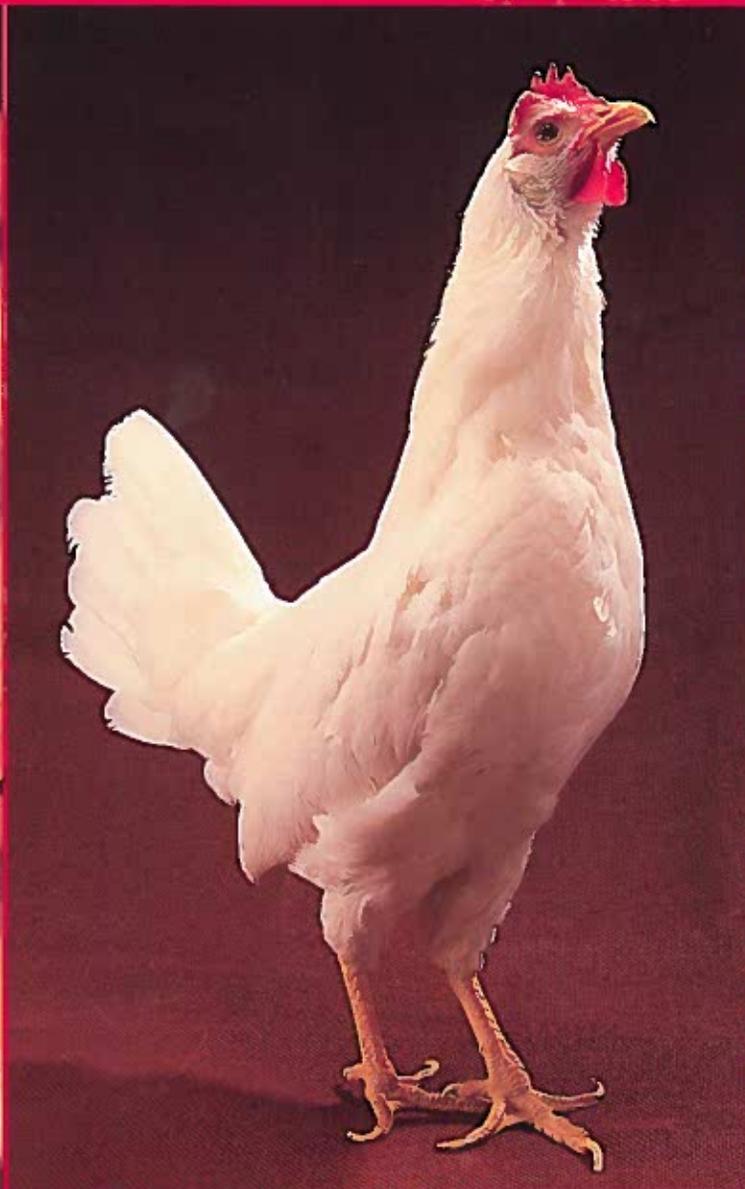
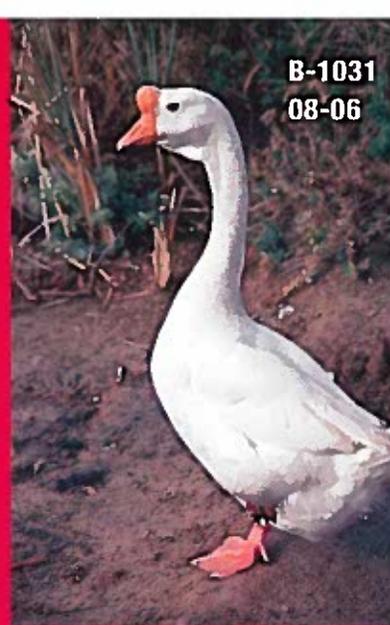


POULTRY DISEASE MANUAL

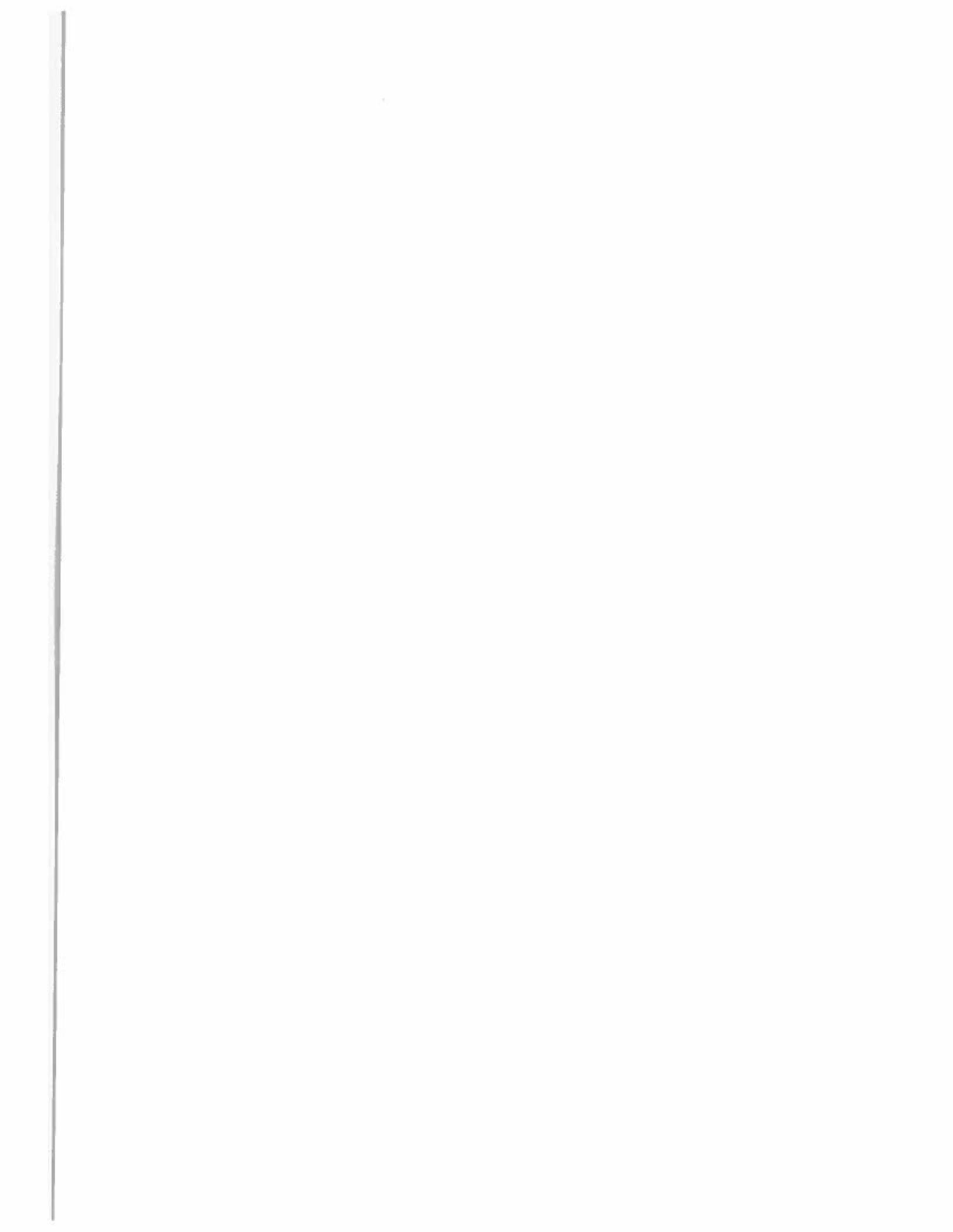
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POULTRY DISEASE MANUAL

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This manual is intended for use by poultry growers (backyard or commercial), veterinarians, poultry service technicians, Extension agents, and others as a reference for identifying and treating certain poultry diseases. Remember that most drugs must be prescribed by a veterinarian and that you should always follow the dosage recommendations.

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The poultry industry in Texas contributes more than \$1.6 billion to the state economy each year. A survey of broiler, egg and turkey producers revealed that, in 2003, the Texas poultry industry produced 4.7 billion table eggs and 615.6 million broilers and turkeys and employed more than 11,560 people. Texas currently ranks sixth in broiler production and seventh in egg production in the U. S.

The disease level of a poultry flock may determine the profitability of the operation. Disease may cause obvious losses such as the death of birds, condemnation in the processing plant, and medication costs, or less obvious but substantial losses from poor growth, poor feed conversion and downgrading.

Nature and Cause of Disease

Diseases have indirect and direct causes; both usually are present when a disease occurs. Indirect causes are conditions that put an animal under stress and lower its resistance to disease. Stress factors include improper temperature, overcrowding, inadequate feed and/or water, overmedication and others. A disease itself may be a predisposing factor, as the onset of one disease will lower a bird's immunity to another disease.

Direct causes of disease are:

- Bacteria
- Viruses
- Parasites
- Fungi
- Nutritional deficiencies
- Chemical poisons

- Overmedication
- Unknown causes

Infectious diseases—those caused by bacteria, viruses or fungi—are the greatest threat to a poultry flock. An infectious disease is a disease caused by living organisms. A contagious disease is one that is readily passed from one individual or host to another. All contagious diseases are considered infectious, but not all infectious diseases are considered contagious. Some protozoan diseases behave much like infectious diseases but are considered parasitic.

When living disease agents enter the body of the host and multiply, they disturb the body's functions and usually cause infection. The disease itself is caused by the toxins or poisons the



infecting agent produces or, in the case of viruses, the injury to the cells and DNA the infecting agent causes. Protozoan diseases also cause mechanical damage to the cells and tissues of the body. It is the combination of mechanical and chemical damage (mostly chemical) that causes the symptoms of the disease.

The ability of an organism to cause disease in a host animal is defined as its **pathogenicity** or **virulence**. Some microorganisms are unlikely to cause disease unless the bird has been exposed to stress factors; these microorganisms are considered pathogenic under certain conditions. Other microorganisms are almost always pathogenic in a specific class or species of bird or animal and are considered to be **specific pathogens** to that group. Some microorganisms tend to be pathogenic among a large variety of classes and species and may even infect humans.

Spread of Infectious Diseases

Infectious diseases can be transmitted by:

- The introduction of diseased birds into a flock
- The introduction of healthy birds that have recovered from a disease but that are still carriers of the disease-causing agent
- Contact with inanimate objects (fomites) that are contaminated with disease-causing agents
- Exposure to poultry carcasses that have not been disposed of properly
- Impure water sources
- Rodents and wild birds
- Insects
- Shoes and clothing of persons moving from flock to flock
- Contaminated feed and equipment

(bags, trucks, hoppers, etc.)

- Contaminated litter
- Airborne infectious agents (most agents cannot be spread long distances in this way)
- A hen passing a disease to eggs and chicks

Immunity and Defense against Disease

Living organisms such as poultry have two types of defenses against disease: 1) natural barriers that impede or prevent the entry of the disease agent into the body; and 2) organs and cells within the body (the immune system) that combat disease agents.

Natural barriers to disease include:

- Intact skin—most microbes cannot pass through intact skin
- Mucous membranes—they form a direct barrier and emit secretions that dilute and wash out infecting agents
- Cilia—these hair-like projections in some mucous membranes trap and remove foreign material

Immunity is the ability to resist and/or overcome harmful agents. The immune system is made up of specialized cells and their products. Most of the functions of the immune system are performed by lymphocytes, their products, or other cells recruited by lymphocytes. The two major types of lymphocytes are **bursa-dependent** or **B-lymphocytes** and **thymus-dependent** or **T-lymphocytes**. Each has a different function.

Newly formed lymphocytes pass through either the **Bursa of Fabricius** or the **thymus gland** for

maturation. These immature lymphocytes are acted on by chemicals of the bursa or the thymus and will eventually be involved in either antibody production (B-lymphocytes) or cell-mediated immunity (T-lymphocytes). The mature lymphocytes are then seeded throughout various body tissues where they divide and produce more lymphocytes like themselves. These new lymphocytes do not need to go through the bursa or the thymus to become mature. Damage to the bursa or the thymus in the early life of the chick will prevent the initial lymphocytes from maturing and thus suppress the bird's immunity. This is why infectious bursal disease (IBD) in young chicks produces immunosuppression, while the clinical form of IBD in older birds does not.

B-lymphocytes produce **antibodies**, which are specific defenses against diseases. This means that antibodies produced in response to Newcastle disease will not affect the virus that causes infectious bronchitis and vice versa. Antibodies are released into the blood plasma and into mucus produced by the intestinal and respiratory tracts. Antibodies in the blood are easy to measure as indicators of immunity and response to vaccines. Antibodies in the mucus of the intestinal and respiratory tracts are not readily measured and are called local antibodies. These local antibodies are usually the first line of defense because many disease-causing agents enter the body via ingestion or respiration. If there are enough of these antibodies they will greatly reduce the number of disease agents the blood plasma antibodies will have to combat once the agents reach the circulatory system.

T-lymphocytes do not produce antibodies. These cells produce chemicals called **lymphokines** that influence other cells in the

immune system to attack a disease agent. The principal cell lymphokines affect is the macrophage. **Macrophages** are scavenger cells that engulf incoming disease agents and destroy them. T-lymphocyte immunity is also referred to as **cell-mediated immunity**. This cell-mediated immunity is especially effective against disease agents that have invaded cells, particularly bacteria, fungi and tumors. Antibodies cannot usually provide defense at the cellular level.

When a disease agent, or antigen, enters the body, it stimulates both antibody (B) and cell-mediated (T) immune responses. Depending on the nature of the antigen and its location within the body, one type of response may be stronger than the other.

Disease resistance is either **innate** or **acquired**. Innate, or inherited, resistance protects a species, breed or individual from infection by a particular antigen. This type of resistance can be either complete or partial. Turkeys have complete innate immunity to laryngotracheitis. Chickens are more resistant to blackhead than turkeys, but can become infected if conditions are right, so they are considered to have partial innate immunity to blackhead.

Acquired immunity comes through vaccination. The antigen (often killed) is introduced via the vaccine and the animal produces antibodies specific to the antigen. If the animal is later infected by a live antigen, the antibodies will identify it and attack it before it can cause disease.

Another type of immunity is **passive immunity** or the transfer of antibodies from one individual to another. This can be done by transferring serum from an immunized individual to an unimmunized individual. Passive immunity also occurs from hen to egg to chick. Passive immu-

nity is short lived, usually lasting just 3 to 6 weeks, so it isn't a good idea to rely on it to protect birds older than that.

Disease Manifestation

Most diseases produce **symptoms** and **lesions**. Symptoms are detectable signs of a disease and can be either general or specific. Examples of general symptoms are droopiness, decreased egg production, lameness, diarrhea, ruffled feathers and loss of appetite. An example of a specific symptom is the flaccid paralysis associated with botulism. Lesions are visible changes in the size, color, shape or structure of an organ. Lesions also can be general or specific. General lesions include enlarged liver, tumors in the intestines, and exudates. An example of a specific lesion is "gray eye," a distortion of the pupil associated with ocular leucosis.



Sanitation Practices for Flock Health

The word sanitation is often overused and misunderstood in the poultry industry. Most people believe sanitation is a catch-all that will prevent all diseases. This is not the case. Sanitation practices that prevent one disease may, in fact, create conditions in which another disease can flourish. Many sanitation procedures should always be applied, while others should be applied only when certain diseases are a problem.

These basic principles of sanitation should always be followed:

- Purchase chicks, poults and hatching eggs only from well-known, reliable sources. The supplier should have a good record of providing healthy stock, developed for a specific purpose.
- Purchase only day-old chicks or hatching eggs. If it is necessary to purchase started birds, select a reliable source.
- Keep birds separate according to source and age.
- Follow an “all-in, all-out” placement program.
- Change the litter and thoroughly clean

and disinfect the house and all equipment between flocks.

- Keep different species of birds separate (e.g., do not mix chickens and turkeys).
- Hatchery supply flocks should be on separate premises from birds grown for market.
- Select a reliable commercial feed. If feed is mixed on the farm, follow a dependable formula.
- Provide an adequate source of drinking water. Do not use surface water such as water from ponds and streams.
- Use a precise vaccination schedule for each flock. The vaccination program should be worked out with the proper poultry disease authorities in your area.
- Do not allow anyone other than the caretaker of the birds to visit the flock or house.
- If disease develops, obtain a reliable diagnosis and perform any eradication measures required for that disease.
- Dispose of dead birds by incinerating or burying them.
- Keep good records on the flock, includ-

ing the vaccination history, medication history, and records of past disease problems.

The National Poultry Improvement Plan (a cooperative industry/state/federal program) recommends these practices for hatching egg sanitation:

- Collect hatching eggs from nests or gathering belts at frequent intervals.
- Use cleaned and disinfected containers to collect the eggs. Be careful not to contaminate them with organisms that may be on hands or clothing.
- Identify all eggs as to breeder flock of origin.
- Do not use dirty eggs as hatching eggs. Collect dirty eggs in a separate container. Eggs that are slightly soiled can be hand cleaned.
- Fumigate the eggs as soon as possible after collection.
- Place sanitized eggs in either new or properly sanitized flats or racks.
- Store eggs at 55 to 70 degrees F. Keep humidity at appropriate levels in storage areas.
- Store eggs for as short a time as possible before setting.
- Clean and sanitize the egg processing area daily. Keep airborne debris, such as dust and feathers, to a minimum. If ink stamps and pads are used, they should be sterile.
- The building that houses the egg processing area should be constructed of materials that are easy to clean and should be located in such a way that cleaning and sanitation are easily performed.
- Walk-in coolers should be cleaned and

sanitized frequently. They also can be fumigated when empty.

- Trucks used to collect eggs should be kept reasonably clean of debris and fumigated whenever possible.
- Use new, or acceptably clean, cases for packing and transportation.

Use the following sanitation practices in the hatchery to prevent and control *Salmonella spp.* and other microorganisms:

- Arrange the hatchery buildings so that there are separate rooms with separate ventilation systems for each of the four main hatchery operations:
 - 1) egg receiving,
 - 2) incubation and hatching,
 - 3) chick holding, and
 - 4) offal disposal and tray cleaning.
- Allow only authorized personnel, who have taken precautions to prevent cross-contamination and introduction of disease, to enter the hatchery.
- Clean and disinfect hatchery rooms, tables, racks and all other equipment daily. Dispose of hatchery waste and offal properly.
- Clean and disinfect hatching trays after each hatch.
- Keep eggs used for hatching clean and free of debris. All eggs should be fumigated either before they are set or within 12 hours of being placed in the incubator.
- Cases used for transporting hatching eggs should be new or clean.
- Day-old chicks should be distributed in new or clean chick boxes or plastic chick delivery trays. Then discard paper linings used to collect chick litter after placement.

- Identify all chicks as to the breeder flock of origin.
- Do not mix the progeny of different breeder flocks unless it is absolutely necessary.

To clean and disinfect poultry houses, egg holding rooms and hatchery rooms:

- Settle dust by spraying lightly with disinfectant.
- Remove all litter and droppings to a location where any microorganisms or disease-causing agents they may contain cannot possibly be disseminated.
- Clean walls, floors and equipment with an approved cleaning agent. Then inspect carefully to make sure these areas are clean before disinfecting them. Disinfect with an approved disinfectant.

To clean and disinfect hatchers:

- Remove trays, controls and fans from the hatchers (if possible) and clean them separately. Scrub the ceiling, walls and floor of the hatcher with a cleaning agent and a hard-bristled brush. Rinse the hatcher thoroughly until no deposits are visible. Pay special attention to the fan opening.
- Replace trays, controls and fans and bring the incubator up to operating temperature.
- Fumigate the hatcher before eggs are transferred.
- If eggs are incubated and hatched in the same machine, cleaning should be done after each hatch.

The fumigation of eggs and incubators is an essential part of a complete hatchery sanitation program. (See Table 1 for concentrations of

formaldehyde gas for specific equipment and areas).

