

## INTRODUCTION:

Contagious Bovine Pleuropneumonia (CBPP) is a highly contagious disease that affects cattle throughout most of sub Saharan Africa. It is consistently ranked amongst the most serious livestock diseases by regional and national authorities and cattle keepers alike, both FAO and AU-IBAR consider improved diagnostic tests and vaccines for CBPP to be a research priority. The disease affects both pastoralist and mixed crop-livestock systems but its impacts are greatest in pastoralist areas. CBPP causes direct impact through mortality and morbidity: up to 15% of infected animals die: lactation yields of infected cows are reduced by up to 90%: meat production is affected through reduced growth rates of infected animals: and infected draught oxen have a much reduced capacity for work. Indirect losses at the household level are incurred through treatment costs (Euro10 - 14 per animal) and movement restrictions: local quarantine and movement control measures imposed in the face of an outbreak can limit access to markets, grazing and water sources, although these are hard to enforce in remote areas. Vaccination campaigns and other control measures stretch under-resourced national veterinary authorities. The persistence of the disease in Africa represents a constant threat to other parts of the world, especially southern Europe where recurrences of CBPP have been recorded during the 1990s. Although CBPP has been successfully eliminated from Europe, North

America and Australia using a combination of strictly enforced movement control and culling, these approaches are considered to be inappropriate in Africa due to the very different socio-economic conditions on the continent.

The objective of this study:

- 1\ Estimate CBPP seroprevalence
- 2\ Identify risk factors associated with CBPP

# CHAPTER ONE

## LITERATURE REVIEW

### 1.1 Introduction and history :

Contagious bovine pleuropneumonia (CBPP) is a contagious disease of cattle caused by *Mycoplasma mycoides* subsp. *mycoides* SC (*Mmm*SC; SC = small colonies). CBPP has been known to occur in Europe since the 16th century but it gained a world-wide distribution only during the second half of the 19th century because of increased international trade in live cattle. It was eradicated from many countries by the beginning of the 20th century through stamping-out policies. However the disease persists in many parts of Africa. The situation in Asia is unclear. There have been no reported outbreaks in Europe since 1999. In natural conditions, *Mmm*SC affects only the ruminants of the *Bos* genus, i.e. mainly bovine and zebu cattle. *Mmm*SC (bovine biotype) has been isolated from buffaloes in Italy (*Bubalus bubalus*), and from sheep and goats in Africa and more recently in Portugal and in India. Among wild animals, one single case has been reported in American buffaloes (*Bison bison*) and none in African buffaloes (*Syncerus caffer*) or other wild ruminants. Wild animals do not play a role in the epidemiology of the disease. CBPP is manifested by anorexia, fever and respiratory signs, such as dyspnoea, polypnoea, cough and nasal discharges. In the case of acute outbreaks under experimental conditions, the mortality rate may be as high as 50% in the absence of antibiotic treatment. When an outbreak first occurs in an area, the mortality will be high but is often lower in the field following the primary outbreak.

CBPP was first reported from Germany in 1693. It spread rapidly over the whole Europe and from there was conveyed to South Africa , Australia , the far East , and the United State via infected cattle . Eradication of CBPP has been achieved during the past century in Europe , North America , South Africa and Australia , but it remains a serious problem in some territories of Africa south of the Sahara , in limited areas of Asia , and recently in parts of China and Mongolia (Haoward , 1994) .

CBPP has been enzootic in the Sudan since the beginning of this century . It is considered one of the most serious diseases of cattle in Sudan , leading to economical losses in forms of debilitation and death of sick animals and adversely affecting foreign trade(Abdlla, 1975; Mohamed Babiker, 2005) .

In the Sudan the disease was first observed in 1875 in Darfur province and latter spread to Khartoum where it caused great losses among cattle (Anon, 1925 ; Mohamed Babiker, 2005 ) .

