The overly thin sheep or goat is a common presentation for veterinary intervention, either as the client’s chief complaint or a finding during other herd work. Poor body condition is a non-specific sign caused by a wide variety of underlying pathophysiologic processes. Definitive diagnosis and successful treatment or herd interventions will be facilitated by using a step-wise, problem-based approach.

**Database: History and physical exam**

Begin with a thorough history and physical examination, including verification of the client’s observations. Signalment, production status, and onset/duration of the weight loss should be taken into consideration, and the patient should be evaluated against its herd mates to identify individual vs. systemic problems. Ask the owner about the herd health program – specifically parasite control protocols, purchased animals, and history of caseous lymphadenitis (CLA), caprine arthritis-encephalitis virus (CAE) or ovine progressive pneumonia (OPP), and Johne’s. Inquire about any past health issues, including pneumonia or scours which may have led to chronic conditions.

Fully evaluate the feeding program, including water sources. What feedstuffs are offered, how much, and how often. Have the forages been tested? Is a mineral offered, and if so, what type. Is there sufficient bunk space or feeder access for the herd? Look at pastures, feeders, hay, and waterers to identify obvious problems with overgrazing, hygiene, quality, or access. Consider the “Four Diets” - the paper ration, the feed as mixed, the feed consumed, and the actual diet digested; where do they vary? How much sorting or waste is occurring? Is the mineral being consumed, and at what rate? Are separate sources of salt or other supplements being offered?

In addition to the history, the minimum database should include a complete physical exam on a representative group of animals, including body condition scoring, and FAMACHA score. Body condition scoring systems for small ruminants typically run on a 1 (emaciated) to 5 (obese) scale and absolutely require a hands-on evaluation for accuracy. Sheep and meat goats are evaluated over their loin; goats store more fat internally than sheep, so a slight allowance may be made especially when scoring leaner breeds. A separate scoring system should be applied to dairy goats.

In addition to the routine physical examination, extra attention should be paid to the oral and pharyngeal cavities, looking for deformities (e.g. malocclusion or cleft palate), dental pathology, or other trauma.

**Problem list – the damnit scheme**

From the evidence collected in this database, it will be necessary to identify, localize, and develop a pathophysiologic hypothesis for the observed problems. In order to avoid prematurely focusing on any specific etiology, the DAMN-IT scheme can be used to help draft a list of possible processes causing the observed weight loss, categorized by physiologic mechanism. The acronym reminds us to consider:

| D  | degenerative, developmental |
| A  | autoimmune                   |
| M  | metabolic, mechanical, mental|
| N  | nutritional, neoplastic      |
| I  | inflammatory, infectious, ischemic, immune, inherited, iatrogenic, idiopathic |
| T  | traumatic, toxic             |

Weight loss may be an expected reaction to the current production stage (breeding male or lactating doe). In the context of emaciation, major mechanisms across all age groups include nutritional, mechanical, and infectious etiologies. Less commonly, cachexia may present secondary to developmental abnormalities, neoplasia, or other chronic inflammatory condition of organ failure.

<table>
<thead>
<tr>
<th>Deenerative</th>
<th>Neonate</th>
<th>Youngstock</th>
<th>Adults and Aged</th>
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<td>Diaphragmatic Hernia</td>
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<td>Goiter</td>
<td>H-P-T Axis disorder</td>
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<td>Metabolic</td>
<td>Pica</td>
<td>Obstruction</td>
<td>H-P-T Axis disorder</td>
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<td>Mechanical</td>
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<td>Incisor loss / Oro-esophageal Pathology</td>
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### Lameness pathology

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<thead>
<tr>
<th>Category</th>
<th>Nutritional</th>
<th>Inflammatory</th>
<th>Infectious</th>
<th>Immune</th>
<th>Inherited</th>
<th>Traumatic</th>
<th>Toxic</th>
<th>Initial plan and follow-up</th>
<th>Therapeutic and client education</th>
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<td>At this point, the signalment, history, physical exam, and oral exam findings should be sufficient to support an initial plan for diagnostic, treatment, and client education. Diagnostic modalities can be used to verify the presumptive diagnosis, further localize the disease process, elucidate the pathophysiologic mechanism, and rule-in/out differentials. Depending on the case, clinical pathology including quantitative fecal floatation, complete blood count (including fibrinogen), and/or serum biochemistry (including GGT) may be indicated.</td>
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<td>Abomasal Emptying Defect (Suffolk)</td>
<td>Oro-pharyngeal Trauma</td>
<td>Organ Damage from Toxicosis</td>
<td>Therapeutic and client education</td>
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<td>Oro-pharyngeal Trauma</td>
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**Neoplastic:**
- Lymphosarcoma
- Msc. Tumors

**Infectious:**
- Scours
- Orf
- Parasitism
- Giardia
- Coccidiosis
- Chronic Illness

**Inflammatory:**
- Rumeno-reticulitis
- Abomasitis or Ulcer

**Immune:**
- Failure of Passive Transfer

**Inherited:**
- Abomasal Emptying Defect (Suffolk)

**Traumatic:**
- Oro-pharyngeal Trauma

**Toxic:**
- Organ Damage from Toxicosis

**Initial plan and follow-up**

**Diagnostic**

At this point, the signalment, history, physical exam, and oral exam findings should be sufficient to support an initial plan for diagnostic, treatment, and client education. Diagnostic modalities can be used to verify the presumptive diagnosis, further localize the disease process, elucidate the pathophysiologic mechanism, and rule-in/out differentials. Depending on the case, clinical pathology including quantitative fecal floatation, complete blood count (including fibrinogen), and/or serum biochemistry (including GGT) may be indicated.

