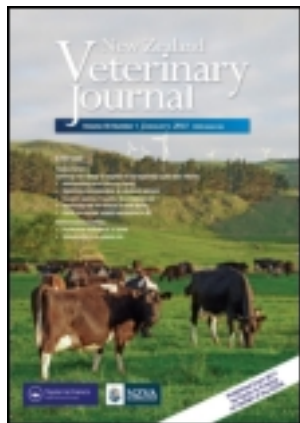


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### Abomasal parasite syndrome in North American elk (*Cervus elaphus canadensis*)

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## Clinical Communication

## Abomasal parasite syndrome in North American elk (*Cervus elaphus canadensis*)

MR Woodbury\*<sup>§</sup> and NMA Parry<sup>†</sup>

### Abstract

**CASE HISTORY:** An 18-month clinical course of chronic ill-thrift, weight loss and emaciation, and eventual death occurred in a group of 520 translocated elk of mixed age and sex. Translocation was carried out without regard to animal welfare or health risks associated with the translocation. Mortality was approximately 84% (436/520) despite supportive nutritional and medical treatment.

**PATHOLOGICAL FINDINGS:** General clinical and post-mortem examinations indicated only Se and Cu imbalances and nutritional inadequacy. Additional purposeful post-mortem examination and histological evaluation of tissue sections from four of the affected adult elk demonstrated elevated abomasal pH and proliferative abomasal lesions as the most significant findings, consistent with Type-II ostertagiosis; intra-lesional nematodes were seen in the abomasum of two animals.

**DIAGNOSIS:** Fading elk syndrome, or abomasal parasite syndrome in elk.

**CLINICAL RELEVANCE:** Abomasal parasite syndrome initiated by Type-II ostertagiosis should be considered as a differential diagnosis in cases of ill-thrift and wasting in elk or elk-red deer hybrids. Changes to the architecture and secretory function of the abomasal wall lead to apparently irreversible digestive pathophysiology and nutritional disease.

**KEY WORDS:** *Abomasum, elk, fading elk syndrome, ill-thrift, Ostertagia spp., wasting*

### Introduction

A condition of chronic ill-thrift characterised by progressive weight loss, and often severe skeletal muscle wasting, has been previously described in North American wapiti (elk) as well as in wapiti-type red deer hybrids farmed in New Zealand (Mackintosh and Orr 1990). North American elk and red deer are both subspecies of *Cervus elaphus*, and in captive circumstances they will interbreed freely, producing fertile hybrid offspring with desirable carcass characteristics for meat production. Although reports of 'fading elk syndrome' are uncommon in the veteri-

nary literature, this disease syndrome should not be confused with prion protein-associated chronic wasting disease (CWD) of cervids. Fading elk syndrome has been associated with a greater susceptibility of farmed elk-red deer crossbreds to gastrointestinal parasitism and Cu deficiency, together with nutritional differences between farmed elk in New Zealand and their native North America (Waldrup and Mackintosh 1992). Typically, no overt cause for disease is recognised in affected animals, although associated clinical features have included hypoalbuminaemia, hypocuprosis, elevated abomasal pH and plasma pepsinogen, as well as diarrhoea (Waldrup and Mackintosh 1993). Affected animals typically respond poorly to anthelmintic and other treatments (Conway 1990; Mackintosh and Orr 1990; Waldrup and Mackintosh 1992; Dougherty 2008).

Type-II ostertagiosis has been described in red deer farmed in Britain (Connan 1991) but a literature search found no references to this disease in elk farmed in North America. Published descriptions of fading elk syndrome in New Zealand contain presumptive evidence of parasitism by hypobiotic forms of *Ostertagia* spp.-like larvae based on the presence of adult nematodes and the typical gross and histological changes in the abomasal wall of affected animals (Conway 1990; Mackintosh and Orr 1990; Waldrup and Mackintosh 1992, 1993). The main aim of this study is to describe the occurrence of fading elk syndrome in a large herd of translocated elk in North America which had direct histological evidence of intramural abomasal parasitism by *Ostertagia* spp. larvae. We also provide an example of the consequences of large-scale translocations of cervids without regard to seasonal, social, nutritional and environmental factors that may affect the outcome.

