

Common Poultry Diseases ¹

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Respiratory Diseases

There are many common and important diseases which can affect the respiratory system (air passages, lungs, air sacs) of poultry (see Table 1). Poultry refers to birds that people keep for their use and generally includes the chicken, turkey, duck, goose, quail, pheasant, pigeon, guinea fowl, pea fowl, ostrich, emu, and rhea. Due to modern systems of management, usually with high poultry densities, these diseases are able to readily spread.

Fowl Pox

Synonyms: chicken pox (not to be confused with chicken pox in humans; the human disease does not affect poultry and vice versa), sore head, avian diphtheria, bird pox

Species affected: Most poultry—chickens, turkeys, pheasants, quail, ducks, psittacine, and ratites—of all ages are susceptible.

Clinical signs: There are two forms of fowl pox. The dry form is characterized by raised, wart-like lesions on unfeathered areas (head, legs, vent, etc.). The lesions heal in about 2 weeks. If the scab is removed before healing is complete, the surface beneath is raw and bleeding. Unthriftiness and retarded growth are typical symptoms of fowl pox. In laying hens, infection results in a transient decline in egg production (see Table 1).

In the wet form there are canker-like lesions in the mouth, pharynx, larynx, and trachea. The wet form may cause

respiratory distress by obstructing the upper air passages. Chickens may be affected with either or both forms of fowl pox at one time.

Transmission: Fowl pox is transmitted by direct contact between infected and susceptible birds or by mosquitos. Virus-containing scabs also can be sloughed from affected birds and serve as a source of infection. The virus can enter the blood stream through the eye, skin wounds, or respiratory tract. Mosquitos become infected from feeding on birds with fowl pox in their blood stream. There is some evidence that the mosquito remains infective for life. Mosquitos are the primary reservoir and spreaders of fowl pox on poultry ranges. Several species of mosquito can transmit fowl pox. Often mosquitos winter-over in poultry houses so, outbreaks can occur during winter and early spring.

Treatment: No treatment is available. However, fowl pox is relatively slow-spreading. Thus, it is possible to vaccinate to stop an outbreak. The wing-web vaccination method is used for chickens and the thigh-stick method for turkeys older than 8 weeks.

Prevention: Fowl pox outbreaks in poultry confined to houses can be controlled by spraying to kill mosquitos. However, if fowl pox is endemic in the area, vaccination is recommended. Do not vaccinate unless the disease becomes a problem on a farm or in the area. Refer to the publication PS-36 (Vaccination of Small Poultry Flocks) for more information on fowl pox vaccinations.

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Newcastle Disease

Synonyms: pneumoencephalitis

The highly contagious and lethal form of Newcastle disease is known as viscerotropic (attacks the internal organs) velogenic Newcastle disease, VVND, exotic Newcastle disease, or Asiatic Newcastle disease. VVND is not present in the United States poultry industry at this time.

Species affected: Newcastle disease affects all birds of all ages. Humans and other mammals are also susceptible to Newcastle. In such species, it causes a mild conjunctivitis.

Clinical signs: There are three forms of Newcastle disease—mildly pathogenic (lentogenic), moderately pathogenic (mesogenic) and highly pathogenic (velogenic). Newcastle disease is characterized by a sudden onset of clinical signs which include hoarse chirps (in chicks), watery discharge from nostrils, labored breathing (gasping), facial swelling, paralysis, trembling, and twisting of the neck (sign of central nervous system involvement). Mortality ranges from 10 to 80 percent depending on the pathogenicity. In adult laying birds, symptoms can include decreased feed and water consumption and a dramatic drop in egg production (see Table 1).

Transmission: The Newcastle virus can be transmitted short distances by the airborne route or introduced on contaminated shoes, caretakers, feed deliverers, visitors, tires, dirty equipment, feed sacks, crates, and wild birds. Newcastle virus can be passed in the egg, but Newcastle-infected embryos die before hatching. In live birds, the virus is shed in body fluids, secretions, excreta, and breath.

Treatment: There is no specific treatment for Newcastle disease. Antibiotics can be given for 3–5 days to prevent secondary bacterial infections (particularly *E. coli*). For chicks, increasing the brooding temperature 5°F may help reduce losses.

Prevention: Prevention programs should include vaccination (see publication PS-36, Vaccination of Small Poultry Flocks), good sanitation, and implementation of a comprehensive biosecurity program.

Infectious Bronchitis

Synonyms: IB, bronchitis, cold

Species affected: Infectious bronchitis is a disease of chickens only. A similar disease occurs in bobwhite quail (quail bronchitis), but it is caused by a different virus.

Clinical signs: The severity of infectious bronchitis infection is influenced by the age and immune status of the flock, by environmental conditions, and by the presence of other diseases. Feed and water consumption declines. Affected chickens will be chirping, with a watery discharge from the eyes and nostrils, and labored breathing with some gasping in young chickens. Breathing noises are more noticeable at night while the birds rest. Egg production drops dramatically. Production will recover in 5 or 6 weeks, but at a lower rate. The infectious bronchitis virus infects many tissues of the body, including the reproductive tract (see Table 1). Eggshells become rough and the egg white becomes watery. (See publication PS-24, Egg Quality, for other causes of poor egg quality.)

Transmission: Infectious bronchitis is a very contagious poultry disease. It is spread by air, feed bags, infected dead birds, infected houses, and rodents. The virus can be egg-transmitted, however, affected embryos usually will not hatch.

Treatment: There is no specific treatment for infectious bronchitis. Antibiotics for 3–5 days may aid in combating secondary bacterial infections. Raise the room temperature 5°F for brooding-age chickens until symptoms subside. Baby chicks can be encouraged to eat by using a warm, moist mash.

Prevention: Establish and enforce a biosecurity program. Vaccinations are available.

