

Camelid parasitology

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Introduction

Like any other animal, camelids are faced with exposure to internal and external parasites. These can be relatively benign or cause severe disease. Where each camelid ends up on this spectrum of severity depends on a number of factors, including type of parasite, number of parasites, and host immunity. Exposure to parasites is a fact of life. Except in very rare cases, we cannot eliminate parasites. Therefore, our primary jobs are to promote immunity and prevent large-scale exposure. We attempt to manage parasitism to reduce or eliminate health effects.

Gastrointestinal tract

Protozoa

New World camelids appear to be susceptible to five or more species of *Eimeria*. These include small coccidia (*E. punoensis*, *E. alpaca*, and *E. lamae*), and large coccidia (*E. macusaniensis* and *E. ivitaensis*). These appear to be specific to New World camelids, but transferable among those species. The prepatent period for small *Eimeria* in camelids appears to be about 15 to 21 days. Over that time, there are two rounds of schizontogeny (asexual reproduction), the first in the lower small intestine and the second in the large intestine. Gametogeny (sexual reproduction) then occurs primarily in the large intestine with destruction of the gut epithelial cells. Clinical signs usually appear with gametogeny, though diarrhea may precede fecal oocyst shedding by a few days. Because of its predilection for large intestinal damage, *Eimeria* are one of the diseases more commonly associated with diarrhea in camelids.

Small coccidia also follow the general pattern described for other herbivores, in that older camelids are generally resistant to disease. Therefore, higher egg counts should be expected in camelids less than one year of age, and up to a few hundred eggs per gram can be considered normal. Counts much higher than 200 eggs per gram are unusual in adults, unless the camelid is otherwise debilitated or in an overcrowded environment.

E. macusaniensis is the largest and slowest maturing of the coccidia. The prepatent period is over one month, and it is common for camelids to show clinical signs of disease (colic, weight loss or poor growth, possibly diarrhea) before they develop patent infections. Diarrhea is less common than other clinical signs, especially in camelids over one year of age. It affects camelids of all ages more so than small coccidia, which infect but rarely cause clinical disease in camelids older than one year. In crias in contaminated environments, prepatent *E. macusaniensis* may be responsible for signs mistakenly attributed to the faster developing small coccidia, because the latter are found in the feces. Large coccidia also appear to be important in creating an environment conducive to *Cl. perfringens*-related disease.

Eimeria ivitaensis is a more recently recognized large coccidial parasite. While not as common or apparently as damaging as *E. macusaniensis*, it has been associated with severe disease in both young and old camelids.

Cryptosporidium is much smaller coccidian parasite, which can infect many species, including man. In addition to the intestinal epithelium, *Cryptosporidium* has been found in the biliary, renal, and respiratory epithelium of some immunocompromised patients of other species. Both *C. parvum* and *C. ubiquitum* have been isolated from camelids. Recently, *Cryptosporidium* has been implicated in several herd outbreaks of diarrhea in crias around the U.S. Adult camelids are less commonly clinically affected, but were found to be positive on fecal examination, and hence a possible source of infections.

Cryptosporidium usually affects younger crias < 21 days old, but has also been found in older crias with diarrhea, and in adults during outbreaks. Larger herds with a large number of births close together are most likely to have outbreaks of cryptosporidiosis.

Giardia

Giardia is a flagellate parasite. *Giardia* cysts can be found in the feces of many normal animals, including camelids, thus causing many to doubt its pathogenicity. It appears to be able to affect many different hosts, and introduction into a camelid herd is usually thought to occur through infected wildlife. Contaminated water courses are considered the usual route, though in moist areas, it is likely that the organism can persist on damp pasture.

Ingested *Giardia* cysts release trophozoites, which attach to small intestinal mucosal cells. Although cell function is impaired, *Giardia* do not reproduce in the cells like *Eimeria*. Number of ingested cysts and hence number of affected host cells is likely to be a predictor of the clinical importance of *Giardia* in a specific situation. The incubation period is approximately five days, but has lasted as long as 21 days in humans. With longer incubation periods, the disease can be seen seven to ten days before patency. The more cysts found in the feces, the more likely that *Giardia* is the cause of diarrhea. *Giardia* is primarily a problem in crias up to about seven months of age. Due to its predilection for the small intestine, diarrhea is not always found in camelids with severe giardiasis.

