29 Observations on the Relative Susceptibility to Disease of Different Species of Deer Farmed in New Zealand

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Abstract

The four major types of deer farmed in New Zealand are European red deer (*Cervus elaphus*), North American wapiti (*C. elaphus spp.*), wapiti X red hybrids, and fallow deer (*Dama dama*). There are also small numbers of sika (*C. nippon*), rusa (*C. timorensis*), sambar (*C. unicolor*), and Père David's deer (*Elaphurus davidianus*). Field observations over the past 20 years have shown a number of species differences in susceptibility to various diseases of deer farmed in this country. Malignant catarrhal fever has never been reported in fallow deer, while wapiti and red deer seem relatively resistant, and sika, rusa and Père David's deer are very susceptible. Fallow deer appear relatively resistant to yersiniosis which commonly affects young red deer. Fallow deer are more susceptible than red deer to sporidesmin, a fungal toxin produced by (*Pithomyces chartarum*) which causes liver damage and "facial eczema." Wapiti and wapiti X red deer appear more susceptible than red deer to a number of conditions including copper deficiency and enzootic ataxia, the fungal endophyte neurotoxicity which causes "ryegrass staggers," internal parasitism, "dietary scours," and "the fading elk syndrome."

Key words: Diseases, farmed deer, relative susceptibility

Introduction

There are approximately 1 000 000 deer on nearly 000 farms in New Zealand, and according to the 988 Department of Statistics survey, the major- are red deer (81%), with fallow (9%), wapiti and apiti X red hybrids (7%) and other species omprising sika, rusa, sambar and Père David's %) making up the rest. Most of the farmed deer figinate from animals captured from wild opulations established throughout New aland in the last 130 years. All the red, fallow, d sika deer were of British origin, although ne came via Australia. The sambar came from Lanka, the rusa came from New Caledonia, hereas the wapiti and white-tailed deer *ocoileus virginianus*) came from North Amer- (Challies 1985). The long sea voyages of the 1800s and early 1900s probably served as a natural quarantine period, and recent importations of deer from the United Kingdom, Europe, and North America have required extensive quarantine residence and testing. Consequently the wild deer in New Zealand are remarkably free of serious diseases, although most populations carry low burdens of helminth parasites. No blood-borne parasitic diseases are recognized, probably due to the lack of suitable insect vectors. The most significant disease to emerge in wild deer populations in certain areas in the past 20 years is tuberculosis (Tb), caused by *Mycobacterium bovis*; it has been isolated from wild red, fallow, and sika deer (de Lisle and Havill 1985). Apart from Tb, none of the important diseases of farmed deer dealt with in this paper have been reported to cause clinical disease in wild deer.

Since deer farming in New Zealand started

TABLE 29.1 Summary of relative disease susceptibility of various deer species farmed in New Zealand

Disease	Very Susceptible	Susceptible	Resistant
Malignant catarrhal fever	WTD, Ru, PD, S	R, W	F
Yersiniosis	—	R, W	F
Facial eczema (sporidesmin)	F	R, W	—
Ryegrass staggers	W	R	F
Enzootic ataxia	W	R	—
Lungworm	R(w), W(w), F(w)	W(a)	R(a), F(a)
Gastrointestinal nematodes	W(w)	W(a), F(w)	R(a), F(a)
Preputial ulceration and prolapse	W	—	R, F
"Dietary scour"	W	—	R, F
"Fading elk syndrome"	W	—	R, F

Species: WTD, white-tailed deer; R, red; Ru, rusa; S, sika; P, Père David's; W, wapiti; F, fallow. Age: w, weaners; a, adults

over 20 years ago, people have tried to farm all of the above species, but some, especially white-tailed deer, have not adapted well to being farmed, whereas red deer, wapiti, and fallow deer have thrived. One of the factors that has influenced this adaptation is their susceptibility to a combination of stress and the diseases that they encounter in the farming environment. This chapter reviews the evidence for species differences in susceptibility to a number of diseases of deer in New Zealand based largely on field observations (Table 29.1).

