MENINGEAL WORM LITERATURE REVIEW WITH IMPLICATIONS FOR ALPACA OWNERS

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MENINGEAL WORM: AN INTRODUCTION

Meningeal worm (*Parelaphostrongylus tenuis*) is a parasite of special concern for many alpaca farmers. Carried by white-tailed deer (*Odocoileus virginianus*) and intermediate slug and snail hosts, "m-worm", as it is commonly known, lives and reproduces in the deer. Although the deer are generally not affected by the parasite, any other animal ingesting an infected slug or snail is usually killed by the activity of the parasite as it travels through the nervous system.

Preventing infection in our alpacas is a critical part of husbandry for alpaca breeders anywhere white-tailed deer are prevalent, as prevention using monthly injections of avermectins is easy – but a cure is often impossible. Even when an infected animal's life can be saved, lingering neurological deficits are common.

LIFE CYCLE

Parelaphostrongylus tenuis is a common parasite of white-tailed deer (WTD) and white-tailed deer only. Even other cervids such as mule deer, fallow deer, moose and elk are aberrant hosts for the parasite, which has been a factor affecting the reintroduction of elk to the east coast [Bender, et al., 2005; Larkin et al., 2003]. The expansion of meningeal worm into historic caribou ranges is considered to be the primary factor causing the decline of caribou in areas where ample suitable habitat remains [Anderson, 1971].

The life cycle of the parasite has been long studied and is generally well known [Anderson, 1972]: WTD harbor adult worms, usually only a breeding pair, in the subdural spaces of the brain. The adult parasites either lay eggs on the dura matter of the brain or deposit them directly into the circulatory system. In the first case, young larvae hatch and penetrate small blood vessels of the brain in which they are carried by the bloodstream into capillaries within the lungs. Those eggs laid directly into the circulatory system are caught up in the capillary structure of the lungs, where the larvae hatch. The L1 larvae in the lungs migrate into the bronchioles and are coughed up, or else they migrate directly into the throat. In both cases they are swallowed by the deer, pass into the digestive system and ultimately out in the feces.

WTD feces have a mucous coating that many species of slugs and snails find appetizing, and the meningeal worm larvae are found in this coating. As slugs and snails pass over the infected deer feces feeding, the L1 larvae burrow into the feet of the gastropods. Although some species of aquatic snails have been experimentally infected with m-worm, no infected snails have been collected from aquatic environments. It is hypothesized that the mucous coating containing the larvae breaks down too rapidly in water for the larvae

to be easily found and consumed by mollusks, as the larvae are washed away when the coating dissolves [Anderson, 1972; Lankester and Anderson, 1968].

Once within the slugs and snails, the L1 larvae continue to develop over the course of several weeks into L3 larvae, which are infective to deer and other mammals. It is believed that L3 larvae can live as long as their gastropod hosts, and was previously thought that survival outside the intermediate host was brief [Anderson, 2002]. However, researchers interested in the interactions between meningeal worm and its gastropod hosts recently observed that at least some of the L3 larvae leave their hosts as they mature, and are deposited in slime trails and on vegetation [Duffy, 2006]. Larvae maintained in 40°F tap water remained viable for up to a year; 80% survived for one month and roughly 25% for six months. Some larvae survived dry at room temperature for 24 hours (extended periods were apparently not investigated), while others survived freezing on lettuce leaves at -4°F for up to a week (again, lengthier periods were presumably not investigated). Although the research raises as many questions as it answers, it is clear that moist vegetation may harbor infective larvae.

If a gastropod infected with L3 larvae is ingested by a WTD, the normal life cycle of the parasite continues, with the larvae entering the bloodstream through the digestive tract walls and from there migrating into the central nervous system (CNS) of the host. In WTD, the larvae spend only a short time within the spinal cord itself before moving into the space surrounding the cord and migrating into the subdural regions of the brain where they mature into adults.

For unknown reasons, in species other than WTD, the meningeal worm does not reach adulthood nor reproduce; it is possible that some chemical signal is missing that triggers these events in the WTD. The larvae continue to migrate throughout the spinal cord and nervous system, causing lesions and other damage to the CNS. Although WTD sustain some damage during the migration of the m-worm larvae, their neural parenchyma apparently regenerates quickly, with no lasting signs of the worms' presence [Ekroade, et al., 1970]. Other species sustain more extensive damage that is not repaired.