Balantidium coli is an amoeba. This has been described in camels and anecdotally in camelids. In the Middle East, camels are postulated to be the reservoir population for human infections. In alpacas, *Balantidium* is thought to cause diarrhea without weight loss in adults, and watery diarrhea in crias.

Identification

Coccidia may be identified by fecal flotation and microscopic examination. The average length and width increase from *E. punoensis* (around 20 x 6 μ), *E. alpaca* (24 x 20 μ), *E. lamae* (36 x 25 μ), to *E. ivitaensis* (88 x 52 μ), and *E. macusaniensis* (94 x 67 μ). Small coccidia float well in most solutions, and appear to float better in saturated saline than Sheather's solution, and are decreased by centrifugation procedures. Thus, a simple suspension or flotation appears best. Large coccidia require a denser flotation solution, such as Sheather's solution, and because low numbers may be important at the beginning of the patent period, concentration techniques such as centrifugation-flotation procedures are often helpful.

On dead animals, impression smears or histopathology may be helpful. It is important to recognize that the gut may appear grossly normal even with severe coccidiosis, or may have white mucosal punctae, hemorrhagic or fibrinonecrotic areas, or rarely edema and cobblestoning of the serosal surface. The distal jejunum and ileum are the areas of highest parasite concentration.

In live animals, diagnosis of prepatent disease is difficult or presumptive. Laboratory abnormalities are likewise non-specific, with evidence of shock and fat mobilization more common than electrolyte loss. Hypoproteinemia is common, but is also ubiquitous with many other camelid disorders. Anemia is rarer. Abdominocentesis usually yields a transudate. Abdominal imaging is also inconclusive: colicky camelids may have ileus and fluid-distended intestine, though usually to a lesser degree than camelids with gastrointestinal obstruction. Thickened bowel walls are rare (<10% of cases). Polymerase chain reaction may be helpful in diagnosing prepatent *E. macusaniensis* infection.

Cryptosporidium and *Giardia* may be seen on routine parasitologic mounts, but use of immunofluorescence for either or acid-fast staining for *Cryptosporidium* improves visibility. Some immunofluorescence kits have a tendency to yield false positives, so performance should be investigated before the kit is put into regular diagnostic use. *Giardia* or *Balantidium* trophozoites may be seen on wet mount of very fresh feces, and cysts can be found using centrifugation in a tube filled with 1.18 SG zinc sulfate solution. Other flotation solutions tend to distort cysts.

Treatment and control

If the camelid has either a confirmed or suspected infestation, treatment may be warranted. The most commonly used anticoccidial medications for small ruminants and camelids in North America are amprolium and sulfa antibiotics. Both are more effective against the immature forms of the parasite, and should not be expected to immediately reduce fecal shedding. Oral sulfa antibiotics are unlikely to be absorbed or effective after the juvenile period. Decoquinatone may be added to feed to reduce coccidia.

Treatment during the prepatent period does appear to reduce subsequent shedding. In countries where they are available, benzeacetonitrile (-azuril) compounds may be an effective alternative, especially as they kill more stages of the organism, and more rapidly reduce shedding. Many affected camelids also require plasma transfusions and antibiotic coverage.

In cases of *Cryptosporidium* infection, there is anecdotal information about some agents, but in general, anticoccidials are not known to have a beneficial effect. *Giardia* may respond to high doses of metronidazole or fenbendazole. Metronidazole may also be effective against *Balantidium*.

Herd control is a tricky issue. Disease in younger animals should be treated similarly to other forms of parasitism. Some degree of shedding may be normal or clinically insignificant prior to the development of immunity. In older animals, the amount of shedding should be lower, but complete lack cannot be expected. Signs of disease, protein loss, diarrhea, or weakness support treatment in herds with known or suspected parasitic issues support the argument for treatment. Other management changes to reduce stress and overcrowding, and clean up the environment are necessary. Herd outbreaks are not common, emphasizing the role of immunocompromise in severe infestations, but have been seen when groups of naïve camelids have been brought to farms with endemic problems. Spot fecals may be helpful of assessing the level of herd shedding, but remember that prepatent disease is common and intermittent shedding may occur. For herd treatments, less aggressive medical treatment is necessary, as the goal is to reduce, but not eliminate the parasite.