The disease disappeared during Mahadi wars in 1889 . In 1912 the disease reappeared again in Kordofan province , the source of the infection was from infected trade animals brought from west Africa , from there the disease spread quickly south words and east words of the province . In 1913 the disease was reported in Nuba mountains , the white Nile ,the Blue Nile Upper Nile and Bahr El Gazal provinces . In 1914 the disease reached Khartoum province and then spread to Kassala province in 1917 and Barber province in 1923 (Anon ,1925 ;



Mohamed Babiker,2005).

Presently the disease is enzootic in the western ,southern and central provinces of the country . Rare or almost no outbreak were reported from eastern and northern provinces and this is may be due to sedentary animals movement and limited numbers in these provinces (Anon,1969; Mohamed Babiker, 2005) .

The disease in Sudan is endemic in many parts of the country. it causes serious `economic losses in the form of exhaustion of the affected animal and finally the death. moreover, it affects adversely the country livestock export to foreign markets and of export and trade to traditional markets in the Middle East (Isam, 2008).

The control measures adopted in Sudan are based on reporting, annual vaccination and restriction of movement during outbreaks. In addition, efforts were exerted from the organization of African unity through its formulation of joint pan African project (JP 15) to control CBPP (Isam, 2008) .

The policy of CBPP control in Sudan is targeting towards reduction to a low level, which justifies eradication by stamping out policy. The only method is to achieve this is by annual vaccination of the national herd. The production of a quality freeze-dried vaccine was the first step to achieve this goal (Isam, 2008).

## **1.2. Aetiology :**

### **1.2.1 Morphology :**

The mycoplasmas are the smallest free-living bacteria. They range from 0.2 – 0.8 micrometers and thus can pass through some filters used to remove bacteria . They have the smallest genome size and, as a result, lack many

metabolic pathways and require complex media for their isolation . The characteristic feature that distinguishes the mycoplasmas from other bacteria is the lack of a cell wall . thus , they can assume multiple shapes including round , pear shaped and even filamentous (Gene Mayer, 2010) . CBPP is caused by *Mycoplasma mycoides* subsp. *Mycoides* Small Colony variant( bovine biotype) this is a member of the mycoides cluster a grouping of six closely related mycoplasmas that are all pathogenic to a greater or lesser degree in ruminants.

Mmm SC type, the causative agent of CBPP belong to:

**Class;** Mollicutes, **Order;** Mycoplasmatales, **Family;** Mycoplasmataceae and **Genus;** mycoplasma (Edward and Freundt, 1969).

MmmSC, like other mycoplasmas, lacks a cell wall and is pleomorphic. In young cultures it tends to appear as branching filaments, and in old cultures as small coccid bodies. It requires special media rich in cholesterol (added serum) for growth. The organism is fragile and survives poorly outside the host. It is sensitive to desiccation and disinfectants. (William Geering; William Amanfu, 2002)

### **1.2.2 Pathogenicity :**

*Mycoplasma mycoid* subsp *mycoides* SC, is considered the most pathogenic of the mycoplasma species. Its virulence is probably the result of a coordinated action of various components of an antigenically and functionally dynamic surface architecture. The different virulence attributes allow the pathogen to evade the hosts immune defense, adhere tightly to the host cell surface, persist and disseminate in the host causing mycoplasmaemia, efficiently import energetically valuable nutrients present in the environment, and release and simultaneously translocate toxic metabolic pathway products to the host cell where they cause cytotoxic effects that are known to

induce inflammatory processes and disease. (Paola Pilo, 2007)