DISTRIBUTION

Given that meningeal worm is a common parasite of WTD, it is possible for meningeal worm to exist anywhere WTD and appropriate secondary gastropod hosts are found. A WTD range map (www.whitetailsunlimited.com/i/p/bk_distribution.pdf) shows that white-tailed deer are more widespread than commonly realized, with recognized subspecies inhabiting almost the entire North American continent. Thankfully, not all subspecies appear to be infected by meningeal worm at the same rate as the common eastern subspecies, and indeed, many subspecies are uncommon and seldom encountered.

For these reasons, meningeal worm has historically not been considered a parasite of concern west of the Mississippi. Likewise, the parasite is less common in the coastal plains of the southeastern United States, where habitat for the intermediate hosts is more widely distributed and difficult to find. Although the parasite occurs there, its distribution is patchy and tied closely to appropriate secondary host habitat.

A study of *P. tenuis* infection of WTD in Oklahoma showed that the parasite was common in and near wooded areas, while grassland areas showed little infection [Kocan et al., 1982]. Alpaca farms located in grassland areas may have less of a problem than areas where woodland tracts are prevalent. However, the authors of the study noted that suitable hosts were available even in areas where meningeal worm was not detected. The open grasslands of Manitoba have apparently historically served as a barrier to the spread of meningeal worm, but there is concern that aspen parklands, particularly in association with human habitation, may permit the translocation of the parasite to the western areas of North America [Anderson, 1972].

In addition, translocation of infected deer has apparently carried meningeal worm to some areas where the parasite had not historically been found [Kocan, et al., 1982]. In short, presence or absence of meningeal worm depends on a number of factors, including human propensity for relocating livestock and wildlife, and historical absence of the parasite is not a guarantee of continued absence.

Pertinently, in most areas studied, WTD numbers are increasing, and eastern subspecies carrying meningeal worm are moving west. Given that meningeal worm causes mortality in other cervid species, tracking the spread of the parasite has been of great concern to fish and wildlife organizations [see for example Whitlaw and Lankester, 1994; Lenarz, 2009]. Numerous studies of hunter killed WTD have been carried out, and the spread of P. tenuis has been documented by actual observation of the adult worms in the meninges of dissected WTD.

In the United States, meningeal worm has long been recognized as a parasite in the northeastern regions. By the 1960s, meningeal worm was well documented in many southeastern states as well, including Alabama, Arkansas, Florida, Georgia, Kentucky, Louisiana, Maryland, Mississippi, North Carolina, Tennessee, Virginia and West Virginia. One 1969 study found almost 50% of deer examined to be infected [Prestwood and Smith, 1968]. Oklahoma WTD populations showed evidence of infection by the 1970s [Carpenter, et al., 1972]. Meningeal worm has reached parts of Texas within the past several years, as evidenced by an outbreak on an alpaca farm in Hempstead, Texas (http://www.texasalpacaranchers.org/health-alerts/meningial-worm).

In more northerly regions, m-worm has been found west of the Mississippi in North Dakota, South Dakota, and western Nebraska [Oates, et al., 2000]. Meningeal worm was found in WTD in Manitoba [Bindernagel and Anderson, 1972] as early as the 1970s, and

had reached eastern Saskatchewan by the early 2000s [Wasel, et al., 2003]. This appears to be the current westward limit of meningeal worm range at this time, but continued westward spread of the parasite it likely.

VECTOR CONTROL

As discussed, meningeal worm passes through two types of obligate host during its life cycle: white-tailed deer and gastropods. Limiting a herd's exposure to meningeal worm therefore depends on management of both vectors. Keeping WTD out of alpaca fields and away from their general area is a good start at limiting the prevalence of meningeal worm, but does not eliminate the risk of infected gastropods entering pastures.

Controlling WTD

WTD are a common sight in most areas of the east coast. They thrive in the patchy habitat created by human activity, and in many areas, their only remaining predator is the automobile. Deer have grown accustomed to living in close proximity to humans and their habitations, and very little will deter a WTD determined to eat your garden or nibble on your shrubbery. The fact that many people actively feed deer only exacerbates the problem.

