

Fading Elk Syndrome Research

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Introduction

The term "fading elk syndrome" has been used to describe a condition of chronic cachexia and illthrift in elk, wapiti and elk/red deer hybrids. This terminology has had broad acceptance among producers and veterinarians. This has led to a somewhat confusing situation in which the terminology has been maintained in spite of specific etiologies and diagnoses. The purpose of this presentation is to share the results of some current research into diseases of elk ongoing at Invermay and to address the terminology of elk diseases.

Differences Between Elk and Red Deer

Although elk and red deer are conspecific (*Cervus elaphus*), elk are not simply large red deer. On a macroscopic scale, there are differences in body size, length of gestation, behavior and antler structure. On a microscopic scale, differences in blood proteins allow the differentiation of red deer and elk by bloodtyping. One of these blood proteins is a copper containing enzyme, superoxide dismutase (SOD) (Tate, et al, 1988). While this difference is not quantifiable using SOD from bloodtyping data, it could suggest a difference in copper metabolism for elk. In New Zealand, elk are known to be more susceptible to clinical copper deficiency (Mackintosh, 1992; Mackintosh et al, 1986). Copper deficiency is apparently rare in elk in North America. One report which does exist (Gogan, et al, 1988) lists emaciation as one of the symptoms noted in the affected animals. The question remains as to whether copper deficiency has a primary or secondary role in chronic cachexia of elk.

Elk are more resistant to MCF than red deer but are more susceptible to ryegrass staggers (lolitrem toxicosis) (Mackintosh, 1992). The difference in resistance to ryegrass staggers may be due to the lack of selective evolutionary pressure on elk in North America as ryegrass is not a native forage of that continent whereas red deer, being of European origin, have been exposed to such selective pressure.

In New Zealand, gastrointestinal parasitism in elk has been associated with weight loss and illthrift (Mackintosh and Orr, 1990; Conway, 1990). The majority of abomasal nematodes identified from parasitized elk in New Zealand (Mason, 1984) are worms which have usually been associated with red deer (Drozd, 1965; Connan, 1991) in Europe, but not elk in North America (Kistner et al, 1982). Because elk have not evolved with these parasites, it is possible that elk, as an abnormal host, are more susceptible to their pathogenic effects. Some of these effects include alteration of abomasal pH and decreased digestive efficiency.

Current Research

Etiology

Examination of several elk and elk hybrids which were "fading" during the winter of 1991 revealed that these animals had abomasal pH levels of above 5.2 (n = 10, average pH = 6.3). This finding has been associated with the presence of *Ostertagia*-type larval infections in the abomasums of these animals. Ostertagiasis is known to cause a rise in abomasal pH (Connan, 1991) and a loss of blood proteins (Mulligan et al, 1963) and copper (Bang, et al, 1990). In addition to the copper loss which occurs with ostertagiasis,

the increase in abomasal pH has a negative effect on the dissolution and uptake of elemental copper from copper oxide wire particles (copper "bullets") (Bang et al, 1990). The rise in abomasal pH is due to the invasion of the fundic glands by the nematode larvae. In the autumn season, it is known that *Ostertagia*-type larvae become "inhibited" in the fundic glands of ruminants (Armour et al, 1969)), presumably as an overwintering strategy by the parasite. In cattle, this stage is quiescent and is referred to as pre-Type II infection (Anderson et al, 1969). These inhibited larvae are more resistant to treatment with anthelmintics (Fisher and Mrozik, 1989). Coincidental to this seasonal phenomenon with the parasites is a seasonal phenomenon in stags, the "roar" or the breeding season. Therefore the authors theorize that, in stags, the catabolic effects of the roar are exacerbated and extended by parasitism. The diagnosis of the parasitic involvement is difficult as the inhibited larvae do not produce ova, and plasma pepsinogen levels, which are commonly elevated with Type II ostertagiasis, are not elevated with pre-type II infection (Chalmers, 1983).

