

## Review on Contagious Caprine Pleuropneumonia and its Status in Ethiopia

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**Abstract:** Goats are thought to have been the first animals to be domesticated for economic purposes in many countries of the world including Ethiopia. In our country, they play a crucial role in food production and can easily be sold in terms of urgent needs such as sickness, death or the payment of school. At present, goat populations in more than 40 countries are affected by CCPP and sporadic outbreaks or cases of CCPP are reported from many more countries from time to time. Contagious caprine pleuropneumonia (CCPP) is a disease of major economic importance in Africa and Asia, posing a major constraint to goat production. It has been reported from almost all regions of Ethiopia and it is more prevalent in the arid and semi-arid lowland areas of Rift Valley, Borana rangelands, South Omo, Afar and other pastoral areas. The disease is caused by members of the *Mycoplasma* genus, usually *Mycoplasma capricolum* subspecies *capricolum* but occasionally also *Mycoplasma mycoides* subspecies *capri* or *Mycoplasma mycoides* subspecies *mycoides*. It is known to be devastating disease of goats, which is characterized by 100% morbidity and 60 to 100% mortality. The main clinical signs are fever, coughing, dyspnea and death. Diagnosis is one of the most important and challenging aspects of the disease as it influences prophylactic and therapeutic regimens and the control strategies for the prevention of global spread. The contagiousness nature of the disease should be taken into consideration to prevent and control focusing on sanitary prophylaxis, medical prophylaxis, treatment and vaccination. Professionals at any country should contribute their best for the proper diagnosis of the disease to reduce the threat to other countries.

**Key words:** Ccpp • Distribution • Ethiopia • Review

### INTRODUCTION

Small ruminants play an important role in the livelihoods and food security of poor families as a source of milk, meat and wool. The great Indian leader and freedom fighter M. K. Gandhi designated goats as “poor man’s cow” [1] emphasizing the importance of small ruminants in poor countries. They can survive in areas where a cow cannot and therefore, replace the cow in importance as they are the means of livelihood for many marginal farmers that contribute significantly to the nutrition and cash income of small farmers in Africa and Asia [2]. Sheep and goats also play a critical role in the livelihoods of the traders. For instance, in the horn of Africa the trade of small ruminants is geographically dispersed in Kenya, Somalia, Djibouti and Ethiopia. The trade of live animals extends into the Middle East and Arabian Peninsula, between 3 to 4 million live sheep and goats being exported every year [3].

In Ethiopia, goats are amongst the chief economically essential animals in livestock sector and they are vital sources of cash income, meat and milk for smallholder farmers in various agro-ecological areas of the country. Large proportions of farmers depend for their livelihood on them due to their comparative advantage of short generation interval and high frequency of multiple births [4]. Despite the presence of a massive goat population and their important socio-economic role, the health of small ruminants in general and goats, in particular, has received little attention so far [5]. Even though goats represent an abundant national resource of the country, their productivity is lowered by numerous factors, including feed shortage, low genetic potential and infectious diseases. Among infectious diseases, contagious caprine pleuropneumonia (CCPP), caused by *Mycoplasma capricolum* subspecies *capripneumoniae* is one of the major constraints of goat production. This disease is highly contagious and characterized by

severe fibrinous pleuropneumonia with high morbidity and mortality and occurs in many countries in Africa and Asia. Though the disease is confined to goats, subclinical cases were reported in sheep and some wild ruminant species.

Clinically the disease is characterized by fever, respiratory distress with coughing, nasal discharge, high morbidity and mortality rates [6]. It is highly contagious severe respiratory disease. It is one of those rampant and highly contagious animal diseases with potential of rapid spread irrespective of national borders [7]. CCPP is a disease that is included in the list of notifiable diseases of the Office of International des Epizootics (OIE) [8]. Generally, it is a major threat to the goat farming industry in developing countries and is pandemic in Africa, the Middle East and Asia [8].

CCPP is a major cause of economic losses in the goat industry globally as it is known to cause up to 100% morbidity and 60–80% mortality rates, reduced milk yield, cost of treatment and vaccination of the disease and indirect loss (hard currency) due to the imposition of trade restrictions [9]. For instance, it is estimated that the total yearly cost of CCPP is about US\$507 million in endemic areas thus involving major economic losses [10, 11].

The presence of CCPP in Ethiopia had been known since 1983; however, laboratory confirmation was done later in 1990 by isolation and identification of *Mycoplasma capricolum* subspecies *capripneumoniae* (F-38) following an outbreak of CCPP in Ogaden, Eastern Ethiopia. Since then the disease has been known to be endemic in different regions of the country [12]. Outbreaks of CCPP have been reported from almost all regions of the country, especially from the lowlands areas which are known for their goat rearing practices. Furthermore, ninety-six outbreaks were reported over a period of four years (2007 to 2011) to the epidemiology unit of the veterinary directorate of the ministry of agriculture from different parts of the country [13]. Similarly, six outbreaks with 289 cases and 93 deaths, all affecting goats, were reported to the OIE in 2014 [14].

