Diseases of Small Poultry Flocks

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The following list is the most common diseases I have diagnosed in backyard or small poultry flocks (mostly chickens).

1. Staphylococcus aureus infection
2. Escherichia coli infection (colibacillosis)
3. Mycoplasma gallisepticum infection
4. Marek’s disease
5. Infectious laryngotracheitis (ILT)
6. Visceral gout
7. Fatty liver
8. Cloacal prolapse
9. Osteomalacia
10. Ascite syndrome
11. Coccidiosis
12. Ascarids
13. Ectoparasites- mites and lice
Staphylococcus aureus Infection

**Definition:** Septicemic infection of many birds characterized by arthritis and tenosynovitis (inflammation of tendon sheath)

**Synonyms:** Staphylococcosis - often associated with bumblefoot or omphalitis (navel ill)

**Cause:** Staphylococcus aureus - Gram-positive coccus

**Epidemiology:** S. aureus is an opportunist that must penetrate skin barrier. Normal inhabitant on skin surface → skin injury → organism penetrates skin to invade blood. Skin injury may be scratches, excessively moist skin, needle or claw punctures

**Transmission:** Ubiquitous on skin and in environment and must penetrate skin. Not directly transmitted from bird to bird

**Clinical signs:** Large variety of diseases: 1. Omphalitis (yolk sac infection), 2. Necrotic dermatitis. 3. Necrotic skin lesions/abscesses 4. Arthritis and tenosynovitis, 5. Osteomyelitis (infection and inflammation of bone)

**Gross lesions:** Swelling on joints or footpad (bumblefoot); breast blister (yellow exudate in sternal bursa); dead chicks with swollen abdomen and crusted navel (omphalitis); osteomyelitis - yellow, necrotic plug in growth plate of bone.

*Staphylococcus aureus infections in foot of chicken (bumblefoot)*
**Diagnosis:** Bacterial culture of joints, bone, or yolk sac is definitive. Differential diagnosis: Pasteurella multocida (fowl cholera) and Erysipelothrix (erysipelas)

**Prevention/Treatment:** Reduce trauma in house- eliminate sharp objects, beak and toe trim, avoid wet litter or leaking drinkers; antibiotics (gram-positive spectrum such as erythromycin) may be useful during outbreak. Prevention is much more important than treatment.

**Escherichia coli Infection (Colibacillosis)**

**Definition:** Infectious disease in which at Escherichia coli is the primary pathogen or a secondary invader causing septicemia, peritonitis, cellulitis, omphalitis, salpingitis and airsacculitis.

**Synonyms:** Escherichia coli infection; coligranuloma, colisepticemia

**Cause:** Escherichia coli, Gram-negative rod; serotypes 01, 02, or 078, but often untypeable

**Epidemiology:** Ubiquitous, present in intestine of birds and mammals and is disseminated in feces. E. coli infections often result from management failures; often a secondary infection. Fecal contamination is most important.

**Transmission:** Birds infected by direct contact with dirty litter and hatches or contaminated egg shells. This is an environmental disease. Not transmitted from bird to bird.

**Clinical signs:** Nonspecific and include ill-thrift, ruffled feathers, enlarged and swollen navel, decreased appetite, depression, diarrhea, pasting of feathers around vent

**Gross lesions:** A variety of lesions depending on system that is affected:

1. **Airsacculitis, perihepatitis and pericarditis** - secondary invader as part of chronic respiratory disease results in white, friable material covering air sacs, liver and pericardial sac. Infection often preceded by Mycoplasma, Newcastle disease, or infectious bronchitis.

2. **Omphalitis** - swollen, red, crusted navels can be caused by contamination of egg shells through dirty setter, fecal covered eggs, excessive moisture during storage of eggs

3. **Septicemia** - hepatosplenomegaly (hepatitis), hemorrhages and necrosis in tissues

4. **Salpingitis/peritonitis** - especially laying hens; oviduct filled with yellow, cheesy exudate
5. Cellulitis ("scabby hip") of broiler chickens: yellow exudate underneath skin of hip, leg and breast
6. Synovitis/arthritis
7. Hypopyon- pus in eye following E. coli septicemia

**Diagnosis:** Histopathology; bacterial culture of affected organs is required for diagnosis.

**Treatment:** E. coli isolates from commercial poultry are often resistant to a variety of antibiotics. This is compounded by the fact that few antibiotics are being put on the market for poultry. Enrofloxacin was effective against this organism, but the antibiotic was removed from food production use in 2006. Regardless, culture and sensitivity may assist treatment if more than one bird is affected.

**Prevention:** Vigorous sanitation program in breeder house, at hatchery and at grow-out facility; Egg fumigation; dust control in house; routinely remove dead birds from house, avoid stress and immunosuppression (IBD).