Strongyles

Camelids are susceptible to infection by many of the same nematodes that affect domestic ruminants. The major stomach (C3) worms include *Haemonchus*, *Ostertagia*, *Teladorsagia*, *Trichostrongylus*, *Camelostrongylus*, and *Marshallagia*. The major small intestinal worms include *Cooperia*, *Nematodirus*, *Trichostrongylus*, *Lamanema*. The major large intestinal worm is *Oesophagostomum*, which is found infrequently. Of these *Lamanema* is reported to be the most pathogenic in South America. The gastric “H.O.T.” complex (initials of the major worm types) worms and the small intestinal *N. battus* appear to be more important in North America. As most of these affect the gastrointestinal tract above the colon, they appear to be more important as causes of weight loss, low protein, anemia, and ill-thrift, versus diarrhea.

Strongyles cause two types of disease. The first type results from grazing heavily contaminated pasture and rapid increase in worm burden. Maturing larvae damage the gastrointestinal wall, leading to a decrease in nutrient absorption and an increase in protein or blood loss. If this build-up occurs rapidly, the disease may occur during larval development, and hence few or no eggs may be present in the feces (prepatent disease).

The second type of disease occurs in the late winter or spring. Larvae ingested towards the end of the previous grazing season arrest their development within the gastrointestinal wall. This is called hypobiosis. Towards the beginning of the next grazing season, something triggers these larvae to continue their development. Thus, there can be an intense period of larval development, leading to severe gut damage and clinical disease. Eggs may not be passed until late in the disease, thus it may also be considered prepatent.

Haemonchus contortus, or the “barber pole” worm deserves special mention. It is far more damaging than similar worms, in part because it feeds on the host’s blood. Blood feeding by L4 larvae and adults may remove 0.05ml blood per worm per day. A typical alpaca blood volume = $0.07 \times 50 \text{ kg} = 3.5\text{L}$. Therefore, each worm removes 0.0014% of blood./day; 1000 worms remove 1.4% of blood/day. The “barber pole” name comes from the double helix of the red, blood-filled intestine and the white, egg-filled ovaries of the adult female worm. Adults live in the acidic part of the third gastric compartment. Females release eggs which hatch out on pasture. Larvae undergo two molts until they become infective L3 larvae. These are susceptible to extreme temperatures, starvation, and desiccation, so they usually gather in water droplets on blades of grass or leafy forage. If eaten, the larvae molt again to L4s and start to feed about 11 days after ingestion on blood from the third compartment wall. It is important to realize

that larvae start the blood feeding. These immature worms do not produce eggs, so it is possible, when large numbers of larvae are ingested over a short period of time, for severe disease to occur several weeks prior to the passage of large numbers of eggs. Eventually the larvae mature to blood-eating adults and the cycle continues.

Clinical signs of severe haemonchosis include the common signs of weakness and ill-thrift, but may specifically include severe anemia. Camelids with single digit packed cell volumes at our clinic frequently are found to have haemonchosis. Most other parasites lead to protein and weight loss, but with only modest red blood cell loss. Because of the anemia, qualitative tests examining oral membrane color may be more useful for diagnosing haemonchosis than they are for other parasitic infections. Remember again that disease caused by larvae may not be accompanied immediately by high fecal egg counts.

The importance of all worm infections appears to be growing with the greater frequency of resistance to common deworming medications. This is a serious problem, especially in the southeastern U.S. Resistant worm populations may be crossing over from ruminants, but also may be arising in camelid herds, especially those using frequent dewormings to prevent diseases such as *Parelaphostrongylus* infection. Drug resistance can be detected in a variety of ways, such as the fecal egg count reduction test (comparing premedication egg counts to those 10 to 14 days later), or the larval development assay. A variety of suggestions have come forth to combat drug resistance, but most have not been evaluated scientifically. These include use of higher doses, use of novel agents, use or alternate methods (such as parasite-eating fungi, copper wires, and toxic plants), breeding for resistance, and pasture management. All have some potential. Regardless of the method chosen, continued surveillance is necessary to check for continued efficacy. As with medications, alternate control strategies have the potential to have diminishing value over time.

Other worms

Strongyloides, *Capillaria (Aonchotheca)*, *Trichuris*, and tapeworms (*Moniezia*) are relatively infrequent findings. *Trichuris* particularly appears to be clinically important. *Capillaria* is known to be an intermittent shedder. Low egg counts can be found in heavily parasitized camelids. *Trichuris* (whipworm) eggs can be hard to detect because they do not float very well in saturated salt solution. Both *Trichuris* and *Capillaria* have lengthy prepatent periods in ruminants, and may cause prepatent disease in camelids. *Trichuris* affect the colon, whereas most of the other worms affect the third compartment or small intestine. By affecting the colon, *Trichuris* are more likely to cause colic, diarrhea, and straining than the other worms.

