

Contagious Agalactia

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& Public Health

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Importance

Contagious agalactia is a mycoplasmal disease of sheep and goats that can cause serious economic losses from mastitis, polyarthritis and keratoconjunctivitis. In lambs and kids, losses due to septicemia and pneumonia can be high. Some outbreaks may affect most of the animals on a farm. Although contagious agalactia has been eradicated from some countries, it may be difficult to control where it is widespread. Antibiotics can result in symptomatic improvement, but treated animals may remain carriers.

Etiology

Contagious agalactia is classically caused by infection with *Mycoplasma agalactiae*. *M. capricolum* subsp. *capricolum*, *M. putrefaciens*, *M. mycoides* subsp. *capri* or *M. mycoides* subsp. *mycoides* large colony (LC) type can also cause this disease. (Note: Some genetic evidence suggests that *M. mycoides capri* and *M. mycoides mycoides* LC may be the same organism, and they may be combined into a single taxon in the future.) Whether *M. putrefaciens* alone causes contagious agalactia in healthy animals, or whether it is pathogenic only when combined with other diseases or environmental stressors is uncertain.

Species Affected

Contagious agalactia primarily affects sheep and goats. Although *M. agalactiae* causes disease in both species, *M. capricolum capricolum*, *M. mycoides capri* and *M. mycoides mycoides* LC primarily affect goats and are rarely found in sheep. *M. putrefaciens* has, to date, been reported only in goats.

Other species may also be infected with these organisms. *M. agalactiae* has been isolated from Spanish ibexes (*Capra pyrenaica*), and antibodies to this organism have been reported in roe deer (*Capreolus capreolus*) and red deer (*Cervus elaphus*). Antibodies to *M. mycoides mycoides* LC and *M. capricolum capricolum* have been found in South American camelids. *M. capricolum capricolum* and a unique mycoplasmal strain that was closely related to *M. mycoides mycoides* LC were reported from captive Vaal rhebok (*Pelea capreolus*) from South Africa, and were implicated in a fatal systemic illness. *M. mycoides mycoides* LC has also been reported in cattle.

Geographic Distribution

Contagious agalactia has been reported in much of the world. This disease is particularly common in the Mediterranean region of Europe, Asia and North Africa. The distribution of the causative agents can vary.

Three isolations of *M. agalactiae* have been reported from the United States, but these North American strains did not seem to cause significant disease. *M. putrefaciens* was isolated from goats in California in 1979, and caused a large outbreak in that state in 1987.

Transmission

Infected animals shed organisms in urine, feces, nasal and ocular discharges, and secretions including milk. *Mycoplasma* spp. can be shed during more than one lactation; between lactations, the organisms can survive in the supramammary lymph nodes. Carriers can remain infectious for months, and in some cases, for more than a year. Animals become infected by ingestion or occasionally by inhalation, as well as through the teat openings. Young animals are usually infected when they drink contaminated milk or colostrum from the dam. Animals may also ingest mycoplasmas shed in other secretions and excretions, either directly or in feed or water. Organisms can enter the teat opening directly during milking, or from fomites such as bedding. Aerosol transmission is possible over short distances.

The organisms that cause contagious agalactia can be transmitted on fomites. Although mycoplasmas are relatively fragile in the environment, most of the organisms that cause this disease can produce biofilms, which protect them from heat and drying, and enhance their survival. In one study, biofilm production was

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particularly abundant for *M. putrefaciens* and *M. agalactiae*, while *M. capricolum capricolum* was unable to produce a significant biofilm. Survival appears to be particularly likely if the temperature is low.

Incubation Period

The incubation period is one to eight weeks.

Clinical Signs

Generally, the clinical signs are more severe in goats. Infections with *M. agalactiae* may be asymptomatic, acute or chronic. Acute cases begin with a transient fever followed by malaise, inappetence and mastitis. The udder is hot and swollen, and the milk is usually greenish–yellow or grayish–blue, with a consistency that is watery at first then becomes lumpy. Lactation diminishes and may completely stop. Eventually, the udder atrophies and becomes fibrosed. Polyarthritis is also common, especially in the tarsal and carpal joints, and may be the major clinical sign in male goats. The severity of the arthritis varies; some animals are merely stiff while others become severely lame and may be unable to stand or walk. Keratoconjunctivitis develops in approximately half of all infections. It is usually transient, but occasionally becomes chronic, and can cause blindness in one or both eyes. Pneumonia is not consistently seen with *M. agalactiae*, but organisms are occasionally isolated from lesions in the lungs. Abortions can occur in chronically infected animals. Granular vulvovaginitis has also been reported in goats.

