



# Respiratory disorders of backyard poultry – Part 2 of 2: Bacterial, Fungal & Other causes

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## CHLAMYDIOSIS

Avian chlamydiosis is a systemic disease in birds, which can also be zoonotic. Formerly the disease was termed psittacosis in psittacine birds and humans, and ornithosis in other birds.

Infection occurs worldwide in poultry with outbreaks more often observed in turkeys and waterfowl than in chickens. Younger domestic birds are generally more susceptible to infection than older birds. However, undiagnosed infection is widespread in poultry and wild birds, with the pathogen (*Chlamydophila psittaci*) being spread via respiratory tract exudates and faeces. Stress may induce activation of the infection and an outbreak of the disease. Transmission occurs by inhalation of contaminated dry dust, in which the organism can survive for a long time. After infection by aerosol, replication of the pathogen begins in the epithelial cells of the upper respiratory tract and subsequently spreads to the epithelia and macrophages throughout the respiratory tract.

Clinical signs:

The highly virulent strains induce anorexia, cachexia, and conjunctivitis. Respiratory distress, yellow-green droppings and a decrease in egg production in laying birds. In birds infected with low virulent strains, the signs are less severe or even absent. Splenomegaly is commonly seen, even on radiographs, and is sometimes accompanied by hepatomegaly.

Diagnosis:

The preferred and most common method of diagnosis is PCR. Swabs from the conjunctiva, pharynx or cloaca of live birds or tissue samples from the air sacs, spleen and liver of dead birds can be submitted for PCR

Treatment:

Due to its zoonotic potential, there may be regulations that have to be adhered to regarding the handling of infected birds. When treatment is allowed, the flock should be treated with tetracycline via the feed (0.4g/kg) for at least 42 days or enrofloxacin for 21 days. Enrofloxacin should not be used in birds whose meat/eggs are intended for human consumption. Supportive care is extremely important.

Prognosis:

In severely affected birds, the prognosis is grave, and birds will die despite treatment. If the entire flock is treated immediately, the prognosis for the other birds is usually good (even though several birds may die despite starting treatment). It needs to be borne in mind, however, that birds may become carriers even after treatment. This may be a risk to other birds within the flock as infection does not confer lifelong immunity. A flock is considered clear if all birds have tested negative on two consecutive occasions (5 days apart).

## MYCOPLASMOSIS

Mycoplasmosis is caused by *Mycoplasma gallisepticum* (a chronic respiratory disease in chickens; infectious sinusitis in turkeys), *M. meleagridis*, *M. synoviae* and *M. iowae*. There are several more *Mycoplasma* spp. Known to occur in poultry, but its role in respiratory disease is minor.

*M. gallisepticum* and *M. synoviae* are of greatest importance and are common in all kinds of backyard poultry. Sources of infection are latently and chronically infected birds. Transmission can be vertical and horizontal. Transmission can occur directly or indirectly, with transmission via aerosol of most importance. Within a flock, the spread of the pathogen is usually slow.

The organisms enter the body via the mucosa of the conjunctiva and/or respiratory tract or are vertically transmitted. These organisms then attach to the epithelial cells and multiply.

Usually, the infection remains latent, due to the ability of the organism to 'hide' from the host immune system by mimicry (absorption or imitation of host cell structures) or due to the variation in antigen-dominant membrane structures.

They pave the way for infection with other pathogens and clinical disease can only occur in the presence of these secondary pathogens. The disease is also possible following impairment of the hosts' defence system by stressors such as transportation or suboptimal conditions. Turkeys are usually more susceptible to the disease than other poultry.

Clinical signs:

The respiratory form of the disease is associated with dyspnoea (which can be severe) and rasping sounds on breathing, as well as nasal discharge and swelling of the infraorbital sinus and eyelids, especially in turkeys. Often poor egg hatchability is seen, and the embryos demonstrate curled toes or dwarfism.

