# **Avian Bacterial Diseases**

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# **Avian Bacterial Diseases**

# **Salmonella** Infections

### **Salmonella Infections**

■ Salmonella infections in poultry flocks can cause acute and chronic clinical diseases but have received greater international attention in recent years because of their role in foodborne outbreaks of human illness.

Human illnesses resulting from the consumption of poultry products contaminated by Salmonella can be expensive for the poultry industry, governments, and affected individuals.

### **Public Health Significance**

Salmonellae are consistently reported to be among the leading international sources of foodborne human disease.

Poultry products are often identified as prominent sources of salmonellae which cause human illness.

Throughout much of the world, eggs and egg-containing foods have been implicated as the principal vehicles for the transmission of *S. Enteritidis* infections.

### **Avian Salmonella Infections**

Two nonmotile organisms are host-specific for avian species:

>Salmonella Pullorum, causing pullorum disease, an acute systemic disease of chicks or poults.

> *S. Gallinarum*, causing fowl typhoid, an acute or chronic septicemic disease that most often affects mature birds.



### Classification

➤The genus Salmonella is a member of the family Enterobacteriaceae. The causative agents of pullorum disease and fowl typhoid have been taxonomically assigned to a single serovar, S. Pullorum-Gallinarum.

### Morphology and Growth

>Salmonellae are straight, Gram-negative, nonspore-forming rods, measuring about  $0.7-1.5 \times 2.0-5.0 \mu m$ .

Salmonellae are facultatively anaerobic and can grow well under both aerobic and anaerobic conditions.



## **Pathobiology**

#### Incidence and Distribution

The actual prevalence of infection or contamination within Salmonella-positive flocks can vary widely.

➤The incidence of Salmonella infection among broiler chicken flocks have ranged from 9% to 57%, with up to 47% of breeding flocks also identified as positive.

Recent surveys for the incidence of Salmonella in egg-type poultry in various nations have generated similarly diverse results, ranging from 12% to 65% of laying flocks and up to 26% of breeding flocks.

### **Pathobiology**

#### Sources, Vectors, and Transmission

Salmonellae can be introduced into poultry flocks from many sources. Feeds containing contaminated animal proteins, vegetable proteins, or cereals, or contaminated by vermin or wildlife, are potential sources of Salmonella in chickens.

>Vertical transmission of salmonellae to the progeny of infected breeding flocks can result from internal or external contamination of eggs.

### **Clinical Signs**

Omphalitis and yolk sac infection are commonly associated with Salmonella infection of newly hatched poultry.

Severe outbreaks of *Salmonella* infection in young chicks can involve rapidly developing septicemia with high mortality and few apparent lesions. Morbidity and mortality can be high during the first 2–3 weeks of life, with significant body weight loss or growth retardation, but signs of disease are infrequent in older birds.

■*S. pullorum* infection causes egg-transmitted disease in young chicks, often associated with white diarrhea and high mortality. Clinical signs include diarrhea with urate staining of the vent, decreased feed consumption, and huddling near heat sources.



#### Isolation and Identification

Sample Selection: Samples from a variety of sources, including tissues, eggs, voided feces, and poultry house environments, are collected and tested to identify Salmonella infection in flocks. Livers and spleens, as filtering organs, are most likely to be contaminated and thereby are the most effective culture targets for identifying infected birds.

Standard Culture Methods (4 steps): nonselective pre-enrichment encourages the growth of very small numbers of salmonellae; selective enrichment allows additional expansion of the Salmonella population; plating on selective agar media yields isolated colonies; colonies with appearances characteristic of salmonellae are subjected to biochemical and serologic tests to confirm their genus and serovar identity.

# Diagnosis

#### Isolation and Identification

>Culture Media: Suggested broth media to pre-enrich samples for salmonellae include trypticase soy broth and buffered peptone water; The selective broth media most often used for *Salmonella* detection are tetrathionate broth and Rappaport–Vassiliadis broth. Among the most commonly used plating media are brilliant green, xylose-lysine-deoxycholate (XLD), xyloselactose-tergitol 4 (XLT4), Rambach, bismuth sulfite, and Hektoen enteric agars.

>Confirmation of Genus and Serovar: The serogroup of isolates can be determined by agglutination tests with polyvalent antisera for somatic O antigens, and the serovar can then be determined by slide agglutination tests with monovalent antisera to specific O antigens and tube agglutination tests with antisera to flagellar H antigens.

