Avian Mycoplasmosis (*Mycoplasma gallisepticum*)

Pleuropneumonia–like Organism (PPLO) Infection, Chronic Respiratory Disease of Chickens, Infectious Sinusitis of Turkeys, House Finch Conjunctivitis

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Importance

Mycoplasma gallisepticum is an economically significant pathogen that can cause significant losses in chickens, turkeys and game birds from chronic respiratory disease, reduced feed efficiency, decreased growth and lower egg production. In addition, the carcasses of birds sent to slaughter may be downgraded. Many countries with modern poultry operations have eradicated this organism from commercial chicken and turkey breeding flocks; however, it can still be an issue in other poultry operations, such as multi-age layer flocks, game bird raising facilities and backyard birds. Since 1994, conjunctivitis caused by one lineage of *M. gallisepticum* has become a significant disease in wild birds in North America. Although other wild birds can be affected, the major impact has been on house finches (*Carpodacus mexicanus*), which have experienced major population declines in some areas.

Etiology

Mycoplasma gallisepticum, a member of the family Mycoplasmataceae (class Mollicutes, order Mycoplasmatales), is one of the most important agents of mycoplasmosis in terrestrial poultry. There are multiple strains of this organism, which can differ in virulence and may also have different host preferences. The house finch lineage is a distinct lineage that has diverged significantly from poultry strains and has become established in wild birds.

Some other mycoplasmas pathogenic in poultry include *M. synoviae*, *M. meleagridis* and *M. iowae*. The diseases they cause are also termed avian mycoplasmosis, but the clinical syndromes can differ.

Species Affected

M. gallisepticum affects chickens, turkeys and various game birds such as ring-necked pheasants (*Phasianus colchicus*), chukar partridges (*Alectoris graeca*), red-legged partridges (*Alectoris rufa*), grey partridges (*Perdix perdix*), bobwhite quail (*Colinus virginianus*), Japanese quail (*Coturnix japonica*) and peafowl (*Pavo cristatus*).This organism has also been detected in ducks and geese, although it does not seem to be a significant pathogen in waterfowl. It has occasionally been found in pet or hobby birds, such as symptomatic yellow-naped Amazon parrots (*Amazona auropalliata*) and asymptomatic pigeons (*Columba livia*).

M. gallisepticum can also infect diverse species of wild birds ranging from passerine birds (order Passeriformes) to flamingos (*Phoenicopterus roseus*) and raptors. Most surveys do not identify this organism to the strain level; however, one lineage has been maintained in house finches (*Carpodacus mexicanus*) in North America since the 1990s. This lineage is also known to occur, sometimes with clinical signs, in American goldfinches (*Spinus tristis*), lesser goldfinches (*Spinus psaltria*), purple finches (*Haemorhous purpureus*), pine grosbeaks (*Pinicola enucleator*), evening grosbeaks (*Coccothraustes vespertinus*) and western scrub-jays (*Aphelocoma californica*). Some of these birds, such as goldfinches, may be additional reservoir hosts. The house finch lineage is likely to be the strain in most other passerine birds with virological evidence of infection with *M. gallisepticum* in North America, such as northern cardinals (*Cardinalis cardinalis*), eastern tufted titmice (*Baeolophus bicolor*), black-capped chickadees (*Poecile atricapillus*), downy woodpeckers (*Picoides pubescens*), cedar waxwings (*Bombycilla garrulus*), common yellowthroat (*Geothlypis trichas*), dark-eyed juncos (*Junco hyemalis*), red-winged blackbirds (*Agelaius phoeniceus*), various corvids and members of several genera of sparrows. However, poultry-associated strains have also been detected sporadically in wild birds, including some passerine birds and corvids. Some authors have speculated that additional unrecognized lineages might circulate subclinically among wild birds in various parts of the world.

Zoonotic potential

There is no evidence that *Mycoplasma gallisepticum* is zoonotic.

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Geographic Distribution

M. gallisepticum can be found worldwide.in poultry The *M. gallisepticum* lineage maintained in finches was first reported in the eastern US in the mid-1990s. Since that time, it has since spread to much of the U.S. and parts of Canada. *M. gallisepticum* infections have also been reported in wild birds in other regions, such as Europe and Asia. However, there is currently no evidence that the house finch lineage occurs outside North America.

Transmission

M. gallisepticum occurs in respiratory and ocular secretions, eggs and semen. It may also be found at other sites such as the cloaca. This organism can enter the body orally and via the respiratory tract or conjunctiva. Aerosol spread occurs over short distances and can be responsible for transmission within a flock. Birds are generally thought to become infected during close contact, and this organism may not always spread across a wall to adjacent pens. However, it has been suggested that windborne transmission between flocks might sometimes be possible. Venereal transmission has also been proposed, although it is not expected to be a major route. Poultry can become infected with *M. gallisepticum* in the egg, and debris from broken eggs may be a source of the organism for other birds. Egg-borne transmission is more frequent in birds infected during laying than in birds infected before they mature. Long-term asymptomatic carriage has been reported in poultry, some game birds and house finches. Subclinically infected birds may later develop clinical signs when they are stressed.