To fumigate eggs before incubation:

- Select a room or cabinet proportionate in size to the number of eggs to be fumigated. The room (cabinet) should be relatively airtight and equipped with a fan to circulate the gas during fumigation.
- Place the eggs in the room on wire racks so gas can circulate around them.
- Circulate the gas within the room for 20 minutes, then expel it. While humidity is not critical, the temperature of the room should be approximately 70 degrees F. If the air is particularly dry, extra humidity may be added.
- Formaldehyde gas used for fumigation is produced by mixing 0.6 gram of potassium permanganate ($KMnO_4$) with 1.2 ml (cc) of formalin (37.5% formaldehyde) for each cubic foot of space to be fumigated.

Eggs that have not been fumigated before setting should be fumigated as soon as possible (no more than 12 hours) after setting.

Fumigating between 12 and 96 hours after setting in the incubator can kill the developing embryos.

To fumigate eggs after setting:

- Determine the volume of the incubator (length x width x height).
- After setting, allow the temperature and humidity levels to reach normal operating levels, then release the fumigant into the incubator.
- Close vents and doors, but keep a fan operating to circulate the gas. Continue

the fumigation for 20 minutes at normal operating temperature and humidity.

- After 20 minutes, open vents to normal operating positions to allow the gas to disperse.

Eggs should be refumigated after they are transferred to the hatcher, once temperature and humidity have reached normal operating levels.

Table 1. Fumigation concentrations.

Fumigation target	Concentration	Time (in minutes)
Hatching eggs (after lay, before set)	3X	20
Eggs in setter	1X or 2X	20
Chicks in hatcher	1X	3
Incubator room	1X or 2X	30
Hatcher (between hatches)	3X	30
Hatcher room, chick room, walk-in coolers (between hatches)	3X	30
Wash room	3X	30
Chick boxes, clean pads	3X	30
Trucks	5X	20

NOTE: Texas Cooperative Extension is not liable for failure on the part of the user to adhere to the Occupational Safety and Health Administration (OSHA) standards for formaldehyde fumigation, published in the Dec. 4, 1987 Federal Register (52 FR 46168, Docket Nos. H-225, 225-A, and 225-B).



Bacterial Diseases (non-respiratory)

Botulism

Cause. Botulism is caused by the ingestion of a neurotoxin produced by the bacterium *Clostridium botulinum*. *C. botulinum* is commonly found in the environment and in the gastrointestinal tract of many animals, and is not in itself considered pathogenic. However, under certain conditions this organism can produce a highly potent neurotoxin that is generally heat resistant. There are different types of botulinum toxin. Types A and C usually affect birds, while type B usually affects humans. Botulism is also called limberneck, bulbar paralysis, western duck sickness and alkali disease.

Susceptibility. All animals of any age are susceptible to botulism toxins. The turkey vulture is the only animal known to be resistant.

Transmission. Chickens usually ingest the toxin with feed. The disease does not spread between live birds. Wild ducks are usually exposed to the toxin in shallow water areas where the water has a high pH and contains decaying vegetation. Storing feed properly virtually eliminates the risk of botulism in commercially reared and backyard chickens.

Clinical signs. Signs of botulism occur within a few hours of ingesting the toxin. The main symptom is paralysis that usually begins at the extremities and progresses toward the trunk of the body. Lethal doses cause death within 12 to 24 hours. If the dose is sub-lethal, the bird will seem listless and sleepy. In pheasants, however, both lethal and sub-lethal doses cause birds to be alert, but paralyzed.

Lesions. There are no visible lesions.

Diagnosis. Botulism is diagnosed from clinical signs, post-mortem laboratory results, or the recovery of birds given botulism antitoxin. A history of botulism in the area is a diagnostic clue.

Treatment. Remove the source of the toxin (i.e., spoiled feed or decaying plant or animal matter). Administer epsom salts in the water system (1 pound per 1,000 birds) to flush the flock. Treat affected birds with botulism antitoxin (intravenously, intramuscularly or subcutaneously). Repeat injections after 12 hours if necessary.

Gangrenous Dermatitis

Cause. Many types of bacteria have been isolated from birds with gangrenous dermatitis, including *Clostridium novyi*, *C. perfringens*, *C. septicum*, *E. coli* and *Staphylococcus aureus*. *C. septicum* and *S. aureus* are found most often and these organisms may have a symbiotic relationship to cause this disease. Other factors that may contribute to the onset of gangrenous dermatitis include bursal damage from infectious bursal disease (IBD), adenoviral infection, vitamin E and selenium deficiencies, and hemorrhagic syndrome. Other names for this disease are necrotic dermatitis and wing rot.

Susceptibility. Chickens and turkeys from 2½ to 16 weeks old are susceptible. Younger birds may have poor feathering. Outbreaks often occur in houses that are excessively warm and humid.

Transmission. This disease is not spread directly from bird to bird. Rather, lesions on the skin caused by trauma are invaded by the bacteria, which is often found in soil, feces and dust. Proper sanitation of the house and floor will dramatically decrease the number of bacteria and the likelihood of this disease occurring.

Clinical signs. When first infected, birds appear depressed and lethargic and may be prostrate. There may be edema at the wingtips, wattles and feet. Birds will not eat or drink and become dehydrated. There may also be a fetid or putrid odor from lesions. Infected birds usually die within 8 to 24 hours. Mortality ranges from 10 percent to 60 percent.

Lesions. Areas of dark, gangrenous skin and necrotic muscle tissue in wings, breasts, thighs and legs are common. Some lesions may produce gas bubbles. Necrosis of the liver is com-

mon, but may not be visible in early stages. The Bursa of Fabricius is usually atrophied.

Diagnosis. Diagnosis is based on flock history, the presence of lesions, and the isolation of bacteria from lesions.

Treatment. Administer antibiotics such as penicillin and tetracycline to eliminate Clostridia. If drugs are used in combination, make sure they are compatible, and do not administer any drug in violation of FDA rules. Eliminating contributing factors such as deficiencies in vitamin E and selenium will help in treatment. To prevent the disease, protect birds from injury, destroy infected birds promptly, and properly clean and sanitize the house.

Necrotic Enteritis

Cause. This disease is caused by the type C toxin of *Clostridium perfringens*. There may be other contributing factors such as coccidiosis, vitamin E or selenium deficiency, infectious bursal disease (IBD), or inclusion body hepatitis. Necrotic enteritis is also known as enterotoxemia and rot gut.

Susceptibility. Necrotic enteritis is an acute infectious disease that affects mainly young broiler-type chickens and turkeys from 4 to 12 weeks old. Older chickens and turkeys, and also quail, are not usually affected by necrotic enteritis, but are more often affected by ulcerative enteritis. *C. perfringens* can cause a similar disease in sheep and cattle.

Transmission. This disease is not contagious. Birds contract the disease when they consume infected droppings. Since Clostridia can survive in soil and feces, proper sanitation is critical, especially in high-density flocks.

Clinical signs. Loss of appetite is a common symptom at the onset on necrotic enteritis. Birds may become acutely depressed and die within hours. Mortality is usually between 2 percent and 15 percent, but losses caused by emaciation, retarded growth and poor feed conversion may be even more costly than mortality.

Lesions. Lesions are usually found in the lower half of the small intestine, although sometimes the entire small intestine is affected. The walls of the intestine become engorged with blood and hemorrhage. The mucous membrane becomes necrotic and may also have a diphtheritic, cauliflower-like appearance. In advanced cases, the liver may become swollen and necrotic. There is also a fetid or putrid smell that exudes from the bird and its feces.

Diagnosis. A presumptive diagnosis is based on history, clinical signs and characteristic lesions. A definitive diagnosis requires isolation and identification of *Clostridium perfringens* type C.

Treatment. Use tetracycline antibiotics, bacitracin, streptomycin, penicillin or novobiocin to eliminate Clostridia. Be sure to follow label directions carefully, administer the correct dosage, and adhere to withdrawal requirements. To prevent the disease from recurring, destroy all birds and sanitize the house. Also correct any factors that predispose birds to this disease.

Ulcerative Enteritis

Cause. *Clostridium colinum* is responsible for ulcerative enteritis, which is also known as UE and quail disease.

Susceptibility. This disease is often severe in quail reared in captivity. It also affects chickens, turkeys and other poultry. Clinical signs usually

appear at 4 to 12 weeks of age, but older birds can be affected also. Ulcerative enteritis is generally associated with pullets and quail, whereas necrotic enteritis affects turkeys, broilers and roasters.

Transmission. The infection is transmitted by caprophagy, or the reingestion of the droppings of infected birds. *C. colinum* is highly resistant to disinfectants and can be found in varying environmental conditions (i.e., cold and hot, wet and dry).

Clinical signs. Birds appear extremely depressed, with loss of appetite and emaciation. Droppings are usually watery and may have a fetid odor. Mortality can be as high as 50 percent if the flock is not treated. The disease will develop from an acute condition to a chronic one.

Lesions. Lesions, which are located in the lower intestine and ceca of the bird, are generally yellow to white, circular and necrotic. If many ulcers form in a small area, the entire area will look like one large lesion with an eroded center. *C. colinum* can be isolated from the liver of chronically infected birds.

Diagnosis. The history and age of a flock, the presence of symptoms and lesions, and the absence of other infections are enough for a field diagnosis. Diagnosis is confirmed by isolating *C. colinum*.

Treatment. The antibiotics of choice are streptomycin, bacitracin or neomycin administered in the feed or drinking water. These antibiotics may be used singly or in combination. Ulcerative enteritis can be prevented by rearing birds in wire cages so they will not have access to droppings. To eradicate the disease, destroy the

infected flock and sanitize the premises before starting with an uninfected flock.

Fowl Cholera

Cause. Fowl cholera is caused by *Pasteurella multocida*, a bipolar, fluorescent, non-motile, rod-shaped bacterium. It is one of the oldest known poultry diseases; Pasteur studied it in the 1880s. There are three strains—1) smooth encapsulated, 2) mucoid, and 3) rough encapsulated. Virulence is highest in type 1 and lowest in type 3. There are also various serotypes associated with different poultry species.

Susceptibility. All domestic fowl species are susceptible to fowl cholera. Wild birds and cage birds are also susceptible. Most cases of fowl cholera occur in birds more than 6 weeks old. Birds that recover from the disease remain carriers for life.

Transmission. *P. multocida* can survive for 1 month in fecal droppings and for 2 to 3 months in decaying carcasses and soil. However, it is very susceptible to disinfectants, drying and sunlight. The bacterium usually enters a bird through the gastrointestinal tract or respiratory system. The incubation period is usually 3 to 9 days. There is no evidence that fowl cholera is transmitted by or through the egg. Reservoirs for *P. multocida* include wild birds, raccoons, opossums, dogs, cats, pigs and contaminated drinking areas.

Clinical signs. Common signs are greenish-yellow diarrhea, increased water consumption due to fever, and decreased feed consumption. In later stages of the disease, birds may become lame, lose weight, and make rattling noises because of congestion in the airway. Death fol-

lows soon after. Heads of dead birds are cyanotic (bluish). The disease is septicemic and is characterized by high morbidity and mortality. Outbreaks are usually acute, but a chronic form of the disease can be present afterwards.

Mortality increases dramatically as the disease becomes chronic.

Lesions. Lesions from *P. multocida* infection are variable. There may be septicemia with hemorrhage in the heart and abdominal lining. The liver is usually dark and swollen.

Diagnosis. Flock history, symptoms and lesions can indicate fowl cholera. Isolating the *P. multocida* bacterium will confirm a diagnosis.

Treatment. Eliminating potential reservoirs is the key to preventing fowl cholera. Proper sanitation of the house and premises is also very important. Vaccinations and other drugs can help, but the disease usually recurs if treatment stops.

Goose Septicemia

Cause. This disease is caused by *Pasteurella septicaemiae*, a gram-negative, encapsulated, non-motile and non-spore-forming bacterium. The organism is specifically different from *P. multocida*, which causes fowl cholera. This disease is also known as goose influenza.

Susceptibility. All ages of geese are susceptible to this disease, but it is very serious in goslings and otherwise immature geese. Swans and ducks are also susceptible.

Transmission. *P. septicaemiae* is transmitted from bird to bird. Infected premises, feed and water help spread the infection. There is no evi-

dence that goose septicemia is spread via the egg. Birds that recover from the disease are carriers for several weeks after their recovery.

Clinical signs. The disease is acute. Coughing, sneezing and discharges from the eyes and nasal passages are common early signs. Depression, general weakening, dehydration, incoordination and death follow soon after the initial signs. The disease causes blood poisoning (septicemia).

Lesions. Most organs are not affected; however, there is usually inflammation of the respiratory tract. There may also be signs of pericarditis and airsacculitis that is cheesy in appearance. The liver may be mottled with areas of necrotic tissue.

Diagnosis. If symptoms and lesions appear in a flock with a history of this disease, a tentative diagnosis can be made. Diagnosis is confirmed by isolating *P. septicaemiae*.

Treatment. Goose septicemia usually can be treated with sulfa drugs. Antibiotics that may be helpful include erythromycin, gentamycin and novobiocin. Remember to follow all regulations for using these drugs and observe the required withdrawal period. Implementing good sanitation practices and biosecurity measures is the way to prevent this disease. Do not place new birds in previously infected pens for at least 2 weeks after the pens have been carefully cleaned and disinfected.

Pasteurella anatipestifer **Infection**

Cause. *P. anatipestifer* is a gram-negative rod that is difficult to isolate and grow in the laboratory, but the bacterium is common in nature.

This disease is similar to goose septicemia and fowl cholera. Other names for this disease are infectious serositis, new duck syndrome and duck septicemia.

Susceptibility. Ducks 1 to 8 weeks old are susceptible. Older ducks may be infected, but usually do not show signs or ill effects.

Transmission. Birds come in contact with the bacterium through infected feed, water and environment.

Clinical signs. Early signs are coughing, sneezing and discharges from the eyes and nasal passages. Greenish diarrhea is also common. These signs are followed by incoordination, coma and death as the bacterium invades the bloodstream and causes systemic septicemia. Mortality ranges from 5 percent to 75 percent.

Lesions. As in goose septicemia, there is inflammation of the respiratory tract, pericarditis and cheesy airsacculitis. The liver and the spleen may be mottled, swollen and necrotic.

Diagnosis. A tentative diagnosis can be made on the basis of flock history, symptoms and lesions. Diagnosis is confirmed when *P. anatipestifer* is isolated from the heart blood, liver and brain. Because this disease is similar to several others, identifying the specific bacterium is important.

Treatment. Sulfa drugs and antibiotics such as erythromycin, streptomycin, novobiocin, gentamycin and tetracycline are helpful. Preventive measures include practicing good sanitation, promptly disposing of infected carcasses, and waiting 2 to 4 weeks after cleaning before new birds are placed in pens.

Salmonella pullorum

Infection

Cause. This disease is an acute or chronic infection caused by the bacterium *Salmonella pullorum*, a gram-negative rod that is resistant in some degree to cold, sunlight (UV), drying and disinfectants. The bacterium can survive in non-sanitized poultry houses for up to 1 year. Another common name for this disease is bacillary white diarrhea (BWD).

Susceptibility. While chickens and turkeys are the most susceptible to this disease, other species of birds also may become infected with *S. pullorum*. Young chickens under 14 days of age are highly susceptible. Birds that recover from the disease continue to shed the organism throughout their lives. This disease has been largely eradicated in commercial broiler, layer and turkey flocks in the U.S. and other developed countries.

Transmission. Transmission of *S. pullorum* is mainly from the hen to the chick via the egg. It also can be transmitted by personnel or equipment, carrier birds, and contaminated houses and premises. Bacteria in the environment enter birds via the respiratory or digestive system.

Clinical signs. The incubation period is 4 to 5 days. Chicks infected with *S. pullorum* begin to die 5 to 7 days after hatching. Mortality will increase for another 4 to 5 days and can be as high as 90 percent. Signs in individual birds include depression, diarrhea, pasty vent, and white feces that may be stained with bile. Surviving birds become asymptomatic carriers and pass the bacterium to their eggs because of a localized infection in the ovary.

Lesions. There may be small necrotic lesions on the liver and white nodes in the heart, gizzard, and walls of the intestine. Very young birds may have no lesions. Adult carriers may have necrosis in the liver and oviducts, which contain cheesy deposits.

Diagnosis. The history of the flock and the presence of symptoms and lesions can be used to make a tentative diagnosis, which is confirmed by the isolation of *S. pullorum*. If *S. pullorum* is confirmed, state and federal regulatory agencies must be notified.

Treatment. The use of antibiotics to treat this disease is not recommended because survivors will be carriers. To prevent *S. pullorum*, test breeder flocks before production to make sure that they are free of this disease. Positive flocks are not used for breeding. In fact, eradicating infected flocks is the best course of action to prevent the disease from spreading. There is no indication that this disease will cross to humans.

Fowl Typhoid

Cause. Fowl typhoid is caused by the bacterium *Salmonella gallinarum*, which is susceptible to sunlight and disinfectants but can survive in soil for many weeks. Another name for this disease is typhoid, but it should not be confused with the typhoid fever that occurs in humans, which is caused by a different organism.

Susceptibility. Chickens 12 weeks old and older are most susceptible to *S. gallinarum* infection. Other poultry species can become infected, but are generally more resistant than chickens.

Transmission. Fowl typhoid is transmitted from hen to egg to chick and also mechanically. There is more risk of mechanical transmission with *S. gallinarum* than with *S. pullorum*.

Clinical signs. The incubation period is normally 4 to 5 days. As with *S. pullorum* infection, loss of appetite, increased thirst, lethargy and yellow-green diarrhea are common signs. The first phase of the infection is acute and usually lasts 5 to 6 days. The secondary (chronic) infection lasts several weeks. Mortality ranges from 5 percent to 50 percent.

Lesions. An enlarged liver with a metallic sheen, an enlarged spleen, and a distended gall bladder are common lesions. There may also be enteritis and pinpoint-size necroses in the liver. Anemia and leukocytosis may occur in the chronic stages of the disease.

Diagnosis. Flock history and the presence of symptoms and lesions are used to make a tentative diagnosis. Fowl typhoid is confirmed by isolating *S. gallinarum*. Note that all cases of fowl typhoid must be reported to state and federal regulatory agencies.

Treatment. Treatment and prevention are the same as for *S. pullorum* infection. Using antibiotics is not recommended because survivors will be carriers. Breeder flocks should be tested before production to make sure they are free of this disease. Positive flocks are not used for breeding and should be eradicated. There is no indication that this disease will cross to humans.

Fowl Paratyphoid

Cause. Paratyphoid is a general term to describe infections caused by species of

Salmonella other than *S. pullorum* and *S. gallinarum*. There are more than 100 species and more than 2,200 serotypes of salmonellae. Many are host-specific and many can cross species. Approximately 40 serotypes are known to infect poultry, with 10 to 20 of them causing 70 percent of the cases. *S. typhimurium*, which is host-specific for chickens and turkeys, causes more than half of the paratyphoid cases in poultry. Another name for these infections is salmonellosis.

Susceptibility. All poultry and most wild birds are susceptible to one or more serotypes of Salmonella. Birds less than 4 weeks old are most likely to have acute infections. Domesticated animals, reptiles, rodents and humans are potential reservoirs and susceptible hosts.

Transmission. Paratyphoid can be transmitted from hen to egg to chick and mechanically. It is often spread by fecal contamination of the egg shell. Infected eggs in the incubator may “blow up” and spread the organism. Because some of these bacteria are able to cross species, paratyphoid is considered a public health issue.

Clinical signs. Outbreaks of paratyphoid usually occur in birds 7 to 21 days old. Mortality in these acute outbreaks peaks 7 to 14 days after infection and can range from 5 percent to 20 percent. Symptoms of paratyphoid include weakness, loss of appetite, increased thirst, diarrhea and pasty vent.