In addition to gastrointestinal parasites, CLA, CAE/OPP, and Johne’s are the big three infectious diseases that commonly present as chronic weight loss in the face of a good appetite. Serology (ELISA or antigen specific AGID) is very useful for diagnosis of CAE and OPP outside of the periparturient period; though there is a significant degree of cross-reaction between the lentiviruses, OPP-based AGID test are less sensitive when used to test for CAE. Serologic testing (SHI) for CLA is typically recommended only as a herd screening tool as it is difficult to interpret at the individual level. Cross-reactions, past infections, and vaccination can all produce titers in individuals without active disease, and occasionally low titers will be observed in animals with active, culture positive abscesses. However, caprine titers ≥1:512 are highly (~95%) correlated with internal abscesses and should have a higher positive predictive value in a high-risk population. Johne's diagnostics, including both serology and fecal PCR, are similarly challenged by low sensitivity, especially early in the infectious course. However, appreciable weight loss is a sign of terminal clinical disease, and either method should perform reliably at this time point.

Imaging, especially abdominal and thoracic ultrasound, can be done to evaluate rumen and GI motility, rule-out pregnancy or advanced uterine tumors, and screen for enlarged mesenteric lymph nodes, and look for evidence of chronic pulmonary consolidation; a more skilled ultrasonographer could also evaluate liver, kidney, and heart for abscesses, parenchymal abnormalities, or valvular insufficiency. Thoracic radiographs may be a better option for assessing pulmonary changes and identifying mediastinal masses. Many conditions, including internal CLA abscesses and tumors, may not be diagnosed until necropsy; scrapie testing should be considered for any remaining open diagnosis of chronic wasting.

**Therapeutic and client education**

Based on the initial database and any timely diagnostics, the initial treatment plan should specifically address the suspected disease process as well as address any additional symptomatic or supportive care requirements. If gross nutritional inadequacies are suspected (or effective access to feedstuffs is limited by overcrowding or lameness), the animal can be challenged with a higher energy or protein diet offered more frequently or with protected access to decrease competition from herd mates. In uncomplicated cases, condition should noticeably improve within two weeks, ideally confirmed by evidence of actual weight gain. Species-appropriate loose mineral should be available ad-lib, other sources of salt (e.g. salt blocks or loose salt) will compete with the mineral mix for consumption. Geriatric patients will benefit from a highly-palatable, easily digestible diet that may need to be chopped or soaked for easier mastication (e.g. complete ration pellets, alfalfa pellets, beet pulp, or Chaffehay). If neonates are failing to thrive, inspect the

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dam’s udder for teat and gland function, mastitis, or trauma. As a rule of thumb, the dam can be expected to raise one lamb/kid per functional half. Although there are many systems that can be made to work for raising orphans, when bottle babies are struggling recommend that the client feed species-appropriate milk or milk replacer at 15-20% of their body weight daily (3 ounces per pound) divided into 3-4 feedings and weigh the kids or lambs daily for a week to assess response.

If parasitemia is present, it is important to investigate the herd deworming protocol and any history of individual deworming in the patient in order to select a suitable drug. Anthelmintic resistance is common, especially in *Haemonchus contortus* populations, and the herd dewormer may not be effective. Fecal egg counts conducted at the time of presentation/deworming and then again 10-14 days later can help identify anthelmintic failures. For animals affected by *Haemonchus*, with a correspondingly low FAMACHA score, the FAMACHA score should improve by one grade within two weeks after deworming. Chronically parasitized animals may benefit from parenteral or oral iron supplementation.

There are no effective treatment options for CAE/OPP, CLA, or Johne’s disease. Chronic pneumonia or chronic coccidiosis cases are also unlikely to respond well to therapy but early intervention or control programs may be indicated for other herd members. Oral or limb pathology should be addressed as appropriate, with a plan for nutritional support during the treatment and recovery period. Rumenotomy is indicated for obstructive disorders in the adult; reported cases of starvation-induced pica in neonates have not responded well to any intervention.

**Additional resources**

Dewormer Chart for Goats. [http://media.wix.com/udcd/adedd98_c7a6cc3b624043a9ee6e86939df37ce71.pdf](http://media.wix.com/udcd/adedd98_c7a6cc3b624043a9ee6e86939df37ce71.pdf)

Dewormer Chart for Sheep. [http://media.wix.com/udcd/adedd98_c7a6cc3b624043a9ee6e86939df37ce71.pdf](http://media.wix.com/udcd/adedd98_c7a6cc3b624043a9ee6e86939df37ce71.pdf)

Henning, E. Dairy Goat Body Condition Scoring Video. [www.youtube.com/watch?v=FC0u1j06y5Y](http://www.youtube.com/watch?v=FC0u1j06y5Y)

Langston University. Body Condition Scores in Goats. [www2.luresext.edu/goats/research/BCS_factsheet.pdf](http://www2.luresext.edu/goats/research/BCS_factsheet.pdf)

On-Line FAMACHA Training, University of Rhode Island Northeast Small Ruminant Parasite Control. [http://web.uri.edu/sheepngoat/famacha/](http://web.uri.edu/sheepngoat/famacha/)

Oregon State University Extension. Body Condition Scoring of Sheep. [http://ir.library.oregonstate.edu/xmlui/bitstream/handle/1957/14303/ec1433.pdf](http://ir.library.oregonstate.edu/xmlui/bitstream/handle/1957/14303/ec1433.pdf)

Intestinal coccidia and strongyles, specifically *Haemonchus contortus* (barber-pole worm), are the two major internal parasites of concern on most domestic small ruminant operations. Clients are often confused about these parasites, deworming protocols, and parasite control programs in general. With the threat of anthelmintic resistance, there is no one-size fits all solution. Instead, tailored solutions must be developed that take into account the production goals and sub-populations, historical performance records, exposure risk factors, epidemiology of the organism of interest, and existing drug resistance patterns. The American Consortium for Small Ruminant Parasite Control website contains an extensive listing of current best practices and timely information on the topic (www.wormx.info). A “Top Ten” client education summary specific to *Haemonchus* control is included at the end of this proceedings.