Liver

Liver flukes

Camelids are susceptible to several species of flukes, including *Fasciola hepatica*, *Fascioloides magna*, and *Dicrocoelium dendriticum*. *F. hepatica* appears to be the most important in the U.S., particularly in the Pacific Northwest, and in England, whereas *D. dendriticum* is the most important fluke in central Europe. Like other internal parasites, they tend to cause an ill-thrift syndrome, with the major difference being that clinical pathology tests point more towards hepatic disease, particularly through increases in gamma-glutamyl transpeptidase activity and bilirubin. Flukes may also contribute to other disorders including clostridial infections and endocarditis.

The primary disease is due to migrating larvae. Thus, egg-shedding may not occur until two to five weeks after the onset of abnormalities.

Worms

Lamanema chavezii is a gastrointestinal worm that has a phase of larval migration through the liver. It causes severe disease in South America, but appears to be rare here. This liver disease may be fatal and prepatent.

Respiratory tract

Lungworms

Lungworm infestations of New World camelids have not been reported in North America, but could potentially occur in areas where camelids are close to infected ruminants or deer. Coughing and respiratory difficulty would be the expected signs.

Nasal bots

Two species of fly, *Oestrus ovis* and *Cephenomyia hominivorax*, deposit larvae on the nose of camelids. The usual hosts for these flies are sheep and deer, respectively. The larvae mature in the nasal passages, and can cause occlusion and irritation. The common signs are respiratory difficulty, snorting, and nasal discharge, sometimes blood-tinged. The maggots can be seen by endoscopy, or suggested by radiographs.

Muscle

Sarcocystis cruzi

Sarcocysts are protozoal invaders of muscle. The definitive hosts are dogs and wild canids. In order to become infected, camelids must ingest something contaminated with infected dog feces. Canine infections are relatively rare in North America, but some imported camelids appear to have arrived with infections. Infected camelids pose no risk to other camelids, and can only spread the disease through their meat. The organisms can cause inflammation of muscle and fascia, which can affect gait or function of specific muscles (extraocular in one case), or potentially be linked to muscle disorders such as megaesophagus. Although camelids are unlikely to receive heavy infections here, stresses may lead to clinical worsening of preexisting infections. The disease is diagnosed through identification of the organism on muscle biopsy.

Nervous system

Parelaphostrongylus tenuis

We are lucky not to have this parasite in the West. The brain worm of white-tailed deer infects snails as its intermediate hosts. Snails transport the larvae and are subsequently eaten by grazing animals. In aberrant hosts, such as camelids, larvae migrate through the brain and spinal cord inducing neurologic disease. Camelids rarely develop patent infections because the long prepatent period (around 90 days) is considerably longer than the average time until development of neurologic signs or death (around 30 to 60 days post-infection). Reported signs include incoordination, hind-end weakness, recumbency, curvature to the neck, blindness, seizures, respiratory paralysis, and death. In many parts of the country, deworming strategies are directed against killing *P. tenuis* larvae before they enter the central nervous system, leading to every 14- day dosing during snail season.

Because of the difficulty in getting eggs, diagnosis is frequently based on compatible signs and geographic location, and the finding of eosinophils within the cerebrospinal fluid. Because of the relatively long period between infection and disease, this disease can be found in camelids transported out of the endemic areas.

Screening for parasites

Alpacas are not uniform in their parasite loads. The old tenet is that 10% of the animals have 90% of the parasites. These animals usually reflect those that have lower immunity (the old, young, and infirm), or have higher exposure (outcasts, dirt-eaters, crias). If the burden is high enough, these animals

will show characteristic signs and should be screened for parasites. If the burden is more moderate, it is harder to identify these animals.

We recommend regular parasite screenings. How often they occur depends on the specifics of the herd. Based on the natural history of most parasites, problems are most likely to occur 1) when there are a large number of younger animals present 2) shortly after mild, wet weather. Considering that most parasites take at least three weeks to develop patent infections, checking feces three+ weeks after the above two events makes sense. In some areas, the condition number two above may be constant, nearly constant, or seasonal.

