Case Report Rapport de cas

Evidence of *Parelaphostrongylus tenuis* infections in free-ranging elk *(Cervus elaphus)* in southern Ontario

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Abstract — The antemortem detection of a *Parelaphostrongylus tenuis* infection in a free-ranging wild elk *(Cervus elaphus)* in southern Ontario is documented. Postmortems on other free-ranging elk that died during 2000–2005 indicated that 59% (17/29) were infected with *P. tenuis,* based on presence of lesions in the brain.

Résumé — Preuves d'infections à *Parelaphostrongylus tenuis* chez le wapiti (Cervus elaphus) en élevage extensif dans le sud de l'Ontario. Cet article décrit la détection antémortem d'une infection à *Parelaphostrongylus tenuis* chez un wapiti en élevage extensif *(Cervus elaphus)* dans le sud de l'Ontario. Des examens post mortem réalisés sur d'autres wapitis en élevage extensif, morts entre 2000 et 2005, ont révélé que 59 % (17/29) présentaient des lésions au cerveau caractéristiques d'infection à *P. tenuis.*

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During 2000 and 2001, 120 elk *(Cervus elaphus)*, also known as wapiti, were translocated from Elk Island National Park (EINP), Alberta, to the Bancroft, Ontario, area as part of a larger restoration project in which 443 elk (from EINP) were released in 4 areas of Ontario during 1998–2001 (1). All but 1 of the elk released near Bancroft were radio-collared and ear-tagged prior to release, as a means for monitoring the location of released elk, as well as their progeny born in Ontario. Mortality signals from the radio-collar alerted researchers that the animal had succumbed, which, in turn, allowed for rapid retrieval of animals, so that postmortems to determine the cause of death could be completed. Radio-telemetry also provided researchers with a means to locate animals for behavioral observation. The purpose of the study was to estimate the prevalence of *P. tenuis* in free-ranging elk in southern Ontario and to evaluate a recently developed ELISA for the detection of antibodies against *P. tenuis.*

Figure 1. The *Parelaphostrongylus tenuis* infected bull elk (Case 1) is pictured on February 1, 2005, with several white-tailed deer at a winter feeding site near Bancroft, Ontario (Photo by R. Rosatte).

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Case 1

During 2003/2004, a group of about 30–40 elk, consisting of animals transported from EINP and their offspring born in Ontario, were observed frequenting the area of Hartsmere, Ontario (approximately 44°5′ N, 77°30′ W) (about 30 km east of Bancroft, Ontario). During October and November 2004, a sick yearling bull elk was observed in this group. By using radio-telemetry, it was determined that during December 2004, the animal left the group, became solitary (abnormal social behavior for a herd animal), and began to frequent a nearby barn.

Eventually, the animal stayed at the barn continuously and was provided with feed, primarily alfalfa (Figure 1).

Case description

Clinical signs exhibited by the yearling bull elk included a loss of fear of humans, ataxia, loss of balance, and a drooping head. Based on the observed clinical signs, a tentative diagnosis of *P. tenuis* infection was made.

On January 11, 2005, he (estimated weight 150–200 kg) was immobilized with an IM injection of 500 mg of tiletamine hydrochloride/zolazepam hydrochloride (Telazole; Fort Dodge Animal Health, Fort Dodge, Iowa, USA) and 300 mg of xylazine hydrochloride (Anased; Vet-A-Mix, Shenandoah, Iowa, USA) in the right upper hind limb area via a 5-mL sterile syringe and 22 g (4 cm) needle, attached to jab stick (an 80-cm section of copper tubing that contained a wooden dowel to apply pressure to the syringe plunger). After the animal was immobilized, 8 mL of blood was collected from the jugular vein into 2, 10-mL sterile blood collection tubes (Vacutainer; Becton Dickinson, Franklin Lakes, New Jersey, USA). The elk was also fitted with a VHF radio-collar and an ear-tag (Flex-Lok; Ketchum Manufacturing, Ottawa, Ontario) for future identification. When processing had been completed, the elk was given 12 mg of yohimbine hydrochloride (Yobine; Lloyd Laboratories, Shenandoah, Iowa, USA), IM, in the right hind limb, as an antagonist, in order to speed recovery from the effects of xylazine. The elk recovered fully from the procedure. The blood sample was centrifuged for 15 min, then a 4-mL plasma sample was collected and stored, for 1 wk, in 2, 2-mL sterile plastic microtubes at -12°C. The frozen sample was then shipped by courier to the Prairie Diagnostic Services laboratory in Regina, Saskatchewan.

