To ensure the health and well-being of pet dogs and cats, examination of feces for parasite eggs, oocysts, and cysts is an important part of the daily routine for most veterinary practices. Many different procedures and techniques are used, each with its own advantages and limitations. Direct fecal smears are useful for detecting motile protozoa, and sedimentation examinations are useful for recovering heavy (e.g., Physaloptera spp) or operculated (e.g., fluke) eggs that do not float well because of the hypertonic effects exerted by the flotation solution. The methods most frequently used to recover parasite eggs, oocysts, and cysts are flotation techniques that rely on the differences in the specific gravity (SG) of the egg(s), fecal debris, and flotation solution.

The SG of most parasite eggs is between 1.05 and 1.23. For parasitic eggs to float, the SG of the flotation solution must be greater than that of the eggs. Ideally, all helminth eggs and protozoan cysts and oocysts would float and still maintain their morphologic integrity while fecal debris would sink in the chosen flotation solution. Flotation solutions are made by adding a measured amount of salt or sugar to a specific amount of water to produce a solution with the desired SG. Common flotation solutions include saturated sodium chloride (NaCl; SG 1.18), sugar (Sheather’s solution; SG 1.27 to 1.33), sodium nitrate (NaNO₃; SG 1.18 to 1.2), magnesium sulfate (MgSO₄; SG 1.2), and zinc sulfate (ZnSO₄; SG 1.2). These solutions are effective, easy to make or commercially available, and relatively inexpensive.

Flotation procedures vary from the simple to the complex. The simplest procedure involves mixing a small amount of feces with flotation solution in a cylinder (shell vial or centrifuge tube) and adding solution until the cylinder is nearly full. The preparation is then allowed to stand until the eggs float to the top, and a sample from the top is removed to a microscope slide using a tool such as a wire loop, straw, needle hub, or glass rod. A refinement of this method involves filling the cylinder until a slight positive meniscus is formed and placing a glass coverslip over it. Again, the cylinder is allowed to stand until the eggs have had time to float to the top, and the coverslip is then removed to a microscope slide and examined. Several commercial apparatuses that use a screen to retain debris from floating to the top are variations of the simple shell vial technique.

A further refinement of the flotation technique involves centrifugation to spin down the debris and allow the eggs to float to the surface of the solution where they can be recovered. If a fixed-angle centrifuge head is used, the centrifuge tubes cannot be filled completely and thus should be removed from the centrifuge after spinning and placed vertically in a test tube rack. If a swing-head centrifuge is used, the tubes can be filled to a slight positive meniscus and covered with 18- or 22-mm² coverslips before centrifuging. When tubes are spun with coverslips in place, care should be taken not to open the centrifuge before it stops spinning, or the coverslips can shift and ruin the preparation. Veterinary hospitals usually use one or more of these methods based on cost, ease of use, availability of hardware, or simply tradition.

The Ovassay method with 1.1-SG ZnSO₄ solution readily floats, hookworm (A. caninum) eggs (SG 1.0559); however, ascarid (T. canis) eggs (SG 1.0900) may not be recovered and whipworm (T. vulpis) eggs (SG 1.1453) are virtually impossible to float with such a solution. This points out the necessity for using care in weighing the salts and measuring water when preparing flotation solutions and for assuring proper SG by testing the solution with an SG hydrometer. When the SG of the salt solution (ZnSO₄) is raised to 1.2, T. vulpis, and T. canis eggs are recovered in the Ovassay but in fewer numbers than with a centrifugation method using either ZnSO₄ or sugar. A centrifugation method will recover significantly higher fecal counts compared with the Ovassay method.

For A. caninum, a centrifugation method using 1.2-SG NaNO₃ solution results in significantly higher fecal egg counts than the simple flotation method, which is allowed to stand for 5 or 10 minutes. A 15- or 20-minute simple flotation method recovers significantly similar fecal counts as compared with the centrifugation method. With low numbers of T. vulpis eggs the 5‘ and 10’ simple floats can miss eggs in 2 out of 3 samples.