Generally, if no animals are showing clinical disease, examining feces from around 20% of the animals should provide reasonable surveillance. This may miss the intermittent heavy shedder, but all animals with a previous history of heavy shedding should be added to or included in the 20%.

With most parasites, we try to quantify the fecal egg count, an estimate of the concentration of worm eggs in the feces. In doing so, we hope that these egg counts correlate well to the number of worms or protozoa in the intestine, and hence the risk of disease. Unfortunately, this is not always the case. Certain parasites cause disease before they shed large numbers of eggs or larvae (*E. macusaniensis*, *T. tenuis*, some strongyles, liver flukes, lung worms).

Which test to run?

Which test to run depends heavily on the parasite we wish to detect. Unfortunately, no one test optimally finds all parasites. Many tests completely miss certain parasites.

1) Direct smear

The simplest test, smearing feces with saline on a slide. This is best for motile protozoa such as *Giardia* and *Balantidium* trophozoites, and decent for most types of worm eggs at high concentrations. It is not particularly good at finding parasites where we worry about low numbers of eggs.

2) Saline flotation

Relies on the difference in specific gravity between saturated (1.18) or more dilute saline and the worm eggs. Saline will not float *T. tenuis*, *E. macusaniensis*, or liver fluke eggs. Many other salts, such as 33% zinc sulfate solution have a similar specific gravity to saline. Saline distorts *Giardia* rapidly.

3) Sucrose flotation

Sheather's solution has a higher specific gravity than saline (1.27), and thus will float all but liver fluke eggs. The high viscosity of this solution may inhibit flotation of small coccidia or *Nematodirus*, but may be better than saline for *Cryptosporidium* and *Giardia*.

4) McMaster counting chamber.

Rather than floating the eggs in a tube onto a coverslip, this involves putting a slurry into a counting chamber. The flotation properties become less important. Most parasites can be detected this way, but it is not particularly sensitive for low levels of infection.

5) Double centrifuge technique

Feces are dissolved in water, preferably overnight, then centrifuged. The pellet is resuspended in a flotation solution, which then gives it the properties of that flotation technique. The advantages to this technique are the longer dissolving period enables more eggs to be released from fecal matter, and also the pelleting concentrates eggs. This technique appears to be the best way to find small numbers of eggs. We have found that extending the flotation time beyond the recommended ten to 15 minutes out to one to four hours further improves the yield.

6) Sedimentation

Feces are dissolved in water and either left to settle or pelleted in a centrifuge. A drop or two of the sediment is placed on a slide for microscopic examination. This technique is used most for fluke eggs, which do not float in most solutions. The Flukefinder is a specialized system for sedimenting fluke eggs.

7) Baermann test

This is most commonly done on fresh feces to identify lungworms. If the sample is allowed to sit a few days, *Strongyloides* and strongyle larvae may also hatch out of their eggs and confound the diagnosis. This test involves suspending a fecal sample in cheesecloth within a funnel or cone-shaped vessel. The vessel is filled with warm water and left to sit for at least eight hours. Free-living larvae will concentrate at the bottom of the cone and can be pipetted out for examination under a microscope.

8) Immunologic methods

Fluorescent antibody and enzyme immunosorbent tests are available to aid in the detection of *Cryptosporidium* and *Giardia*. A systemic (blood) antibody response to flukes occurs within about 30 days of exposure and may be useful in detecting prepatent disease.

Table. Prepatent periods for important camelid parasites

	Prepatent	Patent	
<i>Eimeria punoensis</i>	10 days		Illness rare
<i>E. lamae</i>	15-16 days		Illness with patency
<i>E. alpaca</i>	16-18 days		Illness rare
<i>E. macusaniensis</i>	32-43 days	20-40 days	Illness possible 2-3 weeks before patency
<i>Cryptosporidium</i> spp.	3-7 days	4-5 days	Illness with patency
<i>Giardia duodenalis</i>	5-21 days	months	Illness possible 7-10 days before patency
<i>Balantidium coli</i>			
Strongyle worms	17-36 days		
<i>Nematodirus battus</i>	14-21 days		
<i>Trichuris tenuis</i>	60 days?		
<i>Capillaria</i> spp.	42 days?		
<i>Moniezia</i>	40 days?		
<i>Dictyocaulus</i> spp.	22-25 days?		Prepatent disease possible
<i>Fasciola hepatica</i>	70-84 days?		
<i>Dicrocoelium dendriticum</i>	49-79 days?		

