Diseases of Poultry

Bacterial diseases

Colibacillosis (Coliform infections)

Problems attributed to coliform infections are often caused by strains of the Escherichia coli organism. There is a marked variation in severity. Problems range from severe acute infections with sudden and high mortality to mild infections of a chronic nature with low morbidity and mortality. Infections may result in a respiratory disease from air sac infection, a septicemic (blood) disease from generalized infections, an enteritis from intestinal infection or a combination of any or all of these conditions. The disease may result from a coliform infection alone as in primary infection or in combination with other disease agents as a complicating or secondary infection. Secondary infections commonly occur as a part of the classic air sac disease syndrome as a complication with Mycoplasma gallisepticum infections.

All ages can be affected; however, the acute septicemia in young turkeys and airsacculitis in young chickens is more common in young growing birds. High, early mortality may occur as the result of navel infections.

The symptoms of this disease is caused by the E. coli bacteria and the toxins produced as they grow and multiply. There are many different strains or serological types within the group of E. coli bacteria. Many are normal inhabitants in intestinal tracts of chickens and turkeys and consequently are common organisms in the birds' environment.

A marked variation exists between different strains in their ability to cause disease. Some are
severe and by themselves can cause disease while others are supposedly harmless. All degrees of pathogenicity exist between the two extremes.

The primary routes of invasion by the organism are the respiratory system and the gastrointestinal tract. Omphalitis and infections in young birds may result from entry through the unhealed navel or penetration of the egg shell prior to or during incubation.

The symptoms vary with the different types of infections. In the acute septicemic form, mortality may begin suddenly and progress rapidly. Morbidity may not be apparent and birds in apparently good condition may die. However, in most cases birds are listless with ruffled feathers and indications of fever. Additional symptoms of labored breathing, occasional coughing and rales may be apparent. Diarrhea may be evident. Mortality may be high in recently hatched chicks and poults as a result of navel infection of coliforms.

Extremely acute septicemic infection may result in sudden death with very few, if any, lesions apparent. Common lesions include dehydration, swelling and congestion of the liver and spleen and kidneys and pinpoint hemorrhages in the viscera. Fibrinous to caseous exudate in the air sacs, heart sac and on the surface of the heart, liver and lungs is a characteristic lesion. The intestines may be thickened and inflamed and may contain excess mucus and areas of hemorrhage. Navel infections, similar to those described for omphalitis may be seen in young birds.

Diagnosis by laboratory means is necessary since coliform infection in its various forms may resemble and be easily confused with many other diseases. Isolation and identification of the organism by culture procedures can be accomplished relatively quickly; however, mere isolation is not sufficient to make a diagnosis. One must take into consideration the organ from which the organisms were isolated, the pathogenicity of the particular isolate and the presence of other disease agents.

Management and sanitation practices designed to reduce the number of these types of organisms in the birds' environment are necessary. In addition, reducing stress factors and other disease agents can enhance the ability of birds to defend against harmful infections. Providing adequate ventilation, good litter and range conditions, properly cleaned and disinfected equipment and facilities and high quality feed and water will improve the disease resisting status of the birds. The poultryman must always avoid overcrowding, environmental stresses like chilling or overheating and avoid vaccination or handling stress during periods when the birds are already subjected to stressful conditions. Proper egg handling, good hatchery management and implementing a good sanitation program is necessary to reduce early exposure of chicks or poults to disease organisms. It is always emphasized that problems due to one of the more pathogenic strains may occur even under ideal conditions.

The response of coliform infections to various medications is erratic and often difficult to
evaluate. Under practical conditions, treatment is often disappointing. Drug sensitivity varies with the strain of E. coli causing the condition. Laboratory tests to determine the sensitivity to the various drugs are useful to select the most beneficial drugs. When practical, moving birds to a clean environment may be of more value than medication. For example, when outbreaks occur in growing turkeys in the brooder house, moving to range is often the best treatment.

Mycoplasmosis (CRD, Air sac, Sinusitis)

Organisms in the genus Mycoplasma are a significant cause of respiratory disease in poultry. Of the numerous species of Mycoplasma that have been isolated from domestic poultry, three are of known significance. Mycoplasma gallisepticum is associated with chronic respiratory disease (CRD)/air sac syndrome in chickens and turkeys and infectious sinusitis of turkeys; Mycoplasma meleagridis is associated with airsacculitis in turkeys; and Mycoplasma synoviae is the cause of infectious synovitis in chickens and turkeys.

Chronic respiratory disease (CRD), air sac syndrome and infectious sinusitis of turkeys have a common cause. CRD was first recognized as a chronic but mild respiratory disease of adult chickens. It reduced egg production but caused little or no mortality. Afterward, a condition known as "air sac disease" became a problem in young birds. It caused high mortality in some flocks. Many birds became stunted, feed efficiency was reduced, and many fowl were rejected as unfit for human consumption when processed.

Infectious sinusitis in turkeys produces a sinus swelling under the eye as well as an inflammation of respiratory organs. It is a chronic disease adversely affecting growth and feed conversion. It may also cause significant mortality in young poult's.

A peculiar bacterial-like organism known as Mycoplasma gallisepticum (MG) is common to all three conditions. CRD and sinusitis in turkeys are caused by a pure MG infections while the air sac syndrome is caused by an infection of MG in combination with E. coli. These conditions are triggered by acute respiratory infections such as Newcastle disease or infectious bronchitis.

Mycoplasma gallisepticum is widespread and affects many species of birds. Eradication programs have reduced the incidence in recent years. It is primarily spread through the egg. Infected hens transmit organisms and the chick or poult is infected when it hatches. Organisms may also be transmitted by direct contact with infected or carrier birds.

The true CRD produces slight respiratory symptoms such as coughing, sneezing and a nasal discharge. In the air sac syndrome there is an extensive involvement of the entire respiratory system. The air sacs are often cloudy and contain large amounts of exudate. Affected birds become droopy, feed consumption decreases and there is a rapid loss of body weight.
Infectious sinusitis in turkeys occurs in two forms. When the "upper" form is present, there is only a swelling of the sinus under the eye. In the "lower" form, the lungs and air sacs are involved. The air sacs become cloudy and may contain large amounts of exudate. Both forms of the disease are usually present in the flock and frequently are present in the same bird.

Diagnosis of either condition must be based on flock history, symptoms and lesions. Blood tests are useful in determining whether a flock is infected.

The answer to the MG problem in both chickens and turkeys is eradication of the disease organisms. This goal has been achieved in commercial breeding flocks with voluntary programs conducted by the National Poultry Improvement Plan (NPIP) and National Turkey Improvement Plan (NTIP). The treatment of CRD, air sac syndrome and the lower form of infectious sinusitis is not considered satisfactory. Many antibiotics have been used with varying success. Whether to give treatment is a decision that must be made on each flock based on economic factors. If treatment is attempted, give high levels of one of the broad spectrum antibiotics (Tylosin, aureomycin, terramycin, gallimycin) either in feed, drinking water or by injections. The "upper"; form of infectious sinusitis can be treated with success by injecting antibiotics into the swollen sinus cavity.

**Fowl Cholera**

This disease occurs throughout the country wherever poultry is produced and in recent years has become the most hazardous infectious disease of turkeys. Host range is extensive and includes chickens, turkeys, pheasants, pigeons, waterfowl, sparrows and other free-flying birds.

The causative organism of fowl cholera is Pasteurella multocida. The organism can survive at least one month in droppings, three months in decaying carcasses and two to three months in soil. Pasteurella apparently enters tissues of the mouth and upper respiratory tract. The disease is not transmitted through the egg.