**Coccidiosis**

Coccidiosis is the most common cause of debilitating diarrhea in young ruminants between three weeks and five months of age, with effects ranging from sub-clinical unthriftiness and reduced gains to peracute death. After about five months of age, acquired immune resistance limits the replication and clinical impact of the of the parasite; thus the producer’s major challenge lays in preempting the significant youngstock morbidity and mortality associated with coccidiosis without interfering with the natural acquisition of immunity.

Coccidia are host-species specific, though some species do cross-infect between sheep and goats. Oocysts are regularly shed in the feces at low levels by healthy adult ruminants and at much higher levels by sub-clinically and clinically affected youngstock. Transmission to youngstock is facilitated by high-density housing and manure contamination of feed and water sources; kids and lambs born at the end of a kidding season may be exposed to a very high infectious dose in the first weeks of life. Acute clinical coccidiosis is most common around periods of stress, such as weaning. Stressed or immunocompromised adults are also susceptible to clinical breaks.

In addition to fluid therapy and other ancillary supportive care, treatment options for acute coccidiosis include oral sulfonamides or thiamine agonists such as amprolium. These drugs are coccidiostats which limit replication but do not eliminate the organism. They are primarily effective early in the organism’s reproductive cycle and may appear more effective when used prophylactically for control than in the face of acute disease. Amprolium has been used extra-label in goats at a dose as high as 50 mg/kg, but patients should be monitored closely for signs of polioencephalomalacia. Several triazine coccidiocidal drugs (e.g. diclazuril and toltrazuril) are available overseas for small ruminants but not domestically. Ponazuril (Marquis®) is a related drug which is FDA-approved for treatment of Equine Protozoal Myeloencephalitis in horses and has been used extra-label for treatment of acute coccidiosis in small ruminants (~10 mg/kg orally as a single dose, FARAD recommends a prolonged slaughter withdrawal period of approximately four months).

Control and prevention strategies focus on both reducing avenues for fecal-oral transmission and reducing the number of total oocysts in the environment. Reduce overcrowding, increase cleaning of pens and equipment, and construct feeders that kids cannot climb into. Oocysts are very persistent in the environment and resistant to most disinfectants so it is difficult to reset the clock once a farm has been highly infected. Whitewash, paint, and impervious materials can be used to reduce build-up of oocysts in the facility environment. In herds that struggle with coccidiosis in the youngstock, does and ewes may be started on a coccidiostat in the feed or water from one-month prior to the start of kidding/lambing through weaning and a medicated starter feed should be offered to the youngstock; in the U.S., decoquinate is labeled for both sheep and goats, lasalocid is labeled for sheep, and monensin is labeled for goats. Youngstock may also be treated with amprolium on a 21-day prophylactic cycle.

**Nematodes**

In most of the continental U.S., *Haemonchus contortus* is the most significant nematode parasite of small ruminants. As a rule of thumb – if you can grow corn, you can grow Haemonchus; this includes irrigated pastures in otherwise arid climates. Other clinical-relevant small ruminant nematodes include *Teladorsagia* (formerly *Ostertagia*), *Trichostrongylus*, *Cooperia*, *Oesphagostomum*, *Nematodirus*, and *Trichuris*. Clinical signs of strongyles infestation include varying degrees of anemia, diarrhea, dependent edema (e.g. bottle jaw), production losses, and death. *Haemonchus* is specifically associated with profound anemia but not typically with diarrhea. For this reason, the FAMACHA© test can be applied in locations where *Haemonchus* predominates, while the Five Point Check© system may be more appropriate on operations where other strongyles prevail. FAMACHA© training materials can be found on-line for free through the University of Rhode Island Northeast Small Ruminant Parasite Control website and the citation for the Five Point Check© is included with the references. These nematodes are transmitted in a fecal-oral pattern and larval development and transmission requires access to forage; for the most part, strongyle transmission is interrupted in true dry-lot or indoor environments.

Kelly Still Brooks, DVM, MPH, DACVPM

Iowa State University

Ames, IA
However, larval hypobiosis is common during the winter in more northern locations when pasture conditions are not favorable for larval survival in the environment.

In conjunction with clinical signs, quantitative fecal egg counts (FEC) are useful for estimating the total strongyle parasite load. The different species of strongyle eggs cannot be differentiated on a routine fecal; if needed, speciation can be performed via larviculture, peanut lectin staining, or PCR. Fecal egg counts commonly rise during the periparturient period. Fecal egg count reduction tests (FECRTs), which consist of serial individual FECs taken 10-14 days apart can be used on a herd level to evaluate dewormer efficacy; <95% reduction indicates some degree of resistance is present and ~50% reduction or less is commonly associated with perceived lack of clinical efficacy. The downside to the FECRT is that only one dewormer class can be evaluated per cycle, it takes approximately two weeks to complete a cycle, and serial testing of recommended numbers of animals can become expensive over time. If clients are reluctant to pursue FECRTs, have them record FAMACHA© scores at deworming and two weeks later – scores should improve by at least one grade if the dewormer is having any effect. If no improvement is noted, that strongly suggests that there is a significant degree of resistance to the dewormer and further diagnostics are needed. The DrenchRite® assay offers another alternative to serial FECRTs. This test hatches out larvae from a fresh comingled fecal sample and exposes the larvae to multiple concentrations of the major classes of dewormers (benzimidazoles, levamisole, avermectins and indirectly moxidectin). While the initial cost of this test is high, it may be quicker and less expensive in the long run. The test should be run when FECs are reasonably high and pre-coordination with the lab (Dr. Ray Kaplan, University of Georgia, (706) 542-0742) is necessary to ensure the correct sample is submitted.