Therefore, the objectives of this seminar paper are:

- To review the current study on the disease's history, diagnosis, epidemiology and transmission
- To review the current status of contagious caprine pleuropneumonia in Ethiopia

## Literature Review

**Historical Perspectives of Contagious Caprine Pleuropneumonia:** Contagious caprine pleuropneumonia

(CCPP) was first described in 1873 in Algeria by Thomas and known under the local name of "*boufrida*" because, in the majority of diseased goats, only one lung was affected [15]. Being endemic in most of the places, initially it was not considered as a contagious disease rather climatic conditions were considered for disease occurrence. The major outbreak of CCPP in South Africa in 1881 was the main reason of exploring contagiousness of the disease by Duncan Hutcheon, a colonial veterinary surgeon, as the infection was transported with goats from Turkey [16].

At present, goat populations in more than 40 countries are affected by CCPP and sporadic outbreaks or cases of CCPP are reported from many more countries from time to time. Hence, CCPP is becoming a novel emerging and rapidly spreading disease in most parts of the world [17].

**Etiology:** There are different species of mycoplasmas that are intended to cause contagious caprine pleuropneumonia. The causative agent of contagious caprine pleuropneumonia (CCPP) is *Mycoplasma capricolum* subspecies *capripneumoniae*, which was previously known by the strain name of its type species, F38. On the other hand, experimental infection performed some 44 years ago showed that strains of *Mycoplasma mycoides* subspecies *capri* could be highly pathogenic producing severe pleuropneumonia in large proportions of the experimental goats. Apart from the local edematous reaction at the inoculation site, the gross lesions were confined to lungs, pleura and pericardium. The lung involvements were mainly unilateral. It is unclear if *Mycoplasma mycoides* subspecies *capri* has been recovered from other body sites in natural disease of goats but mastitis induced experimentally. It is believed that *Mycoplasma mycoides* subspecies *capri* has a clear tropism for lung involvement. *Mycoplasma mycoides* subspecies *mycoides* *Large Colony* has one of the widest geographical distributions of ruminant mycoplasmas being found on all continents [18].

**Clinical Signs:** The classical disease as caused by *Mycoplasma capricolum* subspecies *capripneumoniae* is a purely respiratory illness. It is characterized by a fever of 106°F (41°C), coughing and a distinct loss of vigor. Affected goats have labored breathing; later they may grunt or breathe in obvious pain. Frothy nasal discharges and stringy salivation are often seen shortly before death. In acute phase of the disease, which occurs in fully susceptible populations of goats, death occurs within 7 to 10 days of the onset of clinical signs [17].

The clinical signs described for CCPP from different parts of the world have varied enormously. This is not surprising because at least two different mycoplasmas have been regarded as causative agents of the disease. In many field outbreaks, the clinical picture has probably been further complicated by the presence of viruses and other bacteria as part of the etiologic picture. A study to correlate clinical signs and early lesions showed that affected goats died up to a week after contact with affected animals were free from lung lesions or clinical signs; between two and three weeks after contact, lung lesions were generally small and superficial characterized by hyperemia and edema with clinical signs being restricted to an infrequent cough; fever was first seen after nearly 4 weeks which correlated with lung consolidation, the area of which increased as the fever progressed [17].

CCPP causes interstitial, fibrinous pleuropneumonia, interlobular edema and hepatisation of the lung causing high mortality rates of up to 80%. In fully susceptible flocks, morbidity reaches usually 100% and mortality 70 %. A more chronic form of the disease is often seen in endemic areas and may lead to recovery of a higher percentage of infected animals, many of them being carriers of the mycoplasmas. *Mycoplasma mycoides* subspecies *capri* tends to cause a more generalized infection in which septicemia is frequently seen. An acute or per acute septicemia form of the disease involving the reproductive, respiratory and alimentary tracts have been described. In addition, thoracic and reproductive forms of the disease have been attributed to this agent. It is considerably less contagious than *Mycoplasma capricolum* subspecies *capripneumoniae* induced disease and the mortality and morbidity rates are also lower [19].

**Pathogenesis and Pathology:** Tigga, *et al.* [20] reviewed that the exact mechanism of pathogenesis of CCPP is still unclear. The mycoplasmas are extracellular pathogens that adhere to epithelial cell surfaces. Grossly consolidation of lungs is the main finding (100%) in CCPP on pathology often unilateral, followed by alveolar exudation and pleural fluid accumulation (91%) and pleural adhesion (73%) when the microscopic lesions mainly comprise of septal peribronchiolar fibrosis (82%), along with fibrinous pleuritis (64%) and peribronchiolar cuffing of inflammatory mononuclear cells (55%) in pulmonary tissue [21]. *Mycoplasma capricolum* subspecies *capripneumoniae* usually causes acute pathologic changes in young and immunocompromised animals and chronic changes in healthy resistant animals [22].