WTD are excellent jumpers, and can clear most typical farm fencing with ease. Electric fencing may work as a deterrent, but only if the deer are first lured into being shocked. Tying aluminum strips coated with peanut butter to the fence (or coating electric polytape with peanut butter) or using commercially available lure tapes may tempt the deer to touch the fence and be shocked. Some specialists advise first using lures on an electric fence, and then switching to repellents for maximum effectiveness.

Creating an open zone around your pastures may also help reduce their attractiveness to deer. WTD prefer to forage on the edges of fields, and may be reluctant to cross large open areas. As a bonus, this also reduces the likelihood of gastropods migrating into your fields from the areas frequented by deer. In addition, reducing the browse available to your alpacas may further reduce their risk of contracting meningeal worm (discussed below).

One of the best ways to keep WTD out of your fields is to use livestock guard dogs (LGDs), any of several breeds specifically developed to live with livestock animals such as alpacas and to protect them from threats. Great Pyrenees, Maremmas and Anatolian Shepherds are three breeds commonly used by alpaca owners to protect their herds from predators such as coyote and bear. As a bonus, WTD typically stay far from areas patrolled by these dogs.

Gastropod distribution and ecology

Even if deer are not regularly present in your fields, you may still be at risk for meningeal worm, as the proximate source of infection lies with the gastropods that carry the L3 larvae. Dozens of common slug and snail species have been shown to carry infective larvae, and it is likely that more will be found [Maze and Johnstone, 1986; Rowley, et al., 1987; Platt, 1989; McCoy and Nudds, 1997] Meningeal worm larvae are not dangerous to alpacas unless they have matured to the L3 stage in a gastropod; consumption of L1 larvae in deer feces will not result in infection.

Although relatively few individual gastropods are infected by *P. tenuis*, infection rates in WTD remain high, and it is suspected that infection with meningeal worm may alter the behavior of the host mollusks to make it more likely that they will be consumed by WTD [McCoy and Nudds, 2000]. Other parasites such as the common lancet liver fluke Dicrocoelium dendriticum are known to alter their host's behavior.

Adult flukes live in the bile ducts of herbivores, typically ruminants (including camelids). The eggs laid by the adults pass from the bile ducts into the intestines and then into their hosts' feces, from which they are ingested by snails. Over three to four months the eggs mature into cercaria in the snails' respiratory systems. At maturity, they are expelled in balls of slime that are in turn eaten by ants. The cercaria migrate to the ants' brains and abdomen, and cause significant behavioral changes: "ants which normally move into their nests with cold temperatures will move up to the tops of vegetation. The affected insects clamp their jaws onto the plant and remain paralyzed as long as the temperature is below 20°C" [Peacock, 2004]. The ants are therefore much more likely to be consumed by the grazing animals required as the parasite's next host.

While most m-worm studies have concentrated on those mollusks collected in ground surveys, several researchers have discussed the frequent arboreal climbing behavior of many of the host slug and snail species [McCoy and Nudds, 1997]. It is theorized that infected mollusks may climb more openly than uninfected, making them more likely to be eaten by browsing deer. This may be a factor explaining why meningeal worm infection anecdotally appears to be more prevalent in llamas than alpacas. Llamas typically browse more like WTD, and thus may be exposed to more potentially infective slugs and snails. Preventing alpacas and llamas from browsing may help reduce their risk of meningeal worm infection.

Several studies have shown that wintering yards for deer show a much higher rate of meningeal worm infection in the resident mollusks than do grazing areas [Lankester and Peterson, 1996; Slomke, et al., 1995]. It appears that most deer are infected within their first year, many presumably on the wintering grounds, and apparently are typically not reinfected by newer worms after this initial infection [Duffy, et al., 2002; Slomke, et al., 1995]. Eliminating deer wintering areas from near an alpaca farm may significantly

reduce the density of infected mollusks present.

Typical overwinter larval survival in the most northernmost regions of WTD distribution appears to be only in the <25% range, yet approximately three quarters of the deer in those regions are infected. This would appear to support the supposition that most northern deer are infected on their wintering grounds in the fall or else during early spring [Forrester and Lankester, 1998], or that slugs and snails carrying L3 larvae alter their behavior to increase their chances of being consumed by WTD. Despite the low survival rates noted, meningeal worm larvae in deer feces can survive prolonged exposure to temperatures as low as zero degrees Fahrenheit, as well as desiccation of the feces.