Anthelmintic resistance is a major problem in small ruminant herds. Current deworming protocols should utilized a targeted selective treatment approach (deworm only clinically affected animals identified by FAMACHA, 5 Point Check, or FEC), or a strategic deworming approach based on the lifecycle and epidemiology of the parasite of concern (either presumptive or identified via larviculture or PCR). These approaches are designed to minimize production losses while preserving a refugia of non-resistant parasites, decreasing anthelmintic resistance selection pressures. It is especially important to minimize progression of resistance in the breeding and replacement herd, in market animals that are being reared with the permanent herd, and in any animals that will be utilizing pasture used for the permanent herd. In heavily parasitized herds, or those with multi-drug resistant parasites, the herd may need to be subdivided and a tailored approach developed for each class of animal (e.g. market stock vs. replacement stock vs. mature breeding stock). Producers that keep good records will ultimately have a better basis for developing an educated parasite control protocol base on knowing the historical patterns of clinical parasitemia, evidence for (or against) specific drug efficacy, and identifying chronic offenders for culling. Since parasite resilience is a heritable trait, culling chronic offenders can improve overall herd resilience. Remind producers that when they purchase replacements, they purchase both the parasites, and their anthelmintic resistance patterns. As a general guidelines for using anthelmintic drugs in small ruminants: DO NOT routinely rotate dewormer classes, DO use oral routes of administration, and DO dose appropriate for weight – increasing the dose for goats by 1.5-2x the label sheep dose depending on the product used. Anthelmintic dose charts that includes extended withdrawal information are available on-line for goats, sheep, and camelids; the links are included below in the references.

Several non-chemical adjunct control methods may be considered for nematode parasite control. First, evaluate the herd’s nutritional program. We commonly assume that parasites caused the unthrifty animals, but stock reared on a poor plane of nutrition will also be less resilient to strongyle infestation, especially when protein is inadequate. Unfortunately, pasture rotation alone is ineffective for parasite control, but inter-species rotation with non-susceptible species (e.g. cattle or horses) or haying stocked forage can decrease existing parasite loads on pasture. FECs can be reduced by feeding condensed tannin forages (e.g. sericea lespedeza) either via grazing or as supplemented hay or pellet, but these feedstuffs must be incorporated as a significant percentage of the diet. Copper oxide wire particles have also received attention for parasite control in goats (not sheep due to high risk for copper toxicity); although the COWP boluses are safer than copper sulfate drenches, the herds liver copper status should be determined before initiating treatment and then regularly monitored. COWP boluses are increasingly being frequently administered at high levels by dairy goat owners, independent of their veterinarian, increasing the risk for copper toxicosis.

**Tapeworms**

*Moniezia* spp. are the most common tapeworm of sheep and goats in the U.S. Since the segments are easy to see when passed in the feces, they are often a concern to owners. However, they are rarely pathologic and treatment is not typically indicated. One exception may be high levels of infestation in youngstock, where they can be associated with altered gut motility and decreased intakes. Tapeworms are not treated by the common macrocyclic lactone anthelmintics; praziquantel can be used extra-label in sheep and goats.

**Further reading**

Ten to remember: Small ruminant haemonchus control

1. **Resilience.** Instead of eliminating all gastro-intestinal parasites, our main deworming goal is to keep animals healthy and productive despite a moderate parasite burden. While high numbers of parasites can make any animal ill, certain production groups (youngstock and lactating animals) and highly stressed sick individuals are more susceptible to clinical parasitemia. Good overall nutrition and health is an important part of the parasite control program.

2. **70-30.** 70% of your farm’s parasite problem comes from roughly 30% of the animals. In addition to the high-risk groups, this 30% also includes individuals who just can’t handle a moderate parasite load as well as the rest of the flock/herd. These individuals are the biggest contributors to parasite eggs on pasture; culling them can improve overall herd resilience over time, decreasing the need for deworming.

3. **Resistance.** Small ruminants - especially goats - are REALLY good at creating dewormer-resistant parasites. Find a dewormer that works and stick with it until it doesn’t; do not rotate between drugs or you will hasten development of resistance to all the drugs used. If you think your dewormer may no longer work, or if you are unsure what dewormer will be effective in your animals, we can assess the dewormer efficacy in your herd through a Fecal Egg Count Reduction Test or a DrenchRite® Test.

4. **Refugia.** Ideally, the number of eggs on pasture from non-resistant parasites greatly outnumbers those from resistant parasites – this is the refugia. Since our biggest concern in the mid-west is *Haemonchus contortus*, the barberpole worm, we can use a targeted selective treatment protocol (FAMACHA) to identify only clinically ill animals for deworming, allowing the remainder of the flock to pass low levels of eggs that have not been selected for resistance.

5. **Oral Drenching.** Give all dewormers orally with a drench gun; do not use injectable or topical dewormers. Ideally, hold the animal off feed for 24 hours prior to deworming.

6. **Adequate Dose.** Use the correct dose and do not underestimate the animal’s weight. Sheep should be dewormed according the label instructions; goats are usually dewormed at twice the sheep or cattle dose. One key exception is with Levamisole, which can be toxic at higher levels.