Over the past decade a number of studies have been conducted to evaluate and compare the performance of various fecal diagnostic techniques. From 2000 to 2004, students at KSU evaluated 206 fecal samples known to contain hookworm (A. caninum) eggs. When hookworm data were combined, the direct smear technique failed to detect hookworm eggs 72.82% of the time. The Ovassay and centrifugation techniques yielded false-negative results 4.85% and 0.97% of the time, respectively, and recovered more than 50 eggs/slide 36.41% and 74.76% of the time, respectively. Studies evaluated 171 fecal samples known to contain ascarid (T. canis or T. catti) eggs. When all ascarid data were combined, the direct smear technique failed to detect ascarid eggs 85.38% of the time. The Ovassay and centrifugation techniques yielded false-negative results 25.88% and 10.53% of the time, respectively, and recovered more than 50 eggs/slide 1.18% and 42.69% of the time, respectively.

Students evaluated 203 fecal samples known to contain whipworm (T. vulpis) eggs. When all whipworm data was combined, the direct smear technique failed to detect whipworm eggs 92.61% of the time. The Ovassay and centrifugation techniques yielded false-negative results 32.02% and 4.93% of the time, respectively, and recovered more than 50 eggs/slide 2.96% and 23.65% of the time, respectively.

Students also evaluated 53 fecal samples known to contain tapeworm (Taenia sp) oocysts and 26 samples known to contain coccidia (Isospora sp) oocysts. The direct smear technique failed to detect tapeworm eggs 96.15% of the time. The Ovassay and centrifugation techniques yielded false-negative results 76.92% and 11.54% of the time, respectively. When the two sets of coccidia data were combined, the direct smear technique failed to detect coccidia oocysts 94.34% of the time. The Ovassay and centrifugation techniques yielded false-negative results 50.94% and 5.66% of the time, respectively.
Evaluations of centrifugation fecal techniques and IDEXX SNAP® Giardia fecal antigen test kits of puppy fecal samples by 2nd year veterinary students showed that almost half (56/116) of the fecal samples were recorded as positive for Giardia. The direct smear technique detected the fewest number of positives with students recording only 4 positive samples. This data may be artificially low since the fecals were collected several hours prior to laboratory and trophozoites may have been dead at time of examination. Students recorded that the SNAP® Giardia fecal antigen test identified 55 of 116 samples as Giardia positive and ZnSO₄ centrifugation technique recorded 45 of 116 samples as positive.

At a wet lab conducted at the Central Veterinary Conference in 2005 twenty-seven (27) participants returned completed fecal data forms. When a centrifugation fecal flotation technique was compared to passive flotation technique the data demonstrated that centrifugation with either 1.18 sp. gr. ZN₄SO₄ or 1.27 sp. gr. Sheather’s sugar solution routinely recovers more eggs and oocysts than the passive Ovassay technique. Not only did the centrifugation technique recover more eggs and oocysts in addition the participants recorded many more samples as positive with the centrifugation technique. Strikingly only once (T. canis – Ovassay - ZN₄SO₄) did the Ovassay technique recover all parasites in all samples, while only once did the centrifugation technique fail to recover all parasites in all samples. In the group that used 1.18 sp. gr. ZN₄SO₄ solution only 2 of 14 participants recovered Taenia sp. eggs. While in the group using 1.27 sp. gr. Sheather’s sugar solution all 13 participants recovered Taenia sp. eggs using.

Even though the participants knew the samples were positive for Giardia recovery and identification of Giardia sp. oocysts was problematic for the 27 participants regardless of technique. Only 6 of the 27 participants were able to recover and identify Giardia sp. oocysts from a known positive sample. One participant each using the Centrifugation with ZNSO₄, Ovassay with ZNSO₄ and Ovassay with Sugar was able to recover and identify Giardia sp. cysts. Three participants using the Centrifugation with Sugar were able to recover and identify Giardia sp. cysts. All 27 participants had a positive SNAP® Giardia fecal antigen test on the mixed sample.