Quail Bronchitis

Synonyms: none

Species affected: Bobwhite quail are affected. Japanese quail are resistant. The disease is prevalent in the southern states where bobwhite quail are common. Quail bronchitis occurs seasonally as new hatches and broods come along each year.

Clinical signs: Respiratory distress occurs with tracheal rales (rattles), sneezing, and coughing. Feed and water consumption declines dramatically. There can also be conjunctivitis (inflammation of the eye). Loose watery feces are seen in older and sub-acutely affected birds. Nasal discharges are not seen, differentiating quail bronchitis from similar diseases in other poultry (see Table 1).

Transmission: Once infected, quail bronchitis remains on the farm for the duration of the breeding season, infecting each successive brood.

Treatment: There is no specific treatment against quail bronchitis. Quail bronchitis infections are often complicated by concurrent mycoplasma infections. Antibiotics can be used to combat secondary infections. Add tylosin (500g/ton) to the feed for 10 days, withhold the medication for 5 days, and then repeat medication for 5 days. Alternate medication regimens are tylosin (Tylan) or erythromycin (Gallimycin) in the drinking water for the same period of time.

Prevention: There is no commercial vaccine on the market. It is necessary to break the cycle by depopulating and thoroughly cleaning and disinfecting pens and equipment, followed by a 30–90 day quarantine of the facilities.

Avian Influenza

Synonyms: AI, flu, influenza, fowl plague

Species affected: Avian influenza can occur in most, if not all, species of birds.

Clinical signs: Avian influenza is categorized as mild or highly pathogenic. The mild form produces listlessness, loss of appetite, respiratory distress, diarrhea, transient drops in egg production, and low mortality. The highly pathogenic form produces facial swelling, blue comb and wattles, and dehydration with respiratory distress. Dark red/white spots develop in the legs and combs of chickens. There can be blood-tinged discharge from the nostrils. Mortality can range from low to near 100 percent. Sudden exertion adds to the total mortality. Egg production and hatchability decreases. There can be an increase in production of soft-shelled and shell-less eggs (see Table 1).

Transmission: The avian influenza virus can remain viable for long periods of time at moderate temperatures and can live indefinitely in frozen material. As a result, the disease can be spread through improper disposal of infected carcasses and manure. Avian influenza can be spread by contaminated shoes, clothing, crates, and other equipment. Insects and rodents may mechanically carry the virus from infected to susceptible poultry.

Treatment: There is no effective treatment for avian influenza. With the mild form of the disease, good husbandry, proper nutrition, and broad spectrum antibiotics may reduce losses from secondary infections. Recovered flocks continue to shed the virus. Vaccines may only be used with special permit.

Prevention: A vaccination program used in conjunction with a strict quarantine has been used to control mild forms

of the disease. With the more lethal forms, strict quarantine and rapid destruction of all infected flocks remains the only effective method of stopping an avian influenza outbreak. If you suspect you may have Avian Influenza in your flock, even the mild form, you must report it to the state veterinarian's office. A proper diagnosis of avian influenza is essential. Aggressive action is recommended even for milder infections as this virus has the ability to readily mutate to a more pathogenic form.

For more information on avian influenza, refer to publication PS-38 (Avian Influenza in Poultry Species).

Infectious Coryza

Synonyms: roup, cold, coryza

Species affected: chickens, pheasants, and guinea fowl. Common in game chicken flocks.

Clinical signs: Swelling around the face, foul smelling, thick, sticky discharge from the nostrils and eyes, labored breathing, and rales (rattles—an abnormal breathing sound) are common clinical signs. The eyelids are irritated and may stick together. The birds may have diarrhea and growing birds may become stunted (see Table 1).

Mortality from coryza is usually low, but infections can decrease egg production and increase the incidence and/or severity of other diseases. Mortality can be as high as 50 percent, but is usually no more than 20 percent. The clinical disease can last from a few days to 2–3 months, depending on the virulence of the pathogen and the existence of other infections such as mycoplasmosis.

Transmission: Coryza is primarily transmitted by direct bird-to-bird contact. This can be from infected birds brought into the flock as well as from birds which recover from the disease which remain carriers of the organism and may shed intermittently throughout their lives. Birds risk exposure at poultry shows, bird swaps, and live-bird sales. Inapparent infected adult birds added into a flock are a common source for outbreaks. Within a flock, inhalation of airborne respiratory droplets, and contamination of feed and/or water are common modes of spread.

Treatment: Water soluble antibiotics or antibacterials can be used. Sulfadimethoxine (Albon®, Di-Methox™) is the preferred treatment. If it is not available, or not effective, sulfamethazine (Sulfa-Max®, SulfaSure™), erythromycin (gallimycin®), or tetracycline (Aureomycin®) can be used as alternative treatments. Sulfa drugs are not FDA approved for pullets older than 14 weeks of age or for commercial

layer hens. While antibiotics can be effective in reducing clinical disease, they do not eliminate carrier birds.

Prevention: Good management and sanitation are the best ways to avoid infectious coryza. Most outbreaks occur as a result of mixing flocks. All replacement birds on “coryza-endemic” farms should be vaccinated. The vaccine (Coryza-Vac) is administered subcutaneously (under the skin) on the back of the neck. Each chicken should be vaccinated four times, starting at 5 weeks of age with at least 4 weeks between injections. Vaccinate again at 10 months of age and twice yearly thereafter.

Infectious Laryngotracheitis

Synonyms: LT, ILT, trach, laryngo

Species affected: Chickens and pheasants are affected by LT. Chickens 14 weeks and older are more susceptible than young chickens. Most LT outbreaks occur in mature hens. In recent years, LT has also caused significant respiratory problems in broilers greater than 3 weeks of age, especially during the cooler seasons of the year. This is believed to be due to unwanted spread of LT vaccines between poultry flocks.