7. **Emergency Reserve.** It seems like we always find out that our dewormers no longer work at the worst time when animals are critically ill. Plan ahead to have an “emergency only” drug that will work in a pinch – we recommend that you AVOID routinely using Meloxicam (Cydectin) so that this drug will still be effective for use in an emergency, buying time to find a long-term solution.

8. **Package Deal.** When you buy in a new animal, you are also purchasing that animal’s parasites, and any dewormer resistance in those parasites. Eggs from those resistant parasites will infest your pasture and the rest of your herd or flock, accelerating your drug resistance problem.

9. **Dry Lot.** *Haemonchus* needs grass to complete its life cycle. A true dry lot, without any grass or weeds, can be used to help break the parasite cycle or as a quarantine area for new animals.

**Pasture rotation**

While pasture rotation is great for maximizing forages, a typical two- to four-week cycle is actually perfect for growing gastrointestinal parasites. Pastures would need to rest for many months in order to significantly break the parasite cycle. Better options include alternate species grazing such as with cattle or horses (note: sheep, goats, and camelids do share parasites), or haying pasture between grazing cycles.
Although aberrant migration of the deer meningeal worm, *Parelaphostrongylus tenuis* is a commonly recognized cause of neurologic disease in South American camels, *P. tenuis* is less frequently considered as a differential for spinal neuropathies in goats and sheep. In enzootic regions of the U.S. (Atlantic coast west to the Dakotas, Nebraska, Oklahoma, and Texas), meningeal worm should considered as a cause of asymmetric multi-focal neurologic disease in small ruminants as well as camels, as well as for cases of scoliosis, “downers”, and suspected brain lesions in camels. In addition to classic camelid cases, we have observed an uptick in cases of meningeal worm among Midwestern goat herds. It appears that 2015’s unseasonably cool and wet summer has contributed to both greater and earlier exposure to the parasite, especially for goats grazing woodland habitat. These cases involve a range of ages from older pre-weaned kids (4-6 months) to mature animals. Both sporadic cases and herd outbreaks involving as much as 15% of the herd have been observed.

**Epidemiology**

*Parelaphostrongylus tenuis* normally follows an indirect neurotropic lifecycle that passes between the white-tailed deer definitive host and a variety of intermediate host terrestrial gastropods. L1 larvae from deer feces penetrate the foot pad of a snail or slug, where they mature to the infective L3 stage. These L3 emerge from the footpad into the mucous trail environment, where they can survived desiccation and cold stress. After either the gastropod or emerged L3 larvae are ingested by a white-tailed deer, the L3 will migrate through the intestines and peritoneum to the dorsal horn of the spinal cord, and then mature during migration through the subdural space to the cranium. As adults, they enter the dural venous sinuses and deposit eggs on the meninges. Both eggs and embryonated L1 larvae travel via the circulatory system from the venous sinuses to the lungs, are coughed up and swallowed, and expelled in the feces. This life-cycle typically takes four months to complete and little to no clinical disease is observed in infected deer. Camelids, goats and other accidental hosts become infected by ingesting infective L3 larvae present either in the snail or slug, or in the slime trail. Once ingested, normal larval maturation is disrupted, resulting in aberrant migration through the spinal cord and CNS. No eggs are produced, but the neural inflammation and parenchymal damage from aberrant migration causes clinical central nervous system disease with variable signs depending on the anatomic location of the lesions. Clinical disease is most common in the late fall and early winter, roughly forty-five to sixty days after the peak exposure period in the early fall. However, if moderate summer temperature reduce the gastropod’s normal aestivation period, clinical cases may start appearing much earlier in the fall. Exposure to white-tailed deer or common pasture/browse is a common finding in the case history.

**Clinical signs**

Common clinical findings include upper- and lower-motor neuron, and peripheral limb sensory deficits. Cases often present with progressive neurologic deficits that start as hind-end weakness and ataxia which may lead to complete paresis and death. Clients may perceive that the animal is lame, stiff, weak, base-wide, ataxic, slow or reluctant to move, or unable to rise. In the absence of primary brain lesions, most affected animals are initially bright and alert with a good appetite and no fever. Depending on the anatomic locations of the lesions, the physical exam may find evidence of ataxia, hemi- or tetra-paresis, impaired proprioception, decreased nociception, altered reflexes, or scoliosis; advanced cases may be tetraplegic. Acute-onset cranial nerve, vestibular, and brain signs may be identified in atypical camelid cases. In goats, the most consistent findings include asymmetric multi-focal neurologic disease with emphasis on pelvic limb deficits. The clinical signs often progress over the course of several weeks, terminating in death or euthanasia although presumptive sporadic recoveries have been reported. In addition to the neurologic signs, peritonitis and colic symptoms have been reported in goats after high-dose experimental infections and linear pruritic dermatitis lesions have been observed in natural infections.

**Differentials**

Major differentials for typical clinical presentations of *P. tenuis* include all other causes of spinal meningomyelitis. Antemortem differentiation of meningeal worm from other inflammatory and degenerative etiologies may be possible through CSF analysis. An increased CSF white cell to protein ratio is commonly observed with meningeal worm. When present, an eosinophilic pleocytosis is highly specific for cerebrospinal nematodiasis and can be used to rule-out other etiologies. Copper deficiency degenerative myelopathy (enzootic ataxia, swayback) is the most common cause of caprine spinal cord lesions and should be a top differential for hind-end weakness and ataxia occurring in multiple neonatal to weaning-age kids, especially those raised on a sheep concentrate/mineral or high molybdenum-diet. Low liver copper levels are diagnostic for enzootic ataxia. Additional sporadic causes of degenerative myelopathy include compressive lesions from vertebral malformations, trauma, abscess, or neoplasia. Those cases typically present with a more focal anatomic localization than usually observed with *P. tenuis*. Common differentials for inflammatory
spinal neuropathy include lentiviral infection (caprine arthritis-encephalitis virus) as well as any other less-likely viral cause of nonsuppurative meningoencephalomyelitis, such as rabies, pseudorabies, border disease, and West Nile virus. Unfortunately, a non-eosinophilic CSF mononcytosis has been reported in some caprine and camelid meningeal worm cases, which complicates exclusion of viral meningoitis and listeriosis. Other reported causes of spinal cord lesions include congenital anomalies and neoplasia.