### Case history

In May 2005, a herd of 520 male and female elk of mixed ages was transferred in multiple shipments over several weeks between two farms in Maine. The farm of origin was a large elk production facility consisting of large (approximately 5-acre) treeless, flat, fenced pens arranged along a long, straight central alley. There was limited grazing opportunity, and feed consisted of hay forage and a custom-mixed cervid ration distributed into troughs, mostly arranged along the fence dividing the pen and alley. Details of the ration were not recorded in the case history. Water

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ADDL Animal Disease Diagnostic Laboratory  
AHDC-CU Animal Health Diagnostic Center-Cornell University  
CWD Chronic wasting disease  
DM Dry matter

was provided *ad libitum* from automatic stock waterers. Stocking rates were not established but the facility could be described as an intensive livestock operation. The preventative health maintenance programme records consisted of annual vaccination with an unnamed multivalent clostridial vaccine product and biannual anthelmintic treatment by application of a pour-on avermectin product (Eprinex; Merial Limited, Duluth GA, USA) at twice the dose rate recommended for cattle.

The destination farm held a pre-existing herd of 100 elk of mixed ages and sex and, being a hunt ranch-style property, was by contrast extensively managed. The newly acquired animals were mixed without regard to previous social group, sex, and age, and held in variable-sized but large, mostly wooded enclosures with rough terrain and no pasture. They were fed relatively poor (6% protein) hay forage and silage, but were later supplemented with various bulk and bagged formulated concentrate rations (16–21% protein) incorporating trace minerals and salts and, in one instance, a coccidiostat (decoquinat 45.4 g/t; Blue Seal Feeds Inc, Londonderry NH, USA). As health problems and loss of condition became apparent in the animals, changes were made to the diet in response to field necropsy findings which included sarcosporidiosis and mineral imbalances (discussed later). The changes also included supplementation with Brewer's grain, the material remaining after fermenting grain for beer-making. Being highly palatable it is often fed to ruminants to increase levels of dietary protein and water-soluble vitamins. Water was supplied from a natural pond or from a stream on the property.

Elk began dying within 2 weeks of arriving at the destination farm but veterinary assistance was not sought until August 2005, some 3 months after arrival. A veterinarian in private practice, a diagnostician from an animal health laboratory, and the state veterinarian were consulted and all visited the premises at one time or another. Case records showed agreement between the attending veterinarians that the 100 elk already resident on the affected farm when new animals arrived were in good condition and showed no clinical signs of illness. Apparently only translocated animals became sick, and by January 2007, approximately 18 months after they had been moved there, 436/520 had died.

## Clinical findings

Two field necropsies performed by attending veterinarians and one laboratory necropsy at the Animal Disease Diagnostic Laboratory (ADDL; Orono ME, USA) in August 2005 were inconclusive except for gross findings associated with ill-thrift such as poor body condition and depletion of intra-abdominal fat deposits. Differential diagnoses offered for the necropsy at the ADDL were CWD, Johne's disease, heat stress and dehydration. Subsequent histological examination of the brain, and faecal culture, were negative for spongiform changes suggestive of CWD and Johne's disease, respectively.

Three further necropsies were performed in September 2005 at the ADDL and except for poor general body condition (body condition scores were estimated at 1.5/9, 2.0/9 and 2.0/9; Audigé *et al.* 1998), no significant gross or histological lesions were seen in any organ. Serum Se levels determined at the Animal Health Diagnostic Center at the College of Veterinary Medicine, Cornell University (AHDC-CU) from blood samples collected ante mor-

tem indicated a profound Se deficiency (one elk at 1.05  $\mu\text{mol/L}$  and the others below the detection limit of 0.32  $\mu\text{mol/L}$ ; reference range 1.27–2.28  $\mu\text{mol/L}$ ). All reference ranges for trace mineral analysis in elk are taken from Puls (1994).

