated. PPR is not infectious to humans. The role of wildlife on the epizootiology of PPR is unknown at this time (Roger *et al*, 2001 and Abraham *et al*, 2005).

5) *Transmission*

The virus is highly contagious and easilytransmitted by direct contact between the secretions and/orexcretions of infected animals and susceptible animals (Ezeibe*et al.*, 2008). There are several means of transmission between animals: inhalation of aerosols produced by sneezing and coughing of infected animals, direct contact with ocular, nasal, or oral secretions, direct contact with feces and fomites such as bedding, water, and feed troughs, although this is considered unlikely as the virus is inactivated quickly in the environment. Outbreaks are more frequent during the rainy season or the dry, cold season. No carrier state is known to exist (Saliki, 2008 and OIE, 2002).

6) *Morbidity and Mortality*

The morbidity and mortality rates from PPR can be up to 100% in severe outbreaks. In milder outbreaks, morbidity is still high but the mortality rate may be closer to 50%. The mortality rate may range from 0 to 90% according to the animal age, young animals over 3 months of age are no longer protected by colostral antibodies anti PPR being highly susceptible to the infection, while old animals which have survived from precedent outbreaks are protected life long, the breed, dwarf goats seem to be more affected than sahelian breeds, and the species, some findings indicate that sheep are more resistant than goats and others claim the opposite (Taylor *et al.,* 2002 and Diallo, 2003).

Poor nutritional status, stress of movement and concurrent parasitic and bacterial infections enhance the severity of clinical signs. The incidence of PPR in an endemic area is similar to that of rinderpest in that a low rate of infection exists continuously. When the susceptible population builds up, periodic epidemics occur with almost 100% mortality**.** The mortality is usually low in endemic areas, but when associated with other diseases such as capripox, it can approach 100 per cent (Kitching, 1988).

7) *Pathogenesis*

PPR virus, like other morbilliviruses, is lymphotropic and epitheliotropic. Consequently, it induces the most severe lesions in organ systems rich in lymphoid and epithelial tissues. The respiratory route is the likely portal to entry. After the entry of the virus through the respiratory tract system, it localizes first

replicating in the pharyngeal and mandibular lymph nodes as well as tonsil. Viremia may develop 2-3 days after infection and 1-2 days before the first clinical sign appears. Subsequently viremia results in dissemination of the virus to spleen, bone marrow and mucosa of the gastro-intestinal tract and the respiratory system (Abrham, 2005).

8) Clinical signs

PPR covers three essential forms: a per acute form, an acute form and a mild form. The per-acute form is seen in young goats. The incubation period is of two days on average. Then appears a strong hyperthermia (41–42 °C) quickly followed by an attack of the general state (prostration, pilo-erection, anorexia). The animal shows oral and ocular discharges (Figures 3, 4, 5 and 6). In the first days of the disease, constipation can appear. This will be followed very quickly by profuse diarrhea. In all cases, the disease leads towards death within 5-6 days after the beginning of hyperthermia. The evolution of this per acute form is so fast that it does not allow the appearance of other evocative clinical signs of the disease. The acute form, most characteristically, resembles to rinderpest. The incubation period is three to four days and the first clinical signs are identical to those of the per-acute form although they are less intense. The disease develops over a longer period. This allows the appearance of other symptoms absent in the preceding form: thus the congestive lesions of the oral mucosal membrane are replaced by ulcers covered by white necrotic tissues.

The pulmonary sign is manifested by dry cough, which quickly becomes purulent. The ocular and nasal discharges are serous at the beginning and later on, become mucopurulent. Breathing becomes difficult because of the pulmonary attack (broncho-pneumonia) and the partial nasal obstruction by thick mucosal secretions. These signs are in fact the results of bacterial complications, generally by infection with *Pasteurellamultocida*. Because of this form, PPR was for a very long time confused with pasteurellosis.