Major sources of infection include:

Body excreta of diseased birds that contaminate soil, water, feed, etc.,
Carcasses of birds that have died of the disease,
Contaminated water supplies such as surface tanks, ponds, lakes and streams,
Mechanical transmission by contaminated shoes or equipment.

Studies indicate that animals other than birds may serve as reservoirs of infection and actively spread the disease. These animals include raccoons, opossums, dogs, cats, pigs, and vermin.

The disease is seldom seen in chickens under four months of age, but is commonly seen in turkeys under this age. In the peracute form, symptoms may be absent; in the acute form
some birds may die without showing symptoms, but many others are visibly ill before death. Characteristic symptoms include stupor, loss of appetite, rapid weight loss, lameness resulting from joint infection, swollen wattles, difficult breathing, watery yellowish or green diarrhea and cyanosis or darkening of the head and wattles.

Lesions may be lacking in birds dying during peracute outbreaks. When present, lesions may resemble those associated with any acute septicemic bacterial infection, often those of fowl typhoid. Typical lesions may include pinpoint hemorrhages in the mucous and serous membranes and/or abdominal fat; inflammation of the upper third of the small intestine; light, firm "parboiled" appearance of the liver; enlarged and congested spleen; creamy or solid collection of material in joints; and cheesy material in the internal ear and air spaces of the cranium of birds having twisted necks. Turkeys may have pneumonia with solidification of one or both lungs.

A tentative diagnosis may be made on flock history, symptoms and postmortem lesions. A definite diagnosis depends upon isolation and identification of the organism.

Properly administered bacterins are helpful in preventing fowl cholera, particularly in turkeys. Their use must be combined with a rigid program of sanitation. In general, as it applies to the use of bacterins in turkeys, complete protection is unrealistic. Follow the manufacturer's recommendations for use of the bacterin. Vaccination in conjunction with treatment is not recommended.

Sanitation practices that aid in preventing the disease are:

Complete depopulation each year with definite breaks between older birds and their replacements,
Implement a rodent control program,
Dispose of dead birds properly,
Provide safe, sanitary water,
Clean and disinfect all houses and equipment after disposing of flock,
Keep birds confined to the house and away from wild feral birds and animals,
Allow contaminated ranges or yards to remain vacant for at least three months.

Although drugs usually alter the course of a fowl cholera outbreak, affected birds remain carriers and the disease has a tendency to recur when treatment is discontinued. This may necessitate prolonged treatment with drugs added to the feed and water. Sulfa drugs and broad spectrum antibiotics (Penicillin) usually control losses.

**Necrotic Enteritis**

Necrotic enteritis is an acute disease that produces a marked destruction of the intestinal lining of the digestive tract. Common field names (rot gut, crud and cauliflower gut)
accurately describe the condition. The cause of the disease is Clostridium perfringens, a spore-forming, rod-shaped bacterium. Bacterial organisms and their toxins are the primary cause but coccidiosis may be a contributing factor. Most of the damage to the intestinal lining apparently is due to toxins produced by the bacterial organisms.

Little is known about the spread of the disease but transmission is thought to occur by oral contact with the droppings from infected birds. Necrotic enteritis appears suddenly in the affected flock. Apparently healthy birds may become acutely depressed and die within hours. Mortality is usually between two and ten percent, but may be as high as thirty percent in severe outbreaks. Losses due to reduced growth and feed conversion may be more costly than flock mortality.

Lesions of the disease usually involve the lower half of the small intestine, but in some instances the entire length of the tract is involved. The intestine is dilated, contains dark offensive fluid and a diphtheritic cauliflower-like membrane that involves the mucosa. The lining of the intestine will have a coarse Turkish-towel appearance and portions of the lining may slough off and pass out with the intestinal contents. Diagnosis in based upon history, symptoms and findings of the characteristic lesions.

Bacitracin or virginiamycin are effective treatments administered in the feed. Bacitracin can also be given in the drinking water. Supportive vitamin treatment may enhance the effectiveness of the treatments. Preventive medication may be of value on premises where prior infections have been observed. Since coccidiosis may be a contributing factor, attention must be given to an effective coccidiosis control program.

**Ulcerative Enteritis (Quail disease)**

Ulcerative enteritis is an acute or chronic infection of game birds, chickens, turkeys and other domestic fowl. Death losses may be high for young quail or pullets being raised for egg production.

The cause of the disease is Clostridium colinum, a spore forming bacterial rod. The infection spreads by the droppings from sick or carrier birds to healthy birds. The disease organism is very resistant to disinfectants and will persist under varying environmental conditions.

Birds with the acute form may die suddenly while in good flesh, whereas more chronically affected birds become listless, have ruffled feathers, whitish watery diarrhea, and develop a humped-up posture. Such birds usually die in an extremely emaciated condition.

The dropping may be confused with those of birds with coccidiosis and the two diseases are often seen in the same bird. Droppings of birds with only ulcerative enteritis never contain blood.

The postmortem lesions are characteristic. The entire intestinal tract often has button-like
Although the disease is characteristic in nature, anyone suspecting the infection should seek professional confirmation before treatment is started. Bacitracin and penicillin are the most effective drugs in the treatment and prevention of this disease. If bacitracin is used, it should be incorporated in the feed at levels up to 200 grams per ton of feed. Addition of bacitracin to the water at the rate of one teaspoon per gallon aids in controlling an outbreak of the disease. Either method of administering bacitracin will control the disease within two weeks, unless a bacitracin-resistant strain of the disease organism is present. Penicillin is also used to treat the disease if bacitracin is not effective.

Raising birds on wire is an effective preventative measure. Specific drugs (bacitracin or penicillin) fed at low levels, are effective for controlling the disease in operations where the use of wire flooring is impractical.

**Pullorum Disease**

Pullorum disease is an acute or chronic infectious, bacterial disease affecting primarily chickens and turkeys, but most domestic and wild fowl can be infected.

The cause is a bacterium named Salmonella pullorum. This organism is primarily egg transmitted, but transmission may occur by other means such as:

- Infected hen to egg, egg to chick, or chick to chick in incubator, chick box, brooder, or house. Survivors become infected breeders (cycle begins again),
- Mechanical transmission (carried around on clothes, shoes or equipment),
- Carrier birds (apparently healthy birds shed the disease organisms),
- Contaminated premises (from previous outbreaks).

Disease organisms may enter the bird through the respiratory (as in the incubator) or digestive systems. Most outbreaks of acute pullorum disease in chickens or turkeys result from infection while in the hatchery.

Pullorum disease is highly fatal to young chicks or poults, but mature birds are more resistant. Young birds may die soon after hatching without exhibiting any observable signs. Most acute outbreaks occur in birds that are under three weeks of age. Mortality in such outbreaks may approach ninety percent if untreated. Survivors are usually stunted and unthrifty. Infection in young birds may be indicated by droopiness, ruffled feathers, a chilled appearance with birds huddling near a source of heat, labored breathing, and presence of a white diarrhea with a "pasted-down" appearance around the vent. The white diarrhea symptom instigated the term "bacillary white diarrhea" that was commonly associated with this disease at one time. Gross lesions may be lacking in some adult birds.
Diagnosis in young birds is made by isolating the causative organism in the laboratory. In older birds, blood testing may indicate an infection but a positive diagnosis depends upon isolation and identification of the organism by laboratory procedures.

