# Eimeria macusaniensis Infection in New World Camelids

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### **Abstract**

Eimeria macusaniensis is an important coccidian intestinal parasite of New World camelids. It may be the same parasite as *E. camelli*, a similar-appearing intestinal coccidian of Old World camels, but this has not been proven. \**E. macusaniensis* affects all ages of camelid, not just juveniles. Its contribution to illness may not be appreciated, since some camelids become sick up to two weeks before patent infections are detected in their herdmates. Gross lesions are often absent, and histologic lesions can be so segmental that they are missed on routine necropsy.

#### Résumé

Eimeria macusaniensis est un parasite intestinal coccidien important des camélidés du Nouveau Monde. Il pourrait être le même parasite que *E. camelli*, un coccidien intestinal d'apparence semblable des chameaux de l'Ancien Monde, mais ceci n'a pas été prouvé. *E. macusaniensis* affecte les camélidés de tous les âges, et non pas uniquement les juvéniles. Sa contribution à la maladie peut ne pas être prise en compte, étant donné que certains camélidés tombent malades jusqu'à deux semaines avant la détection d'infections patentes chez les autres membres du troupeau. Souvent, il n'y a pas de lésions macroscopiques, et les lésions histologiques peuvent être si segmentaires que même une autopsie régulière ne les détecte pas.

## Introduction

Eimeria macusaniensis is a protozoan parasite of New World camelids. It was first reported in South America in 1978 and in the US in 1988. Early North American reports indicated a low prevalence (<2%) of shedding in healthy camelids. A later study, using a different method of fecal analysis, indicated a higher prevalence in the midwestern states, with up to 22% of camelids under one year old shedding the organism, and about half as many older camelids. In general, these reports suggested that the amount of fecal shedding was low, and did not attribute much pathology to this organism. In most case reports, E. macusaniensis is de-

scribed as a co-infector, usually with other *Eimeria* spp, and the specific contribution to disease caused by this parasite is unclear.

 $E.\ macusaniensis$  is easily recognizable by its size, both in the feces and in intestinal sections. Oocysts are three to four times larger than other Eimeria (90 x 65  $\mu$ ), stain more darkly and are more teardrop-shaped. It is very similar to  $E.\ camelli$  in Old World camelids and various individuals have hypothesized the two parasites are the same, but confirmatory tests have not been done.

Recent work from Germany has elucidated the *E. macusaniensis*' life cycle. Following shedding in the feces of an infected camelid, oocysts sporulate in two to three weeks (faster under higher temperatures). After ingestion of sporulated oocysts by animals, several multiplicative rounds of asexual reproduction occur, which damage the jejunal, and to a lesser degree, ileal mucosa. Eventually sexual reproduction occurs and unsporulated oocysts appear in the feces. The prepatent period is 32 to 36 days, and the patent period is 39 to 43 days. Reinfestation results in a longer prepatent period (37 to 40 days), shorter patent period (about three weeks) and lower oocyst output. These data suggest that acquired immunity plays a role in decreasing output in older, exposed camelids.

#### **Clinical Disease**

Although most reports stress that younger camelids shed more oocysts than older ones, and a report from Peru describes the disease only in young crias, one early North American report acknowledges the presence of significant postmortem lesions in an adult.<sup>3</sup> Our experiences at Oregon State University suggest that all ages of camelid are susceptible to severe infestations. Whether this means that camelids do not develop immunity to this coccidia as readily as other coccidia (unlikely), that exposure is not as widespread thereby allowing older animals to remain naive (possible), that stressors significantly affect immunity to this coccidia in adults (possible), or some other factor, is unknown.

In addition to the propensity to affect older camelids, *E. macusaniensis* infestation sets itself apart from other coccidial infections in lack of signs referable to the GI tract, frequency of signs not directly referable

to the GI tract and frequency of prepatent disease. It is one of the most common causes of weakness, weight loss, hypoproteinemia, or ill thrift in our area. Severe disease and death appear possible within three weeks of initial exposure and two weeks before establishment of patency. Diarrhea and colic are seen in a minority of cases.

# **Diagnostic Testing**

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 GI obstruction. Thickened bowel walls are rare (<10% of cases).

Fecal flotation remains the only ante-mortem test. Prevalence of prepatent infections lessens the value of fecal analysis, and treatment decisions often are made in the face of a negative fecal. Histopathologic samples or impression smears made of affected gut may be diagnostic in postmortem cases or at surgery. Organisms appear to be found most readily in the distal jejunum and ileum. Occasionally there are gross intestinal changes, such as thickening and a cobblestone appearance to the serosa or small white plaque-like lesions on the mucosa.

## **Treatment and Control**

If the camelid has either a confirmed or suspected infestation, treatment may be warranted. The most com-

monly used anticoccidial medications for small ruminants and camelids in North America are amprolium and sulfa antibiotics. Both are more effective against immature forms of the parasite and should not be expected to immediately reduce fecal shedding. Treatment during the prepatent period does appear to reduce subsequent shedding. In countries where they are available, benzeacetonitrile compounds may be an effective alternative. Many affected camelids also require plasma transfusions and antibiotic coverage.

Herd control is a tricky issue. Disease in younger animals should be treated similarly to other forms of coccidiosis. Some degree of shedding may be normal or clinically insignificant prior to development of immunity. In older animals the amount of shedding should be lower, but a complete lack of shedding cannot be expected. Signs of disease, protein loss, diarrhea, or weakness support treatment in herds with known or suspected E. macusaniensis infestation. Other management changes to reduce stress and overcrowding, and clean up the environment, are necessary. Herd outbreaks are uncommon, emphasizing the role of immunocompromise in severe infestations, but have been seen when groups of naive camelids have been brought to farms with endemic problems. Spot fecals may be helpful in assessing 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.

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