Tick-borne diseases are extremely important and emerging diseases in the United States in both humans and animals. The area in which you live will influence the diseases that are circulating in the environment. Although diseases such as Lyme disease has received a great deal of attention, other important diseases including ehrlichiosis, Rocky Mountain spotted fever, anaplasmosis and cytauxzoonosis have been emerging in various areas. A good travel history is imperative given various species of ticks and tick-borne diseases are more common in certain geographical areas. More information on tick-borne disease distribution can be found at http://www.capcvet.org/parasite-prevalence-maps/

Identification of ticks
Tick bodies are divided into two primary sections including fused head and thorax and abdomen. All adult and nymphal forms have 4 pairs legs and no antennae and all larval forms have 3 pairs of legs. The importance of determining larvae vs other stages include to determine the likelihood of tick being infected with various pathogens. Unless transovarial transmission occurs, larvae are unlikely to be infected with pathogens, while nymphs and adults have higher likelihood include with pathogens in transstadial transmission. Whereas hard ticks have scutum, soft ticks do not have scutum. Ticks are great vectors due to their ability to be persistent blood-suckers which attach firmly & feed slowly, long life spans, may be geographically widespread, resistant to environmental conditions, high reproductive potential, and can pass infective agents through egg to next generation and/or through successive stages. Ticks bites in themselves can lead to wounds and inflammation from salivary proteins. Secondary infection and disease can be due to toxicosis, local necrosis, and tick paralysis. Tick bites predispose animals to secondary attacks by myiasis-producing flies.

Soft tick have no scutum are soft, tough, leathery body, do not stay attached—instead take multiple small volumes of blood, and often feed at night.

Soft ticks include Otobius megnini (Spinose Ear Tick) transmits relapsing fever caused by a Borrelia spp. (different than Borrelia burgdorferi which causes Lyme Disease). Spinose ear ticks are more common in western states that are west of 100th meridian have no scutum are soft, tough, leathery body, do not stay attached—instead take multiple small volumes of blood, and often feed at night.

Hard Ticks is largest family of ticks has a scutum (dorsal, hardened plate) that covers entire dorsum of males and forms an anterior shield in females. Hard ticks remain attached until engorged and then fall off to molt or lay eggs. General life cycle include:

- Egg → 6-legged larva → 8-legged nymph → 8-legged adult
- Oviposition (egg laying) occurs off of the host

Nymphs and adults can be identified based on visual exam but often unable to distinguish larvae without microscopic exam. Nymphs and adults are more likely to harbor pathogens than larvae—this is why you need to be able to distinguish larvae (6 legs) from nymphs/adults (8 legs).

Tick species

All dermacentor spp.
- Ornate ticks with eyes
- Basis capitulum (mouth part) is rectangular if viewed from above and has stubby palps
- Resembles Rhipicephalus (both have11 festoons, small rectangular patterns on posterior abdomen)

Dermacentor variabilis (American dog tick)
- Eastern half of U.S. and west coast, but rare in Central US
- Dogs, cats, humans, horses, cattle, fox, rodents, and other mammals
- Can cause tick paralysis in humans, dogs, etc.
- May take as little as 3 months, with favorable conditions, or up to 2 years
- Principal vector of Rickettsia rickettsia - Rocky Mountain Spotted Fever (RMSF) and others in Spotted Fever Group
- Infrequent vectors of tularemia, anaplasmosis, Babesia canis, Cytaxzoic felis

Rhipicephalus sanguineus (brown dog tick)
- Wide distribution
- Rhipicephalus ticks are similar in appearance to Dermacentor, except they have a hexagonal basis capitulum. All stages parasitize on dogs and will attach to other animals, but usually not humans
- Can survive indoors for months to possibly years without a blood meal
- Domestic & kennel problem due to tropical nature of tick and because it cannot survival outdoors in North America
- Vectors Babesia canis voglei, tularemia, Ehrlichia canis, RMSF—Very important vector of RMSF in humans and dogs in southwestern US
All Amblyomma spp.