M. gallisepticum can be transmitted on fomites, and it may remain viable in the environment for several days. Survival was reported to be longer on certain substrates, such as feathers and the contents of eggs. This organism was reported to persist on human skin for a day or two, and on bird feeders for one day. *M. gallisepticum* can form biofilms, which are thought to enhance the environmental survival of mycoplasmas. The extent of biofilm production can differ between strains.

Disinfection

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

Incubation Period

Experimentally infected poultry developed clinical signs in 6-21 days, while incubation periods from 4 to 14 days have been reported in finches. In natural infections, the incubation period is variable, as infected birds may remain asymptomatic until stressed.

Clinical Signs

M. gallisepticum can be carried subclinically in some poultry, while others develop mild to severe respiratory signs. The clinical signs tend to develop gradually, and the course of the disease can be prolonged. However, acute cases can sometimes be seen in young birds, particularly turkeys. Common clinical signs include rales, coughing, sneezing, nasal discharges, dyspnea, and conjunctivitis with a frothy ocular exudate. Conjunctivitis occurs more frequently in turkeys and game birds, and the respiratory signs are also usually more severe in these species. Turkeys and game birds may develop sinusitis, with swelling of the paranasal (infraorbital) sinus. Game birds sometimes die when purulent material in the nasal cavity and mouth prevents them from eating. Avian mycoplasmosis can also result in decreased egg production, and abnormalities (e.g., unusual paleness) may be apparent in some eggs. Embryo mortality may increase. Infrequently reported syndromes include meningoencephalitis in turkeys, and lameness with swelling of the hock in chickens. The neurological signs in outbreaks of meningoencephalitis have varied (e.g., ataxia, hyperesthesia, opisthotonos, tremor, paralysis, somnolence), but torticollis was often prominent.

Conjunctivitis, sometimes accompanied by rhinitis and sinusitis, is the most prominent sign in house finches. Both eyes are usually affected, although the signs are often more severe on one side. The clinical signs in this species may take weeks or months to resolve. Conjunctivitis has also been reported in other passerine birds, including naturally infected American goldfinches, western scrub-jays, pine grosbeaks and evening grosbeaks, and experimentally infected purple finches, tufted titmice and canaries (*Serinus canaria domestica*); however, it is usually less severe and the birds recover more rapidly. Infected wild rooks (*Corvus frugilegus*) from a gamebird facility in Scotland had pericarditis and pneumonia, but a causative role was uncertain, and some other infected corvids have been healthy. Many infections documented in wild birds have not been associated with clinical signs.

Psittacine birds might also be affected. *M. gallisepticum*, *M. iowae* and an unidentified mycoplasma were isolated from a flock of yellow-naped Amazon parrots with upper respiratory signs. The condition appeared to be caused by concomitant infections with mycoplasmas and bacteria, and the precise role of *M. gallisepticum* was not established. Experimental infection with a poultry strain caused severe conjunctivitis in budgerigars (*Melopsittacus undualtus*), although they remained asymptomatic after inoculation with a house finch strain.

Post Mortem Lesions

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The lesions in poultry and game birds may include tracheitis, airsacculitis, pneumonia, salpingitis and mucoid to mucopurulent sinusitis affecting one or both infraorbital sinuses. The lesions are typically more severe in birds co-infected with other pathogens. Gross lesions in the respiratory tract range from very mild changes that are

difficult to detect; to edema of the walls of the air sac, with excess mucus or catarrhal exudates in the nasal passages, trachea and lungs; to caseous exudates. Air sacculitis is not usually prominent in pheasants. Young chickens with infections complicated by *E. coli* may have additional lesions, such as fibrinopurulent pericarditis and perihepatitis. No gross lesions were found in the central nervous system (CNS) of turkeys with encephalitis, although some birds had respiratory lesions. Tenosynovitis and arthritis have been seen rarely in chickens. Finches typically exhibit mild to severe conjunctivitis, which may be accompanied by rhinitis and sinusitis.

Diagnostic Tests

Illnesses caused by *M. gallisepticum* can be diagnosed by isolating the organism from clinical samples or detecting nucleic acids by PCR, and by serology on the flock. Loop-mediated isothermal amplification assays have also been published. *M. gallisepticum* can be recovered in mycoplasma–free chicken embryos or chickens; however, this technique is rarely used since the advent of PCR.

Common sampling sites for culture and PCR include the choanal cleft, oropharynx, conjunctiva, infraorbital sinus, nasal cavity, esophagus, trachea, air sacs and lungs. Organisms may also be found in swabs from the cloaca and phallus, and in embryonated eggs, dead in-shell embryos, and chicks or poults that have broken the shell but failed to hatch. Postmortem samples should be collected from recently dead animals or carcasses frozen soon after death. *M. gallisepticum* can be isolated on various mycoplasma medium, such as Frey's medium. It can sometimes be difficult to recover. Indirect immunofluorescence, immunoperoxidase staining, a growth inhibition test, metabolism inhibition, and PCR or other DNA methods are used to identify cultured organisms. Biochemical tests may be useful in preliminary identification. *M. gallisepticum* can be difficult to distinguish from *M. imitans*, a species that is more commonly found in ducks and geese. If *M. imitans* is a possibility, these two species can be distinguished with tests such as PCR/ restriction fragment length polymorphism (PCR-RFLP) or immunofluorescence using serial dilutions of antisera to the two organisms in parallel.