Clinical signs: The clinical sign usually first noticed is watery eyes. Affected birds remain quiet because breathing is difficult. Coughing, sneezing, and shaking of the head to dislodge exudate plugs in the windpipe follow. Birds extend their head and neck to facilitate breathing (commonly referred to as “pump handle respiration”). Inhalation produces a wheezing and gurgling sound. Blood-tinged exudates and serum clots are expelled from the trachea of affected birds. Many birds die from asphyxiation due to a blockage of the trachea when the tracheal plug is freed (see Table 1).

Transmission: LT is spread by the respiratory route. LT is also spread from flock to flock by contaminated clothing, shoes, tires, etc. Birds that recover should be considered carriers for life. LT may be harbored in speciality poultry such as exhibition birds and game fowl.

Treatment: Incinerate dead birds, administer antibiotics to control secondary infection, and vaccinate the flock. Mass vaccination by spray or drinking water method is not recommended for large commercial or caged flocks. Individual bird administration by the eye-drop route is suggested. Follow manufacturers instructions. In small poultry flocks, use a swab to remove plug from gasping birds, and vaccinate by eye-drop method.

Prevention: Vaccinate replacement birds for outbreak farms. Vaccination for LT is not as successful as for other disease, but is an excellent preventive measure for use in outbreaks and in epidemic areas. Refer to the publication PS-36 (Vaccination of Small Poultry Flocks) for more information on LT vaccinations.

Turkey Rhinotracheitis

Synonyms: TRT, rhino tracheitis

Species affected: Turkeys of all ages are susceptible, but the disease is most severe in young poults. Chickens are susceptible to the virus. Experimentally, guinea fowl and pheasants are susceptible, but waterfowl and pigeons are resistant.

Clinical signs: Respiratory signs in poults include snick-ing, rales, sneezing, nasal exudates (often frothy), foamy conjunctivitis, and sinusitis. Drops in egg production can be as much as 70 percent (see Table 1).

Transmission: Spread is primarily by contact with contaminated environments, feed and water, recovered birds, equipment, and personnel.

Treatment: No drugs are available to combat the virus. Antibiotic therapy is recommended to control secondary bacterial infections.

Prevention: No vaccines are currently available. Prevention is dependent on a comprehensive biosecurity program.

Chlamydiosis

Synonyms: ornithosis, psittacosis, parrot fever

The disease was called psittacosis or parrot fever when diagnosed in psittacine (curve-beaked) birds, and called ornithosis when diagnosed in all other birds or in humans. Currently, the term chlamydiosis is used to describe infections in any animal.

Species affected: Affected species include turkeys, pigeons, ducks, psittacine (curve-beaked) birds, captive and aviary birds, many other bird species, and other animals. Chickens are not commonly affected. Humans are susceptible, especially older and immunosuppressed individuals who are at a higher risk. Chlamydiosis in humans is an occupational disease of turkey growers, haulers, and processing workers in the live-bird areas and of workers in pet-bird aviaries although the incidence is rare. For more information, refer

to publication PS-23 (Avian Diseases Transmissible to Humans).

Clinical signs: Clinical signs in most birds include nasal-ocular discharge, conjunctivitis, sinusitis, diarrhea, weakness, loss of body weight, and a reduction in feed consumption. In turkeys there is also respiratory distress and loose yellow to greenish-yellow colored droppings. Chlamydiosis runs rather slowly through turkey flocks, with a maximum incidence of around 50 percent (see Table 1).

Transmission: The primary means of transmission is through inhalation of fecal dust and respiratory tract secretions. It can also be transmitted on contaminated clothing and equipment. Recovered birds remain carriers and will continue to intermittently shed the infective agent for long periods after clinical signs have subsided. Environmental stress may provoke a reoccurrence of the disease.

Treatment: Chlorotetracycline can be given in the feed (200–400 g/ton) for 3 weeks. Other antibiotics are usually ineffective. Recovered birds are safe for processing. Permanent lesions on the heart and liver are not infectious. FDA withdrawal periods for medications used must be strictly observed to avoid residual chemicals in the tissues.

Prevention: There is no vaccine. Have a good biosecurity program, excluding wild birds as much as possible.

Swollen Head Syndrome

Synonyms: Facial cellulitis, thick head, Dikkop, SHS

Species affected: Chickens and turkeys are the known natural hosts. Experimentally, guinea fowl and pheasants are susceptible but pigeons, ducks, and geese are resistant to the infection. SHS does not presently occur in the United States, but is present in most countries of the world.

Clinical signs: In chicks and poults, there is initial sneezing, followed by reddening and swelling of the tear ducts and eye tissue. Facial swelling will extend over the head and down the jaw and wattles. Adult chickens have mild respiratory disease followed by a few birds having swollen heads. Other signs include disorientation, twisting of the neck, and a significant drop in egg production (see Table 1).

Transmission: The infection spreads by direct contact with infected birds or indirectly by exposure to infectious material.

Treatment: There is no proven medication for swollen head syndrome. The disease is caused by a virus classified as a pneumovirus. A disease closely mimicking SHS is caused by a mixed infection of respiratory viruses and specific bacteria. Antibiotic therapy may be helpful against the bacterial component.

Prevention: A commercial vaccine is available. Swollen head syndrome is considered an exotic disease and a live vaccine is not approved for use in the United States.

Synonyms: MG, chronic respiratory disease (CRD), infectious sinusitis, mycoplasmosis

Species affected: chickens, turkeys, pigeons, ducks, peafowl, and passerine birds.

Clinical signs: Clinical symptoms vary slightly between species. Infected adult chickens may show no outward signs if infection is uncomplicated. However, sticky, serous exudate from nostrils, foamy exudate in eyes, and swollen sinuses can occur, especially in broilers. The air sacs may become infected. Infected birds can develop respiratory rales and sneeze. Affected birds are often stunted and unthrifty (see Table 1).