The gross lesions in classical CCPP are confined to the thoracic cavity. Pea-sized yellowish nodules are seen in the lungs in early cases, whereas in more established cases there is marked congestion around the nodules. The lesions may be confined to one lung or both and an entire lobe may become solidified. The pulmonary pleurae become thickened and there may be adhesions to the chest wall [19]. In CCPP the lung resembles “somewhat granular looking liver”, which is described as massive hepatisation of lungs seen in CBPP. In sharp contrast, *Mycoplasma mycoides* subspecies *Capri* has been reported to cause lesions in a wide variety of organs and to produce lung lesions closely resembling those seen in CBPP. The generalized lesions described include encephalitis, meningitis, lymphadenitis, splenitis, genitourinary tract inflammations and intestinal lesions, none of which are features of classical CCPP. The lung lesions which resemble those seen in CBPP are usually confined to one lung and reflect various stages of fibrinous pneumonia.

Extensive pleuritis is usually present and various stages of hepatisation and marked dilation of interlobular septa is commonly seen. The cardiac and diaphragmatic lobes are the most commonly involved. Some describe this as a mild form of CCPP; others argue that it is not CCPP. Histological examination of the lung tissues may show acute serofibrinous to chronic fibrino-necrotic pleuropneumonia with infiltrates of serofibrinous fluid and inflammatory cells, mainly neutrophils, in the alveoli, bronchioles, interstitial septae and sub pleural connective tissue. Interlobular edema is more prominent. Peribronchial and peribronchiolar lymphoid hyperplasia with mononuclear cell infiltration is also present [19].

**Diagnosis:** Diagnosis is one of the most important and challenging aspects of the disease as it influences prophylactic and therapeutic regimen and the control strategies for prevention of global spread. It might involve microbiological, biochemical, serological and gene-based identification following a clinical tentative diagnosis. Microbiological methods include culture, isolation and identification which are rather conventional ones but are still considered as standard methods of detection of *Mycoplasma capricolum* subspecies *capripneumoniae*. However, the microbiological diagnosis of CCPP is considered difficult for two main reasons; the first being very poor *in vitro* growth of *Mycoplasma capricolum* subspecies *capripneumoniae* and secondly, usual contamination of samples by other easily growing mycoplasmas. In addition, fastidiousness and special requirements of *Mycoplasma capricolum* subspecies

*capripneumoniae* add to the problem of diagnostics. Hence, other diagnostic methods should be relied on Thiaucourt, *et al.* [23].

The incubation period under natural conditions is commonly six to ten days, but may be prolonged (3-4 weeks). Some experimentally infected goats develop fever as soon as three days after inoculation and respiratory signs as early as five days, but others become ill up to 41 days after exposure. CCPP should be suspected in the field when a highly contagious disease occurs in goats characterised by pyrexia of 41°C or greater, severe respiratory distress, high morbidity and mortality and post-mortem lesions of fibrinous pleuropneumoniae with pronounced hepatisation and pleural adhesions [23].

**Clinical Diagnosis:** CCPP is strictly a respiratory disease. Peracute, acute and chronic forms occur in endemic areas. In peracute phase, affected goats may die within 1-3 days with minimal clinical signs. In acute stage, initial signs are high fever (41-43°C), lethargy and anorexia followed within 2-3 days by coughing and labored respiration. The cough is frequent, violent and productive. In the final stages of disease, the goat may not be able to move and stands with its front legs wide apart and its neck stiff and extended. Saliva can drip continuously from the mouth and the animal may grunt or bleat in pain. Frothy nasal discharge and stringy saliva may be seen terminally. Pregnant goats can abort. Acutely affected goats generally die within seven to ten days. In the chronic stage, there is chronic cough, nasal discharge and debilitation [20]. The diagnosis of outbreaks of respiratory diseases in goats and of CCPP in particular, is complicated especially where it is endemic. *Mycoplasma capricolum* subspecies *capripneumoniae* is readily contagious and fatal to susceptible goats of all ages and both sexes, rarely affects sheep and does not affect cattle [24, 25].

**Differential Diagnosis:** CCPP is complicated by other pleuropneumonic diseases causing similar syndromes. PPR, Pasteurellosis and some other *Mycoplasma mycoides* cluster particularly *Mycoplasma mycoides* subspecies *capri*, *Mycoplasma mycoides* subspecies *mycoides LC* and *Mycoplasma capricolum* subspecies *capricolum* can cause a disease that resembles CCPP [20] but may have extrapulmonary signs. That is known as 'MAKePS' syndrome, characterized by mastitis, arthritis, keratoconjunctivitis, pneumonia and septicemia in small ruminants [8].