## **Diagnosis**

Rapid Detection Technologies: Specific antibodies have been utilized in a variety of enzyme immunoassay (EIA) methods to detect Salmonella antigens. An increasingly prominent approach to rapid testing for Salmonella is based on detecting genus-specific or even serovar-specific genetic sequences by hybridization of specific probes with target DNA extracted from samples.

Serologic Diagnosis of Infection: Specific antibodies to salmonellae can be found in infected poultry with high sensitivity using diverse agglutination and EIA methods. Antibodies deposited in egg yolks by infected hens offer uniquely convenient samples for *Salmonella* testing.

### **Intervention Strategies**

### Vaccination

>Administration of either killed or live vaccine preparations can significantly reduce the susceptibility of poultry to Salmonella infection.

Subcutaneous or intramuscular vaccination of laying hens with adjuvanted bacterins induces long-lasting antibody responses and has significantly reduced *S. Enteritidis* isolation from feces, internal tissues, and eggs following subsequent oral challenge.

Live attenuated vaccines must persist in tissues long enough to induce protective immune responses, but should be avirulent and cleared from vaccinated birds within a few weeks of administration.

## **Intervention Strategies**

#### Treatment

➤A variety of antibiotics have demonstrated either prophylactic or therapeutic activity against salmonellae in poultry, in some instances leading to decreased fecal shedding when used as feed additives.

Current control practices for poultry salmonellosis in many nations no longer regularly rely on antibiotics because of both inconsistent performance of these drugs in eliminating Salmonella colonization and concerns that indiscriminate veterinary and agricultural use could promote microbial resistance.

>Both therapeutic and subtherapeutic antibiotic administration can select for drug-resistant strains of salmonellae.

# **Avian Bacterial Diseases**

# Campylobacteriosis

### **Campylobacteriosis**

Thermophilic *Campylobacter* species, primarily *C. jejuni* and *C. coli*, are frequent colonizers of the intestinal tract of domestic poultry species.

Despite the extensive colonization, *Campylobacter* is generally regarded as a commensal in birds, and its infection rarely results in clinical disease or significant pathological lesions.

Fecal colonization leads to consequent carcass contamination in processing plants and foodborne transmission of *Campylobacter* to humans, a significant burden for public health worldwide.



### Classification

The genus *Campylobacter* contains at least 30 valid species and subspecies.

>The family Campylobacteraceae represents a diverse but phylogenetically distinct group within the group of Gram-negative bacteria and placed in the epsilon division of the Proteobacteria.

### **Etiology**

### Morphology and Growth

➤Thermophilic Campylobacter spp. grow optimally at 37° C-42° C on artificial media.

They are slowly growing fastidious organisms and require a microaerobic atmosphere for optimal growth.

>Campylobacter cells are S-shaped spirally curved rods in size of  $0.2-0.8 \mu m$  wide and  $0.5-6.0 \mu m$  long.



# **Pathobiology**

#### Incidence and Distribution

Campylobacter jejuni and C. coli are widespread in avian hosts, especially in commercial chickens.

>On commercial poultry farms, Campylobacter is rarely detected in birds younger than 2–3 weeks of age regardless of production type or species of poultry.

>Typically, the prevalence of Campylobacter increases as the birds grow and reaches to the highest point at the slaughter age for broiler chickens.

#### Sources, Vectors, and Transmission

Many farm-based studies have indicated that horizontal transmission from the environment to poultry houses is the most common source of infection of Campylobacter on poultry farms.

> The current understanding is that vertical transmission of Campylobacter does not occur or occurs very rarely on poultry farms.



#### Clinical Signs

Campylobacter infections in poultry usually produce no clinical signs of disease under natural conditions.

➤A novel Campylobacter species, C. hepaticus, causes spotty liver disease, which is very similar to vibrionic hepatitis and characterized by the presence of multifocal, 1–2 mm grey-white lesions in the liver.





#### Culture-based Isolation and Detection Methods

➤Thermophilic campylobacters are fastidious and slow growing, requiring microaerobic atmosphere (containing 5% O2, 10% CO2, 85% N2) and elevated temperature (42° C) for optimal growth under laboratory conditions, with no growth observed at temperatures below 31° C.