The plasma sample was tested for the presence of antibodies against *P. tenuis* by using excretory-secretory (ES) products derived from infective 3rd-stage larvae of the parasite as coating antigen in an enzyme-linked immunosorbent assay (ELISA), as described by Ogunremi et al (2). The result (obtained on February 4, 2005) of the ELISA performed on the bull elk sample (S), expressed as the ratio of the optical density reading of the samples and of a reference positive (P), or S/P ratio, was 0.986, which translates to an ELISA index of 98.6 units, cut off = 45.0 units, indicating that the bull elk was exposed to *P. tenuis.*

Although field infected elk rarely pass enough *P. tenuis* first-stage larvae to make fecal examination a reliable diagnostic test (3), an attempt was made to recover larvae from the feces of the *P. tenuis*-suspect bull elk by collecting fecal samples at the location of the yearling bull elk on February 18, 2005, and having them screened for *P. tenuis* larvae at Trent University and the Regina laboratory of the Prairie Diagnostic Services, by using the Baermann beaker technique (4). No *P. tenuis* larva was detected; however, the samples were positive for the lungworm *Dictyocaulus.* Historical observations indicate that larval shedding is inconsistent or undetectable in *P. tenuis*-infected elk (5,6). More recently, out of 4 elk inoculated with 6–20 stage 3 larvae, 2 larvae were recovered from only 1 animal that shed larvae and only on day 202 postexposure, despite the examination of feces twice a week (2).

Between February and May 2005, the Bancroft bull elk intermittently demonstrated the same neurological signs, but by June 1, 2005, his condition had deteriorated and he was euthanized. At this time, a 2nd blood sample was collected; it again tested positive for *P. tenuis.* Findings on postmortem examination included meningoencephalomyelitis, characterized by moderate to severe perivascular cuffing of eosinophils, lymphocytes, plasma cells, and macrophages in the brain and spinal cord. Four sections of degenerate larvae (Figure 2a), and 1 of a normal-looking larva (Figure 2b) were present in the meninges of the brain. All larvae were surrounded by mononuclear cells and, in addition, 2 of the degenerate sections were closely associated with eosinophils (1 and 5 eosinophils; Figure 2c). The larvae had the same features as those reported previously in the histopathological sections of the brain of *P. tenuis*-infected elk (7). Areas of hemorrhages were observed in the white matter of the spinal cord and brain. Hemosiderin granules were present inside macrophages and extracellularly in sections of the brain and spinal cord. In the lungs, an adult female worm with eggs (85 × 45 μm) was recovered and identified as a *Dictyocaulus* sp. There was severe pulmonary congestion and infiltration of inflammatory cells, particularly eosinophils, macrophages, and lymphocytes. The central nervous system (CNS) and pulmonary lesions were attributable to *P. tenuis* and *Dictyocaulus,* respectively.

Case 2

During the first 2 wk of September 2005, a yearling (1 1/2 y) cow elk, located near the area utilized by Case 1, demonstrated mobility problems (slow, stumbling, unstable movements), had no fear of humans, and remained in an approximately 10-m × 40-m area.

Case description

On September 14, 2005, the cow elk was observed in the same area and on September 15, 2005, she was found dead. On postmortem examination at the Canadian Cooperative Wildlife Health Centre (CCWHC), Guelph, Ontario the cause of death was confirmed as predation by wild canids. Histologic examination revealed an adult nematode, compatible with *P. tenuis,* in a cerebral sulcus and larvating nematode eggs (compatible with *P. tenuis*) in the meninges (in tissue as well as blood vessels) (Figure 3). The adult nematode was oriented in the section in such a way that it did not allow for an evaluation of the features to classify its genus and species; however, based on visible features, it was likely a metastrongyle. The larvae had evoked an inflammatory response characterized by eosinophilic infiltration. Blood serum samples collected 1 d post mortem revealed a positive *P. tenuis* ELISA S/P ratio of 1.04 and 0.72 (104 and 72 ELISA units). This animal was negative (immunohistochemical staining) for chronic wasting disease (CWD).

