Parasite Control

1. What is parasite control and what does it entail?

Parasite control is based on maintaining parasite populations below which clinical signs are observed. It does not involve the complete elimination of all parasites from a herd for several reasons. A low level of parasites develops immunity in the animals, decreases drug resistance, saves money for the owner, and finally because complete elimination is impossible.

2. How do parasites cause immunity?

An animal exposed to parasites, bacteria, viruses, etc. develops specialized cells that are used to fight infections from these foreign organisms. Some produce life long immunity after a single exposure while others produce immunity for as little as several months. Low level repeated exposure to a foreign organism can stimulate the immune system to continue producing the specialized cells and thereby reduces the severity of infections in the future.

3. Why is drug resistance important?

Drug resistance to anthelmintics (anti-parasite drugs) is becoming more common. Unfortunately these drugs are unable to completely eliminate an entire population of parasites. The few that remain are resistant to the drug and with time reproduce creating a new population that is also resistant. Anthelmintic resistance is very common in sheep and goats and increasing in alpacas and llamas. Resistance is encouraged with indiscriminate use of anthelmintics, shipping animals, open herds, and inadequate biosecurity. Anthelmintics should complement but not replace good management and sanitation practices.

4. How can management and sanitation reduce the need for anthelmintics?

Management strategies include using feed bunkers and eliminating standing water and wet areas around waterers to reduce the favorable environment most parasites need to become infective. Frequent cleanup of the dung pile and pasture rotation reduces the parasite load and possible chances of exposure. Quarantine pens for all incoming animals helps reduce exposure of the existing herd to new parasite species as well as other diseases. Incoming animals are stressed from transportation, new surroundings, and removal from existing herdmates. The stress can cause a mild immunosuppression causing an increase shedding of parasites or other organisms.

We suggest that every 2 months a random sample of fecals from the herd be evaluated to assess parasite control. Concentrate on 3 general groups: crias in the first 3-4 months of life, yearlings, and adults. A sampling of 3-5 animals or 10% of the group should be adequate.
5. **What types of deworming programs are available?**

Deworming can be performed on a seasonal basis and/or as needed. Which you use will vary by your geographical location, open or closed herd, pasture – dry or irrigated, travel, stocking density, etc. Periodic fecal sampling (as described above) including fecal egg counts will provide information as to types and numbers of parasites present. You should review your particular situation with your veterinarian and in combination with fecal egg counts determine the best deworming program for your situation.

A general program for the inland Pacific Northwest would include twice a year treatments of all animals over 2-3 months old twice a year. In the fall after a killing frost, animals would be treated with an ivermectin-type product for intestinal worms, external parasites, and nose bots. Animals should be treated in the spring prior to majority of births with a fenbendazole-type product. The periodic fecal exams would determine if additional dewormings would be needed.

6. **Who to contact for more information?**

- Contact WADDL (509)335-9696 for fecal testing questions.
- Contact WSU-VTH Agriculture Animal Department, Ms. Sallie Bayly, RVT (509-335-0711) to contact a veterinarian regarding management questions.

The following information is a brief overview of the more common parasites seen in alpacas and llamas and some of the treatments available. It is strongly recommended that a veterinarian evaluate if treatment is warranted through examination of the animal or herd, fecal analyses, and fecal egg counts. Use of anthelmintics should not exclude good management and pasture sanitation programs.

**Not all available anthelmintics are listed below. Use is “off-label” as none are currently approved for use in alpacas or llamas.**

**I. PROTOZOA**

A. **Coccidia** (*Eimeria alpacae, E. lamae, E. macusaniensis, E. punoensis*)

A parasite problematic for crias <1 year old and naïve (previously unexposed) or immunosuppressed adults. There is no cross protection between species so adults can be infected and develop clinical disease from a different species. Due to developing drug resistance and the inability to completely eliminate the parasite from animals, treatment is only recommended if oocyst counts are significantly high with the presence of diarrhea.

Infection is through the fecal-oral route and can occur in as little as 4 days if oocysts are exposed in cool, moist pastures. Pasture management is a key factor to reduce exposure to susceptible animals. The oocysts die in warm, dry pasture in 20-30 days but can persist for years in cool, damp environments. The prepatent period (time from ingestion of the oocyst to shedding in feces) is variable among species but ranges from 10 days for *E. punoensis* to 33 days for *E. macusaniensis*.

Oocysts cause diarrhea by damaging intestinal cells. After anthelmintic treatment is finished, feces may remain loose until the intestinal lining is repaired. In severe infections, stunting or ill-thrift with continued diarrhea may occur due to permanent damage to the intestinal lining. Contact your veterinarian if severe diarrhea occurs since dehydration can rapidly lead to death especially in warmer weather conditions.
Treatments Available:
Amprolium (Corid®)
5 day treatment or a 21 day prevention program. There are several formulations available and dosage and delivery vary with each. The product can be used for treating individual animals or via water delivery for herd situations. It is best to treat individual animals in face of outbreak as crias may not drink the water and not all animals will receive the same dose.

Sulfadimethoxine (Albon®)
Available in both liquid and tablets for individual animal treatment.