Complete eradication is the only sound way to prevent pullorum disease. All hatchery supply flocks should be tested and only pullorum-free flocks used as a source of hatching eggs. Purchase chicks or poults from hatcheries that are officially recognized as "Pullorum Clean" by National Poultry Improvement Plan representatives in your state.

Treatment primarily is a salvage operation and does not prevent birds from becoming carriers. Consequently, do not keep recovered flocks for egg production. Among the drugs used to treat pullorum disease are furazolidone, gentamycin sulfate, and sulfa drugs (sulfadimethoxine, sulfamethazine, and sulfamerazine).

**Fowl Typhoid**

Fowl typhoid is an infectious, contagious bacterial disease that is usually acute but sometimes chronic. It affects most domestic and wild fowl including chickens, turkeys, ducks, pigeons, pheasants and other game birds. It must not be confused with typhoid fever in humans that is caused by a distinctly different organism.

The cause in the bacterium, Salmonella gallinarum. Methods of transmission are the same as for pullorum disease, including egg transmission. However, mechanical transmission is more prevalent with this disease than with pullorum disease.

Any age bird can be infected, but the disease primarily occurs in young adults (usually those older than twelve weeks of age). Mortality varies from less than one to about forty percent, but higher mortality has been observed. Signs include sudden or sporadic mortality, listlessness, green or yellow diarrhea (accompanied with pasting of the vent feathers), loss of appetite, increased thirst and a pale, anemic appearance of comb and wattles.

A tentative diagnosis may be made from the history, signs and lesions but final diagnosis must be based on isolation and identification of the causative organism. Lesions observed at necropsy help verify a diagnosis of fowl typhoid. The lesions include an enlarged and mottled spleen, enlarged liver (colored yellow or greenish brown), small pinpoint hemorrhages in muscles and fat surrounding internal organs, and a slimy inflammation of the front third of the small intestine. Small, white plaque-like areas are visible through the walls of the intestine is suggestive of fowl typhoid in turkeys. Blood tests used to detect pullorum reactors are also used to identify fowl typhoid birds.

Prevention and control depend heavily upon basic disease prevention practices including the hatching chicks from disease-free flocks (as determined by pullorum testing), practicing strict sanitation on the farm, providing clean feed and water, and proper disposal of all dead birds.
as approved by the state animal health agency. The causative organism can live outside the bird body for at least six months, thus requiring extra management precautions to break the disease cycle. Following an outbreak, thoroughly clean and disinfect the premises. When feasible, practice range rotation and other special precautions to prevent the carryover of infection to the following flock.

Drugs cannot be depended upon as a means of typhoid prevention and are not recommended for that purpose. Infected birds may be salvaged using the same drugs as used to salvage pullorum infected birds.

**Botulism**

Botulism is a disease caused by the ingestion of a toxin produced by the Clostridium botulinum bacterium. All domestic fowl and most wild birds are susceptible to the toxin's effects. Many human deaths have also been attributed to the consumption of food or water containing the toxin.

Botulism is not a bacterial infection, but a condition produced by a byproduct of the bacteria's growth. The organism is common in nature and is widely dispersed in soils. Ingestion of the organism is not harmful. It becomes dangerous only when conditions are favorable for its growth and subsequent toxin formation. The organism grows best under high humidity and relatively high temperature and in an environment containing decaying organic material (plant or animal). The organism requires an environment in which all atmospheric oxygen is eliminated. The organism cannot multiply in the presence of air. Stagnant pools or damp areas with buried decaying matter are danger areas for toxin development. Botulism results after the decaying animal or plant material containing the toxin is consumed. Decaying carcasses are a frequent source of the toxin, as are many insects feeding in the same tissue. The insects may contain enough toxin to cause the disease in any bird that ingests it. Since the toxin is water soluble, water sources may become contaminated and provide a reservoir for the disease.

The toxin is one of the most potent discovered by scientists. The toxin is relatively heat stable but may be destroyed by boiling. There are different types of the toxin; types A and C cause the disease in birds while type B frequently produces the disease in man.

Weakness is generally the first sign of the illness and is followed by progressive flaccid paralysis of the legs, wings and neck. When neck muscles are affected the head hangs limp, thus causing a condition referred to as "limberneck". Affected birds may have a peculiar trembling, loose feathers that are pulled out easily and dull partly closed eyes. Some birds (turkey) do not develop loose feathers or limberneck symptoms. Because of the paralysis, birds are unable to swallow and mucous accumulates in the mouth. Fatally affected birds may lie in a profound coma appearing lifeless for several hours before death. Significant lesions are not usually observed in affected birds. Examining digestive contents may reveal
insects, decomposed animal or vegetable material or other matter suggesting that the birds have consumed the toxin.

A tentative diagnosis can be made from the history, symptoms and post-mortem findings. As an aid to diagnosis, sick birds may be given water into the crop, kept in a cool environment and treated intravenously with antitoxin. Recovery of a large percentage of the affected birds would substantiate diagnosis.

Prevention should be aimed at eliminating sources of toxin production and preventing access of birds to such materials. These practices include prompt removal of all dead animals from houses and pens, debeaking the birds, controlling fly and insect populations and avoiding access to decaying organic material. Contaminated water supplies are particularly dangerous.

If the disease strikes, locate and remove the source of the toxin and separate all visibly affected birds from the flock for treatment. Place sick birds in a cool shaded area and give fresh water into the crop, twice daily. Mild laxatives may be used for birds that have been exposed but do not show disease symptoms. Epsom salts (one pound per 100 birds) may be mixed into feed. Adding a level teaspoonful of Epsom salts in one ounce of water and placing in the crops of sick birds has been beneficial in many instances. Antitoxin therapy is indicated only in birds that have high individual value since the antitoxin is difficult to obtain and is expensive.

**Infectious Coryza**

Infectious coryza is a specific respiratory disease in chickens that occurs most often in semi-mature or adult birds. Infection may result in a slow-spreading, chronic disease that affects only a small number of birds at one time, or in a rapid spreading disease with a higher percentage of birds being affected. The occurrence of infectious coryza is not widespread and the incidence is relatively low.

The disease is caused by a bacterium known as Hemophilus gallinarum. Outbreaks usually result from the introduction of infected or carrier birds into a flock. Transmission of the infection occurs by direct contact, airborne infection by dust or respiratory discharge droplets and drinking water contaminated by infective nasal exudate. Susceptible birds usually develop symptoms within three days after exposure to the disease. Recovered individuals may appear normal but remain carriers of the organism for long periods. Once a flock is infected, all birds must be considered as carriers.

The most characteristic symptoms of infectious coryza include edematous swelling of the face around the eyes and wattles, nasal discharge and swollen sinuses. Watery discharge from the eyes frequently results in the lids adhering together. Vision may be affected because of the swelling. The disease results in a decrease in feed and water consumption
and an increase in the number of cull birds. An adverse effect on egg production usually occurs in proportion to the number of affected birds.

Diagnosis can be confirmed only by isolation and identification of the causative organism. The organism, Hemophilus gallinarum, is extremely fastidious and often difficult to isolate.

Prevention is the only sound approach in controlling infectious coryza. It usually can be prevented by management programs that eliminate contact between susceptible and infected birds. It requires only separating affected or carrier birds from the susceptible population. In order to prevent the infection, introduce started or adult birds only from sources known to be free of the infection. If infection occurs, complete depopulation followed by thorough cleaning/disinfecting is the only means for eliminating the disease.

A number of drugs are effective for treating the symptoms of the disease although the disease is never completely eliminated. Sulfadimethoxine or sulfathiazole in the feed or water or erythromycin administered in the drinking water can reduce the symptoms of this disease.