- Ornate ticks
- Long mouth parts & commonly 11 festoons—allows one to differentiate from Ixodes spp which lack festoons

Amblyomma americanum (Lone Star Tick)

- Wide distribution, but mainly in southern U.S.
- Large silver spot at apex of scutum on females — hence name “lone star”
- All stages feed on wild & domestic animals, birds, & humans and is significant pest for humans & animals
- Can transmit Coxiella burnetii (Q-fever), tularemia, Ehrlichia chaffeensis, Ehrlichia ewingii, RMSF,
- Vectors agent of Southern Tick Associated Rash Infection (STARI) in humans
- Cause of STARI is currently unknown—may actually be the host reaction to tick saliva—leads to swelling and pain at bite region in people.

All Ixodes spp.

- Inornate ticks and No festoons, has distinct anal groove anterolateral to anal orifice
- Used for identification in NON-ENGORDED tick but can’t see groove in engorged ticks—use mouth parts instead

Ixodes scapularis (Black-Legged Tick)

- Wide distribution, in East, South, and Midwest U.S. Highest populations in upper Midwest and New England/midatlantic states
- Primary Lyme disease (Borrelia burgdorferi) vector in Eastern US and Midwest
- Vectors Babesia microti, Anaplasma phagocytophila

Ixodes pacificus (California Black-Legged Tick)

- Primary Lyme disease vector in the West Coast

Tick-borne diseases

Tick paralysis

Potentially fatal reaction to a paralyzing neuromuscular toxin secreted in the saliva of a female tick late in her feeding. Cattle, sheep, horses, dogs, and humans seem to be most affected.

Clinical signs include: headache, vomiting, general malaise, loss of motor function and reflexes, followed by paralysis that starts in the lower body and spreads to the rest of the body

Respiratory failure and death can result. Signs disappear rapidly when tick is removed, suggesting that the toxin is rapidly excreted or destroyed

Lyme Borreliosis

- Agent: Borrelia burgdorferi
- Vector: Ixodes scapularis (Eastern and Midwestern US), Ixodes pacificus (Western US)
- Animal health: Major cause of canine and equine disease, including endocarditis and joint pain. Most cases occur in the spring and summer, during nymphal emergence, and in late fall and winter, during adult emergence.
- Human health: Acute and chronic diseases including joint pain, heart disease, and neurological disorders. Most cases occur in the spring and summer, during nymphal emergence, and in late fall and winter, during adult emergence.

Rocky Mountain Spotted Fever

- Agent: Rickettsia rickettsia
- Sometimes placed in “Spotted Fever” disease group
- Vector: Dermacentor variabilis
- Geographical distribution: Eastern US mainly. Most frequently reported tick borne disease in the eastern US. Other agents other than R. rickettsia can lead to spotted fever group disease in humans. Clinical signs include flu like symptoms as well as petechial hemorrhage.

Anaplasma phagocytophilum:

- Intracellular rickettsia that causes human granulocytic anaplasmosis
- Infects granulocytes and leads to bleeding, fever, leukopenia,
- Clinical signs/symptoms may be worse with co-infection with Lyme or Babesia
- Vectored by Ixodes scapularis so same geographical distribution as Lyme Disease. Can be transmitted by blood transfusion.
- Diagnosis: clinical signs, PCR (acute cases), serology (chronic), CBC to look for leukopenia, Blood smear to look for morulae in granulocytes.
Cough it Up:  
Respiratory Parasites of Animals  
Richard Gerhold, DVM, MS, PhD  
University of Tennessee  
Knoxville, TN

*Dirofilaria immitis* – See migrating heartworm proceedings  
*Toxoplasma gondii*—See Toxoplasma: microscopic monsters proceedings

**Paragonimus spp. – Lung fluke**  
- Life cycle 1st IH – snail, 2nd IH - crayfish and crabs (crustaceans) DH - mammals (dogs, cats)  
- PPP – 1-2 months  
- Adults – 7-12 x 4 - 6 mm  
- Eggs – 75-118 x 48 - 65 μm  
- Diagnosis - Sedimentation/Sugar Float

**Clinical Signs/Pathologic changes**  
- Respiratory problems  
- Cough  
- Lethargy  
- Pneumothorax

**TREATMENT**  
Praziquantel – higher dose/repeated  
Fenbendazole – 14 days

**CONTROL/PREVENTION**  
Uncooked Crayfish/Crab

**Metastrongyloidea- “Lungworms”**  
- males with a caudal copulatory bursa  
- buccal cavity small  
- usually leave the definitive host as larvae rather than eggs  
- usually live in the lungs of mammals  
- life cycles commonly indirect (snailslug intermediate hosts typical)  
- migratory in definitive host  
- Clinical signs  
  - Coughing  
  - Moderate to severe dyspnea  
  - Loud breath sounds  
  - Fever