Decoquinate (Deccox®)
A preventative product. Add to feed of pregnant females 1 month prior to parturition or when moved onto clean pasture prior to parturition. It can be added to creep feed provided to crias to reduce oocyst production. If coccidiosis has been a problem in a herd, decoquinate should be fed to all growing animals on a daily basis.

B. Cryptosporidium (Cryptosporidium sp.)
Rarely seen but can cause diarrhea in young camelids, especially less than 30 days old. This is zoonotic so humans can get it too! Use good hygiene/sanitation since no treatments are available. Keep the animal dry and well hydrated until the parasite runs its course. Prepatent period is 3-7 days.

II. NEMATODES (ROUND WORMS)
A. Strongyles (Cooperia, Haemonchus, Oesophagostomum, Ostertagia, Trichostrongylus)
There are many types of “strongyles” that cannot be differentiated by egg shape alone. Fortunately the treatment is similar for all. These parasites can cause stunting, weight loss, and diarrhea especially in juvenile animals.

B. Whipworms (Trichuris tenuis)
This parasite causes poor growth, diarrhea, and blood loss. It can severely debilitate crias. This parasite is somewhat resistant to the ivermectin-type products but the benzimidazoles such as fenbendazole and albendazole are effective at the high end of the dose range. This parasite requires 3 weeks in the environment to become infective and is very difficult to remove once present. The prepatent period is unknown.

C. Nematodirus battus, N. helvetianus
Nematodirus spp. eggs are approximately 2 times the size of strongyle eggs. The parasite is a low egg producer so any eggs present indicates a significant infection and should be treated. This parasite can cause poor growth and diarrhea especially in crias. Use the high end dose range.

D. Parelaphostrongylus tenuis
Commonly called “meningeal worm”. The parasite is found in white-tailed deer and requires a snail to continue the life cycle. When camelids and other animals eat an infected snail, the parasite migrates through the animal and penetrate the spinal cord causing paralysis 50-60 days after ingestion death soon after. This parasite is prevalent in the eastern United States but is currently not present in the western states. Prevention includes deer-proof fencing, snail-eating fowl, and the use of ivermectin products every 30 days.
Treatments Available:
Benzimidazoles:
Increasing parasite resistance to many of these products especially in sheep and goats and also in areas of the country with favorable parasite environments.

Products available:
   Fenbendazole (Panacur®, Safe-guard®)
   Albendazole (Valbazen® - not recommended in pregnant animals)

Avermectins:
   Effective against many internal and external parasites. Reduced efficacy with Trichuris.

   Products available in oral or injectable forms:
      Ivermectin (Ivomec® and many others)
      Doramectin (Dectomax®)
      Moxidectin (Cydectin®, Quest®)

   Levamisole (Levasole®, Tramisol®)
      Not recommended in lactating animals.

Pyrantel-Pamoate (Strongid-T®)

III. CESTODES (TAPEWORMS)
Cestodes are rare in camelids at this time.

IV. TREMATODES (LIVER FLUKES)
A. Fasciola hepatica, Fascioloides magna

   Requires a snail as an intermediate host so only at risk in wet areas and the presence of snails. Camelids are good definitive hosts for Fasciola hepatica and pass eggs in their feces when infected. It takes approximately 10-12 weeks after infection before eggs can be detected in the feces. This parasite can cause severe liver damage. Animals are often infected when co-pastured with infected cattle, sheep, or goats.

   Camelids are an aberrant host for Fascioloides magna so eggs are not produced and therefore not detected on fecal examinations. Infection is possible in areas that have infected deer and elk.

   Treatment for either type of liver fluke includes ivermectin-clorsulon combination products or albendazole in non-pregnant animals. Limiting co-mingling with infected domestic ruminants, limiting deer and elk access, and use of snail-eating fowl can reduce exposure.

V. LICE
Lice are commonly seen in the winter months. If the animal has a severe infection, you may see areas of fiber loss from chewing or rubbing as the animal tends to be itchy. Poor growth may be seen as well. These parasites can often be found by parting the fiber along the dorsal midline or rump and looking at the skin for movement at the base of the fiber. Lice are 2-4 mm in length. Sucking lice can be treated with oral or injectable avermectin products. Biting lice are more successfully treated with insecticidal dustings. Treat all animals in a group or they can re-infect each other. Repeat in 3 weeks if using an insecticidal dusting product. Pour-on products do not work well due to the heavy fiber limiting contact and absorption through the skin.

   Lice are species-specific so you cannot get infections from your animals.
VI. Mites (aka. Mange, Scabies)
The prevalence of Sarcoptes, Psoroptes, Chorioptes have decreased with the increased use of ivermectin. Animals with infections will often have areas of alopecia on the face, neck, and perineum. The areas are also itchy. Treat all animals in a group at the same time with injectable or oral avermectin products. Repeat in 2 weeks. These are reportable diseases in some states but not Washington.

VII. Ticks
Ear ticks can cause secondary ear infections, itching, drooping ears. Treatment involves cleaning the ear and use of ivermectin products both systemic and topical in the ear. Other ticks are difficult to detect due to the fiber. In cases of suspected tick paralysis, a thorough examination is required which includes close clipping of the fiber. Treatment is with ivermectin products.

References
3. Foreyt WJ, Jasmer DP. An Illustrated Outline for Veterinary Parasitology (VM537). Washington State University, Department of Veterinary Microbiology and pathology; 2003.