Case 3

During 2000–2005, elk (including elk that originated at EINP, as well as their progeny born in Ontario) that died in southern Ontario were collected opportunistically to determine their state of health, body condition, and cause of death.

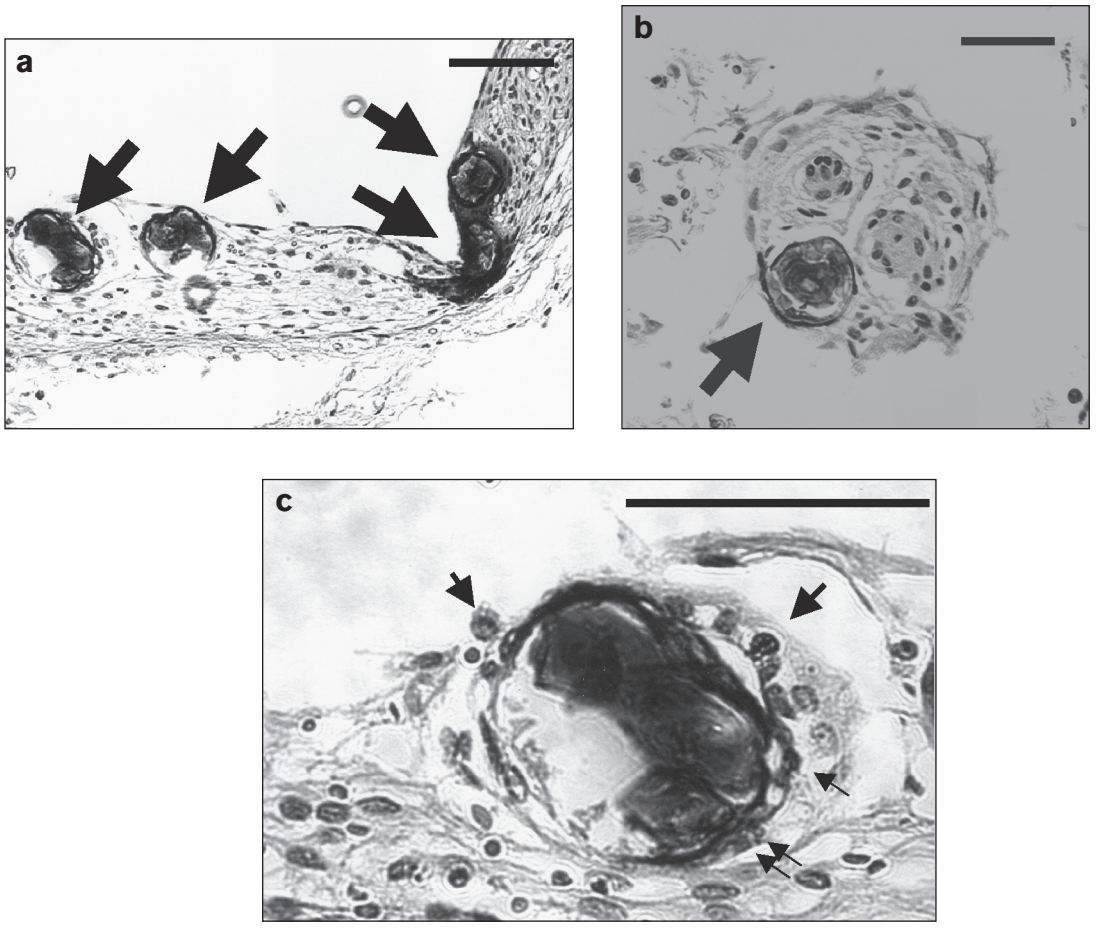

Figure 2. *Parelaphostrongylus tenuis* larvating eggs in the brain meninges of the bull elk (Case 1): (a) degenerate larvae, (b) normal looking larva, (c) larva surrounded by eosinophils. Arrows indicate larvae (a, b) or eosinophils (c). Bar = 20 μm.