Lesions. The bacteria produce endotoxins that cause necrotic foci on the liver and damage to the intestinal wall, as well as enteritis, diarrhea and septicemia. Young birds may not exhibit any lesions.

Diagnosis. If a flock has a history of paratyphoid and if there are symptoms and lesions, a

tentative diagnosis can be made. Diagnosis is confirmed when the causative agent is identified.

Treatment. Sulfa drugs are effective against paratyphoid, but treatment is recommended only to salvage the flock. Prevention requires hatchery, house and flock sanitation.

***Salmonella Arizona* Infection**

Cause. This disease is similar to fowl typhoid, paratyphoid and *S. pullorum* infections. The *S. Arizona* bacterium is very susceptible to disinfectants, but survives easily in soil. There are more than 100 serotypes of this organism, and all of them are infective across species. Other names for this disease are paracolon and Arizona.

Susceptibility. All young chickens and turkeys up to 4 weeks of age are susceptible to this infection. Most *S. Arizona* infections are found in exotic or hobby birds.

Transmission. *S. Arizona* is spread from hen to egg to chick. Most often, egg shells become contaminated by feces and/or cloacal contents that contain the bacteria. Fecal contamination by carrier birds of the feed and water also occurs. Reptiles, wild birds and mammals are believed to be reservoirs for infection in poultry.

Clinical signs. The incubation period for *S. Arizona* infections is 5 to 7 days. Symptoms are the same as for fowl paratyphoid. They include diarrhea, depression, pasty vent and loss of appetite. Turkey poults may develop eye infection and blindness. The mortality rate is high. Birds that survive have chronic intestinal disease that may severely retard their growth.

Lesions. Bulging eyeballs, septicemia, enlarged liver and peritonitis are common with *S. arizona* infections. There may also be blockage of the small intestine, gall bladder distension, and abscesses in the lungs.

Diagnosis. Because the clinical signs of this disease are almost identical to those of paratyphoid, the *S. arizona* bacterium must be isolated for a definitive diagnosis.

Treatment. Treatment of this infection is not recommended. Good sanitation at the hatchery and farm can help prevent it.

Colibacillosis

Cause. This disease is caused by opportunistic intestinal (coliform) bacteria—usually *Escherichia coli*. These bacteria invade and cause a secondary infection when the bird is stressed or has an initial infection caused by another pathogen. An example of initial infection is infectious bursal disease (IBD). Other names for this disease include coligranuloma, coli septicemia and *E. coli*.

Susceptibility. All ages and species of poultry are susceptible to this disease; however, younger birds tend to be more susceptible.

Transmission. Colibacillosis is considered an environmental disease. The infection is not spread directly from bird to bird, but via contact with environmental surfaces contaminated with feces containing the bacteria.

Clinical signs. Depression, decrease in appetite, paleness, thinness and diarrhea are commonly associated with this disease. Septicemia will follow.

Lesions. The most common lesion associated with this disease is enteritis. If the initial infection has been respiratory in nature, this secondary infection also can cause airsacculitis, pericarditis and perihepatitis. If histomoniasis is the primary infection, turkeys often exhibit coligranuloma on the liver, intestines or lungs.

Diagnosis. The presence of symptoms and lesions is enough for a tentative diagnosis. Confirmation of colibacillosis requires the isolation and identification in the laboratory of pathogenic coliform bacteria. Most strains of *E. coli* and other coliforms are naturally present in the gastrointestinal tract of birds; the pathogenic strains can be separated from non-pathogenic strains because they form specific structures of the cell wall called pili. These pili can be seen only under a microscope.

Treatment. Improved sanitation of the environment and of the feeding and watering equipment is the key to preventing this disease. Drugs that can be used against coliforms include sulfa drugs, tetracycline (and its derivatives), novobiocin and neomycin.

Erysipelas

Cause. Erysipelas is caused by *Erysipelothrix insidiosa*, a bacterium that also causes disease in sheep and swine and can cause erysipeloid in humans.

Susceptibility. Turkeys are the most susceptible species of poultry; however, chickens, ducks and gamebirds are also susceptible. Turkeys reared on range tend to be more susceptible than those reared in houses, and the disease usually is found in birds 4 to 7 months old. Because this bacterium can cause disease in humans, care

should be taken when handling infected birds.

Transmission. *E. insidiosa* is a soil-borne disease, so the grounds of the farm are usually the primary source of infection. This disease also can be spread if healthy birds consume the feces of infected birds.

Clinical signs. Cyanosis, listlessness, arthritis, and a yellow-green diarrhea are common symptoms of this disease. There may also be reddish or purplish areas on the skin. The disease is septicemic. Mortality can range from 2 percent to 25 percent.

Lesions. Enteritis, cyanosis, swollen snoods, swollen liver, swollen spleen, hemorrhaging in organs and muscles, and endocarditis may be present. Most lesions resemble those found with fowl cholera.

Diagnosis. Flock history, symptoms and lesions lead to a presumptive diagnosis, with confirmation based on the isolation of *E. insidiosa*.

Treatment. Treatment is mainly directed at turkeys, as this species is the most susceptible. Penicillin is recommended, but tetracycline and erythromycin are also effective. There is a vaccine that will help prevent this disease. Infected premises should be thoroughly cleaned and disinfected before new flocks are introduced.

Omphalitis

Cause. The technical definition of this disease is inflammation of the navel. When the navel closes improperly, bacteria such as *E. coli*, *Pseudomonas* spp., *Salmonella* spp. and *Proteus* spp. may invade, especially if the shell quality is poor. Contributing factors are dirty eggs,

improper incubator and hatchery sanitation, excessive humidity in the incubator, and chilling or overheating of newly hatched chicks. Other common names for this disease are navel ill and mushy chick disease.

Susceptibility. Chicks from eggs laid by older hens are more susceptible because of lower egg shell quality.

Transmission. Omphalitis occurs during the first few days of life and is not transmitted from bird to bird. Transmission takes place if the incubation and chick processing environments are unsanitary. Eggs that explode in the setter and hatcher contribute to the spread of the bacteria that cause this disease.

Clinical signs. Enlarged abdomens, inflamed navels, pasty vents, weakness and lack of body tone are clinical signs. Infected chicks will huddle together and most will die within the first week after hatch. Mortality as high as 15 percent has been reported, but it is normally 1 to 3 percent.

Lesions. Lesions include peritonitis, unabsorbed yolk sac, observable navel infection, dehydration and septicemia.

Diagnosis. Chick age and condition and the presence of lesions indicate a tentative diagnosis. Confirmation depends on isolating and identifying the causative bacterium from the yolk sac.

Treatment. There is no treatment for omphalitis. Chicks with the disease will die within days and unaffected chicks need no medication. The disease can be prevented with proper sanitation of the hatchery and proper handling of eggs. Controlling the temperature at chick placement is very important because temperature stress in

day-old chicks causes symptoms similar to omphalitis and exacerbates the disease.

Staphylococcus aureus **Infection**

Cause. This disease is caused by the bacterium *Staphylococcus aureus*. In avian species, the bacterium liberates beta hemolysin and plasma coagulase. These substances hemolyze the blood and also cause it to coagulate. Septicemia caused by this organism is usually a secondary infection and may coincide with selenium deficiency, gangrenous dermatitis, aplastic anemia or hemorrhagic diseases. Other names for this disease include staph infection, staph septicemia, staph arthritis and bumblefoot.

Susceptibility. All poultry are susceptible to this disease, especially birds 4 to 6 weeks old.

Transmission. *S. aureus* infection is an environmental disease that does not spread from bird to bird. Avian strains of this bacterium are not cross-infective like the human and mammalian strains. This disease can easily become chronic.

Clinical signs. There are three forms of infection by *S. aureus*:

1. **Acute septicemic form** – Symptoms are similar to fowl cholera, including loss of appetite, depression, listlessness and fever. Birds may also have a watery diarrhea.
2. **Chronic arthritic form** – This form will follow the septicemic form. Lameness, breast blisters and painful movement are symptoms. Because movement is limited, birds may be off feed and water so that they become dehydrated and lose weight.
3. **Bumblefoot** – This is a localized *S. aureus* infection of the foot that causes lameness and swelling of the foot pads.

It usually occurs because of puncture wounds to the foot.

The acute form of the disease is characterized by septicemia, while the chronic form is characterized by arthritis or bumblefoot, or both.

Mortality can be as high as 60 percent if an outbreak is untreated, but is usually less than 5 percent.

Lesions. In the acute form, there is septicemia along with swelling and darkening of the liver and other organs. The contents of the intestine may be watery and there may be yellow pus in infected joints. In the chronic form, arthritis, breast blisters, bumblefoot and infections of the earlobes are usually present.

Diagnosis. Tentative diagnosis can be made on the basis of flock history and the presence of symptoms and lesions. The disease is confirmed if the *S. aureus* bacterium is identified.

Treatment. Penicillin, erythromycin and novobiocin are helpful. Good management and isolating chronically infected birds can help prevent the disease.

***Streptococcus* Infection**

Cause. This disease is caused by bacteria from the genus *Streptococcus*, especially *S. zooepidemicus* and *S. faecalis*. *S. zooepidemicus* is usually isolated from infections in mature birds, while *S. faecalis* is usually found in birds 7 to 35 days old. These bacteria release toxins that add to their pathogenicity. Other names for this disease are strep and strep septicemia.

Susceptibility. The disease primarily affects chickens; other species are not normally affected. *Streptococcus* strains are usually adapted to a specific host, so are rarely cross-infectious

between species. The disease usually has low virulence and a low mortality rate.

Transmission. It is thought that contaminated feed and water are the primary source of infection. It is possible that some intestinal strains can be passed from hen to egg to chick.

Clinical signs. The infection can be acute or chronic. Birds with the acute form of the disease tend to be listless and feverish and have a blue to purple color. Birds with the chronic form may show loss of appetite, listlessness and endocarditis. Diarrhea and increased mortality are also signs of infection, although many infected birds do not show clinical signs and go undetected.

Lesions. There are no visible lesions in mature chickens. Chicks will exhibit endocarditis.

Diagnosis. A flock history of *Streptococcus* infection and clinical signs are enough for a tentative diagnosis. Confirmation comes when a *Streptococcus* species is isolated and identified.

Treatment. Penicillin, erythromycin, novobiocin and gentamycin are usually effective in controlling these bacteria. The disease can be prevented through good flock management and correct sanitation, with clean-up between flocks. Infected birds should be culled from the flock.



Viral Diseases (non-respiratory)

Avian Encephalomyelitis

Cause. Avian encephalomyelitis is caused by a picornavirus that propagates in the yolk sac and brain of the chicken embryo. Another name for this disease is epidemic tremor.

Susceptibility. Young chickens from 1 to 6 weeks of age are the most susceptible. Other poultry may become infected, but are more resistant than young chickens. Older chickens may be infected but show no clinical signs.

Transmission. The virus is primarily transmitted from hen to egg to chick. There is also evidence of direct chick-to-chick transmission and environmental transmission. Chicks hatched from infected breeders are infected at hatch. It appears that infected chickens do not remain carriers and are not susceptible to the disease again for a period of time.

Clinical signs. The average incubation period is 10 to 17 days. Tremors of the head and neck are the most common sign. These tremors are best seen after the bird has been shaken energetically. Affected chicks usually have a dull expres-

sion in the eyes, then incoordination, tremors and, finally, paralysis. Feed and water consumption decrease, followed by weight loss. Affected adult birds may show only a drop in egg production.

Lesions. Young birds do not have lesions that are visible to the naked eye; however, most tissues have microscopic lesions.

Diagnosis. A history of the disease in a flock and the presence of clinical signs are enough for a presumptive diagnosis. Definitive diagnosis is made by means of a fluorescent antibody test and/or histopathology.

Treatment. There is no treatment for acute outbreaks and young birds that are affected should be euthanized and disposed of. The disease can be prevented by selecting hatching eggs from breeder flocks that are immune to the disease. Birds become immune by recovering from an outbreak or by vaccination. Reliable vaccines can be made from modified versions of the virus.

Avian Leucosis—Lymphoid Leucosis and Marek's Disease

Cause. Avian leucosis is a complex of two cancer-like diseases—lymphoid leucosis and Marek's disease. These diseases are caused by unrelated viruses.

Lymphoid leucosis is caused by a family of RNA leukoviruses called the lympho/sarcoma group. These viruses are very sensitive to germicides. They are also killed by exposure to room temperature but can survive a long time at freezer temperatures.

The virus that causes Marek's disease is a DNA cell-associated, or type B, herpes virus. This virus can survive at room temperature for a short time but is killed by freezing and thawing. It is also killed by some disinfectants but is resistant to quaternary ammonia and phenols.

Susceptibility. With both lymphoid leucosis and Marek's disease, chickens are more susceptible than other types of poultry and females are more susceptible than males. Young chickens (2 to 16 weeks old) are most susceptible, older birds less so. Stress from other diseases may heighten the severity of these diseases. Males that contract lymphoid leucosis are more susceptible to the development of osteopetrosis. Birds that contract Marek's disease are carriers of the virus for life.

Transmission. The lymphoid leucosis virus is transmitted by contact with infected birds (vector birds or flock mates), by the environment, and from hen to egg to chick. The Marek's disease virus is mainly spread via the air, although it, too, is contagious.

The incubation period for lymphoid leucosis is about 16 weeks. When osteopetrosis, erythroid

leucosis or myeloid leucosis develops, the incubation period can be as short as 3 to 4 weeks. The incubation period for Marek's disease is about 14 days.

Clinical signs. In lymphoid leucosis clinical signs will not be visible until after the bird is 16 weeks of age. Then the bird will be weak and pale and have an enlarged abdomen. The bird becomes emaciated and dies. The clinical signs of Marek's disease include lameness, incoordination, paleness, unthriftiness, weakness, labored breathing and enlarged feather follicles. The eyes have grey irises and constricted, irregularly shaped pupils.

Lesions. Both diseases in the avian leucosis complex cause tumors, which can be either focal (tumor cells reproduce at the location of the tumor) or diffuse (tumor cells pass into the circulatory system and cause enlargement of organs).

Lymphoid leucosis produces focal or diffuse tumors of the liver, spleen and kidneys, and infrequently of the heart, pancreas, gastrointestinal tract (including mesentery) and Bursa of Fabricius. Marek's disease produces tumors of the brain, spinal cord, nerves, liver, spleen, kidneys, heart, lungs and GI tract (including mesentery).

Diagnosis. A presumptive diagnosis can be based on flock history and the presence of lesions. Definitive diagnosis is based on progression of the disease (*see Table 2*). It is very important to differentiate the two forms of avian leucosis from each other and from other diseases.

Treatment. There is no treatment for either lymphoid leucosis or Marek's disease. Good

Table 2. Comparison of lymphoid leucosis and Marek's disease.

Feature	Lymphoid leucosis	Marek's disease
Incubation period	16 weeks (prolonged)	2 weeks (short)
Age of incidence	Over 4 months	Under 5 months
Visceral lesions	Normally liver and spleen	Generalized
Skin lesions	No	Yes
Ocular lesions	No	Yes
Neural lesions	No	Yes
Lesions on Bursa of Fabricius	Yes	No

husbandry techniques can help prevent lymphoid leucosis and vaccination can help prevent Marek's disease.

Avian Pox

Cause. There are three strains of virus that cause avian pox: fowl pox virus, pigeon pox virus, and canary pox virus. Each strain of the virus can infect species other than the one it is named for. Fowl pox virus may infect chickens, turkeys, pheasants, quail and ducks. Pigeon pox virus infects pigeons, chickens and turkeys. Canary pox virus can infect canaries, chickens, pigeons and sparrows. Other names for this disease are fowl pox, canker and avian diphtheria.

Susceptibility. All poultry in all age groups are susceptible, except for newly hatched birds.

Transmission. Avian pox can be transmitted by direct or indirect contact. The viruses found in sloughed-off scabs are highly resistant and may survive for several months. Mosquitoes are also a common vector for this disease. Birds that recover do not continue to carry the virus.

Clinical signs. Avian pox usually spreads slowly through a flock, so clinical signs may not be

readily apparent. The disease usually runs its course in 3 to 5 weeks. Young birds with this disease will have retarded growth. Laying hens will have a drop in production. All infected birds tend to have decreased appetite because of difficulty in eating and breathing.

Lesions. There are two types of lesions and one or both may appear.

- *Cutaneous (dry) pox*—Lesions begin as small, white foci and later develop into wart-like nodules on the skin. These nodules eventually slough off and scabs form before final healing. These lesions are most often seen on areas without feathers (comb, wattles, earlobes, eyes), but may be found on any part of the body.
- *Diphtheric (wet) pox*—Lesions are usually found in the oral cavity and upper respiratory tract. These lesions are diphtheric and will leave an ulcerated or eroded area if removed.

Diagnosis. Presumptive diagnosis is based on flock history and the presence of lesions. Definitive diagnosis is based on laboratory tissue cultures or transmission studies.

Treatment. There is no treatment for avian pox, but it can be prevented by vaccination.

Hen Adenovirus Infection

Cause. The virus that causes this disease belongs to the adenovirus group. These viruses are typically stable when exposed to heat and acid and are resistant to ultraviolet light. The viruses are thought to be opportunistic, infecting birds only if predisposing factors are present.

Susceptibility. Hens that are partially to fully mature are susceptible.

Transmission. Hen adenoviral infection can be spread in many ways, including hen to hen, fecal contamination of feed and water, aerosol or air transmission, and from hen to egg to chick.

Clinical signs. Symptoms are usually so mild that they go unnoticed. Egg production may drop 5 to 10 percent and shell quality may decrease. After 1 to 3 weeks egg production and shell quality stabilize but do not return to previous levels. There may be some respiratory symptoms, but not always.

Lesions. Mild swelling of the liver, kidneys and spleen is common. Microscopic lesions include hepatitis and edema and shrinkage of the mucosa in the shell gland (uterus).

Diagnosis. Presumptive diagnosis is based on flock history and the presence of clinical signs and lesions. Definitive diagnosis is based on laboratory confirmation.

Treatment. There is no treatment for this disease. Vaccines that have been tested have not

been successful. Prevention is accomplished by good flock husbandry and biosecurity.

Infectious Bursal Disease (IBD)

Cause. This disease is caused by a diploornavirus, which has double-stranded DNA. The virus damages all types of lymphocytes, especially lymphocytes of bursal origin. In fact, the virus is dependent on the Bursa of Fabricius. In experiments where the bursa was removed, introducing the virus did not cause disease or immunosuppression. Other names for this disease are gumboro, IBA, IBD, infectious bursitis and infectious avian nephrosis.

Susceptibility. This disease occurs only in chickens. All types and breeds are equally susceptible. Birds may be infected at up to 12 weeks of age, but are usually infected within 24 hours after hatch. If infection occurs after this time, symptoms will usually appear within 2 to 5 weeks.

Transmission. The virus usually spreads from bird to bird or by contact with a contaminated environment. It is also possible that the virus is spread via the air and by vectors such as rodents.

Clinical signs. Symptoms include decreased feed and water consumption, white and watery diarrhea, ruffled feathers, lethargy and vent picking. Morbidity is usually 100 percent, with mortality of up to 15 percent. Recovered birds have immunosuppression because antibodies are produced from B-lymphocytes.

Lesions. The Bursa of Fabricius may be swollen up to three times the normal size. The bursa

also becomes yellow and hemorrhagic. After the initial swelling, the bursa shrinks to an elongated shape, which indicates clinical recovery. Other lesions include rickets-like soft bones and swollen kidneys.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. Definitive diagnosis is based on isolation of the virus.

Treatment. There is no specific treatment for IBD. Vaccination will prevent the disease.