**Diagnosis- Zinc sulfate float and/ or Baermann exam**

**Dictyocaulus** - large lungworm  
D.H. - cattle, sheep, goats, horses, and other herbivores  
*D. viviparus* - cattle  
*D. filaria* – sheep, goats  
*D. arnfieldi* – equids (donkeys)

**Muellerius - hair lungworm**  
- I.H. - snails, slugs  
- D.H. - sheep, goats  
  - ♂ 11 - 14 mm  
  - ♀ 19 - 23 mm

**L1  250-300 um**

**Aelurostrongylus – feline lungworm**  
- I.H. - snails and slug  
- Paratenic Hosts- amphibians, reptiles, birds, rodents  
- D.H. - cats (félidae)
• \( \sigma^4 \) 4 - 6 mm  \( \varphi^9 \) 9 - 10 mm  
• L1 350-400 \( \mu m \)  
• PPP- 6 weeks  
• Treatment (EXTRA LABEL)  
  o Fenbendazole (Panacur -10 days)  
  o +/- Ivermectin/Selamectin  
  o Advantage Multi (Moxidectin) & Profender (Emodepside/Praziquantel)  
  o Prednisone (1 mg/kg PO BID for five days)  

**Angiostrongylus vasorum**  
• fox lungworm/ French Heartworm  
• I.H. - snails and slugs (mollusks)  
• Paratenic Hosts- amphibians, reptiles, birds, rodents  
• D.H. – fox, dog  
• \( \sigma^4, \varphi^9 \) 14 to 20 mm (\( \varphi \) barber pole)  
• L1 310-400 \( \mu m \) - anterior cephalic button with a dorsal spine  
• PPP- 7 weeks  

**Capillarids**  
• Eggs with polar plugs Size 50 - 80 x 20 - 40 \( \mu m \) - often mistaken for *Trichuris* spp eggs  
• Clinical signs include sneezing, coughing, respiratory distress  
• Diagnosis fecal float  
• *Eucoleus aerophilus* – lungworm  
  D.H. - dogs, cats, foxes, raccoons  
• *Eucoleus boehmi* - nasal worm  
  D.H. - dogs, fox, (cat)  
• Treatment– Ivermectin, Fenbendazole, Moxidectin  

**Miscellaneous migrating larvae**  
• Ascarid spp in general have a lung migration during larval phase. High level of infection in young animals can lead to respiratory disease. Can attempt fecal float for diagnosis but may be during pre patent period so may obtain negatives on fecal floats  
• Toxocara canis-dog  
• Toxocara cati-dog  
• Ascaris suum – pig  
• Parascaris -equine
Dirofilaria immitis – heartworm
I.H. – mosquitoes
D.H. - dogs and wild canidae, marine mammals, ferrets, cats
♂ 12-22 cm (6-9 inches)
♀ 25-31 cm (12-14 inches)
Mf 300 - 325 x 6 - 7 μm
PPP 6 months

Life cycle
- Juvenile worm matures to adult over next 3 months in dog.
- Microfilaria produced by young adult worms 6 months post infection (6 month Life Cycle)
- Male worms 6-9 inches, females 12-14 inches
- Lifespan is 5 to 7 years in the dog
- Average infection is 14 worms but can be in excess of 100

Clinical signs
- Cough
- Dyspnea
- Tiring on exercise
- Weight loss
- Classic patient: Active middle-aged dog
- Ascites
- Anemia
- Eosinophilia and thrombocytopenia
- Glomerulonephritis and proteinuria

Reasons for a dog to be AG positive and Knott’s/Filter negative
- 5 month old worms (too young –rem PPP)
- All female worms (single sex)
- Immunological Occult
- Prophylaxis/Drug induced
- Few mf present

Reason for a dog to be MF positive and AG negative
1. Adults dead/mf circulating
2. Ag sequestration/antigen antibody complexes

Time of testing
- The earliest heartworm antigen is detected is 5 months post infection
- With low worm burdens or animals on macrocyclic lactone preventives, antigenemia can be delayed to 9 mos.