Malignant Catarrhal Fever

In New Zealand, malignant catarrhal fever (MCF) of deer and cattle are both caused by a sheep-associated gamma-herpesvirus which appears to be closely related to the wildebeest-associated virus which causes a similar disease in cattle in Africa (Reid et al. 1989) and in deer in zoos in North America (Saio-Kun Wan et al. 1988). The prevalence of infection in deer is not known, but once disease develops it is invariably fatal, although the course varies from hyperacute (< 24 h) to acute (2 to 3 days) to chronic (1 to 4 weeks) (Fennessy 1988). Deer are literally a "dead-end" host, as there appears to be no natural deer-to-deer transmission. It is believed that all infections result from direct or indirect contact with sheep-excreted virus. The majority of cases (70%) in farmed deer occurs between June and September with a peak in July. This is 3 to 4 months earlier than in cattle, but it coincides with the peak stress on deer, especially stag which enter the winter with poor fat reserves (< 5% body weight) following the rut in late autumn.

Throughout New Zealand, approximately 1% of red deer die annually from MCF with the incidence slightly higher in the southern South Island and slightly lower in the northern North Island (Fennessy 1988). This contrasts strongly with the incidence of MCF in other species of deer. White-tailed deer and Père David's deer kept on farms appear highly susceptible. Early attempts to farm white-tailed deer in the low South Island foundered when all 50 live-captured animals succumbed to MCF within a year (R. Brooks, personal communication). Only 11 of the original 70 or so Père David's deer originally imported remain alive 5 years later, the majority having died in the first 2 years from MCF (Orr and Mackintosh 1988). A similar experience occurred with Père David's deer farmed in Scotland (Reid et al. 1987). Hybrids between Père David's and red deer appear to be more resistant with only one of seven F1 hybrids and none of about 20 3/4 red X 1/4 PD hybrids dying of MCF so far.

Rusa and sika deer are also more susceptible to MCF than red deer. In Northland, where MCF occurs only rarely in red deer, a Moluccan rusa deer herd of 20 loses two or three animals annually (Thorley 1990). Similarly, sika deer farms have experienced high mortality due to

MCF, especially in the early days of farming, with losses of up to 29% (Anonymous 1980; Hunter 1981). Under Australian conditions sika, chital *Axis axis*), rusa and white-tailed deer appear to be the most susceptible.

At the other end of the scale it appears that fallow deer are highly resistant to MCF, and despite there being 80 000 to 90 000 fallow deer farmed, there have not been any confirmed cases reported in New Zealand. Wapiti contract MCF but have a lower incidence rate than red deer (C. Mackintosh, unpublished data) and appear to be less susceptible to experimental infection than red deer (R. Oliver, personal communication).

Yersiniosis

Enteric yersiniosis is characterised by a watery green, often blood-stained, diarrhea, and it affects young deer in their first winter. It is caused by the bacterium *Yersinia pseudotuberculosis* and is predisposed to by stressors, especially transport, underfeeding, and bad weather (Mackintosh and Henderson 1984). It appears that almost all young deer are exposed to infection, but with good management they develop only subclinical infection. However, under certain conditions, outbreaks occur and can affect 5% to 40% of a group. Overall around 1% of young red, wapiti and wapiti X red hybrid deer die annually (Mackintosh 1988). Yersiniosis is very uncommon in fallow deer and is rarely reported in other species, probably due to the small numbers farmed.

Sporidesmin Toxicity ("Facial Eczema")

Sporidesmin toxicity is a disease of ruminants which occurs mainly in the warmer northern half of the North Island. It is caused by a toxin, sporidesmin, produced by a fungus *Pithomyces chartarum* which grows on dead plant litter in the pasture especially in warm, humid periods in late summer and autumn (Mortimer 1984). The ingestion of sporidesmin results in severe liver damage often leading to the build-up of the chlorophyll breakdown product, phylloerythrin, in the blood. This is a photodynamic compound and may cause photosensitivity—thus the name "facial eczema," because the lesions are most noticeable on thinly haired areas, especially the face. However, many animals suffer liver damage without any sign of photosensitivity. Elevation of the serum level of the liver enzyme gamma-glutyltransferase (GGT) is a good indicator of the level of damage sustained by the liver. Dosing trials with sporidesmin have shown that red deer are more resistant, but fallow are more susceptible than sheep (Mortimer and Smith 1981; Mortimer 1984). A few isolated cases have occurred naturally in red deer, but serious disease is not uncommon in fallow deer, and the mortality in the natural disease can be high.