**Omphalitis**

Omphalitis may be defined technically as an inflammation of the navel. As commonly used, the term refers to improper closure of the navel with subsequent bacterial infection (navel ill; mushy chick disease).

Apparently, most problems result from mixed bacterial infections including the common coliforms and various species belonging to the genera Staphylococcus, Streptococcus, Proteus, and others. Omphalitis can usually be traced to faulty incubation, poor hatchery sanitation or chilling/overheating soon after hatching (such as in transit). The significance of isolating one of the bacterial species mentioned above is complicated in that many of the same species can be isolated from the yolks of supposedly normal birds immediately after hatching.

Omphalitis occurs during the first few days of life, so it cannot be considered transmissible from bird to bird. It is transmitted from unsanitary equipment in the hatchery to newly hatched birds having unhealed navels.

Affected chicks usually appear drowsy or droopy with the down being "puffed up". They also generally appear to be of inferior quality and show a lack of uniformity. Many individuals stand near the heat source and are indifferent to feed or water. Diarrhea sometimes occurs. Mortality usually begins within 24 hours and peaks by five to seven days.

Characteristic lesions are poorly healed navels, subcutaneous edema, bluish color of the abdominal muscles around the navel and unabsorbed yolk material that often has a putrid odor. Often yolks are ruptured and peritonitis is common.
A tentative diagnosis can be made on the basis of history and lesions. The presence of mixed bacterial infections and absence of any specific disease-producing agent is used for confirming the diagnosis.

Good management and sanitation procedures in the hatchery and during the first few days following hatching are the only sure ways to prevent omphalitis. Broad spectrum antibiotics help reduce mortality and stunting in affected groups, but they do not replace sanitation.

**Erysipelas**

Erysipelas is a bacterial disease caused by Erysipelothrix insidiosa. The disease affects several species of birds including chickens, ducks and geese, but the fowl in which it has been of primary importance is the turkey. Man is susceptible to infection and may contract the disease from infected turkeys. Since this organism is pathogenic for man, care should be taken when handling infected birds or tissues.

Erysipelas in turkeys occurs most often during the fall and winter months and usually affects birds that are four to seven months of age, although any age bird is susceptible. Incidence has often been reported to be higher in males than in females, possibly because fighting males receive numerous skin abrasions that serve as portals of entry for the bacteria. In some instances the incidence is higher in hens than toms because of artificial insemination techniques that provide a means of transmission.

The organism may survive for long periods in the soil and most outbreaks are thought to originate from contaminated soil or premises. Sheep, swine and rodents may be carriers of the disease organisms. Recurrence of the disease on a premise is common. Predisposing or aggravating factors include over-crowding damp or inclement weather and poor sanitation and range management.

The first indication of the disease may be the discovery of several dead birds. Usually several morbid birds can be found; however, most affected birds are visibly sick for only a short period before death. Symptoms are typical of a septicemic disease and include a general weakness, listlessness, lack of appetite and sometimes a yellowish or greenish diarrhea. Occasionally, the snood of toms may be turgid, swollen and purple. Some birds may be found lame with swollen leg joints due to localization of the infection. In breeding flocks, this disease occasionally is associated with decreased fertility and hatchability. Daily morbidity and mortality usually are low; however, in untreated flocks mortality may persist for some time and become excessive.

The most characteristic lesions are small or diffuse hemorrhages located in almost any tissue or organ. Such hemorrhages are commonly observed in the muscles, heart, liver, spleen, fat and other tissues of the body cavities. Hemorrhagic conditions of skin may result in purple blotches. The liver and spleen are usually enlarged, congested and occasionally
contain necrotic foci. Enteritis or inflammation of the intestinal tract is commonly observed, as in most septicemic diseases.

Symptoms and lesions may resemble other diseases so closely that a reliable diagnosis can be made only through isolation and identification of the causative organism.

Good management practices that aid in preventing erysipelas include avoiding the use of ranges previously occupied by swine, sheep or turkeys where erysipelas is known to have existed. Debeaking, removal of the snoods of toms, measures that prevent injury from fighting, avoiding overcrowding and providing well drained ranges will aid in preventing this disease problem.

Bacterins are available and are useful on premises where history indicates that outbreaks may be a problem. The amount and duration of protection is relative to the amount of exposure and may not be sufficient for the entire laying period. Administer bacterins in accordance with the manufacturer's directions.

Move sick birds to a hospital pen for individual treatment and to prevent cannibalism. Moving unaffected birds to a clean range may aid in preventing the spread of the disease but may also contaminate an additional range.

Various antibiotics have shown efficacy in treating erysipelas; however, penicillin is best. Penicillin injections in the leg or breast muscles of visibly sick birds is effective in decreasing mortality. One injection is usually sufficient, but more may be given if necessary. Water and feed medication may be of value under certain conditions.

Parasitic diseases (internal)

Ascarids (Large Intestinal Roundworms)

One of the most common parasitic roundworms of poultry (Ascaridia galli) occurs in chickens and turkeys. Adult worms are about one and a half to three inches long and about the size of an ordinary pencil lead. Thus, they can be seen easily with the naked eye. Heavily infected birds may show droopiness, emaciation and diarrhea. The primary damage is reduced efficiency of feed utilization, but death has been observed in severe infections.

Chickens of three to four months of age show resistance to infection. Specimens of this parasite are found occasionally in eggs. The worm apparently wanders from the intestine up the oviduct and is included in the egg contents as the egg is being formed.

The life history of this parasite is simple and direct. Females lay thick heavy-shelled eggs in the intestine that pass in the feces. A small embryo develops in the egg but does not hatch
immediately. The larvae in the egg reaches infective stage within two to three weeks. Embryonated eggs are very hardy and under laboratory conditions may live for two years. Under ordinary conditions, however, few probably live more than one year. Disinfectants and other cleaning agents do not kill eggs under farm conditions. Birds become infected by eating eggs that have reached the infective stage.

Available drugs remove only the adult parasite. The immature form probably produces the most severe damage. The treatment of choice is piperazine. Many forms of piperazine are produced, and all are effective if administered properly. Piperazine is only effective for treating this parasite. It has no effect on other internal parasites of fowl. Follow the manufacturer's instructions carefully.

The parasite can be controlled by strict sanitation. If the birds are confined, clean the house thoroughly and completely before a new group is brought in. Segregate birds by age groups, with particular care applied to sanitation of young birds. If birds are on range, use a clean range for each group of birds.

**Cecal Worms**

This parasite (Heterakis gallinae) is found in the ceca of chickens, turkeys and other birds.

This parasite apparently does not seriously affect the health of the bird. At least no marked symptoms or pathology can be blamed on its presence. Its main importance is that it has been incriminated as a vector of Histomonas meleagridis, the agent that causes blackhead. This protozoan parasite apparently is carried in the cecal worm egg and is transmitted from bird to bird through this egg.

The life history of this parasite is similar to that of the common roundworm. The eggs are produced in the ceca and pass in the feces. They reach the infective form in about two weeks. In cool weather, this may take longer. The eggs are very resistant to environmental conditions and will remain viable for long periods.

The cecal worm can be effectively treated with fenbendazole. Since the worm itself produces no observable damage and the eggs live for long periods, it is advisable and necessary to keep chickens and turkeys separated to prevent spread of blackhead.

**Capillaria (Capillary or Thread Worms)**

There are several species of Capillaria that occur in poultry. Capillaria annulata and Capillaria contorta occur in the crop and esophagus. These may cause thickening and inflammation of the mucosa, and occasionally severe losses are sustained in turkeys and game birds.