What tests are recommended during annual physical exam?
1. Serology for heartworm antigen  AND
2. Microfilariae concentration test
   a. Same two diagnostic tests are recommended for dogs displaying clinical signs suggestive of heartworm disease
   b. Notes on testing recommendations from AHS
- Antigen testing - most sensitive diagnostic method when screening an asymptomatic dog or seeking verification of a suspected heartworm infection
- But a study conducted on shelter dogs found a 7.1 percent false negative rate because of formation of antigen-antibody complexes.
- AHS now recommends mf testing in tandem with AG to detect dogs that are AG- but mf+
What would you do before treatment?

- Evaluate the dog
  - Already have results from Knott’s & Ag test
- Radiography to assess severity of cardiopulmonary disease
  - Enlarged, tortuous, and often truncated peripheral intralobal and interlobar branches of the pulmonary arteries, particularly in the diaphragmatic (caudal) lobes
  - Pulmonary parenchymal disease, right heart enlargement etc
- Echocardiography

Stabilize dogs presenting with clinical heartworm disease

Treatment AHS/CAPC -3  immiticide dose regimen

- Safety
- Efficacy
- Decreased possibility of needing further melarsomine treatment
- By initially killing fewer worms and completing the treatment in two stages
  - Reduces cumulative impact of worm emboli on severely diseased pulmonary arteries and lungs

Current treatment protocol for positive dogs

First month

- Start macrocyclic lactone (preventive) and continue monthly for life
- Rx Doxycycline 10mg/kg bid for 4 weeks

If dog can not tolerate dose, reduce to 5mg/kg
  - Wolbachia nos will remain low for 3 to 4 mos
    - If dog symptomatic, Rx Prednisone 1mg/kg reducing weekly during 1st month.
    - Begin exercise restriction.

Second month

- Give second dose of heartworm preventive.

Third month

- Give third dose of heartworm preventive.
- Give one injection melarsomine (Day 61).
- Rx Prednisone 1mg/kg reducing weekly.
- Decrease activity level even further. Cage rest in more severe cases.

Fourth month

- Give fourth dose of heartworm preventive
- Give second and third melarsomine injections (Day 90 & 91).
- Rx Prednisone 1mg/kg reducing weekly for four additional weeks.
- Continue exercise restriction for 6 to 8 weeks after last melarsomine injections.
- Antigen test in 6 months
- Knott’s test or other test for microfilariae in 6 months
- Any treatment method utilizing only macrocyclic lactones as a slow-kill adulticide is not recommended!!!
- New information about resistance also prompted the AHS to place additional emphasis on the importance of year-round administration of heartworm preventives.

Diagnostic tests in cats

- Use both antigen and antibody test
- Antigen Test Kits
  - Only detects adult female worms.
  - Average worm burden in the cat is 1-2 worms and is frequently only males.

“Asthma” like syndrome occurs 3-4 months post infection. Antigen test incapable of confirming HW as etiology

- Antigen tests:
  - Detect antigen produced by adult worms (Produced by adult, female worms)
  - First detection at 5-8 months P.I.
- Antibody tests:
  - Detect antibody produced against larval and adult worms
  - First detection at about 3 months
Feline heartworm treatment goals

- Relieve acute signs (usually respiratory) May be due to adult or larval infection
- Control chronic signs (respiratory, vomiting)
  - Prednisone (2mg/kg-decreasing doses one month)
- Prevent reinfection- prophylaxis
- Rid patient of Adults via surgery (possible? advisable?)
Neospora, Toxoplasma, and Coccidia: Microscopic Monsters
Richard Gerhold, DVM, MS, PhD
University of Tennessee
Knoxville, TN

Hosts/Disease
- Cats serve as definitive hosts and numerous mammals and birds are the intermediate hosts
  - Most cats in the wild become infected shortly after weaning
  - Mice are the usual intermediate host and a normal predator-prey relationship exists between the cats and mice that enhances transmission
- Causes toxoplasmosis

Morphology
- Oocysts are unsporulated in fresh feces
  - 12 x 10 μm (11 – 13 x 9 -11)
- Sporulated oocysts contain two sporocysts each with 4 sporozoites (2 x 4 architecture)
- In the environment, sporulation occurs in 1 – 5 days; under favorable conditions, sporocysts can survive about 18 mos.; can survive in fresh and salt water