Culturing Campylobacter spp. from fecal or environmental materials with a high level of background flora requires the use of selective culture media and special culture conditions.



#### Immunology-Based Diagnostic Methods

Enzyme immunoassays (EIA), based on antigen-antibody interaction, have been developed for direct detection of Campylobacter spp. in animal feces or processed food.

#### Nucleic Acid-Based Diagnostic Methods

Molecular techniques, in particular PCR, has been utilized for direct detection of Campylobacter from various sources.

### **Intervention Strategies**

In general, on farm control strategies that have been examined for control of Campylobacter in poultry can be broadly divided in 2 approaches:

> Prevention of flock colonization by use of biosecurity-based interventions.

>Prevention and/or reduction of Campylobacter colonization by nonbiosecurity-based measures such as vaccination, bacteriocins, feed additives, bacteriophages, and competitive exclusion. Currently there are no commercial vaccines available for control of Campylobacter in poultry.

>At present no single measure is completely effective in controlling Campylobacter infections on poultry farms.

# **Avian Bacterial Diseases**

# Colibacillosis

### **Colibacillosis**

Colibacillosis is a localized or systemic disease of production poultry associated with the bacterial pathogen avian pathogenic *Escherichia coli* (APEC).

The disease may present in a number of forms including colisepticemia, air sac disease, peritonitis, swollen head syndrome, and salpingitis among others.

### **Economic Significance**

Colibacillosis is the most common infectious bacterial disease of poultry and that collectively, *E. coli* infections in their various forms are responsible for significant economic losses.

Often collibacillosis is among the most frequently reported diseases in surveys of poultry health or condemnations at processing.

### **Public Health Significance**

Shigatoxin producing *E. coli* (STEC), including *E. coli* O157:H7, an important enterohemorrhagic pathogen of humans, has been isolated from various types of birds and poultry products. More concerning is the possibility that APEC-contaminated poultry and eggs are a foodborne reservoir of ExPEC that cause human urinary tract infections, meningitis, and other

extraintestinal diseases.

### **Etiology**

#### Classification

>E. coli is the type species of the genus Escherichia, family Enterobacteriaceae.

#### Morphology and Growth

>*E. coli* is a Gram-negative, non-acid-fast, uniform staining, non-spore-forming bacillus, usually  $2-3 \times 0.6 \mu m$ . Most strains are motile and have peritrichous flagella.

>*E. coli* grow aerobically or anaerobically on ordinary nutrient media at temperatures of 18–44° C.





#### Antigenic Structure

➤Currently there are approximately 180 O (Somatic), 60 H (Flagellar), and 80 K (Capsular) antigens of *E. coli* according to the Kauffmann scheme. The O antigen determines serogroup; addition of the H antigen and sometimes K antigen determines serotype.

#### Pathogenicity

An embryo lethality test can be used to test avian *E. coli* isolates for virulence.
Eleven 12-day-old chicken embryos are inoculated via the allantoic cavity with 100 cfu of the test organism. Two day mortality is less than 10% for nonvirulent strains, 10–29% for intermediate strains, and more than 29% for virulent strains.
Intravenous and subcutaneous inoculation of chicks correlated with embryo lethality.

# Pathobiology

### Incidence and Distribution

Fluoroquinolone-resistant *E. coli* were vertically transmitted from clinically normal breeders and caused high mortality in chicks.

➤The most important source of egg infection is fecal contamination of the egg surface with subsequent penetration of the shell and membranes.

Colibacillosis often occurs concurrently with other diseases making it difficult to determine the contribution of each agent to the overall clinical disease.

## **Pathobiology**

### Clinical Signs

>Clinical signs vary from inapparent to total unresponsiveness just prior to death depending on the specific type of disease produced by *E. coli*.

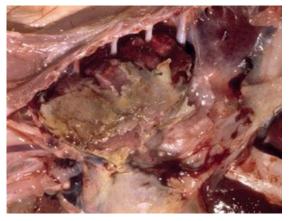
➢All ages are susceptible to colibacillosis, but young birds are more frequently affected and disease severity is greater in young birds.

>Outbreaks can occur in caged layers and coliform salpingitis/peritonitis is a common cause of mortality in breeders.

>Both morbidity and mortality are highly variable depending on the type of disease produced by E. coli.

