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INTERNAL & EXTERNAL PARASITE PROBLEMS OF SOUTH AMERICAN CAMELIDS

In general, camelids are susceptible to the same gastrointestinal parasites as sheep – strongyles such as *haemonchus* and *ostertagia, nematodirus,* whipworms (*trichuris*), and tapeworms less commonly. They are also susceptible to coccidiosis and liver fluke. There are a few other host-specific gastrointestinal nematodes affecting camelids in South America, such as *Lamanema chavezi* and *Graphinema auchenia* but these have not been reported outside South America to the author's knowledge. *Lamanema chavezi* larvae migrate to the liver where they undergo development into L4 larvae before migrating to the bile ducts and back to the intestine. This parasite may account for some of the evidence of parasite migration found within the liver of some necropsied llamas and alpacas that have been exported from South America.

Camelids are not evolutionarily adapted to deal with high levels of gastrointestinal parasites. In the South American Andes, they are grazed under very extensive conditions and the climate is not optimal for parasite persistence on the pasture. As such, it appears that their innate immunity to parasitism is less than sheep. When camelids are kept in temperate climates and under more intensive conditions, they may suffer from clinical disease at levels of infestation that are much lower than you would expect to be clinically significant in sheep.

Parasitic gastroenteritis may result in a variety of clinical signs including ill-thrift, weight-loss, diarrhoea, colic, anaemia, lethargy, anorexia and death. Given the arid climate adaptation of camelids having an extra long spiral colon allowing greater water retention, camelids affected with parasitism often have normal stool. Heavy parasite burdens, particularly where *Haemonchus* is involved, may result in severe anaemia and blood transfusions are frequently required along with treatment of the underlying parasite burden.

Detection of gastrointestinal parasites is achieved by examination of faecal samples for the presence of parasite eggs. Most widely available tests, particularly passive floats, are not sensitive enough for detection of low egg counts. Modified techniques that include centrifugation of samples greatly enhance the sensitivity of the testing procedure, especially for the larger eggs such as *Nematodirus* and more dense eggs such as Trichuris which not only are significant at low egg counts but also are harder to float. Centrifugation as well as the type of float solution used greatly enhances the ability to float these eggs – a concentrated sugar solution with a specific gravity of around 1.028 is the best medium for consistently floating these eggs. The Modified Stoll's test is the test most used by camelid veterinarians.

Development of appropriate farm-specific parasite control strategies are vital for control of gastrointestinal parasitism. These must include pasture management, body condition scoring, checking of pallor and faecal screening for routine monitoring, use of anthelmintics and anticoccidials where required and quarantine measures for incoming stock. Drugs must be given by the correct route and used at the correct dosages based on pharmacokinetic studies: furthermore, if targeted treatment cannot be employed, farms must make use of the refugia concept to slow the development of resistance. Blanket use of anthelmintic agents is not recommended. Removal of faeces from pasture prior to egg hatching is the most effective way to minimise parasite problems and use of anthelmintic agents and should be encouraged.

Coccidiosis is most often a disease affecting youngstock although immunosuppressed adults can also become clinically affected. *Eimeria macusaniensis* and E *ivitaensis* have been associated with significant pathogenicity. During the long pre-patent period of "*E mac*" of 33-34 days, it is capable of causing significant damage to the bowel wall when burdens are high. This may result in acute weight loss and hypoproteinaemia (due to hypoalbuminaemia), normally without changes in PCV, and prior to the appearance of oocysts in faecal samples. Therefore, *E mac* should be a top differential diagnosis in camelids exhibiting weight loss and hypoproteinaemia despite normal faecal results.

Liver fluke causes severe clinical disease in camelids. *Fasciola hepatica* is widespread in wetter parts of the UK and the Pacific Northwest of the US while *Dicrocoelium dentriticum* has been reported in mountainous regions such as Switzerland. There are reports of acute, chronic and fatal forms of fascioliasis. Clinical signs of disease are vague and include reduced appetite, generalised weakness, recumbency and

anaemia. A number of cases have also been diagnosed with mural endocarditis: it is uncertain whether the problem in the liver is predisposing these animals to sepsis and subsequent infections on the heart valves.

The predominant ectoparasite problems are sarcoptic and chorioptic mange. Lice, dermatophilosis and dermatophytosis also occur. The distribution of mange is similar regardless of mite type although the lesions do have some characteristic differences and can be differentiated by evaluation of skin scrapes. Treatment involves combination therapy with the emphasis on topical bathing and acaricide use. Only sarcoptic mange will respond to a course of parenteral ivermectin. Treatment of asymptomatic in-contact animals and environmental management are key to resolution of clinical signs while zinc supplementation may be a useful adjunctive therapy.