**Field Diagnosis:** A highly contagious disease occurring in goats and characterized by severe respiratory distress, high mortality and post-mortem lesions of fibrinous pleuropneumonia with pronounced hepatisation and pleural adhesions warrants a field diagnosis of CCPP. In the field, definite diagnosis of the disease can't be established on clinical signs or on post-mortem examinations alone. In classical acute CCPP, a high mortality and typical early thoracic lesions in goats are highly indicative of *Mycoplasma capricolum* subspecies *capripneumoniae* infection, but all cases of caprine mycoplasmosis need additional laboratory tests to establish a presumptive diagnosis. It may be difficult to distinguish CCPP from an infection by *Mycoplasma mycoides* subspecies *mycoides Large Colony* or *Mycoplasma mycoides* subspecies *mycoides Small Colony*, which have a pulmonary location. In the case of *Mycoplasma mycoides* subspecies *mycoides Large Colony* infection, thickening of the interlobular septa may be evident. These lesions are similar to those observed in the case of CBPP [26]. Samples should be kept cool and shipped on wet ice as soon as possible. If transport to the laboratory is delayed more than a few days, samples may be frozen. Diagnosis must be confirmed by isolation of the agent *Mycoplasma capricolum* subspecies *capripneumoniae*. The causative agent, once isolated, can be identified by immunofluorescence or by growth or metabolic inhibition tests.

**Serological Tests:** Serology has not been widely applied to identify the cause of outbreaks of pleuropneumonia in goats and sheep due to occurrence of false positive results and that acute cases caused by *Mycoplasma capricolum* subspecies *capripneumoniae* rarely show positive titres before death. It is also difficult to rely on serology for the member of *Mycoplasma mycoides* cluster in goat due to frequent occurrence of cross-reacting of antibodies. Such tests are best used on a herd basis rather than for diagnosis in individual animals. Complement fixation test (CFT) remains the most widely used serological test for CCPP. In CCPP, the complement fixation test is the recommended test for detection of *Mycoplasma capricolum* subspecies *capripneumoniae* infection [27].

**Isolation and Biochemical Characterization:** Biochemical characterization and growth inhibition with known antisera are the main tools to identify the species of mycoplasmas although more modern techniques like PCR and restriction enzyme analysis could reveal the confirmative diagnosis [28].

**Nucleic Acid Recognition Methods:** A DNA probe that differentiates *Mycoplasma capricolum* subspecies *capripneumoniae* from others was developed. However, isolation of *Mycoplasma capricolum* subspecies *capripneumoniae* remains the confirmatory test [29].

### Prevention and Control

**Sanitary Prophylaxis:** Contagious caprine pleuropneumonia is most likely to enter a country in infected animals. It is uncertain whether long-term subclinical carriers exist; however, some outbreaks in endemic areas have occurred when apparently healthy goats were introduced into flocks. Outbreaks can be eradicated with quarantines, movement controls, slaughter of infected and exposed animals and cleaning and disinfection of the premises [30]. Some countries have included vaccination in their eradication procedures. In endemic areas, care should be taken when introducing new animals into the flock. Flock testing, slaughter and on-site quarantine may be helpful in controlling the spread of disease. Vaccines help to prevent disease in some countries. Some antibiotics, such as tetracycline or tylosin can be effective if given early [3].

**Medical Prophylaxis:** The current CCPP vaccine contains inactivated *Mycoplasma capricolum* subspecies *capripneumoniae* suspended in saponin has a shelf life of at least 14 months and provides protection for over 1 year. It is available commercially [3].

**Treatment:** Successful treatment varies on the affected site and time course of the disease. Commonly used antibiotics include tetracyclines, tylosin, spiramycin, erythromycin and tiamulin fumarate. In early treatment, the prognosis is good. The prognosis for recovery with prompt treatment is approximately 87% and animals recovered from clinical diseases may remain carrier. Different comparative therapies have been done in different areas of the world. Chakarabart, *et al.* [31] had found tylosin with Bidanzen could bring recovery of 83.5% and tylosin with Bromhexine yielded 91.6% response. They concluded that Bidanzen and Bromhexine could be used against mycoplasma pneumonia as supportive treatment for better and quick recovery.

**Vaccination:** Vaccination has been an important aspect of CCPP prevention and control in countries where it is prevalent since a large population of goats is affected and native populations are at an increased risk of outbreaks. Further, in order to prevent global spread of CCPP and to

overcome ineffectiveness of antibiotics for treating CCPP, vaccines are imperative. An experimental vaccine inactivated with saponin that protects goats against CCPP for approximately a year has been produced in Kenya. The immunity was present for at least six months after vaccination [15].