Diagnosis:

Diagnosis is made by cultivation of the organism using special media. PCR and ELISA can also be used.

Treatment:

Antibiotics such as macrolides (e.g.: tylosin), tetracyclines and fluoroquinolones, may be useful to reduce the severity of the clinical signs, but treatment does not eliminate the infection from carrier birds.

Prognosis:

The clinical prognosis for the flock is usually good. Treatment reduces clinical signs; however, it is nearly impossible to eliminate the pathogen from the flock and recurrence of the disease is likely eventually.

## **ORNITHOBACTERIUM RHINOTRACHEALE**

*O. rhinotracheale* has a worldwide distribution and causes acute to subacute disease in turkeys and chickens. Wild birds can act as sources of infection. Transmission occurs mainly horizontally via direct contact or indirectly by living and inanimate vectors. Vertical transmission is assumed. Birds of all ages are susceptible, although signs of disease are more severe in older birds.

Clinical signs:

Clinical signs are variable and initially include sneezing and nasal discharge, followed by oedema around the infraorbital sinus and dyspnoea of varying degrees. These clinical signs may be associated with depression and a decrease in egg production. Mortality varies from 1-50%. The main lesions on postmortem are pulmonary oedema, consolidation of the lungs, pneumonia, pleuritis and air sacculitis with a foamy, white, yoghurt-like exudate.

Diagnosis:

The history and clinical and pathological signs allow a presumptive diagnosis, which must be verified by cultural isolation of the pathogen. In particular, differentiation from *P. multocida* is important. Suitable samples for laboratory analysis are tracheal swabs from live birds and tissue samples from the lungs and air sacs of dead birds.

Treatment:

As *O. rhinotracheale* can develop resistance to antimicrobials quickly, and there are significant differences in the susceptibility of the pathogen to antibiotics, it is essential to perform a sensitivity test before initiating treatment.

Prognosis:

The prognosis for the flock is usually good and repeated treatment usually leads to reduction of clinical signs. However, in the individual bird, especially if it is chronically and severely affected, the prognosis is poor as the massive fibrinopurulent air sacculitis is difficult to treat, and a full recovery may not be possible.

## **INFECTIOUS CORYZA**

Infectious coryza is a highly contagious, acute respiratory bacterial disease of chickens caused by *Avibacterium paragallinarum*.

The disease occurs worldwide, and chickens of all ages are susceptible with the disease being more severe in mature birds. Pheasants, quails and guinea fowl may also become infected, but turkeys and ducks appear to be refractory.

As the tenacity of the pathogen is very low outside the body, the sources of infection are chronically infected or latently infected carriers who transmit the bacteria via respiratory exudates directly or indirectly (particularly contaminated drinking water). Morbidity is high, and mortality is low. *A. paragallinarum* has a tropism for the cilia-covered epithelia of the upper respiratory tract.

Clinical signs:

The clinical signs of acute infection include clear seromucoid to opaque flocculated nasal discharge, conjunctivitis and facial oedema. Birds sneeze with sticky nostrils, breathe with an open beak, shake their heads and wipe the exudate on the feathers of their wings and back.

Depression, reduced feed and water intake and diarrhoea lead to growth retardation and a reduction in egg production. In chronic cases, birds develop dyspnoea, and the oedema extends to the wattles and eyes.

Diagnosis:

Swabs of exudate squeezed from the sinus of live birds and swabs of the deep sinus cavity of dead birds can be submitted for culture or PCR.

Treatment:

The choice of antimicrobial should be based on culture and sensitivity. Sulphonamides are usually effective. Relapse post-treatment may occur, and the carrier state cannot always be avoided.

Prognosis:

The prognosis for both the individual bird and the flock is usually good when treatment is started immediately. More severely affected birds should be treated individually whilst the rest of the flock can be treated via drinking water.