"Ryegrass Staggers"

The majority of deer in New Zealand are grazed on perennial ryegrass (*Lolium perens*) and clover pastures. Many of the ryegrass cultivars are infected with an endophyte (*Acremonium* sp.) which produces a number of toxins (Lolitrems) especially in the seed head and the base of the plant, and these reach their highest concentrations during hot, dry summers (Mortimer et al. 1984). Ingestion of these toxins leads to "ryegrass staggers" which is characterized by head tremor, muscle tremors, and unsteady gait. The signs become worse if the animal is made to move quickly, and severely affected animals may fall over on their sides and paddle. All domestic livestock, including deer, can be affected and it is much more common in wapiti than in red deer (Mackintosh et al. 1982; Brooks and Cahill 1985; Bringans 1986; Brooks 1986). At Invermay Agricultural Centre, on a number of occasions wapiti have developed ryegrass staggers when grazing alongside red deer that have been apparently unaffected, suggesting that wapiti are more susceptible to the toxins or that their grazing habits may result in the ingestion of larger amounts of toxins. There are no published reports of ryegrass staggers in fallow, rusa, or sika deer.

Copper Deficiency and Enzootic Ataxia

Wapiti and wapiti X red hybrids appear to be more prone to copper deficiency and enzootic ataxia than red deer. Signs of copper deficiency in deer include loss of condition and ill-thrift, dry

dull coats, enlarged limb joints, and brittle bones. Enzootic ataxia, due to demyelination of the spinal cord, occurs in a proportion of copper-deficient deer. This was first reported in New Zealand in a red deer (Wilson et al. 1979), but subsequently wapiti have been shown to be more likely to be affected (Mackintosh et al. 1986a). In a group of 55 2-year-old wapiti, red, and wapiti X red hybrids grazing together as a group at Invermay, five of the wapiti animals developed enzootic ataxia and none of the others were clinically affected. Samples showed red deer had the highest and wapiti the lowest liver copper levels. Copper deficiency problems have not been reported in other deer species farmed in New Zealand.

Liver copper concentrations of less than 100 μmol/kg (wet weight) indicate copper deficiency in red deer, and deer with levels below 60 μmol/kg are likely to develop enzootic ataxia (Mackintosh et al. 1986b).

Nematode Parasites

There is a dearth of published information on the relative susceptibilities of the various breeds of deer to parasites. However, the lungworm (*Dictyocaulus viviparus*) is generally regarded as the most important nematode parasite of young red deer in New Zealand and Britain (Charleston 1980; Fletcher 1982; Watson and Charleston 1985). Although young fallow deer can sometimes develop heavy lungworm burdens under warm wet autumn conditions, they appear to be more resistant than young red deer (G. Asher, personal communication). In Denmark, problems are experienced with lungworm in red deer calves in the autumn and gastrointestinal parasites in fallow deer calves in the late winter (Jorgensen and Vigh-Larsen 1988).

In our experience at Invermay, Canadian wapiti also appear more susceptible to lungworm and take longer to develop age or exposure-related resistance. Watson (1986) reported that wapiti X red hybrids captured from Fiordland, New Zealand, were more susceptible to lungworm than red deer. These hybrids continued to shed fecal larvae throughout the year, and one 5-year-old animal had a heavy lungworm burden (> 1200) at necropsy. In wild populations of Rocky Mountain elk (*C. elaphus nelsoni*), the lungworm is a serious pathogen whereas gastrointestinal parasites only rarely reach pathogenic levels (Worley 1979).

Gastrointestinal parasites are regarded as of little importance in farmed red deer except for recently captured, aged, or debilitated animals. However, there have been a number of reports of abomasal parasites causing clinical disease in adult Canadian wapiti and wapiti X red hybrids which is often associated with elevated pepsinogen levels (Mason 1984; Bringans 1986; Pearce 1988; C.G. Mackintosh, unpublished data).