Complications of parasitic origin, such as coccidiosis, can render difficult the clinical diagnosis. Pregnant females abort in most cases. The evolution of the disease ends most often by death (40-60 %). Those animals surviving the disease remain immunized for the rest of their life. The mild forms are much more frequent than the previous ones and very often they are undetected clinically and are discovery of serosurveillance. At the moment of infection, animals may have a slight and temporary hyperthermia. Sometimes less abundant ocular and nasal discharges may appear. Dried-up purulent discharges around the nostrils of the animal can be observed and this symptom may lead to confusion with the contagious ecthyma (Abrham, 2005).

D. Pathological Lesions

1) Post mortem findings

The carcass of an affected animal is usually emaciated, the hindquarters soiled with soft/watery faeces and the eyeballs sunken. The eyes and nose contain dried-up discharges. Lips may be swollen; erosions and possibly scabs or nodules in later cases. The nasal cavity is **c**ongested (reddened) lining with clear or creamy yellow exudates and erosions. They may be dry

with erosions on the gums, soft and hard palates, tongue and cheeks and into the oesophagus. The lung is dark red or purple with areas firm to the touch, mainly in the anterior and cardiac lobes (evidence of pneumonia). Lymph nodes (associated with the lungs and the intestines) are soft and swollen. Abomasum congested with lining hemorrhages. The pathology caused by PPR is dominated by necrotizing and ulcerative lesions in the mouth and the gastro-intestinal tract (Roeder *et al*., 1994).

The rumen, reticulum and omasum rarely exhibit lesions. Occasionally, there may be erosions on the pillars of the rumen. The omasum is a common site of regularly outlined erosions often with oozing blood. Lesions in the small intestine are generally moderate, being limited to small streaks of hemorrhages and, occasionally, erosions in the first portions of the duodenum and the terminal ileum. The large intestine is usually more severely affected, with congestion around the ileo-cecal valve, at the ceco-colic junction and in the rectum. In the posterior part of the colon and the rectum, discontinuous streaks of congestion "zebra stripes" form on the crests of the mucosal folds. In the respiratory system, small erosion and petechiae may be visible on the nasal mucosa, turbinate, larynx and trachea. Bronchopneumonia may be present, usually confined to the anteroventral areas, and is characterized by consolidation and atelectasis (Abraham, 2005).

2) Histopathology

PPR virus causes epithelial necrosis of the mucosa of the alimentary and respiratory tracts marked by the presence of eosinophilicintracytoplasmic and intranuclear inclusion bodies. Multinucleated giant cells can be observed in all affected epithelia as well as in the lymph nodes (Brown *et al*., 1991). Brown and others (1991) using immunohistochemical methods detected viral antigen in cytoplasm and nuclei of tracheal, bronchial and bronchio-epithelial cell, type II pneumocytes, syncytial cells and alveolar macrophages. Small intestines are congested with lining haemorrhages and some erosion. Large intestines (caecum, colon and rectum) have small red haemorrhages along the folds of the lining, joining together as time passes and becoming darker, even green/black in stale carcasses (Abraham, 2005).

E. Diagnosis

1) Laboratory diagnosis

Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease (OIE, 2008). Swabs of the conjunctival discharges and from the nasal and buccal mucosae; for virus isolation, polymerase chain reaction (PCR) and hematology: whole blood collected in EDTA; preferably collected in early stages of disease and blood and anticoagulant should be mixed gently;for serologic needs, clotted blood can be collected at the end of an outbreak;upon necropsy aseptically collect the following tissues chilled on ice and transported under refrigeration: Lymph nodes (especially the mesenteric and bronchial nodes), Spleen and Lung (especially intestinal mucosae);Set of tissues for histopathology should be placed in 10% formalin (OIE, 2009).