### **1.3. Epidemiology :**

#### **1.3.1 Host range :**

Under natural condition, CBPP occurs in cattle of the species *Bos* and allied animals including buffalo, yak, bison and even reindeer (Hutyra et al., 1938). These authors reported that goats and sheep were susceptible under experimental condition. Many of these reports need to be substantiated. Indeed Provost (1988) reviewing the literature could find no evidence to show that the domestic buffalo, *Bubalus bubalis*, was susceptible under natural or experimental condition. Experimental work in Australia showed that buffaloes could be infected by artificial means but did not spread CBPP to in-contact buffaloes (Newton, 1992). However, (Santini et al. 1992) observed pulmonary lesions and isolated MmmSC from seropositive buffaloes which had been in -contact with CBPP affected cattle in Italy. They concluded that buffaloes were susceptible albeit at a low level and that further research was necessary to clarify the role of buffaloes as a reservoir of infection for cattle. Small ruminants, in particular goats, have also been shown to harbor the MmmSC (Hudson et al., 1971). (Brando. 1975) isolated MmmSC from the milk of sheep with mastitis, as well as from goats with pneumonia in Portugal outside the endemic region of CBPP. The isolation of MmmSC from these sheep did not result in slaughter of the infected flock or imposition of other CBPP control measures. Experiments with goats in contact with cattle infected by the African strain Afade - showing clear

genetic differences with European strains – suggested a lack of susceptibility of these strains for cattle as well as their epidemiological significance are unknown at present and need to be clarified.

Table (1): Member of the *Mycoplasma mycoides* cluster

Name	Main disease	Main (and other)hosts
M. mycoides subsp. Mycoides SC variant	CBPP	Cattle (goats, sheep, buffalo)
M. mycoides subsp. Mycoides LIC variant	Caprine pneumonia, contagious agalactiae	Goats (sheep, cattle)
M. mycoides subsp. capri	Caprine pneumonia	Goats (sheep) but rare
M. capricolum subsp. capricolum	Caprine pneumonia, contagious agalactiae	Goats (sheep, cattle)
M. capricolum subsp. capricolumoniae	CCPP	Goats (sheep)
M bovine group 7 (Bg 7)	Arthritis, mastitis, calf pneumonia	Cattle

### **1.3.2 Transmission :**

CBPP is transmitted primary two different ways or routs. One is by aerosol transmission. Close proximity is necessary for transmission, while occurs primary through breathing in infected droplets from a coughing animal. The second route is direct contact. Direct contact by the introduction of a carrier animal into susceptible herd is the most common cause of outbreaks. Infection from the cow to the unborn calf has been known to occur (Jeam, 2008) .

Chronic carriers are apparently healthy animals that have a localized focus of infection sequestered in a fibrous capsule in their lungs. Such animals are often referred to as "lungers". The organism can persist in such lesions for many months, and in time the fibrous capsule may break down, allowing viable organisms to escape by the bronchi and so infect susceptible in- contact animals. This is particularly prone to occur when chronic carrier animals are subjected to stress, such as when mustered or walked for long distances. (William Geering; William Amanfu, 2002)

### **1.3.3 Morbidity and mortality:**

The attack with CBPP is variable. It is thought to be a highly contagious disease. Morbidity may be reach up to 100%. The mortality rate with CBPP is quite varied and

ranges from 10 to 70 percent in various outbreaks (masiga et al 1996).

As with many sub-acute and chronic infectious diseases, mortality may depend on other inter- current factors such as plan of nutrition, level of parasitism, and general body condition. (Ameera, 2010)

#### **1.4. Symptoms and clinical signs of the disease :**

There is considerable variation in severity of signs observed in cattle affected by CBPP, ranging from acute to chronic and subclinical forms. Respiratory distress and coughing, evident on stimulation of resting animals, are the main signs of CBPP.

##### **1. 4.1 hyper acute form :**

The clinical signs observed in the hyperacute form are much accelerated. The pathological signs are usually characteristic with marked pleural adhesion accompanied by exudative pericarditis (provost et al., 1983). Affected animals may die within a week exhibiting classical respiratory signs.

##### **1.4.2 Acute form :**

The time it takes to become ill depends primary on how healthy the animal was to begin with. In adult animals, loss of energy, lack of appetite, fever (up to 107 F°), and a drop in milk production are the first signs of CBPP. Early signs are followed by a cough which becomes moist if the animal is forced to move quickly. The signs progress to include pain in the chest, difficulty breathing, an increased breathing rate (up to 55 respiration per minute; normal is 20 RPM), moaning

while exhaling and reluctance to move. The photo depicts a coughing animal with neck extended (Jeam, 2008).