Inclusion Body Hepatitis

Cause. Inclusion body hepatitis is an acute infectious disease of chickens caused by a DNA adenovirus. The virus replicates in the nuclei of liver cells, thus causing an inclusion. The disease usually occurs when there is a predisposing condition. Other names for this disease are IBH, aplastic anemia and hemorrhagic syndrome.

Susceptibility. Chickens 3 to 9 weeks old are most commonly affected by this virus, though infection can occur at up to 15 weeks of age. Adult birds show no symptoms if infected as adults.

Transmission. Bird-to-bird contact, environmental exposure, and hen to egg to chick are all common methods of transmission for this virus.

Clinical signs. Depression, fever, aplastic anemia, jaundice and increased mortality are signs of this disease. Mortality usually averages 10 percent, but can be as high as 40 percent. Secondary clostridial and coliform infections are

possible. These secondary infections will usually cause high mortality.

Lesions. Hemolytic anemia, jaundice, swollen liver, mildly swollen spleen and kidneys, and hepatic inclusions are common lesions. Early in the progression of the disease, the virus may be isolated from the liver, kidney, spleen and Bursa of Fabricius.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. Definitive diagnosis is based on isolation of the adenovirus from the affected bird.

Treatment. There is no specific treatment for IBH. Thoroughly cleaning and disinfecting the brooding environment can help prevent it. Germicides that contain formalin or iodine are effective against adenoviruses.

Coronaviral Enteritis of Turkeys

Cause. This disease is a highly contagious enteritis of turkeys caused by a coronavirus. The virus itself is stable at pH 3, which is a highly acidic environment. This disease is not related to "blue comb" of chickens or hemorrhagic enteritis in turkeys. Other names for this disease are transmissible enteritis, TE and "blue comb."

Susceptibility. This disease affects only turkeys. Young turkeys are especially susceptible, but all ages can contract the disease.

Transmission. Ingesting contaminated substances seems to be the primary mode of transmission. This virus is not transmissible via the egg.

Clinical signs. The incubation period is 72 hours. Symptoms include depression, lowered body temperature, loss of appetite, weight loss, decreased production of breeder flocks, watery diarrhea, cyanosis and death. In a young flock mortality can be as high as 50 percent; in older flocks it is usually less than 5 percent. Birds that recover from the disease are carriers and shed the virus for the remainder of their lives.

Lesions. Lesions are found in the intestinal tract and include enteritis, flaccid intestinal walls, separation of the mucosal epithelium from the intestinal wall, nephritis and small spleen.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. Definitive diagnosis is based on a positive fluorescent antibody test in a laboratory.

Treatment. Brooder temperature should be adjusted to comfort young poults. It is helpful to administer water-soluble vitamins in drinking water and antibiotics in feed. Because this disease is highly contagious, its recurrence should be prevented. Recovered birds shed the virus for life, so all infected animals should be destroyed. The rearing house must be thoroughly cleaned and disinfected to ensure that the life cycle of the virus is interrupted.



Viral Respiratory Diseases

Newcastle Disease

Cause. Newcastle disease is a contagious respiratory disease caused by a paramyxovirus. The virus can remain viable in infected litter for approximately 2 months and up to 12 months in infected poultry carcasses. The pathogenicity of the virus ranges from mild to high. It is killed by disinfectants and direct sunlight. Other names for this disease are ND and pneumoencephalitis.

Susceptibility. Birds of all ages are susceptible, especially young chickens. Humans and other mammals are also susceptible. The virus has been known to cause eye infections in people who work with it in laboratories, or who rub their eyes after handling the vaccine.

Transmission. The incubation period for this virus is 5 to 7 days. During that time birds are contagious and shed the virus in their bodily fluids and excretions. The virus can be transmitted by contaminated fomites (equipment, clothing, etc.) and via the air. It spreads rapidly. Newcastle disease is passed from the hen to the egg, but the embryo will die before hatching.

Clinical signs. Respiratory stress, wheezing, nasal discharge, depression, decreased appetite, decreased water consumption, and drop in production are clinical signs. Mortality will range from 10 percent to 80 percent, depending on the virulence of the virus. The disease will run its course in 10 days to 2 weeks; however, production will not come back for 5 to 6 weeks. Even after production has resumed, many eggs will have rough, thin shells and be of low grade.

Lesions. Edema in the face is common, along with airway congestion and hemorrhaging in body tissues. There may be infection in the brain (encephalitis) and exudates in the air sacs and lungs. The undeveloped eggs of infected laying hens will show deterioration.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. Confirmation is based on isolation of the virus and blood tests for the presence of antibodies. This disease has very similar symptoms to other respiratory diseases and should be differentiated by laboratory methods.

Treatment. There is no specific treatment for this disease, although antibiotics can be administered to prevent secondary infections.

Newcastle disease can be prevented by following an annual vaccination schedule.

Exotic Newcastle Disease

Cause. This disease is a foreign form of Newcastle disease. The virus usually arrives via smuggled exotic birds. It can survive for weeks in materials such as bird feces and broken eggs. Other names for the disease are END, Asiatic Newcastle disease (AND), and viscerotropic velogenic Newcastle disease (VVND).

Susceptibility. As with Newcastle disease, birds of all ages are susceptible, as are humans and other mammals.

Transmission. Exotic Newcastle disease is highly contagious. Infected birds shed the virus in all bodily fluids and excretions during the contagious stage of infection—3 to 5 days after infection occurs. Then the virus can be transmitted by contaminated fomites and by the air. Survivors of this disease are highly immune and pass this immunity to subsequent generations.

Clinical signs. Symptoms include listlessness, heavy breathing, wheezing, nasal discharge, watery diarrhea that is bloody or greenish, and a drastic drop in production. However, poultry infected with END may die before any clinical signs or symptoms are evident.

Lesions. As with general Newcastle disease, there is edema of the face. There are also hemorrhages in the mouth, nostrils, and sometimes other areas such as the cloaca. The liver will be dark and swollen. Internal organs hemorrhage

and deteriorate, leading to septicemia and death.

Diagnosis. A tentative diagnosis can be made on the basis of clinical signs and lesions, especially if a flock has had contact with exotic birds. Definitive diagnosis is based on laboratory isolation of the virus to differentiate END from general ND and avian influenza.

Treatment. There is no treatment for this disease. Infected birds should be slaughtered and disposed of in a manner that will keep the disease from spreading. Vaccinating for Newcastle disease and implementing biosecurity measures can help prevent END.

Infectious Bronchitis

Cause. Infectious bronchitis is caused by a coronavirus. The virus does not survive well outside of the body of the chicken and can be easily killed by disinfectants, heat and direct sunlight. There are many strains of this virus, ranging in pathogenicity from mild to severe. Other names for this disease include IB, bronchitis and cold.

Susceptibility. Only chickens are susceptible to this disease. Birds that recover are immune for several months.

Transmission. Many experts consider this the most contagious disease of poultry. All unimmunized birds within a flock will become infected if the virus is introduced. The incubation time is 17 to 36 hours and the disease courses through a flock in 10 to 14 days. This quick spread will occur despite sanitary measures or quarantine. The virus can be spread by the air over long distances; it is also spread

mechanically and by fomites. The virus is not spread via the egg.

Clinical signs. Symptoms include coughing, sneezing, rales, labored breathing, and a watery discharge from the eyes and mouth. Feed consumption decreases sharply and growth slows. Production in laying hens can drop to near zero within 10 to 12 days and it may take up to 5 weeks to return to normal levels. Mortality may be as high as 40 percent in chickens less than 3 weeks old, but is not significant in chickens older than 5 weeks unless there is a secondary infection.

Lesions. Lesions include nasal discharge and exudates in all areas of the respiratory tract.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. Definitive diagnosis is based on isolation of the virus in the laboratory. Other respiratory diseases will be absent in the case of IB.

Treatment. There is no specific treatment for this disease. Antibiotics may be given for 3 to 5 days to prevent secondary infections. It can be prevented by vaccinating birds annually.

Laryngotracheitis

Cause. Laryngotracheitis is caused by a herpes virus. The virus is easily killed by disinfectants or direct sunlight; however, it can survive and remain virulent when it is frozen. There are many strains, each with a different virulence. Other names for this disease include LT, trach and laryngo.

Susceptibility. All chickens and pheasants are susceptible to this disease. Older chickens (≥ 14

weeks) tend to be more susceptible than younger ones; however, some strains of the virus seem to have adapted to young birds, including broilers.

Transmission. This disease is highly contagious. The virus moves within a flock when birds come in contact with infected birds or bird tissue, dead birds, or contaminated fomites (clothing, shoes, equipment, etc.). The incubation time is 2 to 12 days. Birds that recover remain carriers and shedders of the virus for up to 2 years.

Clinical signs. Watery eyes is usually the first symptom noticed in an outbreak of LT. Birds tend to be quiet early in the infection, as breathing is difficult. As the infection progresses egg production drops sharply and birds will cough, sneeze and shake their heads to try to dislodge exudates within the respiratory system. Exudates that are expelled are often tinged with blood and/or serum. These exudates can block the trachea and cause asphyxiation. Mortality is high. Death or recovery usually occurs within 5 to 6 days after the incubation period.

Lesions. Lesions are found mainly in the respiratory tract, where hemorrhaging in the trachea, blood clots, and false membrane formation are common.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. For a definitive diagnosis the virus must be isolated in the laboratory.

Treatment. There is no specific treatment, although vaccinating an infected flock will prevent the spread of the infection. Birds that die should be incinerated. Vaccination can prevent

LT. Attenuated vaccines should be used instead of live, virulent vaccine. If older birds that are infected are to be held over until the disease has coursed, incoming young birds should be vaccinated as soon as possible to help prevent the spread of the disease.

Avian Influenza

Cause. Avian influenza is an infectious respiratory disease. The viruses that cause it are type A influenza viruses. Currently there are 16 hemagglutinin (H) and nine neuramidase (N) types known. These different types can form various combinations such as H5N1 or H7N1. This variability in types makes it difficult to vaccinate against all types of avian influenza. Other names for this disease include AI, flu and influenza.

Susceptibility. All types of poultry and many wild birds are affected by this disease. Humans also are susceptible, but these cases are rare and at this printing have not occurred in the U.S.

Transmission. Avian influenza can be transmitted by bird-to-bird contact, fomites, airborne transmission, ingestion of infected feces, insects, and other animals.

Clinical signs. There are two forms of avian influenza—low-pathogenic AI (LPAI) and high-pathogenic AI (HPAI). LPAI produces weakness, listlessness, reduced feed and water consumption, decreased productivity in laying hens, diarrhea and, in general, low mortality. HPAI produces similar symptoms but also cyanosis of legs and comb, dehydration and acute respiratory stress. Morbidity from HPAI is usually 100 percent and mortality can also be near 100 percent. Mortality usually peaks on about the seventh day of the illness.

Lesions. AI produces typical lesions of respiratory illness, including septicemia, airsacculitis, cyanosis and tracheitis.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. A diagnosis is confirmed by laboratory tests.

Treatment. There is no treatment for AI. Infected flocks should be quarantined and depopulated. Dead birds should be incinerated. Houses should be disinfected thoroughly before new birds are placed in them. Good biosecurity measures and good flock husbandry can help prevent AI.

Quail Bronchitis

Cause. The adenovirus virus that causes quail bronchitis is not related to the virus that causes infectious bronchitis and it can remain viable for a long time. Another name for this disease is QB.

Susceptibility. Bobwhite quail (American) is the only species affected by this virus. The Japanese coturnix quail are resistant. Turkeys and chickens will develop antibodies if exposed to the virus but do not show clinical signs.

Transmission. Quail bronchitis is highly contagious and can be spread by bird-to-bird contact, by fomites, and by aerosol (air) transmission. Incubation time ranges from 4 to 7 days.

Clinical signs. Sneezing, coughing, rales, and decreased feed and water consumption are symptoms of QB. Morbidity is usually 100 percent, and mortality up to 40 percent. The disease runs its course in 3 to 5 weeks, but unless

the rearing facility is completely cleaned out each successive flock will contract the disease from shed virus.

Lesions. Lesions are similar to those found in infectious bronchitis in chickens. They include exudates in the trachea and bronchi, along with tracheitis. There may also be some hemorrhaging and enlargement of the liver.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions and confirmed by laboratory tests.

Treatment. There is no specific treatment for this disease. Antibiotics may be given to reduce the possibility of the secondary mycoplasma infections that usually accompany quail bronchitis. Successive hatches of quail should be kept away from infected birds. The cycle of infection can be broken by depopulating infected birds, cleaning and disinfecting the affected facilities, and then allowing for a 1- to 3-month "drying out" period before the rearing area is repopulated.

Pigeon Paramyxovirus

Cause. This disease is caused by paramyxovirus-1. The virus is considered to be a Newcastle disease virus; however, this paramyxovirus is categorized in a subgroup that is specific to pigeons. Other names for this disease are pigeon Newcastle disease and PMV-1.

Susceptibility. Although it affects mainly pigeons, chickens and turkeys also are susceptible.

Transmission. The virus can be spread from bird to bird, via the fecal/oral route, by fomites

such as clothing, and by airborne means. Incubation time is 2 to 18 days and the disease usually runs its course within 10 to 14 days.

Clinical signs. Symptoms include loss of appetite, depression, greenish diarrhea, and neurological signs such as incoordination and paralysis.

Lesions. Lesions vary, but include encephalitis, tracheitis and pneumonia. There may be cyanosis of muscle and skin.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. A diagnosis is confirmed in the laboratory.

Treatment. There is no specific treatment for this disease. Antibiotics may be given to prevent secondary infections. Vaccination with a killed or inactive poultry Newcastle disease vaccine prevents the disease.



Viral-Like Respiratory Diseases

Chlamydiosis

Cause. This disease is caused by the obligate bacterium *Chlamydia psittaci*, which reproduces in the tissue of the respiratory tract. Other names for this disease are parrot fever, ornithosis and psittacosis.

Susceptibility. This disease affects both wild and domestic birds. Chickens are rarely affected, but can be susceptible. Humans are also susceptible to this disease. Younger birds tend to be more susceptible than older birds, while younger humans are more resistant than older humans. The infection can be serious in humans and may be difficult to diagnose.

Transmission. This disease can be passed from bird-to-bird, via the fecal-oral route, by contaminated clothing and equipment, and by inhalation of dust from feces. The incubation period is 5 to 7 days.

Clinical signs. Symptoms can be acute or chronic and vary with the species infected.

- **Turkeys**—greenish-yellow feces, depression, respiratory difficulty and loss of appetite

- **Parrots and other psittacines (curved-beak birds)**—diarrhea, brain hemorrhage and death
- **Humans**—headache, pneumonia-like symptoms, and fever that can exceed 104 degrees F

Lesions. All affected bird species will have swollen spleen and liver, fibrinous pneumonia and cloudy air sacs. Lesions specific to turkeys include inflammation of the pericardium (membrane around the heart) and plastic-like exudates on the heart and liver.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. Definitive diagnosis is accomplished by laboratory testing.

Treatment. The disease is treated with antibiotics prescribed by a veterinarian. Good husbandry practices and biosecurity can help prevent it. Humans who have been working with turkeys and develop flu-like symptoms should be examined for this disease by a physician.

Mycoplasma gallisepticum **Infection**

Cause. This disease is considered the most pathogenic of the three types of *Mycoplasma* infections. The organism that causes this disease, serotype S-6, is very small and does not have a rigid cell wall. It is susceptible to many antibiotics and is killed by sanitizers, sunlight and disinfectants. Other names for this disease include MG, CRD, infectious sinusitis and mycoplasmosis.

Susceptibility. Most poultry are susceptible to this disease, including chickens, pigeons, turkeys, ducks and peafowl. All ages of susceptible species can contract the disease; however, younger birds tend to be marginally more susceptible than older birds. Chickens seem to endure the disease better than turkeys because the antibodies chickens produce provide some defense.

Transmission. Infected birds shed the disease-causing organism, so the disease can be spread by bird-to-bird contact, inhalation of aerosolized particles, fomites, and from hen to egg to chick.

Clinical signs. Chickens and turkeys show different signs of infection.

- **Chickens**—Typical symptoms include sticky exudates from the nostrils, sinusitis, foamy exudate from the eyes, airsacculitis, yellow exudates in the air sacs, rales and sneezing. If there is no secondary infection, chickens may not show any outward symptoms.
- **Turkeys**—There are two forms of this disease in turkeys—the upper and lower forms. Symptoms of the **upper form** are watery eyes, sinusitis with firm exudates,

rales and unthriftiness. In the **lower form** symptoms are airsacculitis with firm, yellow exudates, and slight rales.

Lesions. Lesions in chickens and turkeys are different.

- **Chickens**—There will be exudates in the trachea, lungs and nasal area, and airsacculitis with cheesy, yellow exudates.
- **Turkeys**—Lesions associated with the lower form of the infection are the same as for chickens. For the upper form, lesions are restricted to the upper respiratory tract and include sinusitis and exudates from the eyes and nostrils and in the trachea.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. Isolation of the organism in the laboratory confirms the diagnosis.

Treatment. There are antibiotics for treating this disease. The one most recommended is tylosin tartrate. Consult a licensed veterinarian about the best treatment for your flock. Infected birds should be eradicated.

Mycoplasma synoviae **Infection**

Cause. This disease is caused by another member of the *Mycoplasma* genus, *Mycoplasma synoviae*. It was originally classified as an infection of the joints, but has recently been shown to be a respiratory infection. There are two forms of this infection: synovitis and respiratory. The organism is susceptible to disinfectants and sunlight and is very closely related to the *M. gallisepticum* and *M. meleagridis* organisms. Other

names for this disease include MS, infectious synovitis, synovitis and silent air sac.

Susceptibility. Chickens and turkeys are the most commonly affected species. All ages of birds can be infected, but the synovital form mainly affects chicks and poults from 4 to 12 weeks of age. The respiratory form of the disease can affect chickens and turkeys of any age.

Transmission. This disease can be spread by bird-to-bird contact, fomites, inhalation of aerosolized particles, and from hen to egg to chick. The incubation period is 5 to 10 days.

Clinical signs. In the **synovitis form** of the disease, symptoms include lameness, swollen joints, breast blisters, weight loss, and decreased feed and water consumption. Symptoms of the **respiratory form** are general respiratory distress and sometimes a green diarrhea. The respiratory form is impossible to differentiate from *M. gal-lisepticum* infection.

Lesions. In the **synovitis form** there is pronounced swelling and infection of the joints. Breast blisters often form because of the pain and difficulty of movement. The liver and spleen may be swollen and there is general dehydration. Lesions of the **respiratory form** may not be easily detected until slaughter. There is usually airsacculitis with exudates, which may cause birds to be condemned at slaughter.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. Definitive diagnosis is based on laboratory tests to differentiate this disease from other *Mycoplasma* infections and from staphylococcal arthritis, infectious tenosynovitis and erysipelas.

Treatment. The disease is treated with antibiotics such as terramycin and streptomycin. These treatments are not always successful, so a veterinarian should be consulted to determine the correct regimen for your flock. Good husbandry practices and the eradication of infected birds will prevent the disease.

Mycoplasma meleagridis **Infection**

Cause. This disease is caused by *Mycoplasma meleagridis*. It has been eradicated in most of the major breeding flocks in the U.S. Other names for this disease include MM, N strain, and H strain.

Susceptibility. Turkeys seem to be the only species of poultry affected by this organism. Turkeys of all ages are susceptible, although young poults tend to be more susceptible than mature birds.

Transmission. The main route of infection is from hen to egg to chick, although there is evidence of lateral transmission and transmission by fomites. Infected birds remain carriers the rest of their lives and tend to be more susceptible to secondary infections.

Clinical signs. Symptoms include general unthriftiness, decreased production, leg abnormalities and respiratory difficulty. Young poults usually have high mortality.