In Ethiopia, National Veterinary Institute (NVI) is also manufacturing inactivated vaccine adjuvanted with saponin from *Mycoplasma capricolum* subspecies *capripneumoniae* strain but the current vaccine supply is found to be far below the requirements of the country. The annual average production capacity of the institute does not exceed 300, 000 doses while the national demand is in million doses as CCPP is prevalent in most goat rearing areas [7].

Different preparations of vaccine trial were conducted at NVI, Ethiopia of which live vaccine trial were carried out from local isolate strain of *Mycoplasma capricolum* subspecies *capripneumoniae* that reported 84.2% seroconversion where as inactivated vaccine develop 68.4% *Mycoplasma capricolum* subspecies *capripneumoniae* antibodies of vaccinated goats [32]. At field trial of inactivated vaccine seroconversion of vaccinated goat were recorded 61.1% of various age groups of goats [5]. The other vaccine trial conducted by Tesgera, *et al.* [33] at the same organization by inactivating whole culture of the bacteria which was prepared from *Mycoplasma capricolum* subspecies *capripneumoniae* was vaccinal seed grown in Mycoplasma specific hayflick media. The culture was inactivated by 37% formalin and adjuvanted by saponin results mean seropositivity of 60.71% in the vaccinated goats.

Generally, during the application of vaccine, coordination between neighboring geographical areas and countries in vaccination is very important to control the spread of disease across the region [34].

### Epidemiology

**Hosts:** CCPP affects only goats of any breed, sex or age worldwide. Younger animals tend to suffer more severe clinical signs than adults. This specificity for both host and tissue is a striking feature of CCPP. Goats are the primary hosts. Sheep may be affected in CCPP outbreaks affecting mixed goat and sheep herds. *Mycoplasma capricolum* subspecies *capripneumoniae* has also been isolated from healthy sheep and their role as a possible reservoir must be considered. Recently CCPP was confirmed in wild ruminants kept in a wildlife preserve in Qatar [35].

For a long time, CCPP has been reported to affect only the domestic goat. Now, it is a threat of wild ungulates both in their natural habitat and in captivity that raised concerns for zoos and for the conservation of some endangered species exposed to goats [36, 37]. Clinical disease and seropositivity have been reported in sheep in contact with affected goats, but the role of sheep as reservoirs of infection is unclear [38].

**Occurrence:** CCPP has been detected in at least 30 countries across Africa, the Middle East and Western Asia. In natural infections, the transmission of the disease is by aerosol. The environment also plays an important role in the appearance, evolution and severity of CCPP. Due to the high sensitivity of mycoplasmas to the external environment, close contact is essential between infected and naive animals for transmission to take place and overcrowding and confinement thus favors their circulation. Stress factors such as malnutrition and long transport can also predispose animals to disease [18].

The disease occurs in many countries in Africa and the Middle East. Exact distribution is not well known and could well include Asian countries. *Mycoplasma capricolum* subspecies *capripneumoniae*, originally known as the F38 biotype, was first isolated in Kenya and subsequently isolated in the Sudan, Tunisia, Oman, Turkey, Chad, Uganda, Ethiopia, Niger, Tanzania, Eritrea and the United Arab Emirates. It was first reported in mainland Europe in 2004 when outbreaks were confirmed in Thrace, Turkey, with losses of up to 25% in some herds [39]. The exact distribution of *Mycoplasma capricolum* subspecies *capripneumoniae* is unknown as CCPP is often confused with other respiratory infections and isolation of the causative agent is difficult.

Contagious caprine pleuropneumonia is a significant disease of goats characterized by: (1) The disease is readily contagious to susceptible goats, (2) Sheep and cattle are not affected, (3) Local edematous reactions do not occur in goats, (4) At subcutaneous injection there is no local reaction at the injection site. In Africa where extensive and traditional husbandry is practiced, pathogens spread when animals meet at watering points and grazing areas. Breed and sex appear not to affect the epidemiology of CCPP, but age is an important factor. Though all age groups are susceptible, mortality is higher among young animals than adults [36].

Contagious caprine pleuropneumonia is transmitted by direct contact through the inhalation of infective

aerosols. Of the two known causative agents, *Mycoplasma capricolum* subspecies *capripneumoniae* is far more contagious. Outbreaks of the disease often occur after heavy rains after the stress of sudden climatic change. It is believed that a long-term carrier state may exist. The incubation period can be as short as 6 to 10 days but may be very prolonged (3-4 weeks) under natural conditions [40].

In natural infections, susceptible goats acquire the organisms by inhalation of contaminated droplets from infected goats while the clinical disease has been reported in nearly 40 countries in Africa and Asia. *Mycoplasma capricolum* subspecies *capripneumoniae* has only been isolated in 13 countries because few have the facilities for isolating and growing mycoplasmas [41].