A common clinical finding in an animal infected with CBPP is the neck outstretched when the animal is coughing as depicted in the photo. Also, it demonstrates that when the animal is standing, the usual posture is with the neck forward, the legs placed far apart, and the elbows turned out (Jeam, 2008) .

#### **1. 4.3 Chronic form :**

Animals that stay sick for long periods of time (chronic) have less obvious signs of pneumonia, but may cough with exercise. These animals are often thin due to extreme weight loss and may have recurrent mild fever. They may appear to recover after several weeks. Calves born with infection commonly have arthritis in several of their joints with or without pneumonia. Joints may be warm and swollen and extremely painful. Animals that do not show signs of illness may still spread CBPP to other cattle. Animals chronically infected with CBPP are often very thin and depressed, as shown above. They may be reluctant to move (Jeam, 2008).

#### **1.5. Pathology :**

##### **1.5.1 Lesions:**

##### **1. 5.1.1 Lungs and Pleura:**

The lungs (almost always only one, the left) and pleura are affected and in most cases, only the diaphragmatic lobe is involved . Affected lobules show various stages of gray and red hepatization and the interlobular septa are greatly distended with serofibrinous exudate<sup>3/4</sup>the classical 'marbled' lung of this disease (Radostits et al., 1994). In acute forms, the yellowish fluid in the chest cavity may solidify and cover the lining of the chest and surface of the lung (the pleura) with a yellow or yellowish-grey fibrin coating resembling an omelet. Accumulation of

fibrin on the pleura causes the lung and chest wall to stick together

(adhesion). In the recovered and chronic form, fluid is rarely seen in the pleural cavity but adhesions between lung lobes and between lungs and the chest wall are commonly found. Infarcts, varying in size from about 10-300 mm, are frequently present in the affected lung tissue, which are the result from thrombosis of inter- or intra-lobular arteries and lymph vessels. The infarcts subsequently become sequestered from the adjacent parenchyma by granulation tissue/fibrous capsule<sup>3/4</sup>the sequestra of carrier cases. The diameter of a sequestrum can vary from 2 to 25 cm and the capsule can be as much as 1 cm thick .

#### **1.5.1.2 Lymphnodes:**

The lymph nodes of the chest are enlarged and edematous, and may contain petechiae and small necrotic foci.

#### **1.5.1.3 Joints and Bursa:**

In calves with poly-arthritis, affected joints are filled with fluid and abundant fibrin.

#### **1.5.1.4 Kidneys:**

In the kidney cortex, white spots of dead tissue of variable size, called infarcts, can sometimes be seen (FAO).

### **1.6. Differential Diagnosis:**

In carrying out CBPP diagnosis, it is necessary to differentiate this disease from other diseases that may present similar clinical signs or lesions. The disease pattern in a herd is as important as the findings in a single animal when carrying out an investigation for CBPP.

#### **1.6.1 Rinderpest:**

The confusion with rinderpest results from the fever and discharges observed from the eyes, nose and mouth. However, the characteristic lesions of rinderpest, which



are essentially erosions in the mouth and throughout the digestive tract, together with the profuse, often bloody diarrhoea in advanced cases, should enable easy differentiation from CBPP. Lung lesions are seen in more chronic cases of rinderpest, consisting of red areas of collapse together with emphysema of lung lobules and the septa separating them. At this stage, the erosive lesions of rinderpest may have healed.

#### **1.6.2 Foot and mouth disease (FMD):**

Salivation, lameness and fever are the cause of confusion.

#### **1.6.3 Haemorrhagic septicaemia (HS):**

This is an acute disease and most affected animals die within 6 to 72 hours after the onset of clinical signs. Buffaloes are particularly susceptible. Oedema of the throat and neck to the brisket is often very pronounced. The lung lesions seen in animals that survive the longest can appear similar to the marbling lesion of CBPP. There may be yellow fluid in the chest cavity and the affected lung may adhere to the rib cage. Thus, in the individual case distinguishing between HS and CBPP can be difficult. Gross pathologic evidence of acute septicaemia and isolation of the causative agent, *Pasteurella multocida* are essential in making a definitive diagnosis of HS.