There are two forms of this disease in the turkey. With the “upper form” the birds have watery eyes and nostrils, the infraorbitals (just below the eye) become swollen, and the exudate becomes caseous and firm. The birds have respiratory rales and show unthriftiness.

With the “lower form”, infected turkeys develop airsacculitis. As with chickens, birds can show no outward signs if the infection is uncomplicated. Thus, the condition may go unnoticed until the birds are slaughtered and the typical lesions are seen. Birds with airsacculitis are condemned.

MG in chicken embryos can cause dwarfing, airsacculitis, and death.

Transmission: MG can be spread to offspring through the egg. Most commercial breeding flocks, however, are MG-free. Introduction of infected replacement birds can introduce the disease to MG-negative flocks. MG can also be spread by using MG-contaminated equipment.

Treatment: Outbreaks of MG can be controlled with the use of antibiotics. Erythromycin, tylosin, spectinomycin, and lincomycin all exhibit anti-mycoplasma activity and have given good results. Administration of most of these antibiotics can be by feed, water or injection. These are

effective in reducing clinical disease. However, birds remain carriers for life.

Prevention: Eradication is the best control of mycoplasma disease. The National Poultry Improvement Plan monitors all participating chicken and turkey breeder flocks.

Synonyms: MS, infectious synovitis, synovitis, silent air sac

Species affected: chickens and turkeys.

Clinical signs: Birds infected with the synovitis form show lameness, followed by lethargy, reluctance to move, swollen joints, stilted gait, loss of weight, and formation of breast blisters. Birds infected with the respiratory form exhibit respiratory distress. Greenish diarrhea is common in dying birds (see Table 1). Clinically, the disease is indistinguishable from MG.

Transmission: MS is transmitted from infected breeder to progeny via the egg. Within a flock, MS is spread by direct contact with infected birds as well as through airborne particles over short distances.

Treatment: Recovery is slow for both respiratory and synovitis forms. Several antibiotics are variably effective. The most effective are tylosin, erythromycin, spectinomycin, lincomycin, and chlorotetracycline. These antibiotics can be given by injection while some can be administered in the feed or drinking water. These treatments are most effective when the antibiotics are injected.

Prevention: Eradication is the best and only sure control. Do not use breeder replacements from flocks that have had MS. The National Poultry Improvement Plan monitors for MS.

Synonyms: MM, N strain, H strain

Species affected: MM affects turkeys of all ages, although poults are affected more severely than mature turkeys. Recently, MM has been shown to infect pigeon, quail and peafowl.

Clinical signs: A drop-off in production and hatchability can be expected in breeder flocks. There can be very high mortality in young poults. Unthriftiness, respiratory distress, stunting, crooked neck with deformity of cervical vertebrae, and leg deformation are common in young birds (see Table 1).

Transmission: Egg transmission is low in the early breeding period, but rises as the age of the flock increases. Infections can be introduced into a flock by contaminated equipment, shoes, and clothing of workers and visitors.

Treatment: Several antibiotics have been effective including tylosin, erythromycin, spectinomycin, and linco-spectinomycin.

Prevention: The best preventive measure is to keep MM-free breeders. The MM-free status of breeders can be confirmed by periodic blood tests through the National Poultry Improvement Plan.

Aspergillosis

Synonyms: brooder pneumonia, mycotic pneumonia, fungal pneumonia, *Aspergillus*. When the source of the disease is the hatchery, the disease is called brooder pneumonia. In older birds, the disease is called aspergillosis.

Species affected: All birds (domestic poultry, pigeons, canary and zoo bird species), animals, humans, and plants are susceptible.

Clinical signs: Aspergillosis occurs as an acute disease of young birds and a chronic disease in mature birds. Young birds have trouble breathing and gasp for air. Characteristically, there are no rales or respiratory sounds associated with aspergillosis. Feed consumption decreases. Occasionally there is paralysis or convulsions caused by the fungal toxin. Mortality in young birds averages 5–20 percent, but may be as high as 50 percent. Mature birds also have respiratory distress, reduced feed consumption, and may have a bluish and dark color of the skin (cyanosis). Nervous disorders, such as twisted necks, may occur in a few birds (see Table 1). Mortality in mature birds is usually less than 5 percent.

Transmission: Aspergillosis is caused by a fungus. The fungus grows well at room temperature and higher. All litter and nest materials (peat moss, peanut hulls, sawdust, peat, bark, straw) have been known to have been contaminated with aspergillus. Feed and water should be suspect when attempting to identify the source of contamination.

Treatment: There is no cure for infected birds. The spread can be controlled by improving ventilation, eliminating the source of the infection, and adding a fungistat (mycostatin, mold curb, sodium or calcium propionate, or gentian violet) to the feed and/or copper sulfate or acidified copper in the drinking water for 3 days. The litter can be sprayed

lightly with an oil-base germicide to control dust and air movement of fungal spores.

Prevention: It is important to thoroughly clean and disinfect the brooding area between broods. Use only clean litter, preferably soft wood shavings. Do not use sawdust, litter high in bark content, or shavings that have been wet.

Viral Diseases (nonrespiratory)

Marek's Disease

Synonyms: acute leukosis, neural leukosis, range paralysis, gray eye (when eye affected)

Species affected: Chickens between 12 to 25 weeks of age are most commonly clinically affected. Occasionally pheasants, quail, game fowl and turkeys can be infected.