Water samples from the farm showed a mild elevation in Fe (37.6  $\mu\text{mol/L}$ ; a level of 5.4  $\mu\text{mol/L}$  was set as the upper desirable limit, as per Northeast Laboratory Services, Waterville ME, USA) but were otherwise unremarkable. Feed analysis (values given on a dry matter (DM) basis) performed by the Deering Hall Analytical Lab, University of Maine, of what was described as a legume/grass mixture from six different enclosures containing elk indicated low levels of crude protein (mean 6.3%, range 4.6–9.0%), total digestible nutrients (mean 58.3%, range 52.8–63.08%), Ca (mean 0.44%, range 0.26–0.69%) and Se (all samples <0.30 mg/kg), and high acid detergent fibre (mean 42.5%, range 39.5–50.4%).

A small number of *Sarcocystis* spp. cysts were identified in skeletal muscle sections from all three animals. The accompanying history indicated approximately 150 of the recently purchased animals were dead. Based on the feed analysis and post-mortem results, a diagnosis of Se deficiency and incidental *Sarcocystis* spp. infestation was made. Veterinary recommendations included parenteral Se and thiamine given once every 3 weeks and repeated three or four times, *ad-libitum* supplemental feeding of a Se-based mineral mix, and the addition of amprolium to the diet in the event that the presence of *Sarcocystis* spp. was clinically significant.

In October 2005, samples of cheek muscle, heart, lung and tongue from two more animals were sent to the ADDL, for histopathological examination, and liver was submitted to the AHDC-CU, for trace mineral analysis. There were deficiencies in Cu (5 and 6 mg/kg DM; reference range 20–120 mg/kg DM) and Mo (<2 mg/kg DM; range 2–4 mg/kg DM), and mildly elevated levels of Zn (95 and 136 mg/kg; reference range 23–80 mg/kg DM). Unlike the previous submissions, Se levels in liver were low (0.42 and 0.25 mg/kg) but within normal values (0.25–1.4 mg/kg DM). With these findings, Cu deficiency was added to the list of diagnoses made in the herd.

Whole blood, blood smears and serum from nine live elk were also submitted for a complete blood cell count, routine ruminant serum chemistry testing and serum Se analysis. All values, including serum Se levels, were within reference ranges. Test records indicated that faeces were submitted with the blood but faecal floatations failed to demonstrate significant numbers of parasite ova beyond "a few strongyle eggs". Consequently, intestinal parasitism by adult worms was not reported as a significant finding.

Animals continued to die throughout 2006 despite nutritional supplementation with a commercial cervid diet formulated with trace minerals (E-Z Pels; Blue Seal Feeds Inc), coccidiostats (Amprolium; CORID Crumbles; Merial Limited), and decoquinat (Calf DC Flakes; Blue Seal Feeds Inc), parenteral treatment with Se/vitamin E (Mu-Se; Burns-Biotech Inc, Oakland CA, USA), and administration of anthelmintic (Ivermectin; Merial Limited). Dose rates were not known. There were many opinions, professional and otherwise, about the causes of the losses numbering into the hundreds of animals of both sexes and all ages, and appropriate remedial measures. Various diet supplements listed above, designed to supplement protein, Se, Cu and other trace minerals, were provided to the elk in succession. In November 2006, whole blood, blood smears and serum from two yearling male elk were submitted to the AHDC-CU for a complete blood



cell count, routine ruminant serum chemistry, analysis of serum Pb, and an agar gel immunodiffusion assay for Johne's disease. All reported values, including Pb levels, were within reference ranges, and the agar gel immunodiffusion assays were negative. Beyond the initial post-mortem examination in August 2005, necropsy test records contained no reference to testing for CWD.

In January 2007, an opinion was sought from one of the authors (MR Woodbury), who suggested a renewal of diagnostic testing efforts. Accordingly, in January and February 2007, four translocated elk showing signs of chronic wasting consistent with those of other affected animals in the herd were subject to euthanasia, and their carcasses submitted for post-mortem examination to the Section of Pathology of the Tufts Cummings School of Veterinary Medicine. All were intact females, estimated at 3–4 years of age. An initial thorough gross evaluation of all organ systems *in situ* was followed by removal and sampling of viscera. Sections of tissue (tongue, oesophagus, abomasum, rumen, reticulum, omasum, duodenum, jejunum, ileum, colon, mesenteric and retropharyngeal lymph nodes, kidney, liver, lung, spleen, heart, pancreas, urinary bladder, thyroid and parathyroid glands, adrenal glands, bone marrow, skeletal muscle, brain, spinal cord) were then collected from each animal, fixed and retained in 10% neutral buffered formalin, embedded in paraffin, cut at 5  $\mu$ m, and stained with H&E for histopathological examination. Following formalin-fixation, portions of the obex, spinal cord and retropharyngeal lymph nodes were also submitted to the National Veterinary Services Laboratories (Ames IA, USA) for prion protein immunohistochemistry.