#### **1.6.4 Bacterial or viral broncho-pneumonia:**

Clinical signs may closely resemble those of acute CBPP. Post mortem examination shows usually both lungs to be affected, fibrinous exudate may be present but not to the same extent as in CBPP. While dark, solid areas of lung may be seen, these are usually restricted to the anterior lobes (not the diaphragmatic lobe as in CBPP) and marbled lungs are not often seen.

#### **1.6.5 Theileriosis (East Coast Fever):**

Coughing, nasal and ocular discharge and diarrhoea are observed. Affected cattle show general enlargement of

superficial lymph nodes and especially those of the head. The lungs contain much clear liquid, which is also present in the chest cavity; the airways in the lung may be filled with white froth. "Cigarette burn-like" ulcers are seen in the abomasal folds. Neither pneumonia nor inflammation of the pleura is present.

#### **1. 6.6 Ephemeral fever:**

In most cases this is a self-limiting disease of short duration; most affected cattle recover quickly, even those which are severely affected. The fever fluctuates with two or more peaks. Pneumonia is not a main feature of the disease but a secondary pneumonia can occur with lung oedema and emphysema in a small proportion of cases. Confusion with CBPP arises from the presence of fever, discharges from the eyes and dripping of saliva from the mouth, lameness and swollen joints (but in animals of all ages, unlike CBPP).

#### **1.6.7 Abscesses:**

- They can be mistaken for sequestra. When cut open the content of abscesses is often offensive smelling, consisting of liquid purulent material. In abscesses a total destruction of the lung tissue occurs.

#### **1.6.8 Tuberculosis:**

Tuberculosis nodules can superficially resemble sequestra but they are degenerative cheese-like lesions, sometimes calcified. The lung tissue is destroyed and the same lesions are also seen in lymph nodes in the chest. The capsule of the tubercular nodules is not well defined when compared to that of the sequestra of CBPP.

#### **1.6.9 Farcy:**

The lung lesions of farcy differ from sequestra as they are filled with foul smelling purulent material, as described for abscesses. Similar lymph node lesions are always present.

#### **1.6.10 Actinobacillosis:**

the pulmonary lesions, when found, could be mistaken for sequestra. Lesions are generalized and seldom present in lungs alone.

**1.6.11 Echinococcal (hydatid) cysts:**

these cysts have a double wall and contain a clear liquid, often calcified when old.

**1.6.12 Foreign body reticulum pericarditis:**

clinically similar to CBPP because of the dyspnoea associated with the disease. Only one animal is usually affected.

**1.7. Diagnosis :**

**1. 7.1 Field Diagnosis :**

Contagious bovine pleuropneumonia is difficult to distinguish clinically from other causes of respiratory disease in cattle. CBPP should be considered in herds with signs of pneumonia (particularly unilateral disease) in adults and polyarthritis in calves. African strains are likely to cause severe disease in naïve animals; however, much milder disease was reported during the recent out-breaks in Europe. Lesions found at necropsy may be help-full in diagnosis; animals displaying severe clinical signs are most likely to show the characteristic lesions([www.cfsph](http://www.cfsph)).

**1.7.2 Laboratory Diagnosis:**

**1.7.2.1 Isolation and Identification of the causative agent:**

In acute cases the causative agent can be isolated from the blood and nasal swabs. If the animal has died, pleural fluid or affected lungs can be collected aseptically for cultural examination in selective media. In chronic

cases cultural examination from the sequestrum in mycoplasma medium usually yields *M. mycoides* (shallali, 1997).

#### **1.1.1.2 Serological Diagnosis :**

##### **1.7.2.2.1 Slide Agglutination Test (SAT):**

The SAT using serum or blood are sensitive in early stages of the disease and suitable for establishing a preliminary diagnosis when large numbers of cattle are involved and for selecting these cattle to be bled for CFT. This test is less suitable for detecting chronic cases. So it is recommended to be used as a herd test rather than on individual animal (shallali, 1997).