Clinical signs: Marek's disease is a type of avian cancer. Tumors in nerves cause lameness and paralysis. Tumors can occur in the eyes and cause irregularly shaped pupils and blindness. Tumors of the liver, kidney, spleen, gonads, pancreas, proventriculus, lungs, muscles, and skin can cause incoordination, unthriftiness, paleness, weak labored breathing, and enlarged feather follicles. In terminal stages, the birds are emaciated with pale, scaly combs and greenish diarrhea (see Table 2).

Marek's disease is very similar to Lymphoid Leukosis, but Marek's usually occurs in chickens 12 to 25 weeks of age and Lymphoid Leukosis usually starts at 16 weeks of age.

Transmission: The Marek's virus is transmitted by air within the poultry house. It is in the feather dander, chicken house dust, feces and saliva. Infected birds carry the virus in their blood for life and are a source of infection for susceptible birds.

Treatment: none

Prevention: Chicks can be vaccinated at the hatchery. While the vaccination prevents tumor formation, it does not prevent infection by the virus.

Lymphoid Leukosis

Synonyms: visceral leukosis, leukosis, big liver, LL

Species affected: Although primarily a disease of chickens, lymphoid leukosis can infect turkeys, guinea fowl, pheasants, and doves, but not on a large scale.

Clinical signs: The virus involved has a long incubation period (4 months or longer). As a result, clinical signs are not noticeable until the birds are 16 weeks or older. Affected birds become progressively weaker and emaciated. There is regression of the comb. The abdomen becomes enlarged. Greenish diarrhea develops in terminal stages (see Table 2).

Transmission: The virus is transmitted through the egg to offspring. Within a flock, it is spread by bird-to-bird contact and by contact with contaminated environments. The virus is not spread by air. Infected chicken are carriers for life.

Treatment: none

Prevention: The virus is present in the yolk and egg white of eggs from infected hens. Most national and international layer breeders have eradicated lymphoid leukosis from their flocks. Most commercial chicks are lymphoid-leukosis negative because they are hatched from LL-free breeders. The disease is still common in broiler breeder flocks.

Infectious Bursal Disease

Synonyms: Gumboro, IBD, infectious bursitis, infectious avian nephrosis

Species affected: chickens

Clinical signs: In affected chickens greater than 3 weeks of age, there is usually a rapid onset of the disease with a sudden drop in feed and water consumption, watery droppings leading to soiling of feathers around the vent, and vent pecking. Feathers appear ruffled. Chicks are listless and sit in a hunched position. Chickens infected when less than 3 weeks of age do not develop clinical disease, but become severely and permanently immunosuppressed (see Table 2).

Transmission: The virus is spread by bird-to-bird contact, as well as by contact with contaminated people and equipment. The virus is shed in the bird droppings and can be spread by air on dust particles. Dead birds are a source of the virus and should be incinerated.

Treatment: There is no specific treatment. Antibiotics, sulfonamides, and nitrofurans have little or no effect. Vitamin-electrolyte therapy is helpful. High levels of tetracyclines are contraindicated because they tie up calcium, thereby producing rickets. Surviving chicks remain unthrifty and more susceptible to secondary infections because of immunosuppression.

Prevention: A vaccine is commercially available.

Equine Encephalitis

Synonyms: EE, EEE, WEE

Note: This disease should not be confused with St. Louis Encephalitis (SLE). Chickens are used as sentinels (test animals) in SLE suspect areas, such as southern Florida. While SLE is also carried by mosquitos, that is where the similarities between the two encephalitis diseases end. Chickens do not get SLE. Refer to Factsheet VM71 (St. Louis Encephalitis—The Role of Chickens) for more information on SLE.

Species affected: Equine encephalitis is a contagious disease of birds (especially pheasants), mammals (especially horses), and people. Birds are the major source of the virus.

Clinical signs: Two forms affect birds: eastern equine encephalitis (EEE) and western equine encephalitis (WEE). The clinical signs are identical and include reduced feed consumption, staggering, and paralysis. Surviving birds may be blind, have muscle paralysis, and have difficulty holding their head up. Damage to the bird's nervous system varies with species. In pheasants, there is pronounced leg paralysis, twisting of the neck, and tremors. Mortality is high. Chukar partridges and turkeys show drowsiness, paralysis, weakness, and death (see Table 2).

Transmission: Infected mosquitoes are the primary source of the virus. The *Culiseta melanuria* mosquito is the primary transmitter of the virus to poultry. Other mosquito species transmit the disease too, but feed mostly on other animals. Cannibalism of sick or dead birds by penmates is a major source of transmission within pens.

Treatment: none

Prevention: Remove the source of infection by establishing mosquito control: keep weeds mowed in a 50-foot strip around bird pens. This removes cover and resting areas for mosquitos. Eliminate mosquito breeding areas. Fog areas with malathion.

It is possible to immunize birds, especially pheasants, with the vaccine prepared for horses. The recommended dose is one-tenth of a horse dose per bird.

Avian Encephalomyelitis

Synonyms: epidemic tremor, AE

Species affected: The disease is most prevalent in chickens less than 6 weeks of age. Pheasants, corturnix quail, and

turkeys are natural hosts as well, but less susceptible than chickens. Ducklings, young pigeons, and guinea fowl can be experimentally infected.

Clinical signs: Signs commonly appear during the first week of life and between the second and third weeks. Affected chicks may first show a dull expression of the eyes, followed by progressive incoordination, sitting on hocks, tremors of the head and neck, and finally paralysis or prostration. Affected chicks are inactive. Some may refuse to walk or will walk on their hocks. In advanced cases, many chicks will lie with both feet out to one side (prostrate) and die. All stages (dullness, tremors, prostration) can usually be seen in an affected flock. Feed and water consumption decreases and the birds lose weight. In adult birds, a transitory drop (5–20 percent) in egg production may be the only clinical sign present. However, in breeding flocks, a corresponding decrease in hatchability is also noted as the virus is egg-transmitted until hens develop immunity. Chickens which survive the clinical disease may develop cataracts later in life (see Table 2).