Case description

Postmortem examinations on 42 elk yielded 3 classes of animals at their time of death: 1) elk that may have been exposed to *P. tenuis* and developed inflammatory lesions in the brain but were in good condition; 2) elk with clinical disease due to *P. tenuis* infection, which included neurological signs, and were in an emaciated state; 3) elk without evidence of *P. tenuis* infection. For some animals, infection was not assessed, due to unsuitability or unavailability of brain tissue for examination. Of the 42 elk that had died due to a variety of causes (found dead, illegally shot, collisions with vehicles, drowning, hit by a train), 13 had died too soon following release (1–5 mo) to have become clinically ill with *P. tenuis* (the elk originated in Alberta where apparently there is no *P. tenuis* infection in wild deer or elk). Of the 29 that survived longer than 6 mo post release, 17 (59%) had lesions in the brain compatible with *P. tenuis* infection. The lesions consisted of mild to moderate perivascular cuffing, primarily with mononuclear cells (lymphocytes and macrophages) with occasional eosinophils present. Hemosiderin was present, typically within macrophages in perivascular locations, usually in the meninges. There was mild, multifocal gliosis and, rarely, small foci of malacia. Greater numbers of females (n = 12) had lesions than males (n = 5) (P = 0.016; chi square = 5.85); however, no difference with respect to the age of elk and occurrence of lesions was found (9 elk were $\leq$ 2 y of age and 8 elk were $\geq$ 3 y of age) (P = 0.88, chi square = 0.02). The sex/age composition of the 17 elk that had lesions was as follows: 35% (6/17) cows $\geq$ 3 y; 12% (2/17) 2-y-old cows; 24% (4/17) yearling cows; 12% (2/17) bulls $\geq$ 3 y; 6% (1/17) 2-y-old bulls; and 12% (2/17) yearling bulls. Of the 12 elk that were not suspect for *P. tenuis,* 3 were adult cows, 3 were adult bulls, 5 were yearling bulls, and 1 was a male calf. None of the 13 elk that died 1–5 mo post release had any evidence of *P. tenuis* infection.

Discussion

Parelaphostrongylus tenuis, also known as meningeal worm or brain worm, rarely causes serious disease in white-tailed deer, the definitive host in Ontario, as deer and *P. tenuis* have coevolved (8). It is, however, one of the most pathogenic nematodes of cervids such as elk, moose *(Alces alces)*, and caribou *(Rangifer tarandus caribou)*, and it can cause severe neurological disease and death in species such as elk. *Parelaphostrongylus tenuis* has probably limited the success of previous elk restorations in eastern North America (3,8,9), although some reintroduced elk herds have persisted on the same range as *P. tenuis*-infected white-tailed deer (10).