Table 1: CCPP prevalence studies in different countries of Africa and Asia continents

Location	Prevalence	Reference
Africa		
Tanzania, Musoma	64.4	[42]
Northern Tanzania	9.2	[43]
Tanzania	52.1	[44]
Mauritius	8	[45]
Kenya, Turkana, Baringo and Kajiado	47.2	[46]
Uganda, Agago and Otuke	20.8	[47]
Asia		
India, Maharashtra	20.2	[48]
India, Madhya Pradesh	10.6	[49]
Afghanistan	0	[45]
Pakistan, Khyber Pakhtunkhwa	3.9	[50]
Pakistan, Punjab	8.5	[51]
Eastern Saudi Arabia	12.1	[37]
East Turkey	37.5	[52]

According to AU-IBAR [53], seven countries reported the occurrence of CCPP in 2011 with Ethiopia, Somalia and Tanzania having reported the disease since 2008. The disease seems to be confined to the central and eastern Africa regions based on the reports received. It is, however, impossible to rule out the presence of CCPP in other parts of the continent due to possible under-reporting of cases and lack of adequate laboratory support to correctly diagnose the disease.

AU-IBAR [53] reported that the disease affected 280 epidemiological units on the continent causing 5, 833 cases and 1, 342 deaths with a case fatality rate of 23%. The highest fatalities were recorded in Somalia with 567 deaths of goats followed by Ethiopia and Chad with 486 and 213 deaths, respectively.

Table 2: Countries reporting CCPP in AU-IBAR (2011): Pan African Animal Health Yearbook

Country	Outbreaks	Cases	Deaths	Slaughtered
Cameroon	4	43	4	6
Chad 17	636	213	49	
Ethiopia	12	1236	486	15
Kenya	12	75	19	0
Somalia	228	3614	567	65
Tanzania	4	223	53	0
Togo	3	6	0	0
Total (7)	280	5833	1342	135

Charting the monthly occurrence reports of CCPP in 2011 revealed that higher incidence of the disease was recorded in the second half of the year, suggesting that risk factors such as movement of goats are more prevalent from July to December in the affected countries. However, CCPP was also reported to have occurred in the first half of 2011 at relatively lower levels [53].

**Transmission:** Contagious caprine pleuropneumonia is a contagious disease. The disease is transmitted during close contact by the inhalation of respiratory droplets. Chronic carriers may exist; but this remains unproven. Some outbreaks have occurred in endemic areas when apparently healthy goats were introduced into flocks. Outbreaks of the disease often occur after heavy rains (e.g. after the monsoons in India), after cold spells or after transportation over long distances. This may be because recovered carrier animals shed the infectious agent after the stress of sudden climatic or environmental changes. Sources of the agent could be infectious aerosols and carrier state is likely but not proven. Inhalation of infected aerosols is the main route of transmission.

The main source of contamination is direct contact with affected animals [22]. Under cold, moist and overcrowded environment pathogen can persist longer and may lead to severe outbreaks. Shorter survival time (3-14 days) in external environment limits transmission of *Mycoplasma capricolum* subspecies *capripneumoniae*. Higher temperature inactivates *Mycoplasma capricolum* subspecies *capripneumoniae* rapidly (within 60 minutes at 56°C and within 2 minutes at 60°C). However, low temperature prolongs survival. *Mycoplasma capricolum* subspecies *capripneumoniae* can survive for 10 years in frozen infected pleural fluid.

**Persistence in Environment and Animals and Affecting Factors:** Persistence in environment and animals may be related to conduciveness of the environment, concentration of pathogen, type of breed, herd density or

immunity of the animals. The duration of infection is quite variable though recent reports suggest shorter persistence (hours to days) in the environment and longer in affected animals (weeks to months) [54].

**Potential Risk Factors for CCPP Transmission:** The occurrence of CCPP is underpinned by risk factors related to environment, production system and immune status of the host population. Livestock mobility and presence of naïve populations in an infected area are major predisposing factors. The presence of chronically infected animals in close proximity with naive animals is also an important factor [34, 36]. In regions where CCPP already occurs, the severity of the disease may depend on the following factors: the proportion of immune animals, as an animal which has survived a previous infection is thought to be protected, the presence of co-existing viral infections like PPR which may favor the development of CCPP, poor climatic conditions, such as a large temperature difference between day and night or an abrupt change of climate, especially during the period between the dry and rainy seasons and stress due to movement over long distances are main factors [15]. Animal movement due to internal insecurity, informal trade, transhumance, watering, grazing and marketing accompanied by porous borders are key points for CCPP spreading [17, 34]. In addition to these, infected goats remain carriers even when treated with the available antibiotics and are a potential risk to the healthy goats in the flock and those they come in contact at feeding areas, watering points and markets [46].