Life cycle stages
- **Sporozoites**- form within oocysts
  - IH ingests oocysts, one means of infection
- **Tachyzoites** (“fast” merozoites)- form rapidly in tissues of intermediate host(s)
  - Tachyzoites form first in epithelial tissues of intestine
  - Disseminate to other tissues for further rapid development
  - Initial, acute infections
  - Within host cells, tachyzoites are contained within a parasitophorous vacuole
- **Bradyzoites** (“slow” merozoites)- develop slowly as immunity develops
  - immune cytokine production is thought to induce differentiation from tachyzoites to bradyzoites
  - form slowly in tissues of intermediate host(s)
  - Can remain viable for life of host, chronic infection (quiescent)
  - Found in large, cyst-like accumulations
  - Bradyzoites are infective upon ingestion

Pre-patent period (PPP) in cat
- Varies with stage ingested
  - 3-5 days when bradyzoites are ingested
  - 5-10 days when tachyzoites are ingested
  - 20-24 days when oocysts (sporozoites) are ingested

Epidemiology
- Seroprevalence of *T. gondii*
  - Serology is not useful in predicting shedding of oocysts by cats → oocysts shed prior to antibody formation
- Infection routes for cats
  - Carnivorism (primary)
  - Transplacental
  - Oocyst ingestion (lowest)
  - Cats can be both definitive and intermediate hosts
    - If intermediate host, usually see lung infections and pneumonia in cats

Human epidemiology
- Fecal-oral ingestion of oocysts (primary way humans are infected in US)
- Ingestion of tachyzoites and/or bradyzoites in undercooked meat and raw milk (goat’s milk esp., unpasteurized), congenital
- Organ transplant
• Blood transfusions (much less common)

Pathology & pathogenesis
• Pathology varies with strain of parasite, age of host, organs invaded, immune status of host, species of host
  o Enteritis
  o Hepatitis
  o Pneumonitis
  o Myocarditis
  o Chorioretinitis
  o Encephalitis
  o Placentitis
  o abortion

Clinical signs of congenital infection
• T. gondii naïve woman stands a 20-50% probability of passing infection to fetus if infected during pregnancy
• Earlier infection, more damaging to fetus

Diagnosis
• Intestinal (cat)
  o Oocysts in fresh feces (possible diarrhea)
  o Few cats shedding at any one time
• Serologic Dx
  o Positive serological result does not correlate with shedding in cats!
  o Can use serology for other mammals and birds→ performed at UTCVM
  o Acute v. past; significant increase in IgG titer in 2-3 week time span- look for rising titers

Control
• Keep cats indoors
• Discourage feral cat colonies and educate owners about Toxoplasma risks due to predation of intermediate hosts
• Keep cats away from livestock
• Keep cats away from sand boxes & public parks, and beaches
• Adequately cook meat
• Freeze meats before eating- freezing kills tissue cys

Neospora caninum
Hosts/Disease
• Causes neosporosis
  o CNS disease in dogs, cats, cattle, sheep, etc.
• In 1998, strain in cattle found to use dogs as DH; dogs can also be infected with tissues stages
• No human cases to date, not considered a human pathogen, not zoonotic
• Causes abortion in cattle, sheep, goats, etc.

Oocysts
• Identical to Toxoplasma
  o Remember only cats shed Toxoplasma oocysts in feces
  • 11 x 12 μm

Life cycle sequence
• Sporozoites (in dog feces, ingested by IH)
  • Tachyzoites (travel to various tissues via blood
  • Bradyzoites (develop in various IH tissues, cysts in brain only)
  » Cyst wall thicker than T. gondii
• DH (dog) eats IH with bradyzoites
  • Bradyzoites initiate asexual schizogony (tachyzoites), eventually a sexual
  • Dogs shed few oocysts, make it difficult to study, much to be learned yet about life cycle

Pathology/Pathogenesis of neosporosis
• CNS & systemic disease in dogs, cats, cattle, sheep, etc. (not humans)
• Can be fatal in dogs, esp. congenitally infected dogs

Clinical Signs (dogs)
• In transplacentally infected puppies hindlimb paresis & weakness are typical presentations
• In adult onset disease:
  o Nodular dermatitis
  o Pneumonia
  o Urine and fecal incontinence
  o Hepatitis
  o Myocarditis
  o Myositis

Clinical signs (cattle)
• Clinical signs seen in calves; only clinical sign in cows is abortion
• Major cause of abortion in U.S.,

