

## CONTAGIOUS BOVINE PLEUROPNEUMONIA

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### Definition [top](#)

Contagious bovine pleuropneumonia (CBPP) is a highly infectious acute, subacute, or chronic disease, primarily of cattle, affecting the lungs and occasionally the joints, and caused by *Mycoplasma mycoides mycoides*.

### Etiology [top](#)

Contagious bovine pleuropneumonia is caused by *M. mycoides mycoides* small-colony type (SC type). *M. mycoides mycoides* large-colony type is pathogenic for sheep and goats but not for cattle. *M. mycoides mycoides* (SC type) survives well only in vivo and is quickly inactivated when exposed to normal external environmental conditions. *M. mycoides mycoides* does not survive in meat or meat

products and does not survive outside the animal in nature for more than a few days. Many of the routinely used disinfectants will effectively inactivate the organism.

### **Host Range** [top](#)

Contagious bovine pleuropneumonia is predominantly a disease of the genus *Bos*; both bovine and zebu cattle are naturally infected. There are many reported breed differences with respect to susceptibility. In general, European breeds tend to be more susceptible than indigenous African breeds (8). There does seem to be some age resistance, for animals less than 3 years of age are less resistant to experimental challenge (5). In zoos the infection has been recorded in bison and yak. Although it has been reported that the domestic buffalo (*Bubalus bubalis*) is susceptible, the disease is difficult to produce experimentally in this species (7).

### **Geographic Distribution** [top](#)

Contagious bovine pleuropneumonia is endemic in most of Africa. It is a problem in parts of Asia, especially India and China. Periodically, CBPP occurs in Europe, and outbreaks within the last decade have occurred in Spain, Portugal, and Italy. Contagious bovine pleuropneumonia was eradicated from the United States in the nineteenth century. It is of historical interest that the Bureau of Animal Industries, which is the forerunner of the USDA's Animal and Plant Health Inspection Service, was formed in 1884 specifically to eradicate CBPP. The United States was declared free of CBPP only 9 years later in 1893. Currently, CBPP is not present in the Western hemisphere.

### **Transmission** [top](#)

Contagious bovine pleuropneumonia is spread by inhalation of droplets from an infected, coughing animal. Consequently, relatively close contact is required for transmission to occur. Outbreaks usually begin as the result of movement of an infected animal into a naive herd. It is widely believed that recovered animals harboring infectious organisms within a pulmonary sequestrum, may become active shedders when stressed. Although this may be a factor in some outbreaks, it has not been substantiated experimentally (9). There are limited anecdotal reports of fomite transmission, but this means of transmission is not generally thought to be a problem.

### **Incubation Period** [top](#)

The time from natural exposure to overt signs of disease is variable but generally

quite long. It has been shown that healthy animals placed in a CBPP-infected herd may begin showing signs of disease 20 to 123 days later (7). Experimentally, subsequent to instillation of large quantities of infective material at the tracheal bifurcation, the incubation period is 2 to 3 weeks.

### Clinical Signs [top](#)

Usually the first abnormality noticed is a depressed, inappetent animal with fever. Coughing may be the next sign (Fig. 36) followed by evidence of thoracic pain and an increased respiratory rate. As pneumonia progresses and animals become increasingly dyspneic, animals are inclined to stand with elbows abducted in an attempt to decrease thoracic pain and increase chest capacity. Auscultation of the lungs reveals any of a wide variety of sounds, depending on how severely the subjacent pulmonary parenchyma is affected.

Crepitations, rales, and pleuritic friction rubs are all possible. Percussion over affected areas reveals dullness. When pulmonary involvement is extensive and severe, there will be very labored respiration and, sometimes, open-mouthed breathing. Occasionally in calves, pneumonia may be accompanied by a polyarthritis. Animals affected in this manner may be very reluctant to move and stand stiffly with a distinctly arched back. Getting up and down may cause obvious discomfort. Large joints (Fig. 37) may be distended and warm on palpation. If joint pain is severe, animals may be so reluctant to bend the joints that they lie in lateral recumbency with legs outstretched. Contagious bovine pleuropneumonia often evolves into a chronic disease. This form, characterized by ill thrift and recurrent low-grade fever, may be difficult to recognize as pneumonia. Forced exercise may precipitate coughing.