Transmission: The virus can be transmitted through the egg from infected hen to chick, accounting for disease during the first week of life. The disease can also be spread through a flock by direct contact of susceptible hatchlings with infected birds, accounting for the disease at 2–3 weeks of age. Indirect spread can occur through fecal contamination of feed and water. Recovered birds are immune and do not spread the virus.

Treatment: There is no treatment for outbreaks. Infected birds should be removed, killed and incinerated. Recovered chicks are unthrifty.

Prevention: A vaccine is available.

Egg Drop Syndrome

Synonyms: egg drop, egg drop syndrome 76, EDS-76

Species affected: The natural hosts for EDS virus are ducks and geese, but EDS has become a major cause of reduced egg production in chickens in many parts of the world. No illness has been observed in ducks or geese. Chickens of all ages and breeds are susceptible. The disease is most severe in broiler-breeders and brown-egg layer strains.

Clinical signs: There are no reliable signs other than the effects on egg production and egg quality. Healthy-appearing hens start laying thin-shelled and shell-less eggs. Once established, the condition results in a failure to achieve egg production targets. Transient diarrhea and dullness occur

prior to egg shell changes. Fertility and hatchability are not affected (see Table 2).

Transmission: It is believed that the syndrome was first introduced into chickens from contaminated vaccine. Vertical transmission occurs from infected breeders to chicks. Newly hatched chicks excrete the virus in the feces.

Treatment: There is no successful treatment. Induced molting will restore egg production.

Prevention: Prevention involves a good biosecurity program.

Infectious Tenosynovitis

Synonyms: viral arthritis, tenosynovitis, teno, reovirus enteritis, reovirus septicemia, malabsorption syndrome, helicopter disease

Species affected: turkeys and chickens

Clinical signs: Several serotypes of the reovirus have been identified. Some localize in the joints (tenosynovitis) while others target respiratory or intestinal tissues (septicemic form) (see Table 2).

The principal sign of tenosynovitis is lameness with swelling of the tendon sheaths of the shank and area extending above the hock (see Table 2). Affected birds are lame, sit on their hocks, and are reluctant to move. Rupture of the tendon can occur in older roaster birds, resulting in permanent lameness of the affected leg. If more than two joints are affected, the entire carcass will be condemned.

Infection can also play a part in broiler stunting, the result of malabsorption syndrome. In chicks, malabsorption due to viral enteritis is called “helicopter disease” because feathering is affected. Wing feathers protrude at various angles. A reovirus is believed to play only a secondary role in this syndrome.

In commercial layer flocks, increased mortality may be the first sign of the septicemia form (see Table 2). Egg production will decrease by about two to three times the mortality rate. For example, a mortality rate of 5 percent will be accompanied by a 10–15 percent drop in egg production. In the septicemic form, joint involvement is present but less pronounced. Affected birds become cyanotic (blue) and dehydrated. The tips of the comb turn purplish. The entire comb darkens as the disease progresses (see Table 2).

Transmission: The infection spreads rapidly through broiler flocks, but less rapidly in caged layers. Spread is by respiratory and digestive tract routes. The virus is shed in the feces.

Treatment: There is no satisfactory treatment available. With hens, tetracycline, molasses, and oyster shell therapy is helpful.

Prevention: A vaccine is available for use in endemic areas or on endemic farms.

Nonrespiratory Bacterial Diseases

Fowl Cholera

Synonyms: avian pasteurellosis, cholera, avian hemorrhagic septicemia

Species affected: Domestic fowl of all species (primarily turkeys and chickens), game birds (especially pheasants and ducks), cage birds, wild birds, and birds in zoological collections and aviaries are susceptible.

Clinical signs: Fowl cholera usually strikes birds older than 6 weeks of age. In acute outbreaks, dead birds may be the first sign. Fever, reduced feed consumption, mucoid discharge from the mouth, ruffled feathers, diarrhea, and labored breathing may be seen. As the disease progresses birds lose weight, become lame from joint infections, and develop rattling noises from exudate in air passages. As fowl cholera becomes chronic, chickens develop abscessed wattles and swollen joints and foot pads. Caseous exudate may form in the sinuses around the eyes. Turkeys may have twisted necks (see Table 3).

Transmission: Multiple means of transmission have been demonstrated. Flock additions, free-flying birds, infected premises, predators, and rodents are all possibilities.

Treatment: A flock can be medicated with a sulfa drug (sulfonamides, especially sulfadimethoxine, sulfaquinonxalene, sulfamethazine, and sulfaquinoxalene) or vaccinated, or both, to stop mortality associated with an outbreak. It must be noted, however, that sulfa drugs are not FDA approved for use in pullets older than 14 weeks or for commercial laying hens. Sulfa drugs leave residues in meat and eggs. Antibiotics can be used, but require higher levels and long term medication to stop the outbreak.

Prevention: On fowl cholera endemic farms, vaccination is advisable. Do not vaccinate for fowl cholera unless you

have a problem on the farm. Rodent control is essential to prevent future outbreaks.

Omphalitis

Synonyms: navel ill, mushy chick disease

Species affected: chickens

Clinical signs: Affected chicks may have external navel infection, large unabsorbed yolk sacs, peritonitis with fetid odor, exudates adhering to the navel, edema of the skin of ventral body area, septicemia and dehydration (see Table 3).

Transmission: Infection occurs at the time of hatching or shortly thereafter, before navels are healed. Chicks from dirty hatching eggs or eggs with poor quality shells, or newly hatched chicks placed in dirty holding boxes, are most susceptible. Chicks removed prior to complete healing of the navel due to improper temperature and/or humidity are also more susceptible. Eggs that explode in the hatching tray contaminate other eggs in the tray and increase the incidence.