**Coliform Omphalitis/Yolk Sac Infection**: Swelling, edema, redness, and possibly small abscesses characterize acute inflammation of the navel; Consequences of yolk sac infection include deprivation of nutrients and maternal antibodies, absorption of toxins, and spread of *E. coli* by extension into the body cavity (peritonitis) or systemically to produce colisepticemia and its sequelae (polyserositis, arthritis).

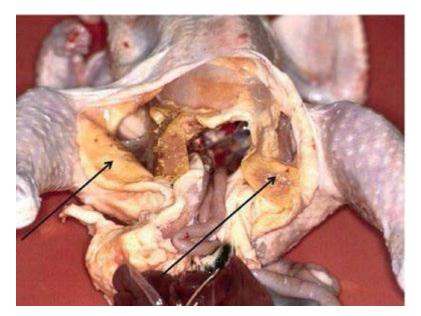




Omphalitis

yolk sac infection

**Coliform Cellulitis**: Coliform cellulitis is characterized by sheets of serosanguineous to caseated, fibrinoheterophilic exudate in subcutaneous tissues.



A caseous sheet of exudate, often referred to as a plaque, is located in the subcutaneous tissues.

**Swollen Head Syndrome**: Swollen head syndrome (SHS) is an acute to subacute cellulitis involving the periorbital and adjacent subcutaneous tissues of the head.



#### Swollen-head syndrome in a broiler chicken

**Diarrheal Disease**: Swollen head syndrome (SHS) is an acute to subacute cellulitis involving the periorbital and adjacent subcutaneous tissues of the head.

**Coliform Salpingitis/Peritonitis/Salpingoperitonitis (Adult):** Inflammation of the oviduct caused by *E. coli* results in decreased egg production and sporadic mortality.





#### Salpingitis

Peritonitis

# Diagnosis

#### Isolation and Identification

Sample Selection: Diagnosis is based on isolation and identification of E. coli from lesions typical of colibacillosis. Care must be taken to avoid fecal contamination of samples.

**Culture Media**: Material should be streaked on EMB, MacConkey, or tergitol-7 agar, as well as noninhibitory media.

**Antigenic Identification**: a pentaplex PCR "quick test" was designed to distinguish APEC from commensal E. coli isolated from the feces of healthy poultry without having to resort to in vivo virulence models.

# **Intervention Strategies**

#### Management Procedures

➢ Reducing the numbers of *E. coli* through water, feed, environmental sanitation, and good air quality, and protecting the flock from factors, especially viral infections that decrease host resistance, will sharply reduce the likelihood of colibacillosis.

### Vaccination

Broilers are rarely vaccinated.

>In breeder hens, a killed autogenous *E. coli* vaccine is effective.

>Live vaccines against *E. coli* are popular amongst egg layer producers and have contributed to reducing *E. coli* peritonitis in layers.

### **Types of Vaccines**

#### Inactivated Vaccines

>provide protection against homologous serogroups, but no cross-protection against heterologous serogroups.

#### Live Vaccines

➤A live vaccine prepared from a naturally occurring, nonpathogenic, piliated E. coli strain (BT-7) was efficacious when used in chickens older than 14 days of age.

#### Recombinant and Mutant Vaccines

➤A recombinant vaccine using S. typhimurium was constructed to produce homologous group B determinants and E. coli O78 antigen.

#### Molecular Vaccines

>Immunization of chickens with Iss, a surface protein common to APEC.

## **Treatment**

### Antimicrobial Drugs

Antimicrobial drugs have been used extensively for reducing losses from colibacillosis since their first introduction for treatment of poultry in the mid 1950s.

➢Growing concern over antibiotic resistance, especially multidrug resistance, has led to changes in the way antimicrobials are used to treat colibacillosis in poultry.

>When selecting an antimicrobial to use for treatment, it is important to determine the susceptibility of the isolate involved in the disease outbreak.

>Water administration of apramycin proved effective in reducing the numbers of organisms in the digestive tract and preventing bacteremia in chickens.

## **Treatment**

### Other Treatments

> The declining use of antibiotics for prevention and treatment of colibacillosis has stimulated interest in alternative methods including prebiotics, probiotics, enzymes, digestive acidifiers, vitamins, immune enhancers, anti-inflammatory drugs, and other antimicrobial products.