Little or no information is available regarding *P. tenuis* infection in southern deer populations and the mechanisms by which they are infected. However, given that temperatures in these areas favor year round mobility of gastropod populations, it may be that grazing areas provide enough opportunities for infection throughout the year without the need for concentrated populations of infected gastropods. The lack of dense concentrations of the slugs and snails in wintering grounds, as well as the paucity of appropriate habitat, may also serve to explain why southern populations of WTD are less frequently infected with m-worm than their northern counterparts.

Controlling gastropods

Given that the majority of the infected gastropods appear to be located in or near deer wintering grounds (in regions where such grounds exist), the overall likelihood of an alpaca encountering an infected slug or snail is relatively low. Alpaca breeders can further reduce the numbers of gastropods found in their pastures by taking a number of measures, from simple to complex.

One method that has been advocated to alpaca breeders is creating physical barriers around fields, given that slugs and snails will avoid crossing surfaces that are potentially harmful. Although gravel is often recommended, coarse gravel actually provides hiding places for slugs and snails by providing shade and trapping water, and is not an adequate deterrent. More effective barriers include bands of diatomaceous earth, lime or gypsum, all of which have desiccant and/or caustic properties [Firpo, 1997]. These barriers need frequent replenishment, however, and are likely to be washed away in rainy weather. Copper and other metals have shown mixed results in deterring gastropods, and are unlikely to be practical on the scale required. There are commercial slug fences available, but these are again impractical for large-scale use.

Making existing landscape features more of a barrier to gastropods may be easier and less expensive to accomplish. Keeping a 10’ or wider boundary of close-cropped vegetation around fields makes it less likely that gastropods will cross into pastures, as short grass

traps less moisture at the ground level, making mollusk travel more difficult. Draining moist areas, removing leaf litter and similar slug habitats near fences will also decrease the numbers of gastropods active around fields.

Many types of poultry are active slug predators, notably Muscovy and runner ducks as well as guinea fowl, which are also avid consumers of ticks. Benefits of keeping free-range poultry include pest disposal, free eggs and some weed control; they also are an endless source of amusement for cria! Negatives may include noise, mess and potentially irritated neighbors. If poultry are not practical, wild predators such as toads, frogs, shrews and many birds can be encouraged to patrol the periphery of pastures by avoiding the use of chemicals.

There are commercial molluscides available for sale. Some are granulated or pelleted baits, while others are spray type applications. These products should be viewed as a last resort. They are generally toxic to more than gastropods, and they must be used frequently to be effective. The most common bait ingredient, metaldehyde, is a known neurotoxicant in mammals; as little as one teaspoon of a bait containing 2% metaldehyde can kill a ten-pound dog [Dolder, 2003]. Considering that grain or bran is a common ingredient in slug baits, it is not unlikely that alpacas or other livestock would ingest the bait if they encountered it. For the health and safety of non-target animals, it is wise to consider non-toxic gastropod controls first.

MENINGEAL WORM PREVENTION

Preventing meningeal worm in an alpaca herd requires both risk assessment and management. The only absolute way to ensure that a herd is not infected with meningeal worm is to adhere to a strict protocol of monthly avermectin injections, making sure that each animal is properly weighed, the dosage properly calculated and that the entire dose goes into the animal. It is potentially possible to attain very good levels of protection using an integrated system of preventative measures to minimize exposure rather than relying on avermectins, but each owner must determine what level of risk is acceptable to them.

How avermectins prevent meningeal worm infection

Ivomec (ivermectin) and Dectomax (doramectin) are popular brand names for two types of avermectins, a class of macrocyclic lactone derivatives discovered in the 1970s to have then revolutionary effects on parasitic nematodes [Geary, 2005]. Avermectins paralyze both the body wall (somatic) as well as the pharyngeal muscles of nematodes by binding to GABA receptors; larvae are rendered both immobile and unable to feed [Geary, 2005].

Avermectins act retroactively by killing larvae already present in the bloodstream during their migration to the central nervous system. In alpacas, their effects at therapeutic levels

peak relatively rapidly, and protection does not persist for extended periods of time as was previously thought. For this reason, dosing intervals have been changed from earlier recommendations. It should be noted that body fat binds avermectins, and in experiments in swine, it has been demonstrated that blood concentrations in fat animals peak more slowly and at lower concentrations [Craven, et al., 2002]. This may be a factor that should be investigated in alpacas, since so many are overconditioned.