Parasitological analysis was also performed on samples of faecal material from all animals. Only an in-house faecal smear was examined from one animal, the first to be submitted for post-mortem examination, but more detailed analyses of faecal samples from the remaining three animals were performed in-house as well as at the AHDC-CU in some cases. Trace element analyses were performed at the AHDC-CU on portions of fresh, unfixed liver that were collected from each animal at the time of post-mortem examination. Samples of fluid from the abomasal lumen were collected from all animals at post-mortem examination (with care taken not to contaminate the sample with rumenal fluid), for rapid and crude analysis of pH using urine dipsticks (Multistix UTS; Bayer, Elkhart IN, USA) (Waldrup and Mackintosh 1993).

At gross post-mortem examination the elk were variably emaciated, and had prominent dorsal spinous processes of the vertebral column and depleted depots of adipose tissue (subcutis, epicardium, perirenal region and bone marrow). The most consistent gross findings were in the abomasum, which was variably thickened and hyperplastic, with the formation of nodules often producing a cobblestone appearance (Figure 1). No significant lesions were found in other tissues, and molar dentition was normal upon sagittal sectioning of the skull.

Histologically, the most significant finding in all animals was abomasal mucosal epithelial proliferation and hyperplasia of mucosal glands (Figure 2) which varied in degree, subjectively, from mild to moderate in three animals to marked in the other animal. Nematodes were also seen (Figures 2 and 3) within the thickened abomasum in two animals, and they were associated with eosinophilic cellular infiltrates in three animals. Microscopic features of the nematode parasites, including external cuticular longitudinal ridges (Figure 3), were consistent with a trichostrongyle species such as *Ostertagia* spp. Scattered sarcocysts were



Figure 1. Photograph of an abomasum from an elk with Type-II ostertagiosis. The mucosal surface is thickened and has a 'cobblestone' appearance (bar=1 cm).

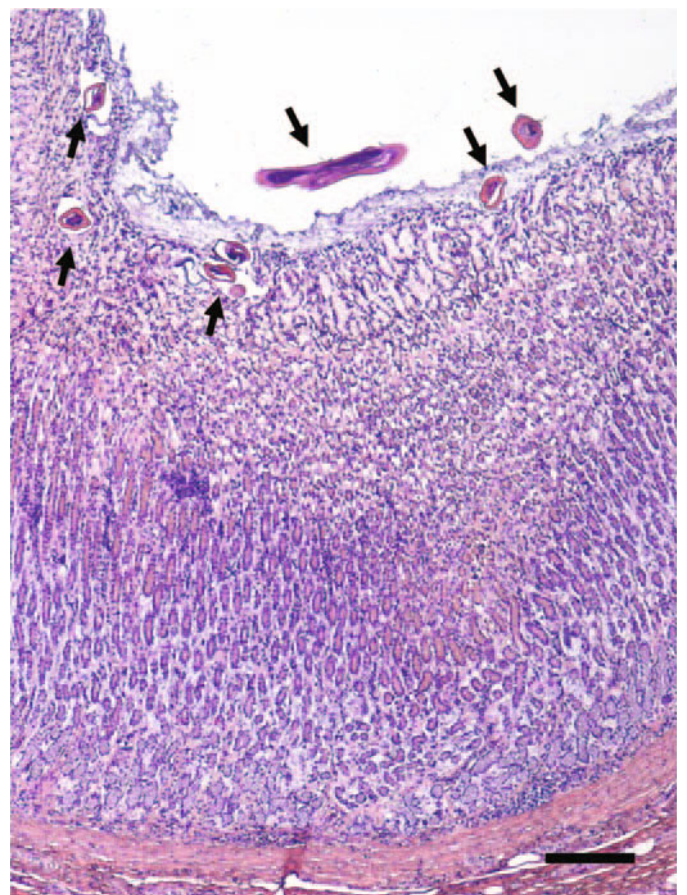


Figure 2. Light photomicrograph of a section of abomasum from an elk with Type-II ostertagiosis. Nematodes (arrows) are present on the luminal surface and are embedded within the superficial mucosa (H&E, bar=75  $\mu$ m).

