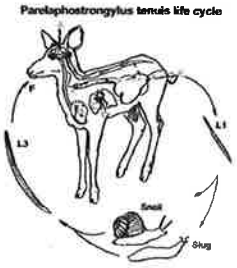
Attachment 2:

John Caywood's 1-22-15 testimony on IDAg's proposed importing of Meningeal worms

Mr. Chairman, Committee Members:

I am John Caywood, an Idaho resident for over 40 years, representing myself.

This year's Elk Industry proposal to further dismantle Idaho's animal health regulations would significantly reduce Administrative Rule protections of our economy, resources and family hunting traditions enjoyed by over 400,000 Idahoans.



Benefits sought by a few dozen shooter bull operators do not justify the significantly increased risk and probable harm to the Public Interest. To put this in perspective, this committee is considering loosening cervid protections while Oregon, Washington and Nevada flatly prohibit elk farming and Montana and Wyoming severely restrict it – all to protect their citizens' Public Interest and sovereign resources.

Key Points:

- The proposed rule's "test and treat" requirement is nonsensical as literature indicates meningeal parasites can neither be detected in live cervids nor effectively treated.[1]
- Scientists have not conclusively established whether other cervids are able to carry and pass on the meningeal worm.[2]
- Idaho's economy and resources shouldn't be at risk of a new exotic worm -- whether from farmed cervids or federally introduced wolves.
- There is insufficient reason to change Rules proven protective of our economy and resources.
- Elk ranchers have abundant cervid sources. They can import meningeal free cervids from 23 jurisdictions -- 17states and 6 provinces.
- "Do no harm." There is no urgent need to change Rules that protect rural economies and resources from this exotic parasite. The Genie should be left in the bottle.
- Reducing animal health protections endangers Idaho's economy and resources.

Meningial Worm Distribution in North America

[1] http://www.petmd.com/blogs/thedailyvet/aobrien/2013/aug/a-passion-for-parasites-meingeal-worm-30746
[2] https://www.courtlistener.com/opinion/666934/pacific-northwest-venison-producers-a-washington-c/

THE SCIENCE ON MENINGEAL WORMS

Disease significance: Mortalities in captive species; failed reintroduction of cervid species .... ; suppression of elk and moose populations; suspected cause of moose population declines in central and eastern North America.

Disease host: White Tailed Deer ... natural host ... rarely clinically affected ... 80% infected in endemic areas.

Aberrant hosts: ... Moose, caribou, mule deer, elk, llamas alpacas, pronghorn, sheep, goats, bison, rarely cattle and horses.

Diagnosis: In aberrant hosts, no ante-mortem diagnosis is available. Post-mortum recovery of adult worms or identification of larvae in neurological tissue is the **only** confirmatory test.

Meningeal worm in elk: Can cause debilitating neurological disease and death, and it has probably limited the sucess of past elk reintroductions into eastern North America. Potentially, elk could introduce meningeal worm to areas where white tailed deer are presently free of infection. **Meningeal worms can be spread through the translocation of infected hosts.**

Domestic animals: In certain areas, meningeal worms cause financial loss to owners of llamas, sheep and goats that share range with white tail deer and it is an important concern in game farm settings. Cases in sheep reported in New Hampshire. Connecticut,West Virginia and Minnesota with morbidity in flocks ranging from 2% to 59%. Cases in goats reported in New York, Texas and Michigan.

Treatment: Includes various anthelminthics. None of these have been tested in controlled studies, but when used with good supportive care, they **may** contribute toward recovery, at least of **lightly infected animals.** Ivermectin will protedt if given within 24 hours after infection. By 10 days, larvae have entered the spinal cord and appear to be protected by the blood brain barrier. Treatment has no effect on adult worms already in the cental nervous system but depresses the number of larvae being passed in feces. **Larvae reappear in feces within a month of treatment.**

Control: Every effort should be made by government regulation and game ranching industry practice to prevent the introduction of meningeal worm into western North America. Ther is no reason to believe that conditions there are unsuitable for transmission if it were to arrive there with infected cervids. White tails represent the greatest threat of accidental introduction, but elk, and possibly other cervids could be responsible. **Fear of spreading this parasite to western North America has led to legislation restricting the translocation of white-tails and other hosts in which the parasite occasionally matures.**

Scientific authorities: The above materials are excerpts from " Extrapulmonary Lungworms Of Cervids, Parasitic Diseases Of Wild Mammals, Second Edition", and " Meningeal Worm, American Association of Zoo Veterinarians Infectious Disease Committee Manual, 2013" Copies of the source materials have been provided also.