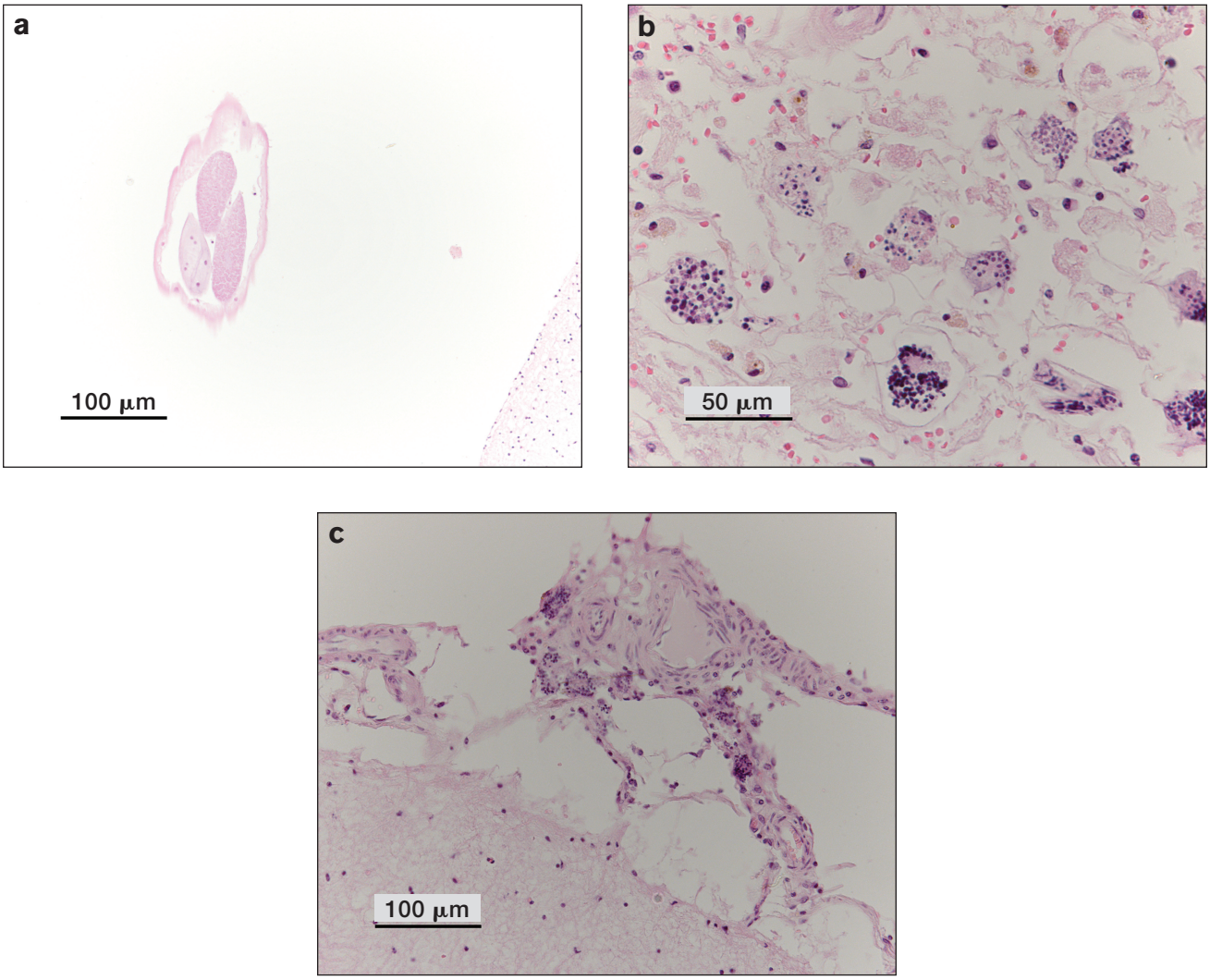

Figure 3. Photos of cross sections of brain from the cow elk (Case 2) showing (a) a submature or adult nematode (compatible with *P. tenuis*) in a cerebral sulcus bar = 100 μm; (b) (c) larvating nematode eggs (compatible with *P. tenuis*) in the meninges (photos by D. Campbell); (b) bar = 50 μm, (c) bar = 100 μm.

It was postulated during 2003–2004 that elk restored to the Bancroft, Ontario, area may have acquired *P. tenuis* infections from deer in the Hartsmere area (Figure 1), as brain lesions, possibly due to parasite migration, were found in elk during postmortem examinations at the CCWHC in Guelph. Similar findings have been reported in other areas where *P. tenuis* is enzootic and elk have been reintroduced. Woolf et al (11) found lesions present in the brain of 37 animals infected with *P. tenuis* in Pennsylvania, although only 11 clinical cases were observed among the 87 elk examined (12). As well, Carpenter et al (7) detected lesions and *P. tenuis* larvae in the meninges of elk sampled in Oklahoma. Since the reintroduction of elk to Ontario, larval or adult meningeal worms have not been found in necropsied elk until now, and despite the strong suspicion of elk acquiring and dying of *P. tenuis,* a serological test (*P. tenuis* ELISA) has not previously been available to assist with the diagnosis (2). Now that a *P. tenuis* ELISA (2) is commercially available, serological testing can be carried out to provide supportive evidence for the role of *P. tenuis* in elk mortality in Ontario.