In the lower intestinal tract there may be several different species but usually Capillaria obsignata is the most prevalent. The life cycle of this parasite is direct. The adult worms may
be embedded in the lining of the intestine. The eggs are laid and passed in the droppings. Following embryonation that takes six to eight days, the eggs are infective to any other poultry that may eat them. The most severe damage occurs within two weeks of infection. The parasites frequently produce severe inflammation and sometimes cause hemorrhage. Erosion of the intestinal lining may be extensive and result in death. These parasites may become a severe problem in deep litter houses. Reduced growth, egg production and fertility may result from heavy infections.

If present in large numbers, these parasites are usually easy to find at necropsy. Eggs may be difficult to find in droppings, due to the small size and time of infection.

Since treatment for capillaria is often lacking, control is best achieved by preventive measures. Some drugs, fed at low levels, may be of value in reducing the level of infection on problem farms. Game birds should be raised on wire to remove the threat of infection. As some species of capillaria have an indirect life cycle, control measures may have to be directed toward the intermediate host. Hygromycin and meldane may be used for control. Additional vitamin A may be of value. Effective treatments that are not approved by the Food and Drug Administration are fenbendazole and leviamisole.

**Tapeworms**

Tapeworms or cestodes are flattened, ribbon-shaped worms composed of numerous segments or division. Tapeworms vary in size from very small to several inches in length. The head or anterior end is much smaller than the rest of the body. Since tapeworms may be very small, careful examination often is necessary to find them. A portion of the intestine may be opened and placed in water to assist in finding the tapeworms.

The pathology or damage tapeworms produce in poultry is controversial. In young birds, heavy infections result in reduced efficiency and slower growth. Young birds are more severely affected than older birds.

All poultry tapeworms apparently spend part of their lives in intermediate hosts, and birds become infected by eating the intermediate hosts. These hosts include snails, slugs, beetles, ants, grasshoppers, earthworms, houseflies and others. The intermediate host becomes infected by eating the eggs of tapeworms that are passed in the bird feces.

Although several drugs are used to remove tapeworms from poultry, most are of doubtful efficacy. In general, tapeworms are most readily controlled by preventing the birds from eating the infected intermediate host. Tapeworm infections can be controlled by regular treatment of the bird with fenbendazole or leviamisole.

**Gapeworms**

The gapeworm (Syngamus trachea) is a round red worm that attach to the trachea
(windpipe) of birds and causes the disease referred to as "gapes". The term describes the open-mouth breathing characteristic of gapeworm-infected birds. Heavily infected birds usually emit a grunting sound because of the difficulty in breathing and many die from suffocation. The worms can easily block the trachea, so they are particularly harmful to young birds.

The gapeworm is sometimes designated as the "red-worm"; or "forked-worm" because of its red color and because the male and female are joined in permanent copulation. They appear like the letter Y. The female is the larger of the two and is one-fourth to one inch in length. The male gapeworm may attain a length of one-fourth inch. Both sexes attach to the lining of the trachea with their mouthparts. Sufficient numbers may accumulate in the trachea to hinder air passage.

The life cycle of the gapeworm is similar to that of the cecal worm; the parasite can be transmitted when birds eat embryonated worm eggs or earthworms containing the gapeworm larvae. The female worm lays eggs in the trachea, the eggs are coughed up, swallowed, and pass out in the droppings. Within eight to fourteen days the eggs embryonate and are infective when eaten by birds or earthworms. The earthworm, snails and slugs serve as primary intermediate hosts for the gapeworm. Gapeworms in infected earthworms remain viable for four and a half years while those in snails and slugs remain infective for one year. After being consumed by the bird, gapeworm larvae hatch in the intestine and migrate from the intestine to the trachea and lungs.

Gapeworms infect chickens, turkeys, guinea fowl, pheasants, chukar partridge, and probably other birds. Young birds reared on soil of infected range pens are at high risk (pen-raised game birds). Some control or reduction in infection density (worms/bird) is achieved by alternating the use of range pens every other year and/or using a pen for only one brood each year. Tilling the soil in the pens at the end of the growing season helps to reduce the residual infection. Treating the soil to eliminate earthworms, snails and slugs is possible but the cost is usually prohibitive.

Gapeworms are best prevented by administering a wormer at fifteen to thirty day intervals or including a drug at low levels continuously beginning fifteen days after birds are placed in the infected pens. One drug that is effective for eliminating gapeworms is fenbendazole, however, its use is not presently approved for use in birds by the Food and Drug Administration.

Parasitic diseases (external)

Poultry Mites
All classes of poultry are susceptible to mites, some of which are blood-suckers, while others burrow into the skin or live on or in the feathers. Others occur in the air passages and in the lungs, liver and other internal organs. Poultry mites cause retarded growth, reduced egg production, lowered vitality, damaged plumage and even death. Much of the injury, consisting of constant irritation and loss of blood, is not apparent without careful examination.

Of primary concern to the poultryman is the Northern Fowl Mite (Ornithonyssus sylviarum) which is a frequent and serious pest of chickens. Heavy infestations result in low condition of the birds and lower egg production, as well as a scabby skin condition. The mite remains on the bird and does more damage than any other species of mite. The mite does not leave the host bird, as do many species of mites, and can be observed on birds in large numbers during daylight hours. It prefers the feathers below the vent and around the tail, but can be found on all parts of the body. The mite is extremely small and a microscope or magnifying glass may be needed to see it.

The female northern fowl mite lays eggs on feathers where the young mites complete their development without leaving the host. Since they remain on the fowl most of the time, treatment of the birds is necessary to destroy the mites.

The Common Chicken Mite (Dermanyssus gallinae) is the most common mite found on all types of poultry. It is a blood-sucker, and when present in large numbers, loss of blood and irritation may be sufficient to cause anemia. Egg production is seriously reduced.

This mite feeds at night, and usually remains hidden in cracks and crevices during the day. It attacks birds at night while they are on the roost. In heavy infestations, some mites may remain on the birds during the day. About a day after feeding, the female lays eggs in cracks and crevices of the house. The eggs hatch and the mites develop into adults within about a week. During cold weather, the cycle is slower. A poultry house remains infested four to five months after birds are removed.

Since the mite feeds on wild birds, these birds may be responsible for spreading infestations. However, it is more likely that spread of the mite is promoted by using contaminated coops. Human carriers are also important. Since these mites do not stay on the birds during the day, apply treatments to houses and equipment as well as the birds.

The Scaly-Leg Mite (Knemidocoptes mutans) lives under the scales on feet and legs of poultry. It also may attach to the comb and wattles. It causes a thickening of scales on the feet and legs that gives the impression that the scales are protruding directly outward, rather than laying flat on the limb. It spends its entire life cycle on the birds and spreads mainly by direct contact.

The Depluming Mite (Knemidocoptes laevis, variety gallinae) causes severe irritation by
burrowing into the skin near the bases of feathers and frequently causes feathers to be pulled out or broken. The mite is barely visible to the naked eye and can be found in follicles at the base of the feathers. The mites crawl around the birds at times, spreading from bird to bird.

The most effective treatment for all mite species is a regular inspection and spraying program of both the birds and their premises. An appropriate solution of permethrin, when sprayed on the birds, will eliminate all mites that infest the bird. The spraying of all facilities will ensure that any mites hiding in cracks and crevices will be destroyed. The treatment should be repeated on a one to two month schedule or whenever populations of the mites are detected.