Histology has been recommended for the diagnosis of *M. gallisepticum* meningoencephalitis in turkeys, together with the identification of organisms in the flock. The microscopic lesions in this disease (multifocal parenchymal necrosis, meningitis, perivascular cuffing and vasculitis) are reported to be consistent and distinctive.

Serology is particularly helpful in screening poultry flocks. It is less useful in individual birds, as nonspecific reactions are common in some tests. Commonly used assays include a rapid serum agglutination (RSA) test, ELISAs and hemagglutination inhibition. The hemagglutination inhibition test is more specific than RSA, which can have false positives, but it is strain specific and less sensitive. Other serological tests have also been used or described.

Treatment

Antibiotics are used to treat poultry with clinical signs, although they may not eliminate the organism from the flock. Some drugs reported to be effective against *M. gallisepticum* include tetracyclines, macrolides (e.g., tylosin, tylvalosin), aminoglycosides, fluoroquinolones and tiamulin.

Control

Disease reporting

Veterinarians who encounter or suspect a *M. gallisepticum* infection should follow their national and/or local guidelines for disease reporting. In the U.S., state reportable disease lists should be consulted for the requirements.

Prevention

Chicken and turkey flocks should be started with chicks, poults or eggs from *M. gallisepticum*–free breeding flocks, and direct or indirect contact with potential sources of this organism, such as backyard poultry and pet birds, should be avoided. Similar measures may be employed in game bird flocks; however, finding *M. gallisepticum*-free breeding stock is more difficult, and game birds released into the wild are likely to become infected. Commercial poultry flocks should be monitored regularly to detect the organism if it is introduced. Infections can be eliminated from a farm by depopulation, followed by thorough cleaning and disinfection of the premises. *M. gallisepticum*-free breeding stock can be obtained by heat or antibiotic treatment of eggs before incubation, combined with screening of the hatched birds. Excellent biosecurity is needed to prevent its reintroduction into these flocks.

When maintaining *M. gallisepticum*-free poultry flocks is impractical, live and/or killed vaccines can help prevent clinical signs. However, some countries have restrictions on vaccine use, and the currently available live vaccines are not generally employed in turkeys. Good hygiene and management, including measures to control co-infections, are also important in minimizing the clinical impact of *M. gallisepticum* infections.

Routine infection control procedures, including good sanitation and disinfection, reduce the risk of transmitting *M. gallisepticum* between birds in wild bird rehabilitation facilities. Regular cleaning and disinfection has also been recommended for backyard bird feeders, as it may reduce the spread of this organism between wild finches. However, one study suggests that *M. gallisepticum* may not survive for more than 24 hours on a feeder.

Morbidity and Mortality

Many countries with modern poultry industries have eliminated *M. gallisepticum* from their primary chicken and turkey breeding flocks. Infected table egg layers and broilers are more common, especially among flocks under continuous production, and game bird flocks are frequently infected. It may be particularly difficult to maintain *M.*

gallisepticum-free flocks in areas with a high concentration of commercial poultry.

The consequences of infection range from asymptomatic carriage to serious illnesses. The clinical signs are usually more severe when the birds are infected concurrently with other pathogens, such as Newcastle disease virus, infectious bronchitis virus or *Escherichia coli*. Their severity is also influenced by management factors such as cold weather, poor nutrition and crowding, as well as the strain of the organism. The morbidity rate is usually high and the mortality rate low in chickens with uncomplicated infections. However, a 10-20% drop in egg production is common during outbreaks. Turkeys and game birds tend to be more severely affected, and the mortality rate can be high. Meningoencephalitis in turkeys seems to be associated with particular strains of *M. gallisepticum*.

House finches seem to be especially susceptible to the house finch lineage of *M. gallisepticum*. In some areas, this disease has apparently caused populations of these birds to drop by as much as 50%. Most of these deaths are thought to be caused by the consequences of conjunctivitis in free-living birds, such as an increased risk of predation and difficulty finding food and water. Under controlled experimental conditions, the morbidity rate can approach 100% in house finches, but the mortality rate has been low (≤ 5%). Other species of wild birds tend to be less severely affected by the house finch lineage and recover more quickly; and some species, such as house sparrows (*Passer domesticus*) and black-capped chickadees, are relatively resistant to the development of clinical signs. The house finch lineage has been found in a few poultry flocks, but it currently seems that it might cause only mild clinical signs in chickens and turkeys.

Internet Resources

The Merck Veterinary Manual

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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*Link is defunct