2) *Diagnostic Techniques*

Agar gel immunodiffusion(AGID) :

Agar gel immunodiffusion (AGID) is a very simple and inexpensive test that can be performed in any laboratory and even in the field. Standard PPR viral antigen is prepared from mesenteric or bronchial lymph nodes, spleen or lung material and ground up as 1/3 suspensions in buffered saline. These are centrifuged at 500 g for 10–20 minutes, and the supernatant fluids are stored in aliquots at –20°C. The cotton material from the cotton bud used to collect eye or nasal swabs is removed using a scalpel and inserted into a 1 ml syringe. With 0.2 ml of phosphate buffered saline (PBS), the sample is extracted by repeatedly expelling and filling the 0.2 ml of PBS into an Eppendorf tube using the syringe plunger. The resulting eye/nasal swab extracted sample, like the tissue ground material prepared above, may be stored at –20°C until used. They may be retained for 1–3 years. Negative control antigen is prepared similarly from normal tissues. Standard antiserum is made by hyperimmunizing sheep with 1 ml of PPRV with a titre of 104 TCID50 (50% tissue culture infective dose) per ml given at weekly intervals for 4 weeks. The animals are bled 5–7 days after the last injection. Standard Rinderpest rabbit hyper immune antiserum is also effective in detecting PPR antigen (OIE, 2008).

Counter immunoelectrophoresis(CIEP):

Counter immunoelectrophoresis (CIEP) is the most rapid test for viral antigen detection. It is carried out on a horizontal surface using a suitable electrophoresis bath, which consists of two compartments connected through a bridge. The apparatus is connected to a high-voltage source. Agar or agarose (1–2%, [w/v]) dissolved in 0.025 M barbitone acetate buffer is dispensed on to microscope slides in 3-ml volumes. From six to nine pairs of wells are punched in the solidified agar. The reagents are the same as those used for the AGID test. The electrophoresis bath is filled with 0.1 M barbitone acetate buffer. The pairs of wells in the agar are filled with the reactants: sera in the anodal wells and antigen in the cathodal wells. The slide is placed on the connecting bridge and the ends are connected to the buffer in the troughs by wetted porous paper. The apparatus is covered, and a current of 10–12 milliamps per slide is applied for 30–60 minutes. The current is switched off and the slides are viewed by intense light: the presence of 1–3 precipitation lines between pairs of wells is a positive reaction. There should be no reactions between wells containing the negative controls (OIE, 2008).

Immunocaptureenzyme-linked immunosorbent assay:

The immunocapture enzyme-linked immunosorbent assay (ELISA) using three monoclonal antibodies(MAb) anti-N protein, allows a rapid differential identification of PPR or Rinderpest viruses, and this is of greatimportance as the two diseases had until recently a similar geographical distribution and may affect thesame animal species (OIE,2008).

3) *Differential diagnosis*

The differential diagnoses include rinderpest (although many reports of ‘rinderpest’ among small ruminants may have been PPR), bluetongue, contagious ecthyma, foot and mouth disease, heartwater, coccidiosis and mineral

poisoning. The respiratory signs can resemble contagious caprinepleuropneumonia (CCPP) or pasteurellosis; pasteurellosis can also be a secondary complication of peste des petits ruminants (Radostitis*et al.,* 2000).

F. Economic significance

PPR causes serious economic losses and remains a major constraint on the development of small ruminant farms. PPR is considered to be one of the main constraints to improve productivity of small ruminants in the regions where it is endemic. Thus its control is a major goal for programs aimed at poverty alleviation (Bailey *et al*, 2005 and Diallo, 2003). It is of great economic importance on the basis of mortalities, morbidity, losses through body wastage, poor food efficiency, loss of meat, milk and milk products and offspring. A consequence of this high mortality was the inclusion of PPR in the list A of the former animal disease classification of the OIE, the world organization for the Animal health. In the new OIE classification it is included in a group of economically important animal diseases, which must be notified to the Organization in all the regions where PPR is endemic (Chauhan*et al.,* 2009).