Additional differentials may be considered with extremely mild or severe presentations; in all cases, eosinophilia in the CSF strongly supports a meningeal worm diagnosis. Advanced tetraparetic and terminal cases of meningeal worm may resemble common causes of “downer” animals, including polioencephalomalacia, listeriosis, pregnancy toxemia, and hypocalcemia. However, the hallmark clinical signs of listeriosis and polioencephalomalacia, (unilateral cranial nerve deficits and bilateral menace deficits concurrent with intact pupillary light reflexes, respectively) are not expected findings with cerebrospinal nematodiosis. Pregnancy toxemia and hypocalcemia can be readily diagnosed through serum β-Hydroxybutyric acid and ionized calcium levels. Less common differentials for downer presentations include brainstem abscess, bacterial or viral meningoencephalitis, tick paralysis, organophosphate toxicity, and lidocaine toxicity. In contrast, mild presentations of meningeal worm may mimic the transient flaccidity or stiff, spastic movement and hyperreflexia observed in the early stages of tetanus, but would not include the third-eyelid spasm and tonic seizures classically associated with tetanus infection. Laminitis, polyradiculoneuritis, myotonia congenita, and nutritional muscular dystrophy (vitamin E and selenium deficiency) may also cause similar abnormalities in gait and posture. Finally, rabies should always be considered with any open neurologic case and small ruminants should be submitted for scrapie testing. In particular, the pruritic skin lesions reported in association with meningeal worm in goats are also characteristic of scrapie. Both of these diseases require postmortem testing for definitive diagnosis.

Diagnostic plan
Definitive diagnosis of *Parelaphostrongylus tenuis* is complicated both by the lack of antemortem tests as well as difficulty in locating the parasite on necropsy. Field diagnosis is based on exposure history, clinical presentation, exclusion of other differentials, cerebrospinal fluid analysis, and response to treatment. At necropsy, gross lesions are uncommon and the nematodes are rarely found on routine examination. Somewhat more frequently, secondary parenchymal changes typical of neural larval migrans may be observed. These relatively characteristic necrotic and eosinophilic inflammatory lesions may include axonal degradation, eosinophilic encephalitis, and hemosiderin-laden macrophages, and gitter cells; occasionally nematode cross-sections are observed in the sections. A nested PCR technique has been applied to formalin-fixed, paraffin-embedded tissues with some success even when the parasite is not observed in the tissue section. Although research efforts are focused on improving antigenic targets for immunodiagnostics and prophylaxis in cameldids, there are no commercially available serologic tests or vaccines.

Treatment protocols
Meningeal worm treatment protocols include extra-label high-dose, extended course fenbendazole, anti-inflammatories, and supportive care along with single dose ivermectin for control of immature larvae. Although avermectin anthelmintic are used in prophylactic control programs, they are actively effluxed from the CNS and are considered ineffective for clinical cases. Typical treatment protocols call for five days of high-dose oral fenbendazole (50 mg/kg) along with a single dose of ivermectin to kill any intestinal and peritoneal L3.

In addition to anthelmintic therapy, anti-inflammatory drugs are indicated to address the existing CNS inflammation from the larval migration and modulate the secondary immune reaction to the dying parasites. Both glucocorticoid steroids and non-steroidal anti-inflammatories have both been used at species-appropriate doses. Glucocorticoids, such as dexamethasone (0.1 mg/kg IM q24 hours for 5 days), are widely used against CNS inflammation. They decrease tissue inflammation and edema, improve vascular permeability, and facilitate neuronal excitability and impulse conduction, and reduce inflammatory chemokine driven neuropathology. Nonsteroidal anti-inflammatory (NSAIDS), most commonly flunixin meglumine (1.1-2.2 mg/kg IV q24 hours for 3 days), non-selectively inhibit cyclooxygenase-mediated inflammatory pathways in the CNS as well as in peripheral tissues. Dimethyl sulfoxide (DMSO, 1 g/kg IV) may be administered to protect against continued oxidative injury to the cellular architecture by free radicles produced by phagocytic leukocytes (including eosinophils) during the inflammatory process.