Colibacillosis: One-day-old White leghorn chicks with omphalitis (left photo) and yolk sacculitis (right photo).
Colibacillosis: Nine-week-old turkey hen with fibrinous pericarditis, perihepatitis and airsacculitis

Colibacillosis: Fibrinous pneumonia in turkey hen
Colibacillosis: The most common cause of salpingitis in laying hens. Note enlarged and exudate-filled oviduct (arrows).

Left: White leghorn pullet with E. coli panophthalmitis; Right: Normal eye
Mycoplasma gallisepticum Infection

**Definition:** A chronic disease of a variety of birds, especially chickens and turkeys, characterized by nasal exudate, coughing and debilitation. Major problem in poultry industry for over 60 years. Monitored by NPIP.

**Synonyms:** “Chronic respiratory disease” of chickens; “Infectious sinusitis” of turkeys; MG, CRD

**Cause:** Mycoplasma gallisepticum; Mycoplasmas are small prokaryotes that lack a cell wall and are covered only by a plasma membrane. These organisms form fried-egg colonies in culture plates and generally require a protein-rich medium that contains 10-15% animal serum. M. gallisepticum, M. synoviae and M. meleagridis have the ability to agglutinate turkey or chicken erythrocytes, a feature that is utilized in the hemagglutination-inhibition assay to detect antibodies to these agents.

**Epidemiology/Transmission:** Transovarian- transmitted from breeder birds to offspring through egg; Horizontal infection via infected aerosols; Chronic respiratory disease of chickens is usually complicated by *Escherichia coli* infection, Newcastle disease virus or infectious bronchitis virus. MG alone produces mild lesions in chickens. MG can be common inhabitant of upper respiratory tract of clinically healthy birds.

**Clinical signs:** Adult laying hens- signs are rare; decreased egg production; decreased feed consumption and increased medication costs  
Broiler chickens- Chronic respiratory disease with coughing, sneezing (snicks), oculonasal discharge, poor feed conversion and air sac condemnations at processing  
Turkey- Infectious sinusitis- swelling of one or both infraorbital sinuses (usually MG alone), with nasal exudate on wings and air sac condemnations at processing

**Gross lesions:** Chickens- Classic triad of fibrinous pericarditis, fibrinous perihepatitis and airsacculitis  
Turkey- Swollen sinuses containing clear, serous fluid, usually MG alone
Infectious sinusitis: MG infection of swollen turkey infraorbital sinuses

Infectious sinusitis: MG infection of turkey infraorbital sinus (incised). Uncomplicated infections consist of a serous to mucoid exudate.

**Diagnosis:** History, gross necropsy, histopathology, serum plate agglutination test, culture or polymerase chain reaction

**Treatment:** usually does not eliminate infection and does not prevent egg transmission of infection. In small flocks the use of injectable tylosin might reduce the extent of clinical signs, but is not approved for birds in production (eggs or meat). For small breeder flocks it may be warranted to blood test for MG (plate agglutination and hemagglutination inhibition tests) and eliminate the positive breeders from the flock.

**Prevention:** Vaccination- chickens only, vaccine can kill turkeys (F strain); killed injectable (bacterin) or live spray vaccine (F strain or Mycovac L). Use and administration of vaccine by small flock owners can be problematic and can cause more problems than it solves because of potential for vaccine reactions. Best practice is to purchase chicks and poults from NPIP Mycoplasma certified-free breeder flocks; avoid swap meets and "exceptional deals."
**Marek’s Disease (MD)**

**Definition:** Marek's disease is herpesvirus infection that causes lymphoma of T lymphocytes. Tumors may occur in the nerves, ovaries, testes, viscera, eye, muscle, and skin. MD is ubiquitous throughout the world. The leg paralysis was often referred to as range paralysis. This is a classic example of vaccines being used to prevent cancer in chickens.

**Cause:** The disease is caused by a cell-associated herpesvirus (double-stranded DNA virus, hexagonal enveloped virus 150-160 nm diameter). There are three serotypes of MD virus: The oncoviruses (tumor-causing) are serotype 1; non-oncogenic viruses are serotype 2; and the herpes virus turkey (HVT) is serotype 3. The virus is intranuclear (cell-associated) and normally cannot live outside the host cell being protected from the environment by host epithelium.

Viral replication occurs in three phases of virus-cell interactions: Productive, latent and transformation.

1. **Productive infection:** Occurs mainly in nonlymphocytic cells with replication of viral DNA, antigen synthesized and viral particles sometimes produced. Full productive infection occurs in feather follicle epithelium that develops large numbers of enveloped and fully infectious virus. Productive restrictive infection occurs in some lymphoid and epithelial cells with viral antigen being produced, but most viruses formed have no envelope and therefore are noninfectious.

2. **Latent infection:** Occurs in lymphocytes, predominantly T cells, but occasionally B cells. The viral genome is not expressed and no virus or tumor-associated antigen is produced.

3. **Transforming infection:** Occurs in only T lymphocytes and is caused by only the virulent serotype 1 Marek’s disease viruses. This transformation in T lymphocytes results in formation of neoplastic lymphocytes with viral antigen expressed, but not virus. MD tumor-associated antigen is expressed on the cell surface. MD viral DNA is not integrated into the host genome.