Diagnosis
• Oocysts in feces of dogs (canids)
• Serology;
  o ELISA
  o IFA
  o Neospora agglutination test (NAT)
  o Also used on bovine sera
• PCR

Public health
• Not considered a human health concern
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- Can cause tick paralysis in humans, dogs, etc.
- May take as little as 3 months, with favorable conditions, or up to 2 years
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- Wide distribution
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- Can survive indoors for months to possibly years without a blood meal
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- Can transmit Coxiella burnetii (Q-fever), tularemia, Ehrlichia chaffeensis, Ehrlichia ewingii, RMSF,
- Vectors agent of Southern Tick Associated Rash Infection (STARI) in humans
- Cause of STARI is currently unknown—may actually be the host reaction to tick saliva—leads to swelling and pain at bite region in people.

Amblyomma maculatum (Gulf Coast Tick)
- Southeastern US in Gulf coast region, but has expanded range recently
- Ornate scutum – often confused with Dermacentor—examine mouth parts to differentiate
- Adults attack nearly all animals & humans and can transmit Hepatozoon americanum Hepatozoonosis—dog must eat tick to be infected with Hepatozoon

All Ixodes spp.
- Inornate ticks and No festoons, has distinct anal groove anterolateral to anal orifice
- Used for identification in NON-ENGORGED tick but can’t see groove in engorged ticks—use mouth parts instead

Ixodes scapularis (Black-Legged Tick)
- Wide distribution, in East, South, and Midwest U.S. Highest populations in upper Midwest and New England/midatlantic states
- Primary Lyme disease (Borrelia burgdorferi) vector in Eastern US and Midwest
- Vectors Babesia microti, Anaplasma phagocytophila

Ixodes pacificus (California Black-Legged Tick)
- Primary Lyme disease vector in the West Coast

Tick-borne diseases
Tick paralysis
Potentially fatal reaction to a paralyzing neuromuscular toxin secreted in the saliva of a female tick late in her feeding. Cattle, sheep, horses, dogs, and humans seem to be most affected.

Clinical signs include: headache, vomiting, general malaise, loss of motor function and reflexes, followed by paralysis that starts in the lower body and spreads to the rest of the body

Respiratory failure and death can result. Signs disappear rapidly when tick is removed, suggesting that the toxin is rapidly excreted or destroyed

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- Vector: Ixodes scapularis (Eastern and Midwestern US), Ixodes pacificus (Western US)
- Animal health: Major cause of canine and equine disease, including endocarditis and joint pain. Most cases occur in the spring and summer, during nymphal emergence, and in late fall and winter, during adult emergence.
- Human health: Acute and chronic diseases including joint pain, heart disease, and neurological disorders. Most cases occur in the spring and summer, during nymphal emergence, and in late fall and winter, during adult emergence.

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- Sometimes placed in “Spotted Fever” disease group
- Vector: Dermacentor variabilis
- Geographical distribution: Eastern US mainly. Most frequently reported tick borne disease in the eastern US. Other agents other than R. rickettsia can lead to spotted fever group disease in humans. Clinical signs include flu like symptoms as well as petechial hemorrhage.

Cytauxzoon felis
- Piroplasm of cats. Bobcats are reservoir host that is transmitted by Amblyomma americanum. Clinical signs: fever, dehydration, icterus, lymphadenomegaly, and hepatosplenomegaly. Treatment with atovaquone plus azithromycin.
- Diagnosis: PCR, blood smear (negative blood smear does not rule out infection) since early stage only see schizonts in macrophages. Prevention: Keep cats indoors!! Use preventative for tick infestation
Anaplasma phagocytophilum:
- Intracellular rickettsia that causes human granulocytic anaplasmosis
- Infects granulocytes and leads to bleeding, fever, leukopenia,
- Clinical signs/symptoms may be worse with co-infection with Lyme or Babesia
- Vectored by Ixodes scapularis so same geographical distribution as Lyme Disease. Can be transmitted by blood transfusion.
- Diagnosis: clinical signs, PCR (acute cases), serology (chronic), CBC to look for leukopenia, Blood smear to look for morulae in granulocytes.