Adjunct therapies include supplementation of key vitamins, fluid and electrolyte support, and attention to key husbandry practices. Vitamin E (α-tocopherol), like DMSO, is a non-specific antioxidant that is also critical to neurocellular health. Vitamin E deficiency is a primary cause of neurologic and skeletal muscle degeneration; supplementation would counter the effect of reduced intake from anorexic animals as well as provide additional neuroprotective anti-oxidant effects. B-complex vitamins, including thiamine (B1) and cobalamin (B12), are vital to metabolic pathways necessary for normal brain and nervous system function. They are normally adequately synthesized by the rumen flora in healthy animals; however, parenteral B-complex supplementation is advisable in the face of decreased appetite or rumen function. Fluid therapy protocols depend on the animal’s requirement, desired route, and fluid types available. Dehydrated, anorexic, and/or severely depressed patients require intravenous fluid therapy for resuscitation and base maintenance requirements (50 mg/kg/day, non-lactating animals). Continuous administration of isotonic non-alkalinizing solutions are
frequently utilized in the hospital setting for this purpose; the fluid base may include 0.9% sodium chloride, acetated Ringer’s solution, or another balanced isotonic electrolyte solution supplemented with potassium chloride (20-40 mEq/L) and/or calcium gluconate (25-50 mL/L) as clinically indicated. Although intravenous fluid therapy can be managed on-farm in the right settings, if protracted, these cases will eventually require supplementation with partial parenteral nutrition, which absolutely requires a higher level of case management in a hospital environment. For more functional patients, intravenous hypertonic saline and/or oral fluid and electrolyte solutions are more practical and less expensive methods of correcting fluid deficits and minor electrolyte imbalances in the field. Hypertonic saline (4-5 ml/kg) is particularly effective at rapidly expanding plasma volume at the expense of the extravascular compartment, but must be accompanied by additional oral or isotonic intravenous fluids. Non-alkalinizing electrolyte solutions are typically indicated for adult ruminants. An appropriate oral electrolyte solution based on sodium chloride, potassium chloride, and calcium chloride salts will provide relatively high levels of chlorine and potassium. Alkalinizes such as bicarbonate, acetate, and propionate, which are commonly included in calf electrolyte solutions, are contra-indicated. Attention to primary husbandry considerations include shelter from sun and inclement weather, and functional access to feed and water. Deep bedding, intentional position changes, and physiotherapy that includes passive range of motion exercises are indicated to limit secondary pressure myopathy, compartment syndrome, formation of decubital lesions, and tendon contraction.

Prognosis
Treatment prognosis is dependent on severity of clinical signs at presentation. Ambulatory animals generally respond to treatment but may retain residual neurologic deficits; prognosis for downer animals is poor. Ambulatory cases typically have a fair prognosis for survival but may not recover all neurologic deficits; when a modified neurologic scoring system is applied (0-5, grade zero indicating no gait deficits, grade five are recumbent), clinical experience indicates that a one-score improvement after treatment is a reasonable expectation for responding animals. Continued improvement more than six months after treatment is not likely. Sporadic recovery has been reported in multiple species.

Prevention and control
Meningeal worm prevention strategies can be group into chemoprophylaxis and management-centric options. Routine administration of avermectin anthelmintic compounds is effective against early larval stages before they migrate into the CNS; unfortunately, this approach facilitates anthelmintic resistance in common gastro-intestinal nematodes such as Haemonchus contortus. Chemoprophylaxis is often administered year-round, but it can be targeted seasonally in some climates; for instance, in locations that experience significant cold freezes and hot, dry summers, 85% of meningeal worm exposure occurs from September to December. Management strategies include efforts to separate livestock from the source. Most producers elect to limit livestock access to wet and marshy areas or other high-risk habitat. In highly endemic areas, deer fencing, vegetative barriers, and molluscicides have been employed, but it is not clear if these methods are effective or should be recommended.

Additional reading
Questions on Q Fever
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Chances are, when Q-fever comes up as part of your professional practice, it is because of an abortion work-up, a trace-back from human illness, or questions from a client on obtaining negative testing for an animal or herd. It has gained new attention due to recent outbreaks of disease in people where it has been implicated in both acute flu-like illness and more rarely in serious chronic disease. As a “new” old disease, advancements in the areas of Coxiella epidemiology and diagnosis unfortunately have not drastically improved our ability to manage the disease due to inherent limitations in the diagnostic testing modalities, limited control options, and zoonotic potential. Small ruminant veterinarians need to be prepared to answer client questions about exposure risks, clinical disease, control and treatment options, and personal safety.

Organism, epidemiology, and zoonotic risk
Q-fever, which is known in animals as coxiellosis, is caused by the gram-negative intracellular bacteria Coxiella burnetii. This agent is ubiquitous in the environment, where it can in a spore-like form for an extended period of time and become airborne, moving a mile or more on wind currents. It is primarily spread through aerosol transmission or direct contact with infected animals or exposure to infective materials, which include abortion or birth products, vaginal discharge, milk, feces, urine, and semen from infected animals.

Clinical disease (abortion, stillbirths, and weak neonates) most commonly occurs in domestic ruminants (sheep and goats, to a lesser extent cattle), but other vectors, including cats, birds, and ticks, have been implicated in human exposure; prevalence as high as 40% has been reported in feral cat populations. Most livestock infections are asymptomatic; in fact, over 75% of domestic cattle dairies are positive for C. burnetii based on bulk tank sampling, though reports of clinical disease are much lower. Unfortunately, shedding patterns are highly variable, both between individual animals and across species, though all species may shed in the absence of clinical signs and for several weeks following parturition or abortion. The highest concentrations of C. burnetii are typically found in ovine abortion products and sheep will continue to shed in the vaginal mucous and feces; however, shedding in milk is more intermittent in sheep compared to the other species; placenta or vaginal fluid rather than milk are the best samples for screening tests. In goats, C. burnetii has also been identified in vaginal secretions from nulliparous and clinically normal goats, though higher numbers of organism are shed after coxiellosis abortion. Goats will persistently shed in the milk for several weeks following parturition or abortion; bulk tank milk PCR is an excellent sample for herd screening, especially in early lactation. Infected cows are least likely of the species to abort and shedding in vaginal fluids is relatively short-term. However, even clinically healthy cows may persistently shed the bacteria in their milk through early to mid-lactation; bulk tank milk is an excellent sample for herd screening. Coxiellosis is a reportable disease in most states.

