

Contagious Agalactia

Caprine Mycoplasmosis

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Importance

Contagious agalactia is a mycoplasmal disease of sheep and goats that can cause serious economic losses from mastitis, arthritis and keratoconjunctivitis. Septicemia and pneumonia also occur during some outbreaks, most often in nursing young animals. Four different organisms, *Mycoplasma agalactiae*, *M. capricolum* subsp. *capricolum*, *M. mycoides* subsp. *capri* and *M. putrefaciens*, can cause contagious agalactia. The latter three are mainly seen in goats. All of these agents are difficult to eliminate once they become established in a herd or flock. Antibiotics are helpful in treating clinical signs, but chronic illnesses may not respond well and treated animals can remain carriers. Although some infected herds have few or no clinical cases for prolonged periods, stressors, decreased immunity or the introduction of susceptible animals into the herd can result in new outbreaks.

Etiology

At one time, contagious agalactia was narrowly defined as the condition caused by *Mycoplasma agalactiae*, but *M. capricolum* subsp. *capricolum*, *M. mycoides* subsp. *capri* and *M. putrefaciens* are now also considered to be causative agents. The organism formerly known as *M. mycoides* subsp. *mycoides* large colony (LC) type has been incorporated into *M. mycoides* subsp. *capri*. More than one agent can be found in some clinical cases.

Species Affected

Contagious agalactia primarily affects sheep and goats. *M. agalactiae* causes disease in both species, but *M. putrefaciens* only seems to affect goats, and *M. capricolum capricolum* and *M. mycoides capri* mostly occur in this species. *M. agalactiae*, *M. capricolum capricolum* and *M. mycoides capri* have been described a few times in symptomatic or asymptomatic cattle. Some South American camelids have antibodies to *M. mycoides capri* and *M. capricolum capricolum*, although there is currently no virological confirmation that they can be infected.

Among wild species, *M. agalactiae* is known to affect Iberian (or Spanish) ibex (*Capra pyrenaica*), Alpine ibex (*Capra ibex ibex*), and chamois (*Rupicapra rupicapra*). It may be maintained in some wild populations of ibex: one outbreak in Alpine ibex and chamois was caused by an unusual and distinct strain of this organism that has not been reported in domesticated sheep or goats. Antibodies to *M. agalactiae* have also been detected in roe deer (*Capreolus capreolus*) and red deer (*Cervus elaphus*). *M. mycoides capri* is known to affect wild Alpine ibex and captive Vaal rhebok (*Pelea capreolus*). *M. capricolum capricolum* can also affect captive Vaal rhebok, was detected in captive Dall's sheep (*Ovis dalli dalli*) and appeared to be responsible for an outbreak in wild markhor (*Capra falconeri*).

Zoonotic potential

In 2014, *M. capricolum capricolum* was isolated from a person with recurrent fever, signs of septicemia and suspected meningitis. As of 2018, this is the only report of a human infection caused by any of the four agents.

Geographic Distribution

Contagious agalactia has been reported in much of the world, although underdiagnosis and underreporting seem to be significant issues. This disease is particularly prevalent in the Middle East and southern Europe, where goats are common. A number of cases have also been documented in parts of Asia, such as Mongolia. Fewer outbreaks seem to occur in the Americas, and there are only sporadic reports of contagious agalactia in the U.S. The distribution of the four causative agents varies between regions.

Transmission

The organisms that cause contagious agalactia are shed in nasal and ocular discharges and milk. They can also occur in other secretions and excretions including urine, feces and semen, and may be present in the external ear canal. Shedding in semen



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can be intermittent. Asymptomatic animals may carry these agents for months to years, and females can shed organisms in milk during more than one lactation. Animals usually become infected by ingestion or inhalation or through the teat opening. Young lambs and kids typically acquire these organisms from their dams when they nurse; both milk and colostrum are infectious. Aerosol transmission can occur over short distances, especially when animals have respiratory signs, and the presence of agents in semen suggests the possibility of venereal transmission.

The agents that cause contagious agalactia can be transmitted on fomites such as feed, drinking water and milking equipment. Ear mites might transmit organisms in the ear canal from animal to animal. Although mycoplasmas are relatively fragile in the environment, they may produce protective biofilms that can enhance their survival. In one study, biofilm production was particularly abundant for *M. putrefaciens* and *M. agalactiae*, while *M. capricolum capricolum* was unable to produce a significant biofilm. Survival in the environment is more likely if the temperature is low.

Disinfection and Inactivation

Mycoplasmas can be inactivated by many disinfectants including 1% sodium hypochlorite, 70% ethanol, iodophors, phenolic disinfectants, peracetic acid, 2% sodium hydroxide (pH 12.4), formaldehyde, glutaraldehyde, and ionic and nonionic detergents. UV irradiation or moist heat of 121°C (250°F) for 20 minutes (autoclaving) are also effective.