Lesions. Lesions include airsacculitis in newly hatched poults and non-specific reproductive tract infections in adults.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and

lesions. Diagnosis is confirmed in the laboratory, where the organism is easily isolated from the respiratory and reproductive systems, the hock joints, and the Bursa of Fabricius.

Treatment. Antibiotics such as tylosin and tetracycline are used to treat birds. Consult a veterinarian to determine the correct regimen for your flock. Prevent the disease with good husbandry practices and the eradication of infected birds.

***Mycoplasma lowae* Infection**

Cause. The *M. iowae* organism was initially placed in the IJKNQR group of avian mycoplasmas. The disease is common in Europe, but few cases have been reported in the U.S. There have been eradication programs in Europe to halt the spread of the disease in turkeys.

Susceptibility. Turkeys of all ages are affected, although older birds seem to be more resistant than poults or embryos. The disease is rare in chickens, but can occur.

Transmission. The organism is thought to be transmitted primarily from hen to egg to chick.

Clinical signs. Symptoms are not usually seen, as most infected embryos die before hatch. In experimentally inoculated poults, symptoms include retardation of growth, inadequate feathering and skeletal deformities.

Lesions. Skeletal deformities, tenosynovitis, lesions in the air sacs, and a rotated tibia are common in birds that survive to hatch.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and

lesions. Laboratory isolation of the organism confirms the diagnosis.

Treatment. There is no specific treatment for this disease. Antibiotics can be helpful, but should be administered only under the supervision of a licensed veterinarian. To prevent the disease, practice good husbandry and eradicate infected birds when the disease is found.



Bacterial Respiratory Diseases

Avian Tuberculosis

Cause. Serovars 1 and 2 of *Mycobacterium avium* are the typical cause of this disease; however, *M. tuberculosis* has been isolated in some cases. *M. avium* is closely related to the organisms that cause human and bovine tuberculosis. It is a very resistant bacterium that can survive in soil for up to 4 years. Other names for this disease are TB and avian TB.

Susceptibility. Many avian species are susceptible, including chickens, ducks, turkeys, geese, pigeons, canaries, parrots, cranes and crows. Adults more than 1 year old are most often affected, although younger birds can become infected if exposed. Other animals susceptible to this disease are swine, sheep, rabbits, calves and rodents.

Transmission. This disease is spread by bird-to-bird, bird-to-animal, and animal-to-bird contact. Animals that eat infected carcasses also can contract the disease. The incubation period seems to be very long, ranging from several weeks to several months.

Clinical signs. This is a chronic, slow-spreading disease. Symptoms include unthriftiness, weight loss, paleness of wattles and combs, diarrhea, emaciation and death.

Lesions. Gross lesions include yellow granulomas in the liver, spleen and other organs. These lesions confirm a diagnosis, because no other disease produces similar lesions.

Diagnosis. Tentative diagnosis is based on flock history and the presence of symptoms. Definitive diagnosis is based on the presence of granulomas and isolation of the *M. avium* and/or *M. tuberculosis* bacteria in the laboratory.

Treatment. There is no treatment for this disease, although some evidence suggests that the causal organism is susceptible to streptomycin. Infected flocks should be depopulated. To prevent this disease practice good husbandry and sanitation and isolate infected flocks.

Infectious Coryza

Cause. Infectious coryza is caused by the bacterium *Hemophilus paragallinarum*. To survive,

this bacterium requires the substance nicotinamide adenine dinucleotide (NAE), also known as a V-factor. Since this substance is not readily available outside of the host's body, the bacterium will not survive for long outside. To grow a culture in the laboratory, another source of NAE must be supplied. Other names for this disease are roup, cold and coryza.

Susceptibility. Chickens are the main species affected by this disease, although pheasants and guineas are also susceptible. Older chickens are more susceptible than younger chickens.

Transmission. The most common mode of transmission is bird-to-bird contact. However, there is evidence that contaminated water and food may also serve as vectors for this organism. Birds that recover from this disease remain carriers. The incubation period ranges from a few hours to 3 days.

Clinical signs. This acute respiratory infection produces depression, nasal discharge, sneezing, and edema of the face and wattles, along with decreased feed and water consumption and decreased egg production. Mortality is usually about 20 percent, but can be as high as 50 percent. The course of the disease is usually 2 to 3 months.

Lesions. There is edema of the face with exudates that usually emit a foul odor and may cause the eyes to stick shut. There may be some evidence of rales in advanced cases.

Diagnosis. Flock history and the presence of clinical signs and lesions indicate that this disease may be present. Diagnosis is confirmed with laboratory tests.

Treatment. Antibiotics and sulfa drugs are used to treat this disease, although sulfa drugs cannot

be used in layer flocks. Drugs should be administered by a licensed veterinarian. Vaccination, good husbandry, isolation of the flock, and good sanitation can prevent the disease.

Turkey Rhinotracheitis

Cause. Turkey rhinotracheitis is caused by the bacterium *Bordetella avium*, which can survive for up to 6 months in the litter and water lines. Other names for this disease are turkey coryza, bordetellosis, TC and *Bordetella rhinotracheitis*.

Susceptibility. Young turkey poults are most commonly infected, although older turkeys and chickens may catch the disease. Secondary infections can cause a relapse. Birds that recover from this disease remain carriers for several weeks.

Transmission. This disease can be spread from bird to bird, by fomites, and by infected litter. The most common method of transmission is contaminated clothing, shoes and equipment. The incubation period is 7 to 9 days.

Clinical signs. There will be high morbidity in an infected flock. Birds exhibit uneven growth, unthriftiness, rales, sneezing, foamy conjunctivitis, nasal discharge and edema of the face. Mortality can reach 15 percent.

Lesions. Lesions are confined to the upper respiratory tract and include rhinotracheitis with mucoid exudates. There may be secondary infections, since this disease compromises the immune system.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. Definitive diagnosis is based on the isolation of *B. avium* in the laboratory.

Treatment. The disease is resistant to most antibiotics. If antibiotics are given, they should be administered only by a licensed veterinarian. Good husbandry and sanitation can prevent the disease. Infected flocks should be eradicated.



Fungal Respiratory Diseases

Aspergillosis

Cause. This disease is caused primarily by *Aspergillus fumigatus*, but it can be caused by other members of the *Aspergillus* family. This fungus is ubiquitous in nature and grows well at 70 degrees F and above. The fungus produces spores that are very resistant to most elimination methods. Other names for this disease include brooder pneumonia, mycotic pneumonia and fungal pneumonia.

Susceptibility. Birds, animals, humans and plants are susceptible to this disease. Young birds, especially chicks, tend to be more susceptible than older birds. Turkey poults and quail are more susceptible than chickens.

Transmission. This disease is transmitted by inhalation of the fungal spores. For this reason, most infections occur because of contaminated environment, particularly incubation equipment and brooder housing. The disease is not considered highly contagious between birds.

Clinical signs. Aspergillosis has two forms, acute and chronic.

- **Acute**—Symptoms include loss of

appetite, gasping, convulsions and sleepiness. Birds that are highly infected usually die within 2 to 4 weeks. Mortality is usually 5 to 20 percent, but can be as high as 50 percent.

- **Chronic**—This form produces loss of appetite, emaciation, cyanosis, gasping and death.

Lesions. Yellow or gray nodular lesions are found in the respiratory tract and cheesy exudates in the lungs and air sacs. There may be encephalitis if the fungus enters the brain.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. Definitive diagnosis is based on laboratory confirmation of the presence of *A. fumigatus* or other *Aspergillus* species.

Treatment. There is no treatment for this disease. Infected birds should be eradicated. Housing and incubation equipment, including chick boxes, should be thoroughly cleaned and fumigated before repopulation. Prevention is accomplished by good husbandry and sanitation.



Protozoan Diseases

Coccidiosis

Cause. This disease is caused by protozoa of the genus *Eimeria*. There are nine species that infect chickens and seven species that infect turkeys. These organisms are considered host-specific, which means that those species that infect turkeys do not infect chickens, and vice versa. The life cycle of these protozoa is very complex and usually lasts 7 to 9 days. The organisms grow and reproduce within the epithelial lining of the intestine. Another name for this disease is coxy.

Susceptibility. Coccidiosis primarily affects young birds because of the immaturity of their immune systems; however, older birds can become infected. It affects most domestic poultry and other birds. Birds that recover have some immunity, but it does not last unless the birds are in constant contact with the organism. This immunity is specific for the coccidial species, so there is no protection from other species that might infect the bird.

Transmission. This disease is spread via the environment, primarily in litter. It is not passed from bird to bird.

Clinical signs. Unthriftiness, diarrhea, weakness, and decreased food and water consumption are common symptoms. Advanced cases cause high mortality.

Lesions. All coccidial species except *E. truncata* produce lesions in the intestines. These lesions include bloody intestinal contents, enteritis, and a mottled intestinal wall. *E. truncata* affects only geese and causes enlargement of the kidneys.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. Definitive diagnosis is based on the isolation of *Eimeria* oocysts in the laboratory.

Treatment. There are many commercial drugs for treating this disease. All sulfa drugs are effective to varying degrees. Other drugs include amprolium, ionophores, nicarbazin, quinolones and robenidine. Follow the manufacturers' directions when administering any of these drugs. Using a commercial coccidiostat in the feed and practicing good husbandry and sanitation can prevent the disease.

Histomoniasis

Cause. This is another protozoan disease; it is caused by *Histomonas meleagridis*. Other intestinal bacteria, such as *E. coli*, work symbiotically with this organism to produce the blackhead lesions. Other names for this disease are infectious enterohepatitis, enterohepatitis and blackhead.

Susceptibility. Turkeys, peafowl, chickens and gamebirds are susceptible to this disease. Birds less than 12 weeks of age tend to be the most susceptible. Turkeys are more susceptible than chickens.

Transmission. *H. meleagridis* is transmitted within the eggs of the cecal worm *H. gallinae*. Birds usually ingest these eggs when they consume the droppings of infected birds.

Clinical signs. Birds exhibit weakness, drowsiness, decreased feed intake, increased water intake, and yellow feces. Up to 50 percent of birds that become sick at 3 to 12 weeks of age will die.

Lesions. Lesions are located in the ceca, lower intestine and liver. Liver lesions are circular and yellow. Peritonitis develops as the infection progresses.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions. Definitive diagnosis is based on isolation of *H. meleagridis* in the laboratory.

Treatment. Many drugs are effective against this disease. Consult a veterinarian for proper administration. Good husbandry practices and good sanitation can help prevent this disease.

Trichomoniasis

Cause. There are two forms of this disease. *Trichomonas gallinae* causes the disease in the upper GI tract, while *Trichomonas gallinarum* causes disease in the lower GI tract. Other names for this disease are trich, canker in pigeons, and frounce in falcons.

Susceptibility. Most species of fowl are susceptible to this disease except for waterfowl. The disease tends to be more severe in younger birds. The upper GI form is more of a danger to pigeons, doves and raptors, while other birds are more susceptible to the lower GI form.

Transmission. Trichomoniasis is spread by bird-to-bird contact and by contaminated litter, feed and fomites. Birds that recover from this disease remain carriers of the organism.

Clinical signs. Symptoms are similar to those of histomoniasis.

- **Upper form**—depression, empty crop, frequent swallowing, open-mouth breathing and neck stretching
- **Lower form**—depression, unthriftiness, yellow diarrhea, weight loss and death

Lesions.

- **Upper form**—ulcers, cheesy exudates, and areas of necrosis in the upper GI tract (mouth, esophagus, crop). There may also be swelling and nodules in the lungs and liver.
- **Lower form**—yellow cecal droppings, necrosis in the ceca, and enlargement of the ceca. There may be other areas of necrosis in the lower GI tract and nodules in the liver.

Diagnosis. If a flock has a history of this disease and birds show symptoms, a tentative diagnosis can be made. Laboratory tests will isolate either *T. gallinae* or *T. gallinarum* to confirm the disease.

Treatment. Consult with a licensed veterinarian before treating birds. Prevent this disease by practicing good husbandry and proper sanitation, and by eliminating reservoirs of both organisms.

Hexamitiasis

Cause. Hexamitiasis is caused by the protozoan *Hexamita meleagridis* in all bird species affected except pigeons. *Hexamita columbae* causes the disease in pigeons. Both protozoa affect mainly the upper intestine and can be found in duodenal and jejunal crypts. Another name for this disease is infectious catarrhal enteritis.

Susceptibility. Turkeys, pheasants, quail, chukar partridges, peafowl and pigeons are susceptible. The disease usually appears in young birds. Turkeys develop immunity to it by 12 weeks of age. This organism is not known to cause disease in chickens, but chickens can convey the organism.

Transmission. Hexamitiasis can be transmitted from bird to bird, by re-ingestion of contaminated feces, or by contaminated feed and water. Birds that recover remain carriers of the organism.

Clinical signs. Symptoms include watery diarrhea, unthriftiness, ruffled feathers and weight loss even though birds continue to eat. Birds have a generally unkempt look. In advanced cases birds become comatose and die. Mortality can reach 90 percent.

Lesions. The intestinal contents is foamy and watery. There also may be hemorrhaging of the cecal tonsil.

Diagnosis. A history of the disease and the presence of symptoms are the basis of a tentative diagnosis, which is confirmed if *H. meleagridis* (*H. columbae* for pigeons) is isolated in the laboratory.

Treatment. Medications effective against histomoniasis are also effective against this disease. Consult a veterinarian to determine the best drugs and administration methods to use. Prevent the disease by eradicating infected birds and by maintaining good sanitation and husbandry practices.

Plasmodium Infections

Cause. The protozoa that cause this disease are of the genus *Plasmodium*. Species of *Plasmodium* tend to infect specific hosts; however, there is some cross-species infection. There are six species of *Plasmodium* that infect domestic poultry. *Plasmodium* infections are commonly referred to as avian malaria.

Susceptibility. All species of birds and mammals, including humans, are susceptible to infection by this protozoan. Most outbreaks of this disease occur in gamebird and wild bird species.

Transmission. *Plasmodium* species are transmitted by mosquitoes.

Clinical signs. The disease causes severe anemia, weakness, loss of appetite and death.

Lesions. These protozoa attack the red blood cells, so the main lesion is anemia. Infected

birds also may have hemorrhaging just under the skin surface and swelling of the liver and spleen.

Diagnosis. Tentative diagnosis is based on the presence of clinical signs and lesions. Definitive diagnosis is based on laboratory isolation of the *Plasmodium* species.

Treatment. There is no specific treatment for this disease in birds, but some drugs may relieve some of the symptoms. Consult a veterinarian before administering any medication. Controlling mosquitoes will help prevent this disease.

Leucocytozoonosis

Cause. Protozoa of the genus *Leucocytozoon* cause this disease and are generally host-specific. Birds that are raised on range are typically infected with this protozoan. The organism has a complex life cycle. There are no other names for this disease.

Susceptibility. Most species of domestic poultry are susceptible, along with waterfowl and wild turkeys. All ages of birds can contract leucocytozoonosis but it tends to cause more damage in younger birds.

Transmission. Black flies are the most common vector for transmission. One species, *Leucocytozoon caulleryi*, is spread by biting midges. Both of these vectors can infect a new host only within 18 days of picking up the organism; thus, a reservoir of infected birds must be present in the area for new outbreaks to occur. Recovered birds remain carriers of the organism.

Clinical signs. Symptoms are similar to malaria, and include lameness, weakness, anemia, fever and loss of appetite. Birds may have green diarrhea and usually die from internal hemorrhaging. Mortality can be as high as 80 percent.

Lesions. Lesions include anemia, lowered white blood cell count (leucocytosis), and enlargement of the liver and spleen.

Diagnosis. Tentative diagnosis is based on the presence of symptoms and lesions and the presence of infected species within the area. If a *Leucocytozoon* species is isolated from the blood, the diagnosis is confirmed.

Treatment. There are drugs that combat this disease, primarily sulfa drugs. Control black flies and midges to reduce a flock's exposure to the disease organism.

Haemoproteus Infection

Cause. Species from the genus *Haemoproteus* cause this malaria-like infection. Like *Plasmodium* and *Leucocytozoon* infections, these infections tend to be host-specific. The organisms reproduce in the red blood cells and in the endothelial cells of the host. This reproduction in the endothelial cells differentiates this disease from true avian malaria. Another name for this disease is pigeon malaria.

Susceptibility. *Haemoproteus* infection affects mainly pigeons, ducks, doves, quail, songbirds, owls and woodpeckers; other wild birds and reptiles also are susceptible. Pigeons and Muscovy ducks tend to be more susceptible and more affected than the other species mentioned. Domestic fowl usually are not affected.

Transmission. As with true avian malaria and other malaria-like diseases, this disease is spread by biting insects. Flies of the genus *Lynchia* spread the disease in most species; biting midges transmit the disease in ducks.

Clinical signs. Symptoms are weakness, anemia, loss of appetite and death.

Lesions. Anemia and enlargement of the liver and spleen are common. Infected birds may also have hemorrhages in the heart and an accumulation of fluid in the lungs. Muscovy ducks also have enlargement of the kidneys.

Diagnosis. If a flock has a history of this disease and exhibits symptoms, a tentative diagnosis can be made. A definitive diagnosis depends on laboratory isolation of the *Haemoproteus* species.

Treatment. Antimalarial drugs can be used to treat this disease. Controlling insects will help prevent it.

Sarcosporidiosis

Cause. This is a parasitic disease caused by protozoa from the genus *Sarcocystis*. The organism requires a definitive host to complete its life cycle, and the opossum has been identified as the host in one or more of the species of *Sarcocystis* that infect ducks. Another name for this disease is sarco.

Susceptibility. Ducks, reptiles and mammals are susceptible to this disease. Other species of poultry can become infected but do not exhibit symptoms or disease. Wild birds are also susceptible and are more affected by this disease

than domestic poultry, with the exception of ducks.

Transmission. Sarcosporidiosis is spread by the ingestion of feces from infected birds. Areas with large opossum populations are of concern because this animal is the definitive host to species of *Sarcocystis* that infect birds.

Clinical signs. There may be unthriftiness and lameness, but most birds infected with this disease do not show outward symptoms.

Lesions. The protozoan forms cysts in the skeletal muscles of the infected animal. There may also be an enlargement of the spleen.

Diagnosis. Definitive diagnosis is based on the presence of cysts and the identification of the *Sarcocystis* organism.

Treatment. There are drugs for treating this disease. A veterinarian can help you determine the correct drug and dosage. Do not use any drugs that contain arsenic, as this chemical is highly toxic to waterfowl. Control predators and rodents and practice good husbandry.



Internal Parasitic Diseases (non-protozoan)

Roundworms

The most prevalent worm parasite that affects poultry is the common roundworm, or *Ascaridia*. These worms are species-specific, so there is only a slight chance of cross-infection between poultry species. *A. galli* is found in chickens; *A. dissimilis* is found in turkeys; *A. columbae* is found in pigeons; and *A. numidae* is found in guineas.

Life cycle and pathogenesis. The life cycle of these worms is relatively simple and may take as little as 35 days to complete. Adult female worms lay eggs that pass out of the host's intestine with the feces. Eggs hatch to the larval stage while in the litter of the poultry house. The optimum temperature for this is 90 to 93 degrees F. The larvae enter new hosts when chickens eat infested litter. The eggs then hatch while in the ventriculus of the bird and the larvae make their way to the intestine, where they invade the mucosal lining to continue development. As this worm develops within the mucosal lining of the intestine, it causes enteritis that is complicated by hemorrhaging. By 34 days after hatch, the worms are sexually mature and

able to lay eggs of their own. When sexually mature, the worms move out of the mucosal lining of the intestine into the intestinal lumen.