Historically, Q-fever has been treated as an occupational disease associated with livestock exposure; high-risk occupations include livestock farming, food animal veterinary medicine, slaughterhouse employees, and animal research laboratory employees. The infective dose for Q-fever is very low. Q-fever is a nationally notifiable disease, diagnosis typically triggers an investigation into potential animal contacts, and then testing of those contacts. However in recent years (2000-2010), more than half of the reported cases of Q-fever in the U.S. did not have any reported contact with livestock. Roughly half of human Q-fever cases result in asymptomatic seroconversion two to three weeks following exposure; approximately 3% of the U.S. population is seropositive. Of those with clinical disease, most experience an acute “flu-like” illness for one to two weeks, characterized by a high (104-105°F) fever, malaise, gastrointestinal signs, and coughing, which may progress to pneumonia; roughly one in four people may experience a post Q-fever fatigue syndrome following recovery from the acute disease. Rarely, chronic Q-fever or other serious complications such as hepatitis, endocarditis, or meningoencephalitis can occur, especially in individuals with heart valve replacement or other underlying risk factors. Pregnant women are generally considered to be at risk for miscarriage or pre-term delivery; although data from the recent Netherlands outbreak did not find evidence for increased obstetrical morbidity, it may depend on the bacterial strain involved.

Diagnostic testing
In all species, diagnostic testing is complicated by inconsistency between clinical infection, serologic status, and organism shedding. It is possible for infected animals to be both seronegative and/or not shedding at the time of sample collection. Titers may persist years past infection; conversely, a significant proportion (10-20%) of animas in infected herds may remain seronegative despite actively shedding the bacteria. For these reasons, serology is best utilized as a herd-level test only; neither negative serology nor negative PCR rule out infection in the individual animal but repeated negative herd tests reduce the likelihood that that the farm is infected. Herd screening tests should be biased towards the highest-risk animals – those that recently kidded, lambed, or aborted; do not include youngstock.

Serologic testing options include complement fixation, ELISA, and indirect IFAs. While complement fixation is used for regulatory export testing, the other tests are more sensitive and should be used for general herd- or flock-level diagnostic testing. Serology can be performed on bulk tank milk samples as a herd-level screening tool; a positive test indicates that herd members have
been exposed and seroconverted. As with most serologic testing, there is a time lag between infection and seroconversion; if used in the face of acute disease, paired samples should be taken two to four weeks apart. When used for herd screening at any herd size, testing thirty animals is sufficient to identify a positive herd at 10% or higher prevalence (0.05% risk of error).

Antigen detection testing options include PCR, immunohistochemistry, and histology; culture requires a Level 3 biosafety laboratory and is not generally performed for routine diagnostics. Histology and/or immunohistochemistry performed on the aborted placenta is the most reliable method of determining if *C. burnetii* is responsible for a specific abortion event. PCR assays can be run on abortion/birth products, vaginal secretions, milk, and feces to detect shedding; a positive PCR result indicates that the animal is both infected and infectious, but due to high levels of subclinical disease, infection with *C. burnetii* may be incidental to the abortion or exposure event. PCR can be performed on bulk tank milk samples as a herd-level screening tool; a positive test indicates that at least one herd member is infected and actively shedding *C. burnetii*.

**Treatment**

There are no known effective treatments to prevent infection, reduce shedding, or “clear” a positive serologic status. Parenteral long-acting tetracyclines (two injections at 20 mg/kg, given 20 days apart) may reduce the number of future abortions in a herd experiencing an acute abortion storm. Oral administration of tetracyclines in feed is ineffective as a herd-level control mechanism.

**Control**

Due to the persistent and ubiquitous nature of *C. burnetii*, our inability to differentiate uninfected animals, and our inability to definitively clear either animals or the contaminated environment, test-and-cull and depopulation schemes are not advised. Basic hygiene and biosecurity measures, including proper disposition of abortion products, is necessary to limit transmission within the herd. Although official quarantine measures are not usually implemented, common sense dictates that producers should not sell or move animals between herds during abortion storms. Vaccines that are used overseas are not available in the U.S.

**Client recommendations**

1. If coxiellosis is diagnosed in the herd or flock, advise owners to report any cases of flu-like illness in the household to their medical practitioner; clinical signs typically appear two to three weeks after exposure.

2. Limit human access to high-risk areas such as kidding and lambing pens or at high-risk times for aerosolization (e.g. cleaning out bedded packs). Limit access of high-risk individuals (immunocompromised, pregnant, or those with heart-valve replacement or disease).

3. Use personal protective equipment for high-risk activities such as kidding/lambing, handling abortion products, and working in contaminated dusty environments. This includes disposable gloves, fitted face mask respirator (N95 or higher, under consultation with a physician), eye protection, and dedicated outer clothing and boots. Follow basic hygiene protocols including washing hands with soap and water and refrain from eating, drinking, or smoking in animal areas.

4. Milk from infected animals should be pasteurized before consumption, further processing, or sale. Current pasteurization standards (145°F for 30 minutes or 165°F for 15 second) will kill *C. burnetii*.

5. Do not move, sell, or buy animals, especially pregnant animals, during an abortion storm or from producers experiencing an abortion storm. Negative tests on individual animals are not sufficient to guarantee that new purchases are uninfected but repeated herd testing can identify low-risk herds or flocks.

6. Appropriately dispose of manure, abortion materials, and deceased animals. Follow local regulations, but typical options include composting (at least 90 days), incineration, deep burial, and disposing bagged material through biohazard or landfill channels. During transport or in arid, windy climates it may help to cover the manure pile to decrease wind-driven aerosolization and spread. Implement an effective pest control program. Do not use high pressure hoses to clean high-risk areas.

7. Once established, low-risk operations should be tested at least annually, all abortions should be presented for diagnostic evaluation, and in the absence of abortion, a subset of normal placentas should be submitted for screening via PCR.

**Additional reading**


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