**Transmission:** Infectious virus is produced only in feather follicle epithelium and spreads by direct or indirect contact between birds. The infectious virus contaminates the premises through infected molted feathers and dander. Birds become infected when they inhale dust containing the virus. Contaminated dust may remain infectious for several months.
Many apparently normal birds are carriers that can transmit the infection. Some birds have been found to shed virus from skin as long as eighteen months. Darkling beetles may also act as a mechanical vector to carry the virus between houses.

**Clinical Signs:** Under field conditions, it is difficult to determine the incubation period. Experimentally, clinical signs and gross lesions generally do not appear until between 3 and 4 weeks post-infection. In acute outbreaks, birds become severely depressed, anorectic and uncoordinated followed by unilateral or bilateral paralysis of legs and wings. Many birds become dehydrated, emaciated, and eventually die. The extremities affected include the legs, wings, and neck. In an infected flock, mortality gradually builds and generally persists for 4-10 weeks. Ocular Marek’s disease is characterized by decreased pupil size and irregular diameter ("grey eye"). A number of factors influence the extent of losses in affected flocks, such as virus strain, dosage, route of exposure, and genetic resistance of the host. Immunosuppression can be a long-term effect.

Marek’s disease: Splay-leg posture of pullet with sciatic neuropathy.

**Gross lesions:** Gross lesions can usually be found in one or more peripheral nerves particularly the sciatic and brachial nerves. Affected nerves are characterized by loss of cross-striations, gray or yellow discoloration, and may be swollen. Lymphoid tumors may be found in the gonads, heart, liver, lung, kidney, spleen, bursa, intestines, muscle, and skin. Skin lesions are not readily seen until feathers are removed at the processing plant. Feather follicles are enlarged and pale. USDA will condemn birds with skin leukosis. The cloacal bursa is usually not involved. "Late Marek’s" can occasionally occur in layers after the onset of egg production.
Marek's disease: Kidneys have been removed and the nerves comprising the pelvic plexus on the right (arrows) are slightly swollen and larger than the nerves on the left.
Eight-week-old Barred Rock chicken with Marek’s disease. Note the enlarged kidney (lymphoma) on the right (arrows). The sciatic nerve on the left (enlarged in inset) is of normal thickness with cross striations while the sciatic nerve on the right (enlarged in inset) is swollen, pale and has lost cross striations.

**Diagnosis:** A presumptive diagnosis is made based on the presence of tumors and the observed paralysis; however, gross necropsy and histopathology are important to make a definitive diagnosis and to differentiate Marek’s disease from other forms of paralysis or recumbency (“knockdown”).

**Treatment:** There is no specific treatment for chickens with Marek's disease. The emphasis is on prevention.

**Prevention:** Vaccination against MD effective in controlling the disease. Marek’s disease vaccine is usually administered on day 1 or injected into the embryo three days prior to hatch. There are three types of vaccines commercially available: the HVT serotype 3, natural occurring avirulent isolates of serotype 2, and nononcongenic strains of serotype 1 (Rispens). MD vaccines often achieve over 90% protection under commercial conditions. The cell-associated HVT vaccine is widely used because it is more effective than the cell-free HVT virus in the presence of
maternal antibodies. At least 1000-1500 plaque-forming units of virus are needed per dose. The HVT dose is often diluted or cut in hatcheries to save money, which may not protect against highly pathogenic MD. In areas where highly pathogenic field strains are prevalent, polyvalent vaccines containing serotype 2 and 3 viruses are commonly used. Chicks are normally vaccinated at the hatchery because they are exposed to the field virus at an early age. Cell-associated vaccines require extreme care in handling and application. Condemnations due to Marek's Disease can often be traced to errors in vaccination. In problem regions all three serotypes of MD virus can be included in vaccines.

Infectious Laryngotracheitis (ILT)

Definition: Infectious laryngotracheitis is an acute, highly infectious viral disease, characterized by conjunctivitis, loud gasping, tracheal plugs and coughing of blood-filled exudate.

Cause: Family *Herpesviridae*, Subfamily *Alphaherpesvirinae*, Genus *Iltovirus*, Species *Gallid herpesvirus 1*, *(infectious laryngotracheitis virus)*, dsDNA virus. ILT is a herpesvirus, a hexagonal shaped, enveloped, ether-sensitive virus that contains DNA. Four major envelope glycoproteins are the major immunogens. The virus replicates in the tracheal and bronchiolar epithelium; viremia does not occur. Field strains have been isolated that vary in their pathogenicity and virulence. Virus is destroyed by most commonly used disinfectants. In some instances infection is caused by vaccine virus increasing in virulence (“heating up”) as it spreads from vaccinated to non-vaccinated birds.