Pasteurization of colostrum at 56°C (133°F) or 60°C (140°F) for 60-120 minutes significantly reduced the number of viable *M. agalactiae* and *M. mycoides capri* in one study. *M. mycoides capri* was eliminated after 1 hour at 60°C, but *M. agalactiae* could still be found after 2 hours. However, the number of organisms in treated colostrum seemed to be less than the infective dose for oral transmission.

Incubation Period

The incubation period is 1-8 weeks.

Clinical Signs

Sheep and goats

Contagious agalactia can be an acute or chronic illness in sheep and goats. Mastitis, arthritis and keratoconjunctivitis are the major clinical syndromes, but all three are not necessarily present in an individual animal. Initially, there may be a fever and nonspecific signs of illness; however, this stage may be mild and it can be overlooked. Clinically apparent mastitis develops soon afterward in lactating females. The udder is hot and swollen, and the milk is usually discolored, often with a yellowish tinge. It may also be watery, granular or clotted. The milk of animals affected by *M. putrefaciens* is reported to have a rotted smell. Lactation diminishes and may completely stop. Permanent damage to the udder, with atrophy and fibrosis, is common. Arthritis or polyarthritis occurs most often in the

tarsal and carpal joints, and can become chronic. Some animals are merely stiff; others become severely lame and may be unable to stand or walk.

Keratoconjunctivitis can be seen in some animals infected with *M. agalactiae*, *M. capricolum capricolum* and *M. mycoides capri*, although it has not yet been reported with *M. putrefaciens*. While ocular signs usually last only a short time, chronic cases are possible, and animals occasionally become blind in one or both eyes. Some animals with contagious agalactia also have diarrhea or respiratory signs ranging from coughing to dyspnea. Septicemia may be seen, most often in nursing lambs and kids. Neurological signs including meningitis have been reported occasionally. Pregnant animals sometimes abort, and congenital polyarthritis was reported in one outbreak caused by *M. agalactiae*. There are sporadic reports of genital lesions such as vulvovaginitis, salpingitis, metritis, balanoposthitis and testicular degeneration. All four organisms can also be carried asymptotically in small ruminants.

Other species

M. agalactiae has been detected infrequently in cattle, either alone or concurrently with cattle pathogens, but whether it causes clinical signs is still unclear. *M. mycoides capri* caused an outbreak of arthritis and septicemia in calves fed unpasteurized goat milk, although some earlier studies had found no clinical signs in experimentally infected calves. It has also been found in a few aborted fetuses. *M. capricolum capricolum* has been detected in dairy cattle with mastitis, and it was implicated in a severe outbreak of arthritis and respiratory disease in neonatal calves. Arthritis and chronic mastitis were seen in some cows during this outbreak, but no causative agent could be identified in these animals. Experimental inoculation of *M. capricolum capricolum* into calves immunosuppressed by African animal trypanosomiasis resulted in arthritis and pneumonia.

Clinical cases in wild relatives of sheep and goats often resemble contagious agalactia in small ruminants, with mastitis, arthritis and/ or keratoconjunctivitis. However, some outbreaks have been severe and killed significant numbers of adult animals. Severe pneumonia and elevated mortality occurred during an outbreak caused by *M. agalactiae* in Alpine ibex and chamois, as well as in an outbreak caused by *M. capricolum capricolum* in markhor. Severe systemic illnesses in captive adult Vaal rhebok, caused by *M. capricolum capricolum* in 2 animals and *M. mycoides capri* in one, were characterized by pulmonary dysfunction, arthritis, diarrhea, neurological signs (ataxia, head tremor) and sepsis. All three rhebok died or were euthanized.

Post Mortem Lesions

Catarrhal mastitis with primary inflammation of the interstitial tissues and enlargement of the mammary lymph nodes is common in females. In the later stages, there may be secondary acinar involvement, fibrosis and/ or parenchymatous atrophy of the udder. Periarticular edema

may be found around the affected joints in animals with arthritis, and the joint fluid can be hemorrhagic or turbid, but the cartilage is usually unaffected. Serous or mucopurulent conjunctivitis or keratitis may also be found. Corneal ulceration occurs occasionally. Evidence of septicemia and/or bronchopneumonia is sometimes apparent, especially in young animals. Generalized peritonitis may be found in animals that die during the acute stage. There are occasional reports of genital lesions including vulvovaginitis, cystic catarrhal metritis and/or salpingitis in females and balanoposthitis or testicular degeneration in males.

Diagnostic Tests

In clinical cases, the causative agent may be found in nasal swabs and exudates, aspirated joint fluid, conjunctival swabs, milk, ear swabs, and internal organs at necropsy. In addition to collecting milk samples from animals with mastitis, samples should be taken from healthy dams when nursing young animals are affected. Organisms can occasionally be detected in blood if the illness is acute. 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 collecting blood, urine, liver and spleen from the carcass if the case was severe. Asymptomatic carriers may be identified by sampling ear swabs, nasal and ocular secretions, milk and semen. Ear swabs seem to be useful for detecting carrier billy goats; however, one study found that asymptomatic rams were more likely to be found by sampling nasal swabs and semen samples.

