

CONTAGIOUS CAPRINE PLEUROPNEUMONIA

- [Definition](#)
- [Etiology](#)
- [Host Range](#)
- [Geographic Distribution](#)
- [Transmission](#)
- [Incubation Period](#)
- [Clinical Signs](#)
- [Gross Lesions](#)
- [Morbidity and Mortality](#)
- [Diagnosis](#)
- [Field Diagnosis](#)
- [Specimens for the Laboratory](#)
- [Laboratory Diagnosis](#)
- [Differential Diagnosis](#)
- [Treatment](#)
- [Vaccination](#)
- [Control and Eradication](#)
- [Public Health](#)
- [References](#)
- [FAD Table of Contents](#)

Definition [top](#)

Contagious caprine pleuropneumonia (CCPP) is an acute highly contagious disease of goats caused by a mycoplasma and characterized by fever, coughing, severe respiratory distress, and high mortality. The principal lesion at necropsy is fibrinous pleuropneumonia.

Etiology [top](#)

For many years the causative agent of CCPP was considered to be *M. mycoides capri* (type strain PG-3) because this was the agent most commonly isolated from goats with CCPP. In 1976, however, MacOwan and Minette (13) reported isolating a new mycoplasma (designated F-38) from a CCPP outbreak in Kenya and demonstrated it to be the cause of a highly contagious form of pneumonia resembling the original description of CCPP by Hutcheon in 1881. McMartin et al. (11) presented very convincing arguments supporting this agent as the cause of

the classical disease, at least in Africa. Both of these mycoplasmas are now considered to cause CCPP, although the infrequency with which *M. mycoides capri* has been isolated from CCPP in recent years (19) suggests that it may be a minor cause of the disease. Neither of these agents occurs in North America. The name *M. capricolum capripneumoniae* proposed for mycoplasma F-38 by Leach et al. (10) is not in common usage. *Mycoplasma mycoides capri* is easily propagated on standard mycoplasma media, but F-38 is much more fastidious and can easily be missed at diagnosis, which may explain its late recognition as the major cause of CCPP.

M. mycoides mycoides has also been isolated from goats with pneumonia. This agent (the so-called large colony or LC variant of *M. mycoides mycoides*) usually produces septicemia, polyarthritis, mastitis, encephalitis, conjunctivitis, hepatitis, or pneumonia in goats. Some strains of this agent will cause pneumonia closely resembling CCPP (15), but the agent is not highly contagious and is not considered to cause CCPP. It does occur in North America. *M. capricolum capricolum*, a goat pathogen commonly associated with mastitis and polyarthritis in goats, can also produce pneumonia resembling CCPP, but it usually causes severe septicemia and polyarthritis. This agent (which does occur in the United States) is closely related to mycoplasma F-38 but can be differentiated from it using monoclonal antibodies (22).

Host Range [top](#)

Contagious caprine pleuropneumonia is a disease of goats, and where the classical disease has been described, only goats were involved in spite of the presence of sheep and cattle. Mycoplasma F-38, the probable cause of the classic disease, does not cause disease in sheep or cattle.

M. mycoides capri, the other agent considered a cause of CCPP, will result in a fatal disease in experimentally inoculated sheep and can spread from goats to sheep. It is however, not recognized as a cause of natural disease in sheep.

Geographic Distribution [top](#)

Contagious caprine pleuropneumonia has been described in many countries of Africa, the Middle East, Eastern Europe, the former Soviet Union, and the Far East. It is a major scourge in many of the most important goat-producing countries in the world and is considered by many to be the world's most devastating goat disease.

The classical disease, as caused by mycoplasma F-38, has not been described in

North America. The reports of CCPP occurring in the United States (23) and in Mexico (1) were erroneous in that, although similar syndromes were described, the agents isolated were misidentified as *M. mycoides capri* and were subsequently shown to be *M. mycoides mycoides* (LC type). Neither mycoplasma F-38 nor *M. mycoides capri* has been isolated in North America

Transmission [top](#)

Contagious caprine pleuropneumonia is transmitted by direct contact through inhalation of infective aerosols. Of the two known causative agents, F-38 is far more contagious. Outbreaks of the disease often occur after heavy rains (e.g., after the monsoons in India) and after cold spells. This is probably because recovered carrier animals start shedding the mycoplasmas after the stress of sudden climatic change. It is believed that a long-term carrier state may exist.