Treatment

It has been known that oral anthelmintics at recommended dosages for cattle have had poor results in the treatment of deer (Connan, 1991) and elk suffering from chronic cachexia ("fading") (Conway, 1990). Absorption of some oral anthelmintics can be dependent upon abomasal acidity (Marriner and Bogan, 1981; Prichard et al, 1985), so the reduction of that acidity would likely reduce absorption and therefore activity. Parenteral administration of an anthelmintic (injectible or pour-on ivermectin) could alleviate this problem. Research at Invermay with red deer indicates that while 1000ug/kg (double the cattle dose) of pour-on ivermectin will control adult abomasal nematodes, that same dosage produced only a 40% reduction of encysted *Ostertagia*-type larvae (Mackintosh, pers. comm.). Therefore even higher doses might be needed for real therapeutic effectiveness.

The use of intra-ruminal sustained-release anthelmintic devices which prevent abomasal infection may be helpful. In ongoing research using Paratect (Registered Trademark, Pfizer Laboratories), an intra-ruminal device containing morantel, early data have shown a weight retention advantage in wapiti hybrid stags from March through May (see Table 1).

Table 1. The liveweight sparing effect of treatment with Paratect (R) in rising 3 yr old hybrid stags from March through May.

Group		Change in Liveweight (mean)
Treated	(n=14)	+5.6 kg
Not treated	(n=14)+	-6.5 kg*

* Statistically significant difference, $p = 0.01$

+ Of the 14 original animals, 2 are deceased and 2 have required treatment for severe cachexia

Clanobutin (Bykahepar, Registered Trademark, Boehringer Ingelheim) was used to treat a limited number (n = 4, with 1 untreated control animal) of cachectic animals having hypoalbuminemia. This drug stimulates the secretory activity of the exocrine digestive glands and has shown clinical effectiveness in the treatment of digestive problems of other ruminants (cattle and sheep). Daily treatment for 3 consecutive days at the rate of 10mg/kg liveweight had no net positive effect with regard to weight gain or serum albumin in the treated animals.

Discussion

It is the recommendation of the authors that the term "fading elk syndrome" should be dropped or at least modified to be more specific relative to etiology. Recent study has led to the hypothesis that definitive mechanisms could be involved. Evidence is increasing that a combination of subclinical parasitism and the "roar" may lead to chronic cachexia in stags. In this situation, stags lose weight due to the breeding season, but abomasal parasitism does not allow the stag to stop the weight loss, which continues beyond that which would be expected due to the roar. It is postulated that the change in abomasal pH due to this parasitism has a negative influence on copper uptake, oral anthelmintics and reduces digestive efficiency. The negative impact on digestive efficiency may be demonstrated by the reported lack of response of cachectic elk to anabolic steroids. Such drugs reduce protein catabolism and increase nitrogen retention in skeletal muscle. Dietary reinforcement with additional protein is recommended for best effect (McDonald, 1980). The proposed mechanism of digestive inefficiency would explain this lack of response as it does not even allow the uptake of sufficient maintenance levels of nitrogen, much less reinforcement levels. As it is known that treatment of cachectic animals is exceedingly frustrating, it would seem that judicious strategies to prevent subclinical parasitism in roaring stags should be implemented.

A similar digestive mechanism involving young growing stock could also be postulated. In stags, the problem lies in excessive weight loss, whereas in growing stock, the primary problem is a failure to perform to their genetic potential (poor weight gain). Problems in growing stock have been alleviated in some cases by vigorous use of anthelmintics (Conway, 1990). Again the idea of prevention by strategic use of parasiticides and adequate nutrition would seem far better than treatment.

Unfortunately the situation with hinds is not as clear. At Invermay, recent work has shown that hinds in lactation are already under stress (Waldrup, unpublished data). As lactation occurs prior to the seasonal "inhibition" of parasitic nematode larvae, a different mechanism is probably at work, as opposed to the suggested mechanism in stags. More research is needed to further elucidate the problem.

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