Ivomec injected at a rate of one cc per 70 pounds or Dectomax at a rate of one cc per 60 pounds, every 28-32 days, has been shown to reliably prevent meningeal worm infection. A cria should receive its first dose at no more than 30 days old to counteract any parasites it may have picked up while mouthing objects and mimicking adult behaviors. There appears to be little or no risk to developing fetuses from Ivomec or Dectomax [Boxsel, 1998], and it is generally recommended that monthly shots be given regardless of a dam's state of pregnancy.

Once larvae have reached the central nervous system, they are no longer vulnerable to avermectins, and the animal must be treated with a different protocol (outlined under Meningeal Worm Treatment, below) [Anderson, 2002]. Avermectins do not typically cross an intact blood-brain barrier; indeed, there is the potential for toxicity and death in an animal with advanced meningeal worm infection if avermectins cross a porous blood-brain barrier into the CNS where GABA receptors are located. For this reason, avermectins should not be used for meningeal worm treatment [Van Amstel, et al., 2009].

Preventing meningeal worm infection without avermectin use

There are many farms, particularly those using organic methods, that are reluctant to dose each animal with chemical dewormers every month. These farms may opt to substantially lower, but not necessarily eliminate, their risk by limiting the exposure of their animals to potentially infected gastropods, as discussed above under vector control. While no one method is foolproof, when taken together, physical means of gastropod population control may reduce risk significantly enough for farms to be comfortable monitoring their herds for signs of infection and only acting to treat if infection is presumed. Other farms may elect to use avermectins, but only during seasons of peak gastropod activity.

Although avermectins are relatively safe drugs for the alpacas themselves, they can have significant impacts on non-target species. While annelid worms do not appear to be adversely affected by avermectin residues in manure, dung beetle larvae and young adults are both killed by avermectin passed in the feces [Strong, 1992; Ridsdill-Smith TJ, 1993]. Manure decomposition in fields housing cattle treated using avermectins was found to be significantly delayed. Other non-target arthropods and insects have also been shown to be affected, and the cumulative effects of agricultural use of avermectins may be significant. For this reason alone, some individuals may prefer to avoid their use.

Farmers confident in their ability to recognize meningeal worm infection early may find that a system of risk management through vector control is comfortable for them. However, it should be noted that llamas appear to be much more susceptible to meningeal worm infection than sheep and goats, which are in turn more susceptible than cattle [Nagy, 2004]; as few as five infective larvae are sufficient to produce death in a llama, while more than 300 are typically required in sheep [Pybus, et al., 1996]. A single mollusk may easily contain five L3 larvae, making the task of vector control that much more daunting. It is not known how many infective larvae can be found on vegetation outside of their gastropod hosts.

*Can meningeal worm and other parasites develop resistance to avermectin*s?

One of the reasons often given for not dosing year around with avermectins is concern that meningeal worm may develop resistance to the wormer. Likewise, it is feared that common gut parasites will develop resistance to avermectins. It has been argued that rotating wormers or using wormers only periodically through the year will prevent both scenarios.

Unfortunately, the second scenario, that of common internal parasites becoming resistant to avermectins, has already happened [Pritchard, 1994; Kaplan, 2004]. There are literally hundreds of scholarly articles available detailing the prevalence of resistance to avermectins in many internal parasites, most pertinently Haemonchus contortus for alpaca owners. Although routine dosing for meningeal worm in alpacas may have contributed to the problem of resistance, many parasite populations had developed resistance before alpacas became common. Given its amazing efficacy when first introduced, Ivermectin was routinely used as a dewormer on a regular basis by many livestock, particularly small ruminant, producers, a practice that rapidly leads to the development of resistant populations.

Given that resistance to avermectins already exists widely in internal parasites and avermectins are therefore of little use in their treatment, there is little reason to reduce the efficacy of avermectins in meningeal worm prevention by avoiding their use. In other words, avermectins are already compromised for other anthelmintic uses in areas where WTD are endemic and avermectins are routinely used for meningeal worm prevention. Avermectins remain valuable for meningeal worm prevention and should be used for this purpose as effectively as possible.