##### **1. 7.2.2.2 Growth Inhibition Test (GIT):**

The DGIT is based on the direct inhibition of the growth of the agent on a solid medium by a specific hyperimmune serum (14). However, cross-reactions within the mycoides cluster are common and great care should be taken to differentiate *MmmSC* (bovine biotype) from *MmmLC* (caprine biotype; LC: large colonies). It is a simple test to perform, but some results require experience to be interpreted: small inhibition zones (less than 2 mm wide), partial inhibition with 'breakthrough colonies', false-negative and false-positive reactions (very rare). The quality of the hyperimmune serum used in this test is critical for good results (OIE, 2008).

##### **1.7.2.2.3 Complement Fixation Test (CFT):**

The Complement Fixation Test (CFT) is a classical serological test designed to measure serum levels of specific antibody to antigens, e. g. bacteria, virus, parasite and fungus.

The CFT, the approved OIE test, although specific, lacks sensitivity. With a positive result being any reaction at 1/10 or higher, the CFT is also not robust. In addition, it requires highly trained staff to perform it accurately and consistently.(Regalla, 1995)

#### **1.7.2.2.4 Enzyme Linked Immunosorbant Assay (ELISA):**

A (c)ELISA has been developed (Le Goff and Thiaucourt, 1998) and validation tests have been performed in Africa (Thiaucourt et al, 1999).

The enzyme linked immunosorbent assay (ELISA) was used to detect antibodies against *M. mycoides* (Onoviran and Taylor Robenson, 1971). The test was found more sensitive than the CFT, slide agglutination serum test and agar gel immune- diffusion test . it could detect *M. mycoides* antibodies in sera of cattle at least 19 month of infection and 25 month after vaccination .

#### **1.7.2.3 Polymerase Chain Reaction (PCR):**

PCR is a rapid and sensitive diagnostic method. It allows detection of *MmmSC* directly in samples of lungs, bronchial lymph nodes, nasal swabs, pleural fluid and blood. Pre-incubation for 24 h of clinical specimens in growth medium may increase test sensitivity.(De Santiset et al., 1997)

In particular, the sensitive nested PCR system has been used for detection of *Mmm SC* from culture and clinical material where the target organism may be low numbers such as in nasal swab samples (Hotzel et al , 1996; Miseres et al, 1997).

### **1.8. Disease Control and Eradication :**

Methods of control depends on the disease status in a given area, state of the country (clean or enzootic), on the mode of animal husbandry(sedentary or nomadic)and the financial status of the country, state or even the owners (shallali, 1997).

Control of CBPP is facing many constraints, which could be summarized in the following:

- 1- Nomadism
- 2- Technical constraints
- 3- Financial constraints
- 4- Civil strife
- 5- Organization and planning constraints
- 6- Political instability

Strategy to control CBPP in Sudan depends on the division of the country into three epidemiological zones according to previous outbreaks of CBPP,

- i. Free zone in the northern region of Sudan
- ii. Surveillance zone in the central region
- iii. An endemic zone in the southern region

Each zone has specific measurements to be adopted for the control of CBPP. The future policy in this aspect is mass vaccination in the endemic zone for at least five years during which surveillance (abattoir and laboratory) will be carried out to monitor progress in reduction of CBPP disease prevalence. The northern region is considered free from the disease since no outbreaks have been reported for more than 20 years (Isam, 2008).

### **1. 8.1 Stamping out :**

Stamping out is certainly the most rapid and effective method of CBPP control (as it is for many other transboundary animal diseases), and international recognition of disease-free status can be more quickly regained for export trading purpose if stamping out is applied. (William Geering; William Amanfu, 2002)

'stamping out' requires the slaughter of either all animals in infected herds or of all animals in a defined infected geographic area, which has been effectively 'sealed off' to prevent animals leaving. However this approach is unlikely to be economically or socially acceptable in many endemic countries.