### Gross Lesions [top](#)

The gross pathologic features of CBPP are quite characteristic (3). If the animal dies, there is usually extensive and marked inflammation of the lung and associated pleuraar layering of yellow fibrin which, with time, becomes fibrosed, often resulting in adhesions between parietal and visceral pleurae. Not uncommonly, within an affected lung will be found a sequestrum - a focus that has undergone coagule (Fig. 38). In severe cases there can be abundant fluid in the thoracic cavity. The inflammation is not uncommonly unilateral (Fig. 39). The initial focus can be in any part of the lung and, in fatal cases, usually has spread locally and extensively to include a sizable segment. The affected pulmonary parenchyma is odorless and often has all stages of lesions with both acute and chronic inflammatory changes adjacent to one another. The predominant gross change is consolidation, or thickening, of individual lobules, which become

encased in markedly widened interlobular septa, resulting in the very characteristic marbled appearance (Figs. 40, 41). Interlobular septa become distended first by edema, then by fibrin, and finally by fibrosis. The overlying pleura may be very thickened by an irregulative necrosis (Fig. 42) and is effectively sealed off from the rest of the lung. Such sequestra may even be found in recovered animals. It has been shown that *M. mycoides mycoides* (SC-type) can survive within these sequestra for months or possibly longer (9).

### **Morbidity and Mortality** [top](#)

The attack rate with CBPP is variable. It is not thought to be a highly contagious disease. With increased confinement of animals, morbidity rises. The mortality rate with CBPP is quite varied and ranges from 10 to 70 percent in various outbreaks. As with many subacute and chronic infectious diseases, mortality may depend on other intercurrent factors such as plane of nutrition, level of parasitism, and general body condition

### **Diagnosis** [top](#)

#### **Field Diagnosis** [top](#)

Clinical diagnosis of CBPP is difficult. At postmortem the gross lesions of CBPP are somewhat distinct. Often there is an extensive deposition of fibrin and a large quantity of straw-colored fluid in the thoracic cavity with a prominent marbling of pulmonary parenchyma. Generally, all stages of pathologic changes, from acute through to chronic, are present in one animal. In some chronic cases the nodules of inflammation may not be readily apparent from the pleural surface but can be palpated within the parenchyma. Unlike many other pneumonias, CBPP is often unilateral.

#### **Specimens for the Laboratory** [top](#)

From a live animal, nasal swabs, transtracheal washes, or pleural fluid obtained by thoracic puncture all provide good samples for isolation attempts. From a dead animal that has had severe clinical disease, the best specimens to submit are affected lung, swabs of major bronchi, tracheo-bronchial or mediastinal lymph nodes, and joint fluid from those animals with arthritis. All samples should be collected aseptically and, if possible, placed in transport medium (heart infusion broth, 20 percent serum, 10 percent yeast extract, benzylpenicillin at 250 to 1000 IU/ml). 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 (1). Blood should be collected for serum.

## Laboratory Diagnosis [top](#)

A definitive diagnosis is made by isolating and identifying the organism. Serology is helpful in the diagnosis of CBPP. Because CBPP is a subacute to chronic disease, most animals will have developed antibodies by the time of clinical disease.

## Differential Diagnosis [top](#)

Clinically, CBPP may be confused with other pneumonic conditions, most especially bovine pasteurellosis. However, bovine pasteurellosis would likely spread much more rapidly and consequently the epidemiologic picture may be distinct.

## Treatment [top](#)

*Mycoplasma mycoides mycoides* (SC-type) is susceptible to a variety of antimicrobials, including streptomycin, oxytetracycline, and chloramphenicol. However, antimicrobial therapy may only serve to slow the progression of the disease or may even in some cases favor the formation of sequestra. In the case of chronically affected animals or subclinically affected carriers, the organisms may be in an inaccessible location within an area of coagulative necrosis, which by definition is not served by a blood supply.

## Vaccination [top](#)

A modified live vaccine is available for use in enzootic areas. A major drawback of this vaccine is that it generates an unpredictable local reaction. For this reason it is often given in the tail tip, which may become necrotic and slough. Immunity subsequent to vaccination is generally good and lasts at least 12 months. The CBPP vaccine is often given in combination with the vaccine for rinderpest.

## Control and Eradication [top](#)

### Prevention [top](#)

Because CBPP is a chronic disease that may exist subclinically in carrier animals, it is important to maintain sufficient regulatory restrictions to prevent its introduction in apparently healthy animals. Serologic testing of susceptible animals for importation is a recommended safeguard.

## Containment and Eradication [top](#)

Successful control of the spread of CBPP rests on removing susceptible animals from any possible contact with CBPP-infected animals, whether they are clinically affected or subclinical carriers only. On-farm quarantine of suspicious and contact animals would be very advantageous in stemming the spread of the disease. In an outbreak situation, testing, slaughter, and quarantine would be the methods of choice.

### Public Health [top](#)

There is no evidence to indicate that humans are susceptible to this disease.

### GUIDE TO THE LITERATURE [top](#)

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