Transmission: This is primarily a human traffic-associated problem. Birds are infected by inhaling virus or via the intraocular route. Rapid virus replication occurs after the virus infects the upper respiratory tract. Transmission is rapid. Mechanical transmission can occur by use of contaminated equipment, clothing, shoes and litter. Some birds that recover from an infection can act as carriers (for up to 16 months virus is latent in dorsal root ganglia) and intermittently shed the virus. Because the virus spreads slowly birds can be vaccinated during an outbreak.

Clinical Signs: In acute infections, there may be nasal discharge, moist rales, coughing and gasping. The neck may be extended (“pump handle respiration”). The eyelids can be red with increased ocular drainage. In severe cases there is marked labored breathing and coughing of blood-stained mucus, which covers the wing and breast feathers as a result of the bird shaking the head with violent coughing. Blood and yellow exudate in the trachea cause death by suffocation. In mild cases, birds are unthrifty with red, swollen eyelids, and nasal discharge.
Mortality may vary from 5 to 70% depending on the severity of the disease. Most chickens recover in 2 weeks. Although textbooks state that egg production can decrease, in my experience the affects on egg production and feed consumption are often negligible.

Lesions: Eyelids can be swollen and red. Gross lesions are in the trachea and larynx. In severe cases, the trachea contains clotted blood. White fibrinonecrotic plugs can fill the glottis and trachea. In milder cases, the only lesions observed are mild tracheitis with mucus in the lumen, and swollen, red eyelids (conjunctivitis).

Extended neck ("pump handle") respiration demonstrated by adult leghorn chicken with infectious laryngotracheitis

Infectious laryngotracheitis: Firm white exudate (necrofibrinous tracheitis) plugs the tracheal glottis of white leghorn chickens. An important differential diagnosis for this lesion is poxvirus infection (wet pox).
**Diagnosis:** A presumptive diagnosis can be made on the history and the presence of blood and caseous/fibrinous plugs in the trachea. Since the clinical signs and lesions of the milder form resemble other respiratory diseases, such as wet pox, infectious bronchitis, Newcastle disease and avian influenza, a positive diagnosis must be made by histopathology of the trachea or virus isolation. The virus is cultivated on the chorioallantoic membrane of 10-day-old chicken embryos.

**Treatment:** If the disease is diagnosed early in an outbreak, vaccinating the entire flock and adjacent flocks may reduce the overall mortality. The administration of broad-spectrum antibiotics may help reduce secondary infections.

**Prevention:** In endemic areas, keep unauthorized personnel out of the poultry house, in addition vaccinating all breeder/layer birds at the proper time. Two types of ILT vaccines are most commonly used: Tissue culture-origin is administered by eyedrop at 1-day of age and a chick embryo origin vaccine, which is administered by drinking water or spray. Layers should be vaccinated prior to beginning egg production. Commercial layers are usually vaccinated by eyedrop or spray at 7-8 weeks of age, again by spray or drinking water at 12-14 weeks. Extra care must be taken to assure good vaccination technique avoid problems with vaccination reaction to ILT vaccines. Be sure to get vaccine to all birds in the flock because the vaccine virus tends to increase in pathogenicity (“heat up”) as it passes from bird to bird, and can eventually cause disease in nonvaccinated birds. For this reason, I rarely recommend ILT vaccination of small flocks.

**Miscellaneous/Metabolic Diseases**

**Visceral gout/urolithiasis:** Gout/urolithiasis is a condition very commonly seen in older layer flocks and is related to kidney failure. On occasion, gout can be a very significant part of flock mortality, sometimes as high as 0.5 % per week, but is often an insignificant cause of low-grade mortality. Kidneys can be damaged by low phosphorus diets at any age, water deprivation at housing, high vitamin D3 in the ration, or excessive calcium before sexual maturity (15 to 16 weeks). A nephrotropic infectious bronchitis (e.g., Australian T or Italian strain) can cause similar lesions. Losses due to gout tend to be chronic with the number of affected birds depending on the severity of the kidney insult. The strain of bird can also affect the severity and incidence of gout. Birds with gout usually show no clinical signs before death or may be extremely thin. The lesions of gout are associated with the accumulation of urates (uric acid is the primary nitrogenous excretory product of birds) on the surfaces of the internal organs (visceral gout) as well as within joint spaces and along synovial membranes (articular gout). The urates are gritty
and white as opposed to inflammatory exudate from bacterial infections, such as colibacillosis, which are yellow and friable. Portions of kidney are atrophic or absent and contralateral portions are often swollen (compensatory hypertrophy). Birds can be treated with varying success by adding ammonium sulfate or ammonium chloride to the ration but these treatments may cause wet droppings and deterioration of shell quality. Gout can be prevented or minimized by providing the proper nutrition of calcium and phosphorus throughout growing (1% calcium and 0.50 to 0.45% available phosphorus), starting layer levels of calcium feeding at the proper time (one week prior to first egg), and avoiding water deprivation at housing.