Preputial Ulceration and Prolapse

Three cases of severe preputial prolapse and swelling, which necessitated surgical removal, occurred in a group of ten Canadian wapiti bulls at Invermay over a 2-year period. All three cases occurred just prior to or early in the rut (Mackintosh 1990). At least two other cases have occurred in wapiti on other farms (N.S. Beatson and L. Hodgkinson, personal communication). There have been no reported cases of this condition in other species of deer in New Zealand. The prolapses were preceded by chronic ulceration and eversion of the preputial lining. In cattle and sheep, preputial ulceration is associated with high protein diets and is caused by ammonia which is produced by urease-forming micro-organisms from urine. The etiology of this condition in wapiti is probably similar and may also relate to physiological changes occurring in males at that time of year. Why it should occur so frequently in wapiti and not in red deer grazing the same pastures is not known.

Dietary Scour

Experience in New Zealand and Canada has shown that Canadian wapiti are prone to a "dietary scour" when grazed on lush pastures in spring and autumn. There is some suggestion that wapiti require more roughage than red deer, and the provision of lucerne (or alfalfa) hay at these times of year can alleviate this problem (Bringans 1986, 1987).

Preliminary investigations at Invermay also suggest that some food-related factor, possibly a

ngal mycotoxin, may also be involved in this etary scour" because elevated serum enzyme els indicative of liver damage often accomny this condition. If animals are removed from ture and fed on hay as soon as signs of rrhea appear, they usually recover quickly. ed deer grazed side-by-side with wapiti under ese conditions have been unaffected (C. Mackosh, unpublished data).

Fading Elk Syndrome"

his syndrome is peculiar to Canadian wapiti lk) and wapiti hybrids, and unless treated rly it is usually fatal. It is characterized by rogressive weight loss, low serum albumin vels, and may be with or without scour (Orr et al. 1990). It has been seen in New Zealand Bringans 1987; Fennessy et al. 1988) and anada (M. Bringans, personal communication), nd it is also called "chronic ill-thrift in elk" and, roneously, "wasting elk disease." It is definitely ot the same etiology as the chronic wasting isease described in North American elk which is type of spongiform encephalopathy and is used by a scrapie-like agent (Williams and oung 1982). The etiology is complex and may volve "dietary scour," copper deficiency and arasitism. It is also not associated with Johnes isease.

Discussion

isease is the end result of an interaction between infectious agent (or agents), the environment, nd the host animal (Mackintosh and Beatson 985). In the farming environment, stress plays especially important part in the equation riffin 1989). Deer are still in the process of mestication, and stressors such as handling, reased stocking density, increased social interion, reduced vegetative cover, contact with her domestic livestock, and exposure to varienvironmental influences contribute to ining their disease susceptibility. Some breeds with these stressors better than others. e-tailed and Père David's deer appear more ly strung" than red deer, which may conte to their increased susceptibility to MCF; ver, fallow deer are also very flightly, but are highly resistant to this disease. Therefore, other factors, such as their exposure to disease agents during their evolution and selection for resistance, have probably been important. For example, Père David's, white-tailed, rusa, and sika deer, evolved in parts of China, North America, southeast Asia, and Japan, respectively, where they would not have had exposure to domestic sheep or antelope and, therefore, have had no selection pressure for resistance to MCF. It is likely that fallow and red deer have had more exposure to sheep during their evolution in the Middle East and Europe and consequently have developed some resistance to MCF.

Pasture-based farms provide a relatively restricted range of environmental conditions. Animals that naturally depend heavily on browse are probably more likely to develop certain deficiencies or have increased susceptibility to diseases associated with a pasture-based diet. For example, the susceptibility of wapiti to ryegrass staggers, copper deficiency, and the "fading-elk syndrome" probably relates to their requirements for higher levels of roughage or browse in their diet (Hofman 1985). A naturally higher level of grazing in the wild may have helped red deer to become more resistant to fungal endophytes and parasites. Hybridization between red deer and Père David's, wapiti, and sika deer may produce animals that adapt better to a farming environment than purebred deer of these latter species.

In the past, veterinarians and farmers in New Zealand have tended to assume that all farmed deer, irrespective of species, have similar susceptibilities to various diseases. The diagnostic laboratories have also tended to collect data on deer as a whole rather than by species. However, the realization that farmed deer of different species and subspecies respond differently to various diseases will help improve the diagnosis, treatment, and prevention of these conditions in deer. This knowledge may also assist new farmers to choose the best type of deer to suit their particular farming environment.

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