Young poultry are especially susceptible to infestation by *Ascaridia*. Birds will develop immunity by the time they are 2 to 3 months old. Birds infested with roundworms become unthrifty because they have a harder time converting and absorbing food. If the infestation is severe, there may be blockage of the intestine.

Diagnosis and control. Diagnosis is based on a necropsy of suspect birds. There are many deworming medications for treating *Ascaridia*. Be sure to use only wormers approved by USDA and FDA and follow the directions on the product to ensure that the worm's life cycle is disrupted. If there are any questions, consult with a licensed veterinarian. Note that since this worm is transmitted via the litter, there are usually few problems in birds reared on wire; however, flies can be carriers of the eggs, so there is a possibility of infestation.

Cecal Worms

Species from the genus *Heterakis* are commonly referred to as cecal worms. While not as widespread as roundworms, they can cause problems such as unthriftiness and a decrease in appetite and growth. The most common problem with infestation by *Heterakis* is that this worm is part of the life cycle of *Histomonas meleagridis*, which causes blackhead.

Life cycle and pathogenesis. The cecal worm, like the roundworm, is spread by birds ingesting infested feces from the litter. If the cecal worms come into contact with *H. meleagridis*, then this infection will be passed along as well. While infestation with cecal worms does not cause too much of a problem, other than decreased efficiency, the blackhead organism can cause major problems within a flock. There is one exception to the low pathogenicity of cecal worms, and that is infestation of pheasants by *H. isolonche*. This cecal worm may cause up to 50 percent mortality.

Diagnosis and control. Diagnosis is based on a necropsy of suspect birds. There may be some formation of granulomas in the liver. Some drugs will disrupt the life cycle of this parasite. Be sure to use only drugs that are approved by the FDA and USDA, and to precisely follow the manufacturer's instructions. Consult a veterinarian for information not included with the instructions, and for questions about treating blackhead. To prevent this disease, the worm's life cycle must be disrupted and proper sanitation practices used.

Gapeworms

This worm, *Syngamus trachea*, infests the trachea of birds. It is referred to as the gapeworm

because it causes an open-mouthed breathing commonly called "the gapes." This parasite affects many poultry species, including chickens, turkeys, guineas, pheasants and partridges. Gapes in ducks and geese is caused by another organism, *Cyathostoma bronchialis*.

Life cycle and pathogenesis. The life cycle of this worm is very similar to the life cycle of the cecal worm. Most birds become infected when they consume the larval stage of the worm, which is in the litter, or when they ingest earthworms that have eaten the larvae. Female gapeworms lay eggs while in the trachea. These eggs are coughed up and swallowed, and thus pass through the GI tract and are eventually deposited on the litter. Earthworms are an intermediate host of the gapeworm. The larvae hatch within the earthworm and enclose themselves within the muscle tissue. They survive in these enclosures for up to 4½ years. If the infected earthworm is then eaten by poultry, the larvae migrate from the intestine of the bird to the lungs, and finally to the trachea to repeat the life cycle. Clinical signs of infestation with gapeworms include weakness, open-mouthed breathing, grunting caused by labored breathing, and shaking of the head to try to extricate the worms. Many birds die of suffocation as the trachea becomes blocked. This is especially common in young birds, whose wind pipes are smaller.

Diagnosis and control. Diagnosis is based on clinical signs and the presence of the worm within the trachea. This worm is usually not a problem for birds raised in wire cages above the litter. There are drugs available to treat this parasite; however, gapeworms can become resistant to a particular drug if it used for a long period of time. Therefore, consult a veterinarian before

administering any de-worming drugs. To prevent gapeworm infestation, keep facilities clean, practice good husbandry, and remove earthworms from the grow-out area.

Capillaria Worms

At present, seven species of worms from the genus *Capillaria* are known to affect domestic poultry and gamebirds. Those that affect the crop and esophagus are *C. annulata* and *C. contorta*. Species that affect the small intestine are *C. obsignata*, *C. bursata*, *C. caudinflata* and *C. columbae*. *C. retusa* affects the ceca of the bird. Birds infected with this parasite quickly become culls.

Life cycle and pathogenesis. *Capillaria* worms can have a life cycle either with or without an intermediate host, depending on the worm species. The worms are spread through the flock by the ingestion of litter that contains worm eggs. The eggs must be in the embryonic stage to be infective; this stage is reached 24 to 30 days after the eggs are deposited onto the litter. The main intermediate host, if there is one, is the earthworm. Clinical signs of infestation with *Capillaria* worms include diarrhea, emaciation, hemorrhagic enteritis, decreased performance, mottled yolks and death. If the infection is located in the crop, the crop will be devoid of feed, have excess mucus, and show a thickening of the crop wall. If the infection is in the intestine or cecum, there will be inflammation of the area but usually no other visible lesions. In some cases there is hemorrhage in infested parts of the body.

Diagnosis and control. Tentative diagnosis is based on clinical signs. Diagnosis is confirmed if there are lesions and *Capillaria* worms are

found in the GI tract. There is no good treatment for infestation by this parasite, although some antibiotics may be of use. Consult a veterinarian. Keep the grow-out area clean and free of earthworms.

Tapeworms

Tapeworms are flattened, ribbon-shaped, segmented worms. There are many species of the genus *Raillietina* that infect poultry; however, not all tapeworms belong to this genus. Tapeworms can be very short ($\frac{1}{8}$ inch) or very long (12 inches). They grow by adding new segments. When these segments mature, they break off and are passed into the litter and eaten by an intermediate host. Poultry ingest the tapeworm by eating the intermediate host.

Life cycle and pathogenesis. All tapeworms that affect poultry have an indirect life cycle. They must have an intermediate host, examples of which include snails, slugs, beetles, earthworms, grasshoppers, flies and other insects. After a segment of tapeworm is passed out of the primary host, it is ingested by the intermediate host. The segment contains immature larvae. These larvae mature to a certain point while in the intermediate host, but must return to the primary host to complete the life cycle. Usually this occurs when the intermediate host is consumed by the primary host. Once this happens, the tapeworm will attach itself to the interior wall of the intestine and begin producing new segments. Tapeworms absorb nutrients from the host but cause no visible lesions or damage to the intestine. Clinical signs of infestation include unthriftiness and weight loss with no decrease in appetite.

Diagnosis and control. Definitive diagnosis is based on finding tapeworm segments in the litter or in the bird's intestine. There are many drugs for controlling tapeworms once they have infested a flock. Consult with a veterinarian to determine the best treatment. Intermediate hosts should be eradicated. Since most tapeworms rely on only a few intermediate hosts, the species of tapeworm should be identified so that proper insecticides can be used.



External Parasitic Diseases

Lice

More than 40 species of chewing lice can infest poultry. Domestic poultry are not infested by sucking lice, but gamebirds and wild birds can have either variety.

Life cycle and pathogenesis. Lice are wingless insects. The females lay 50 to 300 eggs, which they bond to the feathers of the bird. Louse eggs are usually laid in clusters, so are readily visible. Eggs hatch from a few days to a couple of weeks after being laid. It takes 4 to 6 weeks for the immature louse to develop fully, thus allowing for many generations to hatch and become mature within a year. Lice usually spend their entire life cycle on one bird, but may move (usually to a younger bird) if their population becomes too dense. Lice feed on the scales, feathers and scabs on the skin. Some ingest blood if it is readily available from a tear in the skin. Lice irritate the bird's skin, causing it to peck and scratch. This can cause tears in the skin and reduce productivity. Infested birds have an unkempt appearance and ruffled feathers. Egg production in heavily infested flocks may be decreased by 10 percent to 20 percent.

Diagnosis and control. Diagnosis is based on the appearance of the birds, increases in pecking and scratching, and by finding louse eggs on the feathers. Lice are susceptible to most insecticides but only a few products can be used on poultry. These insecticides can be applied as dusts or as sprays. Good husbandry and sanitation practices can prevent problems with lice.

Chicken Mite

Chicken mites (*Dermanyssus gallina*) are common in backyard flocks but not in commercial flocks, because of the differences in management. They are true mites—members of the arachnid, or spider, family. Thus, they have eight legs and are easily distinguished from lice, which are insects with six legs. Chickens are the favored host for this parasite; however, turkeys, pigeons and wild birds can become infested. Other names for this mite are red mite, gray mite and roost mite.

Life cycle and pathogenesis. Chicken mites feed on the blood of the host, so after feeding they will appear red. Unlike lice, chicken mites

do not live continuously on the animal they parasitize. Instead, they live in cracks and crevices in the ceiling, walls and floors of the poultry house. Chicken mites feed at night and so are not usually seen on the birds during the day. Female chicken mites will feed just before laying eggs. The eggs hatch into nymphs within 2 to 3 days. After another 1 to 2 days, the nymphs molt and begin to feed. The entire life cycle can take as little as 10 days, so many generations may be hatched within a year. If the poultry house is unheated, the mites will enter a dormant stage for the winter. If the house is heated, the mites will stay active year round. They are also able to enter a dormant stage for up to 5 months if the house is empty. Because these mites feed on blood, the clinical signs for infestation include anemia, weakness and general malaise. Production in laying hens and feed conversion will suffer as well. Other diseases are reportedly vectored by the chicken mite, including fowl cholera and the spirochete *Borrelia anserina*.

Diagnosis and control. Tentative diagnosis is based on the presence of clinical signs. Definitive diagnosis is based on isolating and identifying the mite. Chicken mites must be differentiated from northern fowl mites before control measures are undertaken. The infested house should first be cleaned and sanitized; then an approved miticide should be applied. Good husbandry and sanitation practices can prevent a mite infestation.

Northern Fowl Mite

This mite (*Ornithonyssus sylvarium*) is the most common mite affecting the poultry industry. Unlike the chicken mite, this mite is found in both backyard flocks and commercial opera-

tions, which makes it very important economically. The northern fowl mite can affect chickens, turkeys and most gamebirds, particularly pheasants. There is particular concern in caged-layer complexes. Also unlike the chicken mite, this parasite will feed during the day and at night. These mites are arachnids, so they are distinguishable from insect infestations.

Life cycle and pathogenesis. The northern fowl mite has a very short life cycle, usually less than 1 week. It spends its entire life on the host. Eggs are deposited on the feathers and hatch within 24 hours. The mite reaches maturity just 4 days after hatch and starts the life cycle over again. This short life cycle means that infestations can reach very high numbers within a short time. The mite can survive away from the host for 3 to 4 weeks. Infestations are more severe in the colder months and may actually decline during the summer. The northern fowl mite feeds on blood. Large infestations cause anemia very quickly and the anemia may be severe enough to kill the birds. Other signs of infestation include reduced production, lowered immunity, weight loss and soiled vent.

Diagnosis and control. Accurate identification is the key to controlling this parasite. The mites can be readily seen on the vent area of the bird. Consult an entomologist for help in identifying them. Pesticides must be used to control infestations and there are many available. Follow instructions and rotate the use of pesticides, as mites can become resistant. Most pesticides that control this mite must be applied to the bird as a wettable powder or an emulsion spray. A veterinarian or entomologist can help you determine the most effective treatment regimen.

Chigger Mite

Chiggers (*Neoschongastia americana*) affect birds raised on open range, such as turkeys and gamebirds. They are not usually a problem in commercial chicken operations. Mature chiggers are covered with hair that gives them a silky appearance. Larvae have less hair and may be red in color. These parasites feed in groups on many areas of the bird and produce red, scabby lesions that take about 3 weeks to heal.

Life cycle and pathogenesis. Chiggers feed only in the larval stage and they feed only once. The chigger feeds by injecting an enzyme into the host that digests surrounding tissue. It then takes up the digested tissue. A larva may feed from 1 day to 1 month. It then drops to the ground and matures to the adult stage. Newly hatched larvae live in soil and vegetation waiting for a host to come by. Chigger bites become itchy and slightly inflamed, which causes the bird to scratch and peck and tear the skin. Such birds may be downgraded at processing.

Diagnosis and control. Diagnosis is based on the presence of the chigger larvae on the skin of the bird, usually the thighs, breasts, vent area and undersides of the wings. There is no specific treatment, since the birds that contract this parasite are raised on range. The range can be treated with pesticide, however. Consult a veterinarian and an entomologist before attempting to apply pesticide to the entire range.

Scaly Leg Mite

This mite burrows under the scales on the feet of poultry. It usually affects wild birds and is not typically a problem in commercial flocks.

Life cycle and pathogenesis. Scaly leg mites begin laying eggs very soon after burrowing under the scales. Egg laying continues for approximately 2 months. The eggs hatch in about 5 days and the life cycle is completed in about 2 weeks. The mites' burrowing causes itching and irritation. The scales become raised and may come loose, causing scabs to form. This can also cause blood loss and red blotches on the legs. Eventually, infested birds become lame, lose their appetite, show a decrease in production, and die.

Diagnosis and control. A tentative diagnosis can be made if birds have raised scales. Definitive diagnosis is based on identifying the mite under a microscope. The most common control method is to dip the birds up to the shanks in diesel fuel or kerosene. You should consult with a veterinarian and an entomologist before performing this or any other type of control measure. Scaly leg mites can be prevented with good husbandry and sanitation practices.

Bed Bug, Fowl Tick and Black Fly

Bed bug. This common external parasite of poultry lives, breeds and lays eggs in nests and in the walls and roofs of the poultry-rearing area. Bed bugs feed on the blood of the host and feed only at night. They prefer a warm environment, so are not usually a problem during winter except in heated facilities. Although infestations usually cause anemia in birds, this parasite may go unnoticed until people who are caring for the birds develop welts and itching from bed bug bites.

Fowl tick. This parasite is also referred to as the blue bug or chicken tick. It is only rarely associ-

ated with poultry today. Like other ticks, it feeds on the host's blood, generally at night. Large infestations can cause anemia, decreased appetite and death. Fowl ticks can also vector fowl spirochaetosis, which is fatal to chickens, turkeys, geese and guineas. The life cycle of this parasite takes about 40 days, but the tick can live for as long as 2 years without feeding. Eradication is difficult, so an entomologist and a veterinarian should be consulted before attempting control measures.

Black fly. Black flies also feed on the blood of the host. They are important because they can carry and transmit the organism that causes leucocytozoonosis. The life cycle of these parasites requires swiftly moving water, so they are not usually a problem unless there is a stream or river near the grow-out area.



Nutritional and Metabolic Diseases

Rickets

Cause. Rickets is the insufficient calcification of bone caused by a deficiency of calcium, phosphorus or vitamin D₃. Calcium deficiency is caused by a lack of this mineral in the feed, or by a deficiency of vitamin D₃ that prevents the bird from absorbing the calcium it ingests. Phosphorus deficiency produces the same results.

Susceptibility. Young poultry, often 6 weeks of age or younger, are affected by this disease.

Clinical signs and lesions. Rickets causes abnormal skeletal development. Leg and long bones are soft and springy. If it is caused by calcium or vitamin D₃ deficiency, birds will become paralyzed in advanced stages of the disease. If it is caused by phosphorus deficiency, birds do not become paralyzed.

Diagnosis. Diagnosis is based on feeding history, feed analysis, and the presence of clinical signs and lesions. In vitamin D₃ or calcium deficiency, the epiphyseal plate of long bones is abnormal or wide. In a deficiency of phospho-

rus, the epiphyseal plate is thin to normal. Rickets-like symptoms also have been seen in birds with infectious bursal disease. Therefore, it is important to have the feed analyzed to determine whether the condition is secondary or caused by improper feed formulation.

Treatment. Rickets is treated by correcting feed-related problems to make sure birds have adequate amounts of the minerals they need. After the feed inadequacy has been addressed, water-soluble vitamins containing vitamin D₃ also can be given for 3 to 7 days. Moldy feed or feed with mycotoxins should not be fed to birds.

Osteomalacia and Caged-Layer Fatigue

Cause. Osteomalacia is rickets in adult birds. It is usually caused by a temporary deficiency of calcium during high production (high production = 80 percent or higher). If a hen is not receiving enough calcium from the diet for eggshell formation, she will take the calcium from her bones. The bones then become soft,

especially the femur. The femur may break, resulting in a bird that cannot stand and is thus suffering from caged-layer fatigue. Caged-layer fatigue is a disease found only in caged layer hens. The exact cause is not known, but a disturbance to the hen's mineral/electrolyte balance is the main suspect.

Susceptibility. Older hens are susceptible to this condition.

Clinical signs and lesions. Soft bones and leg breaks are the common signs of this disease.

Diagnosis. Diagnosis is based on feeding history, feed analysis, and the presence of typical clinical signs and lesions.

Treatment. Calcium should be added to the feed as a top dressing for several days. These large particles will be picked up by the hens without their consuming extra feed. Osteomalacia can be prevented by feeding a proper diet. Caged-layer fatigue may be caused by the lack of exercise that single hens have when caged. Hens in multiple-hen cages do not suffer from this condition because they get exercise when competing for food and water.

Vitamin A Deficiency

Cause. Young poultry require vitamin A (as well as other vitamins) for the proper growth and repair of epithelial tissue, one of the basic components of the skin, mucous membranes and glandular tissue that are the bird's initial defense against disease. If the starter feed is deficient in this nutrient, the chicks will be stunted and more susceptible to disease. Another name for this disease is nutritional roup.

Susceptibility. Both chicks and turkey poults are susceptible, but turkey poults tend to be more susceptible than chicks.

Clinical signs and lesions. Signs and lesions include droopiness, general unthriftiness, swelling of the eyes, nasal discharges, keratinization of epithelial tissues, and urate deposits in the kidneys, ureter and cloaca.

Diagnosis. Flock history and the presence of clinical signs and lesions indicate the possibility of vitamin A deficiency. Feed analysis will confirm the diagnosis.

Treatment. This condition is treated by feeding a correct formulation. In addition, a water-soluble vitamin A supplement should be added to the drinking water for 5 to 7 days. If severe deficiency has caused kidney damage, this treatment may not be 100 percent effective. Birds stressed by other diseases may need more vitamin A than the recommended minimum and should receive a supplement.

Perosis

Cause. Perosis is caused by a deficiency of the mineral manganese (Mn). Choline, niacin and biotin are also involved. Another name for this deficiency is slipped tendon.

Susceptibility. All young poultry are susceptible to this deficiency.

Clinical signs and lesions. Manganese deficiency causes deformation of the leg and leg weakness in young poultry. There is a flattening and enlargement of the hock area, which is followed by slipping and rotation of the Achilles tendon out of the hock joint. This causes the

shank and foot to remain flexed to the point that the foot will extend away from the body of the bird. A decrease in egg production, along with thinner shells and decreased hatchability, are also common. Breeder flocks with a manganese-deficient diet will have an increase in embryonic death.

Diagnosis. A tentative diagnosis can be made on the basis of clinical signs and lesions. Definitive diagnosis is made by feed analysis.

Treatment. Affected birds cannot be helped, but increasing the amount of manganese in the feed or adding a supplement to drinking water will prevent more birds from becoming affected. In general, poultry require 35 to 50 ppm of manganese in the feed. Supplements that increase manganese up to 75 ppm can be offered. Manganese concentrations of 100 ppm or more are not economical.

Vitamin B₁ Deficiency

Cause. Vitamin B₁ is also known as thiamine. Overuse of amprolium, a coccidiostat, will produce a temporary deficiency, as it competes with thiamine in biochemical reactions. Another name for this condition is polyneuritis.

Susceptibility. All poultry are susceptible to this deficiency.

Clinical signs and lesions. Short periods of vitamin B₁ deficiency cause extreme excitability, flightiness and loss of appetite. Long periods of deficiency cause degeneration of the nervous system, which produces lameness that cannot be treated and eventual muscle paralysis.

Diagnosis. If the clinical signs and lesions are present, a tentative diagnosis can be made. Definitive diagnosis is based on feed analysis.