Visceral gout: Chaulk-like urate deposits on pericardial sac and liver capsule

**Fatty liver syndrome:** Fatty liver is caused by an imbalance of energy (positive energy gain) and protein intake. Fatty liver is observed most often in caged laying hens and occasionally in breeder turkey hens. Caged layers are particularly prone to fatty liver because of minimal exercise accompanied by high calorie intake; however, I am seeing more cases of fatal fatty liver in large, obese backyard hens, which are likely the dominant birds who control access to the feed. The liver is enlarged, pale orange, soft, friable and is easily fractured. **Rupture of the fatty liver with hemorrhage** into the abdominal cavity is a common cause of death in laying hens.
Treatment with choline chloride, vitamin K, biotin, and vitamin E in the feed for two weeks has been used to control mortality with varying results- these treatments are generally ineffective and not recommended. Fatty liver is positive-calorie problem and is prevented by use of an adequate diet with proper energy and protein levels- reduce these levels in the feed to prevent obesity in hens.

Fatty liver syndrome: Pale, fatty and friable liver is susceptible to rupture and hemorrhage.

Cloacal Prolapse: The cloaca (vent) of the laying hen everts when an egg is laid. The cloaca can become permanently everted and inflamed if is traumatized by other hens (cannibalism, pecking, peckout) or if the egg being laid is particularly large relative to the cloacal lumen. In small flocks one can see cloacal prolapsed in hens with small body conformation relative to the egg size (e.g., bantam chickens). Birds dying from peckout/prolapse will show hemorrhage around the vent area and most of the intestinal tract can be absent as a result of removal by other birds. Factors affecting the severity and incidence of cloacal prolapse include strain of bird, quality of ration, amount of floor, feeder, or drinker space, high light intensity, and large egg size. Young birds early in lay will be more susceptible to cloacal prolapse because the cloacal lumen has not become fully expanded to accept relatively large eggs. Low light intensity and providing adequate floor, drinker, and feeder space can decrease aggression in the house. Additionally,
vent trauma and cloacal prolapse in floor-raised chickens can be decreased by offering perches and obstacles/distractions (straw bales or plastic drinking jugs) to provide protection for hens, and maintaining adequate nest:hen ratios (1 nest: 4 hens) to decrease fighting for nest space.

Osteomalacia: Also known as cage layer fatigue in laying hens, this nutritional disease is characterized by decreased mineral in bone resulting in soft, pliable bones including the beak, curved keel bone and beading of the ribs at the costochondral junction. High-producing hens maintained in cages can show paralysis during the period of peak egg production. The birds carry approximately 200 grams of calcium in their skeletal system and use 2 grams per day to produce an eggshell. The condition has caused by impaired calcium flux related to the high output of calcium into the shell. Depletion of calcium in the medullary bone of the long bones is usually
seen in caged birds, suggesting that reduced activity/exercise is a predisposing factor. Affected birds can be found on their sides in the back of the cage. At the time of initial paralysis, birds can appear healthy and will have a shelled egg in the oviduct. Death occurs from starvation or dehydration, a failure of the birds to reach the feet or water. A high incidence of cage layer fatigue can be prevented if pullets receive a high calcium diet (minimum of 3.5% calcium) at least two weeks prior to the first oviposition. Older caged layers are also quite susceptible to bone breakage, particularly during transport to processing.

Soft bones occurring in young birds is referred to as rickets. This disorder is usually caused by abnormal Ca/P ratios in the ration, low vitamin D3 levels in the ration, or intestinal malabsorption associated with enteric diseases. The birds are often recumbent, but bright and alert with extremely pliable beaks and long bones.

Rickets: Nonambulatory turkey poult

Osteomalacia: Laying hen with deformed (S-shaped) keelbone
Ascites Syndrome and Right Ventricular Failure of Broiler Chickens

**Definition:** Ascites = accumulation of clear (serous transudate) fluid in the abdomen. Ascites syndrome is a noninfectious, metabolic disease of broiler chickens and ducklings characterized by pulmonary hypertension, right-sided heart failure and accumulation of excessive fluid (transudate) in the abdomen. This is a form of congestive heart failure in chickens. Birds are predisposed to right-sided heart failure because of a muscular flap that forms part of the right atrioventricular valve.

**Epidemiology:** Originally described in broiler chickens raised at high altitudes (decreased PO$_2$). Ascites is commonly observed in a small percentage of broilers at processing and mortality can approach 1-2% or greater in some flocks. Respiratory infection or poor ventilation can promote this problem.

**Clinical Signs:** Sudden death of previously healthy birds, usually at 3-5 weeks of age, in a flock with good rate of weight gain. Live birds are smaller than normal, reluctant to move, have respiratory distress and a distended abdomen. Combs and wattles are cyanotic (pale to bluish).

**Gross lesions:** Cyanotic combs and wattles; The abdomen is distended by abundant clear, yellow fluid and fibrin. Fibrin clots often surround the liver, which is small. Do not confuse with chronic respiratory disease. Lungs are dark red and wet (edema). The pericardial sac can be filled with clear fluid (hydropericardium). The right ventricle is dilated with thin, flaccid wall and there can be degeneration of the of the right atrioventricular valves as evidenced by firm, white nodules (endocardiosis). The right ventricle is predisposed to failure because of a septal valve that is composed entirely of cardiac muscle rather than fibrous connective tissue.
Four-week-old broiler chicken in respiratory distress (facial cyanosis, prostrate with neck extended) in terminal stages of ascites syndrome.