The orientation of the adult nematode worm that was found in a histological section of the brain of the cow elk did not allow for classification beyond family, but anatomical features, such as the multinucleated cells in the intestine, place it in the metastrongyles. However, the possible species of this group that are likely to be found in the brain of an elk in eastern Ontario is somewhat limited to *P. tenuis.* Thus, the logical conclusion was that the adult nematode was "compatible with *P. tenuis.*" This phrase is commonly used by pathologists when a definite diagnosis is not possible but is suspected, based on lesions, physical appearances, geographic location, and other etiologies. In addition, *P. tenuis* infections in elk, based on clinical signs, lesions, and the presence of adult nematodes and larvae in neural tissue, have been documented in numerous references (8,12).

It seems reasonable to conclude that the source of the *P. tenuis* infection for the elk was via ingestion of infected gastropods that had acquired their infections via consumption of, or penetration by, first stage *P. tenuis* larvae shed in the feces of white-tailed deer. The bull elk is known to have wintered during 2003–2004

in the yarding areas east of Bancroft (13), where a high density of deer is found and most of their fecal samples, (82%) have dorsal-spined larvae, presumably *P. tenuis* (14). Furthermore, the infected bull elk likely spent the summer/fall of 2004 in the same general area as the other members of its social group (as determined by radio-telemetry) and probably acquired the infection in the vicinity of the deer yarding areas near Hartsmere. In a previous study in Minnesota, *P. tenuis* larvae found in gastropods had reached the infective 3rd stage by July–October (15). However, only a small proportion of gastropods will be infected; 1% of gastropods were found to be infected with *P. tenuis* in Algonquin Park, Ontario, north of the Hartsmere area (16).

The severity and outcome of the *P. tenuis* infection in cervids such as elk is correlated with the infective dose of larvae. Significant doses (> 125, 3rd stage larvae) can result in neurological symptoms and death. Elk receiving moderate numbers (25–75 larvae) developed neurological signs, some died, and some shed larvae. However, elk that were exposed to small numbers (15 larvae) did not develop clinical signs or shed larvae (10). Thus, the ingestion of low doses of larvae may partially explain the survival of some eastern elk populations. The impact of *P. tenuis* on the Bancroft elk population will most likely be related to many factors, including 1) the prevalence of *P. tenuis* in resident deer populations, 2) the range overlap between deer and elk, 3) the abundance and type of gastropods found on deer and elk range, 4) the number of *P. tenuis*-infected gastropods that elk ingest, 5) the age of elk at the time of infection, 6) the amount of damage caused by worms within the CNS, 7) immunity to infection, and 8) the ability of elk to survive low level infections of *P. tenuis.*

Other studies have noted clinical signs in elk due to *P. tenuis* infection similar to those in this study; however, those diagnoses were made postmortem. Meningeal worms were found postmortem in the brains of elk in Oklahoma that exhibited signs prior to death such as ataxia and circling (7). In Pennsylvania, Woolf et al (11) observed clinical signs such as ataxia, circling, tameness, and head/neck tilt in captive elk that were confirmed on postmortem examination to be infected with *P. tenuis.* Neurological signs in elk in Kentucky and Michigan were also attributed to *P. tenuis* infection (17,18). It should also be noted that Woolf et al (11) found that although there was a high number of *P. tenuis*-infected elk on a preserve in Pennsylvania, many did not exhibit any clinical signs. Thus, a diagnosis based only on clinical signs may provide an underestimate of the true prevalence of *P. tenuis* in a wild elk population.