Treatment. Supplementary thiamine added to the feed and drinking water will eliminate symptoms if the deficiency has not caused the birds to lose all appetite. If the deficiency is severe enough to cause loss of appetite, birds will need thiamine injections to cause them to resume eating. If the cause of the deficiency is overuse of amprolium, the amount used should be reduced to an acceptable level.

Vitamin B₂ Deficiency

Cause. Vitamin B₂ is also known as riboflavin. It is an essential vitamin, but there are only a few natural sources of this nutrient. Another name for this deficiency is curled toe paralysis.

Susceptibility. All young poultry are susceptible to this deficiency.

Clinical signs and lesions. A vitamin B₂ deficiency causes the toes of young birds to curl inward toward the center line of the body. One or both feet may have curled toes. The curling is most visible when the legs are extended. Other symptoms are diarrhea, stunting and high mortality. If the riboflavin deficiency is severe, birds usually begin dying at 8 to 10 days of age, before the toes begin to curl. Turkeys brooded with infrared brooders also have curling of the toes, but not because of riboflavin deficiency; in this case the curling is not correctable or preventable by supplementation.

Diagnosis. Riboflavin deficiency would be suspected if birds have curling toes and other symptoms. Feed analysis will confirm it.

Treatment. Starter feeds are usually fortified with riboflavin. If more than five in 1,000 birds exhibit symptoms, additional riboflavin should be given via water-soluble vitamins in the drinking water. Feed can be reformulated to prevent this condition.

Vitamin E Deficiencies

A deficiency of vitamin E causes or contributes to three diseases in young chickens. It is the primary cause of encephalomalacia, and is a secondary contributor to exudative diathesis and nutritional muscular dystrophy.

Encephalomalacia. This condition is often called "crazy chick disease." It is characterized by incoordination, stumbling, staggering and paralysis. The deficiency causes degeneration of the cerebellum, or coordination center, of the brain. Vitamin E is an antioxidant and prevents damage to the cerebellum by toxic peroxides that are formed during the digestive process. The presence of clinical signs is enough evidence for a tentative diagnosis. If there are lesions in the cerebellum, the diagnosis is confirmed. Chicks affected by this condition are not treatable, but increasing the vitamin E in the diet will prevent new cases.

Exudative diathesis. This condition is caused by deficiencies in both vitamin E and selenium and occurs mainly in birds 5 to 8 weeks old. It causes capillaries to become permeable, which leads to edema and swelling. The severe edema causes the skin to weep, especially under the wings and on the inner surface of the thigh. This weepage causes scabs to form and often leads to a gangrenous condition. Tentative diagnosis is based on clinical signs; definitive diagnosis is based on feed analysis. To correct this

condition, selenium and vitamin E should be added to the feed to recommended levels.

Nutritional muscular dystrophy. This condition is caused by a combined deficiency of vitamin E and cystine. It causes the deterioration of breast and leg muscles. This condition is rare and does not usually occur under normal rearing conditions. It can be treated by adding vitamin E and cystine to the diet in recommended amounts.

Fatty Liver Syndrome

Cause. The primary cause is believed to be excessive feed intake, but because fatty liver syndrome affects only caged layers, a lack of exercise could contribute. There may be a hereditary factor in some leghorn strains. Aflatoxin intake also has been shown to increase fat in hens.

Susceptibility. Caged layer hens are susceptible to this condition, with high-production birds affected the most.

Clinical signs and lesions. Obesity and an enlarged, fatty liver are the most common symptoms. Deposits of fat also may be found along the viscera, in the lower abdomen, and under the skin. Fatty deposits along the pelvic area require the hen to strain more when laying, and the enlargement of the liver makes it susceptible to injury. When birds die from this condition it is almost always because the liver ruptures and hemorrhages.

Diagnosis. Caged layers with clinical signs and lesions should be suspected of having this condition.

Treatment. If the cause of the condition is the presence of aflatoxin, the affected feed should be replaced. If aflatoxin ingestion is not the cause, switching to a lower energy feedstuff is required. This can be accomplished by replacing approximately 15 percent of the corn in the diet with a low-energy feedstuff. Additional treatments include fortifying the feed with choline, vitamin B₁₂ and vitamin E.



Miscellaneous Diseases

Viral Arthritis

Cause. This disease is caused by an RNA reovirus. Several serotypes have been identified. Other names for this disease are infectious tenosynovitis, VA, teno, ruptured tendon, RT, reovirus enteritis and reovirus septicemia.

Susceptibility. Chickens seem to be the only poultry affected by this condition. All ages and breeds of chickens are susceptible.

Transmission. This disease is highly infectious. The virus is spread by direct bird-to-bird contact, either by fecal-oral or respiratory routes. The possibility of egg transmission has been suggested, but at this time there is no direct evidence of it.

Clinical signs. In the tenosynovitis form of this disease, the main symptoms are lameness and swelling of the area around the shank and foot. In the septicemic form of the disease, there is increased mortality and a drop in production, along with some effects on the joints.

Lesions. In older birds (such as roasters and breeders) with the tenosynovitis form, there may be tears and ruptures of the gastrocnemius tendon. In the septicemic form, there is cyanosis, hemorrhage, peritonitis and vasocongestion. There may also be swelling of the liver, spleen and kidneys.

Diagnosis. Tentative diagnosis can be made on the basis of flock history and the presence of clinical signs and lesions. A definitive diagnosis is made if the reovirus is isolated in the laboratory.

Treatment. There is no treatment for this condition. However, there is a vaccine that can be used in areas with previous or continued outbreaks. Along with vaccination, the housing facility should be completely sanitized and fumigated before it is repopulated.

Hemorrhagic Enteritis

Cause. This disease is caused by a type II adenovirus. Other names for it are HE and bankruptcy gut.

Susceptibility. Turkeys 4 to 13 weeks old are susceptible.

Transmission. The virus is introduced into the bird by the ingestion of infected litter. The incubation period is 2 to 6 days; after incubation, the virus replicates in the spleen of the infected bird.

Clinical signs. This disease is an acute intestinal disorder that causes paleness, drowsiness and increased mortality. Birds may not exhibit any symptoms before death occurs. The disease usually runs its course in 3 weeks, but the virus will remain viable in the rearing area or house for several months.

Lesions. Inflammation, hemorrhage and necrosis of the intestinal tract can be seen. There may also be invasive lesions in the liver and spleen.

Diagnosis. Tentative diagnosis is based on the presence of clinical signs and lesions. Definitive diagnosis is based on the isolation of the adenovirus in the laboratory.

Treatment. Antibiotics can be administered to control secondary infection. There is a vaccine that will prevent this disease. Vaccination should be combined with good husbandry and sanitation practices.

Hemorrhagic Anemia Syndrome

Cause. The direct cause is unknown, but microbial toxins, nutrient deficiency, drug toxicity, or a combination of these are all suspected. Other names for this disease are hemorrhagic disease, aplastic anemia and hemorrhagic syndrome.

Susceptibility. Chickens 3 to 15 weeks old are susceptible, with most cases occurring between 3 and 7 weeks.

Transmission. The mode of transmission is unknown.

Clinical signs. Symptoms include diarrhea, weakness, depression, paleness, ruffled feathers, weight loss and death. Mortality can be as high as 40 percent.

Lesions. There may be massive hemorrhaging in many muscles and organs, along with anemia.

Diagnosis. There is no definitive diagnosis because the cause is unknown. Presumptive diagnosis is based on flock history and the presence of clinical signs and lesions.

Treatment. There is no specific treatment for this disease, and no known means of prevention.

Candidiasis

Cause. The cause of this disease is *Candida albicans*. This yeast organism resides in the soil and is resistant to many disinfectants. Colonies grown on media will emit an odor similar to baking yeast. Other names for this disease include thrush and vent gleet.

Susceptibility. All poultry species of all ages are susceptible to infection by this organism.

Transmission. The yeast can be ingested when birds come in contact with contaminated feed, water, equipment or litter.

Clinical signs. Symptoms include ruffled feathers, paleness, anemia, a white crust around the vent, and stunting of growth.

Lesions. This disease affects the digestive tract. It causes white, thickened areas in the crop and proventriculus; inflammation of the vent area; and pits or erosions in the gizzard.

Diagnosis. If there is a history of this disease in a flock and birds have clinical signs and lesions, this disease can be tentatively diagnosed. It is confirmed by isolating *Candida albicans* in the laboratory.

Treatment. There are many anti-fungal and anti-mycotic drugs that can be used. Consult a veterinarian before starting any drug regimen. Copper sulfate is one of the compounds used to treat this disease. However, even small amounts of copper can be toxic to ruminant animals, so treated litter should not be used as fertilizer in areas where ruminants will be foraging. This disease can be prevented by adding mold-inhibiting agents to feed and sanitizers to water.

Mycotoxicosis

Cause. This condition is caused by the intake of mycotoxins—toxic substances produced by molds. There are many types of molds that produce these substances, but there are three main groups that concern poultry production. These are aflatoxins, ochratoxins and fusariotoxins. Aflatoxins are produced by *Aspergillus flavus*; ochratoxins are produced by *Aspergillus ochraceus*; and fusariotoxins are produced by many different species of *Fusarium* molds. All of these molds grow on the typical feed ingredients used to make poultry feed. Damp, cool weather usually exacerbates the growth of

molds, increasing the amount of mycotoxin produced.

Susceptibility. Poultry of all ages are susceptible, as are other animals (including humans).

Transmission. Mycotoxins are ingested with feed.

Clinical signs and lesions. There are a variety of symptoms and lesions associated with the different types of mycotoxins; however, most birds exhibit unthriftiness, drops in production, weight gain and paleness.

Diagnosis. Tentative diagnosis is based on flock history and the presence of clinical signs and lesions in the absence of any other identifiable disease. Definitive diagnosis is based on isolating the particular mold in the laboratory. Consult your veterinarian if you think your flock has ingested mycotoxins.

Treatment. The first step in treatment is to remove the source of the mycotoxin—the contaminated feed. Birds will improve almost immediately. Anti-fungal drugs may be administered, along with vitamins A, D, E and K. Mycotoxicosis can be prevented by testing feed ingredients frequently and observing the flock closely. Birds kept in confined housing without access to outside feeding areas have only one source of the toxin. Birds on range may come in contact with areas where these molds grow. Using an anti-fungal or anti-mycotic product in these areas will be helpful.



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Airsacculitis

Exudates
in air sacs

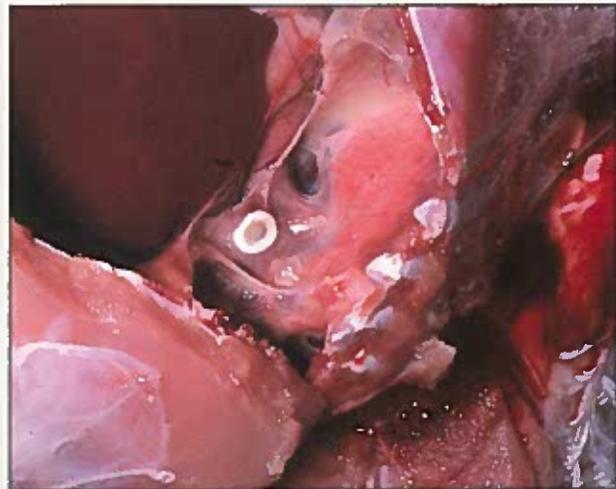
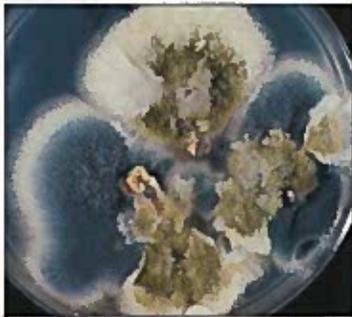


Aspergillosis

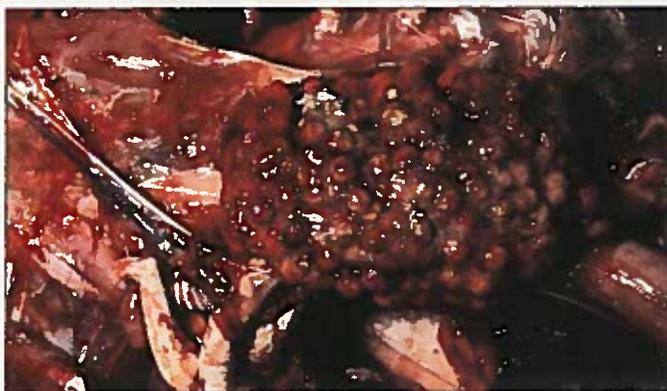


Fungal growth
in the spleen

Aspergillus flavus
growth in a
Petri dish



Fungal growth on internal organs



Fungal nodules in a turkey



Fungal growth in the lungs

Avian Influenza



Unsanitary conditions and death from Avian influenza



Listlessness and depression



Micrograph of avian influenza virus



Disposal of carcasses

Avian Leucosis

Lymphoid Leucosis



Tumors on internal organs



Tumors on liver



Small foci in liver



Discoloration and foci in liver

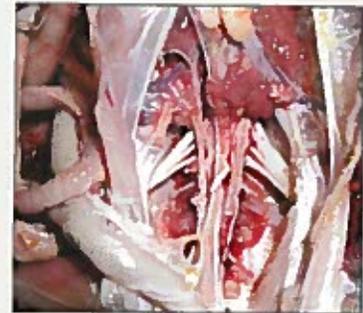
Marek's Disease



Lameness caused by Marek's disease



Skin lesions



Enlarged left pelvic nerve plexus



Skin tumors



Lameness and depression

Avian Pox



Avian pox lesions on a turkey's head



Avian pox lesions

Diphtheritic
mouth lesions
from quail pox



Swollen head from fowl pox



Pox lesions on neck

Pox lesions
on foot



Botulism

Depression and
listlessness



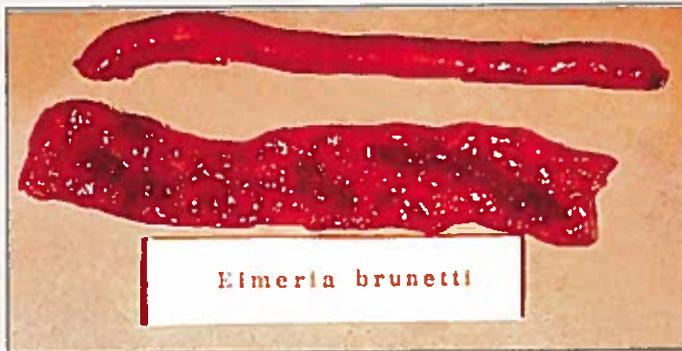
Depression



Coccidiosis



Bloody and watery intestinal con-
tents



Hemorrhagic
intestine

Eimeria brunetti

Bloody
intestinal
contents



Eimeria necatrix



E. acervulina

Foci on outer surface of the intestine

Coccidiosis cont.



Oocyst



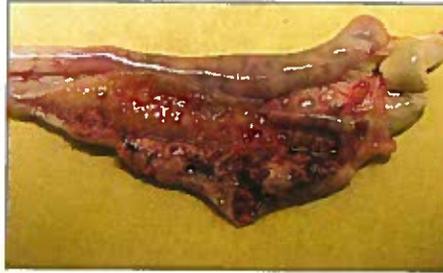
Necrotic ceca



Hemorrhagic intestine



Swollen intestine



Hemorrhagic intestine with lesions



Swollen ceca



Hemorrhagic cecum

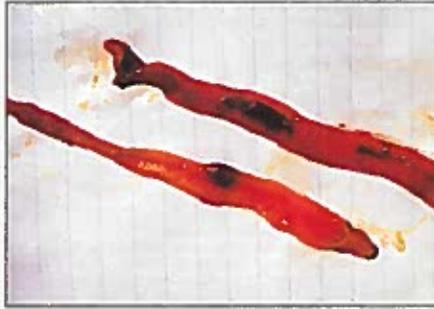
Erysipelas



Hemorrhagic and swollen skin

Exotic Newcastle Disease

Bloody intestine



Watery and bloody digestive contents



Hemorrhagic digestive tract



Watery and hemorrhagic intestines



Swollen ventriculus



Mass death in a poultry house



Hemorrhage in cecal tonsil



Hemorrhage in larynx with ulcers



Ulceration at esophageal-proventricular junction



Hemorrhage in proventriculus

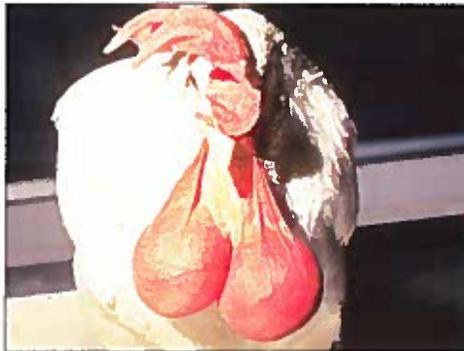
Fowl Cholera



Swollen face and wattles



Swollen face and wattles in a broiler breeder



Extensively swollen wattles

Gangrenous Dermatitis



Black necrotic skin



Necrotic and hemorrhagic skin



Emaciation and hemorrhage

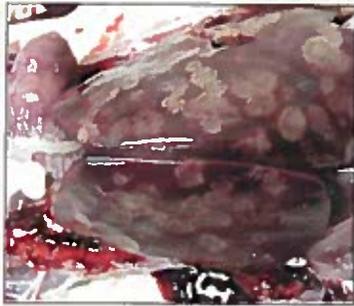
Histomoniasis (Blackhead)



Foci on liver



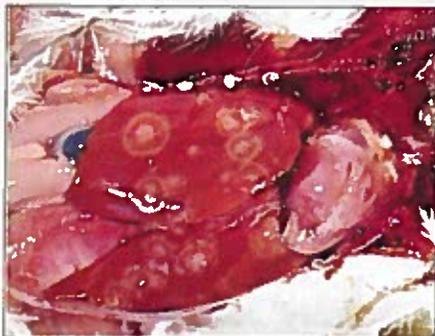
Weakness and depression caused by histomoniasis



Foci and lesions on liver



Caseous cecal core and ulceration of mucosa



Foci on liver



Enlarged and hemorrhagic ceca



Enlarged ceca

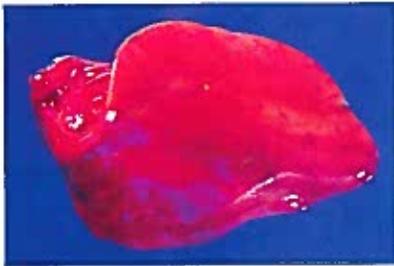
Inclusion Body Hepatitis



Dark inclusion bodies with swelling



Swollen and discolored liver



Swollen liver with inclusion bodies



Swollen and discolored liver

Infectious Bronchitis

Swelling in facial area with watery discharge from eyes



Hemorrhagic lungs

Misshapen and soft-shelled eggs



Underdeveloped yolks in a breeder hen

Infectious Bursal Disease



Depression and unkempt appearance



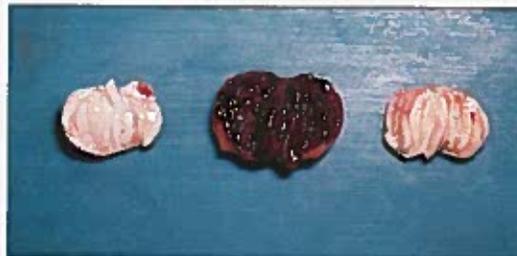
Yellow colored Bursa of Fabricius



Swollen Bursa of Fabricius



Swollen kidneys



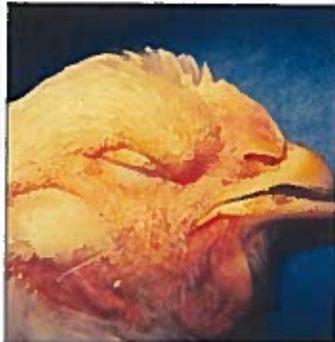
Swollen and hemorrhagic Bursa of Fabricius