As part of a weeklong clinical Parasitology training program, veterinarians participated in a wet-lab evaluating fecal examination techniques. Three classes were offered during 2010, 2011 and 2012, for a total of 9 classes that included 56 participants. Fecal samples were collected from dogs at the local animal shelter, verified as positive for various parasite diagnostic stages and mixed to form composite samples. While species of parasites in fecal samples varied, all 9 classes evaluated samples that contained A. caninum, T. canis and T. vulpis eggs. Each participant conducted a direct smear, an Ovassay using a 1.18 sp. gr. ZNSO₄ solution, a centrifugation procedure using 1.18 sp. gr. ZNSO₄ solution and a centrifugation procedure using 1.24 sp. gr. sugar solution. Using the direct smear technique, participants recovered T. canis, T. vulpis and A. caninum eggs 30.4% (17/56), 26.8% (15/56) and 30.4% (17/56) of the time, respectively. The Ovassay recovered T. canis, T. vulpis and A. caninum eggs 57.1% (32/56), 41.1% (23/56) and 87.5% (49/56) of the time, respectively. The centrifugation procedure with ZnSO₄ recovered T. canis, T. vulpis and A. caninum eggs 94.6% (53/56), 85.7% (48/56) and 100% (56/56) of the time, respectively. The centrifugation procedure with ZnSO₄ recovered T. canis, T. vulpis and A. caninum eggs 94.6% (53/56), 85.7% (48/56) and 100% (56/56) of the time, respectively. When the Ovassay technique was used, only 33.3%, 11.1% and 44.4% of the time did every participant recover T. canis, T. vulpis and A. caninum eggs, respectively. When participants used the centrifugation procedure with sugar solution, every participant in every class recovered eggs of T. vulpis and A. caninum and 77.8% of the time every participant recovered eggs of T. canis.

Addition of Fecal Antigen Testing to your Standard Diagnostic Procedures. New methodology for detecting protein biomarkers secreted or excreted by nematodes in the intestinal lumen. Unique biomarkers now available for ascarid, hookworm, and whipworm. These biomarkers are produced by the worms and not the eggs. Beneficial because they can overcome issues with misidentification, spurious eggs from coprophagy and even prepatent periods.

REFERENCES

While often the same products are used to combat ticks as are used to combat fleas, there are substantial differences between flea and tick control. One of the major differences is in the number of species that confront a dog. While there is one predominant flea species that infests dogs in North America, the Cat flea (*Ctenocephalides felis*), there are at least 10 different tick species that may be encountered. There can be remarkable regional variability in the number and diversity of tick species that infest dogs. While practitioners in Hawaii may only deal with one tick species infesting dogs (Brown Dog tick, *Rhipicephalus sanguineus*), practitioners in New Mexico may encounter three different species, in California six different species and in Kansas up to seven different tick species. This wide diversity in tick species means that ticks occur at different times of the year, are associated with different reservoir hosts and carry and transmit different diseases.

Over the past few decades there has been a change in the distribution and abundance of certain tick species in North America. Two of the best documented are *Amblyomma americanum* and *Ixodes scapularis*. Since both these ticks are important vectors of human and animal pathogens these changes in distribution and abundance have had a marked effect upon both human and animal health. Various factors have contributed to tick population movement including; changes in agricultural practices, reforestation, wildlife conservation, relocation and restocking, climate fluctuations and decreased environmental pesticide application.

Specific factors that have contributed to the increased range of *A. americanum* include increased habitat via reforestation and its wide host range that includes deer, small mammals, birds and man. The White-Tailed Deer is considered a preferred host for *A. americanum*, and all life stages will feed successfully upon White-Tailed Deer. Another species that utilizes similar habitats and is an excellent host for larvae and nymphs is the wild turkey. Areas with high White-Tailed Deer and wild turkey populations can have remarkably large populations of *A. americanum*. Similar to *A. americanum* the distribution of *I. scapularis* is linked to the distribution and abundance of the white-tailed deer.

*Ixodes scapularis* is widely distributed in the Eastern and Central U.S. in at least 35 state. Its distribution is from Florida to Maine, west into far eastern South Dakota, and south through eastern Kansas into central Texas. *Ixodes scapularis* is also located in central and eastern Canada.

Seasonal activity varies by geographic region, but larval activity is generally highest in August and September. Larvae attach to and feed on a wide variety of small mammals, including mice, chipmunks and shrews. Larvae also feed on birds and lizards. The white-footed mouse (*Peromyscus leucopus*) is of particular importance in the tick life cycle and disease transmission, because it serves as a good host for larval *I. scapularis* and it is a major reservoir of *Borrelia burgdorferi*.

Immature ticks typically engorge for 2 to 4 days before dropping off to molt in moist protected areas such as under leaf litter in forested habitats. Larvae over-winter and then molt to nymphs in the spring. Nymphs will feed for 3 – 4 days on a variety of hosts including mice, squirrels, chipmunks, raccoons, opossums, skunks, shrews, cats, birds, and humans. Nymphs occur primarily from May through July in the North. Adults occur most commonly from October through December. Adults that do not find a host will quest again, typically from March to May. Adults feed for 5 – 7 days, primarily on white-tailed deer, but also on bobcats, cattle, coyotes, dogs, foxes, horses, humans, opossums, raccoons and other mammal.