Poultry Lice

The primary effects of lice on their hosts are the irritations they cause. The birds become restless and do not feed or sleep well. They may injure themselves or damage their feathers by pecking or scratching areas irritated by lice. Body weight and egg production may drop.

All lice infesting poultry and birds are the chewing type. Mites may be confused with lice, but mites suck blood. In general, each species of lice is confined to a particular kind of poultry, although some may pass from one kind to another when birds are closely associated. Chickens usually are infested with one or more of seven different species; turkeys have three common species.

All species of poultry lice have certain common habits. All live continuously on feathered hosts and soon die if removed. The eggs are attached to the feathers. Young lice resemble adults except in color and size. Lice differ in preferred locations on the host, and these preferences have given rise to the common names applied to various species.

In general, the incubation period of lice eggs is four to seven days, and development of the lice between hatching and the adult stage requires about twenty-one days. Mating takes place on the fowl, and egg laying begins two to three days after lice mature. The number of eggs probably ranges from fifty to three-hundred per female louse.

As the name suggests, the Head Louse (Cuclolotogaster heterographe) is found mainly on the head, although it occurs occasionally on the neck and elsewhere. It usually is located near the skin in the down or at the base of the feathers on the top and back of the head and beneath the beak. In fact, the head of the louse often is found so close to the skin that poultrymen may think it is attached to the skin or is sucking blood. Although it does not suck blood, the head louse is very irritating and ranks first among lice as a pest of young chickens and turkeys. Heavily infested chicks soon become droopy and weak and may die before they are a month old. When the chickens become fairly well feathered, head lice decrease but may increase again when the fowls reach maturity.
This louse is oblong, grayish and about 1/10-inch long. The pearly-white eggs are attached singly to the down or at the base of the small feathers on the head. They hatch within five days into minute, pale, translucent lice resembling adults in shape.

The Body Louse (Menacanthus stramineus) of chickens prefers to stay on the skin rather than on the feathers. It chooses parts of the body that are not densely feathered, such as the area below the vent. In heavy infestations, it may be found on the breast, under the wings and on other parts of the body, including the head.

When the feathers are parted, straw-colored body lice may be seen running rapidly on the skin in search of cover. Eggs are deposited in clusters near the base of small feathers, particularly below the vent, or in young fowls, frequently on the head or throat. Eggs hatch in about a week and lice reach maturity within twenty days.

This is the most common louse infesting grown chickens. When present in large numbers, the skin is irritated greatly and scabs may result, especially below the vent.

The Shaft Louse or small body louse (Menopon gallinae) is similar in appearance to the body louse, but smaller. It has a habit of resting on the body feather shafts of chickens where it may be seen running rapidly toward the body when feathers are parted suddenly. Sometimes as many as a dozen lice may be seen scurrying down a feather shaft.

Since the shaft louse apparently feeds on parts of the feathers, it is found in limited numbers on turkeys, guinea fowl and ducks kept in close association with chickens. It does not infest young birds until they become well feathered.

The same control measures used to eliminate mite populations is effective for treating lice. It is more important to apply the insecticides directly to the bird's body rather than the premises.

Fowl Tick (Blue Bug)

The Fowl Tick (Argas persicus) may be a serious parasite of poultry if it becomes numerous in poultry houses or on poultry ranges. The tick is a blood-sucker, and when present in large numbers it results in weakened birds, reduced egg production, emaciation and even death. The fowl tick is found throughout most of the South and is extremely hardy. Ticks have been kept alive without food for more than three years. The ticks will feed on all fowl.

Fowl ticks spend most of their lives in cracks and hiding places, emerging at night to take a blood meal. Mating takes place in the hiding areas. A few days after feeding, the female lays a batch of eggs. In warm weather the eggs hatch within fourteen days. In cold weather they may take up to three months to hatch. Larvae that hatch from the eggs crawl around until they find a host fowl. They remain attached to the birds for three to ten days. After leaving the birds they find hiding places and molt before seeking another blood meal. This is
followed by additional moltings and blood meals.

Ticks are difficult to eradicate and methods employed must be performed carefully. It is not necessary to treat the birds, but houses and surrounding areas must be treated thoroughly.

**Chiggers, Red Bugs or Harvest Mites**

These pests (Trombicula splendens, Trombicula alfreddugesi, and Neoschongastia americana americana) attack chickens and turkeys, as well as humans. Normally these small mites feed on wild animals, birds, snakes and lizards. Only the larvae of chiggers attack poultry or animals; adult mites feed on plants.

Larvae usually attach to the wings, breasts and necks of poultry. They inject a poisonous substance that sets up local irritation and itching. After a few days, the larvae become engorged and drop off. Injury to grown fowl may not be apparent or noticed until the bird is dressed; then the lesions are readily apparent and greatly reduce the carcass value. Young chickens or turkeys may become droopy, refuse to eat and die. Due to methods of raising poultry, turkeys are more affected than chickens.

**Control of External Parasites**

There are many insecticides available to help control external poultry parasites. The most effective broad spectrum insecticide is permethrin. Permethrin has a significant residual activity, thus making it ideal for treating facilities and equipment. At reduced concentrations it can be applied to the bird. Follow all manufacturers recommendations when using all insecticides.

**Viral diseases**

**Avian Pox**

Avian pox is a relatively slow-spreading viral disease in birds, characterized by wart-like nodules on the skin and diphtheritic necrotic membranes lining the mouth and upper respiratory system. It has been present in birds since the earliest history. Mortality is not usually significant unless the respiratory involvement is marked. The disease may occur in any age of bird, at any time.

Avian pox is caused by a virus of which there are at least three different strains or types; fowl pox virus, pigeon pox virus and canary pox virus. Although some workers include turkey pox virus as a distinct strain, many feel that is identical to fowl pox virus.

Each virus strain is infective for a number of species of birds. Natural occurring pox in
chickens, turkeys and other domestic fowl is considered to be caused by fowl pox virus.

Fowl pox can be transmitted by direct or indirect contact. The virus is highly resistant in dried scabs and under certain conditions may survive for months on contaminated premises. The disease may be transmitted by a number of species of mosquitoes. Mosquitoes can harbor infective virus for a month or more after feeding on affected birds. After the infection is introduced, it spreads within the flock by mosquitoes as well as direct and indirect contact. Recovered birds do not remain carriers.

Since fowl pox usually spreads slowly, a flock may be affected for several months. The course of the disease in the individual bird takes three to five weeks. Affected young birds are retarded in growth. Laying birds experience a drop in egg production. Birds of all ages that have oral or respiratory system involvement have difficulty eating and breathing. The disease manifests itself in one or two ways, cutaneous pox (dry form) or diphtheritic pox (wet form).

Dry pox starts as small whitish foci that develop into wart-like nodules. The nodules eventually are sloughed and scab formation precedes final healing. Lesions are most commonly seen on the featherless parts of the body (comb, wattles, ear lobes, eyes, and sometimes the feet).

Wet pox is associated with the oral cavity and the upper respiratory tract, particularly the larynx and trachea. The lesions are diphtheritic in character and involve the mucous membranes to such a degree that when removed, an ulcerated or eroded area is left.

Fowl pox is readily diagnosed on the basis of flock history and presence of typical lesions. In some cases, laboratory diagnosis by tissue or transmission studies is necessary.

There is no treatment for fowl pox. Disease control is accomplished best by preventative vaccination since ordinary management and sanitation practices will not prevent it. Several kinds of vaccines are available and are effective if used properly.