Olsen and Woolf (12) and Woolf et al (11) found a higher prevalence of *P. tenuis* infections in yearling and 2.5-y-old elk (60%) than in calves and elk > 3.5 y (25%) in a preserve in Pennsylvania. More recently, Larkin et al (17) showed that 73% of elk dying of *P. tenuis* in Kentucky were < 3 y. In this study, only 53% of wild elk sampled in the Bancroft area and suspected of being infected with *P. tenuis* were ≤ 2.5 y (of those, 35% were yearlings). As noted by Woolf et al (11), a high prevalence of *P. tenuis* in younger-aged elk could affect the productivity of the herd and limit population growth over the long term. The true impact of *P. tenuis* in Ontario remains to be seen, as elk were only recently (2000/2001) introduced to the Bancroft area. However, the impact could be minimal in Ontario, as, in Michigan, *P. tenuis* accounted for only 3% of elk mortalities, much lower than mortality due to harvesting (58%), illegal kills (22%), other diseases (7%), and malnutrition (4%) (18).

During a preliminary validation exercise of the ELISA, all 12 elk experimentally infected with *P. tenuis* obtained from the CNS tested positive for antibodies to *P. tenuis* (Ogunremi et al, unpublished observations). As a confirmation of infection, adult worms were recovered at necropsy from the CNS of the animals, starting 4 mo after inoculation (10), but all were serologically positive starting as early as 1 mo postinoculation and lasting until the termination of the experiment. While serological cross-reactivity has been observed between sera from *Dictyocaulus*-infected elk and white-tailed deer and the somatic antigens of *P. tenuis* (2,19), no such cross-reactivity was observed against the ES products of *P. tenuis* stage 3 larvae (2,20). Specificity of the *P. tenuis* ELISA was found to be 97.2% (95% confidence interval, = 95.8–98.6%) among 579 elk sourced from *P. tenuis*-free areas where the prevalence of *Dictyocaulus* was estimated at about 12% (21,22). Based on the above estimate of specificity and an apparently high sensitivity, albeit on a small sample size (12 out of 12 animals; unpublished observations), it is reasonable to conclude that the use of larval ES products in the ELISA may provide strong evidence that an elk may have been exposed to *P. tenuis.*

One may question whether diseases, parasites, or deficiencies, other than *P. tenuis,* may have been responsible for the signs and clinical condition in the bull elk. Since neurological signs were evident in the bull elk, the brain was tested for rabies virus by the fluorescent antibody test and found to be negative. Chronic wasting disease is also a cause of neurological signs in elk. As in *P. tenuis* infections, elk affected with CWD show abnormal head posture and loss of fear of humans. Excessive salivation is commonly seen in animals terminally ill, but it is not a common finding in *P. tenuis*-infected elk (23–25), and was not observed in this bull elk. Importantly, none of the pathognomonic signs of CWD, namely spongiform degeneration of grey matter neuropil, intraneuronal vacuolation, and astrocytic hypertrophy and hyperplasia (24,26) were observed in the brain of this animal. Both meningitis and encephalitis, which are absent in CWD infections (24), were observed in this elk. Trace mineral deficiency, particularly of copper, resulting in ataxia has been reported in a conspecific species (red deer *Cervus elaphus* subsp.). However, the effects of copper deficiency are different from those of *P. tenuis* infection: there are accompanying skeletal deformities (27,28); many animals in the same social group are usually affected (28); and the main CNS lesion is neural demyelination (29). In a recent report, behavioral observations in Michigan over a period of 14 y of 100 radio-collared elk belonging to different age and gender classes failed to reveal any neurological signs other than those due to *P. tenuis* infection (18). Given the above evidence, we feel confident the bull elk was infected with *P. tenuis.*

In the past, the only technique available for the detection of *P. tenuis* in live hosts was the Baermann funnel technique, which relies on the recovery of 1st stage larvae in the feces of infected

animals. The more efficacious modified technique (4) may lead to the recovery of up to 13% of *P. tenuis* larvae actually present in feces. The probability of recovery of larvae from infected elk using these techniques is low, as elk intermittently shed only low numbers of 1st stage larvae in feces (2,6,8). This may be related to the fact that field-infected elk may harbor only a few (1–3) adult worms (7). As well, unisexual infections may be present in infected animals, making larval shedding impossible (30). Furthermore, worms may die, or the elk may die, before infections become patent (8). As demonstrated for moose in which infected animals also harbor a few parasites (31), the newly developed *P. tenuis* ELISA that uses ES antigen may be useful for detecting meningeal worm infections. However, a presumptive diagnosis based on serological methods could have limitations and an adequate field of validation of data based on a range of elk populations harboring closely related parasites is required. The availability of more field samples, especially those obtained antemortem from animals showing clinical symptoms suggestive of a *P. tenuis* infection, should provide further opportunity to assess the dependability of the *P. tenuis* ELISA as a diagnostic tool and its utility in wildlife management and for monitoring the prevalence of *P. tenuis* in elk.