also incidentally present in the heart in all four animals, as well as the tongue of two. There was no histological evidence of transmissible spongiform encephalopathy in the brain or brainstem, and additionally protease-resistant prion protein (PrP<sup>res</sup>) was not detected by immunohistochemistry in the obex, spinal cord or retropharyngeal lymph node. A diagnosis of prion-related CWD was thus precluded.

In-house parasitological analysis by centrifugal faecal floatation was performed and revealed moderate numbers (five to 10 per



**Figure 3.** Light photomicrograph of a section of abomasum from an elk with Type-II ostertagiosis. Nematodes are embedded within the superficial mucosa. Note the external cuticular ridges (arrow) of the parasite (H&E, bar=40  $\mu$ m).

slide) of trichostrongyle eggs, as well as very small numbers (one to five per slide) of operculated eggs, suggestive of trematode infection in one animal. Analyses of samples from the remaining three animals produced variable results. In one animal, approximately 1,200 trichostrongyle epg of faeces and approximately 900 trichostrongyle epg of abomasal contents were seen. In another animal, approximately 10,650 trichostrongyle epg of faeces and approximately 2,100 trichostrongyle epg of abomasal contents were seen. Paradoxically, in samples forwarded to the AHDC-CU, no parasites were detected in faeces from either of these animals. In-house parasitology was not performed on samples from one animal but faecal material collected from this animal was forwarded to the AHDC-CU for quantitative analysis, and revealed 30 *Eimeria* spp. oocysts per gram, nine *Nematodirus* spp. epg, 89 *Trichuris* spp. epg, and one Capillarid-type epg.

Hepatic trace element analyses (Table 1) revealed that all animals were severely Cu- and Se-deficient, and had marginal levels of Zn. Additionally, in one animal, Fe levels were in the low reference range but were elevated in another animal, although the significance of this was unknown. Abomasal fluid pH levels were elevated, at approximately pH 5 (reference range 1.5–4.3).

## Discussion

Ill-thrift and emaciation are relatively non-specific but not uncommon clinical signs and are associated with various diseases

**Table 1.** Summary of trace element levels (mg/kg dry matter; DM) following necropsy of four North American elk from a herd of 520 translocated deer of mixed age and sex.

Animal	Trace element levels in liver (mg/kg DM)			
	Cu	Zn	Se	Fe
143	11	58	0.07	662
157	9	76	0.17	405
213	8	530	0.83	1,832
251	9	77	0.44	1,004
Reference range	80–600	92–400	1.00–7.00	400–1,000

in ruminants. Among the causes of such signs in ruminants are transmissible spongiform encephalopathy, Johne's disease, intestinal parasitism, and chronic or severe dental disease (Maas 2002). No evidence of any of these conditions was found in any of the animals in the investigation presented here. The low number of initial post-mortem examinations conducted in the face of such large losses of stock was perplexing. The veterinary advice offered, albeit based on evidence from insufficient animals, implicated relatively common ruminant nutritional and trace mineral problems in that geographical area and placed too much significance on the presence of *Sarcocystis* spp. cysts which, unless found in very large numbers, would be considered an incidental finding in elk (Haigh and Hudson 1993). The new owner was confident that the health status of the purchased animals would be improved by following veterinary advice and by providing the supplements and medications suggested. It is regrettable that early veterinary advice was based on insufficient diagnostic effort and also failed to take into account the unsatisfactory nature of the translocation of the herd. An experienced livestock owner would have known that maintenance of social structure, gradual nutritional changes, segregation by age and gender, and consideration of pregnancy and lactation status of females should all have been borne in mind prior to the movement of this herd. For example, the social order of elk herds is matriarchal and hierarchical in nature, and mixing of established groups results in the re-assortment of dominance and interruption of normal behaviour such as feeding, to the detriment of the subordinate animals in the group, until a new herd structure can be established (Haigh and Hudson 1993).