Incubation Period [top](#)

The incubation period can be as short as 6 to 10 days but may be very prolonged (3-4 weeks) under natural conditions

Clinical Signs [top](#)

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 (e.g., pasteurella) as part of the etiologic picture.

The classical disease as caused by mycoplasma F-38 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 bleat in obvious pain. Frothy nasal discharges and stringy salivation are often seen shortly before death. In the acute disease, which occurs in fully susceptible populations of goats, death occurs within 7 to 10 days of the onset of clinical signs. 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 carriers of the mycoplasmas.

M. mycoides capri tends to cause a more generalized infection in which septicemia is frequently seen. An acute or peracute septicemic form of the disease involving the reproductive, respiratory and alimentary tracts has been described. In addition, thoracic and reproductive formes of the disease have been attributed to

this agent. The disease is considerably less contagious than F-38-induced disease, and the mortality and morbidity rates are also lower.

Gross Lesions [top](#)

The gross lesions in classical CCPP are confined to the thoracic cavity (11). 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 involve both, and an entire lobe may become solidified. The pulmonary pleura becomes thickened, and there may be adhesions to the chest wall. Hutcheon emphasized that the lesions of CCPP do not resemble those of contagious bovine pleuropneumonia (CBPP) in that "no thickening of the interlobular tissue" occurs, a classical lesion of CBPP. He described a CCPP-diseased lung as resembling a "somewhat granular looking liver", which is his description of the massive hepatization seen in CCPP lungs.

In sharp contrast, *M. mycoides capri* has been reported to cause lesions in a wide variety of organ systems 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 a feature 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 hepatization and marked dilation of interlobular septa is commonly seen (Fig. 43). The cardiac and diaphragmatic lobes are the ones most commonly involved. Some describe this as a mild form of CCPP; others argue that it is not CCPP.

Morbidity and Mortality [top](#)

Morbidity can be 100 PERCENT and mortality may be in the range of 70 percent to 100 percent (19). Gathering or increased confinement of animals facilitates the spread of the disease.

Diagnosis [top](#)

Field Diagnosis [top](#)

A highly contagious disease occurring in goats and characterized by severe respiratory distress, high mortality, and postmortem lesions of fibrinous pleuropneumonia with pronounced hepatization and pleural adhesions warrants a field diagnosis of CCPP.

Specimens for the Laboratory [top](#)

From a dead animal that has had severe clinical disease, the best specimens to submit are affected lung, swabs of major bronchi, and tracheobronchial or mediastinal lymph nodes. 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](#)

Diagnosis must be confirmed by isolation of the agent (F-38). The causative agent, once isolated, can be identified by immunofluorescence or by growth or metabolic inhibition tests. Several serological tests can be used in the laboratory for the detection of antibodies to mycoplasma F-38. These include complement fixation (CF), passive hemagglutination (PH), and enzyme-linked immunosorbent assay (ELISA). The latex agglutination test (20) is a very convenient field test for detecting antibodies in whole blood or in serum.

Differential Diagnosis [top](#)

Clinically, CCPP may be confused with other pneumonic conditions such as pasteurellosis and peste des petits ruminants.

Treatment [top](#)

The mycoplasmas are sensitive to several broad-spectrum antibiotics (notably the tetracyclines, tylosin, and tiamulin). Although early treatment can be effective, chemotherapy and chemoprophylaxis have not played important roles in CCPP control programs.

Vaccination [top](#)

A crude vaccine prepared from goat lung was used to vaccinate goats in South Africa after the original outbreak of CCPP in the late 1800's. A combination of this vaccine and other control methods eliminated the disease from the country.

Vaccines to *M. mycoides capri* have been used with little success. This is probably because the disease is usually caused by mycoplasma F-38, first recognized in

1976. Since that time both live attenuated and inactivated F-38 vaccines have been tested with varying degrees of success. The most promising of the experimental vaccines is the lyophilized saponin-inactivated F-38 vaccine shown in field tests to confer 100 percent protection to contact exposure (21). This vaccine could be of inestimable value in many countries of Africa.

Control and Eradication [top](#)

Sufficient regulatory restrictions should be maintained to prevent introduction of CCPP into apparently healthy animals. Serologic testing of susceptible animals for importation is a recommended safeguard.

Successful control of the spread of CCPP rests on removing susceptible animals from any possible contact with CCPP-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](#)

Human infection with these mycoplasmas has not been reported.

GUIDE TO THE LITERATURE [top](#)

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