Ascites Syndrome: Broiler chicken with abundant clear fluid distending the abdomen

Ascites Syndrome: Cross-section of heart, broiler chicken. Marked dilation of right ventricle (arrows). Bar = 1 cm.
Predisposing factors:
Broiler chickens that are bred for high feed efficiency with rapid rate of growth and muscle development

Broiler chickens have lower ratio of total lung volume:body weight compared to ancestor (Red Jungle Fowl) or white leghorn chicken.

Continuous availability (ad libitum) of high energy and high quality feeds typical of grower ration

Pathogenesis (How does ascites occur?)

Cornish breeds
- High rate of gain
- High energy feed

Rapid increase in muscle mass
- High oxygen demand for muscle growth

Increased oxygen consumption
- Slower growth rate of heart and lungs relative to skeletal muscle

Hypoxia (decreased PO$_2$ in blood)

Increased cardiac output = Increased amount of blood pumped by heart
- Increased blood flow to lungs
- Increased number of circulating red blood cells (polycythemia)
- Increased hemoglobin inside red blood cells

Pulmonary hypertension (increased pulmonary blood pressure)
- Increased workload on right ventricle to pump blood to lung
  - Dilated right ventricle with thickened wall

Failure of overworked right ventricle (right-sided heart failure)
- Systemic hypertension

Loss of fluid across liver capsule into body cavity

Fluid accumulation in air sacs and lung (suffocation)
Prevention:

Decrease energy level in feed to slow down growth rate. Try limiting feed during the grow-out or turning off house lights at night. The birds should be acclimated to dark-out periods at an early age to minimize panic.

If mortality is >1-2% from ascites look for other complicating factors such as high sodium levels in feed or water, vitamin E or selenium deficiency, respiratory infection, furazolidone or coal tar toxicity

Maintain good air quality with adequate ventilation, especially during winter

Coccidiosis (*Eimeria* spp.)

Cause: Protozoan parasite of the intestinal tract of poultry

Remains one of the most common and expensive diseases of poultry despite improved chemotherapy, management and genetics

Most coccidia affecting chickens are in genus *Eimeria* (up to 9 species)

Species-specificity: The species of coccidia that infect chickens do not infect turkeys and vice versa.

Most field outbreaks involve infection with two or more species of *Eimeria*.

Infections more common in birds raised on a floor environment, but infections can occur in caged birds (e.g., pullets and layers) to produce substantial mortality

Life cycle

Initiated by ingestion of sporulated oocysts (eggs) and usually takes 4-6 days to complete life cycle

Involves both asexual (schizogony or merogony) and sexual (microgametes fertilize macrogametes) stages

Asexual and sexual stages occur in different sites of intestinal tract depending the species of *Eimeria*. 
Eimeria: A single, mature oocyst (egg) contains four sporocysts, and each sporocyst contains two sporozoites (eight sporozoites in each oocyst)

**Sporulated oocysts in poultry house**

- Oocysts consumed by bird

**Gizzard: oocysts crushed to release sporozoites**  DAY 1

**Intestine and cecum epithelium (species dependent)  DAY 2-5**

- Asexual Stage: Sporozoites invade epithelial cells and divide to produce numerous schizonts (cysts) filled with merozoites
- At least two generations of asexual development (first and second generation schizonts containing first and second generation merozoites)
- Merozoites are final product of asexual stage

**Intestine and cecum epithelium**  DAY 5-7

- Sexual Stage: Macrogametes and microgametes produced inside epithelial cells
- Microgametes (small) are released to seek out and fertilize the larger macrogametes to form zygote
- Zygote matures into oocysts
- Oocysts rupture from epithelial cell to enter intestinal lumen

**Feces**

Oocysts undergo further maturation (sporulation) in environment to become infectious. Sporulation takes less than 48 hours and occurs more rapidly in warm, moist litter

**Clinical signs**

- Usually turkeys and broilers
- Range-reared, cage-free layers are at increased risk for coccidiosis
- Pale combs and wattles from blood loss in gut
- Ruffled feathers, depression, blood in droppings, shivering
- Mortality rate may increase, particularly in caged pullets
- Decreased egg production in birds at reproductive age
Gross lesions

Chickens: Lesions vary depending on what species of Eimeria are involved

**Eimeria acervulina**: white “tiger-striping” of upper small intestine (duodenum)

**Eimeria necatrix**: Mid and distal small intestines (jejunum and ileum): White spots (large schizonts) in mucosa with mucus and blood in lumen

**Eimeria tenella**: Hemorrhage in colon and cecum progressing to cores of coagulated blood in lumen of cecum.

**Eimeria tenella** causes marked cecal hemorrhage in chickens.

**Eimeria maxima**: Orange, mucoid material in lumen of jejunum

**Eimeria brunetti**: Mucus and blood in ileum and colon; Can occasionally see cores of blood in cecum.

**Diagnosis**

Diagnosis is made by observing gross lesions and confirming presence of intestinal oocysts by histopathology. Alternatively, segments of infected intestine can be opened and the mucosa gently scraped with a glass coverslip. Place the coverslip on a glass microscope slide and identify the oocysts cytologically.
Oocysts of Eimeria as observed on a mucosal scraping (40X objective).