## **FOWL CHOLERA**

Fowl cholera is a contagious disease of domestic poultry as well as wild birds, caused by *Pasteurella multocida*. It occurs as either a peracute to acute septicaemic disease or as a chronic localized disease. Amongst poultry, the disease occurs mainly in free-range turkeys, chickens and waterfowl, with young mature birds affected most severely and most often. Chronic latent carriers of the pathogen are the major source of the infection. Transmission occurs via direct or indirect contact. The virulence of *P. multocida* varies from apathogenic to highly virulent. Strains of lower virulence result in disease processes restricted to the respiratory tract. The clinical signs here include tracheal rales, dyspnoea, nasal and ocular discharge, swelling of the sinuses, wattles, leg or wing joints, foot pads and sternal bursa and torticollis.

Diagnosis:

Confirmation of the diagnosis should be made by isolation of *P. multocida*. Preferred samples for culture include swabs of the squeezed mucus from the nostrils of living birds and specimens of the liver, viscera and other localized lesions in dead birds.

Treatment:

Treatment with sulphonamides and other antibiotics is possible to reduce the severity of the clinical signs and mortality rate. Birds with acute disease should receive intensive treatment, including intravenous antibiotics. The duration of treatment should be long (approximately 10 days), but it should be noted that recurrence of the disease may occur after cessation of treatment.

Prognosis:

The prognosis for birds with *P. multocida* infection is uncertain as recurrence of infection is common.

## **COLIBACILLOSIS:**

Colibacillosis (colisepticaemia) is an important, worldwide systemic disease caused by *E. coli* and can affect all birds. Colibacillosis in the respiratory tract develops when *E. coli* colonizes the host following

damage to the respiratory mucosa by other infectious and non-infectious agents (e.g. ammonia). Lesions from the resulting disease, which is commonly called air sac disease, chronic respiratory disease (CRD), multi-causal respiratory disease or swollen head syndrome (SHS), are prominent in the trachea, lungs and air sacs and are characterized by a serofibrinous inflammation. To confirm the diagnosis, isolation and identification of all involved pathogens are essential.

### **ASPERGILLOSIS**

The main causative agents for avian aspergillosis are members of the genus *Aspergillus*, particularly *A. fumigatus* and *A. flavus*. The spores of these organisms can survive in the environment for years. Many, if not all, birds are susceptible to aspergillosis. Sources of infection include litter, food, moist bedding, faeces, dust and incubators as well as feeders and drinkers. Infection occurs by inhalation of fungal conidia. In confined birds, morbidity and mortality may reach up to 50%; free-ranging birds are more resistant. Aspergillosis can also be acquired in ovo by penetration of the fungus through the eggshell during incubation. The disease occurs mainly in young birds, especially after hatching, and seldom in older animals, unless the bird's immune system has been compromised.

Clinical signs:

The disease is characterized by exudative necrotizing and/or granulomatous inflammatory processes, primarily in the lower respiratory tract. Young birds present with high-grade dyspnoea and gasping, but usually no breathing sounds. With chronic disease, there are often only non-specific clinical signs whilst respiratory signs become obvious only shortly before death. At necropsy, catarrhal to fibrinous inflammatory lesions and/or yellowish-grey, miliary to pea-sized granulomas are observed in the lungs and air sacs. Sometimes mould growth is macroscopically visible.

Diagnosis:

Clinical signs together with culture are used to confirm the diagnosis.

Treatment:

The main treatment measures include sanitation of the housing, daily cleaning and disinfection of drinkers and feeders, the addition of copper sulphate to the drinking water (1-3g/l), and fumigation with thiabendazole or enilconazole.

Oral or parenteral antifungal drugs are useful in affected birds, but legislation regarding the use of antifungals in food-producing animals should be reviewed before starting treatment. Voriconazole is the drug of choice (10mg/kg po q12h for 3 weeks, followed by 10mg/kg po q24h for 3 weeks). Itraconazole is a good alternative drug.