Vaccination of broilers is not usually required unless the mosquito population is high or infections have occurred previously. The chicks may be vaccinated as young as one day of age by using the wing-web method and using a one needle applicator. All replacement chickens are vaccinated against fowl pox when the birds are six to ten weeks of age. One application of fowl pox vaccine results in permanent immunity.

Newcastle Disease

Newcastle disease is a contagious viral infection causing a respiratory nervous disorder in several species of fowl including chickens and turkeys. Different types or strains of the virus (varying in their ability to cause nervous disorder, visceral lesions and death) have been recognized.
The most severe strain is called viscerotropic velogenic Newcastle disease (VVND) and is kept from birds in the U.S. by enforcement of strict quarantines at our national borders. It is often referred to as "Exotic Newcastle Disease" and infection of susceptible fowl with this form usually causes high mortality. Due to the reduced chance that poultry in this country will become infected with this disease form, it will not be discussed.

A milder form of the disease is called "mesogenic" Newcastle disease and is the most serious strain found in the U.S. This is the form that is referred to as Newcastle disease in this discussion.

Newcastle disease is highly contagious. All birds in a flock usually become infected within three to four days. The virus can be transmitted by contaminated equipment, shoes, clothing and free-flying birds. During the active respiratory stage, it can be transmitted through the air. The virus is not thought to travel any great distance by this method. Recovered birds are not considered carriers and the virus usually does not live longer than thirty days on the premises.

Signs of Newcastle disease are not greatly different from those of other respiratory diseases. The signs most frequently observed are nasal discharge, excessive mucous in the trachea, cloudy air sacs, casts or plugs in the air passages of the lungs and cloudiness in the cornea of the eye.

The disease in young chickens begins with difficult breathing, gasping and sneezing. This phase continues for ten to fourteen days and may be followed by nervous symptoms. If nervous disorders develop, they may consist of paralysis of one or both wings and legs or a twisting of the head and neck. The head often is drawn over the back or down between the legs. Mortality may vary from none to total loss of the flock.

In adult chickens, respiratory symptoms predominate. Only rarely do nervous disorders develop. If the flock is laying, egg production usually drops rapidly. When this occurs, it takes four weeks or longer for the flock to return to the former production rate. During the outbreak, small, soft-shelled, off-colored and irregular-shaped eggs are produced. Mortality in adult birds is usually low but may be fairly high from some virus strains.

In turkeys, the symptoms are usually mild and may be unnoticed unless nervous disorders develop. During an outbreak, turkeys will produce eggs with a chalky white shell. Reduced production in breeder flocks is the main economic loss from this disease in turkeys.

The flock history, signs of a respiratory nervous disorder and other typical lesions often may be sufficient to allow a tentative diagnosis. Usually, however, the disease cannot be differentiated from infectious bronchitis and some of the other respiratory infections, except by laboratory methods.
Vaccination is practiced widely and is the recommended method for prevention. Several types of vaccines are available but the most successful and widely used is the mild live virus vaccine known as the B1 and La Sota types. The vaccines may be used by drops into the nostril or eye, addition to the drinking water or applied in spray form.

Broiler chickens are usually vaccinated when seven to ten days of age. Chickens kept for egg production are usually vaccinated at least three times. The vaccine is given when birds are approximately seven days, again at about four weeks and a third time at about four months of age. Revaccination while in lay is commonly practiced.

Vaccination is not widely used in turkeys. It is used to protect egg producing breeder flocks. One dose of the mild type vaccine is given after selecting breeder birds.

There is no treatment for Newcastle disease. The disease does not always respect even the best management programs, but good "biosecurity" practices will help reduce the possibility of exposure to Newcastle disease virus.

**Infectious Bronchitis**

Infectious bronchitis is an extremely contagious respiratory disease of chickens characterized by coughing, sneezing and rales (rattling). It is caused by a virus that affects chickens only. Other fowl or laboratory animals cannot be infected with this virus. Several distinct strains of the virus exist.

Infectious bronchitis is considered the most contagious of poultry diseases. When it occurs, all susceptible birds on the premises become infected, regardless of sanitary or quarantine precautions. The disease can spread through the air and can "jump" considerable distances during an active outbreak. It can also be spread by mechanical means such as on clothing, poultry crates and equipment. The disease is not egg transmitted and the virus will survive for probably no more than one week in the house when poultry are not present. It is easily destroyed by heat and ordinary disinfectants.

The infection is confined to the respiratory system. Symptoms are difficult breathing, gasping, sneezing and rales. Some birds may have a slight watery nasal discharge. The disease never causes nervous symptoms. It prevails for ten to fourteen days in a flock and symptoms lasting longer than this are from some other cause.

In chickens under three weeks of age, mortality may be as high as thirty or forty percent. The disease does not cause a significant mortality in birds over five weeks of age. Feed consumption decreases sharply and growth is retarded.

When infectious bronchitis occurs in a laying flock, production usually drops to near zero with a few days. Four weeks or more may be required before the flock returns to production. Some flocks never regain an economical rate of lay. During an outbreak, small, soft-shelled,
irregular-shaped eggs are produced.

Infectious bronchitis is difficult to differentiate from many of the other respiratory diseases. For this reason, a definite diagnosis usually requires a laboratory analysis.

Infectious bronchitis is highly contagious and does not always respect sanitary barriers. Vaccinate chickens being retained as layers. Whether broilers should be vaccinated depends upon many factors and is an individual decision. Numerous vaccines are available commercially. Most of them represent a modified or selected strain of the infectious bronchitis virus. The vaccine used should contain virus known to be present in the area. All vaccines contain live virus and those that give the best protection are also capable of producing symptoms and reducing egg production. The vaccine virus will spread to other susceptible birds. Vaccine is usually added to the drinking water, but may be dropped into the eye or nostril or used as a spray.

There is no treatment for this disease. In young chickens it is helpful to increase the brooder temperature and provide as nearly ideal environmental conditions as possible.

**Quail Bronchitis**

Quail bronchitis is a contagious, highly fatal disease in young bobwhite quail. The virus causing this disease also infects chickens and turkey. This agent is also known as a CELO (Chick Embryo Lethal Organism) virus. It has been isolated from chicken eggs but does not produce a recognizable disease in chickens or turkeys. This agent may play a part in respiratory diseases and in infertility problems, but its importance must be established by additional research. It is important because it is one of the agents that may be isolated from birds with respiratory symptoms and is difficult to separate from other agents such as infectious bronchitis virus.

**Lymphoid Leukosis**

Characteristically, lymphoid leukosis is a disease of adult chickens; however, the disease appears to be increasing in importance for turkeys and game birds. Although the virus of lymphoid leukosis can produce various responses (blood, bone, lymph), the lymphoid tumor response is the most common.

The disease is transmitted in a variety of ways. The causative viral agent is passed out of the body of infected birds via eggs and feces. The virus may be transmitted mechanically from infected birds to susceptibles by blood-sucking parasites or by man in such procedures as fowl pox vaccination.

Lymphoid leukosis characteristically produces lymphoid tumors, particularly in the liver and spleen. The tumors may also affect other visceral organs such as ovary and lungs. Affected birds may die without preliminary symptoms, but the disease usually is chronic in nature and
affected birds show loss of appetite, progressive emaciation and diarrhea. Clinically affected birds invariably die. Losses due to the disease are most severe shortly after onset of egg production, but losses will continue for as long as the flock is retained. Total loss may approach twenty percent during the life of a flock.

Clinical diagnosis of lymphoid leukosis is based upon flock history and disease manifestations. The lymphoid disease cannot be readily distinguished from the visceral response to Marek’s disease; however, there are some features that aid in differential diagnosis.