PPRV was recently detected in Kenya in 2006 in the Turkana district.The disease rapidly spread to 16 districts, including severalwhere it has been associated with severe socioeconomic consequencesfor food security and has impacted on the livelihoods of thelocal population. Mortality rates varied according to age with100 % mortality in kids, 40 % in young animals and 10 % in adultanimals. Between 2006 and 2008 it is estimated that more than5 million animals were affected across the 16 Kenyan districtswith more than half of the infected animals succumbing to disease.The annual loss attributed to PPRV in Kenya is currently thoughtto be in excess of 1 billion Kenyan shillings (US$15 million;UK 10.5 million). Vaccination and quarantine have been usedto stop the continued spread of PPRV in Kenya. However, inadequatefunding, limited stocks of available vaccine, shortage of trainedstaff to coordinate vaccination programs, tribal clashes,drought and the mobility of the pastoral communities involvedhave made the task more problematic (Anonymous, 2008). In the 2008 outbreak in Kenya, the cost of vaccines used is estimated at € 4.8 million out of a total vaccination campaign cost of €12 million. The important direct economic losses caused by PPR are often further aggravated by the sanitary measures imposed by authorities in controlling animal movement and trade restriction on their by-products. Because of the high negative economic impact in countries affected by PPR, this disease is one of the priorities of the FAO Emergency Preventive System (EMPRES) program. The presence of PPR can have a serious impact on livestock production and trade. Economic losses are due to loss of production, death, abortion and cost of controlling the disease. The presence of the disease can limit local trade and export (FAO, 2004).

G. Treatment

There is no treatment for PPR. However, mortality rates may be decreased by the use of drugs that control the bacterial and parasitic complications. Specifically, oxytetracycline and chlortetracycline are recommended to prevent secondary pulmonary infections (OIE, 2000).

H. Prevention and control

In the past, the rinderpest vaccine has been used. However, this practice is being phased out to avoid confusion during retrospective serologic studies. A homologous PPR vaccine is now available and gives strong immunity. There are also genetically engineered recombinant vaccines undergoing limited field trials (OIE, 2002).

Sanitary prophylaxis, epidemic outbreak situations: when the disease appears in previously PPR-free zones or countries: rapid identification, humane slaughter and disposal of affected animals and their contacts; carcasses burned or buried, strict quarantine and control of animal movements, effective cleaning and disinfection of contaminated areas of all premises with lipid solvent solutions of high or low pH and disinfectants as described above; includes physical perimeters, equipment and clothing, careful consideration to use of vaccine; strategic ring vaccination and/or vaccination of high-risk populations, monitoring of wild and captive animals; endemic outbreak situations: when is continually circulating: most commonly employed control mechanism is vaccination, antibodies have been demonstrated 4 years after infection; immunity is probably life-long, monitoring of wild and captive animals; especially avoiding contact with sheep and goats, protective vaccination of zoologic species may be considered and exposed or infected animals should be slaughtered and the carcases should be burned with deep burial. Methods applied for rinderpest eradication may be appropriate for PPR. These include the following quarantine, slaughter, proper disposal of carcasses and contact fomites, decontamination of facilities and equipment and restrictions on importation of sheep and goats from infected areas (Saliki, 2008 and OIE, 2002).

IV. EPIDEMIOLOGY OF PPR IN ETHIOPIA

PPR is one of the constraints of small ruminant production in Ethiopia. PPR entered Ethiopia in 1989 in the southern Omo River valley from where it moved east to Borana then northwards along the Rift Valley to Awash. The disease then spread northwards into the central Afar Region and eastwards into the Ogaden. In 1997, a survey conducted at DebreZeit abattoir demonstrated high sero prevalence rates of 85.7 percent in animals from the pastoral areas, 43.2 percent from sedentary farms and 32.9 percent from mixed farms. Strains of PPR virus that cause only sub-clinical disease have been identified in several areas of the country (Sileshi*et al*., 2009).

PPR was suspected on clinical grounds to be present in goat herds in Afar region of Eastern Ethiopia in 1977 (Pegram*et al*., 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. PPR was characterized by ocular and nasal discharges, mouth lesions, pneumonia, gastro enteritis and diarrhea. The disease in this outbreak caused more than 60% mortality. The disease became endemic in goats (Abraham and Berhan, 2001, Gopilo*et al*., 1991, and Roeder *et al*., 1994).