Due to the nature of meningeal worm infection in alpacas, the parasite cannot develop resistance to avermectins in camelid hosts. The worm does not reproduce in aberrant hosts, so there is no mechanism to pass resistance on to a new generation of parasites, even if an individual worm were not susceptible to the medication. The use of avermectins to prevent meningeal worm infection in their herds will not create resistance in *P. tenuis*.

However, the development of avermectin resistance in meningeal worm living in WTD is theoretically possible. Some deer farms use avermectins to control external parasites, and the potential exists for meningeal worms to be selected for avermectin resistance inadvertently. Given the life cycle of the parasite in the deer, however, this seems unlikely. The parasites spend almost their entire life span within the brain, where avermectins do not penetrate. They are only exposed to avermectins during their initial migration to the CNS, and only a single pair of worms is resident in each deer.

Thankfully, avermectin use appears to be sporadic in deer husbandry, and the majority of farms in North America do not raise WTD. So although the hypothetical possibility of resistance developing exists, it appears that an incredible chain of events would be required. At this time, no reports of avermectin resistant P. tenuis could be found in the literature.

SYMPTOMS OF INFECTION

Symptoms of meningeal worm infection are often initially quite subtle, beginning with mild ataxia, poor coordination and subtle behavioral changes. An owner may often sense that something "just isn't right" about an animal before the classic symptom of rear motor dysfunction develops. At this stage, owners note poor coordination, difficulty walking and a reluctance or inability to rise. If the disease has progressed to the point where the alpaca is unable to rise without assistance, the prognosis for recovery is poor (10-20%, per Anderson, 2002).

Experiments in which llamas were intentionally inoculated with meningeal worm showed that symptoms typically manifested in the rear limbs within 45-53 days post inoculation [Rickard, et al., 1994]. Experiments on alpacas at UMass during their vaccine trials (see Meningeal Worm Vaccine, below) showed onset of weakness in the rear limbs at between two to two and a half months [Hoyt, 2008]. Loss of coordination in the rear limbs progressed to the front limbs over time. Additional symptoms may include stiffness, muscular weakness, head tilting, an arching neck, circling, blindness, weight loss, depression, and/or seizures [Anderson, 2002].

One farm that experienced a large scale outbreak of meningeal worm found that the onset of symptoms was significantly later than that reported in the llama experiments published in the literature, and more in line with those reported from the alpaca experiments at UMass [N. Padgett, pers. comm.]. It is possible that this reflects an actual species difference (the average onset of symptoms varies in deer species, for example); a difference in experimental versus on farm conditions (e.g., number of larvae ingested); or other cause. For a detailed discussion of Dr. Padgett's experience with a meningeal worm outbreak, please click here.

One experiment in which mule deer were inoculated with meningeal worm showed an initial, early stage in which lameness in a single leg was noted (5-7 days post infection), followed by a remission of symptoms, and then by the rear limb degeneration associated in alpacas with the parasite (25-40 days post inoculation in mule deer) [Tyler et al, 1980]. It was speculated by the authors that the second onset of symptoms correlated with the migration of larvae from the neural parenchyma into the subdural space; most adult mule deer typically did not survive this phase, although fawns did. Neurological symptoms abated in the survivors until a second phase at approximately 54-71 days post infection. Those animals that did survive the initial onset of neurological symptoms later succumbed to this second onset of symptoms, when it was hypothesized that the increasing size of the larvae and a presumed return to the neural parenchyma created more damage than the host can survive. The presence of "waves" of symptoms appears to be common in aberrant hosts, and early (5-7 days) lameness has also been noted in WTD.

Unfortunately, most meningeal worm infections in alpacas are not detected until there is significant loss of motor function. This is due to many factors, including the ability of the animal to mask signs of discomfort; owners' failure to detect subtle symptoms, or inability to observe their animals regularly; and the fact that numerous other diseases and disorders may present the same initial symptoms (e.g., polioencephalomalacia, trauma, etc.).

Given the early onset of symptoms in other animals studied, notably transient lameness at 5-7 days, it may behoove alpaca owners to watch any animal presenting with transient lameness or stiffness very closely, particularly if a proximate cause cannot be easily determined. It is possible that alpacas also show this initial early onset of symptoms, and unexplained transient lameness followed by additional symptoms several weeks later could be an strong indicator for meningeal worm treatment.