M. mycoides mycoides LC, *M. mycoides capri*, *M. capricolum capricolum* and *M. putrefaciens* cause similar clinical signs, but are reported mainly in goats. Most infections with these organisms are acute or hyperacute. Some infected goats, especially nursing kids, develop pneumonia and/or septicemia with fever, prostration, anorexia and generalized malaise. The mortality rate in these animals may be high, and other clinical signs may not be seen. Sudden death has also been reported in some infected herds. Other goats develop mastitis, arthritis and keratoconjunctivitis, although ocular signs have not yet been documented with *M. putrefaciens*. Pregnant animals may abort. *M. putrefaciens* has been linked to genital lesions in both sexes.

Infections with *M. mycoides mycoides* LC and *M. capricolum capricolum* are reported infrequently in sheep. *M. mycoides mycoides* LC has been found in sheep with vulvovaginitis and balanoposthitis. *M. capricolum capricolum* has also been linked to genital lesions. *M. putrefaciens* has been not documented in this species.

Post Mortem Lesions

At necropsy, the lesions may include signs of septicemia, pneumonia, mastitis, arthritis and/or ocular disease. Catarrhal mastitis with primary inflammation of the interstitial tissues is often seen in females. In the later stages, there may be secondary acinar involvement, fibrosis

and/ or parenchymatous atrophy. Arthritis with periarticular edema may be noted, particularly in the carpal joints. The synovial membranes may be hyperemic, and the joint fluid can be hemorrhagic or turbid. Serous or mucopurulent conjunctivitis, keratitis or, less frequently, corneal ulceration may also be present.

Some organisms have also been associated with genital lesions including vulvovaginitis, cystic catarrhal metritis and/or salpingitis in females, and balanoposthitis or testicular degeneration in males.

Morbidity and Mortality

Contagious agalactia is mainly reported in milking sheep and goats. Animals are particularly susceptible at the beginning of lactation. When an organism is first introduced into a susceptible flock or herd, the morbidity rate is approximately 30-60%; in some cases, most of the susceptible animals become ill. The mortality rate varies and is often 20% or less. However, it may initially reach 40-70%, especially in young animals with septicemia. *M. putrefaciens* may be less virulent than *M. mycoides mycoides* LC or *M. capricolum capricolum*, but severe, highly fatal disease has been reported when *M. putrefaciens* and *M. agalactiae* occurred together in goats. In one outbreak caused by concurrent infection with these two organisms, 80% of the herd died or was euthanized due to severe debilitation. Once an organism has become established in the herd, infections tend to be subacute or chronic.

Diagnosis

Clinical

Contagious agalactia should be suspected in a flock or herd with mastitis and decreased milk production, keratoconjunctivitis and arthritis, particularly when these signs develop near the time of parturition. Acute septicemia can also be seen, especially in young animals, and is more difficult to diagnose.

Differential diagnosis

Contagious agalactia must be distinguished from other causes of pneumonia, mastitis and/or arthritis including *Manheimia haemolytica*, *Streptococcus* spp., *Staphylococcus* spp., *Escherichia coli*, *Klebsiella pneumoniae*, caprine arthritis encephalitis virus, *Erysipelothrix rhusiopathiae* and other organisms.

Laboratory tests

Contagious agalactia can be diagnosed by isolating the causative organism. The *Mycoplasma* species that cause contagious agalactia can grow on most mycoplasma media, producing colonies within 3-4 days. Typical fried egg colonies are seen on solid media. Isolates can be identified with biochemical tests and serology. Serological tests used for identification include the growth inhibition (GIT), film inhibition (FIT), indirect fluorescent antibody (IFA) and dot immunobinding tests. Polymerase chain reaction (PCR)

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assays are increasingly used to identify isolates. Clone purification of colonies is often necessary before an isolate can be identified by GIT or FIT; definitive diagnosis using these tests takes at least two weeks. PCR, IFA and the dot immunobinding test do not require cloning, and can be done rapidly.

PCR techniques can identify organisms directly in clinical samples including nasal, conjunctival and synovial exudates, milk, and tissue samples. Positive PCR results should be confirmed by isolation and identification of the organism, especially in areas where contagious agalactia is not usually found. Negative PCR results are not considered definitive.