American Association of Zoo Veterinarians Infectious Disease Committee Manual 2013

MENINGEAL WORM (*Parelaphostrongylus tenuis*)

Animal Group(s) Affected	Transmission	Clinical Signs	Severity	Treatment	Prevention and Control	Zoonotic
Ungulates, notably cervids	Oral - Ingestion of infected intermediate host which includes numerous terrestrial mollusk species (i.e., snails and slugs)	Neurologic	Ranges from mild lameness to recumbency and death. Severity is typically worse in young animals and may vary between species.	High doses of anthelmintics combined with anti-inflammatories; supportive therapy	Prophylactic anthelmintic administered every 4-6 wks; exclusion of the natural host (white-tailed deer); elimination or control of mollusk population	No

Fact Sheet compiled by: Rae Gandolf and Julie Ter Beest

Sheet completed on: 1 January 2011; updated 9 October 2012

Fact Sheet Reviewed by: Murray Lankester, Priya Bapodra

Susceptible animal groups:

Natural host: The white-tailed deer (*Odocoileus virginianus*) serves as the natural host and is rarely clinically affected; they can shed numerous dorsal-spined larvae in their feces. Approximately 80% of white-tailed deer are infected in endemic regions.

Aberrant or dead-end hosts: Other cervid species (moose, caribou, mule deer, elk, Sika deer); camelids (camels, llamas, alpacas); pronghorn; some bovids (many antelope species, bighorn sheep, Angora goats, bison, rarely domestic cattle); and rarely equids (reported in domestic horses) may show severe clinical signs. Overall, these species rarely shed larvae in their feces.

Disease significance: Mortalities in captive species; failed reintroduction of cervid species such as caribou; suppression of elk and moose populations; suspected cause of moose population declines in central and eastern North America.

Causative organism: *Parelaphostrongylus tenuis,* an extrapulmonary lungworm nematode

Life cycle: The natural host (white-tailed deer) acquires the infection through accidental ingestion of mollusks infected with 3rd stage larvae. The larvae migrate from the gastrointestinal tract along spinal nerves and into the spinal cord where they develop to the last larval state. Adult worms then locate on the meninges and in the cranial venous sinuses where they lay eggs. The eggs pass into the venous circulation, develop into 1st stage larvae in lung capillaries, and then migrate into the lung tissue. These larvae are expectorated, swallowed, and passed in the feces. Mollusks acquire larval infection when crawling over feces and the parasite develops into the infective 3rd stage larvae within this intermediate host.

In the aberrant host, infection is acquired by the same route. However, migration of the larvae in the spinal cord tends to be non-directional and larvae often die before reaching the brain. The aimless migration and larval death result in more local tissue damage as compared to the natural host. Larvae infrequently develop into reproductive adults in the aberrant host.

Zoonotic potential: None reported

Distribution: Predominantly associated with deciduous and deciduous-coniferous forests of eastern and central North America, concurrent with white-tail deer populations. It is uncertain why deer of the southeast

MENINGEAL WORM (*Parelaphostrongylus tenuis*)

coastal plains region and of western North America are not infected.

Incubation period:
Natural host: pre-patent period 82-137 days, inversely proportional to infection dose.
Aberrant host: signs typically appear in 30-60 days, as short as 5 days reported in experimental infections.

Clinical signs: Neurologic signs are associated with intracranial or spinal cord inflammatory lesions caused by parasite migration. Signs may range from single limb lameness or rear limb weakness to head tilt, ataxia, circling, blindness, progressive loss of motor function and death. Ocular symptoms associated with migration of larvae into the uvea have been reported.