Prognosis:

The prognosis for the flock is poor. Antifungal treatment is only possible for individual birds rather than the entire flock because it can take several weeks to complete the course of medication. The prognosis of the individual bird is always guarded.

### **HEAT STRESS**

Temperatures above the comfort zone (>28°C), especially in combination with high humidity (>80%) can cause heat stress in poultry.

As birds do not have sweat glands, they use non-evaporative cooling (radiation, conduction, convection) and evaporative cooling via the respiratory tract as a means of heat dissipation. In cases where this physiological system still does not prevent the body temperature from rising, birds may die from respiratory, circulatory or electrolyte imbalances.

### **VITAMIN A DEFICIENCY**

Vitamin A deficiency causes keratinization of the epithelia on glands and mucosa. In the respiratory tract, the process begins with the exudation of seromuroid to milky white caseous masses in the eyes, nose, sinuses, trachea and bronchi. It is difficult to differentiate this condition from infectious bronchitis and infectious coryza. Later in the disease process, the epithelium is replaced by a stratified squamous keratinizing epithelium, giving the surface a dry, dull and uneven appearance.

### **REFERENCES:**

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2. Poland G., Raftery A. 2019. *Backyard poultry medicine and surgery*.
3. Smith et al. 2009. *Basic poultry production and management*. UP course notes.



# MULTIPLE-CHOICE QUESTIONS

## QUESTION 1

**What is the preferred method of diagnosing Chlamydiosis?**

- a. IHC
- b. Culture
- c. ELISA
- d. HIT
- e. PCR

## QUESTION 2

**You diagnose *C. psittaci* in a backyard flock. You start treating the flock with tetracycline in feed. How long should treatment continue?**

- a. 21 days
- b. 57 days
- c. 42 days
- d. 5 days
- e. 36-72 hours

## QUESTION 3

**What is the dose of tetracycline in feed?**

- a. 0.4g/kg
- b. 1mg/kg
- c. 40mg/kg
- d. 4g/kg
- e. 0.4mg/kg

## QUESTION 4

**Which of the following is true regarding Mycoplasmosis?**

- a. Transmission is mostly via contaminated drinking water
- b. Turkeys are refractory to the disease
- c. There are breed predispositions to the disease
- d. Antibiotics are ineffective
- e. Turkeys are more susceptible to the disease when compared to other poultry

## QUESTION 5

**Which of the following is false?**

- a. Supplementing Vitamin C helps with a bird's immunity to *O. rhinotracheale*
- b. Tracheal haemorrhage on necropsy makes *O. rhinotracheale* a likely diagnosis
- c. Most cases of *O. rhinotracheale* require air sac intubation
- d. *O. rhinotracheale* only occurs in South Africa
- e. All of the above

## QUESTION 6

**Which is the causative agent of Infectious Coryza?**

- a. *Avibacterium paragallinarum*
- b. *E. coli*
- c. *Pasteurella multocida*
- d. *Mycobacterium tuberculosis*
- e. *Ornithobacterium rhinotracheale*

## QUESTION 7

**Which is the causative agent of Fowl Cholera?**

- a. *Avibacterium paragallinarum*
- b. *E. coli*
- c. *Pasteurella multocida*
- d. *Mycobacterium tuberculosis*
- e. *Ornithobacterium rhinotracheale*

## QUESTION 8

**What is an alternative name for Colibacillosis?**

- a. Fowl cholera
- b. Anatepistifer disease
- c. Swollen head syndrome
- d. Infectious coryza
- e. Gape

## QUESTION 9

**At what dose should copper sulphate be added to drinking water for Aspergillosis?**

- a. 1-3g/l
- b. 2-4g/l
- c. 1-3mg/l
- d. 13g/l
- e. 13mg/l

## QUESTION 10

**When should heat stress be suspected?**

- a. Deaths in multiple birds
- b. Open-beak breathing
- c. Reduced food intake
- d. Warm, humid conditions
- e. All of the above

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