**Threat to Other Countries:** CCPP is emerging as a serious threat to other countries which either never encountered this highly contagious disease or are at risk of contracting the disease because of regular trade with the affected countries or the adjacent neighborhood by geographical location [22]. For proper reporting, identification of etiological agent is important.

**Economic Losses:** Small ruminants particularly goats (also known as poor man's cow or the rural bank), contribute significantly to the nutrition and cash income of small farmers in Africa and Asia, the two regions occupied with the largest concentration (about 72.9%) of the poor peoples in the world [2]. Africa, the Middle East and Asia regions those are home to over 80% of the world's sheep and goat population and for more than 330 million of the world's poorest people play an important role in the livelihoods and food security [3].

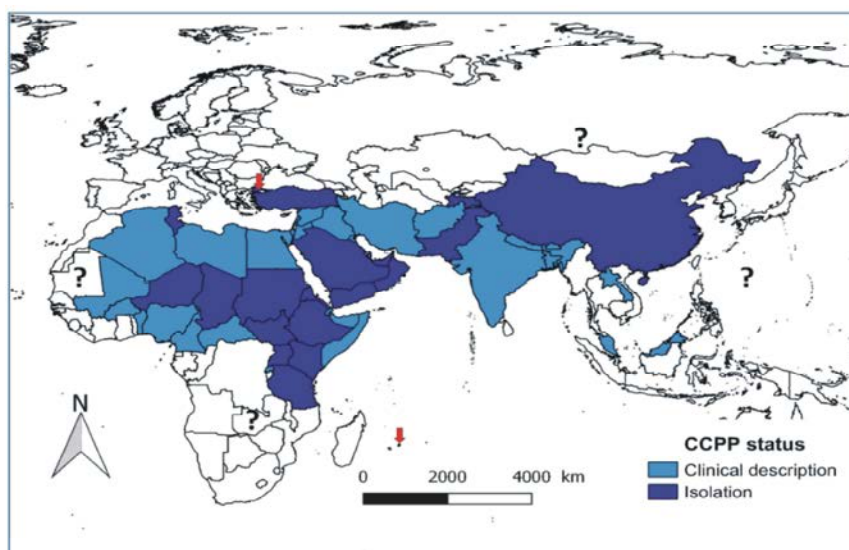


Fig. 1: A map showing the status of CCPP almost in all regions of Ethiopia from Africa continent

Being important commodities to a large segment of the world's population as a source of meat, milk and skin, goats are affected by CCPP which is a disease of major economic importance in Africa and Asia, posing a major constraint to goat production. CCPP is considered as one of the most severe and highly infectious disease of goats. It results in heavy economic losses to countries involved in goat farming especially in Africa, Asia and the Middle East. The disease is one among the mycoplasmal infections resulting in significant losses in almost 40 countries and morbidity and mortality can be as high as 100% especially in exotic breeds. In naive and native herds, 100% morbidity and 80% mortality have been noted. It is estimated that the total yearly cost of CCPP is about US\$507 million in endemic areas thus involving major economic losses. Economic losses are both by morbidity, mortality and decline or loss of production performance in addition to costs involved in prevention, control and treatment [10, 11]. Morbidity causes constraints in livestock management, overburdens with costs of treatment and imposes restriction on trade or transport. Mortality causes direct loss by death of the valuable animals. Diseased animals are usually culled in developed countries but not possible in underdeveloped and developing countries like India [10, 11].

**Status of Contagious Caprine Pleuropneumonia in Ethiopia:** In Ethiopia, CCPP has been suspected to occur for a long period, especially in areas found at the vicinity of endemic areas of Kenya and Sudan. It has been confirmed to be present in Ethiopia since 1980s. CCPP has been reported from almost all regions of Ethiopia

including Tigray, Afar, Dire Dawa, SNNP, Oromia, Benishangul Gumuz and Amhara Regional States. It is more prevalent in the arid and semi-arid lowland areas of Rift Valley, Borana rangelands, South Omo, Afar and other pastoral areas of Ethiopia where about 70% of the national goat population exist. Various authors reported different sero-prevalence rates from different parts of the country. *Mycoplasma capricolum* subspecies *capripneumoniae* induced CCPP-like clinical diseases was not observed in sheep from Ethiopia. There were mixed infection with *Mycoplasma mycoides* subspecies *mycoides* Large Colony and *Mycoplasma ovipneumoniae*. Sheep are naturally resistant to CCPP diseases. The isolation of *Mycoplasma capricolum* subspecies *capripneumoniae*, however, proves the role of sheep as reservoir of infection [55].