Prevention/treatment

Coccidia can be attacked at three stages

1. Anticoccidial drugs that kill (coccidiocidal) or decrease the growth rate (coccidiostat) of coccidia at days 1-4 of infection

   Shuttle programs: Change of coccidiostats within the grow-out period of a single flock. One product used in starter ration and a different product used in grower ration in order to reduce build-up of resistant coccidial strains
   For example, use Nicarb or Coban for 3-4 weeks and then switch to Avatec for 5th week up to withdrawal

   Product rotation: Change products up to twice a year (e.g., spring and fall) to reduce build-up of resistant strains

2. Development of immunity to coccidia, which is usually greatest during the asexual stages. This is most important in layer breeders and floor-raised/range-reared layers.

   Live, attenuated coccidial vaccines contain up to 6 species of Eimeria

3. Killing oocysts in environment or preventing contact with viable oocysts (cage system for pullets)
**Roundworms = Ascariasis**

**Cause:** *Ascaridia galli*: a nematode residing in the upper small intestine of chickens and turkeys

Young birds (<3 months) are most susceptible to intestinal damage and light-weight breeds (e.g., White Leghorns) are more susceptible to infection than heavy meat-type breeds (e.g., White Rock)

Malcom Reid (University of Georgia): Layers harboring 10-14 adult worms in intestinal tract had 5-7% reduction in egg production

Infection can occasionally occur in caged layers that have been exposed to contaminated flies.

Direct Life Cycle (no intermediate host): About 30 days to complete cycle

- **Embryonated egg** (takes 10-12 days in environment; faster if warm and moist litter)
  - Lumen of proventriculus or duodenum
    - Larvae hatch and live in lumen of duodenum for 9-10 days
  - Wall of duodenum
    - Larvae migrate in wall of small intestine for 8-9 days to cause tissue damage and hemorrhage
  - Lumen of duodenum
    - Young adult (male and female) re-enters lumen of intestine
  - Lumen of small intestine
  - Feces
    - Mature adult produces eggs by **30 days after infection**

Eggs contaminate environment and can remain infective for up to 160 weeks on the ground. Resistant to low temperatures

**Clinical Signs**

- Depression, weight loss, diarrhea
- Decreased growth
- Lowered egg production in heavy infection

**Gross lesions**

Worms in small intestine can occasionally cause death by blockage/impaction
Intestinal Roundworms in a floor-raised pullet

Reddened intestinal mucosa (enteritis)
Thin bird; atrophy of breast muscle, decreased body fat
Ascarid can migrate to oviduct and penetrate egg prior to shell formation

Diagnosis

Necropsy: identification of worms that are large, yellow-white, and 5-11 cm long
Fecal flotation (eggs float in zinc sulfate or sodium nitrate solution)

Prevention/Treatment

Confinement and cage rearing has reduced problems with most intestinal parasites
Deep litter (4-6 inches of wood shavings) to reduce exposure to parasite eggs
Proper clean up between flocks
Treat pullets before they are transferred to laying house
Treatment: Piperazine

Ectoparasites

Mites and lice are the most common ectoparasites in poultry. Ectoparasites can cause decreased production and uniformity and also provide opportunity for transmission of other diseases.

Feather mites
Synonyms: Acariasis; Dermanyssus gallinae = the red roost mite or red chicken mite

Causes: The two common mites of poultry are the Northern fowl mite (Ornithonyssus sylviarum) and the red chicken mite (Dermanyssus gallinae).

Northern fowl mite (Ornithonyssus sylviarum): This mite can cause economic loss as a result of
decreased egg production, increased feed consumption, reduced weight gains, and reduced seminal fluid production in roosters. Roosters tend be more heavily infested than the hens. The entire life cycle is completed in five days and adults can live off of the host for three to four weeks; however, in most instances the mite is on the host bird for the entire life cycle and at all times of the day. Northern fowl mite disease is worse during cold months of the year when birds have close contact.

*Ornithonyssus sylviarum*: Mites and mite excreta on the skin and vent feathers of a white leghorn chicken.

*Ornithonyssus sylviarum*: Mites pepper the wing feathers of a white leghorn chicken. Left = adult mite (10x).

*Red roost mite (Dermanyssus gallinae)*: This mite feeds on blood and is primarily active at night (night
feeder), and during the day will leave birds to reside in nest boxes and cracks and crevices of perches and slats in the poultry house.