Ehrlichia canis
- Intracellular rickettsia that causes canine ehrlichiosis
- Infects monocytes and leads to fever, anorexia, lethargy, thrombocytopenia, lymphadenopathy, edema, bone marrow suppression.
- The acute stage is mainly due to a vasculitis. *E. canis* replicates in monocytes. The infected monocytes bind to vascular endothelial cells and leads to a vasculitis
- Transmitted by Rhipicephalus sanguineus –worldwide distribution
- Diagnosis: clinical signs, PCR (acute cases), serology (chronic), CBC to look for leukopenia, Blood smear to look for morulae in monocytes
- Don’t treat animals that are clinically normal but are only seropositive—potential false positive due to positive predictive value.
- Treatment with doxycycline or minocycline
Wildlife Diseases Important for Practitioners to Know
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Wildlife diseases can be important for both wildlife populations as well as potentially important for livestock or domestic animal and public health. A subset of diseases will be discussed that practitioners should know

a. Chronic wasting disease (CWD) or cervids- important prion disease of deer family (cervids) and leads to transmissible spongiform encephalopathy. Only cervids can be naturally infected and incubation periods can be months to years depending on host genotype. Clinically signs include weight loss, salivation, ataxia, poor hair coat, etc. Diagnosis can be performed by ELISA or immunohistochemistry. Current models suggest that CWD can lead to significant focal population decreases in various cervid species and every effort should be made to minimize the chance of the disease being introduced into areas where it does not exist.

b. Tularemia- bacterial disease of rabbits, beavers, and various rodents. The disease can progress quickly and lead to death. Lesions consists of pinpoint areas of necrosis in the liver and abscesses of the lymph nodes. The bacteria can infect humans and can be quickly fatal. Transmission can occur by aerosolization, ingestion, and vector borne.

c. Plague- Similar to Tularemia but seen in western US in ground squirrels and similar species. Cat, dogs, and humans can be infected by direct contact or via aerosolization, ingestion, and vector borne.

d. Hemorrhagic disease of cervids. Caused by Epizootic hemorrhage disease (EHD) and bluetongue (BT) viruses. Multiple serotypes of both viruses exist. The virus is transmitted by Culicoides midges. Clinical signs include edema, hemorrhage, conjunctivitis, lethargy and death. Lesions can include erosions of oral cavity and rumen. Can lead to large focal population declines in certain regions of US where disease does not occur regularly. Disease is seen in later summer. Evidence exists that cattle can be infected with the EHD virus and lead to blisters and vesicles. Sheep are susceptible to BT virus. No evidence of human infection

e. West Nile virus- Virus found in birds (particularly corvids –crows and blue jays). Can lead to sudden death or chronic disease depending on the bird species and likely other factors. Virus is transmitted from mosquitoes so care to reduce mosquito breeding areas is important in controlling the disease. WNV is zoonotic and can lead significant morbidity and mortality in humans. Dead bird surveillance is often important in determining geographical hot spots of the virus.

f. *Baylisascaris procyonis* - roundworm of raccoons but dogs can also act as definite host and shed eggs in feces. Aberrant hosts that ingest larvated eggs can have visceral and neural larval migrans and can lead to neurological disease and death. The parasite has been a major impact in restorations of Allegheny wood rats. Chickens, quail, mice, rodents and other animals can have disease. Furthermore humans can be infected and several humans mortalities have occurred due to the parasite. Efforts should be made to make areas around houses and livestock to be unattractive to raccoons. These efforts include not leaving pet food outside, keeping compost piles away from house and have lid on compost pile, keeping houses and attics closed off from wildlife and educating clients not to feed wildlife and not to keep wildlife as pets.

*Echinococcus* spp.– Tapeworm parasitic disease of ruminates and carnivores or of rodents and carnivores. Eggs are shed by carnivores and are zoonotic if eggs are ingested. Eggs are identical to Taenia tapeworms of dogs. Lesions in ruminants consist of fluid filled sacs in the lungs and liver. The geographical distribution of the parasite may be expanding.

h. Avian influenza- viral disease of birds. Waterfowl are the natural host for low path avian influenza viruses. Viruses may mutate to high path and cause infection in poultry leading to significant mortality and morbidity. There is potential for zoonotic infection of flu in humans and there is continued concern about avian influenza virus mutating to become an epidemic level disease of humans.

i. Feeder diseases of wild birds: includes salmonella, avian pox, aspergillosis, and trichomonosis. Lesions can consist of caseous material in the oral cavity and confirmatory testing is needed to determine exact cause. Feeders and waterers should be cleaned every two weeks with 10% bleach solution to minimize disease transmission.