The organisms that cause contagious agalactia can be isolated on most mycoplasma media, although *M. agalactiae* is reported to grow better on media with organic acids such as pyruvate and isopropanol (e.g., PRM medium). Colonies can be identified to the species level with biochemical tests, genetic tests such as PCR, and serological tests such as growth and metabolic inhibition, film inhibition, indirect fluorescent antibody (IFA) and dot immunobinding. PCR tests can also detect organisms directly in clinical samples. Combining PCR and culture is recommended for optimal sensitivity and specificity. A loop-mediated isothermal amplification (LAMP) test for *M. agalactiae* has also been published. Immunohistochemical staining of tissues may be employed occasionally.

Serology can be useful as a herd test, but false negative results are relatively common, especially in chronically affected animals, asymptomatic carriers and chronically infected herds. Cross-reactivity with other mycoplasmas can also be an issue. Test availability varies with the organism, and assays for *M. putrefaciens* are not widely available. Complement fixation and ELISAs are the most commonly used serological tests. Combining ELISAs with immunoblotting is reported to improve sensitivity. In areas free of contagious agalactia, a serological diagnosis should be confirmed by isolation of the organism.

Treatment

Antibiotics can result in clinical improvement, but they may not be effective in some chronic cases. Fluoroquinolones, tetracyclines and macrolides are the most commonly used agents. Susceptibility to individual drugs may differ between organisms. Treatment may not eliminate the infection from carriers.

Control

Disease reporting

Veterinarians who encounter or suspect contagious agalactia should follow their national and/or local guidelines for disease reporting. Although this disease occurs in the U.S., all four organisms are federally reportable. State or federal reportable disease lists should be consulted for details.

Prevention

Control methods in different countries range from culling of affected herds to antibiotic treatment and vaccination. Contagious agalactia is often introduced in an infected animal, and keeping a closed herd is helpful. Infected herds may be quarantined to prevent them from spreading the agents. Control programs should be established for artificial insemination centers, with screening of animals on entry and periodic testing of semen by culture and PCR. Organisms can be shed in semen without obvious effects on semen quality.

Once they become established in a herd, the mycoplasmas that cause contagious agalactia are difficult to eliminate. Regular herd tests, with culling or isolation of infected animals, can be helpful, and good management and hygiene may reduce transmission within the herd. The premises and equipment should be cleaned and disinfected regularly, and sick animals should be isolated. Cleanliness and infection control measures are especially important during milking. Separating young animals from milking animals reduces their exposure. If it is feasible, newborns may be separated from the dam and fed pasteurized colostrum and milk. Commercial or autogenous vaccines for *M. agalactiae*, *M. capricolum capricolum* and/or *M. mycoides capri* are available in some countries. Inactivated vaccines generally provide only short-term protection, and some studies report that their efficacy may be poor. Live vaccines may be more effective, but are not permitted in many areas.

In regions that are free of contagious agalactia, infected herds are usually quarantined and euthanized. The premises should be cleaned and disinfected before restocking.

Morbidity and Mortality

Contagious agalactia is typically most severe in young animals and lactating females, although other animals, including adult males, are also affected. The prevalence of clinical cases usually peaks at the beginning of lactation and again when the young are removed and machine milking is initiated. Clinical cases are reported to be more severe in goats.

The severity of the illness can also be influenced by the specific organism and its strain, and the breed of animal.

The initial exposure of a flock or herd to these organisms often results in an outbreak, with clinical signs commonly affecting 30-60% of the animals. Once the agent has become established in the herd, infections tend to be subacute or chronic, and clinical cases may be infrequent. There can be periods when the disease seems to be absent. However, new outbreaks may follow stressors (e.g., transport), periods of reduced immunity or the introduction of susceptible herd replacements. The mortality rate is often 10-30% in lactating females affected by *M. agalactiae*, but it can be as high as 50% in cases caused by some other organisms. Particularly high case fatality rates may be seen in nursing lambs and kids.

Relatively little is known about contagious agalactia in animals other than sheep and goats. While clinical cases seem to be uncommon in cattle, a few outbreaks in young calves had high case fatality rates. Reports of contagious agalactia in wild ruminants range from asymptomatic carriage to severe illnesses. An outbreak of pneumonia in markhorses in Tajikistan, thought to be caused by *M. capricolum capricolum*, killed an estimated 20% of the population. Some organisms are thought to be acquired from nearby domesticated small ruminants, but a distinct strain of *M. agalactiae* seems to be maintained in wild Alpine ibex in France, and may be unusually virulent for these animals. Three clinical cases in Vaal rhebok, caused by *M. capricolum capricolum* or *M. mycoides capri*, were also fatal or resulted in euthanasia. These infections were apparently acquired from other animals at the zoo.

Internet Resources

[The Merck Veterinary Manual](#)

[United States Animal Health Association.
Foreign Animal Diseases](#)

[World Organization for Animal Health \(WOAH\)](#)

[WOAH Manual of Diagnostic Tests and Vaccines for
Terrestrial Animals](#)

[WOAH Terrestrial Animal Health Code](#)

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