There are documented antemortem cases based on clinical signs. To our knowledge, this is the 1st documented report of antemortem diagnosis of *P. tenuis* infection in which a serological method was used in an individual free ranging elk in the wild. The potential impact of *P. tenuis* on restored eastern elk populations (32) is of concern, as failures of previous introduction attempts have been attributed to *P. tenuis* infections (33). This is supported by other observations indicating that *P. tenuis* was responsible for reduced growth and mortalities in elk populations in Pennsylvania and Kentucky, respectively (17,34). Given the observations reported in this study, it would not be prudent to transport elk from eastern North America to the west, where *P. tenuis* is currently absent [see Lankester (8) for a map of the current range of *P. tenuis*]. In addition, managers should be aware that moving elk from areas free of *P. tenuis* to eastern North America, where *P. tenuis* is present, could result in significant mortalities as elk will not have had sufficient time to co-evolve with this parasite, as white-tailed deer have done in the east.

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Book Review
Compte rendu de livre

Canine and Feline Geriatric Oncology: Honoring the Human-Animal Bond

Villalobos A, Kaplan L. Blackwell Publishing, Ames, Iowa, USA, 2006. 370 pp. ISBN 0-8138-0266-0. USA$79.99.

This refreshing 1st edition approaches the practice of veterinary oncology in geriatric patients with respect to communication, ethics, and philosophy. This is not a "how-to" book; it is written to assist clinicians in decision-making for their patients and in tackling this emotional and often difficult area of veterinary medicine. Interspersed throughout the text are short clinical vignettes of patients Noodles, Scrappy, Sunshine, and many others relayed from a compassionate standpoint. For detailed instruction on clinical management of small animal oncology cases, the authors refer readers to an extensive list of previously published general oncology reference texts and Web sites for more in-depth information on specific tumor types in dogs and cats.

The book is laid out in a 3 main sections. Part 1 addresses basic oncology principles; carcinogenesis, tumor suppressor genes, apoptosis, the impact of aging on cancer causation, awareness, risk factors, and the metastatic cascade. This section displays a helpful chart on common terminology and abbreviations for basic cancer types based on tissue of origin. The authors expand on the cancer terminology and conclude with a review of therapeutic modalities. An interesting comparative aging chart for cats and dogs is included in Chapter 1. A highlight of this section is a chart of frequently encountered treatment modalities with reference to adjuvant and specialized therapy. Recommendations for the specific needs and concerns of geriatric patients, in particular with respect to the human animal bond are also included.

Part 2 addresses warning signs of cancer by body system and tumor type. Several excellent photographs of common clinical presentations are provided in this section. Basic cancer types and their biological behavior are described, followed by recommendations on generating an accurate diagnosis. A comprehensive discussion of available diagnostic tests utilizing clinical pathology, histopathology, and diagnostic imaging is included as well as how they translate to staging for cancer patients. The TNM (Tumor, Node, Metastasis) staging schematic is presented in this section.

Part 3 is the largest section, and provides a detailed account of treatment modalities for geriatric cancer patients, including surgery, chemotherapy, and radiation therapy. Pain control for cancer patients, is covered in this section and the authors do an excellent job of illustrating the WHO pain ladder, types of pain in cancer patients, and in providing practical tips on the clinical management of pain in veterinary patients. The last part of this section addresses decision-making in patients with recurrent cancer, recommendations for terminal patients, euthanasia, and considerations for care after death.

The appendices provide specific tumor protocols, handouts for clients, and body lesion maps.

Overall, this book is an enjoyable, easy read for veterinary students, staff, and clinicians that is user friendly and informative.

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