The decision to transport animals in May, when pregnant females would have been in their last month of pregnancy and males would have been in velvet antler, constitutes a welfare issue that was never addressed (Anonymous 1996). The mortality rate reported here only included adult animals. Apparently, there were a large but unknown number of deaths of neonatal calves subsequent to translocation of the pregnant females. The stress of the move, changes to the diet, a new environment and the need to establish a new social hierarchy caused by mixing groups would be sufficient to adversely affect the dams' ability to provide nutrition to newborn calves. Transported males were handled similarly to the females, and had apparently similar rates of mortality.

Findings in the final four animals submitted for post-mortem examination were similar, and their clinical histories were similar to those of other members of the herd that had died. Since the whole herd was subjected to the same environmental conditions, treatments and feedstuffs, the post-mortem findings suggest that the lesions in those particular animals were representative of what occurred across the herd.

The most significant gross post-mortem and histological finding in the necropsied cases from January 2007 was the proliferative change of the abomasal mucosa. This finding, together with the associated history and clinical signs, is consistent with well-documented lesions and physiological after-effects of Type-II ostertagiosis in ruminants (Myers and Taylor 1989). As no evidence of significant underlying systemic disease was found to otherwise explain the chronic wasting in these animals, the possibility of parasitism inciting the clinical problems was therefore considered.

*Ostertagia* spp. are arguably the most important gastrointestinal nematodes of cattle and other ruminants. They localise and affect the abomasum, producing intensive economic losses especially in young beef and dairy cattle (Gibbs and Herd 1986). It is, howev-



er, a problem that appears undiminished with increasing age and immune competence of the host and, at least in cattle, *Ostertagia* sp. is one of the few parasites that occur in adults as well as young animals (Selman *et al.* 1976). This organism has a simple direct life cycle that consists of two stages, *viz* the free-living stage on pasture, and the parasitic stage (Myers and Taylor 1989).

In cattle and presumably elk, two types of disease are caused by *Ostertagia* spp.-like nematodes. Type-I disease typically occurs in calves and young animals that have high burdens of adult worms in winter and spring. Disease follows rapid infection with large numbers of third-stage larvae from heavily contaminated pastures in the autumn and winter after weaning (Myers and Taylor 1989). Type-II disease in cattle occurs in the autumn and winter, in particular in females calving for the first or second time, and coincides with the stress of calving and emergence of thousands of hypobiotic fourth-stage larvae from the abomasum. In cattle at this time, severe diarrhoea, loss of weight, and even death may result (Myers and Taylor 1989). Severe diarrhoea was apparently not a major feature of disease in the affected elk herd, although comments recorded from the owner included references to "loose faeces".

Hypobiosis, or arrested development, occurs in many genera of nematode parasites of grazing ruminants, and its main feature is the temporary delay of development of the parasite in the host (Fernandez *et al.* 1999). In trichostrongyles, this usually occurs at the fourth larval stage, and the phenomenon has predominantly been studied in *O. ostertagi*. The exact cause of the arrested development is poorly understood. It is generally considered, at least in cattle, that numerous factors are responsible (Williams 1983; Armour and Duncan 1987), including the host immune response, density-dependent regulatory mechanisms of worm populations, and evolutionary adaptation (Armour 1978). In the northern temperate climates of the Northern Hemisphere, where this case occurred, it is also agreed that low temperature in the autumn and early winter is a determining factor for inhibition of *Ostertagia* spp. in cattle (Myers and Taylor 1989).

The elevated abomasal pH and pronounced trace mineral imbalances additionally detected in these animals are consistent with the secondary effects that such *Ostertagia* spp.-induced mucosal damage initiates (Simpson 2000). Ostertagiosis has been reported to cause elevated abomasal pH in farmed red deer and cattle, negatively affecting the animal's digestive efficiency, not only by reducing abomasal pepsin activity but also by restricting bioavailability and uptake of dietary Cu (Connan 1991; Waldrup and Mackintosh 1993; Simpson 2000; Dougherty 2008). The elevated pH in these cases may also interfere with uptake of oral anthelmintics, further adding to the problem (Waldrup and Mackintosh 1992; Dougherty 2008).