Treatment: There is no specific treatment for omphalitis. Most affected birds die in the first few days of life. Unaffected birds need no medication.

Prevention: Control is by prevention through effective hatchery sanitation, hatchery procedures, breeder flock surveillance, and proper preincubation handling of eggs. Mushy chicks should be culled from the hatch and destroyed. If chick mortality exceeds 3 percent, the breeder flocks and egg handling and hatching procedures should be reviewed.

Pullorum

Synonyms: bacillary white diarrhea, BWD

Species affected: Chickens and turkeys are most susceptible, although other species of birds can become infected. Pullorum has never been a problem in commercially grown game birds such as pheasant, chukar partridge, and quail. Infection in mammals is rare.

Clinical signs: Death of infected chicks or poults begins at 5–7 days of age and peaks in another 4–5 days. Clinical signs including huddling, droopiness, diarrhea, weakness, pasted vent, gasping, and chalk-white feces, sometimes stained with green bile. Affected birds are unthrifty and stunted because they do not eat (see Table 3). Survivors

become asymptomatic carriers with localized infection in the ovary.

Transmission: Pullorum is spread primarily through the egg, from hen to chick. It can spread further by contaminated incubators, hatchers, chick boxes, houses, equipment, poultry by-product feedstuffs, and carrier birds.

Treatment: Treatment is for flock salvage only. Several sulfonamides, antibiotics, and antibacterials are effective in reducing mortality, but none eradicates the disease from the flock. **Pullorum eradication is required by law.** Eradication requires destroying the entire flock.

Prevention: Pullorum outbreaks are handled, on an eradication basis, by state/federal regulatory agencies. As part of the National Poultry Improvement Program, breeder replacement flocks are tested before onset of production to assure pullorum-free status. This mandatory law includes chickens, turkeys, show birds, waterfowl, game birds, and guinea fowl. In Florida, a negative pullorum test or certification that the bird originated from a pullorum-free flock is required for admission for exhibit at shows and fairs. Such requirements have been beneficial in locating pullorum-infected flocks of hobby chickens.

Necrotic Enteritis

Synonyms: enterotoxemia, rot gut

Species affected: Rapidly growing young birds, especially chickens and turkeys 2–12 weeks of age, are most susceptible. Necrotic enteritis is a disease associated with domestication and is unlikely to threaten wild bird populations. Necrotic enteritis is primarily a disease of broilers, roasters and turkeys. Ulcerative enteritis, on the other hand, commonly affects pullets and quail.

Clinical signs: Initially there is a reduction in feed consumption as well as dark, often blood-stained, feces. Infected chickens will have diarrhea. Chronically affected birds become emaciated. The bird, intestines, and feces emit a fetid odor (see Table 3).

Transmission: Necrotic enteritis does not spread directly from bird to bird. Bacteria are ingested along with infected soil, feces, or other infected materials. The bacteria then grow in the intestinal tract. Infection commonly occurs in crowded flocks, immuno-suppressed flocks, and flocks maintained in poor sanitary conditions.

Treatment: The clostridia bacteria involved in necrotic enteritis is sensitive to the antibiotics bacitracin, neomycin,

and tetracycline. However, antibiotics such as penicillin, streptomycin, and novobiocin are also effective. Bacitracin is the most commonly used drug for control of necrotic enteritis. As with all drugs, legality and withdrawal time requirements must be observed.

Prevention: Prevention should be directed toward sanitation, husbandry, and management.

Ulcerative Enteritis

Synonyms: quail disease

Species affected: Captive quail are extremely susceptible and must be maintained on wire-bottom pens or on preventive medications. Chickens, turkeys, partridges, grouse, and other species are occasionally clinically affected.

Clinical signs: In quail, the disease is acute with high mortality. In chickens, signs are less dramatic. Acute signs are extreme depression and reduction in feed consumption. Affected birds sit humped with eyes closed. Other signs included emaciation, watery droppings streaked with urates, and dull ruffled feathers (see Table 3). Accumulated mortality will reach 50 percent if the flock is not treated.

Transmission: Birds become infected by direct contact with carrier birds, infected droppings or contaminated pens, feed and water. Bacteria are passed in the droppings of sick and carrier birds. Infection can be spread mechanically on shoes, feed bags, equipment, and from contamination by rodents and pets.

Treatment: Bacitracin and neomycin can be used singly or in combination. Other antibiotics and drugs such as tetracyclines, penicillin, Lincomycin, and Virginomycin are also effective. Consult a veterinarian for dose, route, and duration of treatment.

Prevention: Ulcerative enteritis is difficult to prevent in quail. When quail have access to their own droppings, this disease commonly occurs. To eradicate, depopulate stock, thoroughly clean and disinfect, and start over with young, clean stock.

Botulism

Synonyms: limberneck, bulbar paralysis, western duck sickness, alkali disease

Species affected: All fowl of any age, humans, and other animals are highly susceptible. The turkey vulture is the only animal host known to be resistant to the disease.

Clinical signs: Botulism is a poisoning causing by eating spoiled food containing a neurotoxin produced by the bacterium *Clostridium botulinum*. Paralysis, the most common clinical sign, occurs within a few hours after poisoned food is eaten. Pheasants with botulism remain alert, but paralyzed. Legs and wings become paralyzed, then the neck becomes limp. Neck feathers become loose in the follicle and can be pulled easily (see Table 3).

If the amount eaten is lethal, prostration and death follow in 12 to 24 hours. Death is a result of paralysis of respiratory muscles. Fowl affected by sublethal doses become dull and sleepy.

Transmission: Botulism is common in wild ducks and is a frequent killer of waterfowl because the organisms multiply in dead fish and decaying vegetation along shorelines.