TREATMENT PROTOCOL

For many years, the protocol known as the "Buckeye Blast," developed by Dr. David Anderson while at Ohio State University, was the recommended treatment for meningeal worm infection. Today, this protocol is still being used, although time and experience have modified it somewhat. The most critical ingredient is fenbendazole (Safeguard), which kills the parasites present in the CNS. The recommended dose is 50 mg per kilogram bodyweight for five days. Although lower doses (20 mg/kg) have also been shown to be effective in many cases, owners may wish to err on the side of caution given how often much of a dose ends up on the animal, rather than in it.

Flunixin (Banamine) is recommended in addition to the fenbendazole as an anti-inflammatory agent. Much of the damage caused by the parasites is created by inflammation and swelling where they have been active in the nervous system, and Banamine helps to mitigate these issues. The recommended dose is 1 mg per kilogram bodyweight, twice daily for three days, then once daily for an additional three days. Although omeprazole (Gastrogard) was previously recommended due to the possible ulcerative properties of Banamine, it has been shown that a) oral Gastrogard is not effective in camelids [Poulsen, 2005] and b) Banamine is unlikely to be ulcerative in this time frame [Evans, 2005]. Injectable avermectins are not recommended as part of the treatment protocol, as they cause further damage if they cross the blood-brain barrier [Van Amstel, et al., 2009].

Vitamins are also often included in the treatment protocol, as many serve to help protect and/or promote regrowth of the nerves damaged by the parasite. Thiamine, vitamin E and additional B complex vitamins can all be administered to the alpaca under treatment. Vitamin E is fat soluble and care needs to be taken not to overdose, but the B vitamins are water soluble, and any excess is excreted by the alpaca in urine.

Recently, the methyl form of vitamin B12 (methylcobalamin) has been shown to be effective in promoting nerve regeneration due to injury, diabetic neuropathy and other causes [Yagihashi, 1982; Watanabe, 1994; Yamazake, 1994; Jacobs, 2009]. This vitamin may be beneficial for meningeal worm survivors. No information concerning appropriate dosing for alpacas currently exists, but extrapolating from information on the use of this vitamin in dogs and horses indicates that 2 mg per kilogram bodyweight would not appear to be unreasonable.

CAN A VACCINE BE DEVELOPED?

High on any East Coast alpaca breeder's wish list is a meningeal worm vaccine. Elk and other cervids have been shown to develop a complex immune response to meningeal worm infection [Bienek, et al. 1998; Neumann, et al., 1994], as have goats [Dew, et al., 1992]. These types of responses indicate that, at least for these species, a test for infection or a vaccine is theoretically possible.
Research into a possible vaccine for alpacas is ongoing in the University of Massachusetts Camelid Studies Program (http://camelidstudies.org/CamelidResearch.html), but given the unique nature of the camelid immune system, it is not clear if a viable vaccine can be produced.

Camelids "are the only known mammals that seem to possess functional homodimeric heavy-chain antibodies besides the classical heteromeric antibodies composed of heavy (H) and light (L) chains" [DeGendst, et al., 2005]. The reactions of the camelid immune system to pathogens are therefore not as predictable as those of other species.

Recent work by immunologists studying *P. tenuis* infected camelids showed that "Diseased animals, infected with P*arelaphostrongylus tenuis*, did not produce antigen-specific HCAbs; rather, they produced the conventional isotype, IgG1, exclusively" [Daley, et al., 2005]. This does not bode well for the possibility of developing a vaccine using traditional techniques. One can only hope that the in vivo studies being conducted by Dr. Purdy at UMass will show more promising results.

Many infected deer, upon dissection, have been found to harbor either a single pair of male and female worms, or else single sex populations [Duffy, et al., 2002]. The worms that infect a WTD in first year are hypothesized to "initiate a protective immunity that restricts further infection and establishes a limited, threshold number of adult worms" [Slomke et al., 1995] in the adult deer. The mechanism by which this is accomplished, once known, may have interesting implications for the search for an alpaca vaccine.

ACKNOWLEDGEMENTS
I would like to thank Drs. Ann Merriwether, PhD, Neil Padgett, MD, and Matthew Townsend, DVM, for their helpful comments on an earlier draft of this paper. Any errors remain solely my own.

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