While recent pharmaceutical advances have been made in control of flea reproduction, such advances in the area of tick control are lacking. With the exception of the brown dog tick *Rhipicephalus sanguineus*, our ability to manage tick reproduction is limited, if not almost non-existent. As discussed previously in most flea infestations we have the opportunity to control flea reproduction by either killing fleas before they can reproduce or killing flea eggs. However, it is not just because we have effective residual insecticides, insect growth regulators or insect development inhibitors that we are successful, it is also due in large part to the fact we can often target the primary reproductive host, the flea infested dog or cat. And interestingly, failures in flea control often occur when flea infested feral pets or flea infested urban wildlife invade the owners yards.

But when dealing with most 3-host ticks the problem is that the majority of the reproducing ticks are not on the dogs or cats, but on their nature wildlife hosts. Since we are limited in our ability to manage ticks on wildlife, reinfestations are a common occurrence and protracted use of acaracides as preventives is routine in many areas.

Since tick control can be extremely difficult and because they are vectors of a variety of bacterial and protozoal diseases, veterinarians should have an understanding of the ecology of the tick(s) encountered in the area in which they practice. Veterinarians need to be educated on the various aspects of tick ecology, disease transmission and control methodologies so that they can then educate their staff and pet owners.

Numerous studies demonstrate the high level of efficacy of the various acaracides but the residual activity is rarely 100% and the efficacy of products varies between and as well as within species, even in the same laboraotry. Evaluations of acaracides under natural or field conditions further illustrates that while efficacy is good it is not 100%.
In a field efficacy trial conducted in Kansas U.S.A, an imidacloprid (8.8% w/w)-permethrin (44.0% w/w) formulation was evaluated on dogs against naturally occurring populations of *Amblyomma americanum*. When dogs were walked in a naturally tick infested environment the 48-hour post-exposure efficacy of imidacloprid-permethrin formulation was 93.5%, 98.9%, 94.6%, 94.1% and 96.6% on days 3, 7, 14, 21 and 28 respectively, post-treatment.\(^14\)

Variation in product efficacy occurs. In two studies conducted at K-State, different results were found when evaluating the efficacy of acaricides against *Dermacentor variabilis* infestations in dogs from two different regions of the USA.\(^9\)-\(^12\) In the first study, the efficacy of imidacloprid–permethrin and fipronil–(s)-methoprene formulations were evaluated against a *D. variabilis* isolate from California. The 48-h post-infestation efficacy on day 30 post-treatment was 92.0% and 83.2%, respectively, for the imidacloprid–permethrin and fipronil–(s)-methoprene formulations. In the second study, the 48-h post-infestation efficacy on day 30 for the imidacloprid–permethrin and fipronil–(s)-methoprene formulations against a *D. variabilis* isolate from Oklahoma was 17.5% and 75.7% respectively.

One combination spot-on product that produces more prolonged and pronounced efficacy is fipronil-amitraz. In a study conducted at K-State, the efficacy against *Dermacentor variabilis* 30 days after treatment was 99.4%.\(^13\)

Recently a new class of insecticide/acaricide has provided the first orally administered approach to tick control. Afoxolaner, fluralaner and sarolaner are members of the isoxazoline class and work by inhibiting insect GABA and Glutamate-gated chloride channels leading to hyper-excitation and death of insects and arachnids.\(^17\)-\(^19\)

While product efficacy is often excellent in most studies, significant variation in efficacy can occur and 100% control is rarely achieved. Therefore, it can be expected that under natural conditions in areas where dogs are being frequently exposed to ticks pet owners will see ticks on treated dogs. We might also expect that efficacy in real world situations might be lower due to such factors as bathing and swimming, differences between dog breeds and haircoat types and frequency and correctness of product application.

Since 100% tick kill is rarely achievable, perceived efficacy of acaricides may be directly related to the numbers of ticks to which dogs are exposed. If a dog is treated with one of these highly efficacious acaricides and encounters just a few ticks it is likely all those ticks will be killed. However, if tick exposure is considerably larger, we can expect a few ticks to be observed on these dogs and pet owners may perceive a lack of efficacy. Therefore, in areas where tick populations are increasing the perception may be that the products are not as effective as they once were.