Small ruminants in this country mainly thrive on free-range pasture land, shrubs and forest cover. Due to the shrinkage in pasture land and forest

area, these animals move to long distance in search of fodder and water during dry season. This phenomenon is common due to different summer and winter grazing grounds depending upon the altitude. PPR is transmitted through direct contact between infected animal and susceptible population. During nomadism, animals come in contact with local sheep and goat population from where they pick up the infection or spread disease if nomadic flock is pre-exposed. Therefore, migratory flocks play an important role in transmission epidemiology of PPR. Movement of animals and introduction of newly purchased animals from the market also play an important role in transmission and maintenance of the virus. Although seasonal occurrence of PPR virus outbreaks is disputed, disease transmission is certainly affected by animal movement for which socioeconomic factors and variations in agro climatic conditions are responsible. Large group of animals move to large areas and even between different districts. With the start of rains, the movement of animals is restricted due to the easy availability of local fodder. Nutritional status of the animals also gets improved during the rains. This may reduce disease transmission after the start of rains and during the period of easy availability of fodder. Similar observations were also recorded in tropical humid zone of Southern Nigeria during a period of 5 years of observations (Taylor, 1984). In Ethiopia, PPR outbreak was reported in the pastoralist areas of Yabello*woreda*in September 2008, and later in Dire and Moyale*woredas*in March 2009. The disease has also been found to existing in Afar and Keyeryou pastoral areas in Ethiopia. However, these reports were not confirmed through laboratory tests. As a result, further diagnostic tests are being conducted through the Yabello regional and Sebeta federal veterinary diagnostic laboratories. The result of test 1472 sera with competitive ELISA was used to estimate the sero-prevalence, sera selected from the serum bank established by national animal health and research center (NAHRC) that comprise 4000 sera collected during the year 2000 and sera collected during active search for disease, tested with ELISA. Sero positivity of 13.4%, 4.3% and 4% was detected in east showa, north wollo and south wollo zone, respectively. The wide variation in sero-positivity betweenwollo area and east showa was attributed, to the presence of different patient of disease detected natural and ecological characteristics of the two area (Elzein*et al*., 2001) (Table 3)

V. CONCLUSION

Peste des petits ruminants (PPR) is a highly contagious viral disease which is threatening the production industry of nearly billions of sheep and goats in Africa, Asia, the Middle and Near East. PPR is also one of the most important economical diseases in Ethiopia, since it had been confirmed in goats in 1991. PPRV exhibits the typical characteristics of the *Morbillivirus*genus in the *Paramyxoviridae*family. Its economic importance has been highlighted by an international study which has identified it as one of the priority animal diseases to be considered in poverty alleviation policy in areas where it is endemic. It has been well documented by different authors that small ruminants are the only hosts of PPRV. Goats are severely affected while sheep undergo a mild form of the disease. However, considering the fact that, this virus can infect and cause disease in cattle and camels in some

unknown circumstances. PPR has therefore even higher priority, particularly in the current situation where vaccination against rinderpest in cattle has been stopped. For most of the countries where it is endemic, the disease control measure easy to be implemented is the vaccination. There is an effective attenuated PPR vaccine which provides a lifelong immunity to inoculated animals.

Based on the above conclusive remarks the following are recommended:

- Advice farmers/pastoralists to keep newly purchased sheep and goats separate from other animals for about three weeks.
- Advise farmers/pastoralists to isolate animals with signs of PPR immediately and to move their healthy animals to other clean area.
- Arrange with the Office of Agriculture and Rural Development to vaccinate all sheep and goats that have been in contact with sick animals. Observe the vaccinated animals very closely every day. If any animal shows signs of PPR put it with the sick ones that have been isolated.
- Arrange with the community and the Office of Agriculture & Rural Development for annual vaccination of sheep and goats against PPR.
- Notification to Authorities State and federal veterinarians should be immediately informed of any suspected cases of PPR.
- Quarantine and restrictions on movement of sheep and goats from affected areas. The affected area should be quarantined by avoiding introduction of healthy animals.
- Proper disposal of carcasses of shoats dying of the disease (burned or buried) and disinfecting contact fomites. Most common disinfectants (phenol, sodium hydroxide, alcohol, ether, and detergents) can be used.
- Focused "ring vaccination" in surrounding areas where outbreaks have been detected.
- Further study should be conducted on the epidemiology, economic importance, and prevention of PPR.

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