Post mortem, gross, or histologic findings Lesions in the aberrant host consist primarily of histologic changes in the brain and spinal cord. They may include meningitis and encephalitis; perivascular cuffing and infiltrations of eosinophils, lymphocytes, and plasma cells; calcified remains of worms; worm tracks; focal traumatic malacia caused by developing nematodes; gliosis; disruption of the ependyma; neuronal and myelin degeneration. Eggs and larvae may be found associated with the eyes or the roots of cranial nerves, on the leptomeninges, and in brain tissue.

Diagnosis:
Natural host: Modified Baermann technique for retrieving 1st stage larvae from feces. Larvae must then be differentiated from related species using PCR. However, there are limited species of dorsal-spined larvae and they are easy to retrieve, allowing for presumptive diagnosis. In addition to white-tailed deer, moose and elk may shed the larvae in low numbers.
Aberrant hosts: No ante-mortem diagnosis is available. Post-mortem recovery of adult worms or identification of larvae in neurologic tissue is the only confirmatory test. A nested PCR assay has been developed to confirm larval identification in the case of verminous migration in horses. A commercial serum ELISA detecting antibodies against 3rd stage larvae in cervid species was briefly available in Canada to aid in diagnosis of ante-mortem cases; results have been reported for moose and elk, but this test is not currently available.

Material required for laboratory analysis: Post mortem: spinal cord and brain
Antemortem: plasma or serum (aberrant hosts), feces (white-tailed deer, moose and elk)

Relevant diagnostic laboratories:
ELISA: Prairie Diagnostic Services, Regina, Saskatchewan, Canada

Treatment: High dose fenbendazole (20-50mg/kg orally once daily for 5 days) and or high dose ivermectin (0.3-0.4mg/kg SC daily for 3-5 days), or levamisole, in addition to supportive therapies including non-steroidal or steroidal anti-inflammatory drugs, vitamin E, and vitamin B complex. Early initiation of treatment is key to success.

Prevention and control:
Captive species: Administration of anthelmintics every 4 -6 weeks to target 3rd stage larvae before they migrate to neural tissue; minimize exposure of captive animals to mollusks by establishing gravel roads or other vegetation breaks to act as snail and slug barriers; use molluscicides with caution due to potential for environmental toxicity; allow non-susceptible species to initiate grazing on new or overgrown pastures; reduce white-tailed deer population and build fences to exclude them.
Free-ranging species: Control of white-tailed deer population to reduce exposure.

Suggested disinfectant for housing facilities: Molluscicides (copper sulfate, metaldehyde, sodium pentachlorophenate) may be used against the intermediate host with caution, as they are potential environmental toxins.

Notification: None

Measures required under the Animal Disease Surveillance Plan: None.

American Association of Zoo Veterinarians Infectious Disease Committee Manual 2013

MENINGEAL WORM (*Parelaphostrongylus tenuis*)

Measures required for introducing animals to infected animal: As no direct transmission of the parasite occurs, and species susceptible to clinical disease do not typically pass larvae, infected animals do not pose a direct threat to un-infected animals. However, white-tailed deer should generally be considered as infected, and exposure of susceptible species to white-tailed deer should be avoided as possible.

Conditions for restoring disease-free status after an outbreak: This disease is endemic in white-tailed deer populations of eastern North America.

Experts who may be consulted:

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Attn. Senate Ag. Affairs Committee

The Idaho Elk Breeders Association helped to draft the legislation last year, and was involved in the negotiated rule making process for the rules before you today. The legislation increased our annual inventory fees, and initiated fees on imports as well as exports to help the Idaho State Department of Ag. operate and maintain the domestic cervidae program. We also worked with ISDA to reduce our testing % (for cwd) to 10% on animals we intentionally cull, any animals that die of unknown causes will still have to be tested 100%

The changes to the import rules are to help lift restrictions on animals being imported from east of the 100th meridian. Montana already has removed this restriction. As elk are a dead end host (the minangeal cannot complete its life cycle), and deworming to eliminate the adult stages of the worm would be required prior to import. The risk is greatly minimized that any worms could be imported. The parasite already exists in moose populations in Idaho.

In conclusion, The IEBA supports both of the proposed rule changes before you today.

Sincerely,

William D. Miller

02.04.21 - Rules Governing The Importation Of Animals (Cervidae)

02.04.19 - Rules Governing Domestic Cervidae