Essential oils often have a substantial inhibitory effect on *E. coli* in vitro and in the lower intestinal tract of chickens.

>Bacteriophage administration provides another possible alternative to antibiotic medication for controlling colibacillosis.

# **Avian Bacterial Diseases**

# **Fowl Cholera**

### **Fowl cholera**

Fowl cholera is a contagious disease affecting both domestic and wild birds and is caused by *Pasteurella multocida*.

It usually appears as a septicemic disease associated with high morbidity and mortality, but chronic or benign conditions often occur.

Fowl cholera is of major economic importance wherever poultry are raised.

### **Etiology**

### Classification

Pasteurella multocida is the causative agent of fowl cholera, which belongs to the genus Pasteurella and family Pasteurellaceae.

➢P. multocida is a Gram-negative, nonmotile, nonspore-forming rod occurring singly, in pairs, and occasionally as chains or filaments.

➢Conventional serotyping of *P. multocida* has focused on capsular and somatic antigens. Five capsular types (A, B, D, E, and F) are currently recognized. Somatic serovar specificity was found to be linked to the lipopolysaccharide (LPS) and 16 Heddleston somatic serovars have been described.

# Pathobiology

#### Incidence and Distribution

>Fowl cholera is typically more prevalent in late summer, fall, and winter.

➢Most reported outbreaks of FC in commercial birds involve chickens, turkeys, ducks, or geese.

#### Sources, Vectors, and Transmission

>No evidence that P. multocida was transmitted through the egg.

>Most species of farm animals may be carriers of *P. multocida*.

Contaminated crates, feed bags, or any equipment used previously for poultry may serve in introducing FC into a flock.

### **Clinical Signs**

#### Acute Disease

Signs of infection in acute FC are often present for only a few hours before death.

Signs that often occur are fever, anorexia, ruffled feathers, mucous discharge from the mouth, diarrhea, and increased respiratory rate.

Cyanosis often occurs immediately prior to death and is most evident in unfeathered areas of the head, such as comb and wattles.

>Fecal material associated with the diarrhea is initially watery and whitish in color but later becomes greenish and contains mucus.



#### Chronic Disease

>Wattles, sinuses, leg or wing joints, foot pads, and sternal bursae often become swollen.

Exudative conjunctival and pharyngeal lesions may be observed, and torticollis sometimes occurs.

> Tracheal rales and dyspnea may result from respiratory tract infections.

#### Morbidity and Mortality

➢In naturally infected chickens, mortality usually ranges from 0% to 20%, but greater losses have been reported.

>Reduced egg production and persistent localized infection often occur.



### Isolation and Identification

➢P. multocida can be isolated readily from viscera of birds that die of acute FC and usually from lesions of chronic cases.

➢Bone marrow, heart blood, liver, meninges, or localized lesions are preferred for culturing.

>A range of PCR assays for the detection or identification for *P. multocida* have been described.

➢Use of commercial MALDI-TOF instruments for the identification of bacteria in diagnostic laboratories is now common.

# **Intervention Strategies**

#### Management Procedures

➤The primary source of infection is usually sick birds or those that have recovered and still carry the causative organism, or other carriers, such as rodents or cats.

Measures should be taken to prevent association of wild birds with the flock.

### Vaccination

>Vaccination should be considered in areas where FC is prevalent, but it should not be substituted for good sanitary practice.

### **Types of Vaccines**

#### Inactivated Vaccines

Commercially produced bacterins are available and usually contain whole cells of Heddleston serovars 1, 3, 4 and 3X4 emulsified in an oil adjuvant.

#### Live Vaccines

>Vaccination of chickens and turkeys with live vaccines induces protection against heterologous serovar challenge.

#### Field Vaccination Protocols and Regimes

➢Bacterins, live vaccines, or both are used, and usually 2 doses are given: the first at 8–10 weeks of age and the second at 18–20 weeks of age.

Some of the more commonly used vaccination programs consist of administering a live vaccine in the wing web at 10−12 weeks of age followed by either another live vaccine in the wing web or a bacterin at 18−20 weeks.

### **Treatment**

### Antimicrobial Drugs

Sensitivity testing should be performed because P. multocida isolates vary in susceptibility to chemotherapeutic agents and resistance to treatment may develop, especially during prolonged use of these agents.