Conflicting results were obtained from faecal assays on the same four animals by two different laboratories, and the extent of intestinal parasitism by adult forms was not noted at necropsy despite finding parasite ova in the faeces of at least one animal. However, the results support the usefulness of pooled or herd sampling rather than individual animal sampling to detect intestinal parasites. Acid digestion assay of the abomasum may have yielded quantitative information about the extent of parasitism but was not requested. Parasitism by adult forms was not an obvious finding in any of the necropsies performed and anthelmintic therapy had not appreciably improved the condition of these elk or of others in the herd. This is consistent with findings in reports of Type-II

ostertagiosis in cattle as only adult nematodes are expelled by anthelmintics, whereas a proportion of inhibited larvae are unaffected (Armour 1970; Smith and Perreault 1972). Studies in red deer have shown that once encysted, even high doses of anthelmintics fail to remove larvae from the abomasal wall (Waldrup and Mackintosh 1992; Connan 1997). However, subsequent studies by Waldrup *et al.* (1998) reported that the pour-on formulation of moxidectin and ivermectin at twice the dose rate for cattle were effective against early fourth-stage larvae of *Ostertagia* spp.-type nematodes in both red deer and elk hybrid deer. In cases where the initial infestation with *Ostertagia* spp. parasites is not cleared with routine anthelmintic treatment, larval forms encysted in the abomasal wall can lead to permanent mucosal damage (Simpson 2000). At best, there is a long post-therapeutic period of recovery while the abomasum repairs and the pathogenic effects are reversed. It is likely that the anthelmintics used on the affected farm would have been ineffective in this case even had a correct diagnosis been made earlier in the clinical course.

Overall, the abomasal lesions, elevated abomasal pH and trace mineral abnormalities are consistent with those described in a condition of chronic ill-thrift and often extreme cachexia that has been documented in elk, red deer, and their hybrids, associated with *Ostertagia* spp.-like nematodes (Waldrup and Mackintosh 1992). Because of its progressive nature and poor response to therapy this condition has been termed 'fading elk syndrome' (Conway 1990; Mackintosh and Orr 1990). As is the case with ostertagiosis in other ruminants (Simpson 2000), this syndrome characteristically leads to increased abomasal pH with associated protein-losing enteropathy and Cu deficiency. The ineffective efforts of the new owner to remedy trace mineral deficiency and to provide high levels of nutrition are consistent with published reports of this disease (Dougherty 2008). Care should be taken, however, to differentiate this syndrome from prion-associated CWD, and it is suggested that this condition be renamed 'abomasal parasite syndrome of elk and red deer'.

It is unlikely that the 436 deaths in this translocated herd were all due to abomasal parasite syndrome, and the ultimate demise of the herd was certainly multifactorial in origin. The inherently stressful movement of the elk to a new environment, subsequent mixing of age and established social groups, and a sudden change in the type and quality of feed would be sufficient to permit expression of any latent diseases in the herd. However, the few animals subject to post-mortem examinations early in the course of morbidity yielded only non-specific information and did not show overt signs of infectious disease such as pneumonia or enteritis. Trace mineral imbalance suggestive of dietary deficiency or abomasal parasitism appeared to be the most consistent finding, and affected the animals necropsied in 2005 as well as those examined in 2007. It is possible that this herd was suffering from Type-II ostertagiosis at the time of translocation and that the nutritional, psychological and social stress placed on them after the move exacerbated the problem. It is also possible that the stressed and immunologically depressed elk acquired the parasite after being translocated. The timing of the infection is perhaps not as important as the eventual consequences of the establishment of a Type-II cycle in this elk herd. Certainly, the ultimate results of the infection are consistent with other descriptions of the clinical course of this debilitating condition, except on a much larger scale (Mackintosh and Orr 1990; Waldrup and Mackintosh 1992, 1993; Dougherty 2008).

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