Decaying bird carcasses on poultry ranges, wet litter or other organic matter, and fly maggots from decaying substances may harbor botulism. There is no spread from bird to bird.

Treatment: Remove spoiled feed or decaying matter. Flush the flock with Epsom salts (1 lb/1000 hens) in water or in wet mash. It has been reported that potassium permanganate (1:3000) in the drinking water is helpful. Affected birds can be treated with botulism antitoxin injections.

Prevention: Incinerate or bury dead birds promptly. Do not feed spoiled canned vegetables. Control flies. Replace suspected feed.

Staphylococcus

Synonyms: staph infection, staph septicemia, staph arthritis, bumblefoot

Species affected: All fowl, especially turkeys, chickens, game birds, and waterfowl, are susceptible.

Clinical signs: Staphylococcal infections appear in three forms—septicemia (acute), arthritic (chronic), and bumblefoot. The septicemia form appears similar to fowl cholera in that the birds are listless, without appetite, feverish, and show pain during movement. Black rot may show up in eggs (the organism is passed in the egg). Infected birds pass fetid watery diarrhea. Many will have swollen joints (arthritis) and production drops (see Table 3).

The arthritic form follows the acute form. Birds show symptoms of lameness and breast blisters, as well as painful

movement (see Table 3). Birds are reluctant to walk, preferring to sit rather than stand.

Bumblefoot is a localized chronic staph infection of the foot, thought to be caused by puncture injuries. The bird becomes lame from swollen foot pads (see Table 3).

Transmission: *Staphylococcus aureus* is soil-borne and outbreaks in flocks often occur after storms when birds on range drink from stagnant rain pools.

Treatment: Novobiocin (350 g/ton) can be given in the feed for 5–7 days. Erythromycin and penicillin can be administered in the water for 3–5 days or in the feed (200 g/ton) for 5 days. Other antibiotics and drugs are only occasionally effective.

Prevention: Remove objects that cause injury. Isolate chronically affected birds. Provide nutritionally balanced feed.

Table 1. Possible clinical signs for common respiratory diseases of poultry.

Clinical signs	pox ¹	Newcastle ²	IB ³	Quail Bronchitis	AI ⁴	coryza ⁵	LT ⁶	TRT ⁷	Chlamydiosis	SHS ⁸	MG ⁹	MS ¹⁰	MM ¹¹	Aspergillosis
Coughing	X	X		X	X		X		X	X	X		X	
Sneezing	X	X		X	X	X	X		X	X	X	X	X	
Shaking head		X		X		X	X		X		X	X		
Rales (abnormal breathing sound)	X	X	X	X	X	X	X	X	X	X	X	X	X	
Gasping	X	X	X	X			X		X	X	X			X
Discharge from eyes	X	X	X	X		X	X		X	X	X	X		
Nasal discharge		X	X	X		X	X		X	X	X	X		
Swelling of face and/or wattles		X		X	X	X	X	X	X	X	X	X		
Bluish-purple discoloration of face		X		X	X					X				X
Retarded growth	X	X		X		X	X		X		X	X	X	
Lameness												X		
General diarrhea		X		X	X	X	X		X		X			
Green, watery diarrhea		X		X					X		X	X		
Swollen joints												X		
Paralysis														X
Twisting of head and neck		X		X						X			X	X
Red/white spots on legs and comb					X									
Warts/scabs	X													
Conjunctivitis		X		X	X	X	X	X	X	X				
Prostration		X		X			X				X			

¹Fowl Pox
²Newcastle disease
³Infectious bronchitis
⁴Avian influenza
⁵Infectious coryza
⁶Laryngotracheitis
⁷Turkey rhinotracheitis
⁸Swollen head syndrome
⁹*Mycoplasma gallisepticum*
¹⁰*Mycoplasma synoviae*
¹¹*Mycoplasma meleagridis*

Table 2. Possible clinical signs for common nonrespiratory viral diseases of poultry.

Clinical Signs	Marek's disease	Lymphoid leukosis	Infectious bursal disease	Equine encephalitis	Avian encephalitis	Egg drop syndrome	Infectious tenosynovitis	Septicemia form
Reduced feed consumption			X	X	X			
Labored breathing	X							
Weight loss/stunted growth					X			X
Reduced water consumption			X		X			
Bluish-purple discoloration of the face								X
Enlarged abdomen		X						
Lameness	X						X	
Swollen joints							X	
Twisted necks				X				
Paralysis	X			X	X			
Inactive					X			
Tremors				X				
Incoordination	X			X	X			
Blindness	X							
Paleness	X							
Pale scaly combs	X							
Greenish diarrhea	X	X						X
Diarrhea (general)				X		X		
Watery droppings			X					
Thin-shelled eggs						X		
Shell-less eggs						X		
Reduced egg production						X		X
Increased mortality								X
Dullness						X		
Weakness		X						
Emaciation		X						
Helicopter wings								X
Ruffled feathers			X					
Small comb		X						

Table 3. Possible clinical signs of nonrespiratory bacterial diseases of poultry.

Clinical signs	Fowl cholera	Omphalitis	Pullorum	Necrotic enteritis	Ulcerative enteritis	Botulism	Staphylococcus
Dead birds, no signs of disease	X						
Fever	X						X
Reduced feed consumption	X			X	X		X
Discharge from mouth	X						
Ruffled feathers	X				X		
Labored breathing	X		X				
Weight loss/stunted growth	X		X	X	X		
Lameness	X						X
Swollen joints	X						X
Abscessed wattles	X						
Swollen foot pads	X						X
Twisted necks	X						
Navel infection		X					
Dehydration		X					
Huddling of chicks			X		X		
Droopiness			X				
Diarrhea/pasted vent	X		X	X			X
White feces			X		X		
Blood in feces				X			
Paralysis						X	
Cyanotic							
Foul odor				X			X