Pet owners often view tick infestations of their pets differently than flea infestations.\(^12\) Whether this is due to concerns about tick transmitted diseases or simply a phobia, the presence of a couple of ticks on the pet often elicits a more pronounced negative reaction than the presence of a couple of fleas. A 95% effective flea product may provide great client satisfaction while a similarly effective tick product may be perceived as a failure. Therefore, it is not uncommon that label recommended application of a product does not appear to control the problem. This may be real or perceived, based upon pet owner expectations of product performance. Given pet owner concerns, a need to reduce tick borne disease and lack of 100% efficacy; occasionally additional control measures are needed.\(^12\),\(^14\) If additional control measures are deemed necessary, pet owners need to be educated as to why additional control measures are necessary and notations made in the pets record before extra label uses are conducted.

One of the most common practical attempted solutions to this problem in dogs is to increase the frequency of application. Here increased residual efficacy is the expected outcome, since you are increasing the residual acaricides levels with the shorter application intervals. Additionally, with many 3-host ticks destruction of tick habitat can reduce exposure pressure. Areas that serve as refuge for ticks and wild mammals such as grass, weeds, and brush piles, between runs and along buildings, can be eliminated or treated with an approved acaricide.

In some situations, especially in tropical and subtropical regions and in climate controlled kennels brown dog ticks may infest buildings with ticks crawling up walls, curtains and throughout the home or kennel.\(^14\)-\(^15\) In these situations acaricides may need to be sprayed indoors into cracks and crevices, behind and under furniture or cages and along walls and the ceiling. Following application, make sure the acaricide is dry before you allow animals or humans back into the premises to minimize toxicity problems. Finally, restricting pet access from tick-infested environments may be necessary.

It is apparent that the range and local density of certain tick species has increased in many areas. Whatever the factors it must be recognized that tick infestation pressure may be much higher and associated tick transmitted diseases may be more prevalent in some locations today than in the past. The increase in tick populations means that pets are encountering ticks more frequently, are exposed to more ticks per encounter and clients may be seeing more ticks on their pets than in the past. Since tick products do not kill or repel all ticks instantly, clients may get the false impression that the products are not performing as well as in the past. These situations necessitate that veterinarians set client expectations, before clients set their own unrealistic expectations of control.

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Wolbachia and Heartworm
Michael Dryden, DVM, MS, PhD
Kansas State University
Manhattan, KS

To understand the relationship of Wolbachia to *Dirofilaria immitis*, we must have a detailed understanding of the life history and pathogenesis of canine heartworm.

Adult *Dirofilaria immitis* naturally in pulmonary arteries and occasionally the right heart; occasionally aberrant migrations to other locations in the body. Showing adult worms in right ventricle of a dog’s heart to a client may be an effective tool but it is typically a post-mortem finding. Female *D. immitis* in pulmonary arteries and occasionally the right heart deposit microfilariae into circulation. Microfilariae may survive up 3.5 years in the vascular system. Mosquitoes (> 70 species worldwide; approximately 25 North America) I.H. become infected when they feed on an infected dog and consumes blood containing the microfilariae (L₁). Microfilariae then develop first in mid-gut & then in malpighian tubules of the mosquito from the L₁ – L₂ – L₃ infective stage within 13 to 30 days. L₃ infective larvae then migrate to the salivary glands of the mosquito. Infected mosquito bites dog and deposits infective L₁ larvae in or around the bite wound. L₁ reside in subcutaneous tissues and molt to the L₂ (1.5mm) in subcutaneous tissues within 3 – 12 days. L₂ reside in subcutaneous tissues or muscle of abdomen or thorax and molt to the adult-juvenile adults 1.2- 1.5cm) within 45 – 70 days (Kotani & Powers, 1982). Immature adults (2 – 4cm long) migrate to pulmonary arteries and heart by 70 – 90 days P.I. Worms mature (12 – 30cm) and then male and female *D. immitis* mate and females begin depositing microfilariae (L₁) within 6 months (rarely) but more commonly 7 – 9 months P.I. In dogs adult *D. immitis* may live 5 – 7 years

Pathogenesis – caused by adults is initially primarily a lung disease. Progressive pulmonary hypertension associated with pathological changes in the pulmonary arteries. Vascular endothelium damaged by repeated embolisms of defunct adults and inflammation associated with antigens released from worms. Platelets and WBC adhere to damaged vascular endothelium and release doxycycline and ivermectin, arterial lesions almost completely absent (Kramer et al. 2011).