In the Sudan this can not be done but cattle owner are persuaded to kill their clinically sick animals (Abdalla, 1975) so when the disease is reported in the herd its movement is restricted and segregated in grazing and watering (Ameera, 2002).

### **1. 8.2 Vaccination :**

Vaccination programs as components of a CBPP eradication campaign must be comprehensively and consistently applied until there is evidence from disease surveillance that the disease has either apparently disappeared or at least the incidence has fallen to an extremely low level. (William Geering; William Amanfu, 2002)

In most African countries, for the time being, mass vaccination (or restricted to target key areas) and where possible controls of animal movement remain the most practical option. To obtain the desired results an exercise aimed at controlling CBPP through mass vaccination should endeavour to achieve high immunization coverage using high quality vaccines, which should be administered, at short intervals especially during the initial stages (90 – 92% of the population). (Gedlu, 2004)

Live, attenuated CBPP vaccine are used. Vaccine strains that are currently in use are T1-44 and T1-SR. T1-44 is currently the preferred vaccine in most countries. However, it has been criticized in some countries for causing excessive local reactions in vaccinated animals. It essential that vaccine be procured from reliable manufacturers who adhere to internationally recognized standards of good manufacturing practice and quality assurance for vaccine seed management, viable mycoplasma titer, purity, safety and potency. Freeze-dried vaccine is usually used. However it is essential that adequate cold-chain facilities are available at central and local vaccine storage depots, and from

there to the points of injection in the field. (William Geering; William Amanfu, 2002)

### **1. 8.3 Chemotherapy :**

Another method of control is chemotherapy with broad spectrum antibiotics. This is only recommended for control of severe local reaction at the vaccination site since its use on actual case of CBPP could lead to a high incidence of carrier animals with a sequestra in their lungs. In 1976 the FAO/OIE OAU pane unanimously opposite therapeutic treatment for the actual case of CBPP and strongly recommended that mass drug or antibiotic treatment of CBPP to be discouraged (FAO Report, 1967).

Antibiotic treatment against CBPP is widely used. It is not part of any official control strategy due to suspicion that its use could facilitate developments of sequestra, increase

the number of carrier animals, increase development of resistant strains, and mask the occurrence of clinical disease (Provost et al., 1987). Masking of clinical disease will make diagnosis difficult, which may contribute to unrecognized infections and CBPP transmission. Nevertheless, antibiotics are widely used in pastoralist communities

(Mariner et al., 2006; Twinamasiko et al., 2004; Msami et al., 2001). At a meeting of 22 international experts organized by FAO in 2003 it was recommended that chemotherapy be reconsidered for CBPP control. Both in-vivo and in-vitro studies demonstrating usefulness of antibiotics for treating CBPP have been reported (Hübschle et al., 2006; Twinamasiko et al., 2004; Yaya et al., 2004; Ayling et al., 2000). In an in-vitro experiment, tilmicosin, danofloxacin, oxytetracycline, florfenicol and spectinomycin were found to be effective against a variety of strains of MmmSC isolated from CBPP cases that had occurred in Africa and Europe (Ayling et al., 2000). In a



study carried out in Namibia, it was demonstrated that naïve animals kept in-contact to denofloxacin treated animals with CBPP had significantly fewer lesions, were less likely to die and to develop clinical disease than naïve animals kept in-contact to untreated animals with CBPP. In the same study, MmmSC was isolated from a limited number of in-contact controls kept with the treated animals suggesting low spread of infection (Hübschle et al., 2006). In a different trial, long-acting tetracycline was demonstrated to be effective in limiting clinical severity of the disease but ineffective in the prevention of persistence of viable MmmSC in treated animals (Niang et al., 2007; Yaya et al., 2004). Thus, the direct effect of tetracycline on the individual is positive (less clinical damage), but the indirect effect on the population may be negative (masking of signs leading to transmission). (Niwael, 2009)









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