There is no treatment for lymphoid leukosis. Although the disease cannot be prevented completely, there are certain steps that can be taken to help control the level of infection within a flock. Some steps are:

- Buy resistant strains of birds since genetic resistance is a deterrent,
- Brood in isolation and do not mix birds of different ages, especially through six weeks of age,
- Keep the incubator clean and disinfected,
- Control blood-sucking parasites,
- Good care, limiting stress, and adequate ration will be of benefit.

**Marek’s Disease (Visceral Leukosis)**

Marek’s disease is characteristically a disease of young chickens but older birds can also be affected. In contrast to the lymphoid leukosis tumor response, Marek’s disease may be observed in more diverse locations.

Marek’s disease is caused by a virus belonging to the Herpes virus group. Much is known about the transmission of the virus; however, it appears that the virus is concentrated in the feather follicles and shed in the dander (sloughed skin and feather cells). The virus has a long survival time in dander since viable virus can be isolated from houses that have been depopulated for many months.

The usual mode of transmission is by aerosols containing infected dander and dust. Young birds are most susceptible to infection by Marek’s disease; however, since the incubation period is short, clinical symptoms can appear much earlier than in the case with lymphoid leukosis.

Marek’s disease may produce a variety of clinical responses, all lymphoid in character. These are acute visceral, neural, ocular, skin or combinations of the responses that can be seen.

Marek’s of the visceral type is characterized by widespread involvement with lesions commonly seen in gonads, liver, spleen, kidney and occasionally heart, lungs and muscles. The disease is often acute, with apparently healthy birds dying very rapidly with massive internal tumors. The disease may appear in broiler-age birds but the most severe losses
occur in replacement pullet flocks prior to onset of egg production.

The neural type of Marek's is typified by progressive paralysis of the wings, legs and neck. Loss of body weight, anemia, labored respiration and diarrhea are common symptom. If lesions are present, they are confined to the nerve trunks and plexes enervating the paralyzed extremities. Frequently no gross lesions can be observed.

Ocular (eye) leukosis or "gray-eye" is usually seen in early maturity. Morbidity and mortality are usually low but may approach twenty-five percent in some flocks. It is characterized by the spotty depigmentation or diffuse graying of the iris in the eye. The pupil develops an irregular shape and fails to react to light. Emaciation diarrhea and death follow.

Skin leukosis produces the most severe losses in broilers. The losses result from high condemnations at the processing plant. Enlargement of the feather follicles due to accumulations of lymphocytes is the typical lesion. This is the most infective virus since it is produced in the regions of the feather follicles and is shed with the skin dander.

Acute Marek's disease can be extremely rapid in its course, producing mortality in apparently healthy birds. However, in some cases the lesions may regress and clinically affected birds may make complete recoveries.

Diagnosis is based upon flock history and disease manifestations. Accurate diagnosis may depend on results of laboratory procedures. As is the case with lymphoid leukosis, there is no treatment for Marek's disease.

A vaccine is available that is extremely effective (90% +) in the prevention of Marek's disease. It is administered to day-old chickens as a subcutaneous injection while the birds are in the hatchery. Use of the vaccine requires strict accordance with manufacturer's recommendations in a sterile environment.

**Infectious Bursal Disease (Gumboro)**

Infectious bursal disease is an acute, highly contagious viral disease of young chickens. It is most often found in highly concentrated poultry producing areas. It causes marked morbidity and mortality in affected flocks. Although the disease causes severe losses, its affect on reducing the bird's ability to develop immunity to other diseases may be the most serious effect produced by this disease.

The transmission or spread of the disease can occur by direct contact (bird to bird), contaminated litter and feces, caretaker, contaminated air, equipment, feed, servicemen and possible insects and wild birds. It is extremely contagious.

Birds have ruffled feathers, a slight tremor at onset of the disease, strained defecation, loss of appetite and are dehydrated. Affected birds have a tendency to sit and when forced to
move, have an unsteady gait. Vent picking is common and a whitish diarrhea frequently develops. A sudden rise in body temperature is followed by a drop to subnormal temperature, prostration and death. Birds surviving the initial infection will recover rapidly within two weeks.

Postmortem lesions include dehydration and changes in the bursa, skeletal muscle, liver and kidneys. All affected birds have bursal changes characterized by swelling, change in shape (oblong), color (pink, yellow, red, black) and the formation of a gelatinous film around the bursa. Within a few days the bursa shrinks to half its normal size or smaller.

Diagnosis of infectious bursal disease is based on flock history and postmortem lesions. Laboratory procedures may be used to substantiate the diagnosis.

Vaccines are available but must be carefully used. If given correctly, good immunity can be developed. There is no specific treatment for infectious bursal disease and indiscriminate medication with certain drugs may severely aggravate mortality. Supportive measures such as increasing heat, ventilation and water consumption are beneficial.

PUBLICATIONS

PUBLICATION NUMBER: P2800
Northern Fowl Mite Management

PUBLICATION NUMBER: IS1953
Poultry Farming and Neighbors: The Little Things Are Important

PUBLICATION NUMBER: P3036
Choosing the Right Breed for Your Backyard Flock

PUBLICATION NUMBER: P3034
Modern Broiler House Heating Systems

PUBLICATION NUMBER: P3012
Rotary Drum Composting of Poultry Mortalities

NEWS

2016 Mississippi Commodities Photos
Filed Under: Agriculture, Catfish, Crops, Beef, Dairy, Poultry

December 16, 2016
Agriculture value is part of state's economy

Filed Under: Agriculture, Corn, Peanuts, Rice, Soybeans, Sweet Potatoes, Poultry

STARKVILLE, Miss. -- The estimated $7.6 billion value of Mississippi agriculture increased by 1.8 percent in 2016, helping the industry retain its prominence in the state's overall economy.

Poultry, forestry lead Mississippi agriculture

Filed Under: Agricultural Economics, Poultry, Forestry

STARKVILLE, Miss. -- Poultry remains Mississippi's top agricultural commodity with an estimated value of $2.9 billion, and it shows no signs of slowing down in 2017.

Forestry comes in a distant second with total farm-gate value of $1.4 billion, according to 2016 estimates.

Mississippi State University Extension Service economists just released their estimates for the state's agricultural commodity values in 2016. The top commodities remain poultry and forestry. Soybeans remain in the third spot, dropping 1.7 percent to just over $1 billion.

MSU researchers cool poultry with sprinklers

Filed Under: Poultry

STARKVILLE, Miss. -- The same principle that cools down kids running through a lawn sprinkler on a hot summer day is being tested on chickens in Mississippi State University's commercial poultry houses.

Tom Tabler, Extension poultry specialist with the MSU Extension Service, said keeping chickens cool in the summer is a life-or-death matter. Mississippi summer temperatures often exceed 90 degrees with humidity above 80 percent.

2016 offers bright hopes for state's poultry, eggs

Filed Under: Poultry

STARKVILLE, Miss. -- Mississippi poultry and egg companies are poised for expansions to fill the national gaps caused by the 2015 bird flu outbreaks in other states.

Tom Tabler, poultry specialist with the Mississippi State University
Extension Service, said companies are looking for more broiler growers or additional barns on existing farms.
We are an equal opportunity employer and all qualified applicants will receive consideration for employment without regard to race, color, religion, sex, national origin, disability status, protected veteran status or any other characteristic protected by law.

Produced by Agricultural Communications.

For information about the website contact webteam@ext.msstate.edu.

Copyright © 2017 Mississippi State University Extension Service. All rights reserved.