The use of antimicrobial agents is a highly regulated area and can vary from nation to nation.

➤A general observation is that many *P. multocida* isolates from poultry remain sensitive to traditional agents such as amoxicillin, penicillin, and tetracyclines.

# **Avian Bacterial Diseases**

# **Infectious Coryza**

## **Infectious Coryza**

Infectious coryza (IC) is an acute respiratory disease of chickens caused by the bacterium known as Avibacterium paragallinarum.

■*Av. paragallinarum* typically requires nicotinamide dinucleotide (NAD) for in vitro growth.

The organism can be classified into 3 Page serovars (A, B, and C) or 9 Kume serovars (A-1 to A-4, B, and C-1 to C-4).

# Pathobiology

#### Incidence and Distribution

➢Infectious coryza occurs wherever chickens are raised. The disease is a common problem in the intensive chicken industry.

➢All ages of chickens are susceptible to Av. Paragallinarum, but the disease is usually less severe in younger birds.

### Sources, Vectors, and Transmission

Chronic or healthy carrier birds have long been recognized as the main reservoir of IC infection.

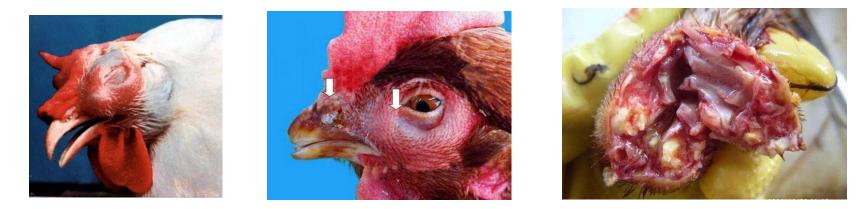
➤IC is not an egg-transmitted disease.

# **Clinical Signs**

The most prominent features of IC are an acute inflammation of the upper respiratory tract including involvement of the nasal passage and sinuses with a serous to mucoid nasal discharge, facial edema, and conjunctivitis.

Swollen wattles may be evident, particularly in males. Rales may be heard in birds with infection of the lower respiratory tract.

■IC is usually characterized by low mortality and high morbidity.





### Isolation and Identification

➤A sterile cotton swab is inserted deep into the sinus cavity where the organism is most often found in pure form. Tracheal and air sac exudates also may be taken on sterile swabs.

➤The swab is streaked on a blood agar plate, which is then crossstreaked with a Staphylococcus culture and incubated at 37 ° C in a large screw-cap jar in which a candle is allowed to burn out.

➤Two PCR tests, one a conventional PCR and the other a real-time PCR, both based on the same target, have been developed for Av. paragallinarum. Both PCRs are specific for Av. paragallinarum, are rapid and able to detect all known variants.

### **Vaccination**

#### Inactivated Vaccines

Commercial IC bacterins are widely available.

Inactivated IC bacterins provide, at best, protection only against the Page serovars included in the vaccine; it is vital that bacterins contain the Page serovars present in the target population.

#### Field Vaccination Protocols and Regimes

➢ Infectious coryza bacterins are generally injected in birds between 10 and 20 weeks of age and yield optimal results when given 3–4 weeks prior to an expected natural outbreak.

➤Two injections given approximately 4 weeks apart before 20 weeks of age seem to result in better performance of layers than a single injection.

Injection of the bacterin into the leg muscle gave better protection than when injected into the breast muscle.

### **Treatment**

### Antimicrobial Drugs

➢ Various sulfonamides and antibiotics are useful in alleviating the severity and course of IC.

>Erythromycin and oxytetracycline are 2 commonly used antibiotics.

# **Avian Bacterial Diseases**

# **Mycoplasmosis**

# **Mycoplasmosis**

Avian mycoplasmosis is a collection of diseases of worldwide distribution caused by bacteria in the genus Mycoplasma affecting several bird species.

They are vertically and horizontally transmitted and the clinical signs vary greatly with the Mycoplasma species, the strain, and the species of bird infected.

Respiratory disease, synovitis, poor performance, skeletal deformities, or embryo mortality have all been associated with Mycoplasma infection.

The primary avian Mycoplasma pathogens of concern to poultry include *M. gallisepticum*, *M. synoviae*, *Mycoplasma iowae* and *M. meleagridis*.