Most filarial nematodes, including all *D. immitis*, harbor symbiotic, intracellular, gram-negative bacteria *Wolbachia pipientis* (Rickettsiales). These bacteria are symbionts that may be necessary for functional reproduction in female *D. immitis* and development in immature stages. *Wolbachia sp.* proteins have been implicated in the pathogenesis of heartworm disease in dogs and cats. *Wolbachia pipientis a* symbiotic, gram negative intracellular bacteria that is closely related to rickettsia. Identified in human and animal filarial nematodes. Heartworm endosymbiont - All *D. immitis* parasites harbor Wolbachia. Wolbachia organisms are maternally transferred from one filarial generation to the next. Bacteria are present in all life stages of the parasite. Wolbachia are released in large numbers at death of parasite and during production and release of microfilariae. Generally, filariae free of Wolbachia after treatment with tetracyclines show inhibition of maturation, survival and reproduction. It has been shown that a combination of doxycycline/ivermectin is microfilaricidal and adulticidal. Weekly doses ivermectin (6mcg/kg) + doxycycline 10mg/kg/day from weeks 0-6, 10-12, 16-18, 22-26, 28-34. No microfilaraemia after week 12. Antigen levels decreased in some dogs by week 24. Ivermectin/doxycycline 78.3% adulticidal effect. Much higher than ivermectin or doxycycline alone (Bazzocchi et al. 2008). In a separate study Italian study – dogs naturally infected with heartworm were administered doxycycline (10mg/kg SID x 30 days) and ivermectin (6mcg/kg every 15 days for 180 days). 100% microfilaria negative by day 90 & 72.7% antigen-negative by day 300 (Grandi et al. 2010). In yet another study Doxycycline administered at 10mg/kg BID for 30-day periods to determine efficacy against larval and immature stages. Group 1, day 0-29; Group 2, day 40-69; Group 3, day 65-94; & Group 4, untreated controls. Dogs in group one 0 no microfilariae and no adult heartworms. Group 2 & 3 98.4% and 69.6% reduction in total worm burden, respectively (McCull 2011). L₁ from mosquitoes fed on dogs treated with doxycycline. Normal appearance and motility, but unable to develop in dogs. Prevented further transmission of disease (McCall 2008). Effect of pretreating with doxycycline before melarsomine. Dogs experimentally infected with adult HW. Group 1 doxy 20mg/kg SID x 30 days, melarsomine at week 12, followed 1 month later by 2 injections 24 hrs apart. Group 2 doxy x 30 days, melarsomine as above, ivermectin 6mcg/kg monthly for 24 wks post infection. Group 3 melarsomine alone. NO pulmonary thromboembolism in any dogs treated with doxycycline. In dogs treated with doxycycline and ivermectin, arterial lesions almost completely absent (Kramer et al. 2011).


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 (snail/slug 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
  - σ 11 - 14 mm
  - μ 19 - 23 mm
- L1 250-300 μm

Aelurostrongylus – feline lungworm
- I.H. - snails and slug
- Paratenic Hosts- amphibians, reptiles, birds, rodents
- D.H. - cats (felidae)
•  ♂ 4 - 6 mm  ♀ 9 -10 mm
•  L1  350-400 µm
•  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
•  ♂ ,♀ 14 to 20 mm  (♀ barber pole)
•  L1  310-400 µm - anterior cephalic button with a dorsal spine
•  PPP- 7 weeks

Capillarids
•  Eggs with polar plugs Size 50 - 80 x 20 - 40 µm - 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 μm 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 intralobar 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 immitticide 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
  - Enteritis
  - Hepatitis
  - Pneumonitis
  - Myocarditis
  - Chorioretinitis
  - Encephalitis
  - Placentitis
  - 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)
  - Oocysts in fresh feces (possible diarrhea)
  - Few cats shedding at any one time
- Serologic Dx
  - Positive serological result does not correlate with shedding in cats!
  - Can use serology for other mammals and birds → performed at UTCVM
  - 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
  - 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
  - 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
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 nymphaal 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

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
- Remains Resembles Rhipicephalus (both have 1 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, Cyttauxzoon fells

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), *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

**Lyme Borreliosis**
- Agent: *Borrelia burgdorferi*
- 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.

**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
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- Diagnosis: clinical signs, PCR (acute cases), serology (chronic), CBC to look for leukopenia.  Blood smear to look for morulae in granulocytes.
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 migrants 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

g. 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.