Infectious Coryza

Cheesy nasal discharge



Swelling of face and wattles



Extreme swelling of face, comb and wattles

Laryngotracheitis



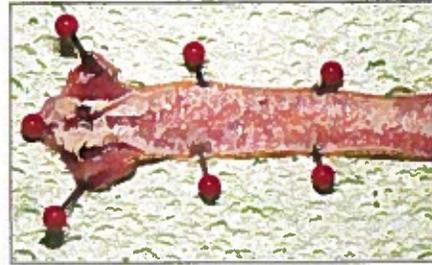
Bloody and swollen airway



Bloody and exudative trachea



CAM inoculation with LT

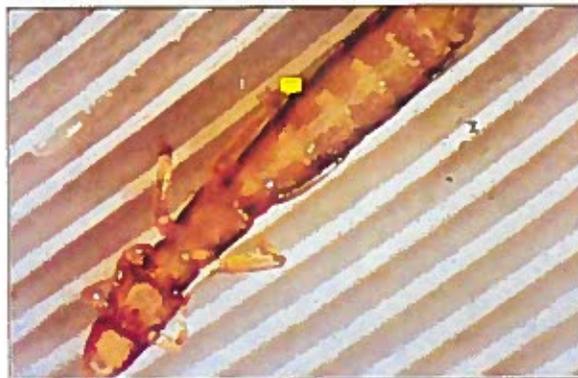


Swollen trachea



Bloody trachea

Lice



Typical louse

Mycoplasma gallisepticum



Sinusitis



Airsacculitis



Swollen sinus with edema



Swollen sinus



Swollen sinus with exudates

Mycoplasma synoviae



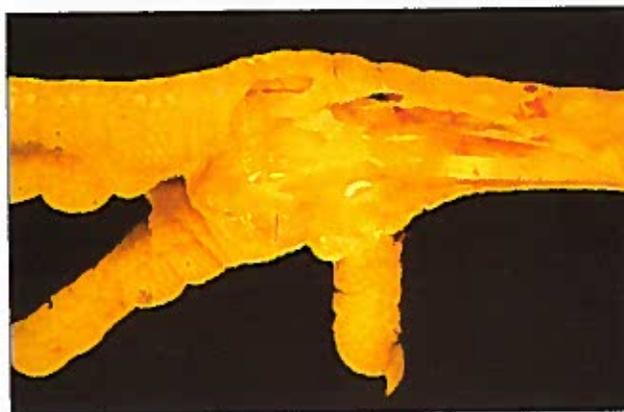
Swollen joint



Sinusitis



Swollen joint



Swollen foot joints

Necrotic Enteritis



Dilated intestine with rough serosa



Dilated lower small intestine



Intestinal mucosa with diphtheritic pseudo-membrane



Rough intestinal mucosal layer



Green diarrhea



Bloody intestinal mucosa

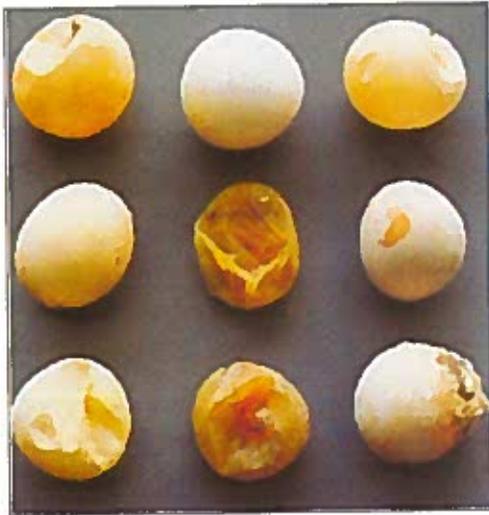


Intestinal mucosa with diphtheritic pseudo-membrane

Newcastle Disease



Depression and discomfort



Misshapen and soft-shelled eggs



Depression



Exudates in air sacs



Swelling of face



Misshapen turkey eggs

Omphalitis



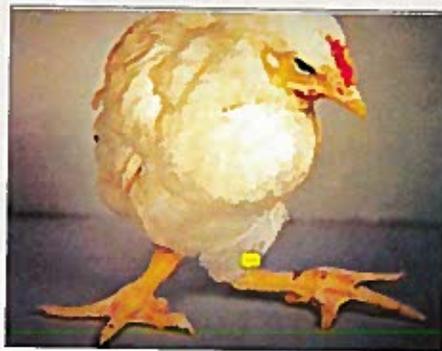
**Swollen
intestinal
area**

**Swollen
navel area**



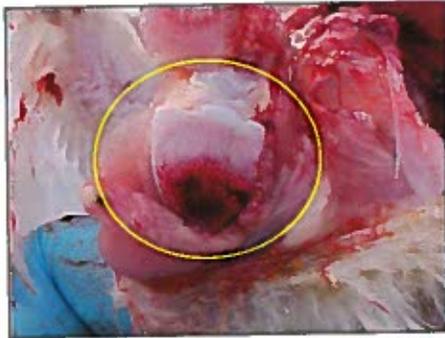
Hemorrhagic navel area

Perosis

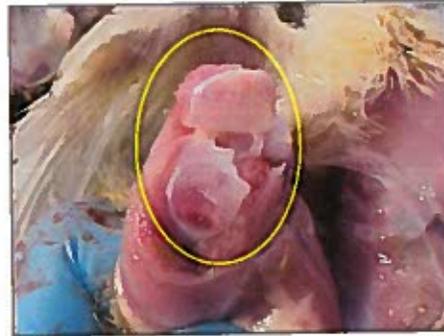


**Typical leg posture for perosis (one or
both legs)**

Rickets



Soft bone growth plates



Cartilaginous growth plate area



Soft bone growth area



Collapsed rib cage with enlarged costochondral junction



Rubbery beak

***Staphylococcus aureus* Infection**

(Bumblefoot)



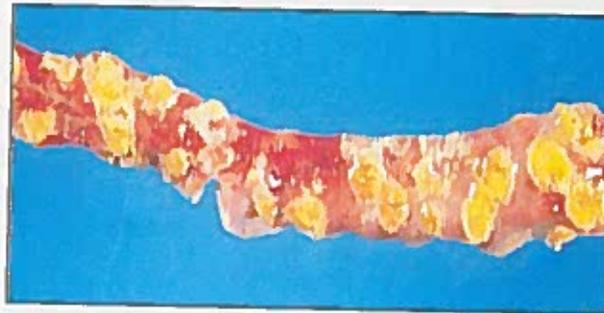
Swollen foot area



Swollen feet

Ulcerative Enteritis

**Ulcers and
hemorrhage
in intestine**



**Ulcerative
intestine**

Vitamin Deficiency

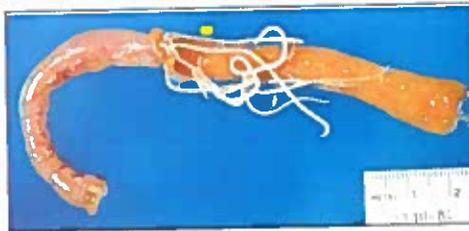


Curled toes (paralysis)



Curled toes

Worms



Roundworms (Ascarids) in intestine



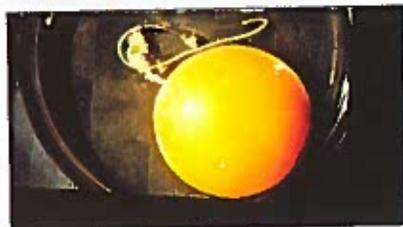
Roundworms



Tapeworm (note segmentation)



Gapeworm in intestine



Roundworm in egg



Appendix B: Glossary

Active immunity – immunity or resistance to disease that has been acquired by host response to a disease agent. It can be acquired by having a disease and recovering or by vaccination.

Acute – a disease that has a short and relatively severe course.

Anemia – a condition in which the blood is deficient in quantity or quality. If deficient in quality, there is a reduction in the hemoglobin content of the blood, in the number of circulating blood cells, or both. Anemia causes paleness of the skin and mucous membranes and loss of energy.

Antibody – an immune substance found in the blood and produced in response to stimulation by an antigen.

Antigen – a substance that, when taken into the body, stimulates antibody production.

Bacteria – microscopic, single-celled organisms that are widely distributed in nature. Bacteria that cause disease are referred to as pathogenic.

Bacterin – a killed suspension of bacterial cells that, when injected into the body, produces an immune response. A bacterin is commonly referred to as an immunizing agent.

Carrier – an apparently healthy animal that harbors disease-causing organisms and can transmit them to other susceptible animals.

Catarrhal – an inflammatory process involving the mucous membranes characterized by an increased flow of mucus.

Chronic – a disease of long duration.

Coccidiostat – a drug that is incorporated into the feed at low levels and fed continuously throughout the life of the bird to prevent coccidiosis.

Congestion – an excessive accumulation of blood or mucus in a part of the body.

Contagious – a disease that is infectious and can be passed from one individual to another.

Culture – *verb*: to attempt to isolate a causative organism from a diseased host. *noun*: a population of microorganisms that have been grown in artificial media.

Cyanosis – bluish discoloration of the skin, easily seen in the comb and wattles of poultry.

Diffuse – as applied to hemorrhage, one that is spread over a considerable area.

Disease – any departure from a normal state of health.

Edema – the presence of abnormal amounts of fluid in tissues.

Etiology – the study of the causes of diseases.

Exudate – fluid that is associated with an inflammatory reaction.

Fomite – an inanimate object that may harbor disease-causing organisms.

Fungi – low-order organisms that usually feed off decaying matter. Some cause disease.

Gross – regarding changes in tissue, a change that can be seen with the naked eye.

Hemorrhage – escape of blood from vessels.

Immune – resistant to a particular disease.

Immunity – the condition of being immune.

Infection – invasion of body tissues by a pathogenic organism, resulting in a state of disease.

Infectious – a disease produced by living organisms, or living organisms capable of causing disease.

Inflammation – a response of tissues to an injury or other irritant, usually resulting in redness and swelling.

'Itis' – a suffix that denotes an inflammatory state. Examples include enteritis (inflammation of the intestines) and airsacculitis (inflammation of the air sacs).

Lesion – a visible change in the shape, size, color or structure of an organ or tissue.

Listless – indifferent to surroundings.

Microscopic – visible only with a microscope.

Morbidity – incidence of disease within a flock or the percentage of diseased animals in a population.

Mortality – incidence of death from a disease within a population; death rate.

Necrosis – death of part of an organ or tissue.

Parasite – an organism that depends on another animal for nutrients and is detrimental to its host.

Pathogen – an organism that is capable of causing disease.

Pathogenicity – the capability (low, medium or high) of a particular organism to cause disease.

Rales – an abnormal respiratory sound; rattling or wheezing.

Serotype – a strain of microorganisms as determined by serological methods.

Sporadic – as applied to a disease outbreak, one that occurs here and there.

Symptom – a detectable sign of a disease.

Toxin – a poisonous substance produced by a living organism.

Tumor – a mass of new tissue that persists and grows independently of its surrounding structures and that has no physiological use.

Vaccine – a suspension of a prescribed amount of disease-causing organism that, when placed inside a living organism, produces an immune response high enough to prevent the disease.

Virulence – as applied to a pathogen, the ability to overcome the immune responses of the host.

Virus – microscopic organisms that must use a host cell to reproduce; some cause disease.



Appendix C: Conversion Tables

Linear (distance) conversion.

	Inches	Feet	Yards	Miles	Centimeters	Meters	Kilometers
1 inch		12	0.0278	0.000016	2.54	0.0254	0.0000254
1 foot	12		0.33	0.00019	30.48	0.3048	0.00031
1 yard	36	3		0.00057	91.44	0.9144	0.0009
1 mile	63,360	5,280	1,760		160,934	1,609.3	1.609
1 centimeter	0.3937	0.0328	0.0109	0.0000062		0.01	0.00001
1 meter	39.37	3.280	1.0936	0.00062	100		0.001
1 kilometer	39,370	3,280	1,093.6	0.621	100,000	1,000	

Area conversion.

	Sq. inches	Sq. feet	Sq. yards	Acres	Sq. miles	Sq. centimeters	Sq. meters
1 sq. inch		0.0069	0.00077	-	-	6.4516	0.00065
1 sq. foot	144		.0111	-	-	929.03	0.0929
1 sq. yard	1,296	9		-	-	8,361.3	0.8361
1 acre	-	43,560	4,840		0.00156	-	4,046.9
1 sq. mile	-	-	3,097,600	640		-	2,589,988
1 sq. centimeter	0.155	0.00108	0.00012	-	-		0.0001
1 sq. meter	1,550	10.764	2.471	0.00025	-	10,000	

Weight conversion.

	Ounces	Pounds	Tons (US)	Metric tons	Grams	Kilograms
1 ounce		0.0625	-	-	28.349	0.0284
1 pound	16		0.0005	0.00045	453.592	0.4536
1 ton (US)	32,000	2000		.09072	907,184	907.2
1 metric ton	35,274	2204.6	1.1023		1,000,000	1,000
1 gram	0.035	0.0022	-	-		0.001
1 kilogram	35.274	2.2046	0.0011	0.001	1,000	

Conversion Tables cont.

Liquid volume conversion.

	Fluid ounces	Pints	Quarts	Gallons	Cubic inches	Cu. feet	Liters	Cu. centimeters (cc)
1 fluid ounce		0.0625	0.0313	0.0078	1.805	0.001	0.0296	29.6
1 pint	16		0.5	0.125	28.875	0.0167	0.4732	473.2
1 quart	32	2		0.25	57.75	0.0334	0.9463	946.3
1 gallon	128	8	4		231	0.1337	3.785	3,785
1 cu. inch	0.554	0.0346	0.0173	0.0043		0.00058	0.0164	16.387
1 cu. foot	957.51	59.844	29.922	7.481	1,728		28.316	28,316
1 liter	33.815	2.1134	1.0567	0.2642	61.026	0.0353		1,000
1 cu. centimeter	0.0338	-	-	-	0.061	-	0.001	

Dry volume conversion.

	Dry pints	Dry quarts	Bushels (US)	Cu. inches	Cu. feet	Cu. yards	Liters
1 dry pint		0.5	0.0156	33.6	0.0195	0.00072	0.5506
1 dry quart	2		0.0313	67.2	0.0389	0.0014	1.1012
1 bushel (US)	64	32		2,150.4	1.2445	0.0461	35.238
1 cu. inch	0.0298	0.0149	-		0.00058	-	0.0164
1 cu. foot	51.428	25.714	0.8036	1,728		0.037	28.3161
1 cu. yard	1,388.56	694.28	21.697	46.656	27		764.53
1 liter	1.816	0.908	0.0284	61.026	0.0353	0.0013	

Working approximations of weights and measures

1 gallon of water weighs 8.34 pounds

1 gram mixed with 1 ton (2,000 pounds) = 1 part per million (ppm)

1 pound mixed with 500 tons = 1 part per million

4 milligrams mixed with 1 gallon of water = 1 part per million

Example: If feed is to be medicated to 200 ppm, then mix 200 grams per ton of feed.

Example: If water is to be medicated to the equivalent of 200 ppm in feed, then mix 400 milligrams per gallon. (This is based on the assumption that water intake is 2X [twice] feed intake.)



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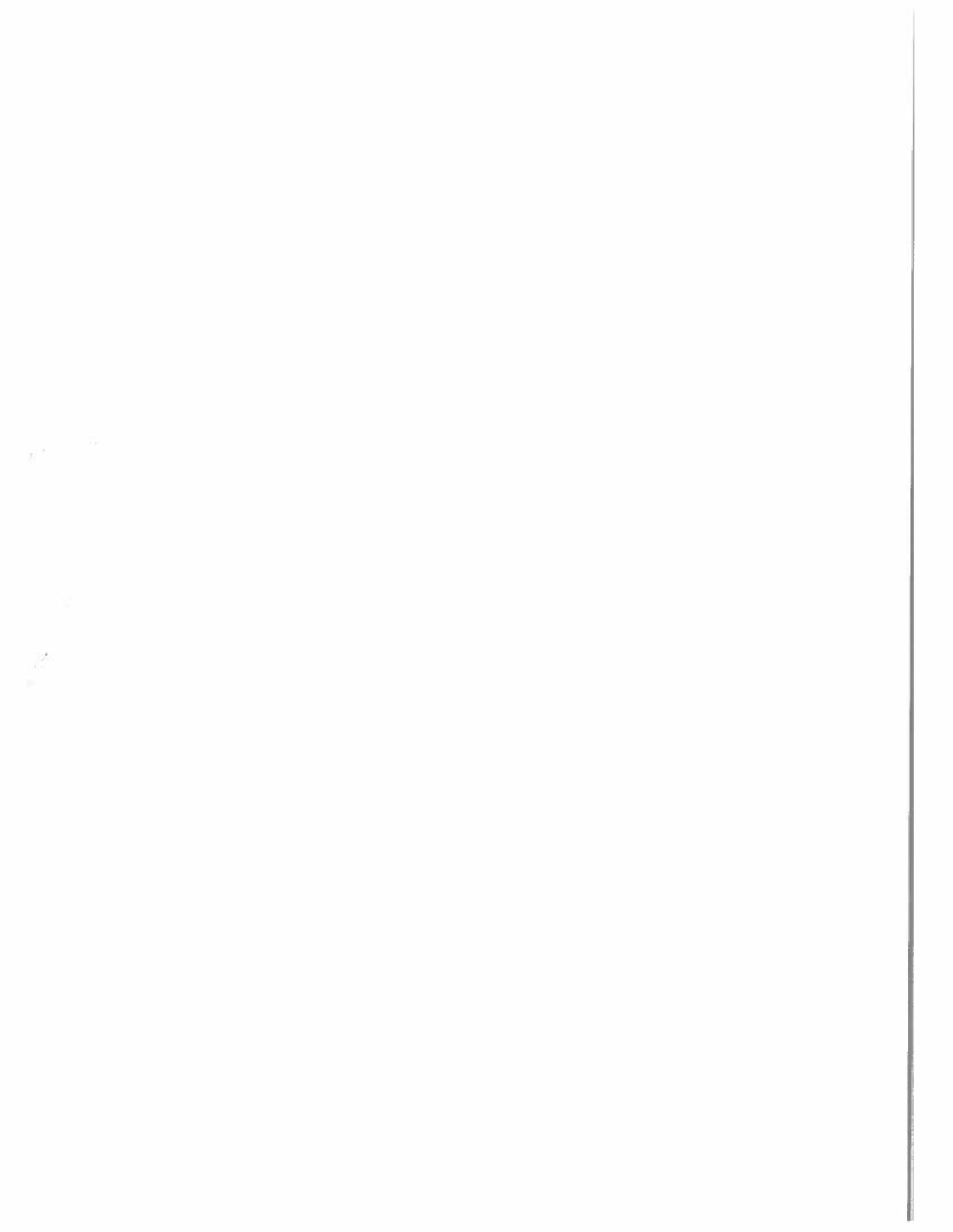
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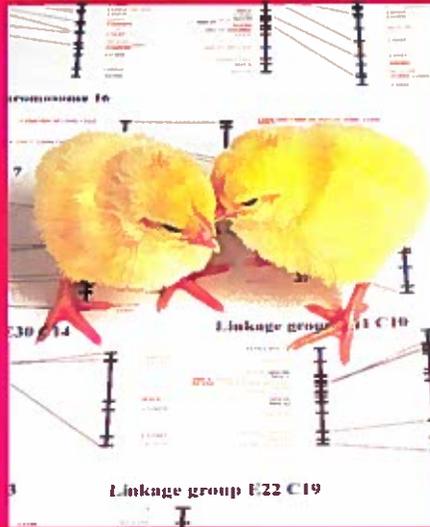
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