### **Mycoplasma gallisepticum Infection**

Mycoplasma gallisepticum (MG) causes chronic respiratory disease (CRD) in chickens and infectious sinusitis in turkeys.

MG is egg transmitted, and economic losses result from processing condemnations, reduced egg production and feed efficiency, and costs of control.

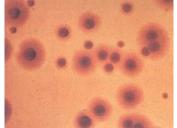
MG is worldwide in distribution and the most pathogenic and economically significant mycoplasmal pathogen of poultry.

# **Etiology**

Morphology and Staining: In Giemsa-stained preparations or darkfield examinations, MG cells generally appear as pleomorphic coccoid or coccobacillary bodies approximately 0.2–0.5 µm in diameter.

Growth Requirements: Mycoplasma species from avian sources generally require a protein-rich medium containing 10%–15% added animal serum.

Colony Morphology: Typical colonies are small (0.1–1.0 mm), smooth, circular, and somewhat flat with a denser central elevation ("fried egg" appearance).





# **Pathobiology**

#### Incidence and Distribution

➤MG infections have resulted in important flock health challenges in chickens and turkeys in all areas of commercial production, and are worldwide in distribution.

#### Sources, Vectors, and Transmission

Horizontal transmission of MG occurs readily by direct or indirect contact of susceptible birds with clinically or subclinically infected birds, resulting in high infection/disease prevalence within flocks.

MG can be transmitted vertically from naturally infected hens to their progeny.

# **Clinical Signs**

The most characteristic signs of naturally occurring MG disease in adult flocks are tracheal rales, nasal discharge, and coughing.

In laying flocks, egg production declines but is usually maintained at a lowered level.

Severe outbreaks with high morbidity and mortality observed in broilers are frequently caused by concurrent infections and environmental factors.

Gross lesions consist primarily of mucosal congestion and catarrhal exudate in nasal and paranasal passages, trachea, bronchi, and air sacs.



### Isolation and Identification

Isolation and identification of the organism is the gold standard for MG diagnosis.

➢For MG culture, swabs taken from the trachea or choanal cleft (palatine fissure) should be inoculated directly to *Mycoplasma* broth and/or agar media.

### Detection of Causative Agent Genetic Material

➢PCR allows rapid, sensitive, and specific detection of MG DNA. Both conventional and real- time PCR techniques are widely used for MG detection, and can be performed directly on clinical samples, without the requirement for culture.



### Serology

Serologic procedures are useful for flock monitoring in MG control programs and to aid in diagnosis when infection is suspected.

➤The serum plate agglutination (SPA) or ELISA tests are used for serologic screening, whereas the HI test is generally used to confirm SPA or ELISA reactors.

Serologic diagnosis should be confirmed by MG isolation and identification and/or by PCR.

# **Intervention Strategies**

#### Management Procedures

➢In situations where preventing MG infection is not considered feasible or economically viable, appropriate antimicrobial therapy may be used as a short-term intervention to reduce morbidity, mortality, production losses, and MG transmission. Vaccination may be considered as a longer term intervention in some situations.

### Vaccination

>Vaccination prior to wildtype exposure is essential. Inactivated, live attenuated, and recombinant MG vaccines are commercially available.



#### Inactivated Vaccines

➤MG bacterin vaccines typically comprise inactivated MG organisms suspended in aqueous oil emulsion or in aluminum hydroxide adjuvants, and are administered by the intramuscular or subcutaneous route.

#### Live Attenuated Vaccines

The 3 commercially licensed live MG vaccines currently in common use worldwide are F strain, ts-11, and 6/85.

#### Recombinant Vaccines

>A recombinant fowlpox-MG vaccine is available.

### **Treatment**

### Antimicrobial Drugs

>MG has shown sensitivity in vitro and in vivo to several antibiotics including macrolides, pleuromutilins, tetracyclines, and fluoroquinolones.

>Various antibiotics, including tylosin, tilmicosin, tylvalosin, tiamulin, valnemulin, oxytetracycline, chlortetracycline, enrofloxacin, danofloxacin, and lincomycin-spectinomycin have demonstrated efficacy for the treatment of MG respiratory diseases, reducing the severity of clinical signs and gross lesions, and lowering mortality and performance losses.

>Tylosin and tetracycline antibiotics are commonly used worldwide for the treatment of MG disease.

# Thank you for your attention!