Serology can be useful as a herd test. Serological tests have been developed for most of the organisms that cause contagious agalactia, but assays for *M. putrefaciens* are not widely available. Complement fixation and enzyme-linked immunosorbent assays (ELISA) are the most commonly used tests. Commercial ELISAs have been developed for *M. agalactiae*, and ELISAs for other causative organisms may be available in individual laboratories. Immunoblotting and indirect hemagglutination may also be used. In areas that are free of contagious agalactia, a serological diagnosis should be confirmed by isolation of the organism.

Samples to collect

Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease.

In live animals, the preferred samples for culture are nasal swabs and exudates, aspirated joint fluid from cases of arthritis, eye swabs from cases with ocular disease, and milk. Milk should be collected from animals with mastitis, or from healthy animals when clinical signs are common in young, nursing animals. Ear swabs may also contain organisms, but the concurrent presence of other *Mycoplasma* species can make isolation difficult. *Mycoplasmas* can be isolated from blood samples during acute disease. PCR can detect organisms in nasal, conjunctival and synovial exudates, milk, and tissue samples.

The optimal samples at necropsy are the udder and its associated lymph nodes, joint fluid, pleural or pericardial fluid, and samples from lung lesions taken at the interface between diseased and healthy tissue. Some authorities also recommend blood, urine, liver, spleen and other organs. Samples for culture should be collected aseptically and placed in transport medium (e.g., heart infusion broth with 20% serum, 10% yeast extract and benzylpenicillin). Samples should be kept cool and transported promptly to the laboratory on wet ice. If samples must be held for more than a few days, they may be frozen.

For serology, serum should be collected from both clinically affected and asymptomatic animals. At least 10

serum samples, preferably from acute and convalescent cases, are collected for complement fixation.

Recommended actions if contagious agalactia is suspected

Notification of authorities

Contagious agalactia is reportable to the World Organization for Animal Health (OIE). Disease notification requirements for OIE member nations and import/export guidelines can be found in the OIE Terrestrial Animal Health Code [<http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/>]. Veterinarians who encounter a case of contagious agalactia should follow their national and/or local guidelines for disease reporting and diagnostic testing.

Notification of authorities in the United States

Contagious agalactia should be reported immediately to state or federal authorities.

Federal: Area Veterinarians in Charge (AVIC):

www.aphis.usda.gov/animal_health/area_offices/

State Veterinarians:

www.usaha.org/Portals/6/StateAnimalHealthOfficials.pdf

Control

In areas that are free of contagious agalactia, infected herds are usually quarantined and euthanized. The premises should be cleaned and disinfected before restocking. Mycoplasmas can be inactivated by many disinfectants including sodium hypochlorite (30 ml of household bleach in 1 gallon of water), 2% sodium hydroxide (pH 12.4), 1% formalin, cresol, sodium carbonate (4% anhydrous or 10% crystalline with 1% detergent), and ionic and nonionic detergents. Eradication has been accomplished in some areas by the euthanasia of infected and exposed flocks or herds.

In endemic areas, good management and hygiene can reduce the transmission of contagious agalactia in a herd. The premises and equipment should be cleaned and disinfected regularly, and sick animals should be isolated. Milking animals should also be separated from young animals. Removal of the newborn from the dam, with feeding of pasteurized colostrum and milk, can be helpful. Regular testing of the flock or herd, with culling or isolation of infected animals, can also help prevent disease introduction and/or reduce its spread. ELISAs, culture and other tests have been useful in screening programs.

Antibiotics can result in symptomatic improvement, but they may not be effective in chronic joint infections or keratoconjunctivitis. Treatment may not eliminate the infection from carriers. Vaccines may be available for some organisms in some areas. Inactivated vaccines generally provide short-term protection. Live vaccines can prevent symptoms, but do not prevent animals from becoming infected or shedding the organism. Vaccine organisms may also be shed in the milk.

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Public Health

There is no evidence that *M. agalactiae* is a threat to human health.

Internet Resources

The Merck Veterinary Manual

<http://www.merckvetmanual.com/mvm/index.jsp>

United States Animal Health Association. Foreign Animal Diseases

http://www.aphis.usda.gov/emergency_response/downloads/nahems/fad.pdf

World Organization for Animal Health (OIE)

<http://www.oie.int>

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

<http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/>

OIE Terrestrial Animal Health Code

<http://www.oie.int/international-standard-setting/terrestrial-code/access-online/>

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*Link defunct as of 2012