**Prevalence of CCPP in Ethiopia:** In Ethiopia, the circulation of CCPP has been suspected for a long period, especially in remote regions those are bordering to the known infected countries with *Mycoplasma capricolum* subspecies *capripneumoniae* like Kenya and Sudan. In 1990 outbreak of CCPP occurred in Ogaden in eastern Ethiopia and in east Shoa province. The causative agents were recovered in pure culture but *Mycoplasma capricolum* subspecies *capripneumoniae* isolated and characterized in the same year from the pleura fluid that was collected during the outbreaks in Gojam in 1982. Since then the disease has been known to be endemic in different regions of the country. Clinical diseases have been identified by various investigators especially in the pastoral areas of the country, Borana [56] and Afar [57].



A meta-analysis of CCPP conducted by Asmare, *et al.* (2016) [58] using more than twelve published articles indicated that the seroprevalence of the disease is 25.7%. Different scholars reported seroprevalence of CCPP from different districts with various proportions [59, 60, 61, 62]. In different years outbreaks of CCPP have been reported from different regions. For example, from 1999 to 2003, 62 outbreaks [63]; from 2007-2010, 94 suspected outbreaks but only one outbreak was confirmed [63] and in 2011, 12 outbreaks have been reported [64].

Table 3: CCPP prevalence studies in different areas in Ethiopia

Location	Prevalence (%)	Reference
Export oriented abattoir, Bishoftu, Ethiopia	31	[63]
Borana zone; Yabello, Dire, Moyale and Liban	19.1	[56]
Southern Ethiopia; Hammer and Benna-Tsemay	15.5	[59]
ELFORA export abattoir	11.8	[66]
Tigray and Afar region	32.7	[60]
Afar Region; Afambo, Assaita, Dubti, Mille, Gewane, Amibara, Dewe and Telalak	22.5	[61]
Southern Ethiopia; Hammer, Benna-Tsemay, Arbaminch and Boreda	18.6	[67]
Borana and Guji zone; Liben, Teltale and Moyale	13.2	[62]
Jijiga zone; Jijiga, Kebribayeh, Tuli Guled	32.6	[68]
Dire Dawa	4.92	[69]
Borana pastoral areas; Arero, Dhas, Yabello districts	31.6	[5]
Afar region; Dubti and Hadar	14.6	[45]

**Temporal Distribution of CCPP Outbreaks in Ethiopia (2007-2016):** Between 2007 and 2016, a total of 175 CCPP outbreaks were reported from different regions and districts to the Epidemiology Directorate of Ministry of Livestock and Fisheries [70]. The highest number of outbreak was reported in 2008 (16.6%) and the least report was recorded in 2016 (1.7%). The outbreak report trend gradually decreased year to year especially from 2012 toward 2016.

**Spatial Distribution of CCPP Outbreaks in Ethiopia:** The highest number of CCPP outbreaks were reported in Oromia region (40%) followed by SNNP (24%), Somali (9.7%), Afar (12.6%), Benishangul Gumuz (4.6%), Tigray (4.6%), Amhara (4%) and Gambella (0.6%) within the ten years period. 64% of the CCPP outbreaks were reported from the two regions, Oromia and SNNP, which have almost half of the country's goat population 13, 720, 656 (46.2%) [71].

**Vaccine Intervention Following CCPP Outbreaks:** From ten years retrospective data, Oromia region used the highest percentage (38.4%) of CCPP vaccine doses

followed by SNNP (25.8%), Amhara (17.7%), Somali (13.7%), Tigray (1.9%), Benishangul Gumuz (1.6%) and Gambella (0.9%) to control the outbreak. Majorities of outbreaks as well as the large percentage of vaccine doses were used in Oromia region while the lowest vaccine usage was recorded in Gambella region. No vaccine intervention was recorded in Afar region despite 12.6% of CCPP outbreak reported for the previous ten years [71].

## CONCLUSION

It has been noted that every year outbreaks of respiratory diseases occur in the goat population with an alarming rate of morbidity and mortality but lack of advanced techniques is a big hindrance in the proper diagnosis of CCPP. Diagnosis of the disease is one of the challenging aspects as it influences prophylactic and therapeutic regimen and the control strategies for the prevention of global spread. However, determination of seroprevalence of the disease and potential risk factors could pave the way in devising control and prevention strategies. From the current review the endemicity of CCPP and the epidemiological nature of the disease should be taken into consideration as it could affect the country from different corners of losses and threat of spread. It is impossible to rule out the presence of CCPP in many parts of the country due to possible under-reporting of cases and lack of adequate laboratory support to correctly diagnose the disease. Therefore, the exact epidemiological picture of CCPP in Ethiopia has to be well established through continuous gap-filling works to exactly put the updated status of the disease and professionals should contribute their best to reveal the truth behind the disease in the country for the benefits of inhabitants.

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