**Clinical signs:** Mites are most often observed in young laying birds and appear as rough, scaley skin, dark, stained feathers around the vent as a result of accumulation of mite eggs, scabs and fecal material. Mites feed on blood and heavy infestations can cause anemia. Decreased egg production can occur.

**Gross lesions:** Mite can be observed on the feathers of the vent, particularly in hens, and over the entire body in roosters. Check more than one bird for the presence of mites because not all birds are infested. Check birds at night if you are looking for the red roost mite (mites are off of the bird during the day).

**Treatment:** In heavy infestations chemical insecticide sprays can be used. The insecticides are available as wettable powders, emusifiable concentrates, soluble concentrates, granules and microencapsulated products. The spray must be at high enough pressure (100-125 PSI with one gallon of water per 100 birds) to penetrate the feathers of the vent region. Commercial acaricidal insecticides(e.g., pyrethrins and permethrins such as Ectobon, Rabon and Ravap) often contain wetting agents to help the active chemical penetrate to the feathers. Instructions on the commercial insecticide sprays should be carefully followed. Caged layers should receive the spray from beneath the cage to increase access to the vent region. Floor raised breeders should be sprayed at night when they are resting on slats. Because the red roost mite commonly spends time off of the bird at night, these infestations require that, in addition to bird treatment, the nests and cracks/crevices of the poultry house should be sprayed or dusted with insecticide.

In small flocks it may be economical to use a carbamate product, commercial rose dust (Sevin Dust). Place 2.5 pounds of 5% Sevin dust in one dust box per fifty birds. Carefully dust the product on the vent region and also sprinkle the dust around nest boxes and along cracks and crevices in the hen house.

**Prevention:** Make sure the house is cleaned, disinfected and free of mites and lice before introducing new birds. New birds introduced to a house should be carefully examined to determine that they are free of ectoparasites. Examine equipment to prevent transmission of mites on clothing, egg flats, crates, etc. Use washable egg flats and steam clean or power wash equipment that is moved into a new house. Maintain biosecurity to keep rodents and wild birds out of houses.

**Scaley-leg mites**

*Knemidocoptes mutans.* This is one of several species of scaley-leg mite that exist on a variety of birds including chickens, turkeys, pheasants, partridges, and many passerine birds. The mite found most often
on older birds and spends the entire life cycle in the skin. A similar mite infection (Knemidocoptes pilae) in psittacine birds causes scaling on the beak and legs. Transmission occurs through direct contact with other birds. Female scaley-leg mites burrow into nonfeathered portions of the body, particularly the scales of the legs, but also the comb and wattles, resulting in marked proliferation of the skin with heavy and irregular keratin crusts. The females deposit eggs for two months after burrowing. Both larval and nymph stages follow after the eggs hatch. The heavy crusting on the leg can interfere with joint flexion and can cause lameness. Severe infections can result in loss of toes, decreased feed consumption and decreased egg production. The disease can be treated by dipping the legs in dilute insecticide (acaricidal) solutions. Birds that are not used for egg or meat production can be effectively treated with a drop of ivermectin solution (Ivomec, off-label treatment) on the comb or wattle. Repeat this treatment two weeks later.

Adult bantam roosters (A and B) with scaley-leg mites: Note the enlarged and deformed shanks and feet with excess accumulation of keratin caused by Knemidocoptes mutans.
Lice  (Pediculosis)

Body lice are larger than mites and can build up in large numbers on affected birds, especially caged layers. Lice spend the entire life cycle on the bird and can live for several months. The lice that infest birds are chewing lice (Mallophaga) rather than blood feeders, feeding on dander and feathers. Laying hens that are infested can have significant decreases in egg production and weight loss.

Causes:
Chicken:  *Cuclogaster* (head louse), *Goniocotes* (fluff louse), *Menacanthus* (body louse)
Turkey:  *Menacanthus*, *Chelopistes*, *Oxylieurus*
Duck and goose:  *Anaticola*, *Trinoton*
Pigeon:  *Columbicola*, *Campanulotes*
Two types of lice (Cuculogaster and Columbicola) found on a floor-raised laying hen. Note lice both on the bird (A) and collected from the bird (B).

**Clinical signs:** Decreased egg production, decreased feed consumption, reduced body weights. Birds may appear irritable or hyperexcitable. Feathers can appear ragged with fluff missing from the feather barbs.

**Gross lesions:** Feathers can appear ragged with fluff missing from the feather barbs. Nits or louse eggs are laid in clusters attached to the base of feathers of the vent.

White leghorn chicken with feather lice: Note the louse eggs (nits) encompassing the shafts of intact (A) and plucked (B) feathers.
**Treatment and Prevention:** This is similar to recommendations made for the Northern Fowl mite, but the louse eggs are resistant to insecticides and a second treatment (spray or dust) is required at two weeks after the first to kill the lice that have recently hatched. Be sure to treat the entire body of the birds because in